

Lior Laver · Baris Kocaoglu
Brian Cole · Amelia J. H. Arundale
Jeffrey Bytowski · Annunziato Amendola
Editors



Basketball Sports Medicine and Science



European Sports Medicine Associates
A section of ESSKA



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Foreword by Coach Mike Krzyzewski

Basketball is one of the most popular sports in the world. Certainly, it has evolved significantly since Dr. James Naismith placed a peach basket on an elevated track in December 1891.

For most of my life, I have been blessed to play or coach this game, including the last 45 years at the highest levels as a head coach serving Army, Duke, and USA Basketball. Of course, our sport has always revolved around the “TEAM” concept at its core. The best basketball teams are formed by many individuals from different backgrounds, successfully working together toward a shared set of values and a common goal.

Those teams are comprised not only of players and coaches but also strong support staffs, including medical personnel, many of whom you will hear from—and hopefully learn from—in this book.

Certainly, high-end performance is contingent on physical health and well-being. We fully understand that our physicians, physical therapists, athletic trainers, performance specialists, and strength and conditioning coaches are integral to the ultimate success of our teams.

At Duke, we are fortunate to have two of the contributors of this book—Dr. Ned Amendola and Dr. Jeff Bytowski—on our medical team. We believe in them. We trust them, and we have incorporated many of their ideas into both our medical preventative and rehabilitation strategies. We have experienced significant injury reductions in recent years. On a daily basis, we utilize data of our players’ workloads in a sophisticated approach to reduce the physical stress on our team. And, in some other cases, we simply adhere to some common sense advice from our medical team to facilitate preventative care.

While we know and trust Ned and Jeff implicitly, the other editors of this book—Lior Laver, Baris Kocaoglu, Brian Cole, and Amelia J. H. Arundale—are equally as accomplished. There is so much to learn from all of them as they will provide the most comprehensive source for basketball’s medical caregivers.

As we know, basketball has evolved significantly, including the medical care provided to athletes around the globe. That basketball medical community, which is so critical to the effectiveness and success of our teams, continues to advance as well. I look forward to using some of the concepts in this book to further advance our program. You can too.

A handwritten signature in black ink that reads "Mike Krzyzewski". The signature is written in a cursive, flowing style with a large initial "M".

Mike Krzyzewski

Duke Basketball Head Coach since 1980

Winner of 5 NCAA championships, 12 Atlantic Coast Conference (ACC) regular season championships, and 15 ACC Tournament championships

USA Basketball Senior National Team Head Coach 2005–2016

Five-time Olympic Gold Medal winner – 1984, 1992 (assistant coach); 2008, 2012, 2016 (head coach)

Two-time FIBA World Cup Gold Medal winner – 2010, 2014

Member of the Naismith Memorial Basketball Hall of Fame

Foreword from the ESSKA Leadership

After Handball and Football, ESSKA's Third Sport-Specific Book—On Basketball Medicine and Science!

ESSKA is proud to expand its book collection with this dedicated book about basketball. It follows the spirit of the European Sport Medicine Associates (ESMA), the fourth section of ESSKA which is fully dedicated to the entire spectrum of sports medicine. After a book on handball medicine and science as well as return to sports aspects in football, this book is the third demonstration of the sport-specific “global vision” ESSKA wishes to promote. ESSKA believes that clinicians and scientists should always have this holistic view in mind when they take care of their athletes. Multi- and interdisciplinary thinking is of utmost importance for the understanding of sports injuries, their prevention, best treatment, and return to sports and performance.

Basketball is undoubtedly one of the most physically demanding team sports and requires a wide variety of different skills. Playing basketball involves a unique assembly of injury risks and related health problems. Therefore, the medical and health care of every basketball team and each player requires an unusual combination of knowledge and skill by every health professional involved. The current book not only provides a deep scientific and clinical foundation of knowledge but addresses every aspect of the health and medical care required in the sport.

What is it that makes basketball so special? It is a fast speed and intense game, with specific player morphotypes and hence some particular biomechanical aspects including very high lever arms in the upper and lower extremities, repetitive jumping with high impact loading on the ankle and knee joints, repetitive loading of the spine through the pelvis, and extreme pivoting elements in this sport which is played on very adherent surfaces. These are some specificities of basketball which lead to characteristic joint loading and frequent subsequent injuries. The game of basketball exposes the players to a very wide panel of injuries from the head and neck to the ankle and foot, as well as the psychological implications of playing a high-profile competitive sport.

The current book provides a compilation of topics representing an excellent tool for coaches, physiotherapists, sports physicians, and scientists as well as orthopedic surgeons—essentially providing a unique perspective for all

basketball personnel—from basic science topics, team building and preparations, injury management and rehabilitation, injury prevention, the special needs of unique and specific groups as well as psychological aspects. It will help the entire “professional perimeter” of the team to strategize, prepare, and address the various aspects involved in the medical coverage of a basketball team’s competition program from the preseason screening through the entire season.

The book provides an overview of a basketball player’s predisposing constitutional abilities to become a “competitive player,” the differences between the various levels of play in basketball and the practical translation of these aspects in terms of physical abilities. The book also provides a strong foundation of knowledge in biomechanics, physiology, endocrinic changes and nutrition as well as other basic essential factors in order to design adequate training programs including important relevant aspects of injury prevention.

Management of sports trauma and life-risking situations begins with good preparation, and these aspects of assembling the medical team and preparing for the various scenarios in basketball are specifically highlighted in the book. Management of trauma and injuries is addressed on all its aspects, from its occurrence and initial management on the court through following management phases and rehabilitation and onto surgical management when necessary. It is important to emphasize that various sports often differ, apart from common types of injuries, in injury management and return to sports aspects which are relevant and specific for each sport, and even the optimal surgical technique and approach (i.e., graft choices for knee ligament reconstructions, surgical techniques for shoulder dislocations, surgical management options in ankle injuries or Achilles tendon ruptures) for a basketball player may differ from the optimal technique in football players, fencers, or in martial arts. Therefore, clinicians would hopefully benefit from this book and gain more knowledge in aspects which would aid in their injury management and decision-making.

The book provides an overview of the state-of-the-art rehabilitation approaches and programs for return to play, injury prevention concepts and programs and load management strategies, as well as valuable and practical tools for implementing these important elements. Finally, the book provides special focus on the psychologic aspects of this fascinating sport as well as a special focus on the retired basketball player.

ESSKA is proud to have such an outstanding team of authors contributing to this project. This book is also unique as it is a first-time collaboration between ESSKA and our good friends from the American Orthopaedic Society for Sports Medicine (AOSSM), coming together as two of the world’s leading orthopedic sports medicine organizations to provide a concise state-of-the-art overview of sports medicine and science in basketball. Over the last years, ESSKA has worked intensively to strengthen the bonds between the two societies. It is with great pleasure that we see this work come to fruition in this huge project.

This book will undoubtedly raise the level of the ESSKA community as high as a basket hoop, and we can say that our great roster of authors, under the leadership of lead editors Lior Laver and Baris Koacoglu along with co-editors Ned Amendola, Brian Cole, Jeff Bytowski, and Amelia J. H. Arundale, have shown great team work from the first JUMP-BALL to produce this outstanding SLAM DUNK of a book !!!

Lyon, France
Luxembourg
Geneva, Switzerland
December 2019

David Dejour
Romain Seil
Jacques Menetrey

Preface

Since its invention in 1891 by Dr. James Naismith, basketball has become one of the most popular sports in the world, with an ever-growing popularity and a constant evolution. When all one needs to play is a ball and a basket, the popularity of the game is no surprise as well as the wide spectrum of participants from kids, “weekend warriors,” amateurs, and onto professional players. Expansions/extensions such as streetball (street basketball) and 3 × 3 basketball (soon to become an Olympic sport) are rapidly attracting more and more participants worldwide. The game itself has become extremely fast pace, with rule changes to accommodate this evolution. These changes, along with the growing numbers of participants worldwide, have brought a growing number of injuries and have thus emphasized the constant need for proper physical and medical coverage and attention for the athletes playing the game.

Basketball is a team sport, and team work is essential for success not only on the court but off the court as well. From the players, coaching staff, logistical staff, and onto the medical staff—team work is the key for a successful team and any successful initiative in the sport. Preparing a book for publication is no different and requires a great team effort.

For this purpose, an excellent multidisciplinary group of individuals has joined forces to form an elite team of authors—each a leader in his own field—in order to produce a unique achievement in the field of basketball medicine, sports medicine, and sports science.

Our goal was to create a comprehensive educational source not only for basketball medical caregivers and scientists but also for all basketball personnel, made easily available and accessible to provide answers in whichever aspect of their interest (physicians, PTs, ATs, rehabilitation specialists, conditioning trainers, and coaches). Our hope is that this source would also serve as a link between the different disciplines and modalities involved in basketball care, creating a common language and improving communication within the team staff and environment.

The unique group of people assembled for this project will undoubtedly bring a great passion not only to the game of basketball but also an equal level of scientific passion. With the help and collaboration of the ESSKA Publications department and in collaboration with the American Orthopaedic Society of Sports Medicine (AOSSM), our aim is that these “ingredients” would combine to produce a result that would be concise and practical as well as innovational and would improve the medical care in basketball

worldwide. We hope that this project would serve as a starting point and a fertile ground for collaboration across the sport of basketball in terms of research and initiatives to improve players' safety and medical care in the future as well as evolve alongside with the evolution of the game.

Tel-Aviv, Israel
Istanbul, Turkey
Chicago, USA
Brooklyn, USA
Durham, USA
Durham, USA

Lior Laver
Baris Kocaoglu
Brian Cole
Amelia J. H. Arundale
Jeffrey Bytomski
Annunziato Amendola

A Word from the Editors/ Acknowledgments

Over the years, we have been fortunate to be surrounded and supported by great medical staff, physicians, physical therapists, athletic trainers, performance specialists, and strength and conditioning coaches. As with any team, leadership, communication, and collaboration are essential to optimize results. Every one of us has learned tremendously from this environment and these interactions, and we are thankful for the professionals we have been fortunate to be around over the years. These professionals have made us better over the years and they still do. This accentuates the importance of teamwork, which is true not only on the field but off the field as well.

Preparing a book for publication is no different and requires a great team effort. A passionate team of international authors have come together to realize this important project and to produce a unique achievement in the fields of basketball, sports medicine, and sports science. An experienced and diverse team of experts, including orthopedic surgeons, primary care team physicians, and rehabilitation specialists, all covering different levels and leagues, from European basketball, college basketball, and the NBA, all came together for this project. We thank all the authors for the time, efforts, dedication, and passion invested in this project.

Editing such an extensive book requires a great effort in order to put together an end product which provides a balanced view and an adequate stage for the various topics covered, while maintaining a structured flow of content which will be useful and easy to navigate through for everyone.

We would especially like to thank and acknowledge Prof. Jon Karlsson, KSSTA editor-in-chief, for his support of this project as well as his contribution as associate section editor in the “basketball injuries” section (Part IV), and Dr. Ron Gilat, for his contribution as associate section editor in the “basketball injuries” section (Part IV).

This book is also a first of its kind collaboration between ESSKA and AOSSM, bringing together two of the world’s leading Orthopedic sports medicine societies. We would like to thank the leadership of both societies for their tremendous support and vision.

While the game of basketball is similar in its essence all over the world, resources also vary between levels of play and not every team can afford the medical and scientific support available at the highest levels. There is a great need for a relevant and reliable basketball-specific source to guide appropriate management of injuries and health problems, as well as to provide a strong frame for scientific and medical support in basketball—wherever it is

played. Our aim with this book was to address this need. The resulting content of the book provides the most comprehensive educational source for basketball medical caregivers, scientists, and all associated basketball personnel.

Our experience has taught us over the years that while knowledge and education are extremely important, leadership is essential for building a strong, sustainable, and successful medical team, and we tried to portray these elements throughout this book. This project was put together by a group of authors that is committed to excellence. Together, they have produced a practical and innovative source of high scientific quality relating to their beloved sport aimed to improve the medical care in basketball worldwide.

We are extremely proud of the end product, and we hope this would be a great impetus to increase the support for basketball science and to improve medical services across the sport.

Sincerely,

Lior Laver, MD

Baris Kocaoglu, MD

Brian Cole, MD

Amelia J. H. Arundale, PT, PhD, DPT, SCS

Jeff Bytomski, DO

Annunziato Amendola, MD

Basketball Sports Medicine and Science - Introduction

Basketball has grown over the years to become one of the most popular sports in the world. One can find a basketball hoop in the backyards of homes and schools in every continent and every corner of the world. History was made in a cold early December day in 1891, when Canadian James Naismith [1], a physical education professor at the International Young Men's Christian Association (YMCA) Training School [2] in Springfield, Massachusetts, USA, nailed a peach basket onto a 10-foot (3.0 m) elevated track, to keep his gym class students active on a rainy day. His purpose was to invent an indoor game to keep his students occupied and at proper levels of fitness during the long New England winters. No doubt, the journey and evolution the game has undergone in over a century have been tremendous.

The new game quickly gained popularity and developed, spreading through schools, colleges, and universities across North America and professional basketball developed soon after. By the 1920s, there were hundreds of men's professional basketball teams all over the United States. In 1946, the Basketball Association of America (BAA) was formed and later, in 1949, merged with the National Basketball League (NBL) to form the National Basketball Association (NBA). By the 1950s, basketball had also become a major college sport. In 1967, the American Basketball Association (ABA) emerged and operated alongside the NBA, until the ABA-NBA merger in 1976 to form current day's NBA, which is the leading professional basketball league in the world in terms of popularity, salaries, talent, and level of competition.

At the international scene, FIBA (International Basketball Federation) was formed in 1932, and originally only oversaw amateur players. Its acronym was derived from the French title: Fédération Internationale de Basket-ball Amateur, and thus "FIBA." Men's basketball was officially first included at the Berlin 1936 Summer Olympics. The first FIBA World Championship for men, now known as the FIBA Basketball World Cup or "Mundobasket," was held in Argentina in 1950. Three years later, in 1953, the first FIBA World Championship for women, now known as the FIBA Women's Basketball World Cup, was held in Chile. Although women's basketball was popular from the very beginning, it was officially added to the Olympics only in 1976, in Montreal, Canada.

Popular national and continental leagues have emerged over the years in Europe (the "Euroleague" is considered the second-best professional league in the world, after the NBA), Australia, Central and South America, Asia, and

Africa. Today, the global popularity of the sport is reflected in the many nationalities represented in the NBA and the Euroleague, including players from all over the world.

The NBA-backed Women's National Basketball Association (WNBA) was only established in 1997; however, amateur and professional women's basketball leagues, both national and continental, have existed worldwide for many years.

The popularity of the game of basketball has enjoyed a substantial growth in recent years. In addition, the development of the game's "siblings" and derivatives such as wheelchair basketball, street basketball (streetball) and 3 × 3 basketball, as well as "show basketball" (i.e., the Harlem Globetrotters) and "slamball" has helped spread the game even more, reaching out to all layers of the population worldwide.

Concurrently with the continuous development of the game and the increased focus on performance optimization in elite sports, significant progress took place in basketball with regard to parameters such as technique, tactics, and intensity, as well as the physical aspect. Over the years, the professional side of the sport has developed as well, alongside the evolution of the game rules, which helped turn it into a fast and very dynamic game, contributing to its attractiveness and growing popularity.

The popularity of the game is shared between genders and is evident in both the men's and women's game at all age groups, which unfortunately is still rarely seen in many other sports, and the women's game received a substantial boost since the establishment of the WNBA.

The future of basketball appears very bright. FIBA now counts 213 national federations worldwide under five confederations, making it one of the biggest international sports federations (www.FIBA.basketball). The introduction of 3 × 3 basketball as an Olympic sport, and the development of wheelchair basketball over the years, which has opened a window into Paralympic sports, is already helping spread the game even more, reaching out to all layers of the population worldwide. With the growing evolution and involvement of the media and social media in the sports, as well as the growing emphasis on fan engagement, the game of basketball is drawing more attention worldwide. Professional leagues for men and women draw thousands of spectators in Europe, Asia, and South America and Australia, and apart from the world and continental championships for national teams, continental club competitions in Europe (i.e., Euroleague and FIBA European champions league), NBA, WNBA, and NCAA for men and women annually feature the world's best teams, competing for substantial prize money and national/international prestige. Finally, the evolution of the game of basketball on all its aspects over the last century has made it the exciting, popular sport it is today. Undoubtedly, the next 100 years will bring ongoing evolution; however, in the meantime, the status of basketball as one of the most popular and exciting sports worldwide is assured.

The evolution and changes the game has undergone over the years have also influenced the physiologic demands of the game as well as the injury profile in the sport, which has emerged as one of the most injury-prone ball sports. The growing need for an appropriate medical and scientific envelope

to support the game became evident over the years; however, the epidemiologic profile of injuries in the sports was lacking, especially when compared to football (soccer), a fact portrayed quite distinctively in the much lower number of epidemiologic studies in basketball compared to football. Even so, over the years high-quality scientific research has been done in basketball, contributing not only to the sport itself, but providing great scientific merit for other sports as well.

Over the years, basketball has emerged as one of the most demanding team sports with regard to the variable skills involved and physical demands. Additionally, participation in the sport of basketball involves a unique constellation of injury risks and related health problems. Therefore, the health and medical care of every basketball team and each individual player requires a special and unique collection of knowledge and skill by the involved medical and health professional. This book aims not only to provide a deep scientific and clinical foundation for basketball professionals, covering every aspect of the health and medical care needed around the sport. This includes basic science topics, medical team assembly and related preparations, injury management and rehabilitation, injury prevention, understanding the special needs of unique and specific groups as well as psychological aspects—all are addressed and covered extensively.

This unique project compiles the work of the top international experts in the field of basketball medicine and basketball science. It is the most comprehensive scientific source to date, aiming to aid and guide medical and all scientific personnel around the sport of basketball, and hopefully will be the reference and starting point of many other joint projects aimed to develop the medical and scientific support for the game in the coming years.

Lior Laver
Baris Kocaoglu
Brian Cole
Amelia J. H. Arundale
Jeff Bytomski
Annunziato Amendola

References

1. The Greatest Canadian Invention. CBC News. Archived from the original on December 3, 2010.
2. YMCA International—World Alliance of YMCAs: Basketball: a YMCA Invention. www.ymca.int. Archived from the original on March 14, 2016. Retrieved March 22, 2016.

Contents

Part I Basic and Applied Sciences

1 Physical and Anthropometric Characteristics of Basketball Players	3
Jay R. Hoffman	
2 Activity and Physiological Demands During Basketball Game Play	13
Zoran Milanović, Emilija Stojanović, and Aaron T. Scanlan	
3 Endocrine Aspects in Performance and Recovery in Basketball	25
Alon Eliakim and Dan Nemet	
4 Biomechanics of Lower Extremity Movements and Injury in Basketball	37
Jeffrey B. Taylor, Eric J. Hegedus, and Kevin R. Ford	
5 Biomechanics of Upper Extremity Movements and Injury in Basketball	53
Jeffrey B. Taylor, Kevin R. Ford, and Eric J. Hegedus	
6 Nutrition and Hydration in Basketball Athletes	65
Jorge Molina-López and Elena Planells	

Part II The Basketball Medical Perimeter - Aspects in Medical Preparation

7 Constructing a Medical Team: The Medical Needs of a Basketball Team	83
Benjamin Oshlag and Benjamin Boswell	
8 The Role of Pre-participation Assessment and Screening in Basketball	91
Mark Rogers and Shan Fairbanks	
9 Medical Coverage of Basketball Events: From Local Competitions to European, World Championships and Olympic Games	103
Ignacio Muro, Ugur Dilicikik, and Baris Kocaoglu	

10	Communication Challenges in Medical Management of a Basketball Team	111
	Kai Fehske, Michael Davison, Christoph Lukas, and Lior Laver	
11	The Role of the Team Physician in Basketball	121
	Jeffrey R. Kovan and Chelsea Gonzalez	
12	Team Medical Coverage in Elite European Basketball	125
	Baris Kocaoglu, Ignacio Muro, and Guy Morag	
13	Team Medical Coverage in College Basketball	135
	John M. MacKnight and Avinash M. Sridhar	
14	Team Medical Coverage in the National Basketball Association (NBA)	145
	Thomas J. Kremen Jr., Grant Schroeder, and David McAllister	

Part III On Court Management in Basketball

15	On Court Examination in Basketball: What the Clinician Should Not Miss	157
	Jeremy Truntzer, Marc Safran, Tahsin Beyzadeoglu, and Geoffrey Abrams	
16	Preparing for Worst-Case Scenarios on Court: How to Best Prepare Your Medical Team	165
	Murat Caglar, İsmail Basoz, Cemre Ayhan, Goktug Firatli, and Baris Kocaoglu	
17	Cardiopulmonary Resuscitation and Cardiorespiratory Arrest in Basketball: Guidelines	175
	Sinan Dagdelen	
18	Immediate Management of Acute Trauma on Court in Basketball	193
	Tekin Kerem Ulku and Ignacio Muro	

Part IV Basketball Injuries and Management

19	Basketball Injuries: Epidemiology and Risk Factors	201
	Kellie K. Middleton, MaCalus V. Hogan, and Vonda Wright	
20	Head, Neck, and Face Injuries in Basketball	215
	Nathan McMurray, Gary E. Means, and Thomas Stocklin-Enright	
21	Concussion Management in Basketball	225
	Kathleen Roberts, Robert G. Hosey, Jeffrey Bytomski, and M. Kyle Smoot	

22	Ophthalmic Trauma in Basketball	239
	C. Ellis Wisely and Terry Kim	
23	Shoulder Injuries in Basketball	251
	Baris Gorgun, Eran Maman, Giacomo Marchi, Giuseppe Milano, Baris Kocaoglu, and Michael Hantes	
24	Management of Shoulder Instability in Basketball Players	265
	David W. Altchek, James B. Carr II, Yiğit Umur Cirdi, Selim Ergün, and Mustafa Karahan	
25	Elbow Injuries in Basketball	281
	Kurt E. Stoll and Grant E. Garrigues	
26	Wrist and Hand Injuries in Basketball	293
	Kostas Ditsios, Triantafyllos Katsimentzas, Polychronis Papadopoulos, and Pericles Papadopoulos	
27	Isolated Finger Injuries in Basketball	303
	Ufuk Nalbantoglu and Okan Tok	
28	Hip and Groin Injuries in Basketball	313
	Lior Laver, Kelechi Okohara, Ben U. Nwachukwu, Omer Mei-Dan, and Shane J. Nho	
29	Knee Injuries in Basketball	333
	Henrique Jones, Gian Luigi Canata, Yaniv Yonai, Christoph Lukas, and Kai Fehske	
30	Management of ACL Injuries in Basketball	351
	Kelechi R. Okoroha, Eric D. Haunschild, Ron Gilat, and Brian Cole	
31	Management of Multi-Ligament Injuries in Basketball	363
	Robert A. Burnett, Nabil Mehta, Kyle N. Kunze, Gilbert Moatshe, Robert F. LaPrade, and Jorge Chahla	
32	Management of Knee Cartilage Injuries in Basketball	379
	Hailey P. Huddleston, Jorge Chahla, Brian Cole, and Adam B. Yanke	
33	Management of Knee Injuries in Adolescent Basketball Players	391
	Bonnie P. Gregory and Jonathan C. Riboh	
34	Anatomical Causes for Patellofemoral Pain in Basketball Players	411
	Pedro Castelhanito, Sebastiano Vasta, and David Dejour	
35	Management of Patellofemoral Disorders in Basketball	423
	Nicholas Tsouris, Charles Popkin, and Elizabeth A. Arendt	

36	Foot and Ankle Injuries in Basketball	445
	Craig C. Akoh, Jie Chen, Mark Easley, and Annunziato Amendola	
37	Management of Chronic Ankle Instability in the Basketball Player	459
	Christopher E. Gross, J. Brett Goodloe, and James A. Nunley II	
38	Management of Cartilage Injuries of the Foot and Ankle in Basketball	467
	Kenneth J. Hunt, Kenneth S. Smith, and Steve Short	
39	Achilles Tendon Ruptures in Basketball	481
	Michael R. Carmont, Annelie Brorsson, Olof Westin, Katarina Nilsson-Helander, and Jón Karlsson	
40	Management of Common Tendinopathies in Basketball	491
	Zahab S. Ahsan and Answorth A. Allen	
41	Back Injuries and Management of low Back Pain in Basketball	509
	Lara W. Massie, Thomas J. Buell, Eyal Behrbalk, and Christopher I. Shaffrey	
42	Osteoarthritis in Basketball Players	519
	Richard Danilkowicz, Brian C. Lau, Thomas Carter, and Annunziato Amendola	
43	Management of Muscle Injuries in Basketball	531
	Gil Rodas, Carles Pedret, David Schmidt, Jordi Puigdellivol, William Garret Jr, and Dai Sugimoto	
44	Stress Fractures and the Stress Reaction Spectrum in Basketball	547
	Henrique Jones, Danica Vance, and Annunziato Amendola	
45	The Role of Orthobiologics in the Management of Tendon and Fascia Injuries in Sports	561
	Ron Gilat, Ferran Abat, Jorge Chahla, Eric D. Haunschild, Lior Laver, and Brian Cole	
46	The Role of Orthobiologics for the Management of Ligament and Muscle Injuries in Sports	587
	Lior Laver, Gonzalo Samitier Solis, Ron Gilat, Montse García-Ballebó, Emilio Lopez-Vidriero, Brian Cole, and Ramon Cugat	
47	The Role of Orthobiologics in the Management of Cartilage and Meniscal Injuries in Sports	605
	Derrick M. Knapik, Ron Gilat, Eric D. Haunschild, Lior Laver, and Brian Cole	

48	In-Season Management of Injuries in Basketball: A Pragmatic Approach	617
	Jeremy Truntzer, Guy Morag, Geoffrey Abrams, and Marc Safran	
Part V Injury Prevention, Rehabilitation and Back to Field Process in Basketball		
49	Shoulder Assessment in Basketball	627
	Wayne J. Diesel, Ido Dana, and Lior Laver	
50	Functional Assessment in Elite Basketball Players	645
	Nicholas D. Potter and Jeffrey B. Taylor	
51	Injury Prevention in Basketball	657
	Annelie Brorsson, Unnur Sædís Jónsdóttir, and Jón Karlsson	
52	Digging Deep into the Etiology of Basketball Injuries: A Complex Systems Approach for Risk Mitigation	665
	Oluwatoyosi B. A. Owoeye	
53	Practical Guidelines for Injury Prevention in Basketball: How to Get it Right	673
	Steve Short	
54	Rehabilitation of Shoulder Injuries in Basketball	687
	Fred Breidenbach, Elizabeth Sargent, and Mathew Failla	
55	Rehabilitation of Trunk, Hip and Groin Injuries in Basketball Players	701
	David M. Clancy, Donald S. Strack, Jeff G. Konin, and Ciaran Dunne	
56	Rehabilitation of Knee Injuries in Basketball Players	711
	Amelia J. H. Arundale	
57	A Biomechanical Perspective on Rehabilitation of ACL Injuries in Basketball	723
	Francesco Della Villa, Stefano Della Villa, and Matthew Buckthorpe	
58	Rehabilitation of Foot and Ankle Injuries in Basketball Players	737
	Jennifer A. Zellers and Karin Grävare Silbernagel	
59	Rehabilitation of Tendinopathy in Basketball	749
	Andrew L. Sprague, Patrick O'Rourke, and Karin G. Silbernagel	
60	On-Court Rehabilitation—From Treatment Table and Return to Play to a Return to Performance	765
	Amelia J. H. Arundale, Maggie Bryant, and Leslie Gartner	

61	Strength Training for Basketball	779
	Lorena Torres Ronda and Francesco Cuzzolin	
62	Training and Performance Differences Between the NBA and FIBA Rules and Major Competition Aspects (Euro, WC, and Olympics)	791
	Francesco Cuzzolin	
63	Post-Exercise Recovery Strategies in Basketball: Practical Applications Based on Scientific Evidence	799
	Thomas Huyghe, Julio Calleja-Gonzalez, and Nicolás Terrados	
64	Load Management in Basketball	815
	Tim J. Gabbett	
65	Practical Considerations for Workload Measurement in Basketball	823
	Aaron T. Scanlan, Jordan L. Fox, Daniele Conte, and Zoran Milanović	
Part VI Special Considerations in Basketball		
66	The Female Basketball Player	835
	Kenzie Johnston, Crystal Higginson, Heather Saffel, Anna Camille Moreno, Kendall E. Bradley, and Alison P. Toth	
67	The Young/Adolescent Basketball Player	847
	Alex B. Diamond and Andrew W. Kuhn	
68	Management of the Sickle Cell Spectrum in Basketball Players	865
	Kimberly G. Harmon	
69	Playing Basketball with a Cardiac Condition: Recommendations and Guidelines	875
	David S. Owens and Jonathan A. Drezner	
70	Inherited Aortic Disease and Sports Participation	891
	Sophia Larson, Jonathan Buber, and Andrew Cheng	
71	Selected Medical Issues in Basketball: Diabetes, Allergies, Asthma, and Dermatologic Issues	907
	Christopher M. Miles and Laura Lintner	
72	Is Overload an Issue in Young Basketball Players?	919
	Andrew Gregory	
73	Doping and Nutrition Supplementation in Basketball	925
	Kai Fehske and Christoph Lukas	
74	Long-Distance Traveling in Basketball: Practical Applications Based on Scientific Evidence	929
	Thomas Huyghe and Julio Calleja-Gonzalez	

75	General Considerations in Basketball: Court Type, Shoes, and Protective Gear	947
	Priscilla Tu and John Travis Nelson	
76	Ethical and Medico-Legal Issues in Injury Management and Return to Sport in Basketball	957
	Jordan Rawlings and Blake Boggess	
Part VII Psychological Aspects in Basketball		
77	The Importance of Sport Psychology in Basketball	969
	Vanessa M. LaBode-Richman and Paul Groenewal	
78	Sport Psychology in Basketball: Performance Under Pressure	983
	Luca Sighinolfi	
79	Perceptual-Cognitive Processes in Basketball—Individual and Team Aspects	995
	Itay Basevitch, Nataniel Boiangin, Camilo Sáenz-Moncaleano, and Gershon Tenenbaum	
80	Psychological Aspects in Return to Sport Following ACL Reconstruction	1005
	Ryan Zarzycki and Clare Ardern	
81	The Retired Professional Basketball Player—Psychological Aspects	1015
	Hidayet Turkoglu and Merve Hosver	

Part I

Basic and Applied Sciences

Physical and Anthropometric Characteristics of Basketball Players

1

Jay R. Hoffman

1.1 Introduction

The popularity of basketball as an international sport has been well-publicized over the past 20 years. Although the rules differ across the various professional and amateur leagues, the sport itself is predominantly played as a high-intensity, strength/power event. Previous descriptions of the physiology of basketball had suggested that the intensity of the game was dependent upon coaching strategy [1, 2]. However, changes in rules and character of the game (e.g., requirement of a shot clock and greater reliance on exploiting the defense to offense transition) have provided a more specific physiological requirement for success. In 1996, Hoffman and colleagues [3] in a 4-year study of elite male college basketball players indicated that components of anaerobic ability (i.e., speed, vertical jump, and agility) were strong predictors of playing time, while a high aerobic capacity was reported to have a negative relationship with playing time. Although additional studies examining performance predictors of playing time are limited, subsequent research has tended to support the importance of strength/power and anaerobic ability to basketball playing performance (e.g., often comparing starters to nonstarters, or different levels of play).

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Pojjskic et al. [4] noted the importance of explosive power and anaerobic capacity as determinants of shooting performance, and a recent study by Garcia-Gil and colleagues [5] in elite female Spanish basketball players indicated that height, wing span, body fat, and time in T-Drill test were significant predictors of playing performance. A recent investigation examining data obtained from the National Basketball Association (NBA) Draft Combine reported that anthropometric measures (specifically values relating to length) were the best predictors of future basketball performance, followed by upper-body strength [6].

Fact Box

Components of anaerobic performance (i.e., speed, vertical jump, and agility) are strong predictors of playing time, while endurance capacity has a negative relationship with playing time.

This chapter will focus on the physical and anthropometric characteristics of basketball players and the relationship that these measures have to basketball performance. When possible, comparisons between positions and leagues will be done. Although focus of discussion will be based upon recent literature, historical perspectives will be provided when appropriate.

1.2 Physical Attributes of the Basketball Player

This section will examine the contribution of various components of fitness and how they impact the basketball player. Data will be discussed relative to both sex and league. In addition, professional basketball will not be grouped together, but rather data from players participating in the NBA will be separated from the results of professional basketball players participating in various teams across the International Basketball Federation (FIBA).

1.2.1 Aerobic Capacity

The maximal oxygen consumption ($\text{VO}_{2\text{max}}$) of male basketball players has been reported to range from $49.8 \text{ mL kg min}^{-1}$ to $63.4 \text{ mL kg min}^{-1}$ [7–11]. These investigations focused on National Collegiate Athletic Association (NCAA) Division II, European and Tunisian professional players. Interestingly, it has been suggested that changes in rules relating to the shot clock (reduction in time allowed to shoot the ball from 30 to 24 s) and moving from two halves to four quarters resulted in a need for greater aerobic capacity [9]. Although evidence is limited, Cormery and colleagues [9] in a 10-year examination of male basketball players playing in the top French professional league reported that the aerobic capacity of guards increased from $51.0 \pm 1.6 \text{ mL kg min}^{-1}$ in athletes before the year 2000 to $63.4 \pm 2.7 \text{ mL kg min}^{-1}$ in athletes playing after the year 2000. No changes in aerobic capacity were noted in forwards and centers during the same time span. In contrast to previous reports [1], guards appeared to have a greater aerobic capacity than either forwards or centers in collegiate and professional level basketball in both men [8, 12, 13] and women [14]. Whether an increase in aerobic capacity among certain positions in basketball is related to changes in the physiological needs of basketball is not clear. Evidence to date, although limited, suggests that aerobic capacity is not a predictor of playing time or performance in elite male basketball players [3].

In contrast to the relationship reported between aerobic capacity and basketball performance in men, aerobic capacity has been suggested to be associated with basketball performance in women [15]. However, as previously indicated investigations examining the relationship between aerobic capacity and basketball performance in both men and women are scarce in the past decade. Interestingly, Ben Abdelkrim and colleagues [7] reported that aerobic capacity was related ($r = 0.67$) to high-intensity activity during a basketball game in Tunisian male basketball players. Whether this is a function of the Tunisian basketball league or a global trend is not well understood. Further research is needed in this area.

1.2.2 Anaerobic Power

Components of anaerobic performance (i.e., speed, vertical jump, and agility) have been demonstrated to be strong predictors of playing time in elite male college basketball players [3]. Power performance in basketball players is most often assessed via a vertical jump. Jump tests are reported as jump height or jump power. The latter test can be performed either on a force plate or predicted from vertical jump height [16]. Different methods of vertical jump assessment have made it difficult to compare between studies or to develop normative data [1].

Studies in the past 10–15 years have been consistent in demonstrating that vertical jump performance can differentiate between starters and nonstarters in NBA players [17] and NCAA Division I women [18], and between different levels of play [12, 19]. A recent study by Spiteri and colleagues [19] indicated that the countermovement jump was able to differentiate between players of different competitive leagues in women's basketball. Basketball players in the Women's National Basketball Association (WNBA) jumped significantly higher ($34.8 \pm 3.3 \text{ cm}$) than athletes in the Australian Women's Basketball League (WNBL) and NCAA Division I women's basketball ($30.6 \pm 3.9 \text{ cm}$ and $32.0 \pm 5.1 \text{ cm}$, respectively). Significant differences have also been reported between positions. Guards tend to jump significantly higher than centers and

power forwards in men [8, 11, 13], but these positional differences may not be seen in women, especially at lower levels of play [20]. However, vertical jump power expression is significantly greater in centers than guards [11, 13], which likely reflects the greater mass seen in these players. However, this difference is lost when power is expressed relative to body mass [8].

Examination of vertical jump data in NBA players reveals interesting results. Figure 1.1 provides a perspective of standing reach height, vertical jump height without a step (preparation step), and maximal vertical jump height with a step. NBA players are only ~44 cm below the baskets rim when standing with their arms outstretched; however, centers are only ~26 cm below the rim. The average NBA player jumping without a step will be ~29 cm above the rim and will be 42 cm above the rim when jumping with a step. It can be clearly seen that much of the NBA game is played “above the rim.” Interestingly, jump height or power has not been demonstrated to be an effective predictor of performance in NBA basketball players [6]. However, that could simply be a function of the exceptional level of jumping ability in most NBA players.

Fact Box

Evidence from the standing reach and vertical jump height of the NBA players clearly indicates that much of the NBA game is played above the rim!

Position comparisons in players participating in the NBA combine from 2000 to 2015 are depicted in Table 1.1. This data was obtained from the NBA Draft Combine (<http://stats.nba.com/draftcombine/>). Standing reach height is significantly different between positions. The standing reach height of point guards was less than all other players, the standing reach height of shooting guards was less than all forwards and centers, while the standing reach height of small forwards were less than both power forwards and centers. Finally, the standing reach height of power forwards was less than centers. When examining total height achieved from a jump with and without a step, results indicated that point guards achieved a total jump height lower than all other positions. Although the total height achieved from jumping by shooting guards was significantly greater than point guards, it was significantly lower than all other positions. No differences were noted in total height achieved from no step and maximum vertical jump height in forwards and centers. Interestingly, when comparing relative jump height between positions (jump height – standing height), no significant differences were observed between positions in either jump height with a step or without a step. Figure 1.2 depicts the range of average jump height (no step or maximal jump height) with standard deviation for 16 years of NBA combine testing. The highest average maximal jump heights have been observed in the last 3 years of the reported testing (2013–2015), in which average maximal jump heights ranged from 89.6 to 91.6 cm, and average jump height with no step ranged from 76.3 to 77.6 cm.

Fig. 1.1 Standing reach height, vertical jump height without a step, and maximal vertical jump height with a step in relation to backboard and rim height in NBA basketball players

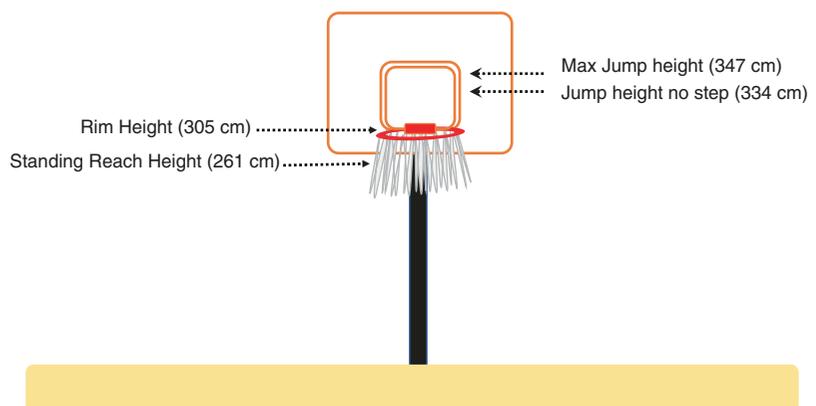


Table 1.1 NBA Combine jump, strength, and speed performance between 2000 and 2015

Position	<i>N</i>	Standing reach (cm)	No step jump height (cm)	Max jump height (cm)	NO step VJ (cm)	Max VJ (cm)	BP reps (185)	3/4 Sprint speed (s)
Point guards	203	245.4 ± 7.6 ^a	320.5 ± 8.6 ^a	335.0 ± 10.2 ^a	74.9 ± 7.6 ^a	89.7 ± 9.1 ^a	8.6 ± 5.2 ^a	3.21 ± 0.10 ^a
Shooting guards	202	256.5 ± 5.8 ^b	332.2 ± 8.1 ^b	345.9 ± 8.1 ^b	75.7 ± 7.4 ^a	89.4 ± 8.1 ^a	9.9 ± 5.3 ^a	3.24 ± 0.10 ^a
Small forwards	191	265.4 ± 6.1 ^c	339.9 ± 7.1 ^c	352.8 ± 8.4 ^c	74.7 ± 7.9 ^a	87.6 ± 8.9 ^a	10.3 ± 5.3 ^a	3.28 ± 0.12 ^a
Power forwards	259	271.3 ± 5.3 ^d	343.7 ± 7.1 ^c	354.3 ± 8.1 ^c	72.4 ± 6.9 ^a	83.3 ± 7.6 ^a	13.0 ± 5.7 ^a	3.33 ± 0.13 ^a
Centers	145	278.9 ± 5.6 ^e	347.2 ± 7.9 ^c	357.1 ± 7.6 ^c	68.6 ± 7.9 ^a	78.5 ± 7.9 ^a	11.7 ± 5.4 ^a	3.42 ± 0.14 ^a

Different letters indicate a significant difference

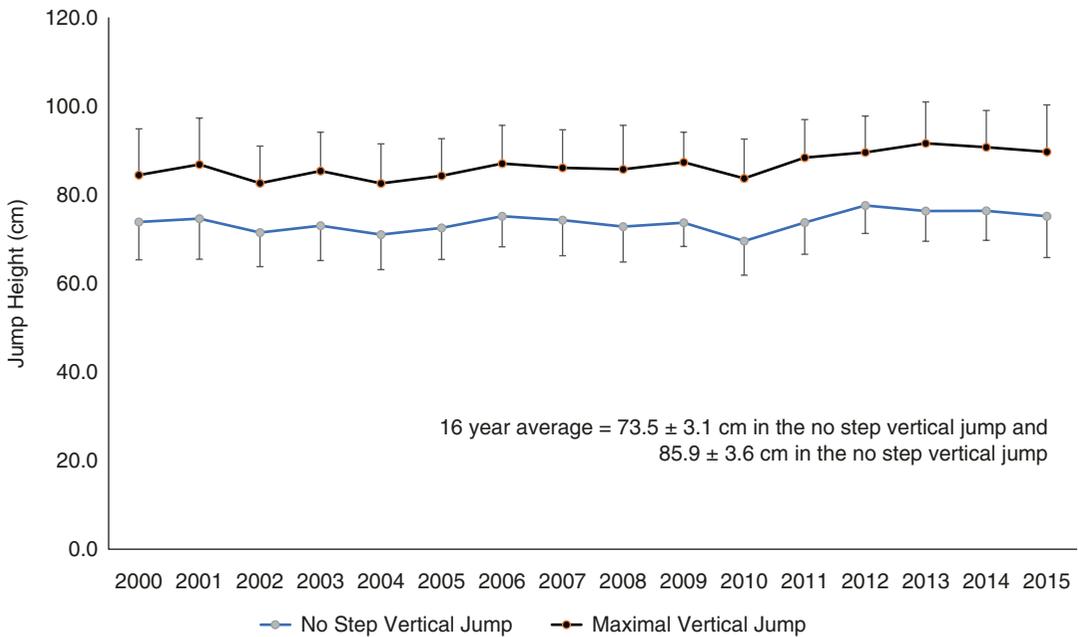


Fig. 1.2 No step and maximal jump height between 2000 and 2015 in professional basketball players participating in the NBA Combine

1.2.3 Strength

Examination of the literature in the past 10–15 years has found a paucity of data reporting maximal strength in college or professional basketball players. It is surprising considering that maximal squat strength (1-RM squat) has been reported to be a strong predictor of playing time in NCAA Division I male basketball players [3]. Hoffman and Maresh [2] reported that the average 1-RM squat in NCAA Division I male athletes was 152.2 ± 36.5 kg. In a position by position analysis

in NCAA Division I basketball players, Latin and colleagues [21] indicated that collegiate forwards (161.9 ± 37.7 kg) were significantly stronger than centers (138.1 ± 32.1 kg) but similar to guards (151.1 ± 35.5 kg). Lower body strength has been suggested to be important for “boxing-out” and positioning during a basketball game [1]. An investigation by Köklü and colleagues [12] comparing first and second division Turkish professional players suggested that isokinetic leg extension and leg flexion strength was unable to differentiate players between the two divisions of basketball play.

Interestingly, the only recent publication that has reported 1-RM strength in collegiate basketball players assessed the front squat and hang (power) clean in both men and women NCAA Division II players [22]. The explosive action of the hang clean and its ability to integrate strength, explosive power, and neuromuscular coordination among several muscle groups suggest that this exercise has similarity to many of the actions common to basketball players [1], and may be a more appropriate exercise to assess in basketball players than the squat. Thus, improving strength in this exercise may provide for a better transfer of strength to the basketball court. Hang clean strength in NCAA Division II men was recently reported to be 44.0 ± 6.1 kg and 23.7 ± 2.7 kg in women [22]. Previously, Latin and colleagues [21] reported maximal strength in the power clean to be 99.2 ± 15.2 kg (range 59.0–137.3 kg) in NCAA Division I male college basketball players, with forwards (105.1 ± 16.9 kg) being significantly stronger than guards (94.5 ± 13.0 kg) but not centers (99.8 ± 13.7 kg).

In a previous discussion of the physiology of basketball, Hoffman [1] indicated that bench press strength was the most common strength testing measure reported in basketball players. This was despite a poor relationship (r 's from -0.04 to 0.14) reported between playing time and upper-body strength [3]. Maximal bench press strength in NCAA Division I college basketball players was reported to be 102.7 ± 18.9 kg [2], with no differences noted between positions (Latin and colleagues [21]). Ben Abdelkrim and colleagues [7] indicated that the 1-RM bench press strength in Tunisian first division basketball players was 92.1 ± 8.3 kg. Recent research has suggested that upper-body strength may be a moderate predictor of future NBA performance [6]. Figure 1.3 provides the average number of repetitions performed by basketball players invited to the NBA combine between the years 2000 and 2015. The average number of repetitions performed was 10.8 ± 5.6 . This is equivalent to a 1-RM bench press of 115.6 kg [23]. Table 1.1 provides a position by position comparison of the number of repetitions performed in the 185 lb bench press

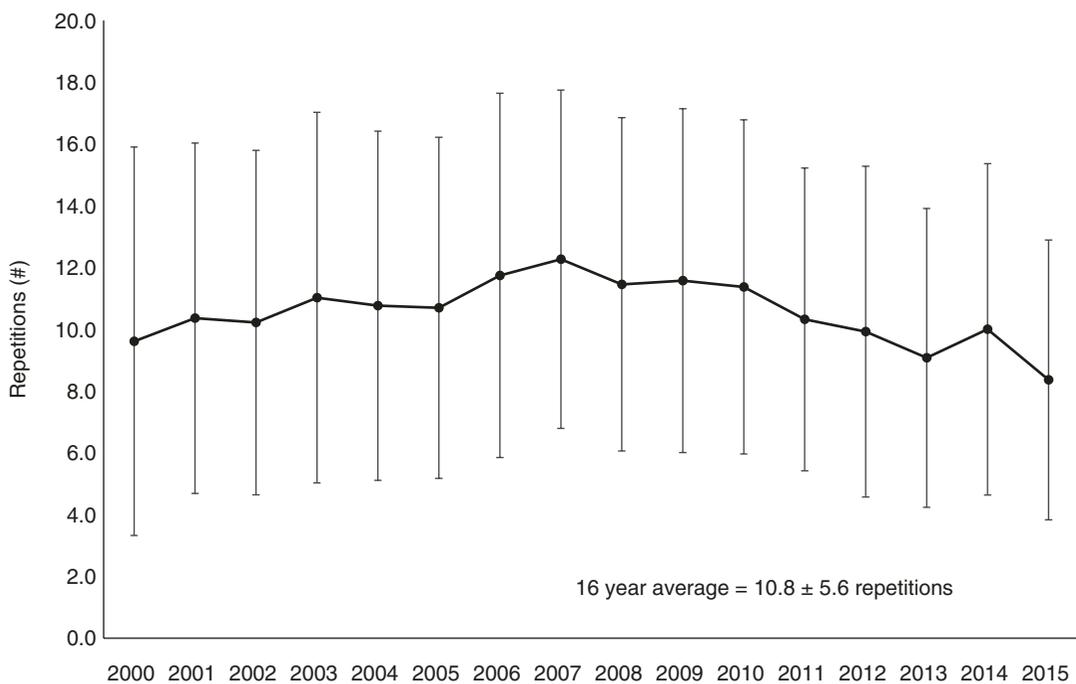


Fig. 1.3 Number of repetitions performed in the 185 lb (84.1 kg) Bench Press test in professional basketball players participating in the NBA Combine (2000–2015)

test. Although power forwards performed the greatest number of repetitions, they were not significantly different than those observed for any other position, and no significant differences in repetitions performed were noted between any position. However, these results should be interpreted in an appropriate context as lower body strength (i.e., 1-RM squat) was not examined or compared in these athletes.

1.2.4 Speed and Agility

Initial research with elite college basketball players indicated that speed (30 m) was a significant predictor of playing time [3]. Speed has also been demonstrated to differentiate between different levels of play in some studies [12, 24] but not all [5, 19, 20]. Ben Abdelkrim and colleagues [7] reported that sprint speed (either 10 m, 20 m, or 30 m) was not related to high-intensity performance in basketball, while sprint performance did not predict future performance in the NBA, as determined from actual basketball performance during the players initial competitive season

[6]. Figure 1.4 depicts 3/4 court sprint speed in athletes participating in the NBA combine from 2000 to 2015. The average sprint speed during this 16-year period was 3.29 ± 0.14 s. No significant changes from this mean were seen at any time. It is likely that sprint speed of these athletes was similar and at a high level to limit this fitness component being an effective predictor of future basketball performance. It is likely when there is a greater variability of performance that sprint speed may be a better predictor.

There does appear to be differences in sprint speed comparisons between positions. Although speed does not appear to be significantly different between positions in basketball players participating in the NBA combine (see Table 1.1), point guards were 0.2 s faster than centers in the 3/4 court sprint (~21.5 m). Others have reported that guards are significantly faster than centers in male Spanish U-15 players, but not in U-16 or U-17 players [25]. Others have also indicated that guards are faster than centers in Turkish men [12], Belgian men [8], and English women basketball [20]. The distances used for assessment in these latter studies were 5-, 10-, and 20-m.

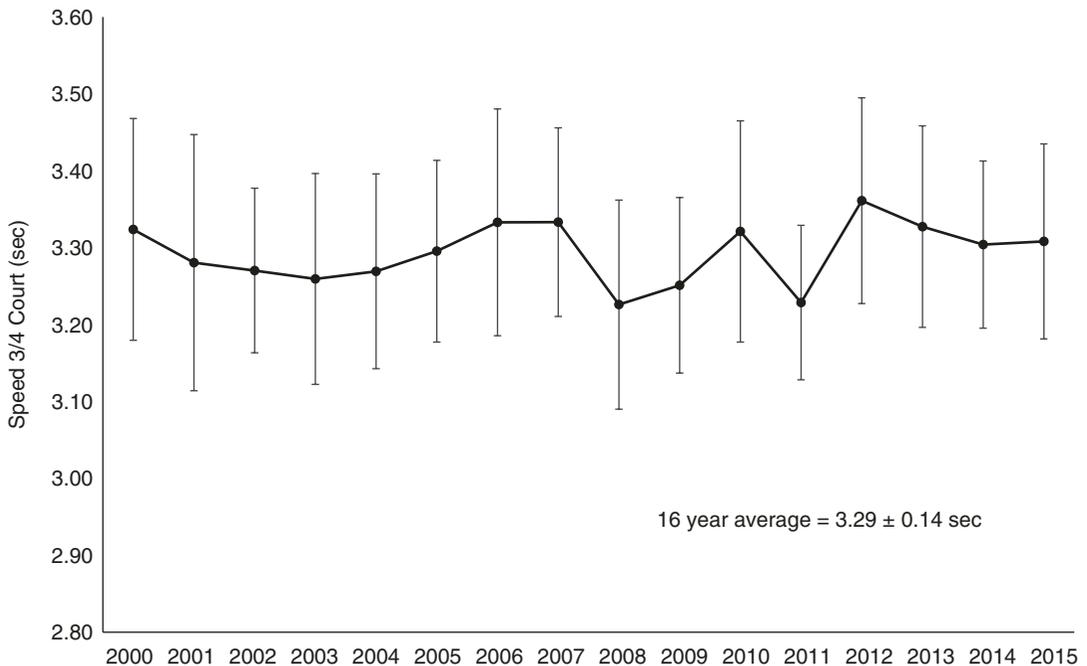


Fig. 1.4 Speed (3/4 court) of professional basketball players participating in the NBA Combine (2000–2015)

Agility has been observed to be more consistent in its relation to basketball performance. Hoffman and colleagues [3] were the first to demonstrate that the T-drill was able to predict playing time in elite male college basketball players. Subsequent studies have reported that the T-drill was a significant performance predictor ($r = 0.34$, $p < 0.05$) in Spanish first division women's basketball [5] and can explain 38.9% of the variability in high-intensity shuttle performance in a basketball game in Tunisian men [7]. Others using change of direction tests to assess agility also reported the effectiveness of these assessments in differentiating levels of play [19, 26]. These results are not surprising considering the rapid changes in movement and direction during the game of basketball.

1.2.5 Anthropometric Measures

A recent study indicated that a matrix of anthropometric measures labeled as *length size*, which included wingspan, hand length, standing reach, hand width, and body weight, was more important for predicting future performance of basketball players in the NBA than power/quickness or upper-body strength [6]. These results probably reflect the physical similarity among the players invited to the combine in speed, strength, and vertical jump ability. Other studies have also demonstrated the importance of anthropometry in differentiating players of differing levels of ability in women [5] and adolescent [24] basketball players. Garcia-Gill and colleagues [5] reported that elite women's Spanish League

basketball teams that were more successful at the end of the competitive season tended to be taller with larger wingspans and a lower body mass index. However, no significant differences were detected for these variables. Skinfold thickness was the only anthropometric variable that differed significantly between the teams, with the lowest skinfold thicknesses observed in the highest ranked teams. Table 1.2 depicts the anthropometric measures, including length size, in basketball players participating in the NBA combine from 2000 to 2015. Each position differed from each other in height, body weight, and wingspan. This is consistent with other studies comparing anthropometric measures between basketball playing positions [25].

Body composition in NBA players also differed between positions (see Table 1.1). Guards and small forwards appeared to be leaner than both power forwards and centers. However, all players were quite lean. An 8- to 9-year study in men and women NCAA Division I basketball players ($n = 127$ and $n = 196$ in the men and women, respectively) revealed that the body fat composition of guards and forwards for the men was $8.6 \pm 3.3\%$ and $14.9 \pm 4.8\%$, respectively [27]. Body fat composition of guards and forwards for the women was $19.2 \pm 6.3\%$ and $24.2 \pm 5.7\%$, respectively. Differences between the guards and forwards for both sexes were significantly different, which was similar to that reported by others examining European professional basketball players [8, 11].

The ability to improve body composition may be limited. Fields and colleagues [27] reported that body composition values remained consistent

Table 1.2 NBA Combine anthropometric assessments between 2000 and 2015

Position	<i>N</i>	Height (cm)	Body weight (kg)	Wingspan (cm)	Body fat (%)	Hand length (cm)	Hand width (cm)
Point guards	203	184.7 \pm 4.8 ^a	84.4 \pm 5.8 ^a	195.1 \pm 7.1 ^a	6.7 \pm 2.5 ^a	21.1 \pm 0.8 ^a	23.4 \pm 1.5 ^a
Shooting guards	202	192.5 \pm 3.3 ^b	92.3 \pm 5.9 ^b	204.2 \pm 5.6 ^b	6.9 \pm 2.2 ^a	21.8 \pm 0.8 ^b	23.6 \pm 1.8 ^a
Small forwards	191	199.4 \pm 3.3 ^c	98.1 \pm 6.4 ^c	210.8 \pm 5.6 ^c	7.4 \pm 2.3 ^a	22.4 \pm 1.0 ^c	24.1 \pm 1.5 ^{a,b}
Power forwards	259	202.9 \pm 3.3 ^d	107.9 \pm 8.3 ^d	215.9 \pm 5.3 ^d	9.0 \pm 3.1 ^b	22.9 \pm 1.0 ^{c,d}	24.9 \pm 1.5 ^{b,c}
Centers	145	208.5 \pm 3.8 ^e	113.5 \pm 9.8 ^e	221.2 \pm 5.8 ^e	10.0 \pm 4.2 ^b	25.4 \pm 0.8 ^d	25.7 \pm 1.8 ^c

Different letters indicate a significant difference

across the athlete's collegiate career. Interestingly, the body composition reported in studies of male basketball players in the past decade is comparable to that previously reported by Latin and colleagues [21], in which NCAA Division I guards had a significantly lower body fat percentage ($8.4 \pm 3.0\%$) than centers ($11.2 \pm 4.5\%$) but not forwards ($9.7 \pm 3.9\%$). These positional differences likely reflect the greater mass needed by centers to play the "low post" position, which involves considerable body contact during box-outs, picks, and rebounding [1].

Take Home Message

Physical and anthropometric measures provide an important resource for coaches to assist in talent identification. At the highest levels of play, the differences in physical performance ability (e.g., strength, power, speed, and agility) may be quite minimal. For those athletes, differences in anthropometric measures such as height, body weight, wingspan, and hand length may take on greater significance.

References

- Hoffman JR. Physiology of basketball. In: McKeag DB, editor. *Handbook on basketball: Olympic handbook of sport medicine*. Oxford, England: Blackwell Publishing; 2003. p. 1–11.
- Hoffman JR, Maresh CM. Physiology of basketball. In: Garrett WE, Kirkendall DT, editors. *Exercise and sport science*. Lippincott, Williams & Wilkins: Baltimore, MD; 2000. p. 733–44.
- Hoffman JR, Tennenbaum G, Maresh CM, Kraemer WJ. Relationship between athletic performance tests and playing time in elite college basketball players. *J Strength Cond Res*. 1996;10:67–71.
- Pojskic H, Sisc N, Separovic V, Sekulic D. Association between conditioning capacities and shooting performance in professional basketball players: an analysis of stationary and dynamic shooting skills. *J Strength Cond Res*. 2018;32:1981–92.
- Garcia-Gil M, Torres-Unda J, Esain I, Duñabeitia I, Gil SM, Gil J, Irazusta J. Anthropometric parameters, age, and agility as performance predictors in elite female basketball players. *J Strength Cond Res*. 2018;32:1723–30.
- Teramoto M, Cross CL, Rieger RH, Maak TG, Willick SE. Predictive validity of National Basketball Association Draft Combine on future performance. *J Strength Cond Res*. 2018;32:396–408.
- Ben Abdelkrim N, Castagna C, Jabri I, Battikh T, El Faza S, El Ati J. Activity profile and physiological requirements of junior elite basketball players in relation to aerobic-anaerobic fitness. *J Strength Cond Res*. 2010;24:2330–42.
- Boone J, Bourgois J. Morphological and physiological profile of elite basketball players in Belgian. *Int J Sports Physiol Perform*. 2013;8:630–8.
- Cormery B, Marcil M, Bouvard M. Rule change incidence on physiological characteristics of elite basketball players: a 10-year-period investigation. *Br J Sports Med*. 2008;42:25–30.
- Narazaki K, Berg K, Stergiou N, Chen B. Physiological demands of competitive basketball. *Scand J Med Sci Sports*. 2009;19:425–32.
- Ostojic SM, Mazic S, Dikic N. Profiling in basketball: physical and physiological characteristics of elite players. *J Strength Cond Res*. 2006;20:740–4.
- Köklü Y, Alemdaroğlu U, Koçak FÜ, Erol AE, Fındıkoğlu G. Comparison of chosen physical fitness characteristics of Turkish professional basketball players by division and playing position. *J Hum Kinet*. 2011;30:99–106.
- Pojskic H, Šeparović V, Užičanin E, Muratović M, Mačković S. Positional role differences in the aerobic and anaerobic power of elite basketball players. *J Hum Kinet*. 2015;49:219–27.
- Smith HK, Thomas SG. Physiological characteristics of elite female basketball players. *Can J Appl Sport Sci*. 1991;16:289–95.
- Riezebos ML, Paterson DH, Hall CR, Yuhasz MS. Relationship of selected variables to performance in women's basketball. *Can J Appl Sport Sci*. 1983;8:34–40.
- Hoffman JR. *Norms for fitness, performance, and health*. Champaign, IL: Human Kinetics; 2006.
- Gonzalez AM, Hoffman JR, Rogowski JP, Burgos W, Manalo E, Weise K, Fragala MS, Stout JR. Performance changes in NBA basketball players vary in starters vs nonstarters over a competitive season. *J Strength Cond Res*. 2013;27:611–5.
- Gonzalez AM, Hoffman JR, Scallin-Perez JR, Stout JR, Fragala MS. Performance changes in National Collegiate Athletic Association Division I women basketball players during a competitive season: starters vs nonstarters. *J Strength Cond Res*. 2012;26:3197–203.
- Spiteri T, Binetti M, Scanlan AT, Dalbo VJ, Dolci F, Specos C. Physical determinants of division I collegiate basketball, Women's National Basketball League, and Women's National Basketball Association Athletes: with reference to lower-body sidedness. *J Strength Cond Res*. 2019;33:159–66.
- Delextrat A, Cohen D. Strength, power, speed, and agility of women basketball players according to playing position. *J Strength Cond Res*. 2009;23:1974–81.

21. Latin RW, Berg K, Baechle T. Physical and performance characteristics of NCAA division I male basketball players. *J Strength Cond Res.* 1994;8:214–8.
22. Townsend JR, Bender D, Vantrease W, Hudy J, Huet K, Williamson C, Bechke E, Serafini P, Mangine GT. Isometric mid-thigh pull performance is associated with athletic performance and sprinting kinetics in division I men and women's basketball players. *J Strength Cond Res.* 2019;33:2665–573.
23. Brzycki M. Strength testing—predicting a one-rep max from reps-to-fatigue. *J Phys Ed Rec Dance.* 1993;64:88–90.
24. Torres-Unda J, Zarrazquin I, Gil J, Ruiz F, Irazusta A, Kortajarena M, Seco J, Irazusta J. Anthropometric, physiological and maturational characteristics in selected elite and non-elite male adolescent basketball players. *J Sports Sci.* 2013;31:196–203.
25. Calleja González J, Mielgo Ayuso J, Lekue JA, Leibar X, Erauzkin J, Jukic I, Ostojic SM, Ponce González JG, Fuentes Azpiroz M, Terrados N. Anthropometry and performance of top youth international male basketball players in Spanish national academy. *Nutr Hosp.* 2018;35:1331–9.
26. Pehar M, Sisic N, Sekulic D, Coh M, Uljevic O, Spasic M, Krolo A, Idrizovic K. Analyzing the relationship between anthropometric and motor indices with basketball specific pre-planned and non-planned agility performances. *J Sports Med Phys Fitness.* 2018;58:1037–44.
27. Fields JB, Merrigan JJ, White JB, Jones MT. Seasonal and longitudinal changes in body composition by sport-position in NCAA division I basketball athletes. *Sports (Basel).* 2018;6:E85.



Activity and Physiological Demands During Basketball Game Play

2

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Basketball is an intermittent team sport with frequent transitions between activities performed at low, moderate and high intensities [1]. Movements completed during basketball gameplay differ with respect to the structure, intensity, distance, frequency and duration at which they are performed. In particular, pronounced changes in movements occur when transitioning between defence and offence [2]. Furthermore, the given movement patterns during basketball gameplay are punctuated with multidirectional running and shuffling bouts, intertwined with frequent jumps occurring almost every minute [3, 4], which is more frequent than most other team sports. Given the novel demands encountered during basketball games, many studies

have quantified the activities and physiological demands encountered to better understand the requirements imposed on players.

It has been highlighted that the movement profiles and physiological demands encountered during basketball games have been augmented following changes in game rules [2]. More precisely, FIBA rule changes in 2000 encompassed shortened offence time after securing possession of the ball from 30 to 24 s and reduced time to advance the ball past the half-court after securing possession from 10 to 8 s, which have promoted higher intermittent demands than before the change in rules [5]. Similarly, collegiate (NCAA) rule changes involved reducing the shot clock from 45 to 35 s in 1993 and then to 30 s in 2015. For this reason, caution should be taken when comparing data concerning the activity and physiological demands encountered by players during games before and after these rule changes in specific competitions.

In addition to rule changes, it is important to consider the methodologies used to quantify game demands in basketball. Original time–motion analyses [3, 5, 6] were based on topographic methods involving drawing of player motion on the court using cartographic coordinate maps to determine the distances travelled. Despite the provision of useful information in early basketball analyses, the cartographic method is based on the subjective, visual interpretation of the researchers, which increases the risk of errors in quantify-

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ing the activity demands in basketball [7]. Since this time, technological advancements have been made to increase the accuracy and objectivity of measurement regarding player activity during games [2]. Semi-automated and automated video tracking systems, radio-wave technology, and microsensors housing accelerometers and other inertial sensors have been extensively used in modern research to assess player activity during basketball games.

While the activities performed during basketball have been quantified using various methodologies, the physiological responses from players have predominantly been quantified using blood lactate concentration and heart rate measures [3, 5, 6, 8–20]. Blood lactate concentration provides insight into the recruitment of rapid glycolysis for energy provision [5], while heart rate is an indirect indicator of oxidative metabolism [16]. Therefore, this chapter will consider the evidence quantifying the activity demands along with the physiological responses in basketball to provide a complete picture of the movement profiles and underpinning internal stress typical of gameplay.

2.1 Activity Demands

2.1.1 Distance Covered

The distance covered during basketball gameplay represents an important parameter for coaches and performance staff to develop specific training programmes to optimally prepare players for the demands of gameplay [21]. Aside from total distance, it is also necessary to quantify the distance covered completing different types of activities performed at different intensities (Fig. 2.1) [2]. A recent systematic review showed studies on this topic have reported that players cover between 4.40 and 7.56 km during basketball games across various competitions and playing levels [2]. However, despite this wide range in data, the average distance identified in the majority of studies is between 5 and 6 km during games lasting 40 min [2].

Examining player responses during total playing time, inclusive of the inherent recovery

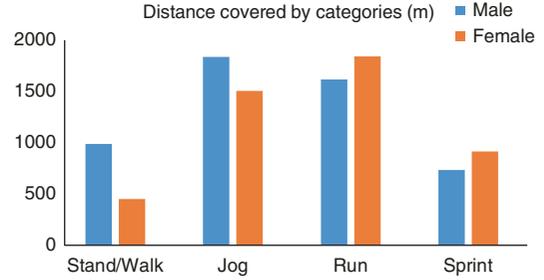


Fig. 2.1 Distance covered performing various types of activity during total game time in male and female basketball players [2]

opportunities available to players across games, is equally important in the development of ecologically valid training plans and testing protocols. Some studies [10, 22] have analysed the live (during active periods of play) and total (during active periods of play and stoppages such as when the ball is out-of-bounds or during free-throws) distance covered together. Analyses show that greater total distance (7558 ± 575 m) is covered during total game time involving 1720 ± 143 m completed undertaking low-intensity activities (standing and walking) [10], compared to only live game time ($5140\text{--}6390$ m) with only $484\text{--}586$ m travelled at low intensities [4, 23] (Figs. 2.2 and 2.3).

Due to the differences in the time–motion analysis procedures adopted across studies and variations in individual playing time within and between teams, relative distance (m/min) measures allow for more accurate comparisons according to sex, playing level and seasonal phase. In this regard, both male and female basketball players, across various competitions and playing levels, cover ~ 130 m/min during live gameplay and ~ 110 m/min during total gameplay [2]. Variations between live and total relative game distance data emphasise the potential impact of stoppages on the recovery of players. Furthermore, relative distance data might reveal temporal changes in activity demands across game quarters that are not detected using absolute distance data. Specifically, systematic analysis [2] reveals significant reductions only in relative distance across game quarters, with fatigue-related mechanisms and tactical play potentially underpinning this trend.

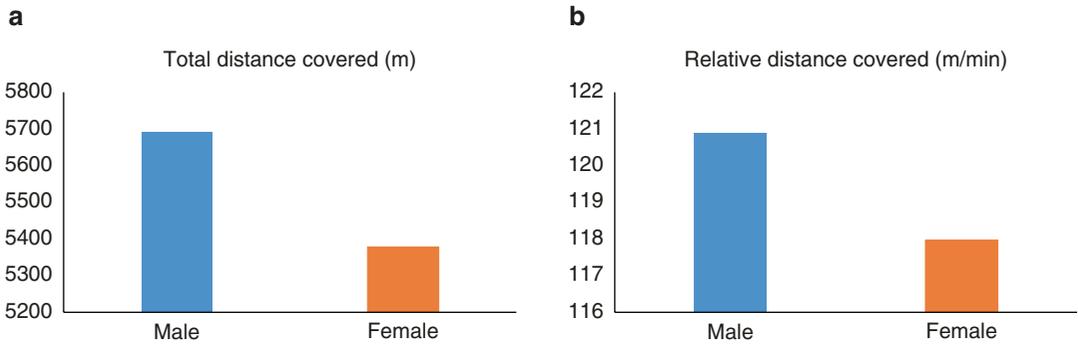


Fig. 2.2 Total absolute (a) and relative (b) distance covered during total game time in male and female basketball players [2]

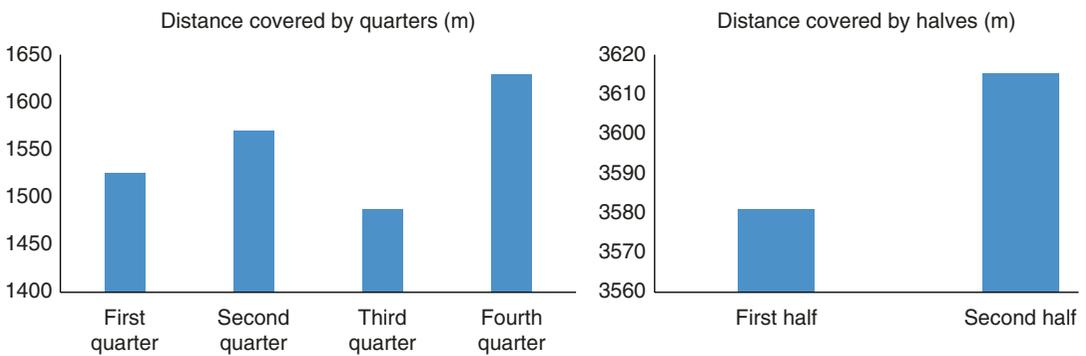


Fig. 2.3 Distance covered relative to playing period during total game time in basketball [2]

Existing analyses also indicate various differences in total distance covered during game-play between playing positions. It is evident that each playing position in basketball holds different role requirements, which appear to induce position-specific activity demands. For instance, Hůlka et al. [11] reported that national, male basketball guards (6635 ± 221 m) cover significantly greater distances during games compared to small forwards (6016 ± 448 m) and centres (5225 ± 659 m). On the contrary, Oba and Okuda [22] observed that point guards (5455 m) and centres (5433 m) demonstrated less overall distances than shooting guards (5566 m), small forwards (5681 m) and power forwards (5897 m) in female basketball players (standard deviation not reported). Furthermore, other analyses have also grouped players as backcourt (guards) or frontcourt (forwards and centres) positions [4, 8, 23]. These data indicate that professional, male, backcourt players (5288–6390 m) travel

greater distances compared to frontcourt players (4976–6230 m) [4, 8, 23]. Reasoning for the positional differences in distance data across studies is that during the transition phase up and down the court, shooting guards and forwards typically travel down the court positioned near the sidelines, increasing the distance travelled. Moreover, centres usually cover less distance during defensive passages being positioned close to the basket compared to other positions involving wider coverage of the court such as when guarding direct opponents trying to create space off the ball via cutting manoeuvres and screens. Conversely, the lower distances covered by point guards and centres reported by Oba and Okuda may relate to these positions transitioning up and down the middle of the court in more direct, linear movement paths [22].

Comparisons in the distance covered according to playing level show some clear differences when activity distance data are analysed according

to intensity. Despite similar total distances covered across different competition levels (e.g. high school vs. university [22], semi-professional vs. professional [4], and national vs. international [9]), pronounced differences were apparent for high-intensity activities [4, 22]. Specifically, professional male basketball players jogged (1.0–3.0 m/s) and ran (3.1–7.0 m/s) for significantly greater distances than semi-professional basketball players, whereas semi-professional basketball players covered larger distances sprinting (>7.1 m/s) than professional players [4]. Various factors may underpin differences in distances covered according to intensity categories between playing levels including team tactics, game structure, player fitness and preparedness, and playing style [4].

Differences in relative distance covered according to the geographical region of various competitions in Australia, Tunisia, Slovenia and Japan reveal some interesting trends that may also be related to the tactics and playing styles adopted by teams. Accordingly, semi-professional and professional male and female players in Australia (126–133 m/min) [4, 8, 23] cover larger relative distances than in other regions, such as professional male players in Slovenia (110 m/min) [24], high school, collegiate and national female players in Japan (93–101 m/min) [22], and junior Tunisian male players (115 m/min) [10]. As indicated earlier, longer games with more frequent stoppages reduce the relative distance covered, making it difficult to establish definitive conclusions given these factors varied across studies.

Despite only one study directly comparing distances covered during games between male and

female basketball players competing at the same playing level, preliminary results indicate no substantial differences in relative distance according to player sex (male, 110–132 m/min, vs. female, 125–136 m/min) [23]. More in-depth analysis of these data revealed that the semi-professional, Australian, male basketball players covered significantly greater distances dribbling the ball, while female players completed significantly larger running distances [23]. Due to the limited data available, basketball coaches and performance staff should interpret sex difference findings with caution when planning sex-specific training plans until more comprehensive evidence is available.

2.1.2 Activity Frequency

Activity frequency during basketball gameplay provides an indication of the intermittent activity profile encountered by players (Fig. 2.4). These data can be used by basketball coaches and performance staff to design precise training plans representative of actual game demands. On average, basketball players change activity types during games every 1–3 s, with the relative rate of changes in activity frequency varying between 21 and 57 movements/min [2] (Fig. 2.5). Activity frequencies have been shown to remain relatively consistent across game quarters (435 vs. 433 vs. 429 vs. 459 movement/min) as well as halves (864 vs. 886 movement/min) in national female basketball gameplay [8].

Basketball playing positions necessitate specific fitness attributes and skills which may yield

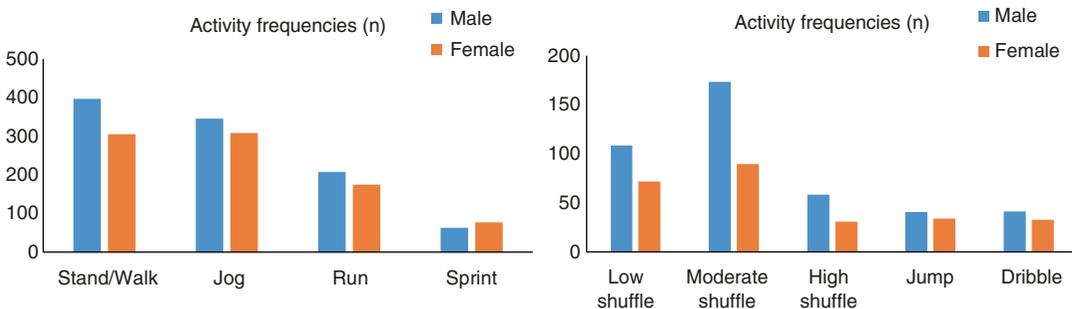


Fig. 2.4 The activity frequencies for various types of activities according to sex during total game time in basketball [2]

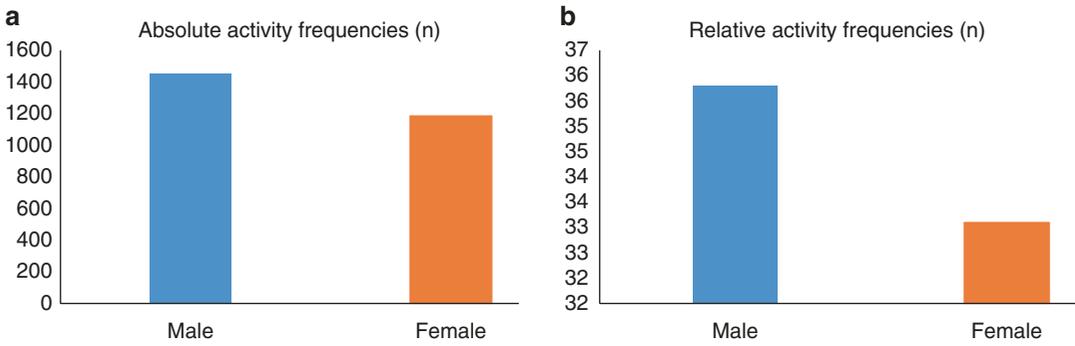


Fig. 2.5 Absolute (a) and relative (b) activity frequencies according to sex during total game time in basketball [2]

variations in the activity frequencies performed during gameplay. In this regard, evidence suggests that backcourt players perform more overall movements [3–5, 8], with significantly more dribbling bouts during games compared to frontcourt players (66 vs. 21) [8].

Comparisons between playing levels show that national male players complete 1000 ± 27 changes in activity types per game, while international players undertake significantly more movements (1105 ± 74) [9]. In contrast, non-significant differences were apparent between semi-professional and professional male players executing 41 movements/min and 57 movements/min, respectively [4]. Further evidence suggests that higher playing levels involve more frequent high-intensity activities such as sprints and high-intensity shuffles. Specifically, Abdelkrim et al. [9] reported that junior international, male, Tunisian basketball players performed significantly more high-intensity activities compared to national players. The greater intermittent demands evident at higher playing levels emphasise the importance of implementing training plans targeting acceleration and deceleration abilities for adequate player preparation to cope with game demands at the highest levels [25, 26].

Geographical comparisons also show some prominent differences between competitions played in various continents with a higher frequency of activities performed by basketball players in Australia [4, 8, 23] compared to players in Europe [13, 27–29] and Africa [3, 9]. The higher frequency of movements in Australian competition may be related to methodological

approaches in the studies conducted on this topic, given additional activities engaging upper-body and dribbling movements were included in the analyses of Australian players but not for other regions.

Differences in physical attributes (body mass, body height and arm span) and fitness characteristics (muscular strength, explosive power, speed, endurance and agility) between male and female basketball players suggest that differences in activity frequencies during games might exist between sexes [21]. Female basketball players [13, 27] demonstrate lower activity frequencies during games compared to male players [3, 5, 9]. The available data show that male basketball players perform 758–2749 movements per game, while female basketball players perform 576–1764 movements [2]. However, direct comparison between semi-professional male and female players at analogous playing levels showed greater dribbling contribution in male players (1.5 vs. 0.8 movements/min), while females performed more frequent running bouts (7.3 vs. 7.0 movements/min) [23].

The lack of clear standardisation procedures across studies likely exerts an important effect on the resultant relative activity frequency data reported across studies and should be acknowledged [23]. Different studies have adopted varied approaches in categorising activities to detect frequencies during games. For example, some studies have incorporated dribbling and activities engaging upper-body such as blocks, jumps and defensive tasks [4, 8, 23], while others have not included these categories and focused solely

on locomotive activity and jumps [2, 3, 5, 9, 12, 13, 27]. Furthermore, some studies categorise activities according to speed [4, 8, 23], whereas others categorise activity based on descriptive criteria [3, 5, 9, 12, 13, 27]. Moreover, the analytical approaches used to quantify the activity demands of players in some studies utilise frame-by-frame methods, which can be much more sensitive to the change of speed and thus detection of new activities (and frequencies) compared to semi-automated video tracking systems or the cartographical method [3, 5, 9, 12, 13, 27]. Therefore, indirect comparisons across studies possess notable limitations, thus highlighting the need to adopt a consensus in the methodological approaches used in determining activity frequency in future research.

2.1.3 Activity Duration

During basketball games, players undertake various types of activities for different proportions of playing time. For instance, 23–66% of playing time is spent standing/walking, 6–36% jogging, 5–33% running, 0–9% sprinting, 2–15% shuffling at low intensities, 7–20% shuffling at moderate intensities, 0–9% shuffling at high intensities, 1–2% jumping and 1–11% dribbling [2]. Despite low-intensity activities occupying the greatest proportion of playing time during basketball games, numerous situations occur

relying on high-intensity movement passages (Fig. 2.6) [9].

Particular attention should be given to positional differences in activity duration data during basketball gameplay, given male backcourt players (sprint: 5.9%; high-intense shuffle: 9.3%) have been shown to spend more time sprinting and performing high-intensity shuffling compared to frontcourt players (sprint: forwards 5.4%, centres 4.5%; high-intense shuffle: forwards 9.2%, centres 7.9%) [3]. Also, female backcourt players spend less time (28.4%) undergoing standing and walking compared to centres (31.8%) [8]. Physical attributes such as height and body mass, as well as reduced aerobic fitness typical of players occupying the centre position compared to other positions, may contribute to the greater proportion of playing time spent engaged in low-intensity activities for these players [30, 31]. Furthermore, temporal comparisons show reduced variability in the proportion of playing time performing high-intensity activities across quarters in male semi-professional players compared to professional players [32]. This finding might have been underpinned by the greater stoppage durations during games and longer periods of low-intensity activities in semi-professional players, enabling greater recovery and maintenance of high-intensity activities.

To date, consistency in findings regarding the proportion of time spent engaged in different activities across different playing levels is lack-

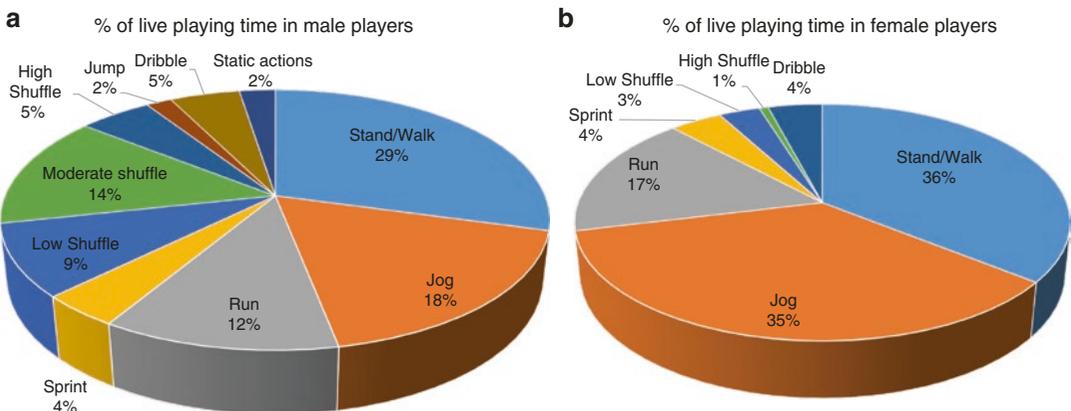


Fig. 2.6 Average activity duration (%) for various types of activities in male (a) and female (b) players during total game in basketball [2]

ing. For instance, some authors have found that high-intensity activities are more expressed in male international players than national players (sprint: 6.0 vs. 4.9%; high-intense shuffle: 9.3 vs. 8.1%) [9], whereas other data indicate that male semi-professional players spend more time sprinting compared to professional players (4.0 vs. 0.4%) [4]. Reasons for variations in findings might relate to the relative discrepancies in the physical preparedness of players across playing levels that are directly compared. Furthermore, discrepancies in decision-making and technical abilities across comparative groups may also underpin the varied findings, given better control of the ball reduces turnovers and subsequently the number of high-intensity transitions up and down the court [4].

While the wide range of activity categories implemented across studies make comparisons difficult, recent systematic evidence [2] demonstrated that the proportion of time spent standing, walking, and jogging is similar regardless of geographical regions. In contrast, the proportion of playing time spent sprinting and engaging in other high-intensity activities differs between geographic regions [2]. Specifically, basketball players in Tunisia execute more sprints and high-intensity shuffle activities than players competing in Australian and European competitions.

Despite different total game durations between many male and female basketball competitions, similarity in the proportions of playing time spent performing various types of activities is apparent. Specifically, standing, walking and running occupy similar proportions of playing time in male and female basketball players irrespective of playing level [6, 23]. However, male players spend significantly more time dribbling than female players (5% vs. 4%) [2].

The proportion of activities performed at lower intensities, especially standing and walking [33], represents important passages in basketball games, given they enable recovery following more intense periods [34]. In this regard, passive recovery is recommended as the most efficient method for restoring myoglobin and haemoglobin oxygen stores, and improving the ability to execute intermittent activity [35]. Indeed, fluctua-

tions in the activity demands across game-play reflect less performance of high-intensity movements and increased dribbling as games approach full time [32]. For this reason, coaches are encouraged to strategically provide recovery opportunities such as substitutions, deliberate fouling and timeouts for the maintenance of high-intensity activities in latter key stages of games. Inconsistencies in the methodological approaches used to quantify activity durations across studies should also be considered when interpreting the existing data, given some studies included additional activities such as sideways movement, striding and static actions [9, 10], while others disregarded movement intensity when quantifying dribbling activity [4, 8, 23]. Moreover, the wide range in thresholds to demarcate activity categories according to speed directly impacts the amount of playing time performing different movement types. For example, some authors combine running and moderate speed running as one category [4, 8, 24], while others use separate categories for these measures becoming more sensitive to detect changes in speed [2, 9].

2.2 Physiological Demands

2.2.1 Blood Lactate Concentration

Blood lactate concentration is frequently used to assess the physiological responses of players during games as it indicates energy provision from rapid glycolytic metabolic pathways [9, 13]. Average blood lactate concentration reported in basketball players during gameplay ranges between 3.2 and 6.8 mmol/L, with the mean blood lactate concentration slightly above 50% of the maximal response [2]. Temporal trends across games reveal that blood lactate concentration decreases in the second half compared to the first half (Fig. 2.7) [13, 14, 36]. The reductions in blood lactate concentration across game periods may be related to a slower game pace due to increased importance being placed on each possession and more frequent stoppages being encountered enabling greater lactate clearance [2].

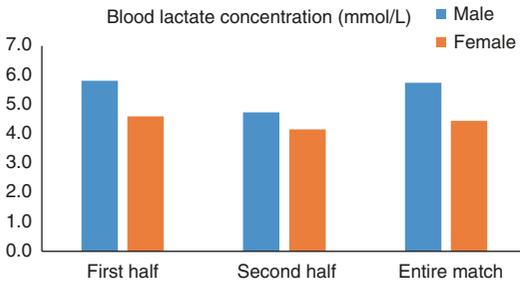


Fig. 2.7 The blood lactate concentration responses to total basketball game time relative to playing halves [2]

Considering backcourt players spend more time engaging in high-intensity activities compared to frontcourt players during games, the higher blood lactate concentrations reported in male (6.4 vs. 4.9 mmol/L) and female (5.7 vs. 4.0 mmol/L) players occupying guard positions compared to other positions may be expected [3, 14]. Likewise, the increased frequency of activities during games at higher playing levels may contribute to the higher blood lactate concentrations evident in international male players compared to national players (6.1 vs. 5.0 mmol/L) [9]. In contrast, similar blood lactate concentrations were reported between international and national female players (5.0 vs. 5.2 mmol/L) [14], which might be due to the lower activity frequencies executed during female gameplay compared to male gameplay [13, 27] nullifying differences in lactate production due to repeated changes in movement intensities.

Interestingly, the highest mean blood lactate concentration reported in basketball players (6.8 ± 2.8 mmol/L) was observed in Australian male players prior to the aforementioned rule changes when total activity distance, frequency and duration were lower compared to modern competitions [2]. In contrast, a recent study [8] reported the lowest blood lactate concentrations in Australian, female basketball players (3.7 ± 1.5 mmol/L). In addition to these data reported for players competing in Australian basketball competitions, male Tunisian (5.0–5.8 mmol/L) and female European players (4.2–5.7 mmol/L) showed more consistent blood lactate concentrations during gameplay [2].

Despite the higher variability among the players in various positions, gender comparisons sug-

gest male and female players experience similar blood lactate concentrations during basketball games. Specifically, average blood lactate concentrations range between 3.2 and 6.8 mmol/L in male players and between 3.2 and 5.7 mmol/L in female players during games.

The relatively high blood lactate concentrations observed in analysed studies indicate an important anaerobic metabolic contribution and a necessity for training prescription aimed at building rapid glycolytic capacity such as high-intensity interval training, shuttle running and repeated-sprint drills. However, it should be noted that development of aerobic capacity during training also plays an important role for energy provision during various game activities, maximising lactate clearance during recovery periods [37], and assisting phosphocreatine regeneration [38]. For these reasons, training plans should stress anaerobic and aerobic pathways for optimal metabolic preparation in players to cope with the array of physiological stress encountered during games. However, the existing basketball literature relies on opportune measurement of blood lactate concentration across games, which should be considered when interpreting the reported findings since blood measures appear unrelated to muscle lactate concentrations [39], whereby lactate clearance is higher in muscles during recovery [39, 40]. Furthermore, the different activity profiles performed directly before blood sampling greatly impact the obtained results and likely create variations in the concentrations reported across players.

2.2.2 Heart Rate

Heart rate (HR) is one of the most commonly implemented parameters for monitoring physiological stress during basketball games. Measurement of player HR responses can serve multiple purposes including training prescription, identifying adaptations in fitness and detecting an intolerance of demands indicative of non-functional overreaching [16]. Furthermore, real-time HR data can inform tactical decisions during games in optimising use of timeouts, substitutions

or deliberate stoppages (e.g. fouls) to amplify recovery opportunity in players [41].

Average HR varies between 132 and 165 beats/min during total game time and between 161 and 186 beats/min during live game time in male and female basketball players [2]. Conversely, average relative HR ranges between 67 and 89% of HR_{max} during total game time and between 82% and 95% of HR_{max} during live game time, indicate that significant cardiovascular stress is placed on players during games [2]. It is also important to determine the percentage of time spent working in different HR intensity zones. It is considered that 85% of HR_{max} is a useful threshold to identify high-intensity activities. In this regard, both male and female players spend ~75% of live playing time in activities eliciting HR responses $\geq 85\%$ of HR_{max} [2]. Across games, HR is significantly lower in the second half than in the first half [12, 15] and in the last quarter when compared to other quarters [3], showing a reduction in cardiovascular intensity with game progression.

Positional analyses show that the highest HR values are recorded in male and female backcourt players compared to frontcourt players in most instances [3, 8, 11, 14–16]. Despite the fact forwards spend more time engaging in high-intensity activities than centres during games, equivalent HR responses have been reported during games across these playing positions [3, 16]. Similar HR between forwards and centres may relate to the higher static movement demands of centres elevating HR throughout games such as when screening, blocking and maintaining inside position against opponents. Accordingly, intense static activities should be included when quantifying activity demands in players where possible since they likely contribute additional physiological stress in players during games.

Similar to positional analyses, HR discriminates players competing at different playing levels [8]. Specifically, international players have demonstrated higher average relative HR ($95 \pm 2\%$ of HR_{max}) compared to national players ($91 \pm 2\%$ of HR_{max}) [2]. International competition also elicited a greater proportion of time spent working at $\geq 85\%$ of HR_{max} in comparison to national competition (international: $77 \pm 4\%$;

national: $70 \pm 6\%$) [9]. These data likely reflect a greater continuity in activity with less frequent intermittent spikes in intensity at lower playing levels [9]. Moreover, comparisons across separate studies show that average HR in professional, female basketball players ranges between 91% and 95% of HR_{max} [14], which is notably higher than semi-professional, female players (82–89% of HR_{max}) [8].

Comparisons between sexes show that average HR is similar in male (84–94% of HR_{max}) and female basketball players (82–95% of HR_{max}) during games, indicating that a similar cardiovascular stress is encountered [2]. However, there is no study directly comparing HR response during match play in male and female basketball players at an equivalent playing level in the same geographical region. Therefore, the similarities in average HR between sexes should be further investigated directly in comparable male and female players in future research. Similar to sex comparisons, HR data across studies show that similar average responses are evident during basketball games played in different geographical regions [2]. Moreover, the proportion of live playing time spent at $\geq 85\%$ HR_{max} is alike in Europe (75%), Australia (75%), and Tunisia (74%) [2], demonstrating similarity in terms of the cardiovascular intensity attained in competitive basketball across the world.

Various factors have been shown to directly affect HR responses during basketball game-play including the initial fitness status of players, intensity of the game, team tactics, playing position, playing level and playing time [2]. In addition, many indirect factors may also affect HR and require consideration such as the nutritional status and hydration of players, psychological states and environmental aspects such as spectator presence and temperature [14]. Team tactics may explain transient changes in HR responses across game periods with increased timeouts and stoppages (e.g. fouls) typically occurring in latter game stages providing players with additional recovery and reducing HR responses. Furthermore, team tactics in the form of substitutions patterns may also impact HR, with substitutions lowering the HR response across games for

example. Even though low- to moderate-intensity activities predominate in basketball games, periodic bursts of high-intensity activities increase HR, which subsequently remains elevated for substantial periods [5]. Moreover, additional activities engaging the upper body such as throws and jumps and maintaining physical resistance against opponents [3, 5], as well as extended demands associated with the changes in activity speed and direction [42], can also exacerbate HR responses during games.

Fact Box

- While low-intensity activities occupy the greatest proportion of playing time during basketball games, frequent situations that rely on high-intensity movement passages also occur.
- Players occupying guard positions and competing at higher playing levels sustain greater activity and physiological workloads than players competing in other playing positions and at lower playing levels.
- Reductions in the proportion of high-intensity activity with game progression likely occur due to tactical strategies as well as fatigue-related mechanisms.

Take Home Message

- Different categorisation of playing positions, activity types and activity intensities across studies makes comparisons difficult and limits the ability to draw definitive conclusions regarding specific activity and physiological demands during basketball gameplay. Therefore, future work examining game demands in basketball needs to follow standardised approaches, especially regarding analysis of player activity.

- Most of the playing time (~75%) during basketball games is spent performing activities eliciting HR responses $\geq 85\%$ of HR_{max} in male and female players.
- Substitutions, deliberate fouling and timeouts may be effectively used to increase stoppage time in the later key stages of games for the maintenance of high-intensity activities.

References

1. McKeag DB. Handbook of sports medicine and science, basketball. Oxford, UK: Wiley; 2008.
2. Stojanovic E, Stojiljkovic N, Scanlan AT, Dalbo VJ, Berkelmans DM, Milanovic Z. The activity demands and physiological responses encountered during basketball match-play: a systematic review. *Sports Med.* 2018;48(1):111–35.
3. Abdelkrim NB, El Fazaa S, El Ati J. Time–motion analysis and physiological data of elite under-19-year-old basketball players during competition. *Br J Sports Med.* 2007;41(2):69–75.
4. Scanlan A, Dascombe B, Reaburn P. A comparison of the activity demands of elite and sub-elite Australian men’s basketball competition. *J Sports Sci.* 2011;29(11):1153–60.
5. McInnes S, Carlson J, Jones C, McKenna MJ. The physiological load imposed on basketball players during competition. *J Sports Sci.* 1995;13(5):387–97.
6. Narazaki K, Berg K, Stergiou N, Chen B. Physiological demands of competitive basketball. *Scand J Med Sci Sports.* 2009;19(3):425–32.
7. Hulka K, Cuberek R, Svoboda Z. Time–motion analysis of basketball players: a reliability assessment of Video Manual Motion Tracker 1.0 software. *J Sports Sci.* 2014;32(1):53–9.
8. Scanlan AT, Dascombe BJ, Reaburn P, Dalbo VJ. The physiological and activity demands experienced by Australian female basketball players during competition. *J Sci Med Sport.* 2012;15(4):341–7.
9. Abdelkrim NB, Castagna C, El Fazaa S, El Ati J. The effect of players’ standard and tactical strategy on game demands in men’s basketball. *J Strength Cond Res.* 2010;24(10):2652–62.
10. Abdelkrim NB, Castagna C, Jabri I, Battikh T, El Fazaa S, El Ati J. Activity profile and physiological requirements of junior elite basketball players in relation to aerobic-anaerobic fitness. *J Strength Cond Res.* 2010;24(9):2330–42.
11. Hůlka K, Cuberek R, Bělka J. Heart rate and time-motion analyses in top junior players during basketball matches. *Acta Gymnica.* 2013;43(3):27–35.

12. Klusemann MJ, Pyne DB, Hopkins WG, Drinkwater EJ. Activity profiles and demands of seasonal and tournament basketball competition. *Int J Sports Physiol Perform.* 2013;8(6):623–9.
13. Matthew D, Delextrat A. Heart rate, blood lactate concentration, and time–motion analysis of female basketball players during competition. *J Sports Sci.* 2009;27(8):813–21.
14. Rodriguez-Alonso M, Fernandez-Garcia B, Perez-Landaluce J, Terrados N. Blood lactate and heart rate during national and international women’s basketball. *J Sports Med Phys Fitness.* 2003;43(4):432.
15. Abdelkrim NB, Castagna C, El Fazaia S, Tabka Z, El Ati J. Blood metabolites during basketball competitions. *J Strength Cond Res.* 2009;23(3):765–73.
16. Vaquera A, Refoyo I, Villa J, et al. Heart rate response to game-play in professional basketball players. *J Hum Sport Exerc.* 2008;3(1). <https://doi.org/10.4100/jhse.2008.31.01>
17. Montgomery PG, Pyne DB, Minahan CL. The physical and physiological demands of basketball training and competition. *Int J Sports Physiol Perform.* 2010;5(1):75–86.
18. Vencúrik T. Differences in intensity of game load between senior and U19 female basketball players. *J Hum Sport Exerc.* 2014;9(1). <https://doi.org/10.14198/jhse.2014.9.Proc1.28>
19. Vencúrik T, Nykodým J. The intensity of load experienced by female basketball players during competitive games. *Int J Med Health Biomed Bioeng Pharm Eng.* 2015;9(7):565–8.
20. Vencúrik T, Nykodým J, Struhár I. Heart rate response to game load of U19 female basketball players. *J Hum Sport Exerc.* 2015;10(1). <https://doi.org/10.14198/jhse.2015.10.Proc1.33>
21. Ziv G, Lidor R. Physical attributes, physiological characteristics, on-court performances and nutritional strategies of female and male basketball players. *Sports Med.* 2009;39(7):547–68.
22. Oba W, Okuda T. A cross-sectional comparative study of movement distances and speed of the players and a ball in basketball game. *Int J Sport Health Sci.* 2008;6:203–12.
23. Scanlan AT, Dascombe BJ, Kidcaff AP, Peucker JL, Dalbo VJ. Gender-specific activity demands experienced during semiprofessional basketball game play. *Int J Sports Physiol Perform.* 2015;10(5):618–25.
24. Erčulj F, Dežman B, Vučković G, Perš J, Perše M, Kristan M. An analysis of basketball players’ movements in the Slovenian basketball league play-offs using the SAGIT tracking system. *Facta Universitatis.* 2008;6:75–84.
25. MacDougall JD, Hicks AL, MacDonald JR, McKelvie RS, Green HJ, Smith KM. Muscle performance and enzymatic adaptations to sprint interval training. *J Appl Physiol.* 1998;84(6):2138–42.
26. Rodas G, Ventura JL, Cadefau JA, Cussó R, Parra J. A short training programme for the rapid improvement of both aerobic and anaerobic metabolism. *Eur J Appl Physiol.* 2000;82(5–6):480–6.
27. Conte D, Favero TG, Lupo C, Francioni FM, Capranica L, Tessitore A. Time-motion analysis of Italian elite women’s basketball games: individual and team analyses. *J Strength Cond Res.* 2015;29(1):144–50.
28. Delextrat A, Badiella A, Saavedra V, Matthew D, Schelling X, Torres-Ronda L. Match activity demands of elite Spanish female basketball players by playing position. *Int J Perform Anal Sport.* 2015;15(2):687–703.
29. Delextrat A, Baliqi F, Clarke N. Repeated sprint ability and stride kinematics are altered following an official match in national level basketball players. *J Sports Med Phys Fitness.* 2013;53:112–8.
30. Jeličić M, Sekulić D, Marinović M. Anthropometric characteristics of high level European junior basketball players. *Coll Antropol.* 2002;26:69–76.
31. Sallet P, Perrier D, Ferret J, Vitelli V, Baverel G. Physiological differences in professional basketball players as a function of playing position and level of play. *J Sports Med Phys Fitness.* 2005;45(3):291.
32. Scanlan AT, Tucker PS, Dascombe BJ, Berkelmans DM, Hiskens MI, Dalbo VJ. Fluctuations in activity demands across game quarters in professional and semiprofessional male basketball. *J Strength Cond Res.* 2015;29(11):3006–15.
33. Castagna C, Abt G, Manzi V, Annino G, Padua E, D’ottavio S. Effect of recovery mode on repeated sprint ability in young basketball players. *J Strength Cond Res.* 2008;22(3):923–9.
34. Piiper J, Spiller P. Repayment of O₂ debt and resynthesis of high-energy phosphates in gastrocnemius muscle of the dog. *J Appl Physiol.* 1970;28(5):657–62.
35. Dupont G, Blondel N, Berthoin S. Performance for short intermittent runs: active recovery vs passive recovery. *Eur J Appl Physiol.* 2003;89(6):548–54.
36. Janeira M, Maia J. Game intensity in basketball. An interactionist view linking time-motion analysis, lactate concentration and heart rate. *Coach Sport Sci J.* 1998;3:26–30.
37. Balsom P, Gaitanos G, Ekblom B, Sjödin B. Reduced oxygen availability during high intensity intermittent exercise impairs performance. *Acta Physiol.* 1994;152(3):279–85.
38. Tomlin DL, Wenger HA. The relationship between aerobic fitness and recovery from high intensity intermittent exercise. *Sports Med.* 2001;31(1):1–11.
39. Krstrup P, Mohr M, Steensberg A, Bencke J, Kjær M, Bangsbo J. Muscle and blood metabolites during a soccer game: implications for sprint performance. *Med Sci Sports Exerc.* 2006;38(6):1165–74.
40. Bangsbo J, Mohr M, Krstrup P. Physical and metabolic demands of training and match-play in the elite football player. *J Sports Sci.* 2006;24(07):665–74.
41. Schönfelder M, Hinterseher G, Peter P, Spitzenpfel P. Scientific comparison of different online heart rate monitoring systems. *Int J Telemed Appl.* 2011;2011:6.
42. Reilly T. Energetics of high-intensity exercise (soccer) with particular reference to fatigue. *J Sports Sci.* 1997;15(3):257–63.



Endocrine Aspects in Performance and Recovery in Basketball

3

Alon Eliakim and Dan Nemet

3.1 Introduction

The efficiency of training depends on the exercise load as well as on the athlete's ability to tolerate it, and imbalance between the two may lead to under- or overtraining. Thus, in recent years, efforts are made to develop objective methods to determine the fine balance between training load and the athlete's tolerability. The endocrine system, by modulation of anabolic and catabolic processes, seems to play an important role in the physiologic adaptation to exercise training [1]. Interestingly, exercise is associated with remarkable simultaneous changes in anabolic (e.g., growth hormone (GH), insulin-like growth factor-1 (IGF-I), and testosterone) and catabolic hormones (e.g., cortisol) and inflammatory cytokines (e.g., interleukin-6 (IL-6)), and the exercise-related response of these markers can be used to gauge exercise load [2, 3]. Measurements of the ratio of testosterone to cortisol [4] as well as IGF-I and IL-6 levels [5] are used occasionally as indicators of the anabolic–catabolic balance in order to determine training strain and to quantify the effects

of training. Dominance of the anabolic response will ultimately lead to greater muscle mass and improved fitness, while prolonged domination of a catabolic response, in particular if combined with inadequate nutrition, may eventually lead to overtraining and injury. It is suggested therefore that the assessment of changes in these antagonistic mediators may assist in quantifying effects of different types of single and prolonged exercise training methods and recovery modalities.

Interestingly, the majority of previous studies were done in individual sports, and relatively few studies examined the changes in anabolic/catabolic hormones in elite team sports during a competitive season in “a real-life” setting. This chapter summarizes the current knowledge on the effect of team sports, and in particular basketball, on these markers and suggests possible ways to use these responses in assisting competitive team-sport athletes and coaches to better evaluate training load and optimize training.

3.2 The Anabolic–Catabolic Systemic-Local Training Model

Previous studies have suggested the hypothesis that a sudden imposition of a training program which is associated with substantial increase in energy expenditure leads initially to an increase in catabolic hormones and pro-inflammatory

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cytokines, and as a consequence, to decreases in IGF-I levels. Furthermore, if the training adaptation is successful, the pro-inflammatory cytokine levels drop, and with that decrease, the suppression of IGF-I diminishes, and an anabolic “rebound” ensues, leading to IGF-I level that exceeds the pretraining level [6]. Exactly how and when this transition/change occurs, and whether the initial catabolic-type stage is necessary for the ultimate anabolic training adaptation (“training effect”) remains unknown.

Interestingly, previous studies have also shown that despite the early decrease in circulating IGF-I levels, training may induce increases in muscle mass. This suggested that the effect of exercise on local muscle tissue growth factors differs from its systemic effects. Moreover, exercise-related increases in local muscle IGF-I occurred also when GH was inhibited [7, 8] emphasizing the GH independence of the “local” IGF-I anabolic adaptations to exercise. Simultaneous central catabolism and local anabolism may hold an advantage early in the adaptation to increased physical activity. This adaptive mechanism may reduce global anabolic function in order to conserve energy sources, but it still allows for local tissue growth in response to exercise training (Fig. 3.1). Consistent with this speculation is what occurs following intense exercise training

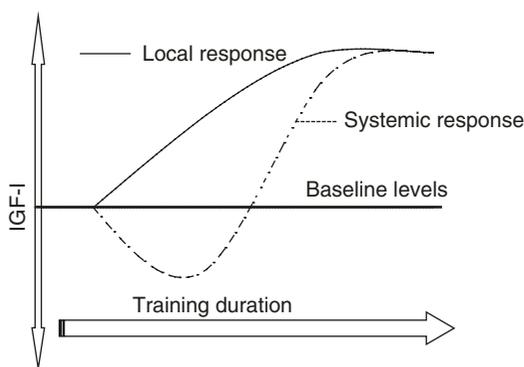


Fig. 3.1 Systemic and local adaptations of IGF-I to exercise training. Systemic IGF-I response is composed of an initial catabolic-type response with a decrease in IGF-I levels, which with proper training is followed by a rebound increase in IGF-I levels. On the other hand, muscle IGF-I usually increases from the early stages of training

in nutritionally, self-deprived, young elite athletes (e.g., female gymnasts [9]), where muscle adaptation occurs despite attenuated somatic growth and reduced circulating IGF-I.

3.2.1 Effect of Single Exercise

Basketball games and practices are characterized by numerous bursts of explosive activities, including jumping, turning, sudden/quick accelerating and decelerating, and short distance sprinting. During a typical match, players perform around 1000 different activity types with change in movement pattern every 2 s, around 100 short sprints (mean duration 1.7 s, every 20 s), between 40 and 65 jumps and around 60 direction changes [10, 11]. Previous studies have consistently suggested that success in basketball appears to be more dependent upon the player’s anaerobic rather than aerobic capabilities, and anaerobic fitness components (e.g., sprint time and jumping height) were found to best discriminate between the league standing of top and bottom teams [12]. Therefore, basketball coaches and players should concentrate on training methods that enhance these fitness components.

Nunes et al. [13] studied the effect of three different resistance training schemes (endurance, strength-hypertrophy, and power) on salivary cortisol and testosterone in female basketball players. Cortisol levels increased after all schemes but mainly following the strength-hypertrophy session, suggesting that high-volume resistance exercise schemes lead to higher metabolic demands and therefore to greater cortisol response. The authors suggested that changes in cortisol can be used as a tool to monitor training strain. There was no change in testosterone levels following resistance training among the female basketball players. Very few studies examined the effect of team sports training of testosterone levels in female athletes. Consistent with Nunes et al. findings [13], no significant changes in circulating testosterone and salivary testosterone levels were found in elite female players following an intense water-polo practice and handball match, respectively [14, 15]. In contrast, serum

testosterone levels were increased among adolescent male and female volleyball players following routine morning practice [16]. Interestingly, baseline and postexercise testosterone levels were significantly higher in males compared with females. However, training was associated with an increase in testosterone levels in both genders, and the response to training was not significantly different between genders. These results suggest that an increase in testosterone levels may play an important role in the anabolic response to exercise in female players as well. In contrast to male athletes, in which the source of the exercise-induced testosterone production is testicular, in female athletes, testosterone is produced mainly by the adrenal gland.

The effect of a single 90-min routine morning practice on salivary cortisol levels was studied among male adolescent basketball and volleyball players [17]. Cortisol levels decreased significantly in both groups representing probably cortisol diurnal variation (higher levels in early morning and decrease throughout the day onto the lowest levels at midnight), and emphasizing some of the interpretation complexity of the hormonal response to exercise.

3.2.2 Effect of Interval Training

Interval training is one of the most common training methods used in anaerobic and aerobic-type sports [18]. The intensity of interval training depends on the running distance (short versus long sprints), running speed (percent of maximal speed), the number of repetitions, and the length of the rest interval between runs. In addition, coaches and athletes very often change the interval training structure, for example, by using either constant running distances (e.g., 4×250 m), increasing distance sessions (e.g., 100–200–300–400 m), decreasing distance sessions (e.g., 400–300–200–100 m), or a combination of increasing–decreasing distance interval sessions (e.g., 100–200–300–200–100 m). While these structure differences may seem negligible, they may involve different physiological demands since in the increasing distance struc-

ture, metabolic demands (e.g., lactate levels) increase gradually and are highest toward the end of practice, while in the decreasing distance protocol, metabolic demands are higher from the start and throughout the session [19].

It was shown that a typical constant distance (4×250 m) interval training led to a significant increase in GH and testosterone levels in elite young national team-level male handball players [19]. Increases in GH and testosterone levels following a brief sprint interval practice suggest exercise-related anabolic adaptations. In addition, the interval training session was also shown to be associated with an increase in the pro-inflammatory marker interleukin-6 (IL-6), indicating its possible role in the postexercise muscle damage healing process and suggesting that anabolic, catabolic, and inflammatory markers may be used to assess interval training load as well. More recently the effect of increasing (100–200–300–400 m) and decreasing distance (400–300–200–100 m) sprint interval training protocols on the balance between anabolic, catabolic, and inflammatory mediators was also studied among handball players [19]. Both sprint interval training types led to a significant increase in lactate, GH, and IGF-I. Interestingly, lactate levels and GH area under the curve (representing total GH levels secreted) were significantly greater in the decreasing distance session. In contrast, rate of perceived exertion (RPE), a quantitative measure of activity/exercise intensity in sports, was higher in the increasing distance session. Thus, despite a similar overall running distance, running speed, and total resting period in the two interval training sessions, the decreasing distance interval was associated with a greater metabolic (lactate) and anabolic (GH) response. Interestingly, these greater metabolic and anabolic responses were not accompanied by an increase in RPE, suggesting that physiological and psychological responses to interval training do not necessarily correlate. When the players were asked to explain why the increasing distance training protocol was perceived as more intense, they replied that the fact that the longest and hardest run (400 m) was only at the end of the session was more difficult to tolerate. Coaches and athletes should be aware

of these differences, as well as the need for specific recovery adaptations after different types of interval training sessions. Differences in physiological and psychological responses to competitive sport training and their influence on the training course and recovery process should also be addressed.

In basketball, small sided games (SSG) are frequently used in training. SSG can be practiced also as interval training. The effect of different tactical tasks (offensive and defensive) and training regimens (short and long) SSG on cortisol and testosterone was previously studied. An increase in cortisol was found following all SSG sessions. However, testosterone increased following a defensive-focused long (3×4 min bouts with 2 min rest in between) practice and decreased following an offensive-focused short (6×2 min bouts with 1 min rest in between) session, suggesting that different regimens lead to different anabolic adaptations [20].

3.2.3 Endocrine Aspects in Recovery

The development of methods to enhance the recovery of elite athletes from intense training and/or competition has been a major target of athletes and their accompanying staff for many years. Quality sleep, for example, is considered essential for optimizing recovery from training. Melatonin (N-acetyl-5-methoxytryptamine) is an important factor in quality sleep. It is synthesized from serotonin in the pineal gland, with sleep being initiated when there is a concomitant rise in melatonin and a decline in body temperature [21].

The use of 30-min red-light whole body irradiation (Photobiomodulation) for 14 nights was shown to be associated with higher melatonin levels, improved sleep quality, and endurance performance among elite Chinese female basketball players [22].

Another widely used modality to treat sports-associated traumatic injuries and for recovery from training and competition that may cause some level of traumatic muscle injury is cryotherapy [23, 24]. However, evidence regarding the

effectiveness and appropriate guidelines for the use of cryotherapy are limited. The effect of cold ice pack application following a brief sprint interval training on the balance between anabolic, catabolic, and circulating pro and anti-inflammatory cytokines in 12 male, elite junior handball players [25]. The interval practice (4×250 m) was associated with a significant increase in GH and IL-6 levels. Local cold-pack application was associated with significant decreases in the anabolic factors IGF-I, and IGF binding protein-3 during recovery from exercise, supporting some clinical evidence of possible negative effects of cryotherapy on athletic performance [25]. These results, along with no clear effect on muscle damage or delayed onset muscle soreness (DOMS), may suggest that the use of cold packs should probably be reserved for traumatic injuries or possibly used in combination with active recovery and not with complete rest. However, this is an example of how exercise-induced changes in anabolic, catabolic, and inflammatory markers may be used to solve the puzzle of optimizing competitive training. Further studies are needed to explore the beneficial use of anabolic, catabolic, and inflammatory markers measurement in many other aspects of recovery from exercise.

3.2.4 Effect of Prolonged Training

Very few studies examined the effect of prolonged training on hormonal status in basketball players. Eight weeks of either continuous running (4.8 km, three times/week) or extensive interval training (4×1.2 km, 3 times/week) were associated with similar improvements in both aerobic and anaerobic power and with increases in GH and cortisol levels [26]. Miloski and collaborators examined the effect of training (5 weeks of intensified training followed by 3 weeks of tapering) on mood state profile and performance among two groups of adolescent basketball players that were differentiated by baseline testosterone levels [27]. There were no mood state differences between the groups; however, low testosterone players displayed a higher fatigue score and lower energy index score during training. Interestingly,

improvements in endurance and agility were not affected by pretraining testosterone levels, suggesting that training-associated changes in anabolic hormones may be more important for greater training effect than baseline testosterone levels.

Measurements of hormones can also assist athletes and coaches in the training preparation for selected competitions. The effect of 4 weeks of training on fitness, self-assessment physical conditioning scores, and circulating IGF-I was investigated in elite professional handball players [28] during their preparation for the junior world championships. Training consisted of 2 weeks of intense training followed by 2 weeks of relative tapering. Circulating IGF-I and physical conditioning scores decreased initially and returned to baseline levels at the end of training. There was a significant positive correlation between the changes in circulating IGF-I and self-assessment physical conditioning scores, suggesting that players' self-assessment might serve as a reliable tool when laboratory assistance is unavailable. Nunes et al. [29] studied the effect of 12 weeks training before an international championship among elite female basketball players. Training included two phases of gradual increase in training overload (weeks 4–6, and weeks 8–10) and two periods of tapering (week 7 and weeks 11–12). They found a significant improvement in physical performance parameters at the end of training prior to the championship. However, in contrast to the previous reported changes in IGF-I, no changes in salivary cortisol and testosterone levels were found.

Tapering down the training intensity prior to a competition is a well-known training methodology to help athletes achieve their best performance [30]. This strategy is indeed associated with a parallel increase in circulating IGF-I levels. Therefore, these measures may assist coaches and athletes in their training preparations. Interestingly, sports that do not plan their training for a specific targeted date, such as many team sports that train in the same relative intensity throughout a regular season (e.g., basketball and soccer), changes in IGF-I levels, and its major binding protein IGFBP-3, were not found [31].

In optimal conditions, during the tapering of training intensity, IGF-I level will increase above baseline levels and will be associated with improved performance; however, this does not always occur. Since IGF-I can be reduced by nutritional imbalance and weight loss, it is possible that a deliberate decrease in body weight in athletes who participate in weight category sports (e.g., judo and wrestling), or even in team-sport players prior to major tournaments, may prevent further increase in this anabolic hormone above baseline levels, and will be associated “only” with a significant return to baseline [6, 32]. This emphasizes the importance of proper nutritional consulting/counseling along the training season. Previous studies in athletes demonstrated training-associated negative correlation between circulating IGF-I and Ghrelin (also known as lenomorelin), a hormone that is secreted by the stomach and pancreas, and is known to stimulate hunger [33]. Moreover, decreases in Ghrelin and Leptin, both known to mediate energy balance, were found following 3-month preseason preparatory training in young female basketball and handball players [34]. These findings suggest that hormonal relationships play a mediating role in training-induced associated energy balance, appetite, body composition, and muscle performance changes.

Interestingly, despite decreases in *circulating* IGF-I and the ratio of testosterone to cortisol during periods of intense training, performance may still improve, as muscle mass increases [35–38]. This suggests that while changes in these measures are good indicators of an athlete's overall well-being and energy balance, they are not necessarily good indicators of an athlete's performance. Presumably, it is the local muscle levels of these hormones (in particular IGF-I), and their autocrine or paracrine secretion, that is more indicative of skeletal muscle performance [8, 39]. Tapering training intensity, however, was found to be associated with both increased IGF-I level and with further improvement of exercise performance in athletes [30, 40].

It is still unknown what should be the permitted decrease of IGF-I levels during periods of heavy training, or what should be the opti-

mal increase of this hormone during periods of tapering down and reduced training intensity. However, we believe that an inability to increase circulating IGF-I levels and/or the testosterone/cortisol ratio before the target competition should be an alarming sign for both the athlete and his/her coach that the athlete's preparedness is not optimal. Collection of baseline and training-related hormonal changes, along with a comparison to the hormonal response in previous seasons, and the knowledge and experience from past success may prove to be of significant importance as well (Fig. 3.2).

One of the most important aspects is the effect of training on the endocrine response to a single practice. The hormonal response to a typical 60 min volleyball practice was assessed before and after 7 weeks of training during the initial phase of the season in elite national team-level male and female players [40, 41]. In male players [41], training resulted in significantly greater GH increase along with significantly reduced IL-6 response to the same relative intensity volleyball practice. In female players [42], training resulted in significantly lower cortisol and IL-6 increase to the same relative intensity volleyball practice. These results suggest that during the

initial phases of the training season, as training progresses, along with the training-associated improvement in power, anaerobic and aerobic characteristics, part of the adaptation to training is that each practice becomes more anabolic and less catabolic/inflammatory. Therefore, hormonal measurements may assist athletes and their coaching staff in assessing training program adaptations throughout different stages of the competitive season.

Finally, serum IGF-I levels were found to be significantly higher among basketball players compared to racehorse jockeys [43]. Whether this finding represents better conditioning of basketball players or simply reflects higher IGF-I levels in taller individuals with a different body composition is unclear.

3.2.5 Effect of Basketball Match

Changes in cortisol levels were assessed before, at halftime and at the end of a basketball game among national elite junior male players [44]. Serum cortisol levels were elevated at the end of the first half and were still elevated at the end of the game.

Inflammatory responses and muscle damage indices following a match were compared between four popular elite level team sports (i.e., soccer, basketball, handball, and volleyball). Soccer produced the greatest increases in cortisol, IL-6, and in muscle damage indices, while volleyball showed the smallest increases compared to the other sports [45]. These findings suggest that basketball match play leads to a relatively moderate stressful and inflammatory response among popular ball sports at the elite level.

Arruda and colleagues [46] examined the influence of the competition venue (home vs. away) on hormonal responses in elite basketball players. Pre-match salivary testosterone levels were found to be higher when playing at home. Pre-match testosterone and cortisol levels and the percent change of these hormones during the match were significantly related to somatic anxiety in particular when playing at home.

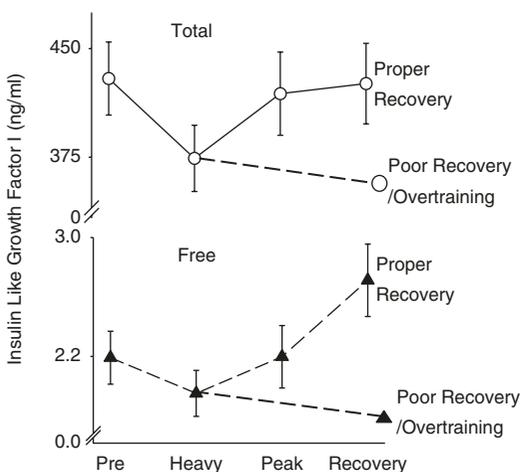


Fig. 3.2 The effect of a training season on total and free IGF-I circulating levels. Following an initial decrease, with proper recovery total IGF-I levels return to baseline, while free IGF-I rebounded reaching an above baseline level. With improper training recovery the IGF-I axis does not recover, leading to unfavorable results/overtraining

The authors concluded that competition playing venue affects player’s anxiety state and hormonal responses prior to elite basketball match, which might influence players’ performance and consequently match outcome. Consistent with that, higher cortisol levels were found prior to playing a difficult match compared to regular training and to playing an easy match [47]. These results suggest that playing against high-level opponent may lead to greater psychological stress, likely because the opponent level is perceived as threatening to the social status in a given hierarchy. In contrast, no differences in match-related cortisol and testosterone increases were found when playing against high-level opponent during a regular season winning game compared to a winning playoff match for the championship [48]. The results suggest that when playing against high-level opponent, players perceive their rival as a status stability threat, regardless of the competition stage.

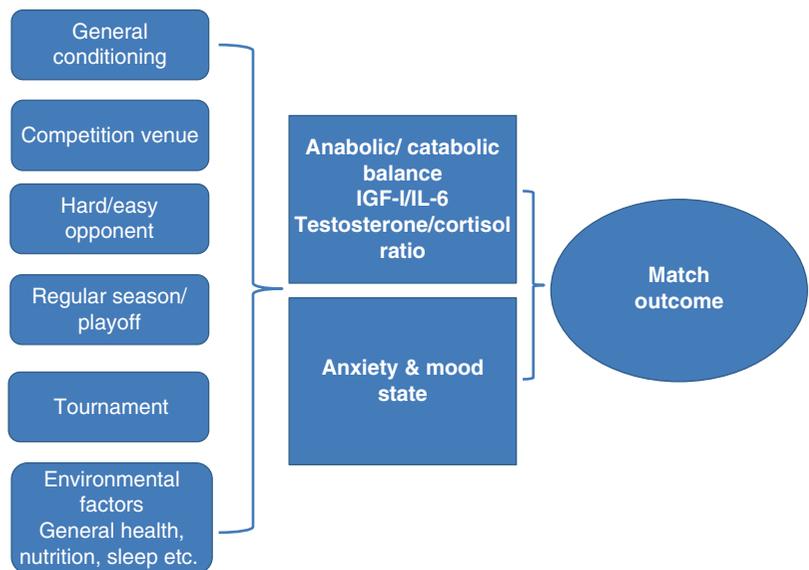
Circulating levels of testosterone and cortisol were measured during different phases in four consecutive seasons in elite professional Spanish league male players with special reference to playing time and players’ position [49]. Hormonal status was found to be playing-position dependent with “power forwards” (PFs) showing the lowest testosterone levels and “small

forwards” (SFs) showing the highest cortisol levels. Players who played 13–25 min per game had the highest testosterone levels and testosterone/cortisol ratio. Calendric analysis revealed that the most catabolic and stressful hormonal state (high cortisol and low testosterone/cortisol ratio) was during the months of March and April, which represented the last third of the regular season. Training staff should be aware that basketball players who play more than 25 min or less than 13 min may need specific training interventions to improve recovery processes or compensate for the lack of stimuli, respectively. In addition, PFs and SFs who play more than 25 min per game may be more susceptible to develop overtraining and even injuries during the last third of the competitive season.

These findings were consistent with a previous study [49] reporting that levels of free testosterone (the bioactive hormone) and free testosterone/cortisol ratio increased from October to December, but were significantly lower during March–April of the competitive season, also supporting the suggestion that players and the coaching staff should be more cautious during this period (Figs. 3.3 and 3.4).

Mental exertion also plays an important role in performance and should be addressed by the athlete and coaches during training and

Fig. 3.3 The exercise—IGF-I success model. Numerous factors are involved in achieving a favorable hormonal response and optimal performance



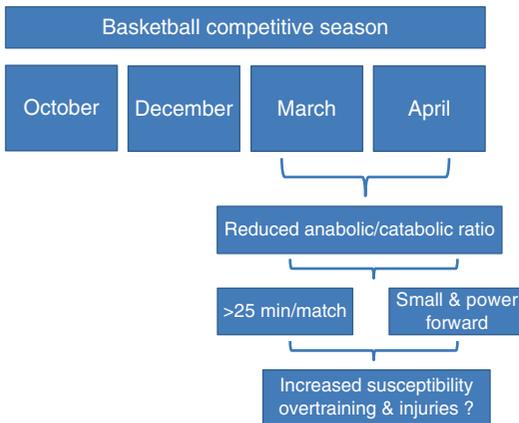


Fig. 3.4 Circulating levels of testosterone and cortisol measured along different phases of the training season suggesting that average match playing time (>25 min) and time of the season (March–April) are associated with catabolic-type hormonal profile which may increase player’s susceptibility to overtraining and injury

competition. Mental fatigue state was found to affect technical performance of basketball players and to modulate endocrine and autonomic responses [50].

Finally, 8 weeks of mindfulness training markedly attenuated the increase in cortisol levels associated with competition period in wheelchair basketball players [51]. Whether this or other methods of relaxation may apply for basketball players of different age groups and gender and at all competitive levels remains to be further studied.

3.2.6 Effect on Performance

Previous studies in Spanish top league basketball players have shown that centers developed greater positive mechanical impulse and achieved higher maximal instantaneous power during jumping and had higher testosterone levels compared to guards [52]. It was suggested that adequate levels of testosterone are a prerequisite for explosive performance but not for aerobic capacity in elite basketball players. A significant correlation was found between resting testosterone levels and vertical jump performance among elite sprinters, handball, volleyball, and soccer male

and female players [53]. The results indicated that testosterone levels can serve as a marker of jumping performance among female players as well, despite the significantly lower levels of testosterone compared with males (less than 10% of the men). In contrast, improvements in endurance and agility were not affected by pretraining testosterone levels in male adolescent basketball players [27], suggesting that during adolescence, a time of global growth, increased muscle mass, and anabolic spurt, other anabolic factors (e.g., GH and IGF) may compensate for the baseline differences of testosterone and lead to beneficial training effect.

Finally, previous studies have found that a higher social rank was associated with higher levels of IGF-I in both men and women, independent of a wide range of known confounders such as age, ethnicity, body weight, nutrition, and exercise [54]. Recently, Bogin et al. [55] studied high-level male and female competitive athletes from different university team sports (men: lacrosse, handball, rugby, and volleyball; women: football, rugby, netball, and volleyball) and assumed that what determines the social rank in this unique social network is the level of success in sports (and not the economic status). Therefore, the athletes were divided to “winners” and “losers.” The main finding of the study was that both pre- and post-competition IGF-I levels were about 11% higher among winners. There was no difference in the competition-related changes in IGF-I levels between the groups, suggesting that it is the baseline levels of IGF-I and not the change in IGF-I levels during the competition that may contribute to winning. This is the first study that relates IGF-I levels with winning. It seems that IGF-I levels integrate the multiple genetic, nutritional, social, and emotional influences to a coherent signal that regulates growth and possibly athletic performance. This suggests a novel cycle: both single practice and prolonged training increase IGF-I levels, which in turn increase the chances of an athlete to win (Fig. 3.5). However, future larger studies that analyze other types of team and individual sports, and that better control for nutritional, training, and doping status are needed to confirm this very interesting finding.

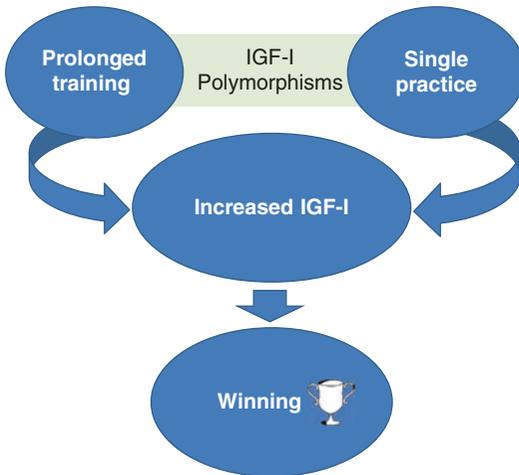


Fig. 3.5 The exercise–training–IGF-I cycle. With proper training, both single practice and prolonged training increase IGF-I levels, which in turn increases the chances of an athlete to win

3.3 Summary

Efforts to find objective parameters to quantify the balance between training load and the athlete's tolerance have been so far partially successful. The complexity of hormonal responses to different exercise types in a variety of sports and during different phases of the competitive season has become increasingly apparent in recent years. The evaluation of the hormonal response to exercise may be used as a helpful objective tool to assess the physical strain of training in team sports like basketball.

Take Home Messages

- The endocrine system, by modulation of anabolic and catabolic processes, seems to play an important role in the physiological adaptation to exercise.
- The magnitude of the hormonal response to exercise is influenced by the intensity and type of sport, the athletes' competitive level, and the timing along the competitive season.
- The balance between anabolic and catabolic responses to exercise may be used to gauge exercise load in team sports.

- Average match playing time (>25 min), playing role (small forwards and power forwards), and time of the season (March–April, last third) are associated with catabolic-type hormonal profile which may increase player's susceptibility to overtraining and injury.
- Following training, along with the training-associated improvement in power, anaerobic, and aerobic characteristics, a single practice becomes more anabolic and less catabolic and inflammatory.
- The anabolic adaptation to exercise may be influenced by recovery modalities (e.g., sleep and cryotherapy).
- A better anabolic profile (e.g., IGF-I and testosterone/cortisol ratio) may be associated with favorable match outcome in team sports like basketball.

References

1. Urhausen A, Kindermann W. The endocrine system in overtraining. In: Warren MP, Constantini NW, editors. *Sports endocrinology*. Totowa, NJ: Humana Press; 2000. p. 347–70.
2. Nemet D, Rose-Gottron CM, Mills PJ, Cooper DM. Effect of water polo practice on cytokines, growth mediators, and leukocytes in girls. *Med Sci Sports Exerc.* 2003;35(2):356–63.
3. Nemet D, Oh Y, Kim HS, Hill M, Cooper DM. Effect of intense exercise on inflammatory cytokines and growth mediators in adolescent boys. *Pediatrics.* 2002;110(4):681–9.
4. Hoffman JR, Falk B, Radom-Isaac S, Weinstein Y, Magazanik A, Wang Y, et al. The effect of environmental temperature on testosterone and cortisol responses to high intensity, intermittent exercise in humans. *Eur J Appl Physiol Occup Physiol.* 1997;75(1): 83–7.
5. Eliakim A, Nemet D, Cooper DM. Exercise, training and the GH-IGF-I axis, Chapter 13. In: Kraemer WJ, Rogol AD, editors. *The endocrine system in sports and exercise. The encyclopaedia of sports medicine*. 1st ed. Oxford, UK: Wiley-Blackwell; 2005. p. 165–79.
6. Nemet D, Pontello AM, Rose-Gottron C, Cooper DM. Cytokines and growth factors during and after a wrestling season in adolescent boys. *Med Sci Sports Exerc.* 2004;36(5):794–800.

7. DeVol DL, Rotwein P, Sadow JL, Novakofski J, Bechtel PJ. Activation of insulin-like growth factor gene expression during work-induced skeletal muscle growth. *Am J Phys.* 1990;259(1 Pt 1):E89–95.
8. Zancanato S, Moromisato DY, Moromisato MY, Woods J, Brasel JA, LeRoith D, et al. Effect of training and growth hormone suppression on insulin-like growth factor I mRNA in young rats. *J Appl Physiol.* 1994;76(5):2204–9.
9. Theintz GE, Howald H, Weiss U, Sizonenko PC. Evidence for a reduction of growth potential in adolescent female gymnasts. *J Pediatr.* 1993;122(2):306–13.
10. McInnes SE, Carlson JS, Jones CJ, McKenna MJ. The physiological load imposed on basketball players during competition. *J Sports Sci.* 1995;13(5):387–97.
11. Ostojic SM, Mazic S, Dikic N. Profiling in basketball: physical and physiological characteristics of elite players. *J Strength Cond Res.* 2006;20(4):740–4.
12. Angel Gomez M, Lorenzo A, Sampaio J, Ibanez SJ, Ortega E. Game-related statistics that discriminated winning and losing teams from the Spanish men's professional basketball teams. *Coll Antropol.* 2008;32(2):451–6.
13. Nunes JA, Crewther BT, Ugrinowitsch C, Tricoli V, Viveiros L, de Rose D Jr, et al. Salivary hormone and immune responses to three resistance exercise schemes in elite female athletes. *J Strength Cond Res.* 2011;25(8):2322–7.
14. Filaire E, Lac G. Dehydroepiandrosterone (DHEA) rather than testosterone shows saliva androgen responses to exercise in elite female handball players. *Int J Sports Med.* 2000;21(1):17–20.
15. Hale RW, Kosasa T, Krieger J, Pepper S. A marathon: the immediate effect on female runners' luteinizing hormone, follicle-stimulating hormone, prolactin, testosterone, and cortisol levels. *Am J Obstet Gynecol.* 1983;146(5):550–6.
16. Eliakim A, Portal S, Zadik Z, Rabinowitz J, Adler-Portal D, Cooper DM, et al. The effect of a volleyball practice on anabolic hormones and inflammatory markers in elite male and female adolescent players. *J Strength Cond Res.* 2009;23(5):1553–9.
17. Bruzda-Zwiech A, Konieczka M, Hilt A, Daszkowska M, Grzegorzczak J, Szczepanska J. Salivary cortisol, alpha-amylase and immunoglobulin a responses to a morning session of basketball or volleyball training in boys aged 14–18 years. *J Biol Regul Homeost Agents.* 2017;31(1):105–10.
18. Kubukeli ZN, Noakes TD, Dennis SC. Training techniques to improve endurance exercise performances. *Sports Med.* 2002;32(8):489–509.
19. Meckel Y, Nemet D, Bar-Sela S, Radom-Aizik S, Cooper DM, Sagiv M, et al. Hormonal and inflammatory responses to different types of sprint interval training. *J Strength Cond Res.* 2011;25(8):2161–9.
20. Sansone P, Tessitore A, Paulauskas H, Lukonaitiene I, Tschan H, Pliauga V, et al. Physical and physiological demands and hormonal responses in basketball small-sided games with different tactical tasks and training regimes. *J Sci Med Sport.* 2019;22(5):602–6.
21. Choy M, Salbu RL. Jet lag: current and potential therapies. *P T.* 2011;36(4):221–31.
22. Zhao J, Tian Y, Nie J, Xu J, Liu D. Red light and the sleep quality and endurance performance of Chinese female basketball players. *J Athl Train.* 2012;47(6):673–8.
23. Barnett A. Using recovery modalities between training sessions in elite athletes: does it help? *Sports Med.* 2006;36(9):781–96.
24. Wilcock IM, Cronin JB, Hing WA. Physiological response to water immersion: a method for sport recovery? *Sports Med.* 2006;36(9):747–65.
25. Nemet D, Meckel Y, Bar-Sela S, Zaldivar F, Cooper DM, Eliakim A. Effect of local cold-pack application on systemic anabolic and inflammatory response to sprint-interval training: a prospective comparative trial. *Eur J Appl Physiol.* 2009;107(4):411–7.
26. Buyukyazi G, Karamizrak SO, Islegen C. Effects of continuous and interval running training on serum growth and cortisol hormones in junior male basketball players. *Acta Physiol Hung.* 2003;90(1):69–79.
27. Miloski B, Aoki MS, de Freitas CG, Schultz de Arruda AF, de Moraes HS, Drago G, et al. Does testosterone modulate mood states and physical performance in Young basketball players? *J Strength Cond Res.* 2015;29(9):2474–81.
28. Eliakim A, Nemet D, Bar-Sela S, Higer Y, Falk B. Changes in circulating IGF-I and their correlation with self-assessment and fitness among elite athletes. *Int J Sports Med.* 2002;23(8):600–3.
29. Nunes JA, Moreira A, Crewther BT, Nosaka K, Viveiros L, Aoki MS. Monitoring training load, recovery-stress state, immune-endocrine responses, and physical performance in elite female basketball players during a periodized training program. *J Strength Cond Res.* 2014;28(10):2973–80.
30. Steinacker JM, Lormes W, Kellmann M, Liu Y, Reissnecker S, Opitz-Gress A, et al. Training of junior rowers before world championships. Effects on performance, mood state and selected hormonal and metabolic responses. *J Sports Med Phys Fitness.* 2000;40(4):327–35.
31. Mejri S, Bchir F, Ben Rayana MC, Ben HJ, Ben SC. Effect of training on GH and IGF-1 responses to a submaximal exercise in football players. *Eur J Appl Physiol.* 2005;95(5–6):496–503.
32. Roemmich JN, Sinning WE. Weight loss and wrestling training: effects on growth-related hormones. *J Appl Physiol.* 1997;82(6):1760–4.
33. Jurimae J, Cicchella A, Jurimae T, Latt E, Haljaste K, Purge P, et al. Regular physical activity influences plasma ghrelin concentration in adolescent girls. *Med Sci Sports Exerc.* 2007;39(10):1736–41.
34. Plinta R, Olszanecka-Glinianowicz M, Drosdzol-Cop A, Chudek J, Skrzypulec-Plinta V. The effect of three-month pre-season preparatory period and short-term exercise on plasma leptin, adiponectin, visfatin, and

- ghrelin levels in young female handball and basketball players. *J Endocrinol Investig.* 2012;35(6):595–601.
35. Eliakim A, Brasel JA, Mohan S, Barstow TJ, Berman N, Cooper DM. Physical fitness, endurance training, and the growth hormone-insulin-like growth factor I system in adolescent females. *J Clin Endocrinol Metab.* 1996;81(11):3986–92.
 36. Eliakim A, Brasel JA, Mohan S, Wong WL, Cooper DM. Increased physical activity and the growth hormone-IGF-I axis in adolescent males. *Am J Phys.* 1998;275(1 Pt 2):R308–R14.
 37. Eliakim A, Scheett TP, Newcomb R, Mohan S, Cooper DM. Fitness, training, and the growth hormone->insulin-like growth factor I axis in prepubertal girls. *J Clin Endocrinol Metab.* 2001;86(6):2797–802.
 38. Scheett TP, Nemet D, Stoppani J, Maresh CM, Newcomb R, Cooper DM. The effect of endurance-type exercise training on growth mediators and inflammatory cytokines in pre-pubertal and early pubertal males. *Pediatr Res.* 2002;52(4):491–7.
 39. Greig CA, Hameed M, Young A, Goldspink G, Noble B. Skeletal muscle IGF-I isoform expression in healthy women after isometric exercise. *Growth Hormon IGF Res.* 2006;16(5–6):373–6.
 40. Izquierdo M, Ibanez J, Gonzalez-Badillo JJ, Ratamess NA, Kraemer WJ, Hakkinen K, et al. Detraining and tapering effects on hormonal responses and strength performance. *J Strength Cond Res.* 2007;21(3):768–75.
 41. Nemet D, Portal S, Zadik Z, Pilz-Burstein R, Adler-Portal D, Meckel Y, et al. Training increases anabolic response and reduces inflammatory response to a single practice in elite male adolescent volleyball players. *J Pediatr Endocrinol Metab.* 2012;25(9–10):875–80.
 42. Eliakim A, Portal S, Zadik Z, Meckel Y, Nemet D. Training reduces catabolic and inflammatory response to a single practice in female volleyball players. *J Strength Cond Res.* 2013;27(11):3110–5.
 43. Codner E, Mericq MV, Maheshwari HG, Inguez G, Capurro MT, Salazar T, et al. Relationship between serum growth hormone binding protein levels and height in young men. *J Pediatr Endocrinol Metab.* 2000;13(7):887–92.
 44. Ben Abdelkrim N, Castagna C, El Fazaa S, Tabka Z, El Ati J. Blood metabolites during basketball competitions. *J Strength Cond Res.* 2009;23(3):765–73.
 45. Souglis A, Bogdanis GC, Giannopoulou I, Papadopoulos C, Apostolidis N. Comparison of inflammatory responses and muscle damage indices following a soccer, basketball, volleyball and handball game at an elite competitive level. *Res Sports Med.* 2015;23(1):59–72.
 46. Arruda AF, Aoki MS, Freitas CG, Drago G, Oliveira R, Crewther BT, et al. Influence of competition playing venue on the hormonal responses, state anxiety and perception of effort in elite basketball athletes. *Physiol Behav.* 2014;130:1–5.
 47. Arruda AFS, Aoki MS, Paludo AC, Moreira A. Salivary steroid response and competitive anxiety in elite basketball players: effect of opponent level. *Physiol Behav.* 2017;177:291–6.
 48. Arruda AFS, Aoki MS, Paludo AC, Drago G, Moreira A. Competition stage influences perceived performance but does not affect rating of perceived exertion and salivary neuro-endocrine-immune markers in elite young basketball players. *Physiol Behav.* 2018;188:151–6.
 49. Schelling X, Calleja-Gonzalez J, Torres-Ronda L, Terrados N. Using testosterone and cortisol as biomarker for training individualization in elite basketball: a 4-year follow-up study. *J Strength Cond Res.* 2015;29(2):368–78.
 50. Moreira A, Aoki MS, Franchini E, da Silva Machado DG, Paludo AC, Okano AH. Mental fatigue impairs technical performance and alters neuroendocrine and autonomic responses in elite young basketball players. *Physiol Behav.* 2018;196:112–8.
 51. MacDonald LA, Minahan CL. Mindfulness training attenuates the increase in salivary cortisol concentration associated with competition in highly trained wheelchair-basketball players. *J Sports Sci.* 2018;36(4):378–83.
 52. Ponce-Gonzalez JG, Olmedillas H, Calleja-Gonzalez J, Guerra B, Sanchis-Moysi J. Physical fitness, adiposity and testosterone concentrations are associated to playing position in professional basketballers. *Nutr Hosp.* 2015;31(6):2624–32.
 53. Cardinale M, Stone MH. Is testosterone influencing explosive performance? *J Strength Cond Res.* 2006;20(1):103–7.
 54. Kumari M, Tabassum F, Clark C, Strachan D, Stansfeld S, Power C. Social differences in insulin-like growth factor-1: findings from a British birth cohort. *Ann Epidemiol.* 2008;18(8):664–70.
 55. Bogin B, Hermanussen M, Blum WF, Assmann C. Sex, sport, IGF-1 and the community effect in height hypothesis. *Int J Environ Res Public Health.* 2015;12(5):4816–32.



Biomechanics of Lower Extremity Movements and Injury in Basketball

4

Jeffrey B. Taylor, Eric J. Hegedus, and Kevin R. Ford

4.1 Introduction

Biomechanics refers to the study of movement of a biological system through the methods of mechanics. This chapter will focus on the biomechanics, both kinematics (motion) and kinetics (forces and torques), during basketball activities. Depending on the quality and precision of data needed, evaluative biomechanical methods can range from inexpensive visual observation with the naked eye to high-tech and expensive three-dimensional motion analysis cameras and force platforms. Movement analysis can focus globally on the athlete in terms of activity demands or more specifically to an athlete's joint loads or muscle function. Thus, biomechanical data can provide insight for coaches and clinicians in their pursuit of optimizing performance or reducing the burden of injury through preventive and rehabilitative practices.

Basketball is a multi-directional team sport that places significant demands on the lower extremity [1]. During sport activity, the lower extremity serves as the primary means of locomotion, positioning an athlete's body in the most optimal location on the court. These movements occur horizontally and vertically, making the ability to efficiently run, shuffle, cut, and jump of utmost importance to the basketball athlete that strives for high performance. Because demands are high, the lower extremities can also be at high risk for injury during basketball competition and practice. The repetitive nature and high volume of movement demands can place the basketball athlete at risk of developing overuse chronic pathologies, while the high intensity of the sport promotes maximal exertion that can lead to acute injury by potentially injurious forces. Basketball is a physical game that can result in various contact-related lower extremity injuries. In efforts to elucidate the lower extremity biomechanical considerations of basketball, this chapter will detail: (1) the global lower extremity movement demands that characterize the sport of basketball, (2) lower extremity joint biomechanics during basketball-specific lower extremity movements, and (3) biomechanical considerations of basketball-related lower extremity injuries.

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4.2 Movement Demands

There has been a substantial amount of evidence reported on the lower extremity movement demands during basketball competition. These demands have been analyzed retrospectively using observational time–motion analysis or computerized analysis of video footage and more recently in real time using wearable technology such as accelerometers, inertial measurement units (IMUs), and global positioning system (GPS) tracking sensors. The most frequently analyzed variables include straight-line running (distance, speeds, frequencies), non-sagittal plane movements (lateral movement demands, accelerations, decelerations), and vertical demands (jump frequency) [1]. These demands are dependent on player position, sex, and age and should influence end-stage rehabilitation and return to play testing after injury which will be discussed in future sections of this chapter.

4.2.1 Total Distance

A standard international level basketball court covers 420 m², requiring athletes to travel significant distances when competing. Evidence indicates that elite male players travel approximately 6150 m per game, while junior males cover approximately 7500 m [1]. Females average slightly less distance than males, with elite females covering an average of 5500–7000 m per game and junior females covering 5500 m [1]. Total distances covered are generally similar between backcourt and frontcourt players, with both covering between 125 and 134 m/min [2–5].

4.2.2 Straight-Line Running

Straight-line running is necessary to quickly cover the length of the court. Basketball players average anywhere between 18 and 105 sprints (≥ 25 km/h) per game, with each sprint averaging 0.5–2.4 s, covering 4–9 m. Though frequent (2–3 per min [6]), these sprints only account for 2–6% of total game time [1]. High-intensity run-

ning (18–24 km/h) accounts for similar distances (~400 m) and 2.4% of total game time [1].

4.2.3 Non-sagittal Plane Movements

The number of activity changes that have been reported over the course of a game are highly variable, ranging from 1000 to 2700 per game in adult males, 1100 in junior males, and 600 to 1750 in adult females [1]. These activity changes range from 32 changes per min in elite males [6] to 40 per min in sub-elite males [5], and 43 per min in female [3]. There have been very few reported positional differences in activity changes.

Specifically, basketball players spend a significant amount of time moving laterally, much of which occurs during defensive activity [1]. Lateral movements occur up to 400 times per match, though approximately 82% occur at low or moderate intensities and speeds [7]. These movements account for approximately 250 m [2], and around >15% of total game time [8, 9]. In general, guards spend more time in lateral movement than forwards and centers [10].

Fact Box

Basketball requires significant multi-directional demands that are characterized by significant lateral movement throughout the course of a game.

4.2.4 Vertical Demands

Basketball is often characterized by the vertical demands associated with shooting, rebounding, and defending. Males (41–56 jumps per game) have been reported to jump more frequently than females (19–43 jumps per game) [1]. These demands account for approximately 2% of total game time [9]. With athletes generally averaging around one jump per min [5]. Positionally, centers have been reported to jump more often

than guards and forwards [10], probably due to the tactical proximity to the basket and their frequent involvement in tasks such as shot-blocking attempts and rebounding on both ends of the court.

4.3 Lower Extremity Biomechanics During Basketball Movements

4.3.1 Jumping and Landing

The analysis of jumping technique is often a focus of basketball athletes in order to improve vertical jump height which influences the skills

of shooting, rebounding, and shot blocking. Jumping can be divided into four main phases: preparatory, propulsive, flight, and landing and further classified as single- compared to double-legged or from standing compared to an approach (Fig. 4.1). Jump height is increased when a preparatory descent is performed prior to the propulsive phase. During the preparatory phase of a countermovement jump (CMJ), the body center of mass is decelerated downward with flexion of the lower extremity joints. This lowering movement creates a muscle stretch before a rapid shortening to accelerate the body vertically and is termed the stretch-shortening cycle (SSC) [11, 12]. The benefit of a preparatory phase on jump performance is well established and often illus-

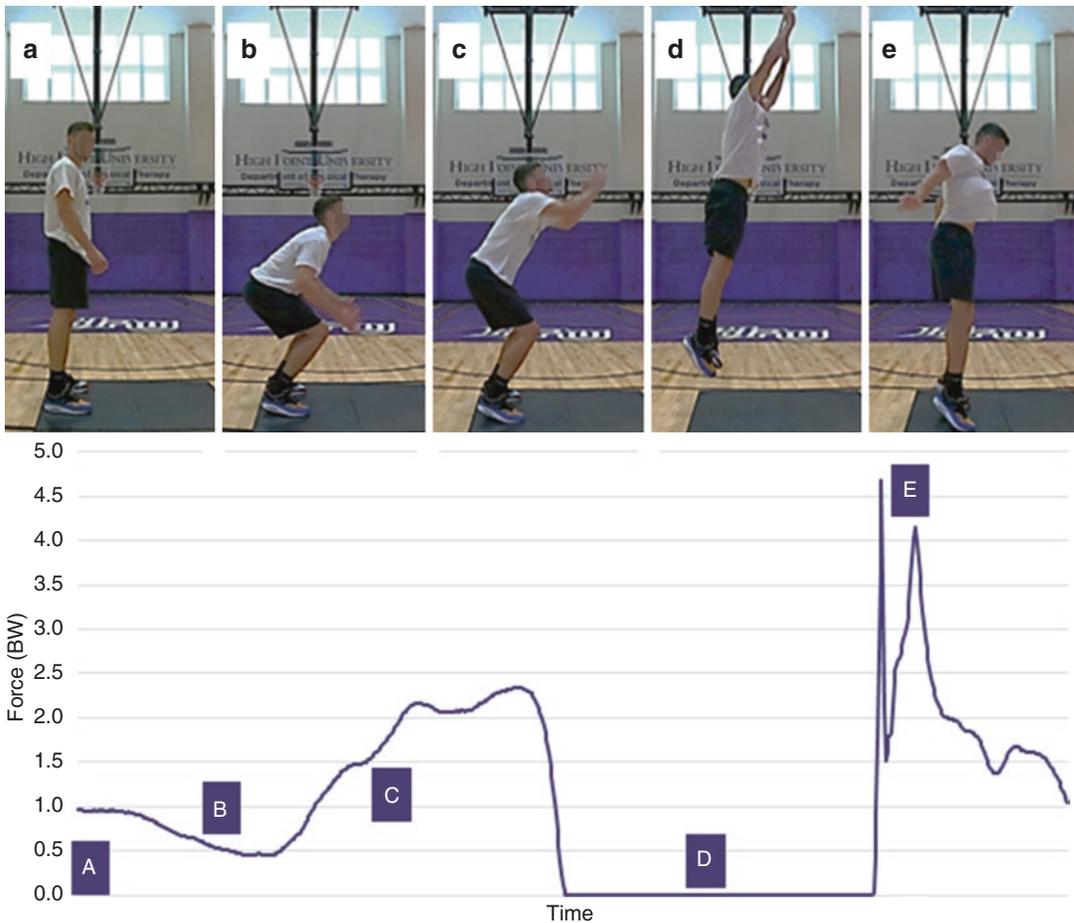


Fig. 4.1 Countermovement jump illustrating the kinematics (top) and vertical ground reaction forces (bottom) during the (A) pre-jump, (B) preparatory, (C) propulsive, (D) flight, and (E) landing phases

trated by comparing a CMJ to a squat jump (SJ) [13]. During the SJ, the athlete begins with the lower extremities in a flexed position and executes the jump without a countermovement. In contrast to the SJ, the hip extensors, knee extensors, and ankle plantarflexors contract eccentrically during the preparatory phase of a CMJ. The coordinated action of the lower extremity places the body center of mass at its lowest vertical position prior to the rapid upward acceleration during the propulsive phase.

The propulsive phase begins at the lowest vertical position of the body center of mass and ends at toe off from the ground. There is rapid hip extension, knee extension, and ankle plantarflexion until a near fully extended position [12, 14–16]. The joint moments, power, and work during this phase of jumping predict jump height in male and female athletes [14, 16]. Specifically, increased knee and hip extensor moments during the propulsive phase are correlated with an increased vertical jump height. This indicates that extensor musculature strength are critical components to be targeted in a strength and conditioning program that aims to improve jumping performance.

The flight and subsequent landing complete the phases of the vertical jump. During the flight phase, the body center of mass reaches its highest vertical position (vertical jump height). Vertical jump height has been reported to vary in both female (22–48 cm) and male (40–75 cm) basketball players [17]. Certainly, the level of skill and the testing methodology likely explain this large variation in jump performance. The landing phase begins immediately when the feet touch the ground and is typically the phase of interest relative to risk of injury. Obviously, the demands on the lower extremity to absorb forces are increased as jump height performance is improved.

The performance and biomechanics of jumping off of or landing on single leg or both legs have been previously investigated in numerous studies [18–21]. From a stationary position, approximately 25% greater height can be achieved off of 2 ft compared to a single foot [18]. However, the difference in jump height is

negligible when a one-stride running approach is added and approximately 10% greater with a single-leg takeoff compared to double-leg takeoff when 3–5 strides are involved [18]. In order to utilize the approach velocity and SSC successfully, a certain level of technical skill and neuromuscular coordination is required by the basketball player [20, 22].

Plyometric training and specifically the use of drop vertical jumps (DVJ) have been advocated to help athletes increase vertical jump height and performance in explosive activities [23–26]. By increasing the drop height, the athlete must transition larger ground reaction forces during the preparatory phase prior to the propulsive phase [16, 27]. Optimal drop heights vary among skill level, sex, and age but typically range from 15 to 30 cm. Additionally, DVJ movements have been used in numerous screening methodologies to identify risk of injury based on altered landing biomechanics [28, 29].

4.3.2 Running, Cutting, and Lateral Movements

Biomechanics of running, cutting, and lateral movements in basketball players are less commonly reported in the scientific literature compared to jumping and landing movements. However, these activities occur much more often during actual basketball games [1]. Additionally, the high ground reaction forces and quick changes of direction may lead to injurious movements [30, 31]. Due to differences in sport demands, basketball players have distinctly different biomechanics during these tasks compared to other athletes [32–34]. Young female basketball players have been reported to exhibit lower ground reaction forces, indicative of potentially lower risk of injury, during a jump-stop cut task compared to soccer players [34]. However, in a similar population, basketball players had less hip and knee flexion and greater external knee abduction moments, movement patterns related to greater risk of knee injury, during a quick laterally directed landing compared to soccer players [32].

4.4 Biomechanical Considerations of Lower Extremity Injuries

Of multi-directional sports, basketball has a relatively high injury rate, ranging from 1.51 injuries per 1000 h of exposure [35] to 8.0 injuries per 1000 athletic exposures [36]. Because basketball players practice more than they compete in games, the majority of injuries occur during practice (67%), but games have a higher injury incidence rate [36]. Though both males and females have high injury rates, males get hurt more frequently but have a lower percentage of time loss injuries than females [35, 36]. While the majority of injuries are a result of contact, up to 48% of injuries may happen via a noncontact mechanism [37]. Injuries are most prevalent in the lower extremity (54–78%), especially at the ankle (30–48%) and knee (18%) [9, 36, 38, 39]. More injuries occur during defending than ball handling, and among specific basketball demands, rebounding (18–29%) and loose balls (24%) account for the most injuries [40, 41]. Epidemiology and biomechanics associated with specific injuries are discussed in the subsequent sections.

4.4.1 Foot and Ankle

4.4.1.1 Ankle Sprain

Ankle sprains are the most common injury in basketball (1.49 and 1.21 injuries per 1000 athletic exposures in men's and women's basketball, respectively) [42] which accounts for the highest ankle sprain injury rate of all sports [43]. Ankle sprains are of major concern because of the high rates of injury recurrence. Basketball players with a history of ankle sprain are five times more likely to suffer a subsequent sprain than players without history of sprain [44]. Sprains of the lateral ligament complex are by far the most prevalent (80–83%), followed by deltoid ligament tears (6–7%) and syndesmotic sprains (7%) [42]. While these injuries typically do not require surgical intervention, they do require considerable rehabilitation in order to return to play. The amount of time loss varies depending on the severity and location of

injury, but 30–33% of ankle sprain injuries result in longer than 7 days lost from sport [42].

Ankle sprains are typically the result of contact with another player (landing on another player's foot), most often in the key area during a rebounding attempt and most often to players playing the center position [42, 45]. Lateral ankle sprains typically occur via sudden and rapid inversion ($>70^\circ$) in low levels of plantarflexion (10° – 35°) and either the presence of internal rotation (resulting in high strain to the anterior talofibular ligament and calcaneofibular ligament) or absence of internal rotation (resulting in isolated strain to the calcaneofibular ligament) [46]. Syndesmotic and deltoid ligament injuries commonly occur at the same time [47]. The most frequent mechanism of syndesmotic injury is foot and ankle external rotation with a dorsiflexed ankle and pronated foot [47].

Multiple intrinsic factors including anthropometrics, neuromuscular performance, and postural stability have been reported as risk factors for ankle sprain in an athletic population [48]. Specifically, a higher body mass index has been connected with greater risk of lateral ankle sprain [49, 50]. Higher-risk has also been reported in athletes with a neuromuscular profile consistent with decreased slow eccentric ankle inversion strength, increased fast concentric plantarflexion strength, and slower reaction time of the peroneus brevis to perturbation [49, 51]. Improving the strength of the gluteus maximus and hip extensors may help mitigate the risk of ankle sprain [52]. Proprioceptively, high levels of postural sway (in all directions), a commonly used measure of balance, may also predict future ankle sprain injury [51].

Primary prevention efforts have been successful in reducing ankle sprains in the basketball population [53], such that seven players need to participate in one neuromuscular training session to prevent one ankle sprain [54]. Specifically, neuromuscular training programs have been reported to significantly improve single-limb balance and postural sway, thus reducing the risk of ankle sprain by up to 35% [54, 55]. Similarly, the use of external support, in the form of bracing or taping, may help reduce the risk of ankle sprain

[53]. In general, over 50% of basketball players wear ankle joint support, and those that do sustain ankle sprains at a significantly lower rate [45]. This is especially successful in athletes with a history of inversion ankle injury [48, 56]. External support systems have been reported to reduce non-weightbearing and weightbearing inversion range of motion [57–59], increase muscle activation and excitability [58, 60], and decrease joint velocity during movement [59, 61], all of which may explain the mechanism for reduced risk of ankle injury when present.

Fact Box

Ankle sprains are the most common injury in basketball. Rehabilitation after sprain is of vital importance to reduce the risk of recurrent injuries.

4.4.1.2 Bony Foot Injury

Foot fractures account for 55% of all lower extremity bony stress injuries, with 18% occurring in the fifth metatarsal [62]. These injuries can be devastating for basketball players because they result in significant time loss (25.1 ± 21.3 games) and place the athlete at high risk for recurrent or subsequent injury [62]. Up to 30% of players may not be able to return to previous level of play after a stress reaction, which increases to 43% when considering fifth metatarsal injury alone [62]. Other common foot stress reaction/fracture sites include the navicular and fourth metatarsal [62].

These types of injuries are typically the result of repetitive overuse and loading, especially from consistent landing and pivoting. While the mechanism of fifth metatarsal injury is not clear, it is thought that larger plantar loads directed to the base of the fifth metatarsal or abnormal relationships between distal and proximal metatarsal loading may place significant bending moments through the metatarsals, leading to stress reaction and failure [63]. These bending moments are especially high during sagittal plane accelerations where a lack of heel strike leads to high forefoot loading [63]. Greater foot length may

lead to higher bending moments across the metatarsal. A cross-sectional study of 220 basketball players reported 14 fifth metatarsal fractures, 100% of which occurred to athletes that played the center position [64]. While foot length was not measured or reported, it can be inferred that because these athletes were the tallest in the study [64], they also had the largest foot length.

While no studies have identified clear-cut ways to reduce the risk of bony foot injuries, reducing overall load or regional-specific plantar load are currently the best options. A recent study investigated the modification of the midsole stiffness of basketball shoes (to reduce bending moments through the metatarsals) reported that increased stiffness resulted in lower plantar forces under the lesser toes, potentially decreasing the risk of fifth metatarsal injury [65]. However, altering biomechanics at one region may alter biomechanics in a different region as illustrated by greater ankle dorsiflexion angles reported in the study, which may ultimately place the Achilles tendon at higher risk for injury [65]. When modifying shoe properties, clinicians need to account for unintended compensations that can occur throughout the kinetic chain.

4.4.2 Knee

4.4.2.1 Anterior Cruciate Ligament Injury

Injuries to the anterior cruciate ligament are commonly seen in the competitive basketball population [66], occurring at a 2–4 times higher rate in women (0.09 4 ACL ruptures per 1000 athletic exposures) than men (0.02 injuries per 1000 athletic exposures) [66, 67]. The majority of ACL injuries occur in game competition during jumping and landing [66, 68]. ACL injuries are of particular concern because of the high risk of concomitant meniscal injury (65%) in basketball players [69]. Depending on the severity of injury, one ACL injury can result in a lifetime cost of \$100,000 [70], and the early development of knee joint osteoarthritis, with up to 48% of injured athletes showing signs of joint degradation within 10 years of injury [71].

The majority (60–70%) of basketball-related ACL injuries occur via a noncontact mechanism [68, 72], with 90% occurring during a single-leg activity [73]. Most injuries occur while attacking, as opposed to defending, with rebounding as the most frequently cited activity causing injury [74, 75]. Video analysis of ACL injuries show that despite the absence of physical contact from an opposing player at initial ground contact, up to 50% of injured female basketball athletes are either part of a collision or pushed immediately prior to injury and a high percentage of injuries occurred with another player in a surrounding 1-m radius [74]. At the time of injury, studies have shown that athletes typically land on the heel or flat portion of the foot, with decreased ankle plantarflexion, decreased knee flexion, and increased hip flexion [76]. Injury to

the ACL occurs within 40 ms of landing, which is represented by rapid knee abduction and internal rotation after initial ground contact [30].

Although ACL injury risk is multifactorial, there is a strong link between lower extremity biomechanics and injury. Dynamic lower extremity valgus (Fig. 4.2) have been prospectively identified as a high-risk movement pattern [29]. Specifically, knee abduction moment, or the external torque pushing the knee into a valgus position, has been reported as the single best predictor of ACL injury in female athletes [29]. Landing with shallow knee flexion angles has also been implicated as a risk factor for ACL injury, potentially due to strong quadriceps contraction, especially if an athlete lands with their foot anterior to the center of mass [73, 77]. Basketball athletes that have previously suffered

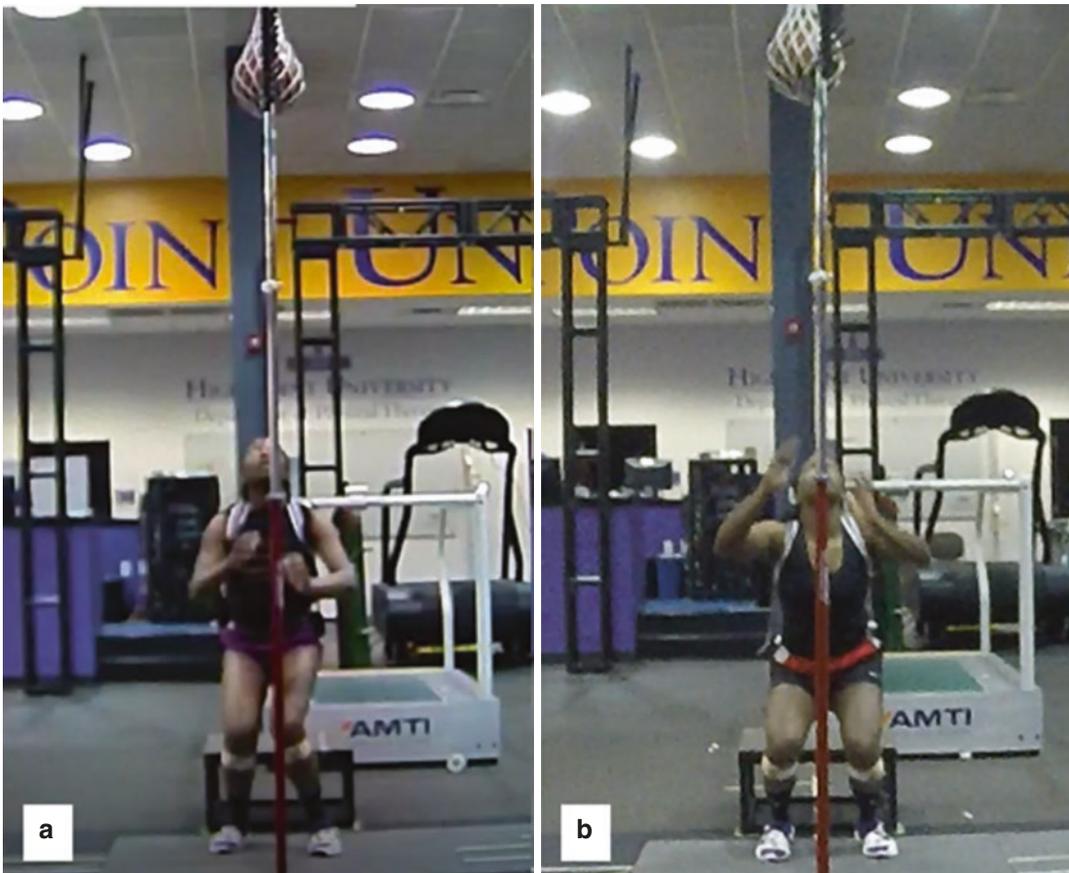


Fig. 4.2 High-risk (a: dynamic lower extremity valgus) and low-risk (b: good control, adequate knee flexion) landing patterns in basketball athletes

an ACL injury are at a significantly higher risk for second injury than uninjured athletes, as 25% of surgically reconstructed patients suffer a second injury (on the same or opposite limb) within 12 months of return to play [78]. Specifically, transverse plane hip kinetics, frontal plane knee kinematics, and sagittal plane knee moments at landing may place a previously injured athlete at risk for second ACL injury [79].

There has also been a reported connection between ACL injury and thigh muscle strength and activation. The primary responsibility of the ACL is to resist anterior tibial translation in respect to the femur [80]. Anterior tibial translation can result from a strong quadriceps contraction, whereas contraction of the hamstrings can act oppositely, generating a protective effect on the ACL. Thus, clinicians often focus on improving the ratio of hamstrings to quadriceps strength. A prospective study has reported that athletes with reduced total hamstrings strength are more likely to sustain an ACL injury [81].

Anterior cruciate ligament injury prevention programs have been reported to successfully reduce the incidence of injury in general populations [82] and team sports such as soccer and handball [83, 84]; however, these efforts have been historically less successful in basketball populations [53, 83, 84], as the first three published studies using women's basketball players resulted in no significant reduction in ACL injury [53]. However, two more recent studies have shown promise. Recent studies by Omi et al. [85] and Bonato et al. [86] are the first to report ACL injury rate reduction after neuromuscular training specific to women's basketball players. One program used a 3x/week, 20 min per session education and neuromuscular training program that emphasized strength, balance, and technique training focused at the hip [85], while the other used a 5-part, 30-min warm-up that focused on general muscular activation, active mobility/flexibility, strengthening, plyometrics, and agility exercises [86]. To date, no studies have reported success at reducing the risk of ACL injury in male basketball players.

Fact Box

Basketball players need to be taught optimal landing techniques, including:

- Good knee stability (don't let knees come together).
- Land on flexed knees.
- Keep hips back so knees do not advance forward in front of toes.

4.4.2.2 Tendinopathy of the Knee Extensor Mechanism

The repetitive demands of basketball can lead to overuse injury. Of all overuse injuries in the sport, 45% occur at the knee [87], specifically from patellar and quadriceps tendinopathy. Patellar tendinopathy (or Jumper's knee) is the second most common basketball injury to ankle sprain and is more common in guards than forwards and centers [64, 88].

Patellar tendinopathy (PT) typically occurs via an insidious onset of anterior knee pain. The risk of developing PT is also multifactorial. Males with a history of prior knee injury and recent high training loads, greater than 20 h per week, are at higher risk for developing PT [89]. Landing mechanics may also place an individual at greater risk for PT injury [90]. While no difference has been identified between athletes with PT and healthy controls, individuals that exhibit ultrasonographic abnormality (a precursor of PT) land with higher angular velocities of hip and knee flexion and a less upright trunk posture that is especially apparent during horizontal jump-stop landings common in the sport [90]. A more flexed position of the knee at initial contact can lead to less displacement of the center of mass, which when combined with higher angular velocities, result in increased stiffness and tendon loading rates. While there have been no reports of successfully using a neuromuscular training program to decrease PT incidence,

clinicians may be able to decrease risk with technique training during various basketball-related tasks or by improving flexibility of the hamstrings, which may decrease the risk of developing PT [89].

4.5 General Considerations for Rehabilitation and Return to Play

There are several general biomechanical considerations which may apply to rehabilitation and return to play that the sports medicine clinician can implement in their practice. We suggest thinking about rehabilitation and return to play in a systematic way to subsequently address local (joint-specific impairments), regional (surrounding joints, functional limitations), and global (whole body disabilities) considerations.

Local interventions are specific to the injury and the patient. For example, a basketball athlete rehabilitating from an ACL reconstruction should initially be focused on regaining quadriceps function, while an athlete rehabilitating from an ankle sprain should be emphasizing improving multi-directional control and stability of the ankle. Local interventions do not have to be solely therapeutic exercise based and can also include evidence-based manual therapy or other modalities to expedite the healing process.

As the clinician begins to address regional interventions, they can begin to get more basketball-specific. The hip complex is crucial to address in these athletes. Basketball players are generally tall [32], many of whom have experienced a rapid growth spurt at some point in their life. These growth spurts can lead to long lever arms (i.e., femur) that may be difficult to control. As stated earlier, lower extremity dynamic valgus is a common biomechanical dysfunction that can lead to injury. Lower hip strength is associated with

a greater lower extremity valgus pattern during single-legged jump landings [91]. Thus, addressing this movement strategy with a focus on strengthening of the proximal hip musculature can promote control of the limb and subsequently reduce future injury risk. Targeted exercises of the gluteus maximus (a hip extensor and external rotator), gluteus medius (a hip abductor), and hamstrings (a hip extensor and knee flexor) can help reduce lower extremity dynamic valgus and balance the sagittal plane tibial translation forces that occur during basketball-specific movements.

Examples of hip-focused interventions can be found in Fig. 4.3. Figure 4.3a–c shows potential lunge progressions. As a strictly sagittal plane exercise, a traditional lunge can target the hamstrings musculature. Adding additionally transverse plane motions (Fig. 4.3a,b) can elicit greater recruitment of the gluteus maximus and other hip rotators needed to combat various abnormal movement patterns. Similarly, Fig. 4.3c increases the frontal plane demands of the lunge. By placing weight in the opposite upper extremity, the gluteus medius of the forward limb is forced to create extra torque to counteract the resistance. Figure 4.3d,e illustrates a sidestepping progression that elicits gluteus medius torque while in a basketball-specific defensive position. Figure 4.3f shows an eccentric hamstring exercise that focuses on recruitment of the athlete's posterior hip and thigh musculature. Other exercises, such as a unilateral straight-knee bridge and single-leg deadlift have been reported to elicit high hamstring muscle activity and could be excellent additions to post-operative protocols [92]. Kettlebell swings and Romanian deadlifts can target the medial hamstrings (i.e., semitendinosus), while supine leg curls and hyperextensions off a table can be used to specifically target the lateral hamstrings (i.e., biceps femoris) [93].

As the athlete progresses through rehabilitation and approaches return-to-play, the global



Fig. 4.3 Examples of posterior and lateral hip-focused exercises, including (a) plie lunge, (b) rotational lunge, (c) contra-lateral weighted lunge, (d, e) resisted side-step progression, and (f) Russian hamstring curls

load demands of the sport must be considered. Earlier in this chapter, we summarized the running, jumping, and change of direction demands in men's and women's basketball. These average values should serve as targets for the clinician, especially when dealing with a lengthy rehab process. Additionally, to adapt a rehabilitation protocol to a basketball athlete, clinicians should emphasize basketball-specific demands,

including exercises in the frontal plane and both double-leg and single-leg jumping and landing activities. Figure 4.4 illustrates examples of these basketball-specific movements. Other examples include the medial and lateral triple hop for distance, which represent a combination of medial/lateral movement and single-leg hopping, making it an ideal late-stage rehabilitation tool for this population [94].

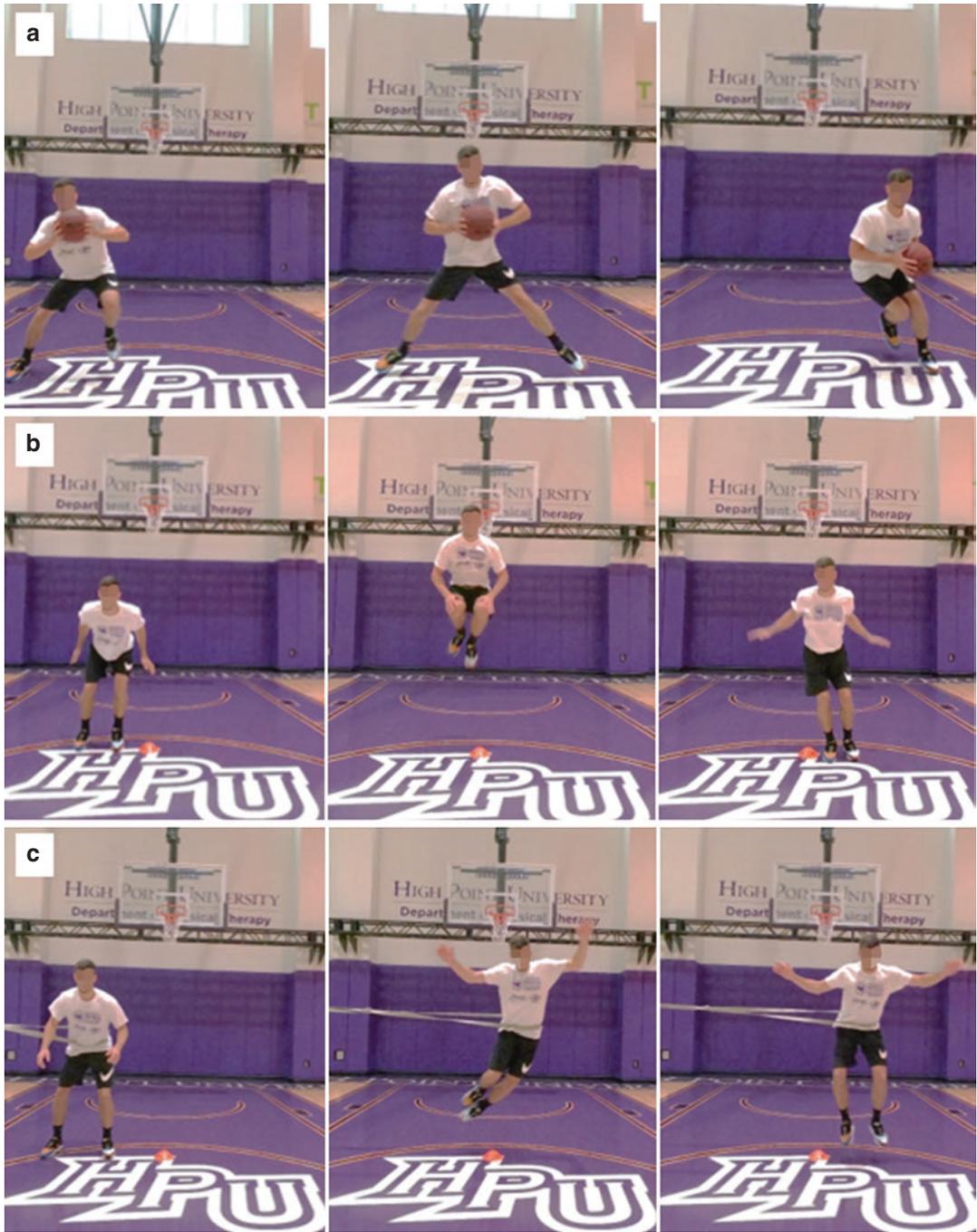


Fig. 4.4 Example laterally biased drills for use during late-stage rehabilitation, including (a) lateral bound, (b) lateral tuck jump, and (c) resisted lateral jump

Fact Box

Clinicians should incorporate local (joint-focused), regional (hip-focused), and global (external load, lateral, and single-leg movements) interventions into their rehabilitation and return to play paradigms.

Take Home Message

In summary, understanding the lower extremity biomechanical demands (load, joint angles, and forces) of a basketball player can help identify those individuals that may be at risk for overuse or noncontact injury. Being able to teach an athlete optimal biomechanics can help reduce injury risk, expedite rehabilitation, and provide for safe return-to-sport.

References

1. Taylor JB, Wright AA, Dischiavi SL, Townsend MA, Marmon AR. Activity demands during multi-directional team sports: a systematic review. *Sports Med.* 2017;47(12):2533–51.
2. Scanlan A, Dascombe B, Reaburn P. A comparison of the activity demands of elite and sub-elite Australian men's basketball competition. *J Sports Sci.* 2011;29(11):1153–60.
3. Scanlan AT, Dascombe BJ, Kidcaff AP, Peucker JL, Dalbo VJ. Gender-specific activity demands experienced during semiprofessional basketball game play. *Int J Sports Physiol Perform.* 2015;10(5):618–25.
4. Scanlan AT, Dascombe BJ, Reaburn P, Dalbo VJ. The physiological and activity demands experienced by Australian female basketball players during competition. *J Sci Med Sport.* 2012;15(4):341–7.
5. Scanlan AT, Tucker PS, Dascombe BJ, Berkelmans DM, Hiskens MI, Dalbo VJ. Fluctuations in activity demands across game quarters in professional and semiprofessional male basketball. *J Strength Cond Res.* 2015;29(11):3006–15.
6. Matthew D, Delextrat A. Heart rate, blood lactate concentration, and time-motion analysis of female basketball players during competition. *J Sports Sci.* 2009;27(8):813–21.
7. McInnes SE, Carlson JS, Jones CJ, McKenna MJ. The physiological load imposed on basketball players during competition. *J Sports Sci.* 1995;13(5):387–97.
8. Ben Abdelkrim N, Castagna C, El Fazaa S, El Ati J. The effect of players' standard and tactical strategy on game demands in men's basketball. *J Strength Cond Res.* 2010;24(10):2652–62.
9. Ben Abdelkrim N, Castagna C, Jabri I, Battikh T, El Fazaa S, El Ati J. Activity profile and physiological requirements of junior elite basketball players in relation to aerobic-anaerobic fitness. *J Strength Cond Res.* 2010;24(9):2330–42.
10. Ben Abdelkrim N, El Fazaa S, El Ati J. Time-motion analysis and physiological data of elite under-19-year-old basketball players during competition. *Br J Sports Med.* 2007;41(2):69–75. discussion.
11. Steben RE, Steben AH. The validity of the stretch shortening cycle in selected jumping events. *J Sports Med Phys Fitness.* 1981;21(1):28–37.
12. Ford KR, Myer GD, Smith RL, Byrnes RN, Dopirak SE, Hewett TE. Use of an overhead goal alters vertical jump performance and biomechanics. *J Strength Cond Res.* 2005;19(2):394–9.
13. Bobbert MF, Gerritsen KG, Litjens MC, Van Soest AJ. Why is countermovement jump height greater than squat jump height? *Med Sci Sports Exerc.* 1996;28(11):1402–12.
14. Aragon-Vargas LF, Gross MM. Kinesiological factors in vertical jump performance: differences among individuals. *J Appl Biomech.* 1997;13(1):24–44.
15. Ford KR, Nguyen AD, Hegedus EJ, Taylor JB. Vertical jump biomechanics altered with virtual overhead goal. *J Appl Biomech.* 2017;33(2):153–9.
16. Ford KR, Myer GD, Brent JL, Hewett TE. Hip and knee extensor moments predict vertical jump height in adolescent girls. *J Strength Cond Res.* 2009;23(4):1327–31.
17. Ziv G, Lidor R. Vertical jump in female and male basketball players—a review of observational and experimental studies. *J Sci Med Sport.* 2010;13(3):332–9.
18. Young W, MacDonald C, Heggen T, Fitzpatrick J. An evaluation of the specificity, validity and reliability of jumping tests. *J Sports Med Phys Fitness.* 1997;37(4):240–5.
19. Wang LI. The lower extremity biomechanics of single- and double-leg stop-jump tasks. *J Sports Sci Med.* 2011;10(1):151–6.
20. van Soest AJ, Roebroek ME, Bobbert MF, Huijing PA, van Ingen Schenau GJ. A comparison of one-legged and two-legged countermovement jumps. *Med Sci Sports Exerc.* 1985;17(6):635–9.
21. Taylor JB, Ford KR, Nguyen AD, Shultz SJ. Biomechanical comparison of single- and double-leg jump landings in the sagittal and frontal plane. *Orthop J Sports Med.* 2016;4(6):2325967116655158.
22. Tai WH, Wang LI, Peng HT. Biomechanical comparisons of one-legged and two-legged running vertical jumps. *J Hum Kinet.* 2018;64:71–6.
23. Bobbert MF, Huijing PA, van Ingen Schenau GJ. Drop jumping. I. The influence of jumping technique on the biomechanics of jumping. *Med Sci Sports Exerc.* 1987;19(4):332–8.

24. Wilk KE, Voight ML, Keirns MA, Gambetta V, Andrews JR, Dillman CJ. Stretch-shortening drills for the upper extremities: theory and clinical application. *J Orthop Sports Phys Ther.* 1993;17(5):225–39.
25. Myer GD, Ford KR, McLean SG, Hewett TE. The effects of plyometric versus dynamic stabilization and balance training on lower extremity biomechanics. *Am J Sports Med.* 2006;34(3):490–8.
26. Myer GD, Faigenbaum AD, Chu DA, Falkel J, Ford KR, Best TM, et al. Integrative training for children and adolescents: techniques and practices for reducing sports-related injuries and enhancing athletic performance. *Phys Sportsmed.* 2011;39(1):74–84.
27. Ford KR, Myer GD, Schmitt LC, Uhl TL, Hewett TE. Preferential quadriceps activation in female athletes with incremental increases in landing intensity. *J Appl Biomech.* 2011;27(3):215–22.
28. Taylor JB, Nguyen AD, Paterno MV, Huang B, Ford KR. Real-time optimized biofeedback utilizing sport techniques (ROBUST): a study protocol for a randomized controlled trial. *BMC Musculoskelet Disord.* 2017;18(1):71.
29. Hewett TE, Myer GD, Ford KR, Heidt RS Jr, Colosimo AJ, McLean SG, et al. Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. *Am J Sports Med.* 2005;33(4):492–501.
30. Koga H, Nakamae A, Shima Y, Iwasa J, Myklebust G, Engebretsen L, et al. Mechanisms for noncontact anterior cruciate ligament injuries: knee joint kinematics in 10 injury situations from female team handball and basketball. *Am J Sports Med.* 2010;38(11):2218–25.
31. Hewett TE, Torg JS, Boden BP. Video analysis of trunk and knee motion during non-contact anterior cruciate ligament injury in female athletes: lateral trunk and knee abduction motion are combined components of the injury mechanism. *Br J Sports Med.* 2009;43(6):417–22.
32. Taylor JB, Ford KR, Schmitz RJ, Ross SE, Ackerman TA, Shultz SJ. Biomechanical differences of multidirectional jump landings among female basketball and soccer players. *J Strength Cond Res.* 2017;31(11):3034–45.
33. Taylor JB, Ford KR, Schmitz RJ, Ross SE, Ackerman TA, Shultz SJ. Biomechanical differences in female basketball and soccer players during multidirectional jump landings. *J Strength Cond Res.* 2017;31:3034–45.
34. Cowley HR, Ford KR, Myer GD, Kernozek TW, Hewett TE. Differences in neuromuscular strategies between landing and cutting tasks in female basketball and soccer athletes. *J Athl Train.* 2006;41(1):67–73.
35. Leppanen M, Pasanen K, Kannus P, Vasankari T, Kujala UM, Heinonen A, et al. Epidemiology of overuse injuries in youth team sports: a 3-year prospective study. *Int J Sports Med.* 2017;38:847–56.
36. Zuckerman SL, Wegner AM, Roos KG, Djoko A, Dompier TP, Kerr ZY. Injuries sustained in National Collegiate Athletic Association men's and women's basketball, 2009/2010–2014/2015. *Br J Sports Med.* 2018;52:261–8.
37. Monfort SM, Comstock RD, Collins CL, Onate JA, Best TM, Chaudhari AM. Association between ball-handling versus defending actions and acute noncontact lower extremity injuries in high school basketball and soccer. *Am J Sports Med.* 2015;43:802–7.
38. Andreoli CV, Chiaramonti BC, Buriel E, Pochini AC, Ejnisman B, Cohen M. Epidemiology of sports injuries in basketball: integrative systematic review. *BMJ Open Sport Exerc Med.* 2018;4(1):e000468.
39. Pasanen K, Ekola T, Vasankari T, Kannus P, Heinonen A, Kujala UM, et al. High ankle injury rate in adolescent basketball: a 3-year prospective follow-up study. *Scand J Med Sci Sports.* 2017;27(6):643–9.
40. Clifton DR, Hertel J, Onate JA, Currie DW, Pierpoint LA, Wasserman EB, et al. The first decade of web-based sports injury surveillance: descriptive epidemiology of injuries in US High School Girls' Basketball (2005–2006 through 2013–2014) and National Collegiate Athletic Association Women's Basketball (2004–2005 through 2013–2014). *J Athl Train.* 2018;53(11):1037–48.
41. Clifton DR, Onate JA, Hertel J, Pierpoint LA, Currie DW, Wasserman EB, et al. The first decade of web-based sports injury surveillance: descriptive epidemiology of injuries in US High School Boys' Basketball (2005–2006 through 2013–2014) and National Collegiate Athletic Association Men's Basketball (2004–2005 through 2013–2014). *J Athl Train.* 2018;53(11):1025–36.
42. Tummala SV, Hartigan DE, Makovicka JL, Patel KA, Chhabra A. 10-Year epidemiology of ankle injuries in men's and women's collegiate basketball. *Orthop J Sports Med.* 2018;6(11):2325967118805400.
43. Roos KG, Kerr ZY, Mauntel TC, Djoko A, Dompier TP, Wikstrom EA. The epidemiology of lateral ligament complex ankle sprains in National Collegiate Athletic Association Sports. *Am J Sports Med.* 2017;45(1):201–9.
44. McKay GD, Goldie PA, Payne WR, Oakes BW. Ankle injuries in basketball: injury rate and risk factors. *Br J Sports Med.* 2001;35(2):103–8.
45. Kofotolis N, Kellis E. Ankle sprain injuries: a 2-year prospective cohort study in female Greek professional basketball players. *J Athl Train.* 2007;42(3):388–94.
46. Panagiotakis E, Mok KM, Fong DT, Bull AMJ. Biomechanical analysis of ankle ligamentous sprain injury cases from televised basketball games: understanding when, how and why ligament failure occurs. *J Sci Med Sport.* 2017;20(12):1057–61.
47. McCollum GA, van den Bekerom MP, Kerkhoffs GM, Calder JD, van Dijk CN. Syndesmosis and deltoid ligament injuries in the athlete. *Knee Surg Sports Traumatol Arthrosc.* 2013;21(6):1328–37.
48. Kerkhoffs GM, van den Bekerom M, Elders LA, van Beek PA, Hullegie WA, Bloemers GM, et al.

- Diagnosis, treatment and prevention of ankle sprains: an evidence-based clinical guideline. *Br J Sports Med.* 2012;46(12):854–60.
49. Kobayashi T, Tanaka M, Shida M. Intrinsic risk factors of lateral ankle sprain: a systematic review and meta-analysis. *Sports Health.* 2016;8:190–3.
 50. Hadzic V, Sattler T, Topole E, Jarnovic Z, Burger H, Dervisevic E. Risk factors for ankle sprain in volleyball players: a preliminary analysis. *Isokinet Exerc Sci.* 2009;17:155–60.
 51. Witchalls J, Blanch P, Waddington G, Adams R. Intrinsic functional deficits associated with increased risk of ankle injuries: a systematic review with meta-analysis. *Br J Sports Med.* 2012;46(7):515–23.
 52. De Ridder R, Witvrouw E, Dolphens M, Roosen P, Van Ginckel A. Hip strength as an intrinsic risk factor for lateral ankle sprains in youth soccer players: a 3-season prospective study. *Am J Sports Med.* 2017;45(2):410–6.
 53. Taylor JB, Ford KR, Nguyen AD, Terry LN, Hegedus EJ. Prevention of lower extremity injuries in basketball: a systematic review and meta-analysis. *Sports Health.* 2015;7(5):392–8.
 54. Eils E, Schroter R, Schroder M, Gerss J, Rosenbaum D. Multistation proprioceptive exercise program prevents ankle injuries in basketball. *Med Sci Sports Exerc.* 2010;42(11):2098–105.
 55. Emery CA, Rose MS, McAllister JR, Meeuwisse WH. A prevention strategy to reduce the incidence of injury in high school basketball: a cluster randomized controlled trial. *Clin J Sport Med.* 2007;17(1):17–24.
 56. Verhagen EA, van Mechelen W, de Vente W. The effect of preventive measures on the incidence of ankle sprains. *Clin J Sport Med.* 2000;10(4):291–6.
 57. Eils E, Demming C, Kollmeier G, Thorwesten L, Volker K, Rosenbaum D. Comprehensive testing of 10 different ankle braces. Evaluation of passive and rapidly induced stability in subjects with chronic ankle instability. *Clin Biomech.* 2002;17(7):526–35.
 58. Lohrer H, Alt W, Gollhofer A. Neuromuscular properties and functional aspects of taped ankles. *Am J Sports Med.* 1999;27(1):69–75.
 59. Vaes PH, Duquet W, Casteleyn PP, Handelberg F, Opdecam P. Static and dynamic roentgenographic analysis of ankle stability in braced and nonbraced stable and functionally unstable ankles. *Am J Sports Med.* 1998;26(5):692–702.
 60. Nishikawa T, Grabiner MD. Peroneal motoneuron excitability increases immediately following application of a semirigid ankle brace. *J Orthop Sports Phys Ther.* 1999;29(3):168–73. discussion 74–6.
 61. McCaw ST, Cerullo JF. Prophylactic ankle stabilizers affect ankle joint kinematics during drop landings. *Med Sci Sports Exerc.* 1999;31(5):702–7.
 62. Khan M, Madden K, Burrus MT, Rogowski JP, Stotts J, Samani MJ, et al. Epidemiology and impact on performance of lower extremity stress injuries in professional basketball players. *Sports Health.* 2018;10:169–74.
 63. Orendurff MS, Rohr ES, Segal AD, Medley JW, Green JR 3rd, Kadel NJ. Biomechanical analysis of stresses to the fifth metatarsal bone during sports maneuvers: implications for fifth metatarsal fractures. *Phys Sportsmed.* 2009;37(2):87–92.
 64. Lopezosa-Reca E, Gijon-Nogueron G, Morales-Asencio JM, Cervera-Marin JA, Luque-Suarez A. Is there any association between foot posture and lower limb-related injuries in professional male basketball players? A cross-sectional study. *Clin J Sport Med.* 2020;30:46–52.
 65. Taylor JB, Nguyen AD, Parry HA, Zuk EF, Pritchard NS, Ford KR. Modifying midsole stiffness of basketball footwear affects foot and ankle biomechanics. *Int J Sports Phys Ther.* 2019;14(3):359–67.
 66. Agel J, Rockwood T, Klossner D. Collegiate ACL injury rates across 15 sports: National Collegiate Athletic Association Injury Surveillance System Data Update (2004–2005 through 2012–2013). *Clin J Sport Med.* 2016;26(6):518–23.
 67. Gornitzky AL, Lott A, Yellin JL, Fabricant PD, Lawrence JT, Ganley TJ. Sport-specific yearly risk and incidence of anterior cruciate ligament tears in high school athletes: a systematic review and meta-analysis. *Am J Sports Med.* 2016;44(10):2716–23.
 68. Piasecki DP, Spindler KP, Warren TA, Andrich JT, Parker RD. Intraarticular injuries associated with anterior cruciate ligament tear: findings at ligament reconstruction in high school and recreational athletes. An analysis of sex-based differences. *Am J Sports Med.* 2003;31(4):601–5.
 69. Granan LP, Inacio MC, Maletis GB, Funahashi TT, Engebretsen L. Sport-specific injury pattern recorded during anterior cruciate ligament reconstruction. *Am J Sports Med.* 2013;41:2814–8.
 70. Mather RC 3rd, Koenig L, Kocher MS, Dall TM, Gallo P, Scott DJ, et al. Societal and economic impact of anterior cruciate ligament tears. *J Bone Joint Surg Am.* 2013;95(19):1751–9.
 71. Oiestad BE, Engebretsen L, Storheim K, Risberg MA. Knee osteoarthritis after anterior cruciate ligament injury: a systematic review. *Am J Sports Med.* 2009;37(7):1434–43.
 72. Benis R, La Torre A, Bonato M. Anterior cruciate ligament injury profile in female elite Italian basketball league. *J Sports Med Phys Fitness.* 2018;58:280–6.
 73. Boden BP, Torg JS, Knowles SB, Hewett TE. Video analysis of anterior cruciate ligament injury: abnormalities in hip and ankle kinematics. *Am J Sports Med.* 2009;37(2):252–9.
 74. Krosshaug T, Nakamae A, Boden BP, Engebretsen L, Smith G, Slauterbeck JR, et al. Mechanisms of anterior cruciate ligament injury in basketball: video analysis of 39 cases. *Am J Sports Med.* 2007;35(3):359–67.
 75. Powell JW, Barber-Foss KD. Sex-related injury patterns among selected high school sports. *Am J Sports Med.* 2000;28(3):385–91.

76. Carlson VR, Sheehan FT, Boden BP. Video analysis of anterior cruciate ligament (ACL) injuries: a systematic review. *JBJS Rev.* 2016;4(11). <https://doi.org/10.2106/JBJS.RVW.15.00116>.
77. Leppanen M, Pasanen K, Kujala UM, Vasankari T, Kannus P, Ayrano S, et al. Stiff landings are associated with increased ACL injury risk in young female basketball and floorball players. *Am J Sports Med.* 2017;45(2):386–93.
78. Paterno MV, Rauh MJ, Schmitt LC, Ford KR, Hewett TE. Incidence of second ACL injuries 2 years after primary ACL reconstruction and return to sport. *Am J Sports Med.* 2014;42(7):1567–73.
79. Paterno MV, Schmitt LC, Ford KR, Rauh MJ, Myer GD, Huang B, et al. Biomechanical measures during landing and postural stability predict second anterior cruciate ligament injury after anterior cruciate ligament reconstruction and return to sport. *Am J Sports Med.* 2010;38(10):1968–78.
80. Markolf KL, Gorek JF, Kabo JM, Shapiro MS. Direct measurement of resultant forces in the anterior cruciate ligament. An in vitro study performed with a new experimental technique. *J Bone Joint Surg Am.* 1990;72(4):557–67.
81. Myer GD, Ford KR, Barber Foss KD, Liu C, Nick TG, Hewett TE. The relationship of hamstrings and quadriceps strength to anterior cruciate ligament injury in female athletes. *Clin J Sport Med.* 2009;19(1):3–8.
82. Taylor JB, Waxman JP, Richter SJ, Shultz SJ. Evaluation of the effectiveness of anterior cruciate ligament injury prevention programme training components: a systematic review and meta-analysis. *Br J Sports Med.* 2015;49(2):79–87.
83. Michaelidis M, Koumantakis GA. Effects of knee injury primary prevention programs on anterior cruciate ligament injury rates in female athletes in different sports: a systematic review. *Phys Ther Sport.* 2014;15(3):200–10.
84. Prodomos CC, Han Y, Rogowski J, Joyce B, Shi K. A meta-analysis of the incidence of anterior cruciate ligament tears as a function of gender, sport, and a knee injury-reduction regimen. *Arthroscopy.* 2007;23(12):1320–1325.e6.
85. Omi Y, Sugimoto D, Kuriyama S, Kurihara T, Miyamoto K, Yun S, et al. Effect of hip-focused injury prevention training for anterior cruciate ligament injury reduction in female basketball players: a 12-year prospective intervention study. *Am J Sports Med.* 2018;46(4):852–61.
86. Bonato M, Benis R, La Torre A. Neuromuscular training reduces lower limb injuries in elite female basketball players. A cluster randomized controlled trial. *Scand J Med Sci Sports.* 2018;28:1451–60.
87. Leppanen M, Pasanen K, Kujala UM, Parkkari J. Overuse injuries in youth basketball and floorball. *Open Access J Sports Med.* 2015;6:173–9.
88. Cumps E, Verhagen E, Meeusen R. Prospective epidemiological study of basketball injuries during one competitive season: ankle sprains and overuse knee injuries. *J Sports Sci Med.* 2007;6(2):204–11.
89. Morton S, Williams S, Valle X, Diaz-Cueli D, Malliaras P, Morrissey D. Patellar tendinopathy and potential risk factors: an international database of cases and controls. *Clin J Sport Med.* 2017;27(5):468–74.
90. Van der Worp H, de Poel HJ, Diercks RL, van den Akker-Scheek I, Zwerver J. Jumper's knee or lander's knee? A systematic review of the relation between jump biomechanics and patellar tendinopathy. *Int J Sports Med.* 2014;35(8):714–22.
91. Dix J, Marsh S, Dingenen B, Malliaras P. The relationship between hip muscle strength and dynamic knee valgus in asymptomatic females: a systematic review. *Phys Ther Sport.* 2019;37:197–209.
92. Bourne MN, Williams MD, Opar DA, Al Najjar A, Kerr GK, Shield AJ. Impact of exercise selection on hamstring muscle activation. *Br J Sports Med.* 2017;51(13):1021–8.
93. Zebis MK, Skotte J, Andersen CH, Mortensen P, Petersen HH, Viskaer TC, et al. Kettlebell swing targets semitendinosus and supine leg curl targets biceps femoris: an EMG study with rehabilitation implications. *Br J Sports Med.* 2013;47(18):1192–8.
94. Hardesty K, Hegedus EJ, Ford KR, Nguyen AD, Taylor JB. Determination of clinically relevant differences in frontal plane hop tests in Women's collegiate basketball and soccer players. *Int J Sports Phys Ther.* 2017;12(2):182–9.



Biomechanics of Upper Extremity Movements and Injury in Basketball

5

Jeffrey B. Taylor, Kevin R. Ford, and Eric J. Hegedus

5.1 Introduction

Whereas the lower extremities provide the means for a basketball athlete to move explosively and position their body all over the court, the upper extremities serve as the main contact point for the ball, necessitating coordination and skill. Indeed, the upper extremities are at times required to perform powerful movements to contain an opposing offensive player, fend off defenders, or create room around the basket to rebound; yet, the primary demands of the upper extremities are based on skill during dribbling, passing, and shooting. This balance of power and precision make it difficult for coaches and clinicians to focus training on one aspect of upper extremity biomechanics in the basketball athlete. Though significantly less work has been reported on upper extremity basketball biomechanics than lower extremity

biomechanics, this chapter will detail (1) the global upper extremity movement demands that characterize the sport of basketball, (2) joint specific biomechanics during basketball-specific upper extremity movements, and (3) biomechanical considerations of basketball-related upper extremity injuries.

5.2 Movement Demands

The predominant upper extremity demands of basketball include shooting, dribbling, defending, and passing. Upper extremity movement demands have been significantly less analyzed than lower extremity movements, partly because upper extremity injuries are less common and partly because technology for lower extremity and global movement tracking has evolved more quickly than that for the upper extremity.

The upper extremities are constantly moving in basketball, whether it be for purposeful basketball-specific movements, during running, or in a defensive- or offensive-ready position. Upper extremity movements are challenging to track and categorize, so researchers have defined these movements as any movement overhead ($>90^\circ$ of shoulder elevation). Basketball players have been reported to perform greater than 200 overhead upper extremity movements per game,

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with backcourt (i.e., guards) performing more than frontcourt (forwards, centers) players [1, 2].

Dribbling demands are also highly position-dependent. Backcourt players dribble more frequently (~60 occurrences per game) than front court players (~20) [1, 2]. For backcourt players, each dribbling episode lasts an average of 4 s, over the course of 12–13 m [1]. In all, backcourt players travel greater than 700 m, while dribbling over the course of a game, accounting for 10–15% of the total distance traveled [1, 2].

Jumping may be a good surrogate measure of upper extremity demands, considering that basketball players rarely jump without upper extremity elevation. Basketball players jump approximately 40 times per game [3]. A more detailed analysis of these jumps reveals an average of up to 20 jumps per game for defending shots (i.e., blocking), 12 jumps for jump shots, 13 jumps for rebounds, and 10 jumps for layups [4].

Basketball players also absorb a lot of physical contact through their upper extremities. Specifically, setting screens while on offense or boxing out while rebounding requires upper extremity contact. Analysis shows that both demands occur more frequently in frontcourt players, with up to 49 instances of screen setting and 72 instances of boxing out per game [4].

5.3 Upper Extremity Biomechanics During Basketball Movements

5.3.1 Shooting

Shooting is the most recognizable basketball skill and arguably the most important, considering that winning involves scoring more points than the other team. Shots can come from short- or long range, but average about 4-m from the basket over the course of a game [5]. Biomechanics can help characterize basketball shots into the following: (1) above head (i.e., jump shot), (2) hook shot, (3) layup, (4) tip-in, and (5) dunk [5]. Most biomechanical analyses have focused on the jump shot.

The jump shot requires shoulder flexion, elbow extension, and wrist flexion coupled with hip, knee, and ankle extension, and can be organized into five phases (Fig. 5.1) [6]. The preparation phase involves the athlete positioning the ball close to the body at waist level with the shooting hand behind the ball and the non-shooting hand held laterally on the ball for support during the shooting motion. During the ball elevation phase, the shoulder flexes 90–135° with the elbow in a position of flexion to position the ball for release. The stability phase occurs while the lower extremities are extending and is charac-

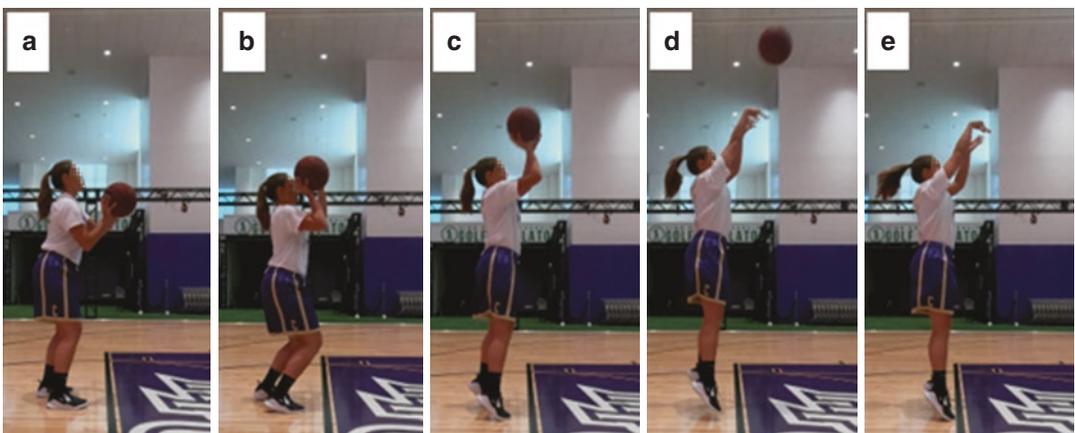


Fig. 5.1 Five phases of the jump shot: (a) preparation phase, (b) ball elevation phase, (c) stability phase, (d) release phase, and (e) inertia phase

terized in the upper extremities by wrist hyperextension. During the release phase, there is rapid and coupled shoulder flexion and elbow extension with wrist flexion. In expert shooters, there is some radioulnar pronation that occurs after full wrist flexion [7]. The final phase, inertia, involves sustained wrist flexion (parallel to the ground) during descending flight and landing.

Biomechanics can significantly impact shot success. The ball trajectory and subsequent angle of entry into the basket are the main factors that determine whether the shot attempt is made or missed [8]. Trajectory can be influenced directly by ball release and indirectly by a number of factors including the distance from the basket, defender presence, body posture, pre-shot movement, field of view, and the expertise level and physical characteristics of the shooter [6].

Extensive analyses of the basketball shot have not concluded in an ideal kinematic pattern for success, but have shown that expert players have less overall variability in their shot mechanics than novice players [6]. Shooters have more success with a greater release angle of the ball (49–60° from parallel) [9], which increases the vertical displacement of the ball and maximizes the rim's width upon entry [6]. Success also depends on ball release velocity, with lower velocities leading to increased success [6]. This is typically accomplished by large wrist flexion angles to produce back spin (~2–3 revolutions during a foul shot). Release height can also affect ball trajectory but is highly dependent on the shooter's size and jumping ability.

Fact Box

Performance of the basketball shot is highly variable, but, in general, is more successful with higher ball release and ball trajectory.

5.3.2 Dribbling

The kinematics of dribbling has not been well studied. Observational analysis suggests that the dribbling motion incorporates simultaneous

shoulder and elbow extension and wrist and finger flexion. Transverse plane motion (shoulder rotation and forearm supination/pronation) are sometimes elicited during select ball handling techniques and change of direction activities. Dribbling can affect global biomechanics by significantly decreasing running speed. Loss of running speed is more marked when dribbling with the nondominant vs. dominant hand and more during change of direction than straight-line running [10, 11].

5.4 Biomechanical Considerations of Upper Extremity Injuries in Basketball

Injuries to the upper extremity account for 10–15% of all basketball-related injuries and are more frequent during game than practice situations [12–15]. In addition, upper extremity injuries may be more common in male than female basketball players. The hand and wrist are the most often injured upper extremity regions (8–9%), followed by the shoulder and elbow [12–14, 16]. In a profile of women's professional basketball players, injuries to the wrist/hand were second only to ankle sprains in frequency of injury [17]. The epidemiology and biomechanics associated with the most frequent basketball-related injuries are discussed in the subsequent sections. Risk factors and preventative strategies for upper extremity injuries in basketball are poorly understood.

5.4.1 Finger Fracture

Finger fractures are more common in women's than men's basketball and have been reported to occur at a rate of 0.09 per 1000 athletic exposures during games [15]. In a study of pediatric basketball injuries presenting to emergency departments, finger fractures were the third most common injury after ankle and finger sprains, and the frequency of all injuries increased with age [18]. Most finger fractures

are traumatic in nature and are caused by ball contact. Specifically, forced flexion of the proximal interphalangeal joint can cause a central slip injury including a dorsal avulsion fracture. If the forced flexion occurs at the distal interphalangeal joint (DIP), this can cause a tear to the terminal extensor tendon or a dorsal avulsion fracture which will lead to mallet finger (or “bony mallet finger” in case of a bony avulsion), a loss of DIP extension, and a flexed posture of that joint [19, 20]. There is some data to suggest that the fourth and fifth digits are most often involved and that most of the fractures are non-displaced or minimally displaced. The average time missed from basketball is about 20–33 days [21, 22].

5.4.2 Thumb Sprain

Thumb sprains occur equally as frequent in men’s and women’s basketball at a rate of 0.09 injuries per 1000 athletic exposures [15, 23]. Most sprains of the thumb are of the ulnar collateral ligament (UCL) which is injured via a radial deviation or abduction force to the metacarpophalangeal (MCP) joint. This force is possible in basketball when falling on an outstretched hand with thumb abducted, getting caught against an opponent jersey, or when having a shot blocked via a sweeping motion. An acute tear of the UCL causes pain, swelling, and instability, and over time, the thumb can subsequently show signs of osteoarthritis [24]. In a study of National Basketball Association (NBA) players over 5 years [22], thumb ligament tears were the least frequent injury of the hand but required the longest time to return to play (average of 67.5 days) and all required surgery.

5.4.3 Wrist Sprain

Wrist sprains occur more in men’s than women’s basketball, with a large majority occurring during game competition (0.15 injuries per 1000 athletic exposures) [25]. The term wrist sprain can encompass a wide range and severity of patholo-

gies including scapholunate ligament tears which can cause wrist instability and limit either the wrist cock or follow-through during shooting. Be careful to rule out a scaphoid fracture which presents with exquisite tenderness in the anatomic snuff box (radial side of wrist) and triangular fibrocartilage injury which presents with pain on the ulnar side of the wrist. Scapholunate tears are the result of forced wrist hyperextension and can happen in basketball with tripping, while landing awkwardly from jumping, when drawing an offensive charge foul, or when diving for a ball and using the wrist/hand to dissipate force. The scaphoid and lunate are in the proximal row of carpals along with the triquetrum and pisiform and are connected by the scapholunate interosseus ligament (SLIL). With wrist extension (cocking phase of shooting), this proximal row rotates into extension and with wrist flexion (release and follow-through), the proximal row rotates into flexion.

5.4.4 Shoulder Injury

The rate of injury of the shoulder is the same in boys and girls high school basketball although boys are more often diagnosed with a sprain/strain and girls with a dislocation/separation [26]. The majority of these athletes, both male and female, return to play in less than 1 week after injury. The most common mechanism of injury for girls’ basketball players is contact with another player (67%) or with the floor (25%). Boys are frequently injured via noncontact events (14%) but by far, the largest number of shoulder injuries in boys is via contact with the playing apparatus (30%) [26]. Shoulder instability injuries can range from subluxation to dislocation. In women’s basketball, shoulder subluxation is the eighth most frequent injury during game competition (0.11 injuries per 1000 athletic exposures) [15]. Acromioclavicular joint injuries can also result from a contact mechanism. Specifically, high inferior or posterior directed forces from a fall, other player, or playing apparatus directly to the acromion can cause tearing of the acromioclavicular ligaments.

Fact Box

Upper extremity injuries in basketball are largely the result of contact that elicits high forces with the joint in a suboptimal position. This contact can occur with the ball, another player, the floor, or the playing apparatus.

5.4.5 Considerations for Upper Extremity Injury Prevention

The majority of upper extremity injuries in basketball are the result of contact; thus, injury prevention is challenging. Clinicians would need to remove the chance of contact, which is unrealistic, or attempt to strengthen the active restraints (i.e., muscles) around the area to potentially withstand the high forces associated with contact mechanisms. Theoretically, preventing upper extremity overuse injuries is more attainable, though evidence is mixed on the risk factors for upper extremity injury and whether neuromuscular-based programs can reduce risk [27]. One randomized controlled trial has shown promise, reporting a 22–28% risk reduction after focusing on the improvement of glenohumeral internal rotation range of motion, external rotator strength, and scapular control [28].

5.5 Considerations for Rehabilitation and Return to Performance

After injury the return to activity by an athlete can be viewed on a continuum progressing from return to participation through return to sport and culminating with return to performance [29]. General considerations of upper extremity rehabilitation and return to performance are similar to the lower extremity in that the rehabilitation professional, in charting the path back to sport and maximum performance, should consider not only tissue healing time and load vs capacity of the tissue but also things like limb dominance, position played, competitive level, time of sea-

son, cardiovascular conditioning, and psychosocial factors. Most rehabilitation programs consist of an initial focus on impairments like pain and lost mobility, include progression to regional functional issues via strengthening, movement correction, and endurance training, and end by addressing global systems through neuromuscular retraining and sport-specific training. Examples of open- and closed-chain exercise progressions can be seen in Figs. 5.2, 5.3, and 5.4. Generally, patient self-report and physical performance measures are used along the continuum to help with the decision to progress the athlete and ultimately to decide on the return to activity continuum. Physical performance tests such as the closed kinetic chain upper extremity stability test (CKCUEST) and unilateral shot put can help with return to play decision-making (Fig. 5.5) [30].

5.5.1 Finger Fracture

Most basketball players with a finger fracture return to sport without limitation and with full healing after a period of immobilization via either splinting or buddy taping [31, 32]. Should surgery be required, most surgeons allow return to play 3–4 weeks after a non-displaced metacarpal fracture although basketball surgeons may be more conservative [33]. Rehabilitation generally consists of reducing pain and swelling with modalities and compression wrapping followed by motion exercises, strengthening exercises, and dexterity training including speed of movement for the fast-twitch muscle-dominated hand.

5.5.2 Thumb Sprain

Grade I and II sprains of the UCL are treated with a thumb spica splint for up to 4 weeks followed by strengthening and range of motion exercises. The timeline can be more aggressive depending on position played and whether the dominant or nondominant hand is injured.

For a Grade III sprain of the UCL (complete tear), most surgeons allow return to pro-



Fig. 5.2 Example of closed-chain upper extremity rehab progression: (a–c) wall push up with neutral wrist position, (d, e) prone push up with neutral wrist position, (f, g) uneven pushup in extreme wrist extension

tected play 2–6 weeks and to unprotected play 6–12 weeks after surgery to repair a thumb UCL tear. Basketball associated surgeons may be more conservative [33]. The majority of surgeons will repair the completely torn UCL immediately, but

almost as many are willing to wait for the end of the season if it is 6 weeks or less away [33]. Patients are generally in a thumb spica cast for 2–4 weeks after surgery followed by a removable thumb spica splint for another 2 weeks. At



Fig. 5.3 Example of open-chain upper extremity dribbling progression: (a) wall dribble, (b) upright dribble, (c) small ball dribble, (d) forward/backward dribble, (e) one-hand cross dribble, (f) figure-eight dribble

4 weeks postoperation, range of motion begins, while at 6 weeks, strengthening can begin with the goal of return to sport by 12 weeks.

5.5.3 Wrist Sprain

Wrist sprain is an inexact diagnosis that may encompass many injuries. Scapholunate tears, scaphoid fracture, and triangular fibrocartilage complex (TFCC) tears are more common basketball injuries to the wrist. The scapholunate joint relies heavily on the SLIL for stability. Whether the choice is made for conservative management or surgical management with postoperative rehabilitation, protection of the joint so that healing can take place is key. In the acute phase

of recovery, maintaining stability of the joint is important. Stability can be maintained by splinting and by motion and strengthening performed in a way that minimizes joint stress. With regard to the scapholunate joint, much attention has been given to exercises involving the dart throwers motion and wrist circumduction. In the dart throwers motion, there is little movement of the scapholunate joint and yet the exercise activates the stabilization-friendly muscles of the extensor carpi radialis longus, extensor carpi radialis brevis, and the flexor carpi ulnaris [34]. Despite making empirical sense, the use of dart throwers motion has not been studied in large trials so caution is recommended [35]. Circumduction has been advocated as being in the same plane as the dart throwers motion.



Fig. 5.4 Example of open-chain upper extremity passing progression: (a) one-hand chest pass, (b) two-hand chest pass, (c) wrap around pass, (d) overhead pass

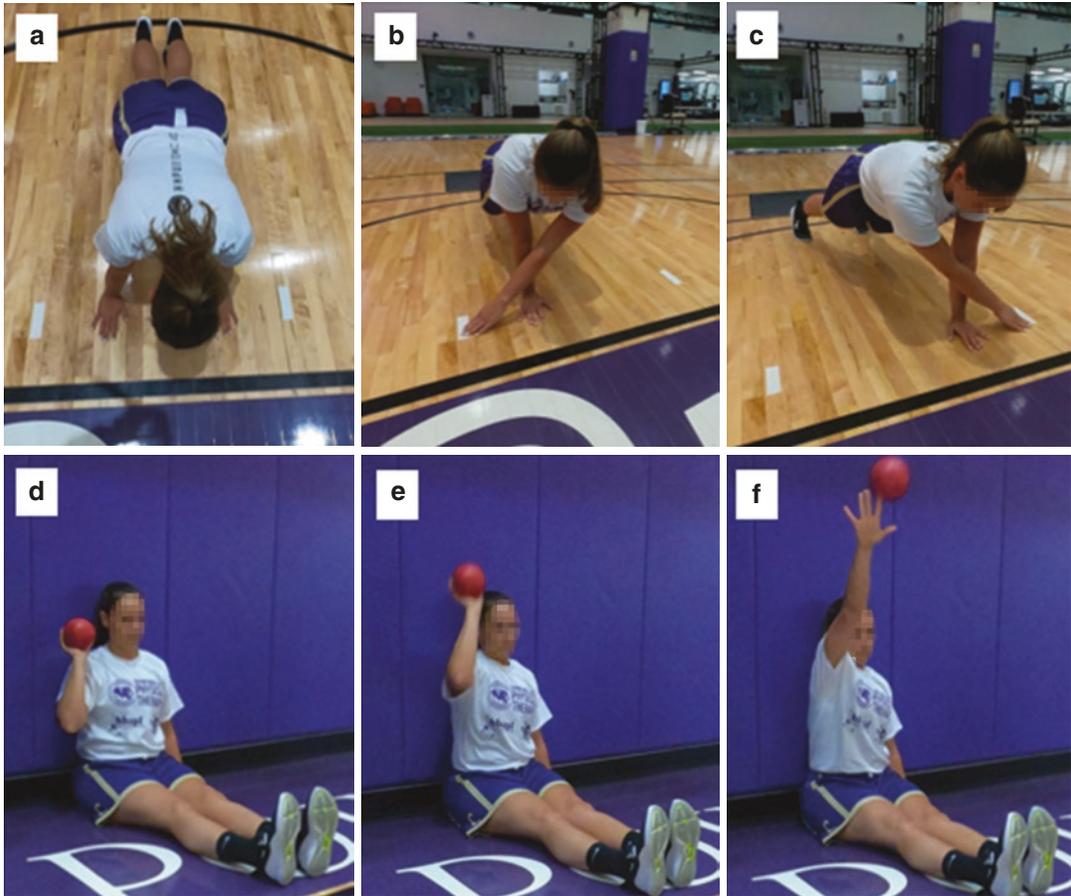


Fig. 5.5 Upper extremity physical performance tests, including: (a–c) closed kinetic chain upper extremity stability test, (d–f) unilateral shot put test

The scaphoid is the most commonly fractured carpal bone and like scapholunate injury, it is caused by a fall on an outstretched hand. Management of this injury depends on the severity and location of the fracture with less severe fractures (distal pole and non-displaced) treated through casting/splinting and more severe fractures treated via internal fixation [36]. If casted, the cast typically remains in place for 8–12 weeks if the middle third is fractured and 12–24 weeks if the proximal third is fractured. If surgery is performed, there is a period of immobilization in either a cast or thumb spica splint. After 2–4 weeks, range of motion is begun.

Tears of the TFCC are generally divided into traumatic and degenerative. Traumatic injuries are often to the periphery and involve the radioulnar ligaments, and degenerative tears affect the articular disc [37]. In basketball, the most likely mechanism is a fall on an outstretched hand. Peripheral injuries often require surgery. As with other injuries of the wrist, there is a post-surgical period of immobilization (4–6 weeks in this case).

The rehabilitation approach for all of the above injuries is generally a period of immobilization based on tissue healing followed by active motion, passive motion, proprioceptive exercises,

strengthening based on proper load, and the addition of functional activities. The rehabilitation specialist is reminded that cardiovascular conditioning in the basketball athlete is very important and with injuries of the wrist, cardiovascular cross-training can be initiated early in the recovery process.

5.5.4 Shoulder Injury

High school athletes with a diagnosis of sprain/strain or subluxation reportedly return to basketball within a week [26]. Research suggesting immediate surgery for episodes of instability has largely been performed on athletes in contact sports [23]. A recent study using return to sport as the criterion for success/failure showed that 78% of those treated with joint mobilization, strengthening, endurance training, and neuromuscular retraining returned to sport within 1 year compared to 92% treated with surgery [38]. The differences in these results are likely multifactorial including age and competitive-level differences, the definition of success/failure, and the severity of the injury, to name a few. Regardless, the rehabilitation should be directed at the entire shoulder complex including the glenohumeral and scapulothoracic joints with a focus of normalization of motion, balance and strength of rotator cuff muscles, and a gradual increase in the sport-specific load [39].

Fact Box

When rehabilitating an upper extremity injury on a basketball athlete, it is important to keep in mind:

1. Stability to promote healing of the injured tissue.
2. Early cross-training to maintain cardiovascular conditioning.
3. Progression to sport- and ball-specific drills.

Take Home Message

In summary, the upper extremity must have the skill and agility to manipulate the ball, and also must have the strength and stability to withstand high contact forces. Incorporating progressive ball-specific exercise during rehabilitation can help in athlete's return to the court.

References

1. Scanlan A, Dascombe B, Reaburn P. A comparison of the activity demands of elite and sub-elite Australian men's basketball competition. *J Sports Sci.* 2011;29(11):1153–60.
2. Scanlan AT, Dascombe BJ, Reaburn P, Dalbo VJ. The physiological and activity demands experienced by Australian female basketball players during competition. *J Sci Med Sport.* 2012;15(4):341–7.
3. Taylor JB, Wright AA, Dischiavi SL, Townsend MA, Marmon AR. Activity demands during multidirectional team sports: a systematic review. *Sports Med.* 2017;47(12):2533–51.
4. Ribeiro RA, Calderani A, Monezi LA, Misuta MS, Mercadante LA. Physical activity demands in elite basketball games. In: 33rd International conference on biomechanics in sports; Poitiers, France; 2015.
5. Erculj F, Strumbelj E. Basketball shot types and shot success in different levels of competitive basketball. *PLoS One.* 2015;10(6):e0128885.
6. Okazaki VH, Rodacki AL, Satern MN. A review on the basketball jump shot. *Sports Biomech.* 2015;14:190–205.
7. Knudson D. Biomechanics of the basketball jump shot—six key teaching points. *JOPERD.* 1993;64(2):67–73.
8. Miller S, Bartlett RM. The effects of increased shooting distance in the basketball jump shot. *J Sports Sci.* 1993;11(4):285–93.
9. Khelifa R, Aouadi R, Shephard R, Chelly MS, Hermassi S, Gabbett TJ. Effects of a shoot training programme with a reduced hoop diameter rim on free-throw performance and kinematics in young basketball players. *J Sports Sci.* 2013;31(5):497–504.
10. Scanlan AT, Wen N, Spiteri T, Milanovic Z, Conte D, Guy JH, et al. Dribble deficit: a novel method to measure dribbling speed independent of sprinting speed in basketball players. *J Sports Sci.* 2018;36(22):2596–602.
11. Ramirez-Campillo R, Gentil P, Moran J, Dalbo VJ, Scanlan AT. Dribble deficit enables measure-

- ment of dribbling speed independent of sprinting speed in collegiate, male, basketball players. *J Strength Cond Res.* 2019. <https://doi.org/10.1519/JSC.0000000000003030>.
12. Andreoli CV, Chiaramonti BC, Buriel E, Pochini AC, Ejnisman B, Cohen M. Epidemiology of sports injuries in basketball: integrative systematic review. *BMJ Open Sport Exerc Med.* 2018;4(1):e000468.
 13. Clifton DR, Hertel J, Onate JA, Currie DW, Pierpoint LA, Wasserman EB, et al. The first decade of web-based sports injury surveillance: descriptive epidemiology of injuries in US High School Girls' Basketball (2005–2006 through 2013–2014) and National Collegiate Athletic Association Women's Basketball (2004–2005 through 2013–2014). *J Athl Train.* 2018;53(11):1037–48.
 14. Clifton DR, Onate JA, Hertel J, Pierpoint LA, Currie DW, Wasserman EB, et al. The first decade of web-based sports injury surveillance: descriptive epidemiology of injuries in US High School Boys' Basketball (2005–2006 through 2013–2014) and National Collegiate Athletic Association Men's Basketball (2004–2005 through 2013–2014). *J Athl Train.* 2018;53(11):1025–36.
 15. Agel J, Olson DE, Dick R, Arendt EA, Marshall SW, Sikka RS. Descriptive epidemiology of collegiate women's basketball injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. *J Athl Train.* 2007;42(2):202–10.
 16. Ito E, Iwamoto J, Azuma K, Matsumoto H. Sex-specific differences in injury types among basketball players. *Open Access J Sports Med.* 2015;6:1–6.
 17. McCarthy MM, Voos JE, Nguyen JT, Callahan L, Hannafin JA. Injury profile in elite female basketball athletes at the Women's National Basketball Association combine. *Am J Sports Med.* 2013;41(3):645–51.
 18. Pappas E, Zazulak BT, Yard EE, Hewett TE. The epidemiology of pediatric basketball injuries presenting to US emergency departments: 2000–2006. *Sports Health.* 2011;3(4):331–5.
 19. Fraser MA, Grooms DR, Guskiewicz KM, Kerr ZY. Ball-contact injuries in 11 National Collegiate Athletic Association Sports: the Injury Surveillance Program, 2009–2010 through 2014–2015. *J Athl Train.* 2017;52(7):698–707.
 20. Carruthers KH, Skie M, Jain M. Jam injuries of the finger: diagnosis and Management of injuries to the interphalangeal joints across multiple sports and levels of experience. *Sports Health.* 2016;8(5):469–78.
 21. Yde J, Nielsen AB. Sports injuries in adolescents' ball games: soccer, handball and basketball. *Br J Sports Med.* 1990;24(1):51–4.
 22. Morse KW, Hearn KA, Carlson MG. Return to play after forearm and hand injuries in the National Basketball Association. *Orthop J Sports Med.* 2017;5(2):2325967117690002.
 23. Dickens JF, Rue JP, Cameron KL, Tokish JM, Peck KY, Allred CD, et al. Successful return to sport after arthroscopic shoulder stabilization versus nonoperative management in contact athletes with anterior shoulder instability: a prospective multicenter study. *Am J Sports Med.* 2017;45(11):2540–6.
 24. Schroeder NS, Goldfarb CA. Thumb ulnar collateral and radial collateral ligament injuries. *Clin Sports Med.* 2015;34(1):117–26.
 25. Dick R, Hertel J, Agel J, Grossman J, Marshall SW. Descriptive epidemiology of collegiate men's basketball injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. *J Athl Train.* 2007;42(2):194–201.
 26. Bonza JE, Fields SK, Yard EE, Dawn Comstock R. Shoulder injuries among United States high school athletes during the 2005–2006 and 2006–2007 school years. *J Athl Train.* 2009;44(1):76–83.
 27. Asker M, Brooke HL, Walden M, Tranaeus U, Johansson F, Skillgate E, et al. Risk factors for, and prevention of, shoulder injuries in overhead sports: a systematic review with best-evidence synthesis. *Br J Sports Med.* 2018;52(20):1312–9.
 28. Andersson SH, Bahr R, Clarsen B, Myklebust G. Preventing overuse shoulder injuries among throwing athletes: a cluster-randomised controlled trial in 660 elite handball players. *Br J Sports Med.* 2017;51(14):1073–80.
 29. Arden CL, Glasgow P, Schneiders A, Witvrouw E, Clarsen B, Cools A, et al. 2016 Consensus statement on return to sport from the First World Congress in sports physical therapy, Bern. *Br J Sports Med.* 2016;50(14):853–64.
 30. Tarara DT, Fogaca LK, Taylor JB, Hegedus EJ. Clinician-friendly physical performance tests in athletes part 3: a systematic review of measurement properties and correlations to injury for tests in the upper extremity. *Br J Sports Med.* 2015;50:545–51.
 31. Rettig AC, Ryan R, Shelbourne KD, McCarrroll JR, Johnson F Jr, Ahlfeld SK. Metacarpal fractures in the athlete. *Am J Sports Med.* 1989;17(4):567–72.
 32. Guss MS, Begly JP, Ramme AJ, Hinds RM, Karia RJ, Capo JT. Performance outcomes after metacarpal fractures in National Basketball Association Players. *Hand (New York, NY).* 2016;11(4):427–32.
 33. Dy CJ, Khmel'nitskaya E, Hearn KA, Carlson MG. Opinions regarding the management of hand and wrist injuries in elite athletes. *Orthopedics.* 2013;36(6):815–9.
 34. Wolff AL, Wolfe SW. Rehabilitation for scapholunate injury: application of scientific and clinical evidence to practice. *J Hand Ther.* 2016;29(2):146–53.
 35. Bergner JL, Farrar JQ, Coronado RA. Dart thrower's motion and the injured scapholunate interosseous ligament: a scoping review of studies examining motion, orthoses, and rehabilitation. *J Hand Ther.* 2019. <https://doi.org/10.1016/j.jht.2018.09.005>.

36. Winston MJ, Weiland AJ. Scaphoid fractures in the athlete. *Curr Rev Musculoskelet Med.* 2017;10(1):38–44.
37. Jaworski CA, Krause M, Brown J. Rehabilitation of the wrist and hand following sports injury. *Clin Sports Med.* 2010;29(1):61–80, table of contents.
38. Shanley E, Thigpen C, Brooks J, Hawkins RJ, Momaya A, Kwapisz A, et al. Return to sport as an outcome measure for shoulder instability: surprising findings in nonoperative management in a high school athlete population. *Am J Sports Med.* 2019;47(5):1062–7.
39. Cools AM, Borms D, Castelein B, Vanderstukken F, Johansson FR. Evidence-based rehabilitation of athletes with glenohumeral instability. *Knee Surg Sports Traumatol Arthrosc.* 2016;24(2):382–9.



Nutrition and Hydration in Basketball Athletes

6

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6.1 Introduction

Achieving optimal athlete nutrition is one of the greatest challenges in developing strategies favoring the adaptations that occur as a result of training and competition. Although scientific evidence on a set of dietary recommendations or nutritional requirements during exercise has been described [1–3], in sports such as basketball, athletes need to maintain a diet in accordance to the general recommendations for macro- and micronutrients in the general population. An individualized approach is needed to meet each athlete's nutritional and hydration needs, assessing nutritional status and offering guidance and advice based on evidence in order to ensure better adaptation to exercise, improve

exercise performance, and enhance the recovery processes which players accumulate throughout the season.

6.2 Physiological Demands

Basketball has established itself as one of the most popular sports in many countries around the globe. In the scientific literature, many publications [4, 5] have addressed the physical and physiological attributes of men and women playing basketball at different competitive levels. During the competitive season, elite basketball players train on a daily basis, often twice a day during the preseason period; play one or two games per week; and take part in international tournaments [6]. Specifically, the National Basketball Association (NBA) comprises 82 regular season games (approximately 3.4 games per week on average, with varying frequencies—often with games on consecutive days), followed by up to 28 playoff games [7]. Basketball is classified within team sports as an intermittent court-based team sport, due to the intermittent nature of the combination of efforts at low and high intensity between offense and defense, with an energy source that alternates energy systems during sport [8]. Current studies [9] have shown that elite basketball players are highly active and engage in a number of intense physical activities (e.g., running, jumping, cutting, and shuffling).

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While specific physiological demands may differ between team sports (e.g., game frequency, season length, specific player position requirements), a common feature is the nature of team sports, with “bursts” of high intensity interspersed with periods of less intense activity or with rest periods [4, 7, 9–12]. In basketball, the comparative analysis of energy demands depending on player position has revealed higher intensity activities in guards compared to forwards and centers (5.9% vs. 5.4% and 4.5% sprinting; 9.3% vs. 9.2% and 7.9% high-intensity shuffling, respectively) [13]. In particular, during 40-min matches, both male and female basketball players cover a distance of 5–6 km at average physiological intensities above lactate threshold and 85% of maximal heart rate (HRmax) [5]. In a recent review [14, 15], significant differences according to playing positions were observed between guards, forwards, and centers, as well as according to playing level where more skilled players experienced greater demands (international versus national: 94.6% vs. 90.8% HRmax, 94.4% vs. 91.8% HRmax). Taking the physiological demands into account, the energy and macronutrient intake in a basketball player’s diet should suffice to cover the energy demands, substrate availability, training adaptations, and competition.

Key Points

- During live playing time across 40-min matches, male and female basketball players cover a distance of 5–6 km at an average physiological intensity above 85% HRmax.
- Basketball combines efforts at low and high intensity between offense and defense, with an energy source that alternates energy systems during sport.
- Player position comparisons show guards to perform more high-intensity activities than forwards and centers.
- Playing level also shows more skilled players to experience greater demands (international versus national).

6.3 Energy Requirements in Basketball

Optimum energy intake is the key to the athlete’s diet and is determined by the intake of macronutrients and micronutrients in order to support optimal physiological functions and body weight in response to the continuous physical demands arising from exercise. In general, an athlete’s energy intake (EI) comes from the consumption of foods, liquids, and supplements that can be recorded using different methods including retrospective food intake questionnaires [16]. An athlete’s energy needs in basketball and team sports in general will depend on the training and competition cycle, the duration and frequency of matches, the length of the season, the training phase, the number of players, and substitutions [17].

Energy balance, therefore, occurs when energy consumption is equal to total energy expenditure (TEE), or the sum of the energy expended as resting energy expenditure (REE), the thermal effect of food, the thermal effect of daily activity, and thermogenesis of resting activity. Spontaneous physical activity is also included in the TEE [18]. On the field, a practical and economic way to assess the daily energy expenditure of an athlete is to use predictive functions such as the Cunningham [19] and Harris–Benedict equations [20]. These equations have allowed the calculation of TEE, taking into account the REE multiplied by activity factor. Nevertheless, categorization of the activity factor could imply error causing us to over- or underestimate the TEE of a basketball athlete. Likewise, TEE estimation based on metabolic equivalents (METs) [21] can be used as another cost-effective tool in order to accurately prescript the energy consumption requirements. The quantification of TEE by METs is widely extended in research, though the trend is changing toward more precise methodologies. In fact, human TEE can be accurately evaluated using the doubly labeled water (DLW) gold standard while avoiding any interference with training activities [22]. DLW is a variant on indirect calorimetry where TEE is derived from the measurement of carbon dioxide production. This method involves enriching the body water

of a subject with heavy hydrogen (^2H) and heavy oxygen (^{18}O), and then determining the difference in washout kinetics between both isotopes, based on the estimation of the rate of CO_2 elimination from the body [23].

Although the indirect estimates of energy intake in team sport athletes were 3660 kcal/day and 2064 kcal/day for males ($n = 819$) and females ($n = 283$), respectively [17], Silva et al. [24, 25] accurately measured TEE and EI in elite junior basketball players and different team sport athletes using the DLW method. The TEE observed was 3897 ± 625 and 3002 ± 466 kcal/day [25]. The follow-up study showed that TEE and REE significantly increased by $15.7 \pm 11.2\%$ and $7.0 \pm 22.2\%$ throughout the annual season. Specifically, basketball players showed an increased TEE in both males and females (532 ± 402 and 372 ± 404 kcal/day, respectively) from the beginning of the season to the competitive period. This would mean an increase in TEE as well as in the energy requirements throughout the season as the latter progresses from the preparatory period to the competitive period. In relation to energy requirements, the average EI values observed were 4764 ± 622 and 3677 ± 330 kcal/day for male and female basketball, respectively.

The abovementioned study characterized the energy balance from the beginning of the season to the main stage of competition, where the energy requirements were expected to increase. As a result, an increase in fat-free mass content was described for those basketball athletes and other included team sports [25]. A deficient energy intake will negatively affect lean tissue (which will be used for fuel), strength, and endurance, and will also result in impaired immune, endocrine, and musculoskeletal function among others aspects [26], which could lead to an increased risk of fatigue, injury, and illness, and to an extended or insufficient recovery process. It is essential to underscore the importance of classifying energy expenditure according to the position of each player, for as we have mentioned, the intensity and volume of actions in basketball differ according to players' position [14, 15].

Key Points

- An athlete's energy needs will depend on the training and competition cycle, the duration and frequency of matches, the length of the season, the training phase, the number of players, and substitutions
- DLW is used as a gold standard for estimating TEE; however, indirect methodologies such as the Cunningham and Harris–Benedict equations and METs also help estimate TEE
- In team sport athletes, TEE observed by DLW methodology was 3897 ± 625 and 3002 ± 466 kcal/day
- The average EI values observed were 4764 ± 622 and 3677 ± 330 kcal/day for male and female basketball players, respectively
- An increase in TEE and REE in male and female basketball players was observed from the beginning of the season to the competitive period

6.4 Macronutrient Status in Basketball

The distribution of each macronutrient in the athlete diet should be defined by the intake of elements such as carbohydrates, proteins, and lipids, present in all foods that form part of a healthy and balanced diet. The scientific literature and organizations, including the International Society of Sports Nutrition (ISSN) and the American College of Sports Medicine (ACSM), provide macronutrient guidelines highlighting the importance of optimizing health and sports performance throughout training and the competitive season [1, 2, 27–30].

6.4.1 Carbohydrate Requirements

Carbohydrates (CHO) have received special consideration in sports nutrition because of their importance for training and competition intensity.

As mentioned, basketball is characterized by an intermittent activity pattern with high-intensity activity bursts followed by rest pauses or periods of low activity. Even when working with the higher intensities, CHO will provide a higher yield of ATP per volume of oxygen that can be delivered to the mitochondria, thereby optimizing exercise efficiency [31]. Carbohydrates serve as the main substrate of the anaerobic and oxidative pathways, providing key fuel for the brain and central nervous system, and are a versatile substrate for muscle work. Considering that CHO are a limited fuel, special attention should be paid to daily dietary intake through a controlled suitable diet (Fig. 6.1).

Dietary guidelines for CHO intake in athletes should be expressed relative to body weight instead of as a percentage of daily energy intake. A recent study [32] showed that CHO intake was successfully maintained across the season in basketball athletes, although lower daily intakes were observed (3.3–4.0 g/kg/day) compared to the recommended amounts for athletes [1, 33]. Another recent study [34] has estimated a CHO intake below 6 g/kg/day for more than half of the junior elite basketball athletes (in 56% of athletes; 95%CI 43–69%), this again falling below the recommendations established by the ASCM or ISSN for CHO (6–10 g/kg/day) [1, 33]. In

this population the CHO distribution was within a range of 48–51% of the total energy intake, even though preseason intakes were greater than in-season intakes, this also being below some previous recommendations [1]. Specifically, basketball players should have a goal carbohydrate intake of >6 g/kg/day and within a range of 55–65% of the total energy intake during the heavy period of training and competition [35]. Comparing these results with those reviewed by Holway et al. [17], we observed that CHO intake was also around 49% of energy intake (Fig. 6.2), although higher values for CHO relative to body mass were reported in males (5.6 ± 1.3 g/kg/day) and females (4.0 ± 0.7 g/kg/day) (Fig. 6.2).

It is important to mention that the ability to sustain the stop-and-go pattern and the possible alterations in exercise performance indices after certain intense periods of the match remains unknown. Some authors [26] indicate that energy and CHO requirements on match days tend to be higher than on weekly training days, though team sport athletes tend to eat less on these days due to game stress, traveling, or match schedules that alter the normal eating pattern [17]. In this way, the role of CHO would become even more important in order for players to be able to better tolerate the basketball match.

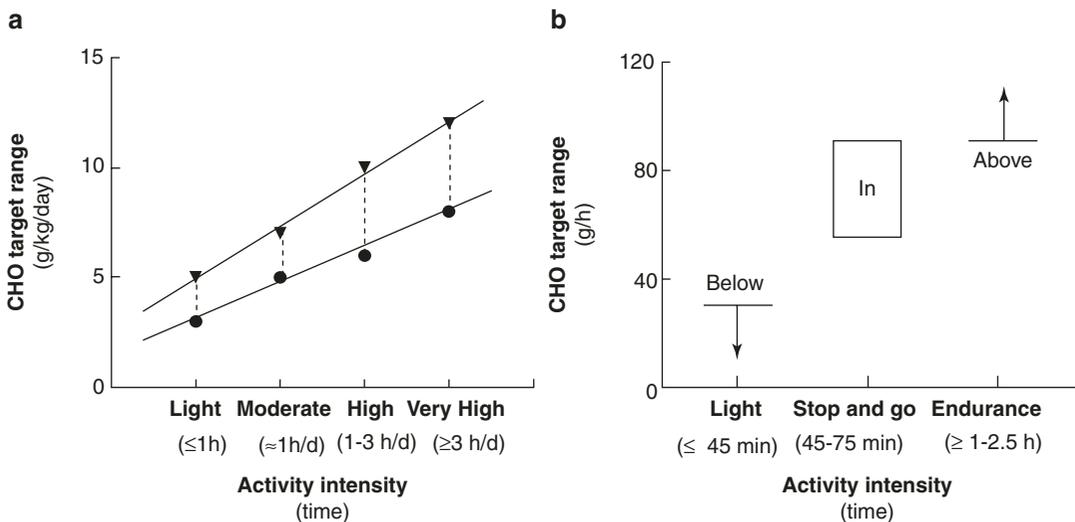
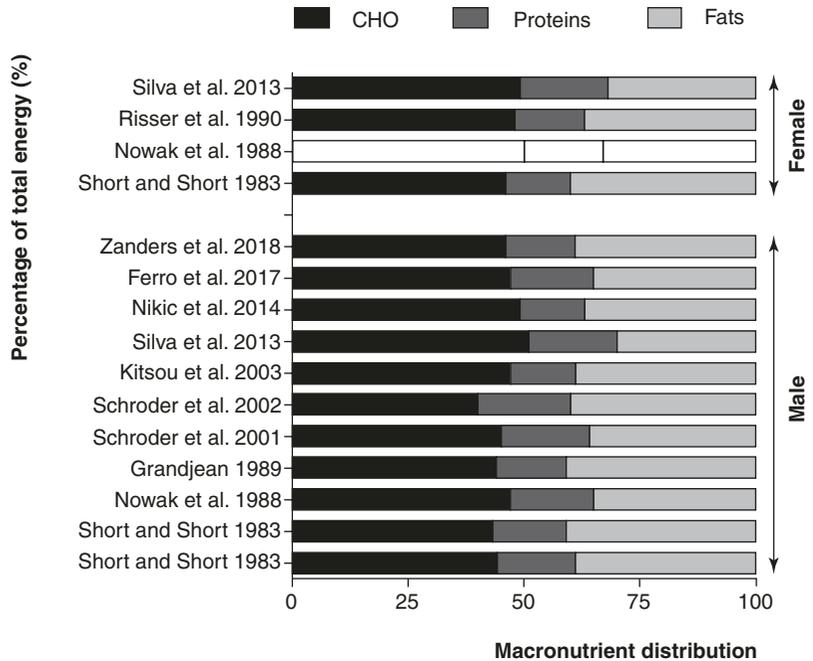


Fig. 6.1 Summary of guidelines for carbohydrate intake by athletes. **(a)** Daily needs for fuel and recovery to provide high carbohydrate availability for different exercise

loads for scenarios. **(b)** Guidelines to promote high carbohydrate availability for optimal performance during competition or key training sessions. (Adapted from [1])

Fig. 6.2 Adequacy macronutrient distribution to total energy intake in basketball athletes [17, 32, 34, 36–38]



The timing of CHO intake over the day and in relation to training can also be adjusted to promote or reduce CHO availability (Fig. 6.3) [35]. An easily digestible meal supplying CHO sources eaten 2–4 h before a game seems to be the best alternative to maintain blood glucose and maximize glycogen stores (low in fat and fiber to facilitate gastric emptying and minimize gastrointestinal distress) [39]. It is assumed that high-glycemic index (HGI) CHO (e.g., instant mashed potato, white rice, sports drink, wholemeal bread, cornflakes, or watermelon) are digested and absorbed more quickly than low-glycemic (LGI) index CHO (e.g., pasta, orange, banana, honey, porridge (from traditional oats) kidney beans, mixed grain bread, yogurt all bran, or milk) [40]. Additionally, Williams and Rollo [40] remarked that athletes may not consume sufficient CHO when recommended to eat LGI foods and so get enough restocking of their glycogen stores. During exercise in basketball athletes, the benefits for exercise performance of ingesting a 6% of CHO-electrolyte solution were evidenced by enhanced physical capabilities during the last quarter [41]. Finally, during the recovery period, additional CHO ingestion (reaching 9 g/kg/day) was compared to a normal CHO intake (5 g/kg

body mass) combined with protein after exhaustive intermittent exercise [42]. In this sense, the consumption of a high-CHO recovery diet made it possible to match performance of the previous day in athletes. In contrast, a normal amount of CHO and an equal energy intake failed to reproduce the performance of the previous day [40]. Therefore, rapidly absorbed CHO together with hydrolyzed whey protein constitute the ideal combination, in 3–4/1 proportion, with 1 g/kg being the recommended amount of CHO [33].

6.4.2 Protein Requirements

Protein is an important nutrient in an athlete's diet, providing a trigger and a substrate for the synthesis of contractile and metabolic proteins, improving structural changes, and contributing to recovery after training sessions [27, 43]. Protein intake may also be a priority in the diet of a basketball player because players differ in weight, height, fat percentage, and muscle mass according to specific playing positions [44, 45].

Protein recommendations in athletes have been previously described by the ACSM and ISSN [1, 3, 33]. Current data suggest that the

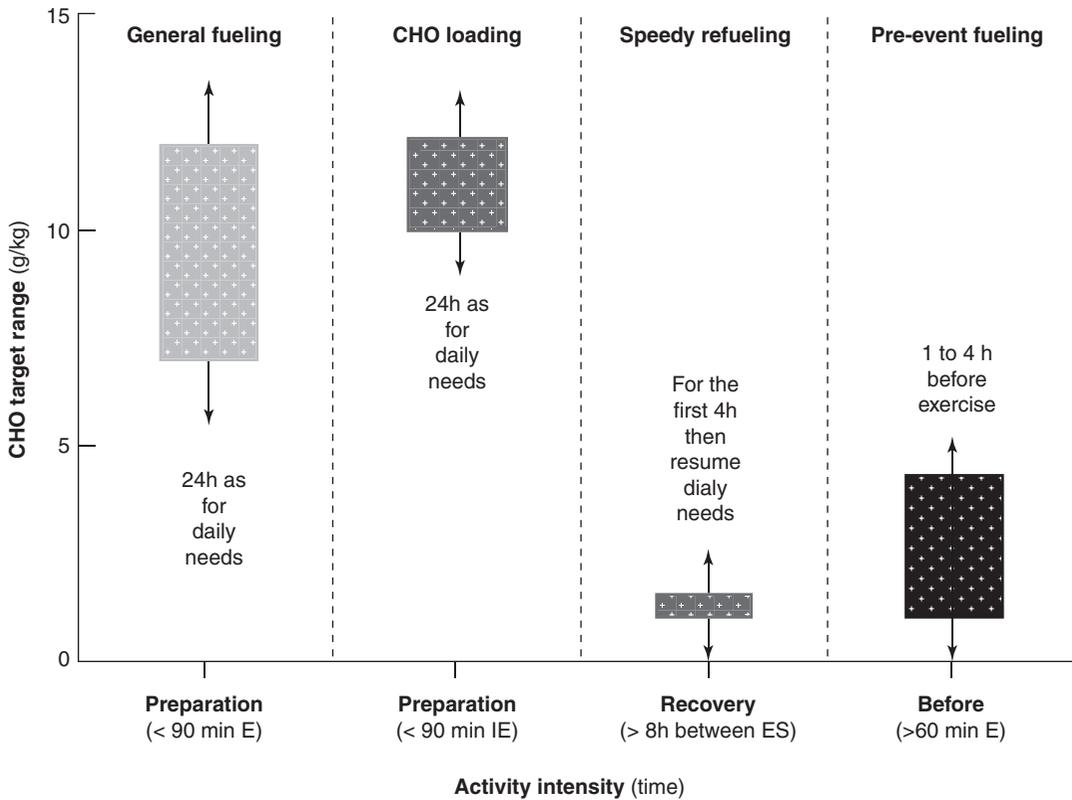


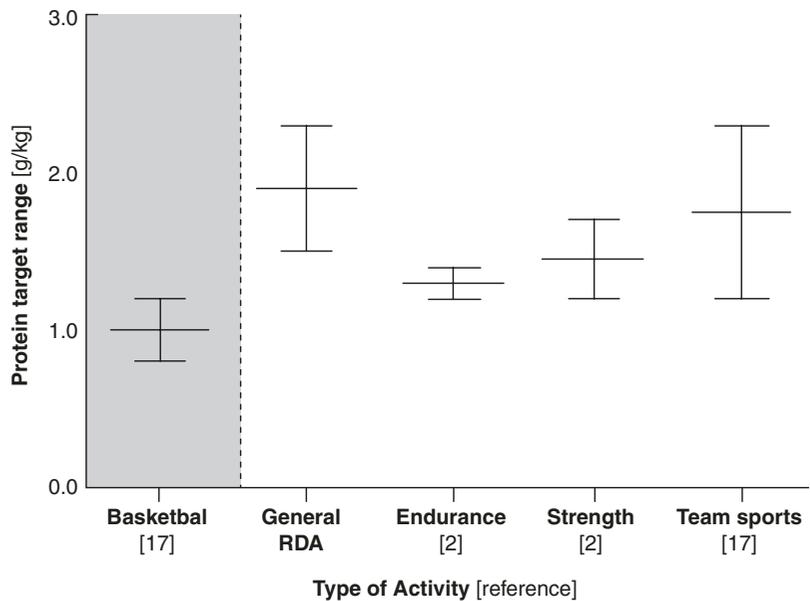
Fig. 6.3 Summary of carbohydrate intake in athletes for acute fueling. (Adapted from [1])

dietary protein intake required to support metabolic adaptation, repair, remodeling, and the renewal of proteins ranges from 1.2 to 2.0 g/kg/day and represents 10–15% of the total energy ingested [33]. Taking the above into account, organisms such as the ADA or the ACSM have established recommendations according to the type of exercise involved (Fig. 6.4). In this respect, endurance disciplines require an intake of 1.2–1.4 g/kg/day, while higher quantities are associated with strength disciplines (1.4–2.0 g/kg/day) [1, 27, 33]. In a systematic review [17], the mean daily protein intake reported for team sport athletes was around 1.2–1.5 g/kg/day for male and female basketball athletes. Similarly, a study carried out in collegiate basketball players [32] showed that daily protein intake tended to change across the season. Specifically, protein intake was lower in the competitive period than during the preparatory phase, with an average intake of around 1.37 g/kg/day, representing

16.6% of total energy intake. Moreover, several studies [46] observed that the contribution of protein to total energy was higher in female than in male team sport athletes (17.8% vs. 16.1%, respectively) (Fig. 6.2). Therefore, the protein intake for basketball or team sport athletes is consistent with the abovementioned recommendations for strength–power disciplines [33].

Protein requirements usually can be covered through the diet and play a key role in combination with CHO. High-quality proteins such as whey, casein, or soy were effectively used for the maintenance, repair, and synthesis of skeletal muscle proteins in response to training [47]. Some studies have shown [1, 43] muscle protein synthesis to be optimized in response to exercise by the consumption of high biological value protein (e.g., egg, beef, pork, concentrated vegetable protein or mixed meals on the stimulation of mammalian target of rapamycin (mTOR) and muscle protein synthesis), providing around 10 g (0.25–

Fig. 6.4 Protein intake recommendations classified by type of activity. (Adapted from [1, 17])



0.3 g/kg body weight) of essential amino acids in the early recovery phase (0–2 h after exercise). Protein intake goals should be considered providing a regular distribution of moderate amounts of high-quality protein across daily training and match distribution, and encompassing most training regimens with annual periodization.

6.4.3 Fat Requirements

Fat provides energy and essential elements of cell membranes and facilitates the absorption of fat-soluble vitamins, including vitamins A, D, and E. Fat is also an important fuel for aerobic exercise. However, at a given exercise intensity and metabolic demand, there can be reciprocal shifts in the proportions of CHO and fat that are oxidized [31]. The ACSM has estimated a range between 20% and 35% of the total energy intake for fats in athletes [1, 33].

Basketball athletes reported high-fat diets reaching approximately 37% of the total energy intake in both the male and female categories [32, 34]. This amount represents an average of 1.37 and 2.12 g/kg/day above the fat recommendations of 0.9–1.1 g/kg/day for males and females, respectively [33]. In other team sports,

fat intake has been estimated to range from 24 to 42% of total energy intake as recorded by 3-day dietary intake questionnaires (reviewed in [17]) (Fig. 6.2). It is important to note that less than 20% of energy from fats affords no benefit in terms of athlete performance. Such a reduction may also compromise the variety of nutrients like fat-soluble vitamins and essential fatty acids [33]. In contrast, although some studies [48] have suggested that a high-fat diet could increase the amount of oxidized fat during exercise, it could also reduce muscular training adaptations, thereby compromising exercise performance.

Therefore, optimal performance in a basketball player will be determined by a diet that is well balanced in terms of macronutrient distribution, which can vary throughout the annual season as well as in relation to player position in the game. As we have observed, the preseason period will require greater amounts of energy as well as a greater contribution of proteins to promote muscle mass building. Subsequently, during the competitive period, the sports nutritionist should work to address the continuous demands of training and competition, adapting the contribution of CHO and protein (55–65% CHO and 10–15% proteins) before, during, and after weekend competitions.

Key Points

- A basketball player needs to consume adequate energy during annual periodization to maintain body weight and health and maximize performance
- A well-balanced diet recommends 6–10 g/kg/day CHO, 1.2–1.7 g/kg/day protein, and 0.9–1.1 g/kg/day fats (55–65% CHO, 10–15% proteins and 20–25% fat, referred to total energy intake)
- The literature shows that basketball players consume a poorly balanced diet, with high protein and fat intake to the detriment of a poor intake of CHO
- Easily digestible CHO sources (2–4 h before a game) are advised. During exercise, basketball athletes benefit from 6% carbohydrate–electrolyte solutions. After the match, rapidly absorbed CHO (1 g/kg being the recommended amount of CHO) together with hydrolyzed whey protein, in 3–4/1 proportion, appears to be a good strategy for recovery
- Special attention should be paid to a balanced diet for preseason and competitive periods within annual basketball periodization

6.5 Micronutrient Intake in Basketball

Vitamins and minerals are also required in many reactions involved with exercise and physical activity, including energy, CHO, fat and protein metabolism, oxygen transfer and delivery, and tissue repair [33]. Moreover, they assist in many metabolic pathways where micronutrients are required, and exercise training may result in biochemical muscle adaptations, synthesis, and repair of muscle tissue during recovery from exercise, and a decrease in oxidative stress promoted by exercise. The intensity, duration, and frequency of training and competition condition whether or not micronutrients are required in greater amounts [49].

6.5.1 Vitamins Adequacy

Water-soluble vitamins comprise the entire B vitamin complex and vitamin C. An adequate intake of B vitamins is important to ensure optimum energy production and muscle tissue construction and repair [50]. For example, thiamine, riboflavin, niacin, pyridoxine (B6), pantothenic acid, and biotin are involved in energy production during exercise [51, 52], while folate and vitamin B12 are necessary for the production of red blood cells and the repair and maintenance of tissues—including the central nervous system. Specifically, an inadequate intake of folic acid has been described in athletes who practice different sports [50, 53, 54], mainly secondary to a deficient intake of total calories, CHO, proteins, and other micronutrients. Some data suggest that exercise may increase the need for these vitamins to as much as twice the current recommended amounts [50, 55]. Although short-term marginal vitamin B deficiencies have not been seen to affect performance, a severe deficiency of vitamin B12 or folic acid or both may result in anemia and decreased endurance performance [53, 56].

Fat-soluble vitamins include vitamins A, D, E, and K, and the body stores them in various tissues, which can result in toxicity if such vitamins are consumed in excessive amounts [57]. Few studies have examined whether exercise increases the need for vitamins in basketball. Additionally, the issue of dietary recommendations for vitamins and particularly for antioxidant vitamins in athletes remains open because the data found in the literature are controversial due to the heterogeneous methods, interventions, and types of exercise involved. One study [38] analyzing vitamin intake reported broad variations among Greek basketball athletes. In this sense, some vitamins were below the recommended values for the healthy population, and this was regarded as a potential limiting factor for athletic performance. On the other hand, elite junior basketball players were seen to have intakes above the recommendations for the majority of the vitamins studied [34]. It should be taken into account that micronutrient intakes below the

recommended levels do not necessarily indicate deficiency. However, since athletes may have nutrient requirements higher than those of the general population, intakes below the reference values theoretically would be inadequate for athletes [29, 54]. The estimation of nutrient density could be useful for assessing the quality of the diet and for identifying insufficient intake considering the total energy ingested.

Antioxidant nutrient requirements and exercise-induced oxidative stress raises the question of the optimal conditions for the adaptation of antioxidants [55]. Although several studies have shown specific vitamins to possess various health benefits (e.g., vitamin E and vitamin C), only a few studies have observed an ergogenic value of vitamins for athletes (reviewed in [55]). For example, vitamin C and E supplementation in maximal exercising basketball players may strengthen the antioxidant defense system by decreasing reactive oxygen species (ROS) [58]. Optimum vitamin C bioavailability is shown to be reached with an intake of 200 mg/day [57]. Consequently, supplementing above this intake threshold has no effect on the vitamin C plasma concentrations. Hypothetically, the decrease in vitamin C and of other antioxidants [59] in the early days of recovery from strenuous or prolonged exercise—particularly if muscle damage and inflammatory responses have been induced—may be associated to increased vitamin C utilization due to sustained oxidative stress in the blood [60].

Recent research [61, 62] suggests that athletes tend to consume enough food to secure an adequate intake of most micronutrients, with some exceptions referred to vitamins A and E. Although vitamin E intake among well-trained athletes is below 12 mg/day, this does not pose a risk of deficiency status [63]. Consequently, the variation in training loads should be taken into account when interpreting vitamin E status [55] since the greater the training intensity, the higher the plasma vitamin E concentrations are [64]. With regard to vitamin A intake, it has been reported that 10–25% of the studied athletes consumed less vitamin A than the dietary reference intakes [65]. Interestingly, in only 5% of the 182

athletes studied were the serum vitamin A levels below 30 mg/dL [66]. Furthermore, more than 70% of elite junior basketball athletes showed insufficient vitamin A levels based on the nutrient density calculations. In this line, the decrease in vitamin A (at biochemical level) was directly related to an increase in oxidative stress promoted by exercise in athletes with a low $\text{VO}_{2\text{max}}$ [67] and a decrease in lipid peroxidation [49].

Although there are no specific recommendations for athletes, it seems that the needs are increased by exercise. Nevertheless, the variety in sports disciplines makes it difficult to define universal requirements for all sports. Supplementation is recommended in case of diagnosed deficiency, although this rarely occurs in healthy athletes who eat a balanced diet. Supplementation with antioxidants appears to be useful in cases of an insufficient intake of vitamins, though no performance benefits have been evidenced in athletes with adequate intakes. In contrast, excess supplementation could result in harmful levels due to prooxidant effects.

6.5.2 Minerals Adequacy

Minerals are required in small amounts and interact with each other to regulate physiological functions [1, 33]. For example, iodine, iron, magnesium, and zinc are important in enhancing the conversion of macronutrients into energy [49]; iron intake is required to form part of hemoglobin and is also necessary for DNA and RNA synthesis [55]; calcium, phosphorus, and magnesium ensure the maintenance of bone mineral density (reviewed in [1]); and selenium and zinc protect cells and tissues against damage by reactive oxygen species, which are increased by exercise [33]. Both the ACSM and the ISSN recommend a well-balanced diet to meet mineral requirements [1, 33]. However, athletes with limited energy intake are at an increased risk of inadequate mineral intake.

A study carried out in elite junior basketball athletes [34] recorded diminished intakes of calcium and zinc (expressed as mg/1000 kcal). An insufficient intake of minerals such as cal-

cium, iodine, magnesium, and zinc has also been reported in both men and women in other team sports [62, 65, 68, 69]. Specifically, female athletes appear to be at highest risk of low bone mineral density linked to low calcium intake. Current calcium recommendations for athletes with eating disorders, amenorrhea, or risk of early osteoporosis are 1500 mg of calcium and 400–800 IU of vitamin D per day [33]. In the same way, the relationship between magnesium status and strength in team sport players has been investigated [70].

Another of the minerals presenting a strong relationship with exercise is iron. In this regard, the iron requirements of endurance athletes increase by approximately 70% with respect to the recommendation of 18 and 8 mg per day for men and women, respectively [33]. Iron deficiency, with or without anemia, can impair muscle function, limiting performance capacity. Relatively low concentrations of ferritin in athletes, and the potential for further decline during the season when the physical load may be at its highest level, suggest hematological monitoring in combination with personal dietary advice [71]. Moreover, high-intensity training loads would provoke changes in macroelements, which would lead to electrolyte disturbances, suggesting that 1 week of high-intensity training would have an impact upon microelement levels—especially selenium and iron [72]. Among other essential trace elements, zinc and copper have crucial functions in regulating the hematological system [33] and act as antioxidant agents, decreasing reactive oxygen species required for copper–zinc SOD activity (copper–zinc superoxide dismutase) in male basketball athletes [73].

The intermittent nature of basketball play makes it difficult to establish recommendations for meeting the actual requirements of the athletes since this sport involves aerobic metabolism as the main energy source, alternating with periods of highly intense activity that require mainly anaerobic metabolism. It would be necessary to control the mineral status over a season in order to assess whether the deficiency is chronic and not sporadic. Previous studies [74] without dietary supplementation have found no change in

the plasma concentrations of any of the minerals in athletes after training. We can conclude that an annual evaluation may be beneficial for athletes, with additional monitoring only when clinically indicated.

Key Points

- The intensity, duration, and frequency of training and competition have an impact on whether or not micronutrients are required in greater amounts
- An adequate intake of water-soluble vitamins (B vitamins and vitamin C) is important to ensure optimum energy production and muscle tissue construction and repair
- Fat-soluble vitamins include vitamins A, D, E, and K, and the body stores these vitamins in various tissues, which can result in toxicity if consumed in excessive amounts
- Although several studies have shown specific vitamins to possess various health benefits (e.g., vitamin E and vitamin C), only a few published studies have reported an ergogenic value of vitamins for athletes
- Diminished intake of minerals such as calcium, iodine, magnesium, and zinc has also been reported in both men and women in basketball and in other team sports
- Basketball makes it difficult to establish recommendations since it involves aerobic metabolism as the main energy source, alternating with periods of highly intense activity requiring mainly anaerobic metabolism

6.6 Hydration

Being well hydrated is a key factor that contributes to optimal health and exercise performance in athletes. Generated as a by-product of muscle work, perspiration helps to dissipate heat. It is

often exacerbated by environmental conditions and, therefore, helps maintain body temperature within acceptable ranges [75]. Likewise, excessive perspiration will lead to a decrease in micronutrients, including sodium and minor amounts of potassium, calcium, and magnesium, and results in an alteration of normal physiological functions.

Dehydration refers to the process where the loss of body fluid leads to hypohydration (Fig. 6.5). The metabolic heat generated by muscle contractions during exercise may eventually lead to hypovolemia (decreased plasma/blood volume), leading to cardiovascular hypotension, increased glycogen consumption, altered metabolic and central nervous system function, and increased body temperature. Specifically, studies on basketball and other team sports suggest that $\geq 2\%$ hypohydration can deteriorate sports performance [76], with a progressive decline as dehydration progresses from 1 to 4% [77], and a reduction in anaerobic performance [78]. In contrast to these findings, Carvalho et al. [79] found that dehydration equivalent to 2.5% of body weight had no effect on performance in

basketball drills, where perceived exertion was observed to be increased.

In relation to match days, game day stress can alter consumption habits, leading to over- or under-hydration [17]. Given these considerations, the ACSM provides a series of hydration recommendations [33, 75]. Specifically, the ACSM recommends that people should drink approximately 5–7 mL/kg body weight (about 2–3 mL/kg) of water or a sports drink at least 4 h before exercise in order to optimize hydration status and to excrete any excess fluid such as urine (Fig. 6.5) [75]. Contrarily, excessive hydration with fluids that expand the extra- and intracellular compartments considerably increases the risk of leaving the competition, and it does not provide a clear physiological benefit for performance. For many team sports, the capacity to sustain high-intensity efforts alternating with rest or lower intensity periods throughout a game is critical to the success of an athlete [76]. Therefore, the type, intensity, and duration of exercise should be considered, as they alter the need for fluids and electrolytes.

Factors related to fluid availability (type and amount), drinking opportunities (per the rules

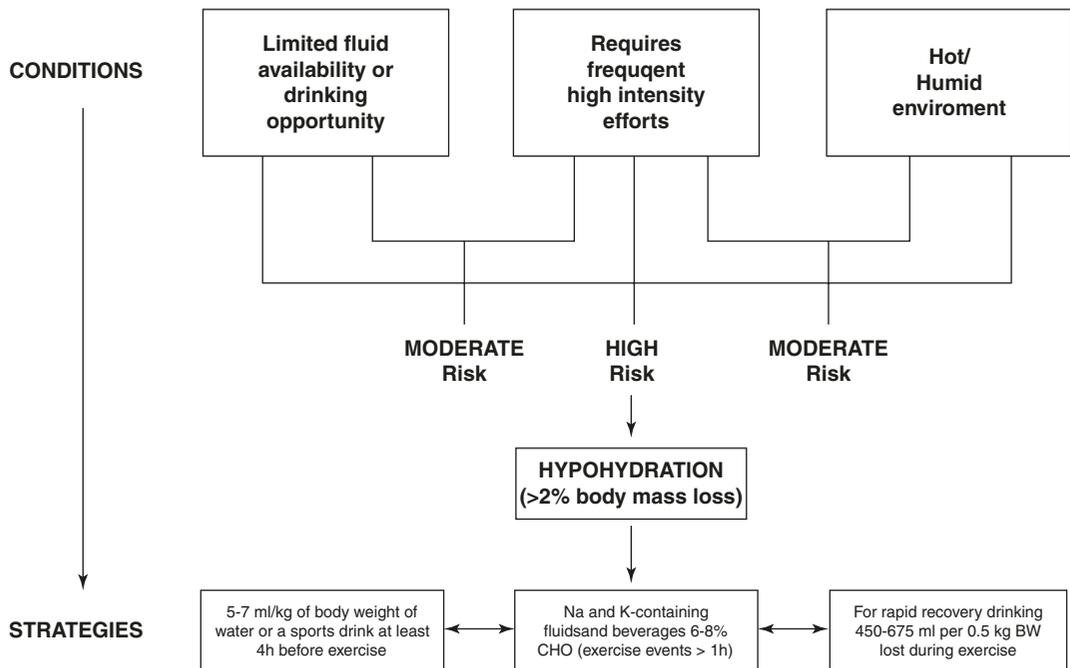


Fig. 6.5 Risk of dehydration and drinking strategies and fluid replacement in team sports. (Adapted from [76])

and structure of the game), exercise duration, hydration education, and personal preferences also play an important role in determining fluid balance [80]. Sodium and potassium-containing fluids help replace perspiration electrolyte loss, while sodium stimulates thirst and fluid retention at the same time that CHO provide a good energy source. In particular, beverages containing 6–8% CHO (e.g., CHO-electrolyte sports drink combining such a CHO source with a fructose-based sugar) are recommended for exercise events lasting more than an hour (Fig. 6.5) [33]. Prolonged exercise or heavy perspiration, where sodium replacement is not covered or there is excessive intake, may result in hyponatremia (serum sodium < 130 mmol/L). Hyponatremia is more likely to develop in athletes who perspire less or consume excess water before, during, or after an event [75]. Therefore, balanced food and drink intake is recommended after exercise to restore hydration status and replace fluids and electrolytes lost during exercise. Rapid and complete recovery from excessive dehydration can be achieved by drinking at least 450–675 mL of liquid per 0.5 kg of body weight lost during exercise. High perspiration rates and fluid balance disturbances have generally been mild, suggesting that drinking opportunities were sufficient to provide most athletes with enough fluid to offset losses [76]. In future studies, it would be helpful to include studies directly comparing the effect of hypohydration in different cohorts, in order to determine who may be more susceptible to the detrimental effects of hypohydration from both a physiological and a performance perspective.

Key Points

- Significant hypohydration ($\geq 2\%$ body mass deficit) has been reported in basketball athletes, with a progressive decline in performance as dehydration progresses from 1% to 4%
- The ACSM recommends that individuals should drink at least 5–7 mL/kg of body weight (about 2–3 mL/kg) of water or a sports drink at least 4 h before exercise

- Sodium- and potassium-containing fluids help replace perspiration electrolyte loss, and beverages containing 6–8% CHO are recommended for exercise events lasting more than an hour
- Rapid and complete recovery from excessive dehydration can be achieved by drinking at least 450–675 mL of liquid per 0.5 kg body weight lost during exercise

6.7 Conclusions

In basketball, as in other team sports, the physical and physiological demands are closely related to the nutritional requirements. This chapter provides important information allowing a better understanding of the nutritional demands and requirements in basketball players. In this sense, future studies should examine the impact of different nutritional strategies in an individualized way with the aim of improving the ability of players to sustain high exercise intensities during training and competition. Furthermore, future research should be conducted to examine the specific physical demands for better understanding of the energy, macronutrient, and micronutrient requirements as well as of the hydration patterns. An overriding reality is that an individual approach is needed to adequately meet each individual athlete's nutritional and hydration needs.

References

1. Thomas DT, Erdman KA, Burke LM. Position of the Academy of Nutrition and Dietetics, Dietitians of Canada, and the American College of Sports Medicine: Nutrition and Athletic Performance. *J Acad Nutr Diet.* 2016;116:501–28. <https://doi.org/10.1016/j.jand.2015.12.006>.
2. Rodriguez NR, Di Marco NM, Langley S. American College of Sports Medicine position stand. Nutrition and athletic performance. *Med Sci Sports Exerc.* 2009;41:709–31. <https://doi.org/10.1249/MSS.0b013e31890eb86>.

3. Kreider RB, Wilborn CD, Taylor L, et al. ISSN exercise & sport nutrition review: research & recommendations. *J Int Soc Sports Nutr.* 2010;7:7. <https://doi.org/10.1186/1550-2783-7-7>.
4. Ziv G, Lidor R. Physical attributes, physiological characteristics, on-court performances and nutritional strategies of female and male basketball players. *Sports Med.* 2009;39:547–68. <https://doi.org/10.2165/00007256-200939070-00003>.
5. Stojanović E, Stojilković N, Scanlan AT, et al. The activity demands and physiological responses encountered during basketball match-play: a systematic review. *Sports Med.* 2018;48:111–35. <https://doi.org/10.1007/s40279-017-0794-z>.
6. Lidor R, Blumenstein B, Tenenbaum G. Psychological aspects of training in European basketball: conceptualization, periodization, and Planning. *Sport Psychol.* 2007;21:353–67. <https://doi.org/10.1123/tsp.21.3.353>.
7. McLean BD, Strack D, Russell J, Coutts AJ. Quantifying physical demands in the National Basketball Association—challenges around developing best-practice models for athlete care and performance. *Int J Sports Physiol Perform.* 2019;14:414–20. <https://doi.org/10.1123/ijspp.2018-0384>.
8. McKeag D. *Handbook of Sports Medicine and Science: Basketball.* Oxford, UK: Wiley; 2008. 225 p. <https://doi.org/10.1002/9780470693896>.
9. Scanlan A, Dascombe B, Reaburn P. A comparison of the activity demands of elite and sub-elite Australian men's basketball competition. *J Sports Sci.* 2011;29:1153–60. <https://doi.org/10.1080/02640414.2011.582509>.
10. Scanlan AT, Dascombe BJ, Reaburn P, Dalbo VJ. The physiological and activity demands experienced by Australian female basketball players during competition. *J Sci Med Sport.* 2012;15:341–7. <https://doi.org/10.1016/j.jsams.2011.12.008>.
11. Narazaki K, Berg K, Stergiou N, Chen B. Physiological demands of competitive basketball. *Scand J Med Sci Sports.* 2009;19:425–32. <https://doi.org/10.1111/j.1600-0838.2008.00789.x>.
12. Puente C, Abián-Vicén J, Areces F, et al. Physical and physiological demands of experienced male basketball players during a competitive game. *J Strength Cond Res.* 2017;31:956–62. <https://doi.org/10.1519/JSC.0000000000001577>.
13. Ben Abdelkrim N, El Fazaa S, El Ati J. Time-motion analysis and physiological data of elite under-19-year-old basketball players during competition. *Br J Sports Med.* 2007;41:69–75.; discussion 75. <https://doi.org/10.1136/bjism.2006.032318>.
14. Abdelkrim NB, Castagna C, Fazaa SE, Ati JE. The effect of players' standard and tactical strategy on game demands in men's basketball. *J Strength Cond Res.* 2010;24:2652–62. <https://doi.org/10.1519/JSC.0b013e3181e2e0a3>.
15. Rodríguez-Alonso M, Fernández-García B, Pérez-Landaluce J, Terrados N. Blood lactate and heart rate during national and international women's basketball. *J Sports Med Phys Fitness.* 2003;43:432–6.
16. Deakin V. Measuring nutritional status of athletes: Clinical and research perspectives. In: Burke L, Deakin V, editors. *Clinical sports nutrition.* 4 ed. Sydney: McGraw-Hill Medical. 2009. p. 18–43.
17. Holway FE, Spriet LL. Sport-specific nutrition: practical strategies for team sports. *J Sports Sci.* 2011;29(Suppl 1):S115–25. <https://doi.org/10.1080/02640414.2011.605459>.
18. McArdle WD, Katch FI, Katch VL. *Exercise physiology: nutrition, energy, and human performance.* Philadelphia: Lippincott Williams & Wilkins; 2010.
19. Cunningham JJ. A reanalysis of the factors influencing basal metabolic rate in normal adults. *Am J Clin Nutr.* 1980;33:2372–4.
20. Harris JA, Benedict FG. A biometric study of human basal metabolism. *Proc Natl Acad Sci U S A.* 1918;4:370–3.
21. Ainsworth BE, Haskell WL, Whitt MC, et al. Compendium of physical activities: an update of activity codes and MET intensities. *Med Sci Sports Exerc.* 2000;32:S498–504.
22. Westerterp KR, Saris WH, van Es M, ten Hoor F. Use of the doubly labeled water technique in humans during heavy sustained exercise. *J Appl Physiol* (1985). 1986;61:2162–7. <https://doi.org/10.1152/jappl.1986.61.6.2162>.
23. Westerterp KR. Doubly labelled water assessment of energy expenditure: principle, practice, and promise. *Eur J Appl Physiol.* 2017;117:1277–85. <https://doi.org/10.1007/s00421-017-3641-x>.
24. Silva AM, Santos DA, Matias CN, et al. Total energy expenditure assessment in elite junior basketball players: a validation study using doubly labeled water. *J Strength Cond Res.* 2013;27:1920–7. <https://doi.org/10.1519/JSC.0b013e31827361eb>.
25. Silva AM, Matias CN, Santos DA, et al. Energy balance over one athletic season. *Med Sci Sports Exerc.* 2017;1. <https://doi.org/10.1249/MSS.0000000000001280>
26. Burke LM, Loucks AB, Broad N. Energy and carbohydrate for training and recovery. *J Sports Sci.* 2006;24:675–85. <https://doi.org/10.1080/02640410500482602>.
27. Phillips SM, Van Loon LJC. Dietary protein for athletes: from requirements to optimum adaptation. *J Sports Sci.* 2011;29(Suppl 1):S29–38. <https://doi.org/10.1080/02640414.2011.619204>.
28. Maughan RJ, Shirreffs SM. Nutrition for sports performance: issues and opportunities. *Proc Nutr Soc.* 2011; 1–8. <https://doi.org/10.1017/S0029665111003211>.
29. Heaney S, O'Connor H, Michael S, et al. Nutrition knowledge in athletes: a systematic review. *Int J Sport Nutr Exerc Metab.* 2011;21:248–61.
30. Burke LM. Fueling strategies to optimize performance: training high or training low? *Scand J Med Sci Sports.* 2010;20(Suppl 2):48–58. <https://doi.org/10.1111/j.1600-0838.2010.01185.x>.
31. Spriet LL. New insights into the interaction of carbohydrate and fat metabolism during exercise.

- Sports Med Auckl NZ. 2014;44:87–96. <https://doi.org/10.1007/s40279-014-0154-1>.
32. Zanders B, Currier B, Harty P, et al. Changes in energy expenditure, dietary intake, and energy availability across an entire collegiate women's basketball season. *J Strength Cond Res*. 2018. <https://doi.org/10.1519/JSC.0000000000002783>
 33. Rodriguez NR, DiMarco NM, Langley S, et al. Nutrition and athletic performance. *Med Sci Sports Exerc*. 2009;41:709–31.
 34. Nikić M, Pedišić Ž, Šatalić Z, et al. Adequacy of nutrient intakes in elite junior basketball players. *Int J Sport Nutr Exerc Metab*. 2014;24:516–23. <https://doi.org/10.1123/ijsnem.2013-0186>.
 35. Burke LM, Hawley JA, Wong SHS, Jeukendrup AE. Carbohydrates for training and competition. *J Sports Sci*. 2011;29(Suppl 1):S17–27. <https://doi.org/10.1080/02640414.2011.585473>.
 36. Ferro A, Garrido G, Villacieros J, et al. Nutritional habits and performance in male elite wheelchair basketball players during a precompetitive period. *Adapt Phys Activ Q*. 2017;34:295–310. <https://doi.org/10.1123/apaq.2016-0057>.
 37. Silva A, Santos D, Matias C, et al. Total energy expenditure assessment in elite junior basketball players. *J Strength Cond Res*. 2013;27:1920–7. <https://doi.org/10.1519/JSC.0b013e31827361eb>.
 38. Kitsou S, Fournounopoulos D, Efstathiou E, et al. Dietary intakes of Greek basketball players. *Nutr Food Sci*. 2003;33:23–7. <https://doi.org/10.1108/00346650310459536>.
 39. Williams C, Serratos L. Nutrition on match day. *J Sports Sci*. 2006;24:687–97. <https://doi.org/10.1080/02640410500482628>.
 40. Williams C, Rollo I. Carbohydrate nutrition and team sport performance. *Sports Med*. 2015;45:13–22. <https://doi.org/10.1007/s40279-015-0399-3>.
 41. Winnick J, Davis J, Welsh R, et al. Carbohydrate feedings during team sport exercise preserve physical and CNS function. *Med Sci Sports Exerc*. 2005;37:306–15. <https://doi.org/10.1249/01.MSS.0000152803.35130.A4>.
 42. Nicholas CW, Green PA, Hawkins RD, Williams C. Carbohydrate intake and recovery of intermittent running capacity. *Int J Sport Nutr*. 1997;7:251–60.
 43. Phillips SM. Dietary protein requirements and adaptive advantages in athletes. *Br J Nutr*. 2012;108(Suppl 2):S158–67. <https://doi.org/10.1017/S0007114512002516>.
 44. Alemdaroglu U. The relationship between muscle strength, anaerobic performance, agility, sprint ability and vertical jump performance in professional basketball players. *J Hum Kinet*. 2012;31:149–58. <https://doi.org/10.2478/v10078-012-0016-6>.
 45. Abdelkrim NB, Chaouachi A, Chamari K, et al. Positional role and competitive-level differences in elite-level men's basketball players. *J Strength Cond Res*. 2010;24:1346–55. <https://doi.org/10.1519/JSC.0b013e3181cf7510>.
 46. Wardenaar F, Brinkmans N, Ceelen I, et al. Macronutrient intakes in 553 Dutch elite and sub-elite endurance, team, and strength athletes: does intake differ between sport disciplines? *Nutrients*. 2017;9. <https://doi.org/10.3390/nu9020119>
 47. Tipton KD, Elliott TA, Cree MG, et al. Stimulation of net muscle protein synthesis by whey protein ingestion before and after exercise. *Am J Physiol Endocrinol Metab*. 2007;292:E71–6. <https://doi.org/10.1152/ajpendo.00166.2006>.
 48. Helge JW, Richter EA, Kiens B. Interaction of training and diet on metabolism and endurance during exercise in man. *J Physiol*. 1996;492:293–306.
 49. Volpe SL. Micronutrient requirements for athletes. *Clin Sports Med*. 2007;26:119–30. <https://doi.org/10.1016/j.csm.2006.11.009>.
 50. Woolf K, Manore MM. B-vitamins and exercise: does exercise alter requirements? *Int J Sport Nutr Exerc Metab*. 2006;16:453–84.
 51. Driskell J. Sports nutrition: vitamins and trace elements. 2nd ed. Boca Raton: CRC Press; 2005. <https://www.crcpress.com/Sports-Nutrition-Vitamins-and-Trace-Elements-Second-Edition/Wolinsky-Driskell/p/book/9780849330223>. Accessed 9 Jul 2017.
 52. Institute of Medicine (US) Standing Committee on the Scientific Evaluation of Dietary reference intakes and its panel on folate, other b vitamins, and choline. Dietary reference intakes for thiamin, riboflavin, niacin, vitamin B6, folate, vitamin B12, pantothenic acid, biotin, and choline. Washington (DC): National Academies Press (US); 1998.
 53. Molina-López J, Molina JM, Chiroso LJ, et al. Effect of folic acid supplementation on homocysteine concentration and association with training in handball players. *J Int Soc Sports Nutr*. 2013;10:10. <https://doi.org/10.1186/1550-2783-10-10>.
 54. Heaney S, O'Connor H, Gifford J, Naughton G. Comparison of strategies for assessing nutritional adequacy in elite female athletes' dietary intake. *Int J Sport Nutr Exerc Metab*. 2010;20:245–56.
 55. Margaritis I, Rousseau AS. Does physical exercise modify antioxidant requirements? *Nutr Res Rev*. 2008;21:3–12. <https://doi.org/10.1017/S0954422408018076>.
 56. Lukaski HC. Vitamin and mineral status: effects on physical performance. *Nutrition*. 2004;20:632–44. <https://doi.org/10.1016/j.nut.2004.04.001>.
 57. Neubauer O, Yfanti C. Antioxidants in athlete's basic nutrition: considerations towards a guideline for the intake of vitamin C and vitamin E. In: Lamprecht M, editor. Antioxidants in sport nutrition. Boca Raton, FL: CRC Press/Taylor & Francis; 2015.
 58. Naziroglu M, Kılınç F, Uğuz AC, et al. Oral vitamin C and E combination modulates blood lipid peroxidation and antioxidant vitamin levels in maximal exercising basketball players. *Cell Biochem Funct*. 2010;28:300–5. <https://doi.org/10.1002/cbf.1657>.
 59. Neubauer O, Reichhold S, Nics L, et al. Antioxidant responses to an acute ultra-endurance exercise: impact

- on DNA stability and indications for an increased need for nutritive antioxidants in the early recovery phase. *Br J Nutr.* 2010;104:1129–38. <https://doi.org/10.1017/S0007114510001856>.
60. Nikolaidis MG, Jamurtas AZ. Blood as a reactive species generator and redox status regulator during exercise. *Arch Biochem Biophys.* 2009;490:77–84. <https://doi.org/10.1016/j.abb.2009.08.015>.
 61. Burkhart SJ, Pelly FE. Dietary intake of athletes seeking nutrition advice at a major international competition. *Nutrients.* 2016;8:638. <https://doi.org/10.3390/nu8100638>.
 62. Grams L, Garrido G, Villacieros J, Ferro A. Marginal micronutrient intake in high-performance male wheelchair basketball players: a dietary evaluation and the effects of nutritional advice. *PLoS One.* 2016;11:e0157931. <https://doi.org/10.1371/journal.pone.0157931>.
 63. Rousseau A-S, Hininger I, Palazzetti S, et al. Antioxidant vitamin status in high exposure to oxidative stress in competitive athletes. *Br J Nutr.* 2004;92:461–8. <https://doi.org/10.1079/BJN20041222>.
 64. Palazzetti S, Rousseau A-S, Richard M-J, et al. Antioxidant supplementation preserves antioxidant response in physical training and low antioxidant intake. *Br J Nutr.* 2004;91:91–100.
 65. Molina-López J, Molina JM, Chiroso LJ, et al. Implementation of a nutrition education program in a handball team; consequences on nutritional status. *Nutr Hosp.* 2013;28:1065–76.
 66. Chen J. Vitamins: effects of exercise on requirements. In: Maughan RJ, editor. *Nutrition in sport.* Oxford, UK: Blackwell Science Ltd.; 2000. p. 281–91.
 67. Izzicupo P, Ghinassi B, D'Amico MA, et al. Vitamin A decreases after a maximal incremental stress test in non-professional male runners with low aerobic performance. *J Biol Regul Homeost Agents.* 2016;30:1223–8.
 68. Heaney S, O'Connor H, Naughton G, Gifford J. Towards an understanding of the barriers to good nutrition for elite athletes. *Int J Sports Sci Coach.* 2008;3:391–401. <https://doi.org/10.1260/174795408786238542>.
 69. Molina-López J, Molina JM, Chiroso LJ, et al. Association between erythrocyte concentrations of magnesium and zinc in high-performance handball players after dietary magnesium supplementation. *Magnes Res.* 2012;25:79–88.
 70. Santos DA, Matias CN, Monteiro CP, et al. Magnesium intake is associated with strength performance in elite basketball, handball and volleyball players. *Magnes Res.* 2011;24:215–9. <https://doi.org/10.1684/mrh.2011.0290>.
 71. Clarke AC, Anson JM, Dziedzic CE, et al. Iron monitoring of male and female rugby sevens players over an international season. *J Sports Med Phys Fitness.* 2018;58:1490–6. <https://doi.org/10.23736/S0022-4707.17.07363-7>.
 72. Wang L, Zhang J, Wang J, et al. Effects of high-intensity training and resumed training on macroelement and microelement of elite basketball athletes. *Biol Trace Elem Res.* 2012;149:148–54. <https://doi.org/10.1007/s12011-012-9420-y>.
 73. Zhao J, Fan B, Wu Z, et al. Serum zinc is associated with plasma leptin and Cu–Zn SOD in elite male basketball athletes. *J Trace Elem Med Biol.* 2015;30:49–53. <https://doi.org/10.1016/j.jtemb.2014.10.005>.
 74. González-Haro C, Soria M, López-Colón JL, et al. Plasma trace elements levels are not altered by submaximal exercise intensities in well-trained endurance euhydrated athletes. *J Trace Elem Med Biol.* 2011;25(Suppl 1):S54–8. <https://doi.org/10.1016/j.jtemb.2010.10.010>.
 75. American College of Sports Medicine, Sawka MN, Burke LM, et al. Exercise and fluid replacement. *Med Sci Sports Exerc.* 2007;39:377–90. <https://doi.org/10.1249/mss.0b013e31802ca597>.
 76. Nuccio RP, Barnes KA, Carter JM, Baker LB. Fluid balance in team sport athletes and the effect of hypohydration on cognitive, technical, and physical performance. *Sports Med.* 2017;47:1951–82. <https://doi.org/10.1007/s40279-017-0738-7>.
 77. Baker LB, Dougherty KA, Chow M, Kenney WL. Progressive dehydration causes a progressive decline in basketball skill performance. *Med Sci Sports Exerc.* 2007;39:1114–23. <https://doi.org/10.1249/mss.0b013e3180574b02>.
 78. Kraft JA, Green JM, Bishop PA, et al. The influence of hydration on anaerobic performance: a review. *Res Q Exerc Sport.* 2012;83:282–92. <https://doi.org/10.1080/02701367.2012.10599859>.
 79. Carvalho P, Oliveira B, Barros R, et al. Impact of fluid restriction and ad libitum water intake or an 8% carbohydrate-electrolyte beverage on skill performance of elite adolescent basketball players. *Int J Sport Nutr Exerc Metab.* 2011;21:214–21.
 80. Garth AK, Burke LM. What do athletes drink during competitive sporting activities? *Sports Med.* 2013;43:539–64. <https://doi.org/10.1007/s40279-013-0028-y>.

Part II

The Basketball Medical Perimeter - Aspects in Medical Preparation



Constructing a Medical Team: The Medical Needs of a Basketball Team

7

Benjamin Oshlag and Benjamin Boswell

7.1 Introduction

Comprehensive medical coverage for a basketball team requires a multidisciplinary team with a cooperative approach toward addressing the various needs of the athletes. Medical issues will frequently arise when dealing with the athletic population, and the team's medical staff should be prepared to deal appropriately with a wide range of conditions, from routine to emergent, when they occur. Additionally, preventative care is an important aspect of team coverage, and yearly pre-participation physical exams (PPEs) allow the medical team to screen and assess athletes regularly. The medical team is also responsible for establishing and maintaining

an effective environment for the care of athletes, including appropriate facilities, equipment, personnel, training, and emergency action plans.

The size and scope of a medical team will vary depending on the resources and level of the basketball team in question, but should be appropriate to manage the medical needs of that team. In some cases, a team may provide direct access and specialty care for all of its athletes' needs, while in others it may fall to the primary medical provider or the athletes and their families to arrange for outside consultants and appointments with specialists.

The medical needs of a particular team will vary as well, including the requirements for facilities and staffing of the medical team. In general, teams should have capabilities that satisfy at least their baseline minimum level of need, which will largely depend on the level of play of that team. Additional factors such as financial restraints or logistical concerns may also affect the overall structure and capabilities of a medical team. Professional teams will usually require the highest level of involvement and coverage, followed by collegiate and then high school teams.

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7.2 High School Teams

There are a wide range of team facilities and training needs for high school teams, as there will be numerous levels of competition, and different state

requirements for athletic training needs and availability. Most teams will have a training room at or near the primary location for practice and games, which will often be shared with other sports. Most teams will have an athletic trainer that covers all, or at least several, sports at that high school, who may or may not be present for each practice/competition. Most, but not all, teams will have an available physician who may or may not be trained in Sports Medicine, but should be available for most primary medical problems and to see athletes on an as-needed basis.

7.3 College Teams

In collegiate athletics, the personnel and facility needs will vary predominantly based on the level of competitiveness, but will generally be higher than most high school teams. Most collegiate teams will have a dedicated athletic trainer or student trainer that is focused primarily on that team. Some teams may have dedicated physical therapists as well. The athletic trainer will be on the sidelines or nearby for all practices and competitions. The training room should be available at all practices and competitions, but this will frequently be shared by other teams. All teams will have a physician available for the needs of that team. Ideally this physician will either be a sports medicine trained primary care physician (PCSM) or orthopedic surgeon. At the higher level Division I universities, the basketball team will have both a PCSM physician and a sports medicine-trained orthopedic surgeon available for all practices and present for all games. At the lower levels, coverage will vary depending on the capabilities and expectations of that university, but will generally fall in between what is seen in high schools and that at the Division I level. Most college teams will have a medical examination room available at or near the sports facility for physician use, but this is not a requirement. The capabilities of this medical facility will vary, but ideally should be equipped for minor procedures such as laceration repairs or splinting. If space is not available, the team physician should have a medical office nearby instead. Most college

teams will also have an affiliated medical center for advanced medical needs.

7.4 Professional Teams

At the professional level, the training and facility needs are far more standard. All teams will have one or more dedicated athletic trainers that work only for that team. They will be present at all practices and competitions, and often present for all team functions. It is also common for teams to have physical therapists and other treatment specialists available to work with athletes as needed. There should be a training room dedicated to that team only, which will not be available to anyone not affiliated with the team. Each team will have a dedicated head PCSM Physician and head sports medicine-trained orthopedic surgeon. One or both of these physicians will also serve as the team's medical director(s), and should be in charge of all services by the medical providers. In the United States, in order for a physician to care for professional athletes, they must be board-certified in their primary specialty, and should also obtain a Certificate of Added Qualifications in Sports Medicine (CAQSM) after successfully completing a Sports Medicine fellowship. Worldwide, providers should be familiar with their national and local requirements for maintaining certification in their specialty. All professional teams should have a medical examination room available for team physician use, with equipment available for primary medical care and basic procedures, and often will have medical imaging available on-site as well. Each team will typically also be affiliated with a nearby medical center to accommodate the advanced medical needs of the athletes. The team's medical director(s) will oversee coordinating the medical care for that team.

7.5 Medical Team Personnel

The primary medical team should include both a primary care sports medicine physician as well as a sports medicine-trained orthopedic surgeon.

Additionally, the medical team should include several other staff with specialty training, including certified athletic trainers and/or physical therapists.

In the United States, primary care sports medicine (PCSM) physicians are doctors with an MD or DO degree who then complete residency training in family medicine, internal medicine, pediatrics, emergency medicine, or physical medicine and rehabilitation, and a 1-year fellowship in primary care sports medicine. PCSM physicians specialize in the treatment of nonoperative orthopedic conditions as well as the general medical needs of the athlete. They provide care for acute injuries including sprains, dislocations, and fractures, chronic overuse injuries, concussions, exercise and nutrition guidance, and return to play decisions, as well as athletes with acute and chronic medical illnesses.

Orthopedic sports medicine surgeons are MDs and DOs who complete an Orthopedic Surgery residency and then a 1-year fellowship in Orthopedic Sports Medicine.

They specialize in the treatment of athletes and sports-related injuries from a surgical perspective although a majority of their care will likely also be nonoperative treatment.

Certified athletic trainers (ATCs) complete a bachelor's or master's degree program from an accredited professional athletic training education program. Athletic trainers collaborate with team physicians to provide care to athletes, with a focus on prevention, examination, diagnosis, treatment, and rehabilitation of acute, subacute, and chronic medical conditions. They also provide organizational and professional health and well-being, developing, administering, and managing facilities to provide health-care services to athletes.

Physical therapists complete a 3-year doctor of physical therapy (DPT) program after earning their undergraduate degree. Additionally, some physical therapists have additional training through residency and fellowship programs specializing in sports-specific therapy and rehabilitation, including recovery from injuries, surgeries, and return to play, and may have a specialty certification in Sports Physical Therapy. Physical

therapists are musculoskeletal movement experts who help patients optimize quality of life and performance through prescribed exercise, hands-on treatment, and education. They are crucial to helping athletes recover from both minor and more significant injuries, and maintaining peak performance throughout the season.

Other medical personnel and specialists can help augment the medical team as resources and athlete needs dictate. These can include sports performance coaches, nutritionists, sports psychologists, chiropractors, massage therapists, and others. In addition, various medical specialists will frequently be needed for specific athlete injuries or complaints, and should be available either as a part of the primary medical team or as easily accessible consultants familiar to the medical team members. These include orthopedic subspecialists, cardiologists, neurologists, otorhinolaryngologists, ophthalmologists, dermatologists, and dentists, and other medical subspecialties as athlete conditions dictate.

The foot and ankle are the most common sites of basketball injuries, representing about 40% of all injuries. Because of this, it is important to have medical staff available who are specialists in this area, including an orthopedic foot and ankle specialist. Knee injuries make up about 15% of all basketball injuries, and can typically be managed by sports medicine physicians and orthopedic surgeons [1–5]. While they are not the most common in terms of occurrence, they account for the largest amount of lost playing time, especially in recovery from injuries that require surgery. Studies have shown that improper landing mechanics confer an increased risk of anterior cruciate ligament (ACL) tears, and have suggested that modifying these mechanics through work with a physical therapist or athletic trainer might help to reduce this risk without compromising performance [6–8]. Arm and hand injuries are slightly less common, making up about 9.6% of total injuries, with the most common being sprains and dislocations of the proximal interphalangeal joint of the fingers. These injuries can typically be managed by the primary medical team as well, although consultation with a hand surgeon

is recommended for complex or significant injuries to the wrist, hand, or fingers [1–5].

About 13.6% of injuries in basketball occur to the head, neck, or face of the athlete. These injuries can include contusions, neck muscle strains, concussions, lacerations, and more significant injuries to the eyes, nose, and mouth. Depending on the type and severity of injury, it may be necessary to seek treatment from a specialist [5, 9].

Simple lacerations occur frequently, and can often be managed by the primary medical team if appropriate supplies are available. These should include a variety of suture size and material, as well as suture kits, gloves, irrigation supplies, gauze, and dressings. Other methods of closing lacerations and controlling bleeding are important as well, especially in more time-sensitive situations where rapid treatment and return to play are desired. Skin glue, wound closure strips (such as Steri-Strips), and topical pro-coagulants or vasoconstrictors can all help control bleeding and get an athlete back to play quickly. These can often be applied by the athletic trainer on the sideline when needed.

An estimated 7500 eye injuries occur during basketball activities in the United States per year. Any significant eye injury should be evaluated promptly by an ophthalmologist. Additionally, the Academy of Ophthalmology “strongly recommend[s]” eye protection for all athletes at risk for eye injury, and eye protection should be required for any athlete with one eye or who have had recent eye surgery or trauma. Red flags for eye injuries that need immediate attention include vision loss, double vision, abnormal or painful eye movements, or abnormal pupils [5, 10, 11].

Nose injuries, including nasal bone fractures, often do not need specific treatment, but depending on the severity may benefit from treatment by an otorhinolaryngologist. Bleeding control is an important immediate consideration for nose injuries, and may initially be managed by the sideline provider or athletic trainer with direct pressure or appropriate nasal packing [5, 9].

Oral injuries are also relatively common, with estimated injury rates ranging from 1 to 14%. Permanent teeth have limited healing ability, and

mouth guards have been shown to reduce the incidence of dental injury. In cases of significant dental trauma, the athlete should be evaluated by a dentist as quickly as possible, as delays in treatment reduce the viability of the injured teeth and limit treatment options. Avulsed teeth can be transported in specialized solutions to help maximize the chances of successful re-implantation. Available solutions include Viaspan and Hank’s Balanced Salt Solution, and should be considered for inclusion in the athletic trainer’s sideline supplies [5, 12–16].

Concussions are an important and increasingly publicized medical injury, and occur frequently in basketball from head and face trauma during practices and games. The mechanism of injury is most often due to direct contact from another player, but can also occur with head strikes against the court or other equipment. Both the athletic trainer and team physician should be well-trained in identification and diagnosis of athletes with a suspected concussion, and protocols should be in place for management, including immediate removal from play and sideline neurologic testing. Athletes with a confirmed concussion should undergo further evaluation by the primary team physician, and a treatment plan developed between the athlete, physician, athletic trainer, and physical therapist as appropriate. Return to play protocols should be established prior to the season by the team physician, and initiated by the physician and/or ATC as the athlete recovers. Athletes with prolonged or severe symptoms may benefit from evaluation by a neurologist with expertise in sports and/or head injuries [5, 9].

7.6 Pre-Participation Physical Exams

The pre-participation physical exam (PPE) is an important part of medical coverage for all athletes and teams. Every athlete should be evaluated annually to address any health concerns and screen for any potential medical issues. These exams should be performed by the team PCSM physician and/or orthopedic surgeon, but can be

assisted by athletic trainers, nurses, and medical assistants to help expedite the process especially with larger groups of athletes.

The overall goal of performing the PPE is to promote the health and safety of the athlete both in their training and in competition. It has been considered a screening tool to evaluate for injuries, illnesses, or other factors that may be preventable for future illnesses or injuries. For the PPE to be effective, it must be sensitive, accurate, affordable, and practical. Current data on the ability for the PPE to satisfy these criteria are lacking, and research demonstrates that PPE has little effect on the overall morbidity and mortality of athletes [17].

The National Federation of State High School Association (NFHS) considers the PPE a prerequisite for participation. The National Collegiate Athletic Association (NCAA) recommends, and most institutions require, a PPE at least on entrance to the program. Youth sports governing bodies do not have a uniform or consistent requirement for a PPE. This large group of young athletes participates with little or no formal screening prior to sports activities other than routine well-child examinations [18]. No uniform requirements exist for professional athletes, so the standard PPE varies between different professional sports organizations.

The primary PPE goals and objectives are to detect potentially life-threatening or disabling medical or musculoskeletal conditions, and to screen for medial or musculoskeletal conditions that may predispose an athlete to injury or illness during training or competition. The secondary goals and objectives are to determine general health, to serve as an entry point into the health-care system for adolescents, and to provide an opportunity for discussions on health and lifestyle issues [18].

The PPE process involves a questionnaire, review of systems, vital signs, and a physical examination. The questionnaire should be reviewed by both the athlete and their guardians. The questions focus on family history and personal history items that may be flags for abnormalities that can place the athlete at risk for illness or injury. A comprehensive personal and family

history is critical to identifying a proportion of asymptomatic athletes at risk for sudden cardiac death (SCD) [18]. The review of systems focuses on all organ systems that can be problematic for the athlete. Vital signs should be reviewed by the examining provider. The physical examination should involve both a general medical evaluation and an evaluation focused on the musculoskeletal system. These aspects of the PPE should be standardized to evaluate the overall health of the athlete.

A focus of the PPE is to evaluate for the risk of sudden cardiac death (SCD). The exact incidence of SCD is unknown in the United States, as studies to date have relied on nonmandatory reporting systems and surveys that may underestimate the true incidence [19]. SCD in athletes occurs more commonly in males, with a male:female difference ranging from 5:1 to 9:1 [20]. Although SCD can occur in any athlete, these deaths occur more frequently in those participating in football and basketball in the United States. A disproportionate amount (>40%) occur in United States African American athletes [18]. Therefore, there should inherently be an emphasis on screening for SCD in this population.

SCD in young athletes is due to a heterogeneous group of structural cardiovascular abnormalities and primary electrical diseases that can go undetected in otherwise healthy-appearing athletes [19, 20]. In the United States, hypertrophic cardiomyopathy (HCM) and congenital coronary artery anomalies are the most common etiologies of SCD. While the combined prevalence of all cardiovascular disorders known to cause SCD in the young athletic population is estimated to be 3:1,000 [21].

The American Heart Association (AHA) recommends the PPE to include auscultation for heart murmurs, palpation of femoral pulses to exclude aortic coarctation, examination for the physical stigmata of Marfan syndrome, and a brachial artery blood pressure taken in the sitting position [21]. Auscultation of the heart should be performed in both the supine and standing positions (or with Valsalva maneuver), specifically to identify murmurs of dynamic left ventricular outflow tract obstruction, most specific for HCM [18].

A highly-debated topic in sports medicine and sports cardiology is the screening electrocardiogram (ECG). The AHA recommended against cardiovascular screening of asymptomatic athletes with the ECG because of a poor sensitivity, high false-positive rate, poor positive predictive value, poor cost-effectiveness, and the total cost of implementation [22]. The governing associations of several US and international professional sports leagues, specifically football and basketball, support the routine use of ECG, in addition to personal and family history and physical examination, in the pre-participation screening of athletes [23]. Recent studies are helping to refine the ECG criteria used to distinguish normal from abnormal findings in athletes in hopes to increase effectiveness and decrease the false-positive rate of ECG screening.

Any athlete with a PPE screening concerning for hypertrophic cardiomyopathy (HCM) or other cardiac abnormality should be referred to a cardiologist for further testing, including ECG, echocardiography, and/or cardiac MRI, before being cleared to return to play. Cardiologists with specific knowledge or experience working with athletes are preferred if available, but appropriate studies can be performed by most cardiologists [5].

7.7 Equipment

The medical team will require several resources in addition to the personnel outlined above. Supplies, equipment, and facilities must be available to help treat and support the athletes.

Equipment should be available for basic management of traumatic injuries at all basketball practices and games. Providers and Athletic Trainers covering games should be prepared for primary management of injuries ranging from minor to severe. All medical personnel should be trained in and practice universal precautions to protect against exposure to biohazardous material.

All providers should be trained in the use of an automatic external defibrillator (AED). If an athlete collapses and is unresponsive, SCD should

be suspected until proven otherwise. Early defibrillation and early CPR are the most significant factors in improving outcomes in these cases, and all providers should be fully prepared to initiate these as soon as possible. An AED should be readily available and easily accessible during all practices and games. Additionally, all medical personnel should complete refresher training prior to the start of each season to maintain their skills, as studies have shown that familiarity with rarely used skills and protocols will deteriorate after only a few months without practice or simulation.

In addition, all basketball practices and competitions should have equipment available to manage a traumatic spinal cord injury. While the utility of routine use of long backboards for immobilization in cases of suspected injury has recently come under question, some equipment for stabilization and transport of athletes with suspected spinal injuries should be available, and all medical personnel should be trained on and familiar with their use.

Athletic trainers covering basketball events should have medical sideline bags that include tools for basic injury management.

Medical providers covering basketball events should also have basic tools for injury management, as well as equipment for more advanced medical care.

It is important to remember to keep medical equipment readily available while traveling with a team. Medical issues can occur while in transit, so essential supplies should not be stored away with the luggage or in another vehicle, in case an incident occurs.

Available imaging modalities will vary depending on resources. There are no universal guidelines for what imaging modalities should be available. Most professional venues will have basic radiographic imaging available on-site, while advanced imaging will typically require transport to a nearby medical center or hospital. High school and college events will have a wide variety of imaging availability, with some having installed or portable X-ray, but others necessitating transport for any imaging. Portable ultrasound has become an increasingly popular

imaging modality that may be available on-site, but will usually be dependent on present medical personnel.

7.8 Emergency Action Plan

Every location utilized for a basketball practice or competition must have an associated emergency action plan (EAP) in place for emergent events. The EAP is a comprehensive plan set in place for each emergency scenario that could occur during a sporting event or practice. It not only outlines initial treatment steps that should take place on the court or in the locker room but will also include detailed protocols including maps and diagrams of relevant facilities, methods of transportation, routes, destinations, emergency contact information, and lists and roles of all personnel involved. For example, in the setting of a player cardiac arrest, the EAP should specify the location of the AED, the entry way for emergency medical personnel (EMS), the method of transportation (ambulance), and the hospital to which the player will be transported. The athletic trainer or athletic director will oversee the coordination of the EAP, in conjunction with the team physician and local emergency medical services (EMS), and should include all services responsible for covering the event. Furthermore, it is the responsibility of the athletic trainer to ensure that all medical personnel are oriented to the execution of the EAP for all foreseeable scenarios, and the plan should be reviewed with physicians, trainers, and the rest of the medical team on a regular basis. The EAP is the framework for how the medical personnel will manage emergency, nonlife-threatening, and life-threatening events, and should be considered of the utmost importance for all sporting events.

7.9 Summary

In constructing a medical team to care for a basketball team, a multidisciplinary approach with cooperation between physicians, athletic trainers, physical therapists, and specialists can help meet the wide-ranging needs of the athletes.

While teams at different levels will have different resources and capabilities, and the ideal situation may not always be achievable, at a minimum the medical team should maintain the personnel, equipment, and facilities to address acute and chronic injuries and illnesses of the athletes, emergent situations, and regular screening physical exams.

References

1. Borowski LA, Yard EE, Fields SK, Comstock RD. The epidemiology of US high school basketball injuries 2005-2007. *Am J Sports Med.* 2008;36(12):2328-35.
2. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the National Basketball Association: a 17-year overview. *Sports Health.* 2010;2:284-90.
3. Huget J. The pathology of basketball. Reports by the Medical Commission of Federation International Basketball Associations; 1999.
4. McKay GD, Goldie PA, Payne WR, Oakes BW, Watson LF. A prospective study of injuries in basketball: a total profile and comparison by gender and standard of competition. *J Sci Med Sport.* 2001;4(2):196-211.
5. Needham M, Asplund C. Basketball. In: O'Connor FG, Casa DJ, Davis BA, Pierre PS, Sallis RE, Wilder RP, editors. *ACSM's sports medicine: a comprehensive review.* Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2013. p. 579-85.
6. Arendt EA, Agel J, Dick R. Anterior cruciate ligament injury patterns among collegiate men and women. *J Athl Train.* 1999;34(2):86-92.
7. Lim BO, Lee YS, Kim JG, An KO, Yoo J, Kwon YH. Effects of sports injury prevention training on the biomechanical risk factors of anterior cruciate ligament injury in high school female basketball players. *Am J Sports Med.* 2009;37(9):1728-34.
8. Myers CA, Hawkins D. Alterations to movement mechanics can greatly reduce anterior cruciate ligament loading without reducing performance. *J Biomech.* 2010;43(14):2657-64.
9. Micheo W. Head and face considerations. *Olympic handbook of sports medicine: basketball.* Oxford, UK: Blackwell; 2003.
10. American Academy of Ophthalmology. Protective eyewear for young athletes. Joint policy statement of the American Academy of Pediatric and American Academy of Ophthalmology; 2013. <https://www.aao.org/clinical-statement/protective-eyewear-young-athletes>
11. Jones NP. Eye injury in sport. *Sports Med.* 1989;7(3):163-81.
12. Azodo CC, Odai DC, Osazuma-Peters N, Obuekwe ON. A survey of orofacial injuries among basketball players. *Int Dent J.* 2011;61(1):43-6.

13. Cohenca N, Roges RA, Roges R. The incidence and severity of dental trauma in intercollegiate athletics. *J Am Dent Assoc.* 2007;138(8):1121–6.
14. Kerr IL. Mouth-guards for the prevention of injuries in contact sports. *Sports Med.* 1986;3(6):415–27.
15. Spinass E, Savasta A. Prevention of traumatic dental lesions: cognitive research on the role of mouthguards during sports activities in paediatric age. *Eur J Paediatr Dent.* 2007;8(4):193–8.
16. Oral Health Policies and Recommendations (Reference Manual). American Academy of Pediatric Dentists. http://www.aapd.org/media/policies_guidelines/g_trauma.pdf
17. Best TM. The preparticipation evaluation an opportunity for change and consensus. *Clin J Sport Med.* 2004;14:107–8.
18. Bernhardt DT, Roberts WO. PPE preparticipation physical evaluation. 4th ed. China: American Academy of Pediatrics; 2010. p. 1–41.
19. Maron BJ, Shirani J, Poliac LC, et al. Sudden death in young competitive athletes. Clinical, demographic, and pathological profiles. *JAMA.* 1996;276(3):199–204.
20. Maron BJ. Sudden death in young athletes. *N Engl J Med.* 2003;349(11):1064–75.
21. Maron BJ, Thompson PD, Ackerman MJ, et al. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update: a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism; endorsed by the American College of Cardiology Foundation. *Circulation.* 2007;115(12):1643–455.
22. Maron BJ, Thompson PD, Puffer JC, et al. Cardiovascular preparticipation screening of competitive athletes. A statement for health professionals from the Sudden Cardiac Death Committee (clinical cardiology) and Congenital Cardiac Defects Committee (cardiovascular disease in the young). American Heart Association. *Circulation.* 1996;94(4):850–6.
23. Harris KM, Sponsel A, Hutter AM, et al. Brief communications: cardiovascular screening practices of major North American professional sports teams. *Ann Intern Med.* 2006;145(7):507–11.

The Role of Pre-participation Assessment and Screening in Basketball

8

Mark Rogers and Shan Fairbanks

8.1 Introduction and Goals of PPE

The pre-participation evaluation (PPE) is a comprehensive history and physical exam performed by a medical provider for amateur and professional athletes of all ages [1, 2]. The origins of the PPE date back to the late 1800s in an attempt to bring physical education to local British primary schools. Extrusive physicians obtained a detailed history about a child's background, living conditions, and medical history prior to starting physical education [3–5]. Continued efforts were made in the United States by physicians during World War II, as mass screenings were performed on draftees by the United States Selective Service. This group found upwards of 25% of draftees unfit for military service. Results of these early screenings led to integration programs in high schools throughout the nation to prepare graduates for service. The United States then created a school health exam becoming

known as the standardized pre-participation evaluation, focusing on “correctable defects,” designed after the military medical evaluation [6, 7]. The American Medical Association (AMA) took notice calling for youth athlete screening [8].

The PPE, in the United States, is balanced by a governing agency of six medical societies including the American Academy of Family Physicians (AAFP), American Academy of Pediatrics (AAP), American Medical Society for Sports Medicine (AMSSM), American College of Sports Medicine (ACSM), American Orthopedic Society of Sports Medicine (AOSM), and American Osteopathic Academy of Sports Medicine (AOASM). The PPE unfortunately was not developed in an evidence-based format [1]. Despite greater than 40 years of active use, there is a lack of outcome-based data leading to an underlying controversy. For a PPE to be effective, the exam should be sensitive, accurate, practical, and cost-effective. Currently, the PPE is unable to meet that criteria [2]. Evidence indicates that despite a thorough PPE, there is little effect on overall morbidity and mortality [1, 9]. Even though there is an absence of evidence to reduce morbidity and mortality from cardiac etiology, examinations continue to be performed throughout the world prior to sport-specific activity [2]. Nonetheless, the PPE is considered the standard of care for athletes, including basketball players, despite the above limitations. Upwards of 75–88% of medical or orthopedic conditions can be detected by history and physical alone. This

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could help medical professionals prepare to prevent further injury and better manage conditions for all athletes.

Greater than 30 million amateur athletes and more than 3 million athletes with special needs will receive medical clearance for sporting activities yearly [2]. The overall goal of the PPE is to not withhold any competitors but promote long-term health and safety [1, 9]. PPE primary objectives include detection of conditions that may predispose an athlete to injury, detection of life threatening or disabling conditions, and meet insurance or legal requirements to play a sport [2]. Secondary goals include maintaining general health considerations (i.e., mental health, immunizations, amenorrhea, and stress fractures), counseling on health-related issues (i.e., sexually transmitted infections, performance-enhancing drugs, and disordered eating), assessing fitness levels for sport-specific activity (i.e., maturity), and serve as an entry point for adolescents to the health-care system [3, 9]. The AAP notes that nearly 10% of adolescents may have a chronic condition which may go undiagnosed due to lack of medical care [1, 2]. Regrettably the conduct, content, and efficacy of PPEs have remained unstandardized across organizations and countries [1].

Ideally, timing of the exam should be performed 6–8 weeks prior to the start of the season. This allows for adequate time for any identifiable problems to be evaluated, treated, and rehabilitated [2, 9]. Timing may be limited by many factors such as age of the participants (if underage, allowing a parent or guardian to be present to assist with the history); availability of health-care professionals to perform exams; and health insurances [10, 11].

The frequency of PPEs has also been called into question as sporting organizations have made variable recommendations. The National Collegiate Athletics Association (NCAA) requires an initial comprehensive PPE prior to matriculation with interval updated history with physical exam during the following years [1]. The American Heart Association (AHA) recommends a PPE when starting a new level of competition, beginning during high school, continuing through college, or pro level with interval cardiac screening every 2 years and annual blood pressure readings [2]. Comparatively the college athletic department

may require a yearly comprehensive exam based on school year with an entrance and/or exit exam.

8.2 Methodology of a PPE

There are many methods to organizing pre-participation exams. PPEs can be performed by a single provider in an office-based setting or a coordinated team-based format. The AAP and AAAP have recently endorsed the pre-participation physical being incorporated into the annual physical exam [9]. Advantages encompass continuity of medical care from the primary care provider (PCP), follow-up care, preventative services, and appropriate record keeping through the electronic health record. This allows examinations to be evenly distributed throughout the year, privacy in a doctor-patient relationship, and allows athletes without a medical home the opportunity to access health care. Disadvantages to the office-based model consist of limited time for appointments, greater cost, narrow physician experience in sports-related illness or injury, and potential lack of communication between PCPs to the designated athletic staff [2, 9].

Team-based approaches to the PPE include multiple health-care providers, usually led by a licensed physician (MD or DO), or advanced care provider [12]. Specialist physicians such as orthopedists, neurologists, and cardiologists may be present to lend their expertise. Additional advantages to the team-based model include time efficiency, cost effectiveness, and communication with pertinent athletic staff. The team may consist of coaches, strength personnel, nurses, medical assistants, athletic trainers, nutritionists, physical therapists, and physiologists [1, 9]. Each person will be designated a particular assignment to assist flow through each station. If group-based examinations must be performed away from the medical home, team physicians and staff are preferred (i.e., university-based staff, Olympic team providers). This allows the team physician and staff to establish relationships with the athletes they will be caring for during the allotted season(s). If performing multi-station PPEs, the AAP and ACSM have recommended closed rooms for confidential review of historical forms and physical exams [1, 2, 9].

8.3 Historical Information

The basic framework of PPEs are usually the same; however minor differences may be experienced based on the demands of certain sports [13]. For instance, the physicality of basketball has evolved and is now becoming accepted as a contact sport, with players suffering more tragic injuries [13]. A thorough medical history remains an essential component of the PPE encounter to identify medical conditions that may interfere with athletic participation. All previous medical issues should be disclosed and addressed. The PPE historical documentation should be completed by the athlete, or jointly by a parent or legal guardian if underage. Emphasis should be placed on cardiovascular, musculoskeletal, and neurologic pathology. The medical history improves the accuracy of the PPE. Medical history including medications, supplements, past surgeries, and positive screening questions should be reviewed by the physician in a private setting, allowing for subsequent questions to determine any further intervention or testing.

8.3.1 Medications and Supplements

All medications prescribed, over the counter medications, and supplements should be reviewed during the evaluation. Routine documentation of all prescribed medications for each athlete, including therapeutic use exemption (TUE), should be available upon request. In competitive sports such as basketball, governing agencies (i.e., NBA, WADA, FIBA) may have a list of prohibited medications and supplements. Reviewing protocols for inappropriate medications and banned substances by medical staff should be performed yearly within the annual or interim PPE [13]. This is also an opportunity to counsel players on the hazards of these substances and provide reminders of which substances are banned [13, 14].

8.3.2 Cardiac History

The cardiovascular portion of the PPE has been a source of debate over the last few years second-

ary to high-profile cardiac deaths of elite athletes in sports like football and basketball [13, 14]. Cardiovascular disorders are a leading cause of athlete death. The risk of sudden cardiac death (SCD) in male, African American college athletes is 6.25 per 100,000. The risk of SCD rises to 19 per 100,000 in NCAA college basketball players [15]. The underlying rationale in cardiac screening is to identify cardiac pathologies and attempt to prevent sudden cardiac death [13]. SCD is usually caused by structural diseases within the heart (i.e., hypertrophic cardiomyopathy, congenital coronary anomalies, myocarditis), valvular pathology (i.e., aortic stenosis or rupture), and arrhythmias (i.e., Long QT, Brugada, Wolff Parkinson White).

The AHA and American College of Cardiology (ACC) have created a risk stratification questionnaire used during PPEs to reduce the risk of sudden cardiac death related to heart conditions associated with physical activity and exercise. Questions include red flag symptoms and family history which are used to detect potential etiologies for SCD [16] (see Table 8.1). Objective findings which may be consistent to SCD include new onset heart murmurs or hypertension. Family history is also extremely important; questions should include any known genetic disorders (i.e., long QTc, HCM), previous death of a family member younger than 35 years old from an unknown cause, or heart disease in close relative prior to 50 years old.

8.3.3 Cardiac Exam

The cardiac exam is indispensable to the PPE and should be comprised of brachial artery blood pressure with an appropriate-sized cuff, in both arms. Elevated blood pressures should be rechecked after a 15-min rest period. All blood pressures greater than 160/100 in adults and greater than the 95th percentile in children should be further investigated with lab work, EKG, and consideration for renal artery duplex for underlying secondary hypertensive etiologies. Participation should be delayed until diagnostic work-up is complete. Upper and lower extremity pulses should be palpated to assist in ruling out coarctation of the aorta and irregular pulses

Table 8.1 AHA screening recommendations

American Heart Association recommendations on screening for cardiovascular abnormalities in competitive athletes
Personal cardiac history:
– Elevated blood pressure
– Excessive dyspnea or fatigue with exercise
– Exertional chest pain or discomfort
– Prior heart murmur
– Unexplained syncope or near syncope with exercise
Family history:
– Disability from heart disease in close relative prior to 50 years of age
– Premature death (sudden or unexpected) before 50 years of age
– Specific knowledge of certain cardiac conditions: hypertrophic or dilated cardiomyopathy, long QT syndrome, Marfan syndrome, or arrhythmias
Physical exam:
– Brachial artery blood pressure, performed in the sitting position
– Height and weight
– Cardiac auscultation with provocative maneuvers
– Physical signs of Marfan’s syndrome
– Femoral pulses, to evaluate for coarctation

Adapted with permission from the American Heart Association, Maron BJ, Thompson PD, Ackerman MJ, et al. Recommendations and Considerations Related to Preparticipation Screening for Cardiovascular Abnormalities in Competitive Athletes: 2007 Update; A Scientific Statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism: endorsed by the American College of Cardiology Foundation. *Circulation*. 2007;115(12):1643–1655

alluding to arrhythmia. Heart auscultation should be performed in a quiet exam room in both the standing and supine positions. Murmurs should undergo provocative maneuvers to help differentiate pathologic from functional murmurs. Any systolic murmurs with grades III to VI in severity, diastolic murmurs, or a murmur louder with valsalva should undergo echocardiogram [9].

The last portion of the cardiac exam should include screening for Marfan syndrome stigmata. Marfan syndrome is an autosomal dominant connective tissue disorder of multiple organ systems (i.e., cardiac, musculoskeletal, ocular) secondary to mutations in FBN1 gene which codes for fibrillin 1 [17]. Most tall athletes do not have Marfan syndrome although certain athletes, including basketball players, may be suspected based on their stature. Phenotypically Marfan syndrome varies significantly. Objective

findings may include chest deformities (pectus excavatum), arachnodactyly, arm span greater than height, scoliosis, ectopic eye lens, systolic murmur exaggerated with provocative maneuvers, Steinberg sign (tip of thumb protrudes while thumb folded inside a fist), wrist sign (thumb and index finger encircle the wrist), and a previous family history [9, 17]. Diagnosing Marfan syndrome should be based on symptom score or revised Ghent criteria [17].

8.3.4 Screening EKG

Pre-participation electrocardiogram (EKG) remain a source of debate as to whether they are worthy of being part of the PPE screen. Many major medical societies agree that EKGs are known to improve the sensitivity of the PPE cardiac screening to near 60% in association with identifying certain cardiac conditions that may cause SCD [13]. Currently, the overall consensus by specialty organizations recommend against routine EKG screening secondary to higher false-positive and false-negative rates. According to those organizations, EKG testing should be reserved for athletes who demonstrate high-risk cardiac behavior on history or physical exam. False-positive EKG testing may actually be induced by normal physiologic changes to an athlete’s heart from prolonged training and improved cardiovascular fitness [9, 18, 19]. A study from the Veneto region of Italy in the 1970s showed a decreased risk of morbidity and mortality by performing routine EKGs on their young athletes. Since this study’s results have not been reproduced, the cost–benefit ratio utilizing screening EKGs to decrease morbidity and mortality lends itself to debate. Although new evidence has been identified in athlete-specific EKG interpretation, the Seattle criteria has compressed false-positive rates below other commonly used screening tests [18]. Performing routine EKGs may prove a benefit to higher risk populations such as African American or Caribbean males, basketball players, and high endurance athletes [18, 19]. More evidence will be needed to determine the future of screening EKGs during PPEs and whether they should become standard practice.

Commonly at the collegiate and professional levels, screening EKGs are performed routinely; however this discussion should be carefully considered by the medical team. This is supported by a recent prospective study using 3620 high school athletes from 2014 to 2017 performed by Williams [20], which compared the AHA-14-point cardiac history to a resting EKG using the Seattle criteria to identify cardiovascular risk factors associated with SCD. Limited echocardiograms were also performed for all screening abnormalities to assist in evaluation of the athlete. Results indicate that 22.5% of student ath-

letes had at least one positive response to the cardiovascular screening questions. The physical exam was abnormal in 9.8% of athletes, and 2.8% of athletes demonstrated an abnormal EKG. The sensitivity (18.8%), specificity (68.0%), and positive predictive value (0.3%) of the AHA 14-point evaluation was substantially lower than the sensitivity (87.5%), specificity (97.5%), and positive predictive value (13.6%) of EKG group [20]. The conclusion from the study is encouraging to support 14-point cardiovascular history alone may not be adequate enough without the assistance of EKG in predicting SCD.



Electrocardiogram (EKG) screening. Picture courtesy of Connor Rogers

8.3.5 Musculoskeletal History

Musculoskeletal injuries remain the forefront of any team physician or health-care professional during a PPE. The goal of the musculoskeletal history and physical exam is to localize bone, muscle, or joint pathology to prevent injury or use the examination as a baseline for return to play activities. Questions should focus on preceding sprains, strains, fractures, dislocations, or tears to the musculoskeletal system that limited the athletes' playing time. The length of time

away from practice or games may help elicit the severity of the injury [2, 9]. If the pain is present from previous injuries, or if the injury is unstable or weak may prompt further rehabilitation before medical eligibility is granted.

Basketball players use explosive maneuvers like running, jumping, shuffling, and cutting on hardwood floors. Being mindful of sport-specific movements may assist a provider's physical exam in attempts to provoke weaknesses, imbalances, or instabilities. Basketball is played with significant forces from the ground through the

lower extremity, pelvis, and torso. As a result, lower extremity injuries are the most prevalent [1]. Providers should ask about previous stress fractures or overuse injuries to the lower extremities. Lastly, questioning about a personal and family history of rheumatologic, degenerative, or other joint conditions may be appropriate.

8.3.6 Musculoskeletal Exam

The musculoskeletal physical exam should focus on major joints overlying the extremities and axial skeleton. In athletes who are asymptomatic with no preceding injuries, a general screening exam from head to toe is justifiable although sensitivity may be as low as 50% [21]. The general musculoskeletal exam consists of inspection, palpation, range of motion, strength, and neurovascular assessment of the extremities. Spine can be evaluated efficiently using range of motion, palpation, and provocative testing [21].

If any abnormalities found within the general screening exam or known injuries, individual joint evaluations should be performed using high-yield special tests. Testing each joint thoroughly may avoid injury and enhance performance but is also time consuming and may be challenging to perform during a routine screening PPE unless a physician has detailed knowledge of sport-specific activity [21–23]. Any positive testing compared from side to side should be further investigated with functional screening or diagnostic testing.

8.3.7 Functional Movement Screening vs. Sport-Specific Testing

Functional movement screening (FMS) is a tool to assist health-care professionals screen patients and athletes to predict injury by monitoring dysfunctional movement patterns. FMS is used to identify asymmetries in the kinetic chain and core to analyze shortcomings in activity which can be corrected through pertinent exercises [22]. The original thought was that strengthening these

deficiencies in the kinetic chain and core would predict and decrease injury. Unfortunately, evidence to support screening athletes during PPEs with functional movement testing is limited and relatively unclear of benefits [22–25].

Basketball remains a popular sport in developed nations. Different age groups have their underlying problems and pathology. Younger basketball players who are developing their musculoskeletal and neuromotor functions may have more difficulty with basketball-specific movements [23]. Basketball players unload energy from the floor through their hips using dynamic lateral movements, quick change of direction, and eccentric loading. These powerful moves put strain on the neuromuscular control of the lower extremities and core. The hip and pelvis serve as a force transfer length between the lower extremity and torso, as such is an at-risk region for injury. The hip, knee, and ankle are predisposed to injuries secondary to overuse, instability, lower crossed syndrome (see Table 8.2 and Fig. 8.1), and ligament and tendon damage. Predicting injuries using FMS has been studied for the 20 plus years. Although FMS has some promise in rehabilitation for lower extremity and core injuries, most data using FMS as a prevention or prediction model for injury is circumstantial at best [24–26].

Identifying injury risk and implementing preventive measures can assist with reducing injury occurrence and may ultimately improve athletic performance. Improper movement patterns often contribute to musculoskeletal injuries [23]. Sport-specific testing, looking at the most common injuries, and mechanisms is the next step in screening. Screening tools that inspect the kinetic chain, functional testing, balance, posture, and flexibility are great for evaluating musculoskeletal deficiencies but may not be indicative to predicting injuries, and are limited based on age, sex, and body mass of the athlete [23, 26, 27].

There is some evidence to using the Y-balance and star excursion balance testing in identifying those at risk for lower extremity injuries but according to Plisky, evidence may be bound only to high school men and women [28]. Single leg squats and the drop box testing have some

Table 8.2 Muscle imbalance/lower crossed syndrome

Lower crossed syndrome (pelvic crossed syndrome)—common in basketball athletes

- Muscle deficiency of the core, imbalance between muscles shortened or lengthened of ventral and dorsal sides
- Caused by strength imbalances in the lower segment
- Overactivity in hip flexors and lumbar extensors which causes weakness in the lower abdomen, gluteus maximus, and medius

Two types

- A: Anterior pelvic tilt and hip flexion, hyperlordosis lumbar spine, hyperkyphosis thoracic spine
- B: Minimal lordosis lumbar spine, minimal kyphosis thoracic spine, anterior head carriage, knee recurvatum

Exam

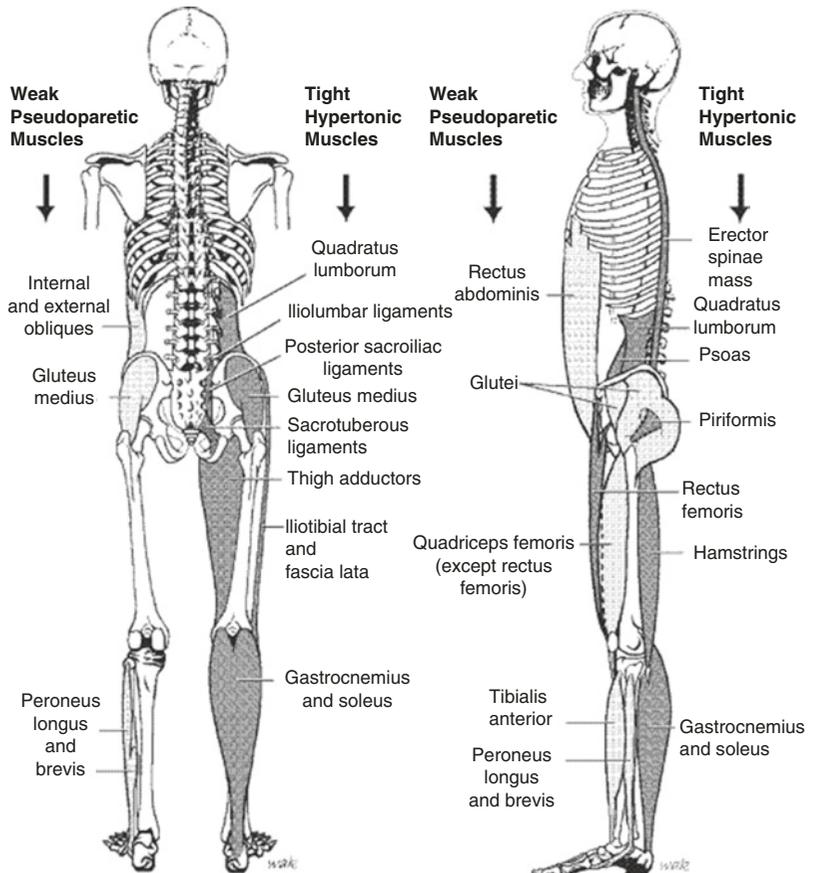
- Inspection of compensatory patterns
- Gait analysis
- Passive muscle testing
 - Thomas test, straight leg test for hamstrings, quadratus lumborum testing, erector spinae flexion, and extension
- Active muscle testing
 - Hip extension (hyperextension phase gait), hip abduction (will see hip abduction and lateral flexion), trunk curl (difference in strength abdominal muscles vs. iliopsoas), gluteus muscle firing pattern

Management

- Rehabilitative exercises targeting posture, core strengthening, and gait training
- Reverse the compensatory pattern
- Manual medicine, trigger point injections
- Ultrasound, laser therapy, dry needling, and/or acupuncture

Adapted from Physiopedia contributors. “Lower Crossed Syndrome.” Physiopedia, Physiopedia, 9. 2019, www.physio-pedia.com/index

Fig. 8.1 Muscle imbalance found in lower crossed syndrome. (Used with permission from Kuchera, M. Postural Considerations in Osteopathic Diagnosis and Treatment. In: Chila, AG, Foundations of Osteopathic Medicine, 3rd Ed. Philadelphia. Lippincott, Williams & Wilkins, 2011) [46]



legitimacy in predicting ACL tears by demonstrating weakness in a valgus stance, but mixed opinions commonly leave them out of the routine PPE [27]. These combinations of muscle testing for neuromuscular deficiencies in basketball players may not be deemed part of the PPE but could be used by strength and conditioning coaches during preseason to assess sport-specific activities and opportunities for improvement [28].



Musculoskeletal screening with single leg squat and gluteus muscle firing pattern testing. Pictures courtesy of Connor Rogers

8.3.8 Common Injuries in Basketball

Injuries like sprains, strains, fractures, and dislocations have become part of almost every sport. Basketball is no different. What started as a non-contact sport created by James Naismith in the late 1800s has now become an everyday physical contact sport [9]. Contact injuries have become more common than noncontact injuries during basketball activity and are more likely to occur during practice [23–29].

Lateral, inversion ankle sprains are the most prevalent injury in basketball players. Treatment is usually conservative and athletes recover quickly within 7–10 days [29]. Other common lower extremity injuries seen in younger basketball players may include jumper's knee or Osgood–Schlatter disease, both of which are overuse injuries to the patella tendon [9, 30]. The developing basketball player who is required to jump upwards of 50 times a game and shuffle from side to side may get irritation along the borders of the anterior knee.

Men and women basketball players get similar injuries as they grow into mature athletes. Adult athletes continue to get bigger, faster, and stronger placing more pressure on the joints and supporting structures. Two-thirds of all injuries to men and women basketball players occur in the lower extremity [31]. Differences in the male to female kinetic chain and wider set hips provoke a larger Q angle to the knee. The anterior cruciate ligament is a commonly injured ligament injured in a noncontact fashion while squatting, jumping, or shuffling [32]. Instability of the ankle due to rapid growth allows higher frequencies of ankle sprains [29, 30].

Traumatic fractures to the lower extremities are common for basketball players due to size, weight, and the forces that have occurred while jumping [9]. Stress fractures of the tibia, tarsal, and navicular bones are the most common type of fracture in basketball players. These stress reactions to the bone are caused by continuous running and launching off the ground and repeatedly landing on the hardwood. Stress fractures are not due to cortical bones fracturing from repeated

use but rather when healthy bone is subject to a level of activity far more intense than what would normally be feasible [33].

8.3.9 Neurological History

The neurological history focuses on detection of previous head or neck injury, sports-related concussions, headaches, radicular symptoms, neuropraxia, previous seizures, or any advanced head imaging. The goal of this portion of the exam is to elicit any neurological conditions that may hinder sports participation, performance, or exclude medical eligibility [9, 34].

Sports-related concussion has been controversial in the sports medicine world. Concussions are a form of traumatic brain injury caused by mechanical forces directed at the brain. This energy may be focused at the head, face, or elsewhere on the body which in turn precipitate neurological dysfunction that will eventually resolve over time. Usually these symptoms are functionally based, and no large structural damage can be visualized [35]. Neurological imaging commonly would be negative although imaging is not recommended with concussions. This mild traumatic injury to the brain is a metabolic process secondary to oxidative stress [36]. When taking a history of a patient with previous concussions, concentration of the total number of concussions, frequency, severity, previous treatments, length of recovery, and most recent concussion should be reviewed. Eligibility due to concussions should not be determined on total number but rather based on symptoms. With each new concussion, symptoms may be prolonged and risk for future concussions rises [37].

8.3.10 Neurological Exam and Baseline Concussion Testing

Conventional neurological exams should include cranial nerve testing, sensation to the upper and lower extremity dermatomes, gross motor exam, and deep tendon reflexes. Concussion baseline

testing may be performed as part of preseason testing or as an adjunct to the PPE. The NCAA does recommend a onetime pre-participation assessment to include cognitive assessment, balance evaluation, symptom score, and concussion history. Most universities have the resources to maintain a database for all athletes specifically for concussion evaluation and management [38]. Baseline vestibular ocular motor screening (VOMS) in contact sport athletes (i.e., football, basketball, wrestling) is not a requirement during PPEs but could assist in return to play activities for athletes.

8.4 Screening Tests

Screening tests are proactive tests used in prevention to detect disease that may be present without symptoms. These further studies should be used as an extension to the regular exam [2, 39]. Although, the AAP and AAFP recommend against routine screening labs during the PPE in asymptomatic athletes because there has been no improvement in outcome. Some athletes who have chronic illnesses require their regular screening labs based on their pathology [9].

The NCAA mandates that all incoming athletes provide blood for a onetime sickle cell screen. Since implementing this testing in mid 2010s exercise induced death due to sickle trait has decreased. Further investigation will need to be completed to determine if this reduction is due to screening for sickle cell or due to education on the topic [2, 9, 40]. Other screening labs such as complete blood count, metabolic profiles, vitamin D, and ferritin may be of benefit in basketball players. Despite positive feedback with early asymptomatic screening by the NCAA, regular use of screening labs is not supported (Fig. 8.2).

8.5 Athletes with Intellectual and Physical Disabilities

A growing community of sports participants that require specialty accommodations are athletes with disabilities. Equal opportunity to



Screening Tests



**** All NCAA student-athletes must have a sickle screen PRIOR to any participation**

Cheerleading and High Techs are exempt, but may have screen if desired/indicated

LAX-Lacrosse; SOC-Soccer; SB-Softball; TK-Track; TN-Tennis; VB-Volleyball; XC-Cross Country, WR-Wrestling

Fig. 8.2 VIRGINIA TECH screening lab protocol during PPE

sports for those with physical or cognitive disabilities has increased interest from legislators. Disabilities include those who are blind, deaf, or amputees. Genetic disorders include Down syndrome, Prader–Willi syndrome, fragile X syndrome, and cerebral palsy, among others. Physical and mental benefits are abundant for disabled athletes [41]. Physically, there is an improvement in cardiovascular function, musculoskeletal strength, increased exercise tolerance, balance, gross, and fine motor skills. Mentally, an athlete will benefit from reduced anxiety, improved self-esteem, encouragement, integration, and support from the community similar to themselves [41, 42].

A PPE for someone with underlying physical or intellectual disability should not deviate much from the current PPE format, except addressing unique features of the specific disability. PPE goals for those with disabilities should be to encourage safe and effective athletic participation with intent on prevention of further disability or death [43]. Specific physical and mental limitations should be documented. All positive

screening questions should be followed with secondary inquiry with greatest concern for sports participation [42, 43].

Examination should also focus on ocular, cardiovascular, neurologic, musculoskeletal, and dermatologic systems. If a prosthetic is present, a prosthetist may augment the evaluation to inspect the limb for proper alignment. Functional exam is more prevalent in this population and individualized for each athlete based on pathology. Diagnostic studies such as cervical spine radiographs of the atlanto-axial joint in athletes with Down syndrome may be considered prior to participation to demonstrate stability [43–45].

Medical eligibility should follow regular pre-participation guidelines. Emphasis should be placed on safe participation of sport and inclusion. Like routine PPEs, the disabled athletes exam has not been standardized, and each athlete's medical, physical, and mental health should be taken into consideration [42]. More subspecialty care may be needed to assist medical providers to allow for competition.

Summary Take Home Points

- Pre-participation evaluations are not evidence-based although PPEs are the gold standard for evaluating an athlete.
- Primary objectives include detection of conditions that may predispose an athlete to injury, detection of life threatening or disabling conditions, and to meet insurance or legal requirements.
- Screening should focus on the cardiovascular, musculoskeletal, and neurological systems.
- History alone using AHA guidelines may not be enough to prevent SCD; EKGs although not routine may benefit basketball players during their PPE.
- Sport-specific screenings may have validity in PPE musculoskeletal screens in prediction of injury as basketball players use tremendous forces through their lower extremities leaving them at high risk for injury.
- Sports-related concussion testing is required by most sport entities although not usual to the PPE.
- Athletes with disabilities require pathology-specific screening for eligibility.

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References

1. Berhardt MD, David T, Roberts WO. PPE preparticipation physical evaluation. 5th version. 5th ed. Itasca: McGraw-Hill and American Academy of Pediatrics; 2019.
2. Mirabelli MD, Mark H. The Preparticipation sports evaluation. *Amer Fam Phys*. 2015;92(5):371–6.
3. Riebe D, Franklin BA, Thompson PD, et al. Updating ACSM's recommendations for exercise preparticipation health screening. *Med Sci Sports Exerc*. 2015;47(11):2473–9.
4. Carter AH. A discussion on the claims and limitations of physical education in school. *BMJ*. 1990;2(1557):995–1001.
5. Neve M, Turner T. What the doctor thought and did: sir James Crichton Browne (1840–1938). *Med Hist*. 1995;39(4):399–432.
6. Gallagher J, Brouha L. Physical fitness: its evaluation and significance. *JAMA*. 1944;125(12):834–8.
7. Kleinschmidt EE. The schools contribute to national defense. 1941. *J Sch Health*. 2001;71(8):378–82.
8. Subcommittee on Classification of Sports Injuries. Committee on the Medical Aspects of Sports, American Medical Association. Standard nomenclature of athletic injuries. Chicago, IL: American Medical Association; 1966.
9. Khodee M, Putakian M, Madden CC. The preparticipation physical evaluation. In: Madden CC, Putakian M, McCarty EC, Young CC, editors. *Netter's sports medicine*. 2nd ed. Philadelphia: Elsevier; 2018.
10. Committee on Practice and Ambulatory Medicine, Bright Futures Periodicity Schedule Workgroup. 2017 Recommendations for preventive pediatric health care. *Pediatrics*. 2017;139(4):e20170254.
11. Krowchuk DP, Krowchuk HV, Hunter DM, et al. Parents' knowledge of the purposes and content of preparticipation physical examinations. *Arch Pediatr Adolesc Med*. 1995;149(6):653–7.
12. Pickham D, Chan G, Carey M. Pre-participation screening for athletes and the role of advanced practice providers. *J Electrocardiol*. 2015;48(3):339–44.
13. Targett S, Bere T, Bahr R. The role of pre-participation assessment (PPA) and screening in handball. In: Laver L, Landreau P, Seil R, Popovic N, editors. *Handball sports medicine*. 1st ed. Heidelberg: Springer; 2018.
14. 2019–2020 NCAA Banned Substances. NCAA. www.ncaa.org/sport-science-institute/topics/2019-20-ncaa-banned-substances.
15. Harmon K, Asif I, Ellenbogen R, Drezner J. The incidence of sudden cardiac arrest and death in the United States high school athletes. *Br J Sports Med*. 2014;48(7):605.
16. Maron BJ, Thompson PD, Ackerman MJ, et al. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update; a scientific statement from the American Heart Association Council on Nutrition, Physical Activity, and Metabolism: endorsed by the American College of Cardiology Foundation. *Circulation*. 2007;115(12):1643–55.
17. Braverman MD, Alan C. Recognizing Marfan syndrome in athletes. *American College of Cardiology*; 2015. www.acc.org/latest-in-cardiology/articles/2015/03/02/13/03/recognizing-marfansyndrome-in-athletes.
18. Asif IM, Prutkin JM. Modern standards of ECG interpretation in young athletes: yield and effectiveness. *J Electrocardiol*. 2015;48(3):292–7.
19. Corrado D, Basso C, Pavei A, et al. Trends in sudden cardiovascular death in young competitive athletes after implementation of a pre-participation screening program. *JAMA*. 2006;296(13):1593–601.

20. Williams E. Performance of the American Heart Association (AHA) 14-point evaluation versus electrocardiography for the cardiovascular screening of high school athletes: a prospective study. *J Am Heart Assoc.* 2019;8(14):e012235.
21. Gomez JE, Landry GL, Bernhardt DT. Critical evaluation of the 2-minute orthopedic screening examination. *Am J Dis Child.* 1993;147(10):1109–13.
22. Cook G, Lee B. Functional movement screen (FMS). Physiopedia. [www.physio-pedia.com/Functional_Movement_Screen_\(FMS\)](http://www.physio-pedia.com/Functional_Movement_Screen_(FMS)).
23. Bond C, Dorman J, Odney T, Roggenbuck S, Young S, Munce T. Evaluation of the functional movement screen and a novel basketball mobility test as an injury prediction tool for collegiate basketball players. *J Strength Cond Res.* 2019;33(6):1589–600.
24. Ugalde V, Brockman C, Bailowitz Z, Pollard C. Single leg squat test and its relationship to dynamic knee valgus and injury risk screening. *PM R.* 2014;7(3):229–35.
25. Warren M, Smith C, Chimera N. Association of the functional movement screen with injuries in Division I athletes. *J Sport Rehabil.* 2015;24(2):163–70.
26. Šiupšinskas L, Garbenytė-Apolinskienė T, Salatkaitė S, Gudas R, Trumpickas V. Association of pre-season musculoskeletal screening and functional testing with sports injuries in elite female basketball players. *Sci Rep.* 2019;9:9286.
27. Hewett TE, Myer GD, Ford KR, et al. Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. *Am J Sports Med.* 2005;33(4):492–501.
28. Plisky P, Rauh MJ, Kaminski TW, Underwood FB. Star excursion balance test as a predictor of lower extremity injury in high school basketball players. *J Orthop Sports Phys Ther.* 2006;36(12):911–9.
29. Tummala SV, Hartigan DE, Makovicka JL, Patal KA, Chhabra A. 10-year epidemiology of ankle injuries in men's and women's collegiate basketball. *Ortho J Sport Med.* 2018;6(11):1–9.
30. Halabchi F, Angoorani H, Mirshahi M, Shahi MHP, Mansournia MA. The prevalence of selected intrinsic risk factors for ankle sprain among elite football and basketball players. *Asian J Sports Med.* 2016;7(3):e35287.
31. Zelisko J. A comparison of men's and women's professional basketball injuries. *AJSM.* 1982;10(5):297–9.
32. Trojan TH, Ragle RB. Injuries in women's basketball. *Conn Med.* 2008;72(3):147–50.
33. Khan M, Madden K, Burrus MT, Sikka R, Bedi A. Epidemiology and impact on performance of lower extremity stress injuries in professional basketball players. *Sports Health.* 2017;10(2):169–74.
34. McCrory P, Meeuwisse W, Dvořák J, et al. Consensus statement on concussion in sport—the 5th international conference on concussion in sport. Held in Berlin, October 2016. *BJSM.* 2017;51(11):838–47.
35. Brooks MA, Snedden TR, Mixis B, Hetzel S, McGuine TA. Establishing baseline normative values for the child sport concussion assessment tool. *JAMA Pediatr.* 2017;171(7):670–7.
36. Tavazzi B, Vagnozzi R, Signoretti S, et al. Temporal window of metabolic brain vulnerability to concussions: oxidative and nitrosative stresses—part II. *Neurosurgery.* 2007;61(2):390–5.
37. Halstead ME, Walter KD, Moffatt K. Council on Sports Medicine and Fitness. Sport-related concussion in children and adolescents. *Pediatrics.* 2018;142(6):e20183074.
38. National Collegiate Athletic Association (NCAA). Concussion Diagnosis and Management Best Practices. NCAA Website. <http://www.ncaa.org/sport-science-institute/concussion-diagnosis-and-management-best-practices>.
39. Erickson CC, Salerno J, Berger S, et al. Prevention of sudden death in the young: what the primary care physician should know. *Pediatr Circ* 2016; 133(10):1006–1026.
40. Taylor WC, Lombardo JA. Preparticipation screening of college athletes: value of the complete blood cell count. *Phys Sportsmed.* 1990;18(6):106–18.
41. Lape EC, Katz JN, Losina E, Kerman HM, Gedman MA, Blauwet CA. Participant reported benefits of involvement in an adaptive sports program: a qualitative study. *PM R.* 2018;10(5):507–15.
42. Special Olympics. Our work. Special Olympics Website. http://www.specialolympics.org/Sections/What_We_Do/What_We_Do.aspx.
43. American Academy of Pediatrics Committee on Sports Medicine and Fitness. Atlantoaxial instability in Down syndrome: subject review. *Pediatrics.* 1995;96(1):151–4.
44. Winell J, Burke SW. Sports participation of children with Down syndrome. *Orthop Clin North Am.* 2003;34(3):439–43.
45. American College of Sports Medicine. Intellectual disability and Down syndrome: special considerations for individuals with Down syndrome. In: ACSM, editor. ACSM's guidelines for exercise testing and prescription. 10th ed. Philadelphia, PA: Wolters Kluwer; 2017. p. 333.
46. Physiopedia Contributors. Lower crossed syndrome. Physiopedia; 2019. www.physio-pedia.com/index.php?title=Lower_crossed_syndrome&oldid=208489.

Medical Coverage of Basketball Events: From Local Competitions to European, World Championships and Olympic Games

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9.1 Introduction

It is really important to know the space where the event will take place to detail, the court or interior space as well as the exterior spaces of the arena. At a local competition we can work in facilities where there has not been any modified spaces and a protocol of established functioning has not changed for a long time.

Nevertheless, at bigger competition we can encounter brand new facilities or newly modified facilities for the event (Table 9.1). It is important to study the blueprints of all levels at the facility. At big competitions, meetings with equipment and services departments have to inform us with details of the infrastructure and distribution of the arena. This is usually complemented with a guided visit throughout all the facility along the entire staff team of the organization [1].

We need to know where the spaces destined to medical assisted are located. Some arenas have a big and equipped medical room; others have a nearly empty small space destined to medical assistance (Fig. 9.1). We have to prepare a room

Table 9.1 Characteristics of competition location

Competition location
• Arena
– Characteristics of infrastructure
– Resources—medical space and material
– Physiotherapy
– Circulation through arena
– Different arena areas
– Press room
– Locker rooms
– VIP-reserved areas

for medical assistance with the necessary materials (Table 9.2).

All types of medical material and commonly used drugs must be available for noncritical care emergency situations (nasal hemorrhages, ocular traumas, and all types of pain). The medical room must be in a perfect state of hygiene, well lit, and ventilated. It has to be situated so as to be directly and rapidly reached by the emergency services outside the arena (ambulances) as well as from the playing court area itself.

The arena also has at least one medical room for the spectators which meets the same requirements as that established for the participants (teams and officials) and has to be located in a different area of the arena. These rooms have to be directly and rapidly accessible from the seating area and to the ambulances coming from outside the arena.

A space where the teams' physiotherapists are able to work will be reserved. If it is possible, this

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Fig. 9.1 Team medical room after practice



Table 9.2 Mandatory equipment list at the arena

Equipment list
• Waiting room with seats
• One toilet with a washbasin and a WC
• Ice machine
• Examination couch 2.40 m long and at least 0.60 m wide with an adjustable revolving stool and a lamp with a mobile arm
• Chair
• Table
• Hangers
• Cabinet for storing medical material
• Sterilized surgery material essential for minor wounds:
• Gauzes or other sterilized dressings
• Antiseptics
• Gauze roller bandages
• Sterilized cotton swabs
• Cellulose dressings
• Suture kit
• Sterilized incise drapes
• Suture thread
• Surgical gloves
• Immobilization splints for the upper and lower extremities
• Compression splints for the upper and lower extremities
• Plaster bandages
• Elastic support bandages
• Adhesive tape
• Band-aids (for minor wounds)
• Local anesthetics

Table 9.2 (continued)

Equipment list
• Treatments of burns (silver sulfadiazine creams)
• Stethoscope
• Sphygmomanometer
• Essentials for critical care:
• Endotracheal cannula
• Laryngoscope
• Mayo's tube
• Manual bag-mask resuscitator
• Plasma expanders
• Intravenous infusion kit
• Anti-allergic medication: corticosteroids
• Cardiorespiratory arrest medication: adrenaline, lidocaine 5%, and atropine, bicarbonate 1 Molar
• Anti-hypertensive medication: Adalat tablets
• Bronchodilator medication: bronchodilator sprays (ventolin puff)
• Oxygen bottle with facemask
• Automated external defibrillator

space has to be located inside or next to the team's locker room. As a minimum, a stretcher has to be installed there and towels and ice bags have to be provided at the end of practices and games (Fig. 9.2).

The best circulation way for the medical assistance within the facility has to be studied. At the court level, the entrance and exit of medical assistance have to be practiced with the medical

assistance teams as well as the path to the medical room and to the ambulance. It has to be taken into an account that in big events the court is a practically closed space, there are led panels, court seats, cameras and photographers, cables and metallic structures for TV cameras. In some occasions a brand-new court is attached onto the older court in the arena, so there is a step that can hinder the stretcher's transportation. The court exit avoiding cameras and TV should be as fast as

possible, and the stretcher presence in front of the media or the audience should be avoided.

The team's doctor can ask for the injured player to go to the locker room in order to be attended (Fig. 9.3). It was previously proved what the best path to take toward the locker room is, as well as from the locker room to the ambulance. The court level assistance toward court seats' audience, team's staff, organization staff, photographers, journalists, and entertainment staff have to follow the same evacuation procedure as the players [2].

The space reserved for the media to be located in while the competition takes place has a particular design, they are provided with desks where they can install their computers, microphones, and cameras. It is not a simple procedure to evacuate a person located there, and it is convenient to plan various alternative plans with the emergency team.

The medical room has to be provided closer to their seating zone, the access from every single public seating area should be proved, the elevators could be used if provided by the facility.

The authorities' zone and spaces reserved for VIPs also need a special plan for evacuation; they tend to be closed areas with a single access and lots of security, which does not facilitate the emergency services' quick entrance and quick exit. In this case it will also be needed to plan various alternative evacuation plans.

At big events the arena's exterior spaces are also used, not only the different security stations with the assigned staff who regulates the tickets,



Fig. 9.2 Field medical setup and equipments

Fig. 9.3 Evaluation of injured player at the field by medical staff



we have to encounter working areas: ticketing, accreditations, protocol, parking, VIP parking. We can encounter sponsors' stands and marketing organized training actions. Without forgetting that since they cross the first security access with their ticket, the audience who suffers from a medical incidence will be attended by our medical team. We have to establish an assistance protocol at the exterior spaces, the evacuation path toward a medical room or straight to the ambulance.

The event security team should know and follow the norm, which limits the alcohol consumption and tobacco use at sports' events; alcohol consumption is one of the most frequent causes for medical assistance.

9.2 Ambulance System

The number of necessary ambulances may vary depending on the event activity. When hiring the ambulances, a contract has to establish a calendar and a schedule with the number and location of every unit. At big events, it has to be taken into account the process of putting everything together and the process of taking it apart when it is over. All the different labors that take place in and out the arena will need the presence of a medical ambulance ALS (Advanced Life Support), which includes a doctor and its nurse presence.

For practice sessions, there would be an ambulance available as well as the court assistance team. If the audience assistance is allowed, it will be necessary to activate the public seating area assistance service.

During games, a medical ambulance have to be available for exclusive players' assistance as well as a certain number of ambulances proportional to the number of spectators expected to attend and the risk level (Fig. 9.4). The previous meetings with security, police, and ambulance service departments will allow estimating the number of audience expected for the game and the fans' type of behavior.

At any time that one vehicle must leave to transport somebody; another replacement vehicle must be on stand-by to arrive immediately. The absence of the emergency ambulance service

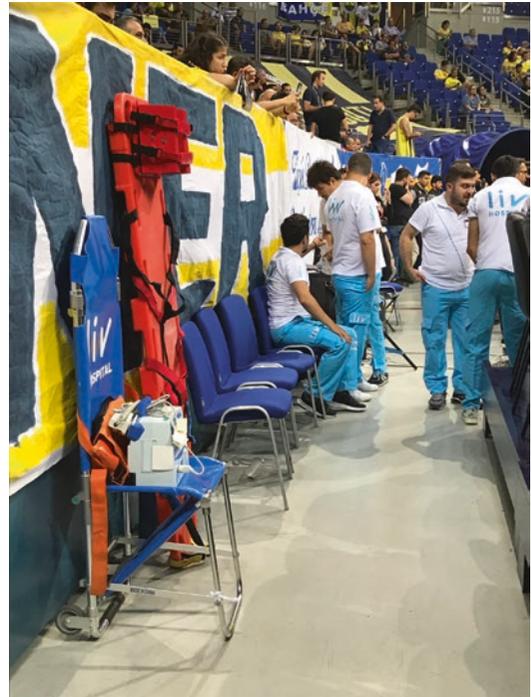


Fig. 9.4 Ambulance team at field near the spectators

with intensive care unit in the arena may be a reason for the suspension of the game.

In every single practice session or game, the ambulances' arrival and departure time have to be controlled as well as the medical assistance team's presence at the facility. It should be verified that they parked at the previously reserved and established parking spots and that the ambulances' emergency exit pathways are clear.

9.3 Designated Hospitals

- Local medical resources—country's sanitary level
 - Climate/season
 - Reference center

The hospital network dedicated to incidents during the event will be defined based on the available health resources at the assigned location. Different countries, big and small cities, rural areas, depending on the location where the sporting event takes place; the health or medical assis-

tance resources may vary on quantity as well as on the assistance level. We have to analyze the different hospital centers and their assistance capacities. If they have a 24-h emergency service, if they can attend serious TBI (traumatic brain injuries) cases—poly-trauma—CVD (cardiovascular diseases), if they have an intense care unit and its capacity [3]. We have to mark the distance toward the arena and the travel time by ambulance.

We have to establish contact with every hospital; inform of the event existence and duration, request a contact person in order to check on patients who are moved to that hospital. If the hospital that meets the best characteristics to treat serious cases is located at a far distance, it may be possible that the need to initiate a helicopter transportation, and we have to reserve an assigned space for the heliport along with establishing a transportation protocol to this point.

In case this happens, at a big city with various hospitals with a great assistance level, ambulance have to be responsible to contact with an operations center, which controls the emergencies and indicate what center is best suited and available for that moment.

The emergency assistance at hospitals could be more or less quick and efficient depending on many aspects [2]:

- The overall country's health status, hospitals more or less modifies, with longer or shorter waiting time, and resources for quick diagnosis.
- Climate and season, emergency services can collapse sometimes for seasonal illnesses or population growth at that zone and time due to holidays, even as a consequence of a big audience attending the sporting event.
- Due to endemic illnesses or public health issues (drinkable water, insects, food hygiene, etc.), the event attending public can become ill and will need medical assistance. The teams, media, organization, and public attending the sporting event should be minimally advised about the particular location's conditions and preventive methods to avoid possible illnesses or health issues. These methods include the vaccines' recommendations by the WHO's (World Health Organisation) information.

For teams, organization's staff, and VIPs, a reference hospital is selected. This hospital can be private or public, and its assistance level will be checked as minimum requirements are needed: a 24-h emergency service, a radiology service with XR (X-ray)–MRI (Magnetic Resonance Imaging)–CT (Computerised Tomography) scan–Ultrasound, a well-equipped lab, and available teams for minor surgeries. If a severe case emergency service or an intensive care unit is not available, patients suffering from these cases will follow the derivation norm established with other hospitals.

In this reference hospital, a priority assistance agreement should be established, reducing the waiting time, diagnosis time, and immediate treatment toward necessary medical tests as well as a very private and confidential assistance, protecting patients from the media.

We should have direct contact with key personal at the hospital, whom will be given a heads up before moving patients and whom we have to follow the complete patient's situation status at all times. They have to be informed that in case of a player, the player will be moved with its own team's doctor, who will have to be consulted and will have to participate on the decision-making process. It will be agreed with the hospital that if a player is moved without its passport or ID, it will be allowed to use its competition accreditation instead as an identification document for the hospital entry and afterwards all details will provide to the medical center.

As an exceptional case, big competitions as the Olympic games with a larger budget can cover all medical assistance expenses. At the majority of sporting events the teams are responsible of their own medical expenses, it is then highly recommended to have a medical insurance that is able to cover the medical assistance expenses at the country where the event is taking place and will allow splitting up costs when a severe accident occurs. It has to be taken into an account that the health-care costs are not the same in all countries, and to make sure insurance can cover a hospital entry. The most common thing to do is to pay all the expenses either with cash or credit card and then send the invoice to

the medical insurance to take care of it. In case of entering at a hospital, it is possible that the medical insurance contacts the hospital and takes care of all expenses immediately.

The organization should state clearly its medical assistance reach; the teams should know that these services come with an economic cost. A nonpayment from the event's invoice can become an issue on the organization and with a difficult solution.

9.4 Volunteers

The big sporting events count on volunteers' participation, which is often an altruistic work. They are distributed throughout the different organization's departments based on their preferences and experience. In the medical area, volunteers are needed for drug tests as player's escorts, the chaperons. In case of doctors or physiotherapeutic professionals, they can complement the medical assistance services at the arena for a better control of possible severe incidents at the seating areas, corridors, exterior zones, and VIP rooms. Their duties are fundamentally of control and warning giving, letting the assigned professionals to act on medical emergencies. Volunteers normally get a working uniform for sporting events as well as drinks and food, and an insurance that covers their expenses in case of an accident.

9.5 Accreditations

At sporting events, accreditations are used to identify all the participants and workers. The bigger the event, the greater is the security level, and importance of adequate accreditation use. At local competitions, we can simplify the circulation between the court/locker rooms zone, and the audience zone, an accreditation will allow the entrance to the court and will not discriminate based on any person's function.

On a big event, we have seen before how the arena is divided by multiple zones: the court, press, seating area, VIP room, etc. Every person gets an accreditation with specific zone access

based on where that person is assigned to work. Medical services need to freely move throughout different areas, which require a specific accreditation that allows medical service's staff to do so. We will need a big number of people working at every competition's area and at all working shifts and an ambulance service that can vary when circulating a vehicle with a medical incidence and be substituted for another, with different staff.

As a conclusion, we will need accreditations with a high security level, but it is difficult to personally give an accreditation to every single worker. For that reason, similarly to what happens with security and police, we use a corporative accreditation where all the medical service staff that has not gotten an accreditation. Depending on the working area you have been assigned to work in at every shift, you could receive an upgrade as a bracelet or sticker added to your current accreditation, which will allow you to enter reserved areas. The same process is applied to medical service volunteers; they usually get a standard accreditation and get an upgrade in order to move around different areas.

Volunteers who are assigned to work at the drug testing process will need access at the drug testing area, court, locker rooms, and pressroom escorting the assigned player all the time. We have to provide accreditations for drug testing assigned agents and their access at the necessary areas in order to accomplish their functions.

9.6 Referees/Entertainment

Sometimes we tend to forget about the importance of the referees and the fact that they can also get injured. We have to make sure referees are able to get medical assistance as quick as possible during games and that they can be assisted at their locker room when they need to. The entertainment has significantly changed over the last years, cheerleading groups have grown with the male presence and choreographies include more jumps and acrobatics; shows that combine ball games and bouncy trampolines are frequent as well as slam dunks' con-

tests using different materials (chairs, tables, people). Court medical service assistance has to be careful and watch out during game breaks since at any moment an accident on court can take place. It is very important that after entertainment events the court is cleaned and cleared in order to prevent and avoid slippery elements or other injury-provoking objects that could cause players to get hurt. Entertainment's timing has to be calculated in order to allow a period of time for clean-up.

9.7 Doping Control Room

The arena should have a doping control room, in a perfect state of hygiene, well-lit and ventilated, and with a waiting area [1, 3]. The doping control room and the material provided therein will be in accordance with the Internal Regulations governing Anti-Doping in the basketball competitions (Fig. 9.5). It is recommended that the game clock



Fig. 9.5 Regular doping room and doping control after a Euroleague game

be displayed inside the doping control room. The doping control room may not be the same room as the medical room. The access door to the room will lock and the key will be under doping agents' possession. Anyone who is missing an accreditation for the doping control room or authorized by the doping agents to access the room will not be allowed in it. We have to make sure there is an adequate availability of drinks at the doping control room.

9.8 Crisis Protocol

9.8.1 Security and Emergency Services Coordination

A crisis protocol has to be established; this plan should be designed to give structure to our communications in the aftermath of a serious incident involving our event. For the purposes of this plan, a serious incident is defined as one in which external emergency services are required, e.g., medical, fire crews, and police.

Examples of potential crises include the following:

- Team member seriously injured or killed
- Spectator(s) injured or killed
- Fights between team supporters resulting with multiple injured

The crisis management team needs the following:

- Crisis team director and spokesperson
- Crisis coordinator—Security coordinator
- Press officer
- Services coordinator
- Medical officer

Our role will be to supervise the response of the medical services, get an extended ambulance support if needed, establish all medical facts surrounding the incident. Travel to the hospital to feedback information on the condition of the injured person(s) to the crisis management team.

9.9 Out-of-Competition Assistance

At sporting events that last multiple days, it could be necessary to organize a medical assistance system out of the competition schedule. The reference hospital information will be provided to all the teams participating in the competition along the medical assigned coordinator's phone number or any organization's member who is available 24 hour in case of help seeking. This information could also be provided within the volunteers who act as team assistants, volunteers who work with the VIP's at protocol, and organization members.

At the Olympic games where all athletes are hosted at the Olympic village, a medical center is available there as well; ready to attend any basic

emergency. When all competition's participant teams share a hotel, a nursing station can be established for emergencies and a physiotherapeutic room for treatments. However, high-standing teams already own those resources for their private use.

References

1. American College of Sports Medicine Expert Panel. Sideline preparedness for the team physician: consensus statement. *Med Sci Sports Exerc.* 2001;33:846–9.
2. Team Physician Manual. International Federation of Sports Medicine (FIMS). Taylor and Francis. Kindle Edition. 2012.
3. Everline C. Application of an online team physician survey to the consensus statement on sideline preparedness: the medical bag's highly desired items. *Br J Sports Med.* 2011;45(7):559–62.



Communication Challenges in Medical Management of a Basketball Team

10

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10.1 Introduction

Basketball is a dynamic contact sport with relatively high rates of injuries. It is indeed the role of the medical team to manage these injuries; however, this is only one layer consisting the medical team's purpose. A simplistic approach would focus on the treatment and management of an injured player until return to health and return to

play (RTP); however, the reality is far more complex—with many players often training and competing with injuries of various degrees, due to the demands of their playing calendar, time within the season and even contract-related constraints. In cases where players are unable to continue training or playing, the RTP decision-making process could be quite challenging—with many factors requiring consideration, of which the future health of the player is only one aspect [1]. It is therefore one of the biggest challenges for the medical team of a professional basketball team, to find the balance between managing the health of the players and optimizing individual and team performance. One of the key elements in optimal management of the professional basketball player's health is communication in a shared decision-making model. This should be supported by an appropriate organizational structure of the medical team in the basketball club or organization.

The sport of basketball has become increasingly popular in recent years, with an ever-growing interest and exposure due to the introduction of social media, fan engagement platforms, and even e-sports. This growing popularity has driven towards a significant increase in professionalism in basketball worldwide, not only from the players' perspective but also in terms of coaching and medical staff. Increased professionalism has raised the standards in every aspect of the sport, and has driven towards better organization of medical services and an expectation for increased specializa-

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tion of basketball medical staff and structure. In the professional setup, the medical staff often consists of experts from multiple health disciplines (i.e. primary care physicians, orthopaedic surgeons, sports and exercise medicine specialists, physiotherapists, athletic trainers, sport therapists, psychologists, nutritionists, strength and conditioning coaches, physiologists and sports scientists) working alongside to provide optimal healthcare for the players as well as assist in optimizing performance. This multitude of clinicians and professionals allows for holistic player care but necessitates a strong organizational approach to avoid speaking in multiple voices. Communication is a key factor to consistently create and maintain a positive environment for the players' benefit.

Good communication is not only about the message transmitted, but more importantly, the message received, and making sure that the gap between the two is as minimal as possible.

There are various categories and forms of communication and more than one may take place at any time, such as verbal communication (face-to-face, telephone), non-verbal communication (body language, gestures, how we dress or act), written communication (e-mails, reports and medical notes) and illustrations (graphs, charts, photos, etc.).

Mehrabian [2] has highlighted the relative importance of verbal and nonverbal messages, reporting that 7% of the understanding of a message arise from feelings and attitudes transferred in the spoken word (verbal communication); 38% of the understanding of a message arise from feelings and attitudes transferred in the way that the words are spoken (paraverbal communication); and 55% of the understanding of a message arise from feelings and attitudes transferred in facial expression (nonverbal communication). The team physician and medical staff members use these skills on a daily basis to communicate, and it is important to create communication opportunities in a team setup, to audit the communication process and to ensure the messages received are indeed the ones transmitted.

Communication could therefore be challenging for the medical team in a competitive sport environment, such as in a professional basketball

team. There is a continuous interaction with the different stakeholders in and around the team (players, coaching staff, strength and conditioning coaches, team physician/s, physiotherapist/s, athletic trainer/s, management, agents, family members) over a competitive period filled with unexpected and often stressful events. The purpose of this chapter is to highlight and describe the communicative challenges the medical team may face with potential solution to optimize communication in a team setting. Successful communication is therefore a combination of several important factors. It is important for the sports clinician to be familiar with the different roles and personnel in the team, their background and experience and their cultural context.

10.2 Communication Within the Medical Team in the Multidisciplinary Setting

The head team physician/clinician or 'head of medical' are usually positioned at the top of the medical organizational pyramid. These professionals are well positioned to take a leadership role in multidisciplinary medical teams providing comprehensive health management of the elite athlete [3]. Their training, background and experience allow them to not only understand the medical needs of the players but also the complexity of their professional environment and the demands of their sport. It is therefore one of the key characteristics of a 'head of medical' to possess the ability to communicate with all stakeholders in and around the team and 'navigate' the team environment in such a way that players, medical professionals and coaching staff all trust and value their opinion and recommendations. Without this ability, it would be very difficult and often impossible for the 'head of medical' to fulfil the role of case manager with success, regardless of his/her medical knowledge and level of judgement. The 'head of medical' is a unique position in the team that translates into a responsibility towards not only the players but also towards the management and coaching staff—often with different interests. The 'head of medical' role is therefore often to find the

balance between the stakeholders' needs and expectations, propose a plan of action which would optimally address the concerns of the different parties, as well as remain medicolegally and morally defensible.

While in the traditional sports medicine model, the team physician/'head of medical' is the primary point of contact for the athlete [4], a more current and improved multidisciplinary model recognizes that the athlete has a variety of 'non-physician' sports medicine practitioners who can provide a primary medical point of contact (i.e. physiotherapists, nutritionists, podiatrists and chiropractors). Whilst this multidisciplinary approach can improve and optimize players' healthcare, it can also create scenarios where players find themselves trying to understand and integrate advice from several different and unsynchronized sources. Different information from multiple sources could lead the athlete to try and find a solution on their own, turn to coaching staff (who are not equipped to make medical decisions), or outside the club entirely. Therefore, a potentially better model is one where the head team physician/clinician acts as a 'case manager'. In this model, the head team physician/clinician is responsible for integrating all the available information and in collaboration with the multidisciplinary team formulating the advice into a recommendation, considering the players' health and performance. While this physician/clinician may not always be the first point of contact for a player, they are responsible for making sure all medical/performance team members are on the same page and passing on the same message to the athlete. This approach aims to put the player in a position to make an informed decision, in discussion with all relevant stakeholders, including the coaching staff [3].

The ability to work together as a team is as important as the qualifications and experience of the medical/performance team. In certain situations, it may be necessary to clearly define roles and responsibilities for each team member. It is not uncommon to have more than one individual in the medical team with similar or overlapping skill sets. Therefore, clearly defined roles can help facilitate communication and eliminate redundancy in work as well as miscommunica-

tions or misunderstanding about a player's plan or treatment. Further, as medical/performance teams interact with players closely, it is essential that they act with the utmost professionalism, thus developing a code of conduct, endorsed by all team members, may be beneficial.

10.3 Communication with the Coaching Staff

In large basketball organizations, with multiple disciplines involved in both medical care, performance and coaching, an integrated performance health management and coaching model [5] is one approach which could be applied to optimize performance in a safe manner. An integrated performance health management and coaching model focuses on how two key departments (Medical/Health and Coaching) can work together to improve performance (Fig. 10.1)—such a model can also be applied in elite basketball [5]. In this model, the two departments ideally function as two independent departments, while working towards one common goal under a single management umbrella: the success and optimal performance of the team. For this to be achieved, the roles and responsibilities of the staff in each department need to be clearly clarified.

The head team physician/'head of medical'/chief medical officer or medical director in the case of larger organizations (or national federations) leads the health department. Their primary responsibility is to oversee the players' health and to work with the coaching department to ensure any health-related decisions are based on the principles of shared decision-making, including informed consent when appropriate. For this to occur, good and continuous communication is necessary in order to achieve a balance between the players' and team's health and performance.

The head coach, who has overall responsibility for all other coaching staff, including the strength and conditioning or fitness coaches, leads the coaching department. In the integrated performance health management and coaching model applied to basketball, communication and organized education should be provided by the

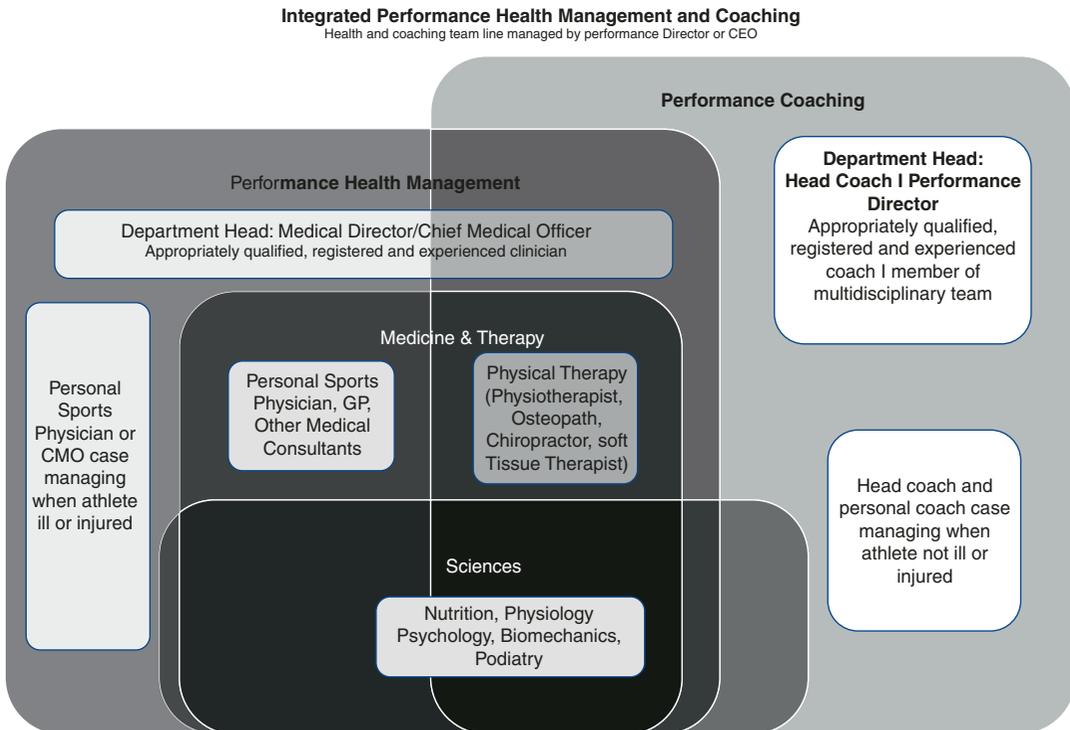


Fig. 10.1 Reproduced with permission [6]. The integrated performance health management and coaching model. All the specialties operate in the performance health and coaching ‘box’. Specialist sports medicine physicians (led by the chief medical officer (CMO) or medical director (MD)) manage health (injury, illness and

prevention); the head coach manages coaching. Both departments are managed by the performance or technical director or chief executive officer (CEO) depending on the structure and size/culture of the organization/club. The health and coaching departments operate in synergy and ‘independently’ with appropriate autonomy at times

coaching staff regarding training principles to achieve particular performance goals, as well as players’ values and preferences. This could be delivered through periodic meetings between the heads of each department, who can then communicate the message to their respective staff, or through joint meetings which include the entire staff from each department. It is important to conduct such inter-departmental meetings prior to the start of the pre-season period in order to best communicate and adjust expectations, strengthen existing inter-departmental working protocols or establish new ones, and present new strategies for each department. Coaches should have a working knowledge of important injuries and illnesses in basketball and an open-minded attitude to receiving information from medical staff about treatment and prevention of injuries and illnesses in basketball. For example, such

inter-departmental communication and collaboration is imperative to strategize and implement injury prevention protocols, from players’ baseline data collection and testing and through implementation into the busy training and playing schedule. In large organizations, the head of performance or performance director has overall responsibility for managing the two departments and ensuring they communicate and work in collaboration to implement the performance strategy of the organization/club.

Confidentiality and adhering to data protection regulations is important when communicating with staff in or outside the medical team. In cases where there is doubt regarding the information about to be shared or requested, it is best to take precaution and adhere to the confidentiality and data protection processes devised in the organization and consult with the relevant person

when necessary. Informed consent may be required from players in certain situations.

Establishing a good and ongoing communication between the medical team and the coaching staff as well as the players is important and essential not only for a positive work environment but also to implement new ideas and concepts more efficiently (i.e. injury prevention concepts and nutrition). A recent study focusing on injury prevention education for rugby coaches has shown a substantial influence on players' training approach and behaviour, favouring injury prevention-related activities/behaviours [7].

As the workload on players continues to increase (e.g. European players, Players in Europe, along with European competitions and international matches, sometimes have to compete in up to 80 games per season, and in the NBA, up to 100 games per season, with play-offs), the topics of nutrition, recovery, load management and injury prevention are increasingly coming into focus.

Therefore, it is extremely important to provide players with information on doping, nutrition and injury prevention as soon as possible at the beginning of each season. Equally important, however, is that these issues are also known by their importance by the coaching staff and that they lead by example.

In a recent study in football, Ekstrand et al. showed that the quality of communication can even positively influence the frequency of injuries [8].

If a severe injury occurs, the team physician has to fulfil the role of a case manager. When the diagnosis is clear, it is a good thing to have a team meeting about the injured player. With the team physician, physiotherapist, athletic trainer, coach and maybe even the nutrition expert or the psychologist coming together, everybody has the same knowledge base afterwards. In this meeting, the diagnosis and its treatment should be explained, the different steps of rehabilitation and the milestones that have to be reached to enter the next step have to be defined and fixed. It is also useful to reflect why the injury occurred, and what kind of preventive activities can prevent another similar case.

10.4 Communication with Administrative/Management Staff

Lack of communication can lead to uncertainty and conflicting interpretations at all the organizational levels within a team. Physicians/clinicians are employed by clubs/organizations and are often hired by administrative/management staff—a fact which might influence clinical decision-making, for example in situations where the club and player have conflicting opinions with regard to a specific injury management or where a clinical decision could impact an athlete's contract. As a general rule, it is advised to establish clear role definitions with internal and external clinical governance (e.g. appraisal and revalidation process by the appropriate external bodies such as the medical council in the countries in which they are practicing, medical associations and league governing bodies) [6].

Physicians/clinicians are often clinically line managed by non-medical team members or non-clinicians. This could pose potential challenges in players' medical confidentiality, ultimate clinical responsibility and access to medical records. It is therefore advised that contractual arrangements detailing their ultimate clinical responsibility are communicated and formally established. Current culture and contracts within sport clubs/organizations should consider and relate to the issue of medical confidentiality.

Further, as most administrative/management staffs do not have a medical background, ensuring that medical-related terms, injuries and treatments are clearly communicated and understood by all parties. Avoiding the use of medical jargon, and instead using accurate terminology, helps in these discussions. Medical teams may find that education regarding anatomy, injuries, surgeries, treatments, risks and long-term implications are beneficial. Education may be provided as information provided on a regular basis for administrative/management staff's knowledge, or provided specific to a particular injury/situation.

10.5 Communication with Players

A basketball team typically consists of 10–15 players, with an often-dynamic roster involving players from secondary or development teams. The composition of a team roster can be very heterogeneous in terms of origin, age, background, language, culture and experience in professional basketball. There could often be variability in regulations, playing rules (i.e. NCAA, FIBA rules and NBA) and even cultural differences which may influence the environment the medical staff and players are exposed to. Given this dynamic environment as well as diversity within teams, diversity awareness and awareness of the variability in communication styles of different athletes and stakeholders are important. Language (verbal and non-verbal), approach to situations and emotional responses may all vary and should be respected by the medical/performance team.

The simplistic interpretation of the current multidisciplinary sports medicine model is that the role of the team physician is to protect the health and welfare of the player [5]. In the reality of elite sports, the player's health is a spectrum ranging from complete wellness to multiple existing injuries and illnesses—especially in the older/aging player. Players are accustomed tolerating this spectrum of health in order to perform consistently. In a holistic approach to viewing players, it is often evident that making the best performance decisions may sometimes compromise a player's 'health and welfare'. Therefore, in an improved model, the head physician's primary role is not only the 'health and welfare' of the player but rather to assimilate information and provide a safe framework for players decisions that balance health and performance goals and to optimize players' careers, with considerations applying to their post-career period. Communication skills and trust are imperative to deliver such information in a consistent manner.

In smaller or non-professional organizations/teams, the team physician/clinician may also find their role expanding to that of nutritionist, parental support (in youth teams), psychologist, massage therapist, or 'kit man'. It can be very challenging to remain professional when players

expect friendship, but it is crucial that the line between 'friend' and 'physician/clinician' remains well defined and transparent. It is important that this distinction is well transferred as part of the communication between the physician/clinician and player.

If a player gets injured, communication is essential for the recovery process and the outcome [9]. The recovery process begins from initial contact between a member of the medical team and the injured player. An injury is a stressful event for players and every word, speaking tone, gesture or facial expression may have a huge effect on injury management, the player's perception, approach and engagement. Players as well as other stakeholders in the team (i.e. coaches, administration staff, club owners/presidents) like expedited information and dislike uncertainty. Keys to a successful rehabilitation include the following: (1) adhering to confidentiality regulations, (2) avoiding uncertainty wherever possible, (3) conveying clear and regular messages so that players and relevant stakeholders are not 'in the dark'. These key elements can be challenging at times and require good continuous communication with all parties.

Predicting a return to play time frame can be tricky and if approached poorly can lead to trust and confidence issues. It is also important to clearly communicate the plans and goals for each phase of the rehabilitation process (which could be dynamic) and continuously manage expectations from all parties through the rehabilitation process to avoid frustration and uncertainty.

Many players have a close relationship with their family members, friends, personal medical/performance staff, or other hired employees. Some players may even rely on their close circle for communication with the team. Similar relationships to an extent may exist between some players and their agents. Medical/performance teams must balance maintaining the player's best interest and respecting these relationships. It is therefore important to understand the context of such relationships and put the player's best interest in first place. Open communication with players' family members, personal medical/performance staff and agents is crucial to

maintain a trusting and ‘two-way relationship’. Information sharing about the athlete therefore must be reciprocal so that all parties are aware of situations that might arise, no one is surprised, and the athlete remains the focal point of care. An organized, clearly defined process involving a player’s close circle, respecting to all involved parties and their roles while maintaining the integrity of the process and shared data regulations, can be helpful achieving optimal communication. This is particularly important when an athlete changes teams, establishing open communication from the outset is crucial for the athlete to trust the new organization/team, but especially the medical/performance team.

When it comes to injury prevention, compliance is a key element and can only be achieved through collaborations across the medical staff, trainers, coaches and, of course, the players. Educating the coaches, strength and conditioning coaches and players about the benefits of injury prevention is an important factor in this process, illustrating how such strategies were able to reduce the frequency of injuries as well as injury severity [10–14]. Without the engagement and cooperation of the coaching department and staff, only suboptimal prevention implementation will be possible, if any. Ideally, an injury prevention strategy and protocol should be developed and agreed upon between medical/performance and coaching staff, which will then be carried out regularly. For example, the 11+ (formerly known as the FIFA 11+) is a prevention programme developed in football but has shown to have injury-reducing effects in basketball [15].

10.6 Doping-Related Issues

Doping regulations in basketball are overseen by the World Anti-Doping Agency (WADA) Code [16] worldwide, and in-competition or training controls will occur, depending on the playing level. This may vary in frequency in different countries.

As a part of the anti-doping control process within a team, baseline information (history, regular medications, medical conditions requiring or

which may require medication or supplement use) should first be collected, which of course is best done during the initial medical examination. For the team physician, it is essential to communicate with each player regarding their medications or supplements. An educational form of communication is recommended in order to raise players’ awareness, establish ground rules, subject them to the control system and process within the team and make the relevant procedures/content known to them, such as the following:

- Which substances/methods are on the doping list?
- What should be done/observed in the case treatment is prescribed?
- What is the procedure of a doping control/test and how is it managed?

It is particularly important to clarify and emphasize to the players that they should not take any medication, substance or dietary supplement without notifying and receiving approval from the team physician or the clinician in the team who is designated to this role.

The medical team should be aware of potential cultural and background differences and be prepared to avoid potential doping issues. For example, in cases where players are recently out of college or playing outside of their home country for the first time, they may be naive and ignorant to doping issues in their new country or league. Another potential issue stems from the fact that more and more states in the USA are legalizing the use of cannabis. Therefore, it is imperative to make the players aware that tetrahydrocannabinol (THC) is on the doping list, so positive testing could lead to a ban. Further, as cannabidiol (CBD) containing products are also becoming more common, education related to what CBD is, purity, possible effects and how it could affect a drug test is necessary.

It is advised that the medical team would be well-educated and remain updated on doping issues as these changes constantly. It is also advised to educate the coaching staff not to administer any substances or supplements, and educate players not to independently use or share such sub-

stances with their teammates without consulting the medical team first. This is a crucial issue and may result in substantial medicolegal and even career ending consequences for medical team and coaching staff members as well as players.

10.7 Second Opinions and Consultant Specialists

Second opinion scenarios can be challenging for both the medical staff and players. If managed inappropriately, such scenarios may lead to conflicting, stressful and even awkward situations at times. Seeking a second opinion can arise from a number of reasons, and does not automatically mean that there is mistrust between the player and the medical team. Good communication leading to an informed and transparent process will help determine the impact of such scenarios on player–medical team relationship. Second opinions are not always initiated by players and can also be initiated by the medical team, for example, in complex or rare injuries or health conditions where the opinion of a specific specialist or more than one specialist is sought after, or in situations where a player has an old or ongoing injury which has been managed by another specialist before joining their current team. It is also important to acknowledge that sometimes other forces around the player (family, agents, friends) may drive for a second opinion as well. In any scenario, players are entitled to choose their physician/clinician. It is, however, important to be aware of and avoid scenarios that could lead to an unwanted outcome, not only in the medical/health aspect but also in terms of the player–medical staff relationship and trust. It is therefore important to avoid an atmosphere which will make the player seek for a second opinion without informing the medical team (i.e. due to fear from the medical team’s reaction or response). This could lead to gaps or even complete lack of information regarding the player’s injury/medical condition, “where did he/she go?”, “which tests were performed?”, “what treatment or course of action was proposed?”, “what was done?”, “was everything in line with the doping or league regulations?”, and many

other questions or unwanted outcomes which may result from a player’s unauthorized or uninformed second opinion.

Undesirable situations can be avoided by laying the ground rules for second opinion situations, discussing such scenarios at the start of the season as a part of a ‘code of conduct’, and by facilitating good communication to eliminate concerns from any player regarding a second opinion process. The process of seeking a second opinion should be managed and overseen by the ‘head of medical’, as an extension of case management, and it is preferable that the request for a second opinion arrives as an official request from the player’s team and medical team. In such a process, transparency and communication are key elements from all sides involved to ensure all the relevant information is being transferred and the medical team can even assist the player in finding the best second opinion (or more, if necessary) for his/her condition. This joint process also ensures that the specialist providing the second opinion is aware of the transparency of the process and that the case is still managed by the ‘head of medical’, hopefully eliminating situations where conflicting messages are presented to the player or messages which could be destructive to the player’s relationship with his/her medical team.

There are certain situations in which consultation is required in a specific specialty which is not covered within the fields of expertise of the medical team (i.e. Dermatology, Cardiology, Neurology, Hand surgery, Obstetrics and Gynaecology). The availability of such specialists can depend on the setup the medical team and medical services are provided for the team. For example, in college basketball, often a university hospital is involved in providing some, if not all, of the medical support for the team (i.e. imaging and blood tests) and the medical staff of the team may be affiliated with the university hospital. In this scenario there is usually easier access to specialists from various fields, if necessary. Similar coverage is provided for professional teams as well, with team physicians who are associated with hospitals, and the hospital provides a variety of services for the team, if and when necessary. While setting up such consultations is usually

done through the team physician, the same rules and code of conduct as applied for second opinions should be applied. Good clear communication is required to avoid gaps in information and recommendations delivery. A key element in the communication between the ‘head of medical’/team physician and specialist in such situations, apart from stressing the importance of accurate and documented information delivery, is to clarify that no treatment should be commenced before informing the ‘head of medical’/team physician as well as stressing the importance of adhering to doping regulations.

10.8 Media

The involvement of the media, on all its aspects, is ever-growing in current day professional sports. There is a constant thirst for immediate information distribution, which can put tremendous pressure on the medical team, coaches, administrative staff as well as players. Such pressure for information can influence decision-making if not handled appropriately. Establishing clear and strict guidelines for conduct with the media beforehand is important to avoid such scenarios. It is important that all team staff are aware, on-board and adhere to these guidelines. Often statements in injury-related scenarios are released by non-medical personnel, and therefore communication between the medical team and media team members is key in these situations, and it is important that the team ‘speaks’ in one voice.

Social media has also become an omnipresent factor in professional sports. Clear regulations on staff social media use must be established and agreed upon, but are important for player/team confidentiality. Education for players on social media use and its impacts may also be beneficial.

10.9 Educating the Medical Team, Coaching Staff and Players

Maintaining a consistent high-performance level by the medical team and adhering to the highest standards of medical care require engaging all members of the medical team in updated educa-

tional activities—both within the team as well as on a personal level. Such educational activities may include periodic life support courses and training, team physician courses, sports-specific conferences and discipline-specific educational activities (nutrition, rehabilitation, sports psychology, etc.). In football and Olympic sports for example, specific programmes and diplomas have been established for these purposes, such as the ‘UEFA football doctor education programme’, the ‘FIFA diploma’ and the ‘IOC Diplomas’ in Sports Medicine and in Nutrition. It can also be useful to utilize the various medical team members’ specialties to educate the other members on issues within their scope and to facilitate decision-making optimization as well as treatment strategies and protocols within the team. In German Basketball, a group of team physicians from the professional leagues has established a group called ‘Basketdocs’ (www.basketdocs.de), who meet twice a year for scientific meetings and workshops and professional exchange.

10.10 Summary

Communication is a key element in optimizing care for athletes in sports. Facilitating good communication channels is essential for the function of the medical team, and for the establishment of trust and a productive working environment—both within the medical team and with players, coaches and other elements in and around the team/organization. Establishing such channels can help in aspects such as injury management; the implementation of preventative and recovery strategies and perhaps more importantly, the engagement and ‘buy in’ of both players and coaches; adhering to doping regulations; and many more essential elements for optimal medical management. Communication is important not only to optimize case management for injuries but also to analyse and learn from injury management processes in order to learn from errors and improve medical care. Overall, engagement of all the elements and departments in the team is crucial for success, and good communication skills and channels are important to achieve the team’s goals.

References

1. Shultz R, Bido J, Shrier I, Meeuwisse WH, Garza D, Matheson GO. Team clinician variability in return-to-play decisions. *Clin J Sport Med.* 2013;23(6):456–61.
2. Mehrabian A. Significance of posture and position in the communication of attitude and status relationships. *Psychol Bull.* 1969;71(5):359–72.
3. Dijkstra HP, Pollock N. The role of the specialist sports medicine physician in elite sport. Managing athlete health while optimising performance—a track and field perspective. *Aspetar Sports Med J.* 2014;3(1):24–31.
4. Dijkstra P, Della Villa S. Sport and exercise medicine: the team approach. In: Brukner & Khan's clinical sports medicine. Volume 1: Injuries / Peter Brukner, Ben Clarsen, Jill Cook, Ann Cools, Kay Crossley, Mark Hutchinson, Paul McCrory, Roald Bahr, Karim Khan. 5th ed. North Ryde, NSW: McGraw-Hill Education (Australia), 2017.
5. Herring SA, Kibler WB, Putukian M. The team physician and the return-to-play decision: a consensus statement-2012 update. *Med Sci Sports Exerc.* 2012;44(12):2446–8.
6. Dijkstra HP, Pollock N, Chakraverty R, Alonso JM. Managing the health of the elite athlete: a new integrated performance health management and coaching model. *Br J Sports Med.* 2014;48(7):523–31.
7. Brown JC, Gardner-Lubbe S, Lambert MI, Van Mechelen W, Verhagen E. The BokSmart intervention programme is associated with improvements in injury prevention behaviours of rugby union players: an ecological cross-sectional study. *Inj Prev.* 2015;21(3):173–8.
8. Ekstrand J, Lundqvist D, Davison M, D'Hooghe M, Pensaard AM. Communication quality between the medical team and the head coach/manager is associated with injury burden and player availability in elite football clubs. *Br J Sports Med.* 2019;53(5):304–8.
9. Della Villa F, Andriolo L, Ricci M, Filardo G, Gamberini J, Caminati D, Della Villa S, Zaffagnini S. Compliance in post-operative rehabilitation is a key factor for return to sport after revision anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc.* 2019. <https://doi.org/10.1007/s00167-019-05649-2>.
10. Benjaminse A, Otten B, Gokeler A, Diercks RL, Lemmink K. Motor learning strategies in basketball players and its implications for ACL injury prevention: a randomized controlled trial. *Knee Surg Sports Traumatol Arthrosc.* 2017;25(8):2365–76.
11. Andersson SH, Bahr R, Clarsen B, Myklebust G. Preventing overuse shoulder injuries among throwing athletes: a cluster-randomised controlled trial in 660 elite handball players. *Br J Sports Med.* 2017;51(14):1073–80.
12. Bonato M, Benis R, La Torre A. Neuromuscular training reduces lower limb injuries in elite female basketball players. A cluster randomized controlled trial. *Scand J Med Sci Sports.* 2018;28(4):1451–60.
13. Omi Y, Sugimoto D, Kuriyama S, Kurihara T, Miyamoto K, Yun S, et al. Effect of hip-focused injury prevention training for anterior cruciate ligament injury reduction in female basketball players: a 12-year prospective intervention study. *Am J Sports Med.* 2018;46(4):852–61.
14. Riva D, Bianchi R, Rocca F, Mamo C. Proprioceptive training and injury prevention in a professional men's basketball team: a six-year prospective study. *J Strength Cond Res.* 2016;30(2):461–75.
15. Longo UG, Loppini M, Berton A, Marinozzi A, Maffulli N, Denaro V. The FIFA 11+ program is effective in preventing injuries in elite male basketball players: a cluster randomized controlled trial. *Am J Sports Med.* 2012;40(5):996–1005.
16. WADA. 2015. <https://www.wada-ama.org/sites/default/files/resources/files/2015-wadc-final-de.pdf>.



The Role of the Team Physician in Basketball

11

Jeffrey R. Kovan and Chelsea Gonzalez

11.1 Who Are Team Physicians?

The team physician is often viewed as the head of a sport's health-care team, and must actively integrate his/her medical expertise with other health-care providers, such as on-site athletic trainers and specialists. According to the American College of Sports Medicine (ACSM) Team Physician Consensus Statement 2013 Update, "The team physician must have an unrestricted medical license and be a D.O. or M.D. who is responsible for treating and coordinating the medical care of athletic team members. The principal responsibility of the team physician is to provide for the well-being of individual athletes—enabling each to realize his or her full potential. The team physician should possess special proficiency in the care of musculoskeletal injuries and medical conditions encountered in sports. The team physician must ultimately assume responsibility within the team structure for making medical decisions that affect the athlete's safe participation." [1]. In addition to specialized musculoskeletal training, team physicians must have a fundamental knowl-

edge of on-field/court/pool emergency situations such as cardiopulmonary resuscitation, cervical neck injuries, and appropriate spine boarding and transport.

Team physicians are trained in a variety of core specialties including, but not limited to, family medicine, internal medicine, pediatrics, physical medicine and rehabilitation, emergency medicine, orthopedics, neurology, and cardiology. A physician with both primary care and sports medicine training likely underwent a subspecialty fellowship program after residency. During that fellowship year, the "primary care sports medicine physician" fine-tuned his/her musculoskeletal exam skills, expanded differential diagnoses relative to sports-related injuries, and learned the roles within the health-care team at community, high school, collegiate, and professional levels. There are also sports fellowships in orthopedics while neurology and cardiology have expanded post-residency training in sports as well.

11.2 Health-Care Team

The most effective strategy to manage the variety of ailments and injuries encountered in sports is to utilize all the resources available to provide the most complete and comprehensive level of care; this management paradigm requires a "gatekeeper" to coordinate the event, assess risk, and ultimately manage the athlete's care

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from start to finish. The relationship between the athletic training staff and the team physician is essential in organizing and creating the health-care team, and the role of mid-level providers, physical therapists, and other ancillary staff should not be undervalued or overlooked. But the certified athletic trainer (ATC) for a basketball team is the health-care member serving at the forefront of an athlete's care. The ATC is often the first medical personnel to evaluate an acute injury or illness and at times, assist the athlete in managing chronic conditions. At the direction of the team physician, the ATC coordinates referrals to the proper specialists, assists with obtaining diagnostic testing, and ensures timely follow-up. They are trained extensively in nonoperative and postoperative rehab protocols and techniques ensuring their athletes make a safe and timely recovery. In tandem, the ATC and team physician act as a bridge between the athletes and their coaching staff, family members, and school or organizations. It is of utmost importance that the team physician and ATC create a relationship founded on respect, trust, and communication.

11.3 Responsibilities and Duties

The team physician in basketball must understand the sport itself to prepare for and thus immediately respond to sport-specific injuries. This requires a knowledge of the typical mechanism of injury seen in basketball and how best to prevent and treat these injuries to allow the athlete to return as quickly and safely as possible. Team physicians, especially those that have been trained in primary care specialties, will often also manage chronic conditions, assess acute illness and injury, and promote preventative medicine to their athletes in the clinical setting.

A team physician's first interaction with an athlete often occurs at the pre-participation physical (PPE). Athletes are often seen by their team physician in the training room or clinic. This office should allow ease, accessibility, and familiarity to the athletes and their ATC. At most levels of organized sport, institutions will require their athletes to have a PPE to assess the level of

risk to participate. Included in the PPE is a thorough review of personal and family history and a detailed physical examination. If the team physician is unable to conduct the PPE themselves, they should personally review and familiarize themselves with their athletes' physical exam notes and records. The PPE preps the physician and ATC on potential illnesses or injuries that may occur and helps to determine the need for additional testing or specialty referral when necessary.

For basketball, the team physician should be vigilant in screening athletes at increased risk of sudden cardiac death. Basketball team physicians must be aware of the high prevalence of hypertrophic cardiomyopathy in African American basketball athletes as well as Marfanoid characteristics, which are particularly prevalent in athletes with tall and lean body habitus (see in more detail in a later chapter), as these athletes are at increased risk for sudden cardiac death from arrhythmia, aortic aneurysm, or dissection. Team physicians should have a basic knowledge of the International Criteria for EKG interpretation in athletes and know when to refer to cardiology for further workup and testing.

Although team physicians may not attend all practices and games, they are often on-call for the athletic trainer and coaching staff for questions and emergencies. (The expectation that the team physician attends practices and games varies from institution to institution.) But, during games and practices, team physicians must stay vigilant of the entire environment, both on- and off-court, as they may be requested to attend to an ill or injured coach, staff, referee, or cheerleader.

Traveling with a team is generally an individual institutional policy, and a team physician may be required to travel to some or all away games depending on their contractual arrangement. In the event of travel, a medical bag with supplies and medication will need to be prepared. Team physicians must understand local, state, and national policy in transporting and dispensing medications during travel as well as their medical liability coverage at venues outside of their own facilities and state. At each new venue, the team physician and ATC should meet with the oppo-

ment team's medical staff to discuss the arena emergency action plan (EAP) as well as locate nearby emergency medical centers, radiology services, and pharmacies. An opponent's medical staff may be smaller or more robust depending on the institution, so frequently a team physician will be called upon to evaluate members of either team.

Another responsibility of the team physician is to develop and implement administrative policy and procedures. This often requires the team physician and ATC to create and develop an emergency action plan (EAP) for practice and game venues, which ensures that urgent situations are handled in a comprehensive and consistent manner. The EAP must be rehearsed by all facility personnel and support staff under the guidance of the team physician and ATC. There are also several additional protocols such as concussion assessment and management, drug-testing, and

return-to-play guidelines that the team physician must be aware of and implement.

Additionally, team physicians may find themselves at the forefront of media attention when it comes to the diagnosis and prognosis of injured athletes. Team physicians must be effective and clear communicators. This skill is of utmost importance to ensure private medical information is kept private in compliance with both HIPAA policy and an athlete's wishes. A release of medical information, signed by the athlete or family (if a minor), is required before any discussion or statement can be made by a health-care provider regarding the athlete's health status.

Reference

1. ACSM Team Physician Consensus Statement. https://www.aafp.org/dam/AAFP/documents/patient_care/fitness/ACSMteamphysicianconsensus.pdf.



Team Medical Coverage in Elite European Basketball

12

Baris Kocaoglu, Ignacio Muro, and Guy Morag

12.1 Introduction

The medical staff in elite basketball includes several professional caregivers. Although the medical staff is responsible of treating all types of injuries and illnesses related to basketball, they are also responsible for the regular treatment of illnesses during winter and summer time as well as treating the families of the players so the players could concentrate on their primary reason for being in the team [1, 2].

The head of the medical staff is usually an orthopedic surgeon who specializes in sports injuries. In the last decade it has been changed from a physician who is interested in sports to a specialist sports medicine physician. It can always be a specialist specialized in Sports medicine, which is less popular in Europe. The team physician has to be specialized in orthopedic sports injuries due to the frequent orthopedic problems occurring in Basketball.

There should be at least one more physician which specializes in internal medicine and take cares of all non-orthopedic issues such as flu, cold issues, gastrointestinal issues, and sleeping problems. Taking care of the player's families is also under the

responsibilities of the medical staff of the team as long as there are foreigner players in the team, which must be focused in playing best for the team [2].

The medical team is mainly composed of one main team doctor, one or two consulting doctors who are specialized at sports medicine and orthopedic, one or two consulting doctors who are specialized at internal medicine fields, one or two physiotherapists, one athletic trainer, one or two physiotherapist assistant (chiropractor and a masseur) and nutritionist [3] (Fig. 12.1).

12.2 Physiotherapists

The next important medical staffs are the physiotherapists. In a team of 12–15 players, the team must have at least two specialized physiotherapists, whereas in busy schedule there is even need for three physiotherapists. Additional chiropractor and a masseur or both can help reduce the load from physiotherapists and help to treat players through the season in very busy schedules where there are 3–4 games in a week [4].

The medical staff should include a mental health coach or sports psychologist who can help the players with stress management during the season as well as helping with specific personal issues and part of rehabilitation after injuries. This staff has to be very professional for sports and has to be a part of the collaboration between coaching staff, medical staff, and player.

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Fig. 12.1 Medical team at the bench



12.3 Nutritionist

A nutritionist is a part of the staff and works very close to the athletic trainer/strength and conditioning coach in order to fit the needs of the players in season during practices and daily home nutrition as well as fitting all needs around the games played. Additionally work is to prepare a menu for all away games. There is a special need for arrangement of meals around the flights and a specific list of food for the hotels that the players will get all their nutrition gradients needed without changing the routine and without being influenced from the traveling in order to keep their body unstressed from changes.

The nutritionist needs to fit each and every player his special diet or routine diet through the season taking into consideration that it should fit all time schedule changes through the season. They need to visit the players each week or two to check the player's body weight and fat percentage according to the plan and together with the strength and conditioning coach change if needed or limit certain energy drinks or bars.

The use of energy drinks, energy bars, protein shakes and additional vitamins and amino acids have become very popular in competitive sports. Although it is the responsibility of the team physician to approve all products consumed by the

players in order to avoid violating WADA regulations, the professional consultant in this issue is the nutritionist who is a part of the medical team and decides the players intake in coordination with the strength and conditioning coach.

12.4 Team Preparation Before the Season

Taking care of a basketball team during a season is challenging. However, a high-quality medical care always begins with a good preseason medical preparation. It needs all caregivers to work in close collaboration and in addition to collaborate with the coaches. Our main goal is to prevent injuries and understand the optimal balance between health management and optimizing performance.

The season starts with the preseason checkups. There is no "gold standard" protocol for preseason examinations. A medical questionnaire, which includes all medical history, injuries, and surgeries, MRI was done in the past, and specific issues are being filled by the players. Each player has to pass the stress test according to "Bruce protocol" in order to be allowed for competition, but for a competitive team which invests millions of dollars it is not enough for the evaluation of the player's medical

Fig. 12.2 Medical team bag at European league



condition. Other than the stress test, we recommend performing a cardiac echo, whereas it is even better to perform stress echo, blood tests which include CBC, full biochemistry, and vitamin levels along with EBV, CMV, and HIV tests and urine analysis.

An orthopedic screening must be done checking all joints, stability, flexibility, and muscle strength as well as previous injured joints or muscles. The screening should be performed twice, one by the head physiotherapist and one by the orthopedic surgeon. After the screening it should be decided if imaging studies such as MRI scan are needed for a previously operated knee or a previous lumbar disc herniation. We recommend adding a muscle strength test—Biodex testing to plan the preseason work up and seasonal strengthening and stretching plan. These are generally performed by strength and conditioning coaches and are an integral part of all preseason checkups. It is extremely important to get all examination done for the evaluation of the player and to build a reconditioning plan to correct or improve muscle imbalances and work on sports-specific muscles and joints involved.

The other important issue is to prepare the medical bag at the field (Fig. 12.2). Outlines of some basic, recommended supplies for basketball are given at Table 12.1. The necessary supplies can

Table 12.1 Medical staff bag coverage

<i>CPR and cervical spine</i>
Automatic external defibrillation device
Rigid cervical spine collar of appropriate sizes for the athletes
<i>Airway and ventilation supplies</i>
Bolt cutters or heavy scissors to remove equipment if necessary
Oropharyngeal (Oral) airways, various sizes
14-Gauge catheter for tension pneumothorax
Nasopharyngeal (nasal) airway
Pocket mask for ventilation
Cricothyroidotomy kit
Bag-valve mask (masks and ventilation bag)
Syringe (10 mL)
Lubricant
Intubation equipment (endotracheal tubes with stylet, large forceps (McGill forceps), laryngoscope with blade (and batteries))
Portable suction (manual)
Oxygen tank and reservoir
<i>Circulation supplies</i>
Intravenous catheters
Intravenous line tubing
Intravenous fluids (Normal saline or Ringer's lactate)
Tape to fix IV catheter in place rubber tubing for tourniquet

(continued)

Table 12.1 (continued)

<i>Miscellaneous</i>
Exam gloves (non-sterile and sterile pairs; latex and non-latex)
Red biohazard bag
Penlight bandage/trauma shears (to cut through clothes, tape)
Stethoscope
Blood pressure cuff
Absorbent 4 × 4 gauze, sterile
Oto/ophthalmoscope with blue filter
Sterile dressings and bandages
Oral/rectal thermometer
Band-aids
Reflex hammer
Tongue depressors
Rapid splint (for arm or leg)
Cotton tip applicators
Eye shield
Athletic tape
Mirror
Alcohol swabs
Fluorescein eye drops and cobalt blue light
Antiseptic solution
Tooth transport medium
Disinfectant
Foil blanket
Lubricant
Urine dipsticks
Salt packets ± electrolyte powder/solution
Tampons
Plastic bags for ice, biohazard
Ice
Prescription pad/forms/paper/pen
Syringes and needles
List of banned substances
Suture kit (needle driver, forceps, scissors, scalpel, suture material, flexible skin closure strips)
For transport
Spineboard with side head supports
Stretcher
Sharps box
Crutches (±wheelchair)
<i>Medications</i>
Antibiotic medication
Injectable Epinephrine (1:1000 solution, prefilled syringes)
Anti-inflammatory medication
Dextrose (50%, prefilled syringes)
Local anesthetic solutions
Nitroglycerine (spray or tablets)
Oral glucose paste or tablets
Aspirin chewable tablets
Beta agonist inhaler
Topical medications (antibiotic, steroid cream, sunscreen)
Aerochamber
Other medications

vary, depending on the nature of the game, the number and severity of injuries expected, the number of athletes participating, and the experience of the physician [5]. A team physician should be familiar with the use of all the equipment in the medical bag.

12.5 Field Triage Algorithm

During routine practices there is need for two physiotherapists, which work together with the strength and conditioning coach, and report directly to the coach if a player is injured, sick, or limited due to any mild orthopedic issue such as strain or contusion. The physiotherapist's work is challenging and demanding. It is a daily challenge to keep all players healthy and ready for the practices and games. It is a maintenance work that should be performed delicately. They should be able to notice small changes in muscle balance, muscle stiffness, or minimal swelling in a joint before the player even starts to complain. They have to take care of the equipment and be ready with protective and/or therapeutic braces as needed and be ready for all sorts of taping depends on injured or sore areas the players complain of.

In any event of an injury the head of the medical team should be informed and the triage begins. If the injury is minimal like a strain or sprain, the head physiotherapist should check the player and initial treatment is started. Then if needed, the player should be evaluated by the orthopedic surgeon (Fig. 12.3). After the quick diagnosis is been made, the treatment plan will be noticed to all medical team members and to the coach to estimate the expected time out of game (Fig. 12.4). The player must be a part of the plan and should get all the information about the estimated process. In case of injuries, non-orthopedic injuries such as eye injuries or face injuries, specific consultants which work with the team should be notified to consult the player immediately. There must be a dentist and an ophthalmologist who work closely with the team since these are the common injuries in basketball other than orthopedic injuries.

During competition, there is need for all physiotherapists and at least one physician on the court. The main goal of the medical team is to evaluate

Fig. 12.3 Physiotherapist can also handle minor sports injuries



Fig. 12.4 Decision for out of the game given by team doctor



the injuries quickly during the game and decide if the player can get back on the court. Any bruise or cuts that bleed can be treated immediately on court or in the locker room and get the player back on court as long as they are superficial. Sprains are being checked bench side as well as muscle strains. It is very challenging to evaluate quickly the nature of injury to rely on the player's feeling in order to prevent an irreversible injury. The physician should also be very well aware of personal character of the player who can under- or overestimate the injuries. The collaboration with the coaches and strength and conditioning coach is extremely important in competition especially when a player returns back from an injury and is limited in minutes playing according to a rehabilitation plan.

In case of a serious injury, which causes a player to be out of competition for few weeks such as hamstrings tear, severe ankle sprains, or meniscal injuries, which may need surgical intervention, there is a return to play algorithm, which medical team should work accordingly.

The first and most important is to diagnose the injury and its severity. Each injury has its own timeframe to heal but is still challenging to treat and bring the player back to the court as soon as possible but more importantly as safe as possible. Treatment starts after the diagnosis, mostly with physiotherapy and measurements to decrease the inflammation, swelling, and pain. Medical team should work on range of motion exercises together with strengthening exercises. The next stage of rehabilitation is to initiate basketball-specific exercises with the strength and conditioning coach who is responsible for bringing the player back to the court. After every injury, the return to practice will start gradually with practice without contact and gradually increase the load in practice time and contact load until the player is ready for full practice. If the progress is without any regressions, then the player should be checked to be sure is ready. Mostly there is no need for further imaging unless there is still mild pain or hesitations of the player or the medical staff. It is recommended to bring a player back to the court when he is totally pain-free and has full range of motion. However, there are situations where the physician in partnership with the coach and the player can allow early

return to court even with some limitations. Those limitations such as limiting playing time should be followed strictly to keep the player safe.

12.6 Medical Coverage During Major European Events

Leading and planning during major Basketball events such as European Championship is of paramount importance in keeping all players and staff medically safe. Any physician who has the responsibility of planning medical care during major events should be very well aware of the framework in which they operate. The setting at these events generally poses challenges which are very different from a hospital or private practice environment.

This part of the chapter aims to outline the general characteristics of the medical service delivery at specific events from a perspective of chief medical officer or local medical team.

Forming a Local Organizing Committee (LOC) for medical care is essential for any major elite event. The main focus of chief medical officer (CMO) and medical LOC is to prepare for the upcoming event days to months before the event starts. Medical LOC should be prepared, the organization regarding Venues/site management, hotel clinics, accreditations, emergency action plans, VIP and spectator medical services.

In all elite events, CMO and LOC should provide guidance to visiting medical staff. A medical information guide sheet should be ready and delivered to visiting medical staff at least 1 week before the event (Fig. 12.5). Medical information sheet should contain all the necessary medical information including the communication details of local medical staff, nearest medical care facilities, public emergency medical services, and closest available pharmacies to hotels and venues.

After the arrival of all teams to organizing city, a detailed medical meeting should be organized. Head of medical delegations, members of LOC, CMO, and team attaches should be present in this meeting in which a detailed overview including maps of the city and health-care provider facilities should be discussed thoroughly (Fig. 12.6). At this stage all the questions of



Medical Care Info Sheet

Chief Medical Officer (On Call):

Name: **Kemal Uzun**
 Telephone: +90 505 318 8888
 E-mail: kuzun@icibad.com
 Emergency number in the host country: 112

Partner/ main Hospital: Acibadem, Hospital Group

Emergency traumatology - orthopaedic medical care:

Available Services:

X-ray: Yes
 Ultrasound: Yes
 MRI: Yes
 CT: Yes

Available Services:

X-ray: Yes
 Ultrasound: Yes
 MRI: Yes
 CT: Yes

Elective traumatology - orthopaedic medical care:

Available Services:

X-ray: Yes
 Ultrasound: Yes
 MRI: Yes
 CT: Yes

Nursing around hotel:

Name: **Acibadem, International Hospital**
 Address: **Yesilköy/maab, Istanbul cad. No.82**
 Telephone: Tel.: +90 212 4684444

Available Services:
 Intravenous systems: Yes
 Intramuscular injections: Yes
 ECG: Yes

Dental Services: (Opening hours: Working days 09.00 – 20.00)

Name: **Acibadem, International Hospital**
 Address: **Yesilköy/maab, Istanbul cad. No.82**
 Telephone: Tel.: +90 212 4684444



Physical Therapy:

Physiotherapy unit of **Acibadem, International Hospital** will be available for teams during final phase during working hours upon request.

Pharmacies:

Closest Pharmacy to **Polat, Renaissance Hotel: (Daytime)**

Name: **Deniz Eczanesi**
 Address: **Istanbul cad. No.29, Babuğköy**
 Telephone: +90 212 6637264
 Opening hours: every day 09.00 – 19.00

Name: **Yesilyurt Eczanesi**
 Address: **Şişli/Boğaziçi cad. No.13, Babuğköy**
 Telephone: +90 212 5731520
 Opening hours: every day 09.00 – 19.00

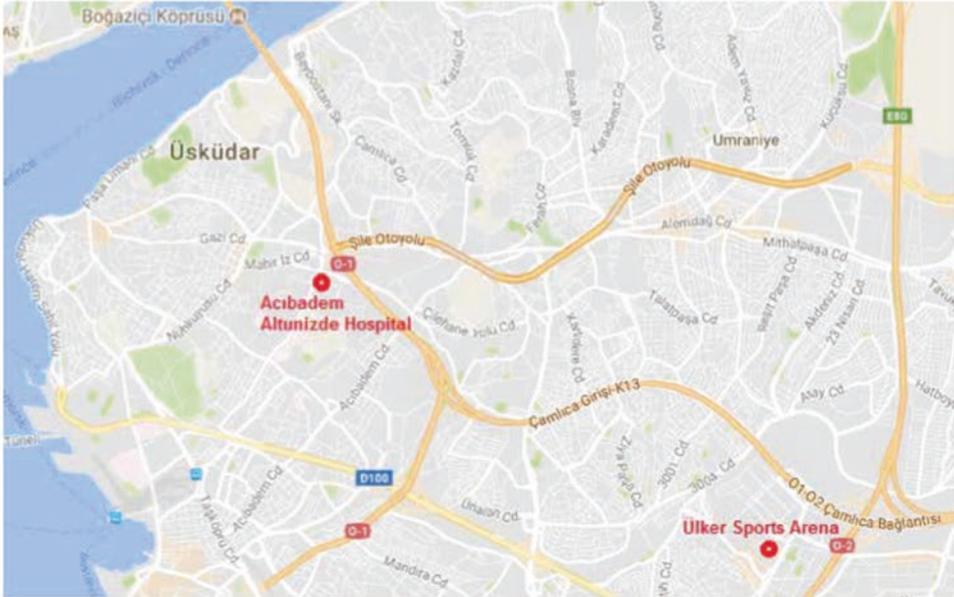
Closest Pharmacies on Night Shift: (Closest to **Polat, Renaissance Hotel)**

Working Hours 19:00- 09:00

10.09.17 İPEK ECZANESİ	Yesilköy Mah. Mekkep Sok. 52/2, Florya, Bakırköy	(212) 574-5750
10.09.17 YESİLÜYÜ ECZANESİ	Yesilköy Mah. Seyit Ali Sok. 55/A, Yesilköy, Bakırköy	(212) 571-7177
11.09.17 ORAL ECZANESİ	Bağcılar Mah. Ahmet Cevdet İsmail Cad. 22-A, Florya, Bakırköy	(212) 580-5911
11.09.17 NUR ECZANESİ	Yesilköy Mah. Ümraniye Sok. 17, Yesilköy, Bakırköy	(212) 663-8608
12.09.17 BİLDÖS ECZANESİ	Yesilköy Mah. Florya Cad. 25/1, Florya, Bakırköy	(212) 4252-2600
12.09.17 SÖĞÜ ECZANESİ	Yesilköy Mah. İstasyon Cad. 17/B, Yesilköy, Bakırköy	(212) 662-3783
13.09.17 FLORYA ECZANESİ	Yesilköy Mah. Tezcan Sok. 15, Florya, Bakırköy	(212) 573-2541
14.09.17 NUR ECZANESİ	Yesilköy Mah. İstasyon Cad. 41/A, Yesilköy, Bakırköy	(212) 573-2092
14.09.17 İPEK ECZANESİ	Yesilköy Mah. Doğruyol Sok. 7, Yesilköy, Bakırköy	(212) 663-1197
15.09.17 İNAN ECZANESİ	Yesilköy Mah. Florya Cad. 23/C, Florya, Bakırköy	(507) 300-4495
15.09.17 YESİLÜYÜ ECZANESİ	Yesilköy Mah. Seyit Ali Sok. 55/A, Yesilköy, Bakırköy	(212) 571-7177
16.09.17 İPEK ECZANESİ	Yesilköy Mah. Mekkep Sok. 52/2, Florya, Bakırköy	(212) 574-5750
16.09.17 YESİLÜYÜ ECZANESİ	Yesilyurt Mah. Şişli/Boğaziçi Cad. 13/B, Yesilyurt, Bakırköy	(212) 571-1520
17.09.17 HİENVA ECZANESİ	Yesilköy Mah. Yesilköy Cad. Fıyın Ahiyentli Merkezi, 48, Florya, Bakırköy	(212) 574-3082
17.09.17 ONUR ECZANESİ	Yesilköy Mah. İstasyon Cad. 6, Yesilköy, Bakırköy	(212) 573-9811

Fig. 12.5 Medical information guide sheet for basketball event

Map 1: Group phase medical map



Map 2: Final phase medical map

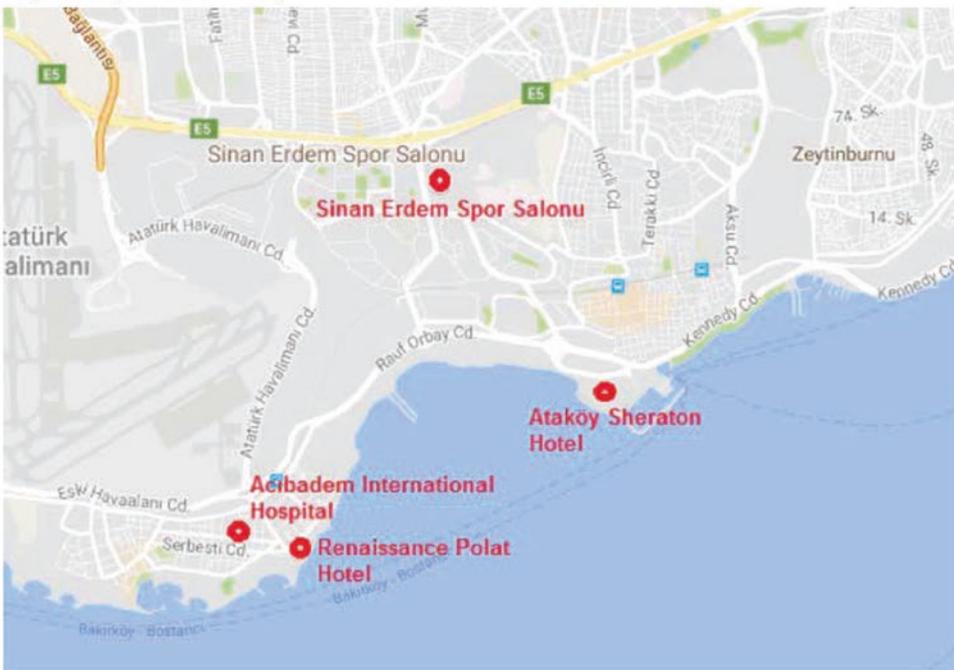


Fig. 12.6 Detailed overview including maps of the city and health-care provider facilities

visiting medical staff must be clearly answered by the members of the LOC remembering the differences in health-care service mentalities in between different countries. At this level, good communication in between CMO, LOC members, team medical staffs, and attaché's should be organized keeping in mind that sport itself is competition area but no competition in medical field should be allowed.

Before and during the games, members of the LOC should be present in venues to provide guidance and give necessary information regarding locations and equipment of treatment rooms, ambulances, and medical personnel.

In case of an injury or emergency, all should be prepared to assist visiting medical staff throughout emergency care, diagnosis, and definitive treatment process.

After the games, LOC should contact visiting medical personnel for assistance of minor injuries if needed.

A nutritionist should be present in LOC to control and supervise the meals in visiting team hotels to prevent and food or water contamination and supply the team's needs.

12.7 Conclusion

Taking care of elite basketball players is challenging and can be very rewarding. It requires teamwork between few disciplines such as medical staff including physicians, physiotherapists, mental coach, nutritionist, and specialist in specific injuries as well as strength and conditioning coach and the head coach and his assistants. All have to work in collaboration with the player himself. The main goal is to prevent injuries, main-

tain good health through the season, diagnose and treat injuries immediately, and finally bring back the player on the court safely. It all starts with preseason checkups and planning strength, stability, and conditioning for the season, continues with daily small interventions during practices by the physiotherapists, strength and conditioning coach, and mental health specialists, and getting ready for competitions. In case of an injury, there is a systematic approach to bring the player quickly and safely back to the court. It can only happen when all abovementioned staff works in collaboration.

Medical coverage during major events should start with a good organization with a very well organized CMO and LOC. Perfect communication between all visiting and local medical committee should be organized keeping in mind that the game itself is the competitive field not the medical services.

References

1. American College of Sports Medicine Expert Panel. Sideline preparedness for the team physician: consensus statement. *Med Sci Sports Exerc.* 2001;33:846–9.
2. Team Physician Manual. International Federation of Sports Medicine (FIMS). Taylor & Francis. Kindle Edition. 2012.
3. Ching BK, Khalili-Borna D. Medical coverage of youth basketball events. *Curr Sports Med Rep.* 2013;12(3):156–61.
4. Kaeding CC, Borchers J, Oman J, Pedroza A. Medical expenditures in division I collegiate athletics: an analysis by sport and gender. *Phys Sportsmed.* 2014;42(3):71–7.
5. Everline C. Application of an online team physician survey to the consensus statement on sideline preparedness: the medical bag's highly desired items. *Br J Sports Med.* 2011;45(7):559–62.



Team Medical Coverage in College Basketball

13

John M. MacKnight and Avinash M. Sridhar

13.1 Review of the Care Team and Their Roles

There are many individuals who comprise the care team for collegiate basketball teams. While the majority of these providers care for all collegiate athletes, those responsible for team medical coverage in collegiate basketball have unique and specialized roles. These providers are detailed as follows:

13.1.1 Team Physicians

The team physician is the primary medical provider for a college athletic team. In addition to providing direct care to athletes during training room appointments, the team physician is often responsible for coordinating care among an array of medical providers that contribute to the comprehensive treatment of the collegiate athlete. Team physicians are present on the sidelines during competition. Working closely with other staff, such as athletic trainers, they are

responsible for the initial diagnostic assessment and treatment plan in the event of an injury during play.

While the team physician is expected to have a focused expertise in musculoskeletal and exercise medicine, they are also responsible for providing primary medical care for student athletes when on campus. Often, both nonoperative primary care sports medicine physicians and orthopedic sports medicine physicians serve as primary team physicians for a collegiate basketball team. These physicians draw from their specialized areas of expertise to diagnose and treat a variety of sports-related injuries.

13.1.2 Athletic Trainers

Athletic trainers are graduates of accredited professional athletic training programs and are capable of evaluating injuries and creating a targeted treatment protocol. Certified athletic trainers focus on injury prevention as well as rehabilitation for those athletes who have sustained injuries. Athletic trainers provide care to college athletes essentially on a daily basis for practice and injury treatment. As the central focus of day-to-day care for their team, athletic trainers have important responsibilities such as providing direct patient care to the athlete and serving as the medical liaison among athletes, coaches, and other medical specialties [1].

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13.1.3 Strength and Conditioning

Strength and conditioning coaches provide comprehensive, sport-specific training regimens for college athletes. In college basketball, these coaches tailor specific programs and adapt training programs to the individual athlete. They formulate personalized training programs that focus on many various components including muscle strength, aerobic and anaerobic endurance, flexibility, coordination, speed, agility, and power [2]. Collaboration with other providers, such as the team physician, athletic trainers, and sports nutrition, is important to provide student athletes with the opportunity to maximize his or her performance potential [2].

13.1.4 Sports Psychology

Sports psychologists have expertise in caring for the mental health of the collegiate athlete. They frequently deal with mental health issues both on and off the court in the realm of collegiate basketball. While common mental health issues that affect college athletes include general mood disorders such as anxiety and depression, sleep disorders, and learning disability, there may be aspects of these conditions that are unique to college basketball players. For example, sports psychologists have used psychological techniques such as progressive relaxation and visual imagery to enhance performance during free throw shooting [3]. Newer psychological concepts, such as the practice of mindfulness, can be taught to college basketball players to improve free throw performance [4]. Ensuring the emotional well-being of all collegiate athletes is at the foundation of optimal general health in addition to peak athletic performance.

13.2 Common Maladies

Due to the inherent musculoskeletal stresses associated with the game of basketball, a number of orthopedic issues are commonly encountered by the treating physician. Some of the most common basketball-associated injuries are discussed below.

13.2.1 Ankle Sprain

The most prevalent orthopedic injury in college basketball is the lateral ankle sprain. With multi-directional movements and the hazards of jumping and landing, sprains of the lateral ankle ligaments are extremely common. The lateral ankle complex is composed of three distinct ligaments: anterior talo-fibular ligament (ATFL), calcaneofibular ligament (CFL), and the posterior talo-fibular ligament (PTFL). Excessive inversion loading of the ankle will injure these ligaments in a sequential manner, with injury to the ATFL classically occurring first and PTFL occurring last. In general, the severity of the ankle sprain equates with the number of ligaments injured and the degree of ankle laxity that is demonstrated on physical exam. A I–III grading system is utilized to describe the injury and to prognosticate on recovery. Key exam features include palpable tenderness over the affected ligaments as well as an assessment for associated bony injury. Particular attention is directed to the distal fibula, proximal fibula as may be involved in a Maisonneuve fracture, and the base of the fifth metatarsal. Anterior drawer and talar tilt testing is useful to assess for joint laxity. Ottawa ankle rules (see Box 1) should be utilized to determine the need for X-rays. General management principles include RICE (Rest, Ice, Compression, Elevation) and protection with activity modification as needed. Gradual return to activity may be undertaken when feeling better, with additional support to the ankle provided by taping, supportive ankle braces, and high-top shoes. Athletes may resume sport when they have restored ROM, normal strength, and good ankle stability. Prevention should focus on strength, flexibility, and proprioceptive training [6].

Box 1: Ottawa Ankle and Foot Rules [5]

An ankle X-ray series is only required if there is any pain in the malleolar zone and ...

- Bone tenderness at the posterior edge or tip of the lateral malleolus

- **OR**

- Bone tenderness at the posterior edge or tip of the medial malleolus

- **OR**

- An inability to bear weight both immediately and in the emergency department for four steps

A foot X-ray series is only required if there is any pain in the midfoot zone and ...

- Bone tenderness at the base of the fifth metatarsal

- **OR**

- Bone tenderness at the navicular

- **OR**

- And inability to bear weight both immediately and in the emergency department for four steps

Anterior and/or posterior tibio-fibular ligament sprains result from higher force loads to the ankle, often with rotation. Pain is felt above the ankle and may extend proximally into the lower leg as a result of injury to the syndesmotic membrane. These “high ankle” sprains are more significant injuries which result in greater degrees of dysfunction and typically take several additional weeks to heal.

13.2.2 Achilles and Patellar Tendinitis

Achilles and patellar tendinitis (“jumper’s knee”) often occur in basketball players as a result of repetitive cutting, jumping, and landing. Chronic tensioning of these tendons results in inflammation and pain that may be function-limiting. Focal tenderness and swelling either in the mid-substance of the tendon or at the bony attachment are common findings. Often an acute increase in physical activity or a change in training regimen may cause these injuries. As overuse syndromes, both generally respond well to RICE, rehabilitation focusing on balanced flexibility and strength,

and activity modification. Some basketball athletes find a counterforce patellar tendon strap to be helpful in changing the loading properties of the tendon for sport. Both conditions generally respond well to the conservative measures above. More violent loading of the Achilles tendon in particular may result in partial or complete tearing with resultant loss of active plantarflexion of the foot and ankle and a positive Thompson/Squeeze test with loss of passive ankle plantarflexion. An acute tear of the Achilles tendon in a basketball player generally necessitates surgical repair to ensure return to play.

13.2.3 Muscle Strains

Basketball players may suffer a variety of muscular injuries as a result of the varied demands of the sport. Muscular strains generally result from transient overload or over-stretching of the muscle with associated muscle fiber disruption. Often the athlete feels a “pull” or “pop” in a muscle group with rapid onset of pain and dysfunction including antalgic gait. The primary muscle groups involved in basketball are the gastrocnemius/soleus complex, hamstrings, quadriceps, and lumbar paraspinal muscles. Physical exam generally reveals loss of range of motion and focal muscle tenderness with spasm and/or hematoma formation. In higher level muscular injuries, a frank defect may be palpable in the area of muscle damage. Muscular injuries generally heal well with time, strengthening, and a progressive pattern of stretching for ROM restoration. Most muscular strains resolve in several weeks, but higher level injuries in muscle groups known to take more time, most notably the hamstrings, may take 6 weeks or longer to heal and allow return to sport.

13.2.4 Anterior Cruciate Ligament (ACL) Tear

ACL tears are devastating season-ending injuries commonly seen in basketball players. Recent data continue to find that women suffer ACL

tears approximately three times more often than men with an incidence of 0.22/1000 athlete exposures [7]. Most female ACL injuries occurred by a noncontact mechanism (60%) versus a contact mechanism for men (59%). These injuries are classically due to noncontact valgus and rotational loads to the knee which create abnormal tensioning of the ACL and subsequent rupture. Often athletes will feel or hear a “pop” when the ACL tears, followed by significant pain, rapid joint swelling due to hemarthrosis, and a sense of instability when loading the knee. Lachman and anterior drawer testing classically reveal increased joint laxity and lack of a firm end point. Management for basketball players is surgical with reconstruction of the ACL being essential to restore sufficient knee stability to return to the physical demands of the sport. Post-operative rehabilitation and return to sport generally takes 9–12 months. Unfortunately, recurrent ACL injury is not uncommon, especially in female collegiate athletes [8].

13.2.5 Finger/Wrist Injuries

Finger and wrist injuries occur commonly in basketball due to ball-induced trauma or from falling onto the outstretched hand. Ball trauma may result in extensor mechanism injuries of the DIP joint (mallet finger) or the PIP joint (boutonniere deformity) as well as fractures and interphalangeal joint dislocations.

Ulnar collateral ligament (UCL) injuries of the thumb metacarpophalangeal joint (“gamekeeper’s thumb” or “skier’s thumb”) create varying degrees of dysfunction based on pain and instability. Dribbling and shooting may both be adversely affected by the injury and the presence of protective bracing. Grade III injuries (complete ligament disruption) of the UCL are generally surgically corrected. Lesser injuries will generally heal well with splint use over 4–6 weeks.

Finally, falling onto the outstretched hand may result in a scaphoid fracture. Pain in the anatomic snuffbox is the hallmark, often with a positive scaphoid shift test. X-rays often do not initially show a fracture; therefore, early manage-

ment should be based on clinical suspicion rather than imaging findings. It is important to immobilize the hand and wrist with a thumb spica splint or cast until a definitive diagnosis is reached. Scaphoid fractures are generally managed operatively in basketball players as there is a high risk for nonunion. The relative severity of the injury, need for surgery, and the ability to place the player in functional bracing all contribute to return to play decisions and timing.

13.2.6 Facial Trauma

Due to the contact risk associated with rebounding or being defended, nasal fractures in basketball are a common facial injury. Nasal fractures generally arise from being struck with an elbow and may occur at the bony bridge of the nose or more inferiorly involving the cartilage. Most nasal fractures heal well without any significant intervention. More aggressive management may be required when the nose is poorly aligned, depressed, or nasal obstruction is present. The examiner should visually evaluate for a septal hematoma which appears as a bluish asymmetric swelling arising from the septal area as seen through the nares. Immediate evacuation of the hematoma is required to avoid septal breakdown. The surrounding facial bones should always be assessed for concomitant injury including the orbits, maxillae, and zygomatic arches. Facial pain, facial asymmetry, loss of extraocular muscle function, or diplopia warrant CT scanning.

13.2.7 Management of Common Illnesses and Impact on Sport

13.2.7.1 Viral Illness

College basketball players are prone to all of the same general medical conditions and illnesses as other athletes. With regard to illness, the suitability for continuing or returning to sport is predicated on common sense principles about readiness and safety for high-level activity. Adherence to these principles ensures that play-

ers are not inappropriately active at a time when it is not safe or prudent for them to do so.

A common construct is to use the “neck up/neck down” division of symptoms as a guide for safe participation. Illnesses with “neck up” symptoms only—sneezing, sore throat, rhinorrhea, sinus pressure, mild headache—allow for continued play. Those illnesses with “neck down” features—fever, chills, sweats, myalgias, significant fatigue/malaise, lymphadenopathy, deep cough, nausea, vomiting, diarrhea—are generally not compatible with high-level exercise and should prompt a hold from basketball activities until resolved.

By far the most common illnesses in basketball players are viral upper respiratory infections (URI). These “common cold” syndromes are generally mild and self-limited, typically lasting 3–5 days. Since these are associated classically with “neck up” features, they allow for ongoing participation with symptomatic management. Viral syndromes associated with “neck down” features require a medical hold from activity until resolved. Most of these more significant illnesses are nonspecific and do not have a well-defined etiology. Influenza is the most common viral syndrome that can be easily diagnosed via testing. Infection with Epstein Barr virus (EBV) causes the classic clinical syndrome of infectious mononucleosis. This condition is associated with a 3- to 6-week course of fevers, significant fatigue, exudative pharyngitis, posterior cervical lymphadenopathy, and varying degrees of hepatosplenomegaly. Because of concerns for possible splenic injury with sport, these athletes are held from all activity for a minimum of 3 weeks from the onset of illness to allow for adequate recovery. In some cases, a number of additional recovery weeks may be required.

13.2.7.2 Concussion

In basketball players, concussions often result from being struck with an inadvertent elbow, hitting the head on the floor during a charge/block collision, or from being undercut during rebounding or jumping. Almost uniformly, athletes suffering a concussion complain of acute onset of headache, nausea with or without vomiting, and

dizziness. Other clinical features may include confusion, fatigue, sound and light sensitivity, poor concentration, and disrupted sleep patterns. The treating physician should rely on consensus guidelines to make the diagnosis and to monitor the natural history of the condition [9, 10]. No athlete should be returned to sport on the day of injury, without exception. Recent research supports the value of low-intensity exercise before complete resolution of the concussion as long as it does not exacerbate symptoms [11]. Once the player is symptom-free, the treating physician should perform a standardized assessment of neurocognitive tests (SCAT5, ImpACT, or equivalent), balance via modified BESS, and oculo-vestibular (VOMS) testing. When all parameters are normal or have returned to athlete-specific baselines, return to basketball is allowed (see Box 2).

Box 2: NCAA Box Concussion Return-to-Play Algorithm [12]

1. Active, targeted treatment includes the following:
 - (a) Progressive aerobic exertion for athletes with prolonged symptoms and impairment,
 - (b) Vestibular therapies that target deficits in gaze stability, proprioception, vestibular ocular reflex, postural control, and dynamic gait,
 - (c) Vision therapies that target deficits such as accommodative and convergence insufficiencies, impaired version eye movements, and ocular misalignments.
2. Return-to-Play Stepwise progression:

Once a concussed student athlete has returned to baseline level of symptoms, cognitive function, and balance, then the return-to-play progression can be initiated as follows in this general outline:

 - (a) Light aerobic exercise such as walking, swimming, or riding a stationary bike. No resistance training. If asymptomatic with light aerobic exercise, then;

- (b) Sport-specific activity with no head impact. If asymptomatic with sport-specific activity, then;
 - (c) Noncontact sport drills and resumption of progressive resistance training. If asymptomatic with noncontact drills and resistance training, then;
 - (d) Unrestricted training. If asymptomatic with unrestricted training, then;
 - (e) Return-to-competition. Medical clearance will be determined by the team physician/physician designee, or athletic trainer in consultation with a team physician.
3. Return to Academics
- (a) Return-to-learn should be managed in a stepwise program that fits the needs of the individual, within the context of a multi-disciplinary team that includes physicians, athletic trainers, coaches, psychologists/counselors, neuropsychologists, administrators as well as academic (e.g., professors, deans, academic advisors) and office of disability service representatives.

13.2.7.3 Cardiac Concerns

College basketball athletes are at increased risk of sudden cardiac death and cardiac complications relative to many other athletes. Though the exact etiology of this predisposition is unknown, it is assumed to relate to the extended high-level aerobic demands of the sport. Screening for pre-existing cardiac conditions is a universal component of the pre-participation evaluation of all collegiate athletes. While cardiovascular disorders are the leading cause of sudden death in young athletes during sports and exercise [13–16], collegiate basketball players, particularly male athletes of African descent, have a higher risk of certain cardiac anomalies that can cause sudden cardiac death [5, 9].

Hypertrophic cardiomyopathy (HCM) is the most common cause of sport-associated sudden cardiac death in the United States, accounting for almost 50% of cases [17]. HCM is defined

by abnormal thickening of the left ventricular or septal walls of the heart, creating either pathologic outflow from the left ventricle or resulting in electrical instability and arrhythmias due to myofibrillary disarray in the myocardial tissue. This autosomal-dominant genetic condition is actually quite common with an incidence of roughly 1:500 in the general population, but varying degrees of penetrance and genetic expression dictate that many who carry the gene will never develop the pathologic condition [18]. Cardiac auscultation may demonstrate a harsh systolic murmur, loudest at the lower left sternal border, which increases with the Valsalva maneuver or dynamic positional changes (squat to stand) and does not radiate to the carotid arteries [19]. It is important to note that some HCM patients do not demonstrate a pathologic murmur. Electrocardiogram (EKG) may demonstrate left axis deviation or left ventricular hypertrophy in the case of HCM [20]. Echocardiogram or cardiac MRI may be required to definitively diagnose this condition and elucidate quantitative information that can be used to restrict or modify an athlete's participation in sport accordingly. Because of the high cardiovascular stresses of basketball, HCM would be more likely to manifest in a basketball player than in many less-demanding sports. Although many collegiate basketball teams perform pre-participation cardiac screenings with EKG and echocardiography [21], it can be difficult to diagnose HCM because it may develop very slowly over years of time, often after the time of initial screening. Unfortunately, sudden death is a common presenting feature.

Marfan's syndrome is a genetic disorder of connective tissue primarily affecting heart, eyes, blood vessels, and bones and characterized by a number of typical phenotypic features. Because of the association of Marfan's syndrome with tall stature, taller basketball players should be considered for this diagnosis. Criteria for a diagnosis of Marfan's are included in Box 3. Depending on the status of their aorta, basketball players with Marfan's can potentially play with close cardiology monitoring to evaluate for progressive aortic root widening with associated risk for aortic rupture.

Box 3: Diagnostic Criteria for Marfan's Syndrome [22]

In the absence of a family history:

1. Ao ($Z \geq 2$) AND EL = MFS
2. Ao ($Z \geq 2$) AND FBN1 = MFS
3. Ao ($Z \geq 2$) AND Syst (≥ 7 points) = MFSa
4. EL AND FBN1 with known Ao = MFS
EL with or without Syst AND with an FBN1 not known with Ao or no FBN1 = ELS Ao ($Z < 2$) AND Syst (≥ 5) with at least one skeletal feature without EL = MASS MVP AND Ao ($Z < 2$) AND Syst (> 5) without EL = MVPS

In the presence of a family history:

5. EL AND FH of MFS (as defined above) = MFS
6. Syst (≥ 7 points) AND FH of MFS (as defined above) = MFSa
7. Ao ($Z \geq 2$ above 20 years old, ≥ 3 below 20 years) + FH of MFS (as defined above) = MFSa

Systemic score

- Wrist AND thumb sign -3 (Wrist OR thumb sign -1)
- Pectus carinatum deformity -2 (pectus excavatum or chest asymmetry -1)
- Hindfoot deformity -2 (plain pes planus -1)
- Pneumothorax -2
- Dural ectasia -2
- Protrusio acetabuli -2
- Reduced US/LS AND increased arm/height AND no severe scoliosis -1
- Scoliosis or thoracolumbar kyphosis -1
- Reduced elbow extension -1
- Facial features (3/5) -1 (dolichocephaly, enophthalmos, downslanting palpebral fissures, malar hypoplasia, retrognathia)
- Skin striae -1
- Myopia > 3 diopters -1
- Mitral valve prolapse (all types) -1

Maximum total: 20 points; score ≥ 7 indicates systemic involvement

Ao Aortic diameter at the sinuses of Valsalva above indicated Z-score or aortic root dissection, EL Ectopia lentis, ELS Ectopia lentis syndrome, FBN1 Fibrillin-1 mutation, FBN1 not known with Ao FBN1 mutation that has not previously been associated with aortic root aneurysm/dissection, FBN1 with known Ao FBN1 mutation that has been identified in an individual with aortic aneurysm, FH Family history, MASS Myopia, mitral valve prolapse, borderline ($Z < 2$) aortic root dilation, skeletal findings, striae, MFS Marfan syndrome, MVPS Mitral valve prolapse syndrome, Syst Systemic score, US/LS Upper segment/lower segment ratio; Z Z-score.

In other parts of the world, most notably in Italy, arrhythmogenic right ventricular cardiomyopathy (ARVC) is the most common cause of sudden cardiac death in athletes. This disorder results from unlinking of desmosomes in the myocardial cells which creates focal areas of myocardial weakness and arrhythmogenic sources due to fibrofatty deposition in the involved areas. This condition is generally not compatible with high-level basketball participation.

A number of other underlying cardiac conditions should be considered in basketball players. Anomalous coronary arteries, myocarditis, a variety of cardiomyopathies, and inherited rhythm disorders such as congenital long QT syndrome and Wolff-Parkinson-White syndrome may present as exercise intolerance, chest pain, inappropriate shortness of breath, syncope, or sudden death. There still exists a debate in the sports medicine community about the benefits and risks of pre-participation cardiac screenings in athletes. That determination is individualized based on the sport and its associated risk for cardiac complications, access to screening methods, assistance in interpreting equivocal findings, and the availability of high-quality cardiology

back-up for additional testing if needed. It is our belief that cardiac screening in basketball players is highly desirable if the above conditions can be met.

13.3 Miscellaneous

13.3.1 Travel

Travel produces a number of challenges for the basketball team physician. Collegiate basketball teams play 30–35 games per season and typically will play 15–18 away games. Depending on the distance traveled and the number of time zones covered, basketball players may experience significant disruptions in their natural body rhythms with regard to practice and game times, meal times, and sleep cycles. The basketball team physician needs to be acutely aware of the variations in schedule associated with road games for their team. Whenever possible, the team physician should utilize outside resources such as a sport nutritionist and sports psychologist to assist with ensuring adequate caloric intake and timing of meals leading up to the time of the game as well as assistance with sleep and relaxation techniques.

One of the other challenges of travel is an increased risk for illness with increased exposure to potentially ill individuals, especially during the cold weather months in a typical basketball season. Steps should be taken to minimize the likelihood of such exposures, and players should be encouraged to maximize their lifestyle management with regard to sleep, caloric intake, and hydration. The rigors of travel also increase the risk of illness simply by creating an added level of fatigue to a group of players who may already be weary from the demands of the season.

13.3.2 Sleep

Perhaps no single lifestyle measure has a greater impact on health and performance in basketball than sleep. As noted above, sleep can be disrupted either because of time zone changes or loss of hours of sleep because of the logistics of travel-

ing from one venue to the next. It is crucial that the basketball athlete understands the importance of adequate sleep for optimizing performance and for recovery after high-level activities. It should be emphasized that a short night of sleep cannot be adequately compensated for after the fact. Particularly with scholastic athletes, this can be a major challenge when academic demands are higher and the individual simply has inadequate time to get the rest they need.

For athletes traveling multiple time zones, the treating physician should anticipate management of the time change [23]. In general, players should try to immediately adapt to the local time of the area that they have traveled to. For significant time zone differences, it is advisable to try to travel at least one additional day prior to game day to allow for better adaptation to local time. Ideally, the normalization of the sleep pattern occurs naturally. In circumstances where athletes are struggling with sleep, safe and conservative measures such as melatonin or sedating antihistamines such as diphenhydramine may aid in falling asleep.

It is also important to realize that each basketball athlete has an innate circadian rhythm where certain times of the day will be preferable for high-level activities, while others will not. Travel may further exacerbate this and may fundamentally force an athlete away from the timeframe that would be ideal for them to be performing. There is no fundamental means of compensating for this, but it is important for the athlete, coach, and medical staff to appreciate that some individuals will not feel well or perform well during times when their circadian rhythms do not favor optimal performance.

13.3.3 Summer Training

Several years ago, the NCAA approved summer practice for collegiate basketball programs. This created a unique set of challenges with regard to balancing the necessary work of team preparation versus providing adequate rest time in what previously had been the off-season. This added physical demand increases the risk of

stress injuries and also can be mentally taxing as collegiate basketball players now get few breaks through the course of the entire year. The basketball team physician should be monitoring both physical and emotional needs of the team during this time frame to ensure that this added level of practice and preparation is not counterproductive.

13.3.4 Mental Health

Mental health concerns have become much more prominent in sport in recent years. Athletes are seeking assistance for their mental health concerns more commonly and are appreciating that they are performing in an increasingly pressured circumstance which lends itself to the development of generalized anxiety, depression, panic attacks, obsessive-compulsive disorder, and related conditions. A basketball team physician should always be on the lookout for fundamental changes in behavior or thoughts within their players. They should create a nurturing environment with an open-door policy to encourage basketball players to address their emotional needs openly and feel comfortable to do so. Whenever possible, additional specialized resources such as a team psychiatrist, team psychologist, or counseling services should be available. It is always important to remember that student athletes face all of the same emotional struggles as the general population and are no less likely to have issues with those mental health concerns over time. Medication use in this population is generally very well tolerated. Selective serotonin re-uptake inhibitor (SSRI) therapy in particular is very useful with regard to management of generalized anxiety or depression. Relaxation techniques, visualization, and other cognitive behavioral therapy interventions not only provide the athlete with significant mental health benefits for life in general but may also translate over to improvements in performance. In some cases, athletes' concerns are purely sport-related, and these can be nicely addressed by the sports psychologist with utilization of the conservative techniques noted above.

13.4 Conclusion

Caring for a college basketball team is an enjoyable yet challenging experience for the sports medicine physician. Because of the broad spectrum of medical and orthopedic concerns that arise in basketball players, it is essential to have a broad working knowledge of illness and injury management in this unique group. Rapid and accurate diagnosis coupled with a methodical and appropriate treatment plan is an essential skill that the basketball team physician may be asked to perform on a daily basis. Whenever possible, the physician should maximize prevention strategies for their team and anticipate issues that may arise relating to travel, academic demands, and the inherent mental health challenges of collegiate sports. Providing such care is the foundation of student athlete success as the physician's thoughtful evaluation, treatment, and return to play protocol ensure both the safety and optimal performance of the collegiate basketball player.

References

1. Romero MG, Pitney WA, Brumels K, Mazerolle SM. Role strain, part 1: experiences of athletic trainers employed in the professional sports setting. *J Athl Train.* 2018;53(2):184–9. <https://doi.org/10.4085/1062-6050-213-16>.
2. Strength and Conditioning (n.d.). <https://virginiasports.com/sports/2019/3/1/strength-and-conditioning.aspx>
3. Hall EG, Erffmeyer ES. The effect of visuo-motor behavior rehearsal with videotaped modeling on free throw accuracy of intercollegiate female basketball players. *J Sport Psychol.* 1983;5(3):343–6. <https://doi.org/10.1123/jsp.5.3.343>.
4. Gooding A, Gardner FL. An investigation of the relationship between mindfulness, Preshot routine, and basketball free throw percentage. *J Clin Sport Psychol.* 2009;3(4):303–19. <https://doi.org/10.1123/jcsp.3.4.303>.
5. Stiell IG, Greenberg GH, McKnight RD, Nair RC, McDowell I, Worthington JR. A study to develop clinical decision rules for the use of radiography in acute ankle injuries. *Ann Emerg Med.* 1992;21:384–90.
6. Minoonejad H, Karimizadeh Ardakani M, Rajabi R, Wikstrom EA, Sharifnezhad A. Hop stabilization training improves neuromuscular control in

- college basketball players with chronic ankle instability: a randomized controlled trial. *J Sport Rehabil*. 2019;28(6):576–83.
7. Agel J, Rockwood T, Klossner D. Collegiate ACL injury rates across 15 sports: National Collegiate Athletic Association Injury Surveillance System Data Update (2004–2005 through 2012–2013). *Clin J Sport Med*. 2016 Nov;26(6):518–23.
 8. Brumitt J, Mattocks A, Engilis A, Isaak D, Loew J. Prior history of anterior cruciate ligament (ACL) reconstruction is associated with a greater risk of subsequent ACL injury in female collegiate athletes. *J Sci Med Sport*. 2019;22:1309–13.
 9. McCrory P, Meeuwisse W, Dvorak J, et al. Consensus statement on concussion in sport—the 5th international conference on concussion in sport held in Berlin, October 2016. *Br J Sports Med*. 2017;51:838–47.
 10. Broglio SP, Cantu RC, Gioia GA, et al. National Athletic Trainers' Association position statement: management of sport concussion. *J Athl Train*. 2014;49(2):245–65.
 11. Leddy JJ, Wilber CG, Willer BS. Active recovery from concussion. *Curr Opin Neurol*. 2018 Dec;31(6):681–6.
 12. NCAA Concussion Diagnosis and Management Best Practices. <http://www.ncaa.org/sport-science-institute/concussion-diagnosis-and-management-best-practices>. Accessed 23 Sep 19.
 13. Harmon KG, Asif IM, Maleszewski JJ, et al. Incidence, cause, and comparative frequency of sudden cardiac death in National Collegiate Athletic Association Athletes: a decade in review. *Circulation*. 2015;132:10–9.
 14. Maron BJ, Doerer JJ, Haas TS, et al. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980–2006. *Circulation*. 2009;119:1085–92.
 15. Maron BJ, Haas TS, Murphy CJ, et al. Incidence and causes of sudden death in U.S. college athletes. *J Am Coll Cardiol*. 2014;63:1636–43.
 16. Harmon KG, Asif IM, Klossner D, Drezner JA. Response to letter regarding article, “incidence of sudden cardiac death in National Collegiate Athletic Association Athletes”. *Circulation*. 2011;124(18):e486. <https://doi.org/10.1161/circulationaha.111.055707>.
 17. Peterson DF, Siebert DM, Kucera KL, Thomas LC, Maleszewski JJ, Lopez-Anderson M, Suchsland MZ, Harmon KG, Drezner JA. Etiology of sudden cardiac arrest and death in us competitive athletes: a 2-year prospective surveillance study. *Clin J Sport Med*. 2018; <https://doi.org/10.1097/JSM.0000000000000598>.
 18. Desai MY, Ommen SR, McKenna WJ, Lever HM, Elliott PM. Imaging phenotype versus genotype in hypertrophic cardiomyopathy. *Circ Cardiovasc Imaging*. 2011;4(2):156–68. <https://doi.org/10.1161/circimaging.110.957936>.
 19. Bickley L, Szilagyi P. Bates' guide to physical examination and history taking. Philadelphia, PA: Lippincott Williams & Wilkins; 2003.
 20. Burns J, Jean-Pierre P. Disparities in the diagnosis of hypertrophic obstructive cardiomyopathy: a narrative review of current literature. *Cardiol Res Pract*. 2018;2018:1–6. <https://doi.org/10.1155/2018/3750879>.
 21. Miars CW, Stamatis A, Morgan GB, Drezner JA. Cardiovascular screening practices and attitudes from the NCAA autonomous “power” 5 conferences. *Sports Health*. 2018;10(6):547–51.
 22. Loeys BL, et al. Revised Ghent criteria for the diagnosis of Marfan syndrome (MFS) and related conditions. *J Med Genet*. 2010;47:476–85.
 23. Bonnar D, Bartel K, Kakoschke N, Lang C. Sleep interventions designed to improve athletic performance and recovery: a systematic review of current approaches. *Sports Med*. 2018;48(3):683–703.



Team Medical Coverage in the National Basketball Association (NBA)

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14.1 Medical Team Members

Each NBA team has a multidisciplinary team of healthcare professionals that work together to provide comprehensive healthcare management with the goal of optimizing players' physical health, mental well-being, and sports performance. Although there is a Director of Sports Medicine for the NBA, his role in day-to-day team coverage for a specific team is limited and beyond the scope of this chapter. Different organizations within the NBA may employ different roles within their network of healthcare professionals; however, there are core elements of each team that are quite similar (Table 14.1). These core elements include a head team physician, as well as other physicians that may vary from team to team. Most NBA team physicians are either primary care physicians or orthopedic surgeons who have completed fellowship training in sports medicine. These physicians as well as the team dentist provide team coverage at every home game throughout the season.

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In addition to this team of physicians, there are also several nonphysician members of the medical team (Table 14.1). These often include a head athletic trainer, an assistant athletic trainer, a physical therapist, a strength and conditioning specialist, a massage therapist as well as a sports performance psychologist. Some organizations also include a director of sports performance that oversees this group and serves as a liaison between management for the organization and the various members of the medical team.

Ad hoc medical team members include a variety of consulting physicians. A cardiology specialist is heavily involved in pre-participation evaluations (refer to pre-participation evaluation section below) as well as to manage any athletes with cardiac pathology. Neurological specialists with expertise in concussion are also involved in pre-participation evaluations including obtaining baseline concussion testing (refer to pre-participation evaluation section below). In addition, they supervise return-to-participation guidelines specific to basketball that are analogous to the return-to-play protocols associated with the management of other sports-related concussions. Most recently, the NBA has implemented requirements and recommended practices with regard to players' mental health and wellness. These requirements include access to psychiatry and psychology providers as well as a mental health emergency action plan (refer to mental health section below).

Table 14.1 Network of healthcare professionals providing care for an NBA team

A. Physician/dentistry team members
Head team physician
Additional team physicians (usually with sports medicine specialization in orthopedic surgery or primary care)
Dentistry
Cardiology (consultant)
Neurology (consultant)
Psychiatry (consultant)
B. Nonphysician team members
Director of sports performance (variable)
Head athletic trainer
Assistant athletic trainer(s)
Physical therapist
Strength and conditioning specialist
Sports performance psychology
Massage therapist

Finally, there are a multitude of orthopedic (foot and ankle, hand, spine, etc.) and non-orthopedic (e.g., ophthalmologists, pulmonologists, general surgeons, etc.) physicians whose opinions are sought under specific circumstances as needed.

It should also be noted that the NBA also encompasses the NBA G-League and as a result the medical guidelines for the NBA also apply to the NBA G-league. Although there may be significant geographic separation between the G-league facilities relative to its affiliate NBA team, G-league athletes will often participate in basketball in both the NBA and the NBA G-league. Thus, the medical team for each of these affiliated organizations has a certain degree of inherent overlap with regard to resources and infrastructure.

14.2 Pre-Participation Evaluations

Pre-participation evaluations (PPEs) include the five components listed in Table 14.2. The medical evaluation is composed of a history, including a family history of sudden death and known cardiac disease, musculoskeletal injury history, neurologic history (i.e., prior history of concussion) as well as a physical exam. The

Table 14.2 Components of the pre-participation medical evaluation

PPE components
Medical evaluation
Orthopedic evaluation
Concussion screening
Cardiac screening
Clinical lab tests

spine, upper extremities, and lower extremities are evaluated for orthopedic pathology with additional evaluation of any known history of musculoskeletal injury or dysfunction. Baseline concussion screening is also required by the NBA. Currently, standardized baseline concussion screening tests include the NBA Concussion Assessment Tool (NBA-CAT) as well as the CogState Computerized Cognitive Assessment Tool. Cardiac screening tests are performed by a cardiologist consisting of standard vital signs, auscultation of heart sounds, a resting electrocardiogram (ECG), as well as a treadmill stress transthoracic echocardiogram. A panel of clinical laboratory tests is performed annually which includes a lipid panel, comprehensive metabolic panel, hemoglobin A1c, thyroid stimulating hormone, ferritin, 25-OH vitamin D, and a complete blood count. Sick cell status is assessed once when the athlete first enters the league. From an infectious disease standpoint, QuantiFERON-TB gold testing is performed to screen for tuberculosis. Players are also recommended for the following vaccines: varicella, measles, mumps, rubella, tetanus and pertussis, hepatitis B, and influenza (Table 14.3). Antibody titers of these diseases are checked to confirm immunity. Further medical, radiologic, or orthopedic evaluation is completed as needed based on the medical history of individual athletes. All of the components of a standard NBA PPE are also performed on the athletes who elect to participate in the annual NBA combine which assesses all athletes who are candidates for that year's NBA draft. It should be noted that, once players are drafted to a team, these PPE tests are not necessarily repeated after the combine, unless there is a medical issue that requires further characterization.

Table 14.3 List of vaccinations recommended for each player in the NBA

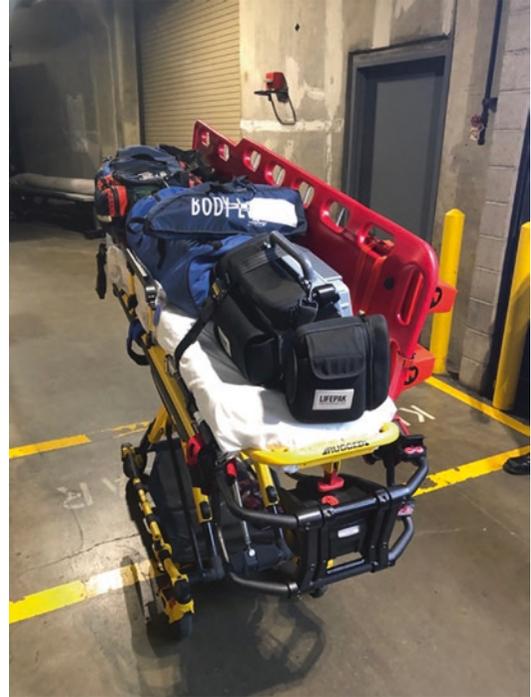
Recommended vaccinations
Measles, mumps, and rubella (MMR)
Tetanus and pertussis
Varicella
Hepatitis B
Influenza

14.3 Emergency Action Plan

Prior to the start of each season, the medical professionals involved with the care of each NBA team are required to review the action plan for medical emergencies that may be encountered throughout the course of a basketball season. This includes issues such as pneumothorax, cardiac arrhythmias, cervical spine injuries, and severe musculoskeletal trauma such as joint fracture dislocations or open fractures. This emergency action plan (EAP) review is typically done in coordination with local paramedics who will be covering games throughout the season. Medical transportation is stationed in close proximity to the basketball court with direct access to surface streets in order to facilitate expeditious transportation of athletes in the event of a medical emergency. Of note, these paramedics/EMTs are dedicated to caring for only the players and coaches. They do not treat medical needs of venue patrons. There are other medical resources available at the venue for the treatment of game spectators. Standard paramedic medical supplies and equipment are available within close proximity to the basketball court. This equipment includes: six lead ECG with cardiac monitoring; pulse oximetry; cardiac defibrillator capabilities; oxygen delivery; intubation equipment; intravenous (IV) access kits; IV fluids; and selected medications as well as a 7-ft spine board (to accommodate athletes with tall stature) with cervical spine stabilization pads (Fig. 14.1).

14.4 Mental Health

The NBA has taken major strides in the recent past to improve mental health resources for its athletes. In addition to creating a series of recom-

**Fig. 14.1** Emergency medical technician equipment on game day

mended practices for mental health care delivery, prior to the start of the 2019–2020 season, the NBA instituted a number of required standards by which each NBA team must abide. For example, NBA teams are required to have a licensed and board-certified consulting psychiatrist who works in collaboration with one to two other mental health clinicians (psychiatrists, psychologists, or social workers) who work with the team and must be available to athletes on a regular basis. Additionally, each NBA medical team must have a written action plan for mental health emergencies that are distributed to all team staff that interact regularly with the players. This plan must include information regarding when each mental health professional is “on call,” and how the consulting psychiatrist may be reached in order to facilitate an emergency room visit or hospital admission, if necessary. In addition to these requirements, the NBA has a number of general recommendations for mental health services, including creating an appropriate physical and emotional environment for care, integrating

mental health with other medical care and mental performance consulting services, as well as educating players on the availability and confidential nature of mental health care. These activities are intended to be conducted in a designated private space within each team’s training facility in order to promote privacy and confidentiality for players engaging these resources. Furthermore, these available integrated mental health services should include player wellness resources addressing issues such as nutrition, sleep, stress reduction, and mindfulness, combined with modalities enhancing both physical and mental recovery.

by a physician are provided as needed by the home team’s healthcare providers. NBA athletes are commonly affected by musculoskeletal injuries. As a result, each NBA game venue is equipped with X-ray imaging capabilities. EMT services are also present at each home game with the resources as described earlier in the “emergency action plan” section. Team physicians routinely accompany the team for post-season playoff games and for selected preseason neutral site games when their presence is requested.

14.5 Game Day Coverage and Resources

The NBA season is travel-intensive (Fig. 14.2, home vs. away game schedule), however, team coverage responsibilities are mitigated by the local team coverage paradigm. Although athletic trainers and other members of the medical team will travel to all regular season away games, it is unusual for team physicians to do so. Instead, medical issues requiring evaluation and treatment

14.6 NBA Season Overview

The NBA basketball season consists of a preseason, a regular season, and the playoffs (Fig. 14.2). As an example, the 2019–2020 season consists of a preseason from September 30, 2019, to October 18, 2019. The regular season is from October 22, 2019, to April 15, 2020, and playoffs are from April 18, 2020, to June 2020. Teams play 5 to 6 games in the preseason with several games being played at international venues. There are 82 games played in the regular season: 41 at home and 41 away. Teams typically play several away games consecutively

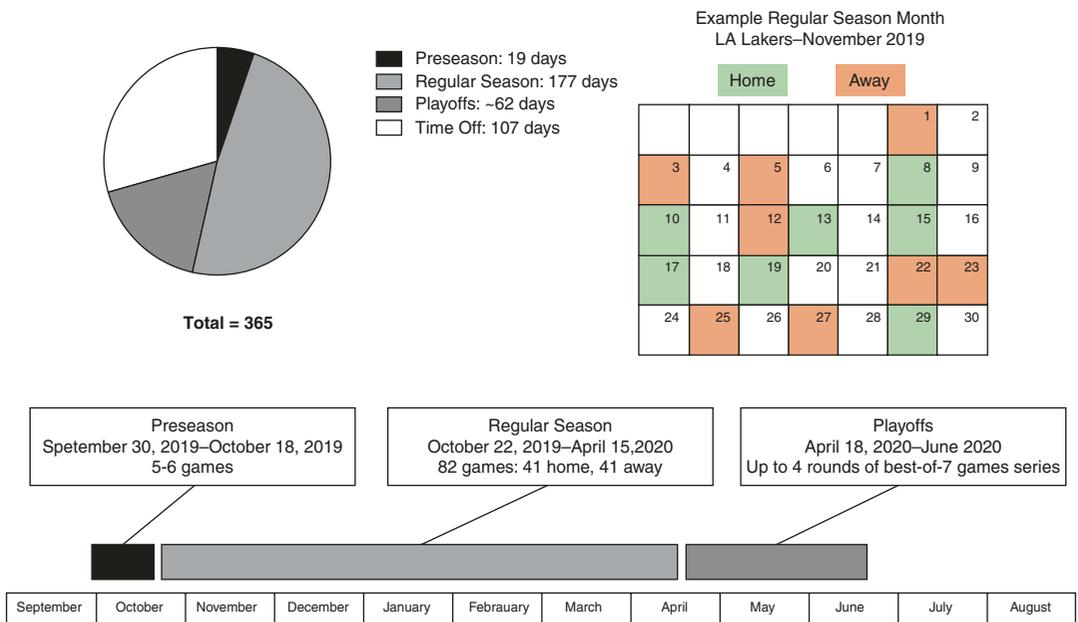


Fig. 14.2 NBA season overview and representative regular season schedule

followed by several games at home. During the regular season, NBA teams never play against the same opponent at the same location for back-to-back games. Figure 14.2 contains a representative sample of home and away games for a typical month during the NBA regular season. Sixteen out of the 30 teams make the playoffs, which begin in April. In total, teams may play up to 4 rounds of 4–7 playoff games (16–28 games total) with a significant amount of traveling during the playoffs.

14.7 Injury Documentation

Injury reporting and research in the NBA have seen major advances in the last few years since the creation of a centralized electronic medical record (EMR) in 2012. Accessed and updated by team physicians and athletic trainers, and audited by the NBA to ensure reporting compliance, this NBA player injury database contains detailed records of injuries and illnesses suffered by players throughout their careers [1]. This allows for medical records to be easily transferred among medical teams when a player is drafted, acquired, or traded. In addition, the EMR system allows for detailed research to be performed characterizing contemporary medical needs and accurately assessing the interventions used to treat the elite athletes that make up this unique population. Prior to 2012, injury data was recorded by and collected from individual teams; however, quality control interventions and auditing were not conducted by the NBA. Given that the majority of large studies of injuries and illnesses in the NBA to date have come from data gathered prior to the introduction of the player injury database in 2012, or even from publicly available injury records, the quality of injury and health research among NBA athletes is expected to greatly improve in the years to come.

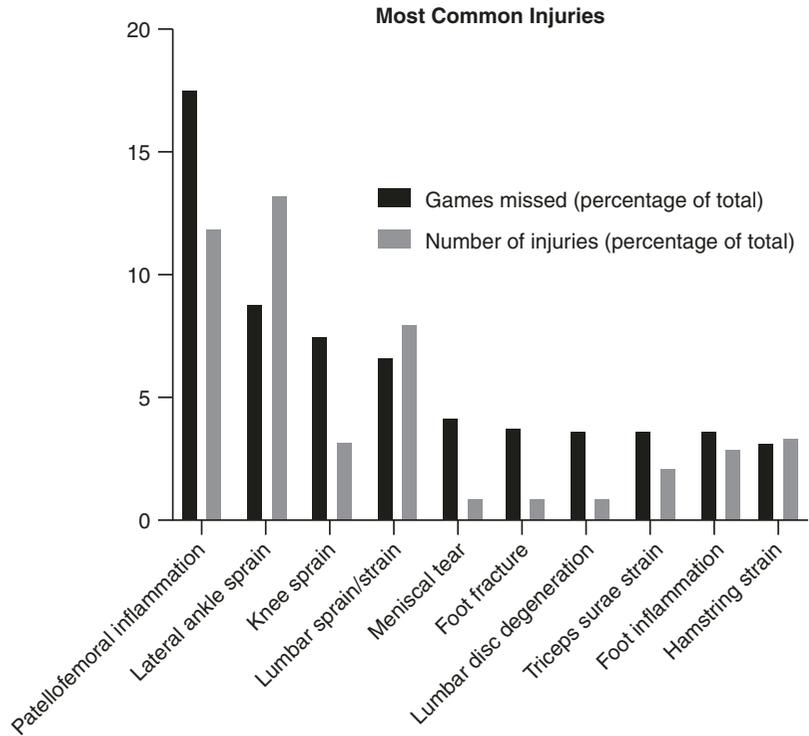
14.8 Common Musculoskeletal Injuries

A review of the available literature regarding injuries in the NBA does provide some insight with regard to the frequency and burden of mus-

culoskeletal injuries in this population. In estimating the impact of these injuries on the NBA, especially from the perspective of a team franchise, a useful metric to consider is the number of games missed as a percentage of games missed for all reasons. In the most recent comprehensive analysis of injuries in the NBA over 17 years, Drakos et al. [2] found the following as the ten most significant causes of games missed among NBA athletes: patellofemoral inflammation (17.5%), lateral ankle sprain (8.8%), knee sprain (7.4%), lumbar sprain/strain (6.6%), meniscal tear (4.1%), foot fracture (3.7%), lumbar disc degeneration (3.6%), triceps surae strain (3.5%), foot inflammation (3.5%), and hamstring strain (3.1%) (Fig. 14.3). It should be noted that the majority of injuries (accounting for the most games missed by players) have a short return-to-play/recovery period, typically consisting of a duration of days to weeks. Meniscal tears, foot fractures, and lumbar disc degeneration on the other hand, often are treated with surgical intervention and are associated with longer recovery periods which have greater impact on the quantity of games missed. The overall team impact of these surgically treated injuries can be even more significant than other injuries, given the relatively small number of players that comprise an NBA team. A similar, often surgically treated injury that was not included in this study: ACL tear represents another highly significant potential contributor to games missed. Minhas et al. reported that ACL reconstructions represented 18.7% of surgical procedures reported in the literature among NBA athletes [3]. As a result, ACL injuries certainly warrant further investigation with regard to interventions that may mitigate the risk of these injuries in the future.

Another metric used in quantifying injury impact in the NBA is the number of injury occurrences as a percentage of all injuries. This is particularly useful information for medical staff aiming to be well prepared to successfully manage the most common basketball injuries. Many of the ten most common injuries also appear as common causes for games missed (Fig. 14.3, black bars); they include lateral ankle sprain (13.2%), patellofemoral inflammation (11.9%),

Fig. 14.3 Most common musculoskeletal injuries in the NBA and percentage of missed games stratified by injury type as reported in the literature [2]



lumbar sprain/strain (7.9%), hamstring strain (3.3%), adductor strain (3.1%), knee sprain (3.1%), foot inflammation (2.9%), quadriceps contusion (2.7%), knee/patella contusion (2.5%), and finger sprain (2.4%) [2].

14.9 Non-Musculoskeletal Injuries and Illnesses

Musculoskeletal injuries are not the only source of morbidity among NBA players. Medical problems including upper respiratory infection, concussion, gastrointestinal distress, and head and face lacerations are the most common documented non-musculoskeletal healthcare issues affecting NBA athletes. Upper respiratory infections (URIs) have been reported to account for 16.7% of all reported injuries and illnesses in the NBA, making them the single most commonly documented injury or illness [4]. URIs are responsible for 2.4% of games or practices missed, which is just below the percentage of missed games as a result of a hamstring strain [4].

Accounts of concussion as the etiology of missed games in the NBA vary widely from 4.3 concussions per season [2] to 14.9 concussions per season [5] (0.6%–2.1% of all injuries). Starting with the 2011 season, the NBA instituted a league-wide concussion protocol which raised the average number of games missed per concussion from 1.6 to 5 [5]. This increase was likely due to increased awareness and caution regarding the risks of playing with a concussion. Upon returning to play after concussions, players were reported to appear to have no difference in performance in their initial five games upon returning to play when compared to their prior performance, and this lack of performance deficiency was also noted in comparison to matched controls [6]. Gastrointestinal distress is a relatively common illness (3.5% of injuries and illnesses); however, this has not been reported to be a significant source of practices or games missed (0.5%) [4]. The importance of NBA medical staff in being prepared to address gastroenteritis, however, is perhaps best exemplified by a 2010 norovirus outbreak that may have affected up to

24 players and staff from 13 different NBA teams [7]. Head and face lacerations represent 2.5% of all injuries and illnesses, but these injuries only result in 0.1% of games and practices missed [4]. Finally, other medical problems that have been described in the NBA population include dehydration during games [8], eye injuries [9], vitamin D deficiency [10], and high rates of inadequate immunity to vaccine-preventable illnesses [11].

14.10 Surgical Treatment of Injuries

Musculoskeletal injuries treated with surgical intervention are relatively uncommon in the NBA. However, surgical treatment can have a significant impact on the individual athletes, including significant time away from basketball participation and can sometimes be career-altering. The ten most studied surgical procedures in the literature among NBA athletes and their influence on return to play are depicted in Fig. 14.4. Despite being relatively rare procedures, ranging from 0.8 per year for Achilles tendon repair to 2.2 per year for ACL reconstruction, NBA medical staff must be prepared to both

appropriately counsel and optimize rehabilitation strategies for players requiring these interventions.

Important metrics for assessing performance in athletes returning from injury include games played upon return and player efficiency rating (PER): the NBA’s primary, normalized metric for quantifying a player’s combined positive and negative statistics. From the standpoint of PER, the two procedures with the worst outcomes are also the procedures with the lowest return to play percentage: (i) Achilles tendon repair and (ii) knee microfracture (Table 14.4). Players undergoing these procedures on average have a significantly lower PER up to 2 and 3 seasons following return to play, beyond which has yet to be studied [3]. Lumbar discectomy and ACL reconstruction procedures also result in reduced PERs for players returning in their first season following injury; however, by seasons 2 and 3 the athlete’s PERs on average are not statistically different from their PERs in the season preceding their injury. Patellar tendon repair, meniscal repair, hip arthroscopy, foot fracture repair, shoulder surgery, and hand/wrist fracture repair procedures on average do not negatively impact PER in athletes to a significant degree in their first season following injury [3, 12, 13].

Fig. 14.4 Injuries treated with surgical intervention in the NBA as reported in the literature [3, 12, 13]

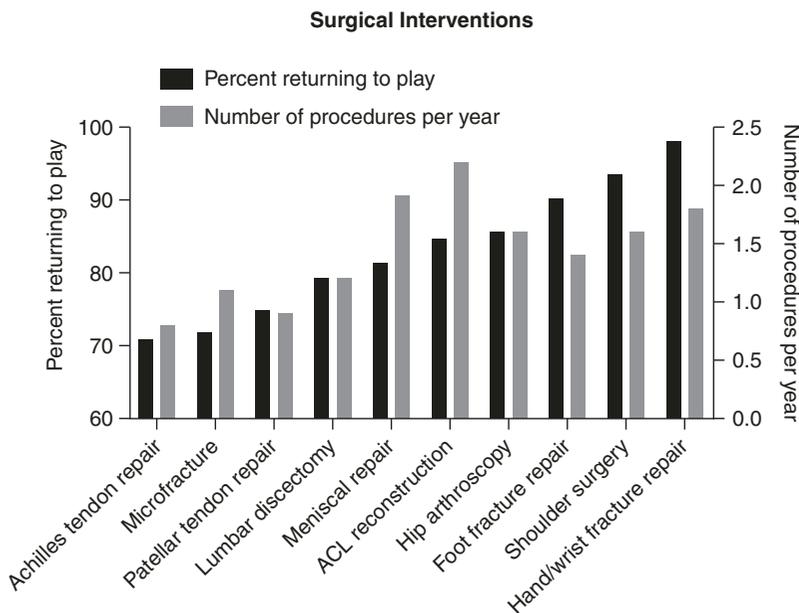


Table 14.4 Percentage of return to play for surgically treated injuries in the NBA as reported in the literature [3, 12, 13]

Surgery	Return to play %	Number of procedures per year	Player efficiency rating (PER) in seasons 2/3 following return ^a	Games played in seasons 2/3 following return ^a
Achilles tendon repair [3]	70.8	0.8	Lower	Lower
Microfracture [3]	71.9	1.1	Lower	Lower
Patellar tendon [12]	75	0.9	No change	No change
Lumbar discectomy [3]	79.4	1.2	No change	No change
Meniscal repair [3]	81.5	1.9	No change	Lower
ACL reconstruction [3]	84.6	2.2	No change	Lower
Hip arthroscopy [13]	85.7	1.6	No change	No change
Foot fracture repair [3]	90.2	1.4	No change	No change
Shoulder surgery [3]	93.5	1.6	No change	No change
Hand/wrist fracture repair [3]	98.1	1.8	No change	No change

^aCompared to their play in the season prior to injury (or in season 2 years prior [12]). Only changes that are statistically significant are noted. (2/3 = season 2 or 3)

Besides PER, games played upon return from injury is another indicator of the injury/procedure burden. Players who undergo Achilles tendon repair, microfracture, meniscal repair, and ACL reconstruction on average play in significantly fewer games up to 2–3 seasons after return when compared to the season prior to injury [3]. For patellar tendon repair, lumbar discectomy, and foot fracture repair, games played rises after an initial decrease of this parameter in the first season of return to play such that, ultimately, there is no significant decrease in games played beyond 2 or 3 years following these procedures [3, 12]. Players undergoing hip arthroscopy, shoulder surgery, and hand/wrist fracture repairs have been noted to not play in significantly fewer games in their first season back from injury compared to games played prior to injury [3, 13].

14.11 Summary

Medical coverage in the NBA involves a multi-disciplinary team. Successful execution of medical care in this environment requires medical providers to incorporate the needs of the organization (NBA), the franchise ownership, the players association, the coaches, as well as the

individual players in order to accomplish the common goal of optimizing the health and performance of all NBA athletes.

References

1. Mack CD, Meisel P, Herzog MM, Callahan L, Oakkar EE, Walden T, et al. The establishment and refinement of the national basketball association player injury and illness database. *J Athl Train.* 2019;54(5):466–71.
2. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the National Basketball Association: a 17-year overview. *Sports Health.* 2010;2(4):284–90.
3. Minhas SV, Kester BS, Larkin KE, Hsu WK. The effect of an orthopaedic surgical procedure in the National Basketball Association. *Am J Sports Med.* 2015;44(4):1056–61.
4. Starkey C. Injuries and illnesses in the National Basketball Association: a 10-year perspective. *J Athl Train.* 2000;35(2):161–7.
5. Padaki AS, Cole BJ, Ahmad CS. Concussion incidence and return-to-play time in National Basketball Association players: results from 2006 to 2014. *Am J Sports Med.* 2016;44(9):2263–8.
6. Yengo-Kahn AM, Zuckerman SL, Stotts J, Zalneraitis BH, Gardner RM, Kerr ZY, et al. Performance following a first professional concussion among National Basketball Association players. *Phys Sportsmed.* [Internet]. 2016;44(3):297–303. <https://doi.org/10.1080/00913847.2016.1200956>.
7. Desai R, Yen C, Wikswo M, Gregoricus NA, Provo JE, Parashar UD, et al. Transmission of norovirus

- among NBA players and staff, winter 2010-2011. *Clin Infect Dis*. 2011;53(11):1115–7.
8. Osterberg KL, Horswill CA, Baker LB. Pregame urine specific gravity and fluid intake by national basketball association players during competition. *J Athl Train*. 2009;44(1):53–7.
 9. Zigelbaum BM, Starkey C, Hersh PS, Donnenfeld ED, Perry HD, Jeffers JB. The National Basketball Association eye injury study. *Arch Ophthalmol*. 1995;113:749–52.
 10. Grieshaber JA, Mehran N, Photopolous C, Fishman M, Lombardo SJ, Kharrazi FD. Vitamin D insufficiency among professional basketball players: a relationship to fracture risk and athletic performance. *Orthop J Sport Med*. 2018;6(5):1–5.
 11. Conway JJ, Toresdahl BG, Ling DI, Boniquit NT, Callahan LR, Kinderknecht JJ. Prevalence of inadequate immunity to measles, mumps, rubella, and varicella in MLB and NBA athletes. *Sports Health*. 2018;10(5):406–11.
 12. Nguyen MV, Nguyen JV, Taormina DP, Pham H, Alaia MJ. A comprehensive return-to-play analysis of national basketball association players with operative patellar tendon tears. *Orthop J Sport Med*. 2018;6(10):1–5.
 13. Schallmo MS, Fitzpatrick TH, Yancey HB, Marquez-Lara A, Luo TD, Stubbs AJ. Return-to-play and performance outcomes of professional athletes in North America after hip arthroscopy from 1999 to 2016. *Am J Sports Med*. 2018;46(8):1959–69.

Part III

On Court Management in Basketball



On Court Examination in Basketball: What the Clinician Should Not Miss

15

Jeremy Truntzer, Marc Safran,
Tahsin Beyzadeoglu, and Geoffrey Abrams

15.1 Introduction

The incidence of basketball injuries has increased recently as the sport has become more physical involving larger, more athletic participants. Multiple studies have reported an incidence of 1.94–9.9 injuries per 1000 player hours (AE) across various levels of play [1, 2]. While the majority of injuries occur to the lower extremities, commonly the head and neck, upper limb, and spine/pelvis can be involved. Concussions and cardiac events also require consideration in basketball players due to the potential for catastrophic outcomes and increased incidence among the basketball demographic. Facial injuries, especially involving the eyes and mouth, can require prompt treatment to improve outcomes. Dislocations of the shoulder and knee should be promptly evaluated and a thorough physical exam performed to help guide primary treatment. Unstable fractures of the hand and wrist similarly warrant early evaluation and immobilization. Some midfoot and forefoot injuries, acute and chronic in etiology, require initial removal from participation and immobilization. This chapter

reviews certain scenarios that mandate on-court examination and provides guidelines for the medical provider.

15.2 Concussions

The physical and vertical nature of basketball makes participants prone to concussions. Rates of concussions range from 0.07 to 0.21 per 1000 athlete exposures depending on level and gender [3, 4]. The mechanism for sustaining a concussion generally includes a direct blow to the head either through contact with an opponent, the ball, or the ground. The diagnosis of concussions is clinical with signs and symptoms of acute neurological dysfunctions, cognitive impairment, and altered mental status. The symptoms can onset rapidly or gradually and resolve in a similar matter. The variation in presentation, as well as the lack of consistent objective diagnostic criteria, makes concussions a challenging diagnosis and therefore requires heightened suspicion. Moreover, making an accurate diagnosis is important to facilitate earlier recovery, reduce risk of early complications, and avoid additional head and musculoskeletal injuries. Given the high implications of appropriately treating a concussion, a conservative management approach is commonly followed which often mandates removal from play to minimize further complications.

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Fact Box

Any player suspected of having sustained a concussion should be removed immediately from competition.

The fundamental starting point for the clinician is to make the diagnosis of a concussion promptly, often requiring on-court evaluation. At all playing levels, a concussion evaluation should be triggered when an athlete is noted to have undergone a forceful impact directly or indirectly to the head associated with: symptom(s) reported by the athlete, visible signs, or any clinical suspicion by the medical staff. The 2017 Berlin Concussion in Sport Group Consensus Statement provided mandatory and discretionary signs of a concussion and appropriate actions tailored to individual sporting federations (Tables 15.1 and 15.2) [5]. It was also noted that a mandatory or discretionary sign in combination with a susceptible mechanism is highly likely to result in the diagnosis of a concussion [6]. Facial lacerations to the eye and bleeding or lacerations to the ear should also initiate concern for a concussion.

When a suspicion of a concussion is present, the athletic trainer or team physician should conduct an initial on-court evaluation. In the case of a collapsed athlete, typically ACLS protocols should be enforced, including evaluation of airway, breathing, and heart function [5]. A physical exam should follow to exclude a cervical spine injury or serious brain injury. Emergency transfer should be initiated with signs of deteriorating mental status or focal neurological findings. Once excluded and the participant is deemed appropriate for further evaluation, a more detailed history and physical examination are necessary. In most cases, the player can be removed from the court for further evaluation.

A number of screens are available for on-site assessment including the Maddocks Questions [7], the Standardized Assessment of Concussion (SAC) [8], the Balance Error Scoring System (BESS) [9], the Sports Concussion Assessment Tool (SCAT5) [10], the King Devick [11], and

Table 15.1 Mandatory signs of a concussion and recommended action

Sign	Action
Loss of consciousness	Remove from field of play. In some sports, the athlete may not return to the game once removed for a mandatory sign. When permanent removal is not required, a mandatory assessment should be conducted in a distraction-free environment to determine return to play.
Lying motionless for >5 seconds	
Confusion/disorientation	
Amnesia	
Vacant look	
Motor incoordination	
Tonic posturing	
Impact seizure	
Ataxia	

Table 15.2 Discretionary signs of concussion and appropriate action

Sign	Action
Clutching the head	Further evaluation is required. The athlete should be removed from the playing environment, undergo an evaluation in a distraction-free setting, and only return to play if the signs are determined to have been from a cause other than concussion.
Being slow to get up	
Suspected facial fracture	
Possible ataxia	
Behavioral change	
Other clinical suspicion	

Adapted from Patricios JS, Ardern CL, Hislop MD, Aubry M, Bloomfield P, Broderick C, et al. Implementation of the 2017 Berlin Concussion in Sport Group Consensus Statement in contact and collision sports: a joint position statement from 11 national and international sports organisations. *Br J Sports Med.* 2018;52(10):635–41

The Concussion Recognition Tool (CRT5) [12]. If available, the Sports Concussion Assessment Tool (SCAT5) should be performed in a distraction-free area by a specialist as recommended by most organization. The Concussion Recognition Tool (CRT5) has also been validated to identify situations where athletes should be removed from sport in settings where a specialist is not available [12].

While it is possible to diagnosis a concussion immediately, often the diagnosis may require

follow-up evaluation up to 48 h due to delayed presentation. As such, removal from play is the standard of care when suspicion of a concussion exists. Even in the “stunned athlete,” who has very short lived, temporary symptoms, concern for a concussion should hold the player from competition until appropriate assessment has been completed. At all levels of play, an athlete diagnosed with a concussion should not be permitted to return to sport on the day the concussion is sustained. Subsequent return to play guidelines, treatment, and prevention is outside the scope of this chapter and covered elsewhere.

15.3 Facial Injuries

Basketball participants following trauma to the head and neck leading to injuries sustained to the eye, nose, and mouth may require prompt evaluation and treatment. A basic understanding of facial anatomy is important along with a properly supplied medical kit to aid in diagnosis.

Due to the absence of facial protection in basketball, injuries to the eye rank among the highest of all sports [13]. If ocular trauma is suspected, examination should commence with review of the symptoms the athlete has experienced in order to develop an appropriate differential diagnosis. Physical examination should follow with testing visual acuity one eye at a time. Assessment of the pupils with penlight and extraocular movements is also required [14]. Cotton swabs and eye-irrigating fluids should be readily available to remove a foreign body. Certain ocular symptoms warrant urgent referral to an ophthalmologist for further evaluation (Table 15.3) [15].

Similarly, if an open globe injury is suspected, a shield should be placed over the eye for protection while limiting pressure on the eye. An urgent evaluation by the ophthalmologist is necessary. Closed globe injuries along with orbital fractures should be triaged according to the discretion of the medical care provider based on the symptoms encountered in a manner similar to Table 15.3. For example, orbital fractures present with varying symptoms including mild pain to reduced vision, diplopia, and loss of extraocular

Table 15.3 Ocular symptoms in athletes which warrant an urgent referral to an ophthalmologist [15]

Symptom	Potential problem
Loss of vision	Various (nonspecific complaint)
Diplopia	Extraocular muscle in orbital fracture Cranial nerve injury
Photophobia	Anterior chamber inflammation (traumatic iritis, microhyphema)
Flashes +/- floaters	Vitreous detachment Retinal tear
Visual field defect	Retinal detachment
Ocular pain with foreign-body sensation	Corneal abrasion
Ocular pain with nausea/vomiting	Increased intraocular pressure

movement. Therefore, not all orbital fractures warrant urgent referral.

Dental and orofacial injuries must also be considered in basketball players [16]. Tooth fractures that only involve the enamel are usually less severe compared to more severe injuries that include the dentin or pulp (nerve). In the case of the former, return to play is generally appropriate. In the case of tooth injury with nerve exposed, the patient should be removed from competition and referred for treatment, given the higher risk for infection. In all cases, it is important to locate all fracture fragments and place in a moist semi-sterile solution for possible reattachment. Following tooth subluxation, return to play is at the discretion of the medical provider, whom may consider use of a mouth guard, especially in contact sports. When luxation of the tooth occurs, the direction of displacement is important [17]. Extrusive luxation can be handled on the sidelines by gently pushing the teeth back into the socket. However, if the tooth can be saved, it should be splinted by a dentist in the correct position. Lateral and intrusive luxation often requires application of local anesthetic or surgical reduction followed by splinting by a specialist. A dental avulsion is the complete traumatic displacement of a tooth. Ideally the tooth is reimplanted within 5 min [18]. The avulsed tooth should be handled by the crown only and quickly rinsed in cold water, saline, or milk prior to

re-implantation. The tooth should not be replanted in a child (with deciduous teeth) or unconscious individual [16]. After replantation, urgent referral to a dentist is encouraged.

Fact Box

Exposure of the pulp (nerve) following a tooth fracture warrants removal from play and prompts treatment by a specialist, given higher risk of infection.

Maxillary and mandibular fractures are relatively uncommon in basketball, but occasionally require urgent management. In some instances, a maxillary fracture can drop posteriorly potentially closing the airway [16]. In this case, a reduction in the maxilla anteriorly is necessary to open the airway. When a mandibular fracture is suspected, proper radiographic imaging is needed and the player should be removed from competition.

15.4 Cardiac

Although relatively uncommon among elite-level athletes, sudden cardiac death (SCD) is a tragic event. Unfortunately, the highest mortality rates observed in NCAA University athletes in the USA were among basketball players [4]. Overall cardiac death was the second most common cause of death, with a rate estimated at 1:43,700 in Division 1 participants, but increases to 1:29,186 in elite athletes. The rate increases to 1:3126 for Division 1 male basketball players. Subsequently, specific screening and protocols have been implemented at higher levels, but medical providers at all levels should be versed regarding on-court management of cardiac events.

Perhaps the most important component of sudden cardiac disease treatment in basketball players is screening and prevention. In most jurisdictions, including high school sports in the USA, a targeted personal history, family history,

and careful physical examination are considered the minimum. The Inter-association Consensus Statement on Cardiovascular Care of College Student-Athletes reported no consensus on universal screening for all athletes with an electrocardiogram (ECG) [19]. Should an athlete test positive for any risk factors, further examination is warranted often including an ECG, echocardiogram, and assessment by a specialist [19]. However, despite no consensus on the benefit of universal ECG testing, among the larger (Power 5) collegiate conferences in the USA, 62% institute routine ECG screening for all athletes, with an increase to 78% for male basketball players [20]. FIBA recommends that all international-level basketball players should undergo the minimal level of testing in addition to a 12 lead ECG, with a similar algorithm for any athlete screening positive. An international summit in 2015, however, noted that in the presence of cardiac symptoms or a family history or inherited cardiovascular disease or premature SCD, a normal ECG should not preclude further assessment [21]. Universal ECG testing has not been adopted secondary to a low specificity with further study necessary [22]. The “Seattle Criteria” was developed to help improve the value of ECG interpretation in the athletic population [23].

Preparedness is also critical to successful management of sudden cardiac events during basketball participation. Site organization including automated external defibrillator (AED) location and emergency protocols should be rehearsed at least annually by the medical team as well as reviewed prior to any participation. Ideally less than 3–5 min will elapse between collapses to shock, if required. Additionally, players at known risk should also be identified, especially at away events.

Fact Box

When a sudden cardiac arrest is suspected, rapid initiation of CPR and prompt administration of an AED greatly improves the chances of survival.

A sudden cardiac arrest (SCA) should be suspected in any athlete that has collapsed and is unresponsive [19]. High suspicion is also warranted in anyone that sustains a non-traumatic collapse. Other signs of SCA include brief seizure activity following collapse and occasional gasping or agonal respirations [24]. In the event of a cardiac event, the emergency protocol should be instituted. Airway, breathing, and circulation should be immediately assessed. Pending the findings, the appropriate measures should be swiftly initiated [25]. Early CPR can double or triple the chance of survival when administered properly [26]. Prompt administration of AEDs is the greatest factor affecting survival from cardiac arrest. Survival from cardiac arrest declines 7–10% for every minute that defibrillation is delayed [27]. As such, an AED should be applied to any patient following SCA as soon as possible for rhythm analysis and shock, if indicated. Urgent transport to an emergency facility should occur as soon as deemed safe.

15.5 Upper Extremities Injuries

The upper extremity in basketball is relied upon for positioning as well as ball handling and therefore is susceptible to acute injuries. Fractures and dislocations of the upper extremity including the shoulder and fingers are most common. Early evaluation is important for both purposes of diagnosis, treatment, and return to play considerations.

Shoulder dislocations represent up to 1.4% of injuries during basketball participation [1, 2, 28]. In many cases, the shoulder can be reduced on-site, especially in players with a history of prior dislocations. First-time dislocations may require sedation or local anesthetic to aid with reduction. Especially with first-time dislocations imaging, usually radiographs, are necessary to rule out associated fracture, which may delay to return to play. In all cases, a thorough neurovascular assessment pre- and postreduction is required to evaluate for concurrent nerve or vascular injury. Unique dislocation patterns should also be considered, given the requirement for alterations in reduction technique. For example, Luxatio Erecta

or inferior shoulder dislocations require extension of the arm at the elbow with overhead traction in the longitudinal direction of the humerus while an assistant may also apply cephalad pressure over the humeral head to help guide it into the joint [29, 30]. Inferior shoulder dislocations are associated with higher rates of brachial plexus and neurovascular injury and should be considered when the patient has extreme discomfort with adduction of the shoulder and, on occasion, the examiner may be able to palpate the humeral head in the axilla.

Hand injuries are common basketball injuries, with the majority involving sprains and ligaments. Severe injuries include fractures and dislocations. Timely and correct evaluation of finger injuries is the priority for the medical staff with some injury patterns requiring special consideration. Mallet fingers should be promptly splinted in extension to reduce long-term complications [31]. Dislocations of the MCP, PIP, or DIP should be reduced as soon as possible prior to increased swelling, if tolerated [32]. All dislocations require follow-up imaging to prevent missed fractures with the potential for long-term disability. Persistent instability or crepitus warrants further evaluation, with return to play postponed until the evaluation is completed. Scaphoid fractures, commonly associated with a fall directly onto an outstretched hand, warrants heightened suspicion, given the long-term sequelae [33]. Tenderness over the snuffbox, volar scaphoid tubercle, and scaphotrapezial joint are sensitive physical exam findings. Immobilization should be initiated until satisfactory imaging is obtained in cases of reasonable concern.

15.6 Lower Extremity Injuries

Fact Box

Knee dislocations are associated with catastrophic outcomes, especially if a concurrent vascular injury is missed. Prompt assessment of neurovascular status is critical if a knee dislocation is suspected.

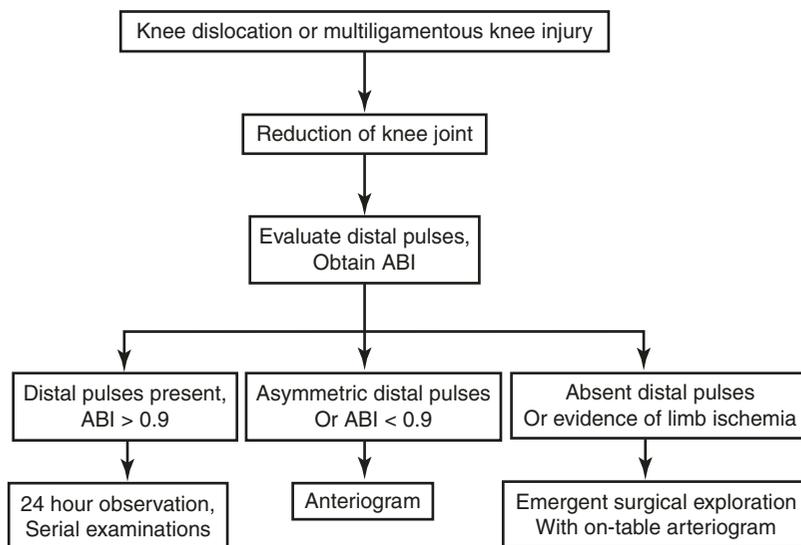


Fig. 15.1 Management of knee dislocation with concurrent vascular injury. Adapted from Nicandri GT, Chamberlain AM, Wahl CJ: Practical management of

knee dislocations: A selective angiography protocol to detect limb-threatening vascular injuries. *Clin J Sport Med* 2009;19(2):125–129

Knee and lower extremity injuries are common in basketball, given the cutting, jumping, and sudden changes of direction required to participate. While ankle sprains are the most common injury encountered among basketball players, knee injuries account for the most time lost and most likely to require surgery. Foot injuries also have a high incidence among basketball players with certain injuries associated with catastrophic outcomes.

Among knee injuries in basketball players, knee dislocations require the most urgent evaluation and decision-making. A knee dislocation by definition is the disruption of the tibiofemoral articulation and can occur in any direction typically involving at least two knee ligaments [34]. Multiple classification systems are used to describe the pattern and severity. Despite the association with high energy mechanisms, approximately 50% of knee dislocations reduce spontaneously before a formal evaluation [35]. When a knee dislocation is suspected, a thorough evaluation is required on-site. Deformity of the knee along with sensory and vascular deficits must be assessed. Knee reduction should be attempted on the court that reverses the deformity; however, occasionally conscious sedation is required after immobilization and removal from the playing area. Repeat examination of the lower

leg should be performed after reduction. If successful, the knee should be splinted between full extension and 20° of flexion. Even in the presence of robust, symmetrical pulses, an ankle brachial index (ABI) is necessary [36]. An abnormal ABI (<0.9) or diminished/absent pulses postreduction warrants further investigation [37, 38]. Normal pulses with an ABI >0.9 still requires at least 24 h of observation based on most recommendations (Fig. 15.1). Treatment of a knee dislocation following initial stabilization and assessment is complicated and outside the scope of this chapter.

Injuries to the knee that also warrant consideration during on-court evaluation include ACL injuries, meniscus tears, and patella tendon ruptures. In all cases, immediate return to play may expose the player to further injury. A brief physical exam in combination with the mechanism of injury is often sufficient to recognize these injuries and determine if the player can safely return to play. Studies report high accuracy with specific physical exam maneuvers for the diagnosis of ACL ruptures and meniscus tears, which also often present with knee swelling and frequently loss of range of motion if a large meniscus tear is present [39, 40]. A defect in the patella tendon, patella alta, or inability/difficulty with straight leg raise should increase suspicion for a patella tendon injury [41, 42].

Additional lower extremity injuries mandate special consideration, given increased incidence among basketball players and carry catastrophic outcomes if missed. Regardless of acute stress-related etiology, talus [43], navicular, and proximal fifth metatarsal fractures (Jones fractures) [44] are associated with poor healing. While early recognition and immobilization are paramount for the above fractures, evidence also supports the role of surgical intervention to improve return to play. Sesamoid stress injuries or fractures also require increased awareness in basketball players, given the higher rate of occurrence and potential for missed diagnosis [45–47]. Any basketball player with concern for an Achilles injury, acute or chronic, also necessitates a complete examination. Complete tears require removal from participation. Early recognition in the case of a partial tear can mitigate the risk of tear completion and significantly reduce the time lost to injury [48].

15.7 Conclusion

The rate of basketball injuries is on the rise with a number of injury patterns that require increased awareness and on-court examination to reduce the probability of a catastrophic outcome. Concussions and sudden cardiac arrest are concerning among all athletes, but occur at higher rates in the basketball population. Facial trauma can necessitate urgent treatment in order to improve outcomes. Certain upper extremity injuries, especially unstable dislocations, may require removal from play and longer absence from play. Knee dislocations, if missed, can be limb-threatening and require thorough initial evaluation. Injuries to the foot and ankle with poorer healing potential also warrant increased awareness. Fortunately, despite a number of injuries that mandate on-court evaluation, most injuries sustained in basketball can be handled off the court.

References

1. Borowski LA, Yard EE, Fields SK, Comstock RD. The epidemiology of US high school basketball injuries, 2005–2007. *Am J Sports Med.* 2008;36(12):2328–35.
2. Dick R, Hertel J, Agel J, Grossman J, Marshall SW. Descriptive epidemiology of collegiate men's

basketball injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. *J Athl Train.* 2007;42(2):194–201.

3. Harmon KG, Clugston JR, Dec K, Hainline B, Herring SA, Kane S, et al. American Medical Society for Sports Medicine position statement on concussion in sport. *Clin J Sport Med.* 2019;29(2):87–100.
4. Harmon KG, Drezner J, Gammons M, Guskiewicz K, Halstead M, Herring S, et al. American Medical Society for Sports Medicine position statement: concussion in sport. *Clin J Sport Med.* 2013;23(1):1–18.
5. Patricios JS, Ardern CL, Hislop MD, Aubry M, Bloomfield P, Broderick C, et al. Implementation of the 2017 Berlin Concussion in Sport Group Consensus Statement in contact and collision sports: a joint position statement from 11 national and international sports organisations. *Br J Sports Med.* 2018;52(10):635–41.
6. Bruce JM, Echemendia RJ, Meeuwisse W, Hutchison MG, Aubry M, Comper P. Development of a risk prediction model among professional hockey players with visible signs of concussion. *Br J Sports Med.* 2018;52(17):1143–8.
7. Maddocks DL, Dicker GD, Saling MM. The assessment of orientation following concussion in athletes. *Clin J Sport Med.* 1995;5(1):32–5.
8. Grubenhoff JA, Kirkwood M, Gao D, Deakynne S, Wathen J. Evaluation of the standardized assessment of concussion in a pediatric emergency department. *Pediatrics.* 2010;126(4):688–95.
9. Mathiasen R, Hogrefe C, Harland K, Peterson A, Smoot MK. Longitudinal improvement in balance error scoring system scores among NCAA division-I football athletes. *J Neurotrauma.* 2018;35(4):691–4.
10. Echemendia RJ, Meeuwisse W, McCrory P, Davis GA, Putukian M, Leddy J, et al. The sport concussion assessment tool 5th edition (SCAT5): background and rationale. *Br J Sports Med.* 2017;51(11):848–50.
11. Dhawan PS, Leong D, Tapsell L, Starling AJ, Galetta SL, Balcer LJ, et al. King-Devick test identifies real-time concussion and asymptomatic concussion in youth athletes. *Neurol Clin Pract.* 2017;7(6):464–73.
12. Echemendia RJ, Meeuwisse W, McCrory P, Davis GA, Putukian M, Leddy J, et al. The concussion recognition tool 5th edition (CRT5): background and rationale. *Br J Sports Med.* 2017;51(11):870–1.
13. Boden BP, Pierpoint LA, Boden RG, Comstock RD, Kerr ZY. Eye injuries in high school and collegiate athletes. *Sports Health.* 2017;9(5):444–9.
14. Rodriguez JO, Lavina AM, Agarwal A. Prevention and treatment of common eye injuries in sports. *Am Fam Physician.* 2003;67(7):1481–8.
15. Micieli JA, Easterbrook M. Eye and orbital injuries in sports. *Clin Sports Med.* 2017;36(2):299–314.
16. Piccininni P, Clough A, Padilla R, Piccininni G. Dental and orofacial injuries. *Clin Sports Med.* 2017;36(2):369–405.
17. Diangelis AJ, Andreasen JO, Ebeleseder KA, Kenny DJ, Trope M, Sigurdsson A, et al. Guidelines for the management of traumatic dental injuries: 1. Fractures and luxations of permanent teeth. *Pediatr Dent.* 2017;39(6):401–11.

18. Kenny DJ, Barrett EJ, Casas MJ. Avulsions and intrusions: the controversial displacement injuries. *J Can Dent Assoc.* 2003;69(5):308–13.
19. Hainline B, Drezner JA, Baggish A, Harmon KG, Emery MS, Myerburg RJ, et al. Interassociation consensus statement on cardiovascular care of college student-athletes. *J Am Coll Cardiol.* 2016;67(25):2981–95.
20. Miars CW, Stamatis A, Morgan GB, Drezner JA. Cardiovascular screening practices and attitudes from the NCAA autonomous “power” 5 conferences. *Sports Health.* 2018;10(6):547–51.
21. Sharma S, Drezner JA, Baggish A, Papadakis M, Wilson MG, Prutkin JM, et al. International recommendations for electrocardiographic interpretation in athletes. *Eur Heart J.* 2018;39(16):1466–80.
22. Asplund CA, O’Connor FG. The evidence against cardiac screening using electrocardiogram in athletes. *Curr Sports Med Rep.* 2016;15(2):81–5.
23. Drezner JA, Ackerman MJ, Anderson J, Ashley E, Asplund CA, Baggish AL, et al. Electrocardiographic interpretation in athletes: the ‘Seattle criteria’. *Br J Sports Med.* 2013;47(3):122–4.
24. Drezner JA, Rao AL, Heistand J, Bloomingdale MK, Harmon KG. Effectiveness of emergency response planning for sudden cardiac arrest in United States high schools with automated external defibrillators. *Circulation.* 2009;120(6):518–25.
25. Travers AH, Perkins GD, Berg RA, Castren M, Considine J, Escalante R, et al. Part 3: adult basic life support and automated external defibrillation: 2015 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. *Circulation.* 2015;132(16 Suppl 1):S51–83.
26. Casa DJ, Guskiewicz KM, Anderson SA, Courson RW, Heck JF, Jimenez CC, et al. National athletic trainers’ association position statement: preventing sudden death in sports. *J Athl Train.* 2012;47(1):96–118.
27. Larsen MP, Eisenberg MS, Cummins RO, Hallstrom AP. Predicting survival from out-of-hospital cardiac arrest: a graphic model. *Ann Emerg Med.* 1993;22(11):1652–8.
28. Agel J, Olson DE, Dick R, Arendt EA, Marshall SW, Sikka RS. Descriptive epidemiology of collegiate women’s basketball injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. *J Athl Train.* 2007;42(2):202–10.
29. Sileo MJ, Joseph S, Nelson CO, Botts JD, Penna J. Management of acute glenohumeral dislocations. *Am J Orthop (Belle Mead NJ).* 2009;38(6):282–90.
30. Kammel KR, Leber EH. Inferior shoulder dislocations (Luxatio Erecta). Treasure Island, FL: StatPearls; 2019.
31. Alla SR, Deal ND, Dempsey IJ. Current concepts: mallet finger. *Hand (N Y).* 2014;9(2):138–44.
32. Morgan WJ, Slowman LS. Acute hand and wrist injuries in athletes: evaluation and management. *J Am Acad Orthop Surg.* 2001;9(6):389–400.
33. Winston MJ, Weiland AJ. Scaphoid fractures in the athlete. *Curr Rev Musculoskelet Med.* 2017;10(1):38–44.
34. Boyce RH, Singh K, Obremskey WT. Acute management of traumatic knee dislocations for the generalist. *J Am Acad Orthop Surg.* 2015;23(12):761–8.
35. Seroyer ST, Musahl V, Harner CD. Management of the acute knee dislocation: the Pittsburgh experience. *Injury.* 2008;39(7):710–8.
36. McCutchan JD, Gillham NR. Injury to the popliteal artery associated with dislocation of the knee: palpable distal pulses do not negate the requirement for arteriography. *Injury.* 1989;20(5):307–10.
37. Nicandri GT, Chamberlain AM, Wahl CJ. Practical management of knee dislocations: a selective angiography protocol to detect limb-threatening vascular injuries. *Clin J Sport Med.* 2009;19(2):125–9.
38. Weinberg DS, Scarcella NR, Napora JK, Vallier HA. Can vascular injury be appropriately assessed with physical examination after knee dislocation? *Clin Orthop Relat Res.* 2016;474(6):1453–8.
39. Kocabey Y, Tetik O, Isbell WM, Atay OA, Johnson DL. The value of clinical examination versus magnetic resonance imaging in the diagnosis of meniscal tears and anterior cruciate ligament rupture. *Arthroscopy.* 2004;20(7):696–700.
40. Leblanc MC, Kowalczyk M, Andruszkiewicz N, Simunovic N, Farrokhyar F, Turnbull TL, et al. Diagnostic accuracy of physical examination for anterior knee instability: a systematic review. *Knee Surg Sports Traumatol Arthrosc.* 2015;23(10):2805–13.
41. Pengas IP, Assiotis A, Khan W, Spalding T. Adult native knee extensor mechanism ruptures. *Injury.* 2016;47(10):2065–70.
42. Lee D, Stinner D, Mir H. Quadriceps and patellar tendon ruptures. *J Knee Surg.* 2013;26(5):301–8.
43. Mayer SW, Joyner PW, Almekinders LC, Parekh SG. Stress fractures of the foot and ankle in athletes. *Sports Health.* 2014;6(6):481–91.
44. Porter DA. Fifth metatarsal Jones fractures in the athlete. *Foot Ankle Int.* 2018;39(2):250–8.
45. Robertson GAJ, Goffin JS, Wood AM. Return to sport following stress fractures of the great toe sesamoids: a systematic review. *Br Med Bull.* 2017;122(1):135–49.
46. York PJ, Wydra FB, Hunt KJ. Injuries to the great toe. *Curr Rev Musculoskelet Med.* 2017;10(1):104–12.
47. McCormick JJ, Anderson RB. The great toe: failed turf toe, chronic turf toe, and complicated sesamoid injuries. *Foot Ankle Clin.* 2009;14(2):135–50.
48. Kadakia AR, Dekker RG 2nd, Ho BS. Acute achilles tendon ruptures: an update on treatment. *J Am Acad Orthop Surg.* 2017;25(1):23–31.



Preparing for Worst-Case Scenarios on Court: How to Best Prepare Your Medical Team

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16.1 Introduction

When an athlete passes out, the team physician must consider both traumatic and medical causes. The priority of the initial assessment should be given to the reversible life-threatening injuries which are damages related to cervical spine, head traumas, airway, or circulation. Head and neck traumas may bring about loss of consciousness. If there is a problem related to the airway and circulation, the athlete may have a limited window of time, as short as 3 or 4 min, before permanent cellular damage occurs. Once threats to cardiorespiratory system are cleared, the athlete is checked for neurological and other life-threatening medical emergencies.

Cardiovascular causes are the most prevalent reason for nontraumatic sudden death in athletes, and they are followed by dehydration, temperature-related illnesses, electrolyte imbalances, and inappropriate drug use. Primary survey must be the first step, which should be done on the field before moving the patient. Using aspects of popular acute cardiac and trauma pro-

ocols, a suggested algorithm is the “C–ABCD” approach to provide a uniform method of examining the downed athlete.

The “C–ABCD” approach stands for cardiopulmonary resuscitation (CPR) and cervical spine (C), airway (A), breathing (B), circulation (C), disability (D) [1, 2].

During the primary survey, the athlete should not be moved instantly if there is no immediate danger, cardiorespiratory emergency, or airway compromise that needs attention. Moving the athlete outside of the field should be done after the completion of each step of the algorithm. In order to avoid unwanted direct contact with body secretions and fluids, medical team should wear gloves while performing intervention.

16.2 CPR (Cardiopulmonary Resuscitation)

In case of encountering a passed-out athlete, the most abruptly developing life-threatening condition is cardiac arrest. Unresponsiveness and irregular breathing may be signs of cardiac arrest. In case of a cardiac arrest caused by sudden arrhythmia, myocardial infarction, shock, or blunt trauma to the chest, CPR with chest compressions should be immediately started without assessing the pulse. CPR can rarely cause harm in individuals who has no cardiac arrest, so it should be started even in a case cardiac arrest is

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Fig. 16.1 Portable defibrillator

assumed [3–5]. Every medical team should have electronic defibrillator for CPR on the field and batteries of defibrillator should be checked before every play and training (Fig. 16.1).

16.3 Cervical Spine Precautions

Immobilization of the cervical spine is the first step intervention of cervical traumas because mishandling of an unstable cervical spine may result with spinal cord injury. In case of accidents that may bring about possible harm to neck or a patient complain of persistent neck pain, a serious injury to the cervical spine must be presumed. After the cardiopulmonary emergency is excluded, the athlete's neck should be preserved with in-line manual support and moved cautiously until neck injury is successfully ruled out.

Immobilization of the neck while other steps of resuscitation are being performed is the responsibility of medical team. Extension or flexion of the neck should be avoided, and in-line



Fig. 16.2 Rigid collar brace application

immobilization of the neck should be maintained until the time that cervical spine is secured and stabilized with a rigid cervical spine collar (Fig. 16.2).

If the athlete is unconscious, the cervical spine protection is crucial, and it should be maintained throughout the primary survey. Holding the head and providing in-line manual immobilization routinely protect the neck. In this maneuver, one person holds each side of the athlete's head to keep the head and neck still. Another preferred way to stabilize the cervical spine is putting a hand on top of each shoulder and stabilizing the head of the player with forearms [5, 6]. Traction should be avoided (Fig. 16.3).

If there is an emergency condition which requires the athlete to be turned over from a prone position, at least four people should collaborate to perform a log-roll maneuver, and if changing position of the athlete is unneeded, log-roll maneuver should not be performed. Assisting three people should be positioned on one side of the player, while the leader provides in-line immobilization to the head and coordinate the timing of the roll with "count of 3" toward the assistants (Fig. 16.4). *The team leader must make sure hands of the athlete are crossed before log-rolling the patient, so that they are in the proper position when the patient is turned on to a supine position. Depending on additional injuries need to be addressed, the athlete may be moved afterwards onto a spine board before transfer [7].*



Fig. 16.3 Management of collapsed athlete



Fig. 16.4 The leader coordinates the roll of the athlete on the “count of 3” toward the assistants

16.4 A—Airway

Airway and ventilation examinations are next to be prioritized in approaching to a collapsed athlete. The medical team should always give a point to respiratory compromise in the event of trauma around the head and neck and when an athlete collapses, the tongue falling back is a very common cause of airway obstruction.

When assessing an injured basketball player, beginning with asking to the athlete what was his/her name is simple and effective way to find

out whether the airway and ventilation are clear or not. *Abnormal breathing sounds, such as stridor, gurgling, whistling, and gasping sounds indicate partial airway obstruction.*

Intercostal retraction, usage of accessory muscles, agitation, and altered level of consciousness are some indicators of severe airway obstruction. Cyanosis signs inadequate oxygenation and may be seen as purplish discoloration over the nail beds and around the lips, but it should be kept in mind that cyanosis is harder to distinguish in athletes with dark skin.

In case of failure to recognize adequate airway, to re-establish secure airway is the most important concern. The first steps are to open the mouth and make sure there is no foreign objects through the airway and that the tongue is not falling backwards occluding the airway. To perform the chin lift, place an index finger under the athlete's mandible and gently pull the lower lip down with your thumb. It is important for a possible cervical spine injury, not to hyperextend or flex the neck when performing the chin lift. In cases of compromised airway by the tongue, the tongue will often fall forward with the chin lift and relieve the obstruction [7].

The airway is placed by positioning the concavity upwards toward the roof of the mouth (upside down) in order to assist the ventilation. The airway inserted along the roof of the hard palate until the soft palate is reached (Fig. 16.5). The device is simultaneously and delicately slid in and rotated 180° so that the concavity is pointing down and positioned over the tongue.

Ventilation with an airway and bag–valve–mask device is possible in most cases and one of the most fundamental skill in basic airway management. *However, if it is not possible to maintain the airway and ventilate the patient, a definitive airway, such as an orotracheal tube, a nasotracheal tube, or a surgical airway (cricothyroidotomy or tracheostomy), is required [7].*

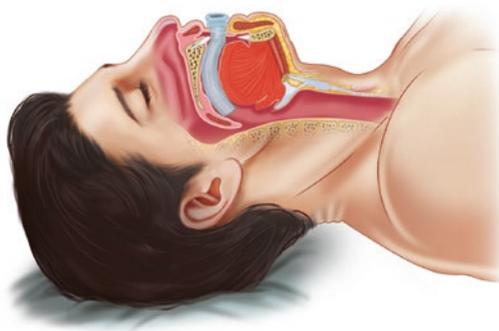


Fig. 16.5 Oropharyngeal airway placement

16.5 B—Breathing

After controlling airway and cervical spine, ventilation should be immediately checked because sufficient oxygenation is necessary to maintain the vital organs. Listening for breath sounds by leaning over mouth of the athlete should be concurrent with watching whether breathing movements of the chest is symmetric or not. Asymmetrical chest movement may point out chest wall injury. In order to be sure about the entry of adequate amount of air with spontaneous breathing or assisted ventilation with a bag–valve–mask device, each lung field should be auscultated with stethoscope [7]. Blood or air in the chest may block transmission of the breath sounds.

In the presence of signs such as difficulty of breathing and decreased breath sounds in auscultation, trachea should be palpated over the anterior neck to see whether it is deviated away to one side of the chest. In case of a deviated trachea, tension pneumothorax should be suspected.

If an athlete is not able to breathe spontaneously, a bag–valve–mask device can be used (Fig. 16.6), and oxygen supplementation can also



Fig. 16.6 Bag–valve–mask device

be administered through the mask. Bag–valve–mask ventilation is performed with one or two people. An appropriately sized mask should cover athlete’s mouth and nose without extending over the chin when it is placed over. If only one person is available, the dominant hand should hold the mask, and the nondominant hand is held over the top of the mask, create a C shape with index finger looking upwards and thumb looking downwards used to ventilate the athlete by squeezing the bag device. Use the remaining fingers of nondominant hand to gently draw the mandible upward and through the mask in order to maintain a good seal between the mask and the athlete’s face, apply pressure by keeping the mask in place (Fig. 16.7). Ventilation rate should be one respiration per every 5 s, or two respirations per every 30 chest compressions if they are also being performed.

Another important indicator of sufficiency of ventilation and patient airway is the blood oxy-



Fig. 16.7 Thumb and index finger and draw the mandible forward by placing the remaining fingers under the angle of the jaw during bag–valve–mask device application

gen saturation and peripheral perfusion. The oxygen saturation and peripheral perfusion can be assessed by pulse oximetry. Finger, toe, or ear-lobe can be used for placement of sensors. Values about 100% are normal, but values which get close or below 92% are worrisome [7].

16.6 C—Circulation

Assessment of the pulse is not one of the first things to do in primary survey because chest compressions should not be delayed, and pulse identification during chest compression is difficult even for an experienced professional. Palpating the carotid artery just under the angle of jaw to assess the cardiovascular system is very reasonable because it can be felt more easily than other peripheral pulses like radial or femoral at lower blood pressures.

If the carotid pulse is absent on each side, and if the athlete demonstrates other signs of shock, cardiac arrest or arrhythmia, CPR has to be initiated immediately [7].

If there is a pulse, physician should assess the hearth rate and rhythm. The rhythm should be regular; every beat should be followed by next beat in rhythm with no skipped or extra beats. The average resting heart rate for an adult is between 60 and 100 beats per minute (bpm) and for children 140 bpm or higher before 5 years of age and around 120 bpm before puberty. But of course it should be kept in mind that trained athletes may have a lower resting heart rate sometimes as low as 40 bpm and because of this fact tachycardia hearth rate values may be lower for these athletes. Athletes have better compensatory mechanisms for hypovolemic event, so it is even more difficult to recognize imminent shock.

If “there is obvious bleeding, application of pressure dressings on the wounds, elevation of the lower extremities above the level of the heart, if there are no complicating injuries to the lower extremities, should be made. Application of a commercial tourniquet can be applied to reduce blood loss from an injured extremity. Intravenous fluids can be administered as soon as possible. Normal saline (0.9%) is the fluid of choice.

Ringer's lactate is an alternative solution. In cases of shock, 1000–2000 cc of normal saline may be given, and the response assessed. If there is absolutely no response, then non-hemorrhagic causes of shock should be considered. If there is significant hemorrhage, the athlete may require blood transfusion at a suitable medical facility.

16.7 D—Disability

A significant head injury should be suspected if the athlete is unable to answer simple questions coherently, or if there is any visual sign of open, depressed, or basal skull fracture, two or more episodes of vomiting and amnesia greater than 30 min. Serious head and neck injury should always be considered in a confused or unconscious athlete. To make the athlete smell strong, scents should be avoided because it can cause reflexive sudden extension of athlete's head.

Bleeding from the ears, nose, or mouth, or bruising around the eyes or base of the skull, suggest a skull fracture and an underlying closed head injury. If the athlete does exhibit any sign of head or neck trauma, athlete should be transferred to a hospital for definitive medical care.

Quick examination of pupils by penlight, asking the athlete a few questions, and assessment of movement in each extremity give much about athlete's neurological status. During the eye examination, physician should look for spontaneous eye movement, size of the pupils, reactivity of the pupils to light, and symmetry of extra ocular movements. Both pupils should react symmetrically to light. Dilation or poor light response of one pupil strongly indicates an intracranial injury which is usually placed on the same side with the enlarged pupil. Abnormal extra ocular movements may indicate cranial nerve palsy and/or additional intracranial injuries. Motor function can be quickly evaluated by asking the athlete to squeeze the physician's fingers and move the toes, owing to this examination physician can also check the athlete's ability to follow commands [7].

By briefly performing eye, verbal, and motor testing in accordance with Glasgow coma scale,

severity of head injuries may be estimated (Table 16.1). If physician consider necessary, the player should be transferred to a hospital (Table 16.2). *The extremities should be quickly examined for fractures, dislocations, or wounds: look for areas of bleeding, swelling, and/or deformity. By palpating and log rolling each limb, physician can easily screen for obvious injury.* In case of injury to an extremity, one of the most important things is to make sure that the athlete's neurovascular status is intact by checking sensation to light touch and pulses distal to the injury.

Prefabricated or vacuum splints can be used to immobilize the fracture until they can be properly evaluated and treated. If fracture causes serious neurovascular abnormality and transfer of the athlete is going to be prolonged, reduction of the fracture may be attempted by an experienced physician. *Alternatively, the injured limb may simply be splinted in a position that decreases tension on the compromised vessels and/or nerves.* If there is an obvious dislocation, an experienced physician can try to relocate the joint on the field, but in case he/she fails, the extremity should be immobilized in a comfortable position as the dislocation may be complicated by a fracture or soft-tissue imposition. If there is a vascular injury, emergent transfer for further evaluation and treatment is strongly required.

Table 16.1 Glasgow coma scale

Eye opening	Grade
Spontaneous	4
To voice	3
To pain	2
None	1
Verbal response	Grade
Oriented	5
Confused	4
Inappropriate words	3
Incomprehensible sounds	2
None	1
Motor response	Grade
Obeys command	6
Localizes pain	5
Withdraws (pain)	4
Flexion (pain)	3
Extension (pain)	2
None	1

Table 16.2 Conditions requiring emergency transfer

Problem	Recommended course of action following “ABCDE” protocol
<i>Airway</i>	
Airway compromise	Immediate transfer (to an appropriate medical facility); provide secure airway before transfer if possible
Anaphylaxis	Administer epinephrine injection intramuscularly; immediate transfer
<i>Breathing</i>	
Respiratory arrest	Start CPR; immediate transfer
Drowning	Start CPR; immediate transfer
Tension pneumothorax	Decompress pneumothorax with large-bore needle in second intercostal space on affected side; chest tube if possible, immediate transfer
<i>Circulation</i>	
Cardiac arrest	Start CPR; defibrillation with AED (Automatic External Defibrillator); immediate transfer
Severe dehydration	Administer fluids; immediate transfer
Internal organ injury (ruptured spleen, liver laceration, fractured kidney)	Administer fluids intravenously; immediate transfer; NPO (Nothing Per Oral); may require surgery
Hidden (internal) bleeding (retroperitoneal bleeding, third space bleeding, pelvic or long-bone fractures)	Administer fluids intravenously; immediate transfer; NPO; may require surgery
Severe bleeding	Control bleeding; administer fluids; immediate transfer
Myocardial infarction	Administer aspirin; immediate transfer
<i>Disability</i>	
Suspected cervical spine injury	Cervical spine immobilization; immediate transfer on spinal board; may require neurosurgical or orthopedic consultation
Any suspected spine injury	Cervical spine immobilization; immediate transfer on spinal board; may require neurosurgical or orthopedic consultation
Loss of consciousness, declining level of consciousness	Cervical spine immobilization; immediate transfer on spinal board; may require CT scan
Confusion, neurological symptoms, worsening headache	Urgent transfer; may require CT scan
Fractures involving skull, pelvis, long bones	Splint the injured extremity; administer fluids if necessary; immediate transfer; NPO; will likely require specialist consultation
Open fracture	Splint the injured extremity with a sterile dressing over the wound; administer fluids if necessary; immediate transfer; NPO; likely requires orthopedic consultation; needs antibiotic coverage
Dislocations	Splint/support the injured extremity; immediate transfer if dislocated; urgent (as soon as possible) transfer if reduced; may require orthopedic consultation
Possible fracture or multiple ligament injury	Splint/support the injured extremity; urgent transfer may require orthopedic consultation
Neurovascular injury in the extremities	Reduce fracture or dislocation; splint/support the injured extremity; urgent transfer; NPO; may require surgical or orthopedic consultation
Acute compartment syndrome (in an extremity)	Splint/support the injured extremity; urgent transfer; NPO; may require orthopedic consultation
<i>Environment/exposure</i>	
Heat illness	Cool patient (water, ice bags); administer fluids; remove excess clothing; immediate transfer
Hypothermia	Warm patient (blankets, dry clothes, use body heat if necessary); remove wet clothing immediate transfer; avoid rapid rewarming if severe hypothermia
<i>Other emergency problems</i>	
Hypoglycemia/diabetic insulin shock	Administer glucose by mouth or intravenously; immediate transfer may require glucagon injection intramuscularly
Hyponatremia	Fluid-restrict athlete; transfer immediately to obtain labs for sodium level and possible hypertonic saline solution

(continued)

Table 16.2 (continued)

Problem	Recommended course of action following “ABCDE” protocol
Seizure	Logroll patient on side of avoid aspiration; immediate transfer, administer benzodiazepine rectally or intravenously (if prolonged seizure of more than 30 min)
Dental injury	Urgent transfer for dental consultation; place any loose teeth or fragments in an appropriate transport medium
Eye injury	Apply eye shield for protection; avoid increasing intraocular pressure; immediate transfer; may require ophthalmology consultation

16.8 First Aid

The team physician should be expert at not only identifying life- or limb-threatening injuries and stabilizing the patient but also administering first aid. The physician should be able to intervene to the minor problems and return the athlete to the competition. In light of this information, the physician should be aware of his/her limitations and be able to decide when to transfer the athlete to a medical center for appropriate investigation and further medical care (Table 16.2).

Skills that are useful on the sidelines include suturing and wound care; taping and splinting joints; applying pressure bandages; applying protective devices such as eye shields and protective padding; and maneuvers such as application of ice and heat, stretching the athlete, and simple massage. It is also useful to comprehend the roles and skills of the other medical team members in order to coordinate them and lead care of the athletes appropriately [7].

16.9 Suturing and Wound Care

The physician should be expert at sterile suture techniques in order to repair simple lacerations because basketball is a game prone to accidents, which precipitate to open wounds. Repairing the laceration requires antiseptic or other cleansing solution, local anesthetic, suture materials, a needle driver, scissors, forceps, and gauze. Sterile adhesive strips or surgical glue can be used to repair small or superficial wounds.

In order to keep the player at the field, the best way to stop bleeding from teeth and lips, and if it

**Fig. 16.8** Taping of the ankle

is not severe, is making pressure on it with gauze, with this choice team doctor could postpone suturing process to the end of the game.

16.10 Athletic Taping

In order to manage and protect soft-tissue and joint injuries, physician should be experienced at basic athletic taping and splinting. Taping can be made of choice in the event of mild sprains, particularly of the ankle, wrist, and fingers, as well as in cases of patellofemoral pain and various foot problems (Fig. 16.8).

16.11 Splinting and Protection

In some cases, rapid immobilization of bone or joint with splint is required. Physician should immobilize the joint distally and proximally by centering the area of injury to prevent movement and then apply the splint.

16.12 Reducing Dislocations

Joint dislocations usually arise from a high-impact trauma. Usually, physician can reduce the dislocation at the field side before pain, muscle spasm, and swelling become too significant. However, attention should be paid to the dislocations because they can have associated neurovascular injuries and require emergency evaluation. Soft-tissue imposition in the joint or by fracture can make the reduction more complex, especially in skeletally immature athletes. That is why, there are limited opportunity to make reduction attempts if success is not achieved initially. The physician should be expert at joint reduction techniques before attempting these maneuvers. The most common joint that dislocates is and more than 90% of shoulder dislocations are anterior. Hip dislocation is an emergency because it can also impair the surrounding vascular structure and can lead to avascular necrosis of the femoral head. Hip fracture usually accompanies (concomitance) fracture of the acetabulum and/or femoral head and usually entails heavy sedation or general anesthesia for reduction at an appropriate facility.

A knee dislocation may be reduced on the field but still because of the possible surrounding neurovascular injury, it needs further evaluation. The patella dislocation usually occurs by a lateral shift of the patella, leaving the trochlea groove of the femoral condyle, often can be reduced on the sideline by extending the knee and manually and force the patella to move into its normal position in the trochlear groove. After reduction, further evaluation should be made to be sure whether there is any associated ligament, neurovascular, or osteochondral injury or not.

The elbow, ankle, or foot may be reduced before urgent transfer to a medical care facility, depending on the training and experience of the physician. Dislocations of the interphalangeal joints of the fingers may be assessed, reduced, and protected with athletic taping or a lightweight aluminum-foam splint. The injured finger can be buddy taped to an adjacent finger, which is used as a natural splint. Return to play

should be decided on an individual basis. A dislocation injury usually comes up with ligament injury and sometimes fractures, and because of this, further evaluation should always be considered, even if the athlete is allowed to return to the game.

16.13 Special Issues

Muscle spasms are one of the worst situations, which an athlete can be faced during game. In this case for example at gastrocnemius and hamstring muscles, fine needling with acupuncture needles to the contacted muscle will relax the muscle and resolve the spasm of the muscle quickly and allows the athlete return to play [8].

Finally, meeting with athletes before travel and competition can make the physician familiar with athletes and their medical situations, and this helps the physician to build a connection with the athletes and foresee any possible special needs or medications. A conscientious, well-prepared team physician can provide a healthy environment, which may increase the confidence of the athletes in their performances and prepared for the worst-case scenarios if they occur [8].

References

1. American College of Sports Medicine Expert Panel. Sideline preparedness for the team physician: consensus statement. *Med Sci Sports Exerc.* 2001;33:846–9.
2. Armstrong LE, Casa DJ, Millard-Stafford M, et al. American College of Sports Medicine position stand. Exertional heat illness during training and competition. *Med Sci Sports Exerc.* 2007;39(3):556–72.
3. Committee on Trauma. Advanced trauma life support for doctors: student course manual, 8th ed. Chicago, IL: American College of Surgeons; 2008.
4. Everline C. Application of an online team physician survey to the consensus statement on sideline preparedness: the medical bag's highly desired items. *Br J Sports Med.* 2011;45(7):559–62.
5. Hazinski MF, Nolan JP, Billi JE, et al. Part 1: executive summary: 2010 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. *Circulation.* 2010;122(16 Suppl. 2):S250–75.

6. Hodge DK, Safran MR. Sideline management of common dislocations. *Curr Sports Med Rep.* 2002;1:149–55.
7. Swartz EE, Boden BP, Courson RW, et al. National Athletic Trainers' Association position statement: acute management of the cervical spine-injured athlete. *J Athl Train.* 2009;44(3):306–31.
8. Team Physician Manual. International Federation of Sports Medicine (FIMS). Taylor and Francis. Kindle Edition; 2012.



Cardiopulmonary Resuscitation and Cardiorespiratory Arrest in Basketball: Guidelines

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17.1 Introduction

The American Heart Association (AHA) published updated cardiopulmonary resuscitation (CPR) guidelines for life support strategy and emergency cardiovascular care in 2015, including revisions on systems of care out of the hospital, in the hospital, and in post-resuscitation settings. The current guidelines emphasize the importance of continuing rescuer education on improving CPR techniques. During the past decade, CPR techniques have been organized as out-of-hospital cardiac arrest (OHCA) and in-hospital cardiac arrest (IHCA), and also management strategies based on basic life support (BLS), advanced cardiovascular life support (ALS), and post-resuscitation stabilization care. An optimal and successful CPR strategy requires several main elements, including an emergency event plan, a dedicated team appropriately educated for resuscitation, and early organization for both emergency medical service and equipment, including an automated external defibrillator.

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17.2 Definition

Sudden cardiac death (SDS) is natural death from any cardiovascular or relevant ancillary causes, heralded by abrupt loss of consciousness within 1 h of the onset of an acute change in cardiovascular status [1, 2]. The one-hour definition is important to satisfy clinical, scientific, legal, and social considerations, and defines the interval between the onset of symptoms signaling cardiorespiratory arrest (CA) and the onset of the CA itself. Cardiopulmonary resuscitation associated with CA was defined, approximately 50 years ago, as the administration of breath ventilation via mouth and closed mechanical chest cardiac compressions in pulseless patients. Since then, CPR and management strategies for cardiovascular life support have significantly advanced. Presently, the main CPR program is described as BLS and further as in-hospital CPR that includes an experienced team, invasive procedures, and facilities, termed adult ALS and pediatric ALS [3].

17.3 Epidemiology

It is not easy to interpret epidemiological studies of SCD because there are differences in definition and difficulties in accessing real data determined as pathophysiological mechanisms and in making decisions based on population and individual risks [4]. Also, as in-hospital and

out-of-hospital epidemiology, the process of care, and treatments have substantial differences between chronic disease evolution and transient events, different forms of epidemiological modeling are needed.

Utstein Collaborators identified 584 citations originating from 50 countries and classified as research articles to analyze OHCA and IHCA [5]. They showed that one third of the citations focused on epidemiology and outcome, including 22% ($n = 126$) for OHCA and 7% ($n = 41$) for IHCA. In many studies, OHCA evaluations and recently published data verified that despite the substantial management strategy each aspect has many differences regarding inclusion criteria, definition, code system, and process element [6]. The survival statistics for OHCA remain disappointing, with an estimated 10% overall survival rate [7]. Among the subgroup of 70% of OHCA that occur in the home, survival is 6%.

It is very difficult to estimate the absolute incidence of SCD throughout the world. According to statistics and the United States database, the estimated sudden death incidence in the overall adult population is 0.1–0.2%/year, accounting for approximately 300,000 SCD/year [8]. A better risk analysis of the common population for SCD identifies a high-risk group: thus, the incidence increases, accompanied by a progressive decrease in the total number of events [9]. Successful interventions in larger population subgroups require identification of specific markers to increase our ability to identify patients who are at particularly high risk for a future event. More than half of all SCDs resulting from coronary heart disease are first clinical events [10], and another 20–30% occur among subgroups of patients with known coronary heart disease profiled at relatively low risk for SCD on the basis of current clinically available markers. Sudden cardiac arrest (SCA) and its most common consequence, SCD, constitute major public health problems, accounting for approximately 50% of all cardiovascular deaths [11], with at least 25% being the first symptomatic cardiac events.

17.4 The Risk of Sudden Cardiac Death and Screening

Disturbances and disorders associated with SCD of athletes and the general population include mainly coronary artery diseases (congenital and acquired), hypertrophic cardiomyopathy, arrhythmia (the principal event is ventricular fibrillation), cardiomyopathies, cardiac arrest, heart attack, valvulopathies, myocarditis, pulmonary embolism, stroke, hyponatremic encephalopathy, hyperthermia, rhabdomyolysis, respiratory failure, and pulseless electrical activity, respectively [12]. Multivariate analyses of selected risk factors (e.g., age, diabetes mellitus, systolic blood pressure, heart rate, electrocardiographic abnormalities, vital capacity, relative weight, cigarette consumption, and serum cholesterol level) have determined that about 50% of all SCDs occur among the 10% of the population in the highest risk decile on the basis of multiple risk factors [13]. Approximately 50% of cardiac arrests occur in individuals without known heart disease, but most suffer from concealed ischemic heart disease [14]. The ratio of coronary disease within the conventional risk factors provides the structural basis for 80% of SCDs in the United States, so that the identification of specific clinical markers of risk for SCD as a specific expression of both coronary heart disease and other cardiovascular disorders has been a goal for many years. Pathological and autopsy studies in SCD victims report epidemiological and clinical observations that coronary atherosclerosis is the major predisposing cause. All other causes of SCD collectively account for no more than 15–20% of cases, but they have provided a large base of enlightening pathological data [9] (Table 17.1).

Familial patterns of SCA risk resulting from genetic variations are very important factors for risk scaling. The various genetic associations can be evaluated in four categories [9] (Table 17.2): inherited uncommon primary arrhythmic syndromes, inherited uncommon structural heart disease associated with SCD risk, acquired or induced arrhythmia risk, and common acquired heart disease associated with SCD risk.

Screening the general population for the risk of SCD is of great importance for saving lives, especially in the high-risk group. Careful evaluation for electrocardiographic (ECG) and echocardiographic signs of inheritable arrhythmogenic diseases seems to be an important part of clinical practice and can contribute to the early identifica-

Table 17.1 Risk factors and causes in sudden cardiac death

<i>Coronary artery diseases:</i> Atherosclerosis, congenital abnormalities, embolism, inflammatory diseases, mechanical obstruction, spasm.
<i>Myocardial hypertrophy:</i> Hypertrophic cardiomyopathy, hypertrophy with pulmonary hypertension/systemic arterial hypertension or valvular heart disease.
<i>Myocardial diseases or heart failure:</i> Congestive heart failure, cardiomyopathy (ischemic, valvular, idiopathic dilated, alcoholic, hypertensive, peripartum mechanical complications), myocarditis.
<i>Cardiac valvular diseases</i>
<i>Inflammatory, infiltrative, neoplastic, and degenerative myocardial pathologies</i>
<i>Congenital heart disease:</i> Aortic or pulmonic valve stenosis, septal defects with severe pulmonary hypertension, Fallot pathologies; after corrective surgery for congenital heart disease.
<i>Electrophysiological abnormalities:</i> Conducting system abnormalities (i.e., fibrotic/calcific, Lenegre's disease, Lev disease, Wolff–Parkinson–White syndrome), QT interval abnormalities, Brugada syndrome, ventricular tachycardia, ventricular fibrillation, supraventricular arrhythmia associated with the AV accessory pathway, catecholaminergic malign arrhythmias, genetic disorders of electrical function/conduction system.
<i>Other occasional causes:</i> Commotio cordis, tamponade, severe unusual physical activity, pulmonary embolism, massive thrombosis, aortic dissection, poisoning, drug overdoses.

Table 17.2 Genetic contributors to sudden cardiac death risk

<i>Primary arrhythmia disorders:</i> Genetic long-QT syndrome, short-QT syndrome, Brugada syndrome, catecholaminergic ventricular tachycardia/fibrillation, genetic electrolyte and metabolic arrhythmogenic effects.
<i>Structural cardiac abnormalities:</i> Inherited cardiomyopathies (i.e., hypertrophic, dilated, restrictive), right ventricular dysplasia, genetic predisposition to acquired coronary artery diseases and/or congestive heart failure.

tion of patients at risk for SCD. Although the existing consensus among experts in Europe and the United States supports pre-participation ECG screening systems in athletes [15], recently a study from Israel reported no change in incidence rates of SCD in competitive athletes following implementation of pre-participation ECG screening programs [16]. In reference to the higher risk of arrhythmias and the worsening of structural or genetic diseases in individuals exposed to intense physical exercise [17], existing recommendations for pre-participation screening in athletes support the consensus that clinical evaluation, personal or family history taking, and a baseline ECG should be performed in this population in Europe.

Although there is no strong consensus concerning the benefit of a prescreening program in the common population, screening family members of sudden death victims is strongly recommended for saving other lives. Inheritable arrhythmogenic disorders (especially channelopathies, cardiomyopathies, familial hypercholesterolemia) can be diagnosed in up to 50% of families with a sudden arrhythmic death victim [18]. In such an event, the first-degree relatives of the victim should be informed of the potential risk of similar events for themselves and should undergo cardiac evaluation [19] (Table 17.3).

Table 17.3 Diagnostic workup for the family of SCD victims

Consultation with expert	Available investigation tests
Family member's history/physical examination	<ul style="list-style-type: none"> • Baseline 12-lead ECG/signal-averaged ECG • 24 or 48 h rhythm Holter monitorization • Cardiac stress test • Provocative test with ajmaline/flecainide to evaluate Brugada syndrome • Echocardiography, cardiac CT/MR for cardiovascular imaging • Genetic molecular testing and counselling if needed • Study of genetic mutations related to malign arrhythmias

SCD sudden cardiac death, ECG electrocardiogram, CT computed tomography, MR magnetic resonance

In patients with documented or suspected ventricular arrhythmias who have survived the threat of sudden death, screening must be done very carefully in terms of clinical history, with noninvasive and invasive evaluations. In patients with documented or suspected ventricular arrhythmias who have survived following the sudden death. Symptoms such as chest discomfort, chest pain, dyspnea, and fatigue, may also be related to underlying structural heart diseases and should not be missed. A family history of SCD and drugs used must be questioned, and a positive family history of SCD is one of the strong independent predictors of susceptibility to ventricular arrhythmias and SCD [20].

Noninvasive tests are applicable and have revealed many inherited and acquired cardiac disorders associated with SCD. A standard ECG may reveal channelopathies (QT syndromes, Brugada syndrome, predictors of ventricular arrhythmias), conduction blocks, and ischemic heart disease, cardiomyopathies, and repolarization abnormalities. Late potentials in the signal:noise ratio of a surface ECG indicate regions of abnormal myocardium with slow conduction, a substrate abnormality that may allow for reentrant ventricular tachyarrhythmias to be detected by signal-averaged ECG. Continuous rhythm Holter recording is appropriate to seek for arrhythmia or intermittent electrical conduction disorders. Implantable devices/loop recorders placed subcutaneously continuously monitor the heart rhythm and record events over a timeframe measured in months to years, and these can record patient activation and catch rare short arrhythmia attacks. The treadmill exercise ECG test is most commonly applied to detect silent ischemia in adult patients with arrhythmia. Exercise-induced ventricular arrhythmias may be a sign of coronary ischemia, but unsustained ventricular tachycardia can be recorded in 4% of asymptomatic adults and is not associated with an increased risk of total mortality [4]. Adrenergic-dependent rhythm disturbances (e.g., ventricular tachycardia) may be triggered by exercise testing because of sympathetic system activation, so that it is useful for diagnostic purposes

and evaluating response to therapy. Transthoracic echocardiography is the most commonly used bedside imaging technique; it is readily available and provides accurate diagnosis of myocardial, valvular, and congenital heart disorders associated with SCD [21]. Besides the evaluation of left and right ventricular structures and function, it is indicated in the workup of the patient's risk for SCD relevant to dilated or hypertrophic cardiomyopathies, right ventricular dysplasia or cardiomyopathies, and ischemic left ventricular wall motion abnormalities. Cardiac and vascular computed tomography (CT) imaging may help in the diagnosis of coronary artery disease and vascular and coronary anomalous cardiac and aortic functions. In some cases, silent myocardial ischemia and microvascular coronary disease cannot be detected by using CT, which is more sensitive for the evaluation of epicardial coronary arteries, and these clinical entities can be revealed by using myocardial perfusion single-photon emission CT (SPECT). Recent advances in magnetic resonance (MR) technology provide excellent image resolution to assess cardiac function and all structures of the heart and vasculatures, allowing for certain quantification of chamber volumes, and ventricular functions, mass, and thickness, particularly to evaluate right ventricular dysplasia and restrictions. Invasive coronary angiography is an important diagnostic technique to diagnose coronary artery disease, vascular abnormalities, hemodynamic measurements, and functional coronary flow abnormalities (e.g., coronary spasm, coronary bridge, slow flow phenomenon, coronary shunts, fistulas, Prinzmetal-type coronary spasm) in patients with malignant ventricular arrhythmia or in survivors of SCD. In cases with electrical conduction abnormalities, reentrant triggered atrio/ventricular arrhythmia, all imaging techniques may represent normal cardiovascular structures and functions, and in such cases an invasive electrophysiological study can be performed to document the inducibility of arrhythmia and conduction abnormalities and to assess the risks of SCD. Also, electrophysiological study can be used to determine prognosis and to pro-

vide a therapeutic guide for patients with cardiomyopathies and inherited primary arrhythmia syndromes. In patients with syncope, which creates a risk for SCD, structural and vascular heart disease, ventricular dysfunction, and malign arrhythmias should be evaluated by using imaging techniques, ECG, Holter monitoring, and sometimes electrophysiologi-

cal studies. In the study published by Middlekauff et al., high recurrence and death rates were observed in syncope patients with heart disease and systolic dysfunction even if the electrophysiological study results were negative [22]. The recommended diagnostic workflow for patients who have survived an aborted cardiac arrest is illustrated in Fig. 17.1 [19].

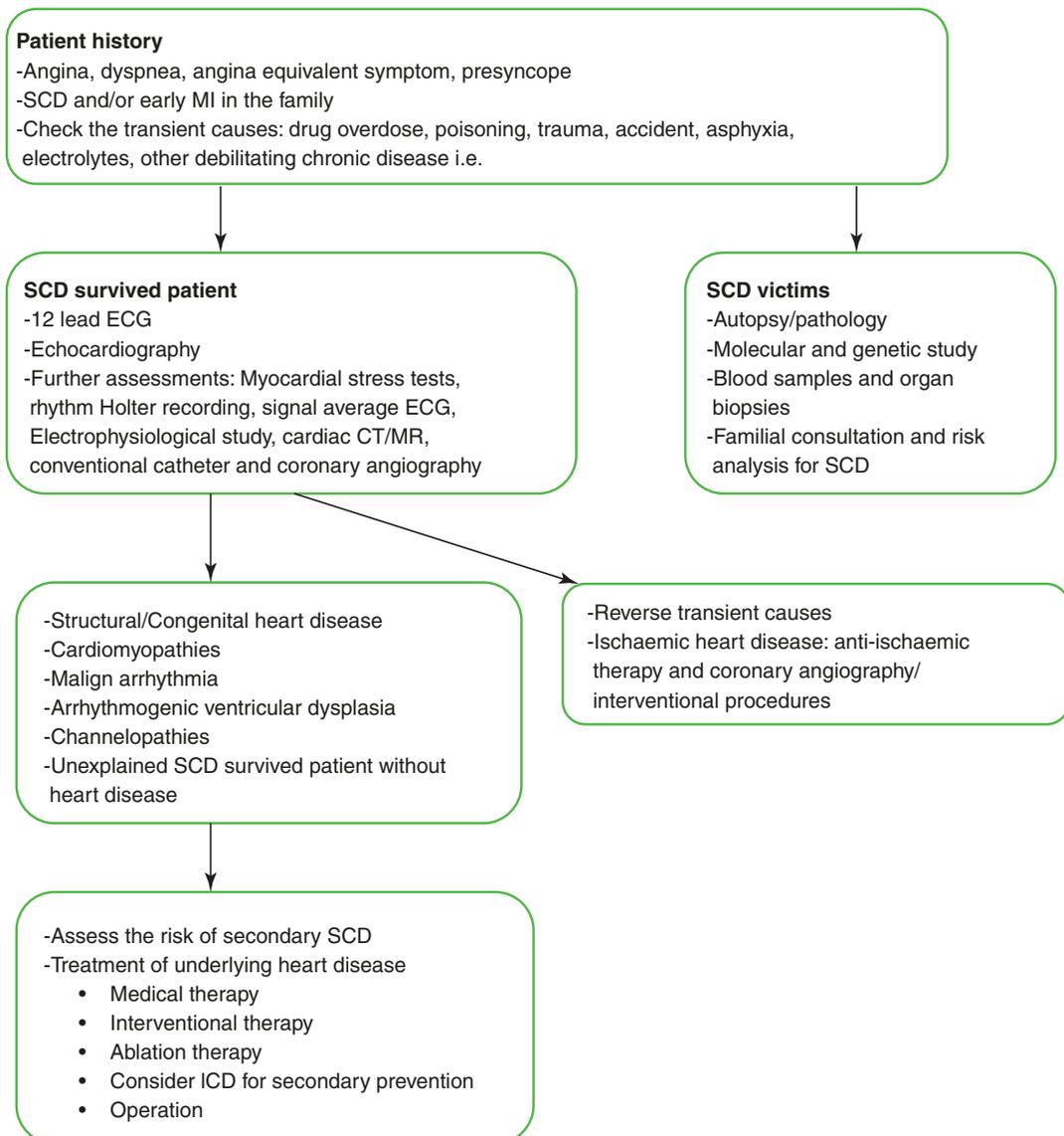


Fig. 17.1 Diagnostic workup in patients with sustained ventricular tachycardia and ventricular fibrillation. *SCD* sudden cardiac death, *MI* myocardial infarction, *CT* com-

puted tomography, *MR* magnetic resonance, *ICD* intracardiac defibrillator

17.5 Consensus Template of CPR

Advances in resuscitation science, new insights into important predictors of outcome from OHCA, and lessons learned from methodological research prompted this review and update of the 2004 Utstein guidelines [19]. Representatives of the International Liaison Committee on Resuscitation (ILCOR) developed an updated Utstein reporting framework iteratively during 2012 through 2014. The original Utstein recommendations focused efforts to report on patients with a nonemergency medical services (non-EMS)-witnessed cardiac arrest of presumed cardiac cause, with ventricular fibrillation at the point of first rhythm analysis. The Utstein 2004 revision broadened this focus to include all EMS-treated [6] cardiac arrests irrespective of first monitored rhythm and whether the arrests were witnessed. In 2004, major changes related to the definition of cardiac arrest (transition from presence/absence of a carotid pulse to signs of circulation), inclusion of defibrillation attempts by bystanders, and extension of the template to include reporting of IHCA in both adults and children were covered in the same template. In 2013, the ILCOR proposed a group form to review and, if necessary, update the Utstein templates for cardiac arrest. A group of studies showed that several core elements have consistently been associated with survival to hospital discharge: witnessed arrest (by a bystander or EMS); bystander CPR; shorter EMS response interval; first shockable rhythm; and return of spontaneous circulation in the field [23]. It is important to recognize of the importance of additional factors associated with the likelihood of survival after OHCA, such as public access defibrillation, dispatcher-assisted CPR, the quality of CPR, post-resuscitation care, variability in “not for resuscitation” order policies and procedures, and accurate prognostication [5]. Implementation, update, and simplification of the ILCOR Utstein templates for cardiac arrest resuscitation audit, registry, and research have been improved since 1990. Still, progress moves on to overcome challenges and stay in the forefront of future implementation, particularly in the balance of

feasibility versus desirability of data elements. In 2010 the Consolidated Standards of Reporting Trials Statement [24] was designed to assist in reporting research by use of a checklist and flow diagram, which facilitates reporting of bystander-witnessed, shockable rhythm as a measure of EMS system efficacy and all EMS-treated arrests as a measure of system effectiveness. The American Heart Association published the “2015 AHA Guidelines Update for Cardiopulmonary Resuscitation and Emergency Cardiovascular Care” including recommendations for adult BLS and CPR quality [25]. That guidelines update was based on the “2015 International Consensus on Cardiopulmonary Resuscitation and Emergency Cardiovascular Care Science with Treatment Recommendations” (CoSTR) developed by the ILCOR [3]. ILCOR is now transitioning to a process of continuous evidence evaluation, with the intent to issue updated systematic reviews and CoSTR statements when prompted by the publication of new evidence. The first topics selected by ILCOR as part of the continuous evidence evaluation process are related to BLS, including dispatch-assisted CPR, the use of continuous versus interrupted chest compressions by EMS providers, and the use of chest compression-only (hands-only) CPR versus CPR using chest compressions with ventilation in both in-hospital and out-of-hospital settings [26]. In 2017 the experts considered and based the CoSTR summary and the ILCOR consensus recommendations in light of the structure and resources of the out-of-hospital and in-hospital resuscitation systems that used AHA guidelines, to plan BLS. Given the advances in understanding of the prognostic determinants of survival in OHCA, the need to revisit and update the 2004 guidelines was evident [27]. According to Utstein consensus, the elements for OHCA were grouped into five domains: system, dispatcher, patients, process, and outcome, and each domain contained core and supplemental elements [5].

The system description defines the characteristics of the population served and the structure of the EMS response. The description includes the number of cases of cardiac arrest attended by

EMS (assessment made by EMS or a bystander), the number of cases for which resuscitation was attempted by EMS, and the reasons why resuscitation was not attempted. A resuscitation attempt is defined as the act of trying to maintain or restore life by establishing and/or maintaining breathing and circulation through CPR, defibrillation, and other related emergency care.

Dispatcher-identified cardiac arrest and dispatcher-assisted CPR have been included as core elements to reflect the impact these processes can have on patient outcome [28]. The system description provides the opportunity to describe operation of the local EMS dispatch. The dispatcher identifies the presence of cardiac arrest before the arrival of EMS and provides telephone CPR instructions to the caller as soon as possible.

Patient variables include patient demographics, comorbidities, pathogenesis, initial presentation, and bystander response. The location of the arrest and whether it was witnessed should be recorded, and cardiac and noncardiac causes of SCD should be separated. Despite all the advances in EMS, CPR studies, and technologies, the separation into cardiac and noncardiac causes is not easy and has proved to be subjective, with some communities reporting the percentage of noncardiac causes of all arrests as only a few percent and others up to 40% [29]. The primary reporting by systems should state the outcomes of all EMS-treated cardiac arrests and those that are bystander witnessed and for which the first monitored rhythm is shockable. Regarding this report, EMS-treated cardiac arrests and the monitored first rhythm represent system effectiveness and efficacy, respectively. Pathogenesis of the SCD should be categorized under the following headings, which also recognize the importance of backward compatibility with existing definitions [5]: medical (presumed cardiac or unknown, other medical causes); traumatic cause; drug overdose; drowning; electrocution; or external asphyxia. If the case has more than one cause of SCD, which is possible (e.g., ventricular fibrillation arrest leading to a fall from a height), the most likely primary cause should be cited.

The first monitored rhythm is the rhythm recorded at the time of first analysis of the monitor or defibrillator after a cardiac arrest. If the automated external defibrillator (AED) does not have a rhythm display, it may be possible to determine the first monitored rhythm from a data storage card, hard drive, or other device used by the AED to record data. If the AED has no data-recording device, the first monitored rhythm should be classified simply as shockable or non-shockable. Bradycardia has been retained as an option to enable appropriate reporting when chest compressions are provided for severe bradycardia with pulses and poor perfusion. When CPR is started because of the absence of signs of circulation despite electrocardiographic evidence of electrical activity (i.e., pulseless electrical activity), it should be recorded as pulseless electrical activity even if the electrocardiographic rhythm is slow. Asystole is defined by a period of at least 6 s without any electrical activity greater than 0.2 mV. In such cases bystander responses are critical to patient outcomes, and all systems should capture the number of cases in which bystander resuscitation is started (chest compressions or standard CPR), whether an AED is deployed, and whether it delivered a shock [5].

The EMS response time and time to first shock are stated in process elements as to whether targeted temperature management was used and whether coronary reperfusion was attempted. Prehospital OHCA process elements include response time to emergency call, defibrillation, time, delivery of any medication and the first time a drug was given, airway control, measuring CPR quality, the number of shocks delivered, and whether a mechanical CPR device used, or if targeted oxygenation/ventilation was achieved, and if coronary reperfusion was attempted.

Finally, survival outcome documentation should be recorded: survival event defined as the return of spontaneous circulation sustained until arrival at the emergency department and transfer of care to medical staff at the receiving hospital. The return of spontaneous circulation is defined according to a clinical assessment that shows signs of life comprising a palpable pulse or a measurable blood pressure; however,

assisted circulation including extracorporeal life support, ventricular assist devices, or mechanical CPR should not be considered to be the return of spontaneous circulation until patient's own self-generated circulation is established [5].

In SCD, patients who survive either to 30 days or to hospital discharge, and also 12-month survival rates, should be recorded to facilitate the collection of information and statistics, which are important to build the healthcare system and facilities. Together with these rates, neurological outcome may be reported using the Cerebral Performance Category [30], modified Rankin Scale (mRS) [31], or equivalent pediatric tools. The Cerebral Performance Category is a 5-point scale ranging from 1 (good cerebral performance) to 5 (dead). The mRS is a 7-point scale ranging from 0 (no symptoms) to 6 (dead). We define survival with favorable neurological outcome as a CPC 1 or 2 or mRS 0 to 3, or no change in Cerebral Performance Category or mRS, from the patient's baseline status. Neurological outcome is very important to evaluate reported outcomes, health-related quality of life, and all stages of CPR quality, so that the outcomes can be used to provide better CPR strategies.

17.6 I-Basic Life Support (BLS)

During the past decade, recommendations for adult BLS have been classified as unchanged and updated (may be updated in wording, class, level of evidence, or any combination of these). The Training Network has also clarified the descriptions of lay rescuers as untrained, trained in chest compression-only CPR, or trained in CPR using chest compressions and ventilation (from ACC/AHA 2015).

BLS includes a series of stages comprising early recognition of SCA, activation of the EMS, early CPR, and rapid defibrillation with an AED. Initial recognition and response to heart attack and stroke are also considered a part of BLS. In 2015 AHA guidelines updated the recommendations for adult BLS guidelines for

lay rescuers and healthcare providers (Fig. 17.2). Untrained lay rescuers are encouraged to take certain steps: ensure scene safety, check for response, shout to or phone someone, check for no breathing or only gasping, and follow the dispatcher's instructions. However, trained lay rescuers may check for no breathing or only gasping, and if none, begin CPR with compressions without the dispatcher's instructions. In the hospital setting, a healthcare provider will activate a resuscitation team as soon as the check for response and adequate breathing and pulse is done, then immediately retrieve AED and emergency equipment, begin CPR, and defibrillate (if shockable rhythm confirmed on the monitor), and provide two-person CPR as help arrives.

17.6.1 Early Recognition and Activation of the Emergency Response System

The AHA guidelines define the laypersons and healthcare providers for the recognition and management of SCD regarding emergency response activation, especially highlighting dispatcher-guided CPR for laypersons in treating OHCA. Following first contact with an unresponsive adult victim, bystanders (lay responders) should immediately call the local emergency number to initiate a response. Healthcare providers should call for nearby help and continue to assess for breathing and pulse simultaneously before fully activating the emergency response system. The pulse should be checked from a larger artery (e.g., the carotid or femoral artery). The elapsed time for the pulse check should be less than 10 s, because the most important step, chest compression, must begin immediately. In this chaos, one should be aware of discriminating normal breathing and occasional gasps when respiration is monitored. In OHCA, erroneous information about respiration can result in failure by dispatchers to identify potential cardiac arrest and failure to instruct bystanders to initiate CPR immediately.

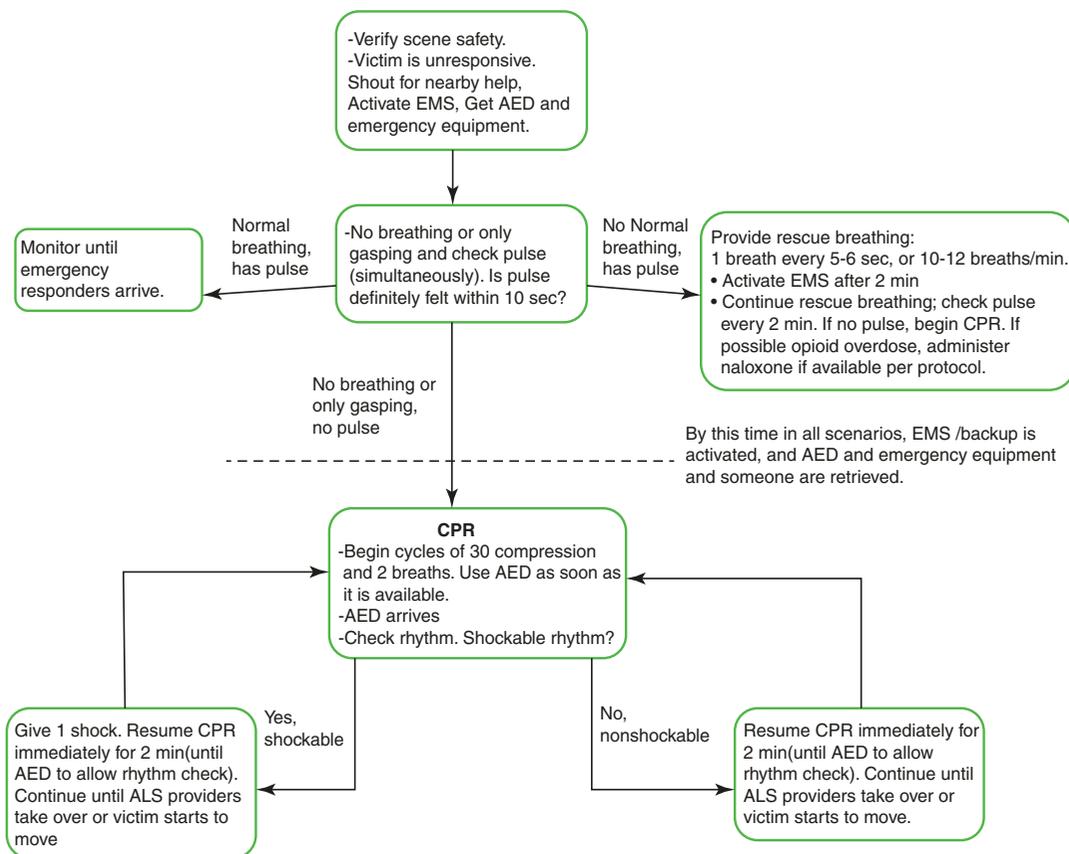


Fig. 17.2 BLS algorithm for adult cardiac arrest. *AED* automated external defibrillator, *BLS* basic life support, *CPR* cardiopulmonary resuscitation, *EMS* emergency

medical service. (Modified from 2015 American Heart Association Guidelines)

17.6.2 Early Cardiopulmonary Resuscitation

In cases with pulse present and normal breathing, the patient is closely monitored and the emergency response system activated as indicated by location and patient condition. If the case presents with pulse and no normal breathing, however, the Food and Drug Administration approves of the use of a naloxone autoinjector by lay rescuers and healthcare providers for patients with suspected opioid-associated cardiopulmonary arrest [32]. In 2015 AHA guidelines recommended for a patient with known or suspected opioid overdose who has a definite pulse but no normal breathing or only gasping (i.e., a respiratory arrest), in addition to providing standard

BLS care, it is reasonable for appropriately trained BLS healthcare providers to administer intramuscular or intranasal naloxone (Class IIa, Evidence-C). In the event of absent pulse, with no breathing or only gasping, the provider should initiate CPR and use an AED as soon as possible, and the emergency response system is activated immediately and a defibrillator and emergency equipment are retrieved. In such cases of pulseless collapse, a blow to the chest, the “thump version,” may be attempted by a properly trained rescuer or healthcare provider. The thump version technique utilizes one to two blows delivered firmly to the junction of the mid- to lower thirds of the sternum perpendicularly to the chest from a height of 20–25 cm. The thump version should be stopped if the patient does not

immediately respond with a spontaneous pulse and begin breathing.

By the way, as the main component of the CPR, chest compressions should not be deferred immediately after the pulse is checked. The key components of chest compression include depth, rate, and degree of recoil. Compressing the chest, the palms of the hands are overlapped and placed longitudinally on the lower half of the sternum. The sternum is depressed at least 5 cm at a rate of 100–120 compressions per minute. Depressing the sternum more than 6 cm is not recommended because of the high risk of thoracic injury with deep depression depth (Fig. 17.3). Each compression needs enough time for the return of complete chest recoil, to allow for venous return and blood flow. Incomplete recoil could increase intrathoracic pressure and reduce venous return, coronary perfusion pressure, and myocardial blood flow. The early CPR is initiated with one or two rescuers, and ideally should be 30 compressions to two breaths in each cycle of CPR [33].

According to new guidelines, the old sequence of CPR steps ABCD (airway, breathing, circulation, and defibrillation) has been modified to CAB (compression, airway, breathing). When CPR begins, minimizing interruptions in chest compressions remains a point of emphasis in that shorter as compared to longer interruptions in chest compressions influence physiological or clinical outcomes. Airway maneuvers should occur quickly, efficiently, and minimize interruptions in chest compressions. Opening the airway can be achieved by using the well-known head

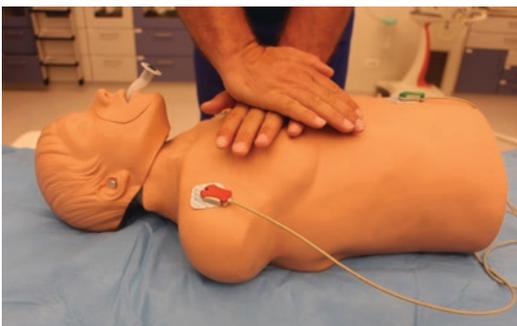


Fig. 17.3 Chest compression. The hands are placed on the lower half of the sternum



Fig. 17.4 The head tilt–jaw thrust maneuver. Displace the mandible forward, tilt the head backward, provide patent airway, and place an airway device

tilt–chin lift maneuver if no evidence of head or neck trauma is present (Fig. 17.4).

After the head is properly placed and the oropharynx cleared, if no airway rescue equipment is available a mouth-to-mouth ventilation effort can be initiated. Various devices may be available at the event scene, including plastic airways, esophageal obturators, the bag-mask device, and endotracheal tubes, and without time delay a trained rescuer inserts the available device quickly and properly. While awaiting emergency rescue personnel, until endotracheal intubation can be carried out, mouth-to-mouth resuscitation and temporary support with bag-mask ventilation is the usual method. When the patient's teeth can be opened enough, nasal or oral airways can be inserted to displace the tongue from the posterior oropharynx to the front. Out-of-hospital studies have demonstrated that chest compression-alone CPR is not inferior to traditional compression-ventilation CPR, yet healthcare providers are still expected to provide assisted ventilation [33]. During the ventilation, rescuer should take care to avoid rapid or forceful breaths, which may reduce preload and cardiac output with excessive positive-pressure ventilation [34]. In the early CPR, oxygen concentration is inhaled in maximum level to provide optimally saturated arterial hemoglobin concentrations. At the beginning, tidal volumes of approximately 400–600 ml are given over 1 s and should produce a visible chest rise, and volume and oxygen saturation readjust according to arterial oxygen saturation and



Fig. 17.5 Bag-mask ventilation

ventilation end-tidal CO₂ pressure. After arrival of an advanced airway facility, a respiratory rate of 10 breaths/min is enough for optimal ventilation because hyperventilation may be detrimental to neurological recovery [5]. Bag-mask ventilation needs training and skill with practice for competency (Fig. 17.5). A bag-mask device can provide positive-pressure ventilation; the health-care provider should be aware of its potential complications (i.e., gastric inflation). So long as the patient does not have an advanced airway in place, the rescuers should deliver cycles of 30 compressions and two breaths during CPR. When an advanced airway is in place during CPR, it might be reasonable for the provider to deliver one breath every 6 s (10 breaths/min) while continuous chest compressions are being performed. High-flow oxygen delivery via a mask with an oropharyngeal airway as part of CPR was reported to be associated with improved survival with favorable neurological outcome. The AHA guideline does not recommend the routine use of passive ventilation techniques but instead positive-pressure ventilation during conventional CPR for adults.

17.6.3 Early AED Defibrillation by First Responders

In 2015, ILCOR stated that observational clinical studies and mechanistic studies in animal models suggest that CPR under conditions of prolonged untreated ventricular fibrillation (VF)

or pulseless ventricular tachycardia (VT) might help restore metabolic conditions of the heart favorable to defibrillation, although some experts suggest that prolonged VF is energetically detrimental to the ischemic heart, justifying rapid defibrillation attempts regardless of the duration of arrest. The time from onset of cardiac arrest to ALS influences clinical outcomes, neurological status, and survival more effectively in patients defibrillated by first responders, compared with outcomes associated with awaiting EMS. The term first responder refers to the person on scene starting the initial CPR and more recently has emerged from trained emergency technicians allowed to carry out defibrillation in conjunction with basic life support to laypersons knowledgeable in CPR with access to AEDs [35]. The current guidelines recommend, for witnessed adult CA, when an AED (Fig. 17.6) is immediately available that the defibrillator be used as soon as possible (Class IIa, Evidence-C). For adults with unmonitored CA or for whom an AED is not immediately available, CPR is initiated while the defibrillator equipment is being retrieved and applied. If indicated, the defibrillator is attempted as soon as the device is ready for use [25]. An available defibrillator is attached to the patient with proper positioning of the electrode pad placement to the right of the upper sternal border below the clavicle and to the left of the nipple with the center in the mid-axillary line (illustrated in Fig. 17.7). According to the type

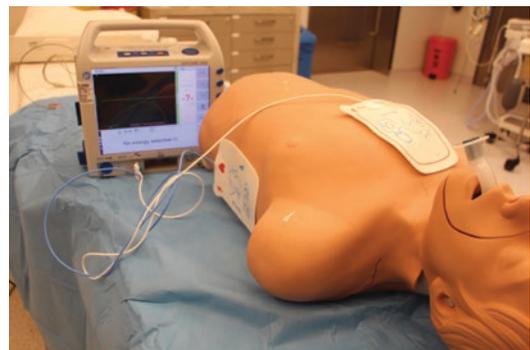


Fig. 17.6 Automated external defibrillator. The right and left electrode pads are attached to the right upper sternal border and left mid-axillary line

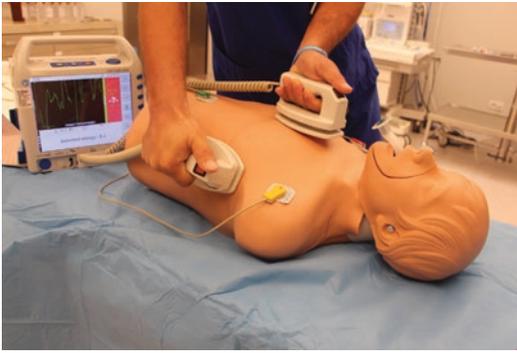


Fig. 17.7 Defibrillation. The defibrillator paddles are attached to the right of the upper sternal border and to the left of the nipple with the center in the mid-axillary line

of defibrillator (monophasic or biphasic), the amount of energy [joules (J)] delivered can change. Monophasic waveform defibrillators deliver a unidirectional energy charge, whereas biphasic waveform defibrillators deliver an in-series bidirectional energy charge. Practical and clinical consensus has showed that bidirectional energy delivery, by using less energy than traditional monophasic waveform shocks (120–200 J vs. 360 J, respectively) is probably more successful in terminating VT and VF, and may therefore cause less myocardial damage. Defibrillation should occur immediately when a VT/VF arrest is recognized; however, failure of the initial shock to provide an effective rhythm is a poor prognostic sign. Delayed defibrillation has slower rates of spontaneous circulation and survival rates to hospital discharge, and each additional minute of delay is associated with worse outcomes [36]. Following failed defibrillation, prompt chest compressions should be resumed in each CPR cycle.

17.7 II-Advanced Cardiac Life Support

The resuscitative sequence is designed to achieve a stabilization of spontaneous circulation and the hemodynamic situation [37]. The implementation of advanced life support (ALS) includes several interventions in addition to basic life support (BLS) to manage SCA. These interven-

tions are mainly related to airway manipulation, medication, arrhythmia management, and transition to post-resuscitation care. Most community-based CPR programs now permit trained emergency technicians to carry out ALS activities. The general goals of ALS are to convert the cardiac rhythm to a hemodynamically effective rhythm, to optimize ventilation, and to maintain circulation. With initiation of ALS, the patient's cardiac rhythm is promptly cardioverted/defibrillated as the first priority if appropriate equipment with AED is immediately available. One cycle period of closed chest cardiac compression immediately before defibrillation enhances the probability of survival, especially if circulation has been absent for 4 min or longer [38]. Besides the initial attempt to restore a hemodynamically effective rhythm, the patient is intubated and oxygenated, and a temporary pacer is placed in case of either a severe bradyarrhythmia or asystole. During the EMS effort for SCD survival, a vein is cannulated to administer emergency medications. The goal of ventilation, mostly required endotracheal intubation, is to reverse hypoxemia and to achieve a high alveolar oxygen pressure for patient ventilation with high oxygen. Following successful CPR, continuous monitoring of end-tidal CO₂ with waveform capnography and arterial O₂ saturation will be helpful during resuscitation and can guide the rescuers in adequacy of chest compressions [39]. If technically available conditions are provided, central venous pressure and O₂ saturation, and arterial pressure, pulse, and rhythm are monitored for further CPR strategy and the optimization of IV medications (Fig. 17.8).

17.7.1 Severe Bradycardia, Asystole, or Pulseless Electrical Activity

Pulseless cardiac arrest comprises VT, VF, asystole, and pulseless electrical activity (PEA). PEA means the heart produces weak electrical activity by itself without producing cardiac contraction and blood outflow, which is named electromechanical dissociation. In such cases, medical administrations may be

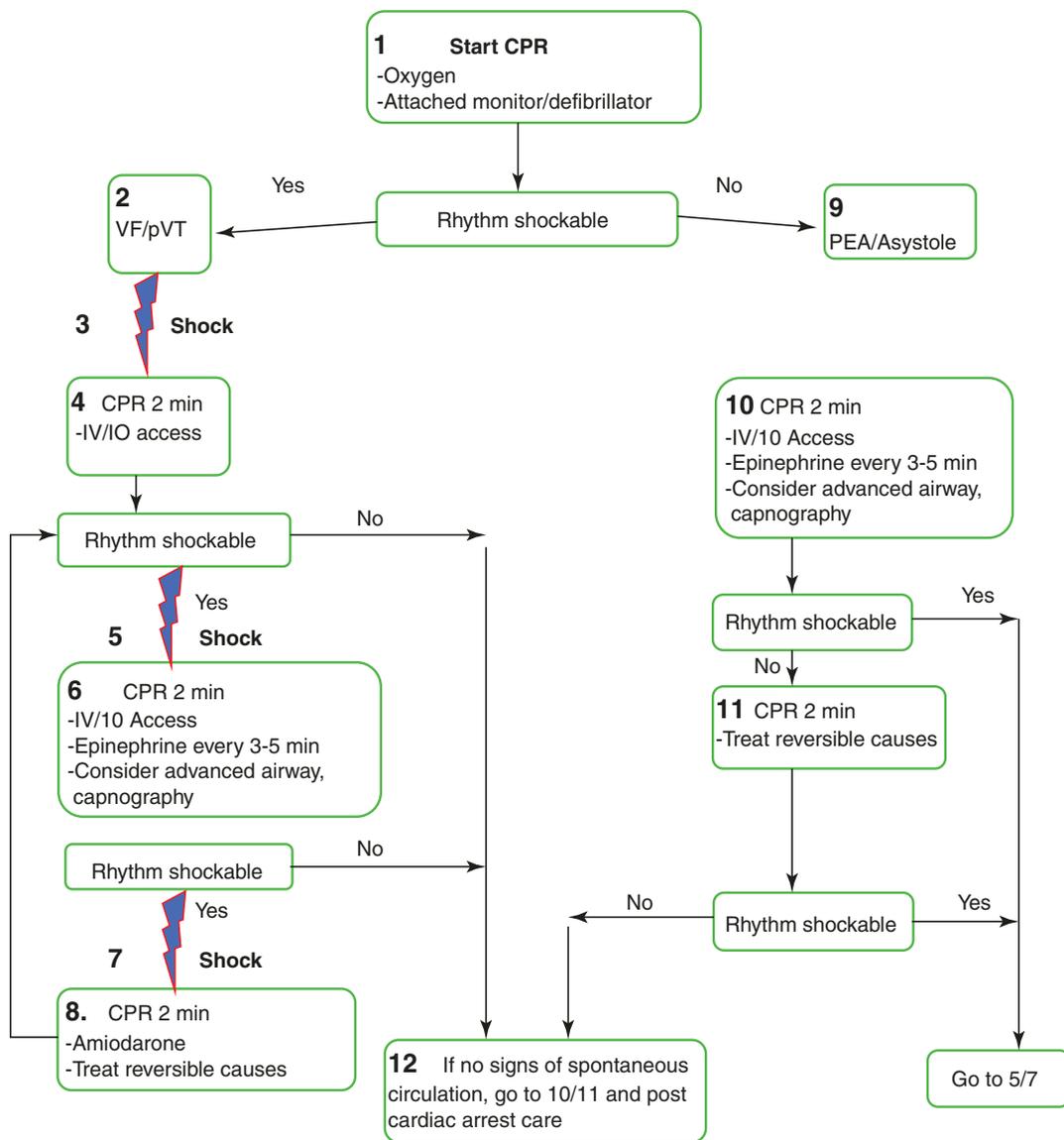


Fig. 17.8 Adult life support algorithm. *CPR* cardiopulmonary resuscitation, *IO* intraosseous, *IV* intravenous, *PEA* pulseless electrical activity, *pVT* pulseless ventricu-

lar tachycardia, *VF* ventricular fibrillation. (Modified from 2015 AHA Guidelines)

insufficient to maintain hemodynamic stability and resume effective rhythm. Providing continued chest compressions and ventilation, intravenous access, obtaining a more definitive airway, and positive inotropic drug therapy should be initiated, and occasionally temporary pacemaker implantation is needed (Fig. 17.9).

17.7.2 Ventricular Fibrillation and Pulseless Ventricular Tachycardia

When initiating CPR, immediately after rescuer witness, an AED is placed on the chest to determine rhythm, and if VF/pulseless VT is detected, a shock is employed (as illustrated in Fig. 17.8,

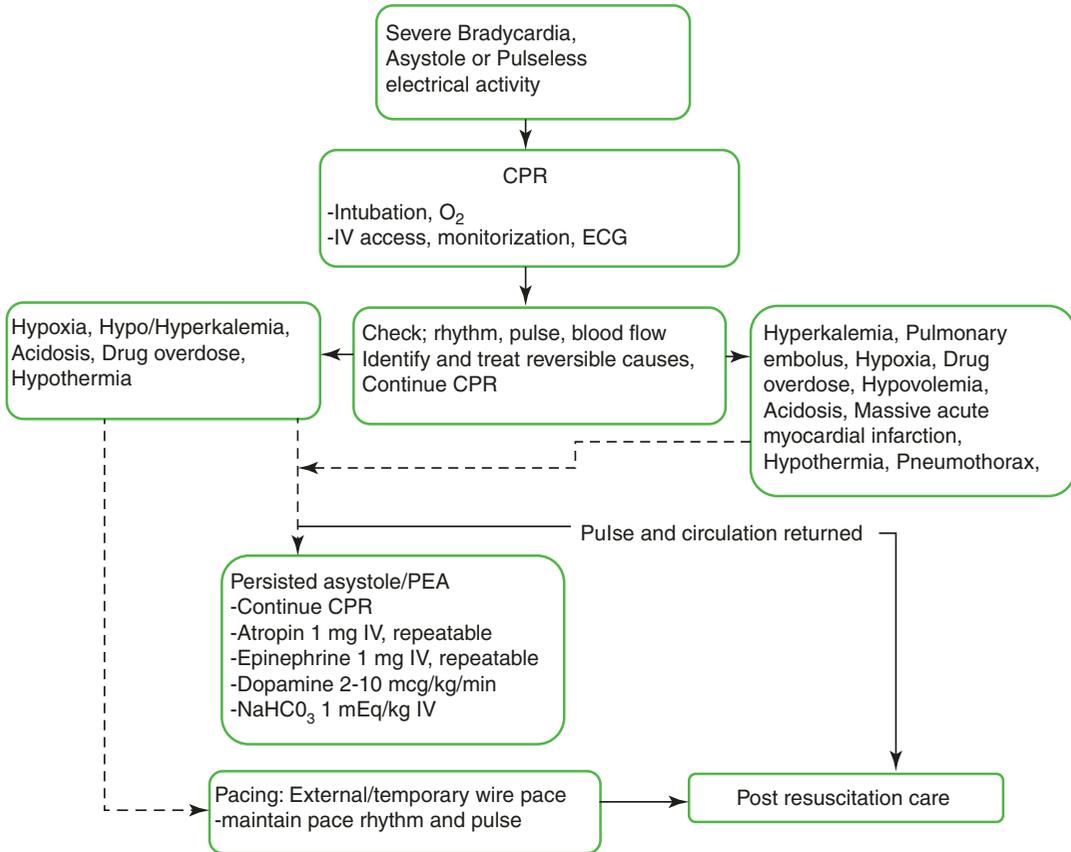


Fig. 17.9 Advanced life support for patients with bradyarrhythmic-asystolic arrests and pulseless electrical activity. *PEA* pulseless electrical activity, *VF* ventricular

fibrillation, *VT* ventricular tachycardia. (Modified from 2015 AHA Guidelines)

step 2). When VF or VT that is pulseless or accompanied by loss of consciousness is recognized on a monitor or by telemetry, defibrillation should be carried out immediately, and then CPR is continued for five cycles or about 2 min, followed by reevaluation of the cardiac rhythm.

An initial shock of 360 J should be delivered by monophasic devices and of 120–200 J by biphasic devices. If VF/VT persists after one to two sets of CPR–defibrillation cycles, intravenously (IV) a vasopressor (i.e., epinephrine 1 mg IV, repeatable every 3–5 min) is administered. Even if VF/VT is restored to an acceptable rhythm, IV bolus administration of lidocaine or amiodarone may improve the likelihood of restoring and maintaining normal rhythm [5].

17.7.3 Pharmaceutical Therapy

During CPR, IV access is of paramount importance, without interruption of CPR and defibrillation. In arrested and shock patients, IV cannulation is not always possible, and the alternative for using drugs may be an intraosseous catheter or endotracheal tube. When using an endotracheal tube, the drug dosage is 2–10 times the recommended IV dose, and it is diluted in 5–10 ml fluid.

Epinephrine, atropine, lidocaine, and amiodarone are among the drugs most commonly used during CPR. Epinephrine is administered and followed by repeated defibrillation, and may be repeated at 3- to 5-min intervals with a defibrillator shock in between; and if needed, vasopressin

has been suggested as an alternative to epinephrine. Epinephrine, dopamine, and dobutamine can increase diastolic blood pressure and thereby restore coronary perfusion pressure. On the other hand, however, epinephrine and vasopressin also increase myocardial oxygen consumption by increasing heart rate and afterload. Amiodarone prolongs repolarization and refractoriness in all cardiac conduction systems by affecting the cardiac sodium and potassium channels and α - and β -receptors. Although amiodarone is one of the most effective drugs for VF/VT, it can exacerbate or induce arrhythmias, especially torsades de pointes, by its proarrhythmic side effect. Administration of amiodarone in patient with VF/VT arrest improves survival when compared with placebo and lidocaine [34]. The usual recommended bolus dose of amiodarone for VF/VT is 300 mg IV, and the dose may be repeated at 150 mg IV.

17.8 III-Post-Cardiac Arrest Stabilization Care

Once return of spontaneous circulation is achieved after resuscitation, patients for both OHCA and IHCA are admitted to an intensive care unit and continuously monitored at least 2 days for post-cardiac arrest stabilization care. Post-cardiac arrest care requires improving cardiopulmonary function and maintaining adequate organ perfusion. Although the patient can be managed in the same center, if needed the patient can be transferred to a larger regional center that is equipped to manage excellent post-cardiac arrest care.

If SCD caused in patients with acute myocardial infarction is complicated by VF/VT, the events are mostly reverted by prompt implementation in the hospital setting. The immediate mortality among patients with hemodynamic or mechanical dysfunction in the setting of complicated acute myocardial infarction ranges from 59% to 89%, depending on the severity of the hemodynamic abnormalities and size of the myocardial infarction. In such cases, post-cardiac arrest management is often difficult and is very

important for survival of the patient. As soon as possible, IV amiodarone and anti-ischemic therapy including invasive procedures should be initiated, and not deferred. Immediate and daily 12-lead ECG is recorded, and if acute ST-segment elevation is noted, the patient should undergo emergent angiography for primary percutaneous coronary intervention [40], regardless of neurological status.

Following post-cardiac arrest care, optimal oxygenation and ventilation by using advanced airway placement should be provided and monitored in the care unit. Desired arterial O₂ saturation and ventilation are aimed at more than 94% and PaCO₂ of 35–40 mmHg, respectively. Advanced airway placement may be considered without hyperventilation. Blood pressure should be kept at or above 90/50 mmHg, especially in patients with coronary and/or cerebral ischemia, and if needed, administration of intravenous fluids and vasoactive drugs can be used for this goal. In the period of post-cardiac arrest care, a chest radiograph, 12-lead ECG, echocardiogram, serum lactate and serial cardiac enzymes are obtained. One of the paramount important steps is neurological recovery and cerebral function at hospital discharge. Following neurological status, cerebral MR and electroencephalogram can be obtained to evaluate neurological recovery, cognitive functions, and epileptic status. Hyperthermia is avoided at all times as this can worsen ischemic brain injury, therefore recommended therapeutic hypothermia of 32–34 °C. Blood glucose concentrations, anemia, electrolyte imbalances and metabolic abnormalities are associated with poor neurological outcome, therefore these parameters should be controlled and maintained in normal range [42].

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Take Home Message

Cardiac arrest is a challenging problem both in the prehospital community and in-hospital emergency medicine with high mortality and morbidity. Cardiopulmonary resuscitation is a life-saving strategy for

treatment applied by laypersons, trained rescuers, and healthcare providers. Although the guidelines cover most related situations, rescuers may be faced with situations for which no clear guidelines exist. Therefore, management of such cases may be difficult and demanding for the whole team approach including trained laypersons, dispatcher's assistants, early call and activation of EMS, early initiation of CPR and defibrillation with available AED, advanced airway ventilation in conjunction with ALS, and finally transfer to hospital to achieve professional support for life and post-cardiac care. Everyone from the layperson to the intensive care unit staff should be aware and ready for immediate implementation of all sequential steps of CPR. Training in the SCR program has paramount importance for this successful action plan; however, iterative education with checking whether all teams and systems are ready, and all needed equipment available as early as possible, is very important for EMS testing.

Public administrators and healthcare system operators should make national emergency preparation plans for SCD victims by defining risky places, high-risk persons, and primary and secondary prevention strategies. According to the preparation plan, trained laypersons/healthcare providers, the EMS organization, and AED should be provided in all places in the shortest period of time.

References

1. Sheppard MN. Etiology of sudden cardiac death in sport: a histopathologist's perspective. *Br J Sports Med.* 2012;46:15–21.
2. Zipes DP, Camm AJ, Borggrefe M, Buxton AE, Chaitman B, et al. ACC/AHA/ESC 2006 guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death executive summary: a report of the American College of Cardiology/American Heart Association Task Force and the European Society of Cardiology Committee for Practice Guidelines (writing committee to develop guidelines for management of patients with ventricular arrhythmias and the prevention of sudden cardiac death) developed in collaboration with the European Heart Rhythm Association and the Heart Rhythm Society. *Eur Heart J.* 2006;27:2099–140.
3. Travers AH, Perkins GD, Berg RA, Castrén M, Considine J, et al.; on behalf of the Basic Life Support Chapter Collaborators. Part 3: Adult basic life support and automated external defibrillation: 2015 international consensus on cardiopulmonary resuscitation and emergency cardiovascular care science with treatment recommendations. *Circulation.* 2015 132(1):51–83.
4. Myerburg RJ, Castellanos A. Emerging paradigms of the epidemiology and demographics of sudden cardiac arrest. *Heart Rhythm.* 2006;3:235–9.
5. Perkins GD, Jacobs IG, Nadkarni VM, Berg RA, Bhanji F, et al.; Utstein Collaborators. Cardiac arrest and cardiopulmonary resuscitation outcome reports: update of the Utstein resuscitation registry templates for out-of-hospital cardiac arrest: a statement for healthcare professionals from a Task Force of the International Liaison Committee on Resuscitation (American Heart Association, European Resuscitation Council, Australian and New Zealand Council on Resuscitation, Heart and Stroke Foundation of Canada, InterAmerican Heart Foundation, Resuscitation Council of Southern Africa, Resuscitation Council of Asia); and the American Heart Association Emergency Cardiovascular Care Committee and the Council on Cardiopulmonary, Critical Care, Perioperative and Resuscitation. *Resuscitation.* 2015;96:328–340.
6. Nishiyama C, Brown SP, May S, Iwami T, Koster RW, et al. Apples to apples or apples to oranges? International variation in reporting of process and outcome of care for out-of-hospital cardiac arrest. *Resuscitation.* 2014;85(11):1599–609.
7. Benjamin EJ, Blaha MJ, Chiuve SE, Cushman M, Das SR, et al.; American Heart Association Statistics Committee and Stroke Statistics Subcommittee. Heart disease and stroke statistics—2017 update: a report from the American Heart Association. *Circulation.* 2017;135:e146–e603.
8. Rosamond W, Flegal K, Furie K, Go A, Greenlund K, et al.; American Heart Association Statistics Committee and Stroke Statistics Subcommittee. American Heart Association Statistics Committee and Stroke Statistics Subcommittee: Heart Disease and Stroke Statistics—2008 update: a report from the AHA Statistics Committee and Stroke Statistics Subcommittee. *Circulation.* 2008;117(4):e25–e146.
9. Bonow RO, Mann DL, Zipes DP, Libby P. Braunwald's heart disease: a textbook of cardiovascular medicine, single volume: expert consult premium edition. In: Myerburg RJ, Castellanos A, editors. *Cardiac arrest*

- and sudden cardiac death. 9th ed. Philadelphia: Elsevier Saunders; 2011. p. 845–81.
10. Myerburg RJ. Sudden cardiac death: exploring the limits of our knowledge. *J Cardiovasc Electrophysiol*. 2001;12:369–81.
 11. Myerburg RJ, Junttila MJ. Sudden cardiac death caused by coronary heart disease. *Circulation*. 2012;125:1043–52.
 12. Vancini RL, Nikolaidis PT, Lira CAB, Vancini-Campanharo CR, Viana RB. Prevention of sudden death related to sport: the science of basic life support—from theory to practice. *J Clin Med*. 2019;8(4):556–75.
 13. Kannel WB, Shatzkin A. Sudden death: lessons from subsets in population studies. *J Am Coll Cardiol*. 1985;5(6):141–9.
 14. Myerburg RJ, Kessler KM, Castellanos A. Sudden cardiac death. Structure, function, and time-dependence of risk. *Circulation*. 1992;85:12–110.
 15. Ljungqvist A, Jenoure P, Engebretsen L, Alonso JM, Bahr R, Clough A, De Bondt G, Dvorak J, Maloley R, Matheson G, Meeuwisse W, Meijboom E, Mountjoy M, Pelliccia A, Schweltnus M, Sprumont D, Schamasch P, Gauthier JB, Dubi C, Stupp H, Thill C. The International Olympic Committee (IOC) consensus statement on periodic health evaluation of elite athletes, March 2009. *Br J Sports Med*. 2009;43:631–43.
 16. Steinvil A, Chundadze T, Zeltser D, Rogowski O, Halkin A, et al. Mandatory electrocardiographic screening of athletes to reduce their risk for sudden death: proven fact or wishful thinking? *J Am Coll Cardiol*. 2011;57:1291–6.
 17. James CA, Bhonsale A, Tichnell C, Murray B, Russell SD, et al. Exercise increases age-related penetrance and arrhythmic risk in arrhythmogenic right ventricular dysplasia/cardiomyopathy-associated desmosome mutation carriers. *J Am Coll Cardiol*. 2013;62:1290–7.
 18. Behr ER, Dalageorgou C, Christiansen M, Syrris P, Hughes S, et al. Sudden arrhythmic death syndrome: familial evaluation identifies inheritable heart disease in the majority of families. *Eur Heart J*. 2008;29:1670–80.
 19. Priori SG, Blomström-Lundqvist C, Mazzanti A, Blom N, Borggrefe M, et al. 2015 ESC guidelines for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death. The task force for the management of patients with ventricular arrhythmias and the prevention of sudden cardiac death of the European Society of Cardiology. *Europace*. 2015;7(11):1601–87.
 20. Friedlander Y, Siscovick DS, Weinmann S, Austin MA, Psaty BM, et al. Family history as a risk factor for primary cardiac arrest. *Circulation*. 1998;97:155–60.
 21. Cheitlin MD, Armstrong WF, Aurigemma GP, Beller GA, Bierman FZ, et al. ACC/AHA/ASE 2003 guideline update for the clinical application of echocardiography—summary article: a report of the American College of Cardiology/American Heart Association task force on practice guidelines (ACC/AHA/ASE Committee to update the 1997 guidelines for the clinical application of echocardiography). *J Am Coll Cardiol*. 2003;42:954–70.
 22. Middlekauff HR, Stevenson WG, Stevenson LW, Saxon LA. Syncope in advanced heart failure: high risk of sudden death regardless of origin of syncope. *J Am Coll Cardiol*. 1993;21:110–6.
 23. Sasson C, Rogers MA, Dahl J, Kellermann AL. Predictors of survival from out-of-hospital cardiac arrest: a systematic review and meta-analysis. *Circ Cardiovasc Qual Outcomes*. 2010;3:63–81.
 24. Begg C, Cho M, Eastwood S, Horton R, Moher D, et al. Improving the quality of reporting of randomized controlled trials: the CONSORT statement. *JAMA*. 1996;276:637–9.
 25. Kleinman ME, Brennan EE, Goldberger ZD, Swor RA, Terry M, Bobrow BJ, Gazmuri RJ, Travers AH, Rea T. Part 5: Adult basic life support and cardiopulmonary resuscitation quality: 2015 American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2015;132(2):414–35.
 26. Kleinman ME, Goldberger ZD, Rea T, Swor RA, Bobrow BJ, Brennan EE, Terry M, Hemphill R, Gazmuri RJ, Hazinski MF, Travers AH. 2017 American Heart Association focused update on adult basic life support and cardiopulmonary resuscitation quality. An update to the American Heart Association guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2017;137(1):e7–e13.
 27. Jacobs I, Nadkarni V, Bahr J, Berg RA, Billi JE, et al.; International Liaison Committee on Resuscitation. International Liaison Committee on Resuscitation. Cardiac arrest and cardiopulmonary resuscitation outcome reports: update and simplification of the Utstein templates for resuscitation registries: a statement for healthcare professionals from a task force of the International Liaison Committee on Resuscitation. *Circulation*. 2004;63:233–249.
 28. Bohm K, Vaillancourt C, Charette ML, Dunford J, Castrén M. In patients with out-of-hospital cardiac arrest, does the provision of dispatch cardiopulmonary resuscitation instructions as opposed to no instructions improve outcome: a systematic review of the literature. *Resuscitation*. 2011;82:1490–5.
 29. Kuusma M, Alasääla. Out-of-hospital cardiac arrests of non-cardiac origin: epidemiology and outcome. *Eur Heart J*. 1997;18:1122–8.
 30. Yasunaga H, Miyata H, Horiguchi H, Tanabe S, Akahane M, Ogawa T, Koike S, Imamura T. Population density, call-response interval, and survival of out-of-hospital cardiac arrest. *Int J Health Geogr*. 2011;10:26–35.
 31. Van Swieten JC, Koudstaal PJ, Visser MC, Schouten HJ, van Gijn J. Interobserver agreement for the assessment of handicap in stroke patients. *Stroke*. 1988;19:604–7.

32. US Food and Drug Administration. FDA news release: FDA approves new hand-held auto-injector to reverse opioid overdose; 2015.
33. Idris AH, Guffey D, Aufderheide TP, Brown S, Morrison LJ, et al.; Resuscitation Outcomes Consortium (ROC) Investigators. Relationship between chest compression rates and outcomes from cardiac arrest. *Circulation*. 2012;125:3004–3012.
34. Aufderheide TP, Sigurdsson G, Pirralo RG, Yannopoulos D, McKnite S, et al. Hyperventilation induced hypotension during cardiopulmonary resuscitation. *Circulation*. 2004;109(16):1960–5.
35. Truong HT, Low LS, Kern KB. Current approaches to cardiopulmonary resuscitation. *Curr Probl Cardiol*. 2015;40(7):275–313.
36. Chan PS, Krumholz HM, Nichol G, Nallamothu BK. American Heart Association National Registry of cardiopulmonary resuscitation investigators. Delayed time to defibrillation after in-hospital cardiac arrest. *N Engl J Med*. 2008;358(1):9–17.
37. ECC Committee. Subcommittees and task forces of the American Heart Association: 2005 AHA guidelines for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2005;112:IV1.
38. Wik L, Hansen TB, Fylling F, Steen T, Vaagenes P, Auestad BH, Steen PA. Delaying defibrillation to give basic cardiopulmonary resuscitation to patients with out-of-hospital ventricular fibrillation: a randomized trial. *JAMA*. 2003;289:1389–95.
39. Sheak KR, Wiebe DJ, Leary M, Babaeizadeh S, Yuen TC, Zive D, Owens PC, Edelson DP, Daya MR, Idris AH, Abella BS. Quantitative relationship between end-tidal carbon dioxide and CPR quality during both in-hospital and out-of-hospital cardiac arrest. *Resuscitation*. 2015;89:149–54.
40. Callaway CW, Donnino MW, Fink EL, Geocadin RG, Golan E, et al. Part 8: Post-cardiac arrest care 2015. American Heart Association guidelines update for cardiopulmonary resuscitation and emergency cardiovascular care. *Circulation*. 2015;132(2):465–82.
41. Marine JE, Shetty V, Chow GV, Wright JG, Gerstenblith G, Najjar SS, Lakatta EG, Fleg JL. Prevalence and prognostic significance of exercise-induced nonsustained ventricular tachycardia in asymptomatic volunteers: BLSA (Baltimore Longitudinal Study of Aging). *J Am Coll Cardiol*. 2013;62:595–600.

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Immediate Management of Acute Trauma on Court in Basketball

18

Tekin Kerem Ulku and Ignacio Muro

18.1 Introduction

More than 500,000 basketball injuries are treated in emergency rooms in United States every year. Popularity and high participation rates of basketball can cause a wide range of injuries. Injury rates are reported to be 8.5–1.1 injuries per 1000-h athletic participation [1].

Basketball is an inherently vertical sport requiring 70–250 jumps actions and direction changes in every 2–3 s per game, which is 2–4 times greater than soccer and volleyball. This multidirectional nature of basketball requires constant acceleration and deceleration. During the past three decades, basketball sustained a variety of changes. After new rules governing shorter shot clock time and longer shooting distance for three points, games become more athletic and aggressive. Therefore, in the past few decades the game evolved from being semi-contact to high contact, bringing higher injury rates.

Luckily majority of basketball injuries are mild to moderate form of sprains, strains, and contusions. Severe injuries occasionally do occur

especially more commonly during elite games. These injuries are usually associated with significant morbidity and subsequent game loss. Due to tremendous exertions performed on lower extremities foot, ankle and knee injuries are the most common ones followed by the lower back, hand, and wrist. Studies showed that lower limb injuries account for 46–68% of all basketball injuries followed by head and neck 5.8–23.7%, upper limb 5.6–23.2%, spine and pelvis 6–14.9%.

Although major injuries are relatively rare, appropriate on-court management of acute injuries are of paramount importance. Management strategies should be based on performing acute life-saving intervention if needed, minimizing zone of injury, preventing further injury, decreasing pain, promoting healing, and allowing safe return to competition.

A variety of health-care professionals with different levels of experience and training are providing health care during games. Despite providing a definitive diagnosis and management of long-term treatment needs different levels of experience, all health-care professionals on court during a game or training should be familiar with acute immediate management of trauma on court.

Acute trauma during a basketball game may affect extremities, head and neck region, or spine. Whichever site is affected, basic principles of providing airway, breathing, and circulation should be the mainstay of treatment whenever necessary.

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FIBA rules governing the role of medical professionals during the event of an injury states that the referee should stop the game and calls the medical professional whenever necessary. However, if medical officer is a doctor, a doctor may enter the court without the permission of an official if, in the doctor's judgment, the injured player requires immediate medical attention.

18.2 Acute Management of Extremity Trauma

Extremity trauma is by far the most common acute injury during basketball games. Lower extremity is the most common affected site. Even though the extremities are primarily composed of musculoskeletal tissue (bones, muscles, tendons, ligaments), health-care professionals should also be prepared to manage injuries to the skin and neurovascular structures.

When an extremity injury occurs first, a history, mechanism of trauma, and severity of pain should be obtained from the player. Athlete should be asked if he/she is able bear weight on the affected extremity. Any feedback about the functional deficits, instability, numbness, or weakness is also important.

The physical examination should be first directed to the entire affected extremity. Team physician should first survey any open injuries, gross deformities, and major soft tissue swelling. Active and passive joint range of motions should be carefully evaluated. Pain localization should be made by carefully palpating the injured area. Any crepitation and pathological motion should be carefully noted. Additional specific tests for ligamentous structures and strength of muscle groups should be performed. Any defects in the major tendon groups should be carefully noted. As a part of the injured extremity survey, a careful neurovascular examination should be performed. Any pallor, diffuse paresthesia, pain, cold, and diminished pulses should alert the physician about extremity ischemia [2]. Fortunately, these are very rare injuries.

Team physician should also be aware of possibility of compartment syndrome in an injured

extremity. Especially lower extremity is more susceptible to compartment syndrome after severe muscle contusions or tibia fractures. Even though compartment syndrome can jeopardize the vascular supply the classical signs of ischemia are usually not common. Instead on physical examination key finding is the pain out of proportion to injury, and marked increase in pain during passive stretching.

Further evaluation may require imaging. On-site access to imaging is very rare. However, some guidelines may aid the physician in determining when radiography is necessary. Ottawa rules are useful tools to assess the need for radiography in knee and ankle injuries [3].

Ottawa Knee Rules Criteria

- Patient over 55 years old or older
 - Isolated tenderness on patella
 - Tenderness at the head of fibula
 - Inability to flex the knee to 90°
 - Inability to bear weight for four steps
- (Presence of one of the following criteria indicates knee series)

Ottawa Foot-Ankle Rules Criteria

- Pain over lateral malleolus
 - Pain over medial malleolus
 - Pain over navicular bone
 - Pain over the base of fifth metatarsal
 - Inability to bear weight for four steps
- (Presence of one of the following criteria indicates knee series)

18.3 Treatment

The health-care professional should apply an effective triage system for treatment on court. One should be able to determine the injuries that can be evaluated for return to play, the injuries that needs removal from the game and stabilization and finally injuries that needs emergency transfer to the hospital. Emergency transfer due to extremity trauma is generally indicated in long bone fractures, open fractures, major joint dislocations,

and injuries causing neurovascular compromise. Whenever the ambulance is not available, the physician should always have a plan to transfer these patients to the nearest medical facility.

Injuries that require removal from the play but do not require immediate transfer of the player like serious musculotendinous injuries, sprains, or small bone fractures like hand or foot can be stabilized and undergo evaluation after the game. Stabilization can be performed using any ready-to-use material such as a brace, tape, bandage, an inflatable splint, or an aluminum enriched foam splint. Most difficult cases are often the moderate injuries with some limitation that might be able to play. However, there are no specific guidelines and each injury must be evaluated on individual basis. The physician must keep in mind general principles of extremity trauma and when in doubt athlete's health must be of paramount importance.

In management of open injuries and lacerations, evaluation should be done to determine if the injury is extending to bone, joint, or neurovascular structures. The lesion should be irrigated with a sterile saline solution to clean out the debris if present and if necessary mechanical debridement may be performed with a sterile sponge. After application of antiseptic solutions, small lacerations can be managed with tissue glues or sterile-strips. However, suturing may treat larger lacerations. Whenever it is appropriate, quick sideline management can be performed with a stapler to keep the player in the game, and wound can be revised after the game if necessary.

Sideline management of closed fractures mainly should focus on relieving pain by stabilizing the extremity and preventing further injury to the adjacent soft tissues especially neurovascular structures. Whenever a markedly displaced fracture is suspected, gentle longitudinal traction should be applied just to relieve and align the tissues. Experienced physicians should only attempt reduction without being too aggressive on court-side. After gentle traction and aligning the extremity, a splint can be applied including fracture site and adjacent joints. A postreduction neurovascular examination should always be performed. Any deterioration in neurovascular status may require emergency transfer. After trac-

tion and splinting elevation and ice application will help to decrease the pain and control excessive inflammation, fingers and toes can be immobilized using buddy taping with a soft tape.

Lower extremity long bone fractures require non-weight-bearing ambulation using stretcher, wheelchair, or crutches depending on severity of injury unless proven otherwise.

Joint dislocations may occur during events. Mostly finger and shoulder dislocations are common. Although very rare in basketball, it should always be kept in mind that knee dislocations and hip dislocations present a medical emergency and requires immediate transfer to nearest medical facility because of high incidence of accompanying neurovascular injury incidence.

Finger dislocations are quite common in basketball, and an experienced physician can perform a sideline reduction. After reduction stability assessment buddy taping can be applied and player can return to play in some occasions. It should be kept in mind that an experienced physician should attempt all reduction maneuvers, and a postreduction stability and neurovascular status assessment should be performed [4].

Initial treatment of ligament sprains depends on involved joint and degree of severity of the sprain. A careful examination shows the grade of sprain. Grade 1 lesions are generally stable with minimal pain, and athlete may return to play after symptomatic care. Most grade 3 sprains show a marked increase in laxity and requires removal from the play and initially a RICE treatment protocol should be initiated. The difficulty of on-court assessment of a sprain usually lies in Grade 2 lesions. Determining whether a grade 2 sprain with a mild to moderate laxity can return to play is somewhat challenging [5, 6]. Best approach would be testing the stability of the affected joint and comparing it with the unaffected site, and if enough stability can be achieved by taping or bracing, then players can be examined on the court side about basic running, jumping, or cutting drills, whenever appropriate player may be allowed to return to play. One main special concern can be type 2 acromioclavicular sprains which are difficult to protect or stabilize and prevent immediate return to play [7].

Muscle contusions are traumatic blunt injuries to muscle bellies mainly in the lower extremity. They create temporary weakness and pain in the affected site, and they can generally be treated by ice and compression. If the athlete is returning to the competition, the area should be generously padded. It should be kept in mind that severe contusions especially at the quadriceps area may develop heterotopic ossification. Whenever a severe injury is suspected to minimize the hematoma formation, the athlete should not be allowed to return to play and should be rested in minimum muscular tension with effected extremity elevated.

Muscle strains are one of the common injuries in basketball. General courtside treatment consists of RICE protocol. None of the suspected players should be allowed to return to play, and lower extremity strains should be allowed to bear weight using crutches.

Complete tendon ruptures cause severe functional limitations and generally require immediate removal from play. The joint that the tendon is crossing can be immobilized with a splint to prevent pain and shortening.

Return to play decisions should always be given by keeping idea of protecting the athlete from further injury in mind. For major injuries like long bone fractures, major joint dislocations, or tendon ruptures, decision of removing the athlete from the competition is obvious. Similarly for mild injuries like grade 1 sprain that do not cause instability or minor contusions, easy decision of keeping the player in competition can be made. However, the main challenge lies in the injuries with moderate level of injury or that cause relative moderate instability. In these cases, the decision should be made on case-by-case basis [8, 9]. Therefore, the main role of the medical professional is deciding whether it is safe for the player to return to play or not.

18.4 Acute Management of Spinal Trauma

Basketball is relatively one of the minor risk sports for spinal injuries. Nevertheless, a study among NBA players shows that lumbar spine

injuries constitute 10.2% and cervical spine 1.6%, a total of 11.8% of all injuries [10]. Although spinal injuries account for a smaller percent of all traumatic basketball injuries, they can lead to devastating consequences.

An optimal outcome for spinal traumatized players requires quick and effective response to injury. Proper pregame preparation is essential for this response. This begins with proper education of team health-care providers both in recognition and management of spinal trauma. Creating a spinal trauma protocol and pregame communication with emergency transfer officials on court is also important. Locating possible medical facilities that player can be transferred will help to provide a quicker transfer, and checking the presence of necessary tools like cervical collar and trauma board is of paramount importance.

- Proper education
- Create a spinal trauma protocol
- Communicate with emergency transfer officials before the game
- Check the presence of appropriate equipment before the game
- Locate possible transfer centers before the game

Team physician or health-care providers should always be attentive to game to be able to identify the mechanism of possible injury. Management begins with a primary survey, standard ABCD Trauma protocol. As soon as a spinal injury is suspected in a basketball player, a neutral cervical stabilization involving cupping the occiput and grasping mastoid process should be performed. Under no circumstance, should traction be applied to the player's head. Player should be moved to neutral position unless motion causes increased pain or loss of function.

If the player is in prone position after injury, player must be returned to supine position using a prone log roll technique, which requires at least four trained personnel to execute. After careful application of cervical collar, a neurologic

screening should be conducted. The evaluation of conscious player should begin by questioning about extremity numbness, painful dysesthesias, and weakness and neck and back pain. A limited examination can identify obvious neurologic deficit if the player is unable to move all or any limbs or has gross weakness numbness or significant pain with palpation. In a player with altered consciousness, it should always be kept in mind that cervical spine trauma can be associated with a head trauma.

After the quick initial evaluation, player should be transferred to trauma board with preferably lift and slide technique with airway easily accessible and transferred to nearest medical facility immediately.

Prone Log Roll Technique

- Requires at least four trained people
- Spine maintained in neutral position by team leader using cross arms technique
- Rescuers synchronously pull the player toward themselves
- Fifth rescuer slides the back board under player during midpoint of the roll

Lift and Slide Technique

- Requires at least five trained people
- Less cervical motion compared to Log Roll Technique
- Spine maintained in neutral position by team leader
- Three rescuers lift the player synchronously lifting 15 cm off ground
- The fifth rescuer slides the board under the player

18.5 Acute Management of Concussion and Head Trauma

A concussion is defined as a complex pathophysiological process affecting the brain, induced by biomechanical forces [11]. Concussions are

potentially serious injuries that may have long-term sequel, which requires careful management. In 2016, FIBA published Concussion Guidelines following 2016 Berlin Concussion Consensus.

According to guidelines any basketball player who has concussion diagnosed by a doctor, must not have any further participation to game or training until medically cleared by a physician experienced in the management of concussion. At the community-level basketball where a doctor is unlikely present, if a concussion is suspected, then player should not participate unless cleared by a doctor.

Whenever a concussion happens, players generally show symptoms of altered mental state, neurological dysfunction, and cognitive impairment. Concussions can sometimes be difficult to diagnose because signs and symptoms can mimic other neurological conditions. Therefore, a doctor is needed to confirm the diagnosis. For multimodal assessment, an experienced medical practitioner should undertake SCAT-5 protocol, which includes symptoms checklist, memory, balance, orientation, coordination, and cognitive functioning assessment.

If any of the following red flags are witnessed (including video review), player should immediately and safely be removed from the play and evaluated by a health-care provider.

Red Flags

- Neck pain or tenderness
- Double vision
- Weakness or tingling or burning in arms or legs
- Severe or increasing headache
- Seizure or convulsion
- Loss of consciousness
- Deteriorating consciousness
- Vomiting
- Increased restlessness or combative behavior

In practical perspective, whenever a trauma to the head witnessed, health-care provider should immediately enter the court, keeping in mind the

basic primary survey if needed, if any of the red flags are present, player should be removed from the play and examined using the SCAT-5 protocol. If a head trauma is witnessed without the presence of any red flag symptoms and initial medical examination reveals no signs of concussion, SCAT-5 assessment can be delayed to half-time or end of the game. However, player should be very carefully watched during the game, and it should be understood that concussion might sometimes evolve and increase by time.

A team doctor should consider performing a detailed thorough neurological examination to exclude more serious damage to brain, head, and neck area; if there are any signs of more serious damage like deteriorating mental status, drowsiness, repeated vomiting, headache or intolerance of light are present, player should immediately be transferred to the nearest medical facility.

Whenever there is any doubt about diagnosis, player should not be allowed to continue game and assessed repeatedly.

18.6 Conclusion

Sideline and on-court management of traumatized basketball player is an important role for health-care professionals providing coverage for events. With a careful history and examination, one should be able to reach a provisional diagnosis. Appropriate early treatment will protect the player from further harm and can hasten recovery. A health-care professional should always keep in mind the medical emergencies requiring urgent transfer to hospital.

References

1. Cumps E, Verhagen E, Meeusen R. Prospective epidemiological study of basketball injuries during one competitive season: ankle sprains and overuse knee injuries. *J Sports Sci Med.* 2007;6(2):204–11.
2. Wani ML, Ahangar AG, Ganie FA, Wani SN, Wani NU. Vascular injuries: trends in management. *Trauma Mon.* 2012;17(2):266–9.
3. Stiell IG, Greenberg GH, Wells GA, McKnight RD, Cwinn AA, Cacciotti T, McDowell I, Smith NA. Derivation of a decision rule for the use of radiography in acute knee injuries. *Ann Emerg Med.* 1995;26(4):405–13.
4. Skelley NW, McCormick JJ, Smith MV. In-game management of common joint dislocations. *Sports Health.* 2014;6:246–55. A review of criteria and methods for common joint dislocation reduction.
5. Gardiner JR, Madaleno JA, Johnson DL. Sideline management of acute knee injuries. *Orthopedics.* 2004;27:1250–4.
6. Mangine RE, Minning SJ, Eifert-Mangine M, Colosimo AJ, Donlin M. Management of the patient with an ACL/MCL injured knee. *Am J Sports Phys Ther.* 2008;3:204–11.
7. Wascher DC, Bulthuis L. Extremity trauma: field management of sports injuries. *Curr Rev Musculoskelet Med.* 2014;7(4):387–93.
8. Creighton DW, Shrier I, Shultz R, Meeuwisse WH, Matheson GO. Return-to-play in sport: a decision-based model. *Clin J Sport Med.* 2010;20:379–85.
9. Schupp CM. Sideline evaluation and treatment of bone and joint injury. *Curr Sports Med Rep.* 2009;8:119–24.
10. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the national basketball association: a 17-year overview. *Sports Health.* 2010;2(4):284–90.
11. McCrory P, Meeuwisse WH, Aubry M, Cantu B, Dvorak J, Echemendia RJ, et al. Consensus statement on concussion in sport—the 4th international conference on concussion in sport held in Zurich, November 2012. *Br J Sports Med.* 2013;47(5):1–11.

Part IV

Basketball Injuries and Management



Basketball Injuries: Epidemiology and Risk Factors

19

Kellie K. Middleton, MaCalus V. Hogan,
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19.1 Introduction

Over the last century, basketball has become one of the most popular sports in the world [1–3]. More people play basketball—including those who play recreationally, on organized teams, for high schools, colleges, and professionally—than any other sport in the United States [4]. This past year, 540,769 boys and 399,067 girls participated in high school basketball during the 2018–2019 academic year [5]. Since the first National Collegiate Athletic Association (NCAA) basket-

ball championships (1939 for men and 1982 for women), men and women’s college basketball has become one of the most popular sports to play and watch.

Once noncontact, the game has evolved along with the athleticism of the players since Naismith’s original “Thirteen Rules.” Basketball is a fast, intense, and aggressive sport. Consequently, basketball players have one of the highest overall injury rates among participants in non-collision sports [6, 7]. Injury severity can manifest in a change in activity or lifestyle, games missed, and/or impact one’s livelihood if playing professionally. Understanding the epidemiology of such injuries is important to all health-care providers and professionals affiliated with team sports.

The purpose of this chapter is to provide an overview of the most frequently reported acute injuries in basketball. Acute injuries are defined as a trauma resulting from a specific, identifiable event compared to an overuse, gradual-onset-type injury, with no specific, identifiable etiology [8]. This chapter will cover the epidemiology of common basketball injuries, mechanisms of injury, and risk and protective factors for each level of play and an overview of the most common injuries encountered while playing basketball.

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19.2 Descriptive Epidemiology and Mechanisms of Injury with Predictive Factors by Level of Play

Many studies have evaluated the types and frequency of injuries associated with playing basketball [9–15]. Some studies focus on children, adolescents, and high school students [16, 17]; others analyze injury rates in only collegiate student-athletes [18] or professional athletes [10, 19–21]. A general consensus of their findings follows.

19.2.1 High School Athletes

According to a descriptive epidemiologic study in 2007 assessing basketball-related injuries in American high schools, of the 409,958 injuries, the most common body sites injured were the foot and ankle (39.7%), knee (14.7%), head/face/neck (13.6%), arm/hand (9.6%), and hip/thigh/upper leg (8.4%) [22]. Upper extremity injuries only accounted for 12–13% of the injuries sustained. Though the authors included chronic injuries in their assessment, they report that the majority of injuries were new (83.6%) as opposed to a flare or recurrence of a prior injury.

The most frequent injury diagnoses reported by Borowski et al. were ligament sprains (44.0%), muscle/tendon strains (17.7%), muscle contusions (8.6%), fractures (8.5%), and concussions (7.0%). Fractures most commonly occurred in the lower arm/hand (42.7%), head/face/neck (23.9%), and foot/ankle (19.0%) regions [22].

With regard to gender differences, Borowski et al. found that girls were more likely to sustain a knee injury and that boys were more likely to sustain an injury to their trunks and foot/ankle regions [22]. However, a more recent data surveillance study revealed that the most commonly injured body parts for girls were ankle, knee, and head/face [15] compared to ankle, head/face, and knee in high school boys [23]. See Table 19.1 for a detailed comparison of injury locations.

Although the difference was not significant, girls were more likely to sustain injuries requir-

Table 19.1 Locations of injuries in high school girls and boys basketball according to the High School Sports-related Injury Surveillance Study, United States, 2005–2006 and 2006–2007 academic years

Types of injuries	High school girls (%) <i>n</i> = 211,535	High school boys (%) <i>n</i> = 198,829
Head, face, and neck	14.2	12.8
Trunk	4.0	7.1
Upper arm, shoulder	2.5	2.8
Lower arm, hand	9.5	9.4
Hip, thigh, upper leg	8.7	8.2
Knee	18.2	10.6
Lower leg	4.9	3.4
Ankle, foot	35.9	43.2
Other ^a	2.2	2.4

^aOther includes respiratory illness, internal organ damage, and so forth [23]

ing surgery than their male counterparts, particularly for ligament sprains (10.2% and 2.9%, respectively) and muscle/tendon strains (10.1% and 5.0%, respectively). They were over two times more likely to require surgery for knee ligament injury (injury proportion ratio; IPR 2.74, 95% CI 1.21–6.23, $P < 0.01$). Boys were found to be 4.43 times (95% CI, 1.25–15.68 $P < 0.01$) more likely to require surgery for head/face/neck fractures [22].

According to Borowski et al., one-quarter of all injuries sustained in high school athletes occurred while rebounding (25.1%), while on defense (14.8%), ball handling/dribbling (8.9%), and shooting (8.5%) [22]. Boys were more likely to sustain an injury while rebounding (IPR 1.72; 95% CI, 3.98–20.63; $P < 0.01$), and girls were more likely to be injured while handling the ball (IPR 1.68; 95% CI, 1.10–2.55; $P = 0.02$). Another study found that 22.6% of girls' injuries occurred during rebounding and 20.1% while playing defense in competitions [23].

Currently, there is no consensus regarding the effect of position on injury rates in high school athletes. Among male and female high school basketball players, Borowski et al. found that the guard position accounted for the most injuries (50.3% and 45.9%, respectively) followed by the

forward position (34.7% and 40.8%, respectively) and the center position (14.1% and 13.0%, respectively) [22]. They found that injury type and location did not vary by position: guards, forwards, and centers all sustained “ligament strains” and “ankle/foot” injuries most frequently [22]. Clifton et al. found that an ankle sprain due to contact with another player was the most frequent injury among all positions for both boys and girls [15, 23].

Fact Box

Noncontact knee sprain was the third most common injury among female high school and college guards [23].

19.2.2 The National Collegiate Athletic Association (NCAA)

Compared to high school basketball players, female and male collegiate basketball players had a higher total injury rate (IRR 2.73, 95% CI 2.60–2.86 and IRR 3.43, 95% CI 3.28–3.59, respectively) [15, 23].

Similar to high school athletes, NCAA Division I, II, and III basketball players were also most likely to sustain injuries to their lower extremities with lower extremity injuries accounting for 60% of all game and practice injuries from 1988 to 1989 through 2003 to 2004 seasons [24, 25].

Fact Box

Male collegiate basketball ranks fourth behind football, wrestling, and soccer in overall time-loss injury rates [26].

Collegiate athletes were more likely to be injured during game competition than practice [24, 27]. Injury rates during games were found to be twice that of those during practice: men’s and women’s basketball players experienced 9.9 and

6.75 injuries per 1000 athlete exposure (AE), respectively, during a game compared to 4.3 and 2.84 injuries per 1000 AE, respectively, during practice.

Mirroring the injury rates found at the high school level, the most common injury experienced by male and female collegiate basketball players is lateral ankle sprain. Knee internal derangement is the second most common injury followed by patellar tendinopathy and muscular strains [13, 18]. Dick et al. reported the same injury profile breakdown with the most common injuries to occur during men’s NCAA games being ankle sprains (26.2%), knee internal derangement (7.4%), patellar injuries (2.4%), upper leg contusion (3.9%), and concussion (3.6%) [25]. For women, the breakdown for injuries during game competition reported by Agel et al. is as follows: ankle ligament sprains (24.6%), knee internal derangements (15.9%), concussions (6.5%), and patellar problems (2.4%). See Fig. 19.1 for comparison of major injury locations in collegiate basketball players (both men and women). The most common practice injuries reported include ankle ligament sprains (26.8%), knee internal derangements (6.2%), and patellar injuries (3.7%) [24].

At the high school level, girls were more likely to sustain injuries requiring surgery compared to boys. Zuckerman et al. found that this trend continues at the collegiate level with the severe injury rate being higher for women compared to men (RR = 1.93; 95% CI 1.24–2.99): 9.4% of female NCAA basketball injuries were severe versus 5.1% of male injuries. However, the rate of injuries requiring surgery did not significantly differ between men and women (0.30 vs. 0.32/1000 AE; RR = 0.92; 95% CI 0.68–1.24) [13].

For all positions on men’s and women’s teams, the most frequent injury was ankle sprain due to contact with another player [15, 23]. For collegiate guards, the second most frequent injury was concussion (secondary to collision with another player). Among centers and forwards, knee sprain was the second most common injury [23]. When looking solely at ankle injuries, male and female guards experienced the highest rate of ankle injuries in competition (43.30% and 50.1%,

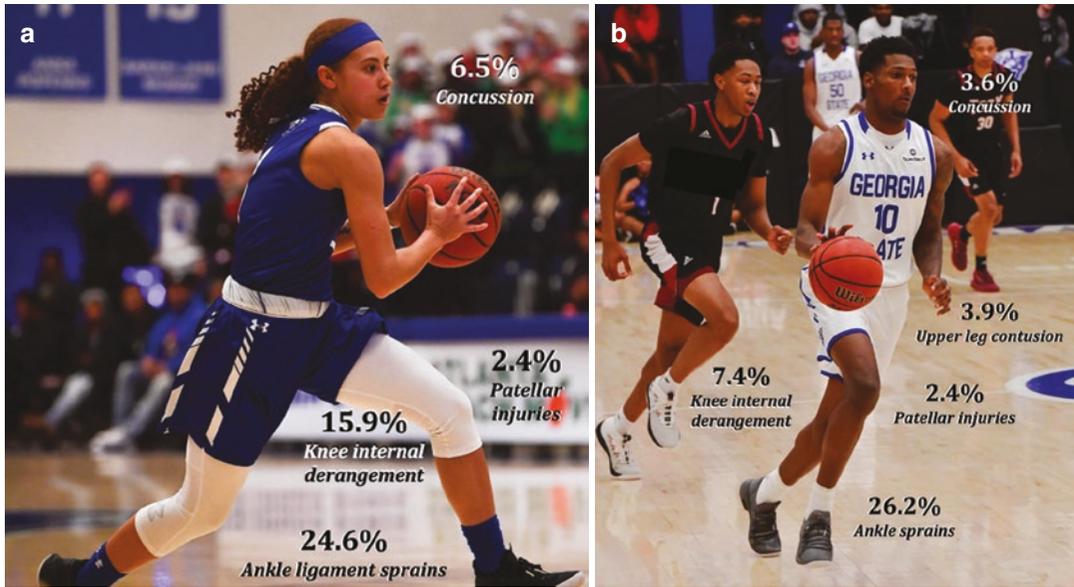


Fig. 19.1 Locations of injuries in NCAA women's (a) and men's (b) basketball injuries during game competition according to Agel et al. [24] and Dick et al. [25], respectively. Photographs provided by Alonza Ashe

respectively), followed by forwards (34.60% and 28.9%, respectively), and centers (17.86% and 16.1%, respectively). Guards had significantly higher injury rates than forwards and centers for both collegiate leagues [28].

According to a recently published 10-year NCAA surveillance study, the most common activity during ankle injury for women was rebounding (30.32%, Fig. 19.2) followed by general play (28.63%), defending (13.05%), and shooting (10.63%) [28]. For men, the most common activities were rebounding (34.36%), general play (23.57%), defending (13.10%), and shooting (11.63%). More than half of the ankle injuries sustained by women and men from 2004 to 2014 were secondary to contact (61.26% and 67.41%, respectively) [28]. Interestingly, women were found to be injured more often through acute noncontact mechanisms (IPR 1.20, 95% CI 2.06–1.35) and less often because of contact (IPR 0.88, 95% CI 0.81–0.95).

Overall, Tummala et al. found that 41.2% and 43.8% of all women's and men's ankle injuries, respectively, resulted in a time loss of less than 3 days. More than 60% of ankle injuries resulted in a time loss of less than 7 days and around 30% of injuries resulted in time loss of greater than

7 days [28]. Lastly, women were more likely than men to have a recurrent ankle injury (IPR 1.77, 95% CI 1.22–2.57).

19.2.3 Professional Leagues

Although fewer studies have been conducted on injury rates in professional basketball players over the last two centuries [20, 21, 29, 30], improved data surveillance has helped with injury evaluation at the professional level. Compared to NCAA basketball, professional athletes compete on a longer court, for more minutes per game, and for a longer season. Such factors could contribute to the increased number of injuries experienced at the professional level. However, falsely reporting injuries to keep players on injured reserve (allowing teams to carry more players on their rosters) could also affect the accuracy of reporting at the professional level. Nonetheless, professional basketball players in the USA and abroad tend to experience a higher injury rate compared to other levels [20, 21, 29, 30]. The incidence of injury in National Basketball Association (NBA) players was found to be 19.1 per 1000 AEs by Drakos et al. [21].



Fig. 19.2 The most common activity during ankle injury for collegiate women and men basketball players was rebounding (30.32% and 34.36%, respectively) based on a 10-year NCAA surveillance study. More than half of all

the ankle injuries sustained from 2004 to 2014 were secondary to contact (61.26% for women and 67.41% for men) [28]

When compared to the Women's National Basketball Association (WNBA), NBA players were found to have a lower game-related injury rate (19.3 vs. 23.9 injuries per 1000 AE) [29, 31].

Fact Box

There was *no* correlation between injury rate and age, height, weight, or years of NBA experience for male professional basketball players reported over the last 30 years in the NBA [20, 21].

Again, lower extremity injuries are the most common, accounting for over 60% of all injuries at the professional level [21, 27]. The incidence of ankle sprain (3.2 per 1000 AE) is more than two times as common as any other injury in the NBA [21]. Lateral ankle sprains, specifically, are the most common injury diagnosis in both the NBA and WNBA [21, 29].

Not much data is available on injury rate breakdown by position player in the NBA. According to an analysis of Brazilian professional basketball players, centers were found to have the highest number of injuries (44.1%) followed by forwards (35.3%) and guards (20.6%) [32]. Compared to other positions, centers sustained more hand, chest, and abdominal trauma and had a higher number of ankle sprains. They were most likely to be injured when in the paint (key area), where more physical contact occurred during rebounding or offensive drives [32]. Among Greek female professional players, the largest number of injuries occurred in and around the key area (56.3%, $P = 0.007$). Among the player positions examined, centers had the highest rate of injury with small forwards having the lowest rate of injury [33].

McKay et al. found that among elite Australian female basketball players, ankle injuries were responsible for more than half of the total time missed due to an injury [34]. In NBA players, patellofemoral injuries were the most common

reason for games missed (17.5% of all causes) [21]. As reported by Drakos et al., patella and knee injuries (e.g., internal derangement) resulted in more games missed than injuries to the ankle and the lumbar spine (the two most commonly injured areas) [21].

Fact Box
 Drakos et al. found that lumbar spine injuries (making up 10.2% of the injuries) were responsible for almost as many NBA games missed as ankle injuries (6729 vs 6838 games, respectively) [21].

have been linked to ankle injuries [37, 38]. The most frequent traumatic mechanism in low-ankle sprains is an inversion injury with the foot in slight plantar flexion, most commonly occurring through contact (e.g., when a player steps on the foot of another player). It can also result from an awkward land directly on the court or a twisting injury sustained while cutting, turning, or pushing off.

In a 10-year injury surveillance study of ankle injuries in the NCAA, the most common diagnosis for male and female players was an injury to the lateral ligamentous complex (LLC; low-ankle sprain) including the anterior talofibular ligament (ATFL) and the calcaneofibular ligament (CFL). In men, 79.97% of ankle injuries involved the LLC, 7.24% involved the deltoid ligament, and 7.01% involved the anterior tibiofibular syndesmosis (high ankle sprain) [28]. The majority of women experienced LLC tears as well (83.47%). See Fig. 19.3 for X-ray and magnetic resonance images corresponding with an LLC injury. Syndesmotic injuries were the second most common injury in women (7.05%) followed by deltoid ligament tears (5.57%).

Inversion injuries result in sprain or disruption of the ATFL most commonly followed by the calcaneofibular ligament (CFL). Such injuries can occur with concomitant injuries that may be radiographically occult, including chondral or

19.3 Common Types of Basketball Injuries

19.3.1 Foot and Ankle Injuries

19.3.1.1 Ankle Sprains

Among male and female basketball players of all ages, lower extremity injuries predominate, with the ankle being the most frequently injured anatomic site and low- or lateral ankle sprains representing the most common injury. Studies indicate that basketball players change motion every 2.0–2.82 s and jump up to 35–46 times per game [35, 36]. Frequent direction changes and jumping

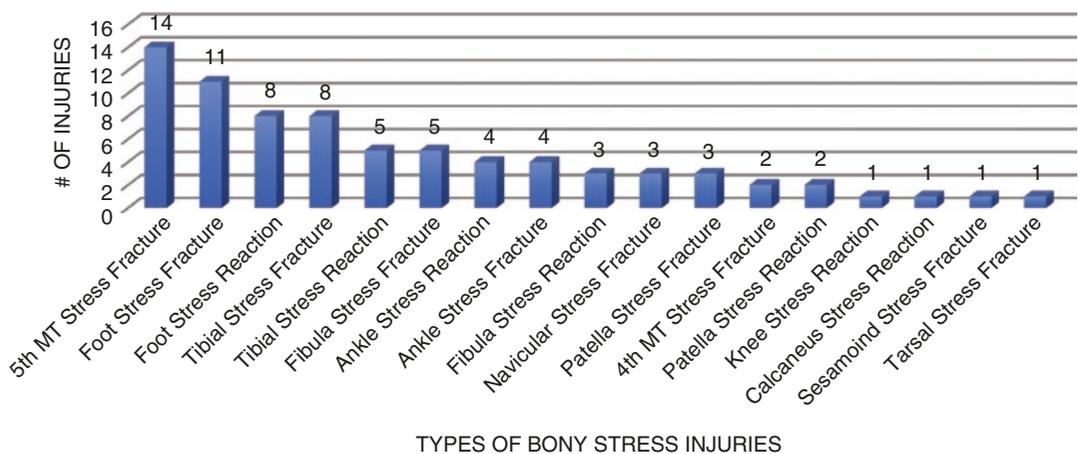


Fig. 19.3 Types of bony stress injuries of NBA athletes from 2005 to 2015 [45]

osteochondral lesions of the talar dome [39]. Additionally, fractures of the anterolateral process of the calcaneus and os trigonum and tears to the superior peroneal retinaculum (SPR) with subluxation of the peroneal tendons can sometimes mimic a lateral ankle sprain. Thorough clinical evaluation of both ankles along with advanced imaging can help identify related pathologies.

Eversion injuries are less common; however, they also occur secondary to dorsiflexion with foot eversion or external rotation. These injuries, often resulting in high ankle sprains, may be more serious because of injury to the deltoid ligament, the anterior tibiofibular ligament, and the interosseous membrane with disruption of the syndesmosis.

Fact Box

For both male high school and collegiate athletes, the most frequent injury across all positions on the court was ankle sprain secondary to player–player contact.

Preventative measures at all levels of play have focused primarily on ankle inversion injuries. Players generally wear mid- or high-top sneakers, and many are encouraged to tape their ankles or wear braces. External ankle support has been shown to decrease the risk of ankle sprains [40, 41]. Studies have shown that external ankle support in the form of a brace or taping may prevent injury [29, 42, 43]. Kofotolis et al. found that in the Greek WNBA, most injuries occurred when players did not wear ankle supports [33]. Given that a history of strain or sprain on the same side of the ankle is a strong predictor for reinjury, athletes who have a history of ankle sprain would benefit from wearing external support on that ankle. Studies have shown that athletes with a prior sprain who wear a brace or tape have a lower incidence of ankle sprain [29, 40, 42, 44–48].

19.3.1.2 Fractures and Stress Fractures of the Foot

Fractures of the anterolateral process of the calcaneus occur when the foot is abducted and plantar flexed. This mechanism places tension on the bifurcate ligament, which connects the anterolateral calcaneus to the cuboid and navicular [49]. Acute dorsiflexion with strong contraction of the peroneal muscles can also result in tears to the SPR. In this case, a small fragment of bone may avulse from the fibula where the SPR was previously attached (seen as a flake sign on plain radiographs). With disruption of the SPR, the peroneal tendons are allowed to sublux anteriorly out of their groove.

Avulsion fractures of the navicular and base of the fifth metatarsal can also present with symptoms similar to low-ankle sprains. Avulsion fractures of the fifth metatarsal usually result from inversion stress. For example, when a player goes up for a rebound and lands on the foot of another player. The same strong contraction that contributes to the rupture of the SPR can result in an avulsion of the peroneus brevis attachment at the base of the fifth metatarsal. Though not as common, an avulsion at the origin of the extensor digitorum brevis (EDB) muscle from the lateral calcaneus can also occur.

Stress injuries are also common occurrences in the NBA. In a study by Khan et al. in 75 NBA players, 76 lower extremity bony stress injuries were found. Over half (55%) involved the foot with most injuries occurring during regular season [50]. Specifically, the most commonly reported injury was a stress fracture to the fifth metatarsal (18.4%) followed by other stress fractures of the foot (14.5%) as seen in Fig. 19.4 [45]. The least reported stress fractures to the foot were to the calcaneus sesamoids and the tarsal bones [45]. Over a third (38.2%) of all stress injuries reported by Khan et al. were managed surgically. Fifth metatarsal fractures were treated surgically 100% of the time with open reduction internal fixation. Despite all being surgically managed, Khan et al. found that fifth metatarsal stress fractures resulted in the inability to return to play in 42% of the players [45].



Fig. 19.4 Mortise view X-ray of the right ankle following an inversion injury. Soft tissue shadows noted, but no bony avulsions. MRI demonstrates a full-thickness ATFL

tear at the talar insertion and signal within the deltoid ligament suggesting partial tearing. Signal in adjacent lateral sided soft tissues

19.3.2 Achilles Tendon Injuries

In the NBA, a majority of Achilles tendon ruptures occur early in the season [51, 52]. The most common mechanism of injury is taking off from a stopped position just prior to toe-off in a dorsiflexed foot. Achilles tendon rupture is a devastating injury with low return to play rates for athletes who underwent Achilles surgery [53]. Compared with other surgical procedures, NBA players who underwent Achilles tendon repair had a significantly greater decline in post-operative performance outcomes at 1- and 3-year postoperative time points and had shorter career length compared with other procedures [53]. Despite outcomes and low return to play rates in professional basketball players following Achilles tendon repair, surgery is still the

most promising option available in this patient population.

19.3.3 Acute Knee Ligament Injuries

19.3.3.1 Anterior Cruciate Ligament (ACL) Tears

ACL disruption is a serious injury with significant loss of playing time and a long post-op rehabilitation program. Busfield et al. demonstrated that 22% of NBA players failed to return to competition and nearly half (44%) of those who did return experienced a lower player efficiency rating [54].

Most orthopedic injuries affect male and female basketball players equally; however, ACL injuries in basketball players show a strong

female preponderance [55]. In a review of injuries sustained on the University of Connecticut's basketball teams, women experienced ACL injuries at two to four times the rate of their male counterparts [27]. In a metaanalysis of the incidence of ACL injury as a function of gender and sport, the female to male ratio was highest 3.5:1 in basketball compared to 2.67:1 in soccer, and 1:1 in alpine skiing [55]. Multiple studies have evaluated potential reasons for the gender differences in ACL injuries.

The mechanism of injury of ACL injuries is more commonly noncontact, deceleration, and sudden change in direction that may cause abnormal rotation of the tibia. Valgus knee collapse also contributes, which occurs more frequently in women [56]. Increased knee abduction leads to an increase in knee abduction moment, which has demonstrated high sensitivity and specificity for ACL injury risk [57]. Attributing factors of the increased risk of ACL injuries in female athletes include a predisposition of women to land with increased knee abduction [58, 59]. The latter factor was found to be correctable with dynamic proprioceptive training [60].

It is important to note that in skeletally immature athletes, a common pattern of ACL injury is an avulsion fracture of the tibia, or less commonly the femur [61]. The chondro-osseous junction is the weakest part of the ACL complex in such patients.

19.3.4 Core Injuries

19.3.4.1 Pelvis and Hip

Soft tissue injuries to the groin and hip are moderately common in basketball players. In a 16-year longitudinal study of injuries in men's NCAA basketball, "pelvis, hip, and upper leg" injuries in the form of musculotendinous strains and contusions accounted for approximately 10% of game-related injuries and 11% of injuries sustained in practice [25]. In all data, thigh injuries were more prevalent, specifically musculotendinous strains and contusions to the adductor, rectus abdominis, hamstring, and thigh (quadriceps) muscle groups [21, 25, 31].

Jackson et al. found that strains were most frequent during the first month of the season (pre-season) with the cumulative risk being related to the length of each season and the length of a player's career [62].

Today, there is heightened awareness and understanding of intra-articular hip pathology; however, most athletic-related injuries to the hip in basketball players are extra-articular [62]. Intra- and extra-articular hip pathologies in basketball players are current topics of ongoing research.

19.3.4.2 Lower Back

Lumbar spine injuries are relatively common in basketball players and account for a substantial proportion of missed games. Interestingly, a large percentage of these injuries are classified as "muscle strains" since this is the most common presenting symptom. Muscle strains and contusion present with back pain without radiation to the lower extremities and are treated conservatively. Unless a player has radicular symptoms indicative of a symptomatic herniated disk, magnetic resonance imaging plays a limited role in lower back injuries.

In young athletes, pars interarticularis defects are an important source of lumbar pain. Pars defects with or without resultant spondylolisthesis can be detected on plain films and are frequently diagnosed by advanced imaging. Careful evaluation of the posterior spinal elements (especially L5) is critical.

In the NBA, lumbar spine injuries accounted for 6.8% of all injuries over a 10-year period. This translated into 11% of all days missed by a player for an injury. Cervical spine injuries accounted for 1.3% of the injuries, followed by sacral spine injuries (0.6%), and thoracic spine injuries (0.5%) [20].

19.3.5 Upper Extremity Injuries

19.3.5.1 Hand Fractures

Injuries of the hand and lower arm predominate over injuries to the shoulder and elbow. Injuries to the hand can be particularly devastating to

basketball players since dominant and nondominant hand function and dexterity are critical to play.

The fingers and thumb represent the most likely site of an acute orthopedic fracture in basketball players. In 2017, Morse et al. published a study assessing players on the inactive list (IL) who missed games due to hand injury or those who underwent surgery as a result of hand injury. One hundred and thirty-seven injuries were identified with 71.5% of the injuries occurring to the fingers and 28.5% of the injuries occurring to the hand [63]. Per the authors' findings, there are major injury patterns to the hand in NBA players: thumb ligament tears (e.g., ulnar collateral ligament injuries) [21, 63], metacarpal fractures, and phalangeal fractures. There was no relationship found between injury type and position [63].

The proximal interphalangeal (PIP) joints are the most frequently injured sites. At the PIP, injuries can occur to the joint capsule and can result in the disruption of ligaments and tendons as well as intra-articular fractures. Volar dislocation or forced flexion at the PIP joint can lead to acute rupture or chronic attenuation of the triangular ligament at the distal end of the central slip. Such injuries are common in basketball players [64]. Injury leads to a boutonniere deformity, in which lateral bands migrate volarly with resultant PIP joint flexion and hyperextension at the distal interphalangeal (DIP) joint.

At the DIP joint, terminal extensor tendon and flexor digitorum profundus (FDP) tendon injuries, as well as fracture/dislocations, have been reported [65]. Though more common in baseball, mallet finger injuries (the disruption of the terminal extensor tendon from the distal phalanx) often occur in basketball as a result of the ball jamming the fingertip [66]. Forceful hyperextension of the DIP joint leads to FDP avulsion, as seen with a jersey tearing away from a finger. This injury is most commonly seen in football and rugby players; however, it can also be seen when a player gets his or her finger caught on a basketball net. Additionally, "dunk lacerations" can occur secondary to the

impact of a player's hand with sharp edges of the rim or with the flange connecting the rim to the backboard [67].

As mentioned previously, injuries to the metacarpophalangeal (MCP) joints also occur, most commonly in the thumb with ulnar collateral ligament (UCL) injury. The mechanism of injury for thumb UCL injuries is abduction moment at the thumb MCP joint, which occurs most frequently with falls onto outstretched hands with the thumb abducted. Carpometacarpal (CMC) dorsal dislocations are more common on the ulnar side of the hand.

19.3.5.2 Shoulder

Shoulder injuries are relatively uncommon in basketball. At the high school level, the incidence of shoulder injuries during two seasons was 0.47 injuries per 10,000 exposures for boys and 0.45 injuries per 10,000 exposures in girls [68]. In the NBA, the most frequently identified shoulder injury diagnoses were "glenohumeral sprain," "acromioclavicular joint sprain," and "rotator cuff inflammation" [20].

19.3.6 Non-Orthopedic Injuries and Illnesses

A detailed review including epidemiology of non-orthopedic injuries is beyond the scope of this chapter. However, it is important to note the impact of non-musculoskeletal pathologies with regard to the athlete's overall health.

Injuries to the head, face, and neck often occur with a collision. Such injuries including nasal and facial fractures, eye injuries, and dental injuries made up to 13.6% of high school basketball injuries [69]. Nasal fractures represent 1.5% and 1.7% of injuries in the NBA and men's NCAA basketball, respectively. Eye injuries include eyelid lacerations, corneal abrasions, and severe orbital fractures with injury to the globe [70]. Orofacial (including dental) injuries are common in basketball [70]. In intercollegiate athletes, Cohenca et al. found a fivefold increase in risk for dental trauma in basketball players compared to football players. Authors speculate that this

finding is due to mouth guard protection in football [71].

In collegiate women's basketball, concussions accounted for 6.5% of all injuries during games and 3.7% of injuries during practice [24]. Players were three times more likely to sustain a concussion during a game versus in practice. Concussions were less frequently reported in men's NCAA competition (3.6% during games) [25] and in the NBA [20].

With respect to medical, non-orthopedic causes of missed games in basketball athletes, skin infections and illnesses related to the upper respiratory tract, gastrointestinal (GI) tract, and the eye were the most common.

Among all high school athletes, skin infections that resulted in time loss were reported at an incidence rate of 2.27 per 100,000 athlete exposures with the largest number occurring in wrestling (73.6%), followed by football (17.9%) [72]. Boys' basketball made up 1.9% and girls' basketball made up 0.6% of the skin infections [72]. Skin infections among boys' basketball players were primarily bacterial in origin (>50%), with the remaining consisting of tinea lesions (~20%) and unspecified organisms (>20%) [72]. The body part affected was not reported specifically for basketball, but among all athletes, skin infection occurred most commonly on the head/face (25.3%), followed by forearm (12.7%), upper aspect of arm (8.0%), lower leg (7.0%), knee (6.8%), and thigh/upper leg (5.7%) [72]. Return to play varied, but the most common timeframe was within 3–6 days following identified infection. Minimal data was available for professional basketball, but it was reported in the NBA that dermatologic problems occurred in less than 5% of athletes, accounting for less than 1% of games missed [20]. Data among collegiate-level athletes was not found.

Upper respiratory infection accounted for 16.7% of all injuries and illnesses in the NBA, second only to sprains (20.9%) [20]. Gastrointestinal, dental, and eye problems accounted for less than 6% combined resulting in less than 2% of games missed [20]. There was a report of an H1N1 influenza outbreak among professional basketball athletes from 18 different

countries (218 teams, 3024 players) during the 2009 H1N1 pandemic [73]. Among 29 teams in the USA, five cases were reported, and it was found that the number of players per team presented as a borderline risk factor for the emergence of H1N1 cases (OR = 1.19, 95% CI: 1.00–1.41, $p = 0.056$) [73].

Stress may also contribute to illness and games missed. In a study on women's collegiate basketball athletes, a moderately positive correlation was found between strain (overall stress demanded on the athlete for a period of a week) and monotony (defined as the variability of practices for the entire season) [74]. However, no correlation existed between the number of illnesses in relation to weekly training loads. More illnesses were reported during midterm exams and at the completion of the semester, which suggests that other life stressors play a role in collegiate athletes but not necessarily physical demand itself [74].

Lastly, and most importantly, health-care providers should understand the risk of sudden cardiac death (SCD) in basketball players of all ages. In most cases, SCD in basketball players is the result of a congenital or structural anomaly such as hypertrophic cardiomyopathy, Marfan syndrome, or myocarditis [75]. SCD is often reported in young athletes at the high school and collegiate level [76]. Prevention requires an awareness of all health-care professionals taking care of athletes and careful medical screening in prospective players [77].

19.4 Conclusion

Basketball is one of the most popular sports in the world with participation from all age and skill levels. Despite the numerous benefits to playing such a team sport, basketball players are prone to certain types of injury. The most common injuries experienced by a basketball player are those of the lower extremity, specifically the ankle. The understanding of basketball injury epidemiology is an important step in the development of evidence-based interventions and education aimed at injury prevention.

References

1. The Triangle (Springfield College Magazine). Original 13 Rules of Basketball. <https://springfield.edu/where-basketball-was-invented-the-birthplace-of-basketball> (1982). Accessed 3 Sep 2019.
2. United States Department of Labor. Sports and exercise. <https://www.bls.gov/spotlight/2008/sports/> (2008). Accessed 3 Sep 2019.
3. National Federation of State High School Associations. Participation Statistics. High school athletics participation survey. https://members.nfhs.org/participation_statistics (2018–2019). Accessed 3 Sep 2019.
4. The Sporting Goods Manufacturers Association (SGMA). U.S. Trends in Team Sports. https://www.sfia.org/reports/294_2012-U.S.-Trends-in-Team-Sports-Report (2012). Accessed 28 Aug 2019.
5. National Federation of State High School Associations. Participation Statistics. High school athletics participation survey. https://members.nfhs.org/participation_statistics (2019). Accessed 3 Sep 2019.
6. Whiteside PA. Men's and women's injuries in comparable sports. *Phys Sportsmed*. 1980;8(3):130–40.
7. DeHaven KE, Lintner DM. Athletic injuries: comparison by age, sport, and gender. *Am J Sports Med*. 1986;14:218–24.
8. Smoljanovic T, Bojanic I, Hannafin JA, Hren D, Delimar D, Pecina M. Traumatic and overuse injuries among international elite junior rowers. *Am J Sports Med*. 2009;37(6):1193–9.
9. Cumps E. Prospective epidemiologic study of basketball injuries during one competitive season: ankle sprains and overuse knee injuries. *J Sports Sci Med*. 2007;6(2):204–8.
10. Deitch JR, Starkey C, Walters SL, Moseley JB. Injury risk in professional basketball players: a comparison of Women's National Basketball Association and National Basketball Association athletes. *Am J Sports Med*. 2006;34(7):1077–83.
11. Herzog MM, Mack CD, Dreyer NA, Wikstrom EA, Padua DA, Kocher MS, et al. Ankle sprains in the National Basketball Association: 2013–2014 through 2016–2017. *Am J Sports Med*. 2019; <https://doi.org/10.1177/0363546519864678>.
12. Rodas G, Bove T, Caparrós T, Langohr K, Medina D, Hamilton B, et al. Ankle sprain versus muscle strain injury in professional men's basketball: a 9-year prospective follow-up study. *Orthop J Sports Med*. 2019;7(6) <https://doi.org/10.1177/2325967119849035>.
13. Zuckerman S, Wegner A, Roos K, Djoko A, Dompier T, Kerr Z. Injuries sustained in National Collegiate Athletic Association men's and women's basketball 2009/2010–2014/2015. *Br J Sports Med*. 2018;52(4) <https://doi.org/10.1136/bjsports-2016-096005>.
14. Ekhtiari S, Khan M, Burrus T, Madden K, Gagnier J, Rogowski JP, et al. Hip and groin injuries in professional basketball players: impact on playing career and quality of life after retirement. *Sports Health*. 2019;11(3):218–22.
15. Clifton DR, Hertel J, Onate JA, Currie DW, Pierpoint LA, Wasserman EB, et al. The first decade of web-based sports injury surveillance: Descriptive epidemiology of injuries in US high school girls' basketball (2005–2006 through 2013–2014) and National Collegiate Athletic Association women's basketball (2004–2005 through 2013–2014). *J Athl Train*. 2018;53(11):1037–48.
16. Emery CA, Meeuwisse WH, McAllister JR. Survey of sport participation and sport injury in Calgary and area high schools. *Clin J Sports Med*. 2006;16:20–6.
17. Messina DF, Farney WC, DeLee JC. The incidence of injury in Texas high school basketball: a prospective study among male and female athletes. *Am J Sports Med*. 1999;27:294–9.
18. Meeuwisse WH, Sellmer R, Hagel BE. Rates and risks of injury during intercollegiate basketball. *Am J Sports Med*. 2003;31:379–85.
19. Henry JH, Lareau B, Neigut D. The injury rate in professional basketball. *Am J Sports Med*. 1982;10:16–8.
20. Starkey C. Injuries and illnesses in the NBA: a 10-year perspective. *J Athl Train*. 2000;35:161–7.
21. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the NBA: a 17-year overview. *Sports Health*. 2010;2(4):284–90.
22. Borowoski LA, Yard EE, Fields SK, Comstock RD. The epidemiology of US high school basketball injuries, 2005–2007. *Am J Sports Med*. 2008;36(12):2328–35.
23. Clifton DR, Onate JA, Hertel J, Pierpoint LA, Currie DW, Wasserman EB, et al. The first decade of web-based sports injury surveillance: descriptive epidemiology of injuries in US high school boys' basketball (2005–2006 through 2013–2014) and National Collegiate Athletic Association men's basketball (2004–2005 through 2013–2014). *J Athl Train*. 2018;53(11):1037–48.
24. Agel J, Olson DE, Dick R, Arendt EA, Marshall SW, Sikka RS. Descriptive epidemiology of collegiate women's basketball injuries: NCAA Injury Surveillance System, 1988–1989 through 2003–2004. *J Athl Train*. 2007;42:202–10.
25. Dick R, Hertel J, Agel J, Grossman J, Marshall SW. Descriptive epidemiology of collegiate men's basketball injuries: NCAA Injury Surveillance System, 1988–1989 through 2003–2004. *J Athl Train*. 2007;42:194–201.
26. Powel JW, Dompier TP. Analysis of injury rates and treatment patterns for time-loss and non-time-loss injuries among collegiate student-athletes. *J Athl Train*. 2004;39:56–70.
27. Trojian TH, Ragle RB. Injuries in women's basketball. *Conn Med*. 2008;72:147–50.
28. Tummala SV, Hartigan DE, Makovicka JL, Patel KA, Chhabra A. 10-year epidemiology of ankle injuries in men's and women's collegiate basket-

- ball. *Orthop J Sports Med.* 2018;6(11) <https://doi.org/10.1177/2325967118805400>.
29. Deitch JR, Starkey C, Walters SL, Moseley JB. Injury risk in professional basketball players: a comparison of WNBA and NBA athletes. *Am J Sports Med.* 2006;34:1077–83.
 30. Lombardo S, Sethi PM, Starkey C. Intercondylar notch stenosis is not a risk factor for ACL tears in professional male basketball players: an 11-year prospective study. *Am J Sports Med.* 2005;33:29–34.
 31. Zelisko JA, Noble HB, Porter M. A comparison of men's and women's professional basketball injuries. *Am J Sports Med.* 1982;10(5):297–9.
 32. Moreira P, Gentil D, César O. Prevalence of injuries of Brazilian Basketball National Team during the 2002 Season. *Rev Bras Med Esporte.* 2003;9(5):258–62.
 33. Kofotolis N, Kellis E. Ankle sprain injuries: a 2-year prospective cohort study in female Greek professional basketball players. *J Athl Train.* 2007;42(3):388–94.
 34. McKay GD, Payne WR, Goldie PA, Oakes BW, Stanley JJ. A comparison of the injuries sustained by female basketball and netball players. *Aust J Sci Med Sport.* 1996;28:12–7.
 35. Matthew D, Delextrat A. Heart rate, blood lactate concentration, and time-motion analysis of female basketball players during competition. *J Sports Sci.* 2009;27(8):813–21.
 36. McInnes SE, Carlson JS, Jones CJ, McKenna MJ. They physiological load imposed on basketball players during competition. *J Sports Sci.* 1995;13(5):387–97.
 37. Nelson AJ, Collins CL, Yard EE, Fields SK, Comstock RD. Ankle injuries among US high school athletes 2005–2006. *J Athl Train.* 2007;42(3):381–7.
 38. McKay G, Goldie P, Payne W, Oakes B. Ankle injuries in basketball: injury rate and risk factors. *Br J Sports Med.* 2011;35(2):103–8.
 39. Taga I, Shino K, Inoue M, Nakata K, Maeda A. Articular cartilage lesions in ankles with lateral ligament injury: an arthroscopic study. *Am J Sports Med.* 1993;21:120–7.
 40. Olmsted LC, Vela LI, Denegar CR, Hertel J. Prophylactic ankle taping and bracing: a numbers-needed-to-treat and cost-benefit analysis. *J Athl Train.* 2004;39:95–100.
 41. Leanderson J, Wykman A, Eriksson E. Ankle sprain and postural sway in basketball players. *Knee Surg Sports Traumatol Arthrosc.* 1993;1:203–5.
 42. McGuire TA, Greene JJ, Best T, Levenson G. Balance as a predictor of ankle injuries in high school basketball players. *Clin J Sports Med.* 2000;10:239–44.
 43. Thacker SB, Stroup DF, Branche CM, Gilchrist J, Goodman RA, Weitman EA. The prevention of ankle sprains in sports: a systematic review of the literature. *Am J Sports Med.* 1999;27:753–60.
 44. Gomez E, DeLee JC, Farnley WC. Incidence of injury in Texas Girls' High School Basketball. *Am J Sports Med.* 1996;24:684–7.
 45. Payne KA, Berg K, Latin RW. Ankle injuries and ankle strength, flexibility, and proprioception in college basketball players. *J Athl Train.* 1997;32:221–5.
 46. Arendt E, Dick R. Knee injury patterns among men and women in collegiate basketball and soccer: NCAA data and review of literature. *Am J Sports Med.* 1995;23:694–701.
 47. Colliander E, Eriksson E, Herkel M, Sköld P. Injuries in Swedish elite basketball. *Orthopedics.* 1986;9:225–7.
 48. Beynon BD, Murphy DF, Alosa DM. Predictive factors for lateral ankle sprains: a literature review. *J Athl Train.* 2002;37:376–80.
 49. McDermott EP. Basketball injuries of the foot and ankle. *Clin Sports Med.* 1993;12:373–93.
 50. Khan M, Madden K, Burrus MT, Rogowski JP, Stotts J, Samani MJ, et al. Epidemiology and impact on performance of lower extremity stress injuries in professional basketball players. *Sports Health.* 2018;10(2):169–74.
 51. Lemme NJ, Li NY, Kleiner JE, Tan S, DeFroda SF, Owens BD. Epidemiology and video analysis of Achilles tendon ruptures in the NBA. *Am J Sports Med.* 2019;47(10) <https://doi.org/10.1177/0363546519858609>.
 52. Amin NH, Old AB, Tabb LP, Garg R, Toossi N, Cerynik DL. Performance outcomes after repair of complete Achilles tendon ruptures in NBA players. *Am J Sports Med.* 2013;41:1864–8.
 53. Minhas SV, Kester BS, Larkin KE, Hsu WK. The effect of an orthopaedic surgical procedure in the NBA. *Am J Sports Med.* 2016;44(4):1056–61.
 54. Busfield BT, Kharrazi FD, Starkey C, Lombardo SJ, Seegmiller J. Performance outcome of ACL reconstruction in the NBA. *Arthroscopy.* 2009;25:825–30.
 55. Prodromos CC, Han Y, Rogowski J, Joyce B, Shi K. A meta-analysis of the incidence of ACL tears as a function of gender, sport, and a knee injury reduction regimen. *Arthroscopy.* 2007;23:1320–5.
 56. Krosshaug T, Nakamae A, Boden BP, Engebresten L, Smith G, Slauterbeck JR, et al. Mechanisms of ACL injury in basketball: video analysis of 39 cases. *Am J Sports Med.* 2007;35:359–67.
 57. Hewett TE, Ford KR, Hoogenboom BJ, Myer GD. Understanding and preventing ACL injuries: current biomechanical and epidemiological considerations—Update 2010. *North Am J Sport Phys Ther.* 2010;5:234–51.
 58. Renstrom P, Ljungqvist A, Arendt E, Beynon B, Fukubayashi T, Garrett W, et al. Non-contact ACL injuries in female athletes: an International Olympic Committee current concepts statement. *Br J Sports Med.* 2008;42:394–412.
 59. Hewett TE, Myer GD, Ford KR, Heidt RS Jr, Colosimo AJ, McLean SG, et al. Biomechanical measures of neuromuscular control and valgus loading of the knee predict ACL injury risk in female athletes: a prospective study. *Am J Sports Med.* 2005;33:492–501.
 60. Hewett TE, Myer GD, Ford KR, Paterno MV, Quatman CE. The 2012 ABJS Nicolas Andry Award: the sequence of prevention: a systematic approach to prevent ACL injury. *Clin Orthop Relat Res.* 2012;470:2930–40.

61. Gaca AM. Basketball injuries in children. *Pediatric Radiol.* 2009;39:1275–85.
62. Jackson TJ, Starkey C, McElhiney D, Domb BG. Epidemiology of hip injuries in the NBA a 24-year overview. *Orthop J Sports Med.* 2013;1:3.
63. Morse KW, Hearn KA, Carlson MG. Return to play after forearm and hand injuries in the NBA. *Orthop J Sports Med.* 2017;5(2) <https://doi.org/10.1177/2325978117690002>.
64. Weiland AJ. Boutonniere and pulley rupture in elite baseball players. *Hand Clin.* 2012;28(3):447.
65. Wilson RL, McGinty LD. Common hand and wrist injuries in basketball players. *Clin Sports Med.* 1993;12:265–91.
66. Chauhan A, Jacobs B, Adnoga A, Baratz ME. Extensor tendon injuries in athletes. *Sports Med Arthrosc Rev.* 2014;22(1):45–55.
67. Kirk AA. Dunk lacerations—unusual injuries to the hands in basketball players. *JAMA.* 1979;242–5.
68. Bonza JE, Fields SK, Yard EE, Comstock DR. Shoulder injuries among US high school athletes during the 2005–2006 and 2006–2007 school years. *J Athl Train.* 2009;44:76–81.
69. Randazzo C, Nelson NG, McKenzie LB. Basketball-related injuries in school-aged children and adolescents in 1997–2007. *Pediatrics.* 2010;126:727–33.
70. Guyette RF. Facial injuries in basketball players. *Clin Sports Med.* 1993;12:247–64.
71. Cohenca N, Rogers RA, Roges R. The incidence and severity of dental trauma in intercollegiate athletes. *J Am Dent Assoc.* 2007;138:1121–6.
72. Ashack KA, Burton KA, Johnson TR, Currie DW, Comstock RD, Dellavalle RP. Skin infections among US high school athletes: a national survey. *J Am Acad Dermatol.* 2016;74(4):679–84. e1. <https://doi.org/10.1016/j.jaad.2015.10.042>.
73. Kousoulis AA, Sergentanis TN, Tsiodras S. 2009 H1N1 flu pandemic among professional basketball players: data from 18 countries. *Infez Med.* 2014;22(4):302–8.
74. Anderson L, Triplett-McBride T, Foster C, Doberstein S, Brice G. Impact of training patterns on incidence of illness and injury during a women's collegiate basketball season. *J Strength Cond Res.* 2003;17(4):734–8.
75. Maron BJ, Gohman TE, Aeppli D. Prevalence of sudden cardiac death during competitive sports activities in Minnesota high school athletes. *J Am Coll Cardiol.* 1998;32:1881–4.
76. Drezner JA, Rogers KJ. Sudden cardiac arrest in intercollegiate athletes: detailed analysis and outcomes of resuscitation in nine cases. *Heart Rhythm.* 2006;3:755–9.
77. Subasic K. Athletes at sudden risk for cardiac death. *J Sch Nurs.* 2010;26:18–25.



Head, Neck, and Face Injuries in Basketball

20

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20.1 Facial Injuries

Between 3% and 29% of all facial injuries are the result of playing sports. Though basketball is technically not a contact sport, inadvertent falls, collisions between players, and impacts with the ball result in facial injuries. Head to head impact and elbow strike to the face are common mechanisms of facial fracture [1]. Facial injuries are the third most common area of injury among United States high school athletes with declining prevalence of facial injury at the collegiate and professional levels [2]. Many of these injuries can be easily treated courtside or in the training room, but some will require emergent transfer to a higher level of care.

20.1.1 Nasal Injury

Nasal injuries in basketball include nasal bone fracture, septal hematoma, nasal laceration, and common epistaxis. Following a nasal injury, it is important to ensure there is not concomitant air-

way compromise, concussion, eye injury, or leakage of cerebrospinal fluid [3]. The nose has extensive arterial blood supply from branches of both internal and external carotid arteries. Sensation is supplied through branches of the fifth cranial nerve.

20.1.2 Nasal Bone Fracture

Fracture of the nasal bones is common in non-contact sports. Nasal bone fractures are the most common type of facial fracture [4]. Physical examination of an athlete with a suspected nasal fracture should include the following: careful observation of the nose from multiple angles to inspect for deformity, palpation of bony vault to assess for crepitus or step-off, and observation for septal deviation, mucosal laceration, epistaxis, or clear-fluid leak. High-resolution ultrasonography can be considered for courtside evaluation of nasal fractures. Ultrasonography has been shown to be superior to plain radiographs in detecting nasal fractures [4].

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Description of nasal fractures

Laterality	Fracture type	Nasal bone fracture position	Septal fracture	Fracture of adjacent facial bones
<ul style="list-style-type: none"> – Unilateral – Bilateral 	<ul style="list-style-type: none"> – Open vs closed – Displaced vs nondisplaced – Simple vs comminuted 	<ul style="list-style-type: none"> – Deviated from midline – Medial vs lateral – Impacted or telescoped – Open book (lateral splay of nasal bones) 	<ul style="list-style-type: none"> – Cartilaginous – Bony septal fracture 	<ul style="list-style-type: none"> – Orbital rim – Nasoorbitoethmoidal – Midface fracture involving zygoma or maxilla – LeFort-type fracture – Mandibular fracture

Adapted from Marston et al. (2017)

Nasal bone fractures can be reduced immediately by an experienced courtside physician. Evaluation by the surgeon should take place approximately 3–5 days following injury [3]. This delay allows reduction in post-injury swelling and facilitates a better examination by the surgeon. Immediate manual reduction in an isolated, minimally displaced nasal fracture is an option. In this case, the physician can apply medially directed, broad pressure with the thumbs along the lateralized portion of nasal bone until satisfactorily reduced. The attempt at reduction should stop if the patient cannot tolerate the procedure or satisfactory results cannot be obtained and the patient should be referred to a surgeon. Analgesia with intranasal pledgets soaked with lidocaine and oxymetazoline as well as infraorbital nerve block will improve tolerance of closed reduction procedure [5]. Nasal fractures continue to have significant mobility for at least 2 weeks following injuries, and many surgeons recommend 6–8 weeks prior to return to sport. Facial protective devices may provide adequate protection from reinjury and can potentially allow a player to return earlier once initial swelling has resolved [3, 6].

20.1.3 Nasal Laceration, Epistaxis, and Septal Hematoma

Nasal lacerations should be copiously irrigated. Superficial skin lacerations can be primarily repaired with fine (5-0 or 6-0) nonabsorbable suture. Care should be taken to determine depth of laceration and involvement of cartilage and intranasal mucosa. Deeper lesions will require

layered closure and should be performed by clinicians skilled in those techniques [4].

Septal hematoma is a potential injury that should not be missed during evaluation. A septal hematoma is a collection of blood in between the septal cartilage and perichondrium; this hematoma separates the cartilage from its nutrient source leading to septal cartilage avascular necrosis if uncorrected within 3 days. Septal cartilage avascular necrosis can lead to significant morbidity including septal abscess, septal perforation, or cosmetic deformity (saddle nose, columellar retraction, and nasal base widening) [4]. Inspection of the nasal septum should be performed with adequate lighting and preferably a nasal speculum. Septal hematomas appear as large blue- or purple-colored areas of fluctuance on one or both sides of the nasal septum [3]. Drainage of a septal hematoma needs to be performed by an otorhinolaryngologist or other skilled physician within 2 days of injury [3].

Epistaxis is very common following a blow to the nose. It most commonly occurs as a result of injury to Kiesselbach's plexus in the anteroinferior aspect of the nasal septum. Bleeding can be controlled by placing direct pressure distal to the nasal bones for 15 min. Ice packs applied to the face and nose may assist with controlling the bleeding. The application of anterior packing soaked with decongestants (i.e., oxymetazoline or phenylephrine) may help control persistent bleeding [7]. Athletes may return to play once bleeding is controlled. Uncontrolled anterior epistaxis or posterior epistaxis may require placement of a nasal balloon catheter or posterior packing. Additionally, uncontrolled bleeding may require transfer to emergency department

for thrombogenic aids and possible surgical intervention [7].

20.1.4 Facial Fractures

Fractures to the midface are uncommon injuries in basketball [1, 7, 8]. Examination should include palpation of the infraorbital rims. The zygomatic arches should be examined for widening of the midface, which can be associated with fractures. The maxillary teeth should be grasped and distracted to assess for any maxillary instability [7]. Any crepitus or deformity should prompt referral to an emergency department capable of appropriate imaging and surgical management. Mandibular fractures are also uncommon in basketball. If suspicion for mandibular injury exists, the athlete should be examined for mandibular tenderness, malocclusion, swelling, trismus, and intraoral bleeding. Examination of the teeth may reveal palpable and visible step-offs [7]. Mandibular fractures should be referred to oral and maxillofacial surgery for reduction and fixation [7]. Mandibular fractures typically require at least 8–12 weeks to heal prior to return to sport. A custom fabricated mouthguard may allow return to sport on the earlier end of that spectrum [7, 9]. It is unclear whether custom or over the counter face shields provide adequate protection of unhealed maxillofacial fractures [6].

20.1.5 Dental Injuries

Dental injuries are relatively common in basketball compared to other sports [10]. The aim of treatment for dental injuries include retaining the tooth in the dental arch, maintaining viability of dental pulp, preventing root resorption, and restoring tooth to form, function, and aesthetics [11]. Superficial injuries to teeth such as chips or fractures in enamel typically are not painful. These injuries can be returned to play with a mouthguard and routine referral to dentist for possible repair of damaged tooth. Deeper injuries to teeth involving the dentin or pulp typically are

associated with pain and risk the viability of the involved tooth. An attempt should be made, pain permitting, to align loose or displaced teeth prior to referral to a dentist. Completely avulsed teeth should promptly be replaced if possible. The avulsed tooth should be handled gently by the crown, taking care not to manipulate the root in any way. The avulsed tooth should be mildly washed with sterile saline solution prior to attempted reimplantation within 15–20 min. The reimplanted tooth should be splinted with available materials (aluminum foil, gum, putty, etc.) until patient can be seen by a dentist. If the tooth cannot be reimplanted, then it can be transported to the dentist in Hank's balanced salt solution (Save-A-Tooth), cool milk, saliva, or normal saline [11].

Lip lacerations are also relatively common. Simple lacerations can be repaired following irrigation with interrupted sutures. If bleeding is controlled, then athletes may resume play. Lacerations crossing the vermilion border of the lip should have close approximation of the border. This suture should be placed first to ensure alignment. Lip sutures are generally removed after 5 days to ensure a cosmetic result. Lacerations to the tongue should also be repaired with close attention to margin approximation to avoid a bifid appearing epithelial cleft [11].

20.2 Ocular Injuries

Approximately one-third of all eye injuries sustained in the United States is related to sport [12]. Given the contact nature of basketball, traumatic ocular injuries are relatively common, with an estimated incidence of 1 injury per 10 athlete years in NCAA men's college basketball players [13]. In fact, among all sports, basketball is the most common cause of ocular injuries, accounting for 22.6% of total sports-related ocular trauma, ahead of baseball and softball, which are second and third, respectively [14]. Most injuries are due to direct trauma from the hands or elbows of other players. A thorough physical examination is key to accurately diagnosing these injuries. Most common ocular injuries in basketball

include eyelid lacerations, periorbital contusions, and corneal abrasions. Less common injuries include traumatic hyphema, globe rupture, and orbital fractures [15]. Some injuries can be managed on the court, while others require more in-depth evaluation in a clinic or emergency department setting. Some injuries will require referral for ophthalmology evaluation.

20.2.1 Eyelid Lacerations

Facial lacerations are a common occurrence in basketball. Simple eyelid lacerations can be repaired in standard fashion by medical personnel; however, eyelid lacerations must be carefully investigated on physical examination to assess for complicating factors which would require referral. Lacerations involving the eyelid margins, tarsal plate, levator palpebrae muscle, orbicularis oculi muscle, orbital fat, lacrimal sack, or lacrimal duct should be referred for ophthalmology evaluation, as many of these injuries will require microsurgery in the operating room [16]. A course of antibiotics is typically indicated perioperatively.

20.2.2 Periorbital Contusion

Direct trauma to the eye often results in periorbital contusion. While this condition is benign, medical personnel should rule out complicating factors, such as injury to the globe or facial fractures, including orbital or midface fractures. Physical examination should include testing of ocular motion and visual acuity as well as palpation of the orbital rim and midface to assess for the presence of fractures. If no complicating factors are present, the athlete may generally return to play without restriction. Management includes icing and anti-inflammatory medication as needed.

20.2.3 Corneal Abrasions

Corneal abrasions are most frequently caused by trauma from another player's finger. These inju-

ries are more common in women's basketball compared to men's basketball, which may be related to fingernail length [17]. Diagnosis is made by fluorescein dye staining of the cornea and visual inspection under Wood's lamp. Most corneal abrasions heal without complication within 72 h. Small abrasions can be treated with ophthalmologic ointment for comfort. Large corneal abrasions and corneal abrasions in athletes who wear contact lenses should be treated with antibiotic ointment. Contact lenses should not be worn until corneal abrasion has healed. Large corneal abrasions causing decreased visual acuity should be referred for ophthalmology follow-up.

20.2.4 Orbital Fracture

Orbital fractures are uncommon injuries in basketball; however, they still comprise a significant proportion of total maxillofacial fractures. One study showed that orbital fractures accounted for 17% of all maxillofacial injuries across all sports [18]. Orbital fractures most commonly involve the orbital floor. Medial floor involvement is also quite common. These fractures are colloquially referred to as "blowout" fractures. Athletes with orbital fractures often have periorbital ecchymosis, orbital rim tenderness or step-offs, and pain with extraocular movements. Limited upward gaze suggests entrapment of the inferior rectus muscle. Proptosis and visual changes suggest retrobulbar hematoma. All athletes suspected of orbital fracture should be referred urgently for ophthalmologic and maxillofacial evaluation as they require surgical consideration. These athletes should be advised to wear facial protection, such as a rigid, transparent face shield, while participating in sport until fractures are completely healed.

20.2.5 Traumatic Hyphema

Direct trauma to the eye can cause shearing of microvasculature in the anterior chamber of the eye, which can result in a hyphema. Traumatic iritis is the most common cause of hyphema.

Physical examination will reveal layering of blood in the anterior chamber between the cornea and iris. Initial management should focus on the management of the athlete's symptoms using analgesics and antiemetics. Other suggested management strategies, including application of eye patch, use of cycloplegics, and avoidance of laying supine, have been suggested but lack evidence [19]. Most hyphemas will resolve with no intervention. However, these injuries should all be referred for ophthalmology evaluation, as a minority will be complicated by re-bleeding, staining of the cornea, and optic nerve atrophy [20].

20.2.6 Globe Rupture

Globe rupture in basketball is typically caused by high force blunt trauma. These injuries can be challenging to diagnose, as the ruptured portion of the globe is not also visible. Pain, visual loss, hyphema, pupil irregularity, and 360° subconjunctival hemorrhage should raise concern for possible globe rupture [21]. Seidel testing is often positive, as indicated by the application of fluorescein dye with subsequent leakage from the globe. CT imaging can confirm the suspected diagnosis. These patients should be immediately fitted with a rigid eye shield. Anti-emetics and analgesics should also be administered expeditiously, as Valsalva maneuvers, including vomiting, can potentially worsen the injury. Manipulation of the eye should be kept to a minimum. Emergent ophthalmologic consultation is indicated for all suspected globe injuries as they typically require operative repair. Delay in repair may be complicated by infectious endophthalmitis, which can lead to long-term visual deficits, including blindness.

20.3 Ear Injuries

Although not thought of as a major source of injuries in basketball players, ear injuries do occur and the sports physician should be equipped to manage the most common injuries. The major-

ity of ear injuries in basketball players are traumatic in nature and include ear lacerations and auricular hematomas. Tympanic membrane perforations are rare but have also been reported [22]. Recognition of complicating factors and subsequent referral for otolaryngology evaluation should be a priority.

20.3.1 Auricular Hematoma

Auricular hematomas occur when blunt trauma to the auricle, or pinna, of the ear results in the accumulation of blood in the subperichondrial space. As the perichondrium supplies the auricular cartilage with nutrients, auricular hematomas can lead to compromise of the auricular cartilage, which can result in cartilage infection and necrosis. Necrosed cartilage is subsequently replaced by fibrocartilage, which leads to the tissue deformity commonly referred to as "cauliflower ear." Management of this condition is drainage of the hematoma, which can be performed either by scalpel incision or by needle drainage although the latter method can be difficult once the hematoma coagulates. After the hematoma is drained, a bolster dressing should be sutured in place for 1 week to prevent reaccumulation of the hematoma. Bolsterless techniques, including use of magnets, have also been suggested, although these are less commonly employed. Complications are uncommon, and these injuries rarely require specialty referral [23].

20.3.2 Ear Lacerations

Simple ear lacerations can typically be repaired in the usual fashion by the sports physician. However, concomitant injuries including middle ear injuries and skull and facial fractures must be ruled out. Referral to otolaryngology or plastic surgery should be considered in the presence of complicating factors, including extensive skin or cartilage injury. Although facial lacerations in general have low incidence of infection, ear lacerations, particularly ear lacerations with cartilage involvement, should

be treated with a course of antibiotics in addition to repair.

20.3.3 TM Perforation

Tympanic membrane perforation, also referred to as ear drum rupture, is common in certain sports, particularly aquatic sports; however, it is relatively rare in basketball. The mechanism of injury in basketball is typically a direct blow to the ear. Diagnosis is made by direct visualization using an otoscope. The most common site of rupture is the pars tensa. Small perforations can be managed by most medical personnel. Treatment includes analgesics and avoidance of water in the affected ear. Large perforations, classically described as involving 25% or more of the total surface area of the tympanic membrane, and perforations complicated by infection or hearing loss should be referred for otolaryngology evaluation. Small, uncomplicated perforations should heal without intervention in as little as 2 weeks and within 6 weeks [22]. Antibiotic ear drops are typically only indicated in cases of concurrent infection.

20.4 Neck Injuries

Neck injuries in basketball can range from simple muscular strains to catastrophic spinal cord injuries (SCI). Head or neck injuries in NCAA basketball comprise 13.9% of injuries sustained across all NCAA sports, with the majority of these injuries being ocular or maxillofacial [24]. The incidence of upper extremity injuries in NCAA basketball is less common, 40%, relative to lower extremity injuries, which account for 60%. The sections below detail common neck injuries, as well as a brief description of spinal cord injuries (SCI) preparedness and management. In-depth description of specific cervical fractures is not discussed in this chapter as it is beyond the scope of this text.

20.4.1 Neck Strains

The incidence of neck strains in basketball is unknown, which may be due to underreporting.

Players may develop the injury from a single acute event or a compensatory mechanism. The acute mechanism is usually from muscular stretch. Resulting neck pain may worsen over the next several days. Neck pain has a broad differential and a thorough history and physical examination are warranted by medical personnel. Any player suffering from this type of injury will usually complain of unilateral pain that is located lateral to the cervical spinous processes and not associated with any distal radiation or upper extremity weakness. If these symptoms are present, one must consider cervical spine injury, which is discussed later in the chapter.

Upon examination of a neck strain, the examiner may find increased hypertonicity, tenderness to palpation, and range of motion may be decreased unilaterally. A thorough neurologic exam including testing of gross sensation, muscular strength, and cranial nerve function is warranted. If neurologic testing is negative, mechanism of injury is low energy, and no additional red flags are present, imaging is not typically warranted. NEXUS criteria, a decision aid for cervical imaging in trauma, may be used to determine the need for imaging. NEXUS includes five criteria to rule out the need for imaging if negative; these include absence of posterior cervical midline tenderness, painful distracting injury, focal neurologic deficit, intoxication, or altered mental status [25].

When considering the differential for neck pain, one can rule out many red-flag diagnoses; however keeping the differential broad will ensure these are not missed. A broad differential includes fracture (i.e., pars, spondylolysis, or spondylolisthesis), muscular strain, ligamentous disruption, brachial plexus injury, nerve entrapment, thoracic outlet syndrome, vascular disruption, or infection. Once these have been ruled out and the player is diagnosed with a cervical strain, treatment may be initiated. Conservative therapy is usually effective for cervical strains. This includes application of ice during the acute phase for 24–48 h, motion restriction to non-tender range of motion, and use of anti-inflammatory medications. The initial goal is pain control which is followed by return to non-painful range of motion and gentle stretching, which can then

be followed by gradual return to full range of motion. Once full range of motion is achieved and non-painful, the player can begin to return to full participation while continuing to work on strengthening the cervical paraspinal musculature [26].

20.4.2 Cervical Spine Injuries

Spinal cord injuries can be life threatening, yet rarely occur in basketball. In the United States, approximately 9% of spinal cord injuries (SCIs) are sports-related [27]. Nonetheless, adequate recognition of the injury and subsequent treatment are vital to athlete's safety. Thorough preparation and prompt recognition of SCIs are keys to proper management if the situation arises where there is a concern for SCI. The following section offers a brief description of key management preparation and execution when SCI is suspected.

20.4.3 Emergency Action Plan

Prior to encountering a scenario where SCI is suspected, medical personnel caring for a sports team should develop an Emergency Action Plan (EAP). The EAP is focused around communication of the plan for catastrophic events (i.e., cardiac, pulmonary, or SCI events). This plan ensures that in the case of SCI on the court, all team member know their role, especially when spine boarding an athlete. A detailed map of the facility entrances, exits, emergency vehicle route, and location of automated external defibrillators (AEDs) is crucial. With these plans in place and all medical personnel aware, the medical team will be prepared to efficiently execute care in these crucial situations.

20.4.4 On-Court Management

If the situation arises where there is concern for SCI, the EAP will be initiated. The initial evaluation of player involves Basic Life Support (BLS) principles. This means ensuring the patient has a

patent airway, is actively breathing, and has adequate circulation. Once these are addressed, the medical staff may begin utilizing Advanced Cardiac Life Support (ACLS) or Advanced Trauma Life Support (ATLS), depending on the situation. In addition, a thorough neurologic examination will be included in this portion of the exam. Given these principles of evaluation, it is important to ensure courtside personnel are trained in BLS as well as ACLS and ATLS. After prompt immobilization, patients with suspected unstable cervical fractures or spinal cord injuries should be transported to the nearest emergency department for advanced imaging. Immobilization techniques vary among institution, yet the 6-man lift is favorable to the log roll for immobilization [27]. Removal of equipment prior to transport is also variable among institutions, and it is recommended that medical teams discuss their approach while organizing their EAP. On the basketball court equipment is limited; however, players may still be wearing facial protection. Further definitive treatment will depend on the advanced imaging results.

20.4.5 Return to Play (RTP)

Athlete return to play following SCI is case-dependent. Brachial plexopathy, referred colloquially as "stingers," are not frequently encountered in basketball. These types of injuries are well studied with well-established return to play protocols. SCIs, however, are based upon the extent of the spinal cord involvement and the sequelae experienced by the athlete. Return to unrestricted play following SCI is not well established in the literature, given the wide variability of sequelae [28]. Therefore, a team approach and shared decision-making with the athlete would likely be best until further return to play criteria are developed.

20.5 Conclusion

Basketball players sustain minor injuries to the head and neck relatively frequently. Courtside medical personnel should be able to recognize

and treat the most common head, neck, and face injuries which occur in basketball, including epistaxis, facial lacerations, nasal bone fractures, dental trauma, corneal abrasions, periorbital contusions, and neck strains. These minor injuries can oftentimes be managed by courtside medical personnel, and the player can subsequently be returned to play safely. More serious injuries to the head and neck are less common, but prompt recognition and treatment can limit morbidity. To this end, courtside medical personnel should also be able to recognize and manage nasal septal hematoma, maxillofacial fractures, globe rupture, cervical fractures, and spinal cord injury. Emergency action plans should be utilized and rehearsed regularly with incorporation of serious head and neck injury scenarios. Medical personnel should avoid distraction from less serious injuries as this may lead to under or delayed diagnosis of more serious head, neck, or facial injuries.

References

- Murphy C, O'Connell JE, Kearns G, Stassen L. Sports-related maxillofacial injuries. *J Craniofac Surg.* 2015;26:2120–3.
- Trojian TH, Cracco AC, Hall M, Mascaro M, Aerni G, Ragle R. Basketball injuries: caring for a basketball team. *Curr Sports Med Rep.* 2013;12(5):321–8.
- Patel Y, Goljan P, Pierce TP, Scillia A, Issa K, McInerney VK, Festa A. Management of nasal fractures in sports. *Sports Med.* 2017;47:1919–23.
- Marston AP, O'Brien EK, Hamilton GS. Nasal injuries in sports. *Clin Sports Med.* 2017;36:337–53.
- Lu GN, Humphrey CD, Kriet JD. Correction of nasal fractures. *Facial Plast Surg Clin N Am.* 2017;25:537–46.
- Gandy JR, Fossett L, Wong BJ. Face masks and basketball: NCAA division I consumer trends and a review of over-the-counter face masks. *Laryngoscope.* 2016;126:1054–60.
- Leinhart J, Toldi J, Tennison M. Facial trauma in sports. *Curr Sports Med Rep.* 2017;16:1.
- Delilbasi C, Yamazawa M, Nomura K, Lida S, Kogo M. Maxillofacial fractures sustained during sports played with a ball. *Oral Surg Oral Med Oral Pathol Oral Radiol Endod.* 2004;97:23–7.
- Viozzi CF. Maxillofacial and mandibular fractures in sports. *Clin Sports Med.* 2017;36:355–68.
- Collins CL, McKenzie LB, Ferketich AK, Andridge R, Xiang H, Comstock RD. Dental injuries sustained by high school athletes in the United States, from 2008/2009 through 2013/2014 academic years. *Dent Traumatol.* 2016;32:121–7.
- Inouye J, McGrew C. Dental problems in athlete. *Curr Sports Med Rep.* 2015;14:27–33.
- Feist RM, Farber MD. Ocular trauma epidemiology. *Arch Ophthalmol.* 1989;107(4):503–4.
- Marton K, Wilson D, McKeag D. Ocular trauma in college varsity sports. *Med Sci Sports Exerc.* 1987;19(2 Suppl):S53.
- Haring RS, Sheffield ID, Canner JK, Schneider EB. Epidemiology of sports-related eye injuries in the United States. *JAMA Ophthalmol.* 2016;134(12):1382–90. <https://doi.org/10.1001/jamaophthalmol.2016.4253>.
- Youn J, Sallis RE, Smith G, Jones K. Ocular injury rates in college sports. *Med Sci Sports Exerc.* 2008;40:428–32.
- Brown DJ, Jaffe JE, Henson JK. Advanced laceration management. *Emerg Med Clin North Am.* 2007;25:83–99.
- Heimmel MR, Murphy MA. Ocular injuries in basketball and baseball: what are the risks and how can we prevent them? *Curr Sports Med Rep.* 2008;7:284–8.
- Antoun JS, Lee KH. Sports-related maxillofacial fractures over an 11-year period. *J Oral Maxillofac Surg.* 2008;66:504–8.
- Gharaibeh A, Savage HI, Scherer RW, Goldberg MF, Lindsley K. Medical interventions for traumatic hyphema. *Cochrane Database Syst Rev.* 2011;(1):CD005431. <https://doi.org/10.1002/14651858.CD005431.pub2>.
- Walton W, Hagen V, Stanley, Grigorian R, Zarbin M. Management of traumatic hyphema. *Surv Ophthalmol.* 2002;47(4):297–334.
- Pokhrel PK, Loftus SA. Ocular emergencies. *Am Fam Physician.* 2007;76:829–36.
- Eagles K, Fralich L, Stevenson JH. Ear trauma. *Clin Sports Med.* 2013;32:303–16.
- Roy S, Smith LP. A novel technique for treating auricular hematomas in mixed martial artists (ultimate fighters). *Am J Otolaryngol.* 2010;31:21–4.
- Dick R, Hertel J, Agel J, Grossman J, Marshall SW. Descriptive epidemiology of collegiate Men's basketball injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 Through 2003–2004. *J Athl Train.* 2007;42(2):194–201.
- Hoffman JR, Mower WR, Wolfson AB, Todd KH, Zucker MI. Validity of a set of clinical criteria to rule out injury to the cervical spine in patients with blunt trauma. National Emergency X-Radiography Utilization Study Group. *N Engl J Med.* 2000;343(2):94–9.

26. Mjaanes JM, Lee J. Cervical strains, 5-minute sports medicine consult. Philadelphia, PA: Walters Kluwer Health Lippincott Williams & Wilkins; 2011.
27. Puvanesarajah V, Qureshi R, Cancienne JM, Hassanzadeh H. Traumatic sports-related cervical spine injuries. *Clin Spine Surg*. 2017;30(2):50–6.
28. Molinari RW, Pagarigan K, Dettori JR, Molinari RJ, Dehaven KE. Return to play in athletes receiving cervical surgery: a systematic review. *Global Spine J*. 2016;6(1):89–96. Published online 2016 Jan 5. <https://doi.org/10.1055/s-0035-1570460>.



Concussion Management in Basketball

21

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Fact Boxes

- The mechanical force that causes SRC can be from a direct hit to the head, or from a hit to another area of the body where the force is transmitted to the head.
- The symptoms from SRC can happen immediately or evolve over time (minutes to hours).
- There is a large range of clinical signs and symptoms, and the resolution of these is typically sequential.

- The diagnosis of SRC should be made by a licensed medical professional.
- All athletes should undergo pre-participation evaluations along with education on concussions prior to the start of the season.
- Studies have shown that delayed concussion reporting can result in a longer return-to-play time versus immediate reporting.

- Although previously thought that athletes should rest until the symptoms of concussion have resolved, new data shows that sub-symptom exercise should be incorporated after 24–48 h of rest.
- The Buffalo Concussion Treadmill Test (BCTT) is when the athlete walks on a treadmill with increasing speed and incline until concussive symptoms return—this heart rate is used to determine the target heart rate for the exercise prescription.
- Prescribing subsymptom threshold aerobic exercise within 1 week of concussion results in a reduced incidence of delayed recovery beyond 30 days.

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- Persistent post-concussive symptoms are symptoms that continue beyond the expected recovery window (more than 2 weeks in adults, more than 4 weeks in children).
- Repetitive SRC can lead to increased length of recovery, the possibility of sustaining a concussion through a less forceful injury, as well as increased severity of the concussive symptoms.
- CTE is considered a progressive neurodegenerative disease and with symptoms including behavior and mood problems and impaired cognitive function, and there is currently no direct link between it and concussions.

21.1 Introduction

Sport-related concussion (SRC) has become an important topic of concern for the public, media, and sports medicine physicians alike. Large sporting bodies are turning to medical professionals including physicians and researchers to lead the way in keeping athletes safe while participating in sport. SRC is associated more so with sports such as football or ice hockey; however, youth league through the professional-level basketball athlete is also at risk. This chapter will focus on SRC in basketball.

21.2 Description/Definition

The definition of SRC has been evolving for 50 years [1]. The separation of SRC from concussion due to other causes, such as motor vehicle collisions, has been important to both sports medicine physicians and sport governing body organizations so that appropriate guidelines for keeping athletes safe can be determined. The most recent definition as discussed at the fifth International Conference on Concussion in Sport places SRC on the spectrum of traumatic brain injury (TBI), the cause of TBI being induced by a

biomechanical force [2]. This, however, does not describe what is actually known regarding what is happening in the brain. Current research is focused on the following areas of the definition including structural change, physiologic change, and grading of severity [1–3]. The current definition was founded using expert opinions, the lowest level on the hierarchical system of classifying evidence when practicing evidence-based medicine [4].

Common features have been found in the literature that can be helpful in further defining SRC. These include the following:

- The mechanical force that causes SRC can be from a direct hit to the head, or from a hit to another area of the body where the force is transmitted to the head.
- SRC has not been shown to result in a structural change on imaging studies and therefore is thought to be more of a physiological disturbance.
- The symptoms from SRC can happen immediately or evolve over time (minutes to hours).
- There is a large range of clinical signs and symptoms (could or could not involve loss of consciousness (LOC)), and the resolution of these is typically sequential.
- These signs and symptoms cannot be explained by drug, alcohol, medication use, other injuries, or other comorbid conditions [1–3].

The two main focuses of the definition are on the areas of biomechanics and clinical signs and symptoms [1–3]. Discussed in further detail in the below section on pathogenesis, most of the biomechanical data that we currently have are from studies done using helmet-based technology. Using this technology, studies are focusing on impact location, linear and rotational acceleration, and head motion [5]. Unfortunately, at the current time the data from these studies varies widely and has not been able to be used as part of the diagnostic process. At some point, this data will also have to be extrapolated to the multitude of sports where concussions occur on a frequent basis and there is no use of helmets—including

basketball. The clinical signs and symptoms of a concussion are also widely variable, further complicating the ability to both define and diagnose SRC. Clinical signs typically involve physical, cognitive, and emotional symptoms that can vary both in presentation and severity. Creating a set of clinical criteria would be an important step in providing some clarity to a confusing disease process.

21.3 Epidemiology

SRC has been reported to affect about 300,000 Americans every year, the majority occurring in children and adolescents less than 20 years old [6]. This is thought to be an underestimate due to the difficulty in identifying SRC and subtle injuries. There have been studies done that show around 10% of adolescent non-athletes sustain concussions in comparison to 20% of adolescent athletes; however, this number varies depending on the source [7]. Data in the years leading up to 2011 reported around 11.6 million American children were playing the sport of basketball [6]. The number of male and female athletes participating in high school basketball has stayed fairly stable over the 10 years from 2005 to 2014, while the number of collegiate athletes has increased over this time [8].

Basketball is typically listed in the top 5 for incidence of concussion when classified by sport [9–11]. This information has been able to be obtained due to the start of Web-based sports injury surveillance programs across multiple levels including high school and the NCAA. When reviewing the current literature on concussions for high school basketball from 2005 to 2014, injury rate for practice in girls was 0.11 (injury rate/1000 AEs) and competition 0.74, whereas for boys was 0.08 and 0.33, respectively. This puts concussions at the fourth and fifth leading cause of injury in practice for both girls and boys high school basketball behind ligament sprains, muscle/tendon strains, and fractures, and the second leading cause of injury in competition behind only ligament sprain [8, 12]. In collegiate basketball from

2005 to 2014, injury rate for practice in women was 0.35 and competition 1.00, whereas for men was 0.45 and 1.26, respectively. This is similar to what is seen in high school basketball with concussion being in the top 5 leading cause of injury in practice and again second to only ligament sprain in competition [8, 12]. This data can be further divided into Collegiate by Division, practices in preseason, regular season, and post-season; however, the data remain similar across the board—concussions are consistently one of the major causes of injury to basketball players.

From 2006 to 2014, there were an average of 15 concussions per season in the National Basketball Association [13]. Data looking at athletes entering the WNBA combine from the years 2000 to 2008, of 500 professional women's basketball players 7.1% had suffered a concussion [14]. It is difficult to compare this data to that of the NBA; however, it is known that due to the longer NBA season, compared to collegiate basketball, that men's professional basketball players are more likely to experience injury [13].

21.4 Pathophysiology/ Biomechanics

The pathophysiology of concussion is not fully understood at this time. Most of the studies performed have been done using animal models; however, with the increased use of Diffusion Tensor Imaging and Functional Magnetic Resonance Imaging, more research has been able to be performed including comparison studies of imaging on animal models and humans [15]. It is currently thought to include many events such as neurometabolic changes, ionic flux and energy mismatch, cerebral blood flow changes and even pituitary dysfunction [15–17]. This cascade starts at impact, with this impact being broadly defined as being less severe than an impact that would cause cranial fracture or hemorrhage, and with the impact taking place on the head or elsewhere on the body where the force is transmitted to the head [2].

This impact delivered to the brain leads to acceleration and deceleration forces on the neuronal structures which set off a complex cascade of neurochemical and neurometabolic events: stretching of neuronal cell membranes and axons, and unregulated outflow of ions through what were once regulated ion channels. With the release of neurotransmitters such as glutamate, there is also simultaneous increase in the activity of the Na/K ATP-dependent pump to work to return to ionic balance. Unfortunately, this results in depleted energy stores with the increased use of glucose, all of which is thought to be the cause of post-concussive symptoms. Often these are self-limited, but repetitive injury could result in more prolonged deficits [15]. Along with this process, it is postulated that there is an injury-related decrease in resting cerebral blood flow which adds to the energy mismatch. This decrease in cerebral blood flow has been shown in a few studies to return to normal as the athlete is recovering from post-concussive symptoms [16, 17].

Using the proposed pathophysiologic changes discussed above, research is shifting into studies looking at biomechanics to attempt to improve the process of diagnosing concussions, staging them by severity and creating appropriate return to play protocols. Most biomechanical studies have been performed in sports in which athletes are wearing helmets because of the use of helmet-based technology and sensor systems. These studies look at the location of impact on the head, frequency, and the motion of the head after impact. Even when looking at large meta-analysis studies of linear and rotational forces exerted by certain impacts, there was difficulty in showing that impacts above a certain force always resulted in a concussion. There were many instances in which extreme forces resulted in no diagnosis, and lower forces resulted in a concussion diagnosis. Other factors that played into these studies include the differences in forces when looking at males versus females, as well as forces affecting the pediatric versus the adult population. When looking at the epidemiologic data reported on concussions in basketball, there are many ways that these concussions take place including player

to player contact as well as player to floor contact. The most common cause found in recent studies is player to player contact, frequently being elbow-to-head contact during rebounding [5, 18]. Unfortunately, there are not a lot of biomechanical studies on player to player contact in basketball to look at forces, impact location, and motion after impact due to players not wearing helmets. Some studies have looked into mouth-guard technology as well as using human head modeling to begin to gather data that could be better extrapolated to sports without helmets. Still yet are studies in the early stages of examining blood biomarkers [2, 3].

It is unlikely at this current time that we are able to use this data in a meaningful way to help with diagnosis, staging severity, predicting outcomes, or determining appropriate return to play. Much more work is needed to be able to use this data in the clinical setting, and especially in sports not involving helmets, athletes of both sexes, and athletes of all ages, such as basketball.

21.5 Diagnosis

As discussed when examining the definition of SRC, it is a complicated injury in which there is no “one size fits all” group of symptoms athletes present with, level of impact force is not black and white, and there are also no proven diagnostic tests including imaging scans or blood work to make a final diagnosis. In this section, we will discuss how to recognize and make the diagnosis of concussion from the sideline and in the office, appropriate physical exam techniques and helpful tools that can be used to aid your clinical judgment.

The diagnosis of SRC happens on the court, ideally by a licensed medical professional. There are many signs and symptoms that the medical professional should be aware of observing athletes during practice and competition from the sidelines. The diagnosis of concussion is difficult and currently based on clinical assessment by the provider. There are many tools available which are unvalidated and therefore medical

professionals will need to rely mostly on history and physical exam.

Before the season begins, all athletes should undergo a preparticipation physical evaluation, and part of this should include a detailed concussion history and the presence of conditions such as a learning disorder, ADHD, mood disorder, headache disorder, and what medications the athlete is currently taking as these can complicate both the diagnosis and management of SRC [2, 3, 19]. This should also include putting into place an Emergency Action Plan (EAP) should a situation arise on the court where an athlete would need immediate emergency services. There are also baseline tests that can be performed which include a symptom checklist, cognitive evaluation, and balance testing which is currently recommended by the NCAA for all athletes [19]. Baseline testing is helpful in some situations; however, it is not considered the standard of care for SRC. Part of the process of the preseason evaluation should include educating athletes on concussion including handouts with information about concussions [3]. Most athletes do not understand the risks associated with concussions and want to do whatever they can to return to the court with their teammates. Studies have shown that delayed concussion reporting can result in a longer return-to-play time versus immediate reporting [20].

After an athlete has sustained an initial injury concerning for concussion, they should be immediately evaluated for an injury that could require a higher level of care. The evaluation should focus on whether or not the athlete has lost consciousness, the development of tonic posturing, a balance disturbance, or cervical spine trauma. All of these would constitute an emergent situation where the athlete should be immediately transported to the nearest Emergency Department as outlined in your EAP. Other signs should also signal transfer to a higher level of care including a neurological deficit on your exam, recurring emesis, and worsening headache or mental status over time. It is important to remember that SRC is an evolving process, and symptoms can change over minutes to hours, highlighting the need for serial examination.

If there are no concerns for need for emergent transfer, the athlete should be evaluated by the medical professional on the sideline. Remember that this sideline evaluation should be performed with the goal of screening for a possible SRC, and not making a final diagnosis. If after the brief screening on the sideline, a concussion is no longer suspected then the medical professional can determine the appropriate timing for the athlete to return to play. As SRC is an evolving injury, serial exams can be performed prior to making official decision on returning the athlete to the activity. If the sideline screening is concerning for SRC, then a full evaluation should take place in a quiet and distraction-free environment such as the medical training room or the locker room. It is important to remember to be extremely cautious when making the decision to return to play from the sideline, keeping the athlete from returning to the game or practice if there is any slight suspicion for concussion is important [2, 3, 21, 22].

After the athlete has been removed from play, the evaluation should begin with a brief history of the event from the athlete and whether or not the athlete is experiencing symptoms such as a headache, feeling of being in a fog, or emotional symptoms (extreme anger, tearful). The athlete should be evaluated for balance or gait unsteadiness, slowed reaction times, drowsiness. Speech patterns and information processing should be inferred during this time of general assessment. A complicated aspect of the sideline diagnosis is the need to rely on the athlete to provide honest answers to symptom-based questions. It can be helpful if there is concern from the medical professional about the athlete's description of symptoms, to move the athlete to a quiet area away from the court, such as the locker room or training room, and perform a more thorough exam. Also, if the athlete is experiencing any of the previous listed clinical findings and SRC is suspected, a full concussion assessment should be performed [2, 3, 21, 23]. Other important points to remember are that if there is not a licensed medical professional at the event, and SRC is suspected, the athlete should be removed from play and not allowed to return until evaluated by a medical professional. An athlete

that has been diagnosed with SRC should never return to activity on the same day as their diagnosis. This can be important in basketball where teams could be playing in multiple games, or having multiple practice sessions in the same day.

If you are evaluating the athlete for the initial encounter in an office-based setting, this should include a comprehensive history of the injury as well as initial symptoms and any change in symptoms since time of injury. The office-based physical exam should include some evaluation in the domains of neurocognition, vestibular ocular function, gait, balance, cervical spine, along with a full neurologic exam. The above evaluation should help the clinician confirm the diagnosis of SRC versus other causes from similar symptom combinations. If the athlete's symptoms have resolved, a discussion of return to learn/work as well as return to graduated activity may be implemented. If the athlete's symptoms are still present, it is important to provide guidance on symptom trajectory, early treatment interventions, and continued abstention from sport.

Many clinical tools exist to aid in the diagnosis of concussion. There is currently no definitive evidence on the performance of sideline tests, and expert opinion is again the best level of evidence we have in most areas of concussion evaluation and treatment. Currently expert opinion leans heavily toward multimodal testing such as the Sport Concussion Assessment Tool or SCAT, which is currently in its fifth iteration (however important to note that studies have not been done comparing this version to previous iterations to determine superiority) [2, 3, 23–25]. Unfortunately, all sideline screening tools are laden with the high risk of bias because of the marked heterogeneity in diagnostic accuracy. Discussed below are some of the most common and most studied concussion assessment tools. These should always be used in conjunction with good clinical judgment and assessment by a trained medical professional.

21.5.1 Sport Concussion Assessment Tool (SCAT)

The SCAT has been around since the 2000s and has gone through multiple revisions since that

time with the most current being the SCAT5 which came about with the fifth International Consensus Conference on Concussion in Sport. The SCAT combines multiple approaches including an immediate/acute assessment section which includes indications for emergency management, a rapid neurological screen, a graded symptom checklist, standardized assessment of concussion (SAC) cognitive testing—immediate and delayed word recall and repeat digits backwards, Maddocks questions, balance assessment with a modified Balance Error Scoring System (mBESS). The SCAT5 is designed to only be used by medical professionals as it is not designed to be used separately from the assessment of the athlete by the trained professional. There is a component referred to as the Concussion Recognition Tool 5 (CRT5) that has been developed for non-medically trained lay persons to identify possible SRC. The SCAT5 should take no less than 10 minutes to administer and is recommended for athletes aged 13 and older, with a separate Child SCAT5 for athletes aged 5–12. With changes to the word recall list and digits backwards testing to increase the number of words and proactively randomize the numbers given, there are hopes that this will improve the ceiling effect that is seen in older athletes, or athletes who have been rumored to memorize word lists. Unfortunately, there is little information on this test being used with athletes with disabilities, or athletes that speak a language other than English [2, 3, 24].

21.5.2 Balance Error Scoring System (BESS)

The BESS is a test of balance which involves the athlete completing three 20 second stance trials: double leg (hands on hips with feet together), single leg (using nondominant leg with hands on hips), tandem stance (nondominant foot behind dominant foot) on both firm and foam surfaces. The athlete's eyes should be closed during the test and errors are counted (opening eyes, hands coming off of hips, falling out of position, turning the hips more than 30 degrees, and being unable to return to the original position in more than 5 sec-

onds). This test has been evaluated in studies in basketball athletes of both genders and all ages. There does seem to be differences in athletes testing ability dependent on sport (better performance in sports where balance is extremely important to performance such as gymnastics). It has been shown to have moderate to good reliability [26]. The SCAT5 uses the modified version (mBESS) without the foam surface component. It would be prudent to use the full BESS protocol when available for greater diagnostic accuracy.

21.5.3 King-Devick Test (K-D)

The K-D test is a 2-minute test performed by the athlete reading single-digit numbers that are displayed on cards or on an electronic tablet device. If the time needed to complete this post-injury is longer than previous baseline testing, this indicates a positive test and the athlete should be removed from play. Studies have been done on basketball athletes using the K-D test. Although there is some data that there is a learning curve with improvement in time after practicing the test to get a baseline, there is still data that shows that an increase in length of time to complete the test can be associated with increasing likelihood of SRC [27–29]. A newer alternative to the K-D test is the Mobile Universal Lexicon Evaluation System (MULES). It uses two pages of colored images and shows similar promise in longer time needed to complete the series vs baseline is indicative of possible SRC [30].

21.5.4 Computer-Based Neurocognitive Testing

Tests such as the Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT), Automated Neuropsychological Assessment Matrix (ANAM), and the Concussion Sentinel to name a few are computer-based tests that generate a score over multiple domains including: attention span, working memory, sustained and selective attention time, non-verbal problem solving, and reaction time, as well as a symptom scale. They are typically used by making a comparison of baseline test results to

the results obtained following SRC. A decrease in score is seen following concussion, and there has been some data showing that these tests can be done at time points after resolution of symptoms to determine if the athlete is appropriate to begin return to play protocol [31–34]. There has been debate to the test–retest reliability and validity of computer-based neuropsychological testing. Further research is needed in this area to aid in the clinician’s appropriate use of when to test athletes and how to interpret the data in return to play decisions.

21.5.5 Vestibular Ocular Motor Screen Assessment (VOMS)

This is a rapid evaluation of vestibular and ocular function where the clinician evaluates smooth pursuits, saccades, convergence, fixating on a stationary object while moving the head side to side and up and down, and standing while tracking a moving object by turning the head and torso fully side to side. This test should take around 5–10 minutes to administer. A positive test is when the athlete reports symptom provocation such as worsening headache, dizziness, or nausea after each assessment. This test can be administered serially, over time, as there is no ceiling effect and could be useful if there is a reported worsening of symptoms with the serial testing [35].

21.6 Management/Return to Play/Return to Learn

Symptoms of SRC resolve spontaneously in the majority of adults and older adolescents in around 2 weeks, with younger athletes typically taking longer to recover with return to baseline around 4 weeks. It is important to discuss this with the athlete to set expectations for the recovery process. [21, 22, 36]

The most common predictor of recovery is the number and severity of acute and subacute symptoms following the injury, having more symptoms or more severe symptoms being closely

correlated with a longer time to recovery. Headache or depression following injury is a risk factor that is often associated with increased return to baseline with symptoms sometimes lasting greater than 1 month. The same is true for athletes with a pre-injury history of depression as well as migraine headaches. ADHD and other learning disabilities have not been shown to result in increased time to recovery; however, athletes with these conditions could require increased interventions when planning the return-to-learn process [36, 37].

Some studies have been done to look at different treatment programs including multimodal rehabilitation supervised by physical therapists and vestibular rehabilitation. When reviewing data on multimodal supervised rehabilitation, which includes training in balance, musculoskeletal, aerobic, anaerobic, and sport-specific exercises, it is difficult to determine the benefits due to a wide range of methods used and unclear conclusions on whether or not athletes that participated in this received benefit in decreased time to returning to competition. Vestibular rehabilitation does have a large amount of positive evidence for improving symptoms; however, this should be limited to athletes that are experiencing specific vestibular deficits and should be targeted toward these [20, 38, 39].

The NCAA, NBA, and WNBA all have concussion policies, protocols, and best practice statements that are updated nearly annually. The NCAA has declared a concussion best practice statement and requires all member schools to have a concussion safety protocol that aligns with this. The NBA and WNBA both have policies that include education to all athletes, coaches, and training staff, appropriate evaluation and management, and return-to-play protocols [40–42].

After an athlete has been diagnosed with a concussion, that athlete should not return to activity on that same day, and should not begin a return-to-play program until deemed appropriate by a physician. The mainstay for managing SRC has been prescribed rest until the athlete is symptom-free. The idea being that rest should ease discomfort during the recovery period and also promote recovery for the brain by decreasing

demands. Athletes should be instructed to refrain from both physical and cognitive activities such as workouts, conditioning, weight training, physical education class, reading or other academic work, and limiting screen time including TV, computer, and cell phone use [2, 3, 39, 43].

Recent studies show that the old idea of “rest is best” following concussions until symptoms have completely resolved is not necessarily true. The idea of early subthreshold aerobic exercise individualized to every athlete suffering from concussion now has a significant amount of data touting its outcomes over those of complete rest. It shows that prescribing subsymptom threshold aerobic exercise within 1 week of concussion results in a reduced incidence of delayed recovery beyond 30 days. It was also shown to be safe when compared with a stretching intervention, with no athletes experiencing worsening of symptoms or prolonged recovery times. This is done by performing an exercise tolerance assessment, the most widely used being the Buffalo Concussion Treadmill Test (BCTT), at the initial visit where concussion was diagnosed by a sports medicine physician. A subsymptom threshold exercise prescription should be written to target a heart rate of 80% of what was achieved during the BCTT (when the athlete began to develop symptoms), for 20 min/day, 6–7 days/week. A new heart rate can be determined weekly as long as the participant remains symptomatic [43–46].

21.6.1 Return-to-Play (RTP)

Athletes should be seen and cleared by a physician prior to beginning an RTP protocol. Following the 24–48 hours rest period, athletes should begin with Stage One, symptom limited activity, which includes return to normal cognitive and physical activities that do not exacerbate concussion symptoms as well as a prescription for subsymptom threshold exercise with the BCTT as discussed above. For school-aged basketball athletes, this can include returning to school activities such as class and home, discussed further in the Return-to-Learn section, as well as walking to classes. Once concussion

symptoms are resolved, the athlete can proceed to the next level as long as he/she does not experience any return of concussion symptoms. Each step should take around 24 hours, resulting in completing the RTP protocol in around 1 week. The athlete will go through a progression of increasing physical demands and sport-specific

activities until completed without return of symptoms. The RTP protocol should be individualized and monitored closely. Athletes should appear back to their baseline function which for some elite athletes could be at a higher level than others [2, 3, 44]. An example protocol is outlined below:

- 1 Symptom Limited Activity — Normal activities of daily living. subsymptom threshold exercise prescription.
- 2 Light aerobic exercise — Perform a controlled activity that increases heart rate such as: Walking outside or on a treadmill, riding a stationary bike.
- 3 Basketball specific exercise — Basketball activities that increase heart rate without risk of contact. Dribbling exercises, shoot around, practicing free throws.
- 4 Non-contact Basketball drills — The goal at this stage is to increase the level of coordination and thinking involved such as passing drills, going through non-contact motion of offensive and defensive sets or out of bounds plays.
- 5 Full contact practice — Return to full contact basketball drills and practice.
- 6 Return to Game

Similar steps should be followed in Return-to-Learn. The athlete should be able to return to school without exacerbation of symptoms. Modifications should be allowed for including breaks during the school day, reduced in class and homework assignments, increased time for completing assignments, testing in quiet, distraction-free environment, limiting the use of computers or other screens, avoiding loud places such as the cafeteria or music class. It is important to remember that in athletes that are school-aged, return-to-learn should take place prior to or simultaneously with RTP (i.e., athletes should not be returned to sport if they are unable to return to the classroom) [2, 3].

21.7 Complications

There are multiple long-term complications that can arise from SRC. Some of these include persistent postconcussive symptoms, the risks associated with repetitive SRC, and lower extremity musculoskeletal injuries.

There are significant risks associated with premature return to play and delayed reporting of symptoms including continuing to play after sustaining a concussion such as more severe symptoms and prolonged recovery. In a study that looked at both basketball athletes as well as athletes from other sports, athletes that delayed the reporting of their concussion symptoms had a significantly increased number of symptoms,

severity of symptoms, duration of symptoms, and time lost to concussion. The immediate reporting of symptoms and removal from play also decreased head impacts taking place during the acute period after the original injury [43, 47].

Postconcussion syndrome, or persistent postconcussive symptoms, is defined as symptoms that continue beyond the expected recovery window (more than 2 weeks in adults, more than 4 weeks in children). These can have a significant negative impact on the daily life of the athlete. It was once recommended to continue complete rest during this time; however, more recent data has shown that subsymptom threshold aerobic exercise could be more beneficial in aiding recovery. It has been used both in attempting to prevent prolonged recovery as well as part of prolonged recovery [45, 47].

Repetitive SRC can lead to increased length of recovery, the possibility of sustaining a concussion through a less forceful injury, as well as increased severity of the concussive symptoms. Data has shown that athletes that have sustained one concussion are three times more likely to have another concussion. There has also been data to show an increased risk of lower extremity musculoskeletal injury following a concussion, and this has been shown to be a bigger risk in female athletes than in male athletes. This increased risk is thought to be due to persistent subclinical effects or alterations in neurocognition. Athletes with decreased neurocognitive performance may have a difficult time with anticipating the actions of their opponents leading to difficulty in dodging versus bracing for collision while taking a charge underneath the basket [47, 48].

Chronic Traumatic Encephalopathy (CTE) has become a growing concern in the athletic community. Although no direct link has been able to be established between CTE and concussions, most research is currently focused on linking it to repetitive brain trauma (RBT). CTE is considered a progressive neurodegenerative disease and with symptoms including behavior and mood problems and impaired cognitive function. No large studies have been performed

in basketball athletes and CTE, with most studies having been done using retired National Football League athletes. The data that is currently available does indicate that exposure to RBT is one of the largest risk factors for development of CTE [49].

21.8 Obtaining Rights to Image

21.8.1 Prevention

Although almost every area of study for concussion needs further research from the definition to the appropriate treatment, research looking into prevention strategies should also be high on the list of priorities. Concussion legislation currently exists across all 50 states in the United States, as well as implementation of the CSIG in both national and international sports organizations, including the implementation of a formal concussion protocol in the National Basketball Association [37, 41]. Some of the main strategies for primary prevention have been in both education as well as rule changes in sports. The NCAA has outlined in their guideline for managing concussions that schools provide athletes and coaches with educational materials on concussions and that a form must be signed by every athlete stating that they have reviewed these guidelines [19, 40, 50]. Educational initiatives, such as the HEADS UP campaign created by the CDC, have been attempting to increase the awareness of concussions [51]. Areas for future research focus around the risk factors for concussion (history of prior concussion, female sex), the most common mechanism for concussion (player to player contact), the use of sport-specific equipment in the role of prevention and rule changes (in basketball, changes have been made to the court including the restricted area under the basket) [50, 52]. Although not necessarily primary prevention, the number of certified athletic trainers working in the collegiate setting has also increased around 86% from 1995 to 2005 and has most certainly continued to increase from then [9]. This would ideally lead to improved

player adherence to immediate removal from competition and outcomes following an appropriate Return-To-Play protocol.

21.9 Conclusion

SRC is an important focus for medical professionals, athletes, coaches, and anyone closely involved in sport. In basketball, SRC affects players of all age ranges, both sexes, elite-level athletes in the National Basketball Association and National Collegiate Athletic Association, to youth athletes participating recreationally. Further research needs include all aspects of SRC from definition to management. Important points to remember include to err on the side of caution when suspecting SRC as it is always better to remove the athlete from competition early, and delayed removal can result in increased time for return to competition. Focus on appropriate and supervised Return-to-Play and Return-to-Learn protocols is needed to reduce the long-term risks of concussion including increased risk of musculoskeletal injury and repeat concussions. It will be important to increase the effort placed on primary prevention with education and awareness of the signs, symptoms, and sequelae of SRC for athletes and coaches.

References

1. McCrory P, Feddermann-Demont N, Dvorak J, et al. What is the definition of sports-related concussion: a systematic review. *Br J Sports Med.* 2017;51(11):877–87.
2. McCrory P, Meeuwisse W, Dvorak J, et al. Consensus statement on concussion in sport – the 5th international conference on concussion in sport held in Berlin, October 2016. *Br J Sports Med.* 2017;51(11):838–47.
3. Harmon KG, Clugston JR, Dec K, et al. American Medical Society for Sports Medicine Position Statement on concussion in sport. *Clin J Sports Med.* 2019;29(2):87–100.
4. Burns PB, Rohrich RJ, Chung KC. The levels of evidence and their role in evidence based medicine. *Plast Reconstr Surg.* 2011;128(1):305–10.
5. Brennan JH, Mitra B, Synnot A, et al. Accelerometers for the assessment of concussion in male athletes: a systematic review and meta-analysis. *Sports Med.* 2017;47(3):469–78.
6. Noble JM, Hesdorffer DC. Sport related concussions: a review of epidemiology, challenges in diagnosis and potential risk factors. *Neuropsychol Rev.* 2013;23(4):273–84.
7. Veliz P, Eckner JT, Zdroik J, et al. Lifetime prevalence of self-reported concussion among adolescents involved in competitive sports: a National U.S. Study. *J Adolesc Health.* 2019;64(2):272–5.
8. Clifton DR, Hertel J, Onate JA, et al. The first decade of web-based sports injury surveillance: descriptive epidemiology of injuries in US High School Girls' Basketball (2005-2006 through 2013-2014) and National Collegiate Athletic Association Women's basketball (2204-2005 through 2013-2014). *J Athl Train.* 2018;53(11):1037–48.
9. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. *J Athl Train.* 2007;42(2):311–9.
10. Kerr ZY, Roos KG, Djoko A, et al. Epidemiologic measures for quantifying the incidence of concussion in National Collegiate Athletic Association Sports. *J Athl Train.* 2017;52(3):167–74.
11. O'Connor KL, Baker MM, Dalton SL, et al. Epidemiology of sport-related concussions in high school athletes: National Athletic Treatment, Injury and Outcomes Network (NATION), 2011-2012 through 2013-2014. *J Athl Train.* 2017;52(3):175–85.
12. Wasserman EB, Sauers EL, Register-Mihalik JK, et al. The first decade of web-based sports injury surveillance: descriptive epidemiology of injuries in US High School Boys' Basketball (2005-2006 through 2013-2014) and National Collegiate Athletic Association Men's Basketball (2204-2005 through 2013-2014). *J Athl Train.* 2019;54(2):198–211.
13. Padaki AS, Cole BJ, Ahmad CS. Concussion incidence and return-to-play time in National Basketball Association Players: results from 2006–2014. *Am J Sports Med.* 2016;44(9):2263–8.
14. McCarthy MM, Voos JE, Nguyen JT, et al. Injury profile in elite female basketball athletes at the Women's National Basketball Association Combine. *Am J Sports Med.* 2013;41(3):645–51.
15. Barkhoudarian G, Hovda DA, Giza CC. The molecular pathophysiology of concussive brain injury – an update. *Phys Med Rehabil Clin N Am.* 2016;27(2):373–93.
16. Maugans TA, Farley C, Altaye M, et al. Pediatric sports-related concussion produces cerebral blood flow alterations. *Pediatrics.* 2012;129(1):28–37.
17. Meier TB, Bellgowan PS, Singh R, et al. Recovery of cerebral blood flow following sports-related concussion. *JAMA Neurol.* 2015;72(5):530–8.
18. Zuckerman SL, Totten DJ, Rubel KE, et al. Mechanisms of injury as a diagnostic predictor of sport-related concussion severity in football, basketball, and soccer: results from a regional concussion registry. *Neurosurgery.* 2016;63:102–12.
19. National Collegiate Athletic Association. Interassociation Consensus: Diagnosis and man-

- agement of sport-related concussion best practices NCAA. Accessed June 24, 2019.
20. Asken BM, Bauer RM, Guskiewicz KM, et al. Immediate removal from activity after sport related concussion is associated with short clinical recovery and less severe symptoms in collegiate student-athletes. *Am J Sports Med.* 2018;46(6):1465–74.
 21. Broglio SP, Cantu RC, Gioia GA, et al. National Athletic Trainers' Association position statement: management of sport concussion. *J Athl Train.* 2014;49(2):245–65.
 22. King D, Brughelli M, Hume P, et al. Assessment, management and knowledge of sport-related concussion: systematic review. *Sports Med.* 2014;44(4):449–71.
 23. Patricios J, Fuller GW, Ellenbogen R, et al. What are the critical elements of sideline screening that can be used to establish the diagnosis of concussion? A systematic review. *Br J Sports Med.* 2017;51(11):888–94.
 24. Echemendia RJ, Meeuwisse W, McCrory P, et al. The sport concussion assessment tool 5th edition SCAT5: background and rationale. *Br J Sports Med.* 2017;51(11):848–50.
 25. Alla S, Sullivan SJ, Hale L, et al. Self-report scales/checklists for the measurement of concussion symptoms: a systematic review. *Br J Sports Med.* 2009;43:3–12.
 26. Bell DR, Guskiewicz KM, Clark MA, et al. Systematic review of the balance error scoring system. *Sports Health.* 2011;3(3):287–95.
 27. Galetta KM, Brandes LE, Maki K, et al. The King-Devick test and sports-related concussion: study of a rapid visual screening tool in a collegiate cohort. *J Neurol Sci.* 2011;309:34–9.
 28. Leong DF, Balcer LJ, Galetta SL, et al. The King-Devick test for sideline concussion screening in collegiate football. *J Optom.* 2015;8:131–9.
 29. Galetta KM, Liu M, Leong DF, et al. The King-Devick test of rapid number naming for concussion detection: meta-analysis and systematic review of the literature. *Concussion.* 2015;1(2):CNC8.
 30. Fallon, et al. MULES on the sidelines: A vision-based assessment tool for sportsrelated concussion. *Journal of the Neurological Sciences.* 402:52–6.
 31. Broglio SP, Katz BP, Zhao S, et al. Test-retest reliability and interpretation of common concussion assessment tools: findings from the NCAA-DoD CARE Consortium. *Sports Med.* 2018;48(5):1255–68.
 32. Schatz P, Pardini JE, Lovell MR, et al. Sensitivity and specificity of the ImPACT test battery for concussion in athletes. *Arch Clin Neuropsychol.* 2006;21(1):91–9.
 33. Roebuck-Spencer TM, Vincent AS, Schlegel RE, et al. Evidence for added value of baseline testing in computer-based cognitive assessment. *J Athl Train.* 2013;48(4):499–505.
 34. Broglio SP, Ferrara MS, Macciocchi SN, et al. Test-retest reliability of computerized concussion assessment programs. *J Athl Train.* 2007;42(4):509–14.
 35. Mucha A, Collins MW, Elbin RJ, et al. A brief vestibular/ocular motor screening (VOMS) assessment to evaluate concussions. *Am J Sports Med.* 2014;42(10):2479–86.
 36. Iverson GL, Gardner AJ, Terry DP, et al. Predictors of clinical recovery from concussion: a systematic review. *Br J Sports Med.* 2017;51(12):941–8.
 37. Chrisman SP, Schiff MA, Chung SK, et al. Implementation of concussion legislation and extent of concussion education for athletes, parents, and coaches in Washington state. *Am J Sports Med.* 2014;42(5):1190–6.
 38. Valovich McLeod TC, Kostishak N Jr, Anderson BE, et al. Patient, injury, assessment, and treatment characteristics and return to play timelines after sport-related concussion: an investigation from the Athletic Training Practice Based Research Network. *Clin J Sport Med.* 2019;29(4):298–305.
 39. Schneider KJ, Leddy JJ, Guskiewicz KM, et al. Rest and treatment/rehabilitation following sport-related concussion: a systematic review. *Br J Sports Med.* 2017;51(12):930–4.
 40. NCAA Sports Science Institute Concussion Safety Protocol Checklist. https://www.ncaa.org/sites/default/files/2017SSI_ConcussionSafetyProtocolChecklist_20170322.pdf. Last Updated 2014. Accessed on June 26, 2019.
 41. National Basketball Association Concussion Policy Summary – 2017–2018 Season. <https://official.nba.com/wp-content/uploads/sites/4/2017/12/Concussion-Program-Summary-2017-18.pdf>. Last Updated 2017. Accessed on July 11, 2019.
 42. Official Rules of the Women's National Basketball Association 2019. <https://ak-static.cms.nba.com/wp-content/uploads/sites/27/2019/05/2019-WNBA-Rule-Book-Final.pdf>. Last updated 2019. Accessed on July 11, 2019.
 43. McCrea M, Guskiewicz K, Randolph C, et al. Effects of a symptom free waiting period on clinical outcome and risk of reinjury after sport-related concussion. *Neurosurgery.* 2009;65(5):876–82.
 44. Leddy JJ, Haider MN, Ellis MJ, et al. Early sub-threshold aerobic exercise for sport-related concussion a randomized clinical trial. *JAMA Pediatr.* 2019;173(4):319–25.
 45. McCarty CA, Zatzick D, Stein E, et al. Collaborative care for adolescents with persistent postconcussive symptoms: a randomized trial. *Pediatrics.* 2016;138(4):e20160459.
 46. Leddy JJ, Haider MN, Ellis M, et al. Exercise is medicine for concussion. *Curr Sports Med Rep.* 2018;17(8):262–70.
 47. Manley G, Gardner AJ, Schneider KJ, et al. A systematic review of potential long-term effects of sport-related concussion. *Br J Sports Med.* 2017;51(12):969–77.
 48. Herman DC, Jones D, Harrison A, et al. Concussion may increase the risk of subsequent lower extremity musculoskeletal injury in collegiate athletes. *Sports Med.* 2017;47(5):1003–10.
 49. Asken BM, Sullan MJ, Dekosky ST, et al. Research gaps and controversies in chronic traumatic encephalopathy a review. *JAMA Neurol.* 2017;74(10):1255–62.
 50. Waltzman D, Sarmiento K. What the research says about concussion risk factors and prevention strate-

- gies for youth sports: a scoping review of six commonly played sports. *J Saf Res.* 2019;68:157–72.
51. Centers for Disease Control and Prevention. HEADS UP. <https://www.cdc.gov/headsup/index.html>. Last Updated March 5, 2019. Accessed on June 27, 2019.
52. Ferguson RW, Green A, Hansen LM. *Game Changers: Stats, Stories and What Communities are Doing to Protect Young Athletes.* Washington, DC: Safe Kids Worldwide; 2013. https://www.safekids.org/sites/default/files/documents/ResearchReports/final_sports_study_2013.pdf. Accessed on June 26, 2019



C. Ellis Wisely and Terry Kim

22.1 Introduction

In recent years, many ocular injuries to famous players in the NCAA, NBA, and FIBA ranks have been publicized in the sports media. Specific examples include the shocking footage of traumatic globe luxations (protrusion of the eye from the orbit) for Akil Mitchell of the New Zealand Breakers in 2017 and Allan Ray of Villanova in 2006, Jon Scheyer's retinal tear and traumatic optic neuropathy while playing for the Miami Heat's summer league team in 2010, and Amar'e Stoudemire's retinal detachment in 2008 as a member of the Phoenix Suns. While these dramatic and rare cases are memorable, ocular injuries in basketball are common and are typically benign. According to a 2016 study of sports-related presentations to US emergency departments (EDs), >97% of basketball-related injuries resulted in no visual impairment [1]. A 2017 study in high school and collegiate athletes also demonstrated that >95% of ocular injuries resulted in competition time loss

<21 days [2]. In spite of this, a wide array of ocular injuries can occur in basketball, and it is critical each be evaluated in a systemic fashion to determine when to involve an ophthalmologist.

22.2 Epidemiology of Ocular Injuries

In the United States, more than 40,000 eye injuries occur annually during sporting events [3], and approximately 30,000 sports-related ocular injuries are evaluated each year in US EDs [1]. More than 75% of injuries are sustained by males [1, 4], and most injuries occur in individuals younger than 25 [1, 4, 5]. Among individuals evaluated in the USA, playing basketball is the most common cause of sports-related ocular injury, and basketball accounts for more than 20% of sports-related ocular injuries evaluated in the ED [1, 6]. The vast majority of basketball-related ocular injuries are caused by inadvertent player–player contact (e.g., being poked in the eye with a finger) [6]. A study of ocular injuries in American collegiate athletes determined that men's basketball had the highest event rate per minutes played for ocular injury of any sport, followed by women's basketball with the second-highest event rate [7]. A study of amateur athletes in Israel found that basketball had the second-highest event rate of ocular injury following only soccer [8]. A 2017 epidemiological study of eye injuries in high school and collegiate athletes corroborated the common nature of eye injuries in basketball, identifying

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men's and women's basketball as two of the top three sports most often associated with ocular injury [2]. This same study also found that ocular injury was nearly 3.5 times more likely to occur in competition than in practices [2]. In the professional ranks, a 1995 study of injuries in the NBA determined that eye injuries represented greater than 5% of all injuries to NBA players [9]. In this study, eyelid abrasions and lacerations were found to represent more than 50% of all injuries to the eye and ocular adnexa, and corneal abrasions were the most common injury to the eye itself.

The most common basketball-related injuries in the US pediatric ED setting included corneal abrasion and subconjunctival hemorrhage [4]. Other common injuries included laceration of the eye or ocular adnexa, hyphema, traumatic iritis, and ocular surface foreign body [4]. Among high school and college athletes, the most common ocular injuries were subconjunctival hemorrhages, making up 33–36% of ocular injuries [2]. Abrasions (16–21%) and lacerations (17–29%) were the next most common injuries.

Fact Box

- Eye injuries are common in basketball, and playing basketball is one of the most common causes of sports-related eye injury.
- Men's basketball has one of the highest event rates per minutes played for ocular injury of any collegiate sport.
- Ocular injury is nearly 3.5 times more likely to occur in competition than in practice.
- The most common basketball-related injuries include corneal abrasion and subconjunctival hemorrhage.
- The vast majority of basketball-related injuries result in no visual impairment.

22.3 Prevention

Given that ocular injuries are common in basketball, the medical community has promoted strategies to prevent ocular injuries. In 2004,



Fig. 22.1 Amar'e Stoudemire donning protective eyewear during his tenure with the NBA's New York Knicks. Stoudemire has diligently worn eye protection since his 2008 traumatic retinal detachment. The use of protective eyewear is rare among both professional and amateur basketball players (Reprinted with permission from The Associated Press)

the American Academy of Pediatrics' Committee on Sports Medicine and Fitness worked with the American Academy of Ophthalmology's Eye Health and Public Information Taskforce to develop a joint statement classifying basketball as a high-risk activity for eye injury and recommending eye protection in all pediatric basketball players [5]. Specifically, they recommended all youth basketball players wear impact-resistant eye protection (typically polycarbonate material) and strongly emphasized the recommendation for functionally monocular athletes. Despite these recommendations, the use of eye protection remains unpopular and uncommon among pediatric and adult basketball players (Fig. 22.1). Unsurprisingly, more than 96% of ocular injuries in the NBA occur in players not wearing eye protection [9].

22.4 Sideline Ocular Assessment

Non-ophthalmologist medical staff or athletic trainers are often the first responders in the assessment of ocular injuries. In this section, we provide guidelines for the assessment of ocular injuries with the caveat that even these basic eye screening techniques should ideally be learned

under the direction of an ophthalmologist. All sideline ocular assessments should include the following [10]:

1. Taking a history
 - (a) History of pre-existing ocular conditions, prior ocular injuries, or prior ocular surgery, and any eye medications.
 - (b) Detailed history of present illness with specific questioning about ocular symptoms including the following:
 - i. Mechanism of injury (e.g., finger/elbow to the eye)
 - ii. Changes in visual acuity
 - iii. Eye pain
 - iv. Double vision
 - v. Seeing flashes of light or floaters
 - vi. Loss of a portion of the visual field (sometimes described as a “curtain” over the vision)
2. Visual acuity testing
 - (a) Ideally performed with a Snellen eye chart (at distance) or hand-held vision test card (at near).
 - (b) Test each eye separately with the contralateral eye occluded.
3. Assessment of pupillary light response.
 - (a) Can be performed with a penlight or flashlight.
 - (b) Assess for round pupil shape, direct and consensual responses in each eye, and relative afferent pupillary defects.
4. Gross inspection of the ocular surface and eyelids.
 - (a) Evaluate for lacerations or globe deformity.
 - (b) Consider using topical anesthetic eye drops (e.g., proparacaine or tetracaine) to examine uncomfortable patients.
5. Confrontational visual fields [11].
 - (a) Assess each eye separately with the contralateral eye occluded.
 - (b) Compare testing to any pre-existing deficits reported.

Abnormalities on pupillary examination suggest optic nerve injury, and deficits on confrontational visual fields suggest retinal or optic nerve injury. Abnormalities noted on any of the tests above

should prompt consultation with an ophthalmologist. If the 5-point assessment above is normal, we find most athletes are able to immediately return to play.

A detailed review of assessment for and implications of specific findings above (e.g., relative afferent pupillary defects) is beyond the scope of this chapter. For more detail for those with limited experience conducting eye exams, we suggest the American Academy of Ophthalmology’s *Basic Ophthalmology*, now in its tenth edition. For readers with a basic understanding of the assessment of ocular injuries who seek details on diagnosis and treatment of ocular injuries, *The Willis Eye Manual: Office and Emergency Room Diagnosis and Treatment of Eye Disease*, now in its seventh edition, is our favored reference.

22.5 General Return to Play Guidelines

In a 2015 article on return to play criteria, Canty and Nilan raised several general questions for determining whether an athlete can return to play that are particularly salient for ophthalmic injuries [12]. We explore several of those questions below.

1. Has an accurate diagnosis been established?
 - For ophthalmic injuries, courtside diagnosis is often difficult due to limited availability of slit-lamp biomicroscopy. Oftentimes, ophthalmology consultation for a detailed examination, including a slit-lamp exam, intraocular pressure check, and dilated fundus exam, is required. Thus, like Canty and Nilan, we recommend involving an ophthalmologist prior to return to play for athletes with visual changes or abnormal exam findings [12].
2. How high is the risk for further injury with return to play?
 - Some ocular injuries like hyphema or globe rupture carry a high risk of worsening in severity with early return to play, while other injuries like subconjunctival

hemorrhage carry minimal risk with expedited return. This reinforces the importance of accurate diagnosis of ocular injury. Thus, diagnostic uncertainty should preclude return to play until an ophthalmologist is consulted.

3. Is equipment available that might prevent further injury?
 - Most common ocular injuries including corneal abrasion can be reliably prevented from worsening with protective eyewear. Protective eyewear is also effective for primary injury prevention, with injuries occurring very rarely when protective eyewear is used [9, 10].
4. Does the athlete feel confident returning to play?
 - Common ocular injuries may be temporarily vision limiting, affecting a player's ability to perform upon return. Thus, the athlete's confidence in his ability to return should be assessed.

Fact Box

- Any abnormalities on the sideline ocular assessment should prompt consultation with an ophthalmologist.
- Given the possibility of exacerbating certain ocular injuries, return to play should not occur until an accurate diagnosis of the injury.

22.6 Common Eye Injuries

22.6.1 Corneal Abrasion

A corneal abrasion occurs when the superficial layer of the cornea, the epithelium, is scraped off, typically by trauma from another player's finger. The cornea is the most densely innervated tissue in the body, and corneal abrasions are typically associated with sharp, intense pain. Mildly blurred vision can also be associated depending on the abrasion's proximity to the corneal center. The corneal epithelium plays a role in protecting the deeper corneal layers from pathogens; thus, abrasions pre-

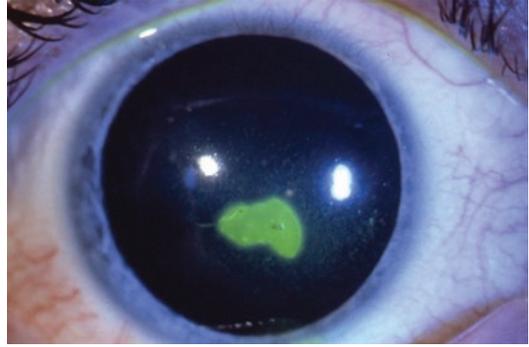


Fig. 22.2 Slit-lamp photograph of an eye with a corneal abrasion under diffuse blue illumination. Note the prominent yellow fluorescein dye staining in the area of denuded corneal epithelium (Reprinted with permission from Slack Incorporated from *The Pocket Guide to Cornea* [13])

dispose patients to infection. Corneal abrasions are best diagnosed by placing fluorescein dye onto the corneal surface and illuminating the cornea with blue light (Fig. 22.2). Fluorescein stains areas where the epithelium has been denuded.

22.6.1.1 Treatment

While corneal abrasions are painful in the short term, they typically heal without intervention and without lasting effects on visual acuity. Additional corneal trauma, which often occurs from reactive eye rubbing, may enlarge the defect and prevent healing. Most ophthalmologists recommend treatment of corneal abrasions with topical antibiotic drops until the epithelial layer has regrown over the defect (often within 1–2 days). Topical antibiotic ointments and artificial tear drops are often added to improve comfort. In some cases, bandage contact lenses can be placed to provide immediate improvement in comfort and vision; however, patients should be monitored for increased risk of corneal infection with contact lens use. We recommend patients with corneal abrasions be followed with serial slit-lamp exams until the epithelial defect has resolved.

22.6.1.2 Return to Play

Corneal abrasion is not a contraindication to an immediate return to play. Ability to return is often determined by patient comfort and visual acuity. Topical anesthetic eye drops may be used to improve patient comfort immediately after

corneal abrasion and are sometimes used to facilitate expedited return to play in the short-term setting. However, topical anesthetics can be toxic to the corneal epithelium, delaying the healing process [14]; thus, we do not advocate their prolonged use. Bandage contact lenses are appropriate for decreasing pain and facilitating return to play in certain cases.

22.6.2 Subconjunctival Hemorrhage

A subconjunctival hemorrhage is a contusion of the conjunctival ocular surface. It occurs when a hemorrhage accumulates between the conjunctiva, the clear epithelial surface tissue of the eye, and the sclera, the rigid white layer of the eye which provides structure to the globe (Figs. 22.3 and 22.4). Subconjunctival hemorrhage can cause mild irritation but is visually insignificant. While

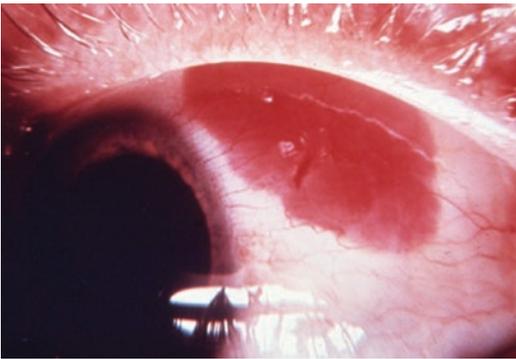


Fig. 22.3 Focal subconjunctival hemorrhage (Image courtesy of the Wills Eye Hospital)

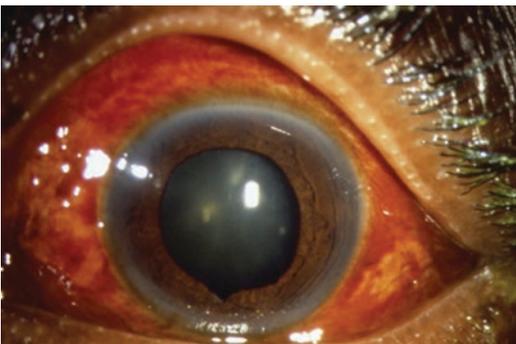


Fig. 22.4 Diffuse, 360-degree subconjunctival hemorrhage. Chemosis (swelling and elevation of the conjunctiva) is also present (Image courtesy of the Wills Eye Hospital)

patients may find the red appearance to be disconcerting, this injury is typically benign. While subconjunctival hemorrhage is common after ocular trauma, it is also common after Valsalva maneuvers including forceful coughing or sneezing.

22.6.2.1 Treatment

No treatment is required for a subconjunctival hemorrhage as it will self-resolve as the blood reabsorbs. Patients should be advised that it can take a few to several weeks for the blood to completely reabsorb, similar to the expected healing time for a contusion elsewhere in the body. If the conjunctiva is elevated by a large hemorrhage, patients often have irritation and complain of a foreign body sensation. We recommend treating these symptoms with frequent lubrication using artificial tear drops and/or ointment.

22.6.2.2 Return to Play

Immediate return to play is typically safe in cases of subconjunctival hemorrhage as there is no significant risk of visual loss. One caveat is that extensive subconjunctival hemorrhage, particularly cases where the hemorrhage extends 360° around the limbus (where the clear cornea meets the sclera), may obscure more severe injuries to deeper ocular tissues such as underlying scleral rupture and open globe injury. Thus, if there is uncertainty in the diagnosis of subconjunctival hemorrhage, return to play should be delayed until after examination by an ophthalmologist.

22.6.3 Ocular Surface Foreign Body

Ocular surface foreign bodies are common in basketball. Eyelashes and displaced contact lenses are two of the most commonly encountered foreign bodies. Patients will typically present with acute onset of ocular irritation.

22.6.3.1 Treatment

While many foreign bodies can be removed grossly at courtside, foreign bodies that are particularly small, trapped beneath the upper eyelid, or embedded in the conjunctiva or cornea usually require the involvement of an ophthalmologist. In cases where ocular surface foreign bodies are

suspected, we suggest first flushing the eye with copious quantities of sterile saline solution to facilitate foreign body removal. If available, we then suggest slit-lamp examination including eyelid eversion. Upper lid eversion is particularly important to expose hidden foreign bodies. If blurred vision or corneal opacity is present, suspicion for an embedded corneal foreign body is raised, and an ophthalmologist should be consulted for foreign body removal.

22.6.3.2 Return to Play

If an athlete is asymptomatic after ocular surface foreign body removal, immediate return to play is indicated. Suspicion for an embedded corneal foreign body is a contraindication to an immediate return to play.

22.6.4 Eyelid Laceration

Facial lacerations are common in basketball, and if they do not involve the eyelids, primary closure may be performed without involvement of an ophthalmologist. If the eyelids are involved, even for superficial lacerations, we recommend consultation with an ophthalmologist. Lacerations involving the medial aspect of the upper or lower eyelid raise concern for the involvement of the canalicular system (tear drainage system), which may require specialized repair (Fig. 22.5). Lacerations involving the eyelid margins raise concern for tear film and corneal surface issues if not accurately reapproximated. Full-thickness lacerations, or lacerations where orbital fat can be seen protruding from the wound, suggest the possibility of injury to the levator muscle or other orbital structures and also require the urgent ophthalmology consultation [10, 15].

22.6.4.1 Treatment

While superficial lacerations can undergo simple primary closure, the complex anatomy of the eyelids necessitates examination by an ophthalmologist if the eyelids are involved. Most eyelid lacerations can be repaired under local anesthesia and may be appropriate for repair in the locker room setting if surgical supplies are available.



Fig. 22.5 Medial lower eyelid laceration involving the canalicular system. The lower punctum can be seen near the medial edge of the lower lid tissue that is separated from the globe. The injury was sustained from an accidental elbow to the face during play. This injury required operative repair with an oculoplastics specialist under general anesthesia

Some full-thickness lacerations, lacerations involving the eyelid margin, and lacerations involving the canalicular system may require repair in the operating room under general anesthesia. Canalicular lacerations often require the involvement of an oculoplastics specialist. If deeper orbital tissues such as orbital fat are seen protruding from the wound, we recommend a CT scan to assess for deeper injuries including orbital bone fractures [15]. Some ophthalmologists routinely recommend oral antibiotics after eyelid lacerations [15]. Tetanus prophylaxis should also be considered.

22.6.4.2 Return to Play

For superficial lacerations, immediate return to play after primary closure is typically appropriate. Recommendations for returning to play should be case-specific depending on the extent of the injury.

22.6.5 Hyphema

A hyphema occurs when blood partially or completely fills the anterior chamber of the eye

(the fluid-filled space between the clear cornea and the pigmented iris, Figs. 22.6 and 22.7). Hyphemas typically occur after blunt trauma. While most hyphemas clear spontaneously without intervention, there are several associated vision-threatening risks. First, blood in the anterior chamber can clog the trabecular meshwork (the drainage system of the eye located in the anterior chamber angle) causing elevated intraocular pressure. Elevated intraocular pressure can lead to acute ocular pain and development of permanent visual loss from glaucoma. Second, in cases where hyphemas are slow to clear, red blood cells can stain the corneal endothelium, leading to permanent corneal opacities and impaired vision. Third, blood in

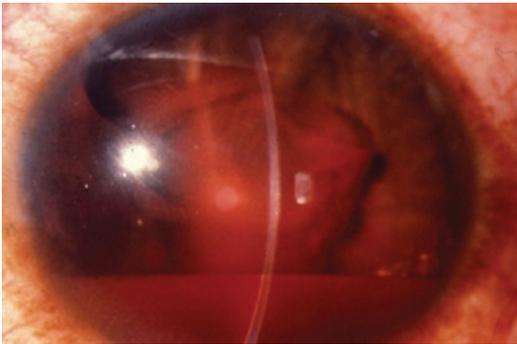


Fig. 22.6 A layered hyphema is seen in the inferior portion of the anterior chamber. Clumped blood products are noted obscuring the pupil in the central visual axis (Image courtesy of the Wills Eye Hospital)

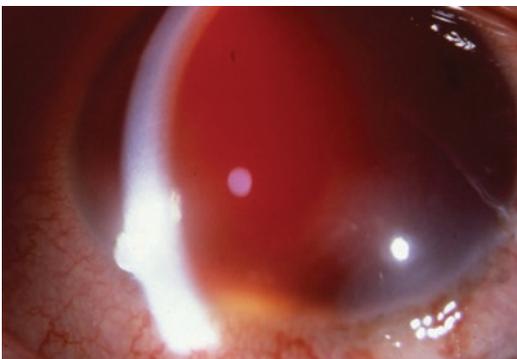


Fig. 22.7 An “8-ball hyphema,” wherein the entire anterior chamber is filled with blood. Eight-ball hyphemas are associated with a higher risk for endothelial blood staining and intraocular pressure spikes

the anterior chamber can lead to secondary inflammation and uveitis, with subsequent development of anterior chamber synechiae. Finally, rebleeding risk is a concern, and risk for rebleeding may persist up to 10 days after initial injury [16].

22.6.5.1 Treatment

Hyphemas should be managed closely by an ophthalmologist, initially with daily exams including intraocular pressure checks. It is important to limit early return to play to prevent the possibility of rebleeds. A rigid eye shield is typically prescribed to prevent the eye from repeat trauma within 10 days of the injury. Hyphemas are also managed with topical medications, typically including steroids and cycloplegics. Steroids prevent reactive inflammation and cycloplegics prevent frequent contraction and expansion of the ciliary muscle, which may exacerbate bleeding. Additionally, elevation of the head of the patient’s bed is often recommended to allow the hemorrhage to settle out of visual axis in gravity-dependent fashion [16].

Fact Box

- Rebleeding with possible progression to an 8-ball or complete hyphema is a serious concern, particularly within the first 10 days after injury.
- Given the potential vision-threatening effects of a rebleed, athletes with hyphemas should be held out of competition for at least 10 days and should wear a rigid eye shield during this period to prevent recurrent trauma.

22.6.5.2 Return to Play

Return to play is unsafe until the hyphema has resolved. In most cases, even if the blood is no longer visible in the anterior chamber, we recommend athletes wait at least 10 days before returning. Due to the rebleed risk, we provide strong recommendations that athletes use protective eyewear for at least a month after injury.

22.6.6 Traumatic Iritis

Iritis is marked by the presence of inflammatory cells (white blood cells) floating in the anterior chamber of the eye. Prominent symptoms include pain, eye redness, tearing, and photophobia. Presentation of traumatic iritis can be delayed after the initial injury, sometimes up to 3 days [15]. Slit-lamp exam is required for diagnosis as the white blood cells circulating in the anterior chamber are microscopic. While court-side history and examination can be suggestive of traumatic iritis, it is nearly impossible to diagnose definitively in this setting. Suspicion for this condition should prompt referral to an ophthalmologist.

22.6.6.1 Treatment

Treatment with topical steroids should be initiated to relieve symptoms and prevent damage to surrounding ocular structures like the iris and the lens by intraocular inflammation. Cycloplegic eye drops are also used to improve patient comfort. Topical steroids should be tapered carefully after resolution of inflammation under the direction of an ophthalmologist.

22.6.6.2 Return to Play

In most cases, we recommend return to play once symptoms have improved with topical steroid therapy, which typically occurs within 1–2 days.

22.6.7 Commotio Retinae

Commotio retinae is defined as an area of relative retinal whitening presenting after traumatic injury (Fig. 22.8). Retinal vasculature typically appears normal around the area of whitening, but adjacent retinal hemorrhages may be seen [15]. Commotio retinae is diagnosed via dilated funduscopy examination by an ophthalmologist. When commotio retinae occurs in the peripheral retina, there are typically no associated symptoms. Thus, many commotio retinae cases likely go undetected. However, cases with injury closer to or in the macula may be accompanied by blurred vision or even permanent visual impairment [17].

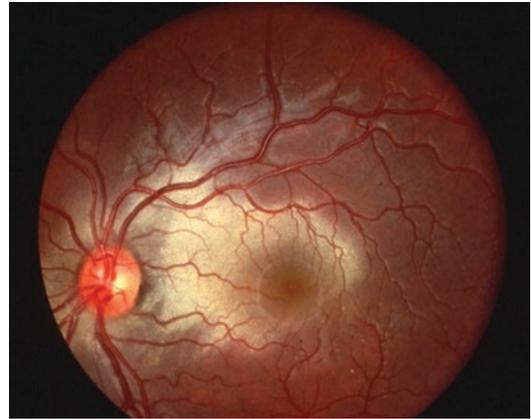


Fig. 22.8 Funduscopy image of a patient with commotio retinae affecting the macula after ocular trauma. Commotio retinae with macular involvement is also referred to as Berlin's Edema (Image courtesy of the Wills Eye Hospital)

Commotio retinae is thought to be a contrecoup injury wherein forces on the anterior globe from blunt trauma are transmitted to the posterior globe, with subsequent damage to the photoreceptor layer of the retina [18]. Whitening is thought to be produced by edema in the retinal pigment epithelium subsequent to photoreceptor damage [15, 18, 19].

22.6.7.1 Treatment

Most cases of commotio retinae resolve without intervention with excellent visual outcomes [17]. Perhaps the most critical aspect of evaluation and treatment of commotio retinae is establishing with certainty that no other concomitant posterior segment injuries such as a retinal tear, retinal detachment, or choroidal rupture are present [15]. Definitively ruling out other retinal injuries typically involves funduscopy examination with scleral depression, and, in certain cases, the involvement of a retina specialist may be required. We recommend providing strict return to clinic precautions for these patients, indicating they should return for urgent ophthalmic examination if they experience any symptoms associated with retinal detachment, including new floaters, flashes of light, a sensation of a "curtain" over their vision, or a new visual field defect.

22.6.7.2 Return to Play

Comotio retinae without visual impairment is not a contraindication to return to play. However, we strongly recommend a thorough dilated eye examination and considering a consultation with a retina specialist prior to return to play.

22.6.8 Ruptured Globe Injury

Ruptured globe injuries can occur from blunt or sharp trauma. While ruptured globes are rarely encountered in basketball in the absence of pre-existing ocular conditions [20] or in patients with a history of ocular surgery, they can be devastating injuries resulting in severe visual impairment. Full-thickness lacerations can be apparent in the cornea or the sclera, and some ruptured globe injuries may be obscured by overlying subconjunctival hemorrhage. The limbus (where the cornea and sclera meet, Fig. 22.9) and the posterior aspect of the extraocular muscle insertions on the sclera are common locations for globe rupture. 360° of subconjunctival hemorrhage, shallowing of the anterior chamber, low intraocular pressure, peaking of the pupil toward the area of injury, or the presence of vitreous in the anterior chamber are all suggestive of ruptured globe injury. Any suspicion for ruptured globe injury should prompt emergent evaluation by an ophthalmologist.

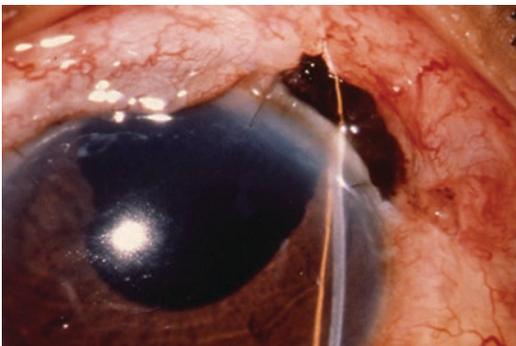


Fig. 22.9 Ruptured globe injury is present at the supero-temporal limbus. Prolapse of intraocular contents (uveal tissue) is noted at the injury site. Note that the pupil is distorted with peaking toward the site of the rupture (Image courtesy of the Wills Eye Hospital)

22.6.8.1 Treatment

In the event of suspicion for ruptured globe injury, further ocular examination or manipulation should be aborted until an operative repair is undertaken. Manipulation of a ruptured globe may result in prolapse of ocular contents, increasing endophthalmitis risk and complicating repair of globe rupture. A rigid eye shield should be placed over the injured eye to protect it from further injury until operative repair. Aggressive pharmacologic control of pain and nausea is recommended to prevent patients from straining and performing Valsalva maneuvers that may exacerbate intraocular tissue prolapse. Orbital CT scan is recommended in most cases to rule out intraorbital or intraocular foreign body [15]. Patients with globe rupture should be transported promptly to a facility capable of performing operative repair within 24 h. Broad-spectrum systemic antibiotics are typically recommended until the time of operative repair in an effort to prevent endophthalmitis [21]. Studies suggest that endophthalmitis rates are approximately 5–12% in cases of open globe injury [21, 22].

22.6.8.2 Return to Play

Any suspicion for globe rupture is an absolute contraindication to return to play, or to any significant physical activity. Athletes suffering open globe injury will likely require a prolonged recovery period prior to return to play, given the high risk of repeat rupture in the postoperative period.

22.6.9 Retinal Tear and Detachment

A traumatic retinal detachment typically occurs via rhegmatogenous mechanism, wherein globe trauma causes a tear or break in the retina, and fluid from the vitreous cavity accesses the subretinal space through the tear, causing elevation of a portion of the retina. There is associated visual loss corresponding to the area where the retina is lifted off of the retinal pigment epithelium. Traumatic retinal tears are rare events in basketball [23], and retinal detachments are even more uncommon.

Symptoms include the development of new floaters, flashes of light, and the sensation of a curtain being pulled over a portion of the visual field. High myopia is a predisposing factor to retinal detachment, and one retrospective study of retinal detachments from basketball-related injuries in a Taiwanese population found that most players who suffered retinal detachments had moderate-high myopia [24]. Diagnosis of retinal tears and detachments requires a dilated fundus exam by an ophthalmologist.

22.6.9.1 Treatment

Patients with suspected retinal tears or retinal detachments based on presentation and symptoms should be urgently referred to a retina specialist. For retinal tears without detachment, laser retinopexy, an office procedure typically employing argon laser to secure the retina surrounding the tear to the retinal pigment epithelium, is recommended within 24–72 h [23] to prevent subsequent detachment of the surrounding retina. If instead a retinal detachment is noted, surgical intervention is typically required. The complex operative management of retinal detachment is beyond the scope of this chapter; however, management may include removal of the vitreous from the central cavity of the eye to decrease traction on the retina, use of laser or cryotherapy to seal breaks in the retina, placement of a scleral buckle (silicone band) around the outside of the eye to provide reinforcement, and placement of a gas bubble inside the eye to help temporarily hold the retina in the proper position.

22.6.9.2 Return to Play

Suspicion for retinal tear or detachment is an absolute contraindication to return to play until the patient has received a thorough dilated fundoscopic exam by an ophthalmologist. Patients who undergo laser retinopexy for retinal tears may be able to return to play within a few days, while patients with retinal detachments requiring surgical repair will typically require a prolonged recovery period of several months.

22.6.10 Orbital Fracture

The globe is surrounded by a series of interconnecting bones that make up the orbit. These bones have significant variations in their thickness and strength. Blunt trauma to the eye and ocular adnexa can result in an orbital blowout fracture. The inferior and medial walls are the weakest and are the most prone to blowout fractures. Symptoms may include pain with eye movement, double vision, pain with palpation of the orbital rim, and ipsilateral cheek numbness. Ocular exam signs associated with orbital fracture include restricted eye movements, enophthalmos (recession of the eye deeper into the orbit), hypoglobus (infraplacement of the eye), and eyelid edema or bruising. Patients with findings suspicious for an orbital fracture should undergo non-contrast CT scan with thin cuts through the orbit.

22.6.10.1 Treatment

A thorough ocular examination should be conducted to rule out concurrent injuries such as an occult open globe injury or traumatic optic neuropathy. Muscle entrapment in the fracture must also be ruled out as urgent surgical repair within 24–48 h may be indicated to prevent muscle ischemia [15]. Repair within 1–2 weeks may be indicated for persistent double vision, large orbital floor fractures, enophthalmos, or hypoglobus. Some ophthalmologists routinely prescribe oral antibiotics for all orbital fractures. We recommend antibiotics in the setting of any concurrent sinus disease on CT scan. Patients should be instructed not to blow their noses due to the potential risk of prolapsing orbital contents through the fracture site. Use of local nasal decongestants such as oxymetazoline should be considered.

22.6.10.2 Return to Play

Careful consideration of the orbital fracture's location and extent, as well as its associated clinical signs and symptoms, should be undertaken before making a recommendation regarding return to play. Most often, athletes should

not return to competition until the fracture has healed, typically resulting in at least a month without play [25].

Take-Home Message

Eye injuries in basketball are common events; however, they are most often self-limited without persistent visual impairment. Protective eyewear is effective in injury prevention and underutilized in basketball. Many aspects of ocular examination and diagnosis require special equipment or the expertise of an ophthalmologist. However, a systematic sideline assessment performed by non-ophthalmologist medical staff can provide valuable information to guide next steps in referral or return to play. Given the possibility of vision-limiting injuries, uncertainty in the diagnosis should prompt referral to an ophthalmologist.

References

- Haring RS, Sheffield ID, Canner JK. Epidemiology of sports-related eye injuries in the United States. *JAMA Ophthalmol.* 2016;134(12):1382–90.
- Boden BP, Pierpoint LA, Boden RG, Comstock RD, Kerr ZY. Eye injuries in high school and collegiate athletes. *Sports Health.* 2017;9(5):444–9.
- Commission UCPS. Sports and recreational eye injuries. Washington, DC: US Consumer Product Safety Commission Report. 2000.
- Miller KN, Collins CL, Chounthirath T, Smith GA. Pediatric sports- and recreation-related eye injuries treated in US Emergency Departments. *Pediatrics.* 2018;141(2):e20173083.
- American Academy of Pediatrics, Committee on Sports Medicine and Fitness, American Academy of Ophthalmology, Eye Health and Public Information Task Force. Joint Policy Statement: Protective eyewear for young athletes. *Ophthalmology.* 2004;111(3):600–3.
- Karlson TA, Klein B. The incidence of acute hospital-treated eye injuries. *Arch Ophthalmol.* 1986;104:1473–6.
- Youn J, Sallis RE, Smith G, Jones K. Ocular injury rates in college sports. *Med Sci Sports Exerc.* 2008;40(3):428–32.
- Yulish M, Reshef N, Lerner A, Pikkel J. Sport-related eye injury in northern Israel. *Isr Med Assoc J.* 2013;15:763–5.
- Zagelbaum BM, Starkey C, Hersh PS, Donnenfeld ED, Perry HD, Jeffers JB. The National Basketball Association Eye Injury Study. *Arch Ophthalmol.* 1995;113:749–52.
- Heimmel MR, Murphy MA. Ocular injuries in basketball and baseball: what are the risks and how can we prevent them? *Curr Sports Med Rep.* 2008;7(5):284–8.
- Rodriguez JO, Lavina AM, Agarwal A. Prevention and treatment of common eye injuries in sports. *Am Fam Physician.* 2003;67:1481–8.
- Canty G, Nilan L. Return to play. *Pediatr Rev.* 2015;36(10):438–47.
- Kim T, Daluovoy MB, editors. *The pocket guide to cornea.* Thorofare: Slack; 2019.
- Moreira LB, Kasetsuwan N, Sanchez D, Shah SS, LaBree L, McDonnell PJ. Toxicity of topical anesthetic agents to human keratocytes in vivo. *J Cataract Refract Surg.* 1999;25:975–80.
- Bagheri N, Wajda B, Calvo C, Durrani A. *The Wills eye manual.* Wolters Kluwer. 2016.
- Gharaibeh A, Savage HI, Scherer RW, Goldberg MF, Lindsley K. Medical interventions for traumatic hyphema. *Cochrane Database Syst Rev.* 2019;1:CD005431.
- Blanch RJ, Good PA, Shah P, Bishop JR, Logan A, Scott RA. Visual outcomes after blunt ocular trauma. *Ophthalmology.* 2013;120(8):1588–91.
- Sipperley MJ, Quigley HA, Gass DM. Traumatic retinopathy in primates: the explanation of commotio retinae. *Arch Ophthalmol.* 1978;96:2267–73.
- Ahn SJ, Woo SJ, Kim KE, Jo DH, Ahn J, Park KH. Optical coherence tomography morphologic grading of macular commotio retinae and its association with anatomic and visual outcomes. *Am J Ophthalmol.* 2013;156(5):994–1001.e1.
- Campagna G, Al-Mohtaseb Z, Khandelwal S, Chang E. Sequential traumatic corneal open globe rupture in a patient with osteogenesis imperfecta type I. *Am J Ophthalmol Case Rep.* 2018;11:35–6.
- Zhang Y, Zhang MN, Jiang CH, Yao Y, Zhang K. Endophthalmitis following open globe injury. *Br J Ophthalmol.* 2010;94(1):111–4.
- Faghghi H, Hajizadeh F, Esfahani M, et al. Posttraumatic endophthalmitis: Report No. 2. *Retina.* 2012;32:146–51.
- Robinson RT, Wadsworth LT, Feman SS. Traumatic retinal tear in a basketball player. *Curr Sports Med Rep.* 2011;10(3):129–30.
- Lee T-H, Chen Y-H, Kuo H-K, et al. Retinal detachment associated with basketball-related eye trauma. *Am J Ophthalmol.* 2017;180:97–101.
- Gilliland GD, Gilliland G, Fincher T, et al. Timing of return to normal activities after orbital floor fracture repair. *Plast Reconstr Surg.* 2007;120(1):245–51.



Shoulder Injuries in Basketball

23

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23.1 Introduction

Basketball is without a doubt the fastest growing sport on every scale from day-to-day play to the competitive area worldwide. As the nature of basketball has evolved from a non-contact sport to one of the most highly physical contact sports, game-related injuries have increased to their current rates [1, 2]. Although injuries of lower extremities are most common amongst basketball players, upper extremity injuries (such as shoulder injuries) may also affect the performance of the player and may even lead to career-ending results [3].

The shoulder is the most mobile joint of the human body with its unique anatomical features.

Having a lot of motional range and function, the glenohumeral joint may be exposed to certain problems in terms of stability. Many structures around the shoulder act together as stabilizers to overcome these problems. Dynamic stabilizers of the glenohumeral joint are composed of muscular structures such as the rotator cuff, biceps, and muscles around the scapulothoracic girdle. Static stabilizers include bony anatomy, labrum, and the joint capsule.

Occasionally, the balance between mobility and stability may be interrupted in professional athletes due to high demands of intensive physical activity, resulting in the glenohumeral joint being susceptible to injuries.

Similar to the other parts of the body, shoulder injury rates are slightly higher during competition than during practice, due to practices being more predictable activities; whereas in competitions, there is a high level of intensity combined with stressful fatigue and unpredictable environments [4, 5]. The consumption of energy during dribbling, jumping, and contact with other players, combined with all the stress factors affecting the player, may lead to the onset of acute and repetitive stress injuries in the shoulder joint. While common acute injuries such as glenohumeral or acromioclavicular separation-dislocations, fractures around the shoulder are usually the result of direct or indirect trauma; chronic injuries such as labral, capsular, rotator cuff, and biceps pathologies are usually the products of overuse and repetitive microtrauma.

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251

The treatment of shoulder injuries requires proper diagnosis based on a detailed history, incorporated with a systemic approach and physical examination, in combination with the knowledge of unique anatomical features of the glenohumeral joint. Imaging modalities are used for both diagnostic and follow-up purposes. Although conservative treatment is the primary method of management in shoulder injuries in basketball players, surgical intervention may be necessary in, especially, failed conservative treatment.

23.2 Epidemiology

Basketball injuries are amongst the most common sports injuries that require medical treatment. Although basketball is one of the leading sports activities associated with injuries, there are few studies in the literature about the epidemiology of various injuries in professional basketball [2, 6–9]. In the study of Podlog et al., it is found that injury rates in basketball have significantly increased over the past few decades, possibly due to the game becoming progressively quicker and more hard-hitting over time [10]. The relationship between game speed and injury incidence is strongly correlated in the study of Norton et al. [11]. However, injuries to the upper extremities in basketball players remain to be less frequent especially compared to the lower extremities [10]. Overall injury rate to the upper extremities of reported players is found to range from 11% to 14% at both the high school and professional level [12, 13]. In upper extremities, injuries to the shoulder are uncommon when compared to the fingers, hands, and arms [14]. Bonza and Fields discovered that in their study group of high school athletes, between the years 2005 and 2007, shoulder injuries occurred in 0.47 of every 10,000 athlete-exposures in boys' basketball, whereas it occurred for 0.45 in female athletes [6]. The same study also confirmed the findings previously reported as injuries were more likely to occur during competition than during practice. In his comparative study of men's and women's professional basketball

injuries, Zelisko et al. found that the women's overall injury frequency in professional levels was 1.6 times that of men, while men had a higher injury rate for shoulder in the same study [4, 15]. Although Deitch et al. found similar results, a slight difference in the frequency of shoulder injuries in Women's National Basketball Association (WNBA) and National Basketball Association (NBA) was noted in his study (3.9% and 3.4% respectively) [8].

23.3 Mechanism and Severity of Injury

The primary mechanism of shoulder injuries in basketball is player-to-player contact [6, 9, 13]. Therefore, the more the player remains in contact with other players (in a game or in practices like 5 on 5 scrimmage), the more he or she will be susceptible to injury. The player-to-player contact is also the most common injury mechanism that requires surgical intervention [6]. The other mechanisms of shoulder injury are contact with floor, basketball, the rim or nonspecific contact, and overuse injuries [9, 13].

The primary diagnosis in acute shoulder injuries are sprain/strains [12]. The others are dislocation/separation, contusion, and fractures. Approximately 60% of shoulder injuries in basketball allow athletes to return to play in less than 1 week [6, 12]. Borowski et al. found that the most frequent injury-related activities during the play are rebounding (25.1%), defending (14.8%), general play (16.9%), ball handling or dribbling (8.9%) and shooting (8.5%) [4]. In the same study, the guard position is found to be the most injury prone position for both males and females in basketball.

23.4 History

A detailed history is crucial for differential diagnosis in professional athletes. The first step of evaluation should begin with obtaining initial information about the patient such as age, medical history, sporting activities, and their activity

Fig. 23.1 A player sustained from shoulder injury on the field



level. Any previous sport-related injury in player's medical history has the potential to serve as an important risk factor for a new injury. Therefore, previous injuries, treatments, medications, physical therapy exercises, injections, and surgeries must be noted. Although most shoulder complaints in athletes are due to overuse and are chronic in nature, acute injuries on the field may also be seen.

The most common symptom of a shoulder injury is pain, which may not always necessarily result in absence from play but can sometimes affect the performance of the athlete (Fig. 23.1). The location and characteristic properties of the pain (radiation, relieving and exacerbating factors, associated symptoms, etc.) point out different injury types. While pain on the top of the shoulder may be a sign of acromioclavicular injury, pain on the posterior of the shoulder may point to cervical etiology. Nocturnal pain may represent rotator cuff injuries.

Instability in the shoulder is another common complaint. The relationship between the instability and trauma must be noted. The duration and frequency of the instability are important as they can affect the performance of the athlete. Provocative motions may give a clue about the direction of the instability. Adducted and internally rotated shoulder instability suggests posterior direction, while abduction and external rotation instability represents an anteriorly instable shoulder. Athletes with labral pathologies may complain about mechanical

symptoms such as catching, locking or snapping, depending on the type and size of the labral lesion. Stiffness is not a common complaint and is usually associated with adhesive capsulitis. It may sometimes be seen in endocrine disorders.

23.5 Physical Examination

The physical examination should begin with observation of posture, shoulder position, and alignment of the trunk. Affected shoulder should be focused after identifying any compensatory movements of the body. As in all musculoskeletal clinical examinations, physical examination of the shoulder includes inspection, palpation, evaluation of range of motion (ROM), muscle strength, and specific tests.

The player should always be evaluated for both shoulders. Any shirts must be removed before the inspection, allowing the visualization of swelling, bruise, and muscular atrophy. Both shoulders must be observed for symmetry and any pathologies thorough active and passive range of motion. Assessing range of motion is a crucial step in shoulder examination. Active and passive forward flexion, abduction, adduction, internal and external rotation capacity of the joint should be evaluated. Any difference in the range of motion between two shoulders must be recorded. Scapular movements should be assessed carefully for synchronization with the

glenohumeral joint. Any muscular atrophy in infraspinatus and supraspinatus fossae may give clues about neuropathy or chronic rotator cuff tear. Scapular winging may be evident upon inspection which is usually due to neuropathic denervation of serratus anterior or trapezius muscles. Any previous incisions in the shoulder region should also be noted.

Palpation should be directed from the painless site to the painful area and include humeral head, glenohumeral, acromioclavicular and sternoclavicular joint lines, bicipital groove, coracoid process, scapula, and the clavicle. While assessing passive range of motion, any crepitation in the shoulder region must be noted. Symmetry, stability, major muscle strength, and examination of rotator cuff muscles should be performed in all patients. While anterior deltoid muscle can be assessed at 90° of forward elevation, middle deltoid fascicles can be evaluated at 90° of abduction. Rotator cuff muscle strength should be assessed, preferably while the patient is seated. Supraspinatus muscle strength is best evaluated by resistance to the examiner's downward force while the patient's shoulder is at 90° of abduction and 30° of forward flexion with internal rotation of the arm and the thumb pointing downwards ("Jobe's" or "empty can" test) [16]. Infraspinatus and teres minor, the main external rotators, can be assessed by resistance to the external rotation of the arm with the elbow flexed at 90°, while the shoulder abduction is at 0° and 90° respectively. Subscapularis muscle pathologies are evaluated by "lift-off" test, in which the patient places the hand to the back in maximal internal rotation of the arm and then tries to lift the hand off against examiner's resistance and fails to perform this maneuver [17]. "Belly-press" test can be also performed to evaluate subscapularis muscle, in which the patient tries to compress through abdomen with the elbow flexed. The "bear-hug" test can reduce pain from long head of biceps influence, often disturbing the belly press test. The patient's shoulder is placed at 90° elevation and internal rotation, with forearm horizontal in front of body and the palm is held on the opposite shoulder. The patient is then asked to press down against physician hand

and hold resisted internal rotation as the physician tries to apply external rotation force to the forearm. The test is considered positive if the patient cannot hold the hand or shows weakness against the force applied.

After the routine examination, specific shoulder tests for unique pathologies should be performed. "Hawkins-Kennedy" test can be used to show impingement [18]. This maneuver can be performed by 90° forward flexion of the arm and forcibly medially rotation of the shoulder. Pain during this maneuver could be the result of subacromial impingement or rotator cuff tendinitis.

Another specific test is "O'Brien's" or "active compression" test, which is used for biceps-labral (especially SLAP, superior labrum anterior to posterior) lesions or acromioclavicular pathologies [19]. This test can be performed by the shoulder brought to 90° of forward flexion and 10–15° of adduction with extended elbow, fully internally rotated arm and pronated forearm. Resistance is applied as the patient tries to elevate the extremity with the thumb pointing downwards. Localization of the pain must be followed carefully during the maneuver. The test is considered to be positive if the patient reports that the pain is relieved with the palm facing up.

The "anterior slide" test can also be used for assessing SLAP lesions [20]. The test is performed in seated position. The patient places the hand on the ipsilateral iliac crest and thumb facing posteriorly, while the examiner applies anterosuperior force on the elbow with the other hand stabilizing the shoulder. The test is considered positive, if it produces pain or clicking sensation.

Biceps pathologies can also produce pain and loss of strength. The "Yergason's" test is used to determine any biceps pathology [21]. This maneuver is performed with the elbow flexed at 90° and the forearm in slight pronation. The patient tries to supinate the forearm against examiner's resistance. The test is considered positive if it produces pain in the bicipital groove.

The "Speed's" test is another maneuver to identify biceps pathologies [22]. The test is performed while the shoulder is elevated forward at 90° with extended elbow and supinated forearm. The patient tries to resist the downward force

produced by the examiner. The test is considered positive if it produces pain in the anterior region of the shoulder.

The last step of the shoulder examination consists of neurologic evaluation. Radiculopathy in the cervical region may present as shoulder discomfort. Pain in the posterior aspect of the shoulder and radiation through the elbow may be signs of cervical pathology. Other nerve injuries such as axillary, suprascapular, or long thoracic may initially present with shoulder pain. In case of neurologic impairment, one should assess a complete upper extremity strength and deep tendon reflexes.

23.6 Imaging

Evaluation of shoulder injuries by imaging should begin with plain radiographs. Routine anteroposterior (AP) view in different rotational positions of the shoulder, axial view, scapular Y, and transthoracic views of the joint allow visualization of bony pathologies around the shoulder (trauma series). These pathologies include dislocation, fractures, and Hill-Sachs lesions. Special views such as Stryker notch (for Hill-Sachs lesions) and West point (for glenoid rim) views are obtained if necessary (Fig. 23.2).



Fig. 23.2 West point x-ray view of the shoulder

Ultrasonographic imaging has been shown to be an effective method especially in the diagnosis of biceps and rotator cuff pathologies; however, it is highly user-dependent [23]. Magnetic resonance imaging (MRI) has been comprehensively used for shoulder injuries in basketball [24]. Performing the MRI with arthrogram has become popular in the last decades and has been utilized with good accuracy and sensitivity for assessing both SLAP lesions, capsular and rotator cuff pathologies. Computerized tomography (CT) with arthrogram can be used alternatively especially for a good bony assessment in addition to soft tissue imaging. CT alone is preferred for identifying Hill-Sachs lesions, greater tuberosity, and glenoid rim fractures.

23.7 Treatment

Among sports injuries, basketball injuries are some of the most common sports injuries that require medical treatment [25]. The objective of treatment to shoulder injuries must be to provide a stable shoulder, minimal recurrence of the injury, and a fast return to preinjury function [26]. Shoulder injuries in basketball are generally treated by conservative means as in other overhead throwing sports. There are rehabilitation protocols defined for the conservative treatment and management of injuries. In acute phase of the injury, the aim is to give time for the healing of damaged tissue and decrease inflammation and pain. Supportive treatment such as passive ROM and stabilization exercises and non-steroid anti-inflammatory drugs (NSAIDs) may be used. After the acute phase, stretching and strengthening exercises can be started. These exercises should be customized according to the weak muscles of the patient. When there is almost no pain, ROM deficit, and weakness in the injured muscle, the patient is allowed to perform intensive strengthening exercises. After these steps are completed, the athlete is allowed to return to training with the team and return to competitions consecutively. If there is no improvement in the pain and performance of the athlete, a re-evaluation and more

aggressive management, such as surgery, may be considered.

23.8 Common Shoulder Injuries in Basketball

The most common shoulder injuries seen in basketball players may be classified as acute and chronic injuries. Acute injuries include glenohumeral dislocation, acromioclavicular separation, clavicle, proximal humerus, and scapula fractures; chronic injuries include rotator cuff tendinitis or tears, internal shoulder impingement, biceps tendon injuries, SLAP lesions, and scapular dyskinesia [27]. Some of these pathologies can occur simultaneously.

23.8.1 Dislocations-Separations around the Shoulder

The shoulder is the most commonly dislocated joint in the human body during sports [26, 28]. Both the static and dynamic stabilizers of the shoulder joint try to keep the humeral head in the glenoid fossa, maintaining a stable joint. However, in different positions of the shoulder, some of the stabilizers fail to resist against traumatic load and dislocation occurs. Glenohumeral dislocations are generally seen anterior and inferiorly in athletes [29]. Although posterior dislocations do occur, there is not enough data in the literature about posterior dislocations in overhead throwing athletes. The primary mechanism responsible for anterior glenohumeral dislocation is the capsulolabral separation from the anterior and inferior glenoid rim, which is called classic Bankart's lesion [26]. It usually results from either a fall on the outstretched arm with externally rotated and abducted shoulder, or a direct force onto the shoulder. The diagnosis is usually achieved by physical examination. The patient typically presents with deformity and limitation of internal rotation. Plain radiographs may help to distinguish the direction of the dislocation and concomitant fractures. Neurovascular examination is crucial because axillary neuropraxia may be seen in 5–35% of first-time

dislocations [30, 31]. Reduction attempt is rarely performed on the field immediately after injury, albeit the reduction maneuvers are not recommended without a previous X-ray, to avoid damages in case of fracture-dislocation. There are many techniques for reduction of the glenohumeral joint. There are different opinions as to which technique is superior in various publications. We only recommend performing the maneuvers in a gentle way, to avoid an enlargement of any lesion (Hill-Sachs, Bankart, etc.). After one or two attempts, it is better to achieve reduction under sedation or general anesthesia.

One of the most common reduction techniques is self-reduction. In this technique, the athlete holds the ipsilateral knee with both hands and leans backwards. Milch technique can also be used in which the patient's hand is placed behind the head, and the physician holds the elbow by applying a slight traction and guiding the humeral head over the glenoid rim [32]. Another technique is traction-countertraction method. The physician's foot is placed against the chest wall of the athlete, and traction is applied at 45° of shoulder abduction. After a successful reduction, the pain is relieved and the deformity is recovered immediately. Plain radiographs should be maintained to confirm the reduction and any associated fractures. Sometimes CT scan may be necessary to evaluate associated bony pathologies such as Hill-Sachs lesions, greater tuberosity, or glenoid rim fractures. MRI provides information about associated injuries such as rupture of rotator cuff or biceps pathologies.

In conservative treatment of glenohumeral dislocations, collected data in the literature has shown no effect of immobilization on the natural history of dislocation [33–35]. Range of motion and strengthening exercises should begin as soon as possible. Between 3 and 6 weeks, isokinetic exercises, between 6 and 8 weeks, plyometric exercises are started. Biceps strengthening is crucial in the management of shoulder dislocation. When the athlete achieves the opposite shoulder's range of motion and at least 90% of the strength, return to sports can be allowed. Recurrent instability of the shoulder is the major problem with anterior shoulder dislocations [26,

36]. Failure of conservative treatment with recurrent instability requires surgical intervention. Arthroscopic and open techniques for the treatment of shoulder instability are described in the literature [26]. The average time to return to sports after an arthroscopic stabilization of shoulder dislocation is 4–6 months [37–39].

Posterior glenohumeral dislocations are much less common and occur with falling onto the adducted, flexed, and internally rotated arm [40] or in player-to-player contact, with trauma on elbow or hand forced in anteroposterior direction. Misdiagnosis, up to 60%, in posterior dislocations is very common [29, 41, 42].

Acromioclavicular joint separation in an athlete may result from a direct fall either onto the acromion or on an adducted arm [27]. Localized tenderness and deformity on the acromioclavicular joint is a good clinical indicator, but radiographs are usually necessary for the diagnosis. Distal clavicle fractures should be kept in mind in differential diagnosis. Although treatment decisions between conservative or surgical options are quite controversial, most of the AC joint separations are treated conservatively with a sling and immobilization. Far displaced AC joint may need a surgical intervention.

23.8.2 Fractures Around the Shoulder

The clavicle is the most commonly fractured bone around the shoulder in basketball [29]. Fractures usually occur by direct falling onto the shoulder or outstretched hand. The diagnosis can be made by plain radiographs. Treatment is either conservative via a sling in minimally displaced fractures or by surgical means in displaced or open fractures. Return to gameplay is allowed after a painless clavicle and radiographic union with full ROM and strength.

Fractures of the proximal humerus do not occur frequently in basketball. In addition to plain radiographs, CT can be used in diagnosis to identify the type of the fracture and decide for the treatment method. These fractures can sometimes be with labral or rotator cuff pathologies. In

displaced or intraarticular humerus fractures, surgical treatment is usually the preferred method.

Scapula fractures involving the body, glenoid neck or rim, coracoid, and acromion are also rare in basketball players. In addition to routine AP, axillary, scapular Y radiographic views, West point view is helpful in determining these fractures. For glenoid rim fractures, bony Bankart lesions with instability should be kept in mind.

23.8.3 Rotator Cuff Tears

Rotator cuff pathologies are common in overhead throwing athletes, but most of them are asymptomatic. In a study by Connor et al., 40% of asymptomatic overhead throwing athletes were found to have rotator cuff tear at any degree in their dominant shoulders [43]. Traumatic rotator cuff tears occur infrequently in athletes younger than 35 years of age [29]. While significant trauma to the shoulder is a predisposing factor for rotator cuff tears in young athletes, it is usually the result of the combination of degeneration, instability, and impingement in older athletes [44, 45]. Chronic overuse may also lead to these kinds of injuries. The diagnosis is mainly based on MRI (Fig. 23.3) in combination with the physical examination and specific shoulder tests as mentioned above. The treatment of rotator cuff tears with partial thickness depends on the size, location, quality of the tendon, and patient's characteristics. Conservative treatment is usually the first choice in managing these pathologies; however, failure of conservative treatment over several months would result in consideration of surgical intervention. Partial tears below 50% thickness could be managed conservatively. If the tear involves between 50% and 75% thickness, surgery should be considered. Full thickness rotator cuff tears have poor prognosis even if treated surgically [46].

23.8.4 Internal Impingement

Internal impingement of the shoulder occurs when a repetitive throwing motion results in

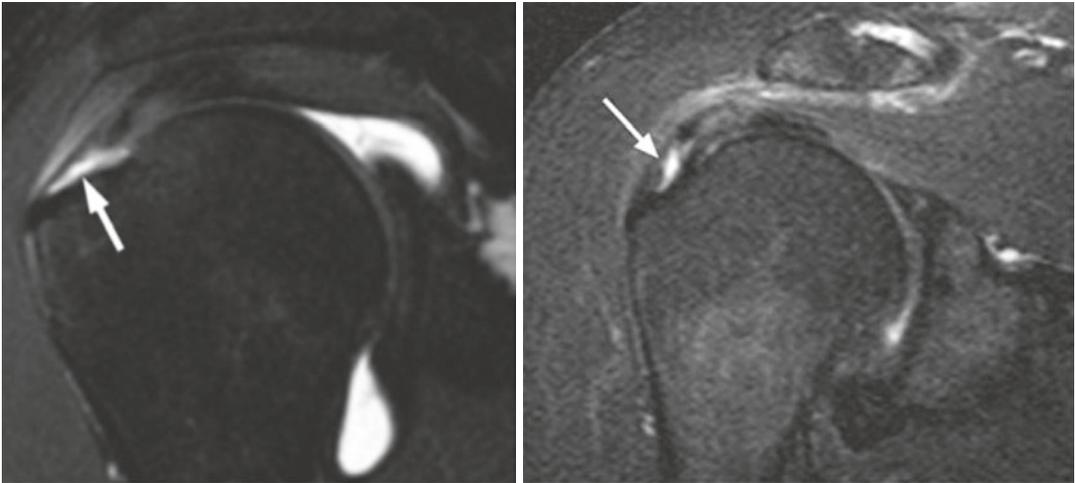


Fig. 23.3 Coronal MRI View of articular rotator cuff tear

anatomic adaptations of the shoulder joint and its surrounding structures [47]. Walch et al., and then Jobe, popularized the concept of internal impingement as a cause of rotator cuff and labrum pathologies in the overhead throwing athletes [48]. The hypothesis is that repeated excessive shoulder abduction and external rotation may lead to impingement of the posterosuperior glenoid and undersurface of the rotator cuff. With the addition of fatigue on the shoulder, anterior humeral translation with microinstability may cause labral abrasion. Treatment is usually non-operative by decreasing the inflammatory process and rebalancing the scapula.

23.8.5 Biceps and SLAP Lesions

During the throwing motion, the overhead throwing athlete places the shoulder in increased horizontal extension over rotation repeatedly. There is an anatomic adaptation to throwing shoulder in the body. The most common adaptive change is the increase in external rotation and decrease in internal rotation at 90° of abduction. This pattern is called “GIRD” (glenohumeral internal rotation deficit) which has been defined in the literature in multiple studies [47, 49, 50]. This adaptation which seems to have beneficial effect on the ath-

lete also can predispose the shoulder to pathologies of biceps, rotator cuff, and the labrum.

Though the exact mechanism of SLAP lesions remains controversial, several mechanisms of injury for SLAP lesions have been defined in the literature in the overhead throwing athletes [47, 51]. One of them is the eccentric load to the biceps–labral complex during throwing, which results in repetitive traumatic injury. Another mechanism includes a fall on the outstretched arm and a combination of compression and subluxation forces on the joint. Internal impingement, which is mentioned above, is the basic mechanism of posterior type II SLAP lesions.

In the clinical evaluation of overhead throwing athletes with SLAP lesions, anterior shoulder pain with “clicking” sensation is usual. There may be associated rotator cuff pathology or subacromial impingement that may give different clinical symptoms. If the internal rotation of both shoulders are different than each other by 25° or more, the athlete may have GIRD, which may be a predisposing factor for SLAP tears. Yergason and Speed’s tests should be performed to check any biceps tendinopathy. O’Brien’s, apprehension, anterior slide, and Jobe’s tests are the specific tests described to help detect SLAP lesions. Radiographic evaluation includes AP, axillary, and scapular Y views. MRI and particularly MRI

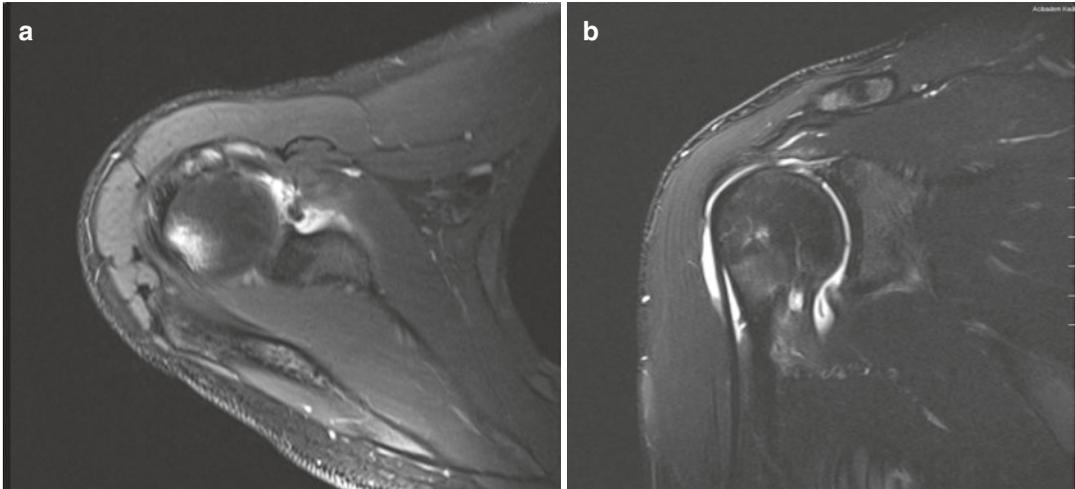


Fig. 23.4 (a) Axial MRI view of traumatic SLAP lesion and labral tear. (b) Coronal MRI view of the same lesion

with arthrogram are the preferred methods of imaging for SLAP lesions (Fig. 23.4).

The treatment should start with nonoperative management. Rest, NSAIDs, and stretching exercises are the first steps of conservative treatment. Treatment of GIRD (90% of throwers with symptomatic GIRD will respond to posterior capsular stretching program) and scapular dyskinesia, if present, is essential in the conservative management of SLAP lesions [17]. In a study of Edwards et al., nearly 70% of the athletes with SLAP lesions who were treated nonoperatively were able to return to play at the same level [52]. Failure of conservative treatment for SLAP lesions may require surgical intervention in overhead throwing athletes.

23.8.6 Scapular Dyskinesia

Scapular dyskinesia is a common pathology seen in overhead throwing athletes which is a cause for pain and dysfunction. It may be seen up to 67–100% in athletes with shoulder injury, and it can cause shoulder injury itself [53]. Whether scapular dyskinesia is a primary pathology or a secondary pathology caused by abnormal shoulder biomechanics is still a matter of debate. The term *SICK* scapula is defined as scapular malpo-

sition and dyskinesia with inferomedial border prominence and pain in the coracoid process with malposition [47]. Scapular instability may result in dysfunction of glenohumeral and scapulothoracic joints. Diagnosis should be made as soon as possible to prevent further injuries to the shoulder. Conservative treatment and prevention are the primary treatment objectives.

23.9 Principles of On-Field Evaluation

When an athlete sustains an acute injury on the field, the medical team should follow an algorithm for evaluation of the athlete to minimize the life-threatening risks of the injury [27]. Obvious extremity injuries should not be the primary concern, instead basic life support should be maintained in all on-field injuries. Once life-threatening emergencies are ruled out, affected extremity can be evaluated in detail. Simple injuries like contusion or minor sprains are usually managed expectantly with or without removal from the play. The examiner should pay attention to extremity-emergent symptoms such as intense pain, deformity, pulselessness, decreased sensation, or discoloration of the affected extremity. If one or more of these symptoms are found, the athlete

may require more urgent or emergent intervention. Once the red flag symptoms have been ruled out, ROM of the affected joint should be evaluated. The examiner can make a preliminary diagnosis with such algorithm.

After the initial diagnosis, the examiner or the medical staff should decide if the player can remain in the game or should be removed from the competition. If an athlete is removed from play, a thorough examination should be performed in a secure environment. The examiner then decides if any further orthopedic evaluation or imaging is necessary or not. It should be kept in mind that the primary objective should be protecting the injured athlete's safety.

There are several key points below for on-field management of shoulder injuries in basketball [3]:

- Proximal humerus or clavicle fractures may be supported by a sling. Unstable fractures seen on the field should be stabilized before removing the athlete from the field. Once stabilization has been completed, the athlete should be taken from the play and requires further evaluation with imaging for a proper treatment decision. Immediate return-to-play is not allowed.
- Glenohumeral dislocations can be diagnosed by deformity and pain on the shoulder with the player unable to move the shoulder joint. When diagnosed, the player should be taken away from the field and assessed again in a secure area such as the training room. The medical staff should decide whether or not an acute reduction attempt is appropriate. If there are red flag symptoms or if the medical staff is not experienced, the reduction maneuvers may be performed in emergency departments. After a successful reduction attempt, the player should be applied a sling. Further evaluation results for associated injuries should be obtained before the decision for treatment is made. Immediate return-to-play is not allowed.
- AC separations are generally difficult for reduction on the field. Once diagnosed, the player should be removed from the competition and the shoulder placed in a sling for sup-

port. Further evaluation will be necessary for treatment decision. Immediate return-to-play is not allowed.

23.10 Summary

With its evolving nature from a finesse sport to a highly physical one, individuals who play competitive basketball have become more susceptible to acute illness or injuries. Although shoulder injuries in basketball are not very common, they can affect the performance of the athlete and may even lead to career-ending results. On average, increases in performance load are positively associated with higher injury risk. Strength and conditioning programs may play a role in prevention of shoulder injuries. In particular, pre-season conditioning programs appear to be effective in reducing these injuries. At least one rest day per week and additional periods of time away from organized sports are recommended for physical recovery, and avoiding burnout. Once injured, proper history and physical examination, along with appropriate imaging modalities, contribute to creating a rational treatment plan. Acute injuries on the field require an evaluation algorithm that will secure the player from further damage and result in a speedy recovery to preinjury levels.

Fact Boxes

- The balance between the mobility and stability might sometimes be interrupted in the shoulder joint causing injuries.
- Shoulder injury rates are slightly higher during competition than practice.
- Acute injuries to the shoulder are usually the result of direct or indirect trauma; while chronic injuries are associated with overuse and repetitive microtrauma.
- In upper extremity, injuries to the shoulder in basketball players are uncommon compared to the fingers, hand, and arm.

- The primary mechanism of shoulder injuries in basketball is the player-to-player contact.
- A detailed history with systematic approach is crucial for the differential diagnosis of the professional athlete.
- Specific shoulder tests for unique pathologies may help the examiner for the diagnosis.
- AP, axillary, and scapular Y views are routine radiographs in the management of shoulder injuries in basketball.
- Conservative management is the mainstay of treatment in shoulder injuries seen in basketball players.
- Axillary neuropathy may be seen up to 35% of first-time anterior shoulder dislocations.
- The clavicle is the most commonly fractured bone around the shoulder in basketball.
- Rotator cuff pathologies are commonly found, yet most of them are asymptomatic in professional athletes.
- GIRD (glenohumeral internal rotation deficit) may predispose the shoulder to biceps, rotator cuff, and labral pathologies in overhead throwers.
- The primary objective of on-field evaluation is to protect the injured athlete's safety.
- Strength and conditioning programs appear to be effective in reducing shoulder injuries in basketball.

References

1. Minhas SV, Kester BS, Larkin KE, Hsu WK. The effect of an Orthopaedic surgical procedure in the National Basketball Association. *Am J Sports Med.* 2016;44(4):1056–61. <https://doi.org/10.1177/0363546515623028>.
2. Starkey C. Injuries and illnesses in the national basketball association: a 10-year perspective. *J Athl Train.* 2000;35(2):161–7.
3. Baker CL, Merkley MS. Clinical evaluation of the Athlete's shoulder. *J Athl Train.* 2000;35(3):256–60.
4. Borowski LA, Yard EE, Fields SK, Comstock RD. The epidemiology of US high school basketball injuries, 2005–2007. *Am J Sports Med.* 2008;36(12):2328–35. <https://doi.org/10.1177/0363546508322893>.
5. Knowles SB, Marshall SW, Bowling JM, Loomis D, Millikan R, Yang J, Weaver NL, Kalsbeek W, Mueller FO. A prospective study of injury incidence among North Carolina High School Athletes. *Am J Epidemiol.* 2006;164(12):1209–21. <https://doi.org/10.1093/aje/kwj337>.
6. Bonza JE, Fields SK, Yard EE, Dawn CR. Shoulder injuries among United States high school athletes during the 2005–2006 and 2006–2007 school years. *J Athl Train.* 2009;44(1):76–83. <https://doi.org/10.4085/1062-6050-44.1.76>.
7. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the national basketball association: a 17-year overview. *Sports Health.* 2010;2(4):284–90. <https://doi.org/10.1177/1941738109357303>.
8. Deitch JR, Starkey C, Walters SL, Moseley JB. Injury risk in professional basketball players: a comparison of women's national basketball association and national basketball association athletes. *Am J Sports Med.* 2006;34:1077–83.
9. Meeuwisse WH, Sellmer R, Hagel BE. Rates and risks of injury during intercollegiate basketball. *Am J Sports Med.* 2003;31(3):379–85. <https://doi.org/10.1177/03635465030310030901>.
10. Podlog L, Buhler CF, Pollack H, Hopkins PN, Burgess PR. Time trends for injuries and illness, and their relation to performance in the National Basketball Association. *J Sci Med Sport.* 2015;18(3):278–82. <https://doi.org/10.1016/j.jsams.2014.05.005>.
11. Norton K, Schwerdt S, Lange K. Evidence for the aetiology of injuries in Australian football. *Br J Sports Med.* 2001;35(6):418–23.
12. Rechel JA, Yard EE, Comstock RD. An epidemiologic comparison of high school sports injuries sustained in practice and competition. *J Athl Train.* 2008;43(2):197–204. <https://doi.org/10.4085/1062-6050-43.2.197>.
13. Dick R, Hertel J, Agel J, Grossman J, Marshall SW. Descriptive epidemiology of collegiate men's basketball injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. *J Athl Train.* 2007;42(2):194–201.
14. Newman JS, Newberg AH. Basketball Injuries. *Radiol Clin N Am.* 2010;48(6):1095–111. <https://doi.org/10.1016/j.rcl.2010.07.007>.
15. Zelisko JA, Noble HB, Porter M. A comparison of men's and women's professional basketball injuries. *Am J Sports Med.* 1982;10(5):297–9. <https://doi.org/10.1177/036354658201000507>.
16. Winter SB, Hawkins RJ. Comprehensive history and physical examination of the throwing shoulder. *Sports Med Arthrosc Rev.* 2014;22:94–100.
17. Gerber C, Krushell RJ. Isolated rupture of the tendon of the subscapularis muscle. Clinical features in 16 cases. *J Bone Joint Surg Br.* 1991;73:389–94.

18. Caliş M, Akgiin K, Birtane M, Karacan I, Caliş H, Tüzün F. Diagnostic values of clinical diagnostic tests in subacromial impingement syndrome. *Ann Rheum Dis.* 2000;59:44–7.
19. O'Brien SJ, Pagnani MJ, Fealy S, et al. The active compression test: a new and effective test for diagnosing labral tears and acromioclavicular joint abnormality. *Am J Sports Med.* 1998;26:610–3.
20. Kibler WB. Specificity and sensitivity of the anterior slide test in throwing athletes with superior glenoid labral tears. *Arthroscopy.* 1995;11:296–300.
21. Rosas S, Krill MK, Amoo-Achampong K, Kwon K, Nwachukwu BU, McCormick F. A practical, evidence-based, comprehensive (PEC) physical examination for diagnosing pathology of the long head of the biceps. *J Shoulder Elb Surg.* 2017;26:1484–92.
22. Bennett WF. Specificity of the Speed's test: arthroscopic technique for evaluating the biceps tendon at the level of the bicipital groove. *Arthroscopy.* 1998;14:789–96.
23. Wiener SN, Seitz WH Jr. Sonography of the shoulder in patients with tears of the rotator cuff: accuracy and value for selecting surgical options. *AJR Am J Roentgenol.* 1993;160:103–7. discussion 109–110
24. Aoyama JT, Maier P, Servaes S, et al. MR imaging of the shoulder in youth baseball players: anatomy, pathophysiology, and impact on treatment. *Clin Imaging.* 57:99. <https://doi.org/10.1016/j.clinimag.2019.05.005>.
25. Hammig BJ, Heewon Y, Bensema B. Epidemiology of Basketball Injuries Among Adults Presenting to Ambulatory Care Settings in the United States. *Clin J Sport Med.* 2007;17(6):446–51. <https://doi.org/10.1097/JSM.0b013e31815aed13>.
26. Burra G, Andrews JR. Acute shoulder and elbow dislocations in the athlete. *Orthop Clin N Am.* 2002;33:479–95.
27. Carr JB 2nd, Chicklo B, Altchek DW, Dines JS. On-field Management of Shoulder and Elbow Injuries in baseball athletes. *Curr Rev Musculoskelet Med.* 2019;12(2):67–71. <https://doi.org/10.1007/s12178-019-09535-9>.
28. Kerr ZY, Collins CL, Pommering TL, Fields SK, Comstock RD. Dislocation/separation injuries among US high school athletes in 9 selected sports: 2005–2009. *Clin J Sport Med.* 2011;21(2):101–8.
29. Owens S, Itamura JM. Differential diagnosis of shoulder injuries in sports. *Orthop Clin North Am.* 2001;32(3):393–8. [https://doi.org/10.1016/S0030-5898\(05\)70208-X](https://doi.org/10.1016/S0030-5898(05)70208-X).
30. Perlmutter GS, Appuzzese W. Axillary nerve injuries in contact sports: recommendations for treatment and rehabilitation. *Sports Med.* 1998;26:351–6.
31. Silliman JF, Dean MT. Neurovascular injuries to the shoulder complex. *J Orthop Sports Phys Ther.* 1993;18:442–8.
32. Russell JA, Holmes EM, Keller DJ, et al. Reduction of acute anterior dislocations using the Milch technique: a study of ski injuries. *J Trauma.* 1981;21:802–4.
33. Henry JH, Genung JA. Natural history of glenohumeral dislocation revisited. *Am J Sports Med.* 1982;10:135–7.
34. Aronen JG, Regan K. Decreasing the incidence of recurrence of first time anterior shoulder dislocations with rehabilitation. *Am J Sports Med.* 1984;12:283–91.
35. Hovelius L. Primary anterior dislocation of the shoulder in young patients: a ten year prospective study. *J Bone Joint Surg Am.* 1987;78:1677–84.
36. Kocher MS, Waters PM, Micheli LJ. Upper Extremity injuries in the Paediatric athlete. *Sports Med.* 2000;30:117. <https://doi.org/10.2165/00007256-200030020-00005>.
37. Arcerio RA, Wheeler JH, Ryan JB, McBride JT. Arthroscopic Bankart repair vs nonoperative treatment for acute, initial anterior shoulder dislocation. *Am J Sports Med.* 1994;22:589–94.
38. Arcerio RA, Taylor DC, Snyder RJ, Uhorchak JM. Arthroscopic bioabsorbable tack stabilization of initial anterior shoulder dislocations: a preliminary report. *Arthroscopy.* 1995;11:410–7.
39. Bacilla P, Field LD, Savoie FH. Arthroscopic Bankart repair in a high demand patient population. *Arthroscopy.* 1997;13:51–60.
40. Moeller JC. Compound posterior dislocation of the shoulder. *J Bone Joint Surg Am.* 1975;57:1006–7.
41. Hawkins RJ, Neer CS, Pianta RM, Mendoza FX. Locked posterior dislocation of the shoulder. *J Bone Joint Surg Am.* 1987;69:9–18.
42. Rowe CR, Zarins B. Chronic unreduced dislocations of the shoulder. *J Bone Joint Surg Am.* 1982;64:494–505.
43. Connor PM, Banks DM, Tyson AB, et al. Magnetic resonance imaging of the asymptomatic shoulder of overhead athletes: a 5-year follow-up study. *Am J Sports Med.* 2003;31:724–7.
44. Weiss JM, Arkader A, Wells LM, Ganley TJ. Rotator cuff injuries in adolescent athletes. *J Pediatr Orthop B.* 2013;22(2):133–7. <https://doi.org/10.1097/BPB.0b013e3283547001>.
45. Tarkin IS, Morganti CM, Zillmer DA, McFarland EG, Giangarra CE. Rotator cuff tears in adolescent athletes. *Am J Sports Med.* 2005;33(4):596–601. <https://doi.org/10.1177/0363546504269033>.
46. Rudzki JR, Shaffer B. New approaches to diagnosis and arthroscopic management of partial-thickness cuff tears. *Clin Sports Med.* 2008;27:691–717.
47. Bakshi N, Freehill MT. The overhead athletes shoulder. *Sports Med Arthrosc Rev.* 2018;26(3):88–94. <https://doi.org/10.1097/JSA.0000000000000200>.
48. Walch G, Boileau P, Noel E, et al. Impingement of the deep surface of the supraspinatus tendon on the

- posterosuperior glenoid rim: an arthroscopic study. *J Shoulder Elb Surg.* 1992;1:238–45.
49. Wilk KE, Macrina LC, Fleisig GS, et al. Correlation of glenohumeral internal rotation deficit and total rotational motion to shoulder injuries in professional baseball pitchers. *Am J Sports Med.* 2011;39:329–35.
50. Ruotolo C, Price E, Panchal A. Loss of total arc of motion in collegiate baseball players. *J Shoulder Elb Surg.* 2006;15:67–71.
51. Rokito SE, Myers KR, Ryu RKN. SLAP lesions in the overhead athlete. *Sports Med Arthrosc Rev.* 2014 Jun;22(2):110–6. <https://doi.org/10.1097/JSA.000000000000018>.
52. Edwards SL, Lee JA, Bell JE, et al. Nonoperative treatment of superior labrum anterior posterior tears: improvements in pain, function, and quality of life. *Am J Sports Med.* 2010;38:1456–61.
53. Burn MB, McCulloch PC, Lintner DM, Liberman SR, Harris JD. Prevalence of scapular dyskinesis in over- head and nonoverhead athletes: a systematic review. *Orthop J Sports Med.* 2016;4(2): 2325967115627608.



Management of Shoulder Instability in Basketball Players

24

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24.1 Introduction

Anatomy: The shoulder is a ball and socket joint that allows for the largest arc of motion of any joint in the human body. This wide range of motion comes at the expense of decreased stability. The delicate balance between motion and stability is achieved by the intricate anatomy of the glenohumeral joint, including both static and dynamic stabilizers [1–3]. Bony static stabilizers include the glenoid, humeral head, and the proximal humerus. The articular conformity of the glenohumeral joint is relatively poor compared to other ball and socket joints. The glenoid is pear-shaped with the inferior aspect forming a true circle [4]. Contrarily, the humeral head is shaped like a sphere and has three times the surface area of the glenoid with only 25–30% of the humeral head articulating with the glenoid at any given position [3]. This poor bony

congruency highlights the need for appropriately functioning soft tissue stabilizers about the shoulder joint.

Soft tissue static stabilizers include the glenoid labrum, glenohumeral ligaments, joint capsule, and negative intra-articular pressure. The labrum is a triangular rim of fibrocartilaginous tissue that deepens the glenoid articulation and increases shoulder stability by 10% [5, 6]. The capsular attachments to the labrum, known as the capsulolabral junction, adds further static stability to the glenohumeral joint. The most important components of the shoulder capsule are the glenohumeral ligaments. Each ligament provides static stability in addition to secondary dynamic stability based on the position of the shoulder. The inferior glenohumeral ligament (IGHL) is the most important ligament when considering anteroinferior and posteroinferior instability of the shoulder. The IGHL has an anterior and posterior band with a capsular bridge that acts like a “hammock” around the inferior proximal humerus (Fig. 24.1) [2, 7, 8]. The posterior band helps resist posterior instability by tightening when the arm is in the adducted, flexed, and internally rotated position. Conversely, the anterior band resists anteroinferior instability with the arm in the flexed, abducted, and externally rotated position. When the capsule and glenohumeral ligaments are intact, a negative-pressure “vacuum effect” is created, which further contributes to static stability of the shoulder [9, 10].

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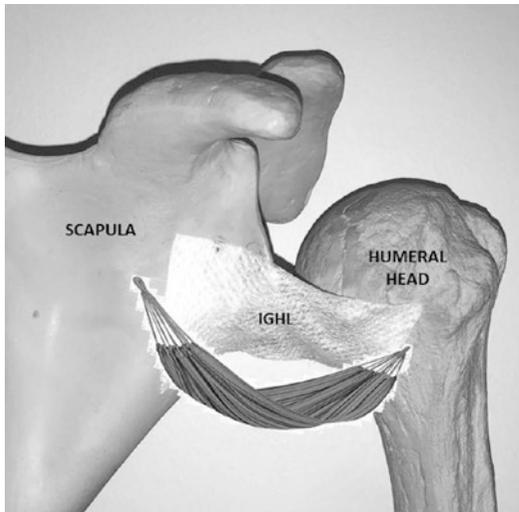


Fig. 24.1 The IGHL acts as a sling or “hammock” to provide anteroinferior stability to the shoulder joint. *IGHL* inferior glenohumeral ligament

Dynamic stabilizers of the glenohumeral joint include the rotator cuff, deltoid, and long head of the biceps tendon. The rotator cuff is the most important dynamic stabilizer. It acts as a dynamic compressor of the humeral head against the glenoid, especially during the initiation of glenohumeral movement. Furthermore, direct attachment of the rotator cuff to the capsule increases articular tension while providing proprioceptive feedback [11, 12].

Pathoanatomy: The Bankart lesion is an anteroinferior labrum tear and is the classic, pathognomonic lesion of anterior shoulder instability. It was first described in 1923 by the English surgeon Arthur Bankart in his report of four cases that each demonstrated this lesion [13]. He stated that “the head shears off the fibrous capsule of the joint from its attachment to the fibro-cartilaginous glenoid ligament. The detachment occurs over practically the whole of the anterior half of the glenoid rim.” Bankart also acknowledged the importance of capsular laxity but maintained that the capsulolabral injury is the sentinel pathologic lesion of anterior shoulder instability, and this concept has largely stood the test of time.

Since its original description, variations of the Bankart lesion have been identified as well. A

“bony Bankart” refers to a Bankart lesion that includes an associated anteroinferior glenoid fracture that becomes detached with the torn labrum. Another variation is the anterior labral periosteal sleeve avulsion (ALPSA) lesion, which occurs when a sleeve of the glenoid periosteum peels off with the Bankart lesion.

As described by Bankart, the anterior capsule and specifically the IGHL become injured and stretched during an anterior dislocation. In severe cases, the IGHL can become detached from its humeral attachment, known as a humeral avulsion of the inferior glenohumeral ligament (HAGL) lesion. This is a severe finding that necessitates surgical management, which will be discussed further in the treatment section. During an anterior dislocation, a Hill-Sachs lesion often develops, which is an impaction injury to the posterior humeral head from contact against the anterior glenoid.

The posterior side of the shoulder is slightly different in its pathoanatomy compared to the anterior side. A traumatic posterior dislocation can result in a labrum tear with an associated capsular injury. However, subtle posterior instability from repetitive trauma is more common because the posterior band of the IGHL is typically not as thick as the anterior band. Since ligamentous support of the posterior capsule is thinner and weaker compared to the more robust anterior side, repetitive trauma may alter the elasticity of the capsule, resulting in a more subtle instability pattern highlighted by capsular laxity.

Spectrum of Instability: Shoulder instability includes a broad spectrum of clinical presentations. Athletes may present after a complete dislocation, subluxation, or subtle micro-instability from repetitive trauma. A complete dislocation occurs when the humeral head no longer articulates with the glenoid surface and remains completely non-articulating. A full dislocation invariably requires the shoulder to be relocated with a reduction maneuver, which may be performed manually by the athlete or by a certified health professional. A subluxation is a partial dislocation that auto-reduces before full dislocation can occur. Athletes will often report a sliding sen-

sation in his or her shoulder but will deny feeling the shoulder locked in a dislocated position. Subtle micro-instability from repetitive trauma usually does not have a traumatic mechanism. Instead, patients will report a gradual sensation of pain and/or not trusting the shoulder because of apprehension despite denying a true dislocation or subluxation event.

Epidemiology: Anterior shoulder instability most commonly occurs in the young and athletic population. Males have a much higher rate of shoulder instability compared to females with most studies reporting a 70–85% male predominance [14–16]. The incidence of anterior shoulder instability has been estimated between 8 and 23.9 per 100,000 person-years [17, 18]. This can be higher in athletes who participate in collision sports, including football, rugby, wrestling, and overhead throwing sports. Moreover, the incidence in military personnel has been estimated as 1.69 per 1000 person-years, which is even higher than contact athletes [17]. Owens et al. investigated 4080 glenohumeral instability events in National Collegiate Athletic Association (NCAA) athletes during a 15-year period [15]. They reported an incidence of 0.12 injuries per 1000 athlete exposures. Dislocations were more likely to occur in male athletes (relative risk 2.67) and during collegiate games as opposed to practice (relative risk 3.50). More than 10 days of time lost from sport occurred after 45% of instability events.

Only a few studies have reported the epidemiology of shoulder instability in high-level basketball players. Recent studies have highlighted that basketball players have an increased risk of shoulder instability compared to many sports. This risk is often underappreciated because basketball is not considered a true contact or overhead sport. However, basketball necessitates low-velocity contact while performing frequent overhead maneuvers, leaving players in a potentially vulnerable position for shoulder injury. Recently, the Multicenter Orthopaedic Outcomes Network (MOON) Shoulder Instability Group investigated 863 patients after surgical management for primary

shoulder instability [14]. They found that 709 patients (82%) were male and the average age was 24 years old. The primary direction of instability was anterior in both male (74%) and female (73%) patients, and posterior instability accounted for 23% of cases. The rate of shoulder dislocation requiring formal closed reduction was highest among male patients less than 20 years old. Moreover, a labrum tear (66%) and Hill-Sachs lesion (41%) were the most frequent concomitant injuries. Trauma from playing sports was the mechanism of injury for 644 patients (75%), and basketball was the second most common sport in which primary shoulder instability occurred, accounting for 13% of all dislocations.

Another recent epidemiological study by Kraeuter et al. compared rates of shoulder instability in high school and college athletes and found basketball to be a high-risk sport for shoulder instability, especially in college athletes [19]. Both male and female college basketball players had a significantly higher risk of shoulder dislocation (relative risk 2.99 and 2.28, respectively) compared to high school basketball players. Player to player contact was the most common mechanism, and dislocations were just as likely to occur in practice or competition.

An epidemiological database study of National Basketball Association (NBA) injuries found that shoulder instability accounted for 57 injuries over a 17-year period, which was only 0.5% of all injuries reported [20]. This seemingly low number is likely due to the extremely high rate of other, more common musculoskeletal injuries in basketball players, including lateral ankle sprains, patellofemoral inflammation, lumbar strains, and hamstring strains. Furthermore, NBA players are elite athletes that likely have the muscle strength, coordination, and proprioception to minimize shoulder instability compared to amateur basketball players. Nonetheless, Minhas et al. investigated the most common orthopedic procedures performed on NBA players and found shoulder instability surgery (46 cases) to be the fourth most frequent surgery performed, accounting for 13.2% of all cases [21].

Fact Box

- The shoulder joint is a minimally constrained ball and socket joint that allows for a wide range of motion at the expense of stability.
- The anteroinferior labrum and anterior band of the IGHL are the most commonly injured structures after an anterior instability event.
- Shoulder instability occurs along a broad clinical spectrum, including complete dislocation, partial dislocation (i.e., subluxation), and repetitive micro-trauma.
- Basketball necessitates low-velocity contact and frequent overhead movements, placing these athletes at a higher, often underappreciated, risk for shoulder instability.

24.2 Diagnosis

Evaluation of an athlete with shoulder instability should begin with a comprehensive history. Determining the type of instability event (i.e., dislocation, subluxation, or repetitive micro-trauma) is extremely important. The patient can often provide valuable information that will help the physician determine the exact nature of the instability event. The physician should always ask if the patient required a shoulder reduction, and if so, how the reduction was performed and who performed the reduction. The patient should also be asked how long the shoulder remained dislocated until the reduction was performed. Hand dominance, history of index injury, mechanism of injury, position of the shoulder at the time of injury, competitive level, position played, number of previous instability events, and goals for return to play should be assessed. Furthermore, the physician should document any other history of contralateral shoulder instability events or previous surgeries to either shoulder.

After acquiring the entire relevant history, a complete, detailed neurologic examination of both upper extremities should be performed. The

examination should begin with inspection for any swelling or obvious deformity. Palpation of the entire shoulder girdle can reveal a concomitant injury to the sternoclavicular (SC) joint, acromioclavicular (AC) joint, clavicle, or proximal humerus. A concomitant fracture can accompany a shoulder dislocation in up to 18% of anterior shoulder dislocations [22]. Next, the examiner should assess both active and passive range of motion of the shoulder in forward flexion, abduction, internal rotation, and external rotation. Rotator cuff strength testing should be performed and any weakness should raise suspicion for possible rotator cuff tear or nerve injury. In a recent review, the incidence of axillary nerve injury after shoulder dislocation varied from 3.3% to 40% with most of the injuries being neuropraxic injuries that resolved spontaneously without special intervention [23, 24]. Every patient should also be assessed for hyperlaxity by testing for a sulcus sign and assessing Beighton's criteria. Special tests should be performed last, including the apprehension test, Jobe relocation test, load and shift test, anterior jerk test, and posterior jerk test. The apprehension test and Jobe relocation test are particularly useful exam maneuvers when assessing anterior instability. A positive apprehension test is particularly useful as it has demonstrated a 96% positive predictive value for a Bankart tear [25].

While anterior instability is a relatively straight forward diagnosis, posterior instability can be more challenging because of its often-subtle presentation. Posterior instability has a wide spectrum of clinical manifestations from subtle subluxation to prominent dislocation. Symptoms include pain, inability to fully participate in athletic events, and inability to reach desired level of activity. Unlike patients who experience anterior instability, patients with posterior instability often present with a chief complaint of posterior shoulder pain as opposed to shoulder instability. Therefore, posterior instability can be an often-overlooked diagnosis. Whenever an athlete reports posterior shoulder pain during physical activity that loads the shoulder joint, a diagnosis of posterior instability should be considered. The diagnosis is often con-

firmed by provocative exam maneuvers, including the posterior jerk test, Kim test, posterior stress test, and the load and shift test [26, 27]. There is a 97% sensitivity to diagnose posterior instability when the Kim test and the jerk test are both positive [27].

Basic and advanced imaging of the affected shoulder should complement the history and physical examination. Any pre- and post-reduction shoulder radiographs must be reviewed. A radiographic shoulder series should include at least a true AP, scapular Y, and axillary views. Additional views, including a Stryker notch and West Point view, can provide further details on Hill-Sachs lesions and Bankart lesions, respectively. Magnetic resonance imaging (MRI) with or without arthrogram should be obtained to further evaluate the shoulder joint and any concomitant pathology. We prefer a non-arthrogram study at our institution to avoid distorting any injured structures in or around the shoulder joint. Special attention should be paid to the glenoid labrum, capsule, IGHL, and rotator cuff when evaluating the MRI. In the setting of a large, bony Bankart lesion or glenoid bone loss, a computed tomography (CT) scan should be obtained to better assess any bony deficiencies. A three-dimensional CT reconstruction of the glenoid and humerus can be particularly helpful in visualizing bone loss on both the glenoid and proximal humerus [28].

By paying careful attention to the patient's history, physical examination, and imaging findings, the treating physician can make an accurate diagnosis. Once the correct diagnosis is determined, the physician can formulate a treatment plan that allows the athlete to safely return to sport with as little time lost from competition as possible. This will be the focus of the remaining sections.

Fact Box

- Evaluation of a basketball athlete with shoulder instability should begin with a complete history, including descriptions of previous and current shoulder injuries.

- The physical examination should start with painless, benign maneuvers and progress to more invasive, special testing at the end of the exam.
- All basketball athletes should be asked about and tested for hyperlaxity at the time of evaluation as hyperlaxity is quite common in this population.
- Advanced imaging is recommended for all patients to better appreciate the injury pattern and to help develop a treatment plan.

24.3 Treatment

The decision for how to treat a basketball player with shoulder instability is dependent on many factors, including the player's age, level of competition, position, time of season, and history of previous dislocations. Educating the patient about various treatment options and mutual decision-making is important for successful treatment. The pros and cons of both nonsurgical and surgical management should be discussed with the patient and his or her family. It is imperative that the physician provides adequate information so an informed decision can be made. Elite athletes may have more complicated personal circumstances, including contract status, monetary incentives, and organizational expectations. Regardless, the physician should not be swayed by these additional factors. Instead, the focus should remain on helping the patient make the best decision that will optimize effective return to play and preserve the long-term health of his or her shoulder.

Regardless of the patient's level of play, the physician's first responsibility is the well-being of the patient, which requires choosing a treatment plan that reduces the risk of future instability events. While once regarded as a relatively benign event, a recurrent shoulder dislocation often causes further harm to the shoulder joint, including increased glenoid and humeral bone loss, worsening labrum tears, and increased

capsular stretch [29, 30]. This can complicate the treatment algorithm and dictate more of a salvage-type procedure in the primary setting, which limits future options if the patient were to have a complication or fail primary surgery.

The first major decision to make is whether to choose nonoperative or operative management. If operative management is chosen, the next major decision is choosing the appropriate surgical procedure, i.e., arthroscopic Bankart repair, open Bankart repair, or a glenoid bone augmentation procedure, such as the Latarjet procedure. The rest of the section will focus on indications and results of the various treatment options for shoulder instability in basketball athletes.

Non-surgical Management: Nonoperative treatment for shoulder instability has shown considerable variability in its effectiveness and remains controversial in the athletic population [15, 31–35]. Conservative management entails an initial period of brief sling immobilization (3–7 days) followed by a graduated physical therapy program focused on regaining shoulder range of motion and strength before returning to sport-specific drills [32, 36, 37]. Return to play is allowed once the patient demonstrates full, pain-free range of motion with full protective strength and no apprehension on physical exam. This usually takes 3–6 weeks but occasionally up to 8 weeks depending on the type of instability pattern [38]. A restrictive brace can be used by the athlete when returning to play, which can provide extra support to the shoulder and limit the athlete's ability to place the arm in a vulnerable position. While bracing is a common practice because it provides subjective improvement in stability, there is no clinical evidence to support its use in preventing recurrent instability [33]. Furthermore, it is our experience that many basketball players find such a brace too restrictive and often decide against wearing one during competition.

The success of conservative management is extremely variable following a first-time, traumatic shoulder dislocation. While conservative management has the potential benefits of avoiding surgery and returning an athlete to play during the same season, it does have a significant risk of sustaining a recurrent instability event. This should not be overlooked and must be heav-

ily considered by the treating physician because each recurrent dislocation risks further damage to the shoulder joint.

Age at the time of dislocation is one of the most important risk factors in determining the success of conservative treatment [34, 39–42]. Sachs et al. demonstrated that age under 25 years is a significant risk factor for re-dislocation following primary anterior shoulder instability [40]. Marans et al. revealed a 100% recurrent dislocation rate in adolescents treated with sling immobilization for 6 weeks [41]. This is consistent with a study from the United States Military Academy that reported a 92% recurrence rate in young athletes following conservative management [34]. Hovelius et al. reported a recurrence rate of 27% in patients older than 30 years but a 72% recurrence rate in athletes younger than 23 years, suggesting that appropriately chosen patients above the age of 30 may be amenable to nonoperative management [42]. Buss et al. showed an 86% return to sport rate without sling immobilization in a group of competitive high school and collegiate athletes, but 37% of the athletes experienced at least one recurrent instability event [32]. These athletes missed an average of 10.2 days (range 0–30 days) of sports participation with 27 of 30 athletes returning to play within 2–3 weeks.

Dickens et al. found that 73% of NCAA athletes were able to return to sport after an instability episode at a median of 5 days after injury [33]. However, 63% had a recurrent dislocation after returning to play. They found that athletes with a subluxation were 5.3 times more likely to return to sport compared to athletes with a dislocation. Similarly, Shanley et al. recently found that high school athletes who sustained a subluxation were three times more likely to successfully return to sport without recurrent instability after conservative management compared to athletes who sustained a dislocation [43].

Another study by Dickens et al. found that only 40% of athletes successfully returned to play without recurrence during a subsequent season following shoulder dislocation compared to 90% of athletes who successfully returned after surgical repair [33]. Athletes were 5.8 times more likely to successfully return for the following season after surgical repair. Return to play rates

were the same between athletes who sustained one or multiple in-season dislocation events.

A recent meta-analysis of randomized clinical trials found a 52.9% re-dislocation rate after conservative management compared to a 7.9% re-dislocation rate after surgical repair [44]. They also found that arm position during immobilization (e.g., external rotation versus internal rotation) had no effect on re-dislocation rates.

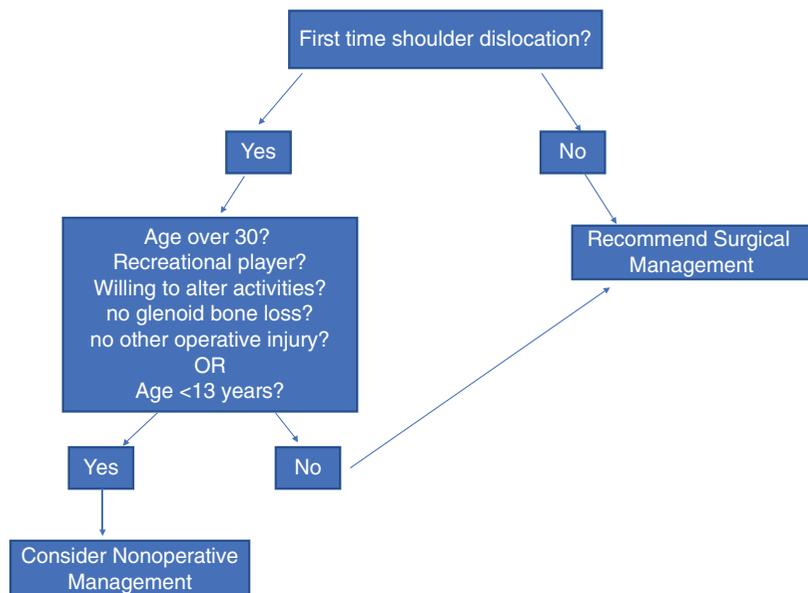
It is our experience that the age of the athlete, physical exam findings, number of previous dislocations, competitive level, and timing within a season are the most important factors to consider when weighing the option of nonoperative management after a primary shoulder dislocation. Only patients who are at a low risk for recurrent dislocation should be indicated for possible conservative management. Therefore, the indications for conservative management are very narrow and include very young patients (i.e., age 10–13), a first-time dislocation, less than 10% glenoid bone loss, intact rotator cuff, and a non-engaging Hill-Sachs lesion. Relative indications for nonoperative management include recreational basketball players over the age of 30 who meet the above criteria and are willing to modify their activities during a trial of conservative management. The role of conservative management is less clear for athletes between the ages of 20 and 30. The recurrence rate for athletes younger than

30 after conservative treatment is three times higher than those having surgical repair [45, 46]. Similarly, we have found that athletes in this controversial age range are usually best treated surgically unless the patient is a casual, recreational player with a first-time dislocation who is willing to modify activities.

Conservative management is a particularly appealing option when a high-level athlete experiences a dislocation in the first half of a season since surgery would require missing the entire season. However, managing an isolated in-season dislocation in the elite athlete remains controversial with no clear guidelines available in the literature. Each case should be managed individually based on the athlete’s history, physical exam, imaging findings, and individual circumstances. Avoiding a lost season should not be the main motivator when managing an athlete after an in-season dislocation.

Contraindications to conservative management include age under 20 years old, presence of a concomitant operative injury such as rotator cuff tear or HAGL lesion, greater than 15% glenoid bone loss, history of more than one dislocation, generalized hyperlaxity, marked apprehension on physical exam, or failure to return to sport after previous conservative management. A decision-making algorithm for conservative management is presented in Fig. 24.2.

Fig. 24.2 Decision-making algorithm for nonoperative versus operative management in a patient presenting after shoulder dislocation



Surgical Management: Early operative treatment for anterior shoulder instability has gained popularity as a growing amount of evidence supports a high rate of successful return to play with decreasing recurrence rates, especially in young and active athletes [38, 44, 47–50]. Many physicians now favor early operative intervention within a few weeks of the initial injury, especially when the patient is not indicated for conservative management. Absolute indications for surgical management include an associated operative injury, including >50% rotator cuff tear, HAGL lesion, >15% glenoid bone loss, >25% humeral head articular surface defect, proximal humerus fracture requiring surgery, irreducible dislocation, or non-concentric reduction. Further indications for operative management include patient age less than 20 years, greater than two dislocations, participation in overhead or contact sports, injury near the end of a season, failure of conservative management, or inability to perform sport-specific drills without apprehension.

Determining the appropriate surgical procedure for each patient is a critical decision that can heavily influence the patient's outcome. Historically, an open Bankart repair was the procedure of choice for anterior shoulder instability. This procedure allows the surgeon to directly visualize the detached labrum and then perform a labrum repair with a formal capsular shift. As shoulder arthroscopy gained popularity, many surgeons attempted arthroscopic Bankart repair with capsulorrhaphy. An arthroscopic approach offers the advantage of preserving the subscapularis attachment and reducing postoperative stiffness, especially loss of external rotation.

Regardless of the chosen technique, the main surgical goal is to restore normal capsulolabral anatomy. This requires (1) strong fixation of the labrum back onto the glenoid face to recreate the normal “bumper” effect of the labrum and (2) appropriate capsulorrhaphy to reduce the distorted capsular volume (Fig. 24.3). This was highlighted in a study by Speer et al., which

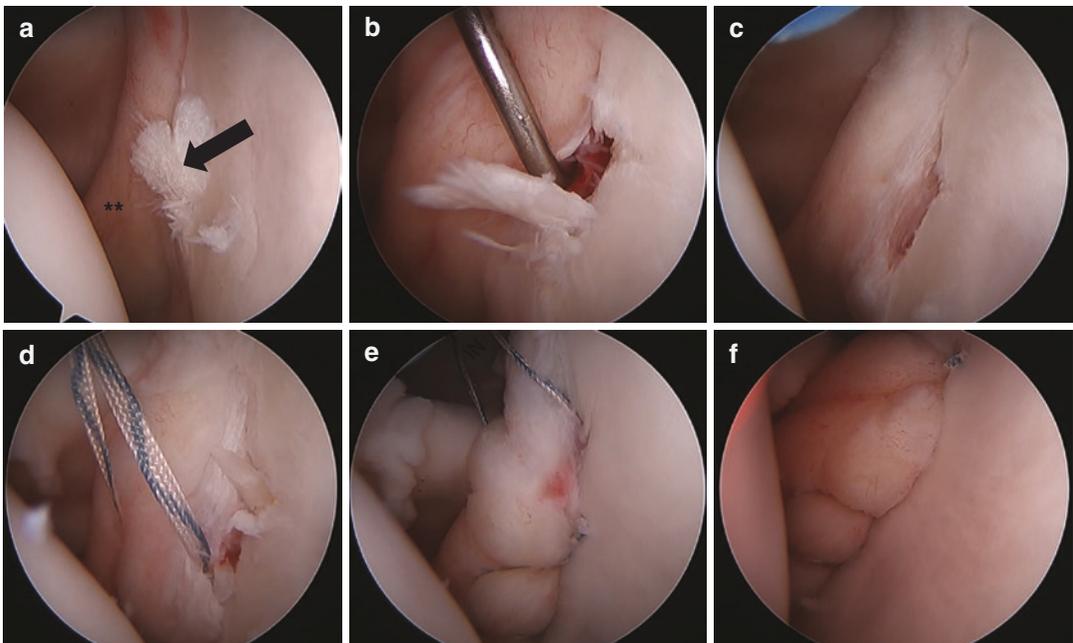


Fig. 24.3 Arthroscopic Bankart repair of a left shoulder in an 18-year-old female. (a) Identification of the Bankart lesion (arrow) and IGHL (double asterisk). (b) Placement of the arthroscopic probe into the Bankart lesion to signify adequate preparation of the labrum from the glenoid surface. (c) Bankart lesion fully prepared for fixation. (d)

Insertion of initial suture tape through the labrum beneath the IGHL. (e) Placement of third suture for Bankart repair. Note that the second suture tape also captured the IGHL to achieve adequate capsular shift. (f) Final construct with labrum “bumper” effect restored with associated reduction of capsular volume. IGHL: inferior glenohumeral ligament

demonstrated that isolated Bankart lesions are less likely to provoke complete dislocation unless there is a capsular injury [51].

Initially, results following an arthroscopic repair were inferior to results after open repair, demonstrating higher re-dislocation rates [52–54]. However, with the evolution of advanced arthroscopic techniques and equipment, multiple modern studies have shown at least equal outcomes between open and arthroscopic Bankart repairs [48, 49, 55–57]. As a result, arthroscopic Bankart repair with capsulorrhaphy is now commonly the surgical method of choice for the majority of surgeons [58–60].

Owens et al. performed a randomized control trial comparing arthroscopic and open Bankart repair in young, competitive athletes who sustained only shoulder subluxations without any associated bone loss [61]. At 2-year follow-up, there was no difference in outcome scores or recurrent subluxation rates based on operative approach. Three patients in each group experienced recurrent subluxations and no dislocations. Outcomes scores were significantly better in patients with three or less subluxations prior to surgery compared to patients with greater than three subluxations prior to surgery, which advocates for early surgical management in this patient population.

While arthroscopic Bankart repair is a very successful surgery when performed for appropriate indications, the open Bankart repair is still a valuable surgery with distinct advantages over an arthroscopic repair, especially in the setting of chronic shoulder instability (>2 shoulder dislocations), revision stabilization surgery, collision athletes, generalized hyperlaxity, and significant capsular stretch that cannot be adequately reduced arthroscopically. In these instances, open procedures have demonstrated superior outcomes compared to arthroscopic repair [44, 62]. A recent meta-analysis by Kavaja et al. demonstrated decreased recurrence rate after open Bankart repair compared to arthroscopic repair in the setting of chronic shoulder instability (5.8% versus 13.4%, respectively) [44]. Similarly, a 2018 study by Su et al. revealed a 42% recurrent instability rate in patients who underwent a revi-

sion arthroscopic Bankart repair [62]. The presence of an engaging Hill-Sachs lesion, age less than 22 years, and ligamentous hyperlaxity were independent predictors of recurrence in their cohort.

To help stratify patients at risk for failing an arthroscopic Bankart repair, Balg and Boileau developed the Instability Severity Index Score (ISIS) to stratify a patient's risk of failure following arthroscopic Bankart repair (Fig. 24.4) [39]. They reported the following risk factors for recurrence of instability following arthroscopic repair: age less than 20 years at the time of surgery, involvement in competitive/contact sports or those involving forced overhead activity, shoulder hyperlaxity, Hill-Sachs lesion present on anteroposterior radiograph with the shoulder in external rotation, and/or loss of the sclerotic inferior glenoid contour. This highlights that the decision to perform an open or arthroscopic repair should be largely dictated by extent of bony involvement, associated risk factors, and ultimately surgeon preference [63, 64].

In the setting of multiple dislocations, revision instability, and/or anterior glenoid bone loss greater than 15%, a glenoid bone augmentation procedure is usually the preferred treatment. The most commonly performed bone augmentation procedure is the Latarjet procedure, which transfers the coracoid process with the attached conjoint tendon to the anteroinferior glenoid. This creates a very stable construct that works through the “triple threat” mechanism of (1) glenoid bone augmentation, (2) sling effect from the conjoint tendon going over a subscapularis split, and (3) capsular shift/closure [65–67]. While the Latarjet procedure can be a very successful procedure, it does alter the normal anatomy and requires a steep learning curve. It also has a larger complication rate (15–30%) with a potentially more severe complication profile, including neurovascular injury, abrasion of humeral head cartilage against the bone block and screws, and bone lysis [68–70]. Many of these complications can be avoided with appropriate surgical experience and technique, and good patient outcomes with low recurrent instability rates are common after the Latarjet procedure [57, 67, 71–74].

Fig. 24.4 The Instability Severity Index Score (ISIS) is a 10-point scale to help determine a patient’s risk for recurrent shoulder instability following arthroscopic Bankart repair. A score greater than 6 imparts a 70% chance of recurrent dislocation after arthroscopic repair (Reproduced with permission from: Balg F, Boileau P. The instability severity index score. A simple preoperative score to select patients for arthroscopic or open shoulder stabilization. *J Bone Joint Surg Br.* 2007;89 (11):1470–7)

Prognostic factors	Points
Age at surgery (yrs)	
≤ 20	2
> 20	0
Degree of sport participation (pre-operative)	
Competitive	2
Recreational or none	0
Type of sport (pre-operative)	
Contact or forced overhead	1
Other	0
Shoulder hyperlaxity	
Shoulder hyperlaxity (anterior or inferior)	1
Normal laxity	0
Hill-Sachs on AP* radiograph	
Visible in external rotation	2
Not visible in external rotation	0
Glenoid loss of contour on AP radiograph	
Loss of contour	2
No lesion	0
Total (points)	10

* AP, anteroposterior

Anterior glenoid augmentation with a free bone block is an alternative option in lieu of a Latarjet procedure or in the setting of a previously failed Latarjet procedure. The most commonly used bone blocks are either iliac crest autograft or distal tibial allograft [75–78]. There are multiple advantages of a free bone block procedure, including maintenance of normal coracoid anatomy and ability to salvage a failed Latarjet procedure. Drawbacks of the procedure include morbidity from iliac crest harvest, loss of the “sling effect” from the conjoint tendon, and bone block resorption, especially with allograft bone [79, 80]. In general, clinical results following glenoid augmentation with a free bone block are similar to results following a Latarjet procedure, making it a viable option in the primary or revision setting [81, 82].

In summary, we recommend meticulous assessment of each patient’s risk profile prior to selecting a surgical procedure. Most basketball athletes are amenable to an arthroscopic procedure, especially after a single shoulder disloca-

tion [30]. Athletes who are younger or have sustained multiple instability events may warrant an open procedure depending on physical exam and imaging findings. In the setting of revision stabilization surgery or significant glenoid bone loss (>15%), a bone augmentation procedure should be performed as arthroscopic repair has demonstrated unacceptably high failure rates. A summary of surgical indications for the various procedures can be found in Fig. 24.5.

Return to Play After Surgery: Many post-surgical rehabilitation protocols allow a safe return to play following surgical correction of shoulder instability. These protocols often follow the same guidelines as conservative programs except the timeline is more prolonged to allow appropriate time for healing and return of full shoulder function. While protocols between surgeons will vary, most follow generally accepted guidelines of a graduated physical therapy program [83–85]. The initial phase focuses on a period of immobilization followed by pendulum exercises and isometric muscle contractions. This

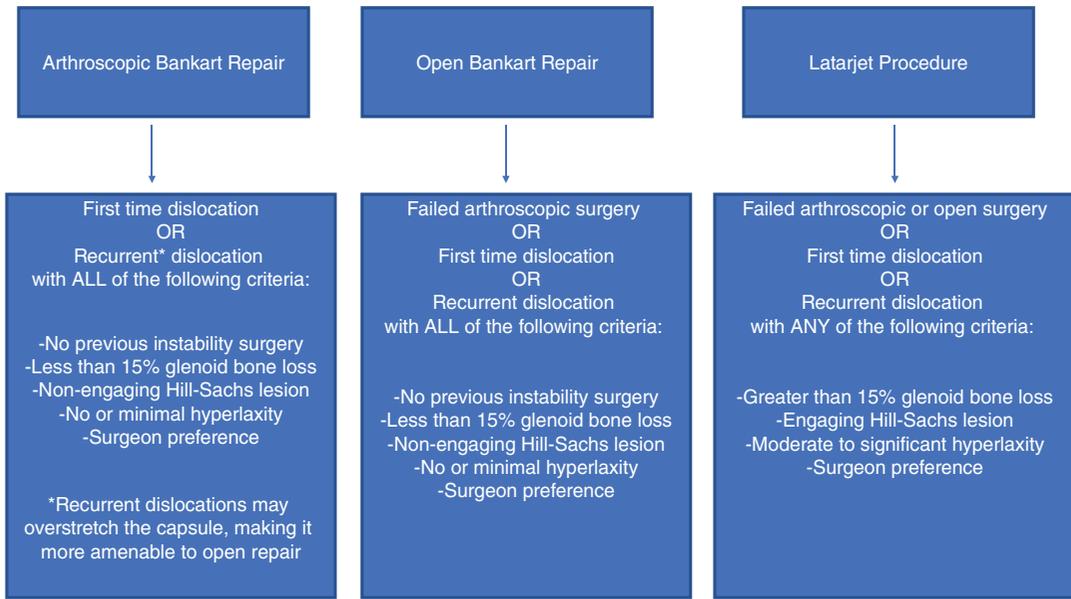


Fig. 24.5 Summary of surgical indications for arthroscopic Bankart repair, open Bankart repair, and the Latarjet procedure

phase lasts about 4 weeks, at which time the athlete progresses to passive range of motion followed by active-assisted range of motion exercises without resistance. This phase also lasts approximately 4 weeks until the athlete transitions to regaining full active range of motion and progressive strength reacquisition. The final phase begins around 3 months after surgery and allows the athlete to progress with plyometric exercises, advanced upper extremity strengthening, and sport-specific exercises. Typically, the timeline for full return to play is 4–6 months, but it can take longer depending on the procedure and unique circumstances of the basketball athlete. It is our recommendation that athletes who must perform overhead movements with unplanned collisions, including basketball players, do not return to athletic competition until 6 months postoperatively.

Return to play rates following surgical shoulder stabilization are quite high with a reported rate between 63% and 93% depending on the type of athlete [14, 16, 21, 38, 47, 52]. Collision and overhead athletes are at the highest risk to experience surgical failure [86, 87]. Basketball players tend to do very well after surgical stabilization

and appropriate rehabilitation. While many studies have investigated results of surgical repair for shoulder instability in contact and overhead athletes, minimal evidence is directly available for basketball players. Minhas et al. reported on 46 NBA players who underwent surgical stabilization for anterior shoulder instability [21]. They demonstrated a 93.5% return to play rate with increased games played and minimal change in post-injury player efficiency rating compared to players who underwent lower extremity surgery.

Fact Box

- The decision for nonsurgical versus surgical management is made based on a patient’s history, physical examination, imaging findings, and overall risk factors for recurrent instability.
- Young age (less than 20 years old), athletic participation that requires contact or overhead movements (including basketball), glenoid bone loss, and history of previous shoulder instability are significant risk factors for recurrent instability.

- Conservative management after a shoulder dislocation is not recommended for most basketball athletes. The high incidence of contact in vulnerable overhead arm positions in basketball makes non-operative management usually doomed to failure.
- Surgical management for shoulder instability is the preferred treatment in young, competitive basketball players. Early surgical intervention is key to prevent additional dislocations in this population.
- Arthroscopic Bankart repair is the most commonly performed procedure for primary, traumatic shoulder instability and allows minimally invasive restoration of normal shoulder anatomy.
- Open Bankart repair or Latarjet procedure are the preferred treatments of choice in the setting of revision surgery or chronic shoulder instability. When there is glenoid bone loss >15% or an engaging Hill-Sachs lesion, then a bone augmentation procedure should be performed.
- Postoperative rehabilitation protocols focus on an initial period of immobilization followed by gradual regaining of the athlete's range of motion, strength, and ultimately sports performance.
- Return to play rates are very high following surgical treatment, and most basketball athletes can return to full sports participation by 6 months postoperatively though timelines vary based on individual circumstances.

24.4 Conclusions

Shoulder instability is a common problem encountered by physicians who treat injured athletes. Basketball is a unique sport that allows for low-velocity contact with the arm in an overhead position. Therefore, basketball athletes are at higher risk for shoulder instability. Recognizing

the pattern of shoulder instability from the athlete's history, physical examination and imaging findings are the first steps in successful management. Understanding that the natural history of shoulder instability is a high propensity for recurrent dislocation, especially in the young athlete, is another vital component to developing a successful treatment plan. Surgical management is often the most appropriate treatment choice, especially in the young basketball player. Choosing the best surgical procedure is based on the patient's age, number of previous dislocations, and presence or absence of glenoid bone loss. Return to play can usually occur within 6 months postoperatively. After appropriate treatment, basketball athletes can expect a high return to play rate at the previous level of competition.

References

1. Murray IR, Goudie EB, Petrigliano FA, Robinson CM. Functional anatomy and biomechanics of shoulder stability in the athlete. *Clin Sports Med.* 2013;32(4):607–24.
2. O'Brien SJ, Neves MC, Arnoczky SP, Rozbruch SR, Dicarlo EF, Warren RF, et al. The anatomy and histology of the inferior glenohumeral ligament complex of the shoulder. *Am J Sports Med.* 1990;18(5):449–56.
3. Codman E. Tendinitis of the short rotators. In: *The shoulder: Rupture of the supraspinatus tendon and other lesions in or about the subacromial bursa.* Boston: Thomas Todd; 1934. p. 78–215. [https://scholar.google.at/scholar?q=E.A.+Codman+\(Ed.\),+The+shoulder,+rupture+of+the+supraspinatus+tendon+and+other+lesions+in+or+about+the+subacromial+bursa,+Krieger+Publishing,+Malabar,+FL+\(1934\),+pp.+313–331&btnG=&hl=de&as_sd](https://scholar.google.at/scholar?q=E.A.+Codman+(Ed.),+The+shoulder,+rupture+of+the+supraspinatus+tendon+and+other+lesions+in+or+about+the+subacromial+bursa,+Krieger+Publishing,+Malabar,+FL+(1934),+pp.+313–331&btnG=&hl=de&as_sd).
4. Huysmans PE, Haen PS, Kidd M, Dhert WJ, Willems JW. The shape of the inferior part of the glenoid: a cadaveric study. *J Shoulder Elb Surg.* 2006;15(6):759–63.
5. Howell SM, Galinat BJ. The glenoid-labral socket. A constrained articular surface. *Clin Orthop Relat Res.* 1989;(243):122–5. <http://www.ncbi.nlm.nih.gov/pubmed/2721051>.
6. Halder AM, Kuhl SG, Zobitz ME, Larson D, An KN. Effects of the glenoid labrum and glenohumeral abduction on stability of the shoulder joint through concavity-compression: An in vitro study. *J Bone Joint Surg Ser A.* 2001;83(7):1062–9.
7. Burkart AC, Debski RE. Anatomy and function of the glenohumeral ligaments in anterior shoulder instability. *Clin Orthop Relat Res.* 2002;(400):32–9. <http://www.ncbi.nlm.nih.gov/pubmed/12072743>

8. Itoigawa Y, Itoi E. Anatomy of the capsulolabral complex and rotator interval related to glenohumeral instability. *Knee Surg Sports Traumatol Arthrosc.* 2016;24:343–9.
9. Habermeyer P, Schuller U, Wiedemann E. The intra-articular pressure of the shoulder: An experimental study on the role of the glenoid labrum in stabilizing the joint. *Arthroscopy.* 1992;8(2):166–72.
10. Hurschler C, Wülker N, Mendila M. The effect of negative intraarticular pressure and rotator cuff force on Glenohumeral translation during simulated active elevation. *Clin Biomech.* 2000;15(5):306–14.
11. Lephart SM, Warner JJP, Borsa PA, Fu FH. Proprioception of the shoulder joint in healthy, unstable, and surgically repaired shoulders. *J Shoulder Elb Surg.* 1994;3(6):371–80.
12. Tibone JE, Fechter J, Kao JT. Evaluation of a proprioception pathway in patients with stable and unstable shoulders with somatosensory cortical evoked potentials. *J Shoulder Elb Surg.* 1997;6(5):440–3.
13. Bankart Blundell AS. Recurrent or habitual dislocation of the shoulder joint. *Br Med J.* 1923;2(3285):1132–3.
14. Trinh TQ, Naimark MB, Bedi A, Carpenter JE, Robbins CB, Grant JA, et al. Clinical outcomes after anterior shoulder stabilization in overhead athletes: An analysis of the MOON Shoulder Instability Consortium. *Am J Sports Med.* 2019;47(6):1404–10.
15. Owens BD, Agel J, Mountcastle SB, Cameron KL, Nelson BJ. Incidence of glenohumeral instability in collegiate athletics. *Am J Sports Med.* 2009;37(9):1750–4.
16. Kraeutler MJ, McCarty EC, Belk JW, Wolf BR, Hettrich CM, Ortiz SF, et al. Descriptive epidemiology of the MOON Shoulder Instability Cohort. *Am J Sports Med.* 2018;46(5):1064–9.
17. Owens BD, Dawson L, Burks R, Cameron KL. Incidence of shoulder dislocation in the United States military: demographic considerations from a high-risk population. *J Bone Joint Surg Ser A.* 2009;91(4):791–6.
18. Zacchilli MA, Owens BD. Epidemiology of shoulder dislocations presenting to emergency departments in the United States. *J Bone Joint Surg Ser A.* 2010;92(3):542–9.
19. Kraeutler MJ, Currie DW, Kerr ZY, Roos KG, Mccarty EC, Comstock RD. Epidemiology of shoulder dislocations in high school and collegiate athletics in the United States: 2004/2005 through 2013/2014. *Sports Health.* 2017;10(1):85–91.
20. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the National Basketball Association: a 17-year overview. *Sports Health.* 2010;2(4):284–90.
21. Minhas SV, Kester BS, Larkin KE, Hsu WK. The effect of an orthopaedic surgical procedure in the National Basketball Association. *Am J Sports Med.* 2015;44(4):1056–61.
22. Grimer RJ, Cool P. The prognosis following acute primary glenohumeral dislocation. *J Bone Joint Surg Br.* 2005;87-B(2):277.
23. Ward JP, Bradley JP. Decision making in the in-season athlete with shoulder instability. *Clin Sports Med.* 2013;32:685–96.
24. Avis D, Power D. Axillary nerve injury associated with glenohumeral dislocation. *EFORT Open Rev.* 2018;3(3):70–7. <https://doi.org/10.1302/2058-5241.3.170003>.
25. Kumar K, Makandura M, Leong NJJ, Gartner L, Lee CH, Ng DZW, et al. Is the apprehension test sufficient for the diagnosis of anterior shoulder instability in young patients without magnetic resonance imaging (MRI)? *Ann Acad Med Singap.* 2015;44(5):178–84.
26. Provencher MT, Leclere LE, King S, McDonald LS, Frank RM, Mologne TS, et al. Posterior instability of the shoulder: diagnosis and management. *Am J Sports Med.* 2011;39(4):874–86.
27. Kim SH, Park JS, Jeong WK, Shin SK. The Kim test: a novel test for posteroinferior labral lesion of the shoulder – a comparison to the jerk test. *Am J Sports Med.* 2005;33(8):1188–92.
28. Bishop JY, Jones GL, Rerko MA, Donaldson C. 3-D CT is the most reliable imaging modality when quantifying glenoid bone loss shoulder. *Clin Orthop Relat Res.* 2013;471(4):1251–6.
29. Duchman KR, Hettrich CM, Glass NA, Westermann RW, Wolf BR, Baumgarten K, et al. The incidence of Glenohumeral bone and cartilage lesions at the time of anterior shoulder stabilization surgery: a comparison of patients undergoing primary and revision surgery. *Am J Sports Med.* 2018;46(10):2449–56.
30. Dickens JF, Slaven SE, Cameron KL, Pickett AM, Posner M, Campbell SE, et al. Prospective evaluation of glenoid bone loss after first-time and recurrent anterior glenohumeral instability events. *Am J Sports Med.* 2019;47(5):1082–9.
31. Hayes K, Callanan M, Walton J, Paxinos A, Murrell GAC. Shoulder instability: management and rehabilitation. *J Orthop Sport Phys Ther.* 2013;32(10):497–509.
32. Buss DD, Lynch GP, Meyer CP, Huber SM, Freehill MQ. Nonoperative management for in-season athletes with anterior shoulder instability. *Am J Sports Med.* 2004;32(6):1430–3.
33. Dickens JF, Owens BD, Cameron KL, Kilcoyne K, Allred CD, Svoboda SJ, et al. Return to play and recurrent instability after in-season anterior shoulder instability: a prospective multicenter study. *Am J Sports Med.* 2014;42(12):2842–50.
34. Wheeler JH, Ryan JB, Arciero RA, Molinari RN. Arthroscopic versus nonoperative treatment of acute shoulder dislocations in young athletes. *Arthroscopy.* 1989;5(3):213–7.
35. Arciero RA, Wheeler JH, Ryan JB, McBride JT. Arthroscopic Bankart repair versus nonoperative treatment for acute, initial anterior shoulder dislocations. *Am J Sports Med.* 1994;22(5):589–94.
36. Brumitt J, Sproul A, Lentz P, McIntosh L, Rutt R. In-season rehabilitation of a division III female wrestler after a glenohumeral dislocation. *Phys Ther Sport.* 2009;10(3):112–7.

37. Burns TC, Owens BD. Management of shoulder instability in in-season athletes. *Phys Sportsmed.* 2010;38:55–60.
38. Watson S, Allen B, Grant JA. A clinical review of return-to-play considerations after anterior shoulder dislocation. *Sports Health.* 2016;8(4):336–41.
39. Balg F, Boileau P. The instability severity index score. *J Bone Joint Surg Br.* 2007;89-B(11):1470–7. <https://doi.org/10.1302/0301-620X.89B11.18962>.
40. Sachs RA, Lin D, Stone ML, Paxton E, Kuney M. Can the need for future surgery for acute traumatic anterior shoulder dislocation be predicted? *J Bone Joint Surg A.* 2007;89(8):1665–74.
41. Marans HJ, Angel KR, Schemitsch EH, Wedgl JH. The fate of traumatic anterior dislocation of the shoulder in children. *J Bone Joint Surg – Ser A.* 1992;74(8):1242–4.
42. Hovelius L, Eriksson K, Fredin H, Hagberg G, Hussenius A, Lind B, et al. Recurrences after initial dislocation of the shoulder. Results of a prospective study of treatment. *J Bone Joint Surg Am.* 1983;65(3):343–9.
43. Shanley E, Thigpen C, Brooks J, Hawkins RJ, Momaya A, Kwapisz A, et al. Return to sport as an outcome measure for shoulder instability: surprising findings in nonoperative management in a high school athlete population. *Am J Sports Med.* 2019;47(5):1062–7.
44. Kavaja L, Lähdeoja T, Malmivaara A, Paavola M. Treatment after traumatic shoulder dislocation: a systematic review with a network meta-analysis. *Br J Sports Med.* 2018;52(23):1498–506.
45. Kirkley A, Griffin S, Richards C, Miniaci A, Mohtadi N. Prospective randomized clinical trial comparing the effectiveness of immediate arthroscopic stabilization versus immobilization and rehabilitation in first traumatic anterior dislocations of the shoulder. *Arthroscopy.* 1999;15(5):507–14.
46. Kirkley A, Werstine R, Ratjek A, Griffin S. Prospective randomized clinical trial comparing the effectiveness of immediate arthroscopic stabilization versus immobilization and rehabilitation in first traumatic anterior dislocations of the shoulder: long-term evaluation. *Arthroscopy.* 2005;21(1):55–63.
47. Elsenbeck MJ, Dickens JF. Return to sports after shoulder stabilization surgery for anterior shoulder instability. *Curr Rev Musculoskelet Med.* 2017;10(4):491–8.
48. Abdul-Rassoul H, Galvin JW, Curry EJ, Simon J, Li X. Return to sport after surgical treatment for anterior shoulder instability: a systematic review. *Am J Sports Med.* 2019;47(6):1507–15.
49. Waterman BR, Burns TC, McCrisky B, Kilcoyne K, Cameron KL, Owens BD. Outcomes after Bankart repair in a military population: predictors for surgical revision and long-term disability. *Arthroscopy.* 2014;30(2):172–7.
50. Fabricant PD, Taylor SA, McCarthy MM, Gausden EB, Moran CJ, Kang RW, et al. Open and arthroscopic anterior shoulder stabilization. *J Bone Joint Surg Am.* 2015;97(1):1–11.
51. Speer KP, Deng X, Borrero S, Torzilli PA, Altchek DA, Warren RF. Biomechanical evaluation of a simulated Bankart lesion. *J Bone Joint Surg.* 1994;76(12):1819–26.
52. Yong GR, Jeong HH, Nam SC. Anterior shoulder stabilization in collision athletes: arthroscopic versus open Bankart repair. *Am J Sports Med.* 2006;34(6):979–85.
53. Mohtadi NGH, Bitar IJ, Sasyniuk TM, Hollinshead RM, Harper WP. Arthroscopic versus open repair for traumatic anterior shoulder instability: a meta-analysis. *Arthroscopy.* 2005;21(6):652–8.
54. Freedman KB, Smith AP, Romeo AA, Cole BJ, Bach BR. Open Bankart repair versus arthroscopic repair with transglenoid sutures or bioabsorbable tacks for recurrent anterior instability of the shoulder: a meta-analysis. *Am J Sports Med.* 2004;32(6):1520–7.
55. Harris JD, Gupta AK, Mall NA, Abrams GD, McCormick FM, Cole BJ, et al. Long-term outcomes after bankart shoulder stabilization. *Arthroscopy.* 2013;29:920–33.
56. Petretera M, Patella V, Patella S, Theodoropoulos J. A meta-analysis of open versus arthroscopic Bankart repair using suture anchors. *Knee Surg Sport Traumatol Arthrosc.* 2010;18(12):1742–7.
57. Ialenti MN, Mulvihill JD, Feinstein M, Zhang AL, Feeley BT. Return to play following shoulder stabilization: a systematic review and meta-analysis. *Orthop J Sports Med.* 2017;5:2325967117726055.
58. Rugg CM, Hettrich CM, Ortiz S, Wolf BR, Baumgarten KM, Bishop JY, et al. Surgical stabilization for first-time shoulder dislocators: a multicenter analysis. *J Shoulder Elbow Surg.* 2018;27(4):674–85. <https://doi.org/10.1016/j.jse.2017.10.041>.
59. Bishop JY, Hidden KA, Jones GL, Hettrich CM, Wolf BRMSG. Factors influencing surgeon's choice of procedure for anterior shoulder instability: a multicenter prospective cohort study. *Arthroscopy.* 2019;35(7):2014–25.
60. Kasik CS, Rosen MR, Saper MGZR. High rate of return to sport in adolescent athletes following anterior shoulder stabilization: a systematic review. *J ISAKOS.* 2019;4(1):33–40.
61. Owens BD, Cameron KL, Peck KY, DeBerardino TM, Nelson BJ, Taylor DC, et al. Arthroscopic versus open stabilization for anterior shoulder subluxations. *Orthop J Sport Med.* 2015;3(1):1–4.
62. Su F, Kowalczyk M, Ikpe S, Lee H, Sabzevari S, Lin A. Risk factors for failure of arthroscopic revision anterior shoulder stabilization. *J Bone Joint Surg Am.* 2018;100(15):1319–25.
63. Phadnis J, Arnold C, Elmorsy A, Flannery M. Utility of the instability severity index score in predicting failure after arthroscopic anterior stabilization of the shoulder. *Am J Sports Med.* 2015;43(8):1983–8.
64. Boughebi O, Maqdes A, Moraiti C, Dib C, Leclère FM, Valenti P. Results of 45 arthroscopic Bankart procedures: does the ISIS remain a reliable prognostic assessment after 5 years? *Eur J Orthop Surg Traumatol.* 2015;25(4):709–16.

65. Yamamoto N, Muraki T, An KN, Sperling JW, Cofield RH, Itoi E, et al. The stabilizing mechanism of the Latarjet procedure: a cadaveric study. *J Bone Joint Surg Am.* 2013;95(15):1390–7.
66. Wellmann M, De Ferrari H, Smith T, Petersen W, Siebert CH, Agneskirchner JD, et al. Biomechanical investigation of the stabilization principle of the Latarjet procedure. *Arch Orthop Trauma Surg.* 2012;132(3):377–86.
67. Mizuno N, Denard PJ, Raiss P, Melis B, Walch G. Long-term results of the Latarjet procedure for anterior instability of the shoulder. *J Shoulder Elb Surg.* 2014;23(11):1691–9.
68. Shah AA, Butler RB, Romanowski J, Goel D, Karadagli D, Warner JJP. Short-term complications of the Latarjet procedure. *J Bone Joint Surg Am.* 2012;94(6):495–501.
69. Gupta A, Delaney R, Petkin K, Lafosse L. Complications of the Latarjet procedure. *Curr Rev Musculoskelet Med.* 2015;8:59–66.
70. Griesser MJ, Harris JD, BW MC, Hussain WM, Jones MH, Bishop JY, et al. Complications and reoperations after Bristow-Latarjet shoulder stabilization: a systematic review. *J Shoulder Elbow Surg.* 2013;22:286–92.
71. Zimmermann SM, Scheyerer MJ, Farshad M, Catanzaro S, Rahm S, Gerber C. Long-term restoration of anterior shoulder stability: a retrospective analysis of arthroscopic bankart repair versus open Latarjet procedure. *J Bone Joint Surg Am.* 2016;98:1954–61.
72. Rossi LA, Bertona A, Tanoira I, Maignon GD, Bongiovanni SL, Ranalletta M. Comparison between modified Latarjet performed as a primary or revision procedure in competitive athletes: a comparative study of 100 patients with a minimum 2-year follow-up. *Orthop J Sport Med.* 2018;6(12):1–7.
73. An VVG, Sivakumar BS, Phan K, Trantalis J. A systematic review and meta-analysis of clinical and patient-reported outcomes following two procedures for recurrent traumatic anterior instability of the shoulder: Latarjet procedure vs. Bankart repair. *J Shoulder Elbow Surg.* 2016;25(5):853–63.
74. Bhatia S, Frank RM, Ghodadra NS, Hsu AR, Romeo AA, Bach BR, et al. The outcomes and surgical techniques of the Latarjet procedure. *Arthroscopy.* 2014;30:227–35.
75. Willemot LB, Akbari-Shandiz M, Sanchez-Sotelo J, Zhao K, Verborgt O. Restoration of articular geometry using current graft options for large glenoid bone defects in anterior shoulder instability. *Arthroscopy.* 2017;33(9):1661–9.
76. Provencher MT, Ghodadra N, LeClere L, Solomon DJ, Romeo AA. Anatomic osteochondral glenoid reconstruction for recurrent Glenohumeral instability with glenoid deficiency using a distal tibia allograft. *Arthroscopy.* 2009;25(4):446–52.
77. Frank RM, Romeo AA, Provencher MT. Glenoid reconstruction with distal tibia allograft for recurrent anterior shoulder instability. *Orthopedics.* 2017;40(1):e199–205.
78. Provencher MT, Frank RM, Golijanin P, Gross D, Cole BJ, Verma NN, et al. Distal tibia allograft glenoid reconstruction in recurrent anterior shoulder instability: clinical and radiographic outcomes. *Arthroscopy.* 2017;33(5):891–7.
79. Wong IH, King JP, Boyd G, Mitchell M, Coady C. Radiographic analysis of glenoid size and shape after arthroscopic coracoid autograft versus distal tibial allograft in the treatment of anterior shoulder instability. *Am J Sports Med.* 2018;46(11):2717–24.
80. Liwski CR, Dillman D, Liwski RS, Wong IH. Donor-specific human leukocyte antigen antibody formation after distal tibia allograft and subsequent graft resorption. *Clin J Sport Med.* 2019;1 <https://doi.org/10.1097/JSM.0000000000000715>.
81. Frank RM, Romeo AA, Richardson C, Sumner S, Verma NN, Cole BJ, et al. Outcomes of Latarjet versus distal tibia allograft for anterior shoulder instability repair: a matched cohort analysis. *Am J Sports Med.* 2018;46(5):1030–8.
82. Liu JN, Gowd AK, Garcia GH, Cabarcas BC, Nicholson GP, Cole BJ, et al. Return to sport following Latarjet versus distal tibia allograft for anterior shoulder instability: a matched cohort analysis. *J Shoulder Elb Surg.* 2019;28(6):e197–8.
83. Bottoni CR, Wilckens JH, DeBerardino TM, D’Alleyrand JCG, Rooney RC, Harpstrite JK, et al. A prospective, randomized evaluation of arthroscopic stabilization versus nonoperative treatment in patients with acute, traumatic, first-time shoulder dislocations. *Am J Sports Med.* 2002;30(4):576–80.
84. Pagnani MJ, Dome DC. Surgical treatment of traumatic anterior shoulder instability in American football players. *J Bone Joint Surg Am.* 2002;84(5):711–5.
85. Popchak A, Patterson-Lynch B, Christain H, Irrgang JJ. Rehabilitation and return to sports after anterior shoulder stabilization. *Ann Joint.* 2017;2:62.
86. Cho NS, Hwang JC, Rhee YG. Arthroscopic stabilization in anterior shoulder instability: collision athletes versus noncollision athletes. *Arthroscopy.* 2006;22(9):947–53.
87. Dickens JF, Rue JP, Cameron KL, Tokish JM, Peck KY, Allred CD, et al. Successful return to sport after arthroscopic shoulder stabilization versus nonoperative management in contact athletes with anterior shoulder instability: a prospective multicenter study. *Am J Sports Med.* 2017;45(11):2540–6.

Kurt E. Stoll and Grant E. Garrigues

25.1 Elbow Anatomy and Biomechanics

The elbow is a modified hinged joint. Hinged motion occurs at the ulnohumeral and radiocapitellar joints, while rotational motion occurs at the radiocapitellar joint and the proximal radioulnar joint. Stability throughout full range of motion is provided by osseous, capsuloligamentous, and muscular structures. The osseous stability provided is secondary to the highly irregular and congruent distal humerus, and proximal radius and ulna [1].

The proximal articular surface of the elbow is comprised of the trochlea and capitellum. The trochlea (L. “pulley”) is shaped like a pulley or spool on the distal end of the humeral shaft [1]. The medial ridge extends more distal than the lateral ridge and creates 6–8° of valgus tilt [1]. The trochlea also has a posterior tilt that prevents posterior translation [2]. The capitellum (L. “little head”) is a hemispheric structure and is lateral to the trochlea [1, 2].

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The distal articular surface of the elbow is comprised of the proximal ulna and radius. The proximal ulna is composed of the coronoid, olecranon, and the greater sigmoid notch. The coronoid plays a significant role in preventing posterior displacement, locking in the coronoid fossa in flexion [1, 3]. Fifty percent of the coronoid is needed to provide stability in extension. The greater sigmoid notch is highly congruent with the trochlea forming a primary constraint of the elbow [1, 2]. The radial head is a concave surface that articulates with the capitellum and is covered by 280° of articular cartilage [1]. The radial head also plays a significant role in valgus stability (3, 5; Fig. 25.1a).

The collateral ligaments are capsular thickenings that enhance elbow stability. The medial collateral ligament (MCL) is comprised of the anterior oblique ligament (AOL), the posterior oblique ligament (POL), and the transverse ligament [4]. The MCL provides restraint to valgus and internal rotatory loads with the AOL being the strongest of the three components [4]. The lateral collateral ligament (LCL) is composed of the lateral ulnar collateral ligament (LUCL), radial collateral ligament (RCL), annular ligament (AL), and accessory lateral collateral ligament (ALCL) [4, 5]. The LCL resists varus and external rotation stresses in conjunction with the capsule and bony architecture. Complete transection of the LCL results in posterolateral rotatory and varus instability [6].

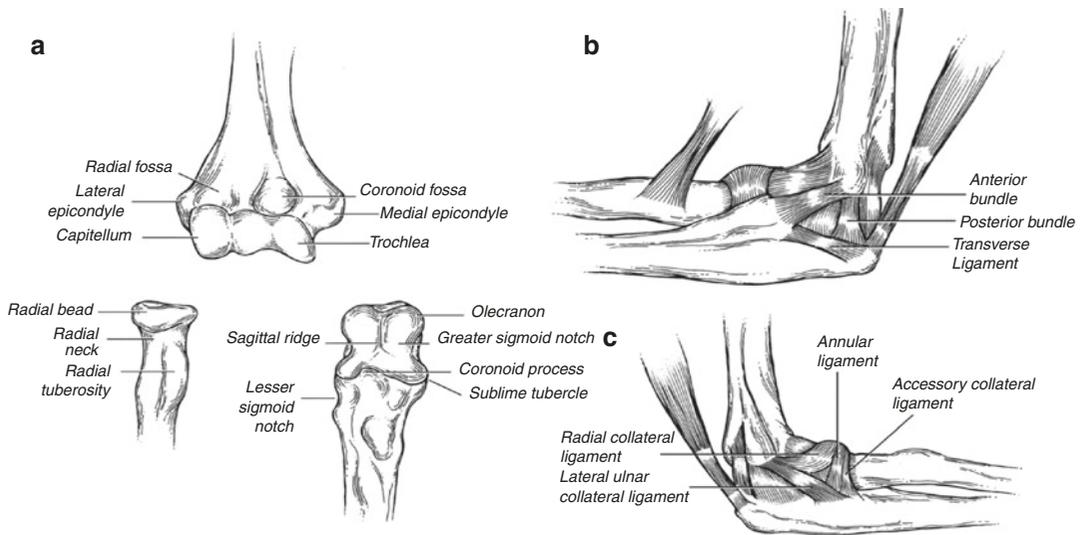


Fig. 25.1 (a) Osseous anatomy of the elbow. (b) Medial collateral ligament complex. (c) Lateral collateral ligament complex (Adapted and permission granted from

Bryce CD, Armstrong AD. *Anatomy and biomechanics of the elbow*. *Orthop Clin North Am*. 2008;39(2):141-54, v)

The muscles that cross the elbow control motion and compress the bony architecture, acting as dynamic stabilizers [2, 4]. The medial flexors of the elbow resist valgus force. Conversely, the lateral extensor muscles resist varus force. The distal biceps tendon crosses the elbow anteriorly and inserts at the posterior/ulnar aspect of the radial tuberosity [7]. The brachialis muscle crosses the elbow and has two heads, the superficial and deep heads. Both heads insert on the ulnar tuberosity, with the deep head inserting proximal to the superficial head [8]. The triceps crosses the elbow posteriorly and inserts on the olecranon. The tendon expands distally and its size correlates with the size of the olecranon [9].

There have been 150 bursae identified in the body, and the olecranon bursa is one of the best known [10]. The olecranon bursa is situated between the olecranon and the skin, and is formed in response to pressure, acting as a lubricating structure for the olecranon [10].

Three major nerves cross the elbow joint. The median nerve crosses anteriorly, medial to the brachial artery and is covered by the bicipital aponeurosis. The nerve then goes deep between the two heads of the pronator teres. The radial nerve emerges between the brachialis and brachioradialis, and it divides at the radiohumeral joint. The ulnar nerve crosses the elbow in the

groove posterior to the medial epicondyle, entering the forearm between the two heads of the flexor carpi ulnaris (FCU). The brachial artery runs on the lateral side of the median nerve in the cubital fossa and then divides into the radial and ulnar arteries [11].

25.2 General Evaluation of the Elbow

25.2.1 Physical Exam of Elbow

A physical exam of the elbow should be performed prior to participation. A thorough physical exam of the elbow, with a fully exposed limb and contralateral limb, is necessary for guiding and interpreting further studies and establishing a proper diagnosis.

The elbow exam begins with inspection. The overall alignment of bilateral elbows should be noted. Due to the bony anatomy, the elbow is aligned in 11–16° of valgus. This angle is referred to as the “carrying angle” and is defined by the long axes of the extended forearm and arm [12, 13]. A decrease in valgus alignment of the elbow can result from prior trauma, such as a pediatric supracondylar humerus fracture [14], and can be detrimental to the overall functioning of the limb.

Next, musculature is examined for atrophy or hypertrophy, providing clues to the chronicity of the injury. The skin is then examined for ecchymosis, rashes, nodules, and prior surgical incisions. A joint effusion can be observed if the soft spot (border of the olecranon, radial head, and lateral epicondyle) is distended [13, 15].

The bony and soft tissue structures should be palpated. Begin by palpating the epicondyles for tenderness. The olecranon and radial head are subcutaneous bony structures that are also easily palpated. The distal biceps tendon is palpated as it courses over the anterior elbow. Disruption of the normal contour of the distal biceps tendon and asymmetric proximal migration of the musculotendinous junction with respect to the antecubital flexion crease may be a sign of a partial or full tendon rupture. Tenderness over the posterior aspect of the medial epicondyle may indicate ulnar neuritis, and tenderness in the soft spot may indicate joint irritation secondary to synovitis, plica, or osteochondritis dissecans of the capitellum [15].

Normal elbow ROM is 145° of flexion, 0° of extension, 75° of pronation, and 85° of supination [15, 16]. Intra-articular pathology, pain, and soft-tissue constraints can limit normal ROM following injury. Crepitus, pain, and mechanical symptoms should be noted during attempted ROM [13, 15]. If there is a limit in full ROM, it should be determined if there is a soft endpoint, suggesting a joint effusion, soft tissue swelling, or capsular tightness [15]; or a firm endpoint that suggests a bony block or loose body. Strength testing should be performed and compared to the other limb.

There are several special tests used about the elbow to provide clues to the underlying pathology. These tests will be described below under the specific injury.

25.2.2 Imaging Evaluation of the Elbow

Following a thorough history and physical exam, a decision should be made as to whether radiographs are needed. Radiographs provide information regarding osseous structure, joint

relationship, and overall alignment [17]. The standard views include an AP and lateral. Oblique views may be obtained to supplement the standard views. The use of MRI is limited in acute injury but is the modality of choice when evaluating soft tissue structures [17]. Ultrasound may be used for evaluating tendon injuries, elbow joint effusions, and intra-articular loose bodies. CT is used to further define osseous anatomy following radiographs.

25.3 Common Basketball-Related Injuries

25.3.1 Elbow Instability

General: The elbow is the second most common joint to suffer dislocation, with an incidence of 5–6 per 100,000 people yearly [18, 19]. Forty-five percent of all elbow dislocations are sports-related, with an estimated 1435 dislocations related to basketball in the United States over a 5-year period [19]. Due to the osseous, capsuloligamentous, and muscular anatomy, the elbow is an inherently stable joint. Ninety percent of dislocations occur posteriorly or posterolaterally, as a result of a fall on an outstretched hand with an extended elbow and a fully supinated forearm [20, 21]. Posteromedial dislocations are rare, also occur after a fall onto an outstretched hand, with the elbow extended and are associated with more severe soft-tissue injuries. There is often a missed anteromedial coronoid facet fracture [20]. A fall onto the hand with the elbow extended results in an axial load through the elbow as the hand impacts the floor. As the body-weight continues to ground with the hand planted, this typically results in external rotation and valgus stress at the ulnohumeral joint [21, 22]. While more recent research has shown that the injury pattern is more variable [23], classically the mechanism begins disrupting what has been called “The Circle of Horii” [24]. Injury begins with disruption of the LCL, moves to the anterior capsule, and finally to the MCL, resulting in a complete dislocation. Although rare in the basketball population, isolated MCL injuries can occur leading to valgus instability.

Initial Assessment and Management: An elbow dislocation may occur in isolation or in conjunction with other injuries of the limb. Therefore, a thorough exam of the shoulder, forearm, and wrist should be done. A dislocated elbow will typically show evident deformity, and the forearm will be in varus and supination [22]. A careful neurovascular exam should be performed. Standard radiographs should be obtained to determine if the dislocation is simple or complex (with associated fracture). After a dislocation is confirmed, a reduction maneuver is required. This is best performed with adequate analgesia which may require conscious sedation in an emergency department in some cases [24]. Reduction is performed by correcting any medial or lateral displacement of the olecranon, followed by flexing the elbow and supinating the forearm while applying longitudinal traction [22, 24]. Following reduction, a repeat clinical examination should be performed, especially a thorough neurovascular exam. The elbow should be taken through a full ROM. The point at which instability first occurs should be noted as should the arc of stable ROM. The elbow should be immobi-

lized. At time of clinic follow-up, varus and valgus instability should be assessed along with posterolateral (PL) instability. PL instability is assessed with the lateral pivot shift test (Fig. 25.2). If elbow instability is thought to be secondary to MCL injury, valgus stress should be applied at 30° of elbow flexion and laxity compared to the uninjured elbow; the “milking maneuver” may also be performed to help identify injury. Radiographs should be repeated to confirm reduction, reevaluate for a complex injury, and ensure there are no loose bodies or other structures entrapped in the joint and blocking concentric reduction. Uncommonly, typically with a very high-energy injury, the elbow may not be able to be reduced by closed reduction and an open reduction must be performed. After radiographs confirm reduction, an MRI arthrogram can assess for LCL and MCL pathology.

Treatment Options: Following reduction, the majority of simple elbow dislocations may be treated nonoperatively. For simple, stable dislocations, early active mobilization starting 2 days after the injury has been shown to be a safe and effective treatment, with improved functional

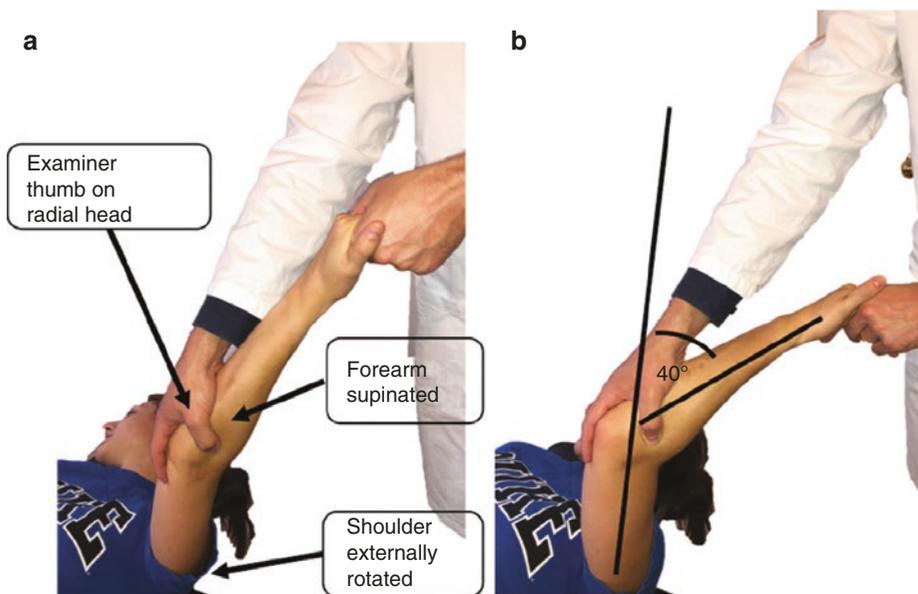


Fig. 25.2 Lateral pivot shift test: The patient is supine on an examination table. (a) The elbow is brought overhead and fully extended. The examiner’s opposite side hand is brought over the dorsum of the forearm and thumb rests over the radial head. The other hand grasps the patient’s hand and supinates the forearm and externally rotates the

shoulder. The elbow is brought from full extension to flexion. While the elbow is being ranged, a valgus stress is applied. The test is abnormal if the patient experiences pain and/or apprehension and/or if there is a palpable dislocation and reduction of the radial head. (b) Reduction of the radial head is usually felt beyond 40° of flexion

outcomes compared to 3 weeks of immobilization [25]. Surgical repair should be considered if the elbow requires more than 45° of flexion to remain reduced [24]. Initial treatment for isolated MCL injuries is typically 6 weeks of rest and physical therapy. If nonoperative treatment fails, MCL reconstruction, the so-called Tommy John Surgery, is indicated.

Rehabilitation: Following the initial immobilization period, an overhead motion protocol has been developed with excellent results [26]. The rehabilitation protocol consists of ROM at the elbow in an overhead and stable position. The athlete performs the exercise in a supine position, shoulder flexed to 90°, adducted to the side and in neutral rotation; this position minimizes varus force and stress on the LCL complex. The athlete then moves through pronation/supination and flexion/extension as tolerated. This phase continues for 3 weeks and then ROM in the upright position begins for an additional 3 weeks. Finally, 6 weeks after injury, strength and endurance exercises begin as well as resumption of normal activities.

Elbow Instability

- The elbow is the second most commonly dislocated joint.
- There are approximately 287 basketball-related dislocations annually in the United States of America. Global numbers are not known.
- 90% of dislocations occur posteriorly or posterolaterally
- Injury pattern typically follows “the circle of Horii”: LCL → anterior capsule → MCL → complete dislocation.
- Surgical repair should be considered if the elbow requires more than 45° of flexion to remain reduced.

25.3.2 Elbow Fractures

General: Fractures about the elbow may involve the distal humerus, radial head, and/or the proximal ulna. Stability of the elbow and the status of

the soft tissues are a critical consideration when treating elbow fractures. Standard AP and lateral radiographs should be obtained. A CT scan often is needed to evaluate the complexity of the injury and to evaluate the integrity of the coronoid and the anteromedial facet, an important stabilizer to prevent posteromedial rotatory instability [27].

Initial Assessment and Management: Elbow fractures in the athlete often are the result of a high-energy mechanism with a fall onto an outstretched arm. Initial evaluation should include a thorough exam of the skin and neurovascular structures. Radiographs should be obtained immediately to evaluate the extent of the injury and the presence of an associated dislocation. Fractures of the distal humerus may be difficult to interpret initially. A radiograph with gentle traction may help the treating surgeon to understand the fracture pattern [28]. Fractures of the radial head that are displaced greater than 2 mm often are associated with ligamentous injury and thus the stability of the elbow may be compromised [29]. A radiocapitellar view of the elbow may be obtained to further evaluate the radial head. This is done by positioning the beam 45° cephalad during a lateral radiograph. Fractures of the proximal ulna can involve the olecranon and coronoid process. Both olecranon and coronoid fractures may be associated with concomitant fractures and/or dislocations. As in fractures of the radial head, associated instability must be considered as this guides surgical and postoperative treatments.

Treatment Options: Generally, the treatment of elbow fractures is to both obtain an anatomic reduction of the articular surface to lower the risk of post-traumatic osteoarthritis and also create a stable construct to allow early ROM, to prevent long-term stiffness. A notable exception is a nondisplaced radial head fracture or small displaced radial head fractures with no block to motion. These injuries can be managed in a sling with active mobilization as early as possible [29] and return of function typically 6 weeks from injury [28].

Rehabilitation: Postoperative ROM is determined by the stability of the elbow found intraoperatively. Furthermore, the length of

immobilization depends on the severity of the fracture. In general, there is a period of immobilization followed by gentle passive and assisted active ROM. Oftentimes, a hinged elbow brace is worn for added stability during ROM exercises. After this, strengthening exercises can begin.

25.3.3 Elbow Tendon Injuries

25.3.3.1 Biceps Tendon Injury

General: The incidence of distal biceps tendon rupture is 1.2 per 100,000 people in the general population [30]. Ruptures are more common in the dominant extremity and the risk is increased with smoking [30]. The use of anabolic steroids increases the risk of bilateral rupture [31]. Ruptures can be classified as partial or complete and also as acute (less than 4 weeks) or chronic. The mechanism is usually an unexpected, eccentric loading of the biceps, with the elbow flexed at 90°, resulting in the tendon avulsing from the radial tuberosity [32].

Initial Assessment and Management: Athletes will typically describe a feeling of a sudden, painful tearing sensation over the front of the elbow [32]. On exam, there typically will be ecchymoses and swelling in the antecubital fossa. A complete tear of the tendon commonly results in proximal migration of the muscle belly from the antecubital crease. This is accompanied by tenderness in the antecubital fossa and an inability to palpate the tendon in the antecubital fossa, indicating a complete rupture. Weakness will be typically demonstrated in supination and, to a lesser extent, with elbow flexion [32]. Special maneuvers include the “hook test,” which involves the examiner using his or her index finger to hook the lateral side of the distal biceps tendon while the athlete actively supinates the elbow [33]. A “biceps squeeze test” has also been validated. This test involves squeezing the biceps muscle belly and observing for passive supination [34]. Radiographs should be obtained to evaluate for other pathology and are usually normal; however, avulsion injuries of the radial tuberosity can be demonstrated [32]. MRI, especially in the FABS (flexion, abduction, supination) position [35], can help delineate between

partial and complete ruptures in the setting of an inconclusive physical exam.

Treatment Options: Nonoperative management consists of immobilization followed by physical therapy. In the athlete, this management is typically reserved for partial ruptures. Nonoperative treatment has an increased risk for activity-related pain and loss of supination strength [36, 37]. For complete ruptures in athletes, anatomic reattachment is indicated in order to allow for return of flexion and supination strength [32].

Rehabilitation: Postoperatively the elbow is immobilized in 90° of flexion and supination for 1 week. Early motion is important to prevent stiffness. After 1 week, the athlete is transitioned to a hinged elbow brace with an extension block which is progressively extended. Strenuous lifting and unrestricted activities are limited for a total of 5 months [32].

25.3.3.2 Triceps Tendon Injury

General: Triceps tendon ruptures are rare and account for only 0.8% of tendon ruptures [38, 39]. The disruption usually occurs as an avulsion from the tendon insertion and less commonly an intramuscular tear occurs [39]. The loss of extension strength from a triceps tendon injury can be devastating to the basketball player [37].

Initial Assessment and Management: History usually involves an elbow extension activity with a sudden, eccentric triceps contraction, such as a fall on an outstretched hand [39]. The provider should inquire about steroid use or metabolic disorders as these can weaken tendons [40]. Acute injuries present with swelling, ecchymosis, and tenderness over the olecranon. There will be weakness in elbow extension on exam. Interestingly, complete tears may have preservation of elbow extension due to an intact lateral tendon expansion [37]. Diagnosis on exam can be difficult given the degree of swelling that often is associated with acute injury [39]. Radiographs should be obtained to evaluate for bony avulsion or associated injuries, and, as described in distal biceps tendon injuries, MRI can aid in determining partial versus complete ruptures.

Treatment Options: Nonoperative treatment can be considered in tears involving less than

50% of the tendon although biomechanical studies have shown a 40% loss of strength with only 2 cm of gapping of the tendon [41]. Therefore, in the athlete, repair of partial and complete tears is indicated in order to regain the strength needed to participate at pre-injury levels [37, 39]. Primary repair should be performed within 3 weeks of injury as after 3 weeks, reconstruction of the tendon will be likely needed [39]. Following repair or reconstruction, 4/5–5/5 strength can be expected as can recovery of 99% of endurance compared to the uninjured arm [39].

Rehabilitation: Nonoperative treatment typically includes immobilizing the elbow in 30° of extension for 4 weeks, followed by ROM exercises for another 4 weeks. Finally, at 8 weeks after the injury, strengthening exercises can commence. This course is not commonly chosen in elite basketball players [37]. Surgical treatment is more commonly performed for full-thickness tears. Following repair, the elbow is immobilized in 30° of flexion for 2 weeks, then ROM exercises are started. Return to sport is permitted after 4–6 months following surgical treatment [37].

Elbow Tendon Injuries

- Biceps tendon ruptures are more common in the dominant extremity.
- Anabolic steroids increase the risk of bilateral distal biceps tendon ruptures.
- A hook test and biceps squeeze test can be diagnostic clues to complete biceps tendon rupture.
- Triceps tendon ruptures are rare and account for only 0.8% of tendon ruptures.

25.3.4 Lateral and Medial Epicondylitis

General: Lateral and medial epicondylitis result from overuse activity, particularly repetitive wrist extension and flexion, respectively [42]. Obesity and tobacco use have been implicated as risk fac-

tors as well [42]. The extensor carpi radialis brevis (ECRB) tendon is the most commonly affected tendon in lateral epicondylitis, but the extensor digitorum communis (EDC) may also be involved [43, 44]. In medial epicondylitis, the flexor-pronator origin is involved [44]. Interestingly, the pathology is the result of microtrauma and degeneration rather than inflammation [44] as the name implies.

Initial Assessment and Management: The diagnosis of lateral and medial epicondylitis is primarily made clinically. Athletes will commonly complain of an achy or burning sensation over the lateral or medial aspect of their elbow. The pain is made worse with activities that involve resisted wrist extension (lateral epicondylitis) or flexion (medial epicondylitis), and grip weakness is a common complaint [42]. The elbow commonly appears normal, but there will be tenderness along the lateral or medial epicondyle. In lateral epicondylitis, the pain often is reproduced with resisted long finger and wrist extension. Further imaging is often not required, but if the diagnosis is in question, plain radiographs may be obtained, and they are generally normal. Classically, ultrasound will show hypoechoic swelling of the tendon origin [45] and MRI will demonstrate an intratendinous signal [46].

Treatment Options: Ninety percent of athletes will have resolution of their symptoms following nonoperative management [42] although there is no established appropriate treatment protocol [47]. Activity modification, physical therapy, and using forearm support bands (the tennis elbow strap) are the first lines of treatment [37]. Therapy should involve eccentric exercises and stretching. Avoidance of lifting activities with the palm down (e.g., reverse curls) is paramount. Other modalities include deep massage with or without the use of ultrasound [37]. The use of a corticosteroid injection is controversial, and there is no evidence that shows improved outcomes when compared to less invasive methods. Interestingly, a randomized controlled trial comparing injections with physical therapy showed a 69% success rate with injection and 91% with therapy [48]. After failed nonoperative treatment, surgery may be considered. Surgery involves excision of the

affected portion of the tendon. Interestingly, when surgery was compared to a sham procedure, both provided relief of symptoms and surgery showed no additional benefit [49].

Rehabilitation: Following surgery, a wrist splint is worn for 2 weeks and physical therapy starts 4 weeks after surgery. Return to weight lifting or sports is allowed 3 months after surgery [37].

Lateral and Medial Epicondylitis

- Overuse injuries that commonly involve the ECRB and flexor mass.
- 90% of athletes will have resolution of their symptoms following nonoperative management.
- The use of a corticosteroid injection is controversial, and there is no evidence that shows improved outcomes.

25.3.5 Olecranon Bursitis (Non-Infectious and Infectious)

General: Bursae (L. “sac”) are fluid-filled sacs that allow smooth gliding of musculoskeletal structures over one another during motion. The olecranon bursa is a superficial bursa. It forms between the ages of 7 and 10 years and protects the posterior elbow. Olecranon bursitis is an abnormal increase in the volume of fluid. If this fluid becomes infected, it is referred to as infectious olecranon bursitis [50]. The incidence of olecranon bursitis is low and is estimated between 0.01% and 0.1% of hospital admissions [10]. However, it is reportedly common among basketball players, although the incidence is unknown. The etiology is most commonly from repetitive trauma or repetitive pressure to the posterior elbow, but it can present from a single fall onto the elbow [37, 50].

Initial Assessment and Management: History of recent falls onto the elbow or repetitive pressure over the elbow should be elucidated. The athlete will often complain of fullness over the back of his or her elbow. On physical exam, the fullness can be observed and palpated [37]. It is

important to rule out an infectious joint by checking for pain with small-arc elbow ROM including pain with pronation and supination. In olecranon bursitis, pain is localized over the olecranon and ROM of the elbow is preserved. Distinguishing between olecranon bursitis and infectious olecranon bursitis can be difficult (Fig. 25.3). Both can present with erythema over the posterior elbow. However, systemic signs of infection can be helpful in diagnosing infectious olecranon bursitis. The gold standard for diagnosing infectious olecranon bursitis is a positive culture [10]. Radiographs should be obtained to rule out a fracture, especially with a history of trauma. Transillumination or ultrasound can be helpful in more chronic cases to identify loculations [10].

Treatment Options: Olecranon bursitis is an inflammatory process and should be treated as such. Avoiding inciting activities (such as resting elbow on a table or armrest), taking anti-inflammatory medications, and compression are the mainstay treatments. Aspiration can be performed to decompress the bursa. In patients with very thin skin, it may be helpful to approach the bursa from proximally to create a longer skin bridge and avoid chronic drainage. A study found that inserting a 16-gauge angiocatheter with a surrounding dressing for 3 days resulted in fewer recurrences when compared to a single



Fig. 25.3 Septic olecranon bursitis: 45-year-old female with chronic olecranon bursitis with 2 weeks of worsening posterior elbow pain and staphylococcus aureus positive cultures from aspiration

aspiration [51]. If nonoperative modalities fail, excision of the bursa is the surgical treatment of choice.

Rehabilitation: The athlete should be informed that resolution of olecranon bursitis can take up to 3 months, and compliance with compression and anti-inflammatory medications is mandatory. Following surgical excision, a splint is worn for 2 weeks followed by a compressive wrap. Return to basketball is typically allowed 6 weeks after surgery [37].

Olecranon Bursitis

- Commonly seen in basketball players although the incidence is low, estimated between 0.01 and 0.1% in the general population.
- Distinguishing between olecranon bursitis and infectious olecranon bursitis can be difficult, and labs, vital signs, and an aspiration can aid in differentiating between the two.

25.3.6 Neuropathies at the Elbow

General: Compressive neuropathies about the elbow may be the cause of pain and weakness in the basketball player [52]. The nerves that may be affected in neuropathies at the elbow include the ulnar, median, and radial nerves.

Initial Assessment and Management: Athletes with compressive neuropathy about the elbow will present with vague pain around their elbow, paresthesia, and weakness depending on the nerve involved and the location of compression. Electrophysiologic testing can be used to help confirm the diagnosis and determine the extent of nerve damage. MRI may be used to assess the soft tissue anatomy surrounding the nerve in question.

The ulnar nerve pierces the intermuscular septum when the nerve travels from the anterior to the posterior compartment at the mid-arm. In about 70% of individuals, the nerve travels through the arcade of Struthers at the intermuscu-

lar septum, where compression can occur [52]. The nerve then descends through the cubital tunnel, deep to the Osborne ligament, and then enters the forearm between the two heads of the FCU muscle. Compression of the ulnar nerve at the elbow results in paresthesia of the ring and small fingers. Often the symptoms are provoked with prolonged elbow flexion, such as talking on a cell phone or while curled up during sleep [52]. Chronic compression may result in atrophy of the intrinsic muscles of the hand. A positive Tinel sign at the cubital tunnel and/or prolonged elbow flexion may help confirm the diagnosis. Furthermore, the ulnar nerve may subluxate or dislocate over the medial epicondyle, which may cause a friction injury and predispose to ulnar neuropathy [53].

The median nerve travels in the antecubital fossa, medial to the brachial artery. Prior to reaching the antecubital fossa, it passes under the ligament of Struthers, which is a band of connective tissue attached to a supracondylar process and the distal humerus, found in 1% of the population. Distal to the antecubital fossa, it goes deep to the bicipital aponeuroses and then passes between the two heads of the pronator teres. Athletes typically present with pain in the proximal, anterior forearm [37]. Paresthesia in the median nerve distribution can be noted. Median neuropathy results in weak pronation, weak flexion, and thenar atrophy [52].

The radial nerve enters the radial tunnel at the radiocapitellar joint. Then, the posterior interosseous nerve (PIN) branches off and enters the supinator muscle and dives under the arcade of Frohse [52]. Compression of the radial nerve can be divided into radial tunnel syndrome and PIN syndrome. Radial tunnel syndrome results in lateral elbow pain that can be mistaken for lateral epicondylitis. In radial tunnel syndrome, there is no associated motor weakness. In contrast, PIN syndrome is a motor neuropathy that results in muscle weakness [52].

Treatment Options: For compressive neuropathies, 3–6 months of nonoperative treatment should be pursued. This may consist of activity modification, anti-inflammatory medications, splinting, and/or injections [37]. If nonoperative

treatment fails or motor symptoms arise, surgical decompression is indicated.

Rehabilitation: Following surgical decompression mobilization should begin within 1 week to avoid elbow stiffness.

25.4 Conclusion

An understanding of the complex elbow anatomy is necessary to properly diagnose and treat basketball-related elbow injuries. Proper diagnosis begins with a thorough history and physical exam and is aided by further studies as needed. Treatment ranges from activity modification and physical therapy to complex fracture and instability procedures. The goals of treatment are to have full and painless range of motion that allows for return to play.

References

- Bryce CD, Armstrong AD. Anatomy and biomechanics of the elbow. *Orthop Clin North Am.* 2008;39(2):141–54, v.
- Karbach LE, Elfar J. Elbow instability: anatomy, biomechanics, diagnostic maneuvers, and testing. *J Hand Surg Am.* 2017;42(2):118–26.
- Morrey BF, An KN. Stability of the elbow: osseous constraints. *J Shoulder Elbow Surg.* 2005;14(1 Suppl S):174S–8S.
- Safran MR, Baillargeon D. Soft-tissue stabilizers of the elbow. *J Shoulder Elbow Surg.* 2005;14(1 Suppl S):179S–85S.
- King GJ, Morrey BF, An KN. Stabilizers of the elbow. *J Shoulder Elbow Surg.* 1993;2(3):165–74.
- Olsen BS, Vaesel MT, Sojbjerg JO, Helmig P, Sneppen O. Lateral collateral ligament of the elbow joint: anatomy and kinematics. *J Shoulder Elbow Surg.* 1996;5(2 Pt 1):103–12.
- Hutchinson HL, Gloystein D, Gillespie M. Distal biceps tendon insertion: an anatomic study. *J Shoulder Elbow Surg.* 2008;17(2):342–6.
- Leonello DT, Galley IJ, Bain GI, Carter CD. Brachialis muscle anatomy. A study in cadavers. *J Bone Joint Surg Am.* 2007;89(6):1293–7.
- Keener JD, Chafik D, Kim HM, Galatz LM, Yamaguchi K. Insertional anatomy of the triceps brachii tendon. *J Shoulder Elb Surg.* 2010;19(3):399–405.
- Reilly D, Kamineni S. Olecranon bursitis. *J Shoulder Elb Surg.* 2016;25(1):158–67.
- Hoppenfeld S, De Boer PG, Hutton R, Thomas HA. Surgical exposures in orthopaedics: the anatomic approach. 4th ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2009. xxii. p. 741.
- Chang CW, Wang YC, Chu CH. Increased carrying angle is a risk factor for nontraumatic ulnar neuropathy at the elbow. *Clin Orthop Relat Res.* 2008;466(9):2190–5.
- Hausman MR, Lang P. Examination of the elbow: current concepts. *J Hand Surg Am.* 2014;39(12):2534–41.
- Labelle H, Bunnell WP, Duhaime M, Poitras B. Cubitus varus deformity following supracondylar fractures of the humerus in children. *J Pediatr Orthop.* 1982;2(5):539–46.
- Smith MV, Lamplot JD, Wright RW, Brophy RH. Comprehensive review of the elbow physical examination. *J Am Acad Orthop Surg.* 2018;26(19):678–87.
- Boone DC, Azen SP. Normal range of motion of joints in male subjects. *J Bone Joint Surg Am.* 1979;61(5):756–9.
- Chen AL, Youm T, Ong BC, Rafii M, Rokito AS. Imaging of the elbow in the overhead throwing athlete. *Am J Sports Med.* 2003;31(3):466–73.
- de Haan J, Schep NW, Tuinebreijer WE, Patka P, den Hartog D. Simple elbow dislocations: a systematic review of the literature. *Arch Orthop Trauma Surg.* 2010;130(2):241–9.
- Stoneback JW, Owens BD, Sykes J, Athwal GS, Pointer L, Wolf JM. Incidence of elbow dislocations in the United States population. *J Bone Joint Surg Am.* 2012;94(3):240–5.
- Cho CH, Kim BS, Rhyou IH, Park SG, Choi S, Yoon JP, et al. Posteromedial elbow dislocations without relevant osseous lesions: clinical characteristics, soft-tissue injury patterns, treatments, and outcomes. *J Bone Joint Surg Am.* 2018;100(23):2066–72.
- Robinson PM, Griffiths E, Watts AC. Simple elbow dislocation. *Shoulder Elbow.* 2017;9(3):195–204.
- Grazette AJ, Aquilina A. The assessment and management of simple elbow dislocations. *Open Orthop J.* 2017;11:1373–9.
- Luukkala T, Temperley D, Basu S, Karjalainen TV, Watts AC. Analysis of magnetic resonance imaging-confirmed soft tissue injury pattern in simple elbow dislocations. *J Shoulder Elb Surg.* 2019;28(2):341–8.
- Hobgood ER, Khan SO, Field LD. Acute dislocations of the adult elbow. *Hand Clin.* 2008;24(1):1–7.
- Iordens GI, Van Lieshout EM, Schep NW, De Haan J, Tuinebreijer WE, Eygendaal D, et al. Early mobilisation versus plaster immobilisation of simple elbow dislocations: results of the FuncSiE multicentre randomised clinical trial. *Br J Sports Med.* 2017;51(6):531–8.
- Schreiber JJ, Paul S, Hotchkiss RN, Daluiski A. Conservative management of elbow dislocations with an overhead motion protocol. *J Hand Surg Am.* 2015;40(3):515–9.
- Ring D, Doornberg JN. Fracture of the anteromedial facet of the coronoid process. Surgical technique. *J Bone Joint Surg Am.* 2007;89(Suppl 2 Pt. 2):267–83.

28. Green DP. Green's operative hand surgery. 5th ed. Philadelphia, PA: Elsevier/Churchill Livingstone; 2005.
29. Kodde IF, Kaas L, Flipsen M, van den Bekerom MP, Eygendaal D. Current concepts in the management of radial head fractures. *World J Orthop.* 2015;6(11):954–60.
30. Safran MR, Graham SM. Distal biceps tendon ruptures: incidence, demographics, and the effect of smoking. *Clin Orthop Relat Res.* 2002;404:275–83.
31. Schneider A, Bennett JM, O'Connor DP, Mehlhoff T, Bennett JB. Bilateral ruptures of the distal biceps brachii tendon. *J Shoulder Elbow Surg.* 2009;18(5):804–7.
32. Ramsey ML. Distal biceps tendon injuries: diagnosis and management. *J Am Acad Orthop Surg.* 1999;7(3):199–207.
33. O'Driscoll SW, Goncalves LB, Dietz P. The hook test for distal biceps tendon avulsion. *Am J Sports Med.* 2007;35(11):1865–9.
34. Ruland RT, Dunbar RP, Bowen JD. The biceps squeeze test for diagnosis of distal biceps tendon ruptures. *Clin Orthop Relat Res.* 2005;437:128–31.
35. Giuffre BM, Moss MJ. Optimal positioning for MRI of the distal biceps brachii tendon: flexed abducted supinated view. *AJR Am J Roentgenol.* 2004;182(4):944–6.
36. Chillemi C, Marinelli M, De Cupis V. Rupture of the distal biceps brachii tendon: conservative treatment versus anatomic reinsertion—clinical and radiological evaluation after 2 years. *Arch Orthop Trauma Surg.* 2007;127(8):705–8.
37. DeLee J, Drez D, Miller MD, Thompson SR. DeLee & Drez's orthopaedic sports medicine : principles and practice. 4th ed. Philadelphia, PA: Elsevier/Saunders; 2015.
38. Anzel SH, Covey KW, Weiner AD, Lipscomb PR. Disruption of muscles and tendons; an analysis of 1, 014 cases. *Surgery.* 1959;45(3):406–14.
39. van Riet RP, Morrey BF, Ho E, O'Driscoll SW. Surgical treatment of distal triceps ruptures. *J Bone Joint Surg Am.* 2003;85(10):1961–7.
40. Lambert MI, St Clair Gibson A, Noakes TD. Rupture of the triceps tendon associated with steroid injections. *Am J Sports Med.* 1995;23(6):778.
41. Hughes RE, Schneeberger AG, An KN, Morrey BF, O'Driscoll SW. Reduction of triceps muscle force after shortening of the distal humerus: a computational model. *J Shoulder Elb Surg.* 1997;6(5):444–8.
42. Taylor SA, Hannafin JA. Evaluation and management of elbow tendinopathy. *Sports Health.* 2012;4(5):384–93.
43. Appelboam A, Reuben AD, Bengler JR, Beech F, Dutson J, Haig S, et al. Elbow extension test to rule out elbow fracture: multicentre, prospective validation and observational study of diagnostic accuracy in adults and children. *BMJ.* 2008;337:a2428.
44. Kraushaar BS, Nirschl RP. Tendinosis of the elbow (tennis elbow). Clinical features and findings of histological, immunohistochemical, and electron microscopy studies. *J Bone Joint Surg Am.* 1999;81(2):259–78.
45. Radunovic G, Vlad V, Micu MC, Nestorova R, Petranova T, Porta F, et al. Ultrasound assessment of the elbow. *Med Ultrason.* 2012;14(2):141–6.
46. Martin CE, Schweitzer ME. MR imaging of epicondylitis. *Skelet Radiol.* 1998;27(3):133–8.
47. Bateman M, Titchener AG, Clark DI, Tambe AA. Management of tennis elbow: a survey of UK clinical practice. *Shoulder Elbow.* 2019;11(3):233–8.
48. Smidt N, van der Windt DA, Assendelft WJ, Deville WL, Korthals-de Bos IB, Bouter LM. Corticosteroid injections, physiotherapy, or a wait-and-see policy for lateral epicondylitis: a randomised controlled trial. *Lancet.* 2002;359(9307):657–62.
49. Krosiak M, Murrell GAC. Surgical treatment of lateral Epicondylitis: a prospective, randomized, double-blinded, placebo-controlled clinical trial. *Am J Sports Med.* 2018;46(5):1106–13.
50. Blackwell JR, Hay BA, Bolt AM, Hay SM. Olecranon bursitis: a systematic overview. *Shoulder Elbow.* 2014;6(3):182–90.
51. Fisher RH. Conservative treatment of distended patellar and olecranon bursae. *Clin Orthop Relat Res.* 1977;123:98.
52. Bencardino JT, Rosenberg ZS. Entrapment neuropathies of the shoulder and elbow in the athlete. *Clin Sports Med.* 2006;25(3):465–87. vi-vii
53. Yang SN, Yoon JS, Kim SJ, Kang HJ, Kim SH. Movement of the ulnar nerve at the elbow: a sonographic study. *J Ultrasound Med.* 2013;32(10):1747–52.



Wrist and Hand Injuries in Basketball

26

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26.1 Introduction

Originally invented in the USA by James Naismith [1], basketball is one of the most beloved and popular sports all over the world. Basketball was developed as a non-contact sport and relies on the concept that “if the offense did not have the opportunity to run with the ball, there would be no necessity for tackling and we would thus eliminate roughness” [2]. However, basketball has transformed over the years into an extremely physical game in which contact is unavoidable. Injuries that occur during basketball are associated with one or more of the following anatomical structures: *bone*: scaphoid, metacarpal, and hook of hamate fractures; *ligaments*: scapholunate ligament and triangular fibrocartilage complex ligament; *tendons*: ECU tendonitis and subluxation.

Prompt diagnosis and appropriate treatment are essential to avoid complications. Diagnosis is based on clinical examination, thorough

physical evaluation, and necessary imaging such as X-rays, computed tomography (CT) scan, or magnetic resonance imaging (MRI). When making treatment decisions, it is important to take into consideration the level of competition and time of season for the athlete. In every case, determining the type of sport, time of season, competitive level, and rules regarding cast wear during play are critical and should be discussed. Each individual athlete requires tailored treatment strategies. It is crucial to take all possible precautions to limit the risk of consequent injury and future posttraumatic arthritis.

26.2 Epidemiology

A few reports exist in the medical literature regarding the epidemiology of basketball injuries. Some examine injuries of professional athletes and college or high school students and others focus on adult non-professional athletes.

Drakos et al. [3] reviewed injuries and medical conditions afflicting athletes competing in the National Basketball Association (NBA) over a 17-year period. They identified 12,594 injuries, of which 1945 (15.4%) involved the upper extremity. In particular, there were 571 (4.5%) wrist injuries with a sprain in 181 ath-

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letes (1.4%), hand or wrist fracture in 100 (0.8%), and hand contusion in 69 (0.5%) athletes. It is remarkable that in 1982, Henry et al. [4] reported 576 injuries that occurred in 71 professional basketball players over 7 years. Over the years, basketball became more popular, and eventually physical contact was increased, resulting in a concomitant rise in injuries. McCarthy et al. [5] reviewed the WNBA medical records for all athletes entering the WNBA in 2000–2008, excluding 2003. The study included 506 athletes, in which 105 hand or wrist injuries (20.8%) were reported.

Conn et al. [6] found basketball to be the most frequently reported sport that resulted in an injury among people age 5 years and older, comprising 25% of all sports injuries that required treatment. They included fractures of the hand, wrist, or fingers (22,000, 40% of all fracture injuries). Andreoli et al. [7] also reviewed with regard to upper limb injuries of the hand, finger, and wrist (1133 injuries, 8.7%). In females, injury to the hand, finger and wrist represented 8.6% (369) of injuries. In males, hand, finger, and wrist injuries comprised 8.4% (386) of all injuries. When investigating by age and skill level, they reported that children and adolescent injuries in the hand, finger and wrist represented 8.9% (662) of injuries. In professionals, the incidence was 8.6% (454) and in masters 7.1% (17).

26.3 Scaphoid Fractures

One of the most frequent fractures of the wrist in basketball athletes is the fracture of the scaphoid. These fractures pose a predicament for both the athlete and doctor. Nowadays, available operative treatments minimize recovery time and promise earlier return to participation in competition [8]. Therefore, surgical fixation of these fractures has replaced casting, the traditional treatment option, in professional basketball players. Scaphoid fractures constitute 69% of all acute wrist fractures and are thus the most common carpal fracture.

Basketball athletes present the highest number of scaphoid fractures (11%) compared to other male athletes [3]. Located in the proximal carpal row, the scaphoid consists of five distinct articular surfaces. These are defined into a proximal pole, waist, and distal pole. The distal part is palmar and the proximal is dorsal.

The mechanism of injury is usually due to a fall on the outstretched hand (FOOSH) injury. During a FOOSH injury longitudinal loading is forced on the scaphoid, delivering a flexion moment that is resisted by the constraining ligaments at the proximal and distal poles and by the radioscapocapitate ligament crossing the waist. Consequently, tension gradually develops on the dorsal cortex of the curved waist. Other mechanisms of injury also exist that do not result in scaphoid fractures at the waist. For example, fractures can occur after contact with a closed fist. These are known as “puncher fractures” [9]. In addition, many athletes report the injury took place after wrist hyperextension from a blocked shot [8].

Accurate medical history informs about the mechanism of injury, and comprehensive wrist examination reveals the extent of the injury. An acute fracture is suspected when swelling or pain exists in the anatomic snuff box, between the first and third extensor compartments at the level of the medial part of the scaphoid or on the palmar scaphoid tubercle. Axial compression of the thumb is another useful clinical test. If all three clinical tests are positive, there is a very high possibility of a scaphoid fracture [10]. In addition, range of motion is examined. Flexion, extension, and radial deviation are compromised after a scaphoid waist fracture. In distal pole fractures, deterioration of range of motion is less significant.

Radiographs should be performed in the following positions: neutral rotation posteroanterior (PA), lateral and additional “scaphoid” view (Fig. 26.1)—approximately 30° of wrist extension and 20° of ulnar deviation in order to achieve an ultimate view of the scaphoid. In more than 30% of cases [10], plain radiographs will be inconclusive for a diagnosis. MRI of the wrist provides the



Fig. 26.1 “Scaphoid” view of a waist fracture of the scaphoid bone

highest sensitivity, specificity, and accuracy (all >95%) in identifying the fracture. Although its role in the diagnosis of scaphoid fractures is disputed, CT scan of all waist and proximal pole fractures of the scaphoid may also be used to evaluate displacement and comminution. For example, the humpback deformity as described by Sanders [11] is better recognized by CT.

Several classifications of scaphoid fractures have been published [12]. The Herbert classification is most commonly used (Table 26.1).

Nondisplaced scaphoid waist fractures are treated by cast immobilization. This has been the standard of care in all non-professional basketball players, and many athletes are still offered this treatment option. Fracture healing with casting achieves as high as 88%–95% success rates in a few studies [13]. Cast immobilization, usually for 6 weeks, is the best treatment option for most fractures of the distal pole of the scaphoid. For waist fractures and some proximal pole fractures, there has been some discrepancy regarding best available treatment. Upfront surgical fixation ensures adequate blood supply to the scaphoid and is usually reserved for professional players. For the amateur athlete, the conservative approach presents a good alternative to operative treatment.

Table 26.1 Herbert classification with treatment

Type	Subtype	Location		Treatment
Type A (stable)	A1	Distal pole		Short-arm thumb Spica cast Long-arm cast (painful in the shorter below-elbow cast) Surgery for professional athletes
	A2	Waist		
Type B (unstable) Middle or proximal thirds of the body of the scaphoid	B1	Distal oblique fracture		Surgery – Internal fixation with screw or plate – Fragment excision (if too small)
	B2	Waist fracture (Most common)	Indicated CT scan (angular deformity)	
	B3	Proximal pole	Indicated CT scan (proximal fragment size)	
	B4	Waist fractures that result from a trans-scaphoid perilunate dislocation		
Type C		Delayed union		Surgery

Immediate treatment of scaphoid fractures with headless screw fixation is the gold standard for professional players [8]. Percutaneous internal fixation of the scaphoid fracture is achieved via a dorsal approach. Anatomic reduction is performed by a reduction maneuver applying extension and ulnar deviation. If not applicable, open reduction through either a dorsal or palmar approach is necessary. Complete displaced proximal pole and waist scaphoid fractures are handled with internal fixation for optimal recovery [14]. Initial internal fixation achieves faster healing, earlier restoration of motion, and hence shorter minimal absence from sporting activities.

To Sum Up—Algorithm [9]

Surgery is indicated for:

- All proximal pole fractures
- Displaced scaphoid fractures (>1 mm)
- Combined injuries and fractures that were neglected for more than 1 month

Acute non-displaced scaphoid waist fractures are subject to discussion with the athlete. Earlier return to sport is achieved after surgery (~2 months) in comparison to conservative treatment (~4 months).

Molded plaster splints are used for immobilization. Removable thumb spica splints are used for waist fractures 1 week after surgery. For extreme proximal pole fractures, cast removal and early motion are not applicable for at least 4 weeks after surgery. Gentle range of motion of the wrist and forearm is initiated when the patient feels no discomfort and continues for 4 weeks [15]. If reduction and fixation are stable after radiographic imaging, strengthening of forearm flexors and extensors begins with wrist curls. Weight-bearing exercises are incorporated in the rehabilitation program once the scaphoid has healed after a minimum of 6 weeks [15]. CT follow-up imaging is performed every 6 weeks to evaluate progress until complete recovery is accomplished. Resumption of usual activities is allowed when there is at least 50% of bone bridging on CT scan,

at least 80% of normal grip strength, absence of pain, and full restoration of joint mobility.

26.4 Hook of Hamate Fractures [16]

Fractures of the hook of the hamate cause ulnar-sided palmar pain and are usually misdiagnosed as a sprain or tendinitis. Consequently, they appear as non-unions at diagnosis. In basketball, these fractures are not common. The mechanism of injury is based on direct hit or fall. Treatment consists of a non-prolonged short-arm cast for a period of 3–4 weeks. If symptoms persist, the ununited hook can be excised.

A symptomatic ununited hook of the hamate fracture presents a risk of tendon dysfunction or rupture and ulnar nerve dysfunction. For this reason, athletes should be carefully evaluated before returning to competition. Excision of the hook of the hamate must be considered early in these situations to diminish these complications.

26.5 Metacarpal Fractures

Metacarpal fractures occur frequently in sports activities and particularly in contact sports using a ball, such as basketball. In the general population, these account for 18% of fractures below the elbow and 10% of all upper extremity fractures [17]. This kind of injury can be caused either from an athlete's fall onto the outstretched hand with a closed fist or a direct hit from the ball.

Metacarpal fractures are classified based on the location of injury into neck, shaft, or base fractures. Depending on morphology, they are classified as transverse, oblique, and spiral. Most fractures are located in the neck because it is the weakest part of the metacarpal. Traditionally, metacarpal fractures were treated conservatively with closed reduction, using either cast or splint immobilization in intrinsic plus position for 4 weeks. Nowadays, professional basketball athletes with comminuted fractures are treated with surgery because they can return faster to previous activity.



Fig. 26.2 Fracture of the neck of the fifth metacarpal and treatment with CRPP

The classic indications for surgical treatment are: (1) in the metacarpal neck, any rotational deformity or neck angulation of the index and long finger greater than 15–20°, ring finger >30–40°, and small finger >70°, (2) in the metacarpal shaft, any rotational deformity or angulation of the index and long finger greater than 10°, ring and small finger >30°. Minimal shortening is accepted in the metacarpal shaft, while as Strauch et al. mentioned for each 2 mm of shortening, the extensor lag is decreased by 7° [18].

Surgical treatment for neck metacarpal fractures includes antegrade, retrograde, or transverse CRPP (closed reduction percutaneous pinning) (Fig. 26.2). On the other hand, shaft fractures can be treated with intramedullary pinning (antegrade or retrograde), lag screw fixation or a plate via a dorsal approach. The plate should be removed immediately after fracture healing because it can cause additional damage and rupture of the extensor tendons.

Base fractures differ from the other types of metacarpal fractures. These fractures occur often with carpometacarpal (CMC) dislocation and are classified into two categories: (1) fractures of the thumb base and (2) other metacarpal base frac-

tures. The thumb base fracture is classified as either extra-articular which is most commonly encountered or intra-articular Bennett and Rolando fractures. The Bennett fracture (Fig. 26.3) is an intra-articular fracture with proximal, dorsal, and radial displacement of the metacarpal shaft (caused by abductor pollicis longus (APL) and thumb extensors), along with supination and adduction of the shaft (caused by adductor pollicis). The Rolando fracture is a comminuted intra-articular fracture shaped as either the letter Y or T. These types of fractures in basketball players are mainly treated surgically with CRPP or ORIF (open reduction internal fixation) with plates or screws based on the size of the fragment. The goal of surgical treatment for these fractures is an articular step-off less than 1–2 mm.

There are few references that address the recovery time needed for an athlete with a metacarpal fracture. For non-displaced fractures, return to play 4–8 weeks after injury is suggested [19]. Other studies recommend that protected play for shaft fractures that were treated either conservatively or surgically may start at the third week after injury. However protected play is rarely possible in basketball [20].



Fig. 26.3 Bennett fracture in a basketball athlete and the appropriate treatment with a screw

26.6 Triangular Fibrocartilage Complex Injuries (TFCC)

The triangular fibrocartilage complex (TFCC) is the main component in providing distal radioulnar joint (DRUJ) stabilization. The TFCC incorporates the dorsal and volar radioulnar ligaments, the articular disc, a meniscus homolog, the ECU subsheath, and the origins of the ulnolunate and ulnotriquetral ligaments. The ulnar side of the wrist, including the TFCC, absorbs 18–20% of the total force applied across the wrist [21]. The periphery of TFCC is rich in vasculature compared to the radial central portion.

At presentation, TFCC injuries have ulnar-sided wrist pain or tenderness. At clinical examination, they display an audible or palpable click on forearm supination and pronation. Injury to the TFCC commonly occurs with extension and pronation of the axial load of the wrist. Plain radiographs (AP and lateral view) that show an ulnar styloid fracture or DRUJ incongruity are an indication of an acute TFCC injury. Recently the use of MRI arthrogram is on the rise, due to its higher sensitivity, specificity, and accuracy in comparison to the classic MRI for TFCC tears [22]. However,

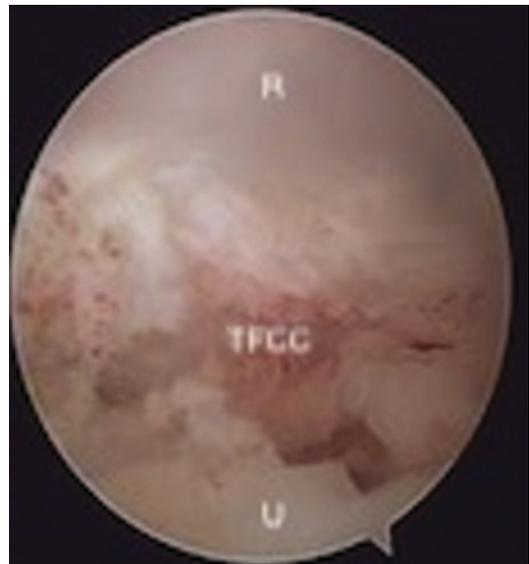


Fig. 26.4 TFCC tear, image during arthroscopy, R: radial bone, U: ulnar bone

wrist arthroscopy is still quintessential in discovering TFCC disorders. In addition, wrist arthroscopy offers both diagnosis and the ability to provide targeted treatment for professional athletes with suspicion of a TFCC disorder (Fig. 26.4).

TFCC injuries are categorized in traumatic (IA, IB, IC, ID) and degenerative (IIA, IIB, IIC, IID, IIE) as classified by Palmer. Peripheral TFCC tears without distal radioulnar joint instability create mechanical manifestations such as catching and locking [23].

Traumatic injuries are treated with sutures and debridement via wrist arthroscopy. Degenerative injuries need a more complex and invasive approach. Corticosteroid injections are offered to athletes with TFCC injuries with minimal instability in order to complete the game season. Delaying peripheral repairs until after the season [21] is an established practice in professional basketball when and if the athlete's situation allows it.

Rehabilitation after debridement of central type IA TFCC tears includes splinting for 1 week followed by range of motion exercises. Recovery is achieved within 4–5 weeks and players are capable of returning to competition.

Rehabilitation for peripheral TFCC disorders for IB, IC, and ID lesions includes 2–6 weeks postoperative immobilization with a splint or cast (to inhibit forearm rotation). After immobilization, gradual range-of-motion and strengthening exercises are initiated for the next 6 weeks. Competitive play is allowed 8–12 weeks after surgery [21].

26.7 Scapholunate Ligament Disruption

The proximal carpal row of the wrist is constructed by the scaphoid, lunate, and triquetrum bones. The scapholunate (SL) and lunotriquetral (LT) interosseous ligaments are responsible for providing fixation of these bones to the wrist. The SL ligament is further divided into the dorsal, proximal, and volar segment. The stability between the scaphoid and lunate is dependent on the dorsal segment of the SL ligament [24]. Additional stability is established through volar and dorsal radio- and ulno-carpal ligaments to the proximal row of the wrist.

Scapholunate ligamentous injuries occur most frequently after wrist extension, ulnar deviation, and carpal supination. Scapholunate injuries are

regularly misdiagnosed in athletes, as simple sprains. Excessive pain over the scapholunate area or instability on examination of the wrist, for example, a positive Watson's test, raises a high suspicion for these types of injuries.

Necessary radiographic imaging for an accurate diagnosis must include a neutral posterior-anterior (PA) and a lateral view of the wrist (Fig. 26.5). These usually reveal a cortical ring sign, a widened scapholunate interval (>3 mm) in PA view or an increased scapholunate angle (>70°) in lateral view. Stress radiographic views such as PA thumb traction, PA closed fist-neutral, and PA closed fist with radial deviation or ulnar deviation also facilitate the diagnosis [25]. Additionally, contralateral wrist comparison radiographs may further provide information about this injury. Classification of scapholunate injuries according to imaging (simple or under stress X-rays) is as follows: pre-dynamic, dynamic, or static.

Standard radiographic views are sufficient in formulating a diagnosis. However, advanced imaging is often recommended before surgery. MRI techniques (Multidetector CT and MRI arthrogram) are essential in unveiling the extent of ligament disruption (complete or incomplete). In professional athletes, the gold standard for diagnosing scapholunate injuries is diagnostic arthroscopy. In 1996, Geissler et al. described an arthroscopic classification system for SL injuries and LT injuries [26] (Table 26.2).

Treating an athlete with scapholunate ligament disruption should be individualized. The management of these injuries in basketball athletes depends on the age of the athlete, the timing in regard to the current season, and the athlete's future expectations.

Conservative management is rarely applied and mostly reserved for non-professional athletes with stable and minor tears of the SL ligament. Treatment includes wrist immobilization for 4–6 weeks followed by physiotherapy. The results of conservative treatment remain controversial.

In professional basketball athletes, surgical treatment is mainly applied. The goal is to restore stability of the wrist and ensure the return of the athlete to pre-injured performance. The choice of surgical technique is based on carpal stability,



Fig. 26.5 Scapholunate disruption with the characteristic “Terry Thomas” sign in AP view

Table 26.2 Greissler arthroscopic classification system for carpal instability

Grade	Description
I	Hemorrhage of interosseous ligament, without gap between carpals
II	The same with Grade I, gap less than width of a probe
III	Incongruity/step-off of carpal alignment is seen in both the radiocarpal and midcarpal spaces. The probe could be passed through the gap between carpals
IV	The same with Grade III. Gross instability with manipulation is noted. A 2.7-mm arthroscope may be passed through the gap between carpals

duration of injury, and the presence of degenerative lesions of the joint.

In partial tears of the SL ligament with pre-dynamic injury, the dorsal SL ligament is intact, and arthroscopy is proposed. In general, arthroscopic debridement, thermal shrinkage, and k-wire stabilization can be performed [27].

In dynamic and recoverable lesions, treatment depends on the duration of the injury. Studies

show that a time interval of 3 weeks since the injury is critical. Up to this time point, surgical treatment of SL ligament with dorsal capsulodesis or dorsal ligamentoplasty can be performed with satisfactory results [28]. For chronic lesion injuries that last over 3–6 weeks, reconstruction of the SL ligament provides better results compared to primary ligament suture or arthroscopic debridement. Several surgical techniques have been proposed for reconstruction of the ligament with a variety of results: (1) *Brunelli technique*, described in 1995, with transferring a slip of the flexor carpi radialis (FCR) through scaphoid tunnel (volar to dorsal) and stabilized to the lunate and the distal radius; (2) “*three ligament tenodesis*” or *modified Brunelli by Garcia Elias*, using as a graft the FCR tendon [29]; (3) *Elsaftawy technique* [30], using a free tendon as graft which restores the scapholunate ligament; (4) *Weiss technique*, utilizing bone-retinaculum-bone autograft [31]; (5) *Ross technique*, using FCR for scapholunotriquetral tenodesis via tunnel through the scaphoid, lunate, and triquetral [32].

26.8 ECU Tendonitis and Subluxation [33]

Another common cause of ulnar side wrist pain in basketball players is ECU tendonitis or tendon subluxation. Injury of the ECU tendon or other pathologic entities such as tendinosis, subluxation, traumatic dislocation, or rupture must be suspected when an athlete presents with this complaint. A single forceful wrist flexion/ulnar deviation/supination event can be responsible for this injury. Physical evaluation and necessary imaging (US or MRI) facilitate the diagnosis.

Acute and chronic tendonitis are managed with conservative treatment. Cold therapy, injection of corticosteroid in the tendon sheath, pulsed ultrasound, and therapeutic massage are all viable options. For traumatic ECU subluxation and dislocations, conservative treatment is the first therapeutic approach. However, if subluxation persists, surgical reconstruction is unavoidable.

26.9 Conclusion

Injuries of the hand and the wrist in basketball athletes are frequent. High clinical suspicion is necessary to diagnose the majority of these conditions. Many injuries are misdiagnosed and treated just as simple sprains. In these cases, diagnosis is often facilitated by specific imaging methods. The treatment of these injuries in basketball athletes is personalized and depends on timing of season, the affected extremity, and the athlete's ability to perform with a splint. There are operative and conservative treatment options available. However, professional basketball athletes are often offered surgical treatment for these injuries because it ensures earlier return to previous state of competition.

References

1. Cantwell JD. The physician who invented basketball. *Am J Cardiol.* 2004;93(8):1075–7. <https://doi.org/10.1016/j.amjcard.2003.12.068>.
2. Frank MG. The encyclopedia of sports. New York: A. S. Barnes.

3. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the National Basketball Association: a 17-year overview. *Sports Health.* 2010;2(4):284–90. <https://doi.org/10.1177/1941738109357303>.
4. Henry JH, Lareau B, Neigt D. The injury rate in professional basketball. *Am J Sports Med.* 1982;10(1):16–8. <https://doi.org/10.1177/036354658201000104>.
5. McCarthy MM, Voos JE, Nguyen JT, Callahan L, Hannafin JA. Injury profile in elite female basketball athletes at the women's national basketball association combine. *Am J Sports Med.* 2013;41(3):645–51. <https://doi.org/10.1177/0363546512474223>.
6. Conn JM, Annest JL, Gilchrist J. Sports and recreation related injury episodes in the US population, 1997–99. *Inj Prev.* 2003;9(2):117–23. <https://doi.org/10.1136/ip.9.2.117>.
7. Andreoli CV, Chiaramonti BC, Buriel E, Pochini ADC, Ejnisman B, Cohen M. Epidemiology of sports injuries in basketball: Integrative systematic review. *BMJ Open Sport Exerc Med.* 2018;4(1) <https://doi.org/10.1136/bmjsem-2018-000468>.
8. Carlson MG. Commentary on scaphoid fractures in basketball. *Hand Clin.* 2012;28(3):281–2. <https://doi.org/10.1016/j.hcl.2012.05.006>.
9. Cassidy C. Should we be repairing all scaphoid fractures in the athlete? *Aspetar, Sport Med J.* 2010;162–64.
10. Benter JP, Mutig K, Hegenscheid K, et al. Diagnostik des Skaphoids [Diagnosis of the scaphoid bone]. *Orthopade.* 2016;45(11):938–44. <https://doi.org/10.1007/s00132-016-3333-y>.
11. Sanders WE. Evaluation of the humpback scaphoid by computed tomography in the longitudinal axial plane of the scaphoid. *J Hand Surg Am.* 1988;13(2):182–7. [https://doi.org/10.1016/S0363-5023\(88\)80045-5](https://doi.org/10.1016/S0363-5023(88)80045-5).
12. Ten Berg P, Drijkoningen T, Strackee S, Buijze G. Classifications of acute scaphoid fractures: a systematic literature review. *J Wrist Surg.* 2016;05(02):152–9. <https://doi.org/10.1055/s-0036-1571280>.
13. Abhinav A, Wildin CJ, Dhukaram V, Dias JJ, Bhowal B. Clinical and radiological outcome of cast immobilisation versus surgical treatment of acute scaphoid fractures at a mean follow-up of 93 months. *J Bone Joint Surg Br.* 2008;90-B(7):899–905. <https://doi.org/10.1302/0301-620x.90b7.20371>.
14. McQueen MM, Gelbke MK, Wakefield A, Will EM, Gaebler C. Percutaneous screw fixation versus conservative treatment for fractures of the waist of the scaphoid. *J Bone Joint Surg Br.* 2008;90-B(1):66–71. <https://doi.org/10.1302/0301-620X.90B1.19767>.
15. Morse KW, Hearn KA, Carlson MG. Return to play after forearm and hand injuries in the national basketball association. *Orthop J Sports Med.* 2017;5(2):1–4. <https://doi.org/10.1177/2325967117690002>.
16. Husband JB. Hook of hamate and pisiform fractures in basketball and hockey players. *Hand Clin.* 2012;28(3):303. <https://doi.org/10.1016/j.hcl.2012.05.012>.

17. Chung KC, Spilson SV. The frequency and epidemiology of hand and forearm fractures in the United States. *J Hand Surg Am.* 2001;26(5):908–15. <https://doi.org/10.1053/jhsu.2001.26322>.
18. Strauch RJ, Rosenwasser MP, Lunt JG. Metacarpal shaft fractures: the effect of shortening on the extensor tendon mechanism. *J Hand Surg Am.* 1998;23(3):519–23. [https://doi.org/10.1016/S0363-5023\(05\)80471-X](https://doi.org/10.1016/S0363-5023(05)80471-X).
19. Dy CJ, Khmel'nitskaya E, Hearn KA, Carlson MG. Opinions regarding the management of hand and wrist injuries in elite athletes. *Orthopedics.* 2013;36(6):815–9. <https://doi.org/10.3928/01477447-20130523-30>.
20. Kovacic J, Bergfeld J. Return to play issues in upper extremity injuries. *Clin J Sport Med.* 2005;15:448–52. <https://doi.org/10.1097/01.jsm.0000188208.00727.0b>.
21. Ko JH, Wiedrich TA. Triangular fibrocartilage complex injuries in the elite athlete. *Hand Clin.* 2012;28(3):307–21. <https://doi.org/10.1016/j.hcl.2012.05.014>.
22. Herold T, Lenhart M, Held P, et al. Indirekte MR-arthrographie des handgelenks bei TFCC-läsionen. *RoFo Fortschritte auf dem Gebiet der Rontgenstrahlen und der Bildgeb Verfahren.* 2001;173(11):1006–11. <https://doi.org/10.1055/s-2001-18318>.
23. Howard TC. The elite athlete: triangular fibrocartilage tears in basketball players. *Hand Clin.* 2012;28(3):325. <https://doi.org/10.1016/j.hcl.2012.05.016>.
24. Rajan PV, Day CS. Scapholunate interosseous ligament anatomy and biomechanics. *J Hand Surg Am.* 2015;40(8):1692–702. <https://doi.org/10.1016/j.jhsa.2015.03.032>.
25. Lee SK, Desai H, Silver B, Dhaliwal G, Paksima N. Comparison of radiographic stress views for scapholunate dynamic instability in a cadaver model. *J Hand Surg Am.* 2011;36(7):1149–57. <https://doi.org/10.1016/j.jhsa.2011.05.009>.
26. William G, Burkett JM. Sports injuries of the hand and wrist. *Sport Med Arthrosc Rev.* 2014;22(1):39–44. http://www.embase.com/search/results?subaction=viewrecord&from=export&id=L27448119;%5Cnhttp://sfx.ub.rug.nl:9003/sfx_local?sid=EMBASE&issn=03063674&id=doi:&atitle=Sports+injuries+of+the+hand+and+wrist&stitle=BR.+J.+SPORTS+MED.&title=British+Journal+of+Spo.
27. Pirolo JM, Le W, Yao J. Effect of electrothermal treatment on nerve tissue within the triangular fibrocartilage complex, scapholunate, and lunotriquetral interosseous ligaments. *Arthroscopy.* 2016;32(5):773–8. <https://doi.org/10.1016/j.arthro.2015.11.050>.
28. Melone CP, Polatsch DB, Flink G, Horak B, Beldner S. Scapholunate interosseous ligament disruption in professional basketball players. Treatment by direct repair and dorsal Ligamentoplasty. *Hand Clin.* 2012;28(3):253–60. <https://doi.org/10.1016/j.hcl.2012.05.002>.
29. Garcia-elias M, Lluch AL, Stanley JK. Three-ligament tenodesis for the treatment of scapholunate dissociation. *J Hand Surg.* 2006;31A(1):125–34.
30. Elsaftawy A, Jablecki J, Jurek T, Domanasiewicz A, Gworys B. New concept of scapholunate dissociation treatment and novel modification of Brunelli procedure—Anatomical study. *BMC Musculoskelet Disord.* 2014;15(1) <https://doi.org/10.1186/1471-2474-15-172>.
31. Morrell NT, Weiss APC. Bone-retinaculum-bone autografts for scapholunate interosseous ligament reconstruction. *Hand Clin.* 2015;31(3):451–6. <https://doi.org/10.1016/j.hcl.2015.04.012>.
32. Ross M, Loveridge J, Cutbush K, Couzens G. Scapholunate ligament reconstruction. *J Wrist Surg.* 2013;2(2):110–5. <https://doi.org/10.2106/jbjs.rvw.o.00060>.
33. Smith DW. ECU tendonitis and subluxation in elite basketball. *Hand Clin.* 2012;28(3):359–60. <https://doi.org/10.1016/j.hcl.2012.05.023>.



Isolated Finger Injuries in Basketball

27

Ufuk Nalbantoglu and Okan Tok

27.1 Introduction

Finger injuries are very common in basketball. Most injuries (>90%) include sprains and volar plate injuries of the proximal interphalangeal (PIP) and metacarpophalangeal joints [1–3]. Direct ball contact with axial load causes dislocations at the PIP joint commonly. Players are also at risk for avulsion of the extensor digitorum when the ball creates an axial load through the fingertip (“mallet finger”) and avulsion of the flexor digitorum profundus when the finger is caught on an opponent’s jersey or on the rim during a slam dunk (“jersey finger”). Boutonnière deformity can result from rupture of the extensor tendon central slip; early recognition of this condition is important. Gamekeepers’ injuries to the thumb usually occur as a result of a fall to the floor or an extension load to the thumb while blocking an opponent.

Hand injuries in basketball, and particularly finger injuries, require knowledge of anatomical structures for accurate examination, diagnosis, and treatment. Most soft tissue injuries of the fingers can be acutely treated by conservative means, but many require definitive care by an

orthopedic or hand surgeon. Missed or misdiagnosed injuries can result in permanent deformity and loss of function. Radiographs are necessary in nearly all cases, and MRI is necessary in some cases to evaluate for fracture or the presence of foreign bodies in cases of fingertip injuries and to evaluate for joint incongruity or fracture in cases of joint or tendon injury.

27.2 Soft Tissue Injuries

Nail and underlying matrix could be damaged by many crush injuries to the fingertips. These kinds of injuries cause hematoma beneath the nail and throbbing pain. Association with tuft fractures of the distal phalanx can also be seen in fingertip injuries because they are typically open fractures and communicate through the nail matrix disruption [4]. A heated paperclip, an 18-gauge needle, or a cautery could be used to create one or multiple holes in the nail for draining the hematoma involving less than 50% of the nail matrix. Soaking the finger in sterile water with peroxide will facilitate drainage. A sterile dressing should then be applied, with a splint or finger bandage in cases involving fracture [4, 5]. Hematoma involving more than 50% of the underlying nail could be a sign for an open fracture. After radiographs, surgical removal of the nail, irrigation, and debridement of the wound, repair of the nail matrix, and replacement of the nail with splinting are recommended [5].

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If nail avulsion is seen without damage to the underlying matrix, the wound should be cleaned and dressed with a non-adherent dressing. If the proximal portion of the nail has also been avulsed from the nail fold and germinal matrix, the patient's cleansed nail or a piece of sterile gauze or foil should be slid under the eponychial fold to prevent adherence [5]. If any part of the sterile or germinal matrix has been torn or lacerated, removal of any remaining nail fragments and repair of the nail bed injury are mandatory [5].

Return to sport in basketball players depends upon whether the player can wear a rigid protective splint and control the ball, and the player's position. Athletes with surgically treated fractures may return to sports between 5 and 10 days with protective taping once soft tissue healing allows.

Simple lacerations may be cleaned and sutured using nonabsorbable monofilament in adults or absorbable suture in children. Grossly contaminated wounds may be cleaned, debrided, and closed. Partial amputations with soft tissue loss measuring less than 1 cm² will heal by secondary intention and may be treated with cleansing and serial dressing changes. Even larger defects will heal well in younger players. Larger wounds involving exposed bone or tendon, nail bed injury, or more proximal amputation should be emergently treated by a hand surgeon [4, 5]. Athletes with surgically treated injuries may return to sports between 5 and 10 days with protective taping once soft tissue healing allows.

27.3 Joint Injuries of the Fingers

Dislocations are usually clinically apparent; they are characterized by pain, limited movement, and digit deformity. If there is any crepitus, bony point tenderness, or open injury, the finger should be radiographed prior to reduction to assess for an associated fracture. Other dislocations can be reduced, splinted, and seen with a post-reduction radiograph. Any irreducible dislocation or dislocation associated with an open wound requires emergent intervention. Local or regional anesthesia may be necessary to obtain adequate pain

relief and relaxation for reduction. Digital blocks are placed by injecting the ulnar and radial web spaces of a digit, anesthetizing the dorsal and volar digital nerves. The local anesthetic should not contain epinephrine, which can cause digital ischemia.

27.4 Proximal and Distal Interphalangeal Joints

Distal interphalangeal (DIP) joint dislocations are uncommon, almost always dorsal, and often are open. These injuries are frequently associated with extensor disruption. If there is no open wound or tendon rupture and closed reduction is possible, extension splinting for 2–3 weeks is recommended. Proximal interphalangeal (PIP) joint injuries are the most common joint injuries in sports, primarily occurring in athletes who participate in contact sports and ball-handling like basketball [6, 7].

27.4.1 Dorsal Dislocations

These injuries are frequently seen in basketball. Dorsal dislocations are most common and caused by hyperextension with axial load. This causes distal volar plate rupture with or without bony avulsion. A true lateral X-ray should be obtained to rule out a fracture [7]. Reduction is usually uncomplicated. Dynamic and static stability should be assessed after reduction, including collateral ligament stability [8]. If there is no associated fracture or ligament injury, splinting in 30° of flexion for 1–2 weeks, followed by buddy taping for sports for 4–6 weeks, is effective. Recurrent dislocation is rare. Swelling and tenderness can persist for several months. Motion exercises as soon as comfort permits can help to prevent stiffness [7].

27.4.2 Volar Dislocations

Volar dislocations of the PIP joint are less common injuries. They are more difficult to reduce

and are associated with more complications than are dorsal dislocations. Compression and rotation with PIP joint flexion cause this type of injury. Pathology usually includes extensor tendon central slip avulsion, volar plate disruption, and collateral ligament tears [7, 8]. If closed reduction is successful, the PIP joint should be splinted in full extension for 3–4 weeks to protect the central slip, allowing the DIP joint to remain free, followed by night splinting for an additional 3–4 weeks. If closed reduction is not possible, open reduction and pinning are necessary [7]. Late development of boutonnière deformity is a potential complication.

27.4.3 PIP Subluxation

Irreducible dislocation of the PIP joint is a typical presentation of this injury. It involves buttonholing of one condyle of the proximal phalanx through a longitudinal rent in the extensor hood between the central slip and lateral band. On lateral radiography, a lateral profile of the proximal phalanx with an oblique profile of the middle phalanx can be seen [8]. Closed reduction buddy taping with full active range of motion is usually sufficient. If closed reduction is not achieved, open reduction is often necessary to disengage the proximal phalanx condyle from the central slip and lateral band.

27.4.4 Collateral Ligament Injuries

Collateral ligament injuries can occur as a result of varus or valgus stress on the joint, which commonly occurs in basketball. Proximal attachment disruption is usually observed. Radial collateral injuries are more common than ulnar side injuries. The index finger is the most commonly involved digit [7]. Typical examination findings are tenderness and ecchymosis. Radiographs should be obtained to confirm or preclude the presence of a fracture. Stability assessment should be done by both examination and radiographs. Most collateral ligament injuries could be treated with buddy

taping, without any restriction in sports. If the tear is associated with significant instability, the digit should be immobilized in a dorsal splint for 3–4 weeks [7].

27.4.5 Metacarpophalangeal Joint

Metacarpophalangeal (MCP) dislocations are relatively rare compared with interphalangeal joints and usually involve dorsal dislocation. Index or small fingers are most commonly affected. Simple dislocations can be treated with immediate closed reduction, and if stability achieved following closed reduction, then buddy taping should be considered and active motion should be allowed immediately. Complex dislocations involve buttonholing of the metacarpal head between the flexor tendon and the lumbrical with volar plate interposition into the dislocated joint. These usually require formal open reduction. After surgical intervention, 3 weeks of activity restriction and controlled activity regaining should be planned.

Fact Box

- Most soft tissue injuries can be treated with conservative methods and cause no out of play period.
- Close follow-up is important for early and better results.
- The goal is motion for the long fingers.
- Some types of injuries that may require surgery must be identified by a specialist.

27.5 Joint Injuries of the Thumb

Thumb interphalangeal (IP) dislocations are unusual injuries and are managed similarly to finger DIP dislocations. Dislocations of the thumb MCP joint are usually dorsal dislocations, resulting from hyperextension at the MCP joint with volar plate rupture. The metacarpal head may slide through the volar plate, where it becomes stuck between the flexor pollicis longus and

flexor pollicis brevis tendons [8]. The volar plate, flexor pollicis longus, or sesamoids may be interposed and prevent reduction, but closed reduction is usually possible, with splinting recommended for 3–4 weeks after reduction.

27.5.1 Skier's Thumb

Also known as gamekeeper's thumb is the acute rupture of the ulnar collateral ligament (UCL) of the thumb MCP joint. Hyper-abduction at the MCP joint causes these injuries. Stener lesions can be seen, in which the torn end of the UCL is displaced superficially to the aponeurosis of the adductor pollicis, preventing healing. Tenderness and swelling over the ulnar side of the thumb MCP joint could be observed with instability of the UCL on radial stress. With the MCP joint in 30° of flexion and at full extension, instability should be evaluated. Stress radiographs can also be used for diagnostic purposes [6, 9–11]. Ultrasound and magnetic resonance imaging (MRI) can directly visualize the torn UCL. Gross instability or a palpable lump suggests a Stener lesion. This lesion may be present in up to 70% of cases [6, 8].

Stable injuries will do well with immobilization for 4 weeks in a thumb spica cast, followed by 2–4 months of protected splinting during athletic competition. Surgical intervention with reconstruction of the UCL is required for any injury with greater than 30–35° of instability in flexion, any instability in extension, a Stener lesion, or large bony avulsion [6, 8–10]. For a safe and fast return to the sport, stability should be regained as soon as possible. A 3-week out-of-play period is needed for a player with torn UCL. Ball contact can be allowed after 3 weeks. Competitive sportive attendance should be delayed for 4–5 weeks according to the player's response to the treatment.

27.5.2 Radial Collateral Ligament Injury

Radial collateral ligament injury could involve proximal or distal portion tears of the ligament. It

is less common than UCL injury but evaluated and treated in a similar manner. Injuries could be associated with volar subluxation of the joint and may require surgical stabilization [7, 8] (Figs. 27.1–27.3).

27.6 Tendon Injuries

27.6.1 Mallet Finger

Mallet finger is also known as drop finger or baseball finger and commonly occurs in basketball players. The extensor mechanism insertion into the distal phalanx is disrupted, resulting from forced flexion of an actively extended DIP joint. A bone fragment may be avulsed with tendon [7, 10, 11]. The patient will have a passively flexed DIP joint with full passive range of motion but inability to actively extend at the DIP joint [4]. Splinting of the DIP joint with mallet finger splints for at least 6 weeks while allowing PIP motion, then up to 4 weeks of nighttime splinting. If there is a large bony fragment avulsed, surgical fixation with reduction and pinning is recommended. Surgical treatment may be necessary for late, chronic cases [7, 10–12]. Players should be out of play for 4–6 weeks.

27.6.2 Boutonnière Deformity

This deformity is caused by avulsion of the extensor mechanism central slip at the middle phalanx and also known as a buttonhole deformity. The head of the phalanx may displace through the defect. The lateral bands then contract, causing late extension deformity at the DIP joint (usually appearing up to 3 weeks later) [7, 10]. This injury usually develops by forced flexion of the middle phalanx while the player is attempting to extend the joint. The examination findings are swelling and pain over the dorsal PIP joint, inability to extend the PIP, and possible hyperextension at the DIP joint [11].

Conservative treatment consists of extension splinting of the PIP for 6 weeks, allowing DIP motion, followed by gradual PIP motion. Chronic cases usually respond to closed treatment as well.

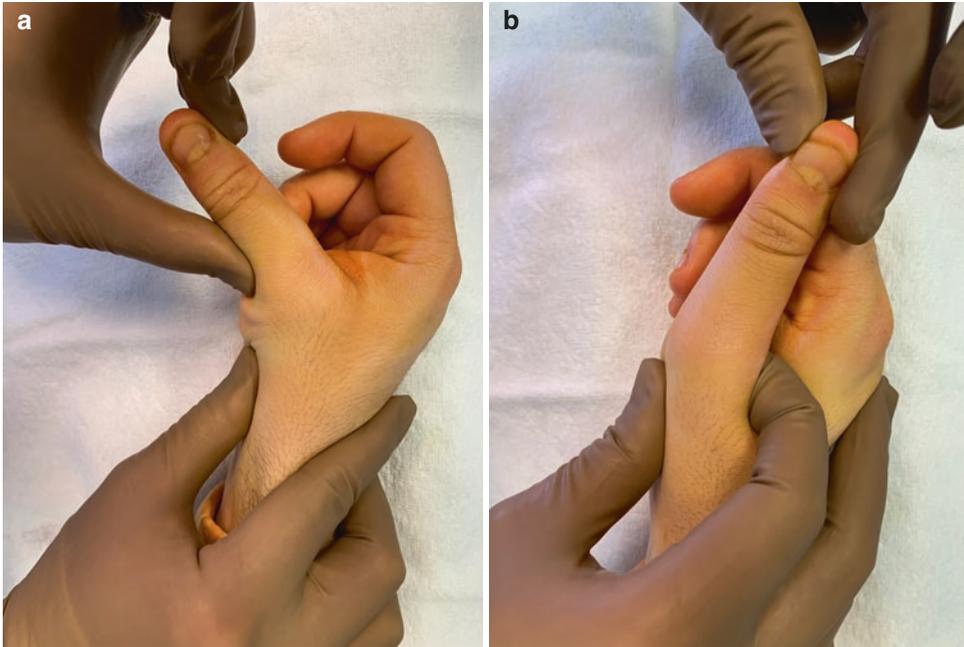


Fig. 27.1 (a) Preoperative image of thumb MCP joint UCL complete injury. (b) Preoperative image of thumb MCP joint RCL complete injury

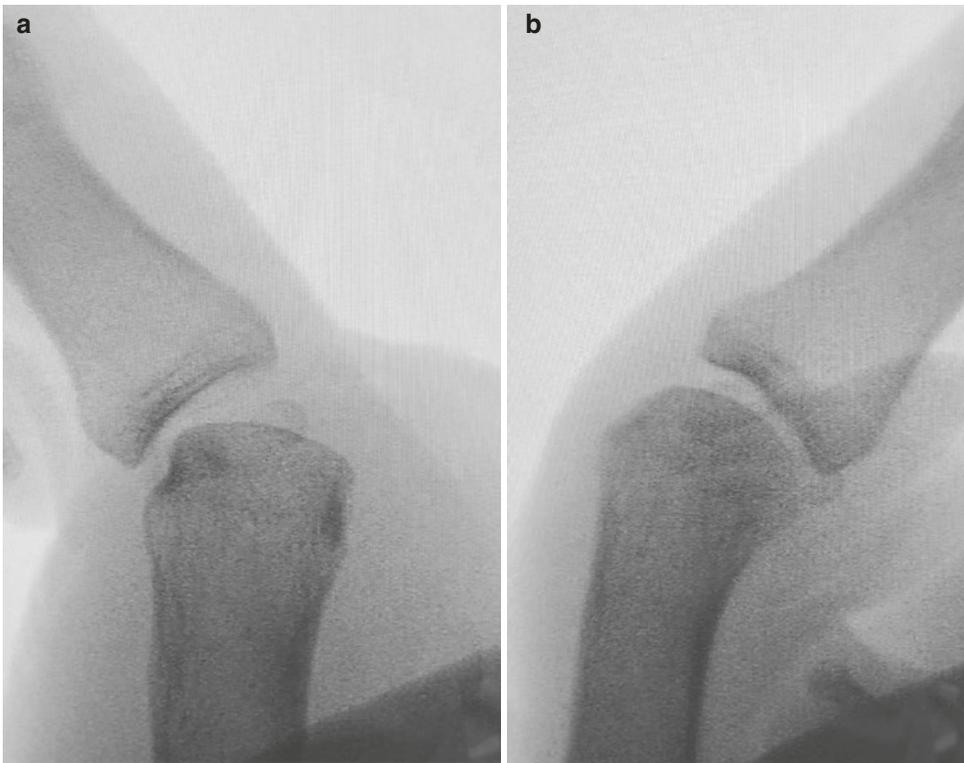


Fig. 27.2 (a) Preoperative fluoroscopy image of thumb MCP joint UCL complete injury. (b) Preoperative fluoroscopy image of thumb MCP joint RCL complete injury



Fig. 27.3 Postoperative fluoroscopy image of thumb MCP joint UCL and RCL reconstruction

Fact Box

- A bilateral clinical examination is important to determine instability and should be preceded by radiography.
- Surgical treatment is a must in complete or unstable injuries.
- Conservative treatment is performed for partial and stable injuries.
- Adequate treatment is essential to prevent chronic instability.

In elite players, early surgical interventions are recommended for safe and fast return to play. Cases associated with large bony fragments should be surgically treated [7, 10, 11]. Out-of-play period can last between 4 and 6 weeks.

27.6.3 Pseudoboutonnière Deformity

Clinically there is a similar appearance to the Boutonnière deformity but a distinctly different mechanism and treatment. Pseudoboutonnière is caused by a hyperextension injury to the DIP joint, disrupting the volar plate and either the radial or ulnar collateral ligament. Tissue contraction causes later development of progressive PIP flexion deformity. Differentiation from a Boutonnière deformity is possible by the absence of tenderness over the PIP central slip and equivalent active and passive ranges of motion at the PIP joint in this injury [8, 10]. Treatment is difficult and often prolonged, with splinting recommended if PIP deformity is less than 45°. Surgical intervention with capsular reconstruction is frequently necessary for major deformities [10]. Out-of-play period can last between 4 and 6 weeks.

27.6.4 Jersey Finger

Jersey finger includes an avulsion of the flexor digitorum profundus (FDP) tendon from its insertion on the volar side of the distal phalanx, usually occurring in basketball players. It most commonly affects the ring finger [7, 10, 13].

Injury is caused by forced DIP extension during maximal FDP contraction, as in grabbing someone's jersey while attempting to tackle. Clinical findings include the inability to actively flex the DIP joint with normal passive range of motion. Radiographs are usually normal if there is no bony fragment avulsed with the tendon [11, 12]. Injuries are treated according to severity, which depends on the degree of tendon retraction. In type I injuries, the tendon slides into the palm. In type II injuries, the tendon slides to the level of the PIP joint. In type III injuries, the tendon slides only to the A4 pulley. These are nearly always associated with a large avulsed bony fragment [10–13]. Surgical treatment is necessary for all kinds of injuries, but the greater the degree of retraction, the more quickly surgical intervention is required. Type I injuries should be addressed within 7–10 days, whereas type III injuries are often successfully treated up to 2–3 months later [7, 10].

Fact Box

- Deciding whether the surgery is needed or not is crucial.
- Early diagnosis, treatment, and close follow-up are necessary for better results.

27.7 Nerve Compression Injuries

Recurrent traumas to the digital nerves cause proliferation of the surrounding fibrous tissue and negatively affect nerve mobility. Players present with paresthesia and dysesthesias in the affected digital nerve dermatome, a positive Tinel's sign over compression area and possible decreased two-point discrimination in the nerve dermatome. Rest, stretching exercises, and anti-inflammatory drugs commonly help relieve the symptoms. Symptoms may take from several days to months to resolve. Surgical decompression or nerve transfer are rarely indicated.

27.8 Phalangeal Fractures

27.8.1 Proximal and Middle Phalanx Fractures

Very common fractures in players participating in contact sports or sports requiring catching of a ball like basketball. Fracture displacement differs according to the mechanism of injury and deforming forces on bone. Proximal phalanx fractures generally have volar angulation with proximal segment flexed by the interosseous muscle contraction and the distal segment extended by pull of the central slip of the extensor mechanism [14, 15]. Deforming forces applied to middle phalanx fractures are generated by both the central slip and the flexor digitorum superficialis tendon resulting in either volar or dorsal angulation depending upon the location of the fracture [14, 15]. Treatment depends upon the stability of the fracture; correction of rotational deformation is crucial. No greater than 10° of angulation in any plane should be allowed.

Nondisplaced and stable fractures can be treated with buddy taping and early range of motion [14]. Careful clinical and radiographic follow-up is required to detect the fracture type and displacement. Even displaced fractures can be treated with closed reduction and immobilization with cast or splint and an outrigger with the affected finger held in the intrinsic-plus position including an adjacent digit to help maintain rotational alignment [14]. In elite players, surgical treatment is recommended for displaced fractures. Fracture immobilization should be limited to 3 weeks prior to initiation of hand-based therapy for regaining the maximal range of motion. Protective splinting should be continued during sport-specific activities until healing and the player's confidence is evident [14, 16, 17]. Intra-articular, unstable, or rotationally misaligned fractures may benefit from open or closed reduction and internal or percutaneous fixation to restore anatomic restoration and rotational control. Rigid fixation should be obtained, and mobilization should start immediately within the first week after surgery to regain range of motion and edema control, permitting an earlier return to sport and a more predictable functional outcome [14, 18]. Protective splinting should be maintained for 4 weeks or until fracture healing and the player's confidence is evident. Simple buddy taping should be continued in sportive activities until range of motion and strength are restored (Figs. 27.4 and 27.5).

27.8.2 Distal Phalanx Fractures

Distal phalangeal fractures comprise 50% of all hand fractures, especially in the thumb and middle finger [19]. Fibrous septa between skin and bone minimize fracture displacement. Evidence of nail bed injury should be evaluated carefully. Treatment is dictated by the presence of soft tissue injury. If the nail bed is injured, the nail bed must be repaired to prevent nail deformity [20]. Immobilization is restricted to the distal interphalangeal joint for a period of 3–4 weeks after which motion is initiated [14]. Persistent tenderness can be seen for greater than 6 months requiring a program of desensitization to allow full



Fig. 27.4 Preoperative fluoroscopy image of 5. Proximal phalanx fracture

function [21]. Loss of continuity of extensor mechanism through bony or tendinous disruption can cause mallet finger deformity. Treatment is almost always conservative with continuous extension splinting of the distal interphalangeal (DIP) joint for at least 6 weeks. After 6 weeks, the removal of the splint several times a day for active range of motion exercises for an additional 2 weeks is needed [15, 16].

27.8.3 Return to Sports after Phalangeal Fractures

Players with stable fractures treated conservatively may return to sports with rigid cast immobilization, thermoplast splint protection, or buddy taping (as specific activities permit) as soon as symptoms allow, often within the first



Fig. 27.5 Postoperative fluoroscopy image of 5. Proximal phalanx fracture

week [22]. Loss of reduction or malrotation must be closely followed-up. Protection should be maintained until radiographic evidence of complete healing is evident and functional recovery of range of motion and strength are complete [16]. Players with surgically treated fractures may return to sports with protective splinting or casting once soft tissue healing is acceptable. Edema control and active motion are typically initiated at 2 weeks and by 4 weeks 75% of motion should have been regained and strengthening can be initiated. Protective splinting should be maintained for sport-specific activities until healing and the player's confidence is evident [14, 22, 23]. Buddy taping should be maintained until strength and motion have been restored.

Fact Box

- Radiography should not be forgotten in finger traumas.
- Rotational deformity must be reduced.
- Closed techniques should be chosen for early recovery.
- Stable fixation is crucial for displaced fractures.

Take Home Message

Finger injuries require a complex and multifactorial process to make the correct diagnosis and to decide when and with which technique the surgery will be performed and when the player is allowed to return to the sport. It is crucial to follow up the players closely and individually and to work with qualified physicians.

References

1. Hamming B, Heewon Y, Bensema B. Epidemiology of basketball injuries among adults presenting to ambulatory care settings in the United States. *Clin J Sports Med.* 2007;17(6):446–51.
2. Messina D, Farney W, DeLee J. The incidence of injury in Texas high school basketball: a prospective study among male and female athletes. *Am J Sports Med.* 1999;27:294–9.
3. Meeuwisse WH, Sellmer R, Hagel BE. Rates and risks of injury during intercollegiate basketball. *Am J Sports Med.* 2003;31:379–85.
4. Idler RS, Manktelow RT, Lucas G, et al. *The hand primary care of common problems.* 2nd ed. New York, NY: Churchill Livingstone; 1990.
5. Fassler PR. Fingertip injuries. Evaluation and treatment. *J Am Acad Orthop Surg.* 1996;4:84.
6. Morgan WJ, Slowman LS. Acute hand and wrist injuries in athletes: evaluation and management. *J Am Acad Orthop Surg.* 2001;9:389.
7. Rettig AC, Coyle MP, Hunt TR. Hand and wrist problems in the athlete. *Am Orthop Soc Sports Med Instr Course 108: AOSSM 28th Annual Meeting, Orlando, FL, 2002.*
8. Kahler DM, McCue FC. Metacarpophalangeal and proximal interphalangeal joint injuries of the hand, including the thumb. *Clin Sports Med.* 1992;11:57.
9. Abrahamson SO, Sollerman C, Lundborg G, et al. Diagnosis of displaced ulnar collateral ligament of the metacarpophalangeal joint of the thumb. *J Hand Surg [Am].* 1990;15:457.
10. Leddy JP. Soft-tissue injuries of the hand in athletes. *Instr Course Lect.* 1998;47:181.
11. Rettig AC. Closed tendon injuries of the hand and wrist in athletes. *Clin Sports Med.* 1992;11:77.
12. Aronowitz ER, Leddy JP. Closed tendon injuries of the hand and wrist in athletes. *Clin Sports Med.* 1998;17:449.
13. Leddy JP. Avulsions of the flexor digitorum profundus. *Hand Clin.* 1985;1:77.
14. Capo JT, Hastings H. Metacarpal and phalangeal fractures in athletes. *Clin Sports Med.* 1998;17:491–511.
15. Henry M. Fractures and dislocations of the hand. In: Bucholz RW, Heckman JD, editors. *Rockwood and Green's fractures in adults.* Philadelphia, PA: Lippincott Williams & Wilkins; 2001. p. 655–748.
16. Posner MA. Hand injuries. In: Nicholas JA, Hershman EB, Posner MA, editors. *The upper extremity in sports medicine.* St Louis, MO: Mosby; 1995. p. 483–569.
17. Strickland JW, Steichen JB, Kleinman WB, et al. Phalangeal fractures, factors influencing digital performance. *Orthop Rev.* 1982;11:39–50.
18. Breen TF. Sport-related injuries of the hand. In: Pappas AM, editor. *Upper extremity injuries in the athlete.* New York, NY: Churchill Livingstone; 1995. p. 451–91.
19. McNealy RW, Lichtenstein ME. Fractures of the bones of the hand. *Am J Surg.* 1940;50:563–70.
20. Simon RR, Wolgin M. Subungual hematoma: association with occult laceration requiring repair. *Am J Emerg Med.* 1987;5:302–4.
21. DaCruz DJ, Slade RJ, Malone W. Fractures of the distal phalanges. *J Hand Surg Br.* 1988;13B:350–2.
22. Alexy C, De Carlo M. Rehabilitation and use of protective devices in hand and wrist injuries. *Clin Sports Med.* 1998;17:635–55.
23. Graham TJ, Mullen DJ. Athletic injuries of the adult hand. In: DeLee JC, Dres Jr D, Miller MD, editors. *DeLee & Drez's orthopaedic sports medicine; principles and practice.* Philadelphia, PA: Saunders; 2003. p. 1381–431.



Hip and Groin Injuries in Basketball

28

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28.1 Introduction

Basketball is a physically demanding, high-speed pivoting sport in which injury risk, especially to the lower limbs, is high, including injuries to and around the hip [1–5]. Basketball players perform repetitive activities that require wide ranges of motion of the hip such as jumping, pivoting, sprinting, and direction changes. These repetitive activities over time may contribute to the development of various injuries in and around the hip. Pathology around the hip is present in both genders and at all ages and levels of play in basketball [1–4]. Hip injuries were found to account for up to 14.6% of all injuries and 4.3% of games missed in National

Basketball Association (NBA) players, with the majority being muscle sprains/strains and contusions [2, 3], and among high school basketball players, hip/thigh injuries were found to represent 8.2% and 8.7% of all injuries for male and female athletes, respectively [1]. While the last two decades have introduced a better understanding of intra-articular hip pathology, the majority of injuries to the hip in basketball are still extra-articular [3, 5]. However, as the understanding and diagnosis of femoro-acetabular impingement syndrome (FAIS) has increased in recent years, it is likely that a number of athletes might have been misdiagnosed as having a “strain” over the years when pain etiology was actually FAIS.

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Limited data exists on the prevalence of surgery due to basketball-related hip injuries. Jackson et al. reported that only 22 of 2852 injuries (0.8%) required surgery over a period of 24 years in the NBA; however, their data excluded offseason surgery and, thus, probably does not provide a true representation of the total burden of hip injuries requiring surgery in basketball.

This chapter addresses the spectrum of the common hip and groin injuries in basketball, diagnosis and management, from soft tissue injuries to extra- and intra-articular impingement, with a focus on FAIS.

28.2 Soft Tissue Hip and Groin Injuries

Despite the increasing focus in recent years on intra-articular hip pathology and FAIS in basketball, the majority of hip and groin injuries in the sport are soft tissue injuries [2, 3]. While at the high-school level, ligament sprains were the most common injury type with 44% [1], the vast majority of soft tissue injuries at the professional level (up to 90%) are strains and contusions, with a higher incidence of hamstrings and adductor strains compared to NFL players in the NBA [2, 3].

Hip and groin pain in athletes have suffered for years from lack of consensus in terms of terminology, definitions, and classification [6]. The Doha agreement, published in 2015 provided uniform definitions for classical groin injuries, including adductor related, iliopsoas related, inguinal related, and pubic related groin pain (Table 1) [7].

Fact Box 1

Hip and groin injuries are common in basketball, accounting for up to 14.6% of all injuries at the professional level. Soft tissue injuries such as muscle strains and contusions represent the most common type of injuries around the hip, with higher incidence of adductor and hamstrings strains at the professional level.

28.2.1 Risk Factors for Groin Injuries

There is very limited data on how acute groin injuries occur in basketball; however, sudden change of direction and lateral acceleration-deceleration movements where the muscles are stretched during forceful contraction have been shown to be common causes in other sports and are common movement patterns in basketball [6].

A systematic review focusing on level 1 and 2 studies highlighted a number of factors associated with increased risk of groin injury in athletes. The most common factor found was previous groin injury, while higher level of play, decreased hip adduction strength (both by itself and relative to abduction), and lower levels of sport specific training were also recognized [8]. Another systematic review and meta-analysis investigating cross-sectional factors differentiating athletes with and without hip and groin pain revealed pain, lower strength on adductor squeeze test, reduced hip internal rotation, and bent knee fall out were frequent findings in athletes with hip and groin pain [9].

In a 24-year epidemiologic overview of hip injuries in the NBA, Jackson et al. highlighted that the majority of hip soft tissue strains occur during the preseason and the first month of the season, with the majority of strains involving the hamstrings and adductors (Fig. 28.1) [3]. This could be explained by the acute surge in loads during this period in comparison to the preceding off-season period [10].

28.2.2 Adductor Muscle Strains

Musculotendinous injuries around the groin are by far the most common type of injuries related to the hip and groin in athletes. For both acute and long-standing groin pain, adductor-related injuries are the most frequent, accounting for up to 64% of all hip and groin injuries [11]. The hip adductor muscles, consisting of six muscles—the adductor longus (AL), magnus, and brevis, gracilis, obturator externus, and pectineus—originate on the pubis and attach on the medial aspect of the femur. The occurrence of adductor injuries

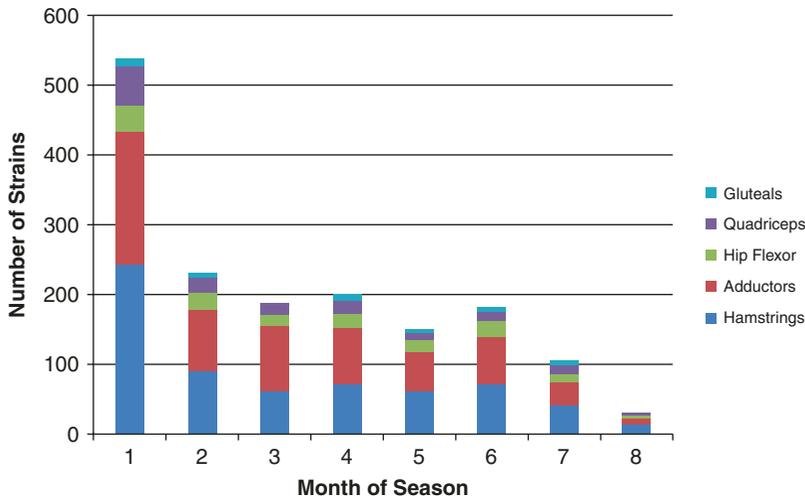


Fig. 28.1 Frequency of hip soft tissue strains in the NBA by month of the season over 24 seasons in the NBA. Month 1 represents the preseason. From: Jackson TJ, Starkey C,

McElhiney D, Domb BG. Epidemiology of Hip Injuries in the National Basketball Association: A 24-Year Overview. *Orthop J Sports Med.* 2013;1(3):2325967113499130

can be acute, with a clear associated mechanism of injury, or chronic, with gradual symptom development. Injuries to the adductor muscles most commonly occur during eccentric contraction, when the muscle contracts while being lengthened or during passive stretch. The adductor longus is most commonly injured during sport activity [12]. It is not surprising that adductor injuries are not uncommon in basketball as sudden direction changes are common in the game both in offense and defense situations, constantly contesting the adductor-abductor muscle groups during play [3].

Mismatch in the abductor and adductor muscle group strength has been identified as a risk factor for adductor strains in athletes [13]. Tyler et al. found that adduction strength was found to be just 78% of abduction strength in those players who sustained an adductor injury and that players were 17 times more likely to sustain an adductor muscle strain if their adductor strength was less than 80% of their abductor strength [13]. Furthermore, they found that preseason hip adduction strength was 18% lower in players who went on to sustain an adductor muscle strain compared to uninjured players.

Clinical signs of adductor-related groin pain include tenderness at the adductor longus and/or

gracilis origin at the inferior ramus pubis as well as groin pain at the same site with palpation or resisted adduction [11]. Other common signs include decreased adductor muscle strength and groin pain on full passive abduction [9]. Magnetic resonance imaging (MRI) and ultrasound are the preferred modalities for evaluating and assessing the location and severity of adductor strains. MRI findings may include avulsion of the adductor muscle from the pubic ramus, edema, or hemorrhage.

Treatment of adductor strains is primarily conservative and depends on the onset (acute or chronic). In the acute setting, management consists of rest, protected weight bearing, and initiation of a strengthening and rehabilitation program. In the chronic setting, muscle weakness and imbalance may be more pronounced and may require a more gradual strengthening strategy. While most protocols begin with passive modalities such as stretching and local treatment, there is evidence that attention to active strengthening consisting of progressive resistive abduction/adduction, core strengthening, balance training, and sports-specific movements can be more effective in treating chronic adductor strains [3]. In the event an athlete fails conservative treatment, surgical management is an appropriate option, particularly in the chronic setting, how-

ever may also be an option in selected acute cases involving fibro-cartilaginous avulsion. Surgical treatment consists of adductor tendon debridement/release [14, 15] and/or repair [16]. If chronic changes are present at the insertion site on the pubis, debridement of the insertion site may be warranted.

Return-to-play following adductor injuries has been previously evaluated in collegiate and professional basketball players. In an epidemiologic study of 621 hip adductor strains among National Collegiate Athletic Association (NCAA) athletes between the 2009–2010 and 2014–2015 academic years, [17] found that among male collegiate basketball players with adductor injuries, 22.9% were restricted from practice or gameplay for 1–6 days, 8.6% for 7–21 days, and 5.7% for >21 days [17]. A similar study of 967 NBA players sustaining hip injuries between the 1988–1989 and 2011–2012 seasons found that an injury to the adductor group resulted in 6.45 ± 7.4 missed practices and/or games [3]. Interestingly, suggesting adductor strains may be more debilitating at the professional level, which could be explained by a more stringent return-to-play protocol in the NBA.

The majority of athletes with adductor-related injuries return to sport within 4–6 weeks. There is, however, evidence that if an elite athlete sustains a groin re-injury, the recovery period for the re-injury is almost twice as long compared to the index injury, emphasizing the importance of managing the injury properly the first time around [18].

Fact Box 2

Adductor injuries represent the most common site of soft tissue injuries around the hip.

The majority of athletes with adductor-related injuries return to sport within 4–6 weeks. It is important to manage these injuries properly and achieve full recovery as the recovery period for a re-injury, if one occurs, is almost twice as long compared to the index injury.

28.2.3 Hip Flexor Muscle Injury/Iliopsoas Tendinitis

Hip flexor muscle injuries and iliopsoas tendinitis can be a common source of injury and irritation in professional basketball players. The iliopsoas muscle is involved in spinopelvic control and hip flexion and is frequently recruited in basketball players leading to increased risk of injuries. Jackson et al. reported an overall incidence of 13.2% for hip flexor muscle or Iliopsoas injury, resulting in 7.7 ± 11.3 days out of sports in a cohort of 967 NBA players over 24 years [3].

Athletes with iliopsoas-related pain may complain of groin pain with hip flexion, tenderness palpating the muscle through the lower abdominal wall and/or just distal to the inguinal ligament medial to the sartorius, and pain on passive stretching during the Thomas test [11]. The iliopsoas may be tight as well as weak and sore when tested isometrically with 90° of hip flexion and may also present as an internal snapping sensation from rubbing over the iliopectineal eminence (“internal snapping hip”). Hip flexor tendinopathy diagnosis is largely clinical; however, ultrasound and MRI may assist in the diagnosis with findings of iliopsoas tissue disruption, edema, neo-vascularization or calcified tissue in the iliopsoas and any findings suggestive of muscle-tendon complex disruption. An ultrasound-guided lidocaine injection into the psoas tendon sheath can aid for diagnostic purposes. Application of steroid should be used with caution.

Conservative measures are the mainstay of treatment for iliopsoas tendonitis, consisting of NSAIDs, physical therapy, rest, massage, and activity modification. These treatments yield symptomatic relief in the majority of athletes. Rehabilitation programs should focus on core musculature and hip flexor strengthening with gradual progression according to clinical milestones. If insufficient symptomatic relief is achieved, ultrasound-guided corticosteroid injection can be considered as a final treatment step [19], however should be used with caution, while other injection options such as platelet-rich plasma (PRP) have gained more popularity in recent years and are considered a safer option.

Although arthroscopic tendon lengthening or release has been shown to have success in athletes [20], this modality should be applied sparingly as there is risk for altering spinopelvic dynamics and sacrificing a secondary stabilizer of the hip, thereby potentially contributing to micro-instability.

28.2.4 Sports Hernia/Athletic Pubalgia/Core Muscle Injury

Although adductor and hip flexor injuries are common soft tissue injuries in professional athletes, lower abdominal musculature and inguinal region dysfunction can be an important cause for pelvic disability. These injuries, often referred to as “sports hernia” or “athletic pubalgia”, have many names including “Gilmore’s groin” and “osteitis pubis,” and more recently, the term “core muscle injury” (CMI) has been coined. While less common than adductor and hip flexor injuries, the extent of this pathology has yet to be described in basketball players. In elite male football, almost 50% of players suffering from inguinal-related groin injury miss more than 4 weeks of training and match play, and injury time is almost double that of adductor injuries [18, 21]. The pathology is thought to involve an injury to the abdominal wall at the fascial attachments of the rectus and adductors onto the pubic symphysis. Some authors have described CMI as an actual tear in the part of the transversalis fascia that forms a portion of the posterior inguinal wall, thereby leading to an incipient posteriorly protruding hernia [22]. CMI is thought to occur from abdominal hyperextension and hip abduction [23], motions such as in abrupt direction changes which are common in basketball. Other reported mechanisms include acute trauma or repetitive microtrauma from overuse.

Presentation often includes proximal adductor pain as well as inguinal canal pain near the rectus abdominis muscle insertion on the pubis [23, 24]. Symptoms typically worsen with activity and resolve with rest. Additionally, these athletes may experience pain with coughing or radiation of pain into the groin and testicular regions, indicating entrapment of surrounding nerves [23].

Symptoms are commonly unilateral, although bilateral symptoms have been reported in up to 43% of athletes [24].

On physical examination, patients may have tenderness on palpation of the pubic tubercle and pubic symphysis. The abdominal obliques and conjoined tendon/rectus abdominis should also be palpated to help differentiate CMI from other etiologies of lower abdominal pain. Resisted hip adduction and palpation of the inferolateral edge of the distal rectus abdominis with resisted sit-up may recreate the patient’s symptom.

Imaging should include radiographs as a first line, with AP pelvis and lateral views of the femur (also to investigate for other causes of groin pain), and may show signs of osteitis pubis, thereby clueing towards CMI. As a next step, MRI can be useful in detecting both rectus abdominis and adductor aponeurosis pathology [25] as well as signs of osteitis pubis and may demonstrate tearing/detachment of other structures from the pubis. Additionally, MRI can rule out concomitant ipsilateral intra-articular hip pathology. Dynamic ultrasound examination may detect weakness of the abdominal wall during maneuvers that increase intra-abdominal pressure (i.e., Valsalva).

First-line management for CMI is conservative with physical therapy focusing on core stabilization, postural retraining, and re-stabilizing the hip and pelvis muscle balance. Gradual RTS can be attempted after a period of rest and activity modification. It is important to note though that CMI can be a very troublesome condition, which takes a long time to recover from, and may not resolve by conservative treatment. Surgical treatment may be considered following conservative treatment failure. A number of surgical techniques have been described, including open or laparoscopic use of a mesh to support the area of weakness and excision/ablation of various nerves. The advantage of the laparoscopic technique is that it allows addressing both sides through the same incisions (whether both sides are symptomatic or as a prophylactic measure for the non-symptomatic side). Overall, surgical treatment has been shown to be successful in treating CMI

in high-level athletes. To our knowledge, the results of surgical treatment for CMI has yet to be described in professional basketball players; however, in a cohort of 22 professional hockey players undergoing external oblique aponeurosis repair and excision/ablation of surrounding neurovascular bundles, Irshad et al. reported 86% of players were able to return to professional hockey [26].

28.3 The Hypermobile Athlete

The hypermobile athlete is a unique population that is predisposed to a specific subset of hip disorders. While more common in dancers or gymnasts that require extreme ROM to perform their athletic tasks, thus placing them at risk for potential hip pathology, it is still important to rule out the condition in basketball players as well. Excessive hip ROM can cause the labrum to become pinched between the femoral head and acetabulum, leading to labral pathology and symptoms consistent with hip impingement. Completing a simple Beighton hypermobility score can aid in the diagnosis, with scores from 5–6/9 and above suggesting a degree of joint laxity and hypermobility [27]. Additionally, high proportions of hypermobile athletes can present with borderline hip dysplasia that will contribute to hip pathology and presentation.

28.4 Intra-Articular Hip Injuries/ Pathologies

Intra-articular hip injuries are the most common source of groin pain in athletes not related to the musculotendinous structures in the groin area. In recent years, intra-articular hip injuries have received increased recognition as an important differential diagnosis in athletes with groin pain with an increasing incidence reported [21]. In elite football, intra-articular hip injuries were found to account for up to 10% of all hip and groin injuries [18]. The most common diagnosis of intra-articular hip pain is femoro-

acetabular impingement syndrome (FAIS) representing symptomatic abnormal premature contact between the proximal femur and the acetabulum [28].

28.4.1 Femoro-Acetabular Impingement Syndrome (FAIS)

Recently, a consensus statement, also referred to as the “Warwick agreement,” defined uniformly accepted definitions and terminology regarding FAIS [28]. It defined FAIS as a motion-related hip disorder consisting of a triad of symptoms, clinical signs, and imaging findings [28]. The pathology is particularly prevalent in athletes who participate in high-impact sports such as basketball which involve constant pivoting motions [3, 29]. FAIS is comprised of two common bony pathomorphologies. Femoral-sided pathomorphologies are termed CAM deformities and acetabular-sided pathomorphologies are termed Pincer deformities. These abnormalities can be present each in isolation or in combination (referred to as “mixed-type FAIS”), as well as associated labral, cartilage, and soft tissue pathology (Fig. 28.2).

CAM patho-morphology refers to a flattening or convexity at the femoral head neck junction, while Pincer patho-morphology refers to either global or focal overcoverage of the femoral head by the acetabulum [30]. Their presence, however, in the absence of appropriate symptoms and clinical signs, does not constitute a diagnosis of FAIS as a substantial proportion in the general population are thought to have CAM or Pincer morphology [31, 32].

28.4.2 Labral Tears

The labrum is a horseshoe-shaped fibrocartilaginous tissue which attaches to the rim of the acetabulum. At its apex, however, the labrum is triangular and functions to maintain hip stability by increasing the depth of the socket, increasing

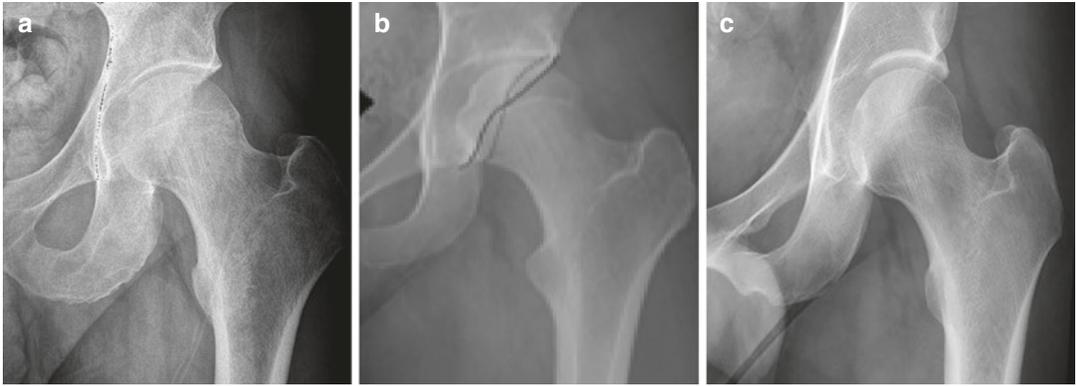


Fig. 28.2 CAM type, Pincer type, and combined type deformities. Anterior-posterior X-ray imaging of the left hip demonstrating isolated CAM-type deformity of the

femoral head (a), isolated Pincer-type deformity of the acetabulum with acetabular retroversion and over-coverage (b), and a combined CAM and Pincer deformity (c)

the surface area of the hip and providing a suction seal. As the labrum ends inferiorly at the anterior and posterior edges of the acetabular fossa, it becomes continuous with the transverse ligament which spans the base of the socket. Similar to the meniscus of the knee the acetabular labrum is vascularized from the periphery and contains nerve fibers which can produce pain upon injury to the structure.

In FAIS a variety of abnormalities of the femur and/or acetabulum combined with rigorous and/or terminal hip motion can produce repetitive collisions that can damage the soft tissue structures (labrum/cartilage) around the acetabulum. CAM-type impingement occurs due to the loss of concavity of the femoral neck junction anterolaterally. This prevents this portion of the femoral head from sliding smoothly under the acetabulum during hip range of motion. As a consequence, the femoral head with its abnormal morphology continuously abuts the acetabulum causing lifting of the labrum due to shear stresses and which may disrupt the chondrolabral junction. Alternatively, traumatic injuries of the labrum also exist. These injuries occur when superphysiologic flexion of the hip causes direct abutment of the femoral neck on the acetabulum. This can cause direct damage to the labrum and acetabular cartilage while creating indirect posterior inferior cartilage damage by leverage [33].

While Labral tears typically occur antero-superiorly in association with FAI or dysplasia, less commonly, labral pathology may occur in isolation in a less common direct anterior location, adjacent to the iliopsoas tendon in the absence of bony abnormalities, and are likely the result of iliopsoas impingement [34–36].

28.4.3 Acetabular Dysplasia

Acetabular dysplasia is a developmental disorder reported to be present in 4.3% of males and 3.6% of females [37]. Dysplasia can lead to hip pain in the young adult and has been recognized as a potential precursor for the development of early hip arthrosis if not recognized and treated early. Hip dysplasia is the result of abnormal hip joint development in early infancy and childhood resulting in abnormal morphology of the acetabulum, femoral head, or both [38]. More commonly there is undercoverage of the femoral head anteriorly or laterally resulting in increased focal contact pressures in the posterosuperior acetabular rim. The labrum is often hypertrophic in dysplastic hip as a result of a physiologic attempt to compensate for the lack of bony coverage. The increased contact pressures can lead to labral and cartilage damage, while decreased contact pressures in the undercovered area can lead to premature cartilage and labral degeneration [39, 40]. Decreased femoral head coverage can lead to

potential instability with increased femoral head translation in the area of acetabular deficiency. Recurrent subluxation/dislocation events can additionally lead to degeneration and cartilage injury, ultimately leading to an increased rate of early arthritis if not treated early.

28.4.4 Pathogenesis of FAIS

Athletes involved in pivoting sports such as basketball are at increased risk for the development of hip pathology [29, 41]. Particularly high-impact activities that involve flexion and rotation of the hip expose the joint to abnormal joint forces. This abnormal mechanical stresses may present in a variety of hip pathologies including FAIS, labral tears, and cartilage injury. High-intensity sport activity causing physal stress surrounding adolescence has also been suggested as a risk factor for the development of the CAM morphology, with observations showing higher FAIS incidence in athletes than in age-matched individuals not participating in high-level sports [42, 43]. Alternatively, FAIS may develop secondary to residual deformity from the sequelae of childhood hip disorders, such as Legg–Calvé–Perthes disease, slipped capital femoral epiphysis (SCFE), and hip dysplasia. When athletes place greater demand on the hip than its natural ROM, compensatory stresses and subsequent pain may develop in the lumbar spine, pubic symphysis, sacroiliac joints, and posterior acetabulum. These compensatory stresses can also be transferred to the peri-articular musculature and pubis, which may lead to muscle injuries of the hip flexors, proximal hamstrings, adductors, iliopsoas and pubic pain.

28.4.5 Peri-Articular and Extra-Articular Hip Impingement

Although hip impingement is classically described as a mechanical condition due to abnormal contact of the femur (CAM) and the acetabulum (PINCER), other forms of impingement have been recently described. **Anterior inferior iliac spine (AIIS) or subspine**

impingement occurs when a prominent AIIS or subspine region contacts the inferior/medial femoral neck when the hip is flexed beyond 90° [44]. This form of impingement, referred by some as extra- or peri-articular impingement, may be related to the acetabulum structure (due to acetabular retroversion) or may arise from bony changes related to prior stresses to the AIIS or prior rectus femoris AIIS avulsion injuries which may heal with a prominent AIIS or subspine region causing impingement [45–48]. This injury, caused by avulsion of the anterior or straight head of the rectus femoris, primarily occurs between the ages of 13 and 23 years when the ratio of muscle to physal strength is the greatest [46, 47].

Ischiofemoral impingement is another form of extra-articular impingement where pain is often associated with hip extension, adduction, and external rotation and commonly presents as lower buttock and inner thigh pain and pain radiating toward the knee [49, 50]. Snapping and clicking are often reported. The pathology stems from reduced distance between the ischial tuberosity and lesser trochanter and injury to the quadratus femoris muscle can be evident on MRI and is the result of repetitive impingement of the muscle between these structures. Ischiofemoral impingement is thought to arise from prior injuries at the ischial tuberosity from proximal hamstring injuries, coxa valga, increased femoral neck anteversion, and previous proximal femoral fractures as well as the result of abductor insufficiency leading to increased hip adduction, and should be conservatively managed initially.

28.4.6 Diagnosis

The clinical examination of the hip in basketball players is a comprehensive assessment that includes a detailed history, a structured physical examination, and clinical tests as well as simple and advanced imaging.

28.4.6.1 History

Obtaining a thorough history is key and will help better characterize the injury and symptoms in

context. Understanding whether symptoms are the result of an acute or overuse injury, direct or indirect trauma as well as characterizing the pain (location/constant/intermittent/mechanical) can provide important input as well as what provokes or alleviates the pain; whether there is a radiating pain element; identifying activity limitations both in activities of daily living (ADL), work, and sport; and also previous treatment/s and response to such treatment/s.

When history does not provide a clear direction, a systematic approach may be warranted. It is important to remember that even when dealing with an otherwise healthy and often young athletic population, more serious conditions (e.g., infection, malignancy, or systemic disease) are possibilities and should always be considered as the hip and groin region is a common sight of referred pain. It is therefore important to ask about weight loss, fatigue, fever, chills, or a history of recent infection.

In an acute injury setup, a precise description of the injury mechanism can be useful. Characterizing the mechanism (i.e., contact or non-contact related), energy and forces involved, the exact movement and action which generated the injury, as well as whether the player could resume activity soon after the injury and the pain pattern following, may be relevant for an accurate diagnosis. Additional focus should be directed at symptoms correlating with the timing of the injury or with current symptoms, such as an accompanying sound or sensation (i.e., snap, click, or pop) or an instability sensation.

In the non-traumatic/non-acute setting, it is important to investigate into the activities undertaken by the player in the period preceding the injury as well as a description of symptoms development, previous similar symptoms, change in the activity load (intensity, duration, frequency); change of surface, technique or equipment; and if symptoms development correlated to such changes.

A history of systemic, urogenital, abdominal, or low-back symptoms should be taken as well. Childhood hip disorders such as Legg–Calvé–Perthes disease, SCFE, developmental dysplasia of the hip (DDH), and septic arthritis

are important to be aware of. Assessing for alcohol or steroid use as well as sickle cell disease is important when avascular necrosis is suspected. Osteoarthritis (OA) can be present in basketball players and is likely to target players toward the end of their career although not only. OA will more likely present with increasing stiffness and a more global pattern of pain localization as opposed to a focal pattern in FAIS and labral tears.

In FAIS, patients often describe a sharp deep groin pain during hip flexion, internal rotation, or adduction movements. Painful clicking is not infrequently reported and may suggest involvement of the hip labrum or an internal snapping hip [51].

Overall, a thorough history taking can help focus the next steps of the diagnostic process, including the clinical examination and requested imaging studies, therefore aiding in a shorter and more effective process.

Fact Box 3

Femoroacetabular impingement syndrome (FAIS) is a motion-related hip disorder consisting of a triad of symptoms, clinical signs, and imaging findings. The actual prevalence of intra-articular hip disorders may have been underestimated over the years. The prevalence of FAIS in basketball has grown with the evolution in understanding and diagnosis of the condition in recent years.

28.4.6.2 Physical Examination

Gait assessment is a key clinical tool for detecting hip pathology or abnormalities in the kinematic chain. Key elements of gait evaluation include foot progression angle (FPA), pelvic rotation, stance phase, and stride length [52]. Abnormal gait patterns associated with hip pathologies include winking gait with excessive pelvic rotation in the axial plane, abductor deficient gait (Trendelenburg gait or abductor lurch), antalgic gait with a shortened stance

phase on the painful side, and short leg gait with dropping of the shoulder in the direction of the short leg. Excessive rotation on FPA could raise suspicion of femoral or acetabular ante/retroversion. Abductor weakness can be identified using the single leg stance test (Trendelenburg test), in which a positive test is determined by the observation of a pelvic drop toward the nonbearing side or shift of more than 2 cm toward the bearing (affected) side. Functional tasks such as squatting (two and single leg), single leg dip and a “step down test” may assist in detecting functional weakness and compensatory strategies. Performing deep squats is often difficult in patients with FAIS as well as sudden stopping/starting and cutting movements. It is important to assess functional tasks in both the frontal and sagittal plane in athletes as pathologic patterns may not always present in simple functional tasks and activities of daily living, as symptoms are mainly related to athletic performance in this population. Therefore, more strenuous activities such as running and jumping may need to be investigated for any unloading or compensating strategies and which may provoke or accentuate their symptoms.

The *seated examination* consists of range of motion in the seated position provides a repro-

ducible and reliable way to assess bilateral internal and external rotation with the ischium square to the examination table. Players with increased femoral anteversion will display increased internal rotation and decreased external rotation, while players with increased femoral retroversion or acetabular retroversion will have decreased internal rotation and increased external rotation (Fig. 28.3).

Supine examination begins with inspection of both legs for any abnormalities or leg length discrepancy. A thorough vascular, lymphatic, and neurologic examination should be performed. Lower back and spinal cord-related symptoms that could present as hip pain should be ruled out. Palpation is performed in key clinical areas that can contribute to hip pain (groin, abdomen, pubic symphysis, adductor tubercle, and greater trochanter). The palpation of the adductor insertion is done with the hip flexed, abducted, and externally rotated, and the knee slightly flexed. Pain on palpation suggests adductor-related groin pain [11]. When adductor-related groin pain is suspected, an adductor squeeze test can be performed (Fig. 28.4).

Hip ROM is recorded for passive flexion, adduction, abduction, and internal and external rotation at 90° of hip flexion. In FAIS, patients often have decreased hip range of motion and hip



Fig. 28.3 Hip range of motion. Internal and external rotation of the hip is checked with the patient supine and the hip flexed to 90°. It can also be checked and compared in the seated and supine positions



Fig. 28.4 The Adductor Squeeze test. There are several adductor squeeze tests however the most sensitive is performed with the patient in the supine position. The examiner stands at the end of the examination table/bed with hands and lower arms between the patient's feet holding them apart. The patient is asked to press the feet together

with maximal force with the feet point straight up (a). A positive test accounts for pain reproduced from the adductor longus insertion site where the patient also was tender at palpation [11, 55]. Another common version of the test is performed with the hips flexed to 45° (b)

muscle strength [54], with flexion and internal rotation mostly affected. The Thomas tests helps assess for hip flexion contractures which can contribute to hip pathology. The hip flexion contracture test or Thomas test is performed by instructing the patient to hold one knee to their chest and passively extend the other knee to the exam table. The inability of the limb to reach the examination table indicates contracture of the hip flexors; however, patients with hyperlaxity or hyper lordosis of the lumbar spine may have a false negative. Assessment of hip flexors or psoas strength is performed using the resisted straight leg raise or Stinchfield test (resisted hip flexion from 10–45° with the knee extended). The psoas may place pressure on the labrum during the maneuver which can cause pain in players with intra-articular pathology or psoas impingement/tendinitis.

The diagnostic process of FAIS remains a challenge due to the relative low diagnostic accuracy of specific clinical tests [56, 57]. The *FADDIR* (Flexion Adduction Internal Rotation) test or *Anterior Impingement Test* is commonly abnormal in athletes with FAIS (Fig. 28.5). However, although very sensitive (high sensitivity), it should be noted that this test is not very specific (low specificity), and thus positive in most patients with an intra-articular



Fig. 28.5 The FADDIR or anterior impingement test is performed in the supine position with flexion to 90°, adduction and internal rotation of the hip, bringing the anterior femoral neck in contact with the anterior rim of the acetabulum, trying to reproduce the patients' symptoms thus indicating a positive test

problem [56]. Therefore, a positive test is not diagnostic on its own for an intra-articular hip joint problem however if negative an intra-articular hip joint problem is not likely [56]. Other tests with less sensitivity and specificity include the FABER (Flexion, Abduction External Rotation) test, also known as Patrick's test [56, 58] (Fig. 28.6) and the squat test [59]. Subspine impingement is tested with straight hip flexion past 90° and is positive with reproduction of the characteristic pain.



Fig. 28.6 (a, b) The FABER (Patrick's) test stands for flexion, abduction, and external rotation. It is considered positive for intra-articular hip pain if it reproduces the patients' typical groin symptoms [58]

28.4.6.3 Imaging

Radiographic abnormalities are not uncommon in basketball [43].

Baseline imaging for a player with hip and groin pain should consist of X-rays. Standard AP pelvis radiographs, preferably with the patient standing [60] with neutral pelvic tilt and leg internal rotation of 15° and a true lateral view are in most cases useful to determine the presence of CAM and/or pincer morphology [61]. Other useful views include the axial cross table view, the modified Dunn view, and the false-profile view and may be preferred by some clinicians in addition to the AP pelvis view and are helpful in identifying femoral head and acetabular abnormalities. CAM morphology is often defined as an alpha angle $>55^\circ$ [28] which is a measure of head-neck sphericity measured in the Dunn view, although in earlier studies it has been

defined as $>50^\circ$. Pincer morphology is often defined as a lateral center edge angle $>39^\circ$ [28] (Fig. 28.7).

However, clinicians should be cautious when interpreting findings of CAM and/or pincer morphology, as their prevalence has been shown to be high in athletes regardless of symptoms [63] and in athletes with adductor-related groin pain [7]. Standard radiographs are also useful to assess for other potential causes of hip and groin pain, such as hip dysplasia, femoral neck stress fractures, or osteoarthritis [28] as well as osseous avulsions femoral neck growth plate injuries in skeletally immature adolescent players when suspected.

Hip dysplasia is defined as a lateral center edge angle $<20^\circ$ while borderline hip dysplasia between 20° and 25° , and these conditions are of relevance

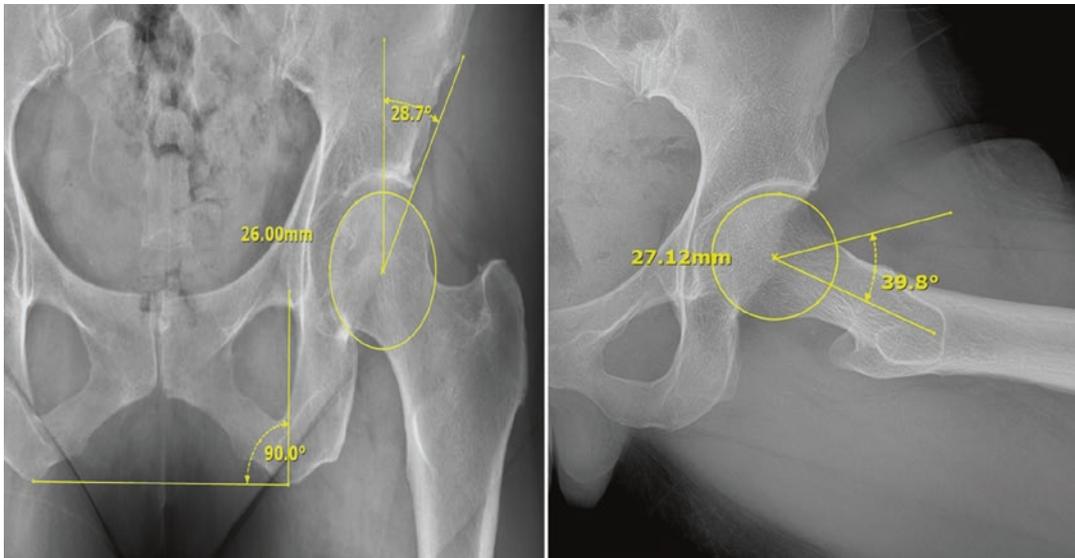


Fig. 28.7 Alpha and lateral center edge angle. The alpha angle is measured in the Dunn view as the angle between (1) a line from the center of the femoral neck to the center of the femoral head and (2) a line from the center of the femoral head to the point where the femoral head–neck

junction extends beyond the margin of the circle [62]. The center edge angle is measured as the angle between (1) a vertical line through the femoral head center and (2) a line between the femoral head center and the lateral edge of the acetabulum [62]

as they may lead to labral and/or acetabular cartilage damage [64]. The acetabular index or Tonniss angle is another measure of hip dysplasia and an angle greater than $10\text{--}14^\circ$ on AP pelvis X-ray also is diagnostic of hip dysplasia (Fig. 28.8).

Ultrasound is useful to confirm/exclude extra-articular soft tissue pathologies in the groin area such as adductor-related injuries/pathologies as well as a means to accurately deliver diagnostic injections. Its limitations stem from being a performer dependent modality.

Advanced imaging modalities may be required for documentation of soft tissue pathology and when indicated, for surgical planning. A 3.0 T MRI is considered the preferred imaging modality for identifying acetabular labral tears and chondral lesions in the hip [28]. However, when a high-resolution MRI is not available, an MRI with intra-articular contrast (MR arthrogram/MRA) can aid in the detection of labral tears and chondral defects or delamination. Caution should however be taken in the clinical interpretation of positive findings as acetabular labral tears may be asymptomatic [32].

MRI can also be useful in assessing the symphysis pubis area for osteolytic changes and sclerosis, with pubic bone marrow edema on MRI adjacent to the symphysis joint being diagnostic and may indicate the level of reactivity of this area.

An ultrasound or fluoroscopy guided intra-articular diagnostic injection is an important aid in the examination of athletes with potential intra-articular hip injuries [28]. Pain relief following such an injection has been suggested to support the diagnosis of FAIS [65].

While radiographs, ultrasound, and MRI have all been used to assess abnormal hip pathology, computed tomography (CT) remains the gold standard in assessment of bony morphology and pathology. CT is particularly useful in evaluating acetabular dysplasia, complex FAI, malalignment syndromes, traumatic hip instability, and sub-spine impingement. CT imaging also enables a full rotational/version profile assessment, and the use of 3D reconstructions on CT allows an accurate understanding of the CAM location, as well as identifying a prominent AIIS.

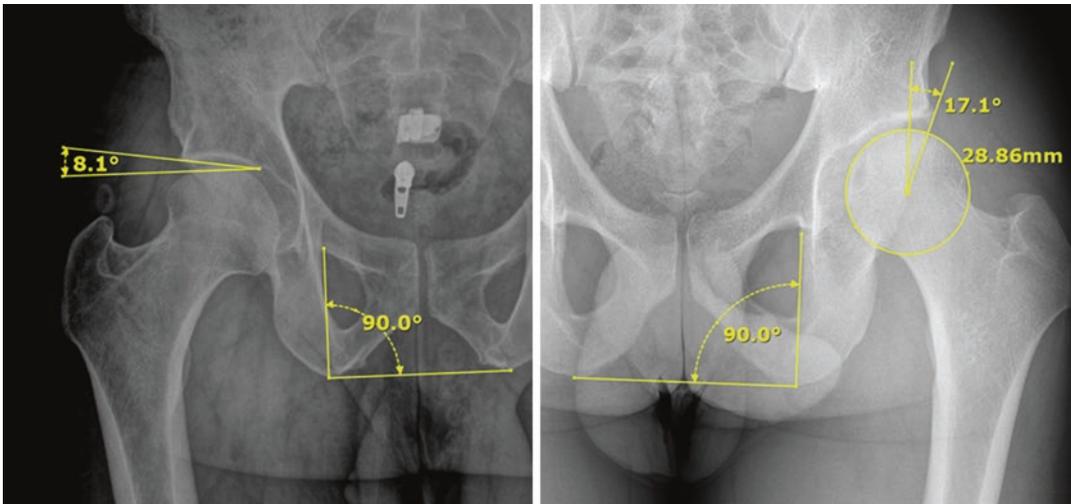


Fig. 28.8 Acetabular index and lateral center edge angle. The acetabular index is formed by a horizontal line connecting both triradiate cartilages (Hilgenreiner line) and a second line which extends along the acetabular roofs. The lateral center edge angle is measured between the line from

the center of the femoral head parallel to the longitudinal axis and the line from the center of the femoral head to the most lateral aspect of the acetabulum or the sourcil edge. The Inter-Ischial line (or alternatively, the Inter Teardrop line) is often used as a reference to avoid any pelvic tilt bias

28.4.7 Treatment of FAIS

Treatment of hip-related pain in athletes continues to evolve. Initial management of hip related pain is usually conservative and most patients, particularly recreational or low-demand athletes may benefit from non-operative care. NSAIDs may be beneficial in cases of muscle contusions, tendinitis or inflammatory causes of hip pain. Local anesthetic injections with or without steroids - bursal, intra-articular, or to the symphysis pubis - can be diagnostic for confirming the cause of hip pain as well as therapeutic in certain instances. Many individuals will benefit from physical therapy after sustaining a hip injury [28]. Athletes with extra-articular pathology are more likely to respond than athletes with intra-articular abnormalities. Therapy should focus on reduction of inflammation, managing pain, regaining dynamic stability, strength, and normalizing joint movement.

If conservative management fails, surgery should be considered. Recent evidence suggests that high-level athletes and patients with intra-articular hip pathology seem to benefit more from operative management [66, 67]. Hip arthroscopy is most commonly performed for intra-articular

conditions such as FAIS, labral tears, cartilage injuries, capsular or synovial disorders, loose bodies, and extra-articular impingement. Labral tears can be repaired and stabilized whenever applicable using suture anchors. If there is labral fraying or a small tear, debridement is an option. If an extensive debridement has been performed as a result of extensive labral damage where repair is not feasible, labral reconstruction could be considered; however, there is lack of evidence on return to sport following this procedure. Focal pincer morphology can be addressed with an acetabuloplasty to remove the impinging bone or alternatively, with debridement of the reciprocal head-neck junction to avoid impingement. If present, subspine impingement can also be debrided [48]. CAM deformities can be addressed with a re-shaping osteochondroplasty of the femoral head-neck junction. As capsulotomy is routinely performed during hip arthroscopy to allow access and maneuverability, concerns may exist with regard to de-stabilizing the hip. Recent literature has suggested that routine and complete capsular closure following capsulotomy is important to restore the natural biomechanics to the hip and optimize outcomes [68–70]. Rehabilitation

following hip surgery can be challenging but is essential in obtaining a positive outcome. The rehab process requires a delicate exercise progression to restore mobility, gait, strength, and neuromuscular control and return to normal activity while preventing excessive anterior hip joint forces that can lead to chronic anterior hip pain. Post-op rehabilitation should focus on addressing muscular and functional hip deficits. The athlete should progress from isolated hip exercises targeting the deep hip stabilizers, into functional activities. Developing hip muscle strength through isolated hip strength exercises such as the Copenhagen Adduction exercise [71], the sliding hip exercise [72], and hip flexion with an elastic band [73] should also be emphasized to increase the load absorption capacity of the hip joint complex. During rehabilitation, the clinician should pay attention to, and address, potential painful competing structures, such as the iliopsoas muscle [74]. Many muscle strengthening exercises and functional exercises can be performed on the court and sport specific tasks which do not involve the lower limb could be performed from very early stages of rehab (i.e., shooting from a seated position). The late phases of rehab should develop toward sport-specific functional tasks with a gradual progression from tasks with no opposition onto full contact. Return to play guidelines should be based on factors such as leg symmetry on hip muscle strength and one-leg jump performance, as well as pain-free capacity to perform complex sport-specific tasks. Clinicians should also be aware that psychological factors such as self-efficacy, motivation and fear of re-injury may be important for successful return to play and should be addressed if indications of such potential barriers exist [75].

28.4.8 Return to Sport

Return to play (RTP) following hip arthroscopy has been previously evaluated [76–83]. Stubbs and colleagues evaluated return to sport and performance outcomes after hip arthroscopy in four pro-level sports, showing an 85.7% RTP rate at an average of 243 days following hip arthroscopy in

28 NBA athletes. No differences in performance were found after RTP [82]. Christian et al. reported an 81% RTP rate in 24 NBA players following hip arthroscopy with a mean RTS time of 175 days [77]. They also found no difference in games played or sport-specific performance scores following return to NBA play after hip arthroscopy. Jack and colleagues evaluated performance and return to sport across four professional sports following hip arthroscopy for FAI [79]. While they reported similar RTP rates for NBA players compared to other sports, they found a decrease in playing time, career longevity, and performance in NHL players following RTP. Ishoi and associates evaluated RTP and sports performance in 189 athletes identified in a Danish Hip Arthroscopy Registry [84]. When more specifically questioning return to sport and performance level, their study found that only 57% of athletes were able to return to their preinjury sport at pre-injury level. Additionally, only 29.6% of athletes reported optimal sports performance including full sports participation. In general, these studies showed a high RTP rate across various professional and amateur sports; however, there is limited data on RTS at pre-injury level [85]. It is likely that in professional sports career longevity and performance following RTP may vary across sports and even playing positions.

Fact Box 4

Initial management of FAIS is conservative and most patients, particularly recreational or low demand athletes, may benefit from non-operative care. However, evidence suggests that high-level players with intra-articular hip pathology seem to benefit more from operative management with good RTS rates.

28.5 Hip Osteoarthritis (OA) in Basketball

While it has been shown that elite athletes are at a higher risk of developing hip and knee OA and undergoing arthroplasty surgery, there is limited

data on OA prevalence in basketball [86]. It has been shown that athletes involved in pivoting sports such as basketball are at increased risk for developing hip pathology which can lead to FAIS, labral tears, and cartilage injury [29, 41]. High-intensity sport activity causing physal stress surrounding adolescence, such as the one encountered in basketball, has also been suggested as a risk factor for the development of the CAM morphology, with observations showing higher FAIS incidence in athletes participating in high-level sports [42, 43]. CAM-type impingement has been highlighted as a risk factor of hip OA [87], and therefore, basketball players with such features could be a population at risk for development of hip OA. A recent survey study by Ekhtiari et al. in 108 retired NBA players showed more than one third (36.3%) of athletes reported current hip and/or groin pain, with 17.6% of athletes receiving injections for hip or groin conditions since retiring from the NBA. Since retiring, 14.7% of the study respondents had undergone total hip arthroplasty. They concluded that retired NBA athletes are at high risk of hip and groin pain after retirement and are more likely to require total hip arthroplasty compared with the general population [88].

28.6 Summary

The hip and groin of basketball players are subjected to high biomechanical loads leading mainly to soft tissue injuries about the hip and pelvis as well as intra-articular injuries which represent a significant burden of injury. The majority of injuries around the hip are soft tissue strains and contusions and are managed primarily with conservative treatment with dedicated rehabilitation protocols and activity modification. For recalcitrant pain and in selected conditions, surgical treatment should be considered. The actual prevalence of intra-articular hip disorders in basketball may have been underestimated over the years. The prevalence of FAIS in basketball has grown with the evolution in understanding and diagnosis of the condition in recent years.

Initial management of FAIS is conservative; however, evidence suggests that high-level players with intra-articular hip pathology seem to benefit more from operative management with good RTS rates.

References

1. Borowski LA, Yard EE, Fields SK, Comstock RD. The epidemiology of US high school basketball injuries, 2005–2007. *Am J Sports Med.* 2008;36(12):2328–35.
2. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the National Basketball Association: a 17-year overview. *Sports Health.* 2010;2:284–90.
3. Jackson TJ, Starkey C, McElhiney D, Domb BG. Epidemiology of hip injuries in the National Basketball Association: a 24-year overview. *Orthop J Sports Med.* 2013;1(3):2325967113499130.
4. Kerbel YE, Smith CM, Prodromo JP, Nzeogu MI, Mulcahey MK. Epidemiology of hip and groin injuries in collegiate athletes in the United States. *Orthop J Sports Med.* 2018;6(5):2325967118771676.
5. Newman JS, Newberg AH. Basketball injuries. *Radiol Clin N Am.* 2010;48:1095–111.
6. Serner A, Tol JL, Jomaah N, Weir A, Whiteley R, Thorborg K, Robinson M, Holmich P. Diagnosis of acute groin injuries: a prospective study of 110 athletes. *Am J Sports Med.* 2015;43:1857–64.
7. Weir A, De Vos RJ, Moen M, Holmich P, Tol JL. Prevalence of radiological signs of femoroacetabular impingement in patients presenting with long-standing adductor-related groin pain. *Br J Sports Med.* 2011;45:6–9.
8. Whittaker JL, Small C, Maffey L, Emery CA. Risk factors for groin injury in sport: an updated systematic review. *Br J Sports Med.* 2015;49:803–9.
9. Mosler AB, Agricola R, Weir A, Holmich P, Crossley KM. Which factors differentiate athletes with hip/groin pain from those without? A systematic review with meta-analysis. *Br J Sports Med.* 2015;49:810.
10. Soligard T, Schweltnus M, Alonso JM, Bahr R, Clarsen B, Dijkstra HP, Gabbett T, Gleeson M, Häggglund M, Hutchinson MR, Janse van Rensburg C, Khan KM, Meeusen R, Orchard JW, Pluim BM, Raftery M, Budgett R, Engebretsen L. How much is too much? (Part 1) International Olympic Committee consensus statement on load in sport and risk of injury. *Br J Sports Med.* 2016;50(17):1030–41.
11. Holmich P. Long-standing groin pain in sportspeople falls into three primary patterns, a "clinical entity" approach: a prospective study of 207 patients. *Br J Sports Med.* 2007;41:247–52; discussion 252.
12. Tyler TF, Silvers HJ, Gerhardt MB, Nicholas SJ. Groin injuries in sports medicine. *Sports Health.* 2010;2(3):231–6.

13. Tyler TF, Nicholas SJ, Campbell RJ, McHugh MP. The association of hip strength and flexibility with the incidence of adductor muscle strains in professional ice hockey players. *Am J Sports Med.* 2001;29(2):124–8.
14. Mei-Dan O, Lopez V, Carmont MR, McConkey MO, Steinbacher G, Alvarez PD, Cugat RB. Adductor tenotomy as a treatment for groin pain in professional soccer players. *Orthopedics.* 2013;36(9):e1189–97.
15. Schilders E, Dimitrakopoulou A, Cooke M, Bismil Q, Cooke C. Effectiveness of a selective partial adductor release for chronic adductor-related groin pain in professional athletes. *Am J Sports Med.* 2013;41(3):603–7.
16. Bharam S, Fegghi DP, Porter DA, Bhagat PV. Proximal avulsion injuries: outcomes of surgical reattachment in athletes. *Orthop J Sports Med.* 2018;6(7):2325967118784898.
17. Eckard TG, Padua DA, Dompier TP, Dalton SL, Thorborg K, Kerr ZY. *Am J Sports Med.* 2017; 45(12):2713–22. Epub 2017 Jul 26.
18. Werner J, Hagglund M, Walden M, Ekstrand J. UEFA injury study: a prospective study of hip and groin injuries in professional football over seven consecutive seasons. *Br J Sports Med.* 2009;43:1036–40.
19. Blankenbaker DG, De Smet AA, Keene JS. Sonography of the iliopsoas tendon and injection of the iliopsoas bursa for diagnosis and management of the painful snapping hip. *Skelet Radiol.* 2006;35(8):565–71.
20. Wettstein M, Jung J, Dienst M. Arthroscopic psoas tenotomy. *Arthroscopy.* 2006;22(8):907.e1–4
21. Orchard JW, Seward H, Orchard JJ. Results of 2 decades of injury surveillance and public release of data in the Australian Football League. *Am J Sports Med.* 2013;41:734–41.
22. Joesting DR. Diagnosis and treatment of sportsman's hernia. *Curr Sports Med Rep.* 2002;1(2):121–4.
23. Larson CM. Sports hernia/athletic pubalgia: evaluation and management. *Sports Health.* 2014;6(2):139–44.
24. Meyers WC, Foley DP, Garrett WE, Lohnes JH, Mandelbaum BR. Management of severe lower abdominal or inguinal pain in high-performance athletes. PAIN (Performing Athletes with Abdominal or Inguinal Neuromuscular Pain Study Group). *Am J Sports Med.* 2000;28(1):2–8.
25. Zoga AC, Kavanagh EC, Omar IM, Morrison WB, Koulouris G, Lopez H, et al. Athletic pubalgia and the "sports hernia": MR imaging findings. *Radiology.* 2008;247(3):797–807.
26. Irshad K, Feldman LS, Lavoie C, Lacroix VJ, Mulder DS, Brown RA. Operative management of "hockey groin syndrome": 12 years of experience in National Hockey League players. *Surgery.* 2001;130(4):759–64; discussion 64–6.
27. Beighton P, Horan F. Orthopaedic aspects of the Ehlers-Danlos syndrome. *J Bone Joint Surg Br.* 1969;51:444–53.
28. Griffin DR, Dickenson EJ, O'Donnell J, Agricola R, Awan T, Beck M, Clohisy JC, Dijkstra HP, Falvey E, Gimpel M, Hinman RS, Holmich P, Kassarian A, Martin HD, Martin R, Mather RC, Philippon MJ, Reiman MP, Takla A, Thorborg K, Walker S, Weir A, Bennell KL. The Warwick Agreement on femoroacetabular impingement syndrome (FAI syndrome): an international consensus statement. *Br J Sports Med.* 2016;50:1169–76.
29. Siebenrock KA, Ferner F, Noble PC, Santore RF, Werlen S, Mamisch TC. The cam-type deformity of the proximal femur arises in childhood in response to vigorous sporting activity. *Clin Orthop Relat Res.* 2011;469(11):3229–40.
30. Ganz R, Parvizi J, Beck M, et al. Femoroacetabular impingement: a cause for osteoarthritis of the hip. *Clin Orthop Relat Res.* 2003;417:112–20.
31. Dickenson E, Wall PD, Robinson B, et al. Prevalence of cam hip shape morphology: a systematic review. *Osteoarthr Cartil.* 2016;24:949–61.
32. Frank JM, Harris JD, Erickson BJ, et al. Prevalence of femoroacetabular impingement imaging findings in asymptomatic volunteers: a systematic review. *Arthroscopy.* 2015;31:1199–204.
33. Leunig M, Nho SJ, Turchetto L, Ganz R. Protrusion acetabuli: new insights and experience with joint preservation. *Clin Orthop Relat Res.* 2009;467(9): 2241–50.
34. Blankenbaker DG, Tuite MJ, Keene JS, del Rio AM. Labral injuries due to iliopsoas impingement: can they be diagnosed on MR arthrography? *AJR Am J Roentgenol.* 2012;199(4):894–900.
35. Cascio BM, King D, Yen YM. Psoas impingement causing labrum tear: a series from three tertiary hip arthroscopy centers. *J La State Med Soc.* 2013;165(2):88–93.
36. Domb BG, Shindle MK, McArthur B, Voos JE, Magennis EM, Kelly BT. Iliopsoas impingement: a newly identified cause of labral pathology in the hip. *HSS J.* 2011;7(2):145–50.
37. Gosvig KK, Jacobsen S, Sonne-Holm S, Palm H, Troelsen A. Prevalence of malformations of the hip joint and their relationship to sex, groin pain, and risk of osteoarthritis: a population-based survey. *J Bone Joint Surg Am.* 2010;92(5):1162–9.
38. Ganz R, Horowitz K, Leunig M. Algorithm for femoral and periacetabular osteotomies in complex hip deformities. *Clin Orthop Relat Res.* 2010;468(12):3168–80.
39. Mavcic B, Iglc A, Kralj-Iglc V, Brand RA, Vengust R. Cumulative hip contact stress predicts osteoarthritis in DDH. *Clin Orthop Relat Res.* 2008;466(4): 884–91.
40. Russell ME, Shivanna KH, Grosland NM, Pedersen DR. Cartilage contact pressure elevations in dysplastic hips: a chronic overload model. *J Orthop Surg Res.* 2006;1:6.
41. Zadpoor AA. Etiology of Femoroacetabular impingement in athletes: a review of recent findings. *Sports Med.* 2015;45(8):1097–106.
42. Agricola R, Heijboer MP, Ginai AZ, Roels P, Zadpoor AA, Verhaar JA, Weinans H, Waarsing JH. A cam

- deformity is gradually acquired during skeletal maturation in adolescent and young male soccer players: a prospective study with minimum 2-year follow-up. *Am J Sports Med.* 2014;42:798–806.
43. Nepple JJ, Vigdorich JM, Clohisy JC. What is the association between sports participation and the development of proximal femoral cam deformity? A systematic review and meta-analysis. *Am J Sports Med.* 2015;43:2833–40.
 44. Hetsroni I, Poultides L, Bedi A, Larson CM, Kelly BT. Anterior inferior iliac spine morphology correlates with hip range of motion: a classification system and dynamic model. *Clin Orthop Relat Res.* 2013;471(8):2497–503.
 45. Mader TJ. Avulsion of the rectus femoris tendon: an unusual type of pelvic fracture. *Pediatr Emerg Care.* 1991;7(2):126.
 46. Rajasekhar C, Kumar KS, Bhamra MS. Avulsion fractures of the anterior inferior iliac spine: the case for surgical intervention. *Int Orthop.* 2001;24(6):364–5.
 47. Rossi F, Dragoni S. Acute avulsion fractures of the pelvis in adolescent competitive athletes: prevalence, location and sports distribution of 203 cases collected. *Skelet Radiol.* 2001;30(3):127–31.
 48. Larson CM, Kelly BT, Stone RM. Making a case for anterior inferior iliac spine/subspine hip impingement: three representative case reports and proposed concept. *Arthroscopy.* 2011;27(12):1732–7.
 49. Lee S, Kim I, Lee SM, Lee J. Ischiofemoral impingement syndrome. *Ann Rehabil Med.* 2013;37(1):143–6.
 50. Taneja AK, Bredella MA, Torriani M. Ischiofemoral impingement. *Magn Reson Imaging Clin N Am.* 2013;21(1):65–73.
 51. Narvani AA, Tsiridis E, Kendall S, Chaudhuri R, Thomas P. A preliminary report on prevalence of acetabular labrum tears in sports patients with groin pain. *Knee Surg Sports Traumatol Arthrosc.* 2003;11:403–8.
 52. Nho SJ, Leunig M, Larson CM, Bedi A, Kelly B. *Hip arthroscopy and hip joint preservation surgery.* New York: Springer; 2015.
 53. Holmich P, Holmich LR, Bjerg AM. Clinical examination of athletes with groin pain: an intraobserver and interobserver reliability study. *Br J Sports Med.* 2004;38:446–51.
 54. Freke MD, Kemp J, Svege I, Risberg MA, Semciw A, Crossley KM. Physical impairments in symptomatic femoroacetabular impingement: a systematic review of the evidence. *Br J Sports Med.* 2016;50:1180.
 55. Delahunt E, Kennelly C, McEntee BL, Coughlan GF, Green BS. The thigh adductor squeeze test: 45 of hip flexion as the optimal test position for eliciting adductor muscle activity and maximum pressure values. *Manual Therapy.* 2011;16:476–80.
 56. Reiman MP, Goode AP, Cook CE, Holmich P, Thorborg K. Diagnostic accuracy of clinical tests for the diagnosis of hip femoroacetabular impingement/labral tear: a systematic review with meta-analysis. *Br J Sports Med.* 2015;49:811.
 57. Reiman MP, Thorborg K, Covington K, Cook CE, Hölmich P. Important clinical descriptors to include in the examination and assessment of patients with femoroacetabular impingement syndrome: an international and multi-disciplinary Delphi survey. *Knee Surg Sports Traumatol Arthrosc.* 2017;25(6):1975–86.
 58. Troelsen A, Mechlenburg I, Gelineck J, Bolvig L, Jacobsen S, Søballe K. What is the role of clinical tests and ultrasound in acetabular labral tear diagnostics? *Acta Orthop.* 2009;80(3):314–8.
 59. Ayeni O, Chu R, Hetaimish B, et al. A painful squat test provides limited diagnostic utility in CAM-type femoroacetabular impingement. *Knee Surg Sports Traumatol Arthrosc.* 2014;22(4):806–11. <https://doi.org/10.1007/s00167-013-2668-8>.
 60. Jackson TJ, Estess AA, Adamson GJ. Supine and standing AP pelvis radiographs in the evaluation of pincer femoroacetabular impingement. *Clin Orthop Relat Res.* 2016;474(7):1692–6.
 61. Reiman MP, Thorborg K, Goode AP, Cook CE, Weir A, Hölmich P. Diagnostic Accuracy of Imaging Modalities and Injection Techniques for the Diagnosis of Femoroacetabular Impingement/Labral Tear: A Systematic Review With Meta-analysis. *Am J Sports Med.* 2017;45(11):2665–77.
 62. Nepple JJ, Prather H, Trousdale RT, Clohisy JC, Beaulé PE, Glyn-Jones S, Rakhra K, Kim YJ. Diagnostic imaging of femoroacetabular impingement. *J Am Acad Orthop Surg.* 2013;21(Suppl 1):S20–6.
 63. Mascarenhas VV, Rego P, Dantas P, Morais F, McWilliams J, Collado D, Marques H, Gaspar A, Soldado F, Consciencia JG. Imaging prevalence of femoroacetabular impingement in symptomatic patients, athletes, and asymptomatic individuals: a systematic review. *Eur J Radiol.* 2016;85:73–95.
 64. McCarthy JC, Lee JA. Acetabular dysplasia: a paradigm of arthroscopic examination of chondral injuries. *Clin Orthop Relat Res.* 2002:122–8.
 65. Khan W, Khan M, Alradwan H, Williams R, Simunovic N, Ayeni OR. Utility of intra-articular hip injections for femoroacetabular impingement: a systematic review. *Orthop J Sports Med.* 2015;3:2325967115601030.
 66. Begly JP, Buckley PS, Utsunomiya H, Briggs KK, Philippon MJ. Femoroacetabular impingement in professional basketball players: return to play, career length, and performance after hip arthroscopy. *Am J Sports Med.* 2018;46(13):3090–6.
 67. Griffin DR, Dickenson EJ, Wall PDH, Achana F, Donovan JL, Griffin J, Hobson R, Hutchinson CE, Jepson M, Parsons NR, Petrou S, Realpe A, Smith J, Foster NE, FASHIoN Study Group. Hip arthroscopy versus best conservative care for the treatment of femoroacetabular impingement syndrome (UK FASHIoN): a multicentre randomised controlled trial. *Lancet.* 2018;391(10136):2225–35.
 68. Dippmann C, Kraemer O, Lund B, et al. Multicentre study on capsular closure versus non-capsular closure during hip arthroscopy in Danish patients with femoroacetabular impingement (FAI): proto-

- col for a randomised controlled trial. *BMJ Open*. 2018;8(2):e019176.
69. Frank RM, Lee S, Bush-Joseph CA, Kelly BT, Salata MJ, Nho SJ. Improved outcomes after hip arthroscopic surgery in patients undergoing T-capsulotomy with complete repair versus partial repair for femoroacetabular impingement: a comparative matched-pair analysis. *Am J Sports Med*. 2014;42(11):2634–42.
 70. Riff AJ, Kunze KN, Movassaghi K, et al. Systematic review of hip arthroscopy for femoroacetabular impingement: the importance of Labral repair and capsular closure. *Arthroscopy*. 2019;35(2):646–56, e643.
 71. Ishoi L, Sorensen CN, Kaae NM, Jorgensen LB, Holmich P, Serner A. Large eccentric strength increase using the Copenhagen Adduction exercise in football: a randomized controlled trial. *Scand J Med Sci Sports*. 2016;26:1334–42.
 72. Kohavi B, Beato M, Laver L, Freitas TT, Chung LH, Dello Iacono A. Effectiveness of Field-Based Resistance Training Protocols on Hip Muscle Strength Among Young Elite Football Players [published online ahead of print, 2018 Oct 29]. *Clin J Sport Med*. 2018;10.1097/JSM.0000000000000649.
 73. Thorborg K, Bandholm T, Zebis M, Andersen LL, Jensen J, Holmich P. Large strengthening effect of a hip-flexor training programme: a randomized controlled trial. *Knee Surg Sports Traumatol Arthrosc*. 2016;24:2346–52.
 74. Sansone M, Ahlden M, Jonasson P, Thomee R, Falk A, Sward L, Karlsson J. Can hip impingement be mistaken for tendon pain in the groin? A long-term follow-up of tenotomy for groin pain in athletes. *Knee Surg Sports Traumatol Arthrosc*. 2014;22:786–92.
 75. Tjong VK, Cogan CJ, Riederman BD, Terry MA. A qualitative assessment of return to sport after hip arthroscopy for femoroacetabular impingement. *Orthop J Sports Med*. 2016;4:2325967116671940.
 76. Casartelli NC, Leunig M, Maffiuletti NA, Bizzini M. Return to sport after hip surgery for femoroacetabular impingement: a systematic review. *Br J Sports Med*. 2015;49(12):819–24.
 77. Christian RA, Lubbe RJ, Chun DS, Selley RS, Terry MA, Hsu WK. Prognosis following hip arthroscopy varies in professional athletes based on sport. *Arthroscopy*. 2019;35(3):837–42, e831.
 78. Ishoi L, Thorborg K, Kraemer O, Holmich P. Return to sport and performance after hip arthroscopy for femoroacetabular impingement in 18- to 30-year-old athletes: a cross-sectional cohort study of 189 athletes. *Am J Sports Med*. 2018;46(11):2578–87.
 79. Jack RA 2nd, Sochacki KR, Hirase T, Vickery JW, Harris JD. Performance and return to sport after hip arthroscopy for femoroacetabular impingement in professional athletes differs between sports. *Arthroscopy*. 2019;35(5):1422–8.
 80. Locks R, Utsunomiya H, Briggs KK, McNamara S, Chahla J, Philippon MJ. Return to play after hip arthroscopic surgery for femoroacetabular impingement in professional soccer players. *Am J Sports Med*. 2018;46(2):273–9.
 81. Mohan R, Johnson NR, Hevesi M, Gibbs CM, Levy BA, Krych AJ. Return to sport and clinical outcomes after hip arthroscopic labral repair in young amateur athletes: minimum 2-year follow-up. *Arthroscopy*. 2017;33(9):1679–84.
 82. Schallmo MS, Fitzpatrick TH, Yancey HB, Marquez-Lara A, Luo TD, Stubbs AJ. Return-to-play and performance outcomes of professional athletes in North America after hip arthroscopy from 1999 to 2016. *Am J Sports Med*. 2018;46(8):1959–69.
 83. Weber AE, Kuhns BD, Cvetanovich GL, Grzybowski JS, Salata MJ, Nho SJ. Amateur and recreational athletes return to sport at a high rate following hip arthroscopy for femoroacetabular impingement. *Arthroscopy*. 2017;33(4):748–55.
 84. Ishøi L, Thorborg K, Kraemer O, Hölmich P. Return to Sport and Performance After Hip Arthroscopy for Femoroacetabular Impingement in 18- to 30-Year-Old Athletes: A Cross-sectional Cohort Study of 189 Athletes. *Am J Sports Med*. 2018;46(11):2578–87.
 85. Reiman MP, Peters S, Sylvain J, Hagymasi S, Mather RC, Goode AP. Femoroacetabular impingement surgery allows 74% of athletes to return to the same competitive level of sports participation but their level of performance remains unreported: a systematic review with meta-analysis. *Br J Sports Med*. 2018;52(15):972–81.
 86. Tveit M, Rosengren BE, Nilsson JÅ, Karlsson MK. Former male elite athletes have a higher prevalence of osteoarthritis and arthroplasty in the hip and knee than expected. *Am J Sports Med*. 2012;40:527–33.
 87. Agricola R, Waarsing JH, Arden NK, Carr AJ, Bierma-Zeinstra SM, Thomas GE, Weinans H, Glyn-Jones S. Cam impingement of the hip: a risk factor for hip osteoarthritis. *Nat Rev Rheumatol*. 2013;9(10):630–4.
 88. Ekhtiari S, Khan M, Burrus T, Madden K, Gagnier J, Rogowski JP, Maerz T, Bedi A. Hip and groin injuries in professional basketball players: impact on playing career and quality of life after retirement. *Sports Health*. 2019;11(3):218–22.



Knee Injuries in Basketball

29

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29.1 Introduction

In 2006, The International Basketball Federation, more commonly known by the French acronym FIBA (Fédération Internationale de Basketball), estimated that 11% of the world's population plays basketball. These numbers have grown substantially since, and new developments in the

game such as 3 on 3 basketball, even being introduced as an Olympic sport, have contributed to its ever-growing popularity.

While the literature has been fairly limited regarding the injury rates of basketball players in Europe, there is abundance of literature on injury rates from US basketball. When comparing injury data between the USA and European players, one has to be aware of the differences between the game played in the USA and the one played in Europe. This is partly due to the different rules maintained by the NBA (National Basketball Association), the NCAA (National Collegiate Athletic Association), and FIBA (Fédération Internationale de Basketball) (e.g., an NBA game is 48-minute long, while the FIBA rules game is 40 minute long) [1]. However, it is clear from the various epidemiologic studies that injuries of the lower extremities are frequent in basketball players. Most epidemiological studies have shown that the majority of acute injuries in basketball are located in the lower extremities, regardless of age, gender, and level of play. A recent study investigating 273 male players of Division I and II basketball teams in Ghana reported the knee to be the most commonly injured anatomic site [2].

Basketball was traditionally considered a non-contact sport but has evolved into an increasingly physical game in which contact is accepted and common [1, 3]. Coaches commonly teach their players to use contact, and players routinely use their bodies to their advantage, including when

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fighting for position, intentionally drawing contact for fouls, and using forearms and elbows to ward off defenders. This evolution has definitely contributed to the number of injuries observed, for example, in the NBA [4].

Physical contact has been shown to be responsible for 52.3% of the game-related injuries in male and 46.0% of injuries in female collegiate players [3]. The most common lower-limb injuries in basketball occur at the ankle and knee. Data collected over 17 seasons in the NBA has shown the knee to be the most common site for injuries (19.1%), although data was collected separately for knee and patellar injuries [4]. Injuries to the patella represented 10.1% of injuries while knee injuries represented 9.0% of all injuries. Knee injuries also resulted in the most games missed [4]. In collegiate basketball, ankle and knee were the most affected with an injury rate of 28.8% and 12.0%, respectively [5]. A recent systematic review of epidemiologic studies in basketball showed knee injuries to be the second most common site of injuries (17.8% of all injuries) after the ankle (21.9%) [6]. A report from a European league over a single season has shown knee overuse injuries to be the most common (23.01%) followed by ankle sprains (15.0%) [1].

29.2 Type and Incidence of Knee Injuries in Basketball

The types of injuries experienced by basketball players reflect the physical demands of the game [4].

In general, sprains (injuries to ligaments) are the most common type of acute injury. Two common serious knee injuries of high importance in basketball are Jumper's knee and anterior cruciate ligament (ACL) injury, due to the significant associated injury burden and time loss from sports.

Jumper's knee, or patellar tendinopathy, particularly affects the proximal end of the patellar tendon in basketball players (around 20% of cases involve the distal insertion of the quadriceps tendon) and accounts for approximately 70% of patellar tendon injuries [1]. Due to its insidious

nature, patellar tendon disorders may be the "silent endemic" among professional basketball players. All players are vulnerable, and particularly those who are 'aerial' players by nature, and depend on frequent and repetitive jumping for their game style [1]. This condition is the leading cause of players missing practices and games, often in multiple, small increments rather than the season-ending magnitude that are common in other, severe and acute injuries [7]. In a survey, independent of the applied treatment strategy, one third of the athletes presenting with patellar tendinopathy were unable to complete a full practice for >6 months [1]. Overuse of the patellar tendon can lead to pain, tenderness, and functional deficit. Patellar tendinopathy, as a typical overuse injury, is related to the intensity of training, jumping performance, and ankle and knee joint dynamics [1].

ACL injury is one of the most severe knee injuries in the sport as it is often season-ending or, at times, even career-ending. Female basketball players have 3–7.8 times increased risk to sustain an ACL injury in comparison to their male counterparts when competing at the same level of competition.

ACL injuries have been reported as non-contact in etiology in 65.2% of male and 80.1% of female college players. Although the majority of ACL injuries arise from a non-contact mechanism, video analysis of 39 ACL injuries identified that one-half of the ACL injuries in women involved the player being pushed or involved in a collision just preceding the time of injury. Authors reported that 71.8% of ACL tears occurred while the injured player was in possession of the ball, and over half (56.4%) occurred while on offense. For female players, 59.1% of ACL injuries occurred during single-leg landings, compared to 35.3% in male players [3, 4, 7].

29.3 Injury Severity of Knee Injuries in Basketball

A key factor for every injury in sports is assessing the duration of time away from sports as well as the chances for return to pre-injury level.

The number of days the athlete will miss define the severity of the injury. Injuries are usually classified as minor (1–7 days), moderate (8–28 days), severe (>28 days), and career-ending injuries [3, 4].

In general, knee injuries are often more severe in nature and therefore are responsible for a significant time loss from play. In the injury statistics in professional German Basketball (top 2 German leagues), the knee is second only to ankle injuries in terms of injury frequency. However, considering the resulting treatment costs, the knee joint is clearly the leader [8]. In the NBA, the knee is only in fourth place in terms of the frequency of injuries, but it is clear number one in terms of missed games [4]. In collegiate basketball players, knee injuries have been reported to cause 18.3 missed days on average [1, 4].

29.4 Significant and Common Knee Injuries in Basketball

29.4.1 Fractures Around the Knee in Basketball

Fractures are not very common in basketball, especially ones which involve the knee. The most common site for fractures around the knee is the patella [9], including osteochondral fractures following patellar dislocations. In addition, the epiphyses of the distal femur and proximal tibia are often involved either in isolation or in association with other intra-articular injuries (Fig. 29.1). Many fractures around the knee are

caused by high-energy trauma, such as falls from significant heights, jumps, or direct collisions with other players. In adolescent players, fractures involving the tibial tuberosity are typical to this population, although rare [10].

29.4.2 Patellar Dislocations in Basketball

Patellar dislocation is part of the patellar instability spectrum, which is defined as an abnormal movement of the patella in the patellofemoral groove. It is characterized by subluxation or true dislocation of the patella, predominantly in the lateral direction. Patients with recurrent patellar instability episodes commonly have specific risk factors. Acute patella dislocation represents 2–3% of all knee injuries [11] and the literature reports recurrence rates of 15–60% [12]. Data from a prospective epidemiologic database collected from emergency departments has shown nearly half (51.9%) of all patellar dislocations occurred during athletic activity, with the highest rates associated with basketball (18.2%), followed by football (6.9%) and American football (6.3%) [13]. Patellofemoral injury was shown to account for 10.1% of all injuries in NBA players [4]. Reports from collegiate sports have shown that patellar dislocation (38.6%) and subluxation (19.8%) represented the highest proportion of injuries that resulted in 14 days or more of time loss from sports and were also two of the most likely injuries to require surgical repair [10]. These findings are similar to the ones reported by Mitchell

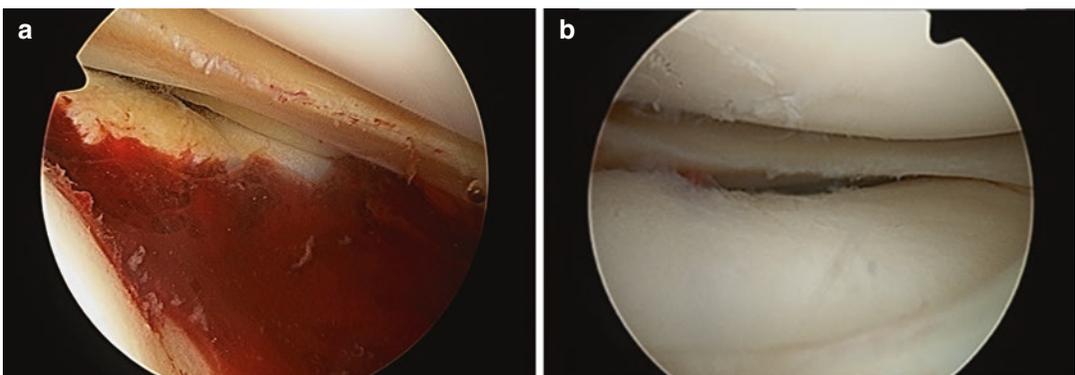


Fig. 29.1 Lateral tibial plateau fracture (Schatzker type III) before reduction (a) and after reduction (b)

et al. [14] in which 20% of high school athletes with patellar dislocation and 14% with a patellar subluxation required more than 3 weeks to return to play. In addition, 37% of dislocation patients and 11% of subluxation patients missed the rest of the season [14].

The injury mechanism is usually an indirect mechanism combining rotation, quadriceps contraction, and valgus. The patella often reduces spontaneously with the extension of the knee.

Such trauma is usually followed by swelling and pain. A direct injury mechanism can also exist from trauma to the medial part of the knee. Patella dislocations are more common in adolescent and young adult players, with the peak incidence of patellar dislocation being between 15 and 19 years of age [13]. Females seem to have a higher risk for patella dislocation.

Various risk factors have been described for patellar instability, and they are extensively discussed in separate designated chapters in this book. They are roughly categorized into osseous/morphologic factors and soft tissue factors. Valgus knee alignment increases the lateral force vector on the patella. Increased femoral anteversion combined with external tibial torsion will also increase this laterally directed force. The patella is a sesamoid bone which is stabilized medially and laterally by the two surfaces of the femoral trochlea. The lateral trochlea ridge is larger, more proximal, and more anterior than the medial trochlea and thus prevents lateral patellar excursion.

Morphological variations such as trochlear dysplasia, meaning a shallow, flattened, or even a convex trochlear groove and lateral femoral condyle hypoplasia decrease the control of the lateral displacement of the patella. Patella alta is another risk factor as this condition leads to a late engagement of the patella in the trochlea during knee flexion. An excessive lateral position of the tibial tuberosity with its patellar tendon attachment is another risk factor and will contribute to lateral displacement of the patella.

Soft tissue risk factors also exist. The medial patellofemoral ligament (MPFL) is a retinacular band located between the superomedial aspect of the patella and the medial femoral epicondyle. It is the primary restraint to lateral patellar dis-

placement, especially during the first degrees of flexion. It is frequently disrupted during a first lateral patellar dislocation. A slack MPFL is therefore a risk factor for recurrent patellar dislocation. Vastus medialis oblique (VMO) is an important dynamic medial patella stabilizer, and weakening of this muscle can predispose to lateral patellar dislocation. It should therefore be one of the main targets for strengthening during physiotherapy for lateral patellar instability [15].

Clinical examination following acute patellar dislocation can be difficult. Knee ROM is usually limited due to pain and effusion which can be significant. Aspiration of the hemarthrosis may sometimes be warranted to facilitate the physical examination. The area around the MPFL and the medial patella are usually painful during palpation. In cases of chronic instability, patients must be assessed in a standing position and observed for morphological abnormalities including genu valgum/increased Q angle, hindfoot valgus, foot pronation, and malposition of the patella or “squinting patella”. Gait and rotation of the hip joint should also be assessed. Several clinical tests may aid in the diagnosis of patellar instability. In the *apprehension test*, a feeling of apprehension is often noted in patellar instability when the patella is pushed laterally by the examiner. A *positive J-sign* represents a sudden lateral patellar shift as it goes over the proximal edge of lateral trochlea ridge when the patient is asked to actively extend the knee from 90°. The *patella glide test* is performed with the knee in full extension and relaxed quadriceps. The patella is translated in the mediolateral plane by the examiner. Patellar displacement is quantified by quadrants and compared to the opposite knee to evaluate the significance of passive lateral patellar displacement.

Radiographs, including standing AP, lateral views at 30° of knee flexion, as well as a sunrise view, are useful in detecting patellar subluxation, osteochondral fractures, or dysplasia. The lateral view allows evaluation of patellar height and identifies trochlear dysplasia. A “crossing sign” has been shown to be present in 96% of patients with history of true patellar dislocation [16]. Dejour’s classification of trochlear dysplasia is based on features from the lateral and axial radiographs [16] (Fig. 29.2). MRI is useful to assess

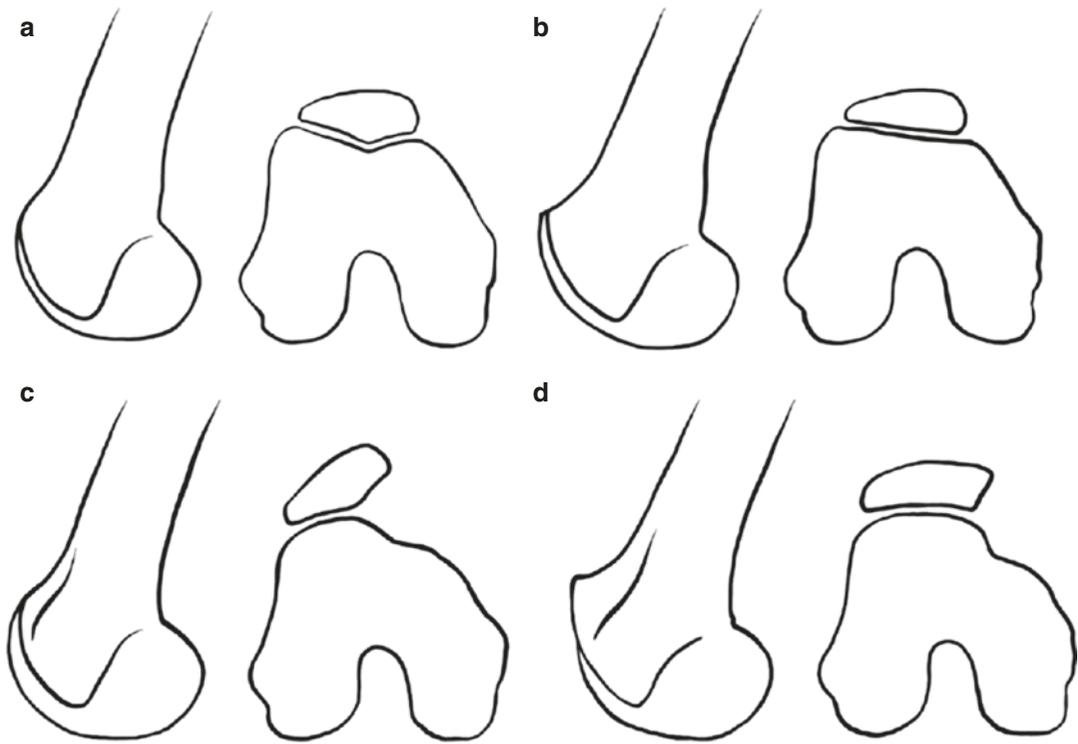


Fig. 29.2 The Dejour trochlear dysplasia classification: Type **a**: Crossing sign, shallow trochlea $>145^\circ$. Type **b**: Crossing sign, supratrochlear spur, flat, or convex trochlea. Type **c**: Crossing sign, double contour (projection of medial hypoplastic facet). Type **d**: Crossing sign, supra-

trochlear spur, double contour, cliff sign (From: Landreau P, Laver L, Seil R (2018) Knee injuries in handball. In: Laver L, Landreau P, Seil R, Popovic N (Hrsg) Handball Sports Medicine. Springer, Berlin, Heidelberg, S 261–278)

the soft tissues, including the MPFL, and cartilage surfaces. Following an acute patellar dislocation, MRI usually shows bone marrow edema on the lateral femoral condyle and medial patella border [17]. Computed tomography (CT scan) allows for bony morphology assessment, especially trochlear dysplasia and patellar shape. It may also be used to quantify the extent of lateralization of the tibial tuberosity, defined by the TT-TG (tibial tuberosity—trochlear groove) distance [16]. A TT-TG distance greater than 20 mm is frequently associated with patella instability and is taken into consideration when considering tibial tuberosity osteotomy as a surgical solution.

Management of patellar dislocation and patellar instability in basketball players is extensively discussed in a separate chapter. The majority of first-time patellar dislocations in basketball are managed conservatively, except in situations

where an osteochondral fracture or a major chondral injury exists, requiring acute fixation, or in the scenario of a persistent patellar dislocation. Operative management is considered in subsequent recurrent dislocations and in cases of failure to improve with nonoperative management, especially in the presence of one or more predisposing factors for patellar instability. For those athletes who have recurrent lateral patella dislocations despite being compliant with an appropriate rehabilitation program, MPFL reconstruction is a surgical option increasing in popularity, in the absence of significant anatomic-morphologic abnormalities [18] (Fig. 29.3).

The management algorithm of patellar instability in basketball players should therefore be based on the risk factors, history of patellar instability, and level of play [19]. Return to basketball following operative treatment depends on several factors including the severity of risk factors and

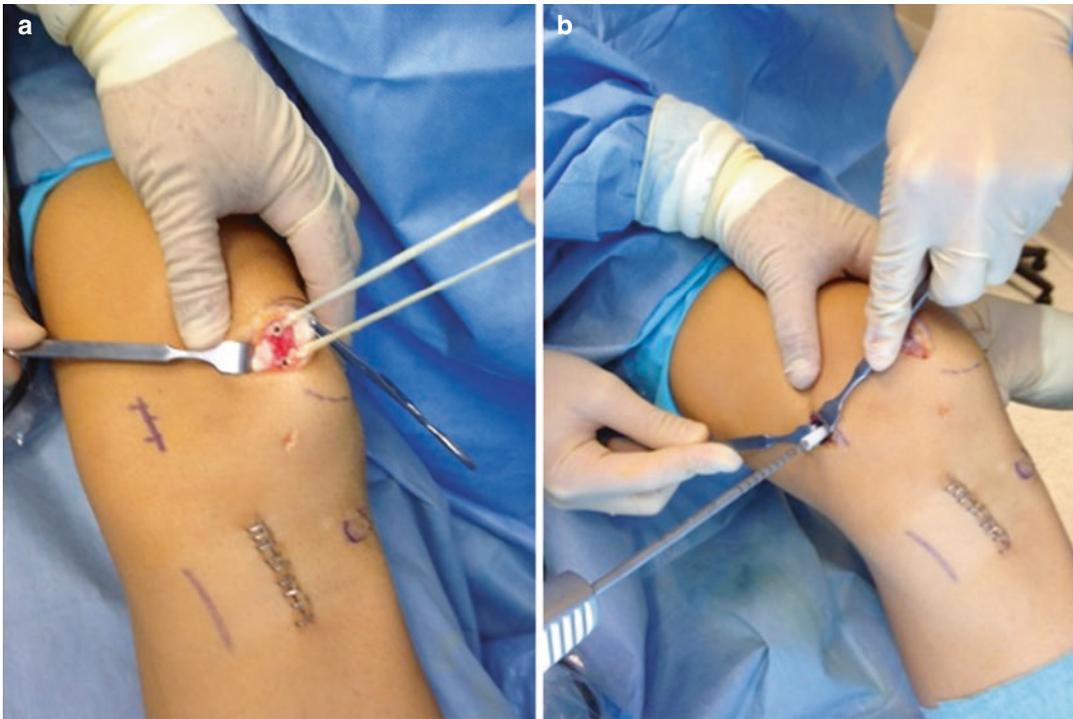


Fig. 29.3 (a) Two anchors in the patella securing the MPFL graft during reconstruction and (b) interferential screw placement to secure the MPFL graft to the femur

type of surgical treatment. RTS is rarely achieved before 6 months following surgery. Although the percentage of patients able to return to sport has been reported to be higher than 90% in some series, return to pre-injury level of sport has been shown to vary widely and is generally considered much lower in athletes (32–82%) [19].

It is important to note that patellar dislocations may occur due to a pre-existing morphologic abnormality in the knee and can also often be caused by high-energy trauma, such as falls and contact injuries.

29.4.3 Knee Dislocations in Basketball

Knee dislocations are rare but may occur in basketball. Approximately 50% of knee dislocations reduce spontaneously. Due to the high risk of neurovascular compromise, the team physician should be alert and suspect such an injury, even

with subtle clinical findings. A peripheral neurovascular examination should always be performed when assessing a patient with a knee injury. It is important to always remember that a normal neurovascular examination in the setting of a knee dislocation does not always rule out a vascular injury and maybe due to collateral circulation masking a popliteal artery injury. Therefore, serial examinations are mandatory. A true or suspected knee dislocation is an orthopedic emergency, and as such, the patient should be transferred to an emergency department immediately.

29.4.4 Ligamentous Knee Injuries in Basketball

29.4.4.1 Anterior Cruciate Ligament (ACL) Injuries in Basketball

An ACL injury is one of the most severe injuries in sports and is more prevalent in cutting and piv-

oting sports such as basketball, accounting for up to 64% of all knee injuries [20–23].

The incidence of ACL injury in basketball has been widely reported, with a meta-analysis of epidemiologic studies showing female and male basketball player ACL tear rates of 0.29 and 0.08 per 1000 athletic exposures (defined as a practice or game), respectively [24]. Rapid cutting or an incorrect landing from a jump are common ACL injury mechanisms in basketball. A substantial amount of ACL injuries occur along with damage to other structures in the knee, such as articular cartilage, meniscus, or other ligaments. Despite their relatively low incidence (<1% of all injuries), ACL lesions are one of the most attention-drawing injuries in sports in general and in basketball in particular. While some decades ago, it was considered a career-ending injury, advances in surgical techniques, optimal anatomic understanding, and rehabilitation have led to improved rates of return to sports even at pre-injury level in many patients. Young players between the ages of 15 and 25 years account for half of all ACL injuries. An athlete who has torn his/her ACL has a 15 times greater risk of a second ACL injury during the initial 12 months after an ACL reconstruction. Risk of ACL injury to the opposite knee is twice that of the reconstructed knee [25].

Fact Box 1

- The incidence of further meniscus lesions could probably be reduced if the torn ACL was surgically reconstructed; however, current evidence does not support the fact that this would be due to the surgical repair rather than to a decrease of involvement in strenuous activities.
- It has not been demonstrated that ACL reconstruction can prevent osteoarthritis [26].

Studies comparing surgical and conservative treatments confirm that ACL reconstruction is not a prerequisite for returning to sports activities, except in children and adolescents. Recent well-

designed studies demonstrate that conservative treatment could give satisfactory results for many patients in non-pivoting sports [27]. Patients should be counseled on realistic expectations and implications when deciding between conservative and surgical treatment. ACL injuries are discussed extensively in other chapters in this book.

29.4.4.2 Posterior Cruciate Ligament (PCL) Injuries in Basketball

PCL injuries are uncommon in basketball [28], however may pose a challenging problem, especially when combined with other ligamentous injuries.

A common mechanism for a PCL injury involves a blow to the front of the knee while the knee is bent and the foot is plantar-flexed; however, hyperextension is also a well-recognized mechanism. PCL injury in sports is often associated with contact. PCL tears are often partial tears with the potential to heal on their own. A review of the literature suggests that partial and some complete isolated PCL tears are best treated with conservative management. Generally only in cases where there is an avulsion fracture, or associated ligamentous injuries should PCL reconstruction be performed. Chronic posterior instability should be treated operatively only if the patients are severely symptomatic. However, complete restoration of knee stability is usually not achieved independent of the different surgical techniques. Very few studies in the literature focus on isolated PCL injury. Recent studies are in general more optimistic with regard to the results than previous reports [28]. There are only a few high-quality studies, which may limit the value of the reported results [29]. Firm recommendations on what treatment to choose cannot be given at this time on the basis of these studies. PCL injuries are discussed extensively in another chapter in this book.

29.4.4.3 Collateral Ligament Injury

Medial Collateral Ligament (MCL) Injuries in Basketball

Collateral ligament injuries are usually generated from forces that push the knee sideways. There are two common MCL tear mechanisms: (1) a

direct contact on the lateral part of the knee with valgus force and (2) a noncontact rotational injury, usually with a combination of flexion, valgus, and external rotation. Collateral ligament injuries can occur in isolation as well as in concomitance with cruciate ligament injuries as part of a multi-ligament injury to the knee [30]. At the collegiate level, rates of MCL injuries have been reported to be 0.87/10,000 athletic exposures (AE) for females and 1.02/10,000 AE for males, while at the high school level rates have been reported to be 0.62/10,000 AE for females and 0.3/10,000 AE for males [31]. Large registry data has shown MCL injury in association with ACL injury in basketball (1.2%) to be much less common than in football (3%), American football (2.8%), and handball (2.7%) [30]. Thorough history taking could aid in identifying the injury mechanism and diagnosis. The physical examination should include inspection and palpation to identify any swelling, deformity, and ecchymosis, keeping in mind that combined injuries are not uncommon. In the supine position, the MCL is palpated from its femoral insertion to the tibia. A valgus stress test should be performed at 0 and 30° of knee flexion. An isolated laxity at 30° is indicative of an isolated lesion of the superficial part of the MCL. Laxity present at 0° of extension indicates a more severe injury involving other knee structures. Hughston classified laxity into three grades [32]: grade I (laxity less than 5 mm), grade II (laxity between 6 and 10 mm), and grade III (laxity more than 10 mm). Theoretically, low-grade MCL injuries can be managed without any additional imaging. Stress radiographs (i.e., TELOS) may still have a role in higher grades of injury, for recovery assessment and in chronic cases, with easy comparison available to the contralateral side, however, they should not be performed in the acute trauma situation. Ultrasound is an excellent and low-cost modality for diagnosis and follow-up, however is operator dependent. With the availability of modern imaging modalities and the pressure of a professional sport environment, MRI allows for an accurate evaluation of the MCL (Fig. 29.4). It is important to note that the MRI tends to overestimate the lesion, and therefore, the treatment must

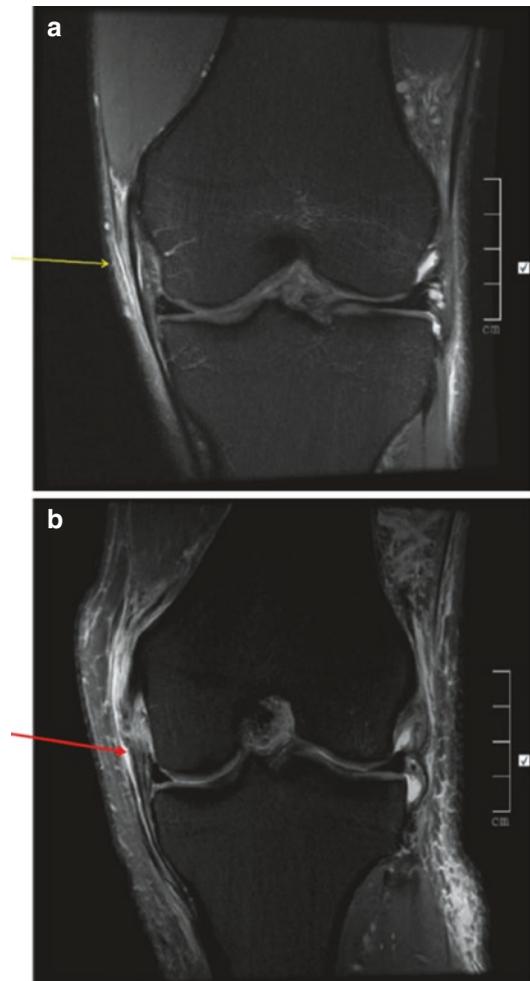


Fig. 29.4 (a) MCL sprain grade I. (b) Complete tear of the MCL, grade III (From: Landreau P, Laver L, Seil R (2018) Knee injuries in handball. In: Laver L, Landreau P, Seil R, Popovic N (Hrsg) Handball Sports Medicine. Springer, Berlin, Heidelberg, S 261–278)

be conducted and led based mainly on clinical examination and progression [33]. If severe medial laxity in full extension exists, MRI will help the surgical decision-making by allowing for an accurate evaluation of the MCL (whether a proximal or the rarer distal MCL tear, which if a complete tear, could result in a “stener-like lesion”) and potential associated meniscus injuries.

The majority of isolated MCL tears are treated conservatively. Grade I injuries (sprain) are treated with RICE with or without a hinged knee brace for a few days until pain and swelling have

improved. Early physiotherapy usually allows return to sport within 2–3 weeks following injury.

A similar treatment is applied for grade II sprains, with the exception that the brace is often applied for a longer period (up to 3 weeks) in order to allow the pain to disappear. For this reason, the recovery of strength and proprioception may take longer. The athlete can usually return to play 6 weeks after the injury. Nonoperative treatment of grade III MCL injury takes longer. In these patients, it is important to protect the knee throughout the healing process with a hinged brace. The minimum period of immobilization is 4 weeks and is gradually discontinued between the fourth and sixth week. During the following 3–6 weeks, the patient can start progressive rehabilitation and sport-specific exercises. Special attention should be given to patients with valgus alignment. Return to sport is rarely possible before 10 weeks in a grade III injury. There are still controversies over the role of surgical management for isolated grade III MCL injuries [34] in athletes. Nonoperative management of a complete tear of the medial ligamentous complex, including the posterior-oblique ligament (POL), may lead to residual rotational instability, especially in a pivoting sport like basketball. Surgery may be considered in full disruption of the medial ligamentous complex or when conservative treatment fails.

Lateral Collateral Ligament (LCL) and Postero-Lateral Corner (PLC) Injuries in Basketball

In basketball, as in other team ball-sports, isolated LCL and PLC injuries are generally less common than MCL injuries. However, the true incidence may be underestimated as these injuries are sometimes misdiagnosed and therefore underreported [35]. Their incidence is much higher when they are combined with other ligamentous lesions such as ACL or PCL injuries and may be present in up to 40% [36]. Misdiagnosis can lead to inappropriate treatment and result in chronic lateral and posterolateral laxity, potentially jeopardizing the athlete's career. In addition, ACL or PCL reconstruction surgery may be compromised if lateral and posterolateral injuries are neglected.

During clinical examination, it is important to compare the injured knee to the opposite knee since some players may have physiological lateral laxity without any symptoms. The majority of cases are the result of a noncontact mechanism, combining hyperextension, varus, and external rotation. But in some cases, it can be the result of a direct medial trauma. This kind of injury mechanism can lead to either isolated or combined ACL and PCL injuries and even result in a knee dislocation in a “worst case scenario.” It is important to remember that some knee dislocations may be missed as they can reduce spontaneously on the field. If such an injury is suspected, a vascular and neurologic assessment should be performed. The clinical examination must focus on assessing the integrity of the peroneal nerve and the popliteal vessels.

LCL and PLC injuries should be suspected in the presence of swelling, hematoma, and tenderness in the posterolateral aspect of the knee, hyperextension of the injured knee, varus laxity at 30° of knee flexion (LCL tear), varus laxity at 0° of knee flexion (suspicious for PLC injury and cruciate ligament injury), a positive *dial test* (more than 10° of side-to-side difference in external rotation in 30° of knee flexion, suggesting a PLC injury) (Fig. 29.5), external rotation recurvatum test (external rotation of the tibia when the knee is in hyperextension), a positive posterolateral drawer test (suspected popliteal tendon and popliteo-fibular ligament injury), and a positive reverse-pivot shift test (reduction of the posterior subluxation of the lateral tibial plateau as the knee is brought close to extension) [37].

Management of LCL and PLC injuries depends on multiple factors such as the degree of laxity, knee alignment, combined injuries, time from injury, and even playing level. The classification system proposed by Hughston [38] is easily clinically applicable and is based on the degree of laxity (grade I, 0–5 mm; grade II, 5–10 mm; grade III, > 10 mm). Laxity must always be compared to the opposite knee. While grade I injuries are usually treated conservatively and grade III injuries are treated surgically, the treatment decision-making in grade II injuries is more complex and challenging, and each case

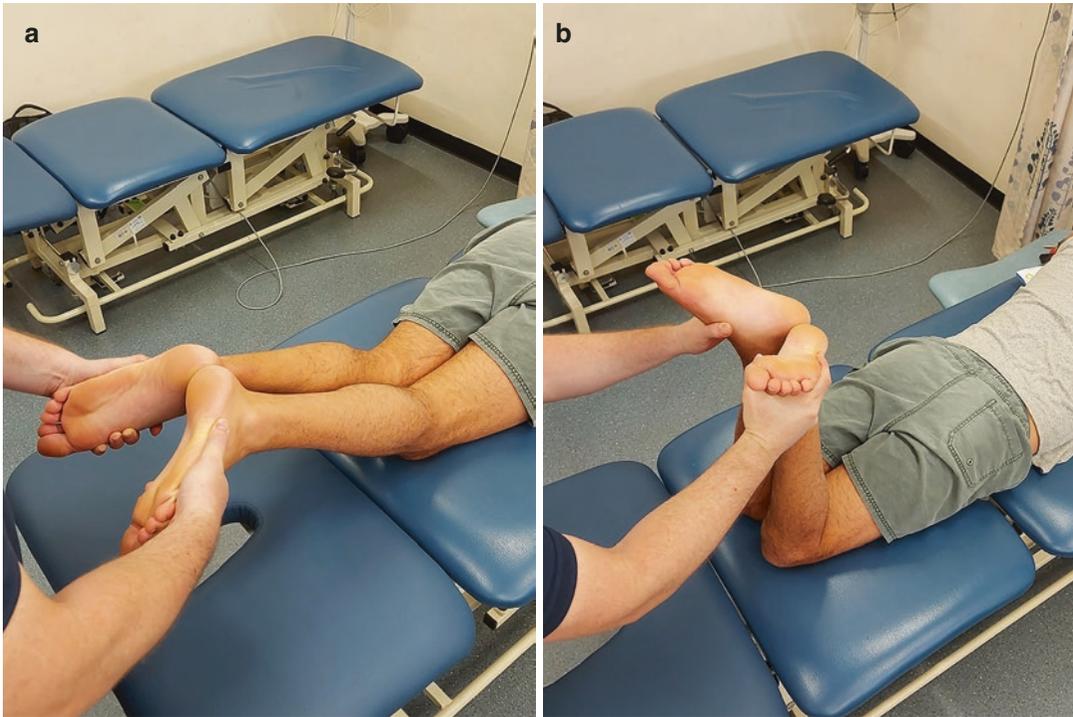


Fig. 29.5 *The dial test.* While the patient is in the prone position, a maximal external a rotation force is applied to the feet while the knees are close together. The test is performed with the knees in 30° (a) and 90° (b) of flexion. A difference of more than 10° of external rotation in the injured knee compared to the uninjured, suggests PLC

injury. If present only in 30°, the test is indicative of an isolated PLC injury. If present both in 30° and 90° – the test is indicative of a combined PCL and PLC injury. If present only in 90° – the test is indicative of PCL instability. Courtesy of Dr. Lior Laver

must be evaluated separately taking into consideration the factors previously mentioned. These injuries and their management are extensively discussed in a separate chapter in this book.

29.4.5 Meniscal Injuries in Basketball

Meniscal injuries are common in basketball. Tears in the meniscus can be traumatic (Fig. 29.6a) as a result of twisting, cutting or pivoting maneuvers, or direct trauma. Degenerative meniscal tears may also occur as a result of arthritic changes or as a result of the biomechanical effects of aging on the meniscus (Fig. 29.6b). Database analysis by Yeh et al. [39] identified 129 isolated meniscal injuries occurring in 21 NBA seasons; of these, 77 (59.7%) involved the lateral meniscus and 52 (40.3%) involved the medial meniscus, with a higher occurrence

recorded in athletes under 30 years of age and in those with a body mass index (BMI) higher than 25 kg/m². They reported a slightly greater involvement of the right knee (53.5%) compared with the left knee (46.5%), although a different distribution was reported by Baker et al. [40], who found right knee involvement in 80% of cases. Furthermore, similar RTS results were shown for medial and lateral injuries, while 25 athletes (19.4%) failed to return to sporting activities [39]. Data from injury profile in elite female basketball athletes at the Women's National Basketball Association (WNBA), combine, collected from 506 players between 2000 and 2008, revealed 10.5% had meniscal injuries [41]. In this study, meniscus surgery was found to be the second most common surgery in college women basketball players entering the WNBA. Meniscal injuries were not found to affect career length in the WNBA, and meniscal injuries were not found

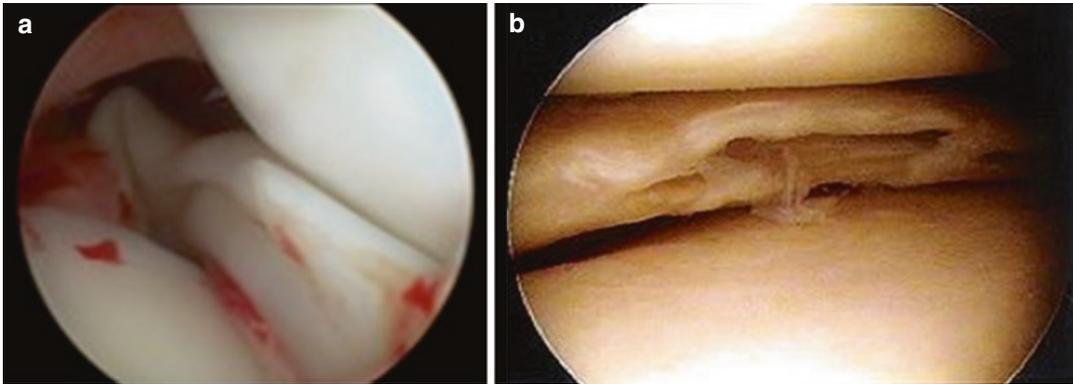


Fig. 29.6 Acute (a) “bucket handle” tear of a medial meniscus and (b) degenerative “fish mouth” tear

to vary between playing positions [41]. Granan et al. have found an increased odds ratio of sustaining a concomitant lateral meniscus injury with ACL injuries in basketball players, analyzing large registry data [30].

Partial or total loss of the meniscus leads to biomechanical abnormalities which may significantly affect sports careers in the short and long term. In 1948 Fairbank [42] reported the clinical outcomes of 107 patients after total meniscectomies and found that the majority had degenerative changes. Similar findings were reported by Pengas et al. concluding that meniscectomy leads to symptomatic osteoarthritis of the knee later in life, with a resultant 132-fold increase in the rate of total knee replacement in comparison to geographical and age-matched peers [43].

The meniscus is composed of three zones: the vascularized red-red zone is located in its periphery, providing good healing potential after repair, the red-white zone has intermediate vascularity, and the white-white (central) zone is avascular. Both menisci provide joint congruency, load transfer, pressure distribution, impact absorption, secondary stabilization, joint nutrition, and lubrication.

The medial meniscus is longer than the lateral meniscus in the anteroposterior direction, and it covers 50% of the medial tibial plateau. The lateral meniscus covers 59% of the lateral tibial plateau [44]. The lateral meniscus carries most of the load transfer on the lateral compartment, while load transmission in the medial compartment is distributed between the cartilage surface

and the medial meniscus [45]. The surface of the lateral tibial plateau is convex, whereas the medial surface is concave. Therefore, lateral meniscectomy will result in proportionally greater contact stress and higher risk of cartilage damage and osteoarthritis compared to the medial compartment [46]. The role of the menisci as secondary knee stabilizers is well recognized [47]. The posterior horn of the medial meniscus helps controlling anterior tibial translation, and the lateral meniscus has a role in controlling internal tibial rotation [47, 48].

Clinically, acute tears present with sudden pain, usually located on the joint line. Effusion may be present, although this is not specific for meniscal tears. Mechanical symptoms may be present, such as clicking or true locking of the knee. In degenerative meniscal lesions, symptoms are of a more chronic nature. Recurrent episodes of pain with effusion are often reported, typically in the absence of an injury. Presence of mechanical symptoms such as clicking and locking with a mechanical block to extension is important to be noted, as it can influence the treatment decision. Some degenerative meniscus tears are asymptomatic and may be an incidental finding on an MRI performed for a different reason. These tears should be left alone. A meniscus tear should be suspected particularly when the patient reports pain localized along the joint line, provoked, or increased by hyperflexion, directional change during walking, crossing legs when seated, or when catching one’s foot on an irregular surface.

Physical examination in suspicion of a meniscal tear should include assessment of gait, mobility, range of motion (ROM), laxity, limb alignment and effusion, as well as assessment of the patellofemoral joint. Specific meniscus tests should be included in the examination [49]:

- *Joint line tenderness* on palpation typically reproduces pain or discomfort.
- *The McMurray test* is performed in the supine position; the knee is extended from fully flexed position while internally rotating the tibia and repeated while externally rotating the tibia. The purpose of this maneuver is to impinge the meniscus between the femur and the tibia. Tenderness and/or crepitation along the joint line during this maneuver indicate a positive sign.
- *The Apley test* is another test causing meniscus compression and grinding of the meniscus between the articular surfaces. It is performed with the patient in a prone position with the knee flexed at 90°. The tibia is compressed on the distal femur, rotated externally and internally to assess the medial and lateral meniscus. The test is considered positive if it produces pain which is less severe or relieved when the maneuver is performed without compression of the tibia (Fig. 29.7).
- *The Thessaly test* has been described more recently [50]. While in single-leg stance, flat footed on the affected knee, the patient axially rotates the knee several times in 5 and then 20° of knee flexion. The test is considered positive when medial/lateral joint line pain or mechanical symptoms are induced.

Among these tests, the joint line palpation has been reported the most sensitive and specific test for isolated meniscus pathology [50, 51].

While in most cases, history and physical examination would allow to suspect isolated meniscal pathology, imaging confirmation should be obtained and can also aid in the management decision-making.

MRI is the most accurate imaging modality for the diagnosis of meniscal lesions [52] and is noninvasive. It also allows good visualization of



Fig. 29.7 The Apley test. Courtesy of Dr. Lior Laver

the surrounding soft tissues and capsular attachments as well as assessing the cartilage and the subchondral bone. High signal within the meniscal substance indicates meniscal pathology. An increased internal signal ending at one of the articular surfaces of the meniscus is a strong indicator of a meniscal tear. The specificity of this sign is improved if the increased signal is visible on more than one consecutive image [53]. Sagittal, coronal, and axial sequences usually allow defining the shape of the tear: vertical, horizontal, radial, or the classic “bucket handle” tear with a “double PCL sign” where the displaced meniscus tissue appears as a second line parallel and anterior to the PCL (Fig. 29.8).

MRI diagnosis of posterior meniscus root tears can be more challenging [54]. A posterior lateral meniscus (LM) root tear usually occurs in association with ACL injuries. Posterior medial meniscal root tears are often of degenerative nature, but they can be acute following isolated axial compression and torsional trauma as well as in cases of multi-ligament injuries. The MRI may show anteromedial meniscal extrusion and some-

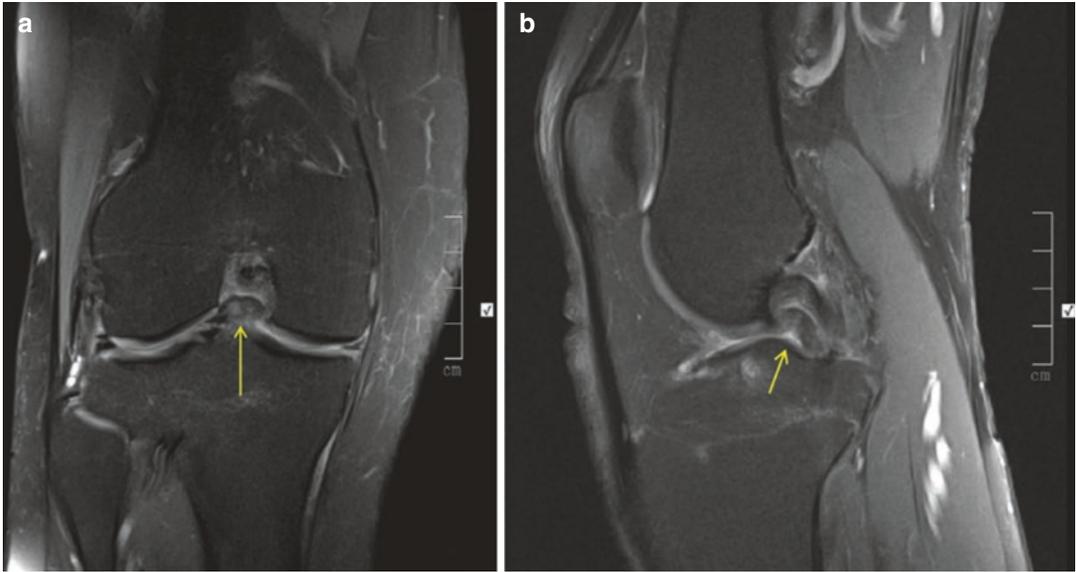


Fig. 29.8 Bucket handle tear of the medial meniscus with (a) meniscal fragment visible in the notch on coronal view. (b) “Double PCL sign” on sagittal view (From: Landreau P, Laver L, Seil R (2018) Knee injuries in hand-

ball. In: Laver L, Landreau P, Seil R, Popovic N (Hrsg) Handball Sports Medicine. Springer, Berlin, Heidelberg, S 261–278)

times the classical “ghost sign” (absence of the posterior horn of the medial meniscus) (Fig. 29.9). The axial view can show a linear defect or edema at the bony insertion of the meniscal root.

While arthroscopic meniscectomy is still frequently performed, recent studies have highlighted the benefit of meniscal repair over partial meniscectomy, when considering clinical outcome and risk of osteoarthritis [55]. The effect of a lateral meniscectomy is less “forgiving” than a medial meniscectomy due to the more significant role of the lateral meniscus in load transfer and the convex morphology of the lateral compartment. This explains the longer delay in RTS following lateral meniscectomy compared to a medial meniscectomy. Therefore, meniscectomy should be considered with great caution, especially for the lateral meniscus. Paxton et al. [55] reported that whereas meniscal repairs have a higher reoperation rate than partial meniscectomies, they are associated with better long-term outcomes. Therefore, meniscus preservation should be attempted, especially in young basketball players. Many factors contribute to the deci-



Fig. 29.9 Posterior medial meniscal root avulsion with the “ghost sign” (absence of the posterior horn of the medial meniscus) (From: Landreau P, Laver L, Seil R (2018) Knee injuries in handball. In: Laver L, Landreau P, Seil R, Popovic N (Hrsg) Handball Sports Medicine. Springer, Berlin, Heidelberg, S 261–278)

sion and choice of treatment, such as age, activity level, location, size, tear pattern, tear chronicity, combined injuries (i.e., ACL injury), and healing potential. Meniscus tears located in the red-red (peripheral) zone should be repaired. This is especially true for those lesions which are repaired in conjunction with ACL reconstructions, since they have a higher healing potential than isolated repairs. In cases of white-white (central) zone tears, a simple partial meniscectomy is usually performed. For tears in the red-white zone, treatment decisions can be more challenging. Healing of these tears could be enhanced using different methods, such as perforations or trephinations reaching into the vascularized area, potentially encouraging cell migration to the tear site. A fibrin clot can be used in combination with rasping of the vascularized parameniscal synovium. More recently, platelet-rich plasma has been used to improve the meniscal tear healing [56]; however, evidence of its efficiency is still lacking.

Complete radial tears lead to complete loss of the biomechanical function of the meniscus. Therefore, repair must be attempted in these cases, especially in young players, even if it is a challenging procedure [57]. Horizontal cleavage tears have been traditionally resected; however, recent studies have shown that repair can lead to good subjective and objective results in the short and long term [58]. Traumatic root tears, which are more often observed in the lateral compartment in combination with ACL tears, should be repaired in young patients as they result complete dysfunction of the meniscus. Transosseous tunnels or an all-inside technique can be used for meniscal root repairs [50]. Meniscus replacement, either by meniscal allograft transplantation or by a scaffold, can be performed in cases of chronic, total, or partial meniscus defects. These situations can occur at the end of a basketball player's career, and currently there is no evidence that surgical procedure can allow professional basketball players to return to the same level of sport [59].

Partial meniscectomy has been shown to provide good short-term results, and players usually return to pre-injury level of performance. However, results seem to deteriorate with time

especially when the lateral meniscus is involved [60]. In general, healing rates after meniscus repair have been shown to provide complete healing in 60% of the cases, partial healing in 25% of the cases, and failure in 15% of the cases [61]. Therefore, meniscus repair must be attempted if potential exists for meniscus healing. The treatment decision can be challenging, especially in high-level professional athletes as the return to sport after an isolated meniscus repair is significantly longer (minimum of 4 months) compared to partial meniscectomy. Treatment decision in professional players is not always straightforward and may rely on factors which are not purely medical or health related. Factors such as time during the season, contract status, player's goals as well as proximity to important or decisive periods of the season may play a role in the treatment decision and should be discussed by all parties before any surgical procedure. The risks and benefits of meniscectomy and repair as well as their short- and long-term effects must be clearly communicated to the player as well as the coaching staff.

29.4.6 Knee Tendinopathies and Tendon Tears in Basketball

Tendinopathies are traditionally considered overuse injuries, involving excessive tensile loading and subsequent breakdown of the loaded tendon. Some tendinopathies may, paradoxically, be considered as "underuse" lesions despite the common beliefs that they are overuse injuries. The traditional concept of tensile failure may not be the essential feature of the pathomechanics of insertional tendinopathy. Tendinopathies are common in basketball, mainly extensor mechanism tendinopathy (patellar tendinopathy/"jumper's knee"; quadriceps tendinopathy) and Achilles tendinopathies, as well as plantar fasciitis.

Both patellar and quadriceps tendinopathies have been historically called "Jumper's knee" due to the high prevalence seen in athletes involved in jumping sports. It has been reported that this injury is prevalent in up to 30% of basketball players. Lian et al. described an overall

“jumper’s knee” prevalence of 14.2% in athletes in their report of 613 athletes with the highest prevalence reported in sports involving high and explosive loading of the knee extensor mechanism such as volleyball (44.6%) and basketball (31.9%) [62]. Zwerver et al. described a “jumper’s knee” prevalence of 11.8% in non-elite basketball players in their report of 891 players [63]. In high school players, it has been reported that 7% of basketball players had current patellar tendinopathy on clinical examination (11% in men, 2% in women) [64].

Repetitive load to the patellar tendon complex in adolescent and middle school athletes (boys aged 12–15 years; girls aged 8–12 years) can lead to *Osgood–Schlatter syndrome* involving the tibial tuberosity. Osgood–Schlatter syndrome is a traction apophysitis of the tibial tubercle due to repetitive strain on the secondary ossification center of the tibial tuberosity [65]. The condition is self-limited, and the vast majority of patients will respond to conservative treatment. While residual deformity may remain, symptoms will resolve, along with closure of the physal plate [66]. Extensor mechanism tendinopathies are extensively discussed in separate chapters dedicated to tendinopathies and patello-femoral disorders.

Tendon rupture occurs mostly due to mechanical stress and intra-tendinous degeneration or pathology and involves rapid loading in an already tensed tendon.

The patellar and quadriceps (Fig. 29.10) tendons, like the Achilles tendon, which is exten-

sively discussed in a separate chapter, are some of the more severe tendon tear injuries in basketball. Patellar tendon rupture can be the ultimate consequence in the spectrum of chronic patellar tendinopathy [67]. There is a complete loss of function, followed by severe effusion and ecchymosis. It is not always preceded by the typical jumper’s knee symptoms; however, these should be regarded when obtaining patient history. Patellar tendon rupture diagnosis is based on clinical examination and history. It is also important to inquire about previous intra- or peritendinous corticosteroid injections. Evidence of a palpable gap between the patella and the tibial tuberosity confirms the diagnosis. The patella often appears in a more proximal position when compared to the contralateral knee, and the patient is unable to actively extend his knee due to extensor mechanism failure. Prior to surgery, ultrasound or MRI is recommended to confirm the diagnosis and to allow for a better understanding of the tear pattern for surgical planning.

Quadriceps tendon tears have a similar clinical onset, however are more common in older players. Clinical symptoms are similar to patellar tendon tears; however, the tendinous gap is either visible or palpable proximally to the patella. Caution should be made not to miss the diagnosis in those with partial quadriceps tendon tears and an incomplete functional loss of the extensor mechanism. In these patients the extensor mechanism is still functioning, but with less power than normal. Radiographs show a high-riding patella

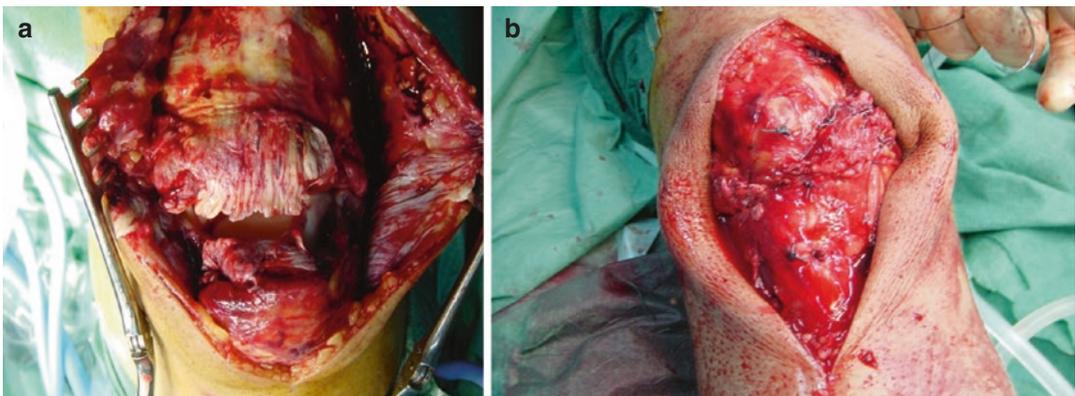


Fig. 29.10 Quadriceps tendon total rupture: (a) before repair, (b) after repair

in cases of patella tendon rupture and patella baja if the quadriceps tendon is torn.

Ultrasound and MRI usually confirm the diagnosis and may provide more information about the quality of the tendon.

Surgical repair is mandatory in case of a complete tear. It consists of suture of the tendon combined with transosseous fixation. In the postoperative period, it is recommended to immobilize the knee in full extension for 6 weeks. Initiating physiotherapy will depend on the quality and strength of the surgical repair. Return to sport is rarely possible before 6 months [67].

Fact Box 2

- Knee extensor mechanism tendinopathies are common in basketball and are traditionally considered overuse injuries, involving excessive tensile loading and subsequent breakdown of the loaded tendon.
- These overuse injuries may result in significant time away from sport; hence, early diagnosis and treatment are important.

29.5 Summary

Knee injuries, both acute and chronic, are very common in basketball and are responsible for the most time away from sport. Management of knee injuries in basketball depends on the severity of the injury, involved anatomic structures, future prognosis, symptomatology, functional disability, age, expectations, and level of play. Prevention programs have been proven to be effective in reducing knee injuries and are easily integrable into warm-up routines. The treatment approach should take into account these factors in order to decide whether a conservative or surgical management is more appropriate. Adequate return to sports decisions and prevention methodologies are key for reduction in the number of basketball injuries.

References

1. Cumps E, Verhagen E, Meeusen R. Prospective epidemiological study of basketball injuries during one competitive season: ankle sprains and overuse knee injuries. *J Sports Sci Med.* 2007;6:204–11.
2. Quarrey J, Davor SF, Kwakye SK. An injury profile of basketball players in Accra, Ghana. *S Afr J Physiother.* 2019;75(1):467.
3. Caine DJ, Harnmer PA, Shiff M. *Epidemiology of injuries in Olympic sports.* Blackwell Publishing Ltd; 2010.
4. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the national basketball association: a 17-year overview. *Sports Health.* 2010;2(4):284–90. <https://doi.org/10.1177/1941738109357303>.
5. Starkey C. Injuries and illnesses in the National Basketball Association: a 10-year perspective. *J Athl Train.* 2000;35(2):161–7.
6. Andreoli CV, Chiamonti BC, Buriel E, Pochini AC, Ejnisman B, Cohen M. Epidemiology of sports injuries in basketball: integrative systematic review. *BMJ Open Sport Exerc Med.* 2018;4(1):e000468.
7. McKay GD, Goldie PA, Payne WR, Oakes BW. Ankle injuries in basketball: injury rate and risk factors. *Br J Sports Med.* 2001;35:103–8.
8. Luig P, Bloch H, Burkhardt K, Klein C, Kuehn N. Der VBG-Sportreport 2017 - Analyse des Unfallgeschehens in den zwei höchsten Ligen der Männer: Basketball, Eishockey, Fußball und Handball. Jedermann Verlag GmbH. 2017:80–99.
9. Trojan JD, Treloar JA, Smith CM, Kraeutler MJ, Mulcahey MK. Epidemiological patterns of patellofemoral injuries in collegiate athletes in the United States from 2009 to 2014. *Orthop J Sports Med.* 2019;7(4):2325967119840712.
10. Benz G, Roth H, Zachariou Z. Fractures and cartilage injuries of the knee joint in children. *Z Kinderchir.* 1986;41(4):219–26.
11. Stefancin JJ, Parker RD. First-time traumatic patellar dislocation: a systematic review. *Clin Orthop Relat Res.* 2007;455:93–101.
12. Fithian DC, Paxton EW, Stone ML, Silva P, Davis DK, Elias DA, White LM. Epidemiology and natural history of acute patellar dislocation. *Am J Sports Med.* 2004;32:1114–21.
13. Waterman BR, Belmont PJ Jr, Owens BD. Patellar dislocation in the United States: role of sex, age, race, and athletic participation. *J Knee Surg.* 2012;25(1):51–7.
14. Mitchell J, Magnussen RA, Collins CL, Currie DW, Best TM, Comstock RD, Flanigan DC. Epidemiology of patellofemoral instability injuries among high school athletes in the United States. *Am J Sports Med.* 2015;43(7):1676–82.
15. LaPrade RF, Engebretsen AH, Ly TV, Johansen S, Wentorf FA, Engebretsen L. The anatomy of the medial part of the knee. *J Bone Joint Surg Am.* 2007;89:2000–10.

16. Dejour H, Walch G, Nove-Josserand L, Guier C. Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc.* 1994;2:19–26.
17. Sanders TG, Morrison WB, Singleton BA, Miller MD, Cornum KG. Medial patellofemoral ligament injury following acute transient dislocation of the patella: MR findings with surgical correlation in 14 patients. *J Comput Assist Tomogr.* 2001;25:957–62.
18. Maffulli N, Caine D. The Epidemiology of Children's Team Sports Injuries. *Med Sport Sci.* 2005;49:1–8.
19. Lion A, Hoffmann A, Mouton C, Theisen D, Seil R. Patellar instability in football players. In: Volpi P, editor. *Football traumatology: new trends.* Switzerland: Springer; 2015. p. 241–52.
20. Deitch JR, Starkey C, Walters SL, Moseley JB. Injury risk in professional basketball players: a comparison of Women's National Basketball Association and National Basketball Association athletes. *Am J Sports Med.* 2006;34(7):1077–83.
21. Mall NA, Abrams GD, Azar FM, Traina SM, Allen A, Parker R. Trends in primary and revision anterior cruciate ligament reconstruction among National Basketball Association team physicians. *Am J Orthop.* 2014;43(06):267–71.
22. Mihata LC, Beutler AI, Boden BP. Comparing the incidence of anterior cruciate ligament injury in collegiate lacrosse, soccer, and basketball players: implications for anterior cruciate ligament mechanism and prevention. *Am J Sports Med.* 2006;34(6):899–904.
23. Rishiraj N, Taunton JE, Lloyd-Smith R, Woollard R, Regan W, Clement DB. The potential role of prophylactic/functional knee bracing in preventing knee ligament injury. *Sports Med (Auckland, NZ).* 2009;39(11):937–60.
24. Prodromos CC, Han Y, Rogowski J, Joyce B, Shi K. A meta-analysis of the incidence of anterior cruciate ligament tears as a function of gender, sport, and a knee injury-reduction regimen. *Arthroscopy.* 2007;23(12):1320–5.e1326
25. Lai CCH, Ardern CL, Feller JA, et al. Eighty-three per cent of elite athletes return to preinjury sport after anterior cruciate ligament reconstruction: a systematic review with meta-analysis of return to sport rates, graft rupture rates and performance outcomes. *Br J Sports Med.* 2017;52:128. <https://doi.org/10.1136/bjsports-2016-096836>.
26. Magnussen RA, Duthon V, Servien E, Neyret P. Anterior Cruciate Ligament Reconstruction and Osteoarthritis: Evidence from Long-Term Follow-Up and Potential Solutions. *Cartilage.* 2013;4(3 Suppl):22S–6S.
27. Delincé P, Ghafil D. Anterior cruciate ligament tears: conservative or surgical treatment? A critical review of the literature. *Knee Surg Sports Traumatol Arthrosc.* 2012;20(1):48–61.
28. Linko E, Harilainen A, Malmivaara A et al. Surgical versus conservative interventions for anterior cruciate ligament ruptures in adults *Cochrane Bone, Joint and Muscle Trauma Group Published Online: 20 Apr 2005.*
29. Kühne JH, Jansson V, Hoppert M, Weippert C, Zimmer M, Refior HJ. Results of surgical treatment of posterior knee instability. *Unfallchirurg.* 1994;97(3):151–8.
30. Granan LP, Inacio MC, Maletis GB, Funahashi TT, Engebretsen L. Sport-specific injury pattern recorded during anterior cruciate ligament reconstruction. *Am J Sports Med.* 2013;41:2814–8.
31. Stanley LE, Kerr ZY, Dompier TP, Padua DA. Sex differences in the incidence of anterior cruciate ligament, medial collateral ligament, and meniscal injuries in collegiate and high school sports: 2009–2010 through 2013–2014. *Am J Sports Med.* 2016;44(6):1565–72.
32. Hughston JC. The importance of the posterior oblique ligament in repairs of acute tears of the medial ligaments in knees with and without an associated rupture of the anterior cruciate ligament. Results of long-term follow-up. *J Bone Joint Surg Am.* 1994;76:1328–44.
33. Marchant MH Jr, Tibor LM, Sekiya JK, Hardaker WT Jr, Garrett WE Jr, Taylor DC. Management of medial-sided knee injuries, part 1: medial collateral ligament. *Am J Sports Med.* 2011;39:1102–13.
34. Sims WF, Jacobson KE. The posteromedial corner of the knee: medial-sided injury patterns revisited. *Am J Sports Med.* 2004;32:337–45.
35. Pacheco RJ, Ayre CA, Bollen SR. Posterolateral corner injuries of the knee: a serious injury commonly missed. *J Bone Joint Surg Br.* 2011;93:194–7.
36. Levy BA, Stuart MJ, Whelan DB. Posterolateral instability of the knee: evaluation, treatment, results. *Sports Med Arthrosc.* 2010;18:254–62.
37. Bahk MS, Cosgarea AJ. Physical examination and imaging of the lateral collateral ligament and posterolateral corner of the knee. *Sports Med Arthrosc.* 2006;14:12–9.
38. Hughston JC, Andrews JR, Cross MJ, Moschi A. Classification of knee ligament instabilities. Part II. The lateral compartment. *J Bone Joint Surg Am.* 1976;58:173–9.
39. Yeh PC, Starkey C, Lombardo S, et al. Epidemiology of isolated meniscal injury and its effect on performance in athletes from the National Basketball Association. *Am J Sports Med.* 2012;40:589–94.
40. Baker BE, Peckham AC, Puppato F, Sanborn JC. Review of meniscal injury and associated sports. *Am J Sports Med.* 1985;13:1–4.
41. McCarthy MM, Voos JE, Nguyen JT, Callahan L, Hannafin JA. Injury profile in elite female basketball athletes at the Women's National Basketball Association combine. *Am J Sports Med.* 2013;41(3):645–51.
42. FAIRBANK TJ. Knee joint changes after meniscectomy. *J Bone Joint Surg Br.* 1948;30B:664–70.
43. Pengas IP, Assiotis A, Nash W, Hatcher J, Banks J, McNicholas MJ. Total meniscectomy in adolescents: a 40-year follow-up. *J Bone Joint Surg Br.* 2012;94(12):1649–54.

44. Bloecker K, Wirth W, Hudelmaier M, Burgkart R, Frobell R, Eckstein F. Morphometric differences between the medial and lateral meniscus in healthy men—a three-dimensional analysis using magnetic resonance imaging. *Cells Tissues Organs*. 2012;195:353–64.
45. Walker PS, Hajek JV. The load-bearing area in the knee joint. *J Biomech*. 1972;5:581–9.
46. Alford JW, Lewis P, Kang RW, Cole BJ. Rapid progression of chondral disease in the lateral compartment of the knee following meniscectomy. *Arthroscopy*. 2005;21:1505–9.
47. Shoemaker SC, Markolf KL. The role of the meniscus in the anterior-posterior stability of the loaded anterior cruciate-deficient knee. Effects of partial versus total excision. *J Bone Joint Surg Am*. 1986;68:71–9.
48. Musahl V, Citak M, O'Loughlin PF, Choi D, Bedi A, Pearle AD. The effect of medial versus lateral meniscectomy on the stability of the anterior cruciate ligament-deficient knee. *Am J Sports Med*. 2010;38:1591–7.
49. Meserve BB, Cleland JA, Boucher TR. A meta-analysis examining clinical test utilities for assessing meniscal injury. *Clin Rehabil*. 2008;22:143–61.
50. Karachalios T, Hantes M, Zibis AH, Zachos V, Karantanas AH, Malizos KN. Diagnostic accuracy of a new clinical test (the Thessaly test) for early detection of meniscal tears. *J Bone Joint Surg Am*. 2005;87:955–62.
51. Fowler PJ, Lubliner JA. The predictive value of five clinical signs in the evaluation of meniscal pathology. *Arthroscopy*. 1989;5:184–6.
52. Muellner T, Weinstabl R, Schabus R, Vècsei V, Kainberger F. The diagnosis of meniscal tears in athletes. A comparison of clinical and magnetic resonance imaging investigations. *Am J Sports Med*. 1997;25:7–12.
53. De Smet AA, Norris MA, Yandow DR, Quintana FA, Graf BK, Keene JS. MR diagnosis of meniscal tears of the knee: importance of high signal in the meniscus that extends to the surface. *Am J Roentgenol*. 1993;161:101–7.
54. Bhatia S, LaPrade CM, Ellman MB, LaPrade RF. Meniscal root tears: significance, diagnosis, and treatment. *Am J Sports Med*. 2014;42:3016–30.
55. Paxton ES, Stock MV, Brophy RH. Meniscal repair versus partial meniscectomy: a systematic review comparing reoperation rates and clinical outcomes. *Arthroscopy*. 2011;27:1275–88.
56. Griffin JW, Hadeed MM, Werner BC, Diduch DR, Carson EW, Miller MD. Platelet-rich plasma in meniscal repair: does augmentation improve surgical outcomes? *Clin Orthop Relat Res*. 2015;473:1665–72.
57. Ra HJ, Ha JK, Jang SH, Lee DW, Kim JG. Arthroscopic inside-out repair of complete radial tears of the meniscus with a fibrin clot. *Knee Surg Sports Traumatol Arthrosc*. 2013;21:2126–30.
58. Sallé de Chow E, Pujol N, Rochcongar G, Cucurulo T, Potel JF, Dalmay W, Ehkirch FP, Laporte C, Le Henaffe G, Seil R, Lutz C, Gunepin FX, Sonnery-Cottet B, d'Arthroscopie SF. Analysis of short and long-term results of horizontal meniscal tears in young adults. *Orthop Traumatol Surg Res*. 2015;101:S317–22.
59. Elattar M, Dhollander A, Verdonk R, Almqvist KF, Verdonk P. Twenty-six years of meniscal allograft transplantation: is it still experimental? A meta-analysis of 44 trials. *Knee Surg Sports Traumatol Arthrosc*. 2011;19:147–57.
60. Chatain F, Adeleine P, Chambat P, Neyret P. Société Française d'Arthroscopie. A comparative study of medial versus lateral arthroscopic partial meniscectomy on stable knees: 10-year minimum follow-up. *Arthroscopy*. 2003;19:842–9.
61. Pujol N, Tardy N, Boisrenoult P, Beaufile P. Long-term outcomes of all-inside meniscal repair. *Knee Surg Sports Traumatol Arthrosc*. 2015;23:219–24.
62. Lian ØB, Engebretsen L, Bahr R. Prevalence of jumper's knee among elite athletes from different sports: a cross-sectional study. *Am J Sports Med*. 2005;33(4):561–7.
63. Zwerver J, Bredeweg SW, van den Akker-Scheek I. Prevalence of Jumper's knee among nonelite athletes from different sports: a cross-sectional survey. *Am J Sports Med*. 2011;39(9):1984–8.
64. Cook JL, Khan KM, Kiss ZS, Griffiths L. Patellar tendinopathy in junior basketball players: a controlled clinical and ultrasonographic study of 268 patellar tendons in players aged 14Y18 years. *Scand J Med Sci Sports*. 2000;10:216–20.
65. Gholve PA, Scher DM, Khakharia S, et al. Osgood Schlatter syndrome. *Curr Opin Pediatr*. 2007;19:44–50.
66. Antich TJ, Brewster CE. Osgood–Schlatter disease: review of literature and physical therapy management. *J Orthop Sports Phys Ther*. 1985;7:5–10.
67. Lee D, Stinner D, Mir H. Quadriceps and patellar tendon ruptures. *J Knee Surg*. 2013;26:301–8.



Management of ACL Injuries in Basketball

30

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and Brian Cole

30.1 Introduction

30.1.1 Incidence

Tears of the ACL are among the most common sports injuries treated by orthopedic surgeons, with an incidence between 30 and 81 per 100,000 people [1, 2]. ACL tears are particularly prevalent in pivoting and cutting sports such as basketball, accounting for up to 64% of all knee injuries [3–6]. The incidence of ACL reconstruction has increased over the recent years, particularly in the young and female athletes, who more commonly undergo reconstruction in order to restore rotatory and translational stability to the knee and maintain performance level [5, 7–9]. The incidence of ACL injury in basketball has also been widely reported, with a meta-analysis of epidemiologic studies showing female and male basketball player tear rates of 0.29 and 0.08 per 1000 athletic exposures (defined as a practice or game), respectively [10].

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30.1.2 ACL Function

The ACL functions as the primary restraint against anterior translation of the knee, providing ~86% of the stability required to prevent anterior translation of the tibia. The ACL also provides rotatory stability, limits hyperextension, and provides secondary coronal stability [11]. Together with the posterior cruciate ligament (PCL), the ACL allows controlled rollback of the femur on the tibia as the knee center of rotation moves posteriorly during flexion. When nearing terminal extension, the tibia externally rotates under the femur, resulting in the tightening of both the ACL and PCL, which stabilizes the knee and allows for stance [11, 12].

30.1.3 Anatomy

The ACL is composed of type I collagen fibers (90%) and type III collagen fibers (10%). The ligament originates from the posterior aspect of the lateral femoral condyle and inserts anterior to the medial tibial eminence, travelling an average distance of 31 ± 3 mm [11, 13]. The ACL is composed of a complex of fascicles which is commonly simplified into two functionally and biomechanically synergistic bundles: the anteromedial bundle, which endures increased tension during flexion, and the posterolateral bundle, which becomes taut in extension [14, 15].

Biomechanical studies have estimated that the ACL has an ultimate strength of between 1730 and 2500 Newtons [16, 17]. The ACL receives blood supply from the middle geniculate artery and innervation from the tibial nerve [18, 19].

30.2 Diagnosis of ACL Tears

30.2.1 History

A thorough history and evaluation is necessary in diagnosing ACL tears and other associated injuries in the basketball athlete. Patients often report an instability event following landing or cutting in a non-contact fashion. Most commonly, the event involves pivoting while accelerating or decelerating while landing forcefully on the heel with slight knee flexion. Although a non-contact mechanism at the time of injury is more common in basketball players, the injury likely includes movement to be perturbed by or contacted by an opposing player earlier in the sequence [20]. Alternatively contact injury mechanism involves a substantial valgus force to the fixed and extended knee [20, 21]. An audible or palpable pop is often described with immediate knee swelling. That athlete is typically unable to continue sports activity.

30.2.2 Physical Examination

Examination should begin with the inspection of the athlete's lower extremity. An effusion is typically representative of an intra-articular pathology. The Lachman test is the key examination maneuver when detecting an ACL injury as it has a high sensitivity and specificity. The patient is placed in the supine position with the knee in approximately 30° of flexion. Instructions on complete relaxation are given while the examiner stabilizes the thigh with one hand and delivers an anterior translation force to the tibia with the other. Amount of translation is noted as well as the presence of a firm end-point. Translation greater than 3 mm compared with the opposite knee and a soft end-point are indicative of ACL

injury. The pivot shift test is an important indicator of rotational instability. The test is performed with the leg in extension and the ankle in internal rotation, a valgus force is then applied to the leg with gentle flexion of the knee. In an ACL-deficient state, this causes an anterior subluxation of the lateral tibia from beneath the lateral femoral condyle. The iliotibial band initially acts as an extensor when its center of rotation is in front of the knee; as the knee is flexed, the ITB becomes a flexor and causes the tibia to be reduced posteriorly. This sudden reduction force is considered a positive test. Although the test is not very sensitive (24%), it is extremely specific (98%). The test is more sensitive in a completely relaxed patient or when performed under general anesthesia (74%) [22]. The anterior drawer test is also valuable in the evaluation of an athlete with an ACL tear, although it is less sensitive and specific than the Lachman test. The test is more useful in patients with chronic ACL tear and is performed with the patients in supine and the knee in 90° of flexion. An anterior force is then applied to the tibia, and the difference in anterior translation between the two knees is evaluated [22]. The above examination maneuvers are very user dependent and are most useful when performed by experienced clinicians, whereas the KT-1000 (or 2000/3000) arthrometer provides a more standardized measurement but is used mostly for research purposes [23].

30.2.3 Imaging

Imaging is an important adjuvant of the clinical examination for an athlete. Although radiographic imaging will not identify ligamentous injury, it allows the assessment of associated bony abnormalities such as avulsion fractures of the tibial spine or anterolateral ligament (Segond fracture). An MRI is useful in confirming ligament injury as well as other soft tissue pathology but is not required for the diagnosis of an ACL rupture (Fig. 30.1). Due to injury mechanism with an acute ACL rupture, the most commonly identified pathology on MRI is ACL rupture, MCL sprain/tear, and posterior lateral meniscus

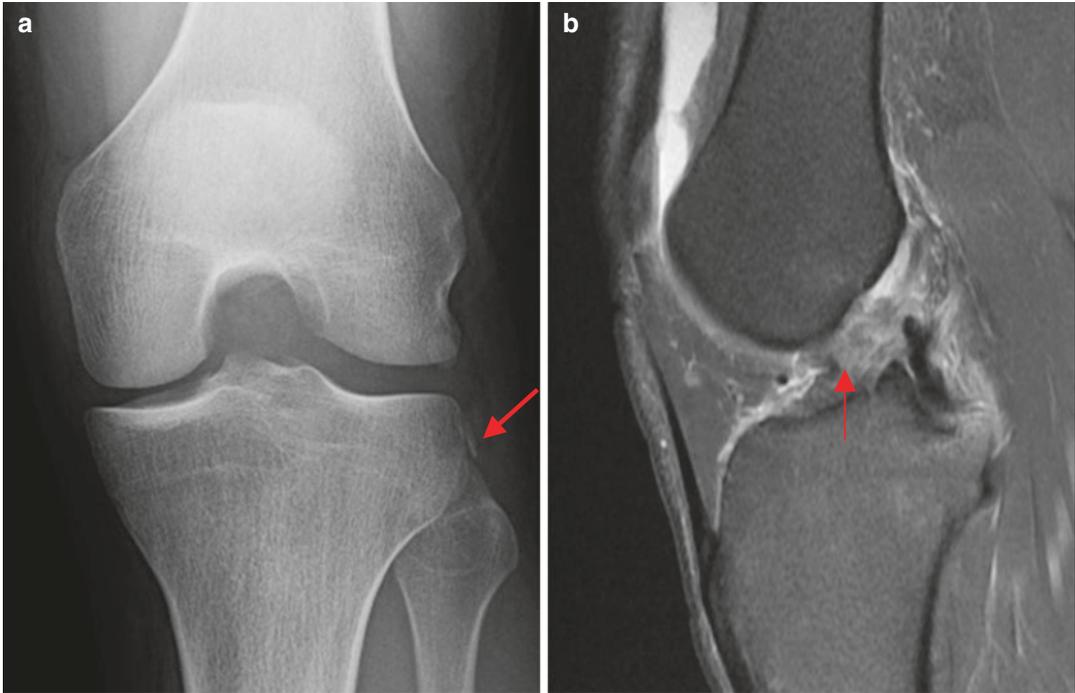


Fig. 30.1 (a) AP X-ray of patient with acutely ruptured ACL. Second fracture indicated by red arrow. (b) MRI demonstrating acute ACL rupture

tear. Bone bruising occurs on the central lateral femoral condyle and the posterior lateral tibial plateau [24]. With chronic ACL tears, the posterior horn of the medial meniscus becomes a more substantial restraint to anterior translation which may subsequently result in tearing. The practitioner should also be aware of any cartilage injury which may need to be addressed during arthroscopy.

30.3 Management of ACL Injury in Athletes

30.3.1 Conservative Management

Although non-surgical management of ACL injuries can be employed in patients without high athletic demand, there is a limited role for non-surgical management of ACL tears in the basketball athlete. This is primarily due to the high translational forces placed on the knee while playing basketball and the requirement of cut-

ting and pivoting motions. Physical therapy prior to surgery, or “pre-hab,” is primarily focused on normalization of range of motion, elimination of effusion, and strengthening the secondary stabilizers of the knee.

30.3.2 Surgical Management: Graft Selection

Following preoperative rehabilitation and normalization of range of motion of the knee (usually 3–4 weeks), surgical management can be employed with a decreased risk of arthrofibrosis [25]. Primary repair of the ACL does not demonstrate adequate healing due to the intra-articular nature of the tendon; therefore, ligament reconstruction is necessary. Various options for ACL grafts exist including: bone–patellar tendon–bone (BTB), hamstring, and quadriceps autografts. Either soft tissue or bony ACL allograft options exist, which eliminate donor site morbidity and decrease operative time. Additionally, graft pres-

ervation and disease transmission are less of a concern with modern day harvesting techniques. However, allografts are less commonly utilized in basketball athletes as they have been found to have a higher re-rupture rate in the younger, more active, athletic population [26].

Autograft reconstruction continues to be the mainstay of treatment in the young athletic patient. BTB autografts utilize bone on each side of the tendon which incorporate more quickly into bony tunnels and have been shown by some to have lower re-rupture rates than hamstring grafts (Fig. 30.2). However, donor site morbidity can present in the form of anterior knee pain, and some studies have shown increased

late stage arthritis [27]. Hamstring grafts demonstrate increased tensile strength in the laboratory when quadrupled and have lower donor site morbidity, but have slower incorporation into the bony tunnels at the tendon–bone interface [28]. Quadriceps autografts are increasingly performed and provide a thicker soft tissue graft option than the hamstring. Patella fractures and quadriceps rupture are potential complications postoperatively that should be discussed. Ultimately, graft choice is a cumulative decision between the patient and the surgeon based on pertinent factors. Commonly in the high-level basketball player, BTB autograft is performed when possible.

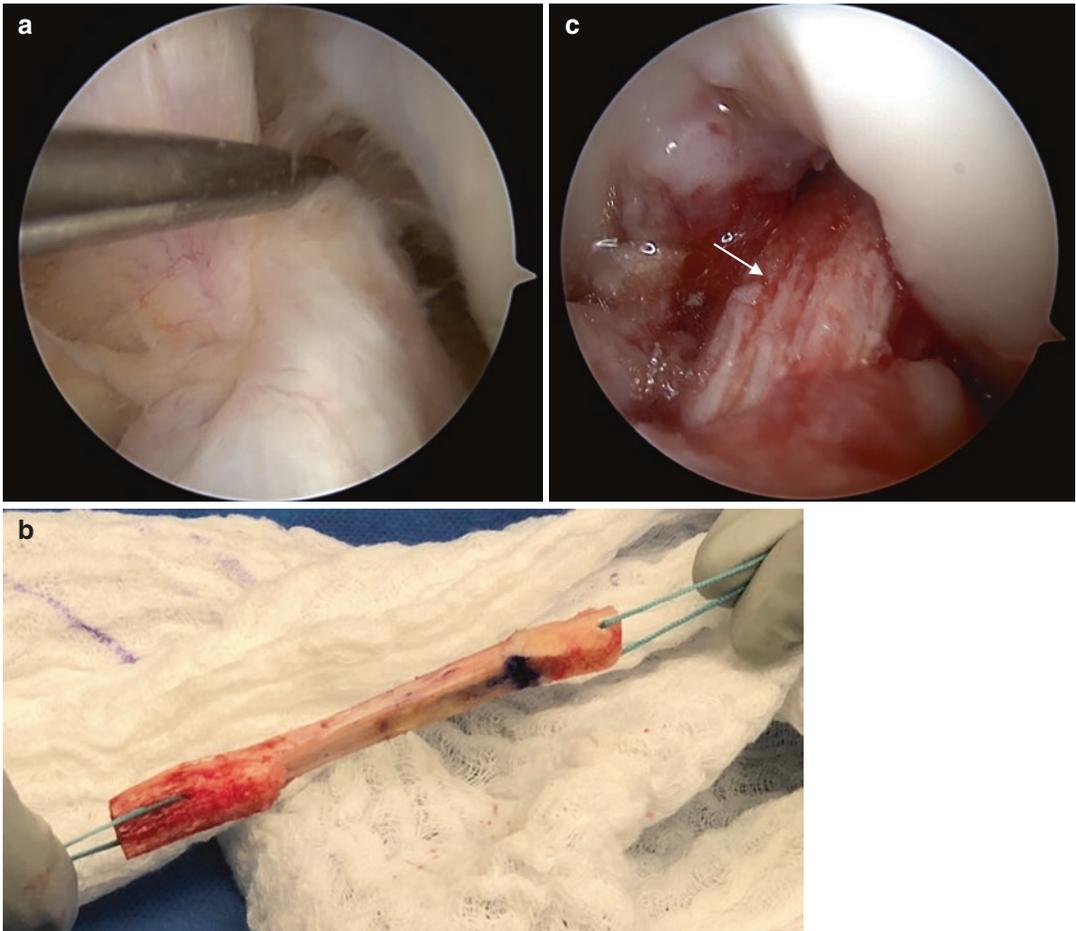


Fig. 30.2 ACL reconstruction with BTB autograft. (a) Arthroscopic view on an acute ACL tear. Detachment of the ACL from its insertion to the femur is demonstrated using a probe. (b) Prepared BTB autograft prior to reconstruction. (c) Reconstructed ACL with a BTB (white arrow)

30.3.3 Surgical Technique and Fixation

Several surgical methods exist when performing ACL reconstruction including trans-tibial drilling, anterior medial portal drilling, two-incision technique, and all-inside methods. Each technique entails pros and cons and is best managed by a physician experienced with the technique. Ultimately, literature has not demonstrated clinical differences between surgical techniques. Additionally, single-bundle or double-bundle ACL reconstruction can be performed.

The anatomic double-bundle technique uses two separate grafts to recreate each natural bundle of the ligament and is aimed at providing more natural knee motions. Although biomechanical studies have found superior results in terms of knee translation and rotation, this has not translated into superior clinical results [29, 30].

30.3.3.1 Fixation

Graft fixation of the reconstructed ligament should be strong enough to withstand close-chain exercises for at least 12 weeks until the bone or tendon is able to incorporate into the bone tunnels. There is a risk of graft slippage or ultimate failure if graft fixation is poor [31]. Many commercially available graft fixation options exist. Patellar bone blocks demonstrate the highest stiffness and fixation strength with interference screw fixation [32]. Optimal screw placement is parallel to the bone block, with screw divergence more than 30° demonstrating increased risk of pullout and failure [32]. Screw length and diameter are also factors which influence fixation strength, whereas composition of the screw (bio-absorbable/metallic) and the use of dilators has not been shown to affect fixation strength [33, 34]. Soft tissue grafts have several options for fixation. These include interference screws, screw and washer constructs, suture posts, tibial staples, and cross-pins and buttons on the femoral side. Screw and washer constructs, cross-pins, and buttons all provide indirect fixation, meaning the graft is suspended in the bony tunnel. All other options provide direct fixation by compressing the graft against the wall of the tunnel.

Fact Box: ACL Injury Management

- For athletes wishing to return to prior level of sport after ACL injury, surgical reconstruction remains the mainstay of treatment.
- Graft selection remains individualized, with most high-level athletes undergoing reconstruction with BTB autografts.
- Proper surgical fixation is critical to surgical outcome, with strongest fixation utilizing patellar bone blocks with screw placement parallel to the blocks.

30.4 Principles of ACL Injury Rehabilitation

Post ACL reconstruction, rehabilitation progression and duration recommendations vary widely from provider to provider. While some providers utilize defined time-points from surgery as milestones to advance rehabilitation, others require patients to complete both objective and subjective benchmarks before advancing. For any athlete, the ultimate goal of a rehabilitation program is to return to sport at pre-injury level, but the optimal therapy program and duration remain highly individualized and dependent on the athlete's level of play, type of sport, and concomitant injuries sustained at the time of ACL rupture. However, based on a systematic review of the current literature on ACL rehabilitation, an evidence-based clinical practice guideline has been created for recommendations [35].

30.4.1 Preoperative Rehabilitation (Pre-Hab)

Prior to undergoing surgery for an ACL tear, there are several milestones that should be achieved. Preoperative rehabilitation focuses on elimination of effusion, normalization of range of motion, and strengthening of the secondary stabilizers of the knee. Preoperative stiffness has been found to closely correlate with postopera-

tive range of motion loss, and therefore obtaining near normal range of motion prior to surgery is essential [36]. Quadriceps weakness/atrophy is another preoperative concern, as quadriceps strength deficits >20% have been identified as a predictor for persistent weakness following surgery [37]. Strengthening of the quadriceps prior to surgical intervention can potentially alleviate this complication.

30.4.2 Postoperative Rehabilitation

30.4.2.1 Initial Phase

Following surgery, athletes are progressed through a structured physical therapy regimen before returning to their sport. Rehabilitation consists of three advancing stages of therapy. As quadriceps activation failure and flexion contracture can occur postoperatively, the initial phase of rehabilitation is centered on quadriceps activation and obtaining full range of motion [38]. Knee extension exercises are used to obtain full extension to 0° by 2–4 weeks. Flexion to 120–130° is obtained by 4–6 weeks using heel-slide exercises. Focus should also be placed on passive mobilization of the patella to ensure good patellofemoral mobility by 4–6 weeks after surgery.

Strength training should focus on quadriceps reactivation with active straight leg knee extensions. Gradually, patients should progress from isometric to concentric quadriceps exercises as tolerated once the quadriceps are reactivated. When the patient has sufficient quadriceps strength, as evidenced by performance of straight leg raises without a lag, they can unlock their postoperative brace and begin advancing their strength training [39]. As strength returns, patients can start progressive weight-bearing until they resume a normal gait pattern, after which crutches can be discontinued. Thereafter, patients should progress to closed kinetic chain exercises (including leg press, squats, step-ups) prior to initiating open kinetic chain exercises approximately 4–6 weeks after surgery [35]. Early precautions should include both closed and open kinetic chain exercises confined to a limited

range of motion, with gradual increases in range of motion during exercise as tolerated.

Another important aspect of early rehabilitation is neuromuscular training, as impaired neuromuscular control can often persist despite recovery of normal strength levels [40]. Training with balance-boards and emphasizing proper mechanics and form during exercises and ambulation are useful adjuncts in regaining neuromuscular control.

30.4.2.2 Intermediate Phase

Once the goals of the immediate postsurgical period have been met—including wound healing, resolution of effusion, voluntary control of quadriceps, restoration of normal gait, knee extension to at least 0°, and knee flexion to at least 120–130°—athletes may advance to the second phase of therapy. The time period to reach these criteria varies but should be considered abnormal if not obtained within 6–8 weeks following surgery.

Many of the principles of this phase follow progressions of the neuromuscular, range of motion, and strength exercises of the initial phase. Neuromuscular training should be continued with increased difficulty, such as transitioning from static to dynamic training and adding perturbation training with varying speed, direction, and amplitude of disturbance. Strength training should gradually progress to closed and open kinetic chain exercises at all ranges of motion. At week 10–12, athletes can resume jogging, assuming they are able to do so with correct form. Sport-specific training can also be initiated at this time with close supervision.

30.4.2.3 Final Phase

Once athletes are able to perform all closed and open kinetic chain exercises without pain and have a Limb Symmetry Index (LSI) greater than 80% for quadriceps and hamstring strength, they can progress to the final phase of rehabilitation before returning to sport. The goal of this phase is to transition back into sports by intensifying sport-specific training. Neuromuscular and perturbation exercises should be intensified and focused on sport-specific movement. Athletes should also increase agility training, with resumption of train-

ing with their sports team in preparation for returning to sports (RTS) and return to play (RTP).

30.4.3 Rehabilitation Considerations in Basketball Players

Much of the investigation in ACL reconstruction rehabilitation has focused on general principles and guidelines for all patients, with few studies focusing directly on rehabilitation in basketball players. One level V commentary published on rehabilitation in basketball players by an NBA trainer [41] outlined a similar three phase progression with early focus on minimizing effusion, allowing for incision closure, and gradual restoration of knee range of motion. In addition to the general intermediate phase exercises outlined above, the author proposed several basketball-specific exercises, including low-intensity cutting drills and single-leg squatting on tilt boards while catching and passing basketballs. With progression to the last rehabilitation phase prior to returning to sport, drills simulating basketball situations can be implemented. This includes ball chase drills, where players chase balls thrown randomly around the court, and sprint-backpedal drills with and without basketball dribbling.

In addition, rehabilitation in basketball players should also emphasize jump-landing training and plyometric-type exercises, focusing on training to improve basketball-specific motions with the potential for ACL injury. The goal of completing these tailored basketball exercises is to progressively expose recovering athletes to in-game movements and scenarios, thus optimizing the neuromuscular and strength training unique to basketball players. Further study is needed to investigate basketball-specific programs before evidence-based recommendations can be made.

30.4.4 Establishing Safe Return-to-Play Criteria Following Rehabilitation

Standardized, objective, and evidence-based criteria for return to sport in basketball, as in all

sports, have yet to be established. A recent systematic review of level I and II studies reporting return-to-play protocols demonstrated that 90% failed to utilize objection criteria and 65% of studies failed to use any criteria for return to sports readiness [42]. There are several obstacles in obtaining unified return-to-play criteria. One barrier is a lack of consensus on what level an athlete can safely return after ACL reconstruction [43]. Another obstacle is that multiple factors contribute to athlete RTP that cannot be controlled uniformly. One such factor is the psychological state of the athlete. Reports have shown that psychological factors can significantly decrease return-to-sport rate and performance despite obtaining equivalent functional outcome measures in athletes [44]. Additionally outcome scores do not necessarily correlate with return-to-sports rates. A recent study evaluating ACL reconstruction outcomes demonstrated that only 44% of athletes RTP despite 90% of the same athletes having normal or near-normal function on objective outcome scores and 85% having normal or near-normal function on IKDC subjective outcome scores [45].

Although limitations to uniform return-to-play criteria exist as detailed above, a recent review on rehabilitation following ACL reconstruction introduced several evidence-based criteria for establishing safe return to play [35]. Before a basketball player can safely RTS, at minimum the following criteria should be met:

- No knee pain with basketball-related movements (running, jumping, pivoting, decelerating, cutting).
- No knee buckling or apprehension of buckling during basketball-related movements.
- Restoration of normal and symmetric gait and running pattern.
- An LSI >90% for both hamstring and quadriceps strength.
- An LSI >90% for a hop test battery (including vertical jump, hopping for distance, single-leg hop-and-hold test, and side hopping) [46].
- Drop jump testing with absence of valgus movement, symmetric knee flexion, and maintenance of upright and lateral truncal posture.

Fact Box: Rehabilitation

- Preoperative rehabilitation is essential in optimizing postoperative restoration of range of motion and strength.
- Postoperative rehabilitation should include range of motion, strength, and neuromuscular exercises with progression to sport-specific exercises.
- Return-to-sport criteria are evolving but should ensure symmetric quadriceps and hamstring strength and absence of pain or buckling with sport-specific movements.

30.5 Outcomes of ACL Reconstruction

30.5.1 Functional Outcomes of ACL Reconstruction

Long-term results of ACL reconstruction have been investigated. At the 20-year follow-up, intact hamstring autografts in both adults and adolescents had favorable subjective outcome scores and return to play rate. However, ACL re-injury rates were significant, particularly in adolescents, with survival rates of 86% and 61% for adults and adolescents, respectively. Moreover, in final follow-up 17% of subjects had radiographic evidence of moderate to severe osteoarthritis based on IKDC radiological grade [47]. A longitudinal evaluation of patellar tendon autografts at mean 10.3 years found similar results, with favorable subjective outcomes, graft survival rates, and common progression of osteoarthritis [48]. A recent study comparing outcomes of hamstring and BTB reconstruction at mean 9 years corroborated the short-term findings, showing insignificant differences in laxity and graft failure. However, patellar tendon autografts were associated with increased rates of arthritis and pain with kneeling.

30.5.2 Repeat Tears of the ACL

A well-described and devastating outcome following successful ACL reconstruction and rehabilitation is an ACL re-tear. Incidence of ipsilateral

graft tear in the reconstructed knee and tearing of the contralateral native ACL are comparable in the first 2 years after ACL reconstruction, occurring in approximately 3% of patients [49]. At 5 years, incidence of tears of the contralateral ACL is significantly higher than that of reconstructed ACLs, with rates of 11.8% and 5.8%, respectively [50]. Several risk factors for re-tear have been identified, including the use of an allograft (5.2× more likely to tear than autografts), younger age, and higher activity levels. There were no significant differences when considering sex, sport played, concurrent meniscus tears, or autograft type [51]. In addition to needing to repeat the significant time and effort in post-surgical rehabilitation, re-tears of the ACL are particularly devastating as they have been shown to have inferior outcomes to primary ACL reconstruction. In particular, patients with repeat ACL ruptures have lower activity levels following rehabilitation, higher incidence of cartilage injury in the medial and patellofemoral compartments of the ipsilateral knee, and higher rates of subsequent recurrent nontraumatic graft injury [52].

30.5.3 Return to Sport Following ACL Reconstruction

With a high number of athletes playing basketball and across all sports opting for ACL reconstruction, the time and success rate of returning to sport have been significantly investigated in recent years. A recent systematic review of ACL reconstruction outcomes analyzed 69 published reports on return rates across many different sports. The investigation found that 81% of athletes were able to return to sport, but only 65% returned to pre-injury level of play, and only 55% were able to return to competitive play [53]. A number of factors associated with favorable outcomes were identified, including younger age, male gender, and a positive psychological response after injury. Interestingly, ACL reconstruction with a hamstring tendon autograft was associated with higher rates of return to competitive sport, whereas repair with a patellar tendon autograft was associated with higher rates of return to pre-injury performance levels [53].

While rates of return to pre-injury sports performance following ACL injury and reconstruction in general have been low, rates among elite and professional athletes have been more favorable. A recent systematic review on return to sport outcomes in elite athletes found a pooled return to sport rate of 83% across several sports with a mean return to sports time ranging from 6 to 13 months [54]. Factors that were associated with higher return to sport rates at pre-injury level included measures suggestive of greater levels of skill and value to elite teams, such as earlier draft selection, a collegiate scholarship, and a higher depth chart position. One study found concomitant meniscus injury to shorten careers of hockey players undergoing ACL reconstruction, but otherwise, no concurrent injuries significantly affected return-to-sport rates across all sports. Similarly, one study found that elite athletes undergoing ACL reconstruction with autografts was associated with higher return to sport rates, but otherwise no investigations iden-

tified significant relationships between graft selection and return to sport rates [54].

The ability to return to sport specifically in elite basketball players after ACL tears and reconstruction has also been investigated. A recent case series and systematic review of professional basketball players undergoing ACL reconstruction found that 11 out of 12 players returned to their prior level of play. Of those in the NBA, eight out of nine returned to play at a mean 9.8 months, with average per game statistical decreases in points, minutes, rebounds, assists, steals, blocks, and turnovers, none of which reaching statistical significance. There was a significant decrease in player efficiency in the first season following reconstruction, but by the second season, performance metrics had all returned to pre-injury levels [55]. Similar findings (Table 30.1) have been identified in other investigations of professional players, with reported return-to-sport rates of 78–86% and similar declines in functional performance upon initial return to sport.

Table 30.1 Investigations comparing return-to-sport rates and performance after ACL reconstruction in elite basketball players

Author (year)	Number of patients	%RTS	Performance compared with pre-injury	Performance compared with control group
Nwachukwu et al. (2017)	12	92	Decrease in player efficiency rating in year 1, returning to pre-injury level in year 2. Insignificant decreases in multiple individual statistics	–
Mehran et al. (2016)	21	–	—	No significant difference
Kester et al. (2016)	79	86	—	Decreases in games started, games played, and player efficiency rating. Mean length of postoperative play is 1.86 years shorter after ACL reconstruction
Minhas et al. (2016)	65	85	Statistically significant decreases in games played for 3 years. Decrease in player efficiency rating at 1 year before returning to pre-injury level at 3 years	–
Harris et al. (2013)	58	86	Decreases in games played per season, points, rebounds, field goal percentage, All-Star selections	Decrease in games played
Namdari et al. (2011)	18	78	Decreases in steals per game and shooting percentage. Insignificant decreases in multiple other individual statistics	No significant difference
Busfield et al. (2009)	27	78	Decreases in player efficiency rating, games played, shooting percentage. Insignificant decreases in multiple other individual statistics	No significant difference

Fact Box: Outcomes

- Both hamstring and BTB autografts provide superior functional outcomes to allografts and should be utilized for young athletes seeking to RTP.
- ACL graft re-tear and contralateral ACL ruptures occur at low rates following ACL reconstruction, particularly in young and active patients.
- RTS rates vary, with more favorable outcomes and return to prior level of play in elite athletes as compared with recreational players.

30.6 Conclusion

ACL rupture remains a common and devastating injury in basketball players, nearly always requiring surgical intervention and lengthy rehabilitation before RTP. Diagnosis should be obtained clinically with a detailed history and physical examination and confirmed with radiographic studies prior to assessing management. Surgical reconstruction with either hamstring or BTB autografts should strongly be considered in any player wishing to return to basketball following an ACL rupture, along with immediate preoperative rehabilitation. Postoperative rehabilitation should follow performance-based progression in range of motion, strengthening, and neuromuscular exercises before initiating gradual return to basketball activity. The success of these interventions in returning players to basketball varies based upon both modifiable and non-modifiable factors, but generally these measures are successful in returning players to their prior level of play.

References

1. Sanders TL, Maradit Kremers H, Bryan AJ, et al. Incidence of anterior cruciate ligament tears and reconstruction: a 21-year population-based study. *Am J Sports Med.* 2016;44(6):1502–7.
2. Frobell RB, Lohmander LS, Roos HP. Acute rotational trauma to the knee: poor agreement between

- clinical assessment and magnetic resonance imaging findings. *Scand J Med Sci Sports.* 2007;17(2):109–14.
3. Rishiraj N, Taunton JE, Lloyd-Smith R, Woollard R, Regan W, Clement DB. The potential role of prophylactic/functional knee bracing in preventing knee ligament injury. *Sports Med (Auckland, NZ).* 2009;39(11):937–60.
 4. Deitch JR, Starkey C, Walters SL, Moseley JB. Injury risk in professional basketball players: a comparison of Women's National Basketball Association and National Basketball Association athletes. *Am J Sports Med.* 2006;34(7):1077–83.
 5. Mall NA, Abrams GD, Azar FM, Traina SM, Allen A, Parker R. Trends in primary and revision anterior cruciate ligament reconstruction among National Basketball Association team physicians. *Am J Orthop.* 2014;43(06):267–71.
 6. Mihata LC, Beutler AI, Boden BP. Comparing the incidence of anterior cruciate ligament injury in collegiate lacrosse, soccer, and basketball players: implications for anterior cruciate ligament mechanism and prevention. *Am J Sports Med.* 2006;34(6):899–904.
 7. Musahl V, Bedi A, Citak M, O'Loughlin P, Choi D, Pearle AD. Effect of single-bundle and double-bundle anterior cruciate ligament reconstructions on pivot-shift kinematics in anterior cruciate ligament- and meniscus-deficient knees. *Am J Sports Med.* 2011;39(2):289–95.
 8. Tashiro Y, Okazaki K, Miura H, et al. Quantitative assessment of rotatory instability after anterior cruciate ligament reconstruction. *Am J Sports Med.* 2009;37(5):909–16.
 9. Mall NA, Chalmers PN, Moric M, et al. Incidence and trends of anterior cruciate ligament reconstruction in the United States. *Am J Sports Med.* 2014;42(10):2363–70.
 10. Prodromos CC, Han Y, Rogowski J, Joyce B, Shi K. A meta-analysis of the incidence of anterior cruciate ligament tears as a function of gender, sport, and a knee injury-reduction regimen. *Arthroscopy.* 2007;23(12):1320–5.e1326.
 11. Cole BJ, Erlund LS, Fu FH. *Soft tissue problems of the knee. Orthopaedic surgery: The essentials.* New York: Thieme Medical; 1999. p. 551–60.
 12. Moglo K, Shirazi-Adl A. Cruciate coupling and screw-home mechanism in passive knee joint during extension–flexion. *J Biomech.* 2005;38(5):1075–83.
 13. Odensten M, Gillquist J. Functional anatomy of the anterior cruciate ligament and a rationale for reconstruction. *J Bone Joint Surg Am.* 1985;67(2):257–62.
 14. Lord BR, El-Daou H, Zdanowicz U, Śmigielski R, Amis AA. The role of fibers within the tibial attachment of the anterior cruciate ligament in restraining tibial displacement. *Arthroscopy.* 2019;35(7):2101–11.
 15. Zantop T, Wellmann M, Fu FH, Petersen W. Tunnel positioning of anteromedial and posterolateral bundles in anatomic anterior cruciate ligament reconstruction: anatomic and radiographic findings. *Am J Sports Med.* 2008;36(1):65–72.

16. Noyes F, Butler D, Grood E, Zernicke R, Hefzy M. Biomechanical analysis of human ligament grafts used in knee-ligament. *J Bone Joint Surg Am.* 1984;66:344–52.
17. Woo SL-Y, Hollis JM, Adams DJ, Lyon RM, Takai S. Tensile properties of the human femur-anterior cruciate ligament-tibia complex: the effects of specimen age and orientation. *Am J Sports Med.* 1991;19(3):217–25.
18. Arnoczky SP. Anatomy of the anterior cruciate ligament. *Clin Orthop Relat Res.* 1983;172:19–25.
19. Toy BJ, Yeasting RA, Morse DE, McCann P. Arterial supply to the human anterior cruciate ligament. *J Athl Train.* 1995;30(2):149.
20. Krosshaug T, Nakamae A, Boden BP, et al. Mechanisms of anterior cruciate ligament injury in basketball: video analysis of 39 cases. *Am J Sports Med.* 2007;35(3):359–67.
21. Boden BP, Dean GS, Feagin JA Jr, Garrett WE Jr. Mechanisms of anterior cruciate ligament injury. *Orthopedics.* 2000;23(6):573–8.
22. Benjaminse A, Gokeler A, van der Schans CP. Clinical diagnosis of an anterior cruciate ligament rupture: a meta-analysis. *J Orthop Sports Phys Ther.* 2006;36(5):267–88.
23. Goldstein J, Bosco JA 3rd. The ACL-deficient knee: natural history and treatment options. *Bull Hosp Jt Dis.* 2001;60(3–4):173–8.
24. Graf BK, Cook DA, De Smet AA, Keene JS. “Bone bruises” on magnetic resonance imaging evaluation of anterior cruciate ligament injuries. *Am J Sports Med.* 1993;21(2):220–3.
25. Johnson RJ, Beynon BD, Nichols CE, Renstrom PA. The treatment of injuries of the anterior cruciate ligament. *J Bone Joint Surg Am.* 1992;74(1):140–51.
26. Borchers JR, Pedroza A, Kaeding C. Activity level and graft type as risk factors for anterior cruciate ligament graft failure: a case-control study. *Am J Sports Med.* 2009;37(12):2362–7.
27. Hospodar SJ, Miller MD. Controversies in ACL reconstruction: bone-patellar tendon-bone anterior cruciate ligament reconstruction remains the gold standard. *Sports Med Arthrosc Rev.* 2009;17(4):242–6.
28. Foster TE, Wolfe BL, Ryan S, Silvestri L, Kaye EK. Does the graft source really matter in the outcome of patients undergoing anterior cruciate ligament reconstruction? An evaluation of autograft versus allograft reconstruction results: a systematic review. *Am J Sports Med.* 2010;38(1):189–99.
29. Maeyama A, Hoshino Y, Kato Y, et al. Anatomic double bundle ACL reconstruction outperforms any types of single bundle ACL reconstructions in controlling dynamic rotational laxity. *Knee Surg Sports Traumatol Arthrosc.* 2018;26(5):1414–9.
30. Mayr HO, Bruder S, Hube R, Bernstein A, Suedkamp NP, Stoehr A. Single-bundle versus double-bundle anterior cruciate ligament reconstruction-5-year results. *Arthroscopy.* 2018;34(9):2647–53.
31. Frank CB, Jackson DW. The science of reconstruction of the anterior cruciate ligament. *J Bone Joint Surg Am.* 1997;79(10):1556–76.
32. Lemos MJ, Jackson DW, Lee TQ, Simon TM. Assessment of initial fixation of endoscopic interference femoral screws with divergent and parallel placement. *Arthroscopy.* 1995;11(1):37–41.
33. Kousa P, Jarvinen TL, Vihavainen M, Kannus P, Jarvinen M. The fixation strength of six hamstring tendon graft fixation devices in anterior cruciate ligament reconstruction. Part I: femoral site. *Am J Sports Med.* 2003;31(2):174–81.
34. Nurmi JT, Jarvinen TL, Kannus P, Sievanen H, Toukosalo J, Jarvinen M. Compaction versus extraction drilling for fixation of the hamstring tendon graft in anterior cruciate ligament reconstruction. *Am J Sports Med.* 2002;30(2):167–73.
35. van Melick N, van Cingel RE, Brooijmans F, et al. Evidence-based clinical practice update: practice guidelines for anterior cruciate ligament rehabilitation based on a systematic review and multidisciplinary consensus. *Br J Sports Med.* 2016;50(24):1506–15.
36. Quelard B, Sonnery-Cottet B, Zayni R, Ogassawara R, Prost T, Chambat P. Preoperative factors correlating with prolonged range of motion deficit after anterior cruciate ligament reconstruction. *Am J Sports Med.* 2010;38(10):2034–9.
37. Eitzen I, Holm I, Risberg MA. Preoperative quadriceps strength is a significant predictor of knee function two years after anterior cruciate ligament reconstruction. *Br J Sports Med.* 2009;43(5):371–6.
38. Hart JM, Pietrosimone B, Hertel J, Ingersoll CD. Quadriceps activation following knee injuries: a systematic review. *J Athl Train.* 2010;45(1):87–97.
39. Cavanaugh JT, Powers M. ACL rehabilitation progression: where are we now? *Curr Rev Musculoskeletal Med.* 2017;10(3):289–96.
40. Stasi SD, Myer GD, Hewett TE. Neuromuscular training to target deficits associated with second anterior cruciate ligament injury. *J Orthop Sports Phys Ther.* 2013;43(11):777. A711.
41. Waters E. Suggestions from the field for return to sports participation following anterior cruciate ligament reconstruction: basketball. *J Orthop Sports Phys Ther.* 2012;42(4):326–36.
42. Harris JD, Abrams GD, Bach BR, et al. Return to sport after ACL reconstruction. *Orthopedics.* 2014;37(2):e103–8.
43. Ellman MB, Sherman SL, Forsythe B, LaPrade RF, Cole BJ, Bach BR Jr. Return to play following anterior cruciate ligament reconstruction. *J Am Acad Orthop Surg.* 2015;23(5):283–96.
44. Gobbi A, Francisco R. Factors affecting return to sports after anterior cruciate ligament reconstruction with patellar tendon and hamstring graft: a prospective clinical investigation. *Knee Surg Sports Traumatol Arthrosc.* 2006;14(10):1021–8.
45. Ardern CL, Webster KE, Taylor NF, Feller JA. Return to sport following anterior cruciate ligament reconstruction surgery: a systematic review and meta-

- analysis of the state of play. *Br J Sports Med.* 2011;45(7):596–606.
46. Thomee R, Neeter C, Gustavsson A, et al. Variability in leg muscle power and hop performance after anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc.* 2012;20(6):1143–51.
 47. Salmon LJ, Heath E, Akrawi H, Roe JP, Linklater J, Pinczewski LA. 20-Year outcomes of anterior cruciate ligament reconstruction with hamstring tendon autograft: the catastrophic effect of age and posterior Tibial slope. *Am J Sports Med.* 2018;46(3):531–43.
 48. Ahn JH, Kim JG, Wang JH, Jung CH, Lim HC. Long-term results of anterior cruciate ligament reconstruction using bone-patellar tendon-bone: an analysis of the factors affecting the development of osteoarthritis. *Arthroscopy.* 2012;28(8):1114–23.
 49. Wright RW, Dunn WR, Amendola A, et al. Risk of tearing the intact anterior cruciate ligament in the contralateral knee and rupturing the anterior cruciate ligament graft during the first 2 years after anterior cruciate ligament reconstruction: a prospective MOON cohort study. *Am J Sports Med.* 2007;35(7):1131–4.
 50. Wright RW, Magnussen RA, Dunn WR, Spindler KP. Ipsilateral graft and contralateral ACL rupture at five years or more following ACL reconstruction: a systematic review. *J Bone Joint Surg Am.* 2011;93(12):1159–65.
 51. Kaeding CC, Pedroza AD, Reinke EK, Huston LJ, Spindler KP. Risk factors and predictors of subsequent ACL injury in either knee after ACL reconstruction: prospective analysis of 2488 primary ACL reconstructions from the MOON Cohort. *Am J Sports Med.* 2015;43(7):1583–90.
 52. Chen JL, Allen CR, Stephens TE, et al. Differences in mechanisms of failure, intraoperative findings, and surgical characteristics between single- and multiple-revision ACL reconstructions: a MARS cohort study. *Am J Sports Med.* 2013;41(7):1571–8.
 53. Ardern CL, Taylor NF, Feller JA, Webster KE. Fifty-five per cent return to competitive sport following anterior cruciate ligament reconstruction surgery: an updated systematic review and meta-analysis including aspects of physical functioning and contextual factors. *Br J Sports Med.* 2014;48(21):1543.
 54. Lai CCH, Ardern CL, Feller JA, Webster KE. Eighty-three per cent of elite athletes return to preinjury sport after anterior cruciate ligament reconstruction: a systematic review with meta-analysis of return to sport rates, graft rupture rates and performance outcomes. *Br J Sports Med.* 2018;52(2):128.
 55. Nwachukwu BU, Anthony SG, Lin KM, Wang T, Altchek DW, Allen AA. Return to play and performance after anterior cruciate ligament reconstruction in the National Basketball Association: surgeon case series and literature review. *Phys Sportsmed.* 2017;45(3):303–8.



Management of Multi-Ligament Injuries in Basketball

31

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31.1 Introduction

Multi-ligament knee injuries (MLI) are complicated and devastating injuries for a basketball player [1]. The lower extremity has been reported to be the most frequently injured extremity in basketball, accounting for 62.4% of all injuries, with knee injuries accounting for 17.8% of all athletic injuries [2]. The incidence of anterior cruciate ligament (ACL) injuries has been reported to be higher in basketball relative to many other sports, with up to 16% of female basketball players likely to suffer an ACL tear during their career [3]. The incidence of MLI in basketball is not known; however, recent studies have reported that sports-related injuries can account for up to 47% of multi-ligament knee injuries in their cohorts [1, 4]. Accordingly, it is important to realize that even though multi-ligament injuries have been associated with high-energy mecha-

nisms, relatively lower-energy sports activities can also result in MLI [5]. Often, MLI in the basketball player occurs due to ligament failure during twisting and pivoting motions while landing, often in low-velocity or non-contact settings. Concurrent knee dislocation (bicusiate involvement) is associated with a high morbidity rate, given its association with potential neurovascular compromise [6].

The diagnostic evaluation of MLI in the basketball player must be thorough because concomitant intra-articular injuries are important to identify and address during surgical reconstruction. Surgical reconstruction is recommended for all athletes to improve knee function and potential to return to previous activity level. Surgical management of these injuries is technically challenging and different aspects of operative decision-making, such as reconstruction versus repair, staging, and ideal timing for surgical intervention, are still up for debate. The available literature reporting outcomes following multi-ligament knee injuries in athletes, and basketball players, specifically, is sparse.

In this chapter, we aim to address both the biomechanical and clinical considerations of multi-ligament knee injuries as they pertain to the basketball athlete by presenting the considerations of diagnosis, treatment, rehabilitation, and outcomes following surgical reconstruction and future options for management of this difficult injury.

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Table 31.1 Schenck's knee dislocation classification system [7, 8]

Classification	Anatomic pattern of injury
KD I	Injury to single cruciate + collaterals
KD II	Injury to ACL and PCL with intact collaterals
KD III M	Injury to ACL, PCL, MCL
KD III L	Injury to ACL, PCL, LCL
KD IV	Injury to ACL, PCL, MCL, and LCL
KD V	Dislocation + fracture

ACL anterior cruciate ligament, PCL posterior cruciate ligament, MCL medial collateral ligament, LCL lateral collateral ligament

The addition of the "C" or "N" modifier indicates concomitant arterial or neural injury, respectively. Dislocation implies rupture of both cruciate ligaments

31.1.1 Classification of Multi-Ligament Knee Injuries

The anatomical pattern of the involved ligaments as described by Schenck et al. [7, 8] is most commonly used when describing multi-ligament injuries (Table 31.1). This classification system is composed of six different patterns of MLI commonly observed and allows for modifiers that indicate whether or not an injury is associated with arterial and/or neural involvement.

31.1.2 Prevalence of Injury and Patient Demographics

Multi-ligament knee injuries in basketball are considered to be low-velocity traumatic events. It has been estimated that the incidence of MLI may be as high as 0.072 per 100-patient years [9]. Therefore, multi-ligament injuries are not as rare as previously thought, especially in the setting of rapid pivoting and low-energy impact activities as observed in basketball. In collegiate athletes, it has been reported that women have a higher susceptibility to ACL injury secondary to multiple extrinsic (body movement, muscular strength, shoe-surface interface, and skill level) and intrinsic (joint laxity, limb alignment, notch dimensions, and ligament size) causes, while intersex differences in posterior cruciate ligament (PCL) and collateral ligament injury rates have not been

reported [10]. This discrepancy appears to be potentiated in basketball. The rate of ACL injury in female basketball players is four times greater than in men ($P < 0.01$) with an increased incidence of meniscal tears ($P < 0.01$) and collateral ligament tears ($P < 0.02$) in female basketball players [10].

The most common overall pattern of MLI involves the medial side of the knee in conjunction with a cruciate ligament tear (KD III-M). Moatshe et al. [11] reported that the incidence of medial- and lateral-sided injuries was 52% and 28%, respectively, in their cohort of 303 multiligamentous injuries. Moreover, isolated bicruciate involvement constituted only 5% of multi-ligament injuries. Robertson et al. [12] also reported a 41% incidence of medial-sided injuries, while lateral-sided injuries accounted for 28% of involvement. Interestingly, Becker et al. [13] reported a high incidence of lateral-sided injuries, which composed 43% of injuries; however, the smaller series of 106 patients from a level-1 trauma center with predominantly high-energy trauma may have accounted for this discrepancy.

Few studies have provided quantitative estimates of the prevalence of sports-related MLI, in particular. In the previously cited study by Moatshe et al. [11], the authors reported that low-energy trauma accounted for 49.7% of knee dislocations as defined by bicruciate involvement, and 44% occurred during athletic participation. Likewise, Engebretsen et al. [4] quantified the prevalence of sports-related multi-ligament injury to be 47% in their cohort of 85 patients with knee dislocations. This suggests that sports-related multi-ligament injury occurs as frequently as those that occur secondary to high-impact energy scenarios, highlighting the importance of considering this pattern of injury when evaluating the knee of an injured basketball player. Collegiate basketball players may have specific injury patterns in comparison to other sports: Tan et al. [14] performed a retrospective cohort study of 2548 collegiate athletes and determined that basketball players were 4.95 times (95% confidence interval [CI], 2.61–8.76) more likely to experience an ACL tear than football players, suggesting

that injury patterns may reflect forces applied to the knee depending on commonly performed movements.

The importance of considering and properly diagnosing MLI in the basketball player should also be emphasized by the incidence of concomitant injuries, which may increase morbidity and return to sport time. Although specific data pertaining to basketball is lacking, the risk of vascular and neurologic injury is significant when a true high velocity knee dislocation occurs. Medina et al. [15] determined that the incidence of neural and vascular injury was 25% and 18%, respectively, in patients who experienced knee dislocation. Becker et al. [13] studied a series of 106 patients and reported a similar incidence of neural and vascular injury at 25% and 21%, respectively. Moatshe et al. [11] reported that vascular injuries occurred in 5% of dislocations, while common peroneal nerve injury occurred in 19%. Strikingly, the authors of this study also found that patients who had a posterolateral corner (PLC) injury had 42 times greater odds of experiencing a peroneal nerve injury and 9.2 times greater odds of experiencing a popliteal artery injury than patients without a PLC injury. Moreover, patients with peroneal nerve injury had 20.6 times greater odds of experiencing concomitant vascular injury. Together, these studies highlight the importance of evaluating for simultaneous neurovascular involvement when evaluating MLI, and the surgeon should not hesitate to pursue additional angiographic imaging in players who suffer associated peroneal nerve injury.

Other studies have reported that additional structural injury may occur up to 72% of the time [16]. Moatshe et al. [11] reported that the incidence of meniscal injury in conjunction with multi-ligament knee injury was 37.3%, while the incidence of concomitant cartilage injury was 28.3%. Krych et al. [17] reviewed a series of 122 knees and found that meniscal or chondral injury was present in 76% of cases. Richter et al. [18] found that meniscal injury was only present in 15% of cases of multi-ligament knee injury, suggesting a lower prevalence. Despite varying ranges of concomitant structural injury when

being evaluated for multi-ligament knee injuries, these studies suggest that surgeons should be cognizant of the possibility of players experiencing additional injury which may influence treatment approach and prognosis.

31.1.3 Diagnosis

Knee injuries suffered during recreational or competitive basketball are considered low energy, and as such there is usually a low concern for injury in other locations beyond the knee. A thorough and systematic history and physical examination is mandatory when treating patients with knee injuries. A video showing the injury can be helpful when available. Physical examination should be focused on swelling alignment, passive and active range of motion, and knee stability should be performed when possible. This examination may be limited by pain. Concomitant intra-articular injury, including meniscal and ligament injuries, can be missed in the acute period.

If a neurovascular injury is identified, it could significantly influence the treatment plan and prognosis. When assessing for vascular involvement, it is important to compare the injured limb to the contralateral side. The examiner should palpate the posterior tibial and dorsalis pedis pulses bilaterally in addition to assessing potential differences in skin color. It is also important to re-examine the patient in the acute period following injury because the vascular changes may not be immediately apparent. A more focused vascular examination may then proceed including capillary refill, pulse symmetry, and neurological exam as this has been reported to be reliable to screen patients for selective arteriography [19]. Vascular injury may also be assessed using the ankle-brachial index (ABI) when the physical exam is equivocal or there is concomitant neurological injury. An ABI <0.9 warrants angiography [20, 21]. If vascular injury is detected, this should guide first treatment steps.

The type of injury can often suggest potential neurovascular complications. In the set-

ting of bicruciate ligament disruption, the risk of vascular and neurologic injury is high, and vascular assessment is often needed [6]. Additionally, the examiner should properly assess the common peroneal nerve and vascular status when suspicious for a PLC injury, given the established association as previously discussed [11].

Diagnostic imaging also plays an essential role in the evaluation of multi-ligament injury. Stress radiographs play an important role in the diagnosis and classification of ligamentous injury, particularly in PCL, posteromedial corner (PMC), and PLC injuries. Moreover, they provide an objective outcome measure for successful reconstruction (Table 31.2, Figs. 31.1 and 31.2) [22]. If there is difficulty in obtaining the stress radiographs secondary to factors such as patient guarding [23–25], it is recommended to use a mini C-arm with examination under anesthesia during surgery. This approach may help identify the degree of knee gapping, if present. Magnetic resonance imaging (MRI) is also imperative as this provides information regarding concomitant potential injuries to the menisci and cartilage in addition to the ligaments of the knee. If a concurrent injury fails to be addressed, such as a collateral ligament injury in conjunction with cruciate ligament injury, then there is an increased risk of graft failure following cruciate ligament reconstruction due to increased force transmission to the graft [26, 27].

Table 31.2 Diagnostic criteria in PCL stress test, valgus stress test, and varus stress test

<i>PCL stress test</i>	
Side-to-side > 8 mm	Grade 3 PCL injury
Absolute difference > 11 mm	Combined PCL & PLC injury
<i>Valgus stress test</i>	
Side-to-side > 3 mm	Grade 3 MCL injury
Absolute difference > 9.8 mm	Posteromedial corner injury
<i>Varus stress test</i>	
Side-to-side > 2.7 mm	Grade 3 FCL injury
Absolute difference > 4 mm	Posterolateral corner injury

31.2 Treatment

Anatomic-based reconstruction is the gold standard for the treatment of multi-ligament knee injury [22, 28, 29]. Dedmond et al. [30] reviewed a series of 132 surgically treated MLI knees compared to 74 knees treated conservatively, reporting an increased range of motion (123° vs. 108°, $P < 0.001$) and higher Lysholm scores (85.2 vs. 66.5, $P < 0.001$) following surgical intervention. Various reconstructive techniques are available to address MLI (Fig. 31.3). Generally, it is preferred to reconstruct completely ruptured ligaments, while repair is preferred in instances of capsular avulsion or hamstring tendon avulsion [22, 31]. When possible, preference is given toward ACL reconstruction with bone–patellar tendon–bone (BTB) autograft; however, BTB allograft is used in cases of older age (>55 years) or patellar tendon compromise appreciated on MRI scan [32]. PCL tears are addressed with an anatomic double-bundle (DB) PCL reconstruction with an Achilles tendon allograft for the anterolateral bundle (ALB) and tibialis anterior allograft for the posteromedial bundle (PMB) [28]. MCL tears are reconstructed with an autograft hamstring reconstruction [33]. Fibular collateral ligament (FCL) tears are amenable to reconstruction with semitendinosus autograft or allograft, while a complete anatomic PCL reconstruction can be performed with a split Achilles tendon allograft.

The sequence of graft fixation depends on the involved ligament reconstruction. While outside the scope of this chapter, this is a heavily debated topic that is rapidly evolving as more outcome data becomes available [22]. Operative variables, such as repair versus reconstruction of the ligament, single versus two-stage surgery and timing of surgical intervention, are addressed in the following sections.

31.2.1 Operative Decision-Making: Repair Versus Reconstruction

Reconstruction of the injured ligaments with autografts and allografts has proven to be superior to repair of the injured intra-articular ligaments.

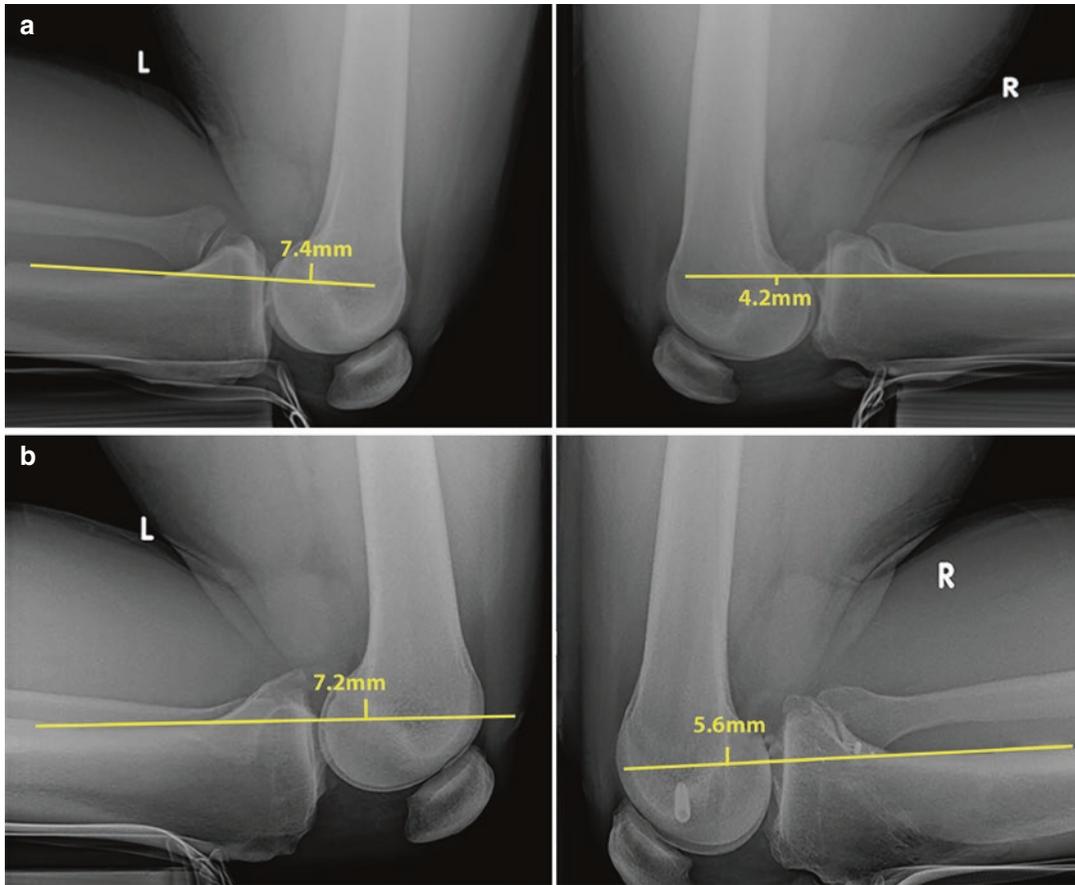


Fig. 31.1 Pre- and postoperative stress radiograph (kneeling) series following double-bundle PCL reconstruction, ACL reconstruction. (a) Preoperative side-to-side kneeling stress radiographs. (b) 12-month

postoperative side-to-side stress radiographs. The right knee demonstrates a side-to-side difference improvement of +1.4 mm 1 year postoperatively. PCL: posterior cruciate ligament, ACL: anterior cruciate ligament

Mariani et al. [36] retrospectively reported on outcomes in 11 knee dislocations treated with repair and 12 dislocations treated with reconstruction [36]. Stability and range of motion were improved with cruciate ligament reattachment compared with direct repair. Moreover, side-to-side total anteroposterior translation was greater following repair (6.67 mm vs. 3.20 mm), suggesting a less stable construct.

Levy et al. [37] reported a higher rate of failure in 10 knees that underwent repair of FCL and PLC with delayed cruciate ligament reconstruction (4/10, 40%) compared to only one failure in 18 knees (6%, $P = 0.04$) that underwent single-stage reconstruction. Stannard et al. evaluated 71 patients with 73 knee dislocations with posteromedial inju-

ries and reported a failure rate of 20% following repair compared to only 5% in the reconstruction group [38]. This contributes to the growing understanding that reconstruction of the collateral ligaments provides improved stability to the construct [37, 39]. A number of studies have demonstrated improved kinematic restoration and superior outcomes with anatomic reconstruction of injured ligaments [31, 40, 41]. Collateral ligament repair may be appropriate in care of bony avulsions, secondary restraints, and capsular injuries [42]. In the setting of multi-ligament knee injuries in basketball players, reconstruction is preferred because it provides the greatest stability, allows for early range of motion, and return to natural knee mechanical structure and function.

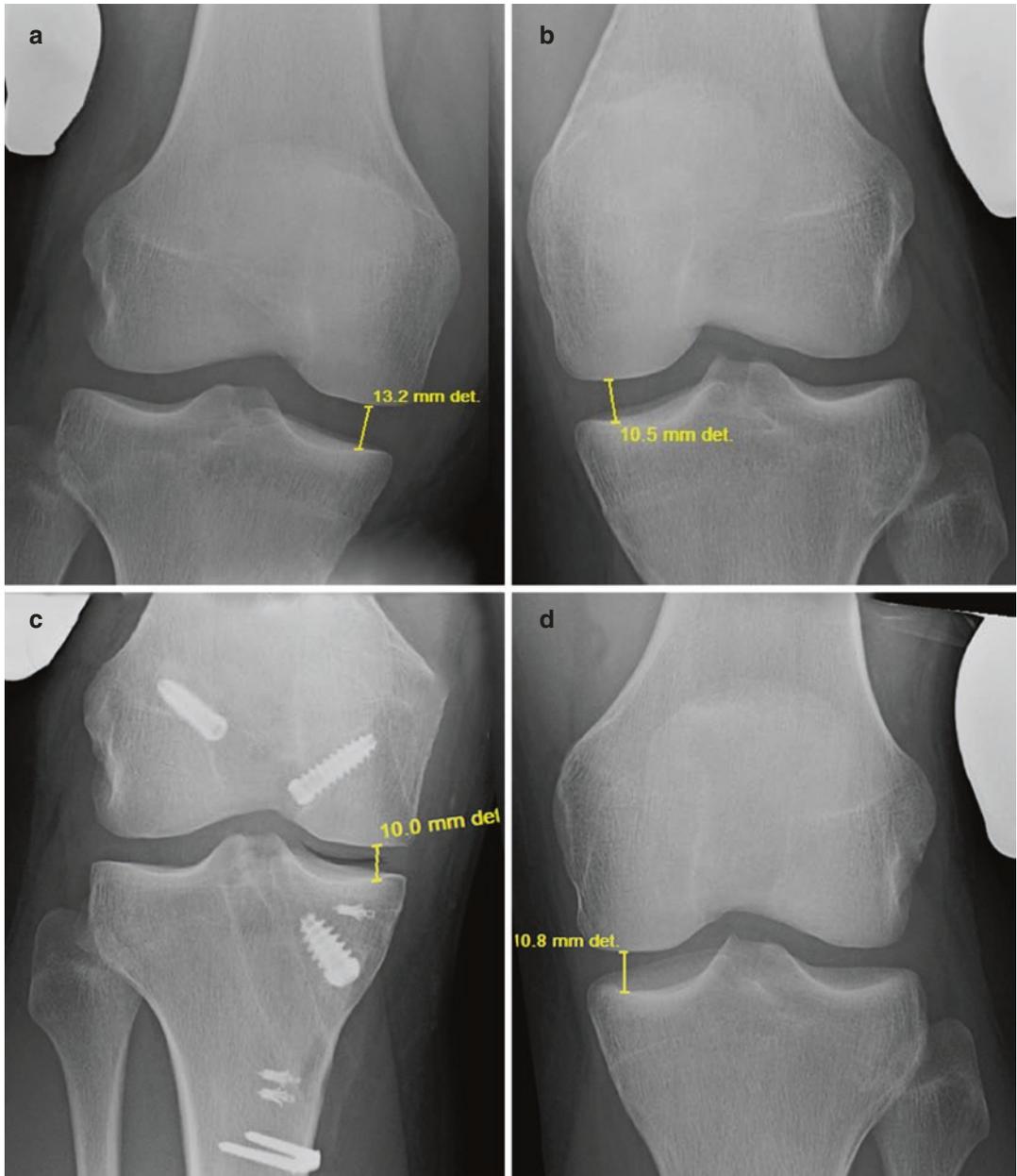


Fig. 31.2 Pre- and postoperative valgus stress radiograph series following a single-stage double-bundle PCL reconstruction, ACL reconstruction, FCL reconstruction, and a sMCL reconstruction. (a) Preoperative valgus stress radiograph of right knee. (b) Preoperative valgus stress radiograph of uninjured (left) knee. (c) 12-month postop-

erative valgus stress radiograph of right knee. (d) 12-month postoperative valgus stress radiographs of left knee. FCL: fibular collateral ligament, sMCL: superficial medial collateral ligament. Stress radiographs demonstrate <1 mm of side-to-side difference after reconstruction

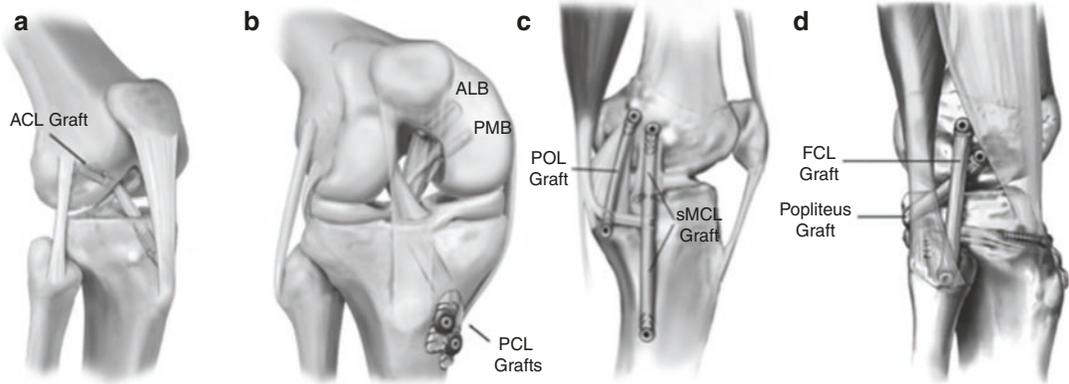


Fig. 31.3 Illustrations of reconstructive techniques utilized in multi-ligament knee reconstruction. (a) Anterolateral view of right knee showing single-bundle ACL reconstruction with a patellar tendon graft. (b) Illustration of double-bundle PCL reconstruction on a right knee using allografts for both the anterolateral and posteromedial bundle. The grafts are fixed on the tibia

using interference screws and washers (ALB: anterolateral bundle, PMB: posteromedial bundle) [34]. (c) Illustration showing a complete medial knee reconstruction (left knee) (POL: posterior oblique ligament; sMCL: superficial medial collateral ligament) [35]. (d) Illustration demonstrating a full posterolateral knee reconstruction (FCL: fibular collateral ligament) [22]

31.2.2 Operative Decision-Making: Single Stage Versus Two Stage

Single- versus multi-stage surgical fixation of the multi-ligament knee injury remains a topic of debate; however, the body of evidence is growing in support of single-stage reconstruction. Traditionally, single-stage reconstruction was criticized as it increased the same day surgery time and required a longer postoperative period of immobilization which led to an increased risk of arthrofibrosis due to delay in rehabilitation process [43–45]. LaPrade et al. [22] reported on a prospective cohort of 194 patients who sustained MLI while participating in sport and were treated with a single-stage ligament reconstruction followed by extensive rehabilitation emphasizing knee motion on postoperative day 1. The authors found that at a mean follow-up of 3.5 years, there was significant improvement in Lysholm (41–90) and WOMAC scores (44–3). The authors found no differences between PCL-based and ACL-based multiple ligament knee injuries. Moreover,

there was no significant difference in the rate of arthrofibrosis when comparing medial- and lateral-based multi-ligament injuries. Other studies have reported benefits of single-stage reconstruction with early rehabilitation, which include less joint instability in all directions, restoration of physiologic force transmission through the joint [26, 27] and greater percentage of patients who return to work [45]. Further studies are needed to elucidate the role of single-stage reconstruction in the basketball athlete, but this provides important insight into the benefit of biomechanically focused knee ligament reconstruction with immediate postoperative rehabilitation regardless of the injury pattern.

31.2.3 Operative Decision-Making: Timing of Surgery

The ideal time for operative intervention remains a topic of discussion when deciding the best treatment course for a basketball player with a multi-

ligament knee injury. A surgical approach within 3 weeks (if swelling has improved and 0–90° of flexion has been achieved) has been advocated as a way to help avoid arthrofibrosis, which has been reported to affect outcomes [4, 36, 43, 46]. Levy et al. performed a systematic review, demonstrating that acute surgical intervention following MLI, as defined as within 3 weeks of injury, is associated with higher mean Lysholm scores (90 vs. 82), higher IKDC scores (47% vs. 31%), and higher sports activity scores (89 vs. 69) on the Knee Outcome Survey [47]. Moatshe et al. [1] reported high risk of cartilage injury if patients are treated in the chronic phase; hence, early surgery is recommended. Alternatively, LaPrade et al. [22] recently found no significant differences in terms of postoperative outcome scores between acute and chronic multi-ligament knee injuries in athletes. Given the option, early surgical intervention is preferred by most providers as it allows for early rehabilitation [31]. In the setting of low-energy trauma, as seen in basketball injuries, early surgical reconstruction provides anatomic stability and rehabilitation potential.

Fact Box 1: Treatment Recommendations

- Ligament reconstruction is superior to repair.
- Single-stage surgery is superior to multistage surgery.
- Early surgical fixation (within 3 weeks) is superior to chronic fixation.

31.3 Rehabilitation

As the surgical treatment options for multi-ligament knee injury have improved, there is an increasing recognition of the importance of postoperative rehabilitation to achieve a successful outcome. Despite variation in individual protocols, there remain four common principles for rehabilitation (periodization concept) of MLI as outlined by Mueller et al. [48]: (1) Protect the surgical reconstruction and restore joint range of motion; (2) manage the scarring process; (3) minimize

muscular atrophy and restore preinjury levels of muscular strength; (4) utilize return to sport testing to guide decision-making. The criteria and timeline for progression through each phase in basketball players is highly variable and depends on the injured tissues, extent of injury, demographics of the patient, desired activity level, and surgeon preference, among other factors.

Fact Box 2: Rehabilitation Principles

- Tissue protection and range of motion
- Manage the scarring process
- Minimize muscular atrophy
- Utilize return to sport testing

In the first (tissue protection) phase, many protocols advocate for immediate active range of motion (ROM) to prevent residual stiffness, which has been reported to be the most common complication after a multi-ligament knee reconstruction [49, 50]. Full range of motion is important to long-term outcomes following surgery, and athletes should aim to reach 0–90° of flexion within the first 2 weeks after surgery. Patient-specific factors such as generalized joint hyperlaxity should be considered during this phase. Aggressive flexion training and muscle stretching should be avoided during the first 4 weeks postoperatively, and extension should be limited to neutral (avoiding hyperextension) for 6 weeks [50]. Patellofemoral mobilization in the nonrestricted range may be started immediately after surgery and should be the initial focus as hypomobility of the proximal pole of the patella can lead to extensor mechanism disruption and knee extensor dysfunction (Fig. 31.4).

There is variability in the literature in terms of weight-bearing after surgery for multi-ligament knee reconstruction. Many protocols call for a period of non-weight-bearing ranging from 4 to 6 weeks postoperatively to limit the load on the reconstructed ligaments and reduce the subsequent risk of graft elongation and joint laxity [48, 51–54]. Others recommend early partial weight-bearing [4, 55] or toe-touch weight-bearing



Fig. 31.4 Patellar mobility exercises initiated early after MLI reconstruction can help to prevent arthrofibrosis and preserve knee range of motion following surgery

[56, 57], while very few protocols recommend weight-bearing as tolerated [58].

While protection of the knee postoperatively is important to preserve the integrity of the repair and allow for healing, complex injuries and extensive surgeries are associated with high risk of developing knee joint stiffness, loss of motion, and arthrofibrosis [45]. Suprapatellar and anterior compartment arthrofibrosis have been shown to increase the patellofemoral contact force by up to 80 N, which can cause significant anterior knee pain [59]. The goal of the second phase of rehabilitation is to manage the scarring process to prevent joint stiffness, which can be debilitating in high-level basketball players. Some authors have advocated for early range of motion to prevent scar tissue formation [39, 60], and in a systematic review of clinical trials, Mook et al. [45] reported that delayed rehabilitation may lead to worse postoperative knee function.

To minimize muscular hypotrophy in the third phase of rehabilitation, patients may perform quadriceps-activating exercises during the tissue-protection phase with the knee in neutral extension and within safe ranges of flexion. Exercises aimed to prevent hypotrophy of the hamstring, calf and gluteal muscles include four-way straight leg raises, ankle pumps, and gluteal sets. Provided the patient does not have significant knee inflammation, the physical therapist may slowly increase the load on surgical tissues and monitor tissue response [50]. Advanced weight-bearing exercises should be performed in

the postoperative brace and can start when the patient has met all criteria for discontinuation of crutches, at least 6 weeks postoperatively [50].

The concept of periodization, first described by Hans Selye, is centered around the principle of dividing the rehabilitation program into smaller phases (periods) to promote increased physiological capacity and avoid exhaustion [61]. Adaptations to this program have been developed for specific injuries and athletes; however, all of these share a common progression through three phases: muscular endurance, strength, and power. The length of each phase should be no shorter than 6 weeks, with the endurance phase starting at approximately 8 weeks postoperatively, when ROM has been entirely restored [61]. Resisted exercises should be gradually introduced, and progress should be constantly re-evaluated to reduce the risk of reinjury, muscle imbalance, and knee inflammation.

Fact Box 3: Phases of Periodization

- Muscular endurance
- Muscular strength
- Muscular power

A treatment algorithm for safe return to basketball (Fig. 31.5) provides the therapist and athlete with objective qualitative and quantitative functional achievements that should be achieved prior to advancement to the next phase of rehabilitation. Muscle strength, power, and endurance are quantified by single hop (Fig. 31.6), squats (Fig. 31.7), box drop, quad index, and range of motion testing. These metrics can help to direct where attention is needed during treatment. Lunging exercises should be implemented cautiously in patients with PCL and PLC injuries as weight-bearing flexion beyond 45° disproportionately loads the PCL versus the ACL [62].

Cycling and treadmill exercises are incorporated once the patient has sufficient quadriceps strength and can walk independently without a brace or crutches. After this point, tissue-specific considerations are not relevant, and the patient can begin sports-specific exercises without

Treatment algorithm for safe return to play in basketball

	ROM	Gait	Physical Exam	Flexibility	Exercises	Lifting	Running	Jumping
Phase 1 Week 0-2	0-90° knee flexion Patellofemoral ROM	Non weight bearing						
Phase 1 Week 2-8	0-10° contralateral side	Protected weight bearing	Swelling within 1 cm of opposite knee					
Phase 3 Week 8-15 (Endurance phase)	Full, active ROM Equal contralateral side	Full weight bearing unassisted x20 mins	Quad index >80%	Anterior reach within 8 cm contralateral leg	10 rep leg press (2.5x body weight) single leg squat from 10' for 15 reps		walk-run program	DorsaVi-pass single leg squat
Phase 3 Week 15-21 (Strength phase)			Quad index: 90% Quad girth within 1 cm Hamstring/quad ratio >60%	Anterior reach within 4 cm contralateral leg	10 rep leg press (2.8x body weight)		Single plane speed and agility	DorsaVi-box drop
Phase 4 Week 21+ (Power phase)			Quadricep within 1 cm contralateral side	Anterior reach within 2 cm contralateral leg	10 rep leg press (3.1x body weight)		Multiplane speed and agility-T test within 90% contralateral side	DorsaVi-single leg vertical jump within 90% contralateral side
Return to sport (9-12 months) Week 21+ Power	Multidisciplinary decision between physician, athletic trainers, coaches Objective tests: Vail Sports Test/SANE/IKDC <5% difference from contralateral side							

Fig. 31.5 Treatment algorithm for safe return to play in basketball. The recovering athlete should meet each of the listed criteria before advancing to the next phase of rehabilitation

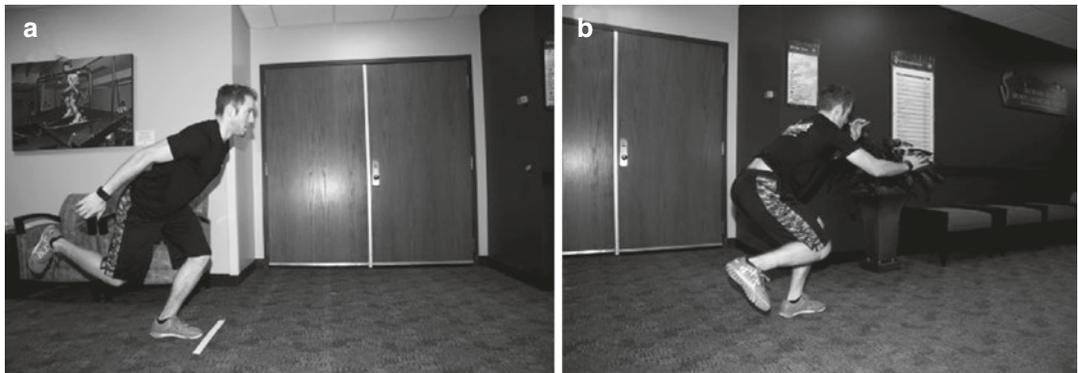


Fig. 31.6 Single hop is an exercise that requires extensive neuromuscular control and is an integral part of the rehabilitation protocol. In (a), the patient is seen eccentrically loading quadriceps, and (b) shows the end of the hop with the patient subsequently landing on the foot that he used to initiate jump

ally loading quadriceps, and (b) shows the end of the hop with the patient subsequently landing on the foot that he used to initiate jump

restrictions of tissue protection in the final rehabilitation phase. For basketball players, lateral movement, jumping, and pivoting exercises should be incorporated to the rehabilitation pro-

gram in a gradual and stepwise manner. In general, these drills should first address single-plane agility with a progression to multiplane agility. The decision to allow an athlete to return to bas-

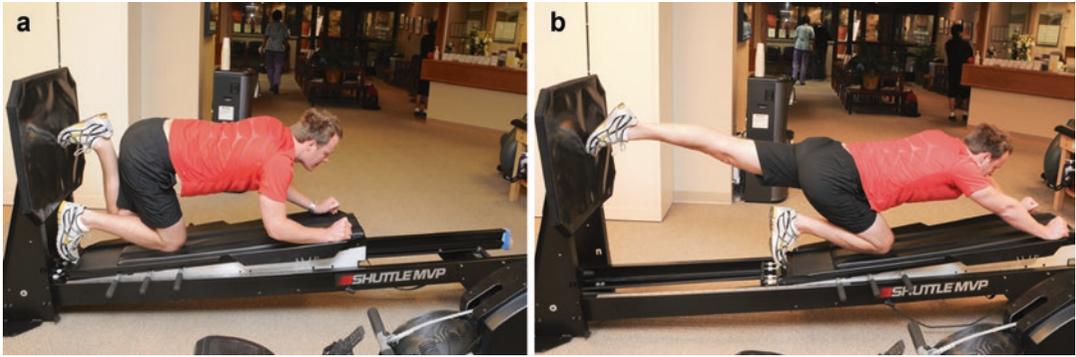


Fig. 31.7 Single-leg squats performed using spring-loaded technology helps to lessen the impact forces of traditional squats as well as quantify the weight distrib-

uted across the joint. In (a), the patient is seen eccentrically loading the quadriceps in deep knee flexion. In (b), the patient is seen maximally extending the knee

ketball is a complex decision that involves input from all members of the athlete's treatment team including the physical therapist, athletic trainers, and surgeon. Return to play testing using an objective scoring system, such as the Vail Sports Test, and outcome scores SANE/IKDC, should be completed and passed prior to the athlete being released to practice or game activity.

31.4 Outcomes

As basketball requires high-intensity multi-directional movements, which put constant dynamic stresses on the knee, injuries to this joint can be devastating to an athlete's career. The compressive forces and shear across the tibiofemoral joint after knee surgery may lead to worse long-term outcomes after the constant cutting, pivoting, and jumping required from basketball. In a review of injuries in the National Basketball Association (NBA) from 1998 to 2005, Drakos et al. reported that injuries to the knee were the fourth most commonly affected structure, but accounted for the highest proportion of total games missed (18.1%) [63].

As previously discussed, the literature is convincing in that operative management is superior to nonoperative management of multi-ligament knee injuries in the general population, and that patients treated surgically are more likely to return to work and sports [18, 30, 64]. In a cohort of 89 patients treated for traumatic

knee dislocation, Richter et al. [18] showed that those who had sports injuries and were 40 years or younger had higher Lysholm and Tegner scores at mean follow-up of 8.2 years postoperatively, though the authors did not stratify by sport played. Good functional outcomes in short-, mid-, and long-term follow-up have been reported after multi-ligament knee surgery in the general population. Moatshe et al. reported improvement in knee function with a median Tegner activity score of 4, average Lysholm score of 84 ± 17 , and an average IKDC-2000 score of 73 ± 19 [65].

Though the literature investigating MLI in basketball players is lacking, the consequences of isolated ACL injuries in these athletes can give insight to how more complex injuries may affect these athletes. Return to play in NBA players after isolated ACL tears has been reported to be between 78% and 84% [66, 67]. NBA players undergoing arthroscopic knee surgery have shorter career lengths and significantly greater decline in postoperative performance outcomes compared with other procedures, with as many as 44% of those who do return to play after ACL reconstruction often experiencing decreased performance while non-injured players demonstrate an increase in performance [66, 67]. Notably, LaPrade et al. [5] published a case report of an NBA player with a combined ACL and FCL knee injury who returned to a preinjury level of competition 9 months after a single-staged reconstruction of the ACL and FCL. As promising as

the results of this report are, multi-ligament knee injuries present increased surgical and rehabilitation challenges compared with isolated ligamentous injuries, and presumably basketball players would be less likely to return to high-level competition and preinjury performance after suffering this complex injury.

Outcomes of multi-ligament knee injuries are generally favorable in highly athletic populations though a large proportion of patients never achieve their preoperative athletic function. Bakshi et al. examined return to play after multi-ligament knee injuries in professional football players, athletes whose sport requires similar biomechanical demands to basketball players. They found that 56% of athletes returned to play at a mean time of 388 ± 198 days, with those suffering ACL/MCL injuries returning sooner than those with combined ACL and PCL/LCL injuries and frank knee dislocations [68]. Hirschmann et al. [69] showed that elite athletes, including basketball players, with complete single-stage reconstruction within 40 days of injury had better outcomes when compared to athletes treated beyond 40 days from injury. Although 19 of 24 athletes returned to their previous sport postoperatively, only eight reached their preinjury sports activity level [69]. Similarly, in a highly-active military population, 54% of patients were able to return to active duty following surgical reconstruction of their knee [70]. Cincinnati Knee Ligament Rating Scale demonstrated an average sport function score of 66 (able to perform at “half speed”) [70].

Given the trauma imparted to the knee following a multi-ligament knee injury, the prevalence of osteoarthritis (OA) following these injuries is high. Moatshe et al. [65] showed that at long-term follow-up (10.0–18.8 years), radiographic osteoarthritis in the operated knee was present in 42% of patients who underwent multi-ligament knee reconstruction. Moreover, researchers showed that patients older than 30 at the time of surgery were at an increased risk of developing OA. As the average length of an NBA career for players is approximately 6 years, [71] post-traumatic osteoarthritis may pose a greater problem to retired players than active ones.

31.5 Future Treatment Options

There is considerable room for optimization of surgical reconstruction for multi-ligament knee injuries in order to enhance outcomes. With the trend of early sport specialization and high-intensity athletics being played at an earlier age, future research should focus on injury patterns and outcomes of multi-ligament knee injury in adolescent basketball players. Similarly, with the aging population and desire for older populations to remain active, future treatment options may focus on enhancing graft healing, particularly in older patients with worse healing potential.

Future treatment considerations may center on finer points of the operation such as graft-tensioning force and optimal tensioning sequence. No consensus currently exists on the optimal tensioning sequence of grafts during multi-ligament knee reconstruction [72]. Further biomechanical studies are required to compare and refine graft-tensioning protocols, and validate arthroscopic reduction landmarks. These studies will translate into large-scale clinical trials to define stability parameters that improve functional results.

Take Home Message

- Multi-ligament knee injuries, while devastating for the basketball athlete, have seen a significant improvement in outcomes with the advent of improved surgical techniques and developed, staged rehabilitation protocols.
- The goal of surgical reconstruction is to restore the native kinematic forces imparted on the knee during high-intensity exercise.
- Ligament reconstruction, single-stage operation, and early surgical intervention are all factors that have been associated with improved outcomes in MLI.
- Rehabilitation is an integral part of the treatment algorithm. Following establishment of strong dynamic neuromuscular control and muscular strength foundation, these programs should start

to incorporate athletic movements that are required on the court such as pivoting and lateral movements prior to return to sport.

- A staged rehabilitation program with phase-specific goals and criteria for progression provide the optimal chance for return to sport.
- While there is still a long way to go in the management of an athlete with MLI, basketball players can expect to see quicker return to sport with better functional outcomes as surgical technique and rehabilitation protocols continue to evolve.

References

1. Moatshe G, Chahla J, LaPrade RF, Engebretsen L. Diagnosis and treatment of multiligament knee injury: state of the art. *J ISAKOS*. 2017;2(3):152–61.
2. Andreoli CV, Chiaramonti BC, Buriel E, Pochini AC, Ejnisman B, Cohen M. Epidemiology of sports injuries in basketball: integrative systematic review. *BMJ Open Sport Exerc Med*. 2018;4(1):e000468.
3. Prodromos CC, Han Y, Rogowski J, Joyce B, Shi K. A meta-analysis of the incidence of anterior cruciate ligament tears as a function of gender, sport, and a knee injury-reduction regimen. *Arthroscopy*. 2007;23(12):1320–5, e6.
4. Engebretsen L, Risberg MA, Robertson B, Ludvigsen TC, Johansen S. Outcome after knee dislocations: a 2–9 years follow-up of 85 consecutive patients. *Knee Surg Sports Traumatol Arthrosc*. 2009;17(9):1013–26.
5. LaPrade RF, O'Brien L, Kennedy NI, Cinque ME, Chahla J. Return to National Basketball Association competition following anterior cruciate ligament and fibular collateral ligament injuries: a case report. *JBJS Case Connect*. 2017;7(4):e81.
6. Wascher DC, Dvirnak PC, DeCoster TA. Knee dislocation: initial assessment and implications for treatment. *J Orthop Trauma*. 1997;11(7):525–9.
7. Schenck RC Jr. The dislocated knee. *Instr Course Lect*. 1994;43:127–36.
8. Schenck R. Classification of knee dislocations. *Oper Tech Sports Med*. 2003;11(3):193–8.
9. Arom GA, Yeranorian MG, Petrigliano FA, Terrell RD, McAllister DR. The changing demographics of knee dislocation: a retrospective database review. *Clin Orthop Relat Res*. 2014;472(9):2609–14.
10. Arendt E, Dick R. Knee injury patterns among men and women in collegiate basketball and soccer. NCAA data and review of literature. *Am J Sports Med*. 1995;23(6):694–701.
11. Moatshe G, Dornan GJ, Løken S, Ludvigsen TC, LaPrade RF, Engebretsen L. Knee dislocations demographics and associated injuries: a prospective review of 303 patients. *Orthop J Sports Med*. 2017;5(5):2325967117706521. <https://doi.org/10.1177/2325967117706521>.
12. Robertson A, Nutton RW, Keating JF. Dislocation of the knee. *J Bone Joint Surg Br*. 2006;88(6):706–11.
13. Becker EH, Watson JD, Dreese JC. Investigation of multiligamentous knee injury patterns with associated injuries presenting at a level I trauma center. *J Orthop Trauma*. 2013;27(4):226–31.
14. Tan Y, Zhou P, Ma C. Analysis of sport-injured pattern of anterior cruciate ligament in the reconstruction period of Chinese college students. *J Sports Med Phys Fit*. 2016;56(11):1346–51.
15. Medina O, Arom GA, Yeranorian MG, Petrigliano FA, McAllister DR. Vascular and nerve injury after knee dislocation: systematic review. *Clin Orthop Relat Res*. 2014;472(9):2621–9.
16. Geeslin AG, LaPrade RF. Location of bone bruises and other osseous injuries associated with acute grade III isolated and combined posterolateral knee injuries. *Am J Sports Med*. 2010;38(12):2502–8.
17. Krych AJ, Sousa PL, King AH, Engasser WM, Stuart MJ, Levy BA. Meniscal tears and articular cartilage damage in the dislocated knee. *Knee Surg Sports Traumatol Arthrosc*. 2015;23(10):3019–25.
18. Richter M, Bosch U, Wippermann B, Hofmann A, Krettek C. Comparison of surgical repair or reconstruction of the cruciate ligaments versus nonsurgical treatment in patients with traumatic knee dislocations. *Am J Sports Med*. 2002;30(5):718–27.
19. Kendall RW, Taylor DC, Salvian AJ, O'Brien PJ. The role of arteriography in assessing vascular injuries associated with dislocations of the knee. *J Trauma*. 1993;35(6):875–8.
20. Mills WJ, Barei DP, McNair P. The value of the ankle-brachial index for diagnosing arterial injury after knee dislocation: a prospective study. *J Trauma*. 2004;56(6):1261–5.
21. Levy BA, Fanelli GC, Whelan DB, Stannard JP, Boyd JL, Marx RG, et al. Controversies in the treatment of knee dislocations and multiligament reconstruction. *J Am Acad Orthop Surg*. 2009;17(4):197–206.
22. LaPrade R, Chahla J, DePhillipo NN, Cram T, Kennedy MI, Dornan GJ, O'Brien LT, Engebretsen L, Moatshe G. Single stage multiple ligament knee reconstructions for sports related injuries: outcomes in 194 patients. *Am J Sports Med*. 47(11):2563–71.
23. Jackman T, LaPrade RF, Pontinen T, Lender PA. Intraobserver and interobserver reliability of the kneeling technique of stress radiography for the evaluation of posterior knee laxity. *Am J Sports Med*. 2008;36(8):1571–6.

24. LaPrade RF, Bernhardson AS, Griffith CJ, Macalena JA, Wijdicks CA. Correlation of valgus stress radiographs with medial knee ligament injuries: an in vitro biomechanical study. *Am J Sports Med.* 2010;38(2):330–8.
25. James EW, Williams BT, LaPrade RF. Stress radiography for the diagnosis of knee ligament injuries: a systematic review. *Clin Orthop Relat Res.* 2014;472(9):2644–57.
26. LaPrade RF, Resig S, Wentorf F, Lewis JL. The effects of grade III posterolateral knee complex injuries on anterior cruciate ligament graft force. A biomechanical analysis. *Am J Sports Med.* 1999;27(4):469–75.
27. LaPrade RF, Muench C, Wentorf F, Lewis JL. The effect of injury to the posterolateral structures of the knee on force in a posterior cruciate ligament graft: a biomechanical study. *Am J Sports Med.* 2002;30(2):233–8.
28. Chahla J, Nitri M, Civitarese D, Dean CS, Moulton SG, LaPrade RF. Anatomic double-bundle posterior cruciate ligament reconstruction. *Arthrosc Tech.* 2016;5(1):e149–56.
29. Griffith CJ, LaPrade RF, Johansen S, Armitage B, Wijdicks C, Engebretsen L. Medial knee injury: Part 1, static function of the individual components of the main medial knee structures. *Am J Sports Med.* 2009;37(9):1762–70.
30. Dedmond BT, Almekinders LC. Operative versus nonoperative treatment of knee dislocations: a meta-analysis. *Am J Knee Surg.* 2001;14(1):33–8.
31. Geeslin AG, LaPrade RF. Outcomes of treatment of acute grade-III isolated and combined posterolateral knee injuries: a prospective case series and surgical technique. *J Bone Joint Surg Am.* 2011;93(18):1672–83.
32. Chahla J, Moatshe G, Cinque ME, Godin J, Mannava S, LaPrade RF. Arthroscopic anatomic single-bundle anterior cruciate ligament reconstruction using bone-patellar tendon-bone autograft: pearls for an accurate reconstruction. *Arthrosc Tech.* 2017;6(4):e1159–e67.
33. Lind M, Jakobsen BW, Lund B, Hansen MS, Abdallah O, Christiansen SE. Anatomical reconstruction of the medial collateral ligament and posteromedial corner of the knee in patients with chronic medial collateral ligament instability. *Am J Sports Med.* 2009;37(6):1116–22.
34. Kennedy NI, LaPrade RF, Goldsmith MT, Faucett SC, Rasmussen MT, Coatney GA, et al. Posterior cruciate ligament graft fixation angles, part 1: biomechanical evaluation for anatomic single-bundle reconstruction. *Am J Sports Med.* 2014;42(10):2338–45.
35. Coobs BR, Wijdicks CA, Armitage BM, Spiridonov SI, Westerhaus BD, Johansen S, et al. An in vitro analysis of an anatomical medial knee reconstruction. *Am J Sports Med.* 2010;38(2):339–47.
36. Mariani PP, Santoriello P, Iannone S, Condello V, Adriani E. Comparison of surgical treatments for knee dislocation. *Am J Knee Surg.* 1999;12(4):214–21.
37. Levy BA, Dajani KA, Morgan JA, Shah JP, Dahm DL, Stuart MJ. Repair versus reconstruction of the fibular collateral ligament and posterolateral corner in the multiligament-injured knee. *Am J Sports Med.* 2010;38(4):804–9.
38. Stannard JP, Black BS, Azbell C, Volgas DA. Posteromedial corner injury in knee dislocations. *J Knee Surg.* 2012;25(5):429–34.
39. Stannard JP, Brown SL, Farris RC, McGwin G Jr, Volgas DA. The posterolateral corner of the knee: repair versus reconstruction. *Am J Sports Med.* 2005;33(6):881–8.
40. Spiridonov SI, Slinkard NJ, LaPrade RF. Isolated and combined grade-III posterior cruciate ligament tears treated with double-bundle reconstruction with use of endoscopically placed femoral tunnels and grafts: operative technique and clinical outcomes. *J Bone Joint Surg Am.* 2011;93(19):1773–80.
41. LaPrade RF, Griffith CJ, Coobs BR, Geeslin AG, Johansen S, Engebretsen L. Improving outcomes for posterolateral knee injuries. *J Orthop Res.* 2014;32(4):485–91.
42. Geeslin AG, Moulton SG, LaPrade RF. A systematic review of the outcomes of posterolateral corner knee injuries, part I: surgical treatment of acute injuries. *Am J Sports Med.* 2015;44:1336.
43. Fanelli GC, Giannotti BF, Edson CJ. Arthroscopically assisted combined posterior cruciate ligament/posterior lateral complex reconstruction. *Arthroscopy.* 1996;12(5):521–30.
44. Jiang L, Wu H, Yan S. Two cases of contact anterior cruciate ligament rupture combined with a posterolateral tibial plateau fracture. *Case Rep Orthop.* 2015;2015:5.
45. Mook WR, Miller MD, Diduch DR, Hertel J, Boachie-Adjei Y, Hart JM. Multiple-ligament knee injuries: a systematic review of the timing of operative intervention and postoperative rehabilitation. *J Bone Joint Surg Am.* 2009;91(12):2946–57.
46. Harner CD, Waltrip RL, Bennett CH, Francis KA, Cole B, Irrgang JJ. Surgical management of knee dislocations. *J Bone Joint Surg Am.* 2004;86-a(2):262–73.
47. Levy BA, Dajani KA, Whelan DB, Stannard JP, Fanelli GC, Stuart MJ, et al. Decision making in the multiligament-injured knee: an evidence-based systematic review. *Arthroscopy.* 2009;25(4):430–8.
48. Mueller BT, O'Brien LT. Multiligament knee injuries in athletes, is it possible to return to play? —a rehabilitation perspective. *Ann Joint.* 2018;3:92.
49. Sisto DJ, Warren RF. Complete knee dislocation. A follow-up study of operative treatment. *Clin Orthop Relat Res.* 1985;198:94–101.
50. Lynch AD, Chmielewski T, Bailey L, Stuart M, Cooper J, Coady C, et al. Current concepts and controversies in rehabilitation after surgery for multiple ligament knee injury. *Curr Rev Musculoskelet Med.* 2017;10(3):328–45.
51. Edson CJ, Fanelli GC, Beck JD. Rehabilitation after multiple-ligament reconstruction of the knee. *Sports Med Arthrosc Rev.* 2011;19(2):162–6.
52. Fanelli GC, Edson CJ, Reinheimer KN, Garofalo R. Posterior cruciate ligament and posterolateral

- corner reconstruction. *Sports Med Arthrosc Rev.* 2007;15(4):168–75.
53. Jenkins PJ, Clifton R, Gillespie GN, Will EM, Keating JF. Strength and function recovery after multiple-ligament reconstruction of the knee. *Injury.* 2011;42(12):1426–9.
 54. Owens BD, Neault M, Benson E, Busconi BD. Primary repair of knee dislocations: results in 25 patients (28 knees) at a mean follow-up of four years. *J Orthop Trauma.* 2007;21(2):92–6.
 55. Irrgang JJ, Fitzgerald GK. Rehabilitation of the multiple-ligament-injured knee. *Clin Sports Med.* 2000;19(3):545–71.
 56. Murphy KP, Helgeson MD, Lehman RA Jr. Surgical treatment of acute lateral collateral ligament and posterolateral corner injuries. *Sports Med Arthrosc Rev.* 2006;14(1):23–7.
 57. Talbot M, Berry G, Fernandes J, Ranger P. Knee dislocations: experience at the Hopital du Sacre-Coeur de Montreal. *Can J Surg.* 2004;47(1):20–4.
 58. Gormeli G, Gormeli CA, Elmali N, Karakaplan M, Ertem K, Ersoy Y. Outcome of the treatment of chronic isolated and combined posterolateral corner knee injuries with 2- to 6-year follow-up. *Arch Ortho Trauma Surg.* 2015;135(10):1363–8.
 59. Mikula JD, Slette EL, Dahl KD, Montgomery SR, Dornan GJ, O'Brien L, et al. Intraarticular arthrofibrosis of the knee alters patellofemoral contact biomechanics. *J Exper Orthop.* 2017;4(1):40.
 60. Noyes FR, Barber-Westin SD. Reconstruction of the anterior and posterior cruciate ligaments after knee dislocation. Use of early protected postoperative motion to decrease arthrofibrosis. *Am J Sports Med.* 1997;25(6):769–78.
 61. Chahla J, Godin J, LaPrade R. Return to play after multiple knee ligament injuries. Return to play in football. Berlin: Springer; 2018. p. 637–47.
 62. Toutoungi DE, Lu TW, Leardini A, Catani F, O'Connor JJ. Cruciate ligament forces in the human knee during rehabilitation exercises. *Clin Biomech (Bristol, Avon).* 2000;15(3):176–87.
 63. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the National Basketball Association: a 17-year overview. *Sports Health.* 2010;2(4):284–90.
 64. Wong CH, Tan JL, Chang HC, Khin LW, Low CO. Knee dislocations—a retrospective study comparing operative versus closed immobilization treatment outcomes. *Knee Surg Sports Traumatol Arthrosc.* 2004;12(6):540–4.
 65. Moatshe G, Dornan GJ, Ludvigsen T, Loken S, LaPrade RF, Engebretsen L. High prevalence of knee osteoarthritis at a minimum 10-year follow-up after knee dislocation surgery. *Knee Surg Sports Traumatol Arthrosc.* 2017;25(12):3914–22.
 66. Minhas SV, Kester BS, Larkin KE, Hsu WK. The effect of an orthopaedic surgical procedure in the National Basketball Association. *Am J Sports Med.* 2016;44(4):1056–61.
 67. Busfield BT, Kharrazi FD, Starkey C, Lombardo SJ, Seegmiller J. Performance outcomes of anterior cruciate ligament reconstruction in the National Basketball Association. *Arthroscopy.* 2009;25(8):825–30.
 68. Bakshi NK, Khan M, Lee S, Finney FT, Stotts J, Sikka RS, et al. Return to play after multiligament knee injuries in National Football League athletes. *Sports Health.* 2018;10(6):495–9.
 69. Hirschmann MT, Iranpour F, Muller W, Friederich NF. Surgical treatment of complex bicruciate knee ligament injuries in elite athletes: what long-term outcome can we expect? *Am J Sports Med.* 2010;38(6):1103–9.
 70. Ross AE, Taylor KF, Kirk KL, Murphy KP. Functional outcome of multiligamentous knee injuries treated arthroscopically in active duty soldiers. *Mil Med.* 2009;174(10):1113–7.
 71. Wilczynski M. Average NBA career length for players – details 2011. 2008. <https://weaksidawareness.wordpress.com/2011/11/22/average-nba-career-length-for-players-details/> Accessed 20 October 2019.
 72. Moatshe G, Chahla J, Brady AW, Dornan GJ, Muckenhirn KJ, Kruckeberg BM, et al. The influence of graft tensioning sequence on tibiofemoral orientation during bicruciate and posterolateral corner knee ligament reconstruction: a biomechanical study. *Am J Sports Med.* 2018;46(8):1863–9.



Management of Knee Cartilage Injuries in Basketball

32

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32.1 Introduction

Treatment of articular cartilage defects of the knee remains a challenging entity, particularly in young high-demand patients. Damaged articular cartilage has limited potential for self-healing and therefore has an increased propensity to progress to osteoarthritis [1, 2]. The prevalence of cartilage lesions in the general population ranges from 13% to 60% and affects an estimated 900,000 patients in the United States [3–6]. However, the prevalence in athletes has been reported to be on average 36% (range 2.4–75%), with 14% of these athletes being asymptomatic at diagnosis and with the patellofemoral compartment (37%) and femoral condyle (35%) being the locations most likely to be affected [7, 8]. In professional basketball athletes, this number is even higher. Three prior studies have reported that the prevalence of focal chondral defects (FCDs) in the national basketball association (NBA) is between 41% and 50% of players and most commonly affects the patellofemoral joint (70–77% of defects) [9, 10]. Magnetic resonance

imaging (MRI) has shown that basketball players have a similar level of undiagnosed, and generally asymptomatic FCDs compared to athletes of other sports [11]. In the general population, the number of surgical procedures to address these cartilage defects is estimated to be approximately 200,000 cases annually [4, 12].

Treatment options for focal chondral defects include non-operative and surgical options. Non-operative treatments are generally considered first-line, especially when no mechanical symptoms are present. A variety of surgical procedures are available; the choice of which surgery to choose is individualized based on the athlete and his or her risk factors and the patient's current time in the season. If conservative measures, such as physical therapy or an intra-articular injection fail, a less-invasive procedure such as a chondral debridement can provide significant symptomatic relief, with minimal down time without altering the opportunity for a more definitive procedure. Other surgical interventions include microfracture, osteochondral autograft transplantation (OCA), osteochondral allograft transplantation (OAT), autologous chondrocyte implantation (ACI), and its newer iterations (matrix ACI) and newer procedures including minced cartilage procedures (DeNovo Natural Tissue (NT), Zimmer Inc., Warsaw, IN), cryopreserved osteochondral allografts (Cartiform, Athrex Inc., Naples, FL; Chondrofix, JRF, Centennial, CO; Prochondrix, AlloSource,

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Centennial, CO), and extracellular matrix scaffolds (BioCartilage, Arthrex Inc., Naples, FL).

The purpose of this chapter is to review focal chondral defects of the knee and their treatment, with special attention on the use and impact of these procedures in basketball players. Initially, this chapter assesses how FCDs are diagnosed using patient history, physical examination, and imaging. Then, non-operative treatments, various surgical techniques and their indications, and postoperative rehabilitation process are investigated. Finally, outcomes of these procedures and their return to sport data and basketball, specifically, are analyzed.

32.2 Diagnosis

FCDs in basketball players are diagnosed through a combination of patient history, physical examination, radiographs, and MRI. An early diagnosis of FCDs is critical, especially in a young basketball player, as increased time from diagnosis to intervention has been shown to increase the risk of worsening cartilage damage and development of osteoarthritis [13].

32.2.1 Patient History

The initial step of diagnosis, as with most sport injuries, is a comprehensive patient history. A typical presentation of an FCD would be a basketball player who presents with continued knee pain and swelling. The symptoms of cartilage injury are generally non-specific, and pain is the most common chief complaint. A high degree of suspicion is important in those who have acute patellar dislocation, ligament injury, or hemarthrosis [12]. Often the pain develops insidiously without an inciting event and presents as intermittent pain that may be worse during specific activity and sports play. This association should be further explored because multiple knee pathologies can present with knee pain. For example, patellar tendonitis which commonly occurs in male basketball players and affects up to 11% of players can present with anterior knee pain local-

ized over the patella, with swelling and stiffness [14]. However, acute pain in association with injury can also occur as approximately half of patellofemoral FCD occurs in the setting of traumatic injury [15]. The location of pain can depend on the location of the FCD, and it can also be diffuse in nature. Pain can be present in addition to intermittent effusion, crepitus, catching, and locking.

A full past medical history is also essential in diagnosing and creating a treatment plan. An understanding of a patient's comorbidities, past surgical history, and history of physical injury is essential. Previous injury, such as ACL injury, is associated with chondral injury [16]. In addition, any prior treatment such as medications, physical therapy, or injections should be noted.

32.2.2 Physical Examination

Aspects of the physical examination can suggest the diagnosis of an FCD. On inspection, one should look for evidence of effusion, deformity, patellar maltracking, and malalignment that may be present. In patients with patellofemoral FCD, the most common type seen in basketball players, in-toeing, valgus alignment, or hip abductor weakness is often observed. On palpation of the joint, tenderness is common over the femoral condyle or tibial plateau, depending on the location of the lesion. Patients usually retain full range of motion. A full knee examination is essential in ruling out other diagnoses such as meniscal tears, ligamentous injury, or extensor mechanism pathologies. While a physical examination must be conducted when evaluating a patient with cartilage injury, the findings are generally non-specific and provide little concrete evidence of the underlying diagnosis.

32.2.3 Radiographic Imaging

Due to the lack of specificity in patient history and on physical examination, both radiographic and MR imaging are necessary in successfully diagnosing a patient with an FCD. When

obtaining X-rays, four views are suggested: bilateral standing anterior posterior (AP), 45° of flexion weight-bearing posterior anterior (PA “Rosenburg”), non-weight-bearing 30° of flexion true laterals, and patella sunrise view (“Merchant”). In addition, physicians should obtain a mechanical axis X-ray. Specifically, long-leg alignment views allow for the determination of the mechanical axis and to evaluate for alignment. These images are necessary to rule out bony defects and determine the alignment of the joint. Radiographs should be evaluated for multiple findings such as radiolucencies, subchondral cysts, sclerosis, fragmentation, loose bodies, joint space narrowing, and physeal status as these can affect the treatment plan.

32.2.4 MRI

MRI can provide more information, and is the most sensitive modality, to evaluate cartilage defects. However, diagnostic accuracy based on MRI compared to arthroscopy has been shown to be in part dependent on the severity of the cartilage defect (e.g., Outerbridge grade 3–4) [17]. Conventional MRI methods include T1-weighted and T2-weighted imaging and can provide morphological and physiological information about a patient’s knee. However, fat-suppressed sequences such as T2-weighted fast spin echo (FSE), and T1-weighted spoiled gradient-echo that allow for enhanced contrast between fluid and cartilage provide improved sequences producing images with intermediate cartilage signal and bright fluid signal [18]. Newer 3D FSE and 3D multi-echo gradient-echo sequences further improve this distinction [19]. Other novel technologies include delayed gadolinium-enhanced MRI for cartilage (dGEMRIC). dGEMRIC is sensitive to glycosaminoglycan distribution in cartilage and allows visualization of areas of glycosaminoglycan depletion; however, it requires a double-dose IV contrast injection. Other techniques include T2 relaxation time mapping, which is sensitive to the cartilage–collagen matrix and water distribution within the articular cartilage, and T1rho

mapping which is sensitive to cartilage proteoglycan depletion [20, 21]. However, whether symptoms correlate with imaging findings should always be considered. A study in basketball players found that 47.5% of the 40 knees included in the study had asymptomatic cartilage lesions on MRI.

32.2.5 Diagnostic Arthroscopy

The most accurate test for diagnosis and grading of an FCD is diagnostic arthroscopy. This allows for visualization of the cartilage defect and allows for determination of lesion size, grade, and location. There are two main grading systems for cartilage defects. The first is the International Cartilage Repair Society grading system (ICRS) [22]. This cartilage grading system ranges from a score of 0 to 4 based on the depth of the defect from nearly normal to penetration beyond the subchondral bone. The other commonly utilized grading system is the Outerbridge cartilage score, which is based on the appearance of the cartilage defect, including the presence of swelling, fragmentation, and erosion [23]. The findings on diagnostic arthroscopy including the severity, size, depth, and location of the lesion will dictate next steps in treatment.

32.3 Conservative Management

Conservative management is generally the initial approach and is used in patients with mild symptoms or small lesions as its goal is to reduce symptoms instead of reversing or fixing the underlying lesion [24]. Types of conservative treatment include analgesics, chondroprotective agents (glucosamine, chondroitin), steroid injections, physical therapy, and knee bracing, and these are especially useful mid-season to allow for players to return to play with symptomatic relief [25]. However, activity modification is also recommended as part of conservative management, which may be a challenge in basketball players. Studies on the role of conservative management in athletes is limited, with one study

showing that 22 of 28 athletes had successful results of conservative treatment but continued to have radiographic chondral abnormalities at follow-up 14 years later [25]. Therefore, depending on the size of the defect, surgery, where return to sport has been studied, may be preferred in high-level athletes when conservative treatment fails.

32.4 Surgical Treatment

Limited literature is available regarding a treatment algorithm specific to basketball players or even athletes in general. Thus, the best way to treat these injuries in this patient population is to treat them the same as in the general population, while managing expectations. Special attention should be given to data showing return to sport after the various cartilage procedures in both basketball players and other professional athletes, although understanding of the sport and season timing is necessary to determine the aggressiveness of treatment at that time. Surgical treatment is generally utilized in patients who are symptomatic, have an acute injury, have loose bodies, and those who fail conservative treatment. There are three main categories of surgical treatment: palliative (debridement and chondroplasty), reparative (microfracture and other bone marrow stimulating techniques), and restorative (MACI, OCA, OAT). The main considerations in deciding on the proper surgical procedure for a cartilage lesion depend on the lesion size, age, and activity level. However, other specific patient factors such as comorbidities and past surgical history also play a role in this decision. In athletes in particular, activity and return to sport ability must be considered. A systematic review analyzing the return-to-sport rates in 1469 athletes found that return-to-sport rates range from 68% in microfracture to 91% in OAT, 74% in ACI, and 84% in OCA. This data is crucial in considering surgical procedural type in a basketball player [26].

The first consideration for the indication of the surgical procedure is cartilage lesion size. Lesions less than 2 cm are typically first addressed with debridement (abrasion chondroplasty) and potentially bone marrow-stimulating techniques,

such as microfracture (which can be augmented with other biological treatments or scaffolds). OAT is also often used for this subset of patients where the chondral lesion is small, especially in those with higher activity levels [27]. As lesions become greater than 2.5 cm, these can be treated with OCA and MACI. Debridement is generally the first-line treatment, especially in lesions <2 cm and if there are flaps or loose tissue [28]. If a rapid return to basketball is necessary, players can undergo a less aggressive procedure such as chondroplasty with a potentially more aggressive procedure, as needed, during off-season. However, this choice greatly depends on the time of the season. If the initial treatment fails, then a more aggressive procedure may be considered. MACI is also more appropriate in those with shallow lesions, especially in the patellofemoral joint (as it is easier to match the shape of the patellofemoral joint) and is thus particularly relevant to basketball players. Newer ACI techniques such as matrix-induced ACI (MACI) can also be used. In addition, OAT or OCA are the suggested treatment in patients with damage to subchondral bone, as these procedures replace the whole osteochondral unit. Osteochondral treatment also gives the benefit of structurally normal cartilage placed immediately for faster return to sport and time to weight-bearing.

The second consideration is defect location. In basketball players, lesions in the patellofemoral area have been reported to be the most common [9]. However, lesions can also occur on the femoral condyles and tibia. OCA has been shown to provide successful results when used for lesions of the femoral condyles or trochlea [28, 29]. In addressing patellofemoral joint lesions and isolated lesions of the patella, ACI, MACI, and OCA have been found to have successful results in numerous studies [30–33]. The most difficult location to adequately treat is lesions on the tibia. Microfracture and local biological augmentation can be used; otherwise, OATs placed in a retrograde manner can be utilized with caution.

Other concurrent issues that must be taken into consideration include ligament pathology, malalignment, and meniscus deficiency. In cases of ligament pathology or meniscus deficiency, a liga-

ment reconstruction or meniscal excision or repair can occur concomitantly with the cartilage procedure. In patients with malalignment, an osteotomy should be considered. An osteotomy, such as a high tibial osteotomy or a distal femoral osteotomy, can be utilized in patients with varus or valgus malalignment, respectively. Furthermore, an anteromedialization, an osteotomy of the tibial tubercle, can be utilized in patients with patellofemoral chondral defects [34].

32.5 Surgical Techniques

32.5.1 Abrasion Chondroplasty

Chondroplasty is one of the most frequently performed arthroscopic procedures. The goal of chondroplasty is to smooth over areas of fragmented and damaged cartilage. This can be performed with a curved shaver that allows for the ability to reach most areas of the knee. The tip of the shaver is then used to gently remove unstable cartilage and the calcified cartilage layer within the cartilage defect while care is taken to not disturb healthy cartilage and underlying subchondral bone [35]. Specialized curettes, such as a D-curette or ring curette, can also be utilized in this situation.

32.5.2 Marrow Stimulation

Microfracture was originally developed by Steadman et al. over 20 years ago to treat small chondral defects [36]. The goal of marrow stimulation is to stimulate cartilage defect healing with pluripotent progenitor cells, cytokine, growth factors, and proteins from within the bone marrow. During this procedure, multiple small holes are made in the subchondral bone to stimulate the cartilage (Fig. 32.1). When performing this procedure, the first step is an examination under anesthesia followed by a 10-point arthroscopy to examine all surfaces of the knee joint and to ensure that only a localized lesion is present. Then the next step is to prepare the osteochondral defect, removing any flaps, and debriding the sur-

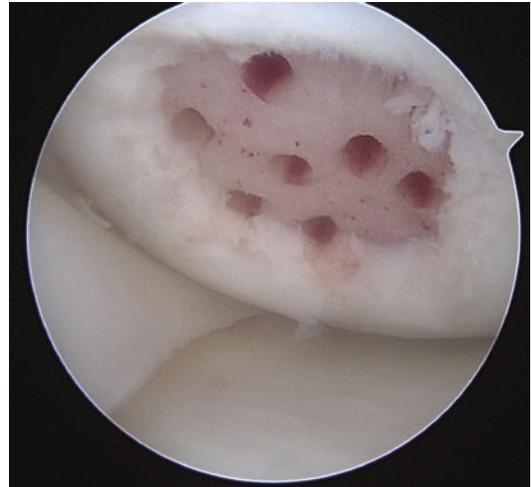


Fig. 32.1 Intraoperative image illustrating marrow stimulation to medial femoral condyle

rounding area the same way as in an abrasion chondroplasty down to the subchondral bone including the calcified cartilage layer. Once this is removed, a microfracture drill using Kirschner wires, fluted drill, wires, or angulated awl is used to create holes 2–4 mm apart, releasing the underlying bone marrow cells into the cartilage defect which can be observed [37, 38]. In addition, nanofracture techniques, which utilize a smaller diameter drill are still being investigated [39].

Newer iterations of marrow stimulation are still being investigated. These newer procedures augment the same microfracture procedure with additional biologics, such as bone marrow aspirate concentrate (BMAC) or platelet-rich plasma (PRP). However, whether these additions provide any long-term benefit in patient outcomes still remains unclear [40, 41].

32.5.3 Osteochondral Autograft Transplantation (OAT)

OAT is generally indicated for patients who have smaller higher-grade lesions and are younger and more active. OAT is performed by removing a small area of healthy cartilage in an area of the joint that is mainly non-load-bearing and placing it into the chondral defect, which can be per-

formed either open or arthroscopically. In surgery, the patient is positioned supine or the limb can be placed in a leg holder with a tourniquet and an examination under anesthesia is performed. A small parapatellar vertical portal incision is then made, and a diagnostic arthroscopy is performed to examine the cartilage surface. During diagnostic arthroscopy, the cartilage defect area is surveyed with a probe to determine defect size and confirm no other cartilage injuries are present.

At the cartilage defect location, a guide pin is placed in the center of the cartilage defect, perpendicularly. A cannulated reamer is then placed over the guide pin, and the guide pin is subsequently removed. The depth of the lesion is measured with a cannulated dilator.

The area of which to harvest the healthy cartilage from is predetermined using MRI. Graft harvest sizes are 6, 8, or 10 mm. Commonly, the harvest graft is taken from the lateral trochlea and lateral femoral condyle. An appropriately sized harvester is then placed perpendicular to the graft harvest location and is inserted into the subchondral bone to a depth of 10–15 mm with a mallet. The harvester is then axially loaded and turned 90° clockwise, then counterclockwise before being removed. A mallet is then used to fragment the graft from the surrounding cartilage, and the plug is removed. The graft is then inspected, with any bony debris removed, and shaved so that it is 1 mm shallower than the cartilage defect. The graft is then replaced into the joint and is gently tapped into place.

32.5.4 Osteochondral Allograft Transplantation (OCA)

OCA is often used in patients with larger (>2 cm) lesions. In the operating room, the patient is positioned supine with a tourniquet. The procedure begins with a knee examination under anesthesia. A lateral or medial parapatellar incision is then made to access the FCD. There are two general techniques that exist for OCA: cylindrical press-fit plugs or free-shell grafts. Whichever technique is used, donor tissue must be size matched to individual patients based on X-ray, CT, or MRI measurement.



Fig. 32.2 Intraoperative image illustrating an osteochondral allograft transplantation to lateral femoral condyle

In the dowel grafting technique, a dowel of similar size to the cartilage lesion is selected. A guidewire is positioned using sizers into the center of the cartilage defect, and the dowel and the socket are drilled to a depth between 5 and 6 mm. The allograft is harvested to the desired size using a reamer from a matching zone and is inserted into the defect [42, 43]. This press-fit technique is often preferred as it eliminates the need for additional fixation (Fig. 32.2). In contrast, in the free-shell technique, a donor graft is matched to the defect site, inserted, and fixed with screws.

Larger defects often require the use of multiple plugs in what is termed “snowman technique” or “mastercard technique.” This involves placing and fixing the first plug, then drilling a second recipient site adjacent to, or partially over the first defect. However, based on prior studies, the snowman technique has been shown to provide inferior results compared to a one-plug technique [43, 44].

32.5.5 Autologous Chondrocyte Implantation (ACI)

Autologous chondrocyte implantation occurs over the course of two procedures with *ex vivo* chondrocyte expansion between procedures.

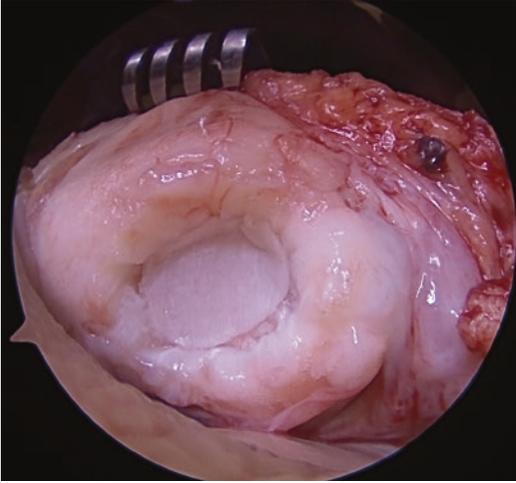


Fig. 32.3 Intraoperative image illustrating an autologous chondrocyte implantation on the patella

The initial procedure is a diagnostic arthroscopy with cartilage biopsy. During this procedure, the lesion size and grade are examined, and 200–300 mg of articular cartilage is harvested from a non-loading bearing surface of the knee. The collected cartilage is then processed via an enzymatic digestion process and is then cultured for 3–6 weeks.

In the second procedure, the harvested chondrocytes are reimplanted into the defect site (Fig. 32.3). It begins with the patient supine with a tourniquet applied. The defect is then debrided with a round-eyed sharp curette to expose subchondral bone. The original ACI technique involves a periosteal flap being sewn over the defect, followed by the injection of cultures chondrocytes underneath the flap (ACI-P), where the flap is harvested from the proximal-medial tibia [45–47]. In contrast, in ACI-C, a synthetic collagen membrane is used. In either case, the flap is positioned over the cartilage defect and sutured into place using 6-0 Vicryl. After the flap is checked to ensure a watertight seal, the cultured cartilage cells are then injected into lesion. In addition, a newer “sandwich” technique with autologous bone grafting can also be utilized, especially in patients with OCD [48].

A newer iteration of the ACI is an alternative technique called matrix ACI (MACI),

which is when a preformed biodegradable porcine type I/III matrix is utilized as a scaffold for the collected and cultured chondrocyte cells. In this procedure, the matrix is inserted into the defect and then fixated to the surrounding cartilage with fibrin glue without the need for suturing.

32.5.6 Novel Techniques

Newer techniques include autologous and allogenic minced cartilage (such as De Novo, biocartilage, and cartiform), which are similar to an MACI in that a collagen-chondroitin scaffold is used to model cultured chondrocytes [49, 50]. Minced cartilage can be utilized instead of cultured chondrocytes. In this case, only one procedure is needed as the cartilage is harvested and reimplanted in the same procedure [50]. In addition, fibrin glue is used to attach the minced pieces of cartilage together and attach the flap in addition to sutures to ensure fixation to the underlying subchondral bone [51]. A cartiform allograft is a cryopreserved osteochondral allograft scaffold that can be used as an alternative to ACI and, similarly to minced cartilage, can be implanted with fibrin glue. Biocartilage is a cartilage scaffold that is hydrated with PRP and can be used to fill defects after a microfracture procedure. All of these newer techniques have limited data supporting their superiority compared to traditional techniques. Future studies are needed to evaluate the benefits and shortcomings of these newer technologies.

32.6 Rehabilitation

32.6.1 Patellofemoral

Rehabilitation for patients who undergo patellofemoral cartilage procedures varies by institution. However, it often includes cryotherapy, elevation, and a brace immediately after surgery. Progressive passive motion and weight-bearing as tolerated can be implemented in the first few days after surgery. Range of motion is then increased with a goal of 90 degrees of flexion in the first 2 or 3 weeks [52].

32.6.2 Tibiofemoral

Patients who undergo cartilage repair of the tibiofemoral joint undergo multiple phases of postoperative rehabilitation. During the first phase, until 1 week postoperative, weight-bearing is restricted to less than 20% of body weight, range of motion (ROM) is restricted from 0 to 30°, and a protective knee brace is used at all times. Patients can progress to full passive motion within 1 week of surgery and then full active range of motion by 3 months post operatively. At 3 weeks patients are allowed to be fully weight-bearing while a brace is utilized until around 3 months postoperatively [53].

32.7 Clinical Outcomes

When deciding on which surgical procedure to use in a basketball player with a chondral defect, outcomes and ability and time to return to sport are of critical importance. Patient understanding and expectations should also be formed by providing all available data on outcomes of cartilage procedures in basketball players and other athletes as outcomes specific to basketball players remain limited. An individual approach should be taken when evaluating return to play as multiple factors influence it beyond surgical choice such as age (>30 years) and BMI (>27 kg/m²) [54]. Furthermore, as elite jumping athlete basketball players are unique from athletes in non-jumping sports, and this should be considered.

32.7.1 Microfracture

Microfracture in basketball players is the most well-studied cartilage procedure with no prior reports on outcomes of isolated chondroplasty in basketball players. Outcomes of microfracture in the general population have been positive. For example, Weber et al. found a statistically significant increase in all patient-reported outcomes (PROs) after a mean follow-up of 5.7 years. Furthermore, similar results have been shown in patients who undergo

microfracture compared to those who undergo ACI at 5- and 10-years postoperatively [55, 56]. In comparison to other sports, basketball players have been shown to have inferior results after microfracture [57].

Three studies have evaluated the success of microfracture in basketball players. The first study evaluated 24 NBA players who underwent microfracture surgery [58]. Sixty-seven percent of the players returned to play after the microfracture procedure. However, abilities after return to sport were found to be decreased compared to preoperatively in terms of both points scored and minutes played. In addition, the study found that patients were 8.15 times less likely to remain in the NBA after the index year. In the second study, 41 NBA players were evaluated after microfracture procedure [59]. Eighty-three percent of these players returned to professional basketball after an average of 9.2 months (4.32–14.08 months). Compared to their preoperative abilities, those who did return to sport had a significantly decreased points scored and steals per game. Furthermore, compared to other NBA players at a similar time point in their career, microfracture patients had significantly fewer points scored per game, games played per season, and free throw percentages. The third study of 24 professional basketball players found that 79% of patients returned to sport and mean time to returning was 30 weeks. However, on average their player efficiency rating decreased by 2.7 and their minutes per game decreased by 3 after surgery [60].

32.7.2 Osteochondral Autograft Transplantation

Osteochondral autograft transplantation has shown success, especially in terms of percentage of players who return to sport. In the general population, OATs has been shown to provide significant benefit in 72% of patients at a mean of 10.2 years of follow-up [61]. An additional study evaluated short- to mid-term outcomes in 112 patients who underwent OAT and found that both the VAS pain (7.14 ± 0.19 vs. 3.74 ± 0.26) and WOMAC (134.88 ± 5.84 vs. 65.92 ± 5.34) significantly

improved at a mean follow-up of 26.2 ± 0.24 months [62]. In comparison to microfracture, a meta-analysis showed that OAT results in a lower risk of failure (11% vs. 32%) and a higher level of patients who return to activity [63].

Furthermore, OAT has been shown to have a higher rate of patients who returned to sport when comparing procedure type: between 89% and 94% [27, 64, 65]. A systematic review found that based on seven articles, the mean time for return to competition after OAT was 5.6 months (3–14 months) [65, 66]. No study specifically investigated the return-to-sport rate and time in basketball players.

32.7.3 Osteochondral Allograft Transplantation

Osteochondral allograft transplantation has been demonstrated to be a successful procedure in both the general population and among athletes. After a mean of 12.3 years of follow-up 75% of patients demonstrated significant improvement in clinical outcomes [66]. A systematic review demonstrated that survivorship was 86.7% at 5 years and 78.7% at 10 years [67–69]. In the general athletic population, return to sport was seen in 72–82% of patients at a mean of 11 months [64, 69–71]. One study evaluated the return to sport in basketball players. The study consisted of 11 basketball players with a total of 14 chondral lesions, the overall rate of return to sport 80%, and the average time to return to play 14 months (6–26 months). The average lesion size was 509 mm² [2] and the location of the lesion varied and included the femoral condyle, trochlea, and patella. Furthermore, this study found that there was no significant decline in athletic performance after return to sport [72].

32.7.4 Autologous Chondrocyte Implantation

Autologous chondrocyte implantation has been shown to have successful outcomes. One study evaluated a cohort of patients at a mean 6.2 years follow-up, and all patients demonstrated significant

improvements in pain and function [73]. Kaplan–Meier survival analysis revealed that the survival rate was 78.2% at 5 years and 50.7% at 10 years.

In terms of return-to-sport outcomes, two systematic reviews have found that return to sport ranges from 82% and 84%, respectively [27, 64]. An additional study found a rate of 73%; however, they found that duration and frequency of exercise significantly decreased postoperatively. An additional study found that 64.5% of patients were able to return to sport at a competitive level [74]. They also showed that previous surgery was the biggest factor that dictated return to sport level in their cohort. No studies investigated the return to play after ACI in basketball players.

32.8 Conclusion

Focal chondral defects are common in athletes, especially basketball players. Symptomatic lesions can be addressed with conservative measures initially, but often surgical intervention is necessary but will depend on where the player is in the season. A range of surgical procedures are used based on chondral size and location, including abrasion chondroplasty, microfracture, OCA, OAT, and ACI. While microfracture has been the most studied technique in basketball players, OAT has been shown to have the highest rate of return to sport in all athletes, although the literature remains limited. Future studies are needed to evaluate other cartilage procedures specifically in basketball players.

References

1. Felson DT, Zhang Y, Hannan MT, et al. The incidence and natural history of knee osteoarthritis in the elderly, the Framingham osteoarthritis study. *Arthritis Rheum.* 1995;38(10):1500–5. <https://doi.org/10.1002/art.1780381017>.
2. Mankin H. The response of articular cartilage to mechanical injury. *J Bone Joint Surg Am.* 1982;64(3):460–6. <https://doi.org/10.2106/00004623-198264030-00022>.
3. Årøen A, Løken S, Heir S, et al. Articular cartilage lesions in 993 consecutive knee arthroscopies. *Am J Sports Med.* 2004;32(1):211–5. <https://doi.org/10.1177/0363546503259345>.

4. Curl WW, Krome J, Gordon ES, Rushing J, Smith B, Poehling GG. Cartilage injuries: a review of 31,516 knee arthroscopies. *Arthrosc J Arthrosc Relat Surg.* 1997;13(4):456–60. [https://doi.org/10.1016/s0749-8063\(97\)90124-9](https://doi.org/10.1016/s0749-8063(97)90124-9).
5. Solheim E, Krokeide A, Melteig P, Larsen A, Strand T, Brittberg M. Symptoms and function in patients with articular cartilage lesions in 1,000 knee arthroscopies. *Knee Surg Sports Traumatol Arthrosc.* 2016;24(5):1610–6. <https://doi.org/10.1007/s00167-014-3472-9>.
6. Widuchowski W, Widuchowski J, Trzaska T. Articular cartilage defects: study of 25,124 knee arthroscopies. *Knee.* 2007;14(3):177–82. <https://doi.org/10.1016/j.knee.2007.02.001>.
7. Everhart JS, Boggs Z, DiBartola AC, Wright B, Flanigan DC. Knee cartilage defect characteristics vary among symptomatic recreational and competitive scholastic athletes eligible for cartilage restoration surgery. *Cartilage.* 2019.;1947603519833144; <https://doi.org/10.1177/1947603519833144>.
8. FLANIGAN DC, HARRIS JD, TRINH TQ, SISTON RA, BROY RH. Prevalence of chondral defects in athletes' knees. *Med Sci Sports Exerc.* 2010;42(10):1795–801. <https://doi.org/10.1249/mss.0b013e3181d9eea0>.
9. Kaplan LD, Schurhoff MR, Selesnick H, Thorpe M, Uribe JW. Magnetic resonance imaging of the knee in asymptomatic professional basketball players. *Arthrosc J Arthrosc Relat Surg.* 2005;21(5):557–61. <https://doi.org/10.1016/j.arthro.2005.01.009>.
10. Walczak BE, McCulloch PC, Kang RW, Zelazny A, Tedeschi F, Cole BJ. Abnormal findings on knee magnetic resonance imaging in asymptomatic NBA players. *J Knee Surg.* 2008;21(1):27–33. <https://doi.org/10.1055/s-0030-1247788>.
11. Hirshorn KC, Cates T, Gillogly S. Magnetic resonance imaging–documented chondral injuries about the knee in college football players: 3-year National Football League Combine Data. *Arthrosc J Arthrosc Relat Surg.* 2010;26(9):1237–40. <https://doi.org/10.1016/j.arthro.2010.01.025>.
12. Kohn D. Arthroscopy in acute injuries of anterior cruciate-deficient knees: fresh and old intra-articular lesions. *Arthrosc J Arthrosc Relat Surg.* 1986;2(2):98–102. [https://doi.org/10.1016/s0749-8063\(86\)80022-6](https://doi.org/10.1016/s0749-8063(86)80022-6).
13. Houck DA, Kraeutler MJ, Belk JW, Frank RM, McCarty EC, Bravman JT. Do focal chondral defects of the knee increase the risk for progression to osteoarthritis? A review of the literature. *Orthop J Sports Med.* 2018;6(10):2325967118801931. <https://doi.org/10.1177/2325967118801931>.
14. Cook J, Khan K, Kiss Z, Griffiths L. Patellar tendinopathy in junior basketball players: a controlled clinical and ultrasonographic study of 268 patellar tendons in players aged 14–18 years. *Scand J Med Sci Sports.* 2000;10(4):216–20. <https://doi.org/10.1034/j.1600-0838.2000.010004216.x>.
15. Gomoll A, Minas T, Farr J, Cole B. Treatment of chondral defects in the patellofemoral joint. *J Knee Surg.* 2010;19(04):285–95. <https://doi.org/10.1055/s-0030-1248121>.
16. Spindler KP, Schils JP, Bergfeld JA, et al. Prospective study of osseous, articular, and meniscal lesions in recent anterior cruciate ligament tears by magnetic resonance imaging and arthroscopy. *Am J Sports Med.* 1993;21(4):551–7. <https://doi.org/10.1177/036354659302100412>.
17. Guettler JH, Demetropoulos CK, Yang KH, Jurist KA. Osteochondral defects in the human knee. *Am J Sports Med.* 2004;32(6):1451–8. <https://doi.org/10.1177/0363546504263234>.
18. Recht M, Bobic V, Burstein D, et al. Magnetic resonance imaging of articular cartilage. *Clin Orthop Relat R.* 2001;391:S379–96. <https://doi.org/10.1097/00003086-200110001-00035>.
19. Yuen J, Hung J, Wiggermann V, et al. Multi-echo GRE imaging of knee cartilage. *J Magn Reson Imaging.* 2017;45(5):1502–13. <https://doi.org/10.1002/jmri.25438>.
20. Gold GE, Chen CA, Koo S, Hargreaves BA, Bangertner NK. Recent advances in MRI of articular cartilage. *AJR Am J Roentgenol.* 2009;193(3):628–38. <https://doi.org/10.2214/ajr.09.3042>.
21. Crema MD, Roemer FW, Marra MD, et al. Articular cartilage in the knee: current MR imaging techniques and applications in clinical practice and research. *Radiographics.* 2011;31(1):37–61. <https://doi.org/10.1148/rg.311105084>.
22. BRITTBORG M, WINALSKI CS. Evaluation of cartilage injuries and repair. *J Bone Joint Surg Am.* 2003;85:58–69. <https://doi.org/10.2106/00004623-200300002-00008>.
23. Outerbridge R. The etiology of chondromalacia patellae. *J Bone Joint Surg Br.* 1961;43-B(4):752–7. <https://doi.org/10.1302/0301-620x.43b4.752>.
24. Wernecke C, Braun HJ, Dragoo JL. The effect of intra-articular corticosteroids on articular cartilage. *Orthop J Sports Med.* 2015;3(5):2325967115581163. <https://doi.org/10.1177/2325967115581163>.
25. Messner K, Maletius W. The long-term prognosis for severe damage to weight-bearing cartilage in the knee: a 14-year clinical and radiographic follow-up in 28 young athletes. *Acta Orthop Scand.* 2009;67(2):165–8. <https://doi.org/10.3109/17453679608994664>.
26. Krych AJ, Pareek A, King AH, Johnson NR, Stuart MJ, Williams RJ. Return to sport after the surgical management of articular cartilage lesions in the knee: a meta-analysis. *Knee Surg Sports Traumatol Arthrosc.* 2017;25(10):3186–96. <https://doi.org/10.1007/s00167-016-4262-3>.
27. Cole BJ, Farr J. Putting it all together. *Operative Tech Orthop.* 2001;11(2):151–4. [https://doi.org/10.1016/s1048-6666\(01\)80025-2](https://doi.org/10.1016/s1048-6666(01)80025-2).
28. Cameron JI, Pulido PA, McCauley JC, Bugbee WD. Osteochondral allograft transplantation of the femoral trochlea. *Am J Sports Med.* 2016;44(3):633–8. <https://doi.org/10.1177/0363546515620193>.
29. Briggs DT, Sadr KN, Pulido PA, Bugbee WD. The use of osteochondral allograft transplantation for primary treatment of cartilage lesions in the

- knee. *Cartilage*. 2015;6(4):203–7. <https://doi.org/10.1177/1947603515595072>.
30. von Keudell A, Han R, Bryant T, Minas T. Autologous chondrocyte implantation to isolated Patella cartilage defects. *Cartilage*. 2017;8(2):146–54. <https://doi.org/10.1177/1947603516654944>.
 31. Ebert JR, Schneider A, Fallon M, Wood DJ, Janes GC. A comparison of 2-year outcomes in patients undergoing tibiofemoral or patellofemoral matrix-induced autologous chondrocyte implantation. *Am J Sports Med*. 2017;45(14):3243–53. <https://doi.org/10.1177/0363546517724761>.
 32. Gomoll AH, Gillogly SD, Cole BJ, et al. Autologous chondrocyte implantation in the Patella. *Am J Sports Med*. 2014;42(5):1074–81. <https://doi.org/10.1177/0363546514523927>.
 33. Rosso F, Rossi R, Governale G, et al. Tibial tuberosity Anteromedialization for patellofemoral chondral disease: prognostic factors. *Am J Sports Med*. 2017;45(7):1589–98. <https://doi.org/10.1177/0363546517690387>.
 34. Ward BD, Lubowitz JH. Basic knee arthroscopy part 4: chondroplasty, meniscectomy, and cruciate ligament Evaluation. *Arthrosc Tech*. 2013;2(4):e507–8. <https://doi.org/10.1016/j.eats.2013.07.011>.
 35. Steadman RJ, Rodkey WG, Briggs KK. Microfracture. *Cartilage*. 2010;1(2):78–86. <https://doi.org/10.1177/1947603510365533>.
 36. Holt K, Sorhaindo M, Coady C, Wong I. Arthroscopic Treatment of Medial Femoral Knee Osteochondral Defect Using Subchondroplasty and Chitosan-Based Scaffold. *Arthrosc Tech*. 2019;8. (Arthroscopy 18 2002):e413–8. <https://doi.org/10.1016/j.eats.2018.11.022>.
 37. Mithoefer K, McAdams T, Williams RJ, Kreuz PC, Mandelbaum BR. Clinical efficacy of the microfracture technique for articular cartilage repair in the knee. *Am J Sports Med*. 2009;37(10):2053–63. <https://doi.org/10.1177/0363546508328414>.
 38. Tahta M, Akkaya M, Gursoy S, Isik C, Bozkurt M. Arthroscopic treatment of osteochondral lesions of the talus: nanofracture versus hyaluronic acid-based cell-free scaffold with concentration of autologous bone marrow aspirate. *J Orthop Surg-hong K*. 2017;25(2):2309499017717870. <https://doi.org/10.1177/2309499017717870>.
 39. Mancò A, Goderecci R, Rughetti A, et al. Microfracture versus microfracture and platelet-rich plasma: arthroscopic treatment of knee chondral lesions. A two-year follow-up study. *Joints*. 2016;04(03):142–7. <https://doi.org/10.11138/jts/2016.4.3.142>.
 40. Arshi A, Fabricant PD, Go DE, Williams RJ, McAllister DR, Jones KJ. Can biologic augmentation improve clinical outcomes following microfracture for symptomatic cartilage defects of the knee? A Systematic Review. *Cartilage*. 2018;9(2):146–55. <https://doi.org/10.1177/1947603517746722>.
 41. Cotter EJ, Hannon CP, Lansdown DA, Frank RM, Waterman B, Cole BJ. Clinical Outcomes of Multiple Osteochondral Allograft Transplantation of the Knee: An Analysis of Snowman Technique and Multifocal Lesions. *Orthop J Sports Med*. 2018;6(7_suppl4):2325967118S0009. <https://doi.org/10.1177/2325967118S0009>.
 42. Pisanu G, Cottino U, Rosso F, et al. Large osteochondral allografts of the knee: surgical technique and indications. *Joints*. 2018;06(01):042–53. <https://doi.org/10.1055/s-0038-1636925>.
 43. Cole BJ, Pascual-Garrido C, Grumet RC. Surgical management of articular cartilage defects in the knee. *J Bone Joint Surg Am*. 2009;91(7):1778–90.
 44. Godin JA, Sanchez G, Cinque ME, Chahla J, Kennedy NI, Provencher MT. Osteochondral allograft transplantation for treatment of medial femoral condyle defect. *Arthrosc Tech*. 2017;6(4):e1239–44. <https://doi.org/10.1016/j.eats.2017.04.010>.
 45. Brittberg M, Lindahl A, Nilsson A, Ohlsson C, Isaksson O, Peterson L. Treatment of deep cartilage defects in the knee with autologous chondrocyte transplantation. *New Engl J Med*. 1994;331(14):889–95. <https://doi.org/10.1056/nejm199410063311401>.
 46. Bartlett W, Skinner J, Gooding C, et al. Autologous chondrocyte implantation versus matrix-induced autologous chondrocyte implantation for osteochondral defects of the knee: a prospective, randomised study. *J Bone Joint Surg Br*. 2005;87-B(5):640–5. <https://doi.org/10.1302/0301-620x.87b5.15905>.
 47. Minas T, Ogura T, Headrick J, Bryant T. Autologous chondrocyte implantation “Sandwich” technique compared with autologous bone grafting for deep osteochondral lesions in the knee. *Am J Sports Med*. 2018;46(2):322–32. <https://doi.org/10.1177/0363546517738000>.
 48. Salzmann GM, Calek A-K, Preiss S. Second-generation autologous minced cartilage repair technique. *Arthrosc Tech*. 2017;6(1):e127–31. <https://doi.org/10.1016/j.eats.2016.09.011>.
 49. Niethammer TR, Pietschmann MF, Horng A, et al. Graft hypertrophy of matrix-based autologous chondrocyte implantation: a two-year follow-up study of NOVOCART 3D implantation in the knee. *Knee Surg Sports Traumatol Arthrosc*. 2014;22(6):1329–36. <https://doi.org/10.1007/s00167-013-2454-7>.
 50. Massen F, Inauen C, Harder L, Runer A, Preiss S, Salzmann G. One-step autologous minced cartilage procedure for the treatment of knee joint chondral and osteochondral lesions: a series of 27 patients with 2-year follow-up. *Orthop J Sports Med*. 2019;7(6):2325967119853773. <https://doi.org/10.1177/2325967119853773>.
 51. Mestriner A, Ackermann J, Gomoll AH. Patellofemoral cartilage repair. *Curr Rev Musculoskelet Med*. 2018;11(2):188–200. <https://doi.org/10.1007/s12178-018-9474-3>.
 52. Edwards PK, Ackland T, Ebert JR. Clinical rehabilitation guidelines for matrix-induced autologous chondrocyte implantation on the tibiofemoral joint. *J Orthop Sports Phys Ther*. 2013;44(2):102–19. <https://doi.org/10.2519/jospt.2014.5055>.
 53. Minhas SV, Kester BS, Larkin KE, Hsu WK. The effect of an orthopaedic surgical procedure

- in the National Basketball Association. *Am J Sports Med.* 2016;44(4):1056–61. <https://doi.org/10.1177/0363546515623028>.
54. Kraeutler MJ, Belk JW, Purcell JM, McCarty EC. Microfracture versus autologous chondrocyte implantation for articular cartilage lesions in the knee: a systematic review of 5-year outcomes. *Am J Sports Med.* 2018;46(4):995–9. <https://doi.org/10.1177/0363546517701912>.
 55. Pellegrino M, Trinchese E, Bisaccia M, et al. Long-term outcome of grade III and IV chondral injuries of the knee treated with Steadman microfracture technique. *Clin Cases Miner Bone Metab.* 2016;13(3):237–40. <https://doi.org/10.11138/ccmbm/2016.13.3.237>.
 56. Schallmo MS, Singh SK, Barth KA, Freshman RD, Mai HT, Hsu WK. A cross-sport comparison of performance-based outcomes of professional athletes following primary microfracture of the knee. *Knee.* 2018;25(Am J Sports Med 14 1986):692–8. <https://doi.org/10.1016/j.knee.2018.04.008>.
 57. Namdari S, Baldwin K, Anakwenze O, Park M-J, Huffman RG, Sennett BJ. Results and performance after microfracture in National Basketball Association Athletes. *Am J Sports Med.* 2009;37(5):943–8. <https://doi.org/10.1177/0363546508330150>.
 58. Harris JD, Walton DM, Erickson BJ, et al. Return to sport and performance after microfracture in the knees of National Basketball Association Players. *Orthop J Sports Med.* 2013;1(6):2325967113512759. <https://doi.org/10.1177/2325967113512759>.
 59. Cernyik DL, Lewullis GE, Joves BC, Palmer MP, Tom JA. Outcomes of microfracture in professional basketball players. *Knee Surg Sports Traumatol Arthrosc.* 2009;17(9):1135–9. <https://doi.org/10.1007/s00167-009-0765-5>.
 60. Pareek A, Reardon PJ, Maak TG, Levy BA, Stuart MJ, Krych AJ. Long-term outcomes after osteochondral autograft transfer: a systematic review at mean follow-up of 10.2 years. *Arthrosc J Arthrosc Relat Surg.* 2016;32(6):1174–84. <https://doi.org/10.1016/j.arthro.2015.11.037>.
 61. Pareek A, Reardon PJ, Macalena JA, et al. Osteochondral autograft transfer versus microfracture in the knee: a meta-analysis of prospective comparative studies at midterm. *Arthrosc J Arthrosc Relat Surg.* 2016;32(10):2118–30. <https://doi.org/10.1016/j.arthro.2016.05.038>.
 62. Baltzer AW, Ostapczuk MS, Terheiden HP, Merk HR. Good short- to medium-term results after osteochondral autograft transplantation (OAT) in middle-aged patients with focal, non-traumatic osteochondral lesions of the knee. *Orthop Traumatol Surg Res.* 2016;102(7):879–84. <https://doi.org/10.1016/j.otsr.2016.06.004>.
 63. Campbell AB, Pineda M, Harris JD, Flanigan DC. Return to sport after articular cartilage repair in athletes' knees: a systematic review. *Arthrosc J Arthrosc Relat Surg.* 2016;32(4):651–68.e1. <https://doi.org/10.1016/j.arthro.2015.08.028>.
 64. Kirsch JM, Thomas JR, Khan M, Townsend WA, Lawton JN, Bedi A. Return to play after osteochondral autograft transplantation of the capitellum: a systematic review. *Arthrosc J Arthrosc Relat Surg.* 2017;33(7):1412–20.e1. <https://doi.org/10.1016/j.arthro.2017.01.046>.
 65. Assenmacher AT, Pareek A, Reardon PJ, Macalena JA, Stuart MJ, Krych AJ. Long-term outcomes after osteochondral allograft: a systematic review at long-term follow-up of 12.3 years. *Arthrosc J Arthrosc Relat Surg.* 2016;32(10):2160–8. <https://doi.org/10.1016/j.arthro.2016.04.020>.
 66. Familiari F, Cinque ME, Chahla J, et al. Clinical outcomes and failure rates of osteochondral allograft transplantation in the knee: a systematic review. *Am J Sports Med.* 2018;46(14):3541–9. <https://doi.org/10.1177/0363546517732531>.
 67. Frank RM, Lee S, Levy D, et al. Osteochondral allograft transplantation of the knee: analysis of failures at 5 years. *Am J Sports Med.* 2017;45(4):864–74. <https://doi.org/10.1177/0363546516676072>.
 68. Nielsen SE, McCauley JC, Pulido PA, Bugbee WD. Return to sport and recreational activity after osteochondral allograft transplantation in the knee. *Am J Sports Med.* 2017;45(7):1608–14. <https://doi.org/10.1177/0363546517694857>.
 69. Crawford ZT, Schumaier AP, Glogovac G, Grawe BM. Return to sport and sports-specific outcomes after osteochondral allograft transplantation in the knee: a systematic review of studies with at least 2 years' mean follow-up. *Arthrosc J Arthrosc Relat Surg.* 2019;35:1880. <https://doi.org/10.1016/j.arthro.2018.11.064>.
 70. Krych AJ, Robertson CM, Williams RJ, Group C. Return to athletic activity after osteochondral allograft transplantation in the knee. *Am J Sports Med.* 2012;40(5):1053–9. <https://doi.org/10.1177/0363546511435780>.
 71. Balazs GC, Wang D, Burge AJ, Sinatro AL, Wong AC, Williams RJ. Return to play among elite basketball players after osteochondral allograft transplantation of full-thickness cartilage lesions. *Orthop J Sports Med.* 2018;6(7):2325967118786941. <https://doi.org/10.1177/2325967118786941>.
 72. Pestka JM, Feucht MJ, Porichis S, Bode G, Südkamp NP, Niemeyer P. Return to sports activity and work after autologous chondrocyte implantation of the knee. *Am J Sports Med.* 2016;44(2):370–7. <https://doi.org/10.1177/0363546515614578>.
 73. Nawaz SZ, Bentley G, Briggs TWR, Carrington RWJ, Skinner JA, Gallagher KR, Dhinsa BS. Autologous chondrocyte implantation in the knee: mid-term to long-term results. 2014;96(10):824–30. <https://doi.org/10.2106/JBJS.L.01695>.
 74. Zaffagnini S, Vannini F, Martino DA, et al. Low rate of return to pre-injury sport level in athletes after cartilage surgery: a 10-year follow-up study. *Knee Surg Sports Traumatol Arthrosc.* 2018;27:1–9. <https://doi.org/10.1007/s00167-018-5255-1>.



Management of Knee Injuries in Adolescent Basketball Players

33

Bonnie P. Gregory and Jonathan C. Riboh

33.1 Introduction

In adolescent basketball players, injuries about the knee can be divided into acute traumatic injuries and non-traumatic overuse injuries. These injuries vary in severity, treatment, and length of time away from activity. Chronic, overuse injuries are increasingly common as young athletes commit to a single sport earlier and earlier [1]. Acute traumatic knee injuries are often more severe and can be divided into ligamentous injuries, extensor mechanism injuries, meniscal injuries, and chondral injuries.

33.2 Anterior Cruciate Ligament Injuries

Anterior cruciate ligament (ACL) injury is by far the most common ligamentous injury in this population that requires surgical intervention, with higher rates in female than male athletes [2, 3]. The prevalence of ACL injuries in basketball is second

only to soccer in adolescent female athletes (4.58%). For adolescent male basketball athletes, the proportion is slightly lower (2.74%), behind football and soccer [4]. Approximately 70–80% of these injuries are non-contact cutting or pivoting injuries, with approximately 70% of patients feeling or hearing a “pop” at the time of injury. This is accompanied by subsequent pain, swelling, and instability in the knee. Often, players are able to bear weight following ACL injury. Female athletes have a twofold to fourfold higher risk of ACL injury with potential contributing factors including biomechanics, alignment, neuromuscular control, hormonal differences, and strength [5–8].

Injury history and physical examination are key to the diagnosis of acute and chronic ACL injuries. An acute knee injury followed by a knee effusion should be carefully evaluated for ligamentous stability as 63% of patients aged 7–18 years with a hemarthrosis were found to have ACL tears [9]. Although examination of an injured knee in a pediatric patient can be more difficult than that of an adult, it is important to assess active and passive range of motion, joint laxity in all planes, presence of an effusion, and pain with palpation of the joint line, physal plate, and collateral ligaments [10]. Specifically, for ACL injury, dynamic tests including the Lachman and the pivot-shift should be performed and compared to the contralateral side. The Lachman test is the most useful diagnostic test for acute ACL injuries [11]. To perform this

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test, the knee is flexed to 30°, and the distal femur is held securely by one hand, while the examiner translates the tibia anteriorly with the other hand. The lack of a solid endpoint and the sense of increased anterior translation compared to contralateral side indicate a positive Lachman test and the presence of ACL injury. The pivot-shift test is pathognomonic and specific for ACL injury; however, it can be difficult to execute in an acutely injured, swollen, and painful knee. The pivot-shift test begins with the injured hip slightly abducted and the knee in full extension. The examiner then flexes the knee while applying a valgus and internal rotational load. A positive test occurs when the tibia reduces posteriorly (from the anteriorly subluxated position) with a visible shift seen at the lateral joint line, as the knee is brought from extension into flexion [12, 13].

Standard radiographs are useful in the acute setting to rule out fracture. The most common fracture noted is the Segond fracture, or small fleck of bone off the proximal lateral tibial plateau that represents avulsion of the lateral capsular ligament, or anterolateral ligament. Tibial spine avulsion fractures can be seen on plain radiographs, and should be carefully identified as their treatment paradigm is somewhat different than ligamentous ACL injuries. Additionally, in skeletally immature patients, radiographs should be evaluated for presence of open physes, which have implications for surgical reconstruction options. When skeletal maturity is in question, clinicians can obtain radiographs of the left hand and wrist to establish the bone age of the patient [14, 15]. Although an MRI is not required for the diagnosis of an ACL tear, it is routinely obtained to assess for concomitant ligamentous injury, meniscal injury, and chondral injury to assist in surgical planning.

The treatment of ACL tears in the adolescent athlete is primarily surgical. Although non-surgical treatment including rehabilitation, strengthening, activity modification, and functional bracing is possible, in general it is not indicated for young, active athletes with a desire to return to sport [16]. The risk of recurrent instability, chondral damage, meniscal injuries, and arthritis with non-operative management of ACL

tears generally make surgical treatment a more attractive option in young patients [10, 17]. There are many different techniques for ACL reconstruction, as well as multiple possible graft types, including autologous bone-patellar tendon-bone, autologous hamstring tendons, autologous quadriceps tendon, and allograft tissue. Autograft tissue is most often used in adolescent as multiple studies have shown significantly higher allograft failure rates (20% vs. 6% in autograft) in patients under 18 year of age [18, 19]. Additionally, recent studies have shown increased failure rate of hamstring autograft in the adolescent athlete population as compared to BTB and quadriceps tendon grafts [20, 21].

A decision to proceed with surgical management of ACL tears in the adolescent athlete requires special attention to surgical technique, graft type, and presence of open physes [10, 22]. Multiple studies have shown risk of growth disturbance or arrest with large tunnels passing through the growth plate [23–28]. As a result, various techniques have been developed to minimize risk of physeal injury in skeletally immature patients. For skeletally immature patient, Tanner stage 1 or 2, a physeal-sparing approach to ACL reconstruction is preferred utilizing iliotibial band autograft [29, 30]. Alternatively, intra-articular, all-epiphyseal ACL reconstruction techniques with hamstring autograft have been used to better restore intra-articular anatomy; however, a risk of physeal injury persists with these techniques [31–33]. For athletes nearing skeletal maturity, or Tanner stage 3, partial (physeal respecting on the femur and transphyseal on tibia) or fully transphyseal techniques with all soft tissue autografts (often hamstring) are utilized. These techniques pay special attention to avoiding hardware placement across the physis, minimizing tunnel size and obliquity across the growth plate to ensure vertical angulation and limit area of physeal impact (ideally <5%) and decrease risk of growth disturbance [34–37]. Finally, for athletes nearly at skeletal maturity, adult ACL reconstruction techniques can be utilized with the similar graft type (BTB autograft, quadriceps tendon autograft, or hamstring autograft) and surgical technique (all-inside, antero-medial, or transtibial placement) options [10, 38].

Fact Box 1: Surgical Decision-Making: ACL Reconstruction

Status of growth plates	Tanner stage	ACL reconstruction
Open	1,2	Physéal sparing <ul style="list-style-type: none"> • Micheli ACL reconstruction with ITB autograft • Intra-articular, all-epiphyseal with soft tissue autograft ACL reconstruction
Open	3	Physéal-respecting <ul style="list-style-type: none"> • Partial transphyseal (femoral all-epiphyseal, tibial transphyseal) • Transphyseal with focus on small, vertical tunnels • Avoidance of implant or bone blocks across the physis
Nearing/at skeletal maturity	4,5	Physéal-ignoring <ul style="list-style-type: none"> • Adult ACL reconstruction • Graft and technique of choice

In lieu of ACL tears, skeletally immature athletes, most commonly age 8–14 years, can sustain tibial spine or eminence fractures via the same injury mechanism with an estimated frequency of 3 in 100,000 [39]. Similar to ACL tears, the athletes who sustain these injuries develop an immediate effusion, pain, and instability of the knee. Unlike the purely ligamentous ACL injuries, these patients are often unable to bear weight and hold the knee in a flexed position with limited ROM [40]. Standard radiographs of the knee (AP and lateral) often demonstrate the displaced tibial spine fragment and are often adequate for diagnosis and treatment. If advanced imaging is indicated and concomitant injury is suspected, MRI is often utilized as the radiation exposure from CT scan is a concern in this age group [40, 41]. Classification of tibial spine fractures includes the initial description of type I–III fractures and addition of type IV fracture [40, 42, 43]. Type I fractures are nondisplaced fractures with excellent bony apposition, which can be treated non-operatively with long-leg casting for 4–6 weeks, followed by progressive weight-bearing, range of motion, and eventual strengthening [40, 44]. Type II represents a partial displaced fracture, with superior displacement of the anterior aspect of the fragment, but maintenance of an intact posterior hinge, creating a beak-like appearance of the fracture on lateral radiographs. Occasionally type II fractures can be treated non-operatively with the reduction of fracture and long-leg casting, if displacement in <5 mm.

Type III fractures are completely displaced fracture, with subtype IIIA involving fracture of only the ACL insertion, while type IIIB are displaced and rotated fracture. Type IV fractures consist of displaced and comminuted fragments. For type II fracture unable to be adequately reduced, as well as type III and IV fractures, surgical intervention is indicated [40]. Surgical management consists of open or arthroscopic reduction and internal fixation of the fracture (Fig. 33.1). Knee arthroscopy is often indicated to evaluate for any entrapped meniscal or concomitant meniscal and chondral or ligamentous injury. Up to 40% of these injuries have associated meniscal, collateral ligament, and chondral injuries which can impact treatment [45]. These injuries can be complicated by loss of motion, arthrofibrosis, growth arrest, and persistent ACL laxity. Postoperative rehabilitation of these injuries is similar to that of nonsurgical patients. The knee is placed in a hinged knee immobilizer, locked in extension with toe-touch weight-bearing for the first 4 weeks, with progression to full weight-bearing by 6 weeks. The brace is discontinued at around the same time when full extension is achieved. Physical therapy is initiated at 2 weeks post-surgery and at 6 weeks progresses with the range of motion, subsequently following an ACL reconstruction protocol [40].

Much research has been conducted on rehabilitation following ACL reconstruction in the adult population, and although protocols vary widely, most protocols aim to address range of motion, strength, and neuromuscular deficits

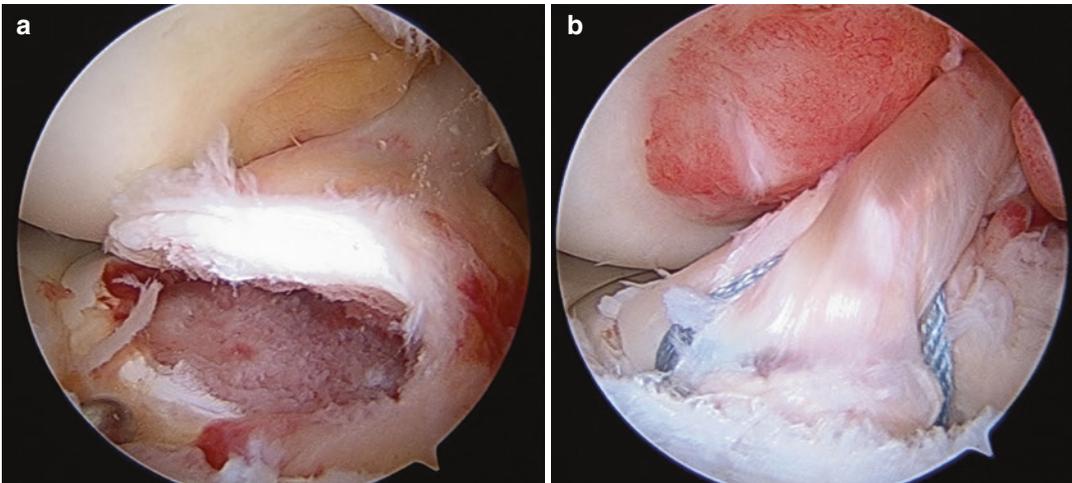


Fig. 33.1 (a) Arthroscopic image of tibial spine avulsion fracture at the time of surgery. (b) Arthroscopic image of tibial spine fracture after reduction and fixation with suture

over the course of 6–9 months to achieve limb symmetry as well as to allow appropriate graft healing time before allowing athletes to return to play. Most authors advocate that athletes complete objective return-to-sport testing prior to clearing them for play. Less research has been done on specific rehabilitation protocols and return to sport in the adolescent patients, but multiple recent studies have shown graft re-injury and contralateral re-tear rates to be remarkably common in children and adolescents, with rates as high as 35% when considering both knees [46–49]. For these reasons, these authors recommend that patients undergo supervised physical therapy following the rehabilitation protocol seen in Table 33.1 and return-to-sport testing for adolescent athletes following ACL reconstruction.

Safe return to basketball following ACL reconstruction can be considered for the adolescent athlete at a minimum of 6 months post-operatively, but optimally at least 9 months post-op, when the athlete demonstrates full range of motion and symmetric isokinetic strength and has passed a battery of physical performance tests (PPTs). Multiple studies have demonstrated significant, incremental decrease in graft re-tear rate in adolescent athletes when return to play is delayed each month after

6 months post-op [47, 50]. At our institution, an evidence-based strength, balance, and neuromuscular testing protocol is followed that includes nine common PPTs (described in Table 33.2 and Appendix), which an athlete must pass prior to being cleared to return to sport.

33.3 Posterior Cruciate and Collateral Ligament Injuries

Less commonly, adolescent athletes can sustain isolated posterior cruciate ligament (PCL), posterolateral corner (PLC), or multiligamentous knee injuries [1]. In fact, isolated PCL injuries are much less common than those with concomitant posterolateral corner (PLC) or other ligamentous injuries [51, 52]. In the case of PCL injuries, athletes often report vague knee pain, stiffness, swelling, or discomfort with activities requiring higher levels of knee flexion and less commonly recall hearing a “pop” [51]. The evaluation of these injuries is similar to that of ACL injuries, but the provider must perform specialized physical examination testing including posterior drawer, posterior sag, quadriceps active test, varus testing, valgus testing, and dial testing, in addition to a normal knee examination

Table 33.1 Pediatric ACL reconstruction rehabilitation

	Weight-bearing	Brace	Range of motion	Exercises/strength
Phase 1 0–6 weeks	0–2 weeks: PWB with crutches 3–6 weeks: WB as tolerated <i>if concomitant meniscal repair toe touch WB with crutches 0–6 weeks</i>	0–2 locked in extension Week 2 unlocked 0–90° with ambulation May remove for sleeping	As tolerated <i>Goal</i> 1 week: Symmetrical hyperextension to contralateral side 90° flexion 6 weeks: Goal Symmetrical ROM	*SLR for quad activation and prone hangs for hamstring stretching Week 2: Strengthening may begin with closed chain exercises. Leg press when ROM is greater than 90 degrees of flexion and quadriceps control improves. Hip and core strengthening involving PREs Proprioceptive exercise. Rocker board and balance exercise may commence when they are able to bear 50% or more weight. Mini squats and other balancing exercises
Phase 2 6–12 weeks	Full WB	D/C PO brace at 6 weeks	Full ROM	May begin pool therapy Continue to progress strengthening from phase 1 adding unilateral strength and balancing exercises Retrograde treadmill walking to assist with quadriceps strengthening Stretching of quadriceps, abductors, hamstrings, and calves
Phase 3 12–24 weeks	Full	Functional brace	Full	Straight ahead jogging Begin plyometric training focus on proper jumping and landing techniques Can move on to lateral jumping once proper technique for vertical has been demonstrated Week 16: Functional exercises may begin along with agility training Week 20: Full speed drills and running
Phase 4 6–8 months	Full	Functional brace	Full	Cutting, pivoting, accelerating, and decelerating drills Dynamic sport drills Advanced agility and plyometric training drills
Return to sport criteria	Full	Functional brace	Full	Symptom-free running Confidence with jumping and landing (double and single leg) Pain-free activities Ability to confidently perform cutting and lateral movements and decelerate and change directions Functional sport test with good results

Rehab is delayed with concomitant meniscal repair procedure. RTS with MD clearance, only after functional sport test and all deficiencies are addressed

battery. Additionally, a comprehensive neurovascular examination should be included, as multiligamentous knee injuries effectively represent knee dislocation events. Standard knee radiographs (AP, lateral, tunnel/notch views) should be obtained, with the addition of lateral stress radiographs to quantify posterior tibial translation. MRI should also be obtained to evaluate

extent of injury and presence of concomitant pathology. Isolated PCL injuries can often be treated non-operatively with a hinged knee brace followed by physical therapy and functional bracing with sport. Operative management of PCL, PLC, and multiligamentous knee injuries is exceedingly rare in adolescents and is beyond the scope of this text [52].

Table 33.2 Description of the neuromuscular testing protocol

Order	Task	Description	Scoring metric	No. of trials/ limb
1	Single-leg squat (flat)	See descriptions/images in Appendix	Time (s) (60 s max)	2
2	Single-leg squat (Bosu)		Time (s) (60 s max)	2
3	Single-leg squat (flat)		No. of repetitions in 60 s	2
4	Single-leg squat (Bosu)		No. of repetitions in 60 s	2
5	Quadrant hop clockwise		No. of repetitions in 30 s	2
6	Quadrant hop counterclockwise		No. of repetitions in 30 s	2
7	Single-leg hop for distance		Distance (cm)	3
8	6 m timed hop		Time (s)	3
9	Triple crossover hop for distance		Distance (cm)	3

Collateral ligament injuries—medial collateral ligament (MCL) or lateral collateral ligament (LCL)—can also occur in the adolescent basketball player. These can occur in isolation but frequently occur in conjunction with ACL, PCL, or multiligamentous knee injuries. When injured, players often complain of knee instability, pain, and difficulty with athletic maneuvers. These collateral ligament injuries are diagnosed and graded based on physical examination testing, with the examiner assessing varus and valgus stability in extension and 30° degrees of flexion. Although physical examination is very helpful in diagnosing medial collateral ligament sprains in particular, true isolated LCL sprains are rare and often represent ACL tears. As such, MRI can be obtained for diagnostic and prognostic purposes. The vast majority of these injuries, whether isolated or in conjunction with other ligamentous injuries, can be treated non-operatively with hinged knee brace with concomitant physical therapy. In fact, non-operative management of MRI-confirmed isolated grade III LCL tears in the NFL resulted in a more rapid return to play than operative management [53]. Surgical management of high-grade isolated MCL injuries remains controversial and often hinges on failure of non-operative management [54].

33.4 Extensor Mechanism Injuries

Acute extensor mechanism injuries in the adolescent basketball player consist of patellofemoral instability events, patellar sleeve avulsion fractures, and tibial tubercle avulsion fractures. Although less common than acute knee ligamentous injuries, these extensor mechanism injuries are a particular risk in basketball due to the athletic demands of jumping.

33.4.1 Patellar Instability

Unlike chronic patellofemoral instability events which are often associated with repeated low-energy patellar dislocations or subluxations in the setting of malalignment or dysplasia, acute patellofemoral instability events usually result from trauma to the knee. Patellofemoral dislocation is a common injury in the pediatric patient, with a rate of 29 per 100,000 patients from 10 to 17 years [55]. Equal by gender, these acute dislocations often occur in knee-to-knee collisions in basketball. Occasionally, they can happen in a noncontact, twisting injury with the knee extended and the foot externally rotated.

On initial physical examination, these patients develop a large effusion, tenderness to palpation at the medial patellofemoral ligament (MPFL) insertion as well as the medial facet of the patella, patellar apprehension and moving apprehension, and increased passive patellar translation laterally. The physician should also make note of limb alignment, the quadriceps angle or “Q” angle, general laxity, and presence of a “J” sign as these are risk factors for patellofemoral instability and play a role in treatment algorithms [56]. These dislocations often self-reduce, as athletes usually reflexively contract the quadriceps muscle which reduces the patella into the trochlea. As a result, these injuries can be missed as dislocations on initial presentation, so a high index of suspicion is warranted. Further work-up consists of initial knee radiographs including AP, lateral, merchant/sunrise views, as well as limb alignment series, to evaluate for patella alta (e.g., Insall-Salvati ratio or Caton Deschamps index), trochlear dysplasia (DeJour classification), and valgus limb alignment. Additionally, MRI is recommended to evaluate for concomitant chondral defects, loose bodies, location of MPFL tear, and determining tibial tubercle-trochlear groove (TT-TG) distance.

Treatment of patellofemoral instability is guided by many of the factors discussed above. First-time dislocators, with no gross malalignment or chondral defects on imaging can be treated with a short course (~6 weeks) of bracing (either hinged knee brace or patellar stabilizing brace) for comfort with concurrent therapy focused on return to full range of motion, followed by a course of physical therapy geared at strengthening of quadriceps, core and hip musculature, and neuromuscular stability. Return to play is based on strength, neuromuscular stability and comfort and can be achieved mostly by 10–12 weeks, but often much sooner. Athletes may continue to use patellar stabilizing sleeve or “J” brace for play. Operative treatment of patellofemoral instabil-

ity is considered for athletes with chondral defects, loose bodies, and multiple dislocations or subluxations.

Surgical options are myriad and, as in other surgeries about the knee in adolescents, dependent on skeletal maturity as distal femoral and proximal tibial physes as well as tibial tubercle apophysis are anatomically relevant in patellofemoral instability surgery. In skeletally immature patients with significant limb malalignment, use of surgical implants for guided growth can be utilized in isolation or in conjunction with the other procedures described below [55]. Knee arthroscopy is indicated for the evaluation of chondral surfaces and excision of loose bodies resulting from dislocations. Medial patellofemoral repair can be utilized in skeletally immature patients as it does not require bone tunnels and poses minimal risk to femoral physis; however, it is biomechanically inferior to both the native ligament and MPFL reconstruction [57]. MPFL reconstruction with hamstring auto or allograft is indicated in recurrent instability, with multiple surgical techniques and fixations methods described in the literature [55, 58]. The use of isolated lateral release was utilized historically, but is no longer considered standard of care. Lateral lengthening is generally used in conjunction with MPFL reconstruction or distal realignment procedure and is reserved for cases with irreducible lateral tilt [55, 59]. Distal realignment procedures include tibial tubercle osteotomy with anteromedialization (for elevated TTTG) or distalization (for patella alta) in skeletally mature patients as well as soft tissue reconstructions such as the modified Roux-Goldthwait, Galeazzi, and Nietosvaara techniques in the skeletally immature patient [55]. Finally, trochleoplasty is often reserved for revision cases in the setting of significant trochlear dysplasia in a skeletally mature patient, although the procedure is increasing in popularity in the USA [60].

Fact Box 2: Surgical Decision-Making: Patellofemoral Instability Treatment Options

Risk factor for instability	Open growth plates	Closed growth plates
Recurrent dislocation	MPFL repair vs. conservative management until skeletally mature	MPFL reconstruction
Valgus alignment	<ul style="list-style-type: none"> Instrumented guided growth 	Address other factors first, consider femoral osteotomy in case of miserable malalignment
Patella alta	Soft tissue distal realignment <ul style="list-style-type: none"> Modified Roux-Goldthwait Galeazzi Nietosvaara 	Tibial tubercle osteotomy with distalization
Lateralization of tibial tubercle (TT–TG > 20)	Soft-tissue distal realignment <ul style="list-style-type: none"> Modified Roux-Goldthwait Galeazzi Nietosvaara 	Tibial tubercle osteotomy with anteromedialization (AMZ)
Severe trochlear dysplasia	Conservative management until skeletally mature	Trochleoplasty

Surgical treatment is followed by a course of immobilization in a hinged knee brace of variable duration depending on the procedure performed and physical therapy focusing on restoring and improving strength and neuromuscular control. Objective return-to-play protocols in the post-operative period are scarce for these procedures, but most surgeons release athletes for return at 6 months post-op.

33.4.2 Extensor Mechanism Fractures

Patellar sleeve avulsion fractures and tibial tubercle avulsion fractures both represent rare injuries representing less than 5% of knee injuries, but can occur in the pediatric and adolescent basketball player [1]. Patellar sleeve fractures happen in slightly younger athletes, aged 8–16 years, more common in males than females, when patellar ossification is nearly complete and represent a separation of the cartilage “sleeve” from the ossific nucleus of the patella. These avulsions occur more commonly at the inferior pole, but can also happen at superior pole of the patella. These injuries result from a forceful contraction of the quadriceps muscle applied to a flexed knee, often representing a non-contact, indirect injury to the knee [61]. Athletes present with severe knee pain, swelling, large effusion, diffuse tenderness, high-riding patella in the case of distal fracture, difficulty extending their knee against

gravity and resistance, and inability to bear weight. Radiographs can confirm the diagnosis by identifying patella alta or baja with small flecks of bone adjacent to inferior or superior pole. MRI or ultrasound may be indicated if the diagnosis is unclear by physical examination and radiographs. Rarely, non-operative treatment in the form of a well-molded, long-leg cylinder cases is indicated for athletes with non-displaced fractures and an intact extensor mechanism [1, 62]. Open reduction and internal fixation followed by immobilization is indicated for the vast majority of these patients [63]. Physical therapy is commenced following immobilization, and return to play is indicated at around 6 months post-op when athlete has regained full strength, mobility, and optimized neuromuscular control. However, no established return-to-play protocol or time frame exists in the literature for these rare injuries.

Tibial tubercle avulsion fractures also represent rare injuries, comprising less than 1% of pediatric fractures, but are seen most frequently in the adolescent basketball player, more commonly in male than female athletes, aged from 12 to 15 years [64, 65]. These fractures occur through the tibial tubercle apophysis with possible extension into the proximal tibial physis, and are due to concentric contraction of the quadriceps muscles during jumping or eccentric contraction of the quadriceps during forces knee flexion [1]. Physeal closure occurs at the proximal tibia from posterior to anterior and proximal

to distal, meaning the tibial tubercle is the last to fuse which accounts for the increased risk of injury at this site in adolescent athletes as they reach skeletal maturity. Athletes present with a knee effusion, soft tissue swelling, decreased range of motion, extensor lag, tenderness at the tibial tubercle, and possibly difficulty bearing weight after experiencing a sudden onset of pain at the initiation of jumping or sprinting [65]. These fractures can be limited to the secondary ossification near the insertion of the patellar tendon or when more severe can propagate proximally through the primary ossification center and through the entire proximal tibial physis. These fractures can be associated with patellar or quadriceps tendon avulsion, meniscal tears, and preoperative compartment syndrome [66]. Standard work-up includes radiographs with an internal rotation view to bring the tibial tubercle in profile and possibly a contralateral view for comparison of the ossification centers in young patients. CT scan can be obtained to evaluate for intraarticular extension in more severe injuries. MRI is rarely used, but can be obtained to evaluate for concomitant meniscal injuries. Often, these patients are admitted for monitoring overnight after injury due to elevated risk of compartment syndrome due to injury to the nearby anterior tibial recurrent artery. Additionally, these injuries can be complicated by recurvatum deformity, leg length discrepancy, stiffness, bursitis, painful hardware, or more significant vascular injury [66].

Non-operative treatment of tibial tubercle fractures is reserved for injuries isolated to the secondary ossification with minimal displacement (<2 mm) or those fractures with acceptable alignment after closed reduction [64]. These are treated in a well-molded long-leg cast in extension for 6 weeks, followed by progressive weight-bearing and physical therapy. Operative intervention is indicated for any displaced fractures and consists of open reduction and internal fixation with possible knee arthrotomy or arthroscopy to evaluate for meniscal injury or intraarticular fracture extension. Following surgery, the patient is kept overnight for compartment checks and then kept non-weight-bearing in a long-leg cast or knee immobilizer for 4–6 weeks, followed by progressive weight-bearing, range of motion and physical therapy. Return to play is initiated

no sooner than 3 months after injury and should be considered only after full, painless range of motion, appropriate strength, and neuromuscular control are achieved.

33.5 Meniscal Injuries

In the pediatric and adolescent population acute meniscal injuries are often associated with other pathology such as ACL tears or tibial eminence fractures or can occur in the presence of a discoid meniscus. Isolated meniscal injuries are more likely to occur in younger patients with lower BMI and are more common in the lateral meniscus than medial. One study found the distribution of meniscal tear type to be: complex (28%), vertical (16%), discoid (14%), bucket-handle (14%), radial (10%), horizontal (8%), oblique (5%), fray (3%), and root detachment (2%) [67]. The most common injury mechanism is a non-contact twisting injury and athletes complain of knee pain and swelling at the time of injury and presence of mechanical symptoms (catching, locking, or giving away sensation) subsequently. On physical examination, patients may have an effusion (larger with concomitant injuries), a block to full extension or ROM (especially in the presence of a flipped bucket handle meniscus tear), medial or lateral joint line tenderness, a positive McMurray test (indicated by pain or an audible pop with flexion and internal or external rotation followed by knee extension), or a positive Thessaly test (indicated by medial or lateral joint line pain when the patient stands flat-footed on a single leg and rotates trunk with the knee flexed to 5° and 20°). Imaging of these injuries consists of plain radiographs and MRI which is the study of choice. Plain films are normal, unless a discoid meniscus is present, then radiographs can demonstrate widening of the lateral joint space and/or squaring of the lateral femoral condyle in the case of lateral discoid menisci (which are much more common than medial).

Treatment of isolated meniscal tears is dependent upon the location and character of the tear. Non-operative management in the form of functional bracing and physical therapy may be attempted in non-displaced partial tears in the red-red zone of the meniscus. Operative

treatment is indicated in the pediatric and adolescent athlete for all tears in the red-white and white-white zones of the meniscus. Meniscal repair should be attempted if at all possible, as there is a high success rate following repair as well as a high long-term morbidity rate and risk of future chondral injury with meniscectomy in this patient populations. Several studies have shown good results (80–87%) with operative treatment of meniscal tears in the pediatric population [68–70].

The presence of a torn discoid meniscus requires special consideration. Discoid menisci are present in 3–5% of the population and located laterally more commonly than medially and often are the causative structure noted in arthroscopy for isolated, symptomatic lateral meniscus tears [71, 72]. The most commonly used classification system of discoid menisci was proposed by Watanabe in 1969 and based on arthroscopic

assessment of meniscal appearance and stability [73]. Type I is a stable, complete discoid meniscus that covers the entire tibial plateau; type II is a stable, partial discoid meniscus that covers up to 80% of the tibial plateau; and type III (i.e. Wrisberg variant) is an unstable discoid meniscus with lack of any posterior meniscotibial attachment [71, 73]. On arthroscopic examination of discoid menisci, 70% had associated tears and peripheral rim instability was noted in 28% [74]. Observation of discoid meniscus lesion is appropriate for minimally symptomatic patients, such as in a patient with a painless, palpable knee clunk or a discoid meniscus found incidentally on knee arthroscopy [71]. Surgical intervention is indicated for symptomatic discoid menisci and consists of limited meniscectomy with saucerization, with emphasis placed on meniscal rim preservation and meniscal repair [71, 74, 75] (Fig. 33.2).

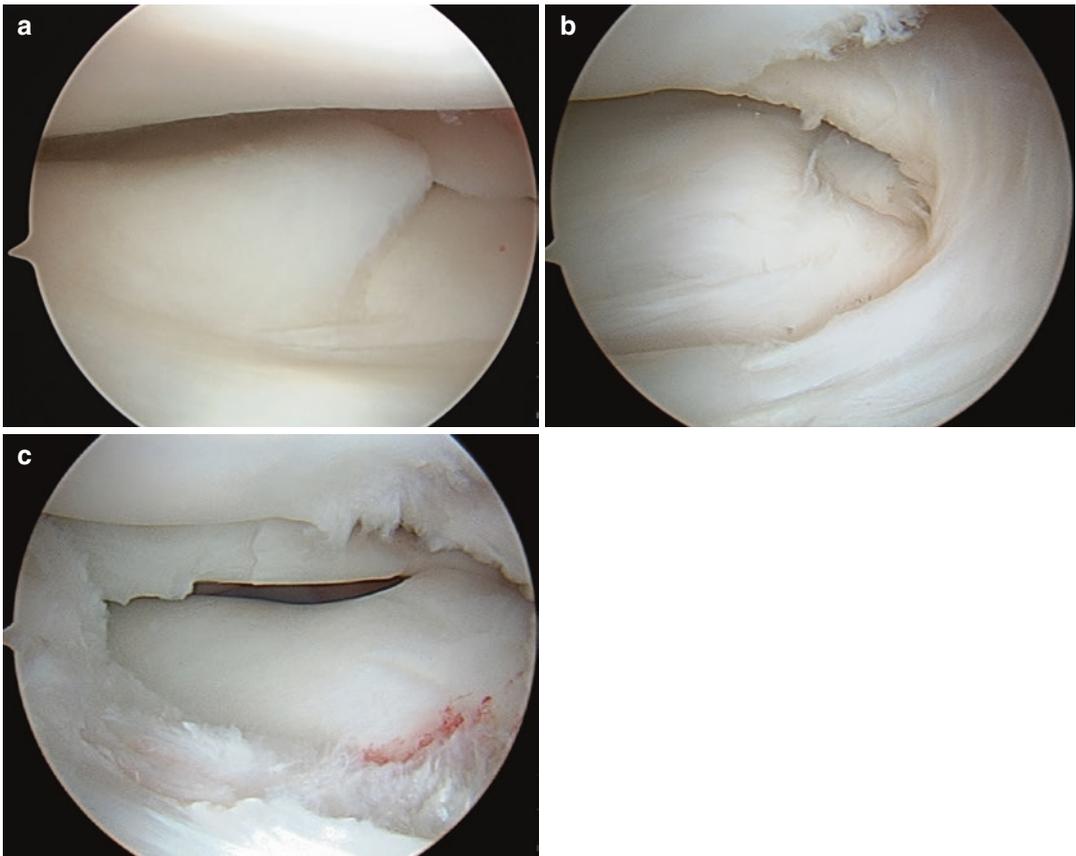


Fig. 33.2 (a) Arthroscopic image of incomplete, discoid lateral meniscus. (b) Arthroscopic image of complete discoid lateral meniscus. (c) Arthroscopic image of discoid lateral meniscus after saucerization and repair

Table 33.3 Meniscal repair, inside-out rehabilitation protocol

	Weight-bearing	Brace	ROM	Exercises
Phase I 0–2 weeks	Partial weight-bearing with crutches	Locked in full extension for sleeping and all activities Off for exercises and hygiene	0–90° when non-weight-bearing	Heel slides, quad sets, patellar mobs, SLR, SAQ <i>No weight-bearing with flexion > 90°</i>
Phase II 2–8 weeks	2–4 weeks: 50% WB with crutches 4–8 weeks: Progress to full WB	2–6 weeks: Unlocked 0–90° off at night Discontinue brace at 6 weeks	As tolerated	Addition of heel raises, total gym (closed chain), terminal knee extensions Activities with brace until 6 weeks; then without brace as tolerated <i>No weight-bearing with flexion > 90°</i>
Phase III 8–12 weeks	Full	None	Full	Progress closed chain activities Begin hamstring work, lunges/leg press 0–90°, proprioception exercises, balance/core/hip/glutes Begin stationary bike
Phase IV 12–20 weeks	Full	None	Full	Progress phase III exercises and functional activities: Single-leg balance, core, glutes, eccentric hamstrings, elliptical, and bike Swimming okay at 16 weeks
Phase V >20 weeks	Full	None	Full	Advance to sport-specific drills and running/jumping once cleared by MD

Postoperative protocol for partial meniscectomy or saucerization without meniscal repair consists of immediate weight-bearing, followed by the initiation of physical therapy at 2 weeks postoperatively, and progression to return to sport after full ROM and strength are achieved [71]. Postoperative rehabilitation for meniscal repair consist of partial weight-bearing in a hinged knee brace, locked in extension for weight-bearing and sleeping, unlocked to 90° for range of motion. Brace is continued with ROM limited to 90° for 6 weeks with progression to full weight-bearing. Physical therapy is then progressed to full range of motion with progression through neuromuscular/proprioceptive exercises and strengthening, with advancement to running, jumping, and sport-specific drills at 5–6 months and return to sports once cleared by physician beginning at 6 months (Table 33.3) [71].

33.6 Chondral and Osteochondral Injuries

Chondral injuries in the adolescent athlete can be associated with other ligamentous or meniscal injuries, as in adults. However, in this population knee osteochondritis dissecans (OCD) is a partic-

ular concern. Defined as a “focal idiopathic alteration of subchondral bone with risk for instability and disruption of adjacent cartilage that may result in premature osteoarthritis,” OCD lesions most commonly occur from age 10 to 15 years [76, 77]. Osteochondritis dissecans lesions occur most commonly in the knee, with the posterolateral aspect of the medial femoral condyle representing the most common location within the knee. Although the mechanism of OCD development is still not fully understood (with hereditary, traumatic, and vascular etiologies discussed most commonly), the cascade of injury is consistent [77]. In the initial stages, there is softening of the overlying articular cartilage with and intact surface, followed by early articular cartilage separation, then partial detachment of articular cartilage lesion, and finally osteochondral separation with loose body formation [77].

Athletes present with vague and poorly localized activity-related pain within the knee and can have recurrent effusions as well. In more advanced stages, athletes can present with mechanical symptoms such as catching or locking. On examination, athletes often have an effusion, decreased ROM, and localized tenderness to palpation in the area of the OCD. Additionally, patients can have a positive Wilson’s test which is indicated by pain

when internally rotating the tibia during extension of the knee between 90° and 30° , then relief of pain with external rotation of the tibia, due to impingement of the tibial spine against the lesion. Plain radiographs can be helpful in making the initial diagnosis, as well as monitoring healing over the course of treatment as well. The radiographic series should include weight-bearing AP, lateral, and tunnel/notch views, which allow visualization of posterior lesions that may be missed on AP views [78]. Radiographs are also used to evaluate lesion location, maturity of growth plate, parent and progeny bone radiodensity, and progeny bone fragmentation or displacement, as outlined by The Research in OsteoChondritis of the Knee (ROCK) Group [76, 79]. MRI is generally indicated in initial diagnosis to better characterize the size of the lesion, status or subchondral bone

and cartilage, stability of the lesion, and evaluate for the presence of loose bodies. However, radiographs can reliably be used for monitoring of OCD lesion healing as demonstrated in one study that showed great interrater reliability when utilizing six radiographic features: articular surface shape, boundary appearance, sclerosis, ossification, lesion size, and overall healing [76, 79]. Full classification of knee OCD lesions is outside the scope of this text but has been well outlined by the ROCK group [79, 80]. In general, prognosis for healing knee OCD lesions is better for younger patients with open distal femoral physes and worse for patients closer to skeletal maturity, with sclerosis on radiographs, presence of synovial fluid behind the lesion on MRI which indicates an unstable lesion, and location within the lateral femoral condyle or patella [76, 79, 81] (Fig. 33.3).

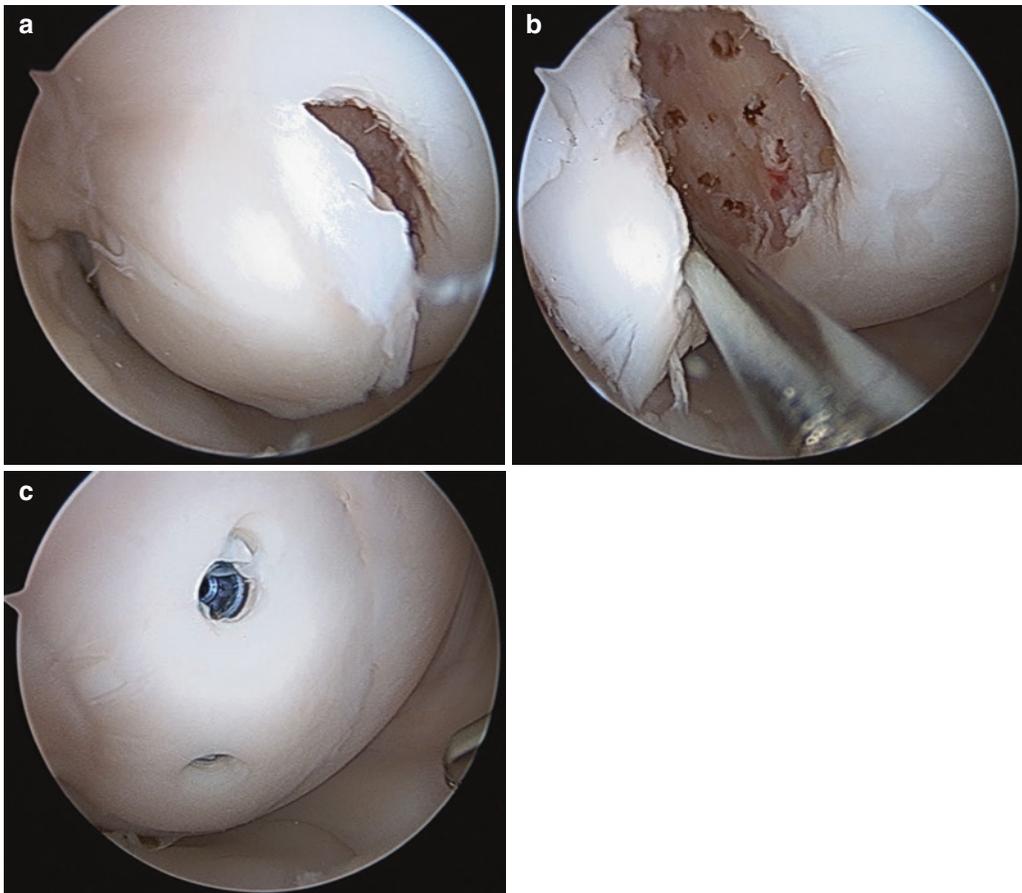


Fig. 33.3 (a) Arthroscopic image of unstable lateral femoral condyle osteochondritis dissecans (OCD) lesion. (b) Arthroscopic image of same OCD after debridement and

marrow stimulation. (c) Arthroscopic image of OCD after reduction and fixation

Table 33.4 Drilling/fixation of osteochondritis dissecans rehabilitation protocol

	Weight-bearing	ROM	Exercises/strength
0–6 weeks	NWB	Full as tolerated	Avoid WB ROM exercises Isometric quad and hamstring strengthening Stretching of quads, hams, and calves
6–12 weeks	Progress to full WB with MD consent after radiographic evaluation	Full symmetrical ROM to contralateral side	Progress strength training in normal fashion guided by pain and symptoms No high-impact activities
12 weeks: Return to sport This phase should take time and not be rushed through and guided by radiographs and MD suggestions	Full		Begin running program Begin impact strengthening Begin functional return-to-sport activities Be aware of pain and symptoms, slow rehab if they are occurring

Treatment for knee OCD lesions is based on the abovementioned factors. Non-operative management consisting of restricted weight-bearing, bracing, and close radiographic follow-up to confirm healing is indicated for stable lesions in patients with open growth plates. This treatment algorithm has shown good (50–75%) healing without fragmentation of the lesion [81]. Operative intervention is indicated for unstable lesions or lesions in older patients. Surgical treatment consists of either subchondral drilling for stable lesions on arthroscopy; open reduction and internal fixation for unstable but fixable lesions with 85% healing rates in the setting of open growth plates; and more advanced cartilage restoration or transplant procedures for large (>2 cm × 2 cm) unfixable lesions [82, 83]. Post-operative protocol varies, depending on the operative procedure performed, but for drilling of OCD lesions, it consists of a period of non-weight-bearing, followed by progression to full-weight-bearing at around 3 months post-op with initiation of impact and gradual return to functional sports activities from then (Table 33.4). Return to full sports is anticipated between 4 and 6 months, after confirmation of radiographic healing of the lesion and clearance from the physician.

33.7 Overuse Injuries

Chronic, overuse injuries of the knee are also quite common in the adolescent basketball players and often present in an acute-on-chronic

nature, persisting for months to years. Traction apophysitis of the tibial tubercle or Osgood–Schlatter syndrome, traction apophysitis of the inferior pole of the patella or Sinding–Larsen–Johansson syndrome, and patellar tendonitis or “Jumper’s knee” are the most common overuse injuries in this patient population [84]. All of these are treated non-operatively in the vast majority of cases, with operative intervention reserved for refractory cases.

Osgood–Schlatter syndrome or tibial tubercle traction apophysitis is marked by self-limited chronic pain in skeletally immature jumpers or sprinters at the anterior aspect of the knee, exacerbated by kneeling. It is more common in males than females, typically present in the 3–4 years prior to cessation of growth (male: 12–15 years, female: 8–12 years), and present bilaterally in 20–50% of patients [84]. On physical examination, athletes have an enlarged tibial tubercle, with associated soft tissue swelling and tenderness to palpation, and often have pain with resisted knee extension. Routine radiographs demonstrate irregularity and fragmentation at the tibial tubercle apophysis; MRI is rarely indicated. First-line treatment consists of rest, ice, activity modifications, NSAIDs, patellar tendon strapping or sleeves to decrease tension on the tubercle apophysis, and quadriceps stretching, with over 90% of patients achieving complete resolution of symptoms. Cast immobilization can be utilized in the case of severe symptoms, unresponsive to the conservative measure described above. Operative intervention in the form of

ossicle excision is reserved for skeletally mature patients with persistent symptoms [85]. Physical therapy focuses on stretching, addressing muscle imbalances across the knee, with gradual, symptom-based return to full participation.

Similarly, Sinding-Larsen-Johansson syndrome is an overuse injury causing anterior knee pain in the skeletally immature athlete. These athletes have tenderness to palpation over the inferior pole of the patella and may also have pain with resisted knee extensions [84]. Radiographs are most often normal, but may show spurring at the inferior pole of the patella. This syndrome is most often self-limited and responds well to activity modification, NSAIDs, and physical therapy. Rarely surgical treatment is indicated, and rarely athletes continue to have symptoms past skeletal maturity.

Fact Box 3: Mainstays of the Treatment of Overuse Injuries in Adolescents

NSAIDs
Rest and activity modification
Offloading apophyses via tendon strapping
Focused stretching program
Initiation of eccentric strengthening exercises

Patellar tendonitis or “Jumper’s knee” is another form of anterior knee pain that can present in adolescence, but can also present in adulthood and represents a degenerative, rather than inflammatory process in the tendon [86]. In the initial phases, athletes complain of focal pain in the patellar tendon following activities; however, as the disease process progresses, athletes also complain of pain during activity as well as without activity, such as with prolonged knee flexion also known as the “movie theater sign”. In the later phases, athletes also begin to experience deterioration of performance. On examination, athletes have focal tenderness over the patellar tendon and pain with resisted extension and may have a positive Basset’s sign (tenderness to palpation at the distal pole of the patella with full knee extension, but no tenderness in full flexion). Radiographs are often normal, but can show traction spurring at the inferior pole of the patella in

some cases. MRI can be obtained in chronic cases, if surgical intervention is being considered, and generally demonstrates tendon thickening, increased signal within the tendon on T1 and T2, and loss of posterior border of the fat pad in chronic cases. Patellar tendonitis is most commonly treated with activity modification, taping or patellar strapping to reduce tension across the tendon, and physical therapy. Corticosteroid injection is generally contraindicated due to risk of patellar tendon rupture. Recently, platelet-rich plasma (PRP) injections have been used in chronic cases with mixed results and are not yet standard of care [87]. Physical therapy focuses on stretching of hamstring and quadriceps muscles, eccentric muscle strengthening program, and use of other therapy modalities [88]. Return to sport is based on symptoms. Similar to other overuse injuries in the knee, surgical intervention is reserved for refractory cases. After surgical debridement, 80–90% of patients return to some level of play, but can anticipate continued aching or pain for 4–6 months following surgery and therapy.

Take Home Message

As the participation in organized team sports continued to increase and athletes continue to narrow to single sports participation at younger ages, physicians should continue to emphasize injury reduction and prevention via preseason physical examination, medical coverage at sporting events, proper coaching and training, and adequate time off from competition and sport both within a given week and over the course of the year. Although the physician’s role in diagnosis and treatment of injuries is invaluable, it is even more important to educate young athletes, parents, and coaches about the prevention of injuries as well as adequate recovery and rehabilitation prior to return to sport following an injury. This will provide young athletes the best chance for continued enjoyment of sporting activities and ensure access to the invaluable mental and physical health benefits that sport participation can provide.

Appendix

<p>Single Leg Stance</p> <p>The subject is asked to balance on a single foot for a maximum of 60 seconds. If he or she is unable to maintain the stance for the full minute, the number of seconds maintained is recorded.</p>		<p>Single Leg Stance on Bosu</p> <p>The subject is asked to balance on a single foot on a Bosu Ball for a maximum of 60 seconds.</p>			
<p>45° Single Leg Squat</p> <p>The subject is asked to balance on a single leg while performing a squat deep enough to create a 45° flexion angle at the knee. The tester pre-determines the angle of the knee with a goniometer and then places a target dowel to ensure adequate depth and alignment. The number of squats performed in 60 seconds is recorded.</p>		<p>45° Single Leg Squat on Bosu</p> <p>A Bosu Ball is placed (flat side up) under the stance leg. Then, the task is performed as described above. If the subject loses his/her balance, he/she may resume within the 60 second period.</p>			
<p>Clockwise Quadrant Hop</p> <p>The subject is asked to hop on a single leg in a clockwise direction for 30 seconds. Then, number of correct hops (at least 50% of the foot surface lands in the correct quadrant) is counted. Each quadrant is a 40cm square.</p>		<p>Counterclockwise Quadrant Hop</p> <p>Same as above, only counterclockwise direction</p>			
<p>Single Leg Hop for Distance</p> <p>The subject is asked to hop as far as possible on one foot without losing his/her balance during landing.</p>		<p>6 Meter Timed Hop</p> <p>The Subject is asked to hop, as quickly as possible, on one foot for a distance of 6 meters</p>		<p>Crossover Hop for Distance</p> <p>The subject is asked to hop as far as possible on one foot while crossing the tape measure three times</p>	

References

1. Siow HM, Cameron DB, Ganley TJ. Acute knee injuries in skeletally immature athletes. *Phys Med Rehabil Clin N Am.* 2008;19(2):319–45. <https://doi.org/10.1016/j.pmr.2007.11.005>.
2. Borowski LA, Yard EE, Fields SK, Comstock RD. The epidemiology of US high school basketball injuries, 2005–2007. *Am J Sports Med.* 2017;36(12):2328–35. <https://doi.org/10.1177/0363546508322893>.
3. Stanley LE, Kerr ZY, Dompier TP, Padua DA. Sex differences in the incidence of anterior cruciate ligament, medial collateral ligament, and meniscal injuries in collegiate and high school sports. *Am J Sports Med.* 2016;44(6):1565–72. <https://doi.org/10.1177/0363546516630927>.
4. Shea KG, Grimm NL, Ewing CK, Aoki SK. Youth sports anterior cruciate ligament and knee injury epidemiology: who is getting injured? In what sports? When? *Clin Sports Med.* 2011;30(4):691–706. <https://doi.org/10.1016/j.csm.2011.07.004>.
5. Leppänen M, Pasanen K, Kujala UM, et al. Stiff landings are associated with increased ACL injury risk in young female basketball and floorball players. *Am J Sports Med.* 2016;45(2):386–93. <https://doi.org/10.1177/0363546516665810>.
6. Leppänen M, Pasanen K, Kulmala JP, et al. Knee control and jump-landing technique in young basketball and floorball players. *Int J Sports Med.* 2016;37(04):334–8. <https://doi.org/10.1055/s-0035-1565104>.
7. Agel J, Arendt EA, Bershadsky B. Anterior cruciate ligament injury in national collegiate athletic association basketball and soccer: a 13-year review. *Am J Sports Med.* 2005;33:524–30.
8. Kernozek TW, Torry MR, Van Hoof H, Cowley H, Tanner S. Gender differences in frontal and sagittal plane biomechanics during drop landings. *Med Sci Sports Exerc.* 2005;37:1003–12.
9. Stanitski CL, Harvell JC, Fu F. Observations on acute knee hemarthrosis in children and adolescents. *J Pediatr Orthop.* 1993;13:506–10.
10. Larsen MW, Garrett WE, Delee JC, Moorman CT. Surgical management of anterior cruciate ligament injuries in patients with open physes. *J Am Acad Orthop Surg.* 2006;14(13):736–44.
11. Gurtler RA, Stine R, Torg JS. Lachman test evaluated. Quantification of a clinical observation. *Clin Orthop Relat Res.* 1987;216:141–50.
12. Fetto JF, Marshall JL. Injury to the anterior cruciate ligament producing the pivot-shift sign. *J Bone Joint Surg.* 1979;61(5):710–4.
13. Galway HR, MacIntosh DL. The lateral pivot shift: a symptom and sign of anterior cruciate ligament insufficiency. *Clin Orthop Relat Res.* 1980;147:45–50.
14. Greulich WW, Pyle SI. Radiographic atlas of skeletal development of the hand and wrist. 2nd ed. Palo Alto, CA: Stanford University Press; 1959.
15. Heyworth BE, Osei DA, Fabricant PD, et al. The shorthand bone age assessment: a simpler alternative to current methods. *J Pediatr Orthop.* 2013;33(5):569–74. <https://doi.org/10.1097/BPO.0b013e318293e5f2>.
16. Dingel A, Aoyama J, Ganley T, Shea K. Pediatric ACL tears: natural history. *J Pediatr Orthop.* 2019;39(Issue 6, Suppl 1):S47–9. <https://doi.org/10.1097/BPO.0000000000001367>.
17. Aichroth PM, Patel DV, Zorrilla P. The natural history and treatment of rupture of the anterior cruciate ligament in children and adolescents. A prospective review. *J Bone Joint Surg Br.* 2002;84(1):38–41. <https://doi.org/10.1302/0301-620x.84b1.11773>.
18. Kaeding CC, Aros B, Pedroza A, et al. Allograft versus autograft anterior cruciate ligament reconstruction: predictors of failure from a MOON prospective longitudinal cohort. *Sports Health.* 2011;3(1):73–81. <https://doi.org/10.1177/1941738110386185>.
19. Engelman GH, Carry PM, Hitt KG, Polousky JD, Vidal AF. Comparison of allograft versus autograft anterior cruciate ligament reconstruction graft survival in an active adolescent cohort. *Am J Sports Med.* 2014;42(10):2311–8. <https://doi.org/10.1177/0363546514541935>.
20. Faunø P, Rahr-Wagner L, Lind M. Risk for revision after anterior cruciate ligament reconstruction is higher among adolescents: results from the Danish Registry of Knee Ligament Reconstruction. *Orthop J Sports Med.* 2014;2(10):2325967114552405. <https://doi.org/10.1177/2325967114552405>.
21. Persson A, Fjeldsgaard K, Gjertsen J-E, et al. Increased risk of revision with hamstring tendon grafts compared with patellar tendon grafts after anterior cruciate ligament reconstruction: a study of 12,643 patients from the Norwegian Cruciate Ligament Registry, 2004–2012. *Am J Sports Med.* 2014;42(2):285–91. <https://doi.org/10.1177/0363546513511419>.
22. Milewski MD, Beck NA, Lawrence JT, Ganley TJ. Anterior cruciate ligament reconstruction in the young athlete: a treatment algorithm for the skeletally immature. *Clin Sports Med.* 2011;30(4):801–10. <https://doi.org/10.1016/j.csm.2011.08.001>.
23. CAMPBELL CJ, GRISOLIA A, ZANCONATO G. The effects produced in the cartilaginous epiphyseal plate of immature dogs by experimental surgical traumata. *J Bone Joint Surg.* 1959;41-A:1221–42.
24. Shifflett GD, Green DW, Widmann RF, Marx RG. Growth arrest following ACL reconstruction with hamstring autograft in skeletally immature patients: a review of 4 cases. *J Pediatr Orthop.* 2016;36(4):355–61. <https://doi.org/10.1097/BPO.0000000000000466>.
25. Chotel F, Henry J, Seil R, Chouteau J, Moyen B, Bérard J. Growth disturbances without growth arrest after ACL reconstruction in children. *Knee Surg Sports Traumatol Arthrosc.* 2010;18(11):1496–500. <https://doi.org/10.1007/s00167-010-1069-5>.

26. Kocher MS, Saxon HS, Hovis WD, Hawkins RJ. Management and complications of anterior cruciate ligament injuries in skeletally immature patients: survey of the Herodicus Society and The ACL Study Group. *J Pediatr Orthop*. 2002;22(4):452–7.
27. Shea KG, Apel PJ, Pfeiffer RP, Traughber PD. The anatomy of the proximal tibia in pediatric and adolescent patients: implications for ACL reconstruction and prevention of physeal arrest. *Knee Surg Sports Traumatol Arthrosc*. 2007;15(4):320–7. <https://doi.org/10.1007/s00167-006-0171-1>.
28. Shea KG, Grimm NL, Belzer JS. Volumetric injury of the distal femoral physis during double-bundle ACL reconstruction in children: a three-dimensional study with use of magnetic resonance imaging. *J Bone Joint Surg Am*. 2011;93(11):1033–8. <https://doi.org/10.2106/JBJS.J.01047>.
29. Ellison AE. Distal iliotibial-band transfer for anterolateral rotatory instability of the knee. *J Bone Joint Surg*. 1979;61(3):330–7.
30. Kocher MS, Garg S, Micheli LJ. Physeal sparing reconstruction of the anterior cruciate ligament in skeletally immature prepubescent children and adolescents. *J Bone Joint Surg*. 2005;87(11):2371–9. <https://doi.org/10.2106/JBJS.D.02802>.
31. McCarthy MM, Graziano J, Green DW, Cordasco FA. All-epiphyseal, all-inside anterior cruciate ligament reconstruction technique for skeletally immature patients. *Arthrosc Tech*. 2012;1(2):e231–9. <https://doi.org/10.1016/j.eats.2012.08.005>.
32. Cordasco FA, Mayer SW, Green DW. All-inside, all-epiphyseal anterior cruciate ligament reconstruction in skeletally immature athletes: return to sport, incidence of second surgery, and 2-year clinical outcomes. *Am J Sports Med*. 2017;45(4):856–63. <https://doi.org/10.1177/0363546516677723>.
33. Lawrence JTR, Bowers AL, Belding J, Cody SR, Ganley TJ. All-epiphyseal anterior cruciate ligament reconstruction in skeletally immature patients. *Clin Orthop Relat Res*. 2010;468(7):1971–7. <https://doi.org/10.1007/s11999-010-1255-2>.
34. Kocher MS, Smith JT, Zoric BJ, Lee B, Micheli LJ. Transphyseal anterior cruciate ligament reconstruction in skeletally immature pubescent adolescents. *J Bone Joint Surg Am*. 2007;89(12):2632–9. <https://doi.org/10.2106/JBJS.F.01560>.
35. Pierce TP, Issa K, Festa A, Scillia AJ, McInerney VK. Pediatric anterior cruciate ligament reconstruction: a systematic review of transphyseal versus physeal-sparing techniques. *Am J Sports Med*. 2017;45(2):488–94. <https://doi.org/10.1177/0363546516638079>.
36. Domzalski M, Karauda A, Grzegorzewski A, Lebidzinski R, Zabierek S, Synder M. Anterior cruciate ligament reconstruction using the transphyseal technique in prepubescent athletes: mid-term, prospective evaluation of results. *Arthroscopy*. 2016;32(6):1141–6. <https://doi.org/10.1016/j.arthro.2015.11.045>.
37. Cruz AI, Lakomkin N, Fabricant PD, Lawrence JTR. Transphyseal ACL reconstruction in skeletally immature patients: does independent femoral tunnel drilling place the physis at greater risk compared with transtibial drilling? *Orthop J Sports Med*. 2016;4(6):2325967116650432. <https://doi.org/10.1177/2325967116650432>.
38. Trivedi V, Mishra P, Verma D. Pediatric ACL injuries: a review of current concepts. *Open Orthop J*. 2017;11(1):378–88. <https://doi.org/10.2174/1874325001711010378>.
39. Edmonds EW, Fornari ED, Dashe J, Roocroft JH, King MM, Pennock AT. Results of displaced pediatric tibial spine fractures: a comparison between open, arthroscopic, and closed management. *J Pediatr Orthop*. 2015;35(7):651–6.
40. Strauss EJ, Kaplan DJ, Weinberg ME, Egol J, Jazrawi LM. Arthroscopic management of tibial spine avulsion fractures: principles and techniques. *J Am Acad Orthop Surg*. 2018;26(10):360–7. <https://doi.org/10.5435/JAOS-D-16-00117>.
41. Ishibashi Y, Rudy TW, Livesay GA, Stone JD, Fu FH, Woo SL. The effect of anterior cruciate ligament graft fixation site at the tibia on knee stability: evaluation using a robotic testing system. *Arthroscopy*. 1997;13(2):177–82.
42. Meyers MH, McKeever FM. Fracture of the intercondylar eminence of the tibia. *J Bone Joint Surg*. 1970;52(8):1677–84.
43. Zaricznyj B. Avulsion fracture of the tibial eminence: treatment by open reduction and pinning. *J Bone Joint Surg Am*. 1977;59(8):1111–4.
44. Kocher MS, Micheli LJ, Gerbino P, Hresko MT. Tibial eminence fractures in children: prevalence of meniscal entrapment. *Am J Sports Med*. 2003;31(3):404–7.
45. Shea KG, Grimm NL, Laor T, Wall E. Bone bruises and meniscal tears on MRI in skeletally immature children with tibial eminence fractures. *J Pediatr Orthop*. 2011;31(2):150–2.
46. Lentz TA, Magill J, Myers H, Eposito V, Reinke E, Messer M, Riboh J. Development of a concise lower extremity physical performance test set for return to sport decision-making in pediatric populations. 2019 American Orthopedic Society of Sports Medicine (AOSSM) Annual Meeting, Boston, MA; 2019.
47. Dekker TJ, Godin JA, Dale KM, Garrett WE, Taylor DC, Riboh JC. Return to sport after pediatric anterior cruciate ligament reconstruction and its effect on subsequent anterior cruciate ligament injury. *J Bone Joint Surg Am*. 2017;99(11):897–904. <https://doi.org/10.2106/JBJS.16.00758>.
48. Wiggins AJ, Grandhi RK, Schneider DK, Stanfield D, Webster KE, Myer GD. Risk of secondary injury in younger athletes after anterior cruciate ligament reconstruction. *Am J Sports Med*. 2016;44(7):1861–76. <https://doi.org/10.1177/0363546515621554>.
49. Greenberg EM, Greenberg ET, Ganley TJ, Lawrence JTR. Strength and functional performance recovery

- after anterior cruciate ligament reconstruction in pre-adolescent athletes. *Sports Health*. 2014;6(4):309–12. <https://doi.org/10.1177/1941738114537594>.
50. Grindem H, Snyder-Mackler L, Moksnes H, Engebretsen L, Risberg MA. Simple decision rules can reduce reinjury risk by 84% after ACL reconstruction: the Delaware-Oslo ACL cohort study. *Br J Sports Med*. 2016;50(13):804–8. <https://doi.org/10.1136/bjsports-2016-096031>.
 51. Bedi A, Musahl V, Cowan JB. Management of posterior cruciate ligament injuries: an evidence-based review. *J Am Acad Orthop Surg*. 2016;24(5):277–89. <https://doi.org/10.5435/JAAOS-D-14-00326>.
 52. Kocher MS, Shore B, Nasreddine AY, Heyworth BE. Treatment of posterior cruciate ligament injuries in pediatric and adolescent patients. *J Pediatr Orthop*. 2012;32(6):553–60. <https://doi.org/10.1097/BPO.0b013e318263a154>.
 53. Bushnell BD, Bitting SS, Crain JM, Boublik M, Schlegel TF. Treatment of magnetic resonance imaging-documented isolated grade III lateral collateral ligament injuries in National Football League athletes. *Am J Sports Med*. 2010;38(1):86–91. <https://doi.org/10.1177/0363546509344075>.
 54. Miyamoto RG, Bosco JA, Sherman OH. Treatment of medial collateral ligament injuries. *J Am Acad Orthop Surg*. 2009;17(3):152–61.
 55. Redler LH, Wright ML. Surgical management of patellofemoral instability in the skeletally immature patient. *J Am Acad Orthop Surg*. 2018;26(19):e405–15. <https://doi.org/10.5435/JAAOS-D-17-00255>.
 56. Bronstein RD, Schaffer JC. Physical examination of knee ligament injuries. *J Am Acad Orthop Surg*. 2017;25(4):280–7. <https://doi.org/10.5435/JAAOS-D-15-00463>.
 57. Mountney J, Senavongse W, Amis AA, Thomas NP. Tensile strength of the medial patellofemoral ligament before and after repair or reconstruction. *J Bone Joint Surg Br*. 2005;87:36–40.
 58. Weinberger J, Fabricant PR, Taylor SA, Mei JY, Jones KJ. Influence of graft source and configuration on revision rate and patient-reported outcomes after MPFL reconstruction: a systematic review and meta-analysis. *Knee Surg Sports Traumatol Arthrosc*. 2017;25:2511–9.
 59. Weeks KD III, Fabricant P, Ladenhauf HG, Green DW. Surgical options for patellar stabilization in the skeletally immature patient. *Sports Med Arthrosc Rev*. 2012;20:194–202.
 60. Nolan JE, Schottel PC, Endres NK. Trochleoplasty: indications and technique. *Curr Rev Musculoskelet Med*. 2018;11(2):231–40. <https://doi.org/10.1007/s12178-018-9478-z>.
 61. Houghton GR, Ackroyd CE. Sleeve fractures of the patella in children: a report of three cases. *J Bone Joint Surg Br*. 1979;61-B(2):165–8.
 62. Sousa PL, Stuart MJ, Prince MR, Dahm DL. Nonoperative management of minimally displaced patellar sleeve fractures. *J Knee Surg*. 2019; <https://doi.org/10.1055/s-0039-1694742>.
 63. Hunt DM, Somashekar N. A review of sleeve fractures of the patella in children. *Knee*. 2005;12(1):3–7. <https://doi.org/10.1016/j.knee.2004.08.002>.
 64. Schiller J, DeFroda S, Blood T. Lower extremity avulsion fractures in the pediatric and adolescent athlete. *J Am Acad Orthop Surg*. 2017;25(4):251–9. <https://doi.org/10.5435/JAAOS-D-15-00328>.
 65. Jakoi A, Freidl M, Old A, Javandel M, Tom J, Realyvasquez J. Tibial tubercle avulsion fractures in adolescent basketball players. *Orthopedics*. 2012;35(8):692–6. <https://doi.org/10.3928/01477447-20120725-07>.
 66. Pretell-Mazzini J, Kelly DM, Sawyer JR, et al. Outcomes and complications of Tibial tubercle fractures in pediatric patients: a systematic review of the literature. *J Pediatr Orthop*. 2016;36(5):440–6. <https://doi.org/10.1097/BPO.0000000000000488>.
 67. Shieh A, Bastrom T, Roocroft J, Edmonds EW, Pennock AT. Meniscus tear patterns in relation to skeletal immaturity: children versus adolescents. *Am J Sports Med*. 2013;41(12):2779–83. <https://doi.org/10.1177/0363546513504286>.
 68. Mosich GM, Lieu V, Ebrahimzadeh E, Beck JJ. Operative treatment of isolated meniscus injuries in adolescent patients: a meta-analysis and review. *Sports Health*. 2018;10(4):311–6. <https://doi.org/10.1177/1941738118768201>.
 69. Liechti DJ, Constantinescu DS, Ridley TJ, Chahla J, Mitchell JJ, Vap AR. Meniscal repair in pediatric populations: a systematic review of outcomes. *Orthop J Sports Med*. 2019;7(5):2325967119843355. <https://doi.org/10.1177/2325967119843355>.
 70. Kraus T, Heidari N, Švehlík M, Schneider F, Sperl M, Linhart W. Outcome of repaired unstable meniscal tears in children and adolescents. *Acta Orthop*. 2012;83(3):261–6. <https://doi.org/10.3109/17453674.2012.693017>.
 71. Kocher MS, Logan CA, Kramer DE. Discoid lateral meniscus in children: diagnosis, management, and outcomes. *J Am Acad Orthop Surg*. 2017;25(11):736–43. <https://doi.org/10.5435/JAAOS-D-15-00491>.
 72. Ellis HB, Wise K, LaMont L, Copley L, Wilson P. Prevalence of discoid meniscus during arthroscopy for isolated lateral meniscal pathology in the pediatric population. *J Pediatr Orthop*. 2017;37(4):285–92. <https://doi.org/10.1097/BPO.0000000000000630>.
 73. Watanabe M, Takeda SJ, Ikeuchi HJ. Atlas of arthroscopy. 2nd ed. Igaku-Shoin: Tokyo; 1969.
 74. Klingele KE, Kocher MS, Hresko MT, Gerbino P, Micheli LJ. Discoid lateral meniscus: prevalence of peripheral rim instability. *J Pediatr Orthop*. 2004;24(1):79–82.
 75. Good CR, Green DW, Griffith MH, Valen AW, Widmann RF, Rodeo SA. Arthroscopic treatment of symptomatic discoid meniscus in children: classification, technique, and results. *Arthroscopy*. 2007;23(2):157–63.
 76. Wall EJ, Milewski MD, Carey JL, et al. The reliability of assessing radiographic healing of osteochondritis dissecans of the knee. *Am J*

- Sports Med. 2017;45(6):1370–5. <https://doi.org/10.1177/0363546517698933>.
77. Kessler JJ, Jacobs JC Jr, Cannamela PC, Weiss JM, Shea KG. Demographics and epidemiology of osteochondritis dissecans of the elbow among children and adolescents. *Orthop J Sports Med.* 2018;6(12):232596711881584–6. <https://doi.org/10.1177/2325967118815846>.
78. Chambers HG, Shea KG, Anderson AF, et al. American Academy of Orthopaedic Surgeons clinical practice guideline on: the diagnosis and treatment of osteochondritis dissecans. *J Bone Joint Surg Am.* 2012;94(14):1322–4. <https://doi.org/10.2106/JBJS.9414ebo>.
79. Ramski DE, Ganley TJ, Carey JL. A radiographic healing classification for osteochondritis dissecans of the knee provides good interobserver reliability. *Orthop J Sports Med.* 2017;5(12):232596711774084. <https://doi.org/10.1177/2325967117740846>.
80. Carey JL, Wall EJ, Grimm NL, et al. Novel arthroscopic classification of osteochondritis dissecans of the knee: a multicenter reliability study. *Am J Sports Med.* 2016;44(7):1694–8. <https://doi.org/10.1177/0363546516637175>.
81. Wall EJ, Vourazeris J, Myer GD, et al. The healing potential of stable juvenile osteochondritis dissecans knee lesions. *J Bone Joint Surg Am.* 2008;90(12):2655–64. <https://doi.org/10.2106/JBJS.G.01103>.
82. Edmonds EW, Polousky J. A review of knowledge in osteochondritis dissecans: 123 years of minimal evolution from König to the ROCK study group. *Clin Orthop Relat Res.* 2013;471(4):1118–26. <https://doi.org/10.1007/s11999-012-2290-y>.
83. Kocher MS, Tucker R, Ganley TJ, Flynn JM. Management of osteochondritis dissecans of the knee: current concepts review. *Am J Sports Med.* 2006;34:1181–91.
84. Chang GH, Paz DA, Dwek JR, Chung CB. Lower extremity overuse injuries in pediatric athletes: clinical presentation, imaging findings, and treatment. *J Clin Imaging.* 2013;37(5):836–46. <https://doi.org/10.1016/j.clinimag.2013.04.002>.
85. Circi E, Atalay Y, Beyzadeoglu T. Treatment of Osgood-Schlatter disease: review of the literature. *Musculoskelet Surg.* 2017;101(3):195–200. <https://doi.org/10.1007/s12306-017-0479-7>.
86. Figueroa D, Figueroa F, Calvo R. Patellar tendinopathy: diagnosis and treatment. *J Am Acad Orthop Surg.* 2016;24(12):e184–92. <https://doi.org/10.5435/JAAOS-D-15-00703>.
87. Drago JL, Wasterlain AS, Braun HJ, Nead KT. Platelet-rich plasma as a treatment for patellar tendinopathy: a double-blind, randomized controlled trial. *Am J Sports Med.* 2014;42(3):610–8.
88. Dimitrios S, Pantelis M, Kalliopi S. Comparing the effects of eccentric training with eccentric training and static stretching exercises in the treatment of patellar tendinopathy: a controlled clinical trial. *Clin Rehabil.* 2012;26(5):423–30.



Anatomical Causes for Patellofemoral Pain in Basketball Players

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34.1 Introduction

Patellofemoral pain (PFP) accounts for 25% of knee problems seen in sport medicine clinics [1]. Running and jumping athletes are frequently affected and up to one fourth of them will stop practicing because of pain [2]. An observational study on 810 adolescent basketball players showed a prevalence of 25% for PFP, specifically ~26% of female and 18% of male athletes were affected [3]. Playing sports means running, jumping, pivoting in a repetitive manner. This exposure to high and repetitive loads can lead to supraphysiologic overload. Running can produce ground reaction forces two to three times the body weight, while during the landing phase of a jump it increases up to 9–10 times the body weight [4]. It has been reported that professional basketball players jump an average of 70 times per match [5], considering the height and the body weights of those athletes, it is easy to expect the high frequency of injuries to the knee. The occurrence of PFP can severely affect the athlete's sport participation and activity level due to the common recur-

rence or chronic course of symptoms. In a multicenter observational analysis, more than a half of study participants reported unfavorable recovery 5–8 years after the initial diagnosis and conservative management for PFP [6].

34.2 Differential Diagnosis of Patellofemoral Pain

In the literature the terms of anterior knee pain and PFP are frequently used as synonyms, which can create some confusion. A large number of different conditions can lead to anterior knee pain, and some of them have an anatomical or functional origin in the patellofemoral (PF) joint. However, it is important to keep in mind that several conditions around the knee, other than PF disorders, can determine pain in the same area (Table 34.1).

Two conditions deserve special consideration. Patellar tendon tendinopathy is a frequent cause of anterior knee pain, especially among athletes involved in jumping sports like basketball and volleyball [7] and an important differential diagnosis for PF joint pain. Several theories on the pathogenesis of patellar tendinopathy have been proposed [8], such as vascular, mechanical, and nervous system causes. Although patellar shape has been proposed as a possible pathogenetic factor, as discussed below in this chapter, chronic repetitive tendon overload seems to be the most common [9].

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Table 34.1 Differential diagnosis of anterior knee pain

Patellar instability <ul style="list-style-type: none"> • Objective patellar instability • Potential patellar instability • Painful patellar syndrome 	Primary and metastatic tumors
Synovial impingement syndromes <ul style="list-style-type: none"> • Pathologic plica • Hoffa syndrome 	Infection
Osteochondrosis <ul style="list-style-type: none"> • Osgood–Schlatter • Sinding–Larsen–Johannsen 	Bursitis <ul style="list-style-type: none"> • Pre-patellar bursitis (“housemaid’s knee”) • Infrapatellar bursitis (“clergyman’s knee”) • Medial collateral ligament bursitis • Pes anserine bursitis
Tendinopathies <ul style="list-style-type: none"> • Patellar tendon • Pes anserine • Semimembranosus 	Localized pigmented villonodular synovitis

Another frequent cause of anterior knee pain is knee osteochondrosis in younger athletic patients [10]. Osgood–Schlatter disease is a condition caused by a traction effect of the extensor mechanism on the secondary ossification center of the tibial tuberosity [11] leading to local tenderness and swelling. A bony prominence in the area of the tibial tuberosity can usually be palpated. It can affect boys and girls with ages between 8 and 12 years involved in jumping sport activities (e.g., basketball) and/or direct contact (e.g., kneeling). The other knee osteochondrosis is the Sinding–Larsen–Johannsen disease [12], a traction injury of the extensor mechanism, but on the inferior pole of the patella. Those patients also present anterior knee pain and a painful palpation of the inferior patellar pole.

In order to make a correct diagnosis of PFP, a detailed clinical examination is fundamental. The patient’s consultation should start with observing the walking pattern and the global posture. A detailed history should be taken, investigating:

- The beginning of the pain (How long ago? Sudden or progressive?)
- How it started (Spontaneously? Trauma?)
- Changes in training (Increase in training intensity? New training strategies? New coach?)
- If the pain is related to specific activities (jumping, climbing, and descending stairs)

The physical examination should be as complete as possible and evaluate:

- Any obvious sign of knee inflammation (swelling)
- Range of motion
- Alignment and symmetry of the lower limbs
- Pain location
- Specific signs of patellar instability
- Muscle status and flexibility (hamstring and quadriceps tightness, anterior pelvic tilt)
- Signs of other conditions (i.e., tenderness to palpation of the inferior pole of the patella or to the tibial tuberosity in cases of patellar tendinopathy or osteochondrosis of the knee)

The correct imaging studies will provide further information after integrating the history and physical examination. They should include at least:

- The anteroposterior weight-bearing X-rays of the knee, preferably of the entire lower limbs, to evaluate coronal alignment and deformities
- A skyline view (merchant), to evaluate patellar alignment and arthrosis
- Lateral views with perfect superimposition of the femoral condyles (essential for assessing trochlear dysplasia and patellar height)

Further imaging such as MRI, CT scan, and ultrasonography could be useful for additional assessment of cartilage, soft tissues, and possible torsional deformities.

34.3 Pathophysiology of Patellofemoral Pain

PFP is a complex clinical entity, with not completely understood pathophysiology yet. Since the 1970s, PFP has been related to patellofemoral malalignment (PFM) [13]; however, that link has weakened over time. In the 1990s Scott Dye came up with the “Envelope of Function” theory [14]. In that theory, Dye proposes that the loss of homeostasis of innervated PF tissues is the mechanism at the basis of patellofemoral pain. The envelope of function is defined as the capacity of the patellofemoral joint to accept and transfer a certain range of load, maintaining the tissues’ homeostasis. Dye identified four zones:

1. The zone of *tissues’ homeostasis*.
2. The zone of *subphysiologic underload*, in case of excessively diminished loads that will lead to disuse alteration.
3. The zone of *supraphysiologic overload*, when an increased load is applied to the joint, but it is insufficient to cause immediate obvious structural damage.
4. The zone of *macrostructural failure* that occurs when an excessive load determines an abrupt damage, such as bone fractures or ligament tears.

The envelope of function corresponds to the zone of homeostasis and is determined by anatomic, kinematic, and physiologic factors. All those factors act interdependently and define the threshold for the loss of homeostasis, with PFP as an indicator of the transition to supraphysiologic overload.

This chapter focuses on the anatomic factors that have important implications in PFP [15]:

1. Bone morphology, lower limb alignment (including coronal and torsional deformities), and foot disorders
2. Static and dynamic soft tissue stabilizers
3. Hip and core stability
4. Cartilage

34.4 Bone Morphology and Lower Limb Alignment

34.4.1 Trochlea

The trochlear shape is of paramount importance in the biomechanics of the patellofemoral joint. A medial and a lateral facet, converging into the trochlear groove, compose the trochlea. Both facets are convex in the sagittal and frontal plane, with the lateral one being higher, larger, and more prominent than the medial one, providing a restraint for lateral patellar subluxation during knee flexion.

Lateral and medial walls are covered by a layer of articular cartilage of 2–3 mm thickness, although it is thinner on the medial side compared to the lateral [16]. The trochlear groove deepens from proximal to distal, and its axis has a downward and outward inclination of 10° compared to the femoral diaphyseal axis; however, there is significant individual variability concerning orientation, length, and curvature of the trochlear groove [17]. The patellar and trochlear groove bone and chondral geometry are of crucial importance in guiding the patella in the distal femur as the knee flexes. The amount of restraint provided seems to increase during flexion, as the groove becomes deeper. At 20 degrees of flexion the force needed to produce a 10 mm lateral displacement of the patella is 74 N, rising to 125 N at 90° [18].

Senavongse and Amis [19] demonstrated that a flat trochlea is more likely to induce lateral patellar translation than an injured medial retinaculum or a released vastus medialis. The same authors stated that static restraints have a less variable and more consistent role in providing patellar stability than dynamic ones [18]. For those reasons, since the first observation of the connection between trochlear dysplasia and patellar instability reported by Brattstroem in the early 1960s [20], trochlear morphology has gained increasing attention, up to 1987 when H. Dejour and G. Walch established the basic principles and diagnostic rationale of patellar

instability, proposing a classification of the trochlear dysplasia [21]. Successively, D. Dejour and Le Coultre defined a more accurate classification based on combined evaluation of axial and lateral radiographs that takes into account three radiographic signs: the supratrochlear spur, the double contour, and the crossing sign (Fig. 34.1). By those signs, the authors established four types of dysplasia [22] (Table 34.2). Trochlear dysplasia has been identified as the predominant factor in inducing patellar instability [23]. In fact, it determines a lack of congruence between the trochlear groove and the patella, during the range of motion. This incongruence is particularly important during the early degrees of flexion, since the proximal

groove is shallower or flat compared to the distal, and the lateral structures tend to overcome the medial ones (Fig. 34.2). This condition can result in dislocation and/or overloading of the lateral facet since the stresses are highly asymmetric and concentrated on the lateral PF articular surface rather than on the entire groove [24]. Over time, PFP can arise because tissues around the PF joint get irritated (loss of tissue homeostasis, Dye’s theory), and degenerative changes can occur on the articular cartilage.

34.4.2 Patella

Patella is the largest sesamoid bone of the human body. It has several functions, such as protecting the deep knee structures, increasing the lever arm

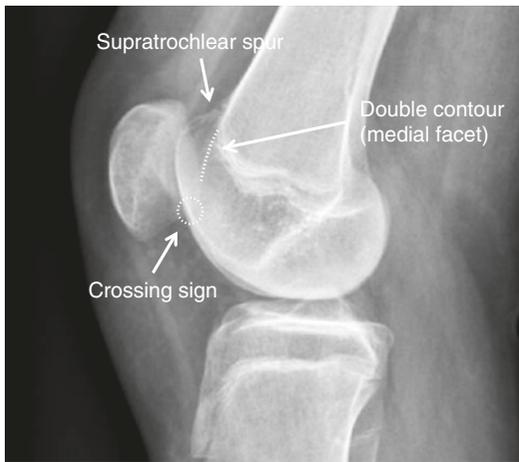


Fig. 34.1 Lateral radiography of a type D trochlear dysplasia. Notice the presence of all three signs used in Dejour’s trochlear dysplasia classification: crossing sign, supratrochlear spur, and the double contour of the medial facet



Fig. 34.2 Axial imaging of a type D trochlear dysplasia. Notice the vertical transition (cliff pattern) between the convex lateral facet and the hypoplastic medial facet

Table 34.2 Dejour’s classification on trochlear dysplasia

	Lateral radiography	Axial imaging—CT or MRI
Type A	– Crossing sign	– Trochlear morphology preserved – Shallow trochlea
Type B	– Crossing sign – Supratrochlear spur	– Flat trochlea
Type C	– Crossing sign – Double contour	<i>Trochlear facet asymmetry:</i> – Convex lateral facet – Hypoplastic medial facet
Type D	– Crossing sign – Supratrochlear spur – Double contour	– Trochlear facets asymmetry – Vertical transition between lateral and medial facet (cliff pattern)

of the extensor apparatus, and providing stability to the knee [25]. Patellar shape and position show an important variability among individuals, which can potentially influence the biomechanics and function of the PFJ.

The variability of the patellar shape in the axial plane was studied by Wiberg [26]. He proposed a classification system based on the configuration of the medial and lateral facets of the articular surface of the patella: type I (with a prevalence of 10%) presents concave medial and lateral facets, both almost equal in size; type II (prevalence of 65%) presents a flat or slightly convex medial facet which is smaller than the lateral facet; type III (25% of all cases) has a considerably smaller and convex medial facet.

There seems to be a correlation between the Wiberg classification and patellar instability. Schiavone-Panni et al. [27] compared the axial patellar configuration of a group of objective patellar instability patients with the normal population and found that the more the patella is tilted, the more the patella is dysplastic; in addition they found a significant association between Wiberg patellar shape type III and the most severe trochlear dysplasia. The authors speculate that increased lateral stresses may produce a Wiberg type III patella. It has been demonstrated as a Wiberg type III patella has a higher incidence of cartilage damage compared to type I and II [28].

In the sagittal plane, three types of patella can be identified according to the Grelsamer classification [29]. This classification is based on the ratio between the length of the patella and the length of the articular surface: type I (most common) exhibit a ratio between 1.2 and 1.5, type II with a ratio greater than 1.5 (long nose) and type III with a ratio less than 1.2 (short nose).

Grelsamer speculated that a patella with a shorter articular surface than normal (type II), as well as patellae with long and short inferior poles (type II and III) can determine abnormal contact stress [29]. However, currently there is no clear evidence that specific patellar shape patterns are linked with a higher risk of pain or instability [30].

Patellar shape in the sagittal plane has also been investigated in relation to patellar tendinopathy (PT), which is a common disease affecting

basketball players (up to 32% in professional players [7]). Johnson and colleagues, on an MRI study on PT, reported that the site of the lesion is more compatible with an impingement-based mechanism (of the inferior pole of the patella against the patellar tendon) rather than a stress overload of the tendon. However, the authors did not find significant differences in the length of the patella, inferior pole or length of the articular surface between the PT group and a matched control group [31].

Conversely, it is well known that patella alta is linked with PFP and an increased risk for instability. In fact, it has been shown that a high riding patella causes a decreased contact between the patella and trochlea during the early degrees of flexion (35–70°) and a higher contact force in deeper flexion (70–120°), compared to normal height patellas. In other words, patella alta demonstrates higher maximal patellofemoral contact force and pressure when compared to a normal riding patella [32], which might explain the relation between patella alta and PFP. In addition, patella alta was related to greater lateral displacement and greater lateral tilt [33], determining a decreased resistance to lateral translation of the patella [34], potentially leading to patellar instability.

Being a predisposing factor for pain and instability, several methods have been proposed to measure patellar height (i.e., Insall-Salvati, Caton-Deschamps, Blackburne-Peel). However, all of them use bony and not cartilaginous reference points, which limits the evaluation of the functional (cartilaginous) engagement of the patella in the trochlea, fundamental to the patellar tracking and stability. This led to the introduction of new methods to evaluate the sagittal position of the patella. One of these methods is the sagittal patellofemoral engagement (SPE) index [35]. This method uses two distinctive sagittal cuts of an MRI to evaluate the functional engagement of the patella over the trochlea (Fig. 34.3). The first cut is taken where the patella shows the longest articular cartilage, which allows the drawing of a patellar length line (PL) that represents the total length of the patellar articular cartilage. The second sagittal

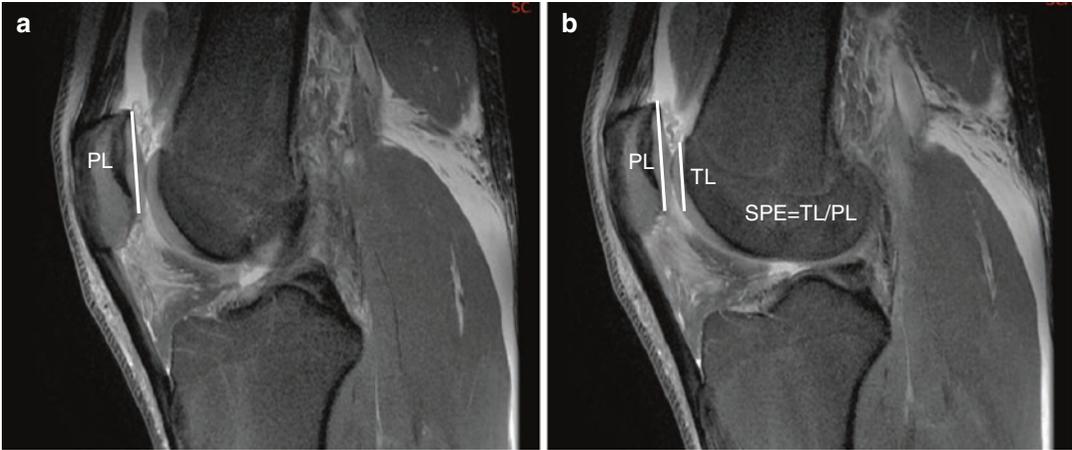


Fig. 34.3 Obtaining the SPE index: (a) patellar length line (PL) is drawn in the MR sagittal cut where the patella shows the longest articular cartilage. (b) The PL line is transferred to the sagittal cut where the trochlear cartilage extends more proximally, and on this cut, the trochlear

line (TL) is drawn parallel to the PL line, starting from the most proximal articular trochlear cartilage and finishing at the distal end of the PL line. The SPE is then calculated as the ratio between TL and PL lines

cut is taken where the femoral trochlear cartilage extends more proximally, and on this cut, a second line is drawn parallel to the PL line, starting from the most proximal articular trochlear cartilage and finishing at the distal end of the PL line, providing the trochlear length (TL). The SPE is then calculated as the ratio between TL and PL. The pathologic cut off value for a normal sagittal engagement was defined at $SPE < 0.45$.

What is most interesting about this method is that there isn't a significant correlation between the SPE index and the Caton-Deschamps index, which means that patients can present patella alta and normal SPE but also normal patellar height and $SPE < 0.45$. It can help to better identify the cases where inadequate functional engagement is present despite the absence of patella alta and help to choose the best treatment options.

34.4.3 Bony Alignment

Bony alignment is also of paramount importance for an optimal PF tracking. The evaluation of lower limb alignment has been classically related to the measurement of the quadriceps angle (Q-angle). Since Insall and colleagues [36] described the measure in its current form, its clinical validity has been questioned concerning nor-

mal values, subject positioning, contraction status of the quadriceps, and validity of the measuring technique [25, 37]. Despite the questions of the utilization of the Q-angle in clinical evaluation of PF instability, its biomechanical importance, however, remains unquestioned [38].

Quadriceps and patellar tendons form an angle, which during muscle contraction determines a lateral displacement of the patella that is counteracted by the lateral wall of the trochlea and the medial soft tissues restraints. The bigger the Q-angle the bigger the lateral forces on the patella [39].

Another index of the PF alignment is the tibial tuberosity–trochlear groove (TT–TG) distance. Described by Goutallier et al. [40], TT–TG distance is a quantitative measure of lateralization of the tibial tuberosity on the proximal tibia with respect to the trochlear groove. From the original technique, based on X-ray with the knee at 30° of flexion and neutral rotation of the hip, the average normal value was 12 mm and the pathologic cut-off value was considered to be 20 mm.

TT–TG distance can also be calculated using axial imaging of CT or MRI, using two cuts: one through the deepest part of the trochlear groove, in its most proximal part (where the notch resemble a Roman arch), and the other through the most proximal part of the tibial tubercle.

In those cuts, two perpendicular lines to the posterior condylar line are drawn, one passing through the deepest part of the trochlear groove and other through the middle of the patellar tendon. The distance between this to parallel lines is the TT–TG distance. Because of different methods of CT or MRI acquisition among published studies, the TT–TG reference values are not unanimously established, ranging from 10 ± 1 to 13.9 ± 4.5 mm [41–43].

34.4.4 Coronal Deformities of the Lower Limb

Coronal (varus-valgus) deformities can alter the biomechanics of the PF joint by increasing the Q-angle and the TT–TG distance. Lower limb deformities determine changes in contact pressure and contact area with resultant PFP or even PF instability.

Valgus malalignment has a significant impact on PF kinematics and patellar tilt, by increasing the Q-angle, and some studies have shown that valgus deformities influence the muscle vector significantly, resulting in increased lateral patellofemoral pressure due to significant lateralization of the patella [44]. Furthermore, it has been shown that varisation osteotomies can produce good results in patients with patellofemoral instability or anterior knee pain [45].

Varus deformity can affect PF joint as well, since there seems to exist an association of proximal tibial rotation with varus deformity, which causes the patella to displace laterally with increased risk of patellar instability, as demonstrated by Fujikawa et al. [46].

34.4.5 Torsional Deformities of the Lower Limb

Abnormal (increased) femoral anteversion can result in PF malalignment and instability. Despite the excessive femoral anteversion, the femoral head will be centered into the acetabulum by the strong soft tissues of the hip, thus internally rotating the whole femur, from proximal to distal,

leading to an increased Q-angle by displacing the patella medially relative to the anterior superior iliac spine and tibial tuberosity [17].

Normal femoral anteversion is usually considered to be about 15° [17], but there is a great variation with the mean value for physiological femoral torsion ranging from 7° to 24° of internal torsion, depending on the measurement technique used [47]. Some studies have also shown a significant gender variation, with slightly less anteversion in male subjects compared to female subjects [17]. Increased internal rotation of the distal femur leads potentially to altered PF kinematic, therefore predisposing to PF pain, arthritis [48], and instability [47].

A study by Lee et al. [49] analyzed the effects of femoral rotation on PF joint contact pressures and found a nonlinear increase in the PF contact pressures with increasing femoral rotation. At 30° rotational deformity of the femur, both the external and internal rotational deformity of the femur showed a significant increase in the tension of the quadriceps tendon and the patellofemoral contact pressures on contralateral facets of the patella. Given the relevant contribution of the femoral torsion to PF kinematics, some authors proposed performing femoral derotational osteotomy in patients with recurrent patellar dislocations caused by significant torsion abnormalities [50].

Primary or secondary external tibial rotation can also affect the patellar alignment, due to a lateral displacement of the tibial tuberosity. The Q-angle and the patellar tracking result abnormal, thus determining PF pain and instability. Turner demonstrated a significantly increased external tibial rotation in patients with PF disorders when compared with controls [51].

34.4.6 Foot Disorders

Although still not strongly demonstrated the cause–effect relationship, it is possible to speculate that foot disorders can contribute to PF pain and instability. It has been largely demonstrated a high incidence of abnormal eversion and/or pronation of the foot in patients affected by PF pain [52, 53].

34.5 Soft Tissue Restraints

During the first degrees of flexion (0–30°) the primary restraints to lateral PF translation are soft tissue structures, mainly the medial patellofemoral ligament (MPFL), followed by the patellofemoral ligament and then the vastus medialis obliquus and the medial retinaculum [54]. By the time the knee goes to further flexion, the patella shifts medially being progressively engaged into the trochlear groove, with the stability relying more on bone contact and not so much on soft tissues. The most important among the static medial restrains is the MPFL, a relatively thin band of retinacular tissue running from the adductor tubercle to the medial border of patella that provides as much as 60% of total restraint at 20 degrees of knee flexion [54]. Congenital abnormalities or traumatic (patellar dislocation) can impair the MPFL function and therefore cause PF instability.

On the lateral side of the knee, the PF stability is aided by the following structures: lateral patellofemoral ligament, joint capsule, iliotibial band (ITB), and lateral retinaculum. According to the anatomical study of Merican and Amis [55], the retinaculum is composed of three different layers: the superficial (deep fascia), intermediate and the deep layer (joint capsule), with the intermediate layer being the most substantial and consisting of derivatives of the iliotibial band and the quadriceps aponeurosis. The authors described extensive interdigitations among the ITB, the quadriceps aponeurosis and the tendon of vastus lateralis obliquus.

It is important to keep in mind that because most of the lateral retinaculum originates from the ITB, tightness of this structure (which has the greatest influence at 20 degrees of knee flexion) will contribute to lateral tracking and tilt of the patella, increasing the stress on the lateral patellar facet and predisposing to malalignment and instability [56].

Besides the static restrains, there are active medial patellar stabilizers, and among them, the vastus medialis is the most important one. It opposes the lateral vector force of the vastus late-

ralis, allowing a more efficient extensor moment at the knee.

The vastus medialis is commonly divided into the oblique portion (VMO) oriented at $47^\circ \pm 5^\circ$ with respect to the femoral axis and, the more vertical component, the vastus medialis longus. The main medial active stabilizing force of the patella is the VMO. Hypoplasia or dysplasia of that muscle is a major cause of dynamic patella instability. However, even in the presence of a normal muscular tissue, an impaired muscle contraction can determine an altered recruitment order between vastus medialis and lateralis originating in patellar instability [57].

34.6 Core Stability and Hip Strength: Looking Beyond the Knee

In addition to the locally defined PF anatomical factors, the role of core stability and hip muscle strength in PF function and pain has gained some attention over the last decades. From an anatomical point of view, the musculoskeletal core of the body refers to the osseous and soft tissue structures of the spine, pelvis, and abdomen, responsible for the lumbopelvic and hip dynamic control. A strong core is fundamental for optimal production, transfer, and control of force and motion throughout the lower limb, which in turn can have a critical role in PF disorders. Poor core stability results in lack of dynamic control on the frontal plane, contralateral pelvic drop, homolateral excessive hip adduction, and internal rotation (dynamic valgus) (Fig. 34.4), which may eventually affect PF kinematics [58]. The actual result of an increased hip adduction and internal rotation is the medial displacement of the knee relative to the foot and internal rotation of distal femur relative to the tibia, which, as seen earlier in this chapter, can contribute to PF pain and instability.

Patients with patellofemoral pain display more weakness in hip abduction and external rotation, allowing for increased hip adduction and internal rotation during functional movements [59]. Contralateral pelvic drop is also



Fig. 34.4 Dynamic valgus in single-leg landing test: homolateral excessive hip adduction and internal rotation

thought to increase tension in the lateral patellar retinaculum, via the iliotibial band, potentially contributing to an increased lateral patellar tracking.

Regarding the sagittal plane, there seems to be a correlation between PF joint pain, lumbar lordosis, and sacral inclination. In a study by Tsuji et al. [60], there was a significant difference in sacral inclination between elderly subjects with and without anterior knee pain, as patients with a lower back lordosis tend to walk with a compensatory knee flexion that can increase PF joint load. The authors called this phenomenon the “knee-spine syndrome.” Although this has not been studied in young adults and active people yet, it seems intuitive that a strong and well-balanced pelvis and lower spine will avoid postural alterations that can eventually be responsible for PF joint symptoms.

34.7 Cartilage

The average patella cartilage thickness is 4.1 mm, being the thickest in the body, due to high joint reaction forces, which can rise to up to 20 times the body weight during sporting activities [61, 62]. Healthy PF articular cartilage is fundamental in energy absorption during daily and sport activities, especially during knee flexion against resistance, like going downstairs or jumping. It would be intuitive to consider cartilage thickness as an important variable associated to PF pain, and it is known that thinner cartilage results in higher stress for the same applied load [63]. However, although individuals with patellofemoral pain exhibit greater patellofemoral joint stress [64], a significant thinner cartilage was reported only in male individuals affected by PFP compared to controls, and not in females, suggesting that cartilage thickness is conceivably a non-dominant factor in the pathogenesis of PFPS, when in absence of concomitant abnormalities.

The medial and central areas of the patella show a richness of intraosseous innervation, significantly higher than the lateral one [65]. Those nerves can deliver nociceptive signals in response to mechanical stimuli secondary to increment of intraosseous pressure (intraosseous edema) or increment of the subchondral bone pressure (that happens when the cartilage fails to absorb the energy, secondary to cartilage lesions or to reduced contact area in PF malalignment) [66].

34.8 Conclusion

Several factors play a role in patellofemoral pain, some of them are clear and with an obvious diagnosis, some others are subtler. Abnormal bone morphology like trochlear dysplasia and patella alta can contribute to a various level to improper PF tracking, but also soft tissue alterations (tightness of the lateral structures, incompetent MPFL, altered patterns in muscle contractures or imbalanced musculature), frontal plane deformities (valgus or varus), torsional deformities (increased femoral anteversion and external tibial rotation),

and hip/core stability are involved in generating PF pain.

Therefore, it is paramount to thoroughly assess the patients, looking not only to the knee but to the whole body in both static and dynamic settings. Only after matching patient's symptoms, history, physical examination, and images, it is possible to draw a correct diagnosis and plan a proper treatment strategy.

References

1. Witvrouw E, Callaghan MJ, Stefanik JJ, et al. Patellofemoral pain: consensus statement from the 3rd international Patellofemoral pain research retreat held in Vancouver, September 2013. *Br J Sports Med.* 2014;48:411–4.
2. Rathleff MS, Rasmussen S, Olesen JL. Unsatisfactory long-term prognosis of conservative treatment of patellofemoral pain syndrome. *Ugeskr Laeger.* 2012;174:1008–13.
3. Foss KDB, Myer GD, Magnussen RA, Hewett TE. Diagnostic differences for anterior knee pain between sexes in adolescent basketball players. *J Athl Enhanc.* 2014;3(1):1814.
4. Ortega DR, Rodríguez Bies EC, Berral de la Rosa FJ. Analysis of the vertical ground reaction forces and temporal factors in the landing phase of a countermovement jump. *J Sports Sci Med.* 2010;9:282–7.
5. McClay IS, Robinson JR, Andriacchi TP, et al. A Profile of ground reaction forces in professional basketball. *J Appl Biomech.* 1994;10:222–36.
6. Lankhorst NE, van Middelkoop M, Crossley KM, et al. Factors that predict a poor outcome 5-8 years after the diagnosis of patellofemoral pain: a multicentre observational analysis. *Br J Sports Med.* 2016;50:881–6.
7. Lian OB, Engebretsen L, Bahr R. Prevalence of jumper's knee among elite athletes from different sports: a cross-sectional study. *Am J Sports Med.* 2002;33:561–7.
8. Schwartz A, Watson JN, Hutchinson MR. Patellar tendinopathy. *Sports Health.* 2015;7:415–20.
9. Schmid MR, Hodler J, Cathrein P, et al. Is impingement the cause of jumper's knee? Dynamic and static magnetic resonance imaging of patellar tendinitis in an open-configuration system. *Am J Sports Med.* 2002;30:388–95.
10. Slotkin S, Thome A, Ricketts C, et al. Anterior knee pain in children and adolescents: overview and management. *J Knee Surg.* 2018;31:392–8. <https://doi.org/10.1055/s-0038-1632376>.
11. Ogden JA, Southwick WO. Osgood-Schlatter's disease and tibial tuberosity development. *Clin Orthop.* 1976;116:180–9.
12. Sinding-Lorsen CMF. A hitherto unknown affection of the Patella in children. *Acta Radiol.* 1987;1(2):171–3.
13. Insall J. "Chondromalacia patellae": patellar malalignment syndrome. *Orthop Clin North Am.* 1979;10:117–27.
14. Dye SF, Stäubli HU, Biedert RM, Vaupel GL. The mosaic of pathophysiology causing patellofemoral pain: therapeutic implications. *Oper Tech Sports Med.* 1999;7:46–54.
15. Post WR, Teitge R, Amis A. Patellofemoral malalignment: looking beyond the viewbox. *Clin Sports Med.* 2002;21:521–46.
16. Zaffagnini S, Becker R, Kerkhoffs GMMJ, Mendes JE, van Dijk CN. ESSKA instructional course lecture book: Amsterdam; 2014.
17. Feller JA, Amis AA, Andrich JT, et al. Surgical biomechanics of the patellofemoral joint. *Arthrosc J Arthrosc Relat Surg.* 2007;23:542–53.
18. Senavongse W, Farahmand F, Jones J, et al. Quantitative measurement of patellofemoral joint stability: force-displacement behavior of the human patella in vitro. *J Orthop Res.* 2003;21:780–6.
19. Senavongse W, Amis AA. The effects of articular, retinacular, or muscular deficiencies on patellofemoral joint stability: a biomechanical study in vitro. *J Bone Joint Surg Br.* 2005;87:577–82.
20. Brattstroem H. Shape of the intercondylar groove normally and in recurrent dislocation of patella. A clinical and x-ray-anatomical investigation. *Acta Orthop Scand Suppl.* 1964;68:1–148.
21. Dejour H, Walch G. Morphologic factors in patellar instability: clinical, radiologic, and tomographic data. *J Lyon Chir Geno.* 1987;25–35.
22. Dejour D, Reynaud P, Lecoultré B. Douleurs et instabilité rotulienne. Essai de classification. *Med Hyg.* 1998;56:1466–71.
23. Dejour H, Walch G, Nove-Josserand L, Guier C. Factors of patellar instability: an anatomic radiographic study. *Knee Surg Sports Traumatol Arthrosc.* 1994;2:19–26.
24. Zaffagnini S, Dejour D, Arendt EA, editors. Patellofemoral pain, instability, and arthritis: clinical presentation, imaging, and treatment. Berlin Heidelberg: Springer-Verlag; 2010.
25. Fox AJS, Wanivenhaus F, Rodeo SA. The basic science of the patella: structure, composition, and function. *J Knee Surg.* 2012;25:127–41.
26. Wiberg G. Roentgenographs and anatomic studies on the Femoropatellar joint: with special reference to Chondromalacia patellae. *Acta Orthop Scand.* 1941;12:319–410.
27. Panni AS, Cerciello S, Maffulli N, et al. Patellar shape can be a predisposing factor in patellar instability. *Knee Surg Sports Traumatol Arthrosc.* 2011;19:663–70.
28. Gudas R, Šiupšinskas L, Gudaitė A, et al. The Patellofemoral joint degeneration and the shape of the Patella in the population needing an arthroscopic procedure. *Medicina (Mex).* 2018;54:21.

29. Grelsamer RP, Proctor CS, Bazos AN. Evaluation of patellar shape in the sagittal plane. A clinical analysis. *Am J Sports Med.* 1994;22:61–6.
30. Servien E, Ait TSS, Neyret P. Study of the patellar apex in objective patellar dislocation. *Rev Chir Orthop Reparatrice Appar Mot.* 2003;89:605–12.
31. Johnson DP, Wakeley CJ, Watt I. Magnetic resonance imaging of patellar tendonitis. *J Bone Joint Surg Br.* 1996;78:452–7.
32. Luyckx T, Didden K, Vandenneucker H, et al. Is there a biomechanical explanation for anterior knee pain in patients with patella alta?: influence of patellar height on patellofemoral contact force, contact area and contact pressure. *J Bone Joint Surg Br.* 2009;91:344–50.
33. Ward SR, Terk MR, Powers CM. Patella alta: association with patellofemoral alignment and changes in contact area during weight-bearing. *J Bone Joint Surg Am.* 2007;89:1749–55.
34. Ward SR, Powers CM. The influence of patella alta on patellofemoral joint stress during normal and fast walking. *Clin Biomech Bristol Avon.* 2004;19:1040–7. <https://doi.org/10.1016/j.clinbiomech.2004.07.009>.
35. Dejour D, Ferrua P, Ntgiopoulos PG, et al. The introduction of a new MRI index to evaluate sagittal patellofemoral engagement. *Orthop Traumatol Surg Res.* 2013;99:S391–8.
36. Insall J, Salvati E. Patella position in the normal knee joint. *Radiology.* 1971;101:101–4.
37. Freedman BR, Brindle TJ, Sheehan FT. Re-evaluating the functional implications of the Q-angle and its relationship to in-vivo patellofemoral kinematics. *Clin Biomech Bristol Avon.* 2014;29:1139–45.
38. Silva DO, Briani RV, Pazzinato MF, et al. Q-angle static or dynamic measurements, which is the best choice for patellofemoral pain? *Clin Biomech Bristol Avon.* 2015;30:1083–7.
39. Loudon JK. Biomechanics and pathomechanics of the patellofemoral joint. *Int J Sports Phys Ther.* 2016;11:820–30.
40. Goutallier D, Bernageau J, Lecudonnet B. The measurement of the tibial tuberosity. Patella groove distanced technique and results (author's transl). *Rev Chir Orthop Reparatrice Appar Mot.* 1978;64:423–8.
41. Schoettle PB, Zanetti M, Seifert B, et al. The tibial tuberosity-trochlear groove distance; a comparative study between CT and MRI scanning. *Knee.* 2006;13:26–31.
42. Sobhanardekani M, Sobhan MRS, Moghadam RN, Nabavinejad S, Ratki SKR. The Normal value of Tibial tubercle trochlear groove distance in patients with Normal knee examinations using MRI. *Acta Med Iran.* 2017;55:573–7.
43. Pandit S, Frampton C, Stoddart J, Lynskey T. Magnetic resonance imaging assessment of tibial tuberosity-trochlear groove distance: normal values for males and females. *Int Orthop.* 2011;35:1799–803.
44. Ficat P, Hungerford D. Disorders of the Patellofemoral joint. 1st ed. Baltimore: Williams & Wilkins; 1977.
45. Frings J, Krause M, Akoto R, Frosch K-H. Clinical results after combined distal femoral osteotomy in patients with patellar maltracking and recurrent dislocations. *J Knee Surg.* 2018;32(9):924–33.
46. Fujikawa K, Seedhom BB, Wright V. Biomechanics of the patello-femoral joint. Part II: a study of the effect of simulated femoro-tibial varus deformity on the congruity of the patello-femoral compartment and movement of the patella. *Eng Med.* 1983;12:13–21.
47. Kaiser P, Schmoelz W, Schoettle P, et al. Increased internal femoral torsion can be regarded as a risk factor for patellar instability - a biomechanical study. *Clin Biomech Bristol Avon.* 2017;47:103–9.
48. Souza RB, Draper CE, Fredericson M, Powers CM. Femur rotation and patellofemoral joint kinematics: a weight-bearing magnetic resonance imaging analysis. *J Orthop Sports Phys Ther.* 2010;40:277–85.
49. Lee TQ, Anzel SH, Bennett KA, Pang D, Kim WC. The influence of fixed rotational deformities of the femur on the patellofemoral contact pressures in human cadaver knees. *Clin Orthop.* 1994;302:69–74.
50. Imhoff FB, Beitzel K, Zakko P, Obopilwe E, Voss A, Scheiderer B, et al. Derotational osteotomy of the distal femur for the treatment of Patellofemoral instability simultaneously leads to the correction of frontal alignment: a laboratory cadaveric study. *Orthop J Sports Med.* 2018;6(6):2325967118775664. eCollection 2018 Jun
51. Turner MS. The association between tibial torsion and knee joint pathology. *Clin Orthop.* 1994:47–51.
52. Barton CJ, Levinger P, Crossley KM, Webster KE, Menz HB. The relationship between rearfoot, tibial and hip kinematics in individuals with patellofemoral pain syndrome. *Clin Biomech Bristol Avon.* 2012;27:702–5.
53. Barton CJ, Levinger P, Menz HB, Webster KE. Kinematic gait characteristics associated with patellofemoral pain syndrome: a systematic review. *Gait Posture.* 2009;30:405–16.
54. Conlan T, Garth WP, Lemons JE. Evaluation of the medial soft-tissue restraints of the extensor mechanism of the knee. *J Bone Joint Surg Am.* 1993;75:682–93.
55. Merican AM, Amis AA. Anatomy of the lateral retinaculum of the knee. *J Bone Joint Surg Br.* 2008;90:527–34.
56. Kang S-Y, Choung S-D, Park J-H, Jeon H-S, Kwon O-Y. The relationship between length of the ilio-tibial band and patellar position in Asians. *Knee.* 2014;21:1135–8.
57. Voight ML, Wieder DL. Comparative reflex response times of vastus medialis obliquus and vastus lateralis in normal subjects and subjects with extensor mechanism dysfunction. An electromyographic study. *Am J Sports Med.* 1991;19:131–7.
58. Boling M, Padua D. Relationship between hip strength and trunk, hip, and knee kinematics during a jump-landing task in individuals with patellofemoral pain. *Int J Sports Phys Ther.* 2013;8:661–9.
59. Boling MC, Padua DA, Alexander Creighton R. Concentric and eccentric torque of the hip musculature in individuals with and without patellofemoral pain. *J Athl Train.* 2009;44:7–13.

60. Tsuji T, Matsuyama Y, Goto M, Yimin Y, Sato K, Hasegawa Y, et al. Knee-spine syndrome: correlation between sacral inclination and patellofemoral joint pain. *J Orthop Sci.* 2002;7:519–23.
61. Porteous A, Sullivan N, Murray J, Eldridge J. How thick is the patella? A reproducible measure of patella width: thickness from adult MRI. *Orthop Proc.* 2013;95-B:46.
62. Schindler OS, Scott WN. Basic kinematics and biomechanics of the patello-femoral joint. Part 1: the native patella. *Acta Orthop Belg.* 2011;77:421–31.
63. Li G, Lopez O, Rubash H. Variability of a three-dimensional finite element model constructed using magnetic resonance images of a knee for joint contact stress analysis. *J Biomech Eng.* 2001;123:341–6.
64. Farrokhi S, Keyak JH, Powers CM. Individuals with patellofemoral pain exhibit greater patellofemoral joint stress: a finite element analysis study. *Osteoarthritis Cartil.* 2011;19:287–94.
65. Barton RS, Ostrowski ML, Anderson TD, Ilahi OA, Heggeness MH. Intraosseous innervation of the human patella: a histologic study. *Am J Sports Med.* 2007;35:307–11.
66. Sanchís-Alfonso V, Roselló-Sastre E, Saus-Mas J, Revert-Ros F. Biological causes of anterior knee pain, 2011; 33–49.



Management of Patellofemoral Disorders in Basketball

35

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35.1 Introduction

Basketball injuries are most prevalent in the lower extremity, especially ankle and knee. Most basketball injuries are orthopedic in nature, including both acute and overuse injuries. The most prevalent overuse injury of the knee is patellar tendinopathy, which comprises up to 30% of knee injuries seen in sports medicine clinics [1–5]. These injuries are typically associated with the movements of basketball—cutting, pivoting, sudden acceleration/deceleration, and jumping, which explains the colloquially used term “jumper’s knee” describing patellar tendinosis. These repetitive basketball movements can cause excessive loading of the extensor mechanism of the knee, primarily at the patellofemoral joint [3, 5–7], and is a frequent cause of anterior knee pain. Chronic anterior knee pain can be troublesome and resistant to treatment;

indeed, up to 53% of athletes attribute anterior knee pain as a factor in the decision to quit their sport [1, 8, 9]. In this chapter, the management of patellofemoral (PF) disorders in basketball will be discussed.

35.2 Epidemiology

Historically, knee is the second most injured body part in basketball for both men and women at the American high school, collegiate, and professional levels [10–14]. More specifically, injuries involving the patella have been reported to be the second most frequent orthopedic injury in American professional basketball (14.8% of all injuries), as well as being the greatest cause of missed games (19.5%) [15]. Looking specifically at patellar tendonopathy, Lian et al. described an overall prevalence of patellar tendonitis (jumper’s knee) of 14.2% among athletes, with the highest prevalence reported in sports involving high-impact ballistic/explosive loading of the knee extensor mechanism such as volleyball (44.6%) and basketball (31.9%) [16]. Zwerver et al. described a prevalence of 11.8% for “jumper’s knee” in non-elite basketball players [17].

Based on data from American intercollegiate athletics and looking at all PF injuries, female athletes experienced significantly more PF injuries than male athletes in similar sports. Patellar

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tendinitis accounted for 49.2% of all PF injuries and was the most common injury in 20 of 25 studied sports, including men's and women's basketball. For PF injuries in all athletes, the majority are due to overuse or gradual use (41.9%) [18]. In surveying American Collegiate basketball (BB), PF injuries in men's BB ranked second for PF injuries among all sports surveyed, with women's BB ranking fourth, with the injury rate per 100,000 athletic exposures being 28 and 25, respectively [16]. Although lateral patellar dislocation is a less common PF injury in basketball, it has been shown to be the most severe. For American collegiate athletes, lateral patellar subluxation resulted in the most time lost (648 days) and lateral patellar dislocation resulted in the highest average time lost per injury (11.42 days per injury). Lateral patellar dislocation/subluxation also represents the highest proportion of PF injuries that resulted in 14 days or more of time lost and was most likely injury to require surgical intervention [18, 19]. This data did not distinguish between primary and chronic injuries.

Due to the incidence and prevalence of patellar tendinopathies, anterior knee pain, including both PF pain and PF chondral lesions, and patellar instability in the sport of basketball, this chapter will focus on these PF disorders.

35.3 Extensor Mechanism Tendinopathy

35.3.1 Background

Overuse of the extensor mechanism causing tendinopathy can occur above and below the patella. The most common location is the superior insertion of the patellar tendon to the patella (65–70% of the cases), followed by the insertion of the quadriceps tendon at the superior pole of the patella (20–25%), and the patellar tendon insertion on the tibial tuberosity (5–10%) [20].

Quadriceps tendinopathy is less common, but must be considered in the differential of anterior knee pain. Treatment follows similar pathways as patellar tendinopathy, which will be reviewed in more detail below.

Patellar tendinopathy is an overuse condition of the patellar tendon frequently affecting athletes who play basketball and other jumping sports [16, 17, 21, 22]. The pain from patellar tendinopathy can hinder performance on the court, prevent participation, and even cause discomfort with basic activities of daily living [23]. While most basketball players are able to return successfully to the court, there remains a subset of athletes who are unable to play [8]. Patellar tendinopathy remains an area of active research as the pursuit of a better understanding of its pathophysiology can optimize treatment decisions for affected basketball players.

Original descriptions of patellar tendinopathy by Blazina [24] were of a “tendonitis” or inflammation, but the condition is degenerative rather than inflammatory, so “tendinosis” is the more appropriate term. Repetitive overuse and inadequate time for healing are risk factors for patellar tendinosis to occur [25]. Micro-tearing of the tendon occurs almost exclusively in the posteromedial aspect of the tendon insertion into the patella. Hallmark findings of patellar tendinopathy include mucoid degeneration, an inability of the tissue to repair itself, and the absence of inflammatory cells [22, 26].

Extrinsic factors are widely agreed upon to be causative risk factors in the development of patellar tendinopathy. Sports like basketball and volleyball, which require explosive jumping, have been shown to have a prevalence of patellar tendinopathy between 30 and 45%, whereas in sports such as cycling can be very limited [16]. Training load and harder training surfaces are also correlated with a higher incidence of patellar tendinopathy [27, 28]. Many authors have studied intrinsic risk factors, such as patella alta, tilt, leg alignment, obesity (BMI), height, and weight, and none have shown a consistent correlation with patellar tendinopathy [22]. However, in the only prospective study on the development of patellar tendinopathy by Witvrouw et al. [29], the authors did show a statistical difference in hamstring and quadriceps flexibility in the athletes in their population who developed patellar tendinopathy.

Despite limited or conflicting literature on modifiable risk factors for patellar tendinopathy

[30], a lower extremity screening examination pre-season with the identification of quadriceps flexibility, hip joint ROM, and knee joint angle during stop-jump task was found to be meaningful predictors of asymptomatic patella tendon abnormalities and could be used as a screening tool to predict asymptomatic athletes at the risk of developing a patella tendon abnormalities [31]. The importance of lumbosacral control of body movement in athletic training and in rehabilitation is consistent with these screening criteria.

35.3.2 Presentation

Pain initially starts after physical activity and can progress to be continuous during activity or present even at rest. Athletes complain of anterior knee pain, localized to the distal pole of the patella and the proximal end of the patellar tendon [25].

Examination of the athlete starts by observing the athlete perform a double-leg squat. The clinician is looking for asymmetry with this examination, as the athlete will guard and not bend down as far on the involved side. The decline squat test can be used to elicit pain from patellar tendinopathy. In this test, a single-leg squat is performed to 30° of knee flexion—a movement that loads the patellar tendon and causes pain in the affected

region [32]. The authors also recommend obtaining a Beighton's score and checking for quadriceps tightness with the Ely's test. A positive Ely's test occurs when the athlete cannot touch his or her heel to the buttock; this is used as a screening test for quadriceps tightness. Palpation of the distal pole of the patella at the insertion of the proximal patellar tendon should be performed. Resisted knee extension of the involved knee can also elicit pain and is frequently positive in athletes with patellar tendinopathy.

35.3.3 Classification

There are several classification systems for tracking the severity of patellar tendinopathy (Table 35.1). The modified Blazina classification was the first utilized; it is entirely based on clinical symptoms. The Blazina classification system includes four progressive stages graded according to disease severity (Table 35.1). Stage I is characterized by pain that occurs only during sports. In Stage II, pain occurs at the beginning of sporting activities, disappears after warm-up, and reappears when fatigue presents. Stage III is characterized by constant pain during activity and at rest with resultant inability to play the sport. Stage IV disease is defined as complete patellar tendon rupture.

Table 35.1 Different classifications for “Jumper’s knee”

Stage/ grade	Blazina classification [24]	Ferretti classification [28]	Roels classification [33]
0		No pain	
I	Pain only following activity without functional impairment	Pain only following intense sports activity with no functional impairment	Pain at the infrapatellar or suprapatellar region following training or event
II	Pain during and following activity with satisfactory performance levels	Moderate pain during sports activity with no sports performance restriction	Pain at the beginning of activity, disappearing after warm-up and reappearing after activity completion
III	More prolonged pain during and following activity with progressively increasing difficulty performing at a satisfactory level	Pain with slight sports performance restriction	Pain during and after activity. The patient is unable to participate in sports
IV		Pain with severe sports performance restriction	Complete rupture of the tendon
V		Pain during daily activity and inability to participate in sport at any level	

The VISA score (Victorian Institute of Sport Assessment) is a questionnaire consisting of eight questions that help identify severity of tendinopathy and can be used to track responsiveness to treatment [34] (Appendix). The basics of the VISA score are as follows: six of the eight questions are scored on a visual analog scale (VAS) 0–10 (10 being optimum, no issues). The other two questions in VISA ask about current activity level and if the athlete is playing with or playing through pain. The VISA is scored out of 100, with a mean score of 95 seen in asymptomatic athletes, 55 seen in athletes playing with pain and a score of 22 for those requiring surgery [34]. This is the most commonly cited outcome measure score for studies looking at the results of different treatment modalities for patellar tendinopathy.

35.3.4 Imaging

Imaging of choice for the diagnosis of patellar tendinopathy includes both MRI and ultrasound. Both imaging modalities can detect thickening of the tendon, partial tearing of the patellar tendon and intratendinous lesions that reside in the posterior and posteromedial aspect of the proximal patellar tendon [35]. Current advantages of the MRI are the ability to rule out other intra-articular causes of pain and to help evaluate for partial thickness patellar tendon tears. Recent work has demonstrated a high correlation of patellar tendinopathy progressing to a partial tear if the axial thickness on MRI cut is greater than 8.8 mm [36] (Fig. 35.1). Furthermore, a novel classification system for patellar tendinopathy has been created based on MRI findings that can help with management decisions (Table 35.2).

35.3.5 Treatment

There are a wide range of treatment modalities available to treat basketball players with patellar tendinopathy, but the optimal treatment algorithm is still a work in progress [21].

Physical therapy with an emphasis on eccentric exercises should be the initial management



Fig. 35.1 MRI image of a 20-year-old basketball player with 1 year history of right knee pain, on presentation to the point he was no longer playing. His Blazina score was 3. VISA score was 29. On the Popkin-Golman classification system for patellar tendinopathy, he was Grade 4. The patient underwent an open debridement and repair

Table 35.2 Popkin-Golman MRI classification system for patellar tendinopathy

Grade	Criteria Tear % on axial MRI	Tendon thickness, mm on axial cut	Patellar tendinopathy
Grade 1	No tears seen on MRI	6	Patellar tendinopathy
Grade 2	Signal seen <25% of axial cuts	8.7 ^a	Patellar tendinopathy
Grade 3	25–50% on axial thickness	10	Partial patellar tendon tear
Grade 4	Tear >50% of axial thickness	>11	Partial patellar tendon tear at risk for poor outcome with conservative treatment>6 months

Adapted from: Popkin CA, Golman M, Wong TT, Wright ML, Lynch TS, Thomopolous S, Ahmad CS (2019) Rethinking Partial Patellar Tendon Tears: A Novel Classification System. AOSSM Annual Meeting July 10, 2019 Boston, Massachusetts

^aTendon thickness of 8.8 mm on axial cut strongly correlated with the finding of a partial patellar tendon tear

in patellar tendinopathy, as it is the only modality of treatment for patellar tendinopathy backed by high-level evidence [37–39]. Eccentric exercises have been found to have positive effects, with minimal adverse reactions, as long as the athlete is removed from the sport [40]. The duration of training should be attempted for at least 3 months. Numerous studies point to the eccentric quadriceps strengthening using a decline board to maximize results [41, 42]. It should also be noted that excellent results may be achieved combining eccentric strengthening with a supplemental protocol. For example, Dimitrios et al. found superior results with the eccentric training combined with a stretching program (89% improvement VISA Score) compared to eccentric training alone (57% improvement VISA score) [39].

Patellar tendon strap and sports tape can also be utilized first line for athletes suffering from symptoms of patellar tendinopathy. A number of studies show that an infrapatellar strap or sports tape can be used to help alleviate pain and can improve symptoms when participating in jumping activities [43–45]. Patellar tendon straps and sports tape are widely available, easy to use, safe, and short-term options for the treatment of initial symptoms of patellar tendinopathy.

The use of extracorporeal shock wave therapy (ESWT) has a role in the treatment of patellar tendinopathy and can be grouped in the first line of treatment options [23]. There is considerable variability in outcomes reported using ESWT. The best outcomes in the literature are from Wang et al. [44] who used 1500 impulses (at 14 kV, 0.18 mJ/mm²) in a single session. A randomized controlled trial done by Zwerver et al. used high doses of ESWT over three weekly sessions (2000 impulses at 4 Hz for maximum up to 0.58 mJ/mm²) and compared this to athletes with patellar tendinopathy who received sham ESWT. They found no differences in the groups [46]. The same group did a follow up, double-blind randomized controlled trial comparing eccentric strengthening and ESWT (1000 impulses at 4 Hz, 0.2 mJ/mm²) to eccentric strengthening and sham ESWT. They also showed no difference

between the two groups looking at changes in VISA scores [47]. Results of ESWT reported by Peers et al. showed improvement in outcomes with low to intermediate dosing (1000 impulses, 4 Hz for 0.08 mJ/mm²) over three weekly sessions [48]. There remains considerable debate as to the usefulness of ESWT, but it is a safe treatment modality with no significant risk.

Platelet-rich plasma (PRP) injections have been shown to have some benefit in the treatment of patellar tendinopathy but have shown wide variability in efficacy [49, 50]. This is likely the result of significant variability in the protocols for the administration of PRP and the lack of high-quality studies demonstrating clear benefit. A recent systematic review showed a protocol with multiple PRP injections may have a role in treatment for athletes with patellar tendinopathy [38]. The role of PRP in the treatment of patellar tendinopathy remains a topic of discussion and an area that requires more study.

There is no role at this time for the use of corticosteroid injections for the treatment of patellar tendinopathy, and they should not be used [23, 32]. Several studies have shown no benefit [42, 51], and steroid injections are not recommended as part of the treatment algorithm for patellar tendinopathy.

The literature reports that 10–15% of cases of patellar tendinopathy do not respond to the above measures and require surgical management [32]. There is a growing body of literature to support the role for surgical treatment for refractory patellar tendinopathy [48, 52, 53]. Most studies did not recommend or advocate surgical management until the athlete had failed conservative measures for more than 6 months [23]. However, it is the opinion of the author that if there are Grade 3–4 MRI changes within the tendon (Table 35.2), and the athlete shows poor flexibility (Beighton's score of 0–2) that these athletes respond poorly to non-operative management and might benefit from early surgical intervention (personal communication, CP, 2019).

There is no agreement on the optimal surgical procedure of choice for refractory patellar

Table 35.3 Patellar tendinopathy treatment options

	Treatment protocol for patellar tendinopathy
First-line treatments	Eccentric quadriceps strengthening and training <ul style="list-style-type: none"> • Use of 25° decline board • Focus also on stretching and flexibility • 2–3× a week for a minimum of 3 months
	Patellar tendon strap or sports tape <ul style="list-style-type: none"> • Studies show reduction in pain with jumping activities
	Extracorporeal shock wave therapy <ul style="list-style-type: none"> • Low to medium dose frequency
Second-line treatment	PRP injections <ul style="list-style-type: none"> • Multiple injection protocols shown better results • Another non-surgical option
Final option after failing >6 months of the above	Surgical debridement <ul style="list-style-type: none"> • Recommend open surgical approach

tendinopathy. Results with both arthroscopic and open approaches are good with most authors noting a significant jump in VISA score and return to sport. There is some variability described in postoperative rehabilitation and return to sport with different studies, but most had the athletes back to their sport within 4 months of the procedure. The authors’ preferred approach at this time is an open procedure, as it allows for thorough debridement of the inferior pole, with an option for use of anchor and suture repair of the tendon and use of 2.0 mm K-wire for drilling the inferior pole for marrow stimulation [36]. A list of treatment options is reviewed in Table 35.3.

35.4 Anterior Knee Pain

35.4.1 Presentation

Athletes with anterior knee pain (AKP) typically describe diffuse ill-defined anterior, peripatellar, or retropatellar knee pain [54, 55]. AKP is commonly thought of as that which originates in anterior knee structures in the absence of an identifiable acute or overuse injury. Insidious onset of pain or repetitive motion activity are

Table 35.4 Differential causes of AKP in children and adult

<i>Apophyseal</i>
Osgood–Schlatter disease
Sinding-Larsen-Johansson syndrome
<i>Bone and cartilage</i>
Articular cartilage injury
Bone tumors, in particular the distal femur, a common site for adolescent tumors
Loose bodies
Osteochondritis dissecans, especially common in the distal femur in the adolescent age group
Patellar instability/subluxation
Patellar stress fracture
Symptomatic bipartite patella
Excessive external tibial torsion
<i>Inflammatory diseases</i>
Rheumatologic
Infectious (i.e., Lyme disease)
<i>Muscle/tendon</i>
Quadriceps tendinopathy
Patellar tendinopathy
<i>Nerve</i>
Complex regional pain syndrome (CRPS)
Neuroma
Saphenous neuritis
<i>Referred pain</i>
Lumbar spine pathology
Hip joint pathology (i.e., slipped capital femoral epiphysis, femoral-acetabular impingement)
<i>Soft tissue</i>
Fat pad impingement
Iliotibial band tendonitis
Pes anserine bursitis
Prepatellar bursitis
Plica irritation

common presentations, but specific or a series of loading events are also noted. AKP has a primary mechanism in overuse injury principles, even though the exact etiology and nature of the pain are poorly understood. An overuse injury is considered repetitive submaximal or subclinical trauma that has over-exceeded tissue’s clinical responsiveness, which leads to pain and/or movement dysfunction.

Because the specific cause of pain in the AKP is indistinct, the clinician’s primary initial task is to rule out other known causes of pain around the knee (Table 35.4). Patients present with stereotypical symptoms, anterior knee pain aggravated both by loaded, flexed-knee activity (stair climb-

ing, jumping, and squatting) and by unloaded, prolonged bent-knee activity such as sitting with a flexed knee.

Patellar retinacular structures extend both medially and laterally from the patella, thereby pain can be associated with medial/lateral/posterior pain. Indeed, despite its common term of “anterior” knee pain, pain in almost every location around the knee can relate to this pain presentation, which can create confusing clinical presentations. AKP is rarely associated with swelling and, when present, one must consider intra-articular damage, most specifically cartilage damage; indeed, the most common form of AKP has no to minimal cartilage damage. The presence of patella cartilage damage is harder to sort out, as patellar cartilage wear is often asymptomatic and metabolically stable and, therefore, is not a source of pain. Giving-way episodes can be reported, typically occurring when one tries to engage the quadriceps muscle, the quad “fatigues,” creating a giving-way episode. It is important for the treating clinician not to confuse these giving-way episodes with ligamentous instability, which typically occurs with planting, pivoting, or jumping activities. Knee “locking” may also be reported. If the knee locks in full extension, it can be a manifestation of an extensor mechanism abnormality; the patient does not want to engage the kneecap in the groove because of pain and therefore keeps the leg straight. If the knee is locked secondary to a loose body or torn meniscus, it is locked in some degree of knee flexion.

Changes in one’s daily routine may play a role in changing one’s envelope of function. Training schedules, i.e., how much load and for how long, may offer important information as to the level of activity and possible overuse. However, this change could be more subtle (e.g., changing schools with increased demands of more walking or hill climbing). Included in “change,” it is important to identify stage of growth and growth history as it relates to muscular control of lengthening lever arms. There may be sex variability in neuromuscular growth spurts with disproportionate height and weight increases [56, 57].

35.4.2 Imaging

Many patients with AKP have normal imaging, and many with abnormal findings on imaging have no AKP. No source of pain should be considered based on imaging alone (e.g., increased TT–TG, patella alta). Radiographic imaging is not necessary to make the diagnosis of anterior knee pain, but certain imaging may help to support the diagnosis (Table 35.5).

The goal of the radiographical assessment in acute anterior knee pain is to determine whether soft tissue or cartilage injury exists. An MRI is the most specific modality for these findings. Basketball athletes are particularly susceptible to chondral lesions of the patellofemoral joint, but may not always be symptomatic. In professional and collegiate asymptomatic male BB athletes, imaging studies have shown trochlear groove lesions up to 25.9% and patella lesions up to 44.4% [58–60].

35.4.3 Treatment

Altered neuromotor control is associated with anterior knee pain, although the exact mechanism is speculative. Restoration of neuromuscular control is the main treatment goal, concentrating on muscle strengthening and improved body move-

Table 35.5 Common imaging tests for evaluation of patellofemoral disorders

Radiographic view	Evaluation
Axial views:Low flexion view and mid flexion view 20° and 60° view	Sulcus angle, patella position in the groove (excessive lateral tilt and/or translation), PF joint space narrowing (in older group)
True lateral view	Trochlear dysplasia, patella height, PF joint space narrowing (in older population), etc.
Standing AP	Tibiofemoral (TF) joint space narrowing, limb alignment
Standing PA flexion	TF joint space narrowing, especially lateral-based disease
Magnetic resonance imaging	Soft tissue, ligament, and cartilage stress injuries to bone
Computed tomography	Fractures Limb version

ment patterns. Extensor mechanism weakness is a characteristic finding in AKP patients, as well as being a risk factor for developing AKP [61]. Historically, quadricep strengthening has been the main focus of rehabilitation of AKP patients. This becomes challenging in an athletic population, where absolute quadricep strength may not be deficit, and further evaluation is prudent.

Hip muscle weakness and impaired gluteal muscle function can lead to increased hip joint adduction and internal rotation during sports-specific body movement patterns [62, 63]. In addition to strength, the dysfunction in neuromuscular control in AKP patients may stem from disordered firing sequences of the muscles [29, 64, 65]. Interventions focused on correcting deficits with hip strengthening, biofeedback or gait retraining, should be included in the treatment of AKP.

More recently, core strength, including hip and abdominal muscles, has been identified as important to include in the management of AKP [66, 67]. Stretching to improve muscle tendon length should be part of the treatment plan. Reduced quadriceps muscle length [29], as well as decreased hip flexibility [68], is a frequent finding in symptomatic patients.

Examining a double- or single-leg squat in both frontal and sagittal planes is another simple clinical office or training room (Fig. 35.2a, b) test to assess and direct treatment. Observing for dynamical alignment gives a snapshot of the forces that the knee may be subjected to with increased force and repetition while participating in sport. In the frontal plane, functional valgus with dynamic activities like the single- and double-leg squat may be a result of weakness of core musculature. In the sagittal plane, increased anterior knee translation over the foot can be indicative of poor squat mechanics and lack of gluteal involvement in this body movement. Gripping of the toes and increased ankle dorsiflexion can often be a sign of anterior muscle recruitment and increase load through the anterior knee.

If one part of the kinetic lower extremity chain is weak or injured, the body often finds ways to accomplish an activity by a “work around” the injured body part. This often initiates faulty body mechanics that can lead to a painful state that centers on the patella and its associated soft tissue structures. Patients with AKP develop strategies to compensate for strength and range-of-motion deficits, which can lead to symptom

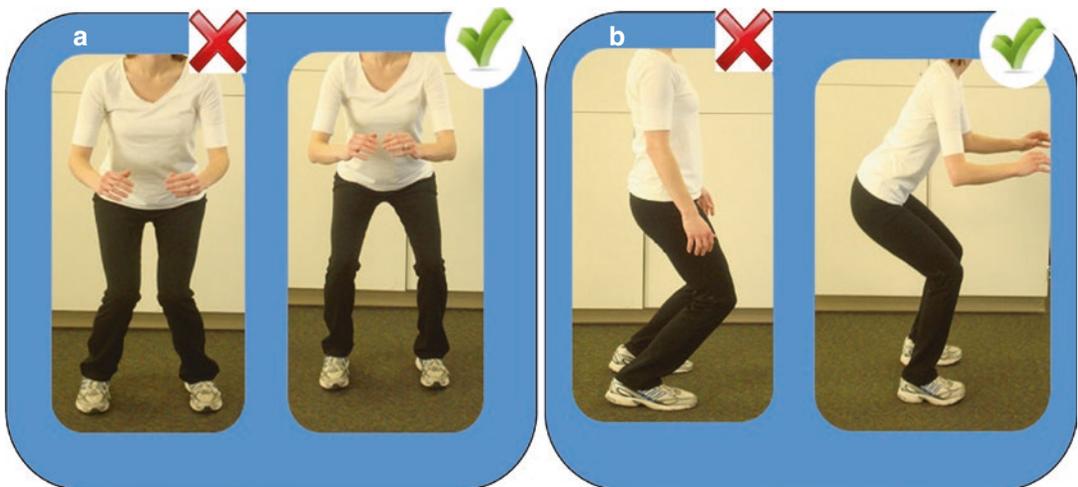


Fig. 35.2 (a) Frontal plane images of a patient performing a squat. **X** marks incorrect body mechanics with functional valgus knee position with squatting. The **✓** image displays proper body movement pattern. (b) Sagittal plane

images of a patient performing a squat. **X** marks incorrect body mechanics with anterior knee excursion and no gluteal engagement with squatting. The **✓** image displays proper body movement pattern

exacerbation. In accomplished athletes, changes in body movement patterns can be subtle, making treatment of AKP difficult for the athlete and the rehabilitation specialist. The main role of the therapist is to recognize these faulty body movement patterns, dissect the underlying muscle-deficit cause, and build an individual program to address this [69]. This approach focuses on movement-centered therapy with a skilled physical therapist. Treatment should be customized to the patient's level of strength and fitness and whenever possible should be made challenging for the athlete. The patients' response to each intervention will help to guide the next step as well as define the need for further intervention.

Treatment for patients with AKP is challenging from both exercise dosage and duration perspective. The challenge to the therapist is to manage this within the envelope of function described by Scott Dye [70]. For those treating the athlete, decisions to include/exclude continued practice and games when activities of daily living (e.g., stair climbing) cause pain are difficult to balance and must be individualized according to time of season, player position, etc. Treatment of AKP in the athlete nearly always involves reduction of activity; prudent reduction of drills and conditioning while undergoing a treatment focused on strength and flexibility deficits is a good starting point. However, if this fails to reduce pain, further activity reduction is merited. Key to "in season" treatment of anterior knee pain is to avoid pain escalation and stay within the athlete's functional envelope. At times full resolution of pain does not occur until off season.

35.5 Patellar Chondral Wear

In BB athletes, PF cartilage is at risk for degenerative changes in both the patella and trochlear. Jumping sports are particularly prone to central trochlear groove cartilage wear. A recent study of PF chondral wear and associated anatomic patellar instability factors found that trochlear lesions were more frequent in men, presented at an older age, and had fewer associated anatomic risk factors. Patellar lesions, conversely, were more frequent in

women, presented at younger age, and were more closely associated with anatomic risk factors [71].

Treatment of cartilage injuries of the PF joint is beyond the scope of this chapter; however, a cautious approach should be used in surgical management of anterior knee pain when there is MR evidence of chondral wear, as these lesions are found in a high percentage of asymptomatic BB athletes [58–60]. For most athletes presenting with anterior knee pain in the absence of an acute injury, with examination findings of a knee effusion, with or without imaging verification of chondral wear, a non-operative approach is recommended. The approach outlined in the section previously provides a framework for individualized treatment. If the chondral wear pattern is laterally based in the PF joint, McConnell taping may be helpful in the rehabilitative process [72].

35.6 Lateral Patellar Dislocations (LPD)

35.6.1 Presentation

An acute LPD is most often a non-contact injury with a planted foot and upper body rotation. The patella dislocates laterally, injuring medial restraining structures, causing a large hemarthrosis. The lateralized patella often reduces spontaneously; less often, the kneecap remains unreduced necessitating a manual reduction. This section will focus on treatment and return to play.

Currently, most patients following first-time LPD are managed non-operatively. Studies demonstrate an unacceptably high redislocation rate in these patients [73–76].

However, studies have not demonstrated significant functional differences using outcome instruments between operative/non-operative management following first-time LPD [77–81].

Though first-time LPD can span all age groups, they are particularly prevalent in the teenage years [82–84]. In addition to presenting at a young age, younger patients with first-time LPD have more anatomic patella instability factors than those presenting with their first injury at a later age [85].

Several recent studies have focused on algorithms to predict risk of re-injury [86–88].

Though no one algorithm has been verified to date, certain anatomic and demographic risk factors have been identified with increased risk of re-injury.

Key Risk Factors for Recurrence of Lateral Patella Dislocation (LPD)

- Open physes
- Previous LPD
- Family history/contralateral LPD
- Hyperlaxity, especially knee hyperextension
- Patella alta
- Trochlear dysplasia

35.6.2 Imaging

Imaging studies are obtained to confirm the diagnosis, recognize associated injuries, and identify anatomic abnormalities that may have predisposed the patient to a patellar dislocation. Objective measurements of patella position and trochlear morphology, often referred to as “anatomic patellar instability risk factors” (APIF), are used to guide surgical planning [84]. More recently, in patients with a number of risk factors, surgical management of first-time lateral patellar dislocation can be advised.

35.7 Management of the Acute Injury

Aspiration may be useful for comfort when swelling is marked [89, 90] and has a possible benefit for articular cartilage [91].

35.7.1 Immobilization Versus Functional Progression

There is a paucity of evidence-based information in the literature concerning the most appropriate immediate treatment of LPD that reduces symp-

toms and minimizes recurrence. There is limited evidence to support knee immobilization; immobilization periods varying from 0 to 6 weeks [77, 92–94].

Rood et al. [95] report the only randomized control study of cast versus taping, which suggested taping had better subjective outcomes in the 18 patients studied. Follow-up was up to 5 years, and there was no recurrence in either group. Maenpaa et al. [96] reported a prospective randomized study comparing immediate immobilization with flexion restriction utilizing a patellar brace and found no difference in recurrence rate at 2 years. A questionnaire survey of the sports subcommittee of ESSKA (16 responses) queried physicians on their preference for non-operative management of primary LPD; nearly all recommended a short period of immobilization [97].

There is no consensus on initial treatment, and to date, our early management of acute LPD lacks an evidence base [75, 93]. There are no studies that categorize patients according to injury variables, including contact versus non-contact injury, the presence or absence of osteochondral fragments, or anatomic variables such as patella alta or trochlear dysplasia. However, some studies suggest that contact injuries or the presence of osteochondral fractures are associated with a lower incidence, whereas patella alta or trochlear dysplasia are associated with a higher incidence of recurrence [82].

35.7.2 Rehabilitation Post Acute Dislocation

Physiotherapy is essential for optimal recovery of function. Rehabilitation should focus on a goal to return to sport around 4–6 weeks, but this varies widely depending on osteochondral injuries, degree of swelling, and pre-injury strength. Functional progression incorporating overlapping stages of physical therapy progression is advised (Table 35.6).

An ISAKOS Consensus task force (2015) on patellar dislocations concluded the following in the management of acute patellar dislocations:

Table 35.6 Functional progression after an acute LPD

• Recovery of normal knee motion and gait mechanics
• Core principles
• Double-leg movement patterns
• Single-leg movement patterns
• Restoration of proper squat mechanics
• Dynamic movements mimicking type and intensity of desired activity
• Eccentric control of quadriceps activity
• Neuromuscular training/proprioception
• Sports-specific training/exercises

- The knee should be protected with crutches and/or limited motion as required until a normal gait pattern is restored.
- The initial management should be focused on reducing swelling and restoring lower limb muscle function. Ideally this is carried out under the guidance of a physiotherapist, certified athletic trainer, or other trained professional. The goal is to restore the envelope of tissue function before progressing to the next phase of rehabilitation.
- Following a new injury, recurrent dislocators with minimal trauma and swelling can be accelerated in the early management phase.
- The use of a knee sleeve for comfort, control of swelling, and/or proprioception confidence is optional.
- There is no evidence to support using specific patellar stability braces for preventing recurrent dislocation [95, 96].
- Adequate performance in PT with sport-specific drills which simulate the intensity and body movement patterns of the athlete’s given sport/activity prior to return to play is recommended.

35.7.3 Return to Play After Patellar Stabilization Surgery

The goal of this chapter is to advise on safe return to play after patellar dislocation and after reconstructive surgery, not to guide the type of surgery to be performed. Surgical procedures to stabilize against recurrent patella dislocations are highly individualized and variable [98]. Surgical proce-

Table 35.7 Return-to-sport study timelines for “isolated” MPFL reconstructions [102]

Study	Stated timeline for return to sport
Ahmad et al. [103]	Running agility after 3 months; full sports at 4 months postoperatively
Christiansen et al. [104]	Controlled sports at 12 weeks; contact sports at 6 months
Gomes [105]	Return to sports at 6 months; some motivated patients return by 4 months
Fernandez et al. [106]	Full activity at 12 weeks
Mikashima et al. [107]	Jogging and “mild” sports at 4 months; full sports at 6 months
Nomura et al. [108]	Jogging and “mild” sports at 8 weeks; full sports at 12 weeks
Ronga et al. [101]	Sports at 6 months
Schöttle et al. [109]	Full activity at 6 months if ROM and quadriceps strength restored
Thaunat and Erasmus [110]	Full sports when quadriceps rehabilitation achieved
Watanabe et al. [111]	Jogging at 12 weeks with good strength, ROM, stability; return to sports at 6 months

ROM range of motion

dures follow a similar functional progression to post dislocation rehabilitation. However, surgery is initially restricted by post-operative pain and is delayed based on what we know of the biology of healing:

- MPFL reconstruction: tendon to bone healing—3 months [99]
- Tibial tubercle osteotomy: bone to bone healing—6+ months; follow-up with radiographs

A recent systematic review of MPFL reconstruction looked specifically at rehabilitation and return-to-sports efficacy. Poor study methodology, as well as limited exercise rehabilitation information, makes it difficult to determine sports efficacy. Table 35.7 reviews specific studies and their stated timelines for return to sport [100].

Though there are a limited number of published outcomes after MPFL reconstruction that discuss a proposed timeline for return to sport, few studies detail when the *patients actually returned to their sport*. One study of 28 patients, largely male (75%), details a cohort of nine

Table 35.8 Physical performance testing elements

Domain tested	Test activity	Recorded value
Anthropometric data	Knee ROM	Degrees of motion
	Joint line circumference	Centimeters around the joint line
	Thigh circumference	Centimeters around the thigh (15 cm proximal to suprapatellar border)
Core stability	Prone plank timed hold (Fig. 35.3a)	Seconds held, maintaining ideal alignment (out of maximum of 60 s)
	Side plank timed hold (Fig. 35.3b)	Seconds held, maintaining ideal alignment (out of maximum of 60 s)
	Single-leg bridge repetitions to fatigue (Fig. 35.3c)	Maximum repetitions to muscle fatigue
Balance	Single-limb balance with eyes closed	Seconds held (out of maximum of 60 s)
	Single-limb stand and reach (Fig. 35.4a)	Centimeters reached with opposite arm of stance limb
	Star excursion balance test (Fig. 35.4b, c)	Centimeters reached with opposite toe from stance limb
Lower extremity muscle strength	Single-limb maximum depth squat (Fig. 35.5a)	Maximum knee flexion angle reached at depth of squat
	Retro step-up/down (Fig. 35.5b)	Maximum step height successfully completed (inches)
Lower extremity muscle endurance	2-min single-leg repeated squat test	Maximum number of squats completed to 60° KF at 60 bpm tempo × 2 min, preserving ideal trunk and limb alignment (max value = 60 squats)
Lower extremity power	Single-limb hop for distance	Maximum distance hopped in centimeters/meters
	6 m timed hop	Maximum speed recorded in seconds
	Triple cross-over hop for distance	Maximum distance hopped in centimeters/meters

patients returning to high-level pivoting activities at a mean of 7.5 months (6–16 month range) [101]. Patients with major osteochondral/chondral lesions with or without repair have a low chance of returning to high-level sports participation. There is little information in the literature that discusses timelines for return to sport for other patellofemoral surgical procedures, in particular tibial tubercle osteotomy.

Table 35.8 details possible physical performance testing activities that can be used to measure or test knee/patellofemoral function.

Table 35.9 outlines general prerequisites for safe return to sport.

Table 35.9 Prerequisites for safe return to sport

• If bony surgery is involved, complete radiographic healing of bone
• No complaints of knee pain or knee instability
• Full or near-full range of motion
• No knee effusion
• Completed neuromuscular training/proprioception
• Satisfactory core strength and endurance
• Acceptable control with dynamic activities (e.g., star excursion balance test)
• Limb symmetry index >85% on hop tests, especially if resuming pivoting sports
• Adequate performance with physiotherapist during sport-specific drills simulating the intensity and movement patterns of the athlete’s given sport
• Athlete demonstrates a psychological readiness to return to sport (e.g., SANE score > 80/100)



Fig. 35.3 (a) This is an illustration of a prone plank, which is used as a measure of bilateral lower extremity core stability in addition to upper body strength. (b) This is an illustration of a side plank, which in this illustration is used as a measure of right-sided core stability, in addition to upper body strength. (c) This is an illustration of a single-leg bridge, which in this illustration is used as a measure of right-sided core stability

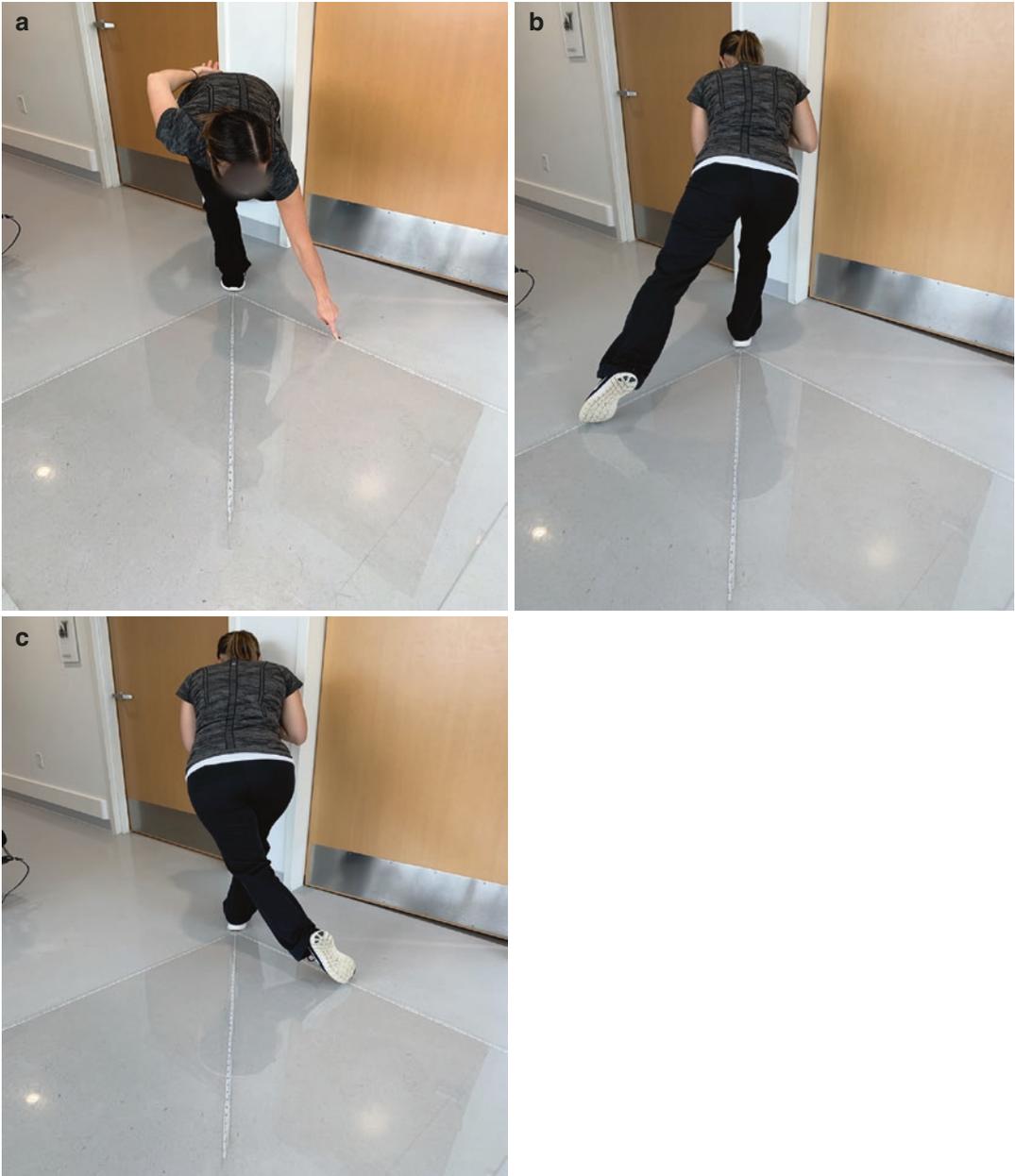


Fig. 35.4 (a) This is an illustration of a single-leg stand and reach which is used as a measure of balance on the right lower limb, reaching with the left arm. (b) This is an illustration of the star excursion balance test reaching for a mark on the side ipsilateral to the side being measured

for balance (right lower limb in this illustration). (c) This is an illustration of the star excursion balance test reaching for a mark on the side contralateral to the side being measured for balance (right lower limb in this illustration)



Fig. 35.5 (a) This is an illustration of a single-leg maximum squat, which is used as a measure of right lower limb strength (primarily quadriceps muscle strength) and balance. (b) This is an illustration of a retro step up, which is

used as a measure of right lower limb strength (primarily quadriceps muscle strength) and core (maintaining a level pelvis)

35.8 Conclusion

Basketball is a sport that demands much of the PF joint, with jumping, sudden deceleration, and pivoting activity. PF overuse injuries and acute contact and non-contact injuries are common.

There is no ideal or correct time for an athlete to return to sport after an injury; return to sport is

patient- and sport-specific and dependent on the athlete's function as assessed by the physiotherapist/athletic trainer/physician. Functional progression incorporating increasing challenge and complexity of sport-specific tasks is recommended. The ultimate goal of management is to minimize recurrence and safely return athletes to previous level of play.

Appendix

VISA SCORE

1. For how many minutes can you sit pain free?

0 mins 100 mins Points

0 1 2 3 4 5 6 7 8 9 10

2. Do you have pain walking downstairs with a normal gait cycle?

strong
severe no pain Points pain

0 1 2 3 4 5 6 7 8 9 10

3. Do you have pain at the knee with full active non-weight bearing knee extension?

strong
severe no pain Points pain

0 1 2 3 4 5 6 7 8 9 10

4. Do you have pain when doing a full weight bearing lunge?

strong
severe no pain Points pain

0 1 2 3 4 5 6 7 8 9 10

5. Do you have problems squatting?

Unable no problems Points 0 1

2 3 4 5 6 7 8 9 10

6. Do you have pain during or immediately after doing 10 single leg hops?

strong severe no pain Points

pain/unable
0 1 2 3 4 5 6 7 8 9 10

7. Are you currently undertaking sport or other physical activity?

- 0 Not at all
- 4 Modified training ± modified competition
- 7 Full training ± competition but not at same level as when symptoms began
- 10 Competing at the same or higher level as when symptoms began

8. Please complete **EITHER A, B or C** in this question.

- If you have **no pain** while undertaking sport please complete **Q8a only**.
- If you have **pain while undertaking sport but it does not stop you** from completing the activity, please complete **Q8b only**.
- If you have **pain that stops you from completing sporting activities**, please complete

Q8c only.

8a. If you have **no pain** while undertaking sport, for how long can you train/practise?

NIL 1-5 mins 6-10 mins 7-15 mins >15 mins

References

- Devereaux MD, Lachmann SM. Patello-femoral arthralgia in athletes attending a sports injury clinic. *Br J Sports Med.* 1984;18(1):18–21.
- Halabchi F, Abolhasani M, Mirshahi M, Alizadeh Z. Patellofemoral pain in athletes: clinical perspectives. *Open Access J Sports Med.* 2017;8:189–203.
- Petersen W, Rembitzki I, Liebau C. Patellofemoral pain in athletes. *Open Access J Sports Med.* 2017;8:143–54.
- Smith BE, Selve J, Thacker D, Hendrick P, Bateman M, Moffatt F, et al. Incidence and prevalence of patellofemoral pain: a systematic review and meta-analysis. *PLoS One.* 2018;13(1):e0190892.
- Swenson DM, Collins CL, Best TM, Flanagan DC, Fields SK, Comstock RD. Epidemiology of knee injuries among U.S. high school athletes, 2005/2006–2010/2011. *Med Sci Sports Exerc.* 2013;45(3):462–9.
- Lewallen L, McIntosh A, Dahm D. First-time patellofemoral dislocation: risk factors for recurrent instability. *J Knee Surg.* 2015;28(4):303–9.
- Mai HT, Alvarez AP, Freshman RD, Chun DS, Minhas SV, Patel AA, et al. The NFL orthopaedic surgery outcomes database (NO-SOD): the effect of common orthopaedic procedures on football careers. *Am J Sports Med.* 2016;44(9):2255–62.
- Kettunen JA, Kvist M, Alanen E, Kujala UM. Long-term prognosis for Jumper's knee in male athletes. A prospective follow-up study. *Am J Sports Med.* 2002;30(5):689–92.
- Scattone Silva R, Nakagawa TH, Ferreira AL, Garcia LC, Santos JE, Serrao FV. Lower limb strength and flexibility in athletes with and without patellar tendinopathy. *Phys Ther Sport.* 2016;20:19–25.
- Clifton DR, Hertel J, Onate JA, Currie DW, Pierpoint LA, Wasserman EB, et al. The first decade of web-based sports injury surveillance: descriptive epidemiology of injuries in US High School Girls' Basketball (2005–2006 through 2013–2014) and National Collegiate Athletic Association Women's Basketball (2004–2005 through 2013–2014). *J Athl Train.* 2018;53(11):1037–48.
- Clifton DR, Onate JA, Hertel J, Pierpoint LA, Currie DW, Wasserman EB, et al. The first decade of web-based sports injury surveillance: descriptive epidemiology of injuries in US High School Boys' Basketball (2005–2006 through 2013–2014) and National Collegiate Athletic Association Men's basketball (2004–2005 through 2013–2014). *J Athl Train.* 2018;53(11):1025–36.
- McCarthy MM, Voos JE, Nguyen JT, Callahan L, Hannafin JA. Injury profile in elite female basketball athletes at the Women's National Basketball Association combine. *Am J Sports Med.* 2013;41(3):645–51.
- Borowski LA, Yard EE, Fields SK, Comstock RD. The epidemiology of US high school basketball injuries, 2005–2007. *Am J Sports Med.* 2008;36(12):2328–35.
- Zuckerman SL, Wegner AM, Roos KG, Djoko A, Dompier TP, Kerr ZY. Injuries sustained in National Collegiate Athletic Association men's and women's basketball, 2009/2010–2014/2015. *Br J Sports Med.* 2018;52(4):261–8.
- Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the national basketball association: a 17-year overview. *Sports Health.* 2010;2(4):284–90.
- Lian OB, Engebretsen L, Bahr R. Prevalence of jumper's knee among elite athletes from different sports. *Am J Sports Med.* 2005;33(4):561–7.
- Zwerver J, Bredeweg SW, van den Akker-Scheek I. Prevalence of Jumper's knee among nonelite athletes from different sports: a cross-sectional survey. *Am J Sports Med.* 2011;39(9):1984–8.
- Trojan JD, Treloar JA, Smith CM, Kraeutler MJ, Mulcahey MK. Epidemiological patterns of patellofemoral injuries in collegiate athletes in the United States from 2009 to 2014. *Orthop J Sports Med.* 2019;7(4):2325967119840712.
- Mitchell J, Magnussen RA, Collins CL, Currie DW, Best TM, Comstock RD, et al. Epidemiology of patellofemoral instability injuries among high school athletes in the United States. *Am J Sports Med.* 2015;43(7):1676–82.
- King D, Yakubek G, Chughtai M, Khlopas A, Saluan P, Mont MA, et al. Quadriceps tendinopathy: a review, part 2-classification, prognosis, and treatment. *Ann Transl Med.* 2019;7(4):72.
- Dan MJ, McMahon J, Parr WCH, Broe D, Lucas P, Cross M, et al. Evaluation of intrinsic biomechanical risk factors in patellar tendinopathy: a retrospective radiographic case-control series. *Orthop J Sports Med.* 2018;6(12):2325967118816038.
- Dan M, Parr W, Broe D, Cross M, Walsh WR. Biomechanics of the knee extensor mechanism and its relationship to patella tendinopathy: a review. *J Orthop Res.* 2018;36(12):3105–12.
- Everhart JS, Cole D, Sojka JH, Higgins JD, Magnussen RA, Schmitt LC, et al. Treatment options for patellar tendinopathy: a systematic review. *Arthroscopy.* 2017;33(4):861–72.
- Blazina ME, Kerlan RK, Jobe FW, Carter VS, Carlson GJ. Jumper's knee. *Orthop Clin North Am.* 1973;4(3):665–78.
- Khan KM, Cook JL, Kannus P, Maffulli N, Bonar SF. Time to abandon the "tendinitis" myth. *BMJ.* 2002;324(7338):626–7.
- Khan KM, Maffulli N, Coleman BD, Cook JL, Taunton JE. Patellar tendinopathy: some aspects of basic science and clinical management. *Br J Sports Med.* 1998;32(4):346–55.
- Visnes H, Bahr R. Training volume and body composition as risk factors for developing jumper's knee among young elite volleyball players. *Scand J Med Sci Sports.* 2013;23(5):607–13.

28. Ferretti A. Epidemiology of jumper's knee. *Sports Med.* 1986;3(4):289–95.
29. Witvrouw E, Lysens R, Bellemans J, Cambier D, Vanderstraeten G. Intrinsic risk factors for the development of anterior knee pain in an athletic population. A two-year prospective study. *Am J Sports Med.* 2000;28(4):480–9.
30. Sprague AL, Smith AH, Knox P, Pohlig RT, Gravare SK. Modifiable risk factors for patellar tendinopathy in athletes: a systematic review and meta-analysis. *Br J Sports Med.* 2018;52(24):1575–85.
31. Mann KJ, Edwards S, Drinkwater EJ, Bird SP. A lower limb assessment tool for athletes at risk of developing patellar tendinopathy. *Med Sci Sports Exerc.* 2013;45(3):527–33.
32. Figueroa D, Figueroa F, Calvo R. Patellar tendinopathy: diagnosis and treatment. *J Am Acad Orthop Surg.* 2016;24(12):e184–e92.
33. Roels J, Martens M, Burssens A. Patellar tendinitis (jumper's knee). *American Journal of Sports Medicine.* 1978;6:362–8.
34. Visentini PJ, Khan KM, Cook JL, Kiss ZS, Harcourt PR, Wark JD. The VISA score: an index of severity of symptoms in patients with jumper's knee (patellar tendinosis). Victorian Institute of Sport Tendon Study Group. *J Sci Med Sport.* 1998;1(1):22–8.
35. Warden SJ, Kiss ZS, Malara FA, Ooi AB, Cook JL, Crossley KM. Comparative accuracy of magnetic resonance imaging and ultrasonography in confirming clinically diagnosed patellar tendinopathy. *Am J Sports Med.* 2007;35(3):427–36.
36. Popkin CA, Golman M, Wong TT, Wright ML, Lynch TS, Thomopoulos S, et al. Rethinking partial patellar tendon tears: a novel classification system. AOSSM Annual Meeting; July 11–14, 2019; Boston, MA; 2019.
37. Larsson ME, Kall I, Nilsson-Helander K. Treatment of patellar tendinopathy--a systematic review of randomized controlled trials. *Knee Surg Sports Traumatol Arthrosc.* 2012;20(8):1632–46.
38. Andriolo L, Altamura SA, Reale D, Candrian C, Zaffagnini S, Filardo G. Nonsurgical treatments of patellar tendinopathy: multiple injections of platelet-rich plasma are a suitable option: a systematic review and meta-analysis. *Am J Sports Med.* 2019;47(4):1001–18.
39. Dimitrios S, Pantelis M, Kalliopi S. Comparing the effects of eccentric training with eccentric training and static stretching exercises in the treatment of patellar tendinopathy. A controlled clinical trial. *Clin Rehabil.* 2012;26(5):423–30.
40. Rudavsky A, Cook J. Physiotherapy management of patellar tendinopathy (jumper's knee). *J Physiother.* 2014;60(3):122–9.
41. Jonsson P, Alfredson H. Superior results with eccentric compared to concentric quadriceps training in patients with jumper's knee: a prospective randomised study. *Br J Sports Med.* 2005;39(11):847–50.
42. Kongsgaard M, Kovanen V, Aagaard P, Doessing S, Hansen P, Laursen AH, et al. Corticosteroid injections, eccentric decline squat training and heavy slow resistance training in patellar tendinopathy. *Scand J Med Sci Sports.* 2009;19(6):790–802.
43. de Vries A, Zwerver J, Diercks R, Tak I, van Berkel S, van Cingel R, et al. Effect of patellar strap and sports tape on pain in patellar tendinopathy: a randomized controlled trial. *Scand J Med Sci Sports.* 2016;26(10):1217–24.
44. Wang CJ, Ko JY, Chan YS, Weng LH, Hsu SL. Extracorporeal shockwave for chronic patellar tendinopathy. *Am J Sports Med.* 2007;35(6):972–8.
45. Dar G, Mei-Dan E. Immediate effect of infrapatellar strap on pain and jump height in patellar tendinopathy among young athletes. *Prosthetics Orthot Int.* 2019;43(1):21–7.
46. Zwerver J, Hartgens F, Verhagen E, van der Worp H, van den Akker-Scheek I, Diercks RL. No effect of extracorporeal shockwave therapy on patellar tendinopathy in jumping athletes during the competitive season: a randomized clinical trial. *Am J Sports Med.* 2011;39(6):1191–9.
47. Thijs KM, Zwerver J, Backx FJ, Steeneken V, Rayer S, Groenenboom P, et al. Effectiveness of shockwave treatment combined with eccentric training for patellar tendinopathy: a double-blinded randomized study. *Clin J Sport Med.* 2017;27(2):89–96.
48. Peers KH, Lysens RJ, Brys P, Bellemans J. Cross-sectional outcome analysis of athletes with chronic patellar tendinopathy treated surgically and by extracorporeal shock wave therapy. *Clin J Sport Med.* 2003;13(2):79–83.
49. Charouset C, Zaoui A, Bellaiche L, Bouyer B. Are multiple platelet-rich plasma injections useful for treatment of chronic patellar tendinopathy in athletes? A prospective study. *Am J Sports Med.* 2014;42(4):906–11.
50. Filardo G, Kon E, Della Villa S, Vincentelli F, Fornasari PM, Marcacci M. Use of platelet-rich plasma for the treatment of refractory jumper's knee. *Int Orthop.* 2010;34(6):909–15.
51. Fredberg U, Bolvig L, Pfeiffer-Jensen M, Clemmensen D, Jakobsen BW, Stengaard-Pedersen K. Ultrasonography as a tool for diagnosis, guidance of local steroid injection and, together with pressure algometry, monitoring of the treatment of athletes with chronic jumper's knee and Achilles tendinitis: a randomized, double-blind, placebo-controlled study. *Scand J Rheumatol.* 2004;33(2):94–101.
52. Bahr R, Fossan B, Loken S, Engebretsen L. Surgical treatment compared with eccentric training for patellar tendinopathy (Jumper's knee). A randomized, controlled trial. *J Bone Joint Surg Am.* 2006;88(8):1689–98.
53. Coleman BD, Khan KM, Maffulli N, Cook JL, Wark JD. Studies of surgical outcome after patellar tendinopathy: clinical significance of methodological deficiencies and guidelines for future studies. Victorian Institute of Sport Tendon Study Group. *Scand J Med Sci Sports.* 2000;10(1):2–11.

54. Ruffin MT, Kiningham RB. Anterior knee pain: the challenge of patellofemoral syndrome. *Am Fam Physician*. 1993;47(1):185–94.
55. Halabchi F, Mazaheri R, Seif-Barghi T. Patellofemoral pain syndrome and modifiable intrinsic risk factors; how to assess and address? *Asian J Sports Med*. 2013;4(2):85–100.
56. Hewett TE, Myer GD, Ford KR. Decrease in neuromuscular control about the knee with maturation in female athletes. *J Bone Joint Surg Am*. 2004;86(8):1601–8.
57. Quatman CE, Ford KR, Myer GD, Hewett TE. Maturation leads to gender differences in landing force and vertical jump performance: a longitudinal study. *Am J Sports Med*. 2006;34(5):806–13.
58. Kaplan LD, Schurhoff MR, Selesnick H, Thorpe M, Uribe JW. Magnetic resonance imaging of the knee in asymptomatic professional basketball players. *Arthroscopy*. 2005;21(5):557–61.
59. Major NM, Helms CA. MR imaging of the knee: findings in asymptomatic collegiate basketball players. *AJR Am J Roentgenol*. 2002;179(3):641–4.
60. Walczak BE, McCulloch PC, Kang RW, Zelazny A, Tedeschi F, Cole BJ. Abnormal findings on knee magnetic resonance imaging in asymptomatic NBA players. *J Knee Surg*. 2008;21(1):27–33.
61. Lankhorst NE, Bierma-Zeinstra SM, van Middelkoop M. Factors associated with patellofemoral pain syndrome: a systematic review. *Br J Sports Med*. 2013;47(4):193–206.
62. Prins MR, van der Wurff P. Females with patellofemoral pain syndrome have weak hip muscles: a systematic review. *Aust J Physiother*. 2009;55(1):9–15.
63. Fukuda TY, Rossetto FM, Magalhaes E, Bryk FF, Lucareli PR, de Almeida Aparecida Carvalho N. Short-term effects of hip abductors and lateral rotators strengthening in females with patellofemoral pain syndrome: a randomized controlled clinical trial. *J Orthop Sports Phys Ther*. 2010;40(11):736–42.
64. Barton CJ, Lack S, Malliaras P, Morrissey D. Gluteal muscle activity and patellofemoral pain syndrome: a systematic review. *Br J Sports Med*. 2013;47(4):207–14.
65. Voight ML, Wieder DL. Comparative reflex response times of vastus medialis obliquus and vastus lateralis in normal subjects and subjects with extensor mechanism dysfunction. An electromyographic study. *Am J Sports Med*. 1991;19(2):131–7.
66. Biabanimoghdam M, Motealleh A, Cowan SM. Core muscle recruitment pattern during voluntary heel raises is different between patients with patellofemoral pain and healthy individuals. *Knee*. 2016;23(3):382–6.
67. Cowan SM, Crossley KM, Bennell KL. Altered hip and trunk muscle function in individuals with patellofemoral pain. *Br J Sports Med*. 2009;43(8):584–8.
68. Hamstra-Wright KL, Earl-Boehm J, Bolgla L, Emery C, Ferber R. Individuals with patellofemoral pain have less hip flexibility than controls regardless of treatment outcome. *Clin J Sport Med*. 2017;27(2):97–103.
69. Monson J, Arendt EA. Rehabilitative protocols for select patellofemoral procedures and nonoperative management schemes. *Sports Med Arthrosc*. 2012;20(3):136–44.
70. Dye SF, Stäubli HU, Biedert RM, Vaupel GL. The mosaic of pathophysiology causing patellofemoral pain: therapeutic implications. *Oper Tech Sports Med*. 1999;7(2):46–54.
71. Ambra LF, Hinckel BB, Arendt EA, Farr J, Gomoll AH. Anatomic risk factors for focal cartilage lesions in the patella and trochlea: a case-control study. *Am J Sports Med*. 2019;47(10):2444–53.
72. Leibbrandt DC, Louw QA. The use of McConnell taping to correct abnormal biomechanics and muscle activation patterns in subjects with anterior knee pain: a systematic review. *J Phys Ther Sci*. 2015;27(7):2395–404.
73. Buchner M, Baudendistel B, Sabo D, Schmitt H. Acute traumatic primary patellar dislocation: long-term results comparing conservative and surgical treatment. *Clin J Sport Med*. 2005;15:62–6.
74. Palmu S, Kallio PE, Donell ST, Helenius I, Nietosvaara Y. Acute patellar dislocation in children and adolescents: a randomized clinical trial. *J Bone Joint Surg Am*. 2008;90(3):463–70.
75. Smith TO, Song F, Donell ST, Hing CB. Operative versus non-operative management of patellar dislocation. A meta-analysis. *Knee Surg Sports Traumatol Arthrosc*. 2011;19(6):988–98.
76. Tompkins MA, Rohr SR, Agel J, Arendt EA. Anatomic patellar instability risk factors in primary lateral patellar dislocations do not predict injury patterns: an MRI-based study. *Knee Surg Sports Traumatol Arthrosc*. 2018;26(3):677–84.
77. Camanho GL, Viegas AC, Bitar AC, Demange MK, Hernandez AJ. Conservative versus surgical treatment for repair of the medial patellofemoral ligament in acute dislocations of the patella. *Arthroscopy*. 2009;25(6):620–5.
78. Nikku R, Nietosvaara Y, Aalto K, Kallio PE. Operative treatment of primary patellar dislocation does not improve medium-term outcome: a 7-year follow-up report and risk analysis of 127 randomized patients. *Acta Orthop*. 2005;76(5):699–704.
79. Petri M, Lioudakis E, Hofmeister M, Despang FJ, Maier M, Balcarek P, et al. Operative vs conservative treatment of traumatic patellar dislocation: results of a prospective randomized controlled clinical trial. *Arch Orthop Trauma Surg*. 2013;133(2):209–13.
80. Pfirrmann CW, Zanetti M, Romero J, Hodler J. Femoral trochlear dysplasia: MR findings. *Radiology*. 2000;216(3):858–64.
81. Sillanpää PJ, Peltola E, Mattila VM, Kiuru M, Visuri T, Pihlajamäki H. Femoral avulsion of the medial patellofemoral ligament after primary traumatic patellar dislocation predicts subsequent instability in men: a mean 7-year nonoperative follow-up study. *Am J Sports Med*. 2009;37(8):1513–21.

82. Fithian DC, Paxton EW, Stone ML, Silva P, Davis DK, Elias DA, et al. Epidemiology and natural history of acute patellar dislocation. *Am J Sports Med.* 2004;32(5):1114–21.
83. Askenberger M, Ekstrom W, Finnbogason T, Janarv P-M. Occult intra-articular knee injuries in children with hemarthrosis. *Am J Sports Med.* 2014;42(7):1600–6.
84. Arendt EA, England K, Agel J, Tompkins MA. An analysis of knee anatomic imaging factors associated with primary lateral patellar dislocations. *Knee Surg Sports Traumatol Arthrosc.* 2017;25(10):3099–107.
85. Askenberger M, Janarv P-M, Finnbogason T, Arendt EA. Morphology and anatomic patellar instability risk factors in first-time traumatic lateral patellar dislocations: a prospective magnetic resonance imaging study in skeletally immature children. *Am J Sports Med.* 2017;45(1):50–8.
86. Jaquith BP, Parikh SN. Predictors of recurrent patellar instability in children and adolescents after first-time dislocation. *J Pediatr Orthop.* 2017;37(7):484–90.
87. Balcarek P, Ammon J, Frosch S, Walde TA, Schuttrumpf JP, Ferlemann KG, et al. Magnetic resonance imaging characteristics of the medial patellofemoral ligament lesion in acute lateral patellar dislocations considering trochlear dysplasia, patella Alta, and tibial tuberosity-trochlear groove distance. *Arthroscopy.* 2010;26(7):926–35.
88. Arendt EA, Askenberger M, Agel J, Tompkins MA. Risk of redislocation after primary patellar dislocation: a clinical prediction model based on magnetic resonance imaging variables. *Am J Sports Med.* 2018;46(14):3385–90.
89. DeHaven KE. Diagnosis of acute knee injuries with hemarthrosis. *Am J Sports Med.* 1980;8(1):9–14.
90. Maffulli N, Binfield PM, King JB, Good CJ. Acute haemarthrosis of the knee in athletes. A prospective study of 106 cases. *J Bone Joint Surg Br.* 1993;75(6):945–9.
91. Rodriguez-Merchan EC. Cartilage damage in the haemophilic joints: pathophysiology, diagnosis and management. *Blood Coagul Fibrinolysis.* 2012;23(3):179–83.
92. Sillanpää PJ, Mattila VM, Mäenpää H, Kiuru M, Visuri T, Pihlajamäki H. Treatment with and without initial stabilizing surgery for primary traumatic patellar dislocation. A prospective randomized study. *J Bone Joint Surg Am.* 2009;91(2):263–73.
93. Smith TO, Davies L, Chester R, Clark A, Donell ST. Clinical outcomes of rehabilitation for patients following lateral patellar dislocation: a systematic review. *Physiotherapy.* 2010;96(4):269–81.
94. Mäenpää H, Lehto MU. Patellar dislocation. The long-term results of nonoperative management in 100 patients. *Am J Sports Med.* 1997;25(2):213–7.
95. Rood A, Boons H, Ploegmakers J, van der Stappen W, Koeter S. Tape versus cast for non-operative treatment of primary patellar dislocation: a randomized controlled trial. *Arch Orthop Trauma Surg.* 2012;132(8):1199–203.
96. Mäenpää HSP, Paakkala A. A prospective, randomized trial following conservative treatment in acute primary patellar dislocation with special reference to patellar braces. *Knee Surg Sports Traumatol Arthrosc.* 2010;18(Suppl 1):119.
97. Lynch AD, Snyder-Mackler L. Post-operative management of surgeries aimed at correcting patellofemoral instability: results of an international surgeon survey. In: Zaffagnini S, Dejour D, Arendt EA, editors. *Patellofemoral pain, instability, and arthritis.* Berlin Heidelberg: Springer-Verlag; 2010. p. 287–96.
98. Arendt EA, Donell ST, Sillanpää PJ, Feller JA. The management of lateral patellar dislocation: state of the art. *J ISAKOS.* 2017;2(4):205–12.
99. Rodeo SA, Arnoczky SP, Torzilli PA, Hidaka C, Warren RF. Tendon-healing in a bone tunnel. A biomechanical and histological study in the dog. *J Bone Joint Surg Am.* 1993;75(12):1795–803.
100. Fisher B, Nyland J, Brand E, Curtin B. Medial patellofemoral ligament reconstruction for recurrent patellar dislocation: a systematic review including rehabilitation and return-to-sports efficacy. *Arthroscopy.* 2010;26(10):1384–94.
101. Ronga M, Oliva F, Longo UG, Testa V, Capasso G, Maffulli N. Isolated medial patellofemoral ligament reconstruction for recurrent patellar dislocation. *Am J Sports Med.* 2009;37(9):1735–42.
102. ISAKOS Orthopaedic Sports Medicine Committee, Almquist F, Arendt EA, Coolican M, Doral N, Erlund L. Guidelines for the evaluation, management and safe return to sport after lateral patellar dislocation or surgical stabilization in the athletic population. ISAKOS Return to Play Consensus meeting; London, England. London, England. May 2012.
103. Ahmad CS, Brown GD, Stein BS. The docking technique for medial patellofemoral ligament reconstruction: surgical technique and clinical outcome. *Am J Sports Med.* 2009;37(10):2021–7.
104. Christiansen SE, Jacobsen BW, Lund B, Lind M. Reconstruction of the medial patellofemoral ligament with gracilis tendon autograft in transverse patellar drill holes. *Arthroscopy.* 2008;24(1):82–7.
105. Gomes JE. Comparison between a static and a dynamic technique for medial patellofemoral ligament reconstruction. *Arthroscopy.* 2008;24(4):430–5.
106. Fernandez E, Sala D, Castejon M. Reconstruction of the medial patellofemoral ligament for patellar instability using a semitendinosus autograft. *Acta Orthop Belg.* 2005;71(3):303–8.
107. Mikashima Y, Kimura M, Kobayashi Y, Miyawaki M, Tomatsu T. Clinical results of isolated reconstruction of the medial patellofemoral ligament for

- recurrent dislocation and subluxation of the patella. *Acta Orthop Belg.* 2006;72(1):65–71.
108. Nomura E, Horiuchi Y, Kihara M. A mid-term follow-up of medial patellofemoral ligament reconstruction using an artificial ligament for recurrent patellar dislocation. *Knee.* 2000;7(4):211–5.
109. Schottle PB, Fucentese SF, Romero J. Clinical and radiological outcome of medial patellofemoral ligament reconstruction with a semitendinosus autograft for patella instability. *Knee Surg Sports Traumatol Arthrosc.* 2005;13(7):516–21.
110. Thaunat M, Erasmus PJ. The favourable anisometry: an original concept for medial patellofemoral ligament reconstruction. *Knee.* 2007;14(6):424–8.
111. Watanabe T, Muneta T, Ikeda H, Tateishi T, Sekiya I. Visual analog scale assessment after medial patellofemoral ligament reconstruction: with or without tibial tubercle transfer. *J Orthop Sci.* 2008;13(1):32–8.



Foot and Ankle Injuries in Basketball

36

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36.1 Introduction

Basketball was invented by James Naismith in 1891 at the International YMCA Training School in Springfield, Massachusetts [1]. The first women's basketball game was played in Springfield, Massachusetts in 1894, and the first Olympics game was played in 1976. Throughout the years, basketball has become a popular sport in the United States as well as across the world. According to the 2004 National Safety Council report, 28.9 million people play basketball every year in the United States [2]. More recently, the High School Athletics Participation Survey conducted by the National Federation of State High School Associations (NFHS) in 2018–2019 reported 399,067 female and 540,769 male basketball participants [3]. Additionally, there are 2192 National Collegiate Athletic Association (NCAA) division I, II, and III basketball teams in the United States [4]. Worldwide, there are an estimated 450 million basketball participants [5].

Basketball has increased in popularity for both men and women due to Title IX legislation promoting gender equality in sports programs [6].

The increase in basketball participation has led to an increase in the incidence of basketball-related injuries [7]. From 1988 to 1997, there has been a 12.4% increase in game-related basketball injuries [8]. Hammig et al. published a retrospective study from the National Hospital Ambulatory Medical Care Survey maintained by the Centers for Disease Control and Prevention [9]. There were 507,000 total basketball-related ambulatory care visits among 20- to 59-year-old subjects from 1997 to 2004. Eighty-eight percent of visits were men and 46% of ambulatory visits were between the ages of 20–29 years. When comparing where patients presented, 59.9% were seen in the physician's office, 33.6% in the emergency department, and 6.5% were seen in another outpatient department. Regarding the geographic region, the South represented the most cases (39.1%), followed by the Midwest (28.4%), Northeast (16.7%), and the West (15.9%).

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36.2 Epidemiology

There are specific injury patterns that are more common in basketball participants [10]. Ankle sprains are the most common injury sustained by basketball players [8, 10–12], followed by patellofemoral inflammation, and lumbar strains [10].

The most common mechanism of ankle sprains is an inversion moment with the ankle plantarflexed during single-legged landing [12, 13]. Drakos et al. retrospectively reviewed 1094 professional players (3843 injuries) within the National Basketball Association (NBA) database from 1988 to 2005 [10]. They found a total of 12,594 injuries, with 62% occurring in the lower extremity and 57.8% occurring during in-season games. The rate of lower extremity injury was 11.1 per 1000 athlete-exposures, with lateral ankle sprains being the most common lower extremity injury (3.2 per 1000 athlete-exposures, 13.2%), accounting for 8.8% of all games missed. Herzog et al. recently studied 389 NBA players with 796 ankle sprains from 2013 to 2017 [11]. They showed that the overall single-season risk of an ankle sprain was 25.8%, with 71% occurring in games and 71.2% involving contact mechanisms. Zuckerman et al. found that from 2009 to 2015, ankle sprains were the most common injury for male (17.9%) and female (16.6%) NCAA basketball players. Borowski et al. reported 1518 injuries (1.94 per 1000 athlete-exposures) in high school basketball players from 2005 to 2006 and 2006 to 2007 years. They found that ankle and foot injuries (39.7%) were the most common site of injury.

Other common foot and ankle injuries in basketball include muscle strains [14], stress fractures [15–18], Lisfranc injuries [19–21], and Achilles [22–24] injuries. Drakos et al. showed that the incidence of other common foot and ankle injuries in the NBA are as follows: triceps surae strain (2.1% injuries, 3.5% games missed), Achilles tendinopathy (1.6% injuries, 1.8% games missed), foot sprain (1.6% of injuries, 1.5% games missed), and foot fracture (0.8% of injuries, 0.6% games missed) [10]. In contrast to most studies, Rodas et al. showed that during nine seasons (2007–2015), there were 463 injuries among Spanish professional basketball players [14]. Muscle sprains (21.2%) were almost twice as common as ankle sprains (11.9%). Khan et al. found that among 75 NBA players (76 stress fracture injuries), the most common location for stress fractures was the fifth metatarsal (18.6%). Other locations included other foot bones

(14.5%), tibia (10.5%), ankle (5.3%), and navicular (3.9%). Boden classified stress fractures as low-risk or high-risk injuries [25, 26]. Low-risk stress fractures usually heal with nonoperative treatment. These injuries include the calcaneus, lateral malleolus, cuneiforms, and cuboid. High-risk injuries have an increased risk for progression, delayed union, or nonunion. These locations include the proximal fifth metatarsal, navicular, sesamoid, talus, and medial malleolus. The pattern of Lisfranc injuries differs based on the sport, with traditional medial column instability being more prevalent in basketball-related injuries [19]. The mechanism for Achilles tendon ruptures is sudden forceful plantarflexion in a fixed plantigrade position during push off [27]. Achilles tendon ruptures have been shown to be devastating injuries in basketball players, with up to 39% of players failing to return to play [22].

36.3 Risk Factors

There are extrinsic and intrinsic risk factors for basketball-related injuries. Extrinsic, basketball is a high-intensity game that requires frequent directional changes and jump-landing. Matthews et al. showed in their study of nine British female basketball players, there was an average of 652 movements per game (35 jumps, 49 sprints, 151 walks, 119 jogs/runs, 298 shuffles) [28]. This corresponded to a change in activity every 2.82 s. For male basketball players, Abdelkrim et al. studied time-motion analysis for 38 elite under-19 year old basketball players in the Tunisian Basketball Union [29]. They found an average of 1050 movements per game (44 jumps, 55 sprints, 110 jogs/runs, 276 stand/walks, and 466 shuffles). Scanlan et al. found that in-game intensity increased in elite Australian men's basketball players when compared to sub-elite players [30]. As a result, overuse injuries can result from submaximal microtrauma without adequate adaptation and recovery [31, 32]. Additionally, basketball schedules for professional players require several back-to-back games and long road trips [33]. Sleep deprivation during the season can affect cardiopulmonary

performance and cognitive performance [34–36]. Mougin et al. found that after sleep deprivation in seven subjects, heart rate and ventilation increased while oxygen consumption decreased during submaximal exercise [36]. Taheri et al. studied the effects of 24-h sleep deprivation in 18 healthy male subjects [35]. They found that reaction time was significantly delayed after sleep deprivation. However, peak anaerobic activity during cycling was not affected. Thus, repetitive loading during basketball activities and lack of recovery decreases cognitive function and increases the risk for sustaining basketball-related injuries [25, 26, 28, 29, 37].

Sport specialization in young athletes is another extrinsic risk factor for sustaining basketball-related injuries. The American Orthopaedic Sports Society Meeting (AOSSM) defines sports specialization as prepubertal athletes participating in a sport for at least 8 months/year at the exclusion of other sports [38]. Sports specialization has become increasingly popular among basketball players. In 2015, the NCAA reported that 49% of male and 55% of female division I basketball players reported specializing by the age of 12 years [39]. Professional players are even more specialized, with 85% of NBA first-round draft picks being specialized in basketball in high school [40]. The emphasis on preparing young athletes for collegiate or professional play has increased the intensity and duration of sports training [41]. Cuff et al. reported that high school athletes who played year-round sports had a 42% increased risk of overuse injury [42]. Additionally, Jayanthi et al. showed that injured athletes reported more organized sports activities (11.2 h/week) compared to uninjured athletes (9.1 h/week) [43]. Although highly specialized athletes did not have an increased risk of acute injuries, their risk of overuse injuries was significantly higher and dose-dependent [43, 44]. Adolescent athletes are particularly susceptible to overuse injuries due to the presence of open physis, which is less resistant to tensile loads than adult bone [41, 45]. Additionally, a lack of lean muscle mass and joint hypermobility can also factor into the increased risk of overuse injuries during sport

specialization. Another concern with sports specialization is that it prevents young players from developing transferable skills and may increase sport burnout [43, 46]. Field et al. found that sports specialization had a greater effect on overuse injuries in females compared to males [47]. Rugg et al. studied 237 first-round draft pick NBA players from 2008 to 2015. They found that multisport athletes sustained fewer major injuries during their career (25% vs. 43%) and had greater career longevity (94% vs. 81%) when compared to single-sport athletes.

Intrinsic factors such as gender differences have been well reported in sport-related injuries [12, 48–52]. Borowski et al. reported an injury rate of 2.08 per 1000 athlete-exposures in females high school basketball players compared to 1.83 per 1000 athlete-exposures in men [12]. Women sustained more concussions and knee injuries while men sustained more foot and ankle injuries. Zuckermann et al. showed that from 2009 to 2015, men had a higher rate of collegiate basketball injuries (2308 injuries, athlete-exposures 7.97 per 1000) compared to women (1631 injuries, 6.54 per 1000 athlete-exposures) [48]. There was a 32% higher relative risk for men sustaining ankle sprains. In contrast, women had a relative risk of 2.84 for sustaining an ACL injury compared to men. Women sustained more overuse injuries in practice (relative risk 1.66) while men sustained more contact injuries during competition (relative risk 1.46) and practices (relative risk 1.72). Detich et al. studied injury rates within the NBA and Women's National Basketball Association (WNBA) from 1996 to 2002. They found that WNBA players sustained more game-related lower extremity injuries (14.6 per 1000 athlete-exposures) compared to NBA players (11.6 per 1000 athlete-exposures).

Gender-related factors such as lean muscle mass, bone density [53, 54], proprioception [55, 56], and neuromuscular control [57–59] may play significant roles in injury rates among male and female athletes. [49] Hewett et al. studied 205 female adolescent soccer, basketball, and volleyball players utilizing three-dimensional kinematic analysis prior to their respective seasons [57]. The authors found that

the nine subjects that sustained ACL injuries had abnormal knee abduction and dynamic valgus during landing tasks. Bone stress injuries are classically associated with women due to the athletic triad [60]. Interestingly, Tenforde et al. found in their study of 239 female collegiate athletes that all female basketball players ($n = 9$) were at low risk for developing stress injuries [54]. In another study, Tenforde et al. also showed that 43% of male athletes with bone stress injuries had low bone mineral density [53]. Low vitamin D levels, thyroid levels, and testosterone levels did not correlate with male athletes developing bone stress injuries. Individuals who have intense running routines are also at risk for sustaining bone stress injuries [61].

Neuromuscular control is also an important factor for sustaining ankle sprains [59, 62]. It has been shown that up to 50% of ankle sprains occur during landing [12, 13]. Van Der Does et al. studied 75 male and female basketball, volleyball, and korfbal players with baseline kinematic screening [59]. The authors found that players with increased ankle dorsiflexion moment during repetitive jumping and unstable landing mechanics were more predisposed to sustaining ankle sprains. Additionally, individuals with chronic ankle instability have continued altered kinematics with decreased knee flexion and dynamic stability during landing testing [62, 63]. Wilkerson et al. studied 30 active adolescents with chronic ankle instability [64]. Isokinetic testing showed that subjects had less inversion strength than eversion strength during peak torque and average torque testing. Similarly, Hartsell et al. studied concentric and eccentric ankle strength in 10 subjects with chronic ankle instability compared with 14 control subjects [65]. They found that eversion and inversion strengths were weaker in subjects with chronic instability. McGuine et al. studied baseline static single-leg balance testing in 210 male and female high school basketball players without a recent ankle sprain [55]. They found that players with high postural sway during single-leg balancing had a ten times increased risk of sustaining an ankle sprain during the season. There were no differences in sustaining

ankle injuries for gender and laterality. Hoch et al. performed a matched control retrospective study on 60 participants with and without chronic ankle instability [56]. They found that individuals with chronic ankle instability had decreased anterior excursion during weight-bearing lunge testing. Although there has been some association with hip abduction weakness correlating with increased ankle sprains [66, 67], McHugh et al. showed that balance ability and hip strength did not correlate with sustaining an ankle sprain [68].

Anatomical factors such as lower extremity alignment has also been thought to contribute to basketball-related injuries. Foot supination, which is more common in the guard position, has been associated with patellar tendinopathy in basketball players [69] as well as recurrent bony stress injuries [61]. Stress fractures to the proximal fifth metatarsal have been associated with varus hindfoot alignment (Figure 36.1) [70–72]. Additionally, stress fractures of the fourth metatarsal have been associated with metatarsus adductus (Figure 36.2) [73]. Nunley et al. reported on 54 athletes with 58 s metatarsal stress fractures and found that there was an association with a short first metatarsal and Achilles contracture [74]. Gallagher et al. found that a second metatarsal length to foot length ratio $< 29\%$ was associated with sustaining ligamentous Lisfranc injuries [75]. Also, Podolnick et al. showed that midfoot cavus as measured by a positive talo-first metatarsal angle and talonavicular angle correlated with ligamentous Lisfranc injuries [76].



Fig. 36.1 Pes cavus foot with peek-a-boo heel sign



Fig. 36.2 Metatarsus adductus with stress fracture of the fifth proximal metatarsal (asterisk) seen on AP (a), oblique (b), and lateral (c) plain radiograph views

De Cesar Netto et al. studied the hindfoot and forefoot alignment of 45 active NBA players (54 feet) with various foot and ankle injuries utilizing weight-bearing cone computed tomography [77]. Most of the players presented with stress fractures of the fifth metatarsal ($n = 15$) and navicular ($n = 8$). The authors concluded that players with symptomatic foot and ankle injuries

tended to have slightly cavus foot alignment. Talar body stress fractures have been associated with excessive subtalar pronation and lateral calcaneus impingement onto the posterolateral talus [78]. Ankle sprains in basketball do not appear to correlate with overall foot alignment, but instead correlate with neuromuscular control during landing activities [69].

36.4 Evaluation and Diagnosis

The initial evaluation of a basketball-related foot and ankle injury should begin with a thorough history. The mechanism and position of the foot at the time of injury can help to formulate a differential diagnosis. Lateral ankle sprains tend to occur with the foot in a plantarflexed and inverted position while a syndesmotic injury occurs with the foot in a dorsiflexed and everted position. A physical examination should include assessing the overall limb alignment. As previously mentioned, genu valgum or cavovarus foot alignment can predispose athletes to certain injury patterns. Careful evaluation of shoe wear to assess midsole flexibility, collar height, and outsole traction can provide useful information for the clinician. If possible, assessing the ability to load the affected limb during ambulation can help determine the severity of injury. Skin inspection for focal swelling and ecchymosis can help correlate the location of injury. Palpation should also be used to localize involved anatomic structures. Special tests such as ankle drawer tests or syndesmosis testing may be difficult to perform in the acute stage of injury and should be delayed for 5 days. Finally, a thorough neurovascular examination of the lower extremity should be performed.

Plain radiographs should be performed if the athlete is unable to bear weight or if there is focal bony tenderness, as indicated by the Ottawa Ankle Rules [79]. Magnetic resonance imaging (MRI) can be performed in patients with chronic ankle pain despite adequate conservative treatment to rule out intra-articular pathology. Additionally, MRI may be useful for detecting subtle Lisfranc, syndesmosis injuries, and bony stress injuries. Weight-bearing computed tomography (CT) scan should be used to characterize Lisfranc injuries and aid in surgical planning.

36.5 Injury Prevention

Basketball injury prevention is multifaceted and involves a multidisciplinary approach. Improving rest, proprioception, neuromuscular control, flexibility, and external support can reduce basketball-

related injuries [80]. Given the recent concerns over the physical demands of the NBA schedule, the league reduced back-to-back games from 19.3 to 17.8 per team [33]. Additionally, long-distance back-to-back games were reduced from 111 to 85 and four games in 5 days were reduced from 2.3 to 0.9 per team.

Proprioception and neuromuscular training can reduce basketball-related injuries. Emery et al. performed a randomized prospective study on 920 high school basketball players in Canada undergoing a home-based balance training program using a wobble board [81]. At 1-year follow-up, there was a 17% relative risk reduction in all lower extremity injuries and 29% relative risk reduction in ankle sprain injuries. Similarly, McGuine et al. performed a randomized control trial on 765 high school soccer and basketball players utilizing a five-phase balance training program [82]. Exercises included single-leg stance, single-leg squat, single-leg stance with a rotating board, and functional dribbling. Athletes who underwent balance training sustained less ankle sprains (6.1%, 1.13 per 1000 athlete-exposures) compared to the control group (9.9%, 1.87 per 1000 athlete-exposures). More recently, the FIFA 11+ warm-up program was developed. Initially developed for female soccer players, this program utilizes core stability, balance, dynamic stabilization, and eccentric hamstring strength. It has been shown to reduce lower extremity injuries by 32%, overuse injuries by 53%, and severe injuries by 45% [83]. Longo et al. studied the effectiveness of the FIFA 11+ program for injury prevention in European male elite basketball players [84]. In their cohort of 121 players during the 9-month season, there was a reduction in overall injuries (0.95 per 1000 athlete-exposures vs. 2.18 per 1000 athlete-exposures) and lower extremity injuries (0.68 per 1000 athlete-exposures vs. 1.4 per 1000 athlete-exposures). However, there was no difference in game injuries, knee injuries, or ankle injuries. The effectiveness of the FIFA 11+ program may be limited by the lack of jump-landing training that is required for basketball. Aerts et al. performed a randomized control trial on 116 amateur players (aged 15–41 years old) who underwent

jump-landing neuromuscular training [85]. The experimental cohort improved their dynamic hip and knee flexion as well as knee valgus during take-off. Gastrocnemius stretching has not been shown to effectively reduce the incidence of foot and ankle injuries [86].

Shoe wear modifications have been implemented to potentially reduce the incidence of foot and ankle injuries in athletes [87, 88]. These modifications include cushioning, midsole hardness, forefoot stiffness, outsole traction, collar height, and shoe mass [89]. Adequate outsole traction and forefoot stiffness are the most important factors for improved athletic performance in basketball [90]. Biomechanically, three quarter top basketball shoes have been associated with 29.4% greater inversion moment resistance when compared to low-top basketball shoes [91]. Additionally, ankle tape has been shown to improve inversion moment resistance by 7.8% [88]. The clinical benefits of high top basketball shoes has not been shown in the literature. Barrett et al. randomized 622 college intramural basketball players with chronic ankle instability into high-top basketball shoes, high-top basketball shoes with an inflatable chamber, and low-top basketball shoes [91]. After 2-month follow-up at the end of the intramural season, there were 15 ankle sprains with no difference in ankle sprains among the three groups. A prospective study by Curtis et al. looked at NCAA division I–III basketball players who sustained lateral ankle sprains during the 2005–2006 basketball season [92]. They found no difference in the rate of ankle sprains in individuals wearing cushioned column or non-cushioned column shoes. Leong et al. compared contact pressures during basketball-specific activities for soft and hard midsole shoes [93]. Although players preferred soft midsole shoes, there was greater contact pressure deviations for soft midsole shoes. Most recently, Helton et al. found that moderate torsional stiffness shoes reduced lower extremity injuries by 49% and overuse injuries by 52% compared to shoes with minimal torsional stiffness [94].

External ankle support can also augment shoe support in basketball players. The goals of external ankle support are to improve dynamic stabil-

ity without impeding athletic performance. Older studies have shown that ankle taping reduces the incidence of ankle sprains and improves proprioception [95, 96]. Sitler et al. studied 1601 United States Military Academy cadets who participated in the 1990–1991 intramural basketball season [97]. They found nearly a threefold reduction in the ankle injury rate for subjects who wore a semi-rigid ankle brace (1.6 per 1000 athlete-exposures vs. 5.2 per 1000 athlete exposures). Eils et al. studied ankle stability conferred by ten different soft, semi-rigid, and rigid ankle braces [98]. In their cohort of 24 subjects with chronic ankle instability, the authors found that semi-rigid ankle braces resisted rapidly induced inversion moment by 31–49%, depending on the brace used. McGuine et al. performed a randomized control trial on 1460 high school male and female basketball players during the 2009–2010 season [99]. Players who were randomized to the lace-up ankle brace group had a 68% reduction in the incidence of ankle sprains when compared to the control group (0.47 per 1000 athlete-exposures vs. 1.41 per 1000 athlete-exposures). The National Athletic Trainers' Association (NATA) 2013 guidelines for ankle sprain management states that ankle bracing and taping should be utilized for recurrent ankle instability rather than prophylactically [100].

36.6 Treatment and Return to Play

Guidelines for the treatment of ankle sprains have been published by NATA in 2013 [100]. The mainstays for ankle sprain treatment include rest, ice, compression, and elevation (R.I.C.E.). Grade I and II ankle sprains should undergo functional rehabilitation rather than immobilization [101, 102]. Grade III ankle sprains should be immobilized for at least 10 days in a boot or below-knee cast and then functional training once the pain and edema improve. Throughout rehabilitation, balance training and neuromuscular training should be utilized. Return-to-play considerations should include subjective patient perception and functional performance testing

during sport-specific tasks. The injured limb's functional performance should measure at least 80% of the uninjured limb prior to return to play. Overall, ankle sprains have a more promising prognosis for return to play. Nelson et al. and Herzog et al. showed that 56% of NBA players sustaining an ankle sprain did not miss any games, with the median games missed being two games [11].

Athletes with Achilles tendinopathy are initially treated conservatively. Treatment modalities include rest, ice, NSAIDs, stretching, and heel lifts. Athletes with flatfoot deformities can use medial longitudinal arch support. Night splints can also be utilized to improve equinus. Achilles tendon corticosteroid injections should be avoided to prevent the risk of an Achilles rupture. In contrast, Athletes with a high-grade partial or complete Achilles rupture should undergo surgical repair. Surgical options include minimally invasive [103, 104], percutaneous [105], and open approaches. Postoperatively, athletes can undergo traditional non-weight-bearing for 4–6 weeks versus accelerated rehabilitation protocol with early weight-bearing [106, 107].

Return to play after orthopedic surgeries in basketball players varies by the nature of the injury and level of play. Achilles injuries have been shown to be one of the most debilitating injuries for basketball players [22, 24, 27, 91]. Minhas et al. studied the effects of orthopedic surgeries on return to play for 348 NBA players [24]. Players that sustained an Achilles tear tended to be the oldest (28.4 years old) and the most experienced (6.5 years) in the injury cohort. Achilles repairs had a marked lower return-to-play rate (70.8%), lower player efficiency rating (−2.46), and less games played (17.1 games) compared to other injuries. The mean time to return to play was 10.5 months [27]. At the 1 and 2 years postoperative period, NBA players performed worse (year 1: 74.8% and year 2: 77.7%) than Major League Baseball (MLB) players (87.8% and 112.4%), respectively [23].

Lower extremities such as ankle sprains and Lisfranc injuries have a better return-to-play prognosis. Herzog et al. studied 389 NBA players with 796 ankle sprains (lateral, medial, and high

over a 4-year period. They found that the median NBA games missed were lateral (2 games), medial (3 games), and high (5 games). Additionally, median days for return to play were lateral (8 days), medial (8 days), and high (24 days). Rodas et al. reported the mean return-to-play time for 59 professional European basketball players was 84 days for ankle sprains [14]. Nelson et al. reported that among high school athletes in the United States, athletes missed less than 7 days of activity (51.7%), 7–21 days (33.9%), and more than 22 days (10.5%) [108]. McGuine et al. specifically studied 23 high school basketball players with ankle sprains [55]. They found that less than 7 days was missed in 56.5% of players, followed by 39.1% and 4.3% of players missing 7–21 and greater than 21 days of activity, respectively.

Porter et al. showed that return to play is determined by the specific Lisfranc injury pattern [19]. Traditional medial column instability, more commonly seen in basketball and football injuries, had a mean return to play of 7.5 months. This contrasts with more severe middle column instability pattern, more common in wakeboarding injuries (return to play 8.4 months). MacMahon et al. retrospectively reviewed 38 patients who underwent primary arthrodesis for a Lisfranc injury [109]. At a mean follow-up of 5.2 years, patients had similar postoperative levels with high impact activity (44.8%), with 97% patient satisfaction despite worse postoperative outcome scores. Mora et al. studied 31 patients (under 56 years old) who underwent open reduction and internal fixation for a Lisfranc injury with screw or plate fixation [110]. At a mean time of 2.9 years follow-up, 94% of the cohort was able to return to some level of recreational sport, with 66% returning to their previous sporting level. Osbahr et al. studied 15 National Football League (NFL) players who sustained midfoot sprains in a 15-year period [111]. All grade 1 sprains underwent nonoperative treatment with a walking boot. Conversely, grade 2 midfoot sprains with latent diastasis underwent either nonoperative or operative treatment. They found that grade 1, 2, and 3 sprains had mean

return-to-play times of 1.9 days, 26.1 days, and 73 days, respectively. Nonoperative subjects had a 92% successful return-to-play rate.

Khan et al. reported on 76 lower extremity bony stress injuries in 75 NBA players from 2005 to 2015 [15]. The injury cohort missed a mean of 25.1 games and 38% of injuries were managed surgically. All proximal fifth metatarsal injuries ($n = 14$) were treated surgically. Overall, 30% of players with stress injuries did not return to play. Forty-three percent of fifth metatarsal stress fractures failed to return to play. Of note, 19.7% of patients sustained subsequent stress injuries. O'Malley retrospectively studied 10 NBA players with proximal fifth metatarsal fractures [18]. Players were treated with intramedullary screw fixation and bone marrow aspirate concentrate (BMAC). They found a radiographic healing rate of 7.5 weeks and mean return to play of 9.8 weeks. Three players sustained refractures and had a rate of metatarsus adductus.

36.7 Conclusion

Basketball-related injuries are common due to the multidirectional nature of the sports. Frequent jump-landing and vigorous play predispose basketball players to injury. Prevention of basketball injuries is multifaceted and includes neuromuscular training, adequate shoe wear, ankle support, and limiting sport specialization. Nonoperative treatment of ankle sprains yields successful outcomes but can lead to neuromuscular deficits if proper functional training is not utilized. The return-to-play outcomes after Achilles tendon ruptures are not as promising, with a significant number of basketball players failing to return to play.

References

1. Cantwell JD. The physician who invented basketball. *Am J Cardiol.* 2004;93(8):1075–7.
2. National Safety Council. National safety council: injury facts, 2004 Edition. Edition. Itasca, IL: National Safety Council; 2004.

3. Participation in high school sports registers first decline in 30 years. September 2019. <https://www.nfhs.org>. Accessed October 6, 2019.
4. National Collegiate Athletic Association. Student-athlete participation 1981–82–2016–17: NCAA sports sponsorship and participation rates report. 2018. Accessed October 7, 2019.
5. FIBA. <https://www.fiba.basketball>. Accessed October 7, 2019.
6. Carpenter LAR. Title IX. Champaign, IL: Human Kinetics.; 2005 Human Kinetics; 2005.
7. Teramoto M, Cross CL, Cushman DM, Maak TG, Petron DJ, Willick SE. Game injuries in relation to game schedules in the National Basketball Association. *J Sci Med Sport.* 2017;20(3):230–5.
8. Starkey C. Injuries and illnesses in the national basketball association: a 10-year perspective. *J Athl Train.* 2000;35(2):161–7.
9. Hammig BJ, Yang H, Bensema B. Epidemiology of basketball injuries among adults presenting to ambulatory care settings in the United States. *Clin J Sport Med.* 2007;17(6):446–51.
10. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the national basketball association: a 17-year overview. *Sports Health.* 2010;2(4):284–90.
11. Herzog MM, Mack CD, Dreyer NA, et al. Ankle sprains in the National Basketball Association, 2013–2014 through 2016–2017. *Am J Sports Med.* 2019;47(11):2651–8.
12. Borowski LA, Yard EE, Fields SK, Comstock RD. The epidemiology of US high school basketball injuries, 2005–2007. *Am J Sports Med.* 2008;36(12):2328–35.
13. McKay GD, Goldie PA, Payne WR, Oakes BW. Ankle injuries in basketball: injury rate and risk factors. *Br J Sports Med.* 2001;35(2):103–8.
14. Rodas G, Bove T, Caparros T, et al. Ankle sprain versus muscle strain injury in professional Men's basketball: a 9-year prospective follow-up study. *Orthop J Sports Med.* 2019;7(6):2325967119849035.
15. Khan M, Madden K, Burrus MT, et al. Epidemiology and impact on performance of lower extremity stress injuries in professional basketball players. *Sports Health.* 2018;10(2):169–74.
16. Finestone A, Milgrom C, Wolf O, Petrov K, Evans R, Moran D. Epidemiology of metatarsal stress fractures versus tibial and femoral stress fractures during elite training. *Foot Ankle Int.* 2011;32(1):16–20.
17. Matheson GO, Clement DB, McKenzie DC, Taunton JE, Lloyd-Smith DR, MacIntyre JG. Stress fractures in athletes. A study of 320 cases. *Am J Sports Med.* 1987;15(1):46–58.
18. O'Malley M, DeSandis B, Allen A, Levitsky M, O'Malley Q, Williams R. Operative treatment of fifth metatarsal Jones fractures (zones II and III) in the NBA. *Foot Ankle Int.* 2016;37(5):488–500.
19. Porter DA, Barnes AF, Rund A, Walrod MT. Injury pattern in ligamentous Lisfranc injuries in competitive athletes. *Foot Ankle Int.* 2019;40(2):185–94.

20. DeOrio M, Erickson M, Uselli FG, Easley M. Lisfranc injuries in sport. *Foot Ankle Clin.* 2009;14(2):169–86.
21. Nunley JA, Vertullo CJ. Classification, investigation, and management of midfoot sprains: Lisfranc injuries in the athlete. *Am J Sports Med.* 2002;30(6):871–8.
22. Amin NH, Old AB, Tabb LP, Garg R, Toossi N, Cerynik DL. Performance outcomes after repair of complete Achilles tendon ruptures in national basketball association players. *Am J Sports Med.* 2013;41(8):1864–8.
23. Trofa DP, Miller JC, Jang ES, Woode DR, Greisberg JK, Vosseller JT. Professional athletes' return to play and performance after operative repair of an Achilles tendon rupture. *Am J Sports Med.* 2017;45(12):2864–71.
24. Minhas SV, Kester BS, Larkin KE, Hsu WK. The effect of an orthopaedic surgical procedure in the National Basketball Association. *Am J Sports Med.* 2016;44(4):1056–61.
25. Boden BP, Osbahr DC, Jimenez C. Low-risk stress fractures. *Am J Sports Med.* 2001;29(1):100–11.
26. Boden BP, Osbahr DC. High-risk stress fractures: evaluation and treatment. *J Am Acad Orthop Surg.* 2000;8(6):344–53.
27. Lemme NJ, Li NY, Kleiner JE, Tan S, DeFroda SF, Owens BD. Epidemiology and video analysis of Achilles tendon ruptures in the National Basketball Association. *Am J Sports Med.* 2019;47(10):2360–6.
28. Matthew D, Delextrat A. Heart rate, blood lactate concentration, and time-motion analysis of female basketball players during competition. *J Sports Sci.* 2009;27(8):813–21.
29. Ben Abdelkrim N, El Fazaa S, El Ati J. Time-motion analysis and physiological data of elite under-19-year-old basketball players during competition. *Br J Sports Med.* 2007;41(2):69–75. discussion 75
30. Scanlan A, Dascombe B, Reaburn P. A comparison of the activity demands of elite and sub-elite Australian men's basketball competition. *J Sports Sci.* 2011;29(11):1153–60.
31. Luke A, Lazaro RM, Bergeron MF, et al. Sports-related injuries in youth athletes: is overscheduling a risk factor? *Clin J Sport Med.* 2011;21(4):307–14.
32. Brenner JS. Overuse injuries, overtraining, and burnout in child and adolescent athletes. *Pediatrics.* 2007;119(6):1242–5.
33. J. Z. NBA schedule more player friendly with fewer back-to-back games. *USA TODAY Sports.* 2015;. Accessed October 12, 2019
34. Leger D, Metlaine A, Choudat D. Insomnia and sleep disruption: relevance for athletic performance. *Clin Sports Med.* 2005;24(2):269–85. viii
35. Taheri M, Arabameri E. The effect of sleep deprivation on choice reaction time and anaerobic power of college student athletes. *Asian J Sports Med.* 2012;3(1):15–20.
36. Mougín F, Simon-Rigaud ML, Davenne D, et al. Effects of sleep disturbances on subsequent physiological performance. *Eur J Appl Physiol Occup Physiol.* 1991;63(2):77–82.
37. McInnes SE, Carlson JS, Jones CJ, McKenna MJ. The physiological load imposed on basketball players during competition. *J Sports Sci.* 1995;13(5):387–97.
38. LaPrade RF, Agel J, Baker J, et al. AOSSM early sport specialization consensus statement. *Orthop J Sports Med.* 2016;4(4):2325967116644241.
39. Results from the 2015 GOALS Study of the Student-Athlete Experience (n.d.). http://www.ncaaorg/sites/default/files/GOALS_convention_slidebank_jan2016_publicpdf 2015. Accessed October 12, 2019.
40. Rugg C, Kadoor A, Feeley BT, Pandya NK. The effects of playing multiple high school sports on National Basketball Association Players' propensity for injury and athletic performance. *Am J Sports Med.* 2018;46(2):402–8.
41. DiFiori JP, Benjamin HJ, Brenner JS, et al. Overuse injuries and burnout in youth sports: a position statement from the American Medical Society for Sports Medicine. *Br J Sports Med.* 2014;48(4):287–8.
42. Cuff S, Loud K, O'Riordan MA. Overuse injuries in high school athletes. *Clin Pediatr.* 2010;49(8):731–6.
43. Jayanthi NA, LaBella CR, Fischer D, Pasulka J, Dugas LR. Sports-specialized intensive training and the risk of injury in young athletes: a clinical case-control study. *Am J Sports Med.* 2015;43(4):794–801.
44. Post EG, Trigsted SM, Riekema JW, et al. The association of sport specialization and training volume with injury history in youth athletes. *Am J Sports Med.* 2017;45(6):1405–12.
45. Bright RW, Burstein AH, Elmore SM. Epiphyseal-plate cartilage. A biomechanical and histological analysis of failure modes. *J Bone Joint Surg Am.* 1974;56(4):688–703.
46. Fabricant PD, Lakomkin N, Sugimoto D, Tepolt FA, Straccolini A, Kocher MS. Youth sports specialization and musculoskeletal injury: a systematic review of the literature. *Phys Sportsmed.* 2016;44(3):257–62.
47. Field AE, Tepolt FA, Yang DS, Kocher MS. Injury risk associated with sports specialization and activity volume in youth. *Orthop J Sports Med.* 2019;7(9):2325967119870124.
48. Zuckerman SL, Wegner AM, Roos KG, Djoko A, Dompier TP, Kerr ZY. Injuries sustained in National Collegiate Athletic Association men's and women's basketball, 2009/2010-2014/2015. *Br J Sports Med.* 2018;52(4):261–8.
49. Carter CW, Ireland ML, Johnson AE, et al. Sex-based differences in common sports injuries. *J Am Acad Orthop Surg.* 2018;26(13):447–54.
50. Deitch JR, Starkey C, Walters SL, Moseley JB. Injury risk in professional basketball players: a comparison of Women's National Basketball Association and National Basketball Association athletes. *Am J Sports Med.* 2006;34(7):1077–83.

51. Zelisko JA, Noble HB, Porter M. A comparison of men's and women's professional basketball injuries. *Am J Sports Med.* 1982;10(5):297–9.
52. Arendt E, Dick R. Knee injury patterns among men and women in collegiate basketball and soccer. NCAA data and review of literature. *Am J Sports Med.* 1995;23(6):694–701.
53. Tenforde AS, Parziale AL, Popp KL, Ackerman KE. Low bone mineral density in male athletes is associated with bone stress injuries at anatomic sites with greater trabecular composition. *Am J Sports Med.* 2018;46(1):30–6.
54. Tenforde AS, Carlson JL, Chang A, et al. Association of the female athlete triad risk assessment stratification to the development of bone stress injuries in collegiate athletes. *Am J Sports Med.* 2017;45(2):302–10.
55. McGuine TA, Greene JJ, Best T, Levenson G. Balance as a predictor of ankle injuries in high school basketball players. *Clin J Sport Med.* 2000;10(4):239–44.
56. Hoch MC, Staton GS, Medina McKeon JM, Mattacola CG, McKeon PO. Dorsiflexion and dynamic postural control deficits are present in those with chronic ankle instability. *J Sci Med Sport.* 2012;15(6):574–9.
57. Hewett TE, Myer GD, Ford KR, et al. Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. *Am J Sports Med.* 2005;33(4):492–501.
58. Brown C, Bowser B, Simpson KJ. Movement variability during single leg jump landings in individuals with and without chronic ankle instability. *Clin Biomech (Bristol, Avon).* 2012;27(1):52–63.
59. van der Does HT, Brink MS, Benjaminse A, Visscher C, Lemmink KA. Jump landing characteristics predict lower extremity injuries in indoor team sports. *Int J Sports Med.* 2016;37(3):251–6.
60. De Souza MJ, Nattiv A, Joy E, et al. 2014 Female Athlete Triad Coalition consensus statement on treatment and return to play of the female athlete triad: 1st International Conference held in San Francisco, CA, May 2012, and 2nd International Conference held in Indianapolis, IN, May 2013. *Clin J Sport Med.* 2014;24(2):96–119.
61. Korpelainen R, Orava S, Karpakka J, Siira P, Hulkko A. Risk factors for recurrent stress fractures in athletes. *Am J Sports Med.* 2001;29(3):304–10.
62. Gribble PA, Robinson RH. Alterations in knee kinematics and dynamic stability associated with chronic ankle instability. *J Athl Train.* 2009;44(4):350–5.
63. Terada M, Pietrosimone B, Gribble PA. Individuals with chronic ankle instability exhibit altered landing knee kinematics: potential link with the mechanism of loading for the anterior cruciate ligament. *Clin Biomech (Bristol, Avon).* 2014;29(10):1125–30.
64. Wilkerson GB, Pinerola JJ, Caturano RW. Invertor vs. evertor peak torque and power deficiencies associated with lateral ankle ligament injury. *J Orthop Sports Phys Ther.* 1997;26(2):78–86.
65. Hartsell HD, Spaulding SJ. Eccentric/concentric ratios at selected velocities for the invertor and evertor muscles of the chronically unstable ankle. *Br J Sports Med.* 1999;33(4):255–8.
66. Nicholas JA, Strizak AM, Veras G. A study of thigh muscle weakness in different pathological states of the lower extremity. *Am J Sports Med.* 1976;4(6):241–8.
67. Beckman SM, Buchanan TS. Ankle inversion injury and hypermobility: effect on hip and ankle muscle electromyography onset latency. *Arch Phys Med Rehabil.* 1995;76(12):1138–43.
68. McHugh MP, Tyler TF, Tetro DT, Mullaney MJ, Nicholas SJ. Risk factors for noncontact ankle sprains in high school athletes: the role of hip strength and balance ability. *Am J Sports Med.* 2006;34(3):464–70.
69. Lopezosa-Reca E, Gijon-Nogueron G, Morales-Asencio JM, Cervera-Marin JA, Luque-Suarez A. Is there any association between foot posture and lower limb-related injuries in professional male basketball players? A cross-sectional study. *Clin J Sport Med.* 2017;30:1.
70. Carreira DS, Sandilands SM. Radiographic factors and effect of fifth metatarsal Jones and diaphyseal stress fractures on participation in the NFL. *Foot Ankle Int.* 2013;34(4):518–22.
71. DeLee JC, Evans JP, Julian J. Stress fracture of the fifth metatarsal. *Am J Sports Med.* 1983;11(5):349–53.
72. Raikin SM, Slenker N, Ratigan B. The association of a varus hindfoot and fracture of the fifth metatarsal metaphyseal-diaphyseal junction: the Jones fracture. *Am J Sports Med.* 2008;36(7):1367–72.
73. Rongstad KM, Tuetting J, Rongstad M, Garrels K, Meis R. Fourth metatarsal base stress fractures in athletes: a case series. *Foot Ankle Int.* 2013;34(7):962–8.
74. Chuckpaiwong B, Cook C, Pietrobon R, Nunley JA. Second metatarsal stress fracture in sport: comparative risk factors between proximal and non-proximal locations. *Br J Sports Med.* 2007;41(8):510–4.
75. Gallagher SM, Rodriguez NA, Andersen CR, Granberry WM, Panchbhavi VK. Anatomic predisposition to ligamentous Lisfranc injury: a matched case-control study. *J Bone Joint Surg Am.* 2013;95(22):2043–7.
76. Podolnick JD, Donovan DS, DeBellis N, Pino A. Is pes Cavus alignment associated with Lisfranc injuries of the foot? *Clin Orthop Relat Res.* 2017;475(5):1463–9.
77. de Cesar NC, Bernasconi A, Roberts L, et al. Foot alignment in symptomatic National Basketball Association Players using weightbearing cone beam computed tomography. *Orthop J Sports Med.* 2019;7(2):2325967119826081.

78. Bradshaw C, Khan K, Brukner P. Stress fracture of the body of the talus in athletes demonstrated with computer tomography. *Clin J Sport Med.* 1996;6(1):48–51.
79. Bachmann LM, Kolb E, Koller MT, Steurer J, ter Riet G. Accuracy of Ottawa ankle rules to exclude fractures of the ankle and mid-foot: systematic review. *BMJ (Clin Res ed).* 2003;326(7386):417.
80. Taylor JB, Ford KR, Nguyen AD, Terry LN, Hegedus EJ. Prevention of lower extremity injuries in basketball: a systematic review and meta-analysis. *Sports Health.* 2015;7(5):392–8.
81. Emery CA, Rose MS, McAllister JR, Meeuwisse WH. A prevention strategy to reduce the incidence of injury in high school basketball: a cluster randomized controlled trial. *Clin J Sport Med.* 2007;17(1):17–24.
82. McGuine TA, Keene JS. The effect of a balance training program on the risk of ankle sprains in high school athletes. *Am J Sports Med.* 2006;34(7):1103–11.
83. Soligard T, Myklebust G, Steffen K, et al. Comprehensive warm-up programme to prevent injuries in young female footballers: cluster randomised controlled trial. *BMJ (Clin Res ed).* 2008;337:a2469.
84. Longo UG, Loppini M, Berton A, Marinozzi A, Maffulli N, Denaro V. The FIFA 11+ program is effective in preventing injuries in elite male basketball players: a cluster randomized controlled trial. *Am J Sports Med.* 2012;40(5):996–1005.
85. Aerts I, Cumps E, Verhagen E, Wuyts B, Van De Gucht S, Meeusen R. The effect of a 3-month prevention program on the jump-landing technique in basketball: a randomized controlled trial. *J Sport Rehabil.* 2015;24(1):21–30.
86. Knapik DM, LaTulip S, Salata MJ, Voos JE, Liu RW. Impact of routine gastrocnemius stretching on ankle dorsiflexion flexibility and injury rates in high school basketball athletes. *Orthop J Sports Med.* 2019;7(4):2325967119836774.
87. Ottaviani RA, Ashton-Miller JA, Kothari SU, Wojtys EM. Basketball shoe height and the maximal muscular resistance to applied ankle inversion and eversion moments. *Am J Sports Med.* 1995;23(4):418–23.
88. Ashton-Miller JA, Ottaviani RA, Hutchinson C, Wojtys EM. What best protects the inverted weight-bearing ankle against further inversion? Evertor muscle strength compares favorably with shoe height, athletic tape, and three orthoses. *Am J Sports Med.* 1996;24(6):800–9.
89. Lam WK, Kan WH, Chia JS, Kong PW. Effect of shoe modifications on biomechanical changes in basketball: a systematic review. *Sports Biomech.* 2019:1–27.
90. Worobets J, Wannop JW. Influence of basketball shoe mass, outsole traction, and forefoot bending stiffness on three athletic movements. *Sports Biomech.* 2015;14(3):351–60.
91. Barrett JR, Tanji JL, Drake C, Fuller D, Kawasaki RI, Fenton RM. High- versus low-top shoes for the prevention of ankle sprains in basketball players. A prospective randomized study. *Am J Sports Med.* 1993;21(4):582–5.
92. Curtis CK, Laudner KG, McLoda TA, McCaw ST. The role of shoe design in ankle sprain rates among collegiate basketball players. *J Athl Train.* 2008;43(3):230–3.
93. Leong HF, Lam WK, Ng WX, Kong PW. Center of pressure and perceived stability in basketball shoes with soft and hard midsoles. *J Appl Biomech.* 2018;34(4):284–90.
94. Helton GL, Cameron KL, Zifchock RA, et al. Association between running shoe characteristics and lower extremity injuries in United States military academy cadets. *Am J Sports Med.* 2019;47(12):2853–62.
95. Garrick JG, Requa RK. Role of external support in the prevention of ankle sprains. *Med Sci Sports.* 1973;5(3):200–3.
96. Miralles I, Monterde S, Montull S, Salvat I, Fernandez-Ballart J, Beceiro J. Ankle taping can improve proprioception in healthy volunteers. *Foot Ankle Int.* 2010;31(12):1099–106.
97. Sitler M, Ryan J, Wheeler B, et al. The efficacy of a semirigid ankle stabilizer to reduce acute ankle injuries in basketball. A randomized clinical study at West Point. *Am J Sports Med.* 1994;22(4):454–61.
98. Eils E, Demming C, Kollmeier G, Thorwesten L, Volker K, Rosenbaum D. Comprehensive testing of 10 different ankle braces. Evaluation of passive and rapidly induced stability in subjects with chronic ankle instability. *Clin Biomech (Bristol, Avon).* 2002;17(7):526–35.
99. McGuine TA, Brooks A, Hetzel S. The effect of lace-up ankle braces on injury rates in high school basketball players. *Am J Sports Med.* 2011;39(9):1840–8.
100. Kaminski TW, Hertel J, Amendola N, et al. National Athletic Trainers' association position statement: conservative management and prevention of ankle sprains in athletes. *J Athl Train.* 2013;48(4):528–45.
101. Kerkhoffs GM, Rowe BH, Assendelft WJ, Kelly K, Struijs PA, van Dijk CN. Immobilisation and functional treatment for acute lateral ankle ligament injuries in adults. *Cochrane Database Syst Rev.* 2002;(3):Cd003762.
102. Beynon BD, Renstrom PA, Haugh L, Uh BS, Barker H. A prospective, randomized clinical investigation of the treatment of first-time ankle sprains. *Am J Sports Med.* 2006;34(9):1401–12.
103. Patel MS, Kadakia AR. Minimally invasive treatments of acute Achilles tendon ruptures. *Foot Ankle Clin.* 2019;24(3):399–424.
104. Rungprai C, Phisitkul P. Outcomes and complications following endoscopically assisted percutaneous Achilles tendon repair. *Arthroscopy.* 2018;34(4):1262–9.
105. Hsu AR, Jones CP, Cohen BE, Davis WH, Ellington JK, Anderson RB. Clinical outcomes and complica-

- tions of percutaneous Achilles repair system versus open technique for acute Achilles tendon ruptures. *Foot Ankle Int.* 2015;36(11):1279–86.
106. Brumann M, Baumbach SF, Mutschler W, Polzer H. Accelerated rehabilitation following Achilles tendon repair after acute rupture – development of an evidence-based treatment protocol. *Injury.* 2014;45(11):1782–90.
107. Willits K, Amendola A, Bryant D, et al. Operative versus nonoperative treatment of acute Achilles tendon ruptures: a multicenter randomized trial using accelerated functional rehabilitation. *J Bone Joint Surg Am.* 2010;92(17):2767–75.
108. Nelson AJ, Collins CL, Yard EE, Fields SK, Comstock RD. Ankle injuries among United States high school sports athletes, 2005–2006. *J Athl Train.* 2007;42(3):381–7.
109. MacMahon A, Kim P, Levine DS, et al. Return to sports and physical activities after primary partial arthrodesis for Lisfranc injuries in young patients. *Foot Ankle Int.* 2016;37(4):355–62.
110. Mora AD, Kao M, Alfred T, Shein G, Ling J, Lunz D. Return to sports and physical activities after open reduction and internal fixation of Lisfranc injuries in recreational athletes. *Foot Ankle Int.* 2018;39(7):801–7.
111. Osbahr DC, O'Loughlin PF, Drakos MC, Barnes RP, Kennedy JG, Warren RF. Midfoot sprains in the National Football League. *Am J Orthop (Belle Mead, NJ).* 2014;43(12):557–61.



Management of Chronic Ankle Instability in the Basketball Player

37

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37.1 Introduction

The basketball athlete faces repetitive rapid acceleration and deceleration, cutting, jumping, and pivoting. Basketball-related injuries are the most common type of sports-related injury in patients under the age of 25 years and second in those aged 25–40 [1]. In 2017, the National Electronic Injury Surveillance System (NEISS) recorded more than 1.46 million individuals who had a sports-related injury treated in an American emergency department [1]. Of those, basketball represented over 34% of total injuries with 500,085 suffering injuries with the most common injury was related to the ankle. In a 17-year study of all injuries in the National Basketball Association, the foot and ankle were most commonly involved with 1850 injuries (14.7% of all injuries) [2]. There were 1123 game-related ankle injuries (17.9%). The foot contributed to 962 more injuries (7.6% of all injuries).

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37.2 Pathomechanism

Lateral ankle sprains generally result from axially loading a plantar-flexed ankle during inversion. When the ankle is in plantar-flexion, the ankle's stability relies on the robust, but often injured soft tissue structures, which, without osseous stability, are more susceptible to injury. The anterior talofibular ligament (ATFL), which is tight in plantar-flexion, is the most commonly injured portion of the lateral ligament complex [3], followed by the calcaneofibular ligament (CFL, tight at ankle neutral), and less frequently, the posterior talofibular ligament (PTFL). A classic basketball injury occurs when a player inverts his ankle on another player's foot while landing after a jump or rebound [4]. However, many ankle sprains occur as the athlete attempts to move around players during cutting or pivoting maneuvers. The focus of the athlete's attention is shifted away from the playing surface, thereby exposing the lower extremity to injury.

Residual disability after ankle sprains is present in 32–76% of patients [5, 6]. Patients may continue to have symptoms, including persistent synovitis or tendinitis, restrictions in the arthrokinematics of the ankle, swelling, muscle weakness, degenerative changes, and recurrent instability.

The etiology of chronic ankle instability is multifaceted, caused by a combination of a mechanical or functional phenomenon [7].

Mechanical instability is defined by a loss of structural support by the ATFL and CFL through frank tear of the ligaments or if the ligaments healed in an elongated state. This structural loss allows for pathologic laxity and excessive motion of the ankle joint. This hypermobility is in contrast to functional instability which is due to a loss of neuromuscular and postural control, proprioception, and muscle strength [8]. In this type of instability, the foot may seem to “give way” after an ankle sprain [9].

In Freeman’s opinion [9], functional instability results from postural stability deficits. However, specific differences in postural control between mechanical and functional instability are not as well-defined as the early literature leads one to believe. In a recent study of a comparison of mechanical and functionally unstable patients and healthy controls, mechanically unstable ankles showed significantly greater postural sway in all directions compared with those in the control group with eyes closed [7]. In the functionally unstable group, no difference was found in postural sway compared with healthy ankles. However, regardless of the etiology, both the types of instability increase the risk of athletic injury.

Fact Box: Pathophysiology of Chronic Lateral Ankle Injury

- Basketball players are prone to lateral ligamentous ankle injuries.
- The ATFL is the most commonly injured lateral ligament.
- Chronic ankle instability is due to a combination of neural, muscular, and structural deficits.

37.3 Epidemiology

Ankle sprains are the most frequent injury sustained in basketball players [10–12]. In a series of professional basketball players reported by Deitch and colleagues [13], lateral ankle sprains were the most common injury diagnosis (13.7%

of all injuries) in both the National Basketball Association (NBA) and the Women’s National Basketball Association (WNBA). In a prospective study spanning 17 NBA seasons from 1988 to 2005, Drakos et al. [2] found that ankle sprains accounted for 11.6% of all games missed, making them the third most common reason for games being missed. The incidence of ankle sprain is more than twice as common as any other injury at 3.4 per 1000 athlete exposures. In a 2-year prospective study of female Greek professional basketball players, most ankle sprains occurred within the 3-point line and occurred during games rather than practice. The highest rate of injury was in centers, followed by guards and forwards [14]. This cohort had an ankle sprain rate of 1.12 per 1000 h of exposure to injury.

Though the incidence of ankle sprains is higher in athletes who have had a prior ankle sprain [15–17], there is conflicting evidence in the literature as to other demographical risk factors for ankle sprains. Among collegiate basketball players, Hosea et al. [18] found a higher rate of grade I ankle sprains in women than in men by 25%, but for higher grade sprains the incidence was similar between groups. However, the study on NBA and WNBA basketball players found no difference in incidence of ankle sprains between genders [13]. A study of high school athletes found that males with a higher body mass index (BMI) had a higher incidence of ankle sprains [16]. Similarly, in a study of 10,393 recreational basketball players, McKay et al. found that the risk of ankle injury was not related to gender, age, height, or weight [17].

37.4 Therapy and Rehabilitation

When undertreated, chronic lateral ankle instability can possibly lead to recurrent sprains, chronic pain, peroneal tendon injuries, osteochondral lesions, and early arthritis. Nonoperative, functional rehabilitation and therapy is the preferred method of treatment for chronic lateral ankle instability, regardless of grade. In the office or training room, an athlete suspected of chronic

instability can mimic several subtle injuries, including an anterior process of the calcaneus fracture, a lateral talar process fracture, a fifth metatarsal stress fracture, osteochondral lesion of the talus, or a peroneal tendon tear or rupture. If after the appropriate amount of rest and rehabilitation the athlete has persistent dysfunction, then further evaluation should be performed, which usually includes MRI evaluation. Findings may include a concomitant injury not previously appreciated, such as an osteochondral lesion of the talus or peroneal tendon pathology, which would warrant further management [19].

Prolonged immobilization following ankle sprains is a common treatment error in the general population [20]. Functional rehabilitation serves as the traditional model for the management of chronic lateral ankle instability in basketball players. There are well-documented deficits in proprioception, balance, and motor control following chronic instability events—functional therapy serves to address these deficits [21–24]. One study specifically found strength deficits existing up to 6 months following injury which can lead to further injury, especially in high demand athletes [25].

Fact Box: Rehabilitation and Therapy for Chronic Ankle Instability

- The traditional method of functional rehabilitation includes immediate range of motion activity, strengthening, proprioceptive exercises, and sport-specific activity.
- Sport-specific training can challenge the somatosensory system of basketball players to enhance proprioception.
- There is growing evidence for new therapeutic modalities including blood flow restriction training.

The four primary aspects of functional rehabilitation are range of motion, strengthening, proprioception, and activity-specific training. Range of motion activity must be initiated as soon as

possible with emphasis on Achilles tendon stretching. Strengthening should target all muscles of bilateral ankles, starting with isometric activity and progressing to dynamic resisted exercises and sport-specific training [26]. As an athlete achieves weight-bearing without pain, proprioceptive training is initiated. Therapists often use various dynamic platforms and surfaces to test an athlete's rehabilitative recovery [27]. Exercises that challenge the somatosensory system with even and uneven surfaces have proven to be effective for reducing ankle sprains in athletes [28]. Basketball-specific drills can integrate passing, ball handling, and multidirectional reactive drills with various stimuli to emphasize external focus of attention during proprioceptive training. Finally, the basketball player must return to sport-specific training with pain-free activity and movements. Treating therapists must create exercises that emulate and challenge the neuromuscular coordination required for basketball-specific movements.

Recently, there has been growing interest toward blood flow restriction training. There is some early evidence supporting promotion of muscular hypertrophy and strength increases with the advantage of lower loading on painful joints and bones [29, 30]. In application with chronic ankle instability, blood flow restriction therapy addresses pain modulation with decreasing the load across the painful, recently injured ankle while diminishing disuse atrophy [31]. At this time, there is no specific research dedicated to blood flow restriction training in basketball players.

37.5 Operative Treatment

Operative options are reserved for basketball players who have failed to respond to nonoperative management. This is a rare occurrence. Surgical management of chronic lateral ankle instability attempts to restore the native ankle anatomy and joint kinematics. The options for anatomic surgical techniques include lateral ankle ligament repair versus anatomic allograft/autograft/braided polyblend suture reconstruc-

tion. The determination of which strategy is used is based on the degree of injury and tissue quality from preoperative imaging.

The most common surgical technique that aims to restore the anatomic lateral ligamentous complex while preserving kinematics is the Brostrom procedure. This procedure includes many modifications over the years that maintain the primary goal of ankle stability and motion preservation. This procedure is designed to repair the ATFL and CFL in their normal anatomic locations [3] (Figures 37.1–37.6) The Brostrom technique has shown excellent results for the treatment of chronic lateral ankle instability with 26 years follow-up [32]. Gould et al. described an augmentation step to the Brostrom repair that incorporates the inferior extensor retinaculum secured to the fibula [33]. This has the effect of reinforcing the ATFL repair, limiting inversion

and stabilization of the subtalar joint. This modification has been shown to increase strength of the repair by 50% [34]. Li et al. found that using



Fig. 37.3 Creation of bleeding surface on fibula



Fig. 37.1 ATFL and CFL exposure



Fig. 37.4 Placement of suture anchor at anatomic location at ATFL



Fig. 37.2 Inspection of peroneal tendons after CFL exposure



Fig. 37.5 Advancement of ligament to fibula



Fig. 37.6 Advancing inferior extensor retinaculum over repair

suture anchors in a variant of the Gould-modified Brostrom procedure was effective in returning high-demand athletes to pre-injury function [35].

Fact Box: Operative Management for Chronic Ankle Instability

- Operative management is reserved for patients who have shown little to no improvement after prolonged physical therapy and strengthening regimen.
- There is good evidence supporting return to pre-injury status for Brostrom procedure with Gould modification as a successful procedure in high-impact elite athletes.
- When there is severe tissue attenuation, anatomic lateral reconstruction with allograft or autograft tendon has been equally effective to standard anatomic repair.

When tissue quality is inadequate, another surgical option is anatomic lateral ligamentous reconstruction. This procedure entails using either autograft or allograft tendon to reconstruct the CFL and ATFL ligaments in anatomic position by using bone tunnels or suture anchors. Appropriate tensioning of the graft is paramount to the success of the surgery with the goal of

emulating the native lateral ligaments. One study used semitendinosus or gracilis non-irradiated allografts for lateral ligament reconstruction with interference screws and found no significant difference in any outcome score when compared to standard anatomic ligamentous repair [36]. Another option is augmenting the native tissue (or after a Brostrom) with a suture tape device. In a recent study by Cho and colleagues, patient-reported functional outcomes significantly improved after lateral ligament augmentation using suture tape. Isokinetic peroneal strength and postural control improved; however, there was no positive effect on proprioception of the ankle [37].

37.6 Return to Sport

When treating basketball players or elite athletes in general, the first question asked of orthopedic surgeons is when they will be able to return to play. The general post-injury protocol consists of 2–4 weeks of immobilization with slow return to rehabilitative exercises and range-of-motion therapy. Following range-of-motion improvement, proprioceptive training is introduced. Broadly speaking, return to play has been considered when an athlete is not experiencing pain or swelling with therapeutic activity. Further assessment of ankle stability, strength, balance, and postural control help guide the treating physicians and therapists for return to sport. Simple protocols are often used to determine readiness for return such as cutting exercises, agility drills or sport-specific activities (such as double jumps in basketball) [35]. Ultimately, readiness for play is based on a multidisciplinary approach with active participation between the treating physician, physical therapist, team training staff, and the player.

In regard to postoperative rehabilitation following a modified Brostrom operation, there is minimal literature guiding athletes and physicians to predicting return to training and sport. Lee et al. retrospectively reviewed 18 elite athletes (including basketball players) who underwent a modified Brostrom procedure following

failed conservative management and found the mean length of time to return to personal training was 2 months, return to team training was 3 months and return to play was 4 months. About 83.3% returned to pre-injury level of sports activity by 4 months and all resumed participation 8 months after lateral ankle repair [38]. At our institutions, we allow the player to return to play after 6 weeks of protected weight-bearing and 8–12 weeks of rehabilitation.

37.7 Injury Prevention

The most important risk factor for an ankle sprain is a previous ankle sprain. One study found a fivefold increase in ankle injuries in previously injured basketball players compared to those with no previous history of ankle injury [17]. This predilection is thought to be due to a combination of mechanical and functional instability. One of the most predictable ways to prevent recurrent or chronic instability is to fully rehabilitate the initial injury by focusing on optimizing both proprioception and peroneal strength [22]. This has been shown to be effectively done through the use of a balance or wobble board (also called ankle disk training) [27].

Given the frequency of lateral ankle sprains and the disability that results (including game time loss and increased risk of re-injury), much attention has been placed on the prevention of these injuries. A Cochrane review of 14 randomized trials with 8279 total subjects was performed to determine the effectiveness of various prevention strategies [39]. The only intervention that showed a significant reduction in recurrent ankle sprains was the use of an external ankle support device (i.e., semi-rigid orthosis, air-cast brace) [40, 41]. For basketball players returning to play, some external bracing should be considered to decrease the likelihood of recurrent ankle sprains and predispose patient to further lateral ligamentous laxity [42].

Although athletic taping is frequently used among basketball players upon return to play after injury, it remains inconclusive as to whether

this modality effectively prevents recurrent ankle sprains [39]. Biomechanical studies have assessed the inversion restraint of ankle taping and have found a decrease in restrictive capacity and inversion resistance with prolonged activity [43, 44, 45]. The downsides of athletic taping include length of application, requirement of a skilled trainer/therapist to apply, and known high cost compared to some external ankle support devices [46].

Fact Box: Injury Prevention

- The most important risk factor for recurrent ankle sprain is previous ankle sprain.
- The only effective intervention that has been proven to decrease ankle sprains is a semi-rigid ankle orthosis external brace.
- Among multiple studies, taping has not been proven to be effective in decreasing recurrent ankle strains and has a poor biomechanical profile over time.
- Pre-habilitation programs are being utilized among elite basketball players to prevent ankle instability events.

Calf-stretching programs [47] and the use of cushioned insoles were not found to effectively reduce the risk of ankle sprains in the Cochrane review. A separate study found that the presence of air cells in the heel of the shoe was associated with a higher rate of injury (4.3 times more likely) while also stating that those who did not stretch before a game were 2.6 times more likely to injure an ankle than players who did [17]. In a prospective study of intramural basketball players, there was no difference in the rate of ankle injury with those who wore high top sneakers [48].

Prehabilitation programs currently utilized by most NBA teams [49] emphasize overall fitness, balance, and prevention strategies, such as landing with a wide-based stance.

37.8 Conclusions

Injuries to the foot and ankle are frequent in both competitive and recreational basketball, and are an important source of significant time lost from play. Because of the rapid acceleration and deceleration, cutting, jumping, and pivoting maneuvers involved in basketball, the foot and ankle are particularly susceptible to both acute and overuse injuries. Prevention strategies, appropriate operative intervention when indicated, and dedicated functional rehabilitation are all important in the management of the injured basketball player for safe and timely return to play.

37.9 Summary

1. The ankle is the most injured joint in basketball.
2. Most ankle sprains are noncontact injuries and may occur while taking an awkward step during cutting or turning maneuvers.
3. The most important risk factor for an ankle sprain is a previous ankle sprain. One of the most predictable ways to prevent recurrent or chronic instability is to fully rehabilitate the initial injury by focusing on optimizing both proprioception and peroneal strength.
4. Surgery should be reserved solely for failed non-operative management. A lateral ligament anatomic repair or reconstruction is the operative treatment of choice.
5. Return to play is based on an individualized, functional rehabilitation protocol that should incorporate sport-specific training to emulate game-type movements.

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References

1. Commission USPCS. NEISS data highlights 2017. Washington DC 2017.
2. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the national basketball association: a 17-year overview. *Sports Health*. 2010;2:284–90.
3. Brostrom L. Sprained ankles. I Anatomic lesions in recent sprains. *Acta Chir Scand*. 1964;128:483–95.
4. Powell JW, Barber-Foss KD. Sex-related injury patterns among selected high school sports. *Am J Sports Med*. 2000;28:385–91.
5. Gerber JP, Williams GN, Scoville CR, Arciero RA, Taylor DC. Persistent disability associated with ankle sprains: a prospective examination of an athletic population. *Foot Ankle Int*. 1998;19:653–60.
6. Bosien WR, Staples OS, Russell SW. Residual disability following acute ankle sprains. *J Bone Joint Surg Am*. 1955;37-A:1237–43.
7. Chen H, Li HY, Zhang J, Hua YH, Chen SY. Difference in postural control between patients with functional and mechanical ankle instability. *Foot Ankle Int*. 2014;35:1068–74.
8. Hertel J. Functional anatomy, Pathomechanics, and pathophysiology of lateral ankle instability. *J Athl Train*. 2002;37:364–75.
9. Freeman MA, Dean MR, Hanham IW. The etiology and prevention of functional instability of the foot. *J Bone Joint Surg Br*. 1965;47:678–85.
10. Dick R, Hertel J, Agel J, Grossman J, Marshall SW. Descriptive epidemiology of collegiate men's basketball injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. *J Athl Train*. 2007;42:194–201.
11. Starkey C. Injuries and illnesses in the national basketball association: a 10-year perspective. *J Athl Train*. 2000;35:161–7.
12. Hootman JM, Dick R, Agel J. Epidemiology of collegiate injuries for 15 sports: summary and recommendations for injury prevention initiatives. *J Athl Train*. 2007;42:311–9.
13. Deitch JR, Starkey C, Walters SL, Moseley JB. Injury risk in professional basketball players: a comparison of Women's National Basketball Association and National Basketball Association athletes. *Am J Sports Med*. 2006;34:1077–83.
14. Kofotolis N, Kellis E. Ankle sprain injuries: a 2-year prospective cohort study in female Greek professional basketball players. *J Athl Train*. 2007;42:388–94.
15. Milgrom C, Shlamkovitch N, Finestone A, et al. Risk factors for lateral ankle sprain: a prospective study among military recruits. *Foot Ankle*. 1991;12:26–30.
16. McHugh MP, Tyler TF, Tetro DT, Mullaney MJ, Nicholas SJ. Risk factors for noncontact ankle sprains in high school athletes: the role of hip strength and balance ability. *Am J Sports Med*. 2006;34:464–70.
17. McKay GD, Goldie PA, Payne WR, Oakes BW. Ankle injuries in basketball: injury rate and risk factors. *Br J Sports Med*. 2001;35:103–8.
18. Hosea TM, Carey CC, Harrer MF. The gender issue: epidemiology of ankle injuries in athletes who participate in basketball. *Clin Orthop Relat Res*. 2000;372:45–9.
19. Renstrom PA. Persistently painful sprained ankle. *J Am Acad Orthop Surg*. 1994;2:270–80.
20. Kerkhoffs GM, Rowe BH, Assendelft WJ, Kelly KD, Struijs PA, van Dijk CN. Immobilisation for

- acute ankle sprain. A systematic review. *Arch Orthop Trauma Surg.* 2001;121:462–71.
21. Konradsen L, Olesen S, Hansen HM. Ankle sensorimotor control and eversion strength after acute ankle inversion injuries. *Am J Sports Med.* 1998;26:72–7.
 22. Willems T, Witvrouw E, Verstuyft J, Vaes P, De Clercq D. Proprioception and muscle strength in subjects with a history of ankle sprains and chronic instability. *J Athl Train.* 2002;37:487–93.
 23. Hertel J. Functional instability following lateral ankle sprain. *Sports Med.* 2000;29:361–71.
 24. Pietrosimone BG, Gribble PA. Chronic ankle instability and corticomotor excitability of the fibularis longus muscle. *J Athl Train.* 2012;47:621–6.
 25. Perron M, Moffet H, Nadeau S, Hebert LJ, Belzile S. Persistence of long term isokinetic strength deficits in subjects with lateral ankle sprain as measured with a protocol including maximal preloading. *Clin Biomech (Bristol, Avon).* 2014;29:1151–7.
 26. Mattacola CG, Dwyer MK. Rehabilitation of the ankle after acute sprain or chronic instability. *J Athl Train.* 2002;37:413–29.
 27. Verhagen E, van der Beek A, Twisk J, Bouter L, Bahr R, van Mechelen W. The effect of a proprioceptive balance board training program for the prevention of ankle sprains: a prospective controlled trial. *Am J Sports Med.* 2004;32:1385–93.
 28. Mohammadi F. Comparison of 3 preventive methods to reduce the recurrence of ankle inversion sprains in male soccer players. *Am J Sports Med.* 2007;35:922–6.
 29. Kubota A, Sakuraba K, Sawaki K, Sumide T, Tamura Y. Prevention of disuse muscular weakness by restriction of blood flow. *Med Sci Sports Exerc.* 2008;40:529–34.
 30. Centner C, Wiegel P, Gollhofer A, Konig D. Effects of blood flow restriction training on muscular strength and hypertrophy in older individuals: a systematic review and meta-analysis. *Sports Med.* 2019;49:95–108.
 31. Faltus J, Owens J, Hedt C. Theoretical applications of blood flow restriction training in managing chronic ankle instability in the basketball athlete. *Int J Sports Phys Ther.* 2018;13:552–60.
 32. Bell SJ, Mologne TS, Sitler DF, Cox JS. Twenty-six-year results after Brostrom procedure for chronic lateral ankle instability. *Am J Sports Med.* 2006;34:975–8.
 33. Gould N, Seligson D, Gassman J. Early and late repair of lateral ligament of the ankle. *Foot Ankle.* 1980;1:84–9.
 34. Aydogan U, Glisson RR, Nunley JA. Extensor retinaculum augmentation reinforces anterior talofibular ligament repair. *Clin Orthop Relat Res.* 2006;442:210–5.
 35. Li X, Killie H, Guerrero P, Busconi BD. Anatomical reconstruction for chronic lateral ankle instability in the high-demand athlete: functional outcomes after the modified Brostrom repair using suture anchors. *Am J Sports Med.* 2009;37:488–94.
 36. Matheny LM, Johnson NS, Liechti DJ, Clanton TO. Activity level and function after lateral ankle ligament repair versus reconstruction. *Am J Sports Med.* 2016;44:1301–8.
 37. Cho BK, Hong SH, Jeon JH. Effect of lateral ligament augmentation using suture-tape on functional ankle instability. *Foot Ankle Int.* 2019;40:447–56.
 38. Lee K, Jegal H, Chung H, Park Y. Return to play after modified Brostrom operation for chronic ankle instability in elite athletes. *Clin Orthop Surg.* 2019;11:126–30.
 39. Handoll HH, Rowe BH, Quinn KM, de Bie R. Interventions for preventing ankle ligament injuries. *Cochrane Database Syst Rev.* 2001:CD000018.
 40. Sitler M, Ryan J, Wheeler B, et al. The efficacy of a semirigid ankle stabilizer to reduce acute ankle injuries in basketball. A randomized clinical study at West Point. *Am J Sports Med.* 1994;22:454–61.
 41. Surve I, Schweltnus MP, Noakes T, Lombard C. A fivefold reduction in the incidence of recurrent ankle sprains in soccer players using the sport-stirrup orthosis. *Am J Sports Med.* 1994;22:601–6.
 42. Dizon JM, Reyes JJ. A systematic review on the effectiveness of external ankle supports in the prevention of inversion ankle sprains among elite and recreational players. *J Sci Med Sport.* 2010;13:309–17.
 43. Gross MT, Bradshaw MK, Ventry LC, Weller KH. Comparison of support provided by ankle taping and semirigid orthosis. *J Orthop Sports Phys Ther.* 1987;9:33–9.
 44. Greene TA, Hillman SK. Comparison of support provided by a semirigid orthosis and adhesive ankle taping before, during, and after exercise. *Am J Sports Med.* 1990;18:498–506.
 45. Laughman RK, Carr TA, Chao EY, Youdas JW, Sim FH. Three-dimensional kinematics of the taped ankle before and after exercise. *Am J Sports Med.* 1980;8:425–31.
 46. Metcalfe RC, Schlabach GA, Looney MA, Renehan EJ. A comparison of moleskin tape, linen tape, and lace-up brace on joint restriction and movement performance. *J Athl Train.* 1997;32:136–40.
 47. Pope RP, Herbert RD, Kirwan JD, Graham BJ. A randomized trial of preexercise stretching for prevention of lower-limb injury. *Med Sci Sports Exerc.* 2000;32:271–7.
 48. Barrett JR, Tanji JL, Drake C, Fuller D, Kawasaki RI, Fenton RM. High- versus low-top shoes for the prevention of ankle sprains in basketball players. A prospective randomized study. *Am J Sports Med.* 1993;21:582–5.
 49. Bernot MP. Medical care of the Atlanta hawks. *J Med Assoc Ga.* 2000;89:21–3.



Management of Cartilage Injuries of the Foot and Ankle in Basketball

38

Kenneth J. Hunt, Kenneth S. Smith,
and Steve Short

38.1 Incidence and Epidemiology

Little is known about the incidence of talus cartilage lesions in elite basketball players. The true incidence and prevalence are difficult to pinpoint because many lesions are asymptomatic or even if they become symptomatic are not picked up on routine radiographs. Data from the US military suggests a recent increase in the incidence of OLTs with a reported rate of 27 OLTs per 100,000 person years [1]. We do know that ankle sprains are exceedingly common in basketball. Recent studies have shown that ankle sprains comprise 13–22% of all musculoskeletal injuries in elite-level basketball players [2, 3]. Another study from Sweden found that 92% of all players had suffered at least one ankle sprain in their career and that 78% had injured their ankle at least once in the last two seasons [4]. In addition, it has been estimated that up to half of all ankle sprains may result in a chondral injury [5].

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There is a clear association between OLTs and both recurrent ankle ligamentous injuries and ankle fractures. Some studies have reported a 38–51% rate of talus cartilage injury diagnosed by arthroscopy at the time of lateral ligament repair/reconstruction in patients with chronic ankle instability [6, 7]. While ankle fractures are much less common than ankle sprains in basketball players, ankle fractures pose an even higher risk for OLT. Hintermann et al. found an articular cartilage injury rate of 79% in 288 patients treated with ORIF for an ankle fracture [8].

Coexistent ankle instability also impacts the severity of OLTs and patient outcomes. In a study of 420 patients who underwent bone marrow stimulation for OLTs, Lee et al. found that osteochondral lesions in an unstable ankle had an increased proportion of lateral lesions and larger lesions (150 mm² or larger) and were more likely to have additional chondral lesions at the medial malleolus [9]. In addition, patients with an unstable ankle had an increased rate of clinical failure and inferior performance in sport and recreational activities.

38.2 Pathophysiology

Osteochondral injuries of the talus typically fall into one of two categories: idiopathic lesions (osteochondritis dissecans) or post-traumatic lesions (relating to an ankle sprain or fracture). The primary focus of this chapter is on the

traumatic lesions although most of the treatment principles are the same between the two groups. In one of the first descriptive articles on the subject, Flick et al. noted a history of trauma in 80% of medial and 100% of lateral talus lesions and while this difference is of questionable importance, what is notable is the high degree of association between all OLTs and trauma [10]. Another more recent systematic review of 582 patients with OLT reported that 76% of lesions had a history of traumatic injury [11].

The majority of OLTs are noted in one of two locations: the anterolateral or posteromedial aspect of the talus. The mechanism for the anterolateral lesion is thought to be ankle inversion and dorsiflexion causing the talar dome to forcibly contact the distal fibula [12]. In contrast, posteromedial lesions are thought to arise from an injury with the ankle in inversion and plantarflexion. In either location, the compressive force to the underlying subchondral bone causes a disruption in the fragile blood supply. This may be due to an acute injury or repetitive microtrauma in the setting of chronic ankle instability. This force can also lead to direct injuries to the articular cartilage such as a compression injury leading to chondrocyte death. In contrast, the force may lead to indirect cartilage damage via changes in the subchondral bone. The alteration in the bony blood supply can cause bone collapse and cyst formation which in turn leads to a change in the mechanical loading of the overlying cartilage. Once the cartilage detaches from the injured subchondral bone, flaps and/or loose bodies can form, which can lead to synovitis and mechanical symptoms in the ankle [13] (Fig. 38.1). The pain associated with OLTs may stem from load-bearing induced increased intra-articular pressure that drives fluid into the subchondral bone inducing pain at the bone nerve endings [14]. As intra-articular pressure recurs and increases, subchondral edema and cysts can develop, worsening the symptoms.

There is no clear link between traumatic OLTs and the development of osteoarthritis in the ankle joint. Synovial fluid studies suggest that the cytokine profile of OLT differs from that of ankle arthritis, with a greater number of pain cytokines and fewer degenerative cytokines. However,



Fig. 38.1 Arthroscopic images illustrating cartilage detachment from the talar surface with flaps which can generate loose bodies, and lead to synovitis and mechanical symptoms in the ankle

there is evidence that larger lesions are associated with an increased risk of edge loading and a shift of ankle contact mechanics [15].

Interestingly, new research has brought to light another potential pathophysiological factor. Telleria et al. (2018) found a statistically significant increase in the rate of vitamin D deficiency in patients presenting with OLTs compared with controls (54% vs 28%) [16]. While we know that vitamin D is critically important to bone mineral density, this is the first study to show a correlation between vitamin D deficiency and OLTs. This study suggests that perhaps a routine ankle sprain in an individual with normal vitamin D levels can develop to an OLT in a patient with compromised bone mineral density. This information is even more important given a recent study found that only 27% of professional basketball players have a sufficient level of vitamin D [17]. Since vitamin D supplementation is low risk, this should be considered as part of a nutritional regimen for basketball players.

38.3 Diagnosis

As with all patient encounters, arriving at the correct diagnosis begins with a good history. While the majority of patients have a history of trauma,

some studies have reported up to 2–3 years between traumatic injury and presentation [18]. However, one should try to elucidate any history of recurrent instability or any symptoms of locking or catching in the ankle. Like most conditions, the primary presenting symptom of patients with OLTs is pain. Stiffness, swelling, and/or catching can accompany the pain in many instances [19]. The physical examination may find tenderness to palpation, especially with hyper-plantarflexion of the ankle, which uncovers the talar dome from the tibial plafond. An effusion may be present. Crepitus, pain, and stiffness can often be seen when taking the ankle through a range of motion. One should perform provocative stability tests of the ankle and compare with the contralateral ankle to rule out concomitant ankle instability. In addition, any hindfoot deformity that could be predisposing to eccentric overload of one side of the talus needs to be noted. Because no single symptom is specific only to OLTs, the differential diagnosis is

quite large and includes common pathologies such as synovitis, ankle instability, ankle impingement, and ankle arthritis.

All patients complaining of ankle pain should undergo weight-bearing radiographs of the ankle including antero-posterior, lateral, and mortise projections. While acute, non-displaced injuries are not often seen on plain films, there are many lesions that can be picked up from radiographs alone [20, 21]. Any displaced lesions, lesions associated with osteonecrosis, or chronic lesions with underlying cysts can usually easily be seen on plain films. However, given the low sensitivity of plain radiographs, if a high degree of suspicion exists for an OLT, one should proceed with advanced imaging even in the setting of normal radiographs (Fig. 38.2 MRI).

Some debate still exists as to which advanced imaging modality is the best to diagnose OLTs. One prospective study found that the sensitivity and specificity of MRI were 96% and 96%, respectively [22]. These values for CT were

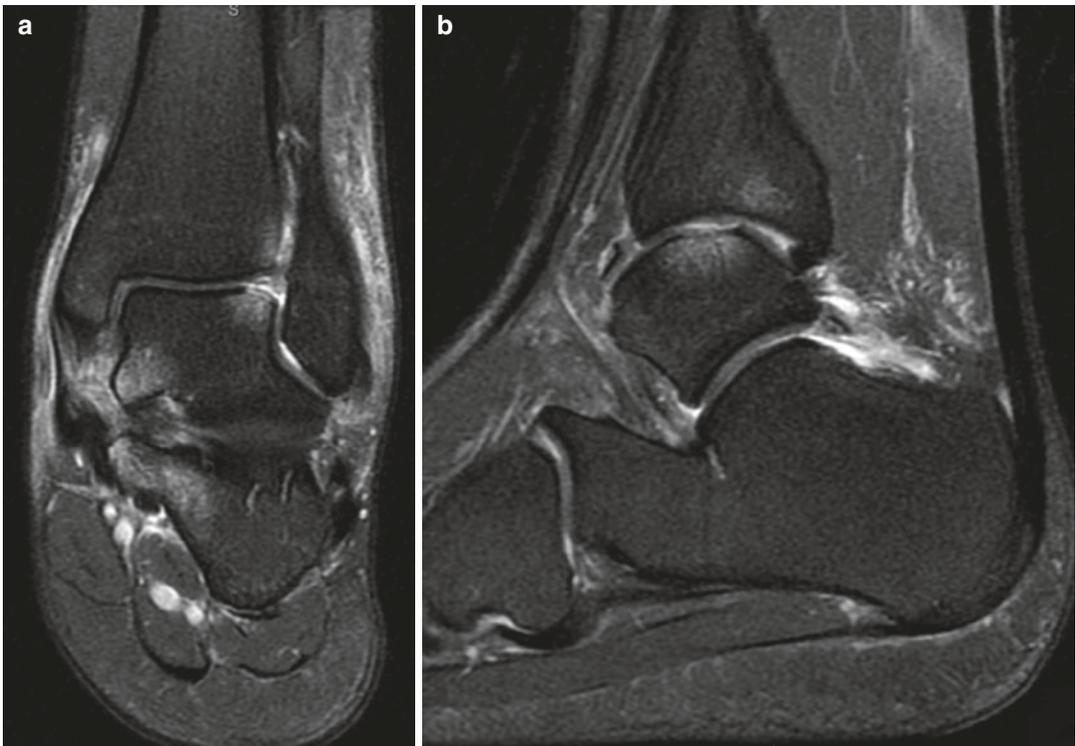


Fig. 38.2 Fat-suppressed proton-density-weighted coronal (a) and sagittal (b) MRI images of a lateral-central OLT

found to be 81% and 99% with no statistically significant difference between the two modalities. However, MRI is much better at evaluating soft tissues and diagnosing other pathologies about the ankle including ligamentous injuries, tendinous injuries, soft tissue cysts, and abnormalities associated with neurovascular structures. For this reason, it is recommended to obtain an MRI and proceed with a CT scan only if needed to better quantify the degree of bony destruction in the event a large cyst is noted on the MRI.

38.4 Classification

There are many different classification systems for OLTs that span over 50 years of clinical advancement. As the first system created in 1959, Berndt and Harty's classification scheme is still the most commonly cited (Table 38.1, Fig. 38.3) [12]. More recently, several researchers have presented either updates to the original

classification or a new system altogether based on CT, MRI, or arthroscopic findings [23–25]. However, none of these classification systems take into account lesion size, which has been shown to be the most important predictor of outcome and one of the most important factors to help guide treatment [26]. As such, these classification systems are not widely used in clinical practice.

38.5 Treatment

38.5.1 Nonoperative Treatment

Nonoperative treatment for OLTs can be quite effective in the right circumstances. While the operative criteria are somewhat controversial, most physicians agree that nondisplaced or incomplete fractures of the talus can be treated without surgery. The typical modalities include boot immobilization, short-term protected weight-bearing, activity modification, and oral NSAIDs [27]. A recent Korean study found good results with nonoperative management in 142 patients treated nonoperatively for symptomatic OLTs [28]. The average patient reported outcomes all improved, only 6% of the lesions grew larger at an average follow-up of 6 years, and only 6% of patients reported limitations with sports activities. However, how well this study translates to the extreme physical demands of a basketball player is difficult to determine.

Table 38.1 Berndt and Harty's OLT classification

Stage 1	Compression of subchondral bone with intact cartilage
Stage 2	Compression of subchondral bone with partial detachment of osteochondral fragment
Stage 3	Compression of subchondral bone with complete detachment of osteochondral fragment
Stage 4	Compression of subchondral bone with complete detachment of osteochondral fragment with displacement from donor site

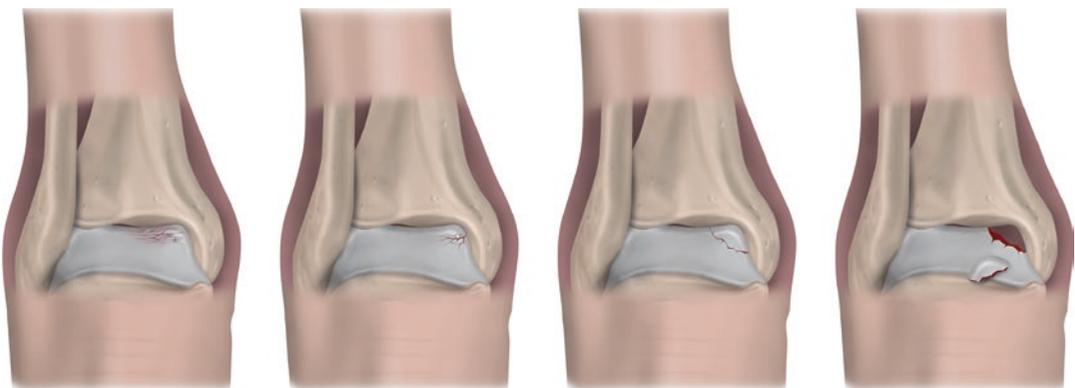


Fig. 38.3 An illustration of the Berndt and Harty's OLT classification. Courtesy and credit to Pontus Andersson (all right reserved). Used with permission

38.5.2 Surgical Management

Surgical management is typically reserved for either acute, displaced, or complete OLTs, large OLTs, and chronic, symptomatic OLTs that have failed nonoperative management and remain symptomatic. There are many surgical techniques that have been described to address both acute and chronic OLTs. The treatment strategies can generally be categorized as cartilage repair, cartilage or regeneration, or cartilage replacement (Table 38.2). It is important to recognize the role of subchondral bone as well, both in terms of pain generation and mechanical stability, when developing a treatment strategy for the athlete. We will here focus on the most commonly used techniques in basketball players.

38.5.2.1 Cartilage-Repair Strategies

Bone marrow stimulation through microfracture or drilling is the most commonly utilized strategy, especially for smaller, well-circumscribed OLT lesions. It is considered the first line of treatment by most surgeons in patients who have failed nonoperative management. The technique typically involves curettage of fibrous material and subchondral bone to create a rounded defect with stable edges (Fig. 38.4).

Perforation of the subchondral bone with a microfracture pick or small diameter drill ostensibly allows bone marrow progenitor cells to fill the OLT lesion and stimulate formation of new cartilage. It is generally recognized that the resulting cartilage is primarily type-I fibrocartilage as opposed to approximating native hyaline cartilage (mostly type-II collagen). Studies of

Table 38.2 Surgical treatment strategies for OLTs

Repair	Regeneration	Replacement
Bone marrow stimulation (microfracture)	Autologous chondrocyte implantation (ACI)	Osteochondral autograft transfer (OAT)
Retrograde drilling	Matrix-induced autologous chondrocyte implantation (MACI)	Osteochondral allograft transfer
	Bone-marrow-derived stem cell transplantation	Allograft cartilage (juvenile or adult micronized)
	Adipose-derived stem cell transplantation	

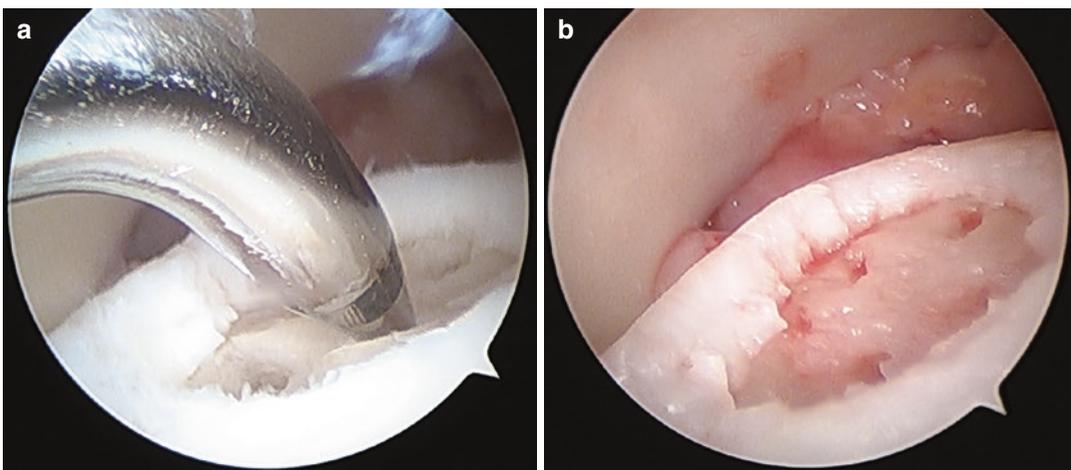


Fig. 38.4 Arthroscopic images illustrating perforation of the subchondral bone with a microfracture pick (a) to achieve subchondral bleeding and stimulation. The micro-

fractures are evenly distributed throughout the lesion to cover the entire defect area (b)

this procedure in the knee have shown that the resulting fibrocartilage is structurally inferior to hyaline cartilage [29]; however, intermediate-term results have shown consistent good functional results for OLTs [18, 30].

There are a number of factors that have been associated with suboptimal results with microfracture. Patient age; OLT chronicity, size, location, and containment; associated joint degeneration; and the presence of subchondral cysts have been identified as prognostic factors. The most consistent data is related to the size of the defect. Chuckpaiwong et al. reported on a large series of patients with OLTs treated with microfracture [31]. They found that all treatment failures had OLT diameters larger than 15 mm. Similarly, Choi et al. found a higher failure rate in lesions that were larger than 150 mm² in area [26]. They concluded that confirming lesion size on MRI may help facilitate better results. Ramponi et al. reviewed 25 clinical outcomes papers evaluating OLTs and concluded that bone marrow stimulation may best be reserved for OLT sizes less than 107.4 mm² in area and/or 10.2 mm in diameter [32]. This corresponds to biomechanical data suggesting that a diameter greater than 10 mm begins to alter the cartilage contact mechanics close to the lesion, increasing edge loading of the OLT [15].

Other factors are less consistently reported. Becher and Thermann reported that older post-traumatic degenerative lesions portended a worse outcome [33]. The presence of subchondral cysts has proven a controversial topic. In some studies, microfracture for OLTs with subchondral cysts has demonstrated poor clinical outcomes, with relatively high failure rates [18]. In other studies, no difference in outcomes has been reported comparing microfracture in patients with subchondral cysts to those without [34]. The location and containment of an OLT impact outcomes as well. In a large series, Choi et al. found that uncontained lesions had worse outcomes with microfracture compared with contained lesions, while that size had little impact [35].

Retrograde Drilling

While it is rare in basketball players, painful talar subchondral bone lesions (e.g., cysts and edema) can be seen with intact overlying cartilage. This may be due to an injury that only damaged bone, or a relatively minor cartilage defect that subsequently healed, leaving only the subchondral lesion. When talar lesions have associated subchondral cysts, but are found arthroscopically to have intact overlying cartilage, retrograde drilling alone can be an effective solution [36, 37]. Various techniques have been described [38]. It is critical that arthroscopy is used to confirm an intact cartilage cap, and during and/or following the retrograde procedure to confirm no extravasation into the joint.

38.5.2.2 Cartilage-Regeneration Strategies

Autologous Chondrocyte Implantation (ACI)

Autologous chondrocyte implantation (ACI) was originally developed for knee cartilage injury and subsequently has been indicated for OLT patients, mostly those with larger lesions or failed prior bone marrow stimulation (BMS) surgery. ACI is a two-stage procedure. First, healthy autograft articular cartilage is harvested from the patient, and the chondrocytes are cultured for several weeks. Then, in a subsequent procedure, viable cultured chondrocytes are implanted into the defect site and covered by a periosteal flap harvested from the distal end of the tibia, facilitating a hyaline-like repair tissue. The primary disadvantage of ACI includes the need for an arthrotomy for exposure, and the need for two surgical procedures, substantially increasing the cost and the potential for morbidity. There have been reported concerns of repair tissue overgrowth [39]. As a result of these disadvantages, ACI is available at a limited number of centers only.

In spite of the limitations, reports on the clinical outcomes of ACI have been largely positive. Giannini et al. reported clinical and MRI outcomes of ACI for OLT at a mean follow-up of

10 years [40]. They reported significant clinical improvement and excellent restoration of the talar articular surface on MRI. In addition, seven of eight athletes in the series resumed sports, five at the same level as prior to injury. In a systematic review, Niemeyer et al. reported a clinical success rate of 89.9% in 213 patients included in 16 level IV studies [41]. Kwak et al. reported that 72% of patients who underwent ACI with a mean lesion area of 200 mm² had good-to-excellent clinical outcomes at a mean follow-up of 70 months and had improved Tegner activity scores [42].

MACI

As advances are made in regenerative techniques for cartilage, there is more promise than ever that simple, effective, and predictable solutions will emerge to cure cartilage injuries in the ankle and avoid long-term adverse consequences of cartilage injury. Matrix-induced autologous chondrocyte implantation (MACI) is a second generation of ACI performed in a single stage. While good results have been shown [43], experience is limited to a select few centers only.

Bone-Marrow-Derived Stem Cell Therapy

Cartilage regeneration with bone-marrow-derived stem cells includes autologous matrix-induced chondrogenesis (AMIC) and other 1-step techniques that utilize bone-marrow-derived cells and/or platelet-rich plasma (PRP) to aid in healing of OLTs. These techniques typically combine microfracture with the addition of autologous iliac crest bone marrow aspirate concentrate, and/or platelet-rich plasma (PRP) to the lesion. The advantages include one-step arthroscopic procedure involving the use of autologous tissue with minimal donor-site morbidity. Outcome data are scarce because of the variety of pathological conditions for which these techniques are used as well as the fact that such data are often reported by the inventors of the techniques. There is limited data on these techniques in the treatment of OLTs in basketball players.

38.5.2.3 Cartilage Replacement Strategies

As a result of concerns regarding the biomechanical properties of the cartilage that results from the above techniques, there are substantial purported advantages to successfully replacing cartilage. To date, most data on replacement techniques is on osteochondral autograft or allograft transplantation. However, this technique is rarely used in a basketball player, and therefore the ICCRA consensus statement on this technique are presented [44].

Newer techniques that implement allograft cartilage have increased considerably in utilization and early results are promising. Allograft transplantation of either particulated juvenile allograft cartilage (Denovo; Zimmer) or micronized adult cartilage (Biocartilage, Arthrex) entails transplantation of fresh cartilage fragments containing live cells within the extracellular matrix. A fibrin adhesive (tissue glue) is used to secure the tissue firmly inside the prepared lesion (Fig. 38.5). The particulated nature of the graft allows arthroscopic application, reducing the need for osteotomies of the malleoli in most cases. There is also no graft contouring, no donor-site morbidity, and the performance of a single-stage procedure. Bone-marrow-derived stem cells can be added to this technique. The primary disadvantages of these techniques is the absence of long-term data.

Coetzee et al. presented a case series of 24 ankles that were treated with DeNovo and followed for an average of 16 months, reporting improvement in clinical outcomes similar to those of bone marrow stimulation, ACI, and MACI [45]. More recent comparisons have shown that this technique does not always result in hyaline cartilage and may not be superior to bone marrow stimulation alone [46]. New cartilage replacement techniques show great promise but are in need of more intermediate and long-term outcomes to prove superiority to other effective techniques which are more cost-effective.

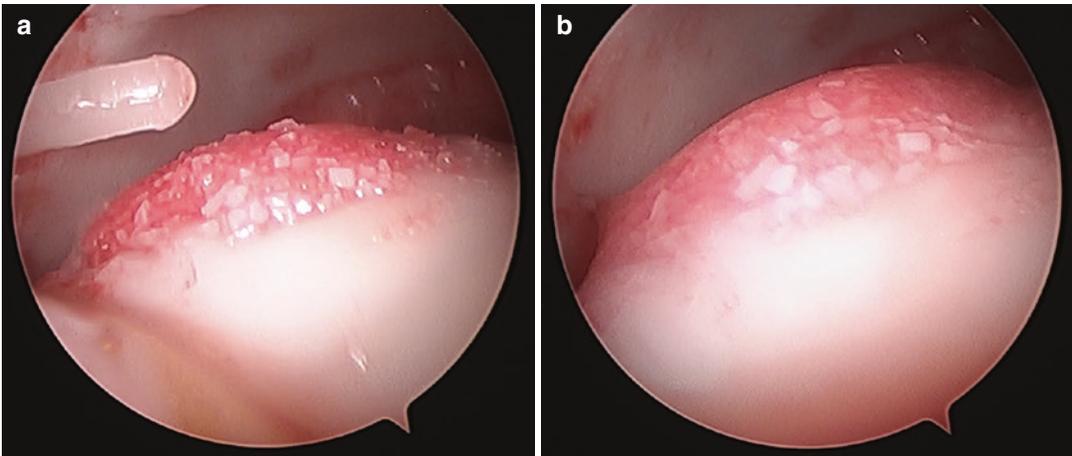


Fig. 38.5 Arthroscopic images illustrating (a) micronized cartilage graft covering OLT defect before and (b) after applying fibrin glue

38.6 Rehabilitation for Safe Return to Basketball

Initial rehabilitative management of cartilage injuries must consider appropriate tissue healing timelines and the natural history of the injury. In these patients, a period of immobilization is necessary to protect the ongoing healing. To maximize the rehabilitation period, focus should be on what can be trained on the athlete, as opposed to what they must be restricted from. A phased rehabilitation protocol is recommended to progress through the appropriate stages and allow return to play. These phases often mimic a modified strength and conditioning periodization program [47, 48].

Periodized rehabilitation can typically be broken down into 3- or 5-phase programs. A 3-phase program is typically defined by acute, sub-acute, and reconditioning phases followed by a return to play [49–51]. A 5-phase program may better address functional impairments and provide a more adequate training stimulus to bridge the gap from returning to play and returning to performance. The initial phases can still typically be defined as acute and sub-acute or proliferation. More functionally, these early phases place an emphasis on general capacity and tissue healing. Describing the final three phases is a matter of

the desired physical adaptations required to recover from injury and restore sport-specific qualities. Common descriptors of these final three phases include accumulation, transmutation or reconditioning, and realization, respectively [47, 52].

Early stage management of injuries consists of a focus on protection and load management. However, early active interventions, as opposed to delayed, may result in superior outcomes [53, 54]. It is recommended that the early management of these injuries follow the POLICE acronym (Protect, Optimally Load, Ice, Compress, and Elevate) [55]. As noted, protecting the lesion is essential in early management, shifting the focus on modified methods to promote local and systemic healing.

In foot and ankle cartilage injuries, the affected limb often requires significant unloading and protection, often non-weight-bearing, due to the nature of the respective tissue damage. Passive and active motion may be allowed depending on the nature of the injury and the course of surgical vs nonsurgical management. Consistent with early protection, local resistance training of the foot and ankle is often limited to open chain activity and required to be performed at sub-threshold resistances. Adverse reactions to this disuse include hypotrophy,

impaired neuromuscular performance, and prolonged function strength and power deficits locally and throughout the affected limb [56–58]. The hypotrophy signaling pathways of immobilization take effect within the first few days of disuse and begin to attenuate around 2 weeks [56].

Utilization of concepts such as the cross-over effect, in which the unaffected limb is resistance trained, has been shown to provide positive adaptations to the contralateral extremity in an attempt to reduce the adverse reactions of disuse [59, 60]. Other exercise modalities such as blood flow-restricted exercise (BFR) may promote an anabolic cascade that simulates heavy loading and improves outcomes, while still protecting the injury from unappropriated forces [61, 62]. Additional benefits of blood flow-restricted exercise may include an increase bony metabolism during an unloading period [63]. If the individual is limited by pain, symptom modification modalities such as joint and soft-tissue-based manual therapies may temporarily improve range of motion, function, and assist in the progression of appropriately dosed exercise [64].

As the athlete progresses beyond the early phases, an increased emphasis is placed on graded, progressive loading. Scheduling these progressions typically takes place in linear or undulated programs. Loads and repetition schemes should attempt to hit a target stimulus to produce mechanotransduction-induced adaptations that include hypertrophy, strength, and power development [52, 65, 66]. Each block of the training process may place a greater focus on different qualities as it respects the tissue and training status and history of the athlete.

Aerobic training is an important consideration for all stages of rehabilitation. The cardiovascular system is quickly and negatively impacted by periods of immobilization. Deconditioning has been identified by increases in resting heart rate by 1–2 beats per minute per day in the initial stages of disuse [67, 68]. In the acute and global capacity phase, an emphasis on long, slow aerobic conditioning may help offset deconditioning as well as satellite cell

availability and promote healing [48, 69]. Long-term development of the aerobic system is also a common method to protect against injury risk [70, 71]. Additional systemic effects such as pain reduction and increased blood perfusion may improve the individuals tolerance to other training stimulus provided in their rehabilitation program [72].

Transitioning to normal running and training may be assisted by hydrotherapy or anti-gravity treadmill running and jumping, and may have a positive improvement in the recovery of articular cartilage and synovial membrane of the ankle [73]. As weight-bearing is allowed and progressed, integration into basketball activity is recommended. Simple plyometric training can be performed as jump shots or layups as opposed to typical clinic-based contact methods. As the volume of sport-specific activity is progressed, long duration cardiovascular activity transitions to tempo, interval, repeat sprint, and agility training variants. Simulating tasks such as defensive shuffling or basic basketball drills with appropriate work to rest ratios and durations are recommended to match training loads as the athlete approaches return to sport. Matching the overall training load to prior levels of competition and demands of play are necessary to maximize performance and reduce the risk of future injury [48].

Take Home Message

Osteochondral lesions of the talus occur most commonly after severe ankle ligament injuries or fractures. Given the long-term functional and degenerative issues that can arise from osteochondral lesions, it is critical to be diligent in the diagnosis and treatment of these injuries in the basketball athlete. New, less invasive treatments are available, which may ultimately provide the medical team with tools to restore or regenerate articular cartilage while allowing safe return to basketball. Rehabilitation of the entire athlete is critical to ensure safe return to high levels of performance.

References

- Orr JD, Dawson LK, Garcia EJ, Kirk KL. Incidence of osteochondral lesions of the talus in the United States military. *Foot Ankle Int.* 2011;32(10):948–54. Epub 2012/01/10. <https://doi.org/10.3113/FAI.2011.0948>.
- Andreoli CV, Chiamonti BC, Buriel E, Pochini AC, Ejnisman B, Cohen M. Epidemiology of sports injuries in basketball: integrative systematic review. *BMJ Open Sport Exerc Med.* 2018;4(1):e000468. <https://doi.org/10.1136/bmjsem-2018-000468>. Epub 2019/01/29. PubMed PMID: 30687514; PMCID: PMC6326319
- Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the national basketball association: a 17-year overview. *Sports Health.* 2010;2(4):284–90. <https://doi.org/10.1177/1941738109357303>. PubMed PMID: 23015949 Epub 2010/07/01. PMCID: PMC3445097
- Leanderson J, Nemeth G, Eriksson E. Ankle injuries in basketball players. *Knee Surg Sports Traumatol Arthrosc.* 1993;1(3–4):200–2. Epub 1993/01/01
- Saxena A, Eakin C. Articular talar injuries in athletes: results of microfracture and autogenous bone graft. *Am J Sports Med.* 2007;35(10):1680–7. Epub 2007/07/28. <https://doi.org/10.1177/0363546507303561>.
- Cha SD, Kim HS, Chung ST, Yoo JH, Park JH, Kim JH, Hyung JW. Intra-articular lesions in chronic lateral ankle instability: comparison of arthroscopy with magnetic resonance imaging findings. *Clin Orthop Surg.* 2012;4(4):293–9. <https://doi.org/10.4055/cios.2012.4.4.293>. Epub 2012/12/04. PubMed PMID: 23205239; PMCID: PMC3504694
- Komenda GA, Ferkel RD. Arthroscopic findings associated with the unstable ankle. *Foot Ankle Int.* 1999;20(11):708–13. Epub 1999/12/03. <https://doi.org/10.1177/107110079902001106>.
- Hintermann B, Regazzoni P, Lampert C, Stutz G, Gachter A. Arthroscopic findings in acute fractures of the ankle. *J Bone Joint Surg Br.* 2000;82(3):345–51. Epub 2000/05/17. <https://doi.org/10.1302/0301-620x.82b3.10064>.
- Lee M, Kwon JW, Choi WJ, Lee JW. Comparison of outcomes for Osteochondral lesions of the talus with and without chronic lateral ankle instability. *Foot Ankle Int.* 2015;36(9):1050–7. Epub 2015/04/15. <https://doi.org/10.1177/1071100715581477>.
- Flick AB, Gould N. Osteochondritis dissecans of the talus (transchondral fractures of the talus): review of the literature and new surgical approach for medial dome lesions. *Foot Ankle.* 1985;5(4):165–85. Epub 1985/01/01
- Tol JL, Struijs PA, Bossuyt PM, Verhagen RA, van Dijk CN. Treatment strategies in osteochondral defects of the talar dome: a systematic review. *Foot Ankle Int.* 2000;21(2):119–26. Epub 2000/02/29
- Berndt AL, Harty M. Transchondral fractures (osteochondritis dissecans) of the talus. *J Bone Joint Surg Am.* 1959;41-A:988–1020. Epub 1959/09/01
- Crawford DC, Safran MR. Osteochondritis dissecans of the knee. *J Am Acad Orthop Surg.* 2006;14(2):90–100. Epub 2006/02/10
- van Dijk CN, Reilingh ML, Zengerink M, van Bergen CJ. Osteochondral defects in the ankle: why painful? *Knee Surg Sports Traumatol Arthrosc.* 2010;18(5):570–80. <https://doi.org/10.1007/s00167-010-1064-x>. Epub 2010/02/13. PubMed PMID: 20151110; PMCID: PMC2855020
- Hunt KJ, Lee AT, Lindsey DP, Slikker W 3rd, Chou LB. Osteochondral lesions of the talus: effect of defect size and plantarflexion angle on ankle joint stresses. *Am J Sports Med.* 2012;40(4):895–901. Epub 2012/03/01. <https://doi.org/10.1177/0363546511434404>.
- Telleria JJM, Ready LV, Bluman EM, Chiodo CP, Smith JT. Prevalence of vitamin D deficiency in patients with Talar Osteochondral lesions. *Foot Ankle Int.* 2018;39(4):471–8. Epub 2018/01/24. <https://doi.org/10.1177/1071100717745501>.
- Grieshober JA, Mehran N, Photopolous C, Fishman M, Lombardo SJ, Kharrazi FD. Vitamin D insufficiency among professional basketball players: a relationship to fracture risk and athletic performance. *Orthop J Sports Med.* 2018;6(5) <https://doi.org/10.1177/2325967118774329>. 2325967118774329. Epub 2018/05/31. PubMed PMID: 29845086; PMCID: PMC5964858
- Robinson DE, Winson IG, Harries WJ, Kelly AJ. Arthroscopic treatment of osteochondral lesions of the talus. *J Bone Joint Surg Br.* 2003;85(7):989–93. Epub 2003/10/01. <https://doi.org/10.1302/0301-620x.85b7.13959>.
- McGahan PJ, Pinney SJ. Current concept review: osteochondral lesions of the talus. *Foot Ankle Int.* 2010;31(1):90–101. Epub 2010/01/14. <https://doi.org/10.3113/FAI.2010.0090>.
- Anderson IF, Crichton KJ, Grattan-Smith T, Cooper RA, Brazier D. Osteochondral fractures of the dome of the talus. *J Bone Joint Surg Am.* 1989;71(8):1143–52. Epub 1989/09/01
- Loomer R, Fisher C, Lloyd-Smith R, Sisler J, Cooney T. Osteochondral lesions of the talus. *Am J Sports Med.* 1993;21(1):13–9. Epub 1993/01/01. <https://doi.org/10.1177/036354659302100103>.
- Verhagen RA, Maas M, Dijkgraaf MG, Tol JL, Krips R, van Dijk CN. Prospective study on diagnostic strategies in osteochondral lesions of the talus. Is MRI superior to helical CT? *J Bone Joint Surg Br.* 2005;87(1):41–6. Epub 2005/02/03
- Ferkel RD, Zanotti RM, Komenda GA, Sgaglione NA, Cheng MS, Applegate GR, Dopirak RM. Arthroscopic treatment of chronic osteochondral lesions of the talus: long-term results. *Am J Sports Med.* 2008;36(9):1750–62. Epub 2008/08/30. <https://doi.org/10.1177/0363546508316773>.
- Hepple S, Winson IG, Glew D. Osteochondral lesions of the talus: a revised classification. *Foot Ankle Int.* 1999;20(12):789–93. Epub 1999/12/28. <https://doi.org/10.1177/107110079902001206>.

25. Pritsch M, Horoshovski H, Farine I. Arthroscopic treatment of osteochondral lesions of the talus. *J Bone Joint Surg Am.* 1986;68(6):862–5. Epub 1986/07/01
26. Choi WJ, Park KK, Kim BS, Lee JW. Osteochondral lesion of the talus: is there a critical defect size for poor outcome? *Am J Sports Med.* 2009;37(10):1974–80. Epub 2009/08/06. <https://doi.org/10.1177/0363546509335765>.
27. Hannon CP, Smyth NA, Murawski CD, Savage-Elliott I, Deyer TW, Calder JD, Kennedy JG. Osteochondral lesions of the talus: aspects of current management. *Bone Joint J.* 2014;96-B(2):164–71. Epub 2014/02/05. <https://doi.org/10.1302/0301-620X.96B2.31637>.
28. Seo SG, Kim JS, Seo DK, Kim YK, Lee SH, Lee HS. Osteochondral lesions of the talus. *Acta Orthop.* 2018;89(4):462–7. <https://doi.org/10.1080/17453674.2018.1460777>. Epub 2018/04/11. PubMed PMID: 29635971; PMCID: PMC6600130
29. Nehrer S, Spector M, Minas T. Histologic analysis of tissue after failed cartilage repair procedures. *Clin Orthop Relat Res.* 1999;365:149–62. Epub 2000/01/11. <https://doi.org/10.1097/00003086-199908000-00020>.
30. Savva N, Jabur M, Davies M, Saxby T. Osteochondral lesions of the talus: results of repeat arthroscopic debridement. *Foot Ankle Int.* 2007;28(6):669–73. Epub 2007/06/27. <https://doi.org/10.3113/FAI.2007.0669>.
31. Chuckpaiwong B, Berkson EM, Theodore GH. Microfracture for osteochondral lesions of the ankle: outcome analysis and outcome predictors of 105 cases. *Arthroscopy.* 2008;24(1):106–12. Epub 2008/01/10. <https://doi.org/10.1016/j.arthro.2007.07.022>.
32. Ramponi L, Yasuni Y, Murawski CD, Ferkel RD, DiGiovanni CW, Kerkhoffs G, Calder JDF, Takao M, Vannini F, Choi WJ, Lee JW, Stone J, Kennedy JG. Lesion size is a predictor of clinical outcomes after bone marrow stimulation for Osteochondral lesions of the talus: a systematic review. *Am J Sports Med.* 2017;45(7):1698–705. Epub 2016/11/18. <https://doi.org/10.1177/0363546516668292>.
33. Becher C, Thermann H. Results of microfracture in the treatment of articular cartilage defects of the talus. *Foot Ankle Int.* 2005;26(8):583–9. Epub 2005/08/24. <https://doi.org/10.1177/107110070502600801>.
34. Han SH, Lee JW, Lee DY, Kang ES. Radiographic changes and clinical results of osteochondral defects of the talus with and without subchondral cysts. *Foot Ankle Int.* 2006;27(12):1109–14. Epub 2007/01/09. <https://doi.org/10.1177/107110070602701218>.
35. Choi WJ, Choi GW, Kim JS, Lee JW. Prognostic significance of the containment and location of osteochondral lesions of the talus: independent adverse outcomes associated with uncontained lesions of the talar shoulder. *Am J Sports Med.* 2013;41(1):126–33. Epub 2012/08/04. <https://doi.org/10.1177/0363546512453302>.
36. Geerling J, Zech S, Kendoff D, Citak M, O'Loughlin PF, Hufner T, Krettek C, Richter M. Initial outcomes of 3-dimensional imaging-based computer-assisted retrograde drilling of talar osteochondral lesions. *Am J Sports Med.* 2009;37(7):1351–7. Epub 2009/04/10. <https://doi.org/10.1177/0363546509332499>.
37. Kono M, Takao M, Naito K, Uchio Y, Ochi M. Retrograde drilling for osteochondral lesions of the talar dome. *Am J Sports Med.* 2006;34(9):1450–6. Epub 2006/04/26. <https://doi.org/10.1177/0363546506287300>.
38. Anders S, Lechler P, Rackl W, Grifka J, Schaumburger J. Fluoroscopy-guided retrograde core drilling and cancellous bone grafting in osteochondral defects of the talus. *Int Orthop.* 2012;36(8):1635–40. <https://doi.org/10.1007/s00264-012-1530-9>. Epub 2012/04/12. PubMed PMID: 22491802; PMCID: PMC3535023
39. Nam EK, Ferkel RD, Applegate GR. Autologous chondrocyte implantation of the ankle: a 2- to 5-year follow-up. *Am J Sports Med.* 2009;37(2):274–84. Epub 2008/12/24. <https://doi.org/10.1177/0363546508325670>.
40. Giannini S, Battaglia M, Buda R, Cavallo M, Ruffilli A, Vannini F. Surgical treatment of osteochondral lesions of the talus by open-field autologous chondrocyte implantation: a 10-year follow-up clinical and magnetic resonance imaging T2-mapping evaluation. *Am J Sports Med.* 2009;37(Suppl 1):112S–8S. Epub 2009/12/16. <https://doi.org/10.1177/0363546509349928>.
41. Niemeyer P, Salzmann G, Schmal H, Mayr H, Sudkamp NP. Autologous chondrocyte implantation for the treatment of chondral and osteochondral defects of the talus: a meta-analysis of available evidence. *Knee Surg Sports Traumatol Arthrosc.* 2012;20(9):1696–703. Epub 2011/11/01. <https://doi.org/10.1007/s00167-011-1729-0>.
42. Kwak SK, Kern BS, Ferkel RD, Chan KW, Kasraeian S, Applegate GR. Autologous chondrocyte implantation of the ankle: 2- to 10-year results. *Am J Sports Med.* 2014;42(9):2156–64. Epub 2014/07/25. <https://doi.org/10.1177/0363546514540587>.
43. Giza E, Sullivan M, Ocel D, Lundeen G, Mitchell ME, Veris L, Walton J. Matrix-induced autologous chondrocyte implantation of talus articular defects. *Foot Ankle Int.* 2010;31(9):747–53. Epub 2010/10/01. <https://doi.org/10.3113/FAI.2010.0747>.
44. Smyth NA, Murawski CD, Adams SB Jr, Berlet GC, Buda R, Labib SA, Nunley JA 2nd, Raikin SM, International Consensus Group on Cartilage Repair of the A. Osteochondral allograft: proceedings of the international consensus meeting on cartilage repair of the ankle. *Foot Ankle Int.* 2018;39(1_suppl):35S–40S. Epub 2018/09/15. <https://doi.org/10.1177/1071100718781097>.
45. Coetzee JC, Giza E, Schon LC, Berlet GC, Neufeld S, Stone RM, Wilson EL. Treatment of osteochondral lesions of the talus with particulated juvenile cartilage. *FootAnkleInt.* 2013;34(9):1205–11. Epub 2013/04/12. <https://doi.org/10.1177/1071100713485739>.
46. Karnovsky SC, DeSandis B, Haleem AM, Sofka CM, O'Malley M, Drakos MC. Comparison

- of juvenile allogeneous articular cartilage and bone marrow aspirate concentrate versus microfracture with and without bone marrow aspirate concentrate in arthroscopic treatment of Talar osteochondral lesions. *Foot Ankle Int.* 2018;39(4):393–405. Epub 2018/01/13. <https://doi.org/10.1177/1071100717746627>.
47. Lorenz DS, Reiman MP, Walker JC. Periodization: current review and suggested implementation for athletic rehabilitation. *Sports Health.* 2010;2(6):509–18. <https://doi.org/10.1177/1941738110375910>. Epub 2010/11/01. PubMed PMID: 23015982; PMCID: PMC3438871
48. Morrison S, Ward P, duManoir GR. Energy system development and load management through the rehabilitation and return to play process. *Int J Sports Phys Ther.* 2017;12(4):697–710. Epub 2017/09/14. PubMed PMID: 28900575; PMCID: PMC5534159
49. Anloague PA, Strack DS. Considerations in the diagnosis and accelerated return to sport of a professional basketball player with a triceps Surae injury: a case report. *J Orthop Sports Phys Ther.* 2018;48(5):388–97. Epub 2018/04/07. <https://doi.org/10.2519/jospt.2018.7192>.
50. Short SM, Anloague PA, Strack DS. Rehabilitation and return to sport following surgical repair of the rectus abdominis and adductor longus in a professional basketball player: a case report. *J Orthop Sports Phys Ther.* 2016;46(8):697–706. Epub 2016/07/05. <https://doi.org/10.2519/jospt.2016.6352>.
51. Wilk KE, Reinold MM, Dugas JR, Arrigo CA, Moser MW, Andrews JR. Current concepts in the recognition and treatment of superior labral (SLAP) lesions. *J Orthop Sports Phys Ther.* 2005;35(5):273–91. Epub 2005/06/22. <https://doi.org/10.2519/jospt.2005.35.5.273>.
52. Lorenz D, Morrison S. Current concepts in periodization of strength and conditioning for the sports physical therapist. *Int J Sports Phys Ther.* 2015;10(6):734–47. Epub 2015/12/01. PubMed PMID: 26618056; PMCID: PMC4637911
53. Deal J, Groth A, Ryan P. Early versus delayed weightbearing after microfracture for osteochondral lesions of the talus. *Foot Ankle Orthopaed.* 2017;2(3.):2473011417S000038 <https://doi.org/10.1177/2473011417s000038>.
54. McCormack R, Bovard J. Early functional rehabilitation or cast immobilisation for the postoperative management of acute Achilles tendon rupture? A systematic review and meta-analysis of randomised controlled trials. *Br J Sports Med.* 2015;49(20):1329–35. Epub 2015/08/19. <https://doi.org/10.1136/bjsports-2015-094935>.
55. Bayer ML, Magnusson SP, Kjaer M, Tendon Research Group B. Early versus delayed rehabilitation after acute muscle injury. *N Engl J Med.* 2017;377(13):1300–1. Epub 2017/09/28. <https://doi.org/10.1056/NEJMc1708134>.
56. Appell HJ. Muscular atrophy following immobilisation. A review. *Sports Med.* 1990;10(1):42–58. Epub 1990/07/01. <https://doi.org/10.2165/00007256-199010010-00005>.
57. Brooks NE, Myburgh KH. Skeletal muscle wasting with disuse atrophy is multi-dimensional: the response and interaction of myonuclei, satellite cells and signaling pathways. *Front Physiol.* 2014;5:99. <https://doi.org/10.3389/fphys.2014.00099>. Epub 2014/03/29. PubMed PMID: 24672488; PMCID: PMC3955994
58. Campbell M, Varley-Campbell J, Fulford J, Taylor B, Mileva KN, Bowtell JL. Effect of immobilisation on neuromuscular function in vivo in humans: a systematic review. *Sports Med.* 2019;49(6):931–50. <https://doi.org/10.1007/s40279-019-01088-8>. Epub 2019/03/23. PubMed PMID: 30900205; PMCID: PMC6548068
59. Cirer-Sastre R, Beltran-Garrido JV, Corbi F. Contralateral effects after unilateral strength training: a meta-analysis comparing training loads. *J Sports Sci Med.* 2017;16(2):180–6. Epub 2017/06/21. PubMed PMID: 28630570; PMCID: PMC5465979
60. Lee M, Carroll TJ. Cross education: possible mechanisms for the contralateral effects of unilateral resistance training. *Sports Med.* 2007;37(1):1–14. Epub 2006/12/28. <https://doi.org/10.2165/00007256-200737010-00001>.
61. Hughes L, Paton B, Rosenblatt B, Gissane C, Patterson SD. Blood flow restriction training in clinical musculoskeletal rehabilitation: a systematic review and meta-analysis. *Br J Sports Med.* 2017;51(13):1003–11. Epub 2017/03/06. <https://doi.org/10.1136/bjsports-2016-097071>.
62. Yow BG, Tennent DJ, Dowd TC, Loenneke JP, Owens JG. Blood flow restriction training after Achilles tendon rupture. *J Foot Ankle Surg.* 2018;57(3):635–8. Epub 2018/02/27. <https://doi.org/10.1053/j.jfas.2017.11.008>.
63. Bittar ST, Pfeiffer PS, Santos HH, Cirilo-Sousa MS. Effects of blood flow restriction exercises on bone metabolism: a systematic review. *Clin Physiol Funct Imaging.* 2018. Epub 2018/03/03; <https://doi.org/10.1111/cpf.12512>.
64. Cleland JA, Mintken PE, McDevitt A, Bieniek ML, Carpenter KJ, Kulp K, Whitman JM. Manual physical therapy and exercise versus supervised home exercise in the management of patients with inversion ankle sprain: a multicenter randomized clinical trial. *J Orthop Sports Phys Ther.* 2013;43(7):443–55. Epub 2013/05/01. <https://doi.org/10.2519/jospt.2013.4792>.
65. Dunn SL, Olmedo ML. Mechanotransduction: relevance to physical therapist practice—understanding our ability to affect genetic expression through mechanical forces. *Phys Ther.* 2016;96(5):712–21. Epub 2015/12/25. <https://doi.org/10.2522/ptj.20150073>.

66. Khan KM, Scott A. Mechanotherapy: how physical therapists' prescription of exercise promotes tissue repair. *Br J Sports Med.* 2009;43(4):247–52. <https://doi.org/10.1136/bjism.2008.054239>. Epub 2009/02/27 PubMed PMID: 19244270; PMCID: PMC2662433
67. Dittmer DK, Teasell R. Complications of immobilization and bed rest. Part 1: musculoskeletal and cardiovascular complications. *Can Fam Physician.* 1993;39:1428–32. 35-7. Epub 1993/06/01. PubMed PMID: 8324411; PMCID: PMC2379624
68. Taylor HL, Henschel A, et al. Effects of bed rest on cardiovascular function and work performance. *J Appl Physiol.* 1949;2(5):223–39. Epub 1949/11/01. <https://doi.org/10.1152/jappl.1949.2.5.223>.
69. Joannis S, Snijders T, Nederveen JP, Parise G. The impact of aerobic exercise on the muscle stem cell response. *Exerc Sport Sci Rev.* 2018;46(3):180–7. Epub 2018/04/18. <https://doi.org/10.1249/JES.000000000000153>.
70. Lisman PJ, de la Motte SJ, Gribbin TC, Jaffin DP, Murphy K, Deuster PA. A systematic review of the association between physical fitness and musculoskeletal injury risk: part 1-cardiorespiratory endurance. *J Strength Cond Res.* 2017;31(6):1744–57. Epub 2017/05/26. <https://doi.org/10.1519/JSC.0000000000001855>.
71. Murphy DF, Connolly DA, Beynnon BD. Risk factors for lower extremity injury: a review of the literature. *Br J Sports Med.* 2003;37(1):13–29. <https://doi.org/10.1136/bjism.37.1.13>. Epub 2003/01/28. PubMed PMID: 12547739; PMCID: PMC1724594
72. Ote Karaca S, Demirsoy N, Gunendi Z. Effects of aerobic exercise on pain sensitivity, heart rate recovery, and health-related quality of life in patients with chronic musculoskeletal pain. *Int J Rehabil Res.* 2017;40(2):164–70. Epub 2017/01/04. <https://doi.org/10.1097/MRR.0000000000000212>.
73. Kunz RI, Coradini JG, Silva LI, Bertolini GR, Brancalhão RM, Ribeiro LF. Effects of immobilization and remobilization on the ankle joint in Wistar rats. *Braz J Med Biol Res.* 2014;47(10):842–9. <https://doi.org/10.1590/1414-431x20143795>. Epub 2014/08/21. PubMed PMID: 25140815; PMCID: PMC4181219



Achilles Tendon Ruptures in Basketball

39

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39.1 Introduction

Basketball is a springing, jumping and sprinting sport with players repetitively jumping to play shots, to make and receive passes, or work sudden sprints together with the start–stop nature of the game. This means that players may be at an increased risk of Achilles tendon injury and have increased difficulty returning from this injury to compete in this sport at the elite level, particularly for senior players [1].

The benefits of exercise are well proven, and a lack of physical activity has been shown to be associated with increased morbidity [2]. Basketball is a well-known community-based sport primarily in North America crossing many cultures.

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39.2 Epidemiology

Achilles tendon ruptures are increasing with a recently quoted incidence of 55 per 100,000. Injuries in the over 60s are increasing most, while those younger than 39 years of age are decreasing slightly [3–5].

There are three acknowledged mechanisms of Achilles tendon ruptures, usually involves an eccentric contraction [6]:

1. Push-off with the ankle going into plantar flexion, with powerful eccentric contraction.
2. Forced dorsiflexion of the plantar flexed foot.
3. Forced unexpected powerful dorsiflexion foot/ankle.

The force on the Achilles tendon is nine times the body weight during running [7]. It must be appreciated that during jumping, the plantar flexors are exposed to double the biomechanical forces as during sprinting [8–10].

The activity in which the rupture was sustained usually reflects local sporting culture, with the majority of patients participating in the most popular sports activities. When Achilles tendon ruptures were studied from a population in a United States, sporting activity was responsible for 68% of the ruptures, of which basketball was the most commonly involved sport accounting for 48% of sports-related ruptures and 32% of all ruptures, respectively [11].

Similar rupture rates have been noted in US military populations (64.9%).

Within the National Basketball Association (NBA), those sustaining rupture are significantly younger than the general population, at mean 28.3 years compared with 45 years, reflective of the age of participation. Players had been playing for an average of 6.8 seasons prior to rupture. Ruptures were most prevalent during early season game play (27.3%) followed by preseason (18.2%) and then late season competition (18.2%) [5, 12].

39.3 The Achilles Tendon Has Been Ruptured, What Are the Management Options?

The aims of management of all patients with Achilles tendon rupture are to return the patient to their required level of physical activity with the least risk of complications of injury and management in an appropriate time period.

The main complication of Achilles tendon rupture is calf muscle weakness, due to tendon elongation and altered muscle function following injury and management. Additional complications of injury and surgical treatment include sural nerve injury, deep venous thrombosis, and although unusual in the sports population, concurrent ankle fracture.

The major treatment decision is whether to choose operative or non-operative management [13]. Since recent studies of operative and nonoperative treatments have showed no difference in re-rupture rates and low re-rupture rates in large nonoperatively managed patient series, there has been a trend to manage the general patient population nonoperatively [14–17]. This led several hospitals to offer only nonoperative treatment for acute ruptures [18]. These studies, however, identified that operatively managed patients were significantly stronger at follow-up [14, 15]. In addition function at high speeds has also been shown to superior in the operatively treated patient [14]. Recent studies continue to show improved

strength and reduced tendon elongation with operative compared with nonoperative repair [19–21].

There has always been the assumption that for high-performance athletes, the best treatment is operative repair following rupture [1]. Initial percutaneous operative techniques reported high rates of iatrogenic sural nerve injury and weaker repairs compared with open repairs, with the benefit, however, of lower wound complication rates [22–25]. Patients undergoing minimally invasive repair were significantly more likely to report satisfactory subjective outcome compared with open surgery. It was also appreciated that current evidence had considerable heterogeneity and a considerable risk of bias [26].

Operative treatment reduces the resultant calf muscle weakness, which can be as much as 30%, tendon elongation, and re-rupture rate. These benefits must be balanced by the risk of complications of operative repair: infection, wound breakdown, iatrogenic sural nerve injury, and adhesions [27]. In a healthy sporting population, infection and wound breakdown are fortunately rare but can occur in the hypovascular area of the tendon. Sural nerve injury may be minimized by modern surgical techniques and if this occurs, it usually results in sensory loss to the lateral side of the foot, with occasional neuralgic pain and neuroma formation [28].

Wound problems may be minimized by the use of mini-invasive and percutaneous techniques [26] although, traditionally associated with tendon lengthening, percutaneous sutures have evolved to locking techniques minimizing this complication [29].

Current advances in techniques aim to ensure secure fixation to the distal stump and/or calcaneus [30], together with locking sutures in the proximal stump [31] (Fig. 39.1). The sutures are placed using percutaneous trans-tendinous passages of a suture needle. This minimizes the risk of iatrogenic sural nerve injury and adhesions formation and optimizes healing by maintaining the integrity of the vascular paratenon and crural fascia [32]. Knots can be avoided by suture anchor

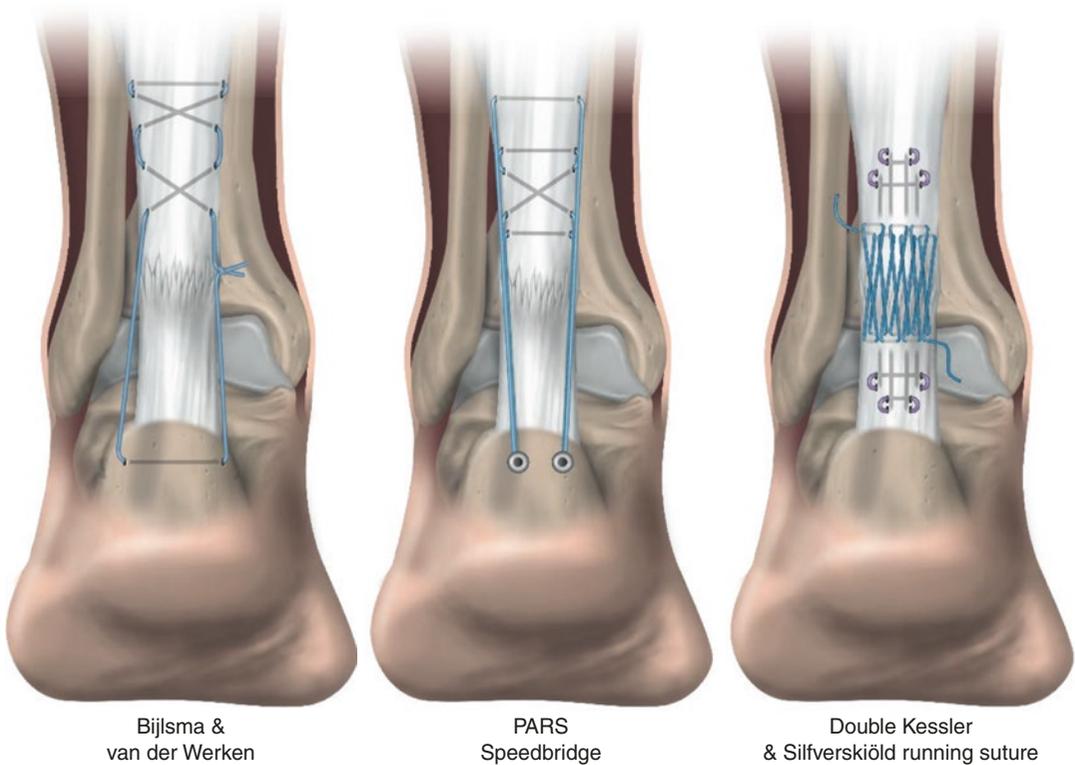


Fig. 39.1 Distal anchorage into the Achilles tendon using the PolyEthylene Ether Ketone (PEEK) anchors permitting deep intra-osseous anchorage, in comparison to the Bijlsma/Van der Werken technique

fixation into the calcaneus. The locking sutures minimize but do not entirely prevent elongation, potentially optimizing the biomechanical properties of the healing tendon [31, 33].

Series using these suture and fixation methods have used only postoperative plaster protection until the wound is healed [30], but using the Bijlsma and van der Werken's technique [34], 3.8% patients suffered from re-ruptures [35].

In addition to operative techniques, nonoperative regimes have also evolved with initial cast protection with early weight-bearing and movement in a supportive brace, in which the ankle is held in a "cast" of polystyrene balls [36]. Following application, the air is extracted by suction, so the balls mold themselves to the ankle. This management method has produced good results in the general population with low re-rupture rates and has also been used in the competitive sporting population.

Fact Box 1

Following Achilles tendon rupture:

Operative treatment reduces the resultant calf muscle weakness, tendon elongation, and re-rupture rate.

Patients undergoing minimally invasive surgery are significantly more likely to report satisfactory outcome.

39.4 Rehabilitation Following Achilles Tendon Rupture

The aim of the rehabilitation process is to restore full weight-bearing with a normal gait pattern, initially walking, and then toward the components of other activities, namely single heel-rise, running, and finally single-leg jumping [37].

The balance between loading the muscle tendon complex and the healing of the rupture site needs to be considered. An imbalance can result in excessive tendon lengthening and may stimulate a thicker tendon. A ruptured Achilles tendon elongates for up to 6 months after surgical repair regardless of early or late weight-bearing in combination with ankle mobilization [38]. Thick tendons do reduce in cross-sectional area from 6 months until reaching a plateau thickness [39].

While there may be pressure to return to sport as soon as possible, perhaps the emphasis should be to return to the best possible level of function and play. Given that rupture may occur at the later stages of an athlete's career, many athletes may only have a few professional seasons remaining and as a consequence good function is required.

Enhanced healing of ruptured tendons with platelet-rich plasma (PRP) treatment in animal models has not been shown in human studies. Case series of patients undergoing this treatment within optimal rehabilitation settings have reported good outcomes, but randomized controlled studies in professional athletes have yet to be performed. Other orthobiologics have been utilized to enhance healing or the use of scaffolds to improve the strength of repair [40].

Rehabilitation following Achilles tendon rupture can be considered in several phases: controlled mobilization, early recovery, late recovery, and return to sport [37]. Despite these distinctions, contributors to return to sport (i.e., strength deficits, psychosocial concerns) should be considered throughout the rehabilitative process [41]. For the later phases of rehabilitation, basketball and jumping sport-specific guidelines have not been published in the context of Achilles tendon rupture; however, guidelines developed for other orthopedic injuries may be modified and applied to this specific population.

Fact Box 2

Rehabilitation following Achilles tendon rupture can be considered in several phases:

Controlled mobilization,
Early recovery,
Late recovery,
Return to sport.

39.4.1 Controlled Motion

The controlled mobilization phase starts directly following injury or surgery. The goal of this phase is to approximate tendon ends and facilitate tendon healing. Of particular concern is the avoidance of tendon elongation. Tendon elongation occurs during the first 8–26 weeks post injury [38, 42, 43] and results in long-term plantar flexor strength deficits [44] as well as changes in biomechanics, particularly with running and jumping [45]. Early weight-bearing has been associated with less risk of tendon elongation [43] although Eliasson et al.'s recent work showed no difference in terms of elongation [38]. Early weight-bearing is performed in plantar flexed positions using weight-bearing casts or boots. Later in this phase, the effects of immobilization can be addressed with joint mobilization techniques to the talocrural and sub-talar joints, taking care not to put the tendon on stretch. Further, general hypotrophy can be addressed with active range of motion and isometric strengthening, avoiding maximum dorsiflexion ranges.

39.4.2 Early Recovery

The early recovery stage begins when the patient is able to ambulate in trainers with an inserted heel wedge, typically around 6–8 weeks. Slow, controlled weight-bearing exercise (such as the bilateral heel-rise) is initiated to gradually load the tendon. Exercises to address balance, range of motion, or strength deficits can also be added at this stage. The goal of this stage is to walk symmetrically without a brace and perform activities of daily living (stairs and walking).

39.4.3 Late Recovery

The late recovery stage starts when the patient is able to perform a unilateral heel-rise with the goal of gradually progressing strengthening and to return to more dynamic activities, such as running. A running progression has been described as being initiated when the patient is able to

complete five unilateral heel-rises at 90% of the available height on the ruptured side. If the patient is unable to achieve this by 16 weeks, running is considered safe to be initiated if the patient was able to raise at least 70% of body-weight during a unilateral heel-rise [37]. Low-speed, low-intensity agility training (i.e., figure-8 jogging) can also be initiated during this phase.

The literature comments upon goals to be attained following Achilles tendon rupture. In Saxena et al.'s study specific targets to be attained are as follows: five sets of 25 repetitions. These are introduced over time starting with three sets of 10 based upon pain tolerance. The calf circumference, measured 10 cm distal to the inferior pole of the patella, should be comparable to the non-injured limb, and the ankle range of motion should aim to be within 5° range of the non-injured side [46]. The difficulties with the use of calf circumference involve the high prevalence of deep venous thrombosis and corresponding calf swelling, which may affect values. Return to play was quoted as being between 12 and 26 weeks following Achilles tendon rupture [46].

Hutchison et al.'s study advised against sports activity in the general population until they could perform at least a single heel raise, sprint using the toe-off phase of gait, perform a horizontal single-leg hop ×3 for more than 75% of the non-injured leg, and lastly perform a vertical hop to greater than 75% of the non-injured side. Return to sports rates were not given in relation to non-operative management [36].

39.5 Return to Play and Performance in Basketball

One of the difficulties of return to play in any sport is the lack of clear definition of return to play (RTP). Ardern et al. have explained that return to play is actually a continuum of sequential progression from a return to participation, a return to sport, and finally a return to performance [41].

Zellers et al. performed a meta-analysis and systematic review of return to play following

Achilles tendon rupture. Overall 108 studies were included, and of these 85 included a measure for determining RTP. The overall rate of RTP in all studies was 80%. Studies with measures describing determination of return to play reported lower rates than studies without specifically described metrics [46].

A recent study by Grassi et al. used a time limit to return to unrestricted practice as the number of days absent from sport. A return to competition is defined as the time from injury to the day of participation for at least 1 min of official match play. A return to the previous level of play was defined as returning to play for at least two seasons after a return to unrestricted training and more than five matches per season in the same division as before the player suffered the index injury [47].

Considering the medical staff, trainers, and facilities available to a professional athlete, a “weekend warrior” should be counseled that even in optimal conditions, players might be unable to return to functional/play after Achilles tendon rupture and those who did so at a decreased level of performance.

Much of the published research on RTP in basketball at the professional level has been obtained from injury reports, press releases, and player profiles. This has the advantage that return to level of play can be determined in real terms, in regard to team selection as well as participation. It must be remembered that studies of individual techniques of management are likely to be heterogenous as many surgeons and therapists are likely to be involved.

One of the disadvantages of this research method is the role of the player participation. The inclusion of a particular player may be very motivational for team dynamics although the injured player may not have actually reached their full recovery of performance.

Performance after a repair of complete Achilles tendon rupture in NBA players ($n = 18$) was assessed over a 23 years period between 1988 and 2011. The average playing experience was 7.6 years. Players who returned missed on average of 55.9 games. The Player Efficiency Rating, determined from the numbers of points made, rebounds, assists, steals, and blocks, etc.,

was reduced by 4.57 ($p = 0.003$) in the first season and by 4.38 ($p = 0.01$) in the second season. When compared with controls, players demonstrated a significant decline in the Player Efficiency Rating during the first season ($p = 0.038$) and second season ($p = 0.081$) after their return. Thirty-nine per cent of players never returned to play [48].

The effect of an Achilles tendon rupture and subsequent repair has been compared with other common procedures in the sports setting [49]. The effect of an orthopedic surgical procedure in the National Basketball Association was studied by comparing ACL reconstruction, Achilles tendon repair, lumbar discectomy, microfracture and meniscus surgery, hand, wrist or foot fracture and shoulder stabilization using archives on public record RTP rate, games played preseason, and player efficiency rating before and after the surgery. Achilles tendon rupture had the lowest incidence of injury, 70.8%, of all the treatments studied, with players having a mean age of 28.4 years. Age ≥ 30 years and a BMI ≥ 27 were predictors of not returning to play. Players undergoing Achilles tendon repair and arthroscopic knee surgery for meniscal and chondral lesions had a significantly greater decline in postoperative performance at the 1- and 3-year time points and had shorter career lengths compared with the other injuries.

Professional athletes from US sports were examined for performance after Achilles tendon repair to compare pre- versus postoperative functional outcomes in respect to performance and return to play. On comparing 25 NBA, 32 NFL, and 5 Major League Baseball (MLB) players, 30.6% of all athletes studied were found to be unable to return to play. Among those who returned to play, game participation averaged 75.4% and 81.9% of the total games played in the season before injury at 1 and 2 years postoperatively. Play time was decreased and athletes performed significantly worse compared with pre-injury levels. When players were compared with matched controls, a player sustaining an Achilles tendon rupture played in fewer games, and had decreased play time and worse performance statistics at 1 year, but not at 2 years postoperatively. When individual sports were compared, NBA players were most

significantly affected [49]. The level of resultant dysfunction clearly has a greater influence on selection and performance depending upon the characteristics of the sport played.

In Lemme et al.'s study, Achilles tendon ruptures in 44 basketball players, with mean age 28.3 years, were identified over a 48-year period, more than one-third of the players (36.8%) either did not return to play or started in fewer than 10 games for the remainder of their career with 21% of injuries leading to retirement [12]. The mean time to return to play was 10.5 months, and the rate of return to play was lower in the NBA (61–71%) compared with that of the NFL players (64–71%) [48]. There was a decreased performance and shortened career length after RTP following Achilles tendon rupture [50].

A systematic review and meta-analysis have been performed to identify return to play (RTP) rates following Achilles tendon rupture and evaluate the measures used to determine RTP. Small cases series and individual case reports were excluded (Table 39.1). A total of 108 studies encompassing 6506 patients were included in this review. Eighty-five studies included a measure for determining RTP. The rate of RTP in all studies was 80% (CI_{95%}: 75–85%). Studies with measures describing determination of RTP reported lower rates than studies without such metrics described, with rates being significantly different between groups ($p < 0.001$). Eighty percent of patients returned to play following Achilles tendon rupture; however, the return to play rates were dependent on the quality of the method used to measure RTP. To further understand RTP after Achilles tendon rupture, a standardized, reliable, and valid method is required [59].

Fact Box 3

The overall return to play rate following Achilles tendon rupture is 80%.

In NBA, 36.8% players did not return to play or started in fewer than 10 games, 21% of ruptures lead to retirement.

The mean time to return to play in the NBA was 10.5 months.

Table 39.1 Outcome in elite athletes and professional sportsmen—in which the specific functional outcome of the athletes had been evaluated

Author year	Number	Mean age	Repair method	Return to play	Notes
Martinelli 2000 [51]	30	30.5	TenoLig®	120–150 days	
Gigante 2008 [52]	40	41	Pc (TenoLig® vs. Bunnell)	No difference between groups	
Parekh 2009 [53]	31	29 NFL	Mixed	36% unable 50% drop performance	
De Carli 2009 [54]	20	39.7	Kakiuchi	76.4% same level	
Maffulli 2011 [55]	17	34	Percutaneous	4.8 ± 0.9 months	11 swelling 4 cramps
Amin 2013 [48]	18	29.7 NBA	Mixed	30% DNR, 11/18 1 season only	
Jallageas 2013 [56]	31	38	Pc vs. open	81% vs. 73.5% same-level play	Overall time 153 days 91–246)
Vadala 2014 [57]	36	29.7	Combined pc/ mini-open	86% within 5 months	
McCullough 2014 [33]	9	25.6 NFL	Pc	78% within 8.9 months	1 athlete 166 days
Byrne 2017 [58]	1	36	Pc	Medal winner 18 weeks	

Psychological aspects are very important during the rehabilitation process and RTS. Information can be used to provide feedback to the player in terms of the ability to cope with specific on-field activities. The sharing of information with athletes can provide consciousness of the ability to RTS and can reduce anxiety and stress and increased their motivation.

With regard to psychological readiness, the “fear of re-injury” hinders return to the pre-injury level. A useful strategy is to adopt Shrier’s Strategic Assessment of Risk and Risk Tolerance (StARRT) Framework. This allows demographic, risk activity and risk tolerance all to be assessed to help determine RTP decisions. Additionally viewing the considerations of the patient, the clinician together with the scientific evidence through an evidence-based practice lens can give additional support and guidance [41].

Take Home Message

Achilles tendon ruptures are common injuries in competitive basketball, with a relatively low rate of return to play. Operative treatment reduces the risk of re-rupture and minimizes calf weakness. For persons playing recreational basketball the majority

will return to play; however, at the top level a third of players will not play at the same level again.

References

1. Caldwell JME, Turner Vosseller J. Maximising return to sports after Achilles tendon rupture in athletes. *Foot Ankle Clin.* 2019;24:439–45.
2. Blair SN. Physical inactivity: the biggest public health problem of the 21st century. *Br J Sports Med.* 2009;43(1):1–2.
3. Huttunen TT, Kannus P, Rolf C, Felländer-Tsai L, Mattila VM. Acute Achilles tendon ruptures: incidence of injury and surgery in Sweden between 2001 and 2012. *Am J Sports Med.* 2014;42(10):2419–23.
4. Lantto I, Heikkinen J, Flinkkilä T, Ohtonen P, Leppilahti J. Epidemiology of Achilles tendon ruptures: increasing incidence over a 33-year period. *Scand J Med Sci Sports.* 2015;25(1):e133–8.
5. Lemme NJ, Li HY, DeFroda SF, Kleiner J, Owens BD. Epidemiology of Achilles tendon ruptures in the United States: athletic and nonathletic injuries from 2011 to 2016. *Orthop J Sports Med.* 2018;6(11):2325967118808238.
6. Arner O, Lindholm A. Subcutaneous rupture of the Achilles tendon; a study of 92 cases. *Acta Chir Scand Suppl.* 1959;116(Supp 239):1–51.
7. Komi PV, Fukashiro S, Jarvinen M. Biomechanical loading of Achilles tendon during normal locomotion. *Clin Sports Med.* 1992;11(3):521–31.

8. Luhtanen P, Komi PV. Mechanical power and segmental contribution to force impulses in long jump take-off. *Eur J Appl Physiol Occup Physiol.* 1979;41:267–74.
9. Mero A, Komi PV. Force-, EMG-, and elasticity-velocity relationships at submaximal, maximal and supramaximal running speeds in sprinters. *Eur J Appl Physiol Occup Physiol.* 1986;55:553–61.
10. Muraki Y, Ae M, Koyama H, Yokozawa T. Joint torque and power of the takeoff leg in the long jump. *Int J Sport Health Sci.* 2008;6:21–32.
11. Raikin SM, Garras DN, Krapchev PV. Achilles tendon injuries in a United States population. *Foot Ankle Int.* 2013;34(4):475–80.
12. Lemme NJ, Li NY, Kleiner JE, Tan S, DeFroda SF, Owens BD. Epidemiology and video analysis of Achilles tendon ruptures in the National Basketball Association. *Am J Sports Med.* 2019;47(10):2360–6.
13. Barfod KW, Nielson F, Nilsson-Helander K, Mattila VM, Tingby O, Boesen A, Troelsen A. Treatment of acute Achilles tendon rupture in Scandinavia does not adhere to evidence-based guidelines: a cross sectional questionnaire based study of 138 departments. *J Foot Ankle Surg.* 2013;52(5):629–33.
14. Willits K, Amendola A, Bryant D, Mohtadi NG, Giffin JR, Fowler P, Kean CO, Kirkley A. Operative versus non-operative treatment of acute Achilles tendon ruptures: a multicenter randomized trial using accelerated functional rehabilitation. *J Bone Joint Surg Am.* 2010;92:767–75.
15. Nilsson-Helander K, Silbernagel KG, Thomeé R, Faxén E, Olsson N, Eriksson BI, Karlsson J. Acute achilles tendon rupture: a randomized controlled study comparing surgical and non-surgical treatments using validated outcomes measures. *Am J Sports Med.* 2010;38(11):2186–93.
16. Soroceanu A, Sidhwa F, Aarabi S, Kaufman A, Glazebrook M. Surgical versus non-surgical treatment of acute Achilles tendon rupture: a meta-analysis of randomized trials. *J Bone Joint Surg Am.* 2012;94(23):2136–43.
17. Ochen Y, Beks RB, van Heill M, Hietbrink F, Leenan LPH, van der Velde D, Heng M, van der Meijden O, Groenwold RHH, Houwert RM. Operative treatment versus nonoperative treatment of Achilles tendon ruptures: a systematic review and meta-analysis. *BMJ.* 2019;364:K5120. <https://doi.org/10.1136/bmj.k5120>.
18. Robertson A, Godavitarne C, Bellringer S, Guryel E, Auld F, Cassidy L, Gibbs J. Standardized virtual fracture clinic management of Achilles tendon ruptures is safe and reproducible. *Foot Ankle Surg.* 2018; <https://doi.org/10.1016/j.fas.2018.10.010>.
19. Olsson N, Silbernagel KG, Eriksson BI, Sansone M, Brorsson A, Nilsson-Helander K, Karlsson J. Stable surgical repair with accelerated rehabilitation versus non-surgical management for acute Achilles tendon ruptures: a randomized controlled study. *Am J Sports Med.* 2013;41(12):2867–76.
20. Heikkinen J, Lantto I, Piilonen J, Flinkkilä T, Ohtonen P, Siira P, Laine V, Niinimäki J, Pajala A, Leppilähti J. Tendon length, calf muscle atrophy and strength deficit after acute Achilles tendon rupture: long-term follow up of patients in a previous study. *J Bone Joint Surg Am.* 2017;99(18):1509–15.
21. Heikkinen J, Lantto I, Flinkkilä T, Ohtonen P, Niinimäki J, Siira P, Laine V, Leppilähti J. Soleus atrophy is common after the nonsurgical treatment of acute Achilles tendon ruptures: a randomized clinical trial comparing surgical and nonsurgical functional treatments. *Am J Sports Med.* 2017;45(6):1395–404.
22. Ma GW, Griffith TG. Percutaneous repair of acute closed ruptured Achilles tendon: a new technique. *Clin Orthop Relat Res.* 1977;128:247–55.
23. Hockenbury RT, Johns JC. A biomechanical in vitro comparison of open versus percutaneous repair of tendon Achilles. *Foot Ankle.* 1990;11(2):67–72.
24. Chan AP, Chan YY, Fong DT, Wong PY, Lam HY, Lo CK, Yung PS, Fung FY, Chan KM. Clinical and biomechanical outcome of minimal invasive and open repair of the Achilles tendon. *Sports Med Arthrosc Rehabil Ther.* 2011;3(1):32.
25. Bradley JP, Tibone JE. Percutaneous and surgical repairs of Achilles tendon ruptures: a comparative study. *Am J Sports Med.* 1990;18(2):188–95.
26. Grassi M, Amendola A, Samuelsson K, Svantesson E, Romagnoli M, Bondi A, Mosca M, Zaffagnini S. Minimally invasive versus open repair for acute Achilles tendon rupture: meta-analysis showing reduced complications with similar outcomes after minimally invasive surgery. *J Bone Joint Surg Am.* 2018;100(22):1969–81.
27. Carmont MR. Achilles tendon rupture: the evaluation and outcome of percutaneous and minimally-invasive repair. *Br J Sports Med.* 2018;52(19):1281–2.
28. Majewski M, Rohrbach M, Czaja S, Oschner P. Avoiding sural nerve injuries during percutaneous Achilles tendon repair. *Am J Sports Med.* 2006;34(5):793–8.
29. Clanton TO, Haytmanek CT, Williams BT, Civitarese DM, Turnbull TL, Massey MB, Wijdicks CA, LaPrade RF. A biomechanical comparison of an open repair and 3 minimally invasive percutaneous techniques during a simulated, progressive rehabilitation protocol. *Am J Sports Med.* 2015;43(8):1957–64.
30. Groetelaers RP, Janssen L, van der Velden J, Wieland AW, Amendt AG, Geelan PH, Janzing HM. Functional treatment of cast immobilization after minimally invasive repair of an acute Achilles tendon rupture: prospective randomized trial. *Foot Ankle Int.* 2014;35(8):771–8.
31. Leicht DJ, Moatshe G, Backus JD, Marchetti DC, Clanon TO. A percutaneous knotless technique for acute Achilles tendon ruptures. *Arthrosc Tech.* 2018;7(2):e171–8.
32. Atinga M, Highland AM, Davies MB. The anatomy of the fascia cruris and implications for Achillon limited open Achilles tendon repair: a case report. *Foot Ankle Int.* 2008;29(8):814–6.
33. McCullough KA, Shaw CM, Anderson RB. Min-open repair of Achilles rupture in the National Football League. *J Orthop Surg Adv.* 2014;23(4):179–83.

34. Bijlsma PS, van der Werken C. Operative treatment of Achilles tendon rupture: minimally invasive technique allowing functional after treatment. *Orthop Traumatol*. 2000;4:285–90.
35. Metz R, van der Heijden GJ, Verleisdonk EJ, Andriks M, van der Werken C. Persistent disability despite sufficient calf muscle strength after re-rupture of surgically treated acute Achilles tendon ruptures. *Foot Ankle Spec*. 2011;4(2):77–81.
36. Hutchison AM, Topliss C, Beard D, Evans RM, Williams P. The treatment of a rupture of the Achilles tendon using a dedicated management programme. *Bone Joint J*. 2015;97-B(4):510–5.
37. Grävare Silbernagel K, Brorsson A, Karlsson J. Rehabilitation following Achilles tendon rupture. In: Karlsson J, Calder J, van Dijk CN, Maffulli N, Thermann H, editors. *Achilles tendon disorders. A comprehensive overview of diagnosis and treatment*. 1st ed. UK: DJO Publications; 2014. p. 151–64.
38. Eliasson P, Agergaard AS, Couppe C, Svensson R, Hoeffner R, Warming S, Warming N, Holm C, Jensen MH, Krogsgaard M, Kjaer M, Magnusson SP. The ruptured Achilles tendon elongates for 6 months after surgical repair regardless of early or late weight-bearing in combination with ankle mobilization: a randomized clinical trial. *Am J Sports Med*. 2018;46(10):2492–502.
39. Schepull T, Kvist J, Aspenberg P. Early E modulus of healing Achilles tendons correlates with late function: similar results with or without surgery. *Scand J Med Sci Sports*. 2012;22(1):18–23.
40. Indino C, D'Ambrosi R, Uselli FG. Biologics in the treatment of Achilles tendon pathologies. *Foot Ankle Clin*. 2019;24(3):471–93.
41. Ardern CL, Glasgow P, Schneiders A, Witvrouw E, Clarsen B, Cools A, Gojanovic B, Griffin S, Khan KM, Moksnes H, Mutch SA, Phillips N, Reurink G, Sadler R, Silbernagel KG, Thorburg K, Wangenstein A, Wilk KE, Bizzini M. 2016 consensus statement on return to sport from the First World Congress in Physical Therapy, Bern. *Br J Sports Med*. 2016;50(14):853–64.
42. Mortensen H, Skov O, Jensen P. Early motion of Achilles after operative treatment of a rupture of the Achilles tendon: a prospective randomized clinical and radiographic study. *J Bone Joint Surg Am*. 1999;81(7):983–90.
43. Kangas J, Pajala A, Ohtonen P, Leppilahti J. Achilles tendon elongation after rupture repair: a randomized comparison of two post operative regimens. *Am J Sports Med*. 2007;35(1):59–64.
44. Grävare Silbernagel KG, Steele R, Manal K. Deficits in heel-rise height and Achilles tendon elongation occur in patients recovering from an Achilles tendon rupture. *Am J Sports Med*. 2012;40(7):1564–71.
45. Brorsson A, Willy RW, Tranberg R, Grävare Silbernagel K. Heel-rise height deficit 1 year after Achilles tendon ruptures relates to changes in ankle biomechanics 6 years after injury. *Am J Sports Med*. 2017;45(13):3060–8.
46. Saxena A, Ewan B, Maffulli N. Rehabilitation of the operated Achilles tendon: parameters for predicting return to activity. *J Foot Ankle Surg*. 2011;50(1):37–40.
47. Grassi A, Rossi G, D'Hooghe P, Aujla R, Mosca M, Smauelsson K, Zaffagnini S. Eighty two per cent of male professional football (soccer) players return to play at the previous level two seasons after Achilles tendon rupture treated with surgical repair. *Br J Sports Med*. 2019; <https://doi.org/10.1136/bjsports-2019-100556>.
48. Amin NH, Old AB, Tabb LP, Garg R, Toossi N, Cernyik DL. Performance outcomes after repair of complete Achilles tendon ruptures in national basketball association players. *Am J Sports Med*. 2013;41(8):1864–8.
49. Trofa DP, Miller JC, Jang ES, Woode DR, Greisberg JK, Vosseller JT. Professional athlete's return to play and performance after operative repair of an Achilles tendon rupture. *Am J Sports Med*. 2017;45(12):2864–71.
50. Minhas SV, Kester BS, Larkin KE, Hsu WK. The effect of an orthopaedic surgical procedure in the National Basketball Association. *Am J Sports Med*. 2016;44(4):1056–61.
51. Martinelli B. Percutaneous repair of the Achilles tendon in athletes. *Bull Hosp Jt Dis*. 2000;59(3):149–52.
52. Gigante A, Moschini A, Verdenelli A, Del Torto M, Ulisse S, de Palma L. Open versus percutaneous repair in the treatment of acute Achilles tendon rupture: a randomized prospective study. *Knee Surg Sports Traumatol Arthrosc*. 2008;16(2):204–9.
53. Parekh SG, Wray WH 3rd, Brimmo O, Sennett BJ, Wapner KL. Epidemiology and outcomes of Achilles tendon ruptures in the National Football League. *Foot Ankle Spec*. 2009;2(6):283–6.
54. De Carli A, Vadalà A, Ciardini R, Iorio R, Ferretti A. Spontaneous Achilles tendon ruptures treated with a mini-open technique: clinical and functional evaluation. *J Sports Med Phys Fitness*. 2009;49(3):292–6.
55. Maffulli N, Longo UG, Maffulli GD, Khanna A, Denaro V. Achilles tendon ruptures in elite athletes. *Foot Ankle Int*. 2011;32(1):9–15.
56. Jallageas R, Bordes J, Daviet JC, Mabit C, Coste C. Evaluation of surgical treatment for ruptured Achilles tendon in 31 athletes. *Orthop Traumatol Surg Res*. 2013;99(5):577–84.
57. Vadalà A, Lanzetti RM, Ciompi A, Rossi C, Lupariello D, Ferretti A. Functional evaluation of professional athletes treated with a mini-open technique for Achilles tendon rupture. *Muscles Ligaments Tendons J*. 2014;4(2):177–81.
58. Byrne PA, Hopper GP, Wilson WT, Mackay GM. Knotless repair of Achilles tendon rupture in an elite athlete: return to competition in 18 weeks. *J Foot Ankle Surg*. 2017;56(1):121–4.
59. Zellers J, Carmont MR, Grävare Silbernagel K. Return to play post Achilles tendon rupture: a systematic review and meta-analysis of rate and measures of return to play. *Br J Sports Med*. 2016;50(21):1325–32.



Management of Common Tendinopathies in Basketball

40

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40.1 Introduction

Tendinopathies are a common cause of disability in the basketball player and account for a substantial proportion of overuse injuries in the sport. The term “tendinopathy” is defined as a clinical syndrome characterized by a combination of activity-related pain, diffuse or localized swelling, and impaired performance [1–3]. Often a sequelae of overuse phenomenon, tendinopathy may less commonly occur as a consequence of medical condition. Midportion and insertional tendinopathy (enthesopathy) should be distinguished as unique clinical entities.

Tendinosis and peritendinitis are two common phenomena that are integral to understanding tendinopathy. Tendinosis entails histopathological disorganization and collagen fiber separation which leads to haphazard proliferation of tenocytes, increase in mucoid noncollagenous matrix, and nerve and vessel ingrowth without evidence of intratendinous inflammation [3, 4].

Peritendinitis is an acute or chronic inflammation of the paratenon which can be induced by repetitive loading activities and is characterized

by local inflammatory infiltration. The insertion site to bone and surrounding bursal structures are commonly susceptible to inflammation as a consequence of their greater density of blood vessels and nerves. The tissue of the tendon proper is primarily aneural and avascular and does not primary exhibit classical inflammatory responses [1, 3, 4].

Tendinopathic tendons often have an increased rate of matrix remodeling, leading to a mechanically less stable tendon that is likely more susceptible to damage [4]. Histologic studies of surgical tendon specimens consistently demonstrate absent or minimal inflammation, but there is a loss of tightly bundled collagen architecture, increase in proteoglycan content, and neovascularization [5]. The inflammatory cascade is thought to play a major role in the initiation of the disease process [6].

The tendons most vulnerable to overuse in the jumping athlete entail the Achilles and patellar tendons, which will be the focus of this chapter.

40.2 Epidemiology

Basketball is a very dynamic sport that requires a combination of agility, speed, technical skill, and strength. As a consequence of many repetitive maneuvers such as jumping and pivoting, the incidence of tendinopathy is quite prevalent. Due to increased participation in recreational sports, the total exposure of hours to sport practice and competition, a known risk factor for tendinopathy, has

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been on the rise. In addition to basketball (12%), patellar tendinopathy is common in volleyball (14%), track and field (7%), and soccer (2.5%) [7].

Although physical activity is the primary source of tendinopathy, it is important to consider medical and pharmacologic causes of tendinopathy. Perturbation of glucose metabolism and atherosclerosis has been identified as underlying factors in tendinopathy [1, 8]. Obesity, hypertension, diabetes mellitus, hypercholesterolemia, and metabolic disease contribute to the incidence of tendinopathy. Inflammatory arthropathies, such as rheumatoid arthritis, contribute to connective tissue healing malfunction and homeostasis [9].

Medication therapy with statins [10] and fluoroquinolone antibiotics [11] can increase the risk for tendinopathy and should be avoided. The use of immunosuppressive corticosteroids, particularly intratendinous injection, can have detrimental effects on tendon metabolism and should be used with caution [1].

40.3 Pathophysiology (Fact Box 1)

The mechanical loading of tendon tissue leads to the upregulation of collagen gene expression and increased synthesis of collagen proteins. This anabolic process has been observed to peak

around 24 h after exercise and remains elevated for up to 80 h [12, 13]. During the first 24–36 h after exercise, there is a net loss of collagen due to degradation of collagen proteins [3]. The interplay between anabolic and catabolic processes of collagen merits a restitution time interval in between exercise to mitigate tendon breakdown.

Repetitive mechanical loading leads to microruptures of collagen fibrils and the production of inflammatory molecules by tenocytes, this is evidenced by increased levels of proinflammatory cytokines [14]. Findings of inflammatory infiltrate consisting of macrophages, mast cells, and lymphocytes suggest a role for the intrinsic immune system in mediating early tendinopathy. Tenocytes and fibroblasts can transform into myofibroblasts under repetitive stress and a proinflammatory environment. Myofibroblasts facilitate tendon healing and are triggered to undergo apoptosis after the mechanical stresses abate. Although, under constant stress that does not permit this apoptosis or tissue hypoxia, myofibroblasts continue to propagate resulting in fibrosis, a feature of chronic tendinopathy [15].

Another feature of tendinopathy is microvascular angiogenesis into the tendon; this is consequence of hypoxia-induced upregulation of vascular endothelial growth factor (VEGF) [16]. This neovascularization has been ascribed as a pain generator as sensory nerve fibers can grow adjacent to the blood vessels and lead to the release of nociceptive substances, including substance P [17]. Sensory nerve ingrowth is observed in repetitive loading as well as response to injury; in non-pathologic tendon, the repair process involves autonomic nerve ingrowth and ultimate decreased nociception with subsequent nerve retraction, whereas in tendinopathy, the ingrowth nerves do not retract leading to increased pain signaling and hyperproliferative changes. Substance P has a role in potentiating tendinopathy with proinflammatory and trophic actions leading to vasodilation, plasma extravasation, and the stimulation of TGF- β in fibroblasts [1].

Fact Box 1 Pathophysiology of Tendinopathy

- Mechanical loading of tendons facilitates protein synthesis and degradation of collagen at the cellular level.
- Adequate tendon unloading (~24 h) after exercise is helpful in preventing net loss of collagen that leaves the tendon vulnerable to injury.
- Tendinopathy is associated with neovascularization and ingrowth of nerves that serve as pain generators.
- Chronic inflammation of a tendon can lead to increased pain signaling and hyperproliferation and fibrosis, which may ultimately become an irreversible process.

40.4 Patellar Tendinopathy

40.4.1 Etiology

Patellar tendinopathy, or “jumper’s knee,” is highly prevalent in sports with a high demand for speed and power, such as basketball, volleyball, and soccer. The hallmark of patellar tendinopathy is progressive patellar tendon pain that leads to recurrent or long-standing impairment of athletic performance [17]. Overall prevalence of patellar tendinopathy has been reported across multiple sports by Lian et al. [18] as 14%. Estimated prevalence of current or prior patellar tendinopathy was 55% in professional basketball players with a mean duration of symptoms of 32 ± 25 months, suggesting that most athletes participate in their sport with active symptoms. The condition affects men approximately twice as frequently as women. Basketball often leads to maximal muscle forces generated eccentrically that are 1.5–2.0 times higher than the maximal isometric forces. Additionally, ground reactive forces can be 10 times body weight in a long jump takeoff [19, 18], explicating the connection between the loading pattern of the knee extensors and jumper’s knee. Cook et al. [20] evaluated patellar tendons of a cohort of elite junior basketball players using ultrasonography and demonstrated that 29% of the tendons had hypoechoic regions with intra and peritendinous changes of collagen disorganization although not all findings correlated with clinical symptomatology.

Quadriceps tendinopathy, a less common entity, is important to consider as part of the extensor mechanism spectrum. The prevalence is estimated to range from 0.2% to 2% in athletic patient populations. This entails approximately one in four patients with extensor mechanism pain who report pain localized to the superior pole of the patella with symptoms more pronounced in deep flexion [18, 21]. Although the patellar and quadriceps tendon work in concert as part of the extensor mechanism of the knee, they differ anatomically as the quadriceps tendon connects muscle to bone while the patellar tendon connects bone to bone.

The quadriceps tendon is comprised of a coalescence of four muscle tendons that may variably contribute to the ultimate insertion onto the superior pole of the patella. Notably, this results in a transition of stiffness that is variable between the quadriceps and patellar tendons [22, 23]. The focus of this chapter is on the more common patellar tendinopathy in the jumping athlete although it is important to consider quadriceps tendon involvement and acknowledge that a rehabilitation protocol taking into account similar principles but localization to the quadriceps tendon is merited [21].

Ferretti et al. have demonstrated a linear relationship between training volume and the prevalence of jumper’s knee and with the firmness of the floor type for training [19]. Additional extrinsic contributors to patellar tendinopathy include inappropriate training equipment, excessive loading, or repetitive high-intensity loading [24]. Patellar tendinopathy most frequently affects the inferior pole of the patella in the deeper portion of the tendon; this zone is subjected to the highest tensile forces during impact activity [25]. Patellar tendinopathy may affect the midportion zone or the insertion at the tibial tuberosity and often irritation of the infrapatellar bursa at the distal insertion of the patellar tendon coexists. Additionally, inflammation and pathology of the Hoffa fat pad may contribute to patient symptoms of tendinopathy and merit consideration in the therapeutic protocol [26, 27].

Anthropometric factors such as high body mass index (BMI), large abdominal circumference, limb-length discrepancy, and flatfoot arch are independent risk factors for the development of patellar tendinopathy [28, 29]. Weakness of the quadriceps muscles and low flexibility of the quadriceps and hamstrings are associated with patellar tendinopathy. Intrinsic factors including patellar malalignment, patella alta, abnormal patellar laxity, muscular tightness, and imbalance have been proposed as risk factors although it is their complex interplay with the aforementioned extrinsic factors that result in tendinopathy [30]. The classification systems of Jumper’s knee are detailed in Table 40.1.

Table 40.1 The different classifications for “Jumper’s knee” [19, 92–94]

Stage/ grade	Blazina classification	Ferretti classification	Roels classification
0		No pain	
I	Pain only following activity without functional impairment	Pain only following intense sports activity with no functional impairment	Pain at the infrapatellar or suprapatellar region following training or event
II	Pain during and following activity with satisfactory performance levels	Moderate pain during sports activity with no sports performance restriction	Pain at the beginning of activity, disappearing after warm-up and reappearing after activity completion
III	More prolonged pain during and following activity with progressively increasing difficulty performing at a satisfactory level	Pain with slight sports performance restriction	Pain during and after activity. The patient is unable to participate in sports
IV		Pain with severe sports performance restriction	Complete rupture of the tendon
V		Pain during daily activity and inability to participate in sport at any level	

40.4.2 Diagnosis

Patellar tendinopathy presents with activity-related anterior knee pain typically localized to the distal pole of the patella or proximal patellar tendon. Pain is insidious and can be precipitated by an increase in sports activity. The pain may initially commence with activity but can progress to being present even at rest. Physical examination yields tenderness to palpation at the distal pole of the patella with the knee in terminal extension; with pain reduced with knee flexion. Load-related pain that increased demand on the knee extensors, such as the single-leg decline squat maneuver to approximately 30° of flexion reproduces the symptoms of patellar tendinopathy [31]. Weakness of the quadriceps and a lack of hamstring flexibility may be observed as well. Pain may improve with repeated loading (the “warm-up” phenomenon), but there is often increased pain the day after energy-storage activities of the patellar tendon. This dose-dependent pain is a key feature, demonstrating that the patient’s pain increases as the magnitude and rate of load application to the tendon increases [25, 32, 33]. Comprehensive evaluation necessitates a thorough examination of the entire lower extremity is necessary to identify relevant deficits at the hip, knee, or ankle/foot region.

Patellar tendinopathy is a primary clinical diagnosis that does not require confirmatory imaging although imaging can be helpful for inclusion/exclusion of potential alternative diagnoses in a convoluted clinical picture. In addition to the inferior pole of the patella, tendinopathy of the extension mechanism of the knee can involve the quadriceps tendon or the distal insertion of the patellar tendon at the tibial tuberosity [25]. Preferred imaging modalities are ultrasonography and MRI. Ultrasonography can locate intratendinous lesions evidenced by hypoechogenicity that may be adjacent to the inferior pole of the patella. Thickening of the tendon, intratendinous calcifications, and erosions in the inferior pole of the patella may be observed. Ultrasonography has a sensitivity of 58% and specificity of 94% for diagnosing patellar tendinopathy [31]. MRI typically demonstrates hyperintensity of the proximal patella tendon with observed tendon thickening as with ultrasonography. MRI provided the added benefit of assessing intra-articular pathology over ultrasonography. The sensitivity and specificity are 78% and 86% for MRI to diagnose patellar tendinopathy, respectively. In the high-level basketball player, MRI should be the first-line imaging option due to the ability to rule out additional pathologies. It is important to note that each imaging modality guides the clinical based on a diag-

nosis of patellar tendinopathy that is diagnosed based on history and physical examination. The main differential diagnosis to be distinguished is patellofemoral pain syndrome and fat-pad syndrome which can often be done on the basis of clinical examination.

40.4.3 Management

40.4.3.1 Nonoperative Treatment (Fact Box 2)

Eccentric Exercises

The mainstay of management of patellar tendinopathy is nonoperative management and the support of high-level evidence [34]. Eccentric

exercises lead the musculoskeletal unit to adapt to the stresses by remodeling the collagen fibers in the patellar tendon. This is likely a consequence of the tendon stretching more during eccentric loading as opposed to concentric loading, resulting in more mechanotransduction of collagen fibers leading to greater number of blood vessels and a larger quantity of collagen ultimately resulting in improved tendon healing [35–37].

Universal positive effects are noted without adverse effects [38]. There are multiple types of eccentric loading exercise programs, and in a systematic review Visnes and Bahr [22] concluded that a particular modality is not isolated. A treatment program should include a decline board and should be performed with a mild level of discomfort for the athlete. Assessing pain irritability is a fundamental part of configuring a training regimen with regard to determining the duration of symptoms aggravation following energy-storage activities. It is suggested that up to 24 h of pain provocation after a training session is acceptable during rehabilitation [25, 32]. A minimum of 20 training sessions, performed at a 15-repetition maximum [39], appears to be necessary to ensure effective therapy with a program duration of 6 weeks to 1 year depending on the severity to reduced symptoms of tendinopathy [35, 40].

It is important to recognize that eccentric exercise for the treatment of patellar tendinopathy may be too aggressive for patients that are highly symptomatic, particularly during the basketball season. As a consequence, rehabilitation should progress in a stepwise fashion. The first phase of this should entail load modification to reduce pain and avoid aggravation of symptoms. Following this, we recommend a gradual progression of rehabilitation from isometric loading, to isotonic loading, to ultimate eccentric loading with a culmination of return to sport-specific training activities [25]. It is important to acknowledge that progression of rehabilitation, particularly with the demands of returning to elite-level competition, may require 6 months or longer.

Fact Box 2 Nonoperative Management Modalities for Tendinopathy

- Eccentric exercises lead to tendon stretching and mechanotransduction of collagen fibers leading to increased vascular inflow and ultimately improved tendon healing.
- Extracorporeal shock wave therapy (ECWT) improves tendon healing by hyperstimulation analgesia to reduce pain signal transmission and the production of growth factors via mechanical stimulation.
- Ultrasound-guided galvanic electrolysis technique (USGET) may treat tendinopathy by electrol destruction of degenerative tissue and facilitating a controlled, localized inflammatory healing response.
- Platelet-rich plasma (PRP) injections have shown clinical improvement in tendinopathy due to improved cellular remodeling. Multiple injections offer an improvement over single injection.
- Corticosteroid injections should be used with caution for the management of tendinopathy.

Extracorporeal Shock Wave Therapy

Extracorporeal shoulder wave therapy (ESWT) is gaining vogue as a safe and promising treatment for patellar tendinopathy. The basis of utility of ESWT is based on the theory that pain relief is achieved by hyperstimulation analgesia, and overstimulation of the painful area leads to a diminished transmission of pain signals to the brain stem. Additionally, that the mechanical load developed by ESWT stimulates tissue regeneration and destroys calcifications via a mechanical disintegration [41, 42]. Mechanical stimulation of the tendon increases the expression of growth factors such as insulin-like growth factor 1 (IGF-1), transforming growth factor β 1 (TGF- β), and interleukin [43]. A 2017 randomized controlled trial comparing ESWT to conservative treatment (physiotherapy, exercise program, non-steroidal anti-inflammatory drugs (NSAIDs), and knee strap) provided some valuable insight to patellar tendinopathy management. Patients did not require local or regional anesthesia and were treated with 1500 impulses of shockwave at 14 kV (equivalent to 0.18 mK/mm² energy flux density) at the point of maximal tenderness. ESWT demonstrated superior pain relief, Victorian Institute of Sport Assessment (VISA) scores, and sonographic vascularity at 2–3 year follow-up [44]. Additionally, recurrence of patellar tendinopathy symptoms occurred in 13% of the ESWT group and 50% of the conservative group ($p = 0.014$). Ultrasonography showed a significant increase in the vascularity of the patellar tendon and a trend of reduction in the patellar tendon thickness with ESWT without any risk of local to systemic complications. The particulars of ESWT use, including energy level, number of treatments, and impulses, are varied among the literature.

Ultrasound-Guided Galvanic Electrolysis Technique

Ultrasound-guided galvanic electrolysis technique (USGET) is a novel therapeutic option that has recently been evaluated in a randomized controlled trial by Abat et al. [45]. They have demonstrated the efficacy and superiority of USGET compared to conventional electrophysiotherapy

when performed with eccentric exercise for patellar tendinopathy management. USGET is a minimally invasive technique that entails treatment at 2-week intervals applying a galvanic current to the area of pathology with a 0.25 × 25 mm stainless steel acupuncture needle. This technique involves puncturing the superficial paratenon, deep paratenon, and intratendinous areas at the inferior pole of the patella with electrical current of 2 milliamps. The authors suggest that this technique is capable of minimally invasively destroying degenerative tissue and facilitating an appropriate localized inflammatory response to trigger biological collagen repair [46]. This technique merits strong consideration as a therapeutic modality for patellar tendinopathy that obviates the need for operative intervention.

Injection Therapy

Corticosteroid injections are not recommended for the treatment of patellar tendinopathy. Multiple randomized controlled trials (RCTs) have shown limited long-term relief and an increase in potential patellar tendon rupture with the use of corticosteroid (triamcinolone and methylprednisolone) injections [47–49]. Corticosteroid injections may normalize lesions in patellar tendons on ultrasonography and may have pronounced clinical effects over a short duration but do not demonstrate sustained benefits beyond 6 months.

Platelet-rich plasma (PRP) is a promising therapy for enhancing tissue repair due to delivery of platelet-derived growth factors and bioactive molecules in hyperphysiologic doses [50]. A 2019 systematic review and meta-analysis reviewed 15 articles describing the use of PRP with good overall results in terms of clinical improvement, return to sports, and pain relief at short-term and long-term follow-up [51]. In vitro and in vivo studies of PRP demonstrate benefits that include improved cellular remodeling and decreased healing time [50, 52–54]. The details of optimal preparation and administration of PRP remain unclear in the literature. Although, there is now evidence to support multiple injections of PRP to yield a significant benefit at long-term

follow-up [55, 56]. Single infiltration of PRP might provide short-term relief but not optimal long-term results. Notably, one study comparing single injection to multiple injections of PRP found no benefit for successive injections [57]. As a consequence, although further investigation is pending—in the case of advanced patellar tendinitis we recommend 2–3 consecutive infiltrations of leukocyte-rich PRP with 5 mL delivered at an average spacing of 10–15 days. It is advised to combine PRP treatment with rehabilitation due to the synergistic effect in recovery.

Cell-Based Therapies

Stem cell-based therapies theoretically could improve the speed and quality of the healing process with the aid of autocrine and paracrine factors to enhance remodeling. This research remains in the preliminary phases including case studies and animal models. This has been evaluated with mononuclear bone marrow cells at 5-year follow-up [58] and skin-derived tenocyte-like cells [59] with initial promising results. Ultimately, further information is needed to indicate their role in patient management.

Hyaluronic Acid

High molecular weight hyaluronic acid (HA) has been hypothesized to have an anti-inflammatory effect as well as potential tendon healing impact at the bone-tendon interface [60]. A mean of two injections of hyaluronic acid has been shown to improve recovery at a mean follow-up of 25.7 months, reporting 54% excellent results and 40% good results. High-level evidence is still lacking to determine the efficacy of HA.

Additional Treatments

A variety of agents have been and are currently being investigated to aid in the management of patellar tendinopathy. Sclerosing agents inhibit blood vessel formation and counteract accompanying vasa nervorum and can have an impact on the known neovascularization that occurs in the pathophysiology of patellar tendinitis. Level IV evidence shows that ultrasound-guided injection of 5 mg/mL polidocanol to the paratenon can improve pain during activity

[61]. There are multiple studies suggesting improvement over placebo [62–64] although the longevity of impact and overall usefulness remain unclear.

Injection of matrix metalloproteinase inhibitor aprotinin has been evaluated in a retrospective case series of 97 patients [65] which demonstrated differential degrees of improvement at approximately 1 year of follow-up. Further clinical data is desired in this treatment regimen.

Glyceryl trinitrate delivers nitric oxide locally and has been shown to play a role in fibroblast proliferation, collagen synthesis, and macrophage angiogenesis. Glyceryl trinitrate can be delivered via a topical patch and has shown promising early data with regard to positive effects on tendinopathy [66, 67].

40.4.3.2 Operative Treatment (Fact Box 3)

Nonsurgical treatment of tendinopathy is successful in approximately 90% of patients. Those who are refractory may ultimately respond to surgical intervention [68]. The objective of surgical intervention is tenotomy of the patellar tendon, excision of pathologic tissue, and induction of the repair process via stimulating the inferior pole of the patella. This can be done with open surgery or arthroscopic intervention.

Open approach entails a midline longitudinal incision from the inferior pole of the patella to the tibial tubercle; the paratenon is incised to expose the patellar tendon. Through a longitudinal incision in the tendon, the posterior degenerative tissue is debrided and the distal 5 mm of the patella are resected to ensure a bleeding surface for healing (Fig. 40.1). The tendon and paratenon are closed with resorbable sutures; a suture anchor may be placed into the patella if needed. Alternatively, a technique using a Beath pin to secure the tendon to the patella [69].

Arthroscopic intervention begins with diagnostic knee arthroscopy to evaluate the patella for chondral lesions. The inferior pole of the patella is identified, and the adjacent synovial tissue is resected, exposing the degenerative tissue in the

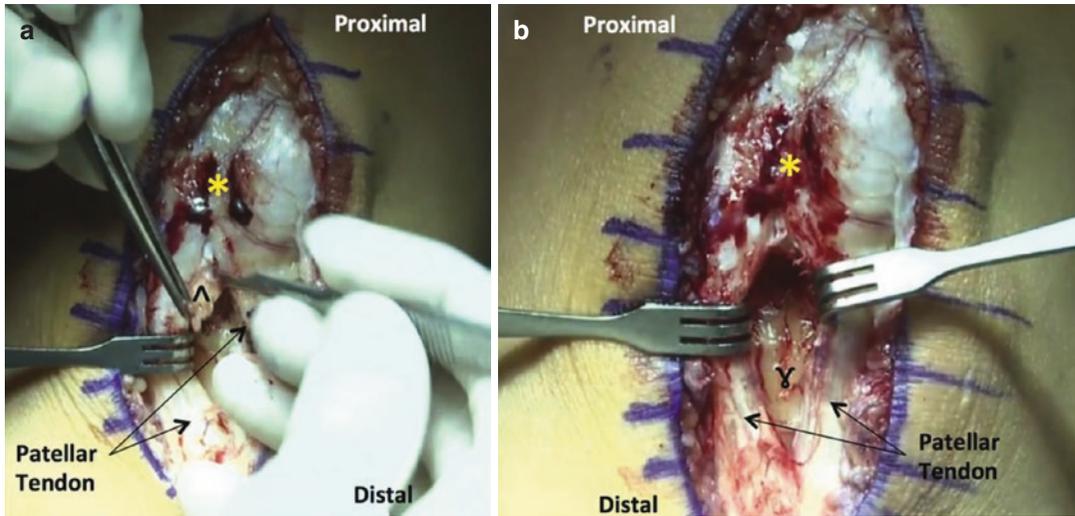


Fig. 40.1 Midline approach to patellar tendon demonstrated with posterior degenerative portion exposed and identified by abnormal color and texture of the tissue. (a) The tissue is excised and carefully debrided with a #15

blade, asterisk denotes the patella. (b) Post excision the infrapatellar fat pad is visualized by the gamma sign (Borrowed from Kruckeberg et al. *Arthroscopy Techniques* 2017)

posterior zone of the proximal patellar tendon, which can be debrided until healthy tissue is identified. The distal pole of the patella is resected of 5 mm using a burr.

Each of these techniques can be augmented with leukocyte-rich PRP injection at the conclusion of the procedure. Postoperative rehabilitation should not restrict weight-bearing or range of motion. After wound healing at approximately 10 days postoperatively, patients should commence initiation of eccentric squats on an inclined board and strengthening. Typical return to sport is 3 months postoperatively when patients can demonstrate functional recovery without pain during strengthening and exercise [28].

40.5 Achilles Tendinopathy

40.5.1 Etiology

The Achilles tendon (AT) structure plays a crucial part in basketball-specific movements, allowing explosive movements by storing and releasing elastic energy. Risk factors associated with Achilles injuries include advancing age, changes in player performance character-

istics, altered foot and ankle biomechanics, gastrocnemius-soleus dysfunction, and anatomic variations [63, 64, 70]. Achilles tendinopathy can be attributed to excessive loading of the tendon during vigorous physical training that is exacerbated by an imbalance between muscle power and tendon elasticity. In particular the strength of the ankle plantar flexors and the amount of dorsiflexion excursion predict an Achilles tendon overuse injury [71]. Lateral ankle instability leads to excessive motion of the hindfoot with resultant excessive compensatory pronation causing a “whipping action” on the AT, predisposing tendinopathy. Forefoot varus is additionally implicated in patients with Achilles tendinopathy. It has been hypothesized that the plantaris tendon may contribute to midportion tendinopathy or chronic paratenon or AT [27]. Extrinsic factors that predispose athletes include changes in training pattern, poor technique, previous injuries, footwear, and environmental factors such as hard, slippery, or slanting training surfaces [72]. Players with Achilles tendinopathy have a better change to return if they are younger in age and early in their professional career [73, 74].

40.5.2 Diagnosis

Achilles tendinopathy can be categorized into two distinct categories as insertional and non-insertional. Non-insertional is localized to the midportion which typically involves an area of 2–7 cm from the distal insertion; it may be localized to the main body of the tendon or to the paratenon. The patient typically presents with activity-limiting pain and chronic swelling of the AT. The diagnosis is mainly based on history and clinical examination. Pain is often a late symptom, with common symptoms of morning stiffness or stiffness after a period of inactivity and a gradual onset of pain during activity. Athletes often experience pain at the beginning and end of training sessions, with a period of diminished discomfort during activity. On physical examination, the both legs should be evaluated simultaneously and is best with the patient standing and prone. Tendinopathy of the main body of the AT involves tenderness, likely thickening/nodularity, and swelling that is decreased or relieved when the tendon is put under tension. Paratendonitis of the tendon is similar in the involvement of the central 1/3 although the swelling and tenderness remain fixed in relation to the malleoli from full dorsi- to plantarflexion. Insertional tendinopathy or Achilles enthesopathy is characterized by pain, swelling, and tenderness of the posterior aspect of the calcaneus that is aggravated by active to passive motion [72, 75].

From an imaging standpoint, X-ray radiography may demonstrate deviation of the soft tissue contours. Insertional Achilles tendinopathy at the calcaneus can result in calcifications and enthesophyte development. Ultrasonography (US) correlates the histopathologic findings very well and offers advantages over the other imaging modalities. Ultrasonography can quantify tendon structure and may demonstrate an enlarged tendon with or without fibrillation or hypoechoic areas. Additionally, power Doppler US can detect neovascularization and can serve as a reliable tool to diagnose and follow Achilles tendinopathy. In the setting that US remains inconclusive, magnetic resonance imaging (MRI) of the tendon can be used and



Fig. 40.2 MRI demonstrating tendinopathy of the main body of the Achilles tendon, localized thickening without involvement of the paratenon (*Borrowed from Maffulli et al 2019. Foot and Ankle Surgery*)

will further characterize the internal morphology of the tendon and surrounding tissues. MRI also clearly delineates paratendinopathy and tendinopathy of the main body of the tendon (Fig. 40.2). If considering surgical intervention, MRI may be valuable in pre-operative planning although the high sensitivity of MRI should be interpreted with caution and correlated directly to patient symptoms [5].

40.5.2.1 Nonoperative Treatment (Fact Box 2)

The management of Achilles tendinopathy lacks evidence-based guidance and long-term morbidity is unpredictable. In a prospective observational study of patients with AT, 29% of patients required surgical intervention during an 8-year follow-up period [76]. Nonoperative management should be implemented for a minimum of 3–6 months prior to surgical consideration [77].

There is a distinct overlap in the nonoperative management of patellar and Achilles tendinopathy. This segment of the chapter is abbreviated as the same principles are in play as previously described for the management of patellar tendinopathy. Anti-inflammatory medications demonstrate a modest improvement in symptoms in the

short term as well as facilitating eccentric strengthening exercises of the gastrocnemius and soleus. There is the caveat of the analgesic effect potentially allowing patient to ignore early symptoms and imposing further damage on the affected tendon. Stretching of the gastrocnemius/soleus complex is essential in these patients as many jumping athletes are predisposed to gastrocnemius contracture, as this can often exacerbate Achilles tendinopathy [77].

Rest and eccentric exercises are the mainstay of therapy, and multiple studies have demonstrated 60%–80% of patients safely continuing with their preferred activity [78–80]. The combination of eccentric exercises with extracorporeal shockwave therapy has been shown to produce a higher success rate as compared to each modality in isolation [81].

Injections of PRP are preferred in succession as opposed to single injections for patellar tendinopathy and other tendinopathies (rotator cuff, lateral epicondylitis) [82]; this principle may be applied to the Achilles with the caveat that recent literature suggests equivocal efficacy in the management of chronic Achilles tendinopathy with PRP as compared to saline injection [83]. Further research with a large, multicenter, blinded, randomized controlled trial with a robust study population and long-term follow-up is needed to confirm these findings [84]. Corticosteroid injections are not recommended as literature has shown limited benefit with potential risk of tendon rupture [47, 85].

High-volume ultrasound-guided injections aim to produce local mechanical effects, aiming to stretch, break, or occlude neo-vessels. The injections can be localized to the area pathology, whether midportion or insertional tendinopathy. Short- and long-term pain relief along with improved daily function (VISA scores) has been observed in 70% of patients with resistant Achilles tendinopathy after injection with 10 mL of 0.5% bupivacaine, 25 mg hydrocortisone acetate, and up to 40 mL of normal saline [86]. Similar improvement in symptomology via pain and stiff values as well as VISA scores has been noted in comparison to ESWT [87].

Unique to Achilles tendinopathy, orthotics are widely used with heel pads alleviating insertional Achilles tendinopathy, but clinical evidence is limited [88]. An AirHeel brace, with applied intermittent compression to minimize swelling and promote circulation, may serve as an alternative to eccentric exercises, particularly in patients with activity-limiting pain. No clinical differences have been found in the comparison of the AirHeel brace and eccentric training program for chronic Achilles tendinopathy [89].

40.5.2.2 Operative Treatment (Fact Box 3)

Approximately one-third of patients with Achilles tendinopathy fail to improve after 3–6 months of conservative management and as a consequence surgical treatment may be indicated [75]. Surgical intervention entails a spectrum from simple percutaneous tenotomy, to minimally invasive stripping of the tendon, to open procedures that may include tendon augmentation. In systematic review of surgical intervention for chronic Achilles tendinopathy, successful results in over

Fact Box 3 Operative Management of Tendinopathy

- Open or arthroscopic debridement of the inferior pole of the patella and pathologic tissue at the postero-superior patellar tendon is the mainstay of surgical management of patellar tendinopathy.
- Gastrocnemius contracture is important to assess and can be routinely treated with gastrocnemius recession.
- Achilles paratendinopathy can be managed with minimally invasive stripping, tendoscopic debridement, or open debridement of the paratenon.
- Open debridement and tubularization of the Achilles tendon or debridement of the insertion on the calcaneus allow for the removal of pathologic tissue, an initiation of a healing response.

70% of cases have been reported with a particular predilection for male patients and those who classify as athletes.

Regardless of the anatomic location of the Achilles tendinopathy, patients must be first assessed for gastrocnemius contracture via the Silfverskiold physical examination maneuver. This entails assessment of ankle dorsiflexion with and without knee flexion, determining the contribution of the triceps surae to limitation of ankle dorsiflexion. Gastrocnemius recession may be an adequate surgical option to address symptoms of refractory Achilles tendinopathy. Our preferred method of gastrocnemius recession involves a medial incision at the muscle-tendon intersection of the gastrocnemius with division of the gastrocnemius fascia while taking care to avoid injury to the sural nerve. Increased ankle

dorsiflexion without increased force on the tendon may alleviate further tendinopathy.

Isolated paratendinopathy can be managed with debridement of the paratenon; this may be done via a minimally invasive stripping technique (Fig. 40.3), endoscopically, or with an open incision. Minimally invasive stripping involves prone positioning with a tourniquet inflated to 250 mmHg. Two longitudinal 0.5 cm skin incisions are made at the proximal origin of the Achilles tendon, just medial and lateral to the origin, and two additional 0.5 cm incisions are made 1 cm beyond at the distal end of the AT. Using small hemostat, the AT is freed of adhesions proximally and a nonabsorbable suture is inserted, passing through the proximal incisions. This is then retrieved distally and used to carefully strip the posterior extent of the tendon from

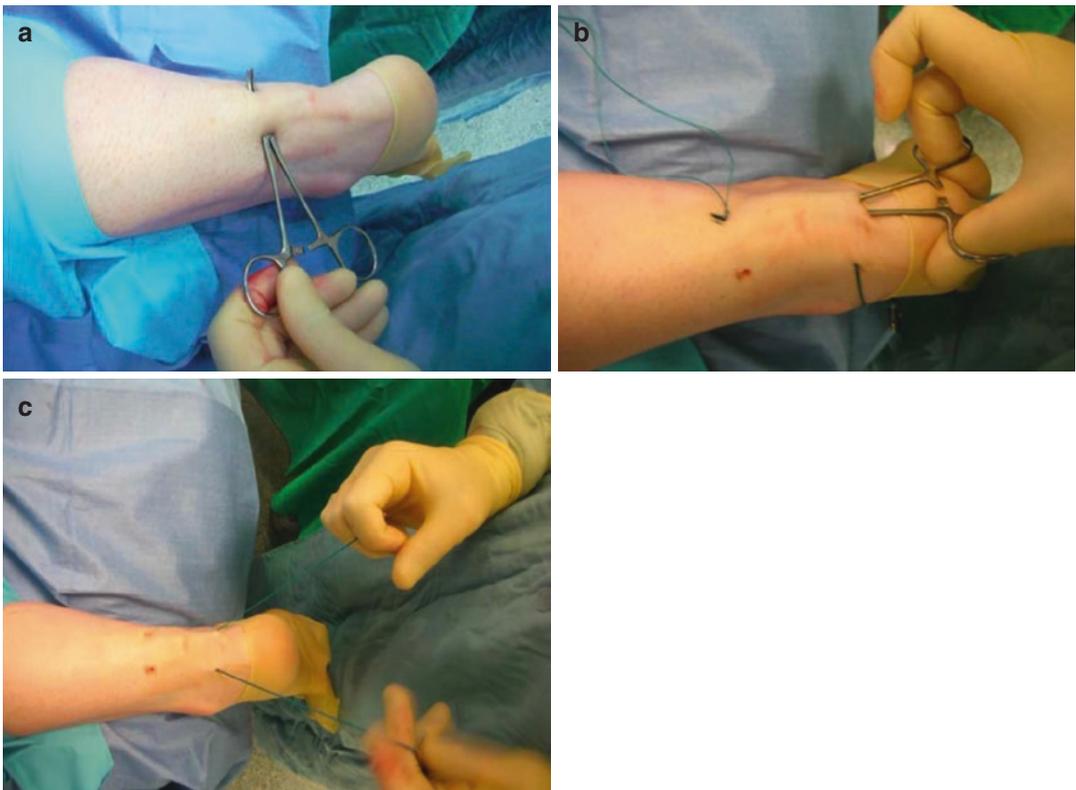


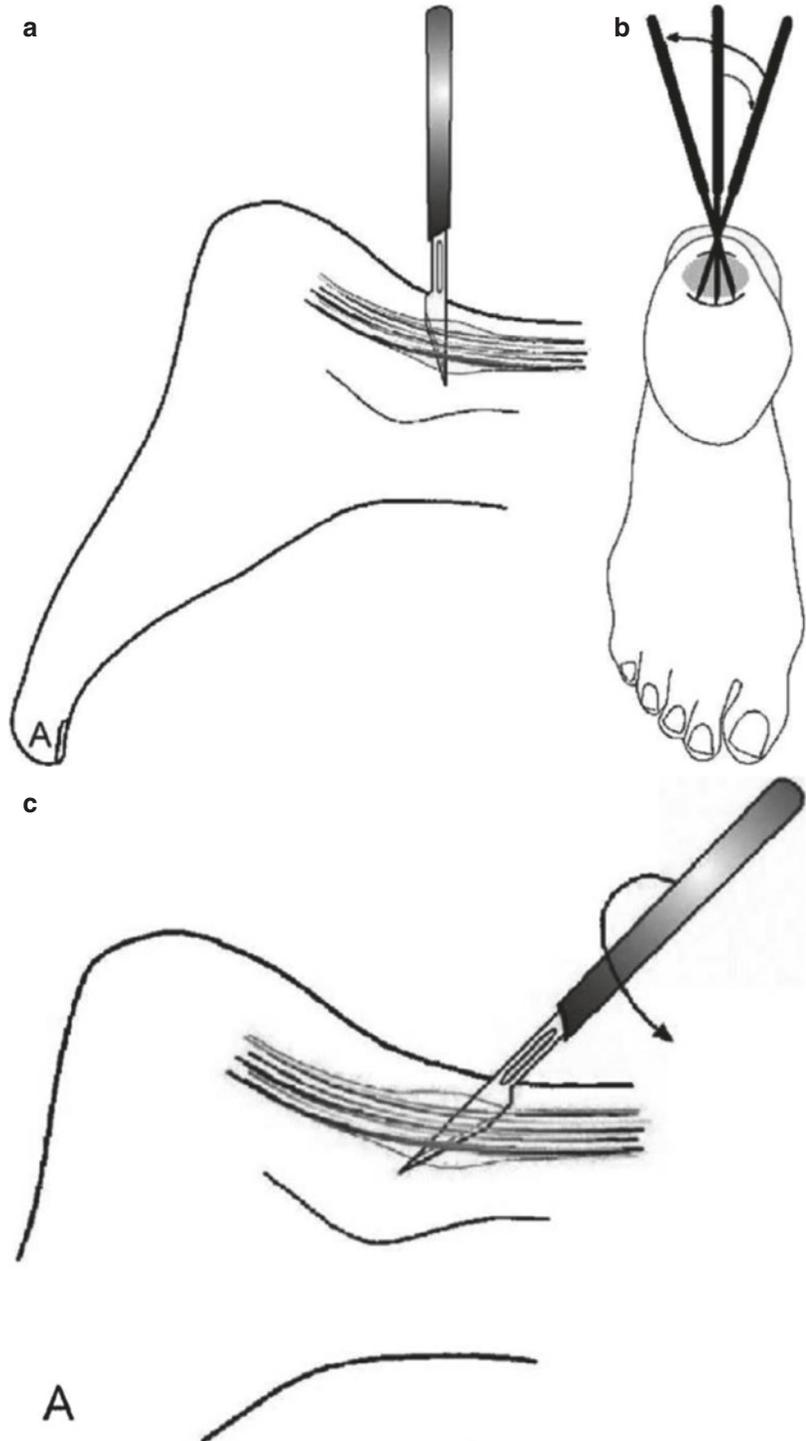
Fig. 40.3 Minimally invasive stripping of Achilles tendon. (a) Hemostat inserted to free AT of peritendinous adhesions. (b) Nonabsorbable suture inserted proximally and retrieved distally. (c) Using gentle sawing manipula-

tion, the suture is translated from proximal to distal, freeing the tendon periphery [75] (Borrowed from Maffulli et al 2019. *Foot and Ankle Surgery*)

proximal to distal [75, 77]. Endoscopic technique involves the use of a 30° arthroscopic camera and a mechanical shaver to break up paratenon adhesions.

Tendinopathy of the midportion of the Achilles tendon can be managed by less invasive percutaneous longitudinal tenotomy (Fig. 40.4) If percutaneous techniques are not successful, a more

Fig. 40.4 With patient lying prone, the area of maximal tenderness is palpated, and ultrasonography identifies swelling and location is marked. (a) #11 scalpel blade is inserted parallel to the long axis of the tendon fibers in the center of the area of tendinopathy. The cutting edge of the blade is directed caudally and penetrates the full thickness of the tendon. With the blade in place, a full passive ankle dorsiflexion is performed. This process is repeated with the blade angled at 45° on the sagittal axis medially and laterally (b). Ultimately the blade is reverse 180° and the entire process repeated (c) (*Borrowed from Maffulli et al 2019. Foot and Ankle Surgery*)



aggressive open debridement with tubularization of the tendon is an option. Typically, if more than 50% of the tendon is pathologic and requires debridement, we advocate for a flexor hallucis longus (FHL) tendon transfer to augment the deficit. Insertional tendinopathy is approached in a similar fashion with debridement and reattachment of the tendon. Debridement or resection of the postero-superior extent of the calcaneus is recommended to facilitate healing. Concomitant tendon transfer is indicated in greater than 50% AT resection. In cases of a nonviable Achilles tendon, with advocate for hamstring autograft as an option for reconstruction of Achilles tendon defects [90].

Surgery for insertional Achilles tendinopathy has shown to be correlated with good functional outcomes and satisfactory return to sports in 71% of patients when the surgical care was tailored to the degree of tendon involvement [91].

Take-Home Message

Management of patellar and Achilles tendinopathy in athletes requires timely diagnosis and patience in management from the provider and patient alike. Nonoperative management should be the goal and the overall mainstay of treatment. Activity modification to alleviate stresses to the affected tendon is a primary element of this approach. Due to limited level 1 evidence for the management of tendinopathy, all modalities available in our armamentarium must be used judiciously. Recent literature suggests that adjunct therapies such as extracorporeal shock wave therapy (ESWT) and platelet-rich plasma injection (PRP) are indicated to accelerate rehabilitation (eccentric exercise) and return to play. Ultimately, surgical modalities for refractory or recalcitrant symptomology are available, yet should remain avoidable with vigilant and aggressive management of tendinopathy.

References

1. Ackermann PW, Renström P. Tendinopathy in sport. *Sports Health*. 2012;4:193–201.
2. Khan KM, Maffulli N. Tendinopathy: an Achilles' heel for athletes and clinicians. *Clin J Sport Med*. 1998;8:151–4.
3. Magnusson SP, Langberg H, Kjaer M. The pathogenesis of tendinopathy: balancing the response to loading. *Nat Rev Rheumatol*. 2010;6:262–8.
4. Arya S, Kulig K. Tendinopathy alters mechanical and material properties of the Achilles tendon. *J Appl Physiol*. American Physiological Society Bethesda, MD. 2010;108:670–5.
5. Maffulli N, Longo UG, Denaro V. Novel approaches for the management of tendinopathy. *J Bone Joint Surg Am*. 2010;92:2604–17.
6. Rees JD, Maffulli N, Cook J. Management of tendinopathy. *Am J Sports Med*. 2009;37:1855–67.
7. Kujala UM, Sarna S, Kaprio J. Cumulative incidence of achilles tendon rupture and tendinopathy in male former elite athletes. *Clin J Sport Med*. 2005;15:133–5.
8. Rechartd M, Shiri R, Karppinen J, Jula A, Heliövaara M, Viikari-Juntura E. Lifestyle and metabolic factors in relation to shoulder pain and rotator cuff tendinitis: a population-based study. *BMC Musculoskeletal Disord*. *BioMed Central*. 2010;11:165–11.
9. Abboud JA, Beason DP, Soslowsky LJ. Emerging ideas: the effect of hypercholesterolemia on tendons. *Clin Orthop Relat Res*. Springer-Verlag. 2012;470:317–20.
10. Marie I, Delafenêtre H, Massy N, Thuillez C, Noblet C, Network of the French Pharmacovigilance Centers. Tendinous disorders attributed to statins: a study on ninety-six spontaneous reports in the period 1990–2005 and review of the literature. *Arthritis Rheum*. John Wiley & Sons, Ltd. 2008;59:367–72.
11. Alves C, Mendes D, Marques FB. Fluoroquinolones and the risk of tendon injury: a systematic review and meta-analysis. *Eur J Clin Pharmacol*. Springer Berlin Heidelberg. 2019;75:1431–43.
12. Miller BF, Olesen JL, Hansen M, Døssing S, Crameri RM, Welling RJ, et al. Coordinated collagen and muscle protein synthesis in human patella tendon and quadriceps muscle after exercise. *J Physiol Lond*. John Wiley & Sons, Ltd (10.1111). 2005;567:1021–33.
13. Heinemeier KM, Olesen JL, Haddad F, Langberg H, Kjaer M, Baldwin KM, et al. Expression of collagen and related growth factors in rat tendon and skeletal muscle in response to specific contraction types. *J Physiol Lond*. John Wiley & Sons, Ltd (10.1111). 2007;582:1303–16.
14. Wang JH-C, Jia F, Yang G, Yang S, Campbell BH, Stone D, et al. Cyclic mechanical stretching of human tendon fibroblasts increases the production of prostaglandin E2 and levels of cyclooxygenase expression:

- a novel in vitro model study. *Connect Tissue Res.* 2003;44:128–33.
15. Tomasek JJ, Gabbiani G, Hinz B, Chaponnier C, Brown RA. Myofibroblasts and mechano-regulation of connective tissue remodelling. *Nat Rev Mol Cell Biol* .. Nature Publishing Group. 2002;3:349–63.
 16. Pufe T, Petersen WJ, Mentlein R, Tillmann BN. The role of vasculature and angiogenesis for the pathogenesis of degenerative tendons disease. *Scand J Med Sci Sports.* John Wiley & Sons, Ltd (10.1111). 2005;15:211–22.
 17. Lian Ø, Dahl J, Ackermann PW, Frihagen F, Engebretsen L, Bahr R. Pronociceptive and antinociceptive neuromediators in patellar tendinopathy. *Am J Sports Med* .. SAGE Publications Sage CA: Los Angeles CA. 2006;34:1801–8.
 18. Lian ØB, Engebretsen L, Bahr R. Prevalence of Jumper's knee among elite athletes from different sports: a cross-sectional study. *Am J Sports Med.* SAGE PublicationsSage CA: Los Angeles, CA. 2017;33:561–7.
 19. Ferretti A, Ippolito E, Mariani P, Puddu G. Jumper's knee. *Am J Sports Med.* 1983;11:58–62.
 20. Cook JL, Khan KM, Kiss ZS, Griffiths L. Patellar tendinopathy in junior basketball players: a controlled clinical and ultrasonographic study of 268 patellar tendons in players aged 14–18 years. *Scand J Med Sci Sports.* John Wiley & Sons, Ltd (10.1111). 2000;10:216–20.
 21. Sprague A, Epsley S, Silbernagel KG. Distinguishing quadriceps tendinopathy and patellar tendinopathy: semantics or significant? *J Orthop Sports Phys Ther.* 2019;49:627–30.
 22. Visnes H, Bahr R. The evolution of eccentric training as treatment for patellar tendinopathy (jumper's knee): a critical review of exercise programmes. *Br J Sports Med.* 2007;41:217–23.
 23. Visnes H, Tegnander A, Bahr R. Ultrasound characteristics of the patellar and quadriceps tendons among young elite athletes. *Scand J Med Sci Sports.* John Wiley & Sons, Ltd (10.1111). 2015;25:205–15.
 24. Häggglund M, Zwerver J, Ekstrand J. Epidemiology of patellar tendinopathy in elite male soccer players. *Am J Sports Med.* SAGE PublicationsSage CA: Los Angeles CA. 2011;39:1906–11.
 25. Malliaras P, Cook J, Purdam C, Rio E. Patellar tendinopathy: clinical diagnosis, load management, and advice for challenging case presentations. *J Orthop Sports Phys Ther.* JOSPT, Inc. JOSPT, 1033 North Fairfax Street, Suite 304, Alexandria, VA 22134-1540. 2015;45:887–98.
 26. Ward ER, Andersson G, Backman LJ, Gaida JE. Fat pads adjacent to tendinopathy: more than a coincidence? *Br J Sports Med.* 2016;50:1491–2.
 27. Abat F, Alfredson H, Cucchiari M, Madry H, Marmotti A, Mouton C, et al. Current trends in tendinopathy: consensus of the ESSKA basic science committee. Part I: biology, biomechanics, anatomy and an exercise-based approach. *J Exp Orthop.* 2017;4:1046–11.
 28. Figueroa D, Figueroa F, Calvo R. Patellar tendinopathy: diagnosis and treatment. *J Am Acad Orthop Surg.* 2016;24:e184–92.
 29. van der Worp H, van Ark M, Zwerver J, van den Akker Scheek I. Risk factors for patellar tendinopathy in basketball and volleyball players: a cross-sectional study. *Scand J Med Sci Sports.* John Wiley & Sons, Ltd (10.1111). 2012;22:783–90.
 30. Witvrouw E, Bellemans J, Lysens R, Danneels L, Cambier D. Intrinsic risk factors for the development of patellar tendinitis in an athletic population. A two-year prospective study. *Am J Sports Med.* 2001;29:190–5.
 31. Warden SJ, Brukner P. Patellar tendinopathy. *Clin Sports Med.* 2003;22:743–59.
 32. Rio E, Moseley L, Purdam C, Samiric T, Kidgell D, Pearce AJ, et al. The pain of tendinopathy: physiological or pathophysiological? *Sports Med.* 5 ed. Springer International Publishing. 2013;44:9–23.
 33. Kountouris A, Cook J. Rehabilitation of Achilles and patellar tendinopathies. *Best Pract Res Clin Rheumatol.* 2007;21:295–316.
 34. Larsson MEH, Käll I, Nilsson-Helander K. Treatment of patellar tendinopathy – a systematic review of randomized controlled trials. *Knee Surg Sports Traumatol Arthrosc.* 2nd ed. Springer-Verlag. 2012;20:1632–46.
 35. Kaux JF, Drion P, Libertiaux V, Colige A, Hoffmann A, Nusgens B, et al. Eccentric training improves tendon biomechanical properties: a rat model. *Br J Sports Med.* BMJ Publishing Group Ltd and British Association of Sport and Exercise Medicine. 2014;48:617.
 36. Stanish WD, Rubinovich RM, Curwin S. Eccentric exercise in chronic tendinitis. *Clin Orthop Relat Res.* 1986;65–8.
 37. Couppé C, Svensson RB, Silbernagel KG, Langberg H, Magnusson SP. Eccentric or concentric exercises for the treatment of Tendinopathies? *J Orthop Sports Phys Ther.* JOSPT, Inc. JOSPT, 1033 North Fairfax Street, Suite 304, Alexandria, VA 22134-1540. 2015;45:853–63.
 38. Rudavsky A, Cook J. Physiotherapy management of patellar tendinopathy (jumper's knee). *J Physiother.* Elsevier. 2014;60:122–9.
 39. Frohm A, Saartok T, Halvorsen K, Renström P. Eccentric treatment for patellar tendinopathy: a prospective randomised short-term pilot study of two rehabilitation protocols. *Br J Sports Med.* British Association of Sport and Exercise Medicine. 2007;41:e7.
 40. Young MA, Cook JL, Purdam CR, Kiss ZS, Alfredson H. Eccentric decline squat protocol offers superior results at 12 months compared with traditional eccentric protocol for patellar tendinopathy in volleyball players. *Br J Sports Med.* British Association of Sport and Exercise Medicine. 2005;39:102–5.
 41. van Leeuwen MT, Zwerver J, van den Akker Scheek I. Extracorporeal shockwave therapy for patellar tendinopathy: a review of the literature. *Br J Sports Med.*

- British Association of Sport and Exercise Medicine. 2009;43:163–8.
42. van der Worp H, van den Akker-Scheek I, van Schie H, Zwerver J. ESWT for tendinopathy: technology and clinical implications. *Knee Surg Sports Traumatol Arthrosc.* Springer-Verlag. 2013;21:1451–8.
 43. Waugh CM, Morrissey D, Jones E, Riley GP, Langberg H, Screen HRC. In vivo biological response to extracorporeal shockwave therapy in human tendinopathy. *Eur Cell Mater.* 2015;29:268–80.. –discussion280
 44. Wang C-J, Ko J-Y, Chan Y-S, Weng L-H, Hsu S-L. Extracorporeal shockwave for chronic patellar tendinopathy. *Am J Sports Med.* SAGE PublicationsSage CA: Los Angeles, CA. 2017;35: 972–8.
 45. Abat F, Sánchez-Sánchez JL, Martín-Nogueras AM, Calvo-Arenillas JI, Yajeya J, Méndez-Sánchez R, et al. Randomized controlled trial comparing the effectiveness of the ultrasound-guided galvanic electrolysis technique (USGET) versus conventional electro-physiotherapeutic treatment on patellar tendinopathy. *J Exp Orthop.* Springer Berlin Heidelberg. 2016;3:34.
 46. Abat F, Gelber PE, Polidori F, Monllau JC, Sanchez-Ibañez JM. Clinical results after ultrasound-guided intratissue percutaneous electrolysis (EPI®) and eccentric exercise in the treatment of patellar tendinopathy. *Knee Surg Sports Traumatol Arthrosc.* Springer Berlin Heidelberg. 2014;23:1046–52.
 47. Fredberg U, Bolvig L, Jensen MP, Clemmensen D, Jakobsen BW, Pedersen KS. Ultrasonography as a tool for diagnosis, guidance of local steroid injection and, together with pressure algometry, monitoring of the treatment of athletes with chronic jumper's knee and Achilles tendinitis: a randomized, double-blind, placebo-controlled study. *Scand J Rheumatol.* Taylor & Francis. 2009;33:94–101.
 48. Kongsgaard M, Kovanen V, Aagaard P, Doessing S, Hansen P, Laursen AH, et al. Corticosteroid injections, eccentric decline squat training and heavy slow resistance training in patellar tendinopathy. *Scand J Med Sci Sports.* John Wiley & Sons, Ltd (10.1111). 2009;19:790–802.
 49. Everhart JS, Cole D, Sojka JH, Higgins JD, Magnussen RA, Schmitt LC, et al. Treatment options for patellar tendinopathy: a systematic review. *Arthroscopy.. YJARS.* W.B. Saunders. 2017;33:861–72.
 50. Vetrano M, Castorina A, Vulpiani MC, Baldini R, Pavan A, Ferretti A. Platelet-rich plasma versus focused shock waves in the treatment of Jumper's knee in athletes. *Am J Sports Med.* SAGE Publications Sage CA: Los Angeles, CA. 2013;41:795–803.
 51. Andriolo L, Altamura SA, Reale D, Candrian C, Zaffagnini S, Filardo G. Nonsurgical treatments of patellar tendinopathy: multiple injections of platelet-rich plasma are a suitable option: a systematic review and meta-analysis. *Am J Sports Med.* 2019;47:1001–18.
 52. Gosens T, Oudsten Den BL, Fievez E, van t Spijker P, Fievez A. Pain and activity levels before and after platelet-rich plasma injection treatment of patellar tendinopathy: a prospective cohort study and the influence of previous treatments. *Int Orthop.* 2nd ed. Springer-Verlag. 2012;36:1941–6.
 53. Filardo G, Kon E, Villa Della S, Vincentelli F, Fornasari PM, Marcacci M. Use of platelet-rich plasma for the treatment of refractory jumper's knee. *Int Orthop..* Springer-Verlag. 2009;34:909–15.
 54. Anitua E, Andía I, Sanchez M, Azofra J, del Mar Zalduendo M, la Fuente de M, et al. Autologous preparations rich in growth factors promote proliferation and induce VEGF and HGF production by human tendon cells in culture. *J Orthop Res..* John Wiley & Sons Ltd. 2005;23:281–6.
 55. Kon E, Filardo G, Delcogliano M, Presti ML, Russo A, Bondi A, et al. Platelet-rich plasma: new clinical application: a pilot study for treatment of jumper's knee. *Injury.* 2009;40:598–603.
 56. Ferrero G, Fabbro E, Orlandi D, Martini C, Lacelli F, Serafini G, et al. Ultrasound-guided injection of platelet-rich plasma in chronic Achilles and patellar tendinopathy. *J Ultrasound.* 2012;15:260–6.
 57. Kaux JF, Croisier JL, Forthomme B, Le Goff C, Buhler F, Savanier B, et al. Using platelet-rich plasma to treat jumper's knees: exploring the effect of a second closely-timed infiltration. *J Sci Med Sport..* Elsevier. 2016;19:200–4.
 58. Pascual-Garrido C, Rolón A, Makino A. Treatment of chronic patellar tendinopathy with autologous bone marrow stem cells: a 5-year-Followup. *Stem Cells Int..* Hindawi. 2012;2011:1–5.
 59. Clarke AW, Alyas F, Morris T, Robertson CJ, Bell J, Connell DA. Skin-derived tenocyte-like cells for the treatment of patellar tendinopathy. *Am J Sports Med.* SAGE PublicationsSage CA: Los Angeles, CA. 2011;39:614–23.
 60. Muneta T, Koga H, Ju Y-J, Mochizuki T, Sekiya I. Hyaluronan injection therapy for athletic patients with patellar tendinopathy. *J Orthop Sci.* 2012;17:425–31.
 61. Alfredson H, Ohberg L. Neovascularisation in chronic painful patellar tendinosis--promising results after sclerosing neovessels outside the tendon challenge the need for surgery. *Knee Surg Sports Traumatol Arthrosc..* Springer-Verlag. 2005;13:74–80.
 62. Hoksrud A, Bahr R. Ultrasound-guided sclerosing treatment in patients with patellar tendinopathy (jumper's knee). 44-month follow-up. *Am J Sports Med.* SAGE Publications Sage CA: Los Angeles, CA. 2011;39:2377–80.
 63. Hoksrud A, Torgalsen T, Harstad H, Haugen S, Andersen TE, Risberg MA, et al. Ultrasound-guided sclerosis of neovessels in patellar tendinopathy: a prospective study of 101 patients. *Am J Sports Med.* 2012;40:542–7.

64. Hoksrud A, Ohberg L, Alfredson H, Bahr R. Ultrasound-guided sclerosis of neovessels in painful chronic patellar tendinopathy: a randomized controlled trial. *Am J Sports Med.* SAGE PublicationsSage CA: Los Angeles, CA. 2006;34:1738–46.
65. Orchard J, Massey A, Brown R, Cardon-Dunbar A, Hofmann J. Successful management of tendinopathy with injections of the MMP-inhibitor Aprotinin. *Clin Orthop Relat Res.* Springer-Verlag. 2008;466:1625–32.
66. Paoloni JA, Appleyard RC, Nelson J, Murrell GAC. Topical nitric oxide application in the treatment of chronic extensor tendinosis at the elbow: a randomized, double-blinded, placebo-controlled clinical trial. *Am J Sports Med.* SAGE PublicationsSage CA: Los Angeles, CA. 2003;31:915–20.
67. Steunebrink M, Zwerver J, Brandsema R, Groenenboom P, van den Akker-Scheek I, Weir A. Topical glyceryl trinitrate treatment of chronic patellar tendinopathy: a randomised, double-blind, placebo-controlled clinical trial. *Br J Sports Med.* BMJ Publishing Group Ltd and British Association of Sport and Exercise Medicine. 2013;47:34–9.
68. Ogon P, Maier D, Jaeger A, Suedkamp NP. Arthroscopic patellar release for the treatment of chronic patellar tendinopathy. *Arthroscopy.* YJARS. W.B. Saunders. 2006;22:462.e1–5.
69. Kruckeberg BM, Chahla J, Ferrari MB, Sanchez G, Moatshe G, LaPrade RF. Open patellar tendon Tenotomy, debridement, and repair technique augmented with platelet-rich plasma for recalcitrant patellar tendinopathy. *Arthrosc Tech.* 2017;6:e447–53.
70. Kudron C, Carlson MJ, Meron A, Sridhar B, Brakke HR. Using ultrasound measurement of the Achilles tendon in asymptomatic runners to assist in predicting tendinopathy. *J Ultrasound Med.* 2019;11:521.
71. Mahieu NN, Witvrouw E, Stevens V, Van Tiggelen D, Roget P. Intrinsic risk factors for the development of Achilles tendon overuse injury: a prospective study. *Am J Sports Med.* SAGE Publications Sage CA: Los Angeles CA. 2017;34:226–35.
72. Longo UG, Ronga M, Maffulli N. Achilles tendinopathy. *Sports Med Arthrosc.* 2018;26:16–30.
73. Amin NH, Old AB, Tabb LP, Garg R, Toossi N, Cerynik DL. Performance outcomes after repair of complete Achilles tendon ruptures in National Basketball Association Players. *Am J Sports Med.* SAGE Publications Sage CA: Los Angeles, CA. 2013;41:1864–8.
74. Amin NH, McCullough KC, Mills GL, Jones MH, Cerynik DL, Rosneck J, et al. The impact and functional outcomes of Achilles tendon pathology in National Basketball Association Players. *Clin Res Foot Ankle.* OMICS International. 2016;4:1–6.
75. Maffulli N, Longo U, Kadakia A, Spiezia F. Achilles tendinopathy. *Foot and ankle surgery: official journal of the European society of foot and ankle surgeons.* 2019. (Article in Press) <https://dx.doi.org/10.1016/j.fas.2019.03.009>.
76. Paavola M, Kannus P, Paakkala T, Pasanen M, Järvinen M. Long-term prognosis of patients with Achilles tendinopathy. *Am J Sports Med.* SAGE PublicationsSage CA: Los Angeles, CA. 2000;28:634–42.
77. Maffulli N, Sharma P, Luscombe KL. Achilles tendinopathy: aetiology and management. *J R Soc Med.* SAGE Publications Sage UK: London, England. 2017;97:472–6.
78. Silbernagel KG, Crossley KM. A proposed return-to-sport program for patients with midportion Achilles tendinopathy: rationale and implementation. *J Orthop Sports Phys Ther.* JOSPT, Inc. JOSPT, 1033 North Fairfax Street, Suite 304, Alexandria, VA 22134-1540. 2015;45:876–86.
79. Mafi N, Lorentzon R, Alfredson H. Superior short-term results with eccentric calf muscle training compared to concentric training in a randomized prospective multicenter study on patients with chronic Achilles tendinosis. *Knee Surg Sports Traumatol Arthrosc.* Springer-Verlag. 2001;9:42–7.
80. Roos EM, Engström M, Lagerquist A, Söderberg B. Clinical improvement after 6 weeks of eccentric exercise in patients with mid-portion Achilles tendinopathy—a randomized trial with 1-year follow-up. *Scand J Med Sci Sports.* John Wiley & Sons, Ltd (10.1111). 2004;14:286–95.
81. Rompe JD, Furia J, Maffulli N. Eccentric loading versus eccentric loading plus shock-wave treatment for midportion achilles tendinopathy: a randomized controlled trial. *Am J Sports Med.* SAGE PublicationsSage CA: Los Angeles, CA. 2009;37:463–70.
82. Chen X, Jones IA, Park C, Vangsness CT. The efficacy of platelet-rich plasma on tendon and ligament healing: a systematic review and meta-analysis with bias assessment. *Am J Sports Med.* 2018;46:2020–32.
83. Zhang Y-J, Xu S-Z, Gu P-C, Du J-Y, Cai Y-Z, Zhang C, et al. Is platelet-rich plasma injection effective for chronic Achilles tendinopathy? A meta-analysis. *Clin Orthop Relat Res.* 2018;476:1633–41.
84. LaBelle MW, Marcus RE. CORR insights®: is platelet-rich plasma injection effective for chronic Achilles tendinopathy? A meta-analysis. *Clin Orthop Relat Res.* 2018;476:1642–4.
85. DaCruz DJ, Geeson M, Allen MJ, Phair I. Achilles paratendonitis: an evaluation of steroid injection. *Br J Sports Med.* British Association of Sport and Exercise Medicine. 1988;22:64–5.
86. Chan O, O'Dowd D, Padhiar N, Morrissey D, King J, Jalan R, et al. High volume image guided injections in chronic Achilles tendinopathy. *Disabil Rehabil.* Taylor & Francis. 2009;30:1697–708.
87. Wheeler P, Tattersall C. Novel interventions for recalcitrant achilles tendinopathy: benefits seen following high-volume image-guided injection or extracorporeal shockwave therapy-A prospective cohort study. *Clinical journal of sport medicine.* *Clin J Sport Med.* 2018;0:1–6. <https://dx.doi.org/10.1097/jsm.0000000000000580>.
88. Lowdon A, Bader DL, Mowat AG. The effect of heel pads on the treatment of Achilles tendinitis: a double blind trial. *Am J Sports Med.* SAGE Publications. 2016;12:431–5.

89. Petersen W, Welp R, Rosenbaum D. Chronic Achilles tendinopathy: a prospective randomized study comparing the therapeutic effect of eccentric training, the AirHeel brace, and a combination of both. *Am J Sports Med.* SAGE Publications Sage CA: Los Angeles, CA. 2017;35:1659–67.
90. Karnovsky SC, Drakos MC. Revision Achilles reconstruction with hamstring autograft and FHL tendon transfer in an athlete. *HSS J.* Springer US. 2017;13:302–6.
91. Hardy A, Rousseau R, Issa S-P, Gerometta A, Pascal-Moussellard H, Granger B, et al. Functional outcomes and return to sports after surgical treatment of insertional Achilles tendinopathy: surgical approach tailored to the degree of tendon involvement. *Orthop Traumatol Surg Res.* 2018;104:719–23.
92. Blazina ME, Kerlan RK, Jobe FW, Carter VS, Carlson GJ. Jumper's knee. *Orthop Clin North Am.* 1973;4:665–78.
93. Roels J, Martens M, of JMTAJ 1978. Patellar tendinitis (jumper's knee). journals.sagepub.com.
94. Ferretti A, Conteduca F, Camerucci E, Morelli F. Patellar tendinosis: a follow-up study of surgical treatment. *J Bone Joint Surg Am.* 2002;84:2179–85.

Best Evidence Based References

- Andriolo L, Altamura SA, Reale D, Candrian C, Zaffagnini S, Filardo G. Nonsurgical treatments of patellar tendinopathy: multiple injections of platelet-rich plasma are a suitable option: a systematic review and meta-analysis. *Am J Sports Med.* 2019;47(4):1001–18.
- Figueroa D, Figueroa F, Calvo R. Patellar tendinopathy: diagnosis and treatment. *J Am Acad Orthop Surg.* 2016;24(12):e184–92.
- Maffulli N, Longo UG, Kadakia A, Spiezia F. Achilles tendinopathy. *Foot Ankle Surg.* 2019.
- Malliaras P, Cook J, Purdam C, Rio E. Patellar tendinopathy: clinical diagnosis, load management, and advice for challenging case presentations. *J Orthop Sports Phys Ther.* JOSPT, Inc. JOSPT, 1033 North Fairfax Street, Suite 304, Alexandria, VA 22134-1540. 2015;45(11):887–98.
- Rompe JD, Furia J, Maffulli N. Eccentric loading versus eccentric loading plus shock-wave treatment for midportion achilles tendinopathy: a randomized controlled trial. *Am J Sports Med.* SAGE Publications, Sage CA: Los Angeles, CA. 2009;37(3):463–70.



Back Injuries and Management of low Back Pain in Basketball

41

Lara W. Massie, Thomas J. Buell, Eyal Behrbalk, and Christopher I. Shaffrey

41.1 Introduction

Low back pain is common among the general population, with a prevalence of up to 80% [1]. However, professional athletes put repetitive, unnatural levels of stress on their spine in the form of dynamic loading (jumping), lateral bending, twisting, shear, and compressive forces [2]. Low back pain is the seventh most common injury found in NCAA men's basketball population, accounting for 2.2% of the injuries sustained in games and 3.6% of the injuries sustained in practice [3]. At the professional level, the trunk and spine was found to be the fourth most prevalent injury region [4].

The success of treatment of low back pain is intrinsically tied to the accuracy of diagnosis, especially given that athletes are likely to return to the pattern of activity which produced their

injury in the first place. Several treatment classification schemes have been described to guide workup for the diagnosis and treatment of low back injuries from a physical therapy and training perspective [5, 6]. These largely emphasize accuracy of diagnosis and identification of psychosocial stressors or psychological stressors which can predispose to lingering symptoms (such as a fear-based mindset) which can be addressed as part of the therapeutic regimen.

In comparison to an older, degenerative population with back pain, the athletic population is more likely to experience an acute disc herniation, a stress fracture of the pars interarticularis, muscular back strain. Identification of red flag symptoms during workup can help delineate which patients are most likely to benefit from advanced imaging rather than rest.

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41.2 Epidemiology in Basketball

A recent systematic review focusing on the epidemiology of sports injuries in Basketball reported that a total of 975 injuries were registered as associated to the trunk and spine, representing 7.5% of all injuries [4]. A higher prevalence of trunk and spine injuries was observed in professionals, and of the total trunk and spine injuries reported, 31% were registered in children and adolescents and 69% in professional players. The trunk and spine was the

fourth most prevalent injury region in professionals. Of the all injuries reported for professionals, 11.1% occurred in the trunk and spine, while only 5% for children and adolescents [4]. Starkey reported that only 6.9% of all injuries occurred in this region in a study on NBA players; however, only injuries to the lumbar and thoracic spine were reported, and not pooled with trunk injuries [7].

Other studies in adolescent basketball players reported trunk and spine injuries accounted for 11.4–13.5% of all injuries [3, 8].

Drakos et al. in their 17-year review of injuries in the NBA reported that lumbar spine injuries accounted for 10.2% of all injuries ($n = 1279$) and were responsible for almost as many games missed ($n = 6729$) as ankle injuries ($n = 6838$) [9].

41.3 Spinal Anatomy

A comprehensive understanding of spinal anatomy is important and can help facilitate accurate diagnosis of the underlying pathology which manifests as low back pain. Briefly, the lumbar spine is composed of five vertebral segments, which surround the spinal canal. It sits above the sacrum, which articulates with the pelvis at the sacroiliac (SI) joints. The lumbar vertebrae articulate posteriorly at the facet joints and anteriorly at the intervertebral discs. Between the superior and inferior facet joints at each level is a bridge of bone, the pars interarticularis, which is subject to repeated stress or congenital nonunion (traumatic or congenital spondylolysis). There are ligamentous attachments anteriorly and posteriorly along the vertebral bodies, as well as between the laminae posteriorly. Any of these anatomical structures can be injured and produce symptoms of low back pain.

Intervertebral discs are composed of a fibrous outer layer (annulus fibrosis, functions as a ligament to restrain movements of the vertebral bodies above and below), as well as a gelatinous nucleus pulposus.

The neural elements (spinal cord, which ends around L1, and lumbar nerve roots, which exit at each neural foramen) are surrounded and pro-

ected by the bony and ligamentous spine but are at risk of impingement or irritation when there is injury or inflammation to the musculoskeletal structures.

Furthermore, the spine is surrounded by muscular attachments anterolaterally (psoas), posteriorly (erector spinae, multifidus, and interspinalis), which also are subject to injury or strain.

An understanding of the normal biomechanics of the lumbar spine is helpful to identify how certain activities can worsen symptoms. For instance, extension of the lumbar spine decreases the diameter of the spinal canal and neural foramina, whereas flexion expands both spaces. Flexion increases pressure on the intervertebral discs, and extension increases pressure on the facet joints. Axial loading (such as landing from a jump) puts a significantly higher pressure on the intervertebral disc than the facets. Bending and twisting stress both the annulus fibrosis of the intervertebral disc and the facet joints and can lead to annular tears.

Biomechanically, basketball players experience significantly more twisting, bending, and axial compressive forces than would be experienced in normal daily activities [7].

41.4 Clinical Examination

History and physical examination are critical in aiding with diagnosis. Information about the onset (gradual vs. sudden), location, timing, and relationship to activity is critical in diagnosis. Gradual onset may be suggestive of a stress injury such as a fracture of the pars interarticularis.

A clear understanding of the location and distribution of the pain (between the low back and lower extremities) is critical. Pain which radiates down the legs is concerning for nerve root irritation and classically follows a dermatomal pattern. Pain that does not follow a classic dermatomal pattern may indicate a peripheral nerve injury, or if it involves both lower extremities, it could be concerning for central involvement with cord compression and myelopathy.

Timing of pain and association with activity is also critical. Pain that is worse with motion, espe-

cially twisting, is indicative of mechanical back pain. Pain arising from the discs may worsen with flexion or with straining. Pain with extension may be associated with spinal stenosis, SI joint dysfunction, or spondylolysis. Lying down often improves discogenic, mechanical, and stenotic symptoms, but back pain that worsens at night should be considered a “red flag symptom” as it may be associated with neoplasm/tumor. Other red flag symptoms for spine pathology that may warrant urgent workup/imaging include bowel, bladder, or sexual dysfunction (associated with neurologic impingement of the conus medullaris/cauda equina). Fever and the inability to find a comfortable resting position warrant further investigation for an infectious source of pain.

Physical examination should assess the overall alignment of the patient’s stance, palpation for any step-offs along the palpable spinous processes or paraspinal muscle spasm, and range of motion in flexion, extension, side bending, and twisting. Patients should be asked to identify the exact location and distribution of their pain and any numbness or tingling radiating into the legs. The patient’s posture and gait should be assessed for any forward flexion (spinal stenosis), wide-based stance (myelopathy), or leaning to one side (often away from the side of nerve root compression).

Neurologic examination, including motor strength, sensory exam, and reflexes, is critical to document. Findings of nerve root compression (lower motor neuron findings), include muscular weakness or numbness in a dermatomal distribution and hyporeflexia. Spasticity, hyperreflexia, clonus, and non-dermatomal numbness/weakness are concerning for spinal cord compression (myelopathy). In cases of suspected spinal cord injury, rectal tone should be documented.

Provocative maneuvers during physical examination are also useful. Lower lumbar radicular/sciatic pain can often be reproduced with the crossed straight leg raise (SLR) test (elevation of the leg $\sim 60^\circ$). Similarly, upper lumbar radicular pain can often be provoked with a femoral stretch test. Flexion, abduction, and external rotation (FABER) of the hip/ Patrick’s test can suggest hip or SI joint dysfunction.

41.5 Radiographic Evaluation and Additional Testing

Any form of imaging provides a structural assessment of the skeletal and neural structures. It is important to remember that the presence of a radiographic abnormality may or may not correlate with the patient’s clinical symptoms. On initial assessment, imaging studies may not be indicated if there are neither neurologic deficits nor other red flag symptoms. Disc herniations and degenerative changes are noted on 35% of MRIs performed on asymptomatic volunteers [10]. Given that the athletic population has been found to have more rapid deterioration of the lumbar intervertebral discs than age-matched counterparts, they may have several abnormal findings on imaging which are incidental [11].

Plain radiographs, especially standing and weight-bearing films, allow for the assessment of vertebral alignment, disc height, facet arthropathy, and foraminal height. Spondylolisthesis, degenerative endplate changes, and congenital anomalies can also be identified. Mobile spondylolisthesis can be assessed on flexion/extension imaging.

Advanced imaging with computed tomography (CT) is helpful in assessing the osseous structures and more definitively visualizing suspected spondylolisthesis/stress fractures. Magnetic resonance imaging (MRI) is more useful for visualizing the soft tissues of the discs, spinal cord, and nerve roots. Short-TI Inversion Recovery (STIR) can be used to identify edema in acute injuries consistent with ligamentous and muscular strain. CT myelography can be used as an alternative to MRI to identify compressive lesions, especially in patients with history of instrumented spine surgery since metallic artifact can severely limit accurate MRI assessment.

Neural function can be assessed with electromyography (EMG) and nerve conduction studies (NCS); however, these tests are performed in the paraspinal muscles and extremities distal to the common site of compression, and the nerve will produce normal firing on stimulation distal to the injury on EMG/NCS testing for several weeks after an acute injury. Thus, EMG/NCS is usually reserved for 4–6 weeks following injury.

41.6 Common Spinal Injuries

41.6.1 Lumbar Strain

Definition: Paraspinal muscle strain. The most common cause of lower back pain [12].

Mechanism of injury: Overuse or overstretching of a muscle, ligament, or muscle-tendon unit. Often occurs with repetitive motions (overuse), misuse, or with trauma to the low back. Can have an associated inflammatory radicular pain.

Clinical symptoms: Back pain without neurologic deficit. Pain is often localized and is consistent with muscular spasm. There may be immediate pain or a delayed development of stiffness and muscular spasm the day after the injury. Pain is exacerbated by stretching and relieved with rest.

Diagnosis: MRI may show bright signal at the site of the tear on the STIR images. Weight-bearing imaging may show straightening of the spine from paraspinal muscle spasm.

Treatment: Rest from athletic activities, with early gentle mobilization. Rest should be limited to 1–2 days. Steroids or nonsteroidal anti-inflammatory drugs (NSAIDs), muscle relaxants. Physical therapy with core strengthening. Stretching. Trigger point injections.

Prognosis: 90% of patients achieve total resolution of their symptoms within 2 months in the general population [12]. Return to play should be delayed until the patient has complete range of motion [13].

41.6.2 Herniated Nucleus Pulposus and Radiculopathy

Definition: Radiculopathy: Numbness, weakness, parasthesias, or pain caused by impingement or damage to a single nerve root.

Mechanism of injury: Protrusion of the nucleus pulposus through the annulus fibrosis (Figs. 41.1 and 41.2), either in continuity (termed “herniation”) or not (“extrusion”). This usually occurs posteriorly along the annulus, either centrally or eccentric to one side. In more advanced cases of degeneration of the nucleus



Fig. 41.1 A computerized illustration of a lumbar intervertebral disc herniation (Courtesy of Thomas J Buell. Used with permission)

pulposus, there can be an annular bulge rather than a disc herniation.

Clinical symptoms: Patients will often have some degree of back pain but a more significant lower extremity pain in the associated dermatome. Often, when a disc bulge or herniation is paracentral, symptoms will be unilateral. Central disc herniations, if large enough, can produce cauda equina syndrome (weakness and urinary retention with overflow incontinence). Patients with severe, unrelenting radicular pain may have herniations more laterally along the annulus which can compress the dorsal root ganglion of the nerve as it exits the foramen. Thoracic disc herniations can produce radicular pain around the chest or abdominal wall, and these patients should also be examined for symptoms of myelopathy.

Diagnosis: Advanced imaging with MRI or CT myelogram is the most sensitive for evidence of compression. Selective nerve root injections can be helpful as both a nonoperative treatment and serve as a diagnostic aid in patients with

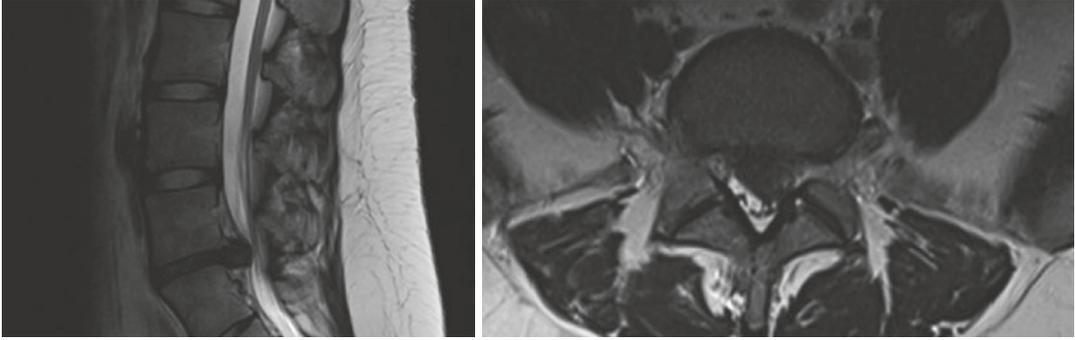


Fig. 41.2 L5-S1 left lumbar disc herniation on MRI

multilevel radiographic changes or nonspecific radicular pain.

Treatment: Nonoperative treatment includes neuroleptic pain medications (gabapentin, pregabalin), nonsteroidal anti-inflammatory medications, a short course of oral corticosteroids, injection of epidural corticosteroids, and physical therapy. Physical therapy treatment regimens may initially begin with extension and isometric exercises, as flexion places additional stress on the intervertebral discs [13]. Surgical intervention is indicated more urgently for neurologic weakness or myelopathy, or after a failure of conservative treatment for radicular pain.

Prognosis: Return to play should be delayed until the patient has pain relief and return of normal range of motion in those managed conservatively, typically 3 months for those playing contact sports [13]. Ninety percent of athletes treated with lumbar discectomy were able to return to play for single-level disease [14], and return to play should be delayed 3 months after surgery to prevent risk of reherniation [15].

41.6.3 Spondylolisthesis and Spondylolysis

Definition: Slippage of one vertebral body relative to another is termed spondylolisthesis. When this occurs due to a defect or break in the pars interarticularis (Fig. 41.3), it is termed spondylolysis (Fig. 41.4). The superior vertebral body will slip forward (anterolisthesis) or backward



Fig. 41.3 A computerized illustration of a pars interarticularis defect/break (Courtesy of Thomas J Buell. Used with permission)

(retrolisthesis) as compared with the inferior body, creating kyphosis and foraminal stenosis at that segment.

Mechanism of injury: Spondylolisthesis can have a variety of causes: Isthmic (caused by a defect in the pars interarticularis after repetitive stress, especially extension), degenerative (caused by disc and facet joint degeneration which leads to ligamentous laxity), traumatic (caused by high impact injuries), pathologic

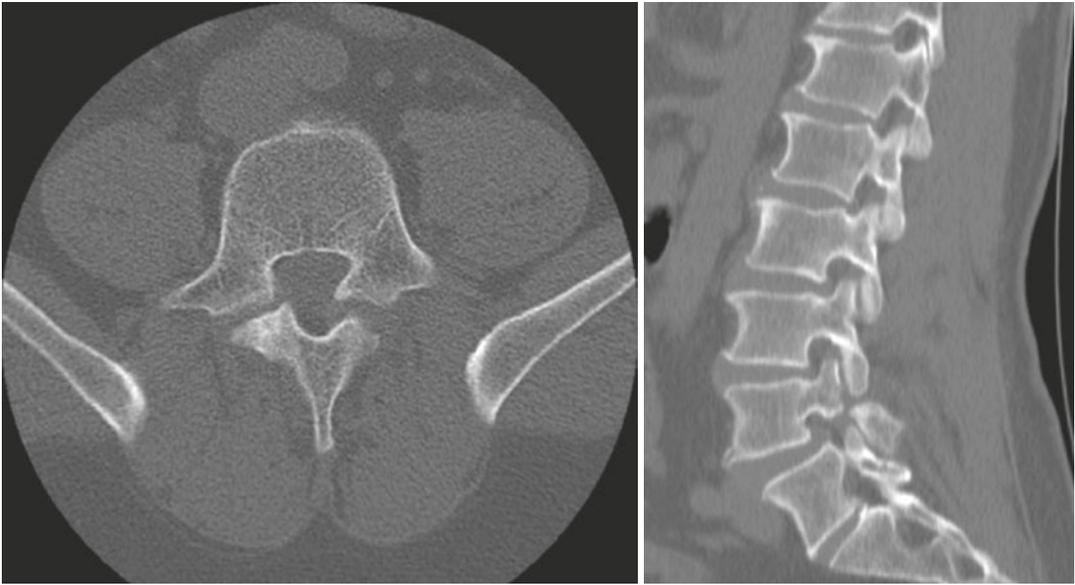


Fig. 41.4 L5 spondylolysis on axial (left) and sagittal (right) CT scan images

(caused by tumors or osteoporotic bone), or dysplastic (congenital abnormalities).

Isthmic spondylolisthesis is the most common. This begins as a stress reaction to repetitive motion (such as hyperextension, twisting, and axial loading as in gymnastics). The stress reaction can progress to a full stress fracture (spondylolysis) and can form a chronic fibrous nonunion (pseudarthrosis) which allows the vertebral body, pedicle, and superior facet to slip forward. Overgrowth of fibrous tissue in the area of the fracture at the pars interarticularis, which forms the posterior/superior corner of the neural foramen, will often contribute to foraminal narrowing and radicular symptoms.

Clinical symptoms: Patients may be asymptomatic initially and unaware of their defect, especially if they have a low-grade spondylolisthesis. Back pain in this condition is often the first symptom, is worse with activity, especially extension. Pain can be generated by the stress reaction in the pars interarticularis, which can progress to a stress fracture, and eventually abnormal slipping motion of the vertebral bodies (spondylolisthesis). There can be associated radicular pain from inflammation during a stress reaction, or, in a mobile

spondylolisthesis, from dynamic compression of the exiting nerve root in the foramen, or from compression of the traversing nerve roots in the lateral recess. Clinical symptoms may be related to the degree of slippage, which is graded into five categories based on how much of the length of the superior vertebral body has slipped forward beyond the inferior body. When the superior vertebrae completely slips forward beyond the inferior level, this is termed spondyloptosis.

Diagnosis: Spondylolisthesis is graded on upright, weight-bearing radiographs. Flexion and extension views will be helpful in assessing for additional motion. Oblique images will more clearly show the pars interarticularis and whether there is a break or fibrous nonunion. CT will also clearly reveal whether there is a bony defect. Supine imaging, such as on CT and MRI, can be compared to the standing radiographs, and if there is a change in the amount of slippage when going from supine to standing, the spondylolisthesis is mobile. Often, on MRI alone, it can be difficult to definitively identify a spondylolysis, especially one which reduces completely when supine, depending on the thickness of the sagittal imaging.

Nuclear imaging with a bone scan can identify a stress reaction which has not yet progressed to a fracture.

Patients may have a palpable step-off on palpation of the spinous processes. They will often demonstrate exaggerated lumbar lordosis and lumbar muscle spasm.

Bertolotti's syndrome consists on the presence of a transition vertebra which can cause a conflict with the sacrum or ilium due to a hyperplastic transverse process or even in changes in the mobility of the lumbar spine which may trigger symptomatic discopathies, leading also to LBP. It affects 3–9% of the population and sometimes can be confused with spondylolysis. Treatment consists of pain management and control, which can be attainable with rest and/or an orthosis. The role of physical rehabilitation is important, not neglecting the sport-specific skills. Surgical treatment is not advisable for this condition.

Treatment: Depends on the stage of injury to the pars interarticularis and grade of spondylolisthesis at the time of diagnosis. Bracing could be considered when a stress reaction is detected, or when an acute stress fracture is visible in CT imaging. If there is sclerosis at the fracture line, bracing will no longer be effective, and there is a fibrous nonunion.

Rest and participation in rehabilitation, once the patient is completely pain-free, are indicated. Surgery is indicated for intractable pain or neurologic symptoms in a mobile spondylolisthesis. Single-level fusion (Fig. 41.5) or direct pars repair are surgical options.

Prognosis: In nonoperative management, athletic activities should be stopped until lumbar extension is pain-free (often 2 months or more) [16]. Direct pars repair for spondylolysis without significant spondylolisthesis may preserve range of motion as compared with lumbar fusion, and in one series of four patients treated with direct pars repair, all had returned to training at 6 months, and competition by 1 year post surgery [17].

41.6.4 Vertebral Stress Fractures

There are many types of stress fractures; the majority of them occur in poor-conditioned ath-

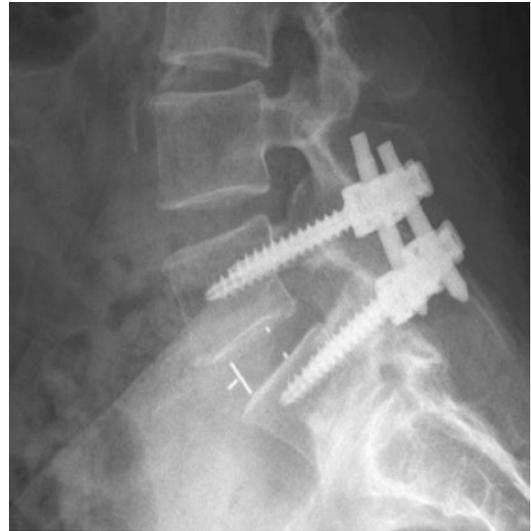


Fig. 41.5 Minimally invasive single-level transforaminal lumbar interbody fusion (MI-TLIF)

letes or in those subjected to heavy loads. While the majority of stress fractures involve the lower extremities, they can occur in vertebral bodies and are not uncommon in basketball. In a recent epidemiologic study focusing on stress fractures in collegiate student-athletes over 10 seasons, lower back/lumbar spine/pelvis region was the third most common location with 12.1% of all stress fractures [18]. Out of these, the rate in male basketball players was reported to be 8.3% while the rate in female players was 4.5%. Spondylolysis can be considered as part of the stress fracture spectrum, but other types include vertebral endplate fractures in adolescents due to growth plate and undeveloped ossification centers and vertebral body stress fractures. Stress fractures result from repeated submaximal loads causing fatigue of the bone structures. These fractures occur when the stress applied to the bone is greater than the bone's capacity to remodel and heal. Bone turnover depends on genetic, hormonal, mechanical, and nutritional factors. The repetitive microdamage and the incapacity to maintain appropriate skeletal repair (fatigue reaction or fracture) are characteristics of stress fractures in the athlete. The true prevalence of stress fractures is unknown since the majority of them are underdiagnosed.

Plain radiographs are usually normal; however, an MRI or a bone scan can detect the fracture as early as few days after its occurrence. A CT scan can also provide good accuracy for detecting vertebral stress fractures. Treatment should consist of a period of rest and physical rehabilitation; in some cases surgical treatment is required in order to stabilize the fracture site with vertebroplasty or kyphoplasty.

41.6.5 Annular Tears

Definition: A tear in the annulus fibrosis, with or without herniation of disc material.

Mechanism of injury: Repetitive rotation. Causes inflammatory radicular pain.

Clinical symptoms: Back pain, with or without inflammatory radicular pain without neurologic deficits. Often will be associated with lumbar paraspinal muscle spasms.

Diagnosis: MRI may show bright signal at the site of the tear on the T2-weighted images. Weight-bearing imaging may show straightening of the spine from paraspinal muscle spasm.

Treatment: Steroids or nonsteroidal anti-inflammatory drugs (NSAIDs), physical therapy with core strengthening.

41.6.6 Inflammatory Syndromes

Definition: Pain arising from inflammation of the facet joints or sacroiliac (SI) joint.

Mechanism of injury: Inflammation of the synovial lining in these joints, with or without cartilage damage. Sacroiliitis is common after repetitive jumping or single leg standing/landing, as is common in basketball.

Clinical symptoms: Facet inflammation: Back pain, especially with extension, rotation, and bending. Sacroiliitis: pain overlying the SI joint, which may be associated with an inflammatory sciatica. Several provocative maneuvers: FABER testing, pain with palpation, and pain with extension may suggest sacroiliac dysfunction.

Diagnosis: Plain radiographs may show degenerative arthritis at the facet joints or SI joint in advanced cases. Facet joint inflammation may

be visible as a site of increased activity on bone scan.

Treatment: Nonsteroidal anti-inflammatory drugs (NSAIDs) and image-guided steroid injections into the affected joint are usually quite effective.

41.7 Return to Play Considerations

Return to play criteria is dependent on the diagnosis and treatment. Any injury with a neurologic component (motor weakness, numbness extending into the arms or legs) requires urgent assessment by a spine specialist.

While no specific guidance exists about return to play after lumbar injuries in basketball, as in other contact sports, players should be pain-free with no neurological deficit. For patients treated with a fusion, evidence of bone fusion should be visible [19].

College basketball players were found to be 1.61 times more likely than age-matched counterparts to have multilevel disc degeneration on imaging, with 81% of basketball players reported experiencing low back pain and 28% reporting radicular pain during their playing career [11]. This may complicate standard criteria for return to play (such as complete pain freedom). However, in general, it is recommended that players would meet the following four criteria for safe return to play: they should be free of pain, have full range of motion, full strength, and no neurologic injury [20]. Successful completion of a structured rehabilitation program tailored to the demands of the player's position is also recommended [21].

41.8 Athletic Performance After Low Back Injuries and Treatment

In an analysis of 61 NBA players diagnosed with lumbar disc herniation, 34 underwent discectomy and 27 were managed conservatively. Players were equally likely to return to play despite operative and nonoperative treatment (77.8% vs. 79.4%) [22]. Players treated operatively played

significantly fewer games and had a significantly decreased player effectiveness rating in the season following their injury; however, there were no differences in the second postinjury season. Patients treated operatively had no significant decrease in their overall career length compared to non-injured patients, whereas injured patients treated nonoperatively did have a significant decrease in their career length (3.25 vs. 5.79 seasons).

Take Home Message

Low back injuries are common in basketball due to the biomechanical stressors associated with play. Accurate diagnosis is the most critical factor for successful treatment and return to play. Acute lumbar strain is the most common injury and carries a good prognosis. However, any chronic pain or injury presenting with neurologic symptoms should prompt evaluation by a qualified spine specialist.

References

1. Frymoyer JW, Cats-Baril WL. An overview of the incidences and costs of low back pain. *Orthop Clin North Am.* 1991;22:263–71.
2. Ong A, Anderson J, Roche J. A pilot study of the prevalence of lumbar disc degeneration in elite athletes with lower back pain at the Sydney 2000 Olympic Games. *Br J Sports Med.* 2003;37:263–6.
3. Dick R, Hertel J, Agel J, Grossman J, Marshall SW. Descriptive Epidemiology of Collegiate Men's Basketball Injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 Through 2003–2004. *J Athl. Train.* 42, 194–201.
4. Andreoli, C. V. et al. Epidemiology of sports injuries in basketball: integrative systematic review. *BMJ Open Sport Amp Exerc. Med.* 4, e000468 (2018).
5. Delitto A, Erhard RE, Bowling RW. A treatment-based classification approach to low back syndrome: identifying and staging patients for conservative treatment. *Phys Ther.* 1995;75:470–85.; discussion 485–489.
6. Fritz JM, George S. The use of a classification approach to identify subgroups of patients with acute low back pain. Interrater reliability and short-term treatment outcomes. *Spine.* 2000;25:106–14.
7. Starkey C. Injuries and illnesses in the national basketball association: a 10-year perspective. *J Athl Train.* 2000;35:161–7.
8. Agel J, Olson DE, Dick R, et al. Descriptive epidemiology of collegiate women's basketball injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. *J Athl Train.* 2007;42:202–10.
9. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the national basketball association: a 17-year overview. *Sports Health.* 2010;2(4):284–90.
10. Jensen MC, Brant-Zawadzki MN, Obuchowski N, Modic MT, Malkasian D, Ross JS. Magnetic resonance imaging of the lumbar spine in people without back pain. *N Engl J Med.* 1994;331:69–73.
11. Hangai M, Kaneoka K, Hinotsu S, Shimizu K, Okubo Y, Miyakawa S, et al. Lumbar intervertebral disk degeneration in athletes. *Am J Sports Med.* 2009;37:149–55.
12. Hart LG, Deyo RA, Cherkin DC. Physician office visits for low back pain. Frequency, clinical evaluation, and treatment patterns from a U.S. national survey. *Spine.* 1995;20:11–9.
13. Eck JC, Riley LH. Return to play after lumbar spine conditions and surgeries. *Clin Sports Med.* 2004;23:367–79.
14. Wang JC, Shapiro MS, Hatch JD, Knight J, Dorey FJ, Delamarter RB. The outcome of lumbar discectomy in elite athletes. *Spine.* 1999;24:570–3.
15. Mochida J, Nishimura K, Okuma M, Nomura T, Toh E. Percutaneous nucleotomy in elite athletes. *J Spinal Disord.* 2001;14:159–64.
16. Sys J, Michielsen J, Bracke P, Martens M, Verstreken J. Nonoperative treatment of active spondylolysis in elite athletes with normal X-ray findings: literature review and results of conservative treatment. *Eur Spine J.* 2001;10:498–504.
17. Reitman CA, Esses SI. Direct repair of spondylolytic defects in young competitive athletes. *Spine J.* 2002;2:142–4.
18. Rizzone KH, Ackerman KE, Roos KG, Dompier TP, Kerr ZY. The epidemiology of stress fractures in collegiate student-athletes, 2004–2005 through 2013–2014 academic years. *J Athl Train.* 2017;52(10):966–75.
19. Schnebel BE. Thoracic and lumbosacral spine injuries. In: *Netter's sports medicine.* 2nd ed. Philadelphia, PA: Elsevier; 2018. p. 415–424.e2. <https://www-clinicalkey-com.proxy.lib.duke.edu/#!/content/book/3-s2.0-B9780323395915000532?scrollTo=%23h10000758>. Accessed 3 Nov 2019.
20. Huang Y-F, Cherng Y-G, Hsu SPC, Yeh C-C, Chou Y-C, Wu C-H, et al. Risk and adverse outcomes of fractures in patients with Parkinson's disease: two nationwide studies. *Osteoporos Int.* 2015;26:1723–32.
21. Li Y, Hresko MT. Lumbar spine surgery in athletes: outcomes and return-to-play criteria. *Clin Sports Med.* 2012;31:487–98.
22. Minhas SV, Kester BS, Hsu WK. Outcomes after lumbar disc herniation in the National Basketball Association. *Sports Health.* 2016;8:43–9.



Osteoarthritis in Basketball Players

42

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and Annunziato Amendola

42.1 Introduction

Osteoarthritis is a condition most commonly associated with the aging population, but it is also the one that is critical for the sports physician to understand in athletes. Arguably the most common of joint disorders and the most prevalent form of arthritis, osteoarthritis is a disease of primarily the articular cartilage, and also includes the adjacent subchondral bone and soft tissues of the joint that poses not only a physical burden to those affected but also a significant financial burden for the health-care system [1]. When dealing with basketball players in particular, it is critical to not only understand how to manage already established osteoarthritis in older players but also how to identify and treat the potentially modifiable conditions in younger players who may be prone to developing osteoarthritis at an advanced rate when compared with non-athletes of similar age. Although data is scarce in terms of overall prevalence of osteoarthritis specifically in basketball players, there are studies suggesting that the anterior knee pain found in up to one-fourth of

youth basketball participants has a patellofemoral component that is suggestive of underlying degenerative changes [2]. While some risk factors for osteoarthritis are non-modifiable, understanding the disease process and potential points of intervention is key. Optimization of the joint health in basketball athletes is critical to both their statistical production on the court and longevity in the sport and should be a high priority for the medical teams.

42.2 Anatomy and Physiology

The underlying mechanism behind osteoarthritis is the degeneration of articular cartilage, which leads to decreased joint lubrication and an increase in force transmission to the subchondral bone. Cartilage is composed primarily of water, and along with collagens, proteoglycans, extracellular matrix, and other proteins, it is able to withstand repeated cyclic loads without damage [3]. The three primary zones that comprise cartilage each contain a varying composition of cells (Fig. 42.1) [4]. The superficial zone, which comprises 10–20% of the total cartilage volume, provides protection from shear stress due to the presence of tightly packed collagen fibers and low levels of aggrecan. This level is also important for the secretion of joint lubrication fluid that is crucial for proper joint function. The middle zone, also named transitional zone, comprises the bulk of articular car-

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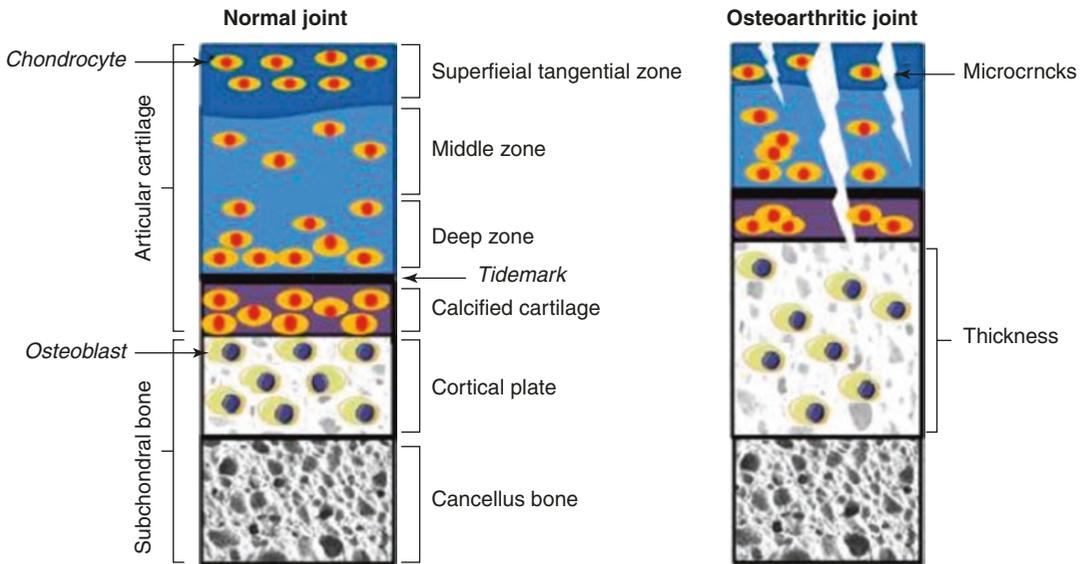


Fig. 42.1 Zones of Cartilage in Normal and Osteoarthritis Joint [4]. Obtained from Sharma AR, Jagga S, Lee S-S, Nam J-S. Interplay between cartilage and subchondral

bone contributing to pathogenesis of osteoarthritis. *Int J Mol Sci.* 2013;14(10):19805–19830

tilage at 40–60% and contains a random collection of collagen fibers that serve to oppose compressive forces. Finally, the deep zone also serves to resist compressive forces with the largest collagen fibrils, and also connects with the underlying calcified zone and makes up approximately 30% of articular cartilage [5].

When cartilage is loaded, fluid shifts occur allowing for a distribution of force that mitigates impact, however, when the load is applied quickly, as with the majority of basketball-related movements, the cartilage is not given ample time to distribute the force effectively [6]. The repetitive nature of this loading in basketball play leads to further cartilage degeneration, which takes the form of zone composition and hydration changes at the cellular level and will manifest as pain and disability in the player [3].

42.3 Biomechanics

The amount of force loading on individual joints and compartments within a joint has been biomechanically determined through multiple studies and can provide insight to the development of

Table 42.1 Joint forces during common activities

Activity	Joint	Forces
Walking	Tibiofemoral	3.4× body weight
Going upstairs	Tibiofemoral	4.3× body weight
Walking downhill	Tibiofemoral	8.5× body weight
Jumping	Patellofemoral	4.6× body weight
Jumping	Tibiofemoral	9.0× body weight
Jumping	Hip	8.4× body weight
Jumping	Tibiotalar	10× body weight

osteoarthritis. The medial compartment of the knee is often faced with the highest contact forces due to the external knee adduction moment and is frequently the first compartment to wear, as the mechanical axis passes through the medial compartment in many knees [7]. The actual numerical forces seen by the tibiofemoral joint have been reported to be 3.4× the body weight when walking, 4.3× when going up stairs, and 8.5× when walking downhill [7]. When jumping, there up to 4.6× body weight in the patellofemoral joint, while it is higher in the tibiofemoral joint; 9×, the hip 8.4× and the ankle 10× body weight [8] (Table 42.1). Since basketball is a jumping sport, repetitive supraphysiologic loads may lead to early degenerative changes in weightbearing joints.

42.4 Risk Factors

The risk factors for osteoarthritis in basketball players can be divided into two broad categories, non-modifiable and modifiable. The non-modifiable risk factors are those that are present in all patients, not just the basketball athlete, and include demographic factors of race, gender, and age as well as genetics (Table 42.2). Epidemiologic studies have suggested that women are at higher risk for osteoarthritis than men, and African Americans are the most at-risk race. In terms of genetic association with osteoarthritis, most studies agree that there is a genetic component involved to developing osteoarthritis; however, the extent of that connection is debated and a wide range of risk statistics have been reported [9, 10]. A player's natural varus/valgus alignment at the knee for example can play a major role in the early progression of osteoarthritis as changes of just 5° can have a clinical impact [11].

Potentially modifiable risk factors again may affect all individuals, but there are some that are more common in high-level athletes, see Table 42.2. The significance of these modifiable risk factors may vary related to the specific sport or even by position within a single sport. For example, in basketball the center is generally larger than a point guard and may be at a higher risk for osteoarthritis based on a larger size and probably higher BMI [9, 12].

The most widely accepted and clear connection between modifiable risk factor and osteoarthritis is prior injury status, which is common in basketball athletes [13–17]. Of all adults diagnosed with osteoarthritis, regardless of activity participation, it is estimated that approximately 12% have sustained an injury that started the pro-

cess, and this is even considered to be a conservative number [14]. When considering the ankle alone, up to 90% of arthritic change may be attributed to a prior traumatic injury which is a staggering number considering the ankle is the most commonly injured joint in athletics. It is estimated that up to 22.6% of all injuries in high school and collegiate athletes involves the ankle, and it is the most common injury in all basketball players [14, 18].

Similarly, meniscal injuries of the knee are very common among basketball athletes, comprising almost 10% of all knee injuries in NBA players. There is a well-studied and documented connection between the increased incidence of arthritis and meniscal injury. Meniscal injuries are a major contributor to early onset osteoarthritis as their loss decreases the contact area of the femur and thus increases the forces transmitted to the articular cartilage [7, 19, 20]. Although studies have shown that both the medial and lateral meniscectomy procedures lead to progression of OA in the respective compartment, there is no statistical difference between the two, while the medial procedure is more common, therefore more medial compartment OA progression is encountered [21]. Taking all knee injuries in account, meniscal injuries represent the fourth most common injury site in NBA players, yet account for the most playing time lost [22, 23].

Estimates of articular cartilage injuries vary considerably depending on the sport, with approximately one-third of distance runners having a chondral lesion on magnetic resonance imaging (MRI), primarily on the lateral patella, in contrast to the 61% of players at the National Football League Combine and 81% of National Collegiate Athletic Association (NCAA) basketball players [7, 24, 25]. A significantly higher number of the distance runners also showed a pattern of bone edema without accompanying chondral lesion, located primarily in the tibia and patella [25]. Basketball places a very high demand on the articular knee cartilage because of the need for constant pivoting and jumping, and cartilage injuries are ubiquitous among high-level basketball players [26]. MRI studies of asymptomatic collegiate and professional basketball

Table 42.2 Modifiable and non-modifiable risk factors for osteoarthritis

Modifiable risk factors	Non-modifiable risk factors
Weight	Age
Prior injury status	Race
Activity	Gender
Nutrition status	Genetics
Bone mineral density	
Gait mechanics	

players have reported incidence of chondral injuries in 81% and 50%, respectively [24, 27].

The root cause of injury leading to cartilage wear is thought to be associated with an alteration in joint biomechanics which leads to cartilage being exposed to further injury [28]. This theory suggests that the joint architecture is constructed in a particular way that is most effective in absorbing and distributing a given load. Injury to the joint disrupts this biomechanical equilibrium and shifts the load to areas of the joint that were not meant to support it based on instability and incongruity. There is a particularly strong link between joint injuries that require surgery, commonly seen in knees and ankles [17]. These injuries place players at a significantly higher risk of osteoarthritis within 10–20 years of injury, even if players remain asymptomatic for years. Please refer to Table 42.2 below for a more complete list of risk factors.

42.5 Presentation in Basketball Athletes

Distinguishing osteoarthritis in the basketball athlete is a difficult task, as pain is the most common initial symptom and can be easily attributed to any number of sports-related activities such as muscle fatigue. Further complicating this particular issue is many of the athletes with early osteoarthritis are asymptomatic, which can lead to further cartilage degeneration as a player continues to perform the same detrimental activities without proper intervention. The treating physician should suspect potential pain related to cartilage injury or early osteoarthritis in the athlete when they complain of persistent dull joint pain, particularly with no identifiable incident such as after noncontact injury mechanism. Complaints of swelling, morning stiffness, and “my joints make noise” should also arouse suspicion as these symptoms are further suggestive of an arthritic process. The most commonly involved joints for basketball players are the knees and ankles, followed by the hips and shoulders, primarily due to the injury incidence and biomechanics as previously described.

The distribution of knee compartment wear may vary based on some of the risk factors out-

lined above and present in either the medial, lateral, patellofemoral compartments, or a combination. Localized pain along the medial or lateral joint line as well as anterior pain may signify the underlying compartment affected, respectively. If a player had a prior meniscal or chondral injury, in whichever compartment the injury occurred is likely to develop arthritis at an advanced rate. Additionally, if a player’s knee is biomechanically disposed due to alignment to placing additional stress on any one of the compartments, as evidenced on plain radiographs, that too will see an advanced wear pattern accompanied by an earlier onset arthritis.

In the ankle, a serious sprain commonly leads to an osteochondral lesion within the tibiotalar joint in 95% of cases as the medial tibial plafond contacts the medial talar dome, starting the arthritic cascade [14]. A medial sided talar lesion is twice as likely as the lateral side with a common ankle sprain, with estimates of 1/2 of the OCDs leading to OA down the road [14]. This injury pattern leads to pain felt “deep” in the ankle, often accompanied with symptoms of instability and sometimes mechanical clicking. Less commonly, the lesion may be seen in tibiofemoral or subtalar regions. The proper identification of athletes who may be experiencing osteoarthritis-related pain based on presentation is important in order to proceed to the proper diagnostic evaluation.

42.6 Diagnosis and Imaging

The diagnosis of osteoarthritis in basketball athletes routinely involves a combination of history, physical examination, and imaging. As outlined above, players will complain of pain and stiffness in the joints. On physical examination, players will generally present with a combination of the following in their painful joint: crepitus, tenderness, or effusion. A history and physical examination is commonly followed by diagnostic imaging, of which there are many useful options that present different diagnostic strengths and weaknesses in assessing osteoarthritis.

Radiographs are widely used as the initial imaging to assess osteoarthritis based on their ease to obtain and interpret, relative cost-effectiveness, and minimal risk to the patient. The five radiographic signs used to establish the diagnosis of osteoarthritis are as follows [29] (Fig. 42.2a, b):

1. Osteophyte formation at the joint margins.
2. Periarticular ossicles.
3. Narrowing of the joint space with subchondral sclerosis.
4. Subchondral cysts.
5. Changes in the shape of bone ends at the joint, such as flattening of the femoral condyles in the knee.



Fig. 42.2 Radiograph of Osteoarthritic Knee. Radiographs demonstrating advanced osteoarthritis in 41-year-old former professional basketball player. (a) AP view with medial compartment narrowing. (b) Merchant view of the patellofemoral view demonstrating mild joint space narrowing and osteophytes

Additionally, when a player presents with symptoms and signs typical for arthritis or the early stages of arthritis, the use of special radiographic views and measurements can be helpful in identifying abnormalities that may predispose to knee injury and ultimately the development or progress of osteoarthritis. One such measurement, the Caton-Deschamps index of the knee, compares the length of the patella articulating surface and the distance from the inferior pole of the patella to the tibia as a means of evaluating the position of the patella. Patella positioning is key for proper knee biomechanics and can be predictive of improper force distribution, potentially accelerating an arthritic process, although many players do have abnormal patella positioning on radiographs and do not progress to arthritis as a result. Radiographs represent just one piece of the overall evaluation and should be evaluated in the proper context. Full-length lower extremity radiographs (hip-knee-ankle angle measurement) also provides valuable biomechanical information in regard to the varus/valgus alignment of the lower extremity. While useful for mild/moderate to severe cases, radiographs are less effective in early osteoarthritis and their utilization alone may lead to a missed diagnosis and opportunity for early intervention.

Not traditionally thought of as a means of arthritis diagnosis and evaluation, ultrasound (US) has emerged as a viable diagnostic option. As a low cost, non-invasive modality that can be performed easily at bedside in the clinic, ultrasound has been used for a number of indications involving arthritis due to its ability to detect changes in multiple tissues. Primarily, US can be effective in the detection of effusion, synovial thickness, synovitis, cartilage architecture, osteophytes, cystic lesions, and for the monitoring of osteoarthritis progression [30]. As a safe, non-invasive modality, US can be used to detect significant findings or evaluate responses to intervention without the side effects other imaging may present. In evaluating early osteoarthritis, changes in the articular cartilage can be observed on US as blurring of cartilage edges, with more advanced disease presenting as joint space narrowing and changes in the echogenicity.

The disadvantage of ultrasound is mainly the operator-dependent nature of the study, the lack of access to all sections of most joints, and the significant learning curve to effectively perform the exam [30–32]. Additionally, a lack of established grading mechanisms for findings such as those detected on plain radiographs and MRI makes interobserver reliability difficult to ascertain [30].

The gold standard diagnostic modality to evaluate focal cartilage injuries in younger athletes is MRI. One of the many roles of MRI is the detection of radiographically occult chondral lesions that can predispose to osteoarthritis if left untreated. Unlike radiographs, MRI does not produce harmful radiation and also allows for superior evaluation of the soft tissue. MRI also allows direct visualization of the other joint structures including ligaments and menisci. Multiple scoring systems have been developed using MRI, of which the International Cartilage Regeneration and Joint Preservation classification for focal cartilage lesions is one of the more widely recognized which allows for ease of interpretation and comparisons between researchers and a clear mechanism to track progression. Additionally, other classification systems such as the ICRS scale exist which rely on direct arthroscopic views of the lesion (Table 42.3).

The quality images for an effective MRI is dependent on field strengths. Field strengths must be at least 1 T and 1.5 T is preferable for morphological and compositional assessment. More modern scanners including 3 T provides a twofold increase in signal-to-noise ratio and spatial resolution, and a fourfold reduction in imaging time. T2-weighted sequences among conventional 2-D spin-echo sequences provide the optimal visualization with cartilage depicted as an intermediate signal (gray) in the backdrop of hyperintense (bright) synovial fluid. The limitations of conventional MRI imaging are cost and potential technical difficulties in performing examination on younger children who are unable to tolerate sitting still for the entirety of the procedure [34].

Novel MRI techniques are being developed for a quantitative assessment of cartilage. They

Table 42.3 ICRS Cartilage Injury Scale [33]

ICRS score	Cartilage findings
0	Normal cartilage
1	Near normal cartilage surface
2	Defect less than 50%, partial thickness
3	Defect greater than or equal to 50%, partial thickness
4	Full thickness defect

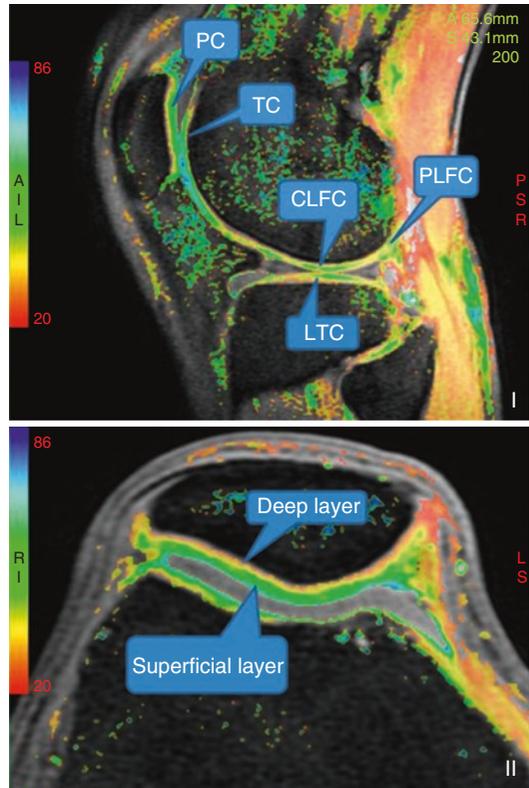


Fig. 42.3 T1 Rho Imaging of Knee Cartilage [35]: (I) sagittal and (II) axial views. Scales on the left with blue indicating healthy cartilage and red indicating decreased proteoglycan content (signaling worn cartilage). PC patella cartilage, TC trochlear cartilage, CLFC central lateral femoral condyle, LTC lateral tibial cartilage, PLFC posterior lateral femoral cartilage. Obtained from Chen M, Qiu L, Shen S, et al. The influences of walking, running, and stair activity on knee articular cartilage: Quantitative MRI using T1 rho and T2 mapping. *PLoS One*. 2017;12(11):e0187008

include collagen assessment (T2 mapping) and proteoglycan assessment (T1 rho, dGEMRIC, Fig. 42.3) [36]. These biochemical assessment techniques are capable of detecting the macro-

molecular changes within the cartilage which occur very early on in the development of actual morphological changes of cartilage thinning and focal defects [37, 38]. These techniques are labor-intensive and currently remain in the research realm, but as techniques are refined, they will become more clinically available in future and may change the diagnostic evaluation and treatment of early osteoarthritis.

42.7 Treatment

The treatment algorithm for osteoarthritis in basketball players is primarily based on symptoms. The initial preference for all basketball players is nonsurgical managements, with surgical options on a condition-specific basis and depending on severity.

Any athlete still participating in competitive basketball should be treated with the least invasive approach possible that will allow for continued participation while limiting the progression of osteoarthritis for the future. The mainstay initial treatments are a combination of non-steroidal anti-inflammatory medications, physical therapy with a focus on quadriceps/hamstrings/and core strengthening, orthotics, and functional bracing if biomechanics are the issue [39–41]. Moreover, low impact exercises that allow for strengthening without additional stress including pool workouts, elliptical machine, and stationary bikes are best for all forms of arthritis, particularly patellofemoral joint arthritis in the knee. If not adequately controlled, the next step in treatment progression may be the use of injectables and orthobiologics.

A corticosteroid injection directly to the symptomatic joint allows for pain-free intervals that may allow a player to make it through the season or an especially symptomatic time frame. These injections are generally given in 3-month intervals with total injection number varying by provider and continued response to treatment. Studies have suggested the long-term use of corticosteroid injections to be safe although a concern about the long-term effect on chondrocytes is a point of discussion and controversy [42]. An

alternative injection option is hyaluronic acid, which is based on the theory that arthritic joints are deficient in this acid and direct supplementation can restore pain-free functionality to the joint. Evidence exists that hyaluronic acid is beneficial for articular cartilage and delays degradation, with minimal side effects [43–47]. These injections are intended to last longer than standard corticosteroid injections although the data on their effectiveness is mixed [48, 49]. An emerging set of therapeutic injections are the orthobiologics, which consist of therapies including platelet-rich protein (PRP), stem cells, autologous conditioned serum, and adipose-derived stromal cell therapy, among others.

PRP therapy is a topic of much discussion and controversy due to its promise as a safe, natural treatment for osteoarthritis. Based on the fact that avascular cartilage requires additional factors to assist in healing and regeneration, PRP provides a local injection of highly concentrated, important natural proteins and growth factors spun from the patient's own blood that may diffuse into the cartilage and assist in healing and repair [50]. Although PRP is still debated in its efficacy, recent meta-analyses and systematic reviews have found PRP injections to provide pain relief and functional benefits for patients when compared with placebo [51]. Similarly, stem cells or bone marrow aspirate concentrate (BMAC) are a treatment modality that also utilize the patient's own cells for regeneration and repair. Using bone marrow aspirate from the iliac crest, or less commonly the proximal humerus or tibia, BMACs are high in mesenchymal stem cells that are one of the primary cells involved in cartilage repair [50]. Stem cell and adipose-tissue-derived therapies have been extensively studied and are a popular topic in the future course of medicine, but to this point the literature on their efficacy in treating osteoarthritis is controversial at best [52, 53]. Despite the current lack of high-level evidence for its use, stem cell treatment presents another potential option for the treatment of osteoarthritis in basketball players that may provide both symptomatic and functional improvement. Orthobiologic treatment options for cartilage damage are extensively discussed in a separate

chapter in this book. In cases where a young athlete has an identifiable cartilage lesion or biomechanical issue that can be addressed to help prevent the onset or progression of arthritis, surgery may be indicated.

As outlined above, a commonly encountered issue in young basketball athletes is osteochondral injury, which is a precursor for early onset osteoarthritis. Depending on the character of the lesion, including size, involved bone, and player demographics, multiple surgical options exist ranging from microfracture to osteochondral allografts and autografts, which may allow return to high-level basketball in up to 80% of the cases [35, 54, 55]. Although used less frequently today, microfracture is a treatment option that allows for early return to play, but players must be counseled on the potential long-term sequela including the early development of osteoarthritis (Fig. 42.4a, b). In athletes with improper lower limb alignment, particularly of the knee, osteotomy may be a solid option although ability to return to basketball is less widely reported and is comprised primarily of case series involving amateur athletes [56]. In cases of severe isolated single compartment osteoarthritis of the knee, unicompartmental (Figs. 42.5a–d) or patellofemoral joint replacement may be considered. Returning to high-level activity following a unicompartmental joint replacement is often dis-

couraged due to concerns over polyethylene wear, component loosening, potential for infection although there are reports of successful return to play at amateur and recreational levels with partial knee replacements [57, 58].

When a player is ready to retire, the treatment algorithm will include more definitive surgical options. While all of the more conservative measures outlined previously still apply, the retired basketball athlete may consider total knee or total hip arthroplasty as a definitive solution. A suggested treatment algorithm is outlined below (Fig. 42.6).

42.8 Summary

While more commonly seen in the older population, osteoarthritis is an irreversible, painful condition that is common in active basketball players and can negatively affect the performance of those affected. Initially reported as nonspecific knee pain, stiffness, and periodic swelling, the symptoms of early osteoarthritis are commonly attributed to the pains of basketball participation. Through patient awareness, early recognition with risk modification and initiation of nonsurgical treatment, a player suffering with osteoarthritis may be able to effectively manage their symptoms while stemming the progression of osteoarthritis.

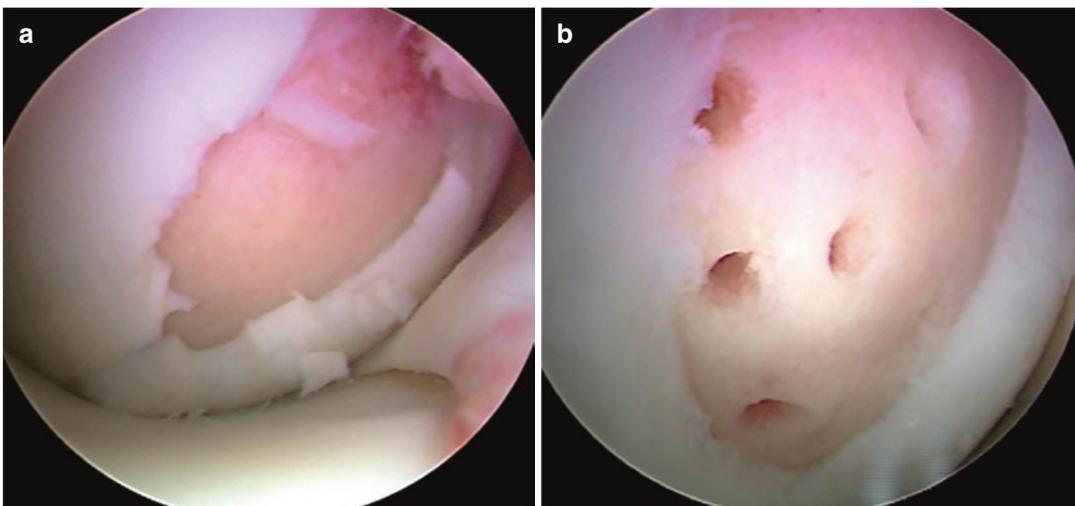


Fig. 42.4 Microfracture: (a) Chondral lesion debridement. (b) Penetration of subchondral bone

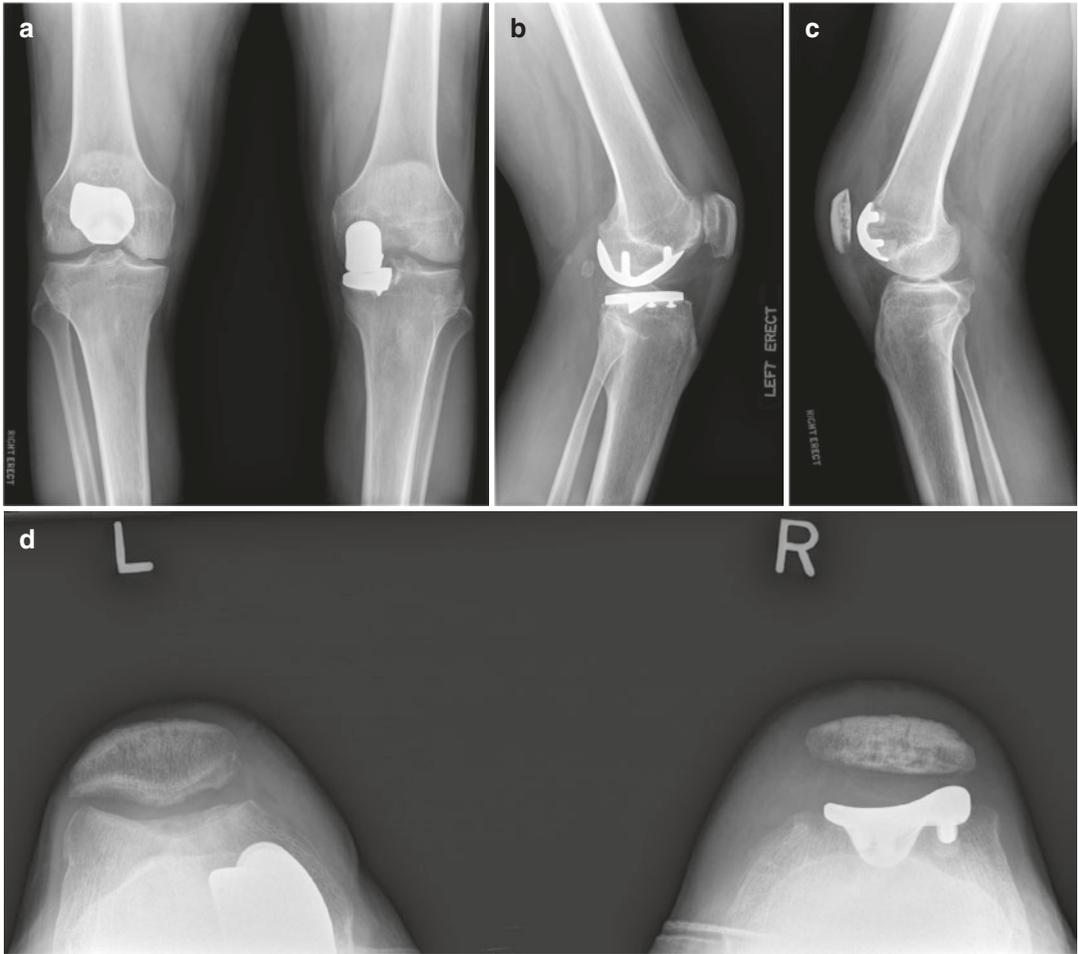


Fig. 42.5 Postoperative anteroposterior, lateral radiographs, merchant views of patellofemoral arthroplasty, and unicompartmental arthroplasty

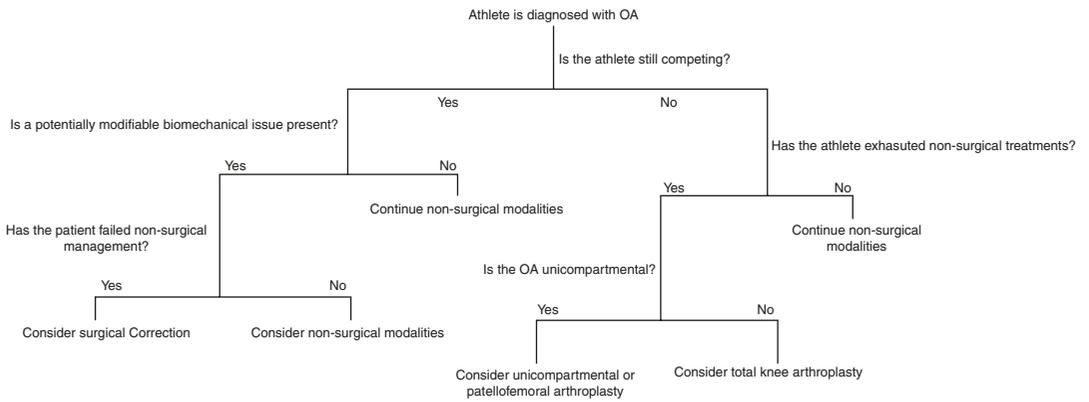


Fig. 42.6 Treatment algorithm for osteoarthritis in basketball players

References

- Hunter DJ, Felson DT. Osteoarthritis. *BMJ*. 2006;332(7542):639–42.
- Foss KDB, Myer GD, Magnusson RA, Hewett TE. Diagnostic differences for anterior knee pain between sexes in adolescent basketball players. *J Athl Enhanc*. 2014;3(1):1814.
- Sophia Fox AJ, Bedi A, Rodeo SA. The basic science of articular cartilage: structure, composition, and function. *Sports Health*. 2009;1(6):461–8.
- Sharma AR, Jagga S, Lee S-S, Nam J-S. Interplay between cartilage and subchondral bone contributing to pathogenesis of osteoarthritis. *Int J Mol Sci*. 2013;14(10):19805–30.
- Grogan SP, Duffy SF, Pauli C, et al. Zone-specific gene expression patterns in articular cartilage. *Arthritis Rheum*. 2013;65(2):418–28.
- Levangie PKNC. Joint structure and function: a comprehensive analysis. 3rd ed. Philadelphia PA: Davis.
- Masouros SD, AMJ B, Amis AA. A biomechanics of the knee joint. *Orthopaed Traum*. 2010;24(2):84–91.
- Cleather DJ, Goodwin JE, Bull AM. Hip and knee joint loading during vertical jumping and push jerking. *Clin Biomech*. 2013;28(1):98–103.
- Johnson VL, Hunter DJ. The epidemiology of osteoarthritis. *Best Pract Res Clin Rheumatol*. 2014;28(1):5–15.
- Vina ER, Kwok CK. Epidemiology of osteoarthritis: literature update. *Curr Opin Rheumatol*. 2018;30(2):160–7.
- Sharma L, Song J, Felson DT, Cahue S, Shamiyeh E, Dunlop DD. The role of knee alignment in disease progression and functional decline in knee osteoarthritis. *JAMA*. 2001;286(2):188–95.
- Reijman M, Pols HA, Bergink AP, et al. Body mass index associated with onset and progression of osteoarthritis of the knee but not of the hip: the Rotterdam study. *Ann Rheum Dis*. 2007;66(2):158–62.
- Conaghan PG. Update on osteoarthritis part 1: current concepts and the relation to exercise. *Br J Sports Med*. 2002;36(5):330–3.
- Delco ML, Kennedy JG, Bonassar LJ, Fortier LA. Post-traumatic osteoarthritis of the ankle: a distinct clinical entity requiring new research approaches. *J Orthop Res*. 2017;35(3):440–53.
- Maffulli N, Longo UG, Gougoulis N, Caine D, Denaro V. Sport injuries: a review of outcomes. *Br Med Bull*. 2011;97:47–80.
- Molloy MG, Molloy CB. Contact sport and osteoarthritis. *Br J Sports Med*. 2011;45(4):275–7.
- Smith MV, Nepple JJ, Wright RW, Matava MJ, Brophy RH. Knee osteoarthritis is associated with previous meniscus and anterior cruciate ligament surgery among elite college American football athletes. *Sports Health*. 2016;9(3):247–51.
- Tummala SV, Hartigan DE, Makovicka JL, Patel KA, Chhabra A. 10-year epidemiology of ankle injuries in men's and women's collegiate basketball. *Orthop J Sports Med*. 2018;6(11):2325967118805400.
- McDermott ID, Amis AA. The consequences of meniscectomy. *J Bone Joint Surg Br*. 2006;88(12):1549–56.
- Yeh PC, Starkey C, Lombardo S, Vitti G, Kharrazi FD. Epidemiology of isolated meniscal injury and its effect on performance in athletes from the National Basketball Association. *Am J Sports Med*. 2012;40(3):589–94.
- Longo UG, Ciuffreda M, Candela V, et al. Knee osteoarthritis after arthroscopic partial Meniscectomy: prevalence and progression of radiographic changes after 5 to 12 years compared with contralateral knee. *J Knee Surg*. 2019;32(5):407–13.
- Cumps E, Verhagen E, Meeusen R. Prospective epidemiological study of basketball injuries during one competitive season: ankle sprains and overuse knee injuries. *J Sports Sci Med*. 2007;6(2):204–11.
- Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the national basketball association: a 17-year overview. *Sports Health*. 2010;2(4):284–90.
- Pappas GP, Vogelsong MA, Staroswiecki E, Gold GE, Safran MR. Magnetic resonance imaging of asymptomatic knees in collegiate basketball players: the effect of one season of play. *Clin J Sport Med*. 2016;26(6):483–9.
- Stahl R, Luke A, Ma CB, et al. Prevalence of pathologic findings in asymptomatic knees of marathon runners before and after a competition in comparison with physically active subjects—a 3.0 T magnetic resonance imaging study. *Skelet Radiol*. 2008;37(7):627–38.
- Klusemann MJ, Pyne DB, Hopkins WG, Drinkwater EJ. Activity profiles and demands of seasonal and tournament basketball competition. *Int J Sports Physiol Perform*. 2013;8(6):623–9.
- Walczak BE, McCulloch PC, Kang RW, Zelazny A, Tedeschi F, Cole BJ. Abnormal findings on knee magnetic resonance imaging in asymptomatic NBA players. *J Knee Surg*. 2008;21(1):27–33.
- Andriacchi TP, Koo S, Scanlan SF. Gait mechanics influence healthy cartilage morphology and osteoarthritis of the knee. *J Bone Joint Surg Am*. 2009;91(Suppl 1):95–101.
- Kellgren JH, Lawrence JS. Radiological assessment of osteo-arthrosis. *Ann Rheum Dis*. 1957;16(4):494–502.
- Iagnocco A. Imaging the joint in osteoarthritis: a place for ultrasound? *Best Pract Res Clin Rheumatol*. 2010;24(1):27–38.
- Keen HI, Conaghan PG. Usefulness of ultrasound in osteoarthritis. *Rheum Dis Clin*. 2009;35(3):503–19.
- Wakefield RJ, Gibbon WW, Emery P. The current status of ultrasonography in rheumatology. *Rheumatology*. 1999;38(3):195–8.
- Brittberg M, Winalski CS. Evaluation of cartilage injuries and repair. *J Bone Joint Surg Am*. 2003;85-A(Suppl 2):58–69.
- Ashraf S, Zahoor A. Magnetic resonance imaging of articular cartilage. *JBJS Rev*. 2016;4(8)

35. Balazs GC, Wang D, Burge AJ, Sinatro AL, Wong AC, Williams RJ 3rd. Return to play among elite basketball players after osteochondral allograft transplantation of full-thickness cartilage lesions. *Orthop J Sports Med.* 2018;6(7):2325967118786941.
36. Chen M, Qiu L, Shen S, et al. The influences of walking, running and stair activity on knee articular cartilage: quantitative MRI using T1 rho and T2 mapping. *PLoS One.* 2017;12(11):e0187008.
37. Klocke NF, Amendola A, Thedens DR, et al. Comparison of T1rho, dGEMRIC, and quantitative T2 MRI in preoperative ACL rupture patients. *Acad Radiol.* 2013;20(1):99–107.
38. Taylor KA, Collins AT, Heckelman LN, et al. Activities of daily living influence tibial cartilage T1rho relaxation times. *J Biomech.* 2019;82:228–33.
39. Feeley BT, Gallo RA, Sherman S, Williams RJ. Management of osteoarthritis of the knee in the active patient. *J Am Acad Orthop Surg.* 2010;18(7):406–16.
40. Krohn K. Footwear alterations and bracing as treatments for knee osteoarthritis. *Curr Opin Rheumatol.* 2005;17(5):653–6.
41. Uthman OA, van der Windt DA, Jordan JL, et al. Exercise for lower limb osteoarthritis: systematic review incorporating trial sequential analysis and network meta-analysis. *BMJ.* 2013;347:f5555.
42. Raynauld JP, Buckland-Wright C, Ward R, et al. Safety and efficacy of long-term intraarticular steroid injections in osteoarthritis of the knee: a randomized, double-blind, placebo-controlled trial. *Arthritis Rheum.* 2003;48(2):370–7.
43. Abate M, Pulcini D, Di Iorio A, Schiavone C. Viscosupplementation with intra-articular hyaluronic acid for treatment of osteoarthritis in the elderly. *Curr Pharm Des.* 2010;16(6):631–40.
44. Abate M, Vanni D, Pantalone A, Salini V. Hyaluronic acid in knee osteoarthritis: preliminary results using a four months administration schedule. *Int J Rheum Dis.* 2017;20(2):199–202.
45. Bannuru RR, Natov NS, Dasi UR, Schmid CH, McAlindon TE. Therapeutic trajectory following intra-articular hyaluronic acid injection in knee osteoarthritis--meta-analysis. *Osteoarthr Cartil.* 2011;19(6):611–9.
46. Bellamy N, Campbell J, Robinson V, Gee T, Bourne R, Wells G. Viscosupplementation for the treatment of osteoarthritis of the knee. *Cochrane Database Syst Rev.* 2006;(2):Cd005321.
47. Wang CT, Lin J, Chang CJ, Lin YT, Hou SM. Therapeutic effects of hyaluronic acid on osteoarthritis of the knee. A meta-analysis of randomized controlled trials. *J Bone Joint Surg Am.* 2004;86(3):538–45.
48. Cooper C, Rannou F, Richette P, et al. Use of intra-articular hyaluronic acid in the management of knee osteoarthritis in clinical practice. *Arthritis Care Res.* 2017;69(9):1287–96.
49. Watterson JR, Esdaile JM. Viscosupplementation: therapeutic mechanisms and clinical potential in osteoarthritis of the knee. *J Am Acad Orthop Surg.* 2000;8(5):277–84.
50. Huebner K, Frank RM, Getgood A. Ortho-biologics for osteoarthritis. *Clin Sports Med.* 2019;38(1):123–41.
51. Riboh JC, Saltzman BM, Yanke AB, Fortier L, Cole BJ. Effect of leukocyte concentration on the efficacy of platelet-rich plasma in the treatment of knee osteoarthritis. *Am J Sports Med.* 2015;44(3):792–800.
52. Shapiro SA, Kazmerchak SE, Heckman MG, Zubair AC, O'Connor MI. A prospective, single-blind, placebo-controlled trial of bone marrow aspirate concentrate for knee osteoarthritis. *Am J Sports Med.* 2016;45(1):82–90.
53. Wakitani S, Imoto K, Yamamoto T, Saito M, Murata N, Yoneda M. Human autologous culture expanded bone marrow mesenchymal cell transplantation for repair of cartilage defects in osteoarthritic knees. *Osteoarthr Cartil.* 2002;10(3):199–206.
54. Cerynik DL, Lewullis GE, Joves BC, Palmer MP, Tom JA. Outcomes of microfracture in professional basketball players. *Knee Surg Sports Traumatol Arthrosc.* 2009;17(9):1135–9.
55. Harris JD, Walton DM, Erickson BJ, et al. Return to sport and performance after microfracture in the knees of National Basketball Association Players. *Orthop J Sports Med.* 2013;1(6):2325967113512759.
56. Voleti PB, Wu IT, Degen RM, Tetreault DM, Krych AJ, Williams RJ 3rd. Successful return to sport following distal femoral Varus osteotomy. *Cartilage.* 2019;10(1):19–25.
57. Hopper GP, Leach WJ. Participation in sporting activities following knee replacement: total versus unicompartmental. *Knee Surg Sports Traumatol Arthrosc.* 2008;16(10):973–9.
58. Kim Y-M, Joo Y-B. Patellofemoral osteoarthritis. *Knee Surg Relat Res.* 2012;24(4):193–200.



Management of Muscle Injuries in Basketball

43

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43.1 General Principles of Muscle Injury

Muscle injury is one of the most serious problems in either professional or amateur sport. In recent years, there has been a significant increase in scientific studies to properly classify and understand muscle injuries, yet there is still not a universal consensus, especially in terms of prognosis of every muscle injury.

Chan et al. [1] and Mueller-Wohlfarth et al. [2] classified muscle injuries based on severity.

This chapter is in memory of Dr. William (Bill) Garrett Jr., who passed away during the preparation of this chapter and contributed tremendously over the years to the development of knowledge around muscle injuries and their management and to sports medicine.

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Recently, Pollock et al. [3] and Valle et al. [4] introduced a novel, valuable concept: the intramuscular or peripheral connective tissue involvement, which was considered as one of the key components when assessing muscle injuries.

In 2018, Balius et al. concluded that studying the exact anatomic and topographical locations of every injury could help physicians to make more specific and precise diagnoses for muscle injuries and facilitate a better understanding of injury recurrence mechanism [5].

It is well known that muscle injuries often occur in the myoconnective junction; however, this knowledge is not enough to accurately evaluate injury severity and precisely estimate the injury prognosis.

In reality, when a basketball player is injured, the first step is to develop a plan of the return to

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play process. To be able to establish an appropriate return to play plan, a correct diagnosis is necessary. This approach implies not only knowing the injured general muscle group or specific muscle area but also knowing how much connective tissue is affected by that injury is important. For instance, if there is a damage on both tendinous and myotendinous components in the same muscle structure, likelihood of reinjury is higher because of the coexisting condition and its aggregate effects [5].

Therefore, it is no coincidence that most of the recent articles on muscle injuries have focused on both the anatomical location and involvement of connective tissue rather than on the severity/degree of the injury to determine its prognosis [6–9].

Thus, starting from this premise, this chapter will address the key elements for the management of muscle injuries in basketball players.

43.2 What Is the Magnitude of Muscle Injuries in the Basketball?

Basketball has one of the highest rates of musculoskeletal injuries of all team sports. However, epidemiological studies are generally lacking, especially professional basketball players, mainly because of methodological challenges in data collection.

In 2018, Andreoli et al. performed an integrative systematic review of the epidemiology of musculoskeletal injuries in basketball [10]. Initially, this research team selected a total of 268 articles. Of which, 11 were eligible for their integrative review. Among a total of 12,960 injuries, most injuries occurred in the lower extremities (63.7%), with 2832 (21.9%) at the ankle and 2305 (17.8%) at the knee joints, while injuries in the upper extremities represented 12–14% of the total injuries. Their conclusion was that basketball-related musculoskeletal injuries were the most prevalent in the lower extremities, with the ankle and knee joints having the highest prev-

alence of injuries regardless of sex and various playing categories [10].

In 2019, Rodas et al. published a descriptive epidemiology study [11]. A total of 59 professional male basketball players were evaluated over nine seasons (2007–2015). They analyzed 463 injuries, of which 207 resulted in time loss and 256 required medical attention based on a total exposure time of 42,678 h. Muscle strains and ankle sprains accounted for 21.2% and 11.9% of all injuries, respectively. The global incidence rate was 10.8 injuries per 1000 player-hours (95% CI, 9.9–11.9). The global injury burden was 53.9 days lost due to injuries per 1000 h for a total exposure time. The incidence rate of muscle strains (2.3; 95% CI, 1.9–2.8) was higher than that of ankle sprains (1.3; 95% CI, 1–1.7). The incidence rate for muscle injuries for the entire study period was 1.8 times higher (95% CI, 1.28–2.49) than that for ankle sprains.

In the same study, regarding the location of muscle injuries, 90% were located in the lower extremities, with distribution including gastrocnemius-soleus (21.7%), quadriceps (20.4%), hamstrings (18.5%), adductor-groin (13.4%), and obliquus-abdominis (8.0%). The same study analyzed the top 5 injured body parts that led to time loss, which were hamstrings (28.5%), gastrocnemius-soleus (26.7%), adductor-groin (14.2%), obliquus-abdominis (12.5%), and quadriceps (8.9%). Rodas and the research group discussed that a tight practice and competition schedules may be a contributing factor for the high muscle injury rates.

In summary, considering the increasingly intense schedule of professional basketball competitions in Europe and the NBA, it seems quite likely that muscle injuries are becoming more prevalent and may cause significant physical burdens in professional basketball players. This must be taken into consideration when injury prevention programs for both pre-season and in-season are designed. Such programs can be beneficial for basketball players to maintain long-lasting, competitive athletic careers.

43.3 Risk Factors and Mechanisms for Muscle Injuries in Basketball - The Complex, Multifactorial, and Dynamic Nature of Muscle Injury

In the field of sports medicine, injury risk factors are often determined by a comparison of certain variables such as physical characteristics, muscular strength, and biomechanical propensities between injured and non-injured athletes [12–15]. Reviewing various research search engines, there was lack in studies that examined musculoskeletal injuries as an outcome variable in basketball-related studies. Instead, there were a few studies that focused on specific injury types and body areas in basketball players.

Khan et al. focused on stress injuries of male professional basketball players in the NBA. Their study concluded that stress-related injuries negatively impact short- and long-term performance and career longevity in NBA players [16]. Another study investigated risk factors of professional basketball players based on the status of ankle injury history (acute or recurrent ankle sprain) [17]. This study did not find specific risk factors of future musculoskeletal injuries but noted that positive test results in single leg balance test was more prevalent in professional basketball players with history or ankle sprain [17]. Pasanen et al. examined the risk factor of low back pain in basketball players and identified that presence of family history in musculoskeletal disorders increases the risk of low back pain two-fold compared to basketball players who do not have any history of musculoskeletal disorders [18]. Additionally, the same study documented that older age was also identified as a risk factor for low back pain in basketball players [18].

As discussed in previous sections, muscle injuries are highly prevalent in basketball players. Rodas et al. performed a 9-year prospective study and descriptively analyzed the type of injuries among professional basketball players [18]. Findings of this study suggested that muscle injuries are more common than ankle sprains in com-

petitive professional basketball players [18]. A systematic review study performed by Green et al. investigated risk factors of calf muscles [19]. Although this study included athletes from various sports such as Australian football, rugby players, and triathlon athletes in addition to basketball players, calf muscles injury risk factors were found, which indicated the presence of previous calf injury history and older chronological ages [19].

To summarize the available evidence, documented risk factors of muscle injuries in basketball players are: presence of previous injury history of the player, presence of injury history in family members, and higher chronological ages [18, 19]. To synthesize the research findings in actual clinical settings, it is a reasonable assumption that basketball players who have a long career are likely older and sustain more injuries than young basketball players who did not have an opportunity to play. Therefore, exposures to basketball participation may play an important role. In addition to athletic exposures, risk factors may vary by sex (males, females), physical fitness level (good, poor), general fatigue status (physical, emotional, mental status), and playing positions (center, guard, and forward). Therefore, further studies are necessary to further investigate specific risk factors based on the aforementioned variables in basketball players.

43.4 Musculoskeletal Screening in Basketball

As previously stated, the most common risk factors of musculoskeletal injuries were the presence of a previous injury, previous injury history of family members, and higher chronological ages [18, 19]. It was well documented that competitive soccer players who have a previous injury history of the lower extremities have a greater likelihood of sustaining another injury at the same body region [20–23]. For instance, soccer players who suffered previous groin injuries showed 2.6–7.3 times greater odds to sustain a future groin injury

[20, 21]. Additionally, Hagglund et al. documented that competitive soccer players who have a history of quadriceps and calf injuries demonstrated 3.1 and 2.3 times higher risk of sustaining the future injuries in the same body parts, respectively [23]. Furthermore, Hagglund et al. reported 3.5 greater risk of hamstring injury in competitive soccer players who had the previous hamstring injury history [22]. Although the sport type is different, the effect of the presence of previous injury history appeared to be substantial. Moreover, a few studies identified specific gene types such as COL5A1 that are associated with a traumatic knee injury such as anterior cruciate ligament (ACL) tear [24–27]. Those gene studies suggested predisposition to certain musculoskeletal injuries within family members due to the inheritance of certain genes. Interestingly, one study highlighted that the familial effects on ACL tear are stronger in males compared to females [28]. In short, musculoskeletal screening should include previous injury histories in both the player and family members. Examining previous injury history from both the player and family member is likely beneficial to identify basketball players who are at-risk or prone to future musculoskeletal injuries.

Another screening consideration for musculoskeletal injuries for basketball players is the utility of an innovative imaging modality such as ultrasound. One study identified that hypoechoic area, recorded by ultrasound examination, was associated with increased risk of onset of patellar tendinopathy [29]. Additionally, several other studies found that hypoechoic area and micro-calculi/calculi formation in patellar tendon were indicators of future patellar tendon abnormalities [30–32]. Moreover, one of the recent studies that examined patellar tendon of asymptomatic competitive jumpers using ultrasound indicated that 29% of the jumpers including basketball players who identified themselves “asymptomatic” has hypoechoic area at their patellar tendons [33]. Basically, ultrasound examination can potentially identify abnormal muscle tissue structures even if athletes report neither pain nor any symptoms at the time of examination.

In summary, identifying a history of previous injury is likely an effective injury prevention

screening method. Research evidence suggested that surveying a history of previous injury should include both the player and family members. Another consideration is to use ultrasound as a screening tool. A few ultrasound-related studies identified abnormal muscle tissues and tendon structures, even in asymptomatic jumping athletes. If abnormal muscle and tendon changes are detected by ultrasound prior to the onset of pain and symptoms, it is possible to provide a preventive intervention before injury occur.

43.5 Diagnosis and Classification/Imaging (US/MRI)

When an athlete is injured, especially with a muscle injury, the main goal is to provide appropriate treatments based on accurate diagnosis so that the athlete can return to optimal performance dynamics as soon as possible, minimizing the reinjury risk substantially. This cannot be achieved if accurate diagnosis was not identified in the early phase. An accurate diagnosis allows the medical staff to understand the exact location and severity of the injury.

Although some clinical studies suggested that imaging tests, especially MRI, are not useful for decision making regarding the return to play process [34–36], medical professionals who work with athletes closely consider imaging tests as one of the essential components for both the initial diagnosis and the injury prognosis along with physical tests and field tests, particularly for muscle injuries. Furthermore, imaging is crucial to confirm and assess the extent of sports-related muscle injuries, which can help guiding specific management strategies and directly influences the prognosis. This is extremely useful when the diagnosis or grade of injury is not clear. Also, this approach is beneficial when recovery turns out to be longer than initially expected, and when interventional or surgical management are considered and required to make further progress [37].

The most routinely used imaging tests in order to diagnose muscle injuries are mainly ultrasound and MRI. Although the ultrasound is a widely

used imaging modality to assess muscle injuries because of its accessibility and low cost compared with MRI [38, 39] and, particularly, its dynamic nature and capability to diagnose most injuries. In order to monitor the injury and identify possible complications, MRI can provide detailed information that is advantageous to determine the precise prognosis of muscle injury in athletes [5].

Usually, both modalities can aid in an adequate diagnosis and monitoring the recovery process, however ultrasound has a few advantages. Conversely, ultrasound may sometimes have “blind points” which can be better identified by MRI (Fig. 43.1).

Ultrasound has a lot of other advantages. It is readily available, inexpensive, and can be easily used repeatedly, which allows us to monitor the images and progress over time. It can also be used to make ultrasound-guided interventions such as intra-articular and extra-articular injections and hematoma evacuations when required. Also, ultrasound provides dynamic exploration capacity (Fig. 43.2).

MRI is considered the gold standard imaging modality to assess the morphology of muscles in athletes. This is due to its capability to visualize soft tissues with excellent contrast and provides high spatial resolution and multiplanar assessment. This is particularly important in cases clinically severe traumatic injuries are suspected. MRI

is probably the best imaging technology to confirm and evaluate the extent, severity, and location of muscle injuries. Furthermore, MRI is better than US for the assessment of muscle injuries in deep muscle compartments [37, 40, 41] (Fig. 43.3).

Muscle injuries in the same macroscopic or topographical location with the same treatment protocol could evolve differently based on the type of specific microscopic or connective tissue involvement. Thus, the amount of involvement of the connective tissue plays an important role in both prognosis and reinjury. Therefore, studying the exact location of the injury will help physicians to make specific and precise diagnoses for muscle injuries, which further allows physicians to have a better understanding of some reinjury patterns [5].

This framework is quite theoretical, however the example presented in Fig. 43.4, it can be seen as an example of the highlights the precision often necessary to be able to establish a correct prognosis for muscle injuries. Fig. 43.4 depicts the different evolution of apparently very similar injuries. The prognosis is very different for an injury that has both muscle tendon involvement and a rupture with retraction of a free tendon (Fig. 43.4a) compared to an injury that is only myofascial without any involvement of the tendon (Fig. 43.4b) or an injury that has a myotendinous component and a complete transverse rupture of the tendon (Fig. 43.4c). This level

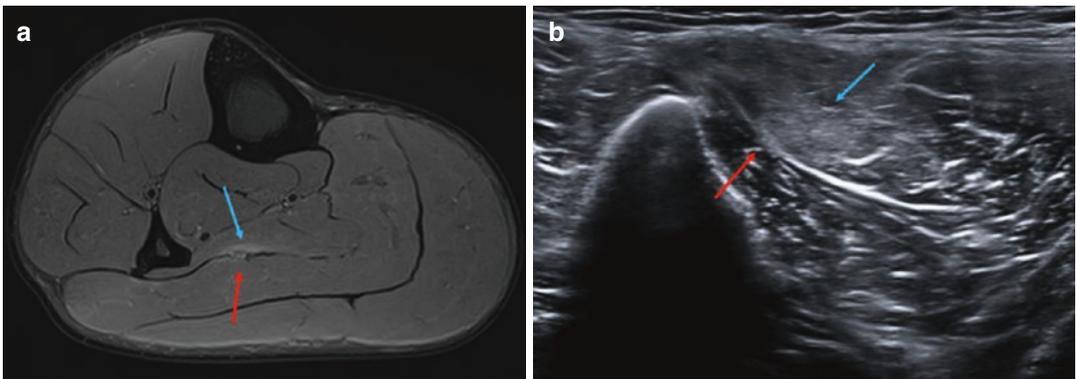


Fig. 43.1 Image of a soleus lateral aponeurosis rupture. (a) Axial T2 FS MRI image showing a subacute rupture of the lateral aponeurosis of the soleus muscle (red arrow) with surrounding edema (blue arrow). (b) Transversal ultrasound view of the same injury. The rupture (red

arrow) is much more difficult to identify with the ultrasound, and it is also difficult to assess the evolution of the scar tissue. The edema (blue arrow) can be assessed perfectly

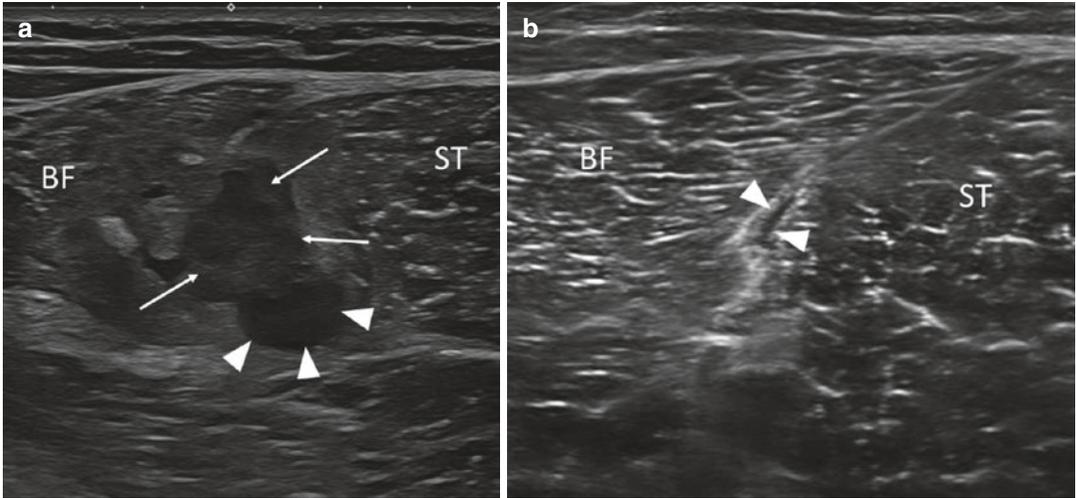


Fig. 43.2 (a) Severe injury of the common tendon of the hamstrings. Transverse ultrasound of the left posterior thigh shows hypoechoic disruption (arrows) with hematoma (arrowheads) in the common tendon. (b) Small injury of the common tendon of the hamstrings. Although

it appears small, it is classified as a severe injury due to the involvement of the common tendon. Transverse ultrasound of the left posterior thigh clearly shows longitudinal split of the common tendon (arrowheads)

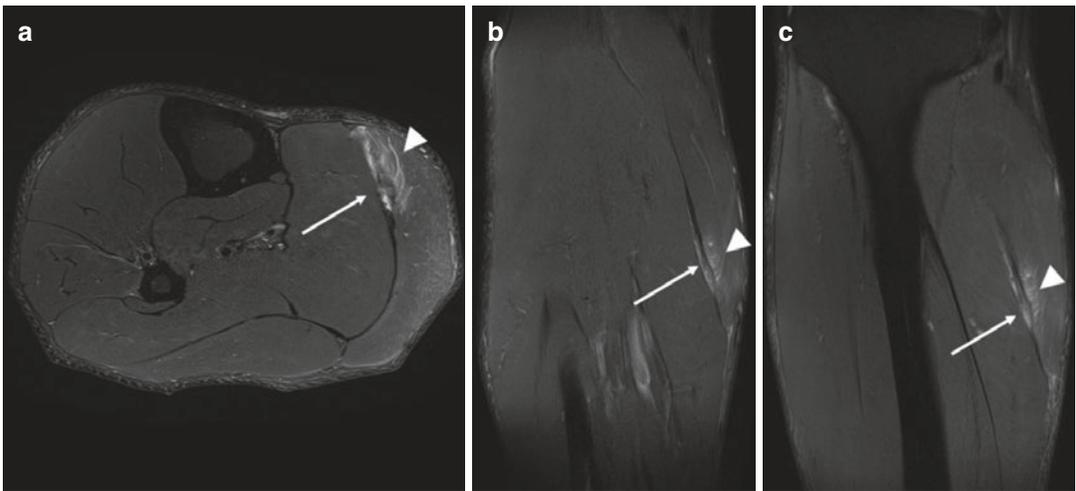


Fig. 43.3 MRI of a subacute and multiple reinjured myotendinous junction (MTJ) of the medial head of the gastrocnemius. Marked thickening and signal intensity changes of the aponeurosis of the distal MTJ of the medial head gastrocnemius (arrows) with partial discontinuity,

soft scar tissue regions, and muscle edema (arrowheads) are noticeable. (a) Axial DP FatSat MRI sequence. (b) Sagittal DP FatSat sequence. (c) Coronal DP FatSat sequence

of precision (especially as in Fig. 43.4c) is only available with MRI.

Although the physical examination still plays a key role in any patient assessment, imaging modalities such as MRI and ultrasound play an integral role for providing an accurate diagnosis

and following-up healing progression. Furthermore, accurate decisions in the return to play process such as return to training (individually and with the team) and competition and possible risks of reinjury may be better estimated based on appropriate imaging.

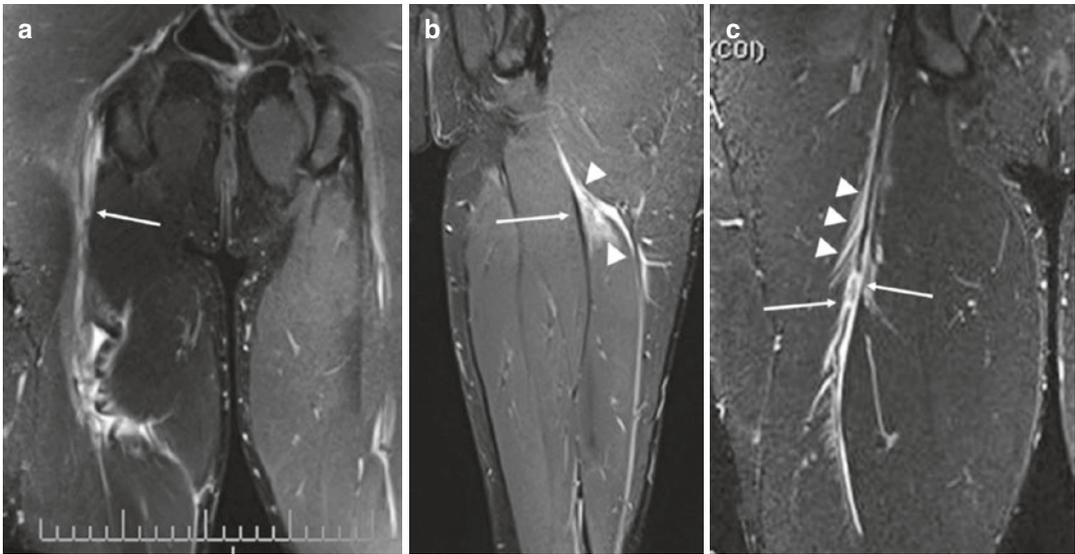


Fig. 43.4 Three different injuries affecting the proximal region of the hamstrings muscle complex with completely different prognosis. All images are coronal T2 FatSat sequences. (a) Complete rupture of the proximal semimembranosus tendon (arrow) with moderate retraction.

(b) Intramuscular rupture with mild myofascial fluid (arrowheads) with an intact common tendon. (c) Myotendinous rupture of the common hamstrings tendon (arrowheads) with two foci of complete transversal rupture of the common tendon itself (arrows)

43.6 Emerging and New approaches in the Treatment of Muscle Injuries: Regenerative and Biological Treatments for Muscle Injuries/Surgery for Muscle Injuries

A series of experimental studies suggested that early, active mobilization after a short period of immobilization/rest (duration: inflammatory period of healing) is an ideal form of therapy for injured muscles. Athletes are encouraged to start active rehabilitation immediately after the inflammatory period (3–5 days). Safe and effective treatment protocols were developed and scientifically tested and showed positive effects without increased risk of reinjury for certain muscle groups such as hamstrings, calf, and quadriceps muscles [42–44].

A few medications such as non-steroidal anti-inflammatory drugs (NSAIDs), corticosteroids, and losartan were proposed to take an assistive role to enhance the healing process in rehabilitation protocols. Although more evidence is neces-

sary, several new emerging rehabilitation technologies such as extracorporeal shockwave therapy (ESWT), shock waves, and hyperbaric oxygen therapy (HBOT) have been recently introduced. At this point, there are no clinical investigations addressing the effects of any of these clinical modalities [44]. The clinical utility of these emerging clinical modalities need to be tested in the near future. Additionally, there is a growing interest in sports medicine and athletic communities in “regenerative medicine” using endogenous growth factors or stem cells therapies delivered directly into the injury site to facilitate optimum healing following injury.

The most popular use of regenerative products is injection of platelet-rich plasma (PRP). It is rationalized that platelets release various growth factors upon activation which are supposed to provide regenerative effects. Our current scientific knowledge about PRP remains at a basic level, and there are many unanswered questions regarding its use in various types of muscle injuries. Currently, there is not a known “optimal” PRP content, nor is there a generally accepted protocol for PRP use regarding volume, number,

and timing of injections. It is unknown whether these factors could influence clinical outcomes as it has never been examined or compared. This requires further investigation in high-quality clinical trials [45].

In addition to the emerging use of PRP in the management of muscle injuries, there is a growing interest for the use of stem cells in muscle injuries. In the use of the stem cells on muscle injuries, diverse types of cells were discussed including muscle-derived stem cells (MDSCs—not to be confused with satellite cells), bone-marrow-derived mesenchymal cells (MSCs), or adipose-tissue-derived stem cells (ADSCs). The proposed mechanism of stem cells is twofold: firstly, administrating stem cells can differentiate into a large portion of cells and facilitate replacing lost or injured tissues. Secondly, mesenchymal cells produce a vast number of growth factors that could augment tissue regeneration. In addition, it is known that MSCs have an immunoregulatory effect (suppression of chronic, detrimental inflammation) on their tissue environment.

However, whether or not this interesting therapeutic approach could play a beneficial role in the treatment of (acute) muscle injuries has not been extensively studied so far. Most available literature focused on degenerative disorders such as muscular dystrophies. Despite promising results found in patients with muscle dystrophies, we currently do not advocate its use for (acute) skeletal muscle injuries because its long-term outcomes including efficacy and safety for athletes have not been determined yet. The use of orthobiologics and regenerative medicine for muscle and tendon injuries in basketball are extensively discussed in separate chapters in this book.

Operation is seldom considered as a first choice of the treatment in muscle injuries. For instance, the phrase “muscle injuries can be healed without intervention” are commonly used as a guiding principle [46]. However, there are exceptions. Certain muscle injuries with highly specific indications may benefit from surgical intervention, even though research evidence is currently limited. Those clinical indications include a complete (grade III) rupture of a muscle with few or no agonist muscles, a tear (grade II)

if more than half of the muscle is torn, or a large intramuscular hematoma.

Currently, new indications for surgical intervention were proposed. For instance, it has been suggested that surgery should be performed immediately after the injury occurs [47]. Additionally, it was theorized that surgery may also be necessary if a series of conservative treatments fail to achieve a satisfactory result. For example, if the player has chronic symptoms or recurrent injuries. Further clinical attention is recommended for the following two muscle groups: hamstrings and quadriceps. For hamstrings, early hamstring surgery was suggested following avulsion of two or three proximal tendons from the ischial tuberosity. Furthermore, a complete tendon rupture of the hamstrings proximal common/central tendon is another indication, especially in professional athletes. In quadriceps injuries, the indications for surgery are not well established; however, chronic pain and functional disability that last for more than a few months after a complete or partial tear is one of the potential indications. Also, surgery might be considered in complete proximal avulsions with significant retraction or complete tears in which there is a significant gap between the tendon junctures of the injured muscles [44].

43.7 A Multidisciplinary and Algorithmic Approach to Muscle Injuries Rehabilitation

Determining the type of rehabilitation program that most effectively promotes muscle tissue repair and functional recovery is essential to minimize the risk of reinjuries as well as to enhance the quality of return to play, and, consequently, optimal performance [43].

Generally, rehabilitation of muscle injuries is usually presented as a general protocol. During the rehabilitative process, certain risk factors for future musculoskeletal injuries need to be considered in accordance with the biology of the muscle injury and healing process. Currently, rehabilitation protocols for soccer or basketball

players do not seem to place a substantial emphasis on the programming and sequencing of training loads or on performance-related factors during and following rehabilitation (e.g., ankle stiffness and horizontal forces). However, those aspects, sport-specific functional rehabilitation, should not be underestimated since it is necessary to optimize injured athletes' return to play based on their unique sports demands.

To support this notion, Mendiguchia et al. introduced valuable evidence [43]; Their study published in 2017 showed that male football players who underwent an individualized, multifactorial, criteria-based algorithm and rehabilitated with a performance- and primary risk factor-oriented training program from the early stages of the rehabilitation process markedly reduced the risk of reinjury compared to a general protocol, which is based on a traditional strength-based training.

The individualized, multifactorial approach Mendiguchia et al. described, included a comprehensive evaluation of health status, participation risk as well as factors involved in the decision

modification. This approach provided clinicians with an evidence-based rationale for RTP decision-making. Furthermore, these risk factors should be considered along with the course of the RTP continuum.

Making an accurate diagnosis is the cornerstone of effective injury management and realistic RTP planning. An accurate diagnosis facilitates a precise estimation of the injury prognosis, and in turn decision-making regarding injury management. Imaging may be used judiciously at this point, and if there is a significant clinical finding in the imaging studies, the course of RTP plans may need to be potentially altered. At FC Barcelona, we work backwards, meaning that we calculate the anticipated time to return to full match play from the targeted date. Understanding biology will help developing productive rehabilitation directions optimizing injury prognosis, and planning a strategy for appropriate loading through the return to play continuum [44]. A general suggested model from FC Barcelona is shown in Fig. 43.5.

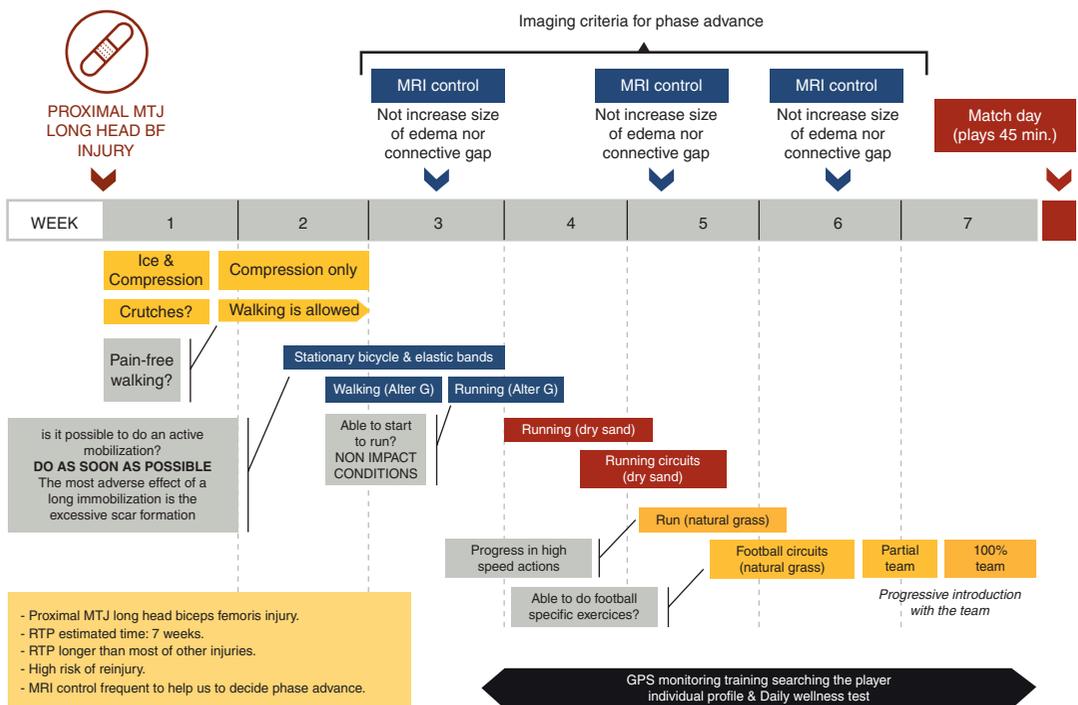


Fig. 43.5 An overview of RTP from a hamstring muscle injury at FC Barcelona, adapted to Basketball players

At FC Barcelona, a five-stage approach to the management of muscle injuries is currently used. Stepwise progression of loading will facilitate effective tissue healings while restoring functional capacity. During the acute phase of management, the clinical focus is to limit the extent of the initial injury and to provide a strong tissue healing foundation in the rehabilitation process. Starting with ice and compression to reduce pain and prevent further damages to the injured and surrounding tissues are key goals during this phase. During the subacute phase, active joint mobilization will facilitate enhancing movement capability and promoting further tissue healing.

In addition to focusing on the injured body parts, the non-injured body parts such as the upper quadrant in case of a lower extremity injury, lumbopelvic stabilization exercises, and other body parts such as the hip musculatures should be focused on. This approach helps minimizing atrophy of other body parts and assists maintaining an overall fitness level.

Also, it is advised that aerobic conditioning is maintained. This can be achieved using elliptical trainers, stationary cycles, aqua jogging, and AlterG treadmill. If the athlete can tolerate, it is ideal to implement aerobic conditioning workouts before progressing to walking and/or running on a treadmill.

Muscle activation with controlled manner can be useful in the early stages. Starting with manual resistance exercises is recommended because the manual resistance exercise can deliver mechanical stimulus to the affected area without overloading the structures. More complex exercises can be performed as a next step of progression as long as they are tolerable for athletes and do not reproduce pain or discomfort. Tissue activation progression should gradually include training contents from isometric to concentric exercises. Exercises with eccentric contractions are usually implemented in the final stage of the progression.

Once a full range of motion was achieved with adequate muscle recruitments, it is important to progress to a gym-based training. In this phase, the main aim is to regain full muscle function, which means regaining full voluntary muscle contraction of the injured muscle throughout a full range of motion.

Also, as early as tolerated, the player should begin a running progression with careful control of volume, intensity, and mechanics. An important aspect of the running progression is to ensure that the loading is progressively and carefully increased. The running could preferably be performed on soft surface. At FC Barcelona, running in the early stages is commenced on dry sand since the metabolic demand is higher on dry sand than on hard surface. Additionally, mechanical impact is lower on soft surfaces compared with that on hard surface; thus, it is an ideal platform for athletes to rehabilitate and transition to linear running on the field. During the running progression, sport-specific drills can be initiated. It is important to begin the sport-specific drills from slow speed. Then, speed, duration, and level of sport-specific drills can be progressed gradually based on the athlete's tolerance level.

Later, the rehabilitative emphasis needs to be placed on incorporating the ball and skill-related functions in every session (or at least as many sessions as possible). One of the practical strategies is to integrate unanticipated movements with speed and timing, simulating actual practice and competition modes. Furthermore, advanced skills and cognitive challenges need to be introduced. Once athlete begins demonstrating enough running, skills, and sport-specific functions, the athlete can transition to the RTP process from rehabilitative phase.

The primary goal during the RTP process is to ensure the player can return safely to targeted activities while minimizing reinjury risk with a 'position specific' focus as the RTP continuum progresses.

Finally, when the player steps on the court again, it is necessary to plan and clarify which training sessions he/she can perform with the team. This decision needs to be updated daily and mutually agreed by the medical and performance staff, as well as the coaches, analyzing the external loads (i.e through GPS) and the internal loads of the player in addition to psychological aspects. By monitoring, analyzing and integrating these factors together, the medical staff can decide when the player is ready to return to full training and match play.

43.8 How to Manage Return to Play for Muscle Injuries - a Dynamic Model; Return to Play for Specific and more Common Muscle Injuries in Basketball

In 2010, Creighton et al. [48] proposed a return to play (RTP) decision-making model. This model helped clarifying the precise processes of RTP for clinicians. Also, providing such a structured model facilitated reducing controversy related to RTP timing, procedure, and operations, which further optimized filling an important gap between clinical practices and research evidence.

In 2015, Shrier et al. [49] introduced the Strategic Assessment of Risk and Risk Tolerance framework. In this framework, it was commonly agreed that any RTP decision-making process should include a balance between an assessment of injury recurrence (reinjury risk) and the acceptable risk tolerance threshold.

In 2017, van der Horst [50] published an article with a consensus statement based on a panel of 58 international soccer experts and defined RTP terminology, definition, medical criteria, responsibilities, and consultation guidelines for RTP following hamstrings injuries in football. The contents mainly regarded a RTP model for hamstrings injuries in soccer. It is unsure that the same approach is applicable for basketball; however, this model is used by FC Barcelona basketball club due to several philosophical overlaps. RTP decision should always be a multidisciplinary process. For RTP readiness assessment of the player after a hamstrings injury, emphasis is placed on pain relief, flexibility assessment, psychological readiness, and functional performance. In certain aspects, RTP scientific evidence was limited until recently. For instance, how imaging modalities such as MRI play a role was notably understudied. However, this RTP model appeared to be a good fit for our clinical practice in FC Barcelona basketball club.

In 2018, a new concept has emerged with the introduction of the FC Barcelona Muscle

injury Guide book [44]. In the core of the concept, following a muscle injury (or any injury for that matter), two main objectives (and at the same time, two challenges) were defined: (1) to return the player to match play as soon as possible and (2) to avoid injury recurrence (reinjury). There is a fine balance between the two main goals, which are complex. Furthermore, the goals depend on the context of each individual player, injury severity, and other circumstances including the timing in the season, the player's role in the team, and expectations from the coaching staff. However, there is a paradigm shift occurring in the way we envision RTP. Instead of a simple concept of "just return to play" in a manner that defines the highly anticipated event occurring at the end of the rehabilitation program, we now consider the "return to play" process starts the moment the injury occurs and continues beyond the point where the player is making his or her return to unrestricted match play with optimal performance (Fig. 43.6). This type of progression is malleable and needs to be individualized, meaning that there is a variation of faster and slower progressions throughout this RTP continuum according to each players needs and limitations [44].

The FC Barcelona Muscle Injury Guide [44] proposed the following approach: The foundation for any RTP estimation begins with an accurate diagnosis. However, it is also essential to treat each player in an individualized course of RTP and consider their intrinsic, extrinsic, and sport-specific factors (regardless of soccer or basketball). We suggest that practitioners should continuously re-evaluate the initial RTP goals throughout the rehabilitation process, depending on how quickly the player progresses along each milestone defined in the RTP continuum. Key indicators of whether or not the player is on-target to meet the anticipated RTP date include regaining baseline strength and flexibility measures, completing high-intensity training sessions comparable to (or even greater than) their anticipated match demands, and demonstrating an appropriate level of basketball-specific cognitive skills and psychological readiness.

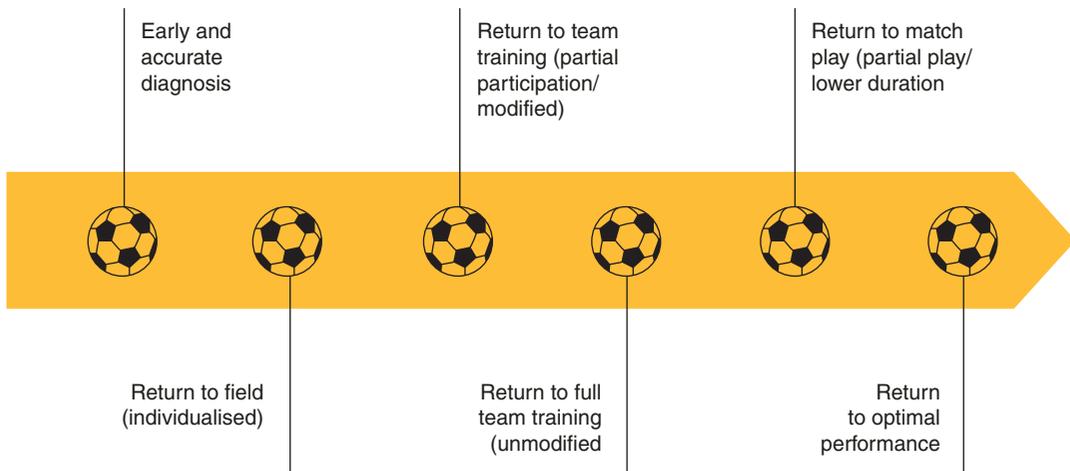


Fig. 43.6 Modified RTP model in football, Arden & Pruna in FCB Muscle Injury Guide, 2018

43.9 A Model for Muscle Injuries Prevention in Basketball/ Barriers and Facilitators to Delivering Injury Prevention Strategies

As previously described in Sect. 8, the concept of “return to play” has been evolving over the years. Simply, returning to sporting activities at the end of the rehabilitation process should not be a goal for clinicians. As discussed in Sect. 3, presence of previous injury history is an influential factor for future musculoskeletal injury. Therefore, rehabilitating athletes from the original injury and simultaneously preventing them from sustaining additional future injuries should be a new, ideal goal for clinicians. As illustrated in Fig. 43.6, return to field/court should be a first step in the “return to optimal performance” phase. The return to field/court, should be individualized based on injury severity, anatomic involvement, previous injury histories, and other factors discussed in Sect. 8 (intrinsic, extrinsic, and sports-specific factors). Following the return to field/court, return to practice (partially and full), return to match (partially and full), and return to optimal performance (Fig. 43.6) should be subsequent steps. Clinicians including physiotherapists/athletic trainers, fitness coaches/strength and conditioning specialists, and orthopedic/pri-

mary care sports medicine physicians should focus on how to maintain the optimal performance level in extended duration during a season. In order to optimize the level of performance for a long time in athletes who are on the way to return to sports following injury, injured anatomic regions should be evaluated periodically. Greater risk of injury recurrence (reinjury) at the same anatomic region is well documented [20–23]. Thus, regular examination of the injured anatomic area using imaging modalities such as ultrasound could be beneficial.

This concept of preventing athletes from additional future injuries needs to be emphasized and shared not only with health-care practitioners, but also with the coaching staff. As it was pointed out in Sect. 3, athletic exposures may play a role in the increased likelihood of musculoskeletal injuries in conjunction with the presence of a previous injury history and older chronological age. Tight practice and match schedules were discussed as a potential underlying mechanism for increased muscle injuries documented in a recent study [18]. Tight practice and match schedules, were often physically and mentally demanding for athletes who are on the way of this continuum of recovery during the RTP process. It is important for the coaching staff to understand that it is not ideal for athletes to skip a step in the continuum of recovery because

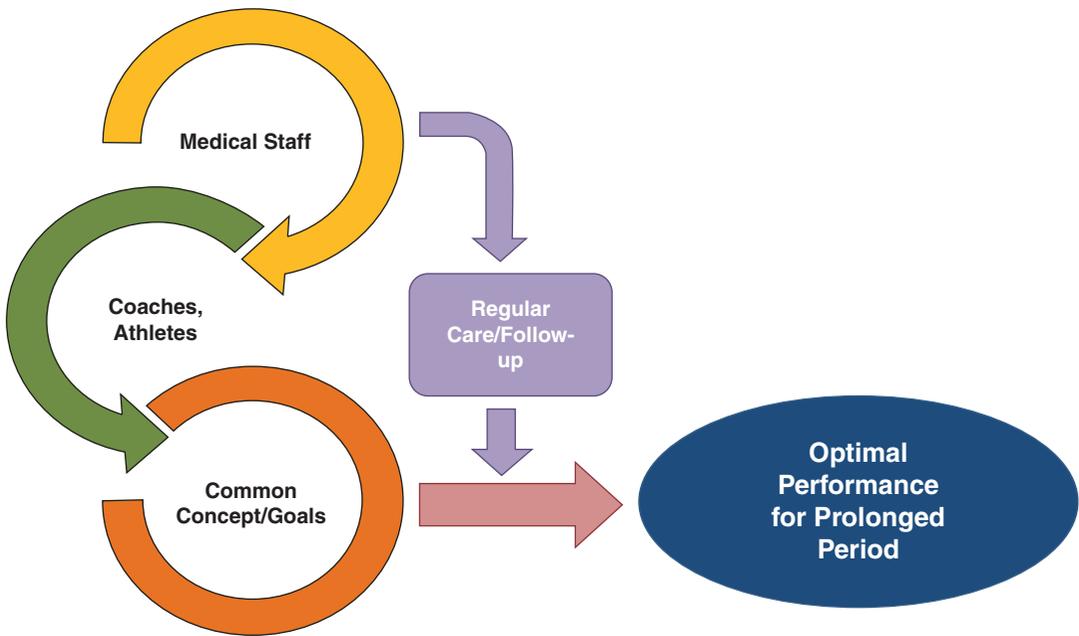


Fig. 43.7 An integrative model for injury prevention for athletes who are engaged in the continuum of return to play following injury

muscle and tendon tissues require appropriate steps in order to heal properly. Athletes need to go through each step of the return to play model (Fig. 43.6). Skipping a step [return to practice (partially and full), return to match (partially and full), and return to optimal performance (Fig. 43.6)] may lead to setbacks such as injury recurrence (reinjury). Health-care practitioners need to take the role of the facilitator of this prevention initiative through positive communications with the coaching staff. A lack of communication between health-care practitioners and the coaching staff may possibly lead to misunderstanding and potentially negative consequences for the athlete. Also, in addition to the health-care practitioners and coaching staff, players need to understand their own higher susceptibility to additional musculoskeletal injuries. Having this awareness likely helps taking reasonable recovery steps to reach optimal performance level (Fig. 43.7). In a circle of well-communicated plans and shared goals, injured players should be able to reach and maintain a high level of performance throughout a season.

Take Home Messages Traditionally, ankle injury has been recognized as the most common pathology in basketball; however, emerging evidence suggests that muscle injuries may be more frequent than ankle injuries.

- Hamstrings and calf are the most common sites for muscle injuries in professional basketball players.
- Muscle injuries require sufficient healing time to promote appropriate recovery and reduced injury recurrence (reinjury).
- Return to play timelines should be realistic, but should not be either too aggressive or too conservative in their approach.
- Following a rehabilitation program, safe return to play and process of return to optimal performance should be prioritized as ultimate goals instead of competitive schedules and team demands.
- Every injury is unique; therefore, return to play programs should be individualized.
- Basketball players can sometimes play with injuries in comparison to soccer players due to specific positions, demands, and situations

during the game; however, risk of injury recurrence (reinjury) should be considered.

- In this context, more research is needed in professional basketball to optimize medical care and management of injuries.

References

- Chan O, Del Buono A, Best TM, et al. Acute muscle strain injuries: a proposed new classification system. *Knee Surg Sports Traumatol Arthrosc.* 2012;20:2356–62.
- Mueller-Wohlfahrt H-W, Haensel L, Mithoefer K, et al. Terminology and classification of muscle injuries in sport: the Munich consensus statement. *Br J Sports Med.* 2013;47:342–50.
- Pollock N, James SLJ, Lee JC, et al. British athletics muscle injury classification: a new grading system. *Br J Sports Med.* 2014;48:1347–51.
- Valle X, Tol Alentorn-Geli E, et al. Muscle injuries in sports: a new evidence-informed and expert consensus-based classification with clinical application. *Sports Med.* 2017;47(7):1241–53.
- Balius R, Alomar X, Pedret C, et al. Role of the extracellular matrix in muscle injuries: Histoarchitectural considerations for muscle injuries. *Orthop J Sports Med.* 2018;6(9):2325967118795863.
- Balius R, Maestro A, Pedret C, et al. Central aponeurosis tears of the rectus femoris: practical sonographic prognosis. *Br J Sports Med.* 2009;43:818–24.
- Comin J, Malliaras P, Baquie P, Barbour T, Connell D. Return to competitive play after hamstring injuries involving disruption of the central tendon. *Am J Sports Med.* 2013a;41(1):111–5.
- Pollock N, Patel A, Chakraverty J, et al. Time to return to full training is delayed and recurrence rate is higher in intratendinous (“c”) acute hamstring injury in elite track and field athletes: clinical application of the British athletics muscle injury classification. *Br J Sports Med.* 2016;50:305–10.
- Prakash A, Entwisle T, Schneider M, et al. Connective tissue injury in calf muscle tears and return to play: MRI correlation. *Br J Sports Med.* 2018;52(14):929–33.
- Andreoli CV, Chiamonti BC, Biruel E, et al. Epidemiology of sports injuries in basketball: integrative systematic review. *BMJ Open Sport Exerc Med.* 2018;4:e000468. <https://doi.org/10.1136/bmjsem-2018-000468>.
- Rodas G, Bove T, Caparrós T, et al. Ankle sprain versus muscle strain injury in professional Men’s basketball. A 9-year prospective follow-up study. *Orthop J Sports Med.* 2019;7(6):2325967119849035. <https://doi.org/10.1177/2325967119849035>.
- Hewett TE, Myer GD, Ford KR, et al. Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. *Am J Sports Med.* 2005;33:492–501.
- Myer GD, Ford KR, Barber Foss KD, et al. The relationship of hamstrings and quadriceps strength to anterior cruciate ligament injury in female athletes. *Clin J Sport Med.* 2009;19:3–8.
- Paterno MV, Schmitt LC, Ford KR. 2010. Biomechanical measures during landing and postural stability predict second anterior cruciate ligament injury after anterior cruciate ligament reconstruction and return to sport. *Am J Sports Med.* 2010;38:1968–78.
- Sugimoto D, Howell DR, Tocci NX, et al. Risk factors associated with self-reported injury history in female youth soccer players. *Phys Sportsmed.* 2018:1–7.
- Khan MK, Madden MT, Burrus JP, et al. Epidemiology and impact on performance of lower extremity stress injuries in professional basketball players. *Sports Health.* 2018;10:169–74.
- Halabchi FH, Angoorani M, Mirshahi MH, et al. The prevalence of selected intrinsic risk factors for ankle sprain among elite football and basketball players. *Asian J Sports Med.* 2016;7:e35287.
- Pasanen K, Rossi KM, Parkkari J, et al. Low Back pain in young basketball and Floorball players. *Clin J Sport Med.* 2016;26:376–80.
- Green B, Pizzari T. Calf muscle strain injuries in sport: a systematic review of risk factors for injury. *Br J Sports Med.* 2017;51:1189–94.
- Arnason A, Tenga A, Engebretsen L, et al. A prospective video-based analysis of injury situations in elite male football: football incident analysis. *Am J Sports Med.* 2004;32:1459–65.
- Engebretsen A, Myklebust HG, Holme I, et al. Intrinsic risk factors for groin injuries among male soccer players: a prospective cohort study. *Am J Sports Med.* 2010;38:2051–7.
- Hagglund M, Walden M, Ekstrand J. Previous injury as a risk factor for injury in elite football: a prospective study over two consecutive seasons. *Br J Sports Med.* 2006;40:767–72.
- Hagglund M, Walden M, Ekstrand J. Risk factors for lower extremity muscle injury in professional soccer: the UEFA injury study. *Am J Sports Med.* 2013;41:327–35.
- Lulinska-Kuklik E, Rahim EM, Domanska-Senderowska, et al. Interactions between COL5A1 gene and risk of the anterior cruciate ligament rupture. *J Hum Kinet.* 2018;62:65–71.
- Lv ZT, Gao ST, Cheng P, et al. Association between polymorphism rs12722 in COL5A1 and musculoskeletal soft tissue injuries: a systematic review and meta-analysis. *Oncotarget.* 2018;9:15365–74.
- O’Connell K, Knight H, Ficek K, et al. Interactions between collagen gene variants and risk of anterior cruciate ligament rupture. *Eur J Sport Sci.* 2015;15:341–50.
- Posthumus M, September AV, O’Cuinneagain D, et al. The COL5A1 gene is associated with increased

- risk of anterior cruciate ligament ruptures in female participants. *Am J Sports Med.* 2009;37:2234–40.
28. Myer GD, Heidt RS, Waits C, et al. Sex comparison of familial predisposition to anterior cruciate ligament injury. *Knee Surg Sports Traumatol Arthrosc.* 2014;22:387–91.
 29. Cook JL, Khan KM, Kiss ZS, et al. Prospective imaging study of asymptomatic patellar tendinopathy in elite junior basketball players. *J Ultrasound Med.* 2000;19:473–9.
 30. Comin J, Cook JL, Malliaras P, et al. The prevalence and clinical significance of sonographic tendon abnormalities in asymptomatic ballet dancers: a 24-month longitudinal study. *Br J Sports Med.* 2013b;47:89–92.
 31. Fredberg U, Bolvig L. Significance of ultrasonographically detected asymptomatic tendinosis in the patellar and achilles tendons of elite soccer players: a longitudinal study. *Am J Sports Med.* 2002;30:488–91.
 32. Visnes H, Tegnander A, Bahr R. Ultrasound characteristics of the patellar and quadriceps tendons among young elite athletes. *Scand J Med Sci Sports.* 2015;25:205–15.
 33. Fazekas ML, Sugimoto D, Cianci AJ, et al. Ultrasound examination and patellar tendinopathy scores in asymptomatic college jumpers. *Phys Sportsmed.* 2018;46:477–84.
 34. Reurink G, Whiteley R, Tol JL. Hamstring injuries and predicting return to play: ‘bye-bye MRI?’. *Br J Sports Med.* 2015;49:1162–3.
 35. van der Made AD, Almusa E, Reurink G, et al. Intramuscular tendon injury is not associated with an increased hamstring reinjury rate within 12 months after return to play. *Br J Sports Med.* 2018;52:1261–6.
 36. Van Heumen M, Tol JL, de Vos R, et al. The prognostic value of MRI in determining reinjury risk following acute hamstring injury: a systematic review. *Br J Sports Med.* 2017;51:1355–63.
 37. Guermazi A, Roemer FW, Robinson P, et al. Imaging of muscle injuries in sports medicine: sports imaging series. *Radiology.* 2017;282(3):646–63.
 38. Nazarian LN. The top 10 reasons musculoskeletal sonography is an important complementary or alternative technique to MRI. *AJR.* 2008;190:1621–6.
 39. Bianchi S, Martinoli C, Abdelwahab IF, Derchi LE, Damiani S. Sonographic evaluation of tears of the gastrocnemius medial head (“tennis leg”). *J Ultrasound Med.* 1998;17:157–62.
 40. Koulouris G, Connell D. Hamstring muscle complex: an imaging review. *Radiographics.* 2005;25(3):571–86.
 41. Hayashi D, Hamilton B, Guermazi A, et al. Traumatic injuries of thigh and calf muscles in athletes: role and clinical relevance of MR imaging and ultrasound. *Insig Imag.* 2012;3(6):591–601.
 42. Bayer ML, Magnusson SP, Kjaer M. Early versus delayed rehabilitation after acute muscle injury. *N Engl J Med.* 2017;377(13):1300–1. <https://doi.org/10.1056/NEJMc1708134>.
 43. Mendiguchia J, Martinez-Ruiz E, Edouard P, et al. A multifactorial, criteria-based progressive algorithm for hamstring injury treatment. *Med Sci Sports Exerc.* 2017;49(7):1482–92. https://doi.org/10.1249/mss.00000_00000001241. [published Online First: 2017/03/10]
 44. FCB Muscle Injury Guide. Prevention of and return to play from muscle injuries. © Barça Innovation Hub; 2017; List editors: Ricard Pruna Thor Einar Andersen Ben Clarsen Alan McCall.
 45. van der Made AD, Reurink G, Tol JL, Marotta M, Rodas G, Kerkhoffs GM. Emerging biological approaches to muscle injuries. In: Gobbi A, et al., editors. *Bio-orthopaedics*. Lyon: © ISAKOS; 2017. https://doi.org/10.1007/978-3-662-54181-4_19.
 46. Järvinen M. Muscle injuries: optimizing recovery. *Best Pract Res Clin Rheumatol.* 2007;21:317–331. https://doi.org/10.1007/978-3-662-54181-4_19.
 47. Lempainen L, Kosola J, Pruna R, Puigdellivol J, Sarimo J, Niemi P, Orava S. Central tendon injuries of hamstring muscles. Case series of operative treatment. *Orthop J Sports Med.* 2018;6(10):2325967118798827. <https://doi.org/10.1177/2325967118798827>.
 48. Creighton DW, Shrier I, Shultz R, et al. Return-to-play in sport: a decision-based model. *Clin J Sport Med.* 2010;20:379–85.
 49. Shrier I. Strategic assessment of risk and risk tolerance (StARRT) framework for return to-play decision-making. *Br J Sports Med.* 2015;49:1311–5. *ort Med.* 2010;20:379–385
 50. van der Horst N, Backx FJG, Goedhart EA, et al. Return to play after hamstring injuries in football (soccer): a worldwide Delphi procedure regarding definition, medical criteria and decision-making. *Br J Sports Med.* 2017;51:1583–91. <https://doi.org/10.1136/bjsports-2016-097206>.



Stress Fractures and the Stress Reaction Spectrum in Basketball

44

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44.1 Introduction

Originally termed “march fractures,” stress fractures were first described by Breithaupt in 1855. The invention of radiography 40 years later led to the description of specific radiographic fracture characteristics [1, 2]. The first known diagnosis of a stress fracture in an athlete occurred in 1934, with the diagnosis of a femoral shaft stress fracture [2]. In 1956, Devas provided the first major publication on stress fractures in athletes, reporting the incidence of fibular stress fractures [1, 2].

Stress fractures are more common in athletes because of the repetitive stress and high physical demands they often endure within their specific sport. It is estimated that the general incidence of stress fractures in athletes is 1%; however, in some sports such as cross country and long distance running, the incidence may be as high as 15% [3]. Basketball players are particularly prone to lower-extremity stress fractures because of their repetitive

high-impact jumping and sprinting. Among NBA players from 2005 to 2015, an average of 6 (± 2.6) stress-related injuries occurred annually [3]. The most common stress injuries involved the foot (55%) with 18.4% of stress injuries occurring at the fifth metatarsal [3] (Fig. 44.1).

In general, stress fractures are described as fatigue injuries caused by repeated submaximal stress [2]. Instead of one event delivering enough force to break the bone, a repetitive application of submaximal forces leads to bone insufficiently and eventual fracture [1, 2]. Stress fractures initially present with pain only during exercise or activity but can progress to pain at rest. The patient’s history often reveals a recent increase in either training volume or intensity.

The treatment for most stress fractures is relatively straightforward and involves nonoperative management with a period of rest and activity modification, optimizing nutrition, Vit D levels and calcium intake. However, in athletes, some stress fractures such as displaced femoral necks, tibial shafts, and fifth metatarsals require operative management due to a higher nonunion rate. Among NBA players between 2005 and 2015, 38.2% of stress fractures were managed surgically with 100% (14/14) of fifth metatarsal fractures treated surgically [3].

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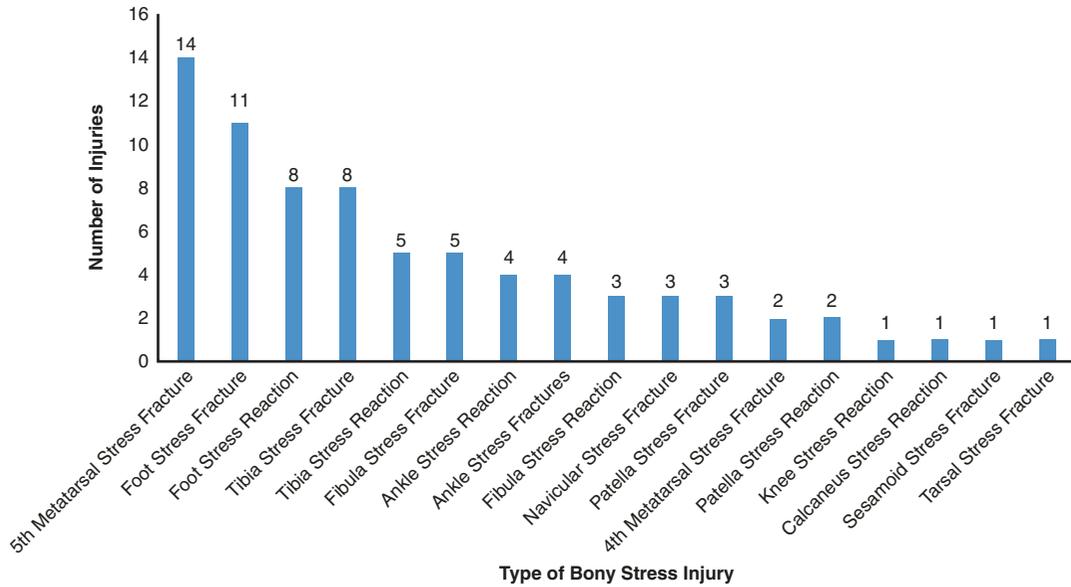


Fig. 44.1 The number and type of stress injuries in the NBA between 2005 and 2015. From: Khan et al. *Epidemiology and impact on performance of lower-*

extremity stress injuries in professional basketball players. Sports Health 2017; 10(2):169–174 (16)

Fact Box 1: Stress Fractures in Athletes [2–4]

- Overall 2–15% incidence in athletes.
- 13–15% incidence in running athletes.
- Foot stress fractures have the highest incidence among NBA players.
- 38% of NBA stress fractures managed surgically.
- Higher incidence in Caucasian athletes.
- Higher incidence among females (10×).
- Higher incidence in female athletes (3.5×) than male athletes.

tures [1]. Without sufficient time for the bone to undergo remodeling and repair before the next insult, the microdamage accumulates, predisposing the bone to a complete fracture [1, 5]. Although a single load is insufficient to cause an acute fracture, the constant and repetitive overloading without time for repair causes this chronic injury. This situation most likely occurs in athletes and military recruits [1, 5].

The other mechanism of stress fractures is associated with the female athlete triad or metabolic bone disease. In this case, you may have normal forces and strain but decreased bone remodeling, thereby predisposing the bone to a stress injury [5].

44.2 Pathophysiology and Etiology

44.2.1 Pathophysiology

Stress fractures are thought to occur by one of the two mechanisms. In healthy metabolic bone, stress fractures can occur from repetitive physiological overloading of the involved bone [1, 5]. The repetitive submaximal forces initially cause elastic deformation to the bone, which progresses to plastic deformity and eventually microfrac-

44.2.2 Risk Factors

Although stress fractures result from repetitive loading, the exact contribution of training factors (e.g., volume, intensity, equipment, and surface) has not been clearly established. Factors such as menstrual disturbances, caloric restriction, decreased bone density, muscle weakness, and leg-length differences are well-known risk factors for stress fractures [6].

Myburgh et al. reported that stress fractures occurred more commonly in athletes who had decreased bone density, lower dietary calcium intake, current menstrual irregularity, and less oral contraceptive use when compared to matched controls with similar training volumes and intensity [7].

Nattiv and Armsey et al. found that genetics, female sex, white ethnicity, low body weight, lack of weight-bearing exercise, amenorrhea, oligomenorrhea, and low calcium and caloric intake were additional risk factors for stress fractures [8]. A decreased testosterone level in male endurance athletes has also been implicated as a risk factor for stress fractures. Athletes found to have low bone mass, and hormonal disturbances may require endocrinologic management [8].

Schnackenburg et al. performed a matched control study on 19 female athletes with tibial stress fractures and demonstrated that stress fracture patients had lower tibial cross-sectional areas and lower trabecular bone mineral densities. They suggested that impaired bone quality of the posterior cortex and decreased muscle strength were associated with stress fractures in female athletes [9].

Giladi et al. identified two anatomic risk factors for tibial stress fractures in military recruits. Recruits with stress fractures had a significantly narrower tibia and increased external rotation of the hip. These two variables were independent and cumulative, and when both risk factors were present, the stress-fracture morbidity was 45% [10].

44.2.3 Incidence

The most common stress fractures in basketball players are those of the lower extremity and include injuries to the foot, the ankle or fibula, the tibia, and the knee/patella [3]. Khan et al. reviewed stress fractures in the NBA from 2005 to 2015 and found that 55% of those involved the foot, 21% involved the ankle or fibula, 17.1% involved the tibia, and 6.6% involved either the patella or knee. The majority of these stress injuries occurred during the regular season, especially within the first 6 weeks [3]. In the national collegiate athletic association (NCAA), an overall incidence of

10.7% was reported in male basketball players and 16.3% in females between 2004 and 2014, with metatarsal stress fractures being the most common (59% of all fractures in males; 46% in females) [11]. Out of these fractures, 19.4% were recurrent in male players, while 20.9% were recurrent in female players [11].

Fact Box 2

- Stress fractures most commonly occur during the regular season with 50% occurring in the first 6 weeks.
- The most common location among basketball players is the foot.

44.2.4 Imaging Studies

Plain radiographs are often the first imaging choice because of the ease of access and low cost [1]. However, stress fractures may not show up on radiographs for the first 2–4 weeks after injury. The first radiographic finding may be a localized periosteal reaction or an endosteal cortical thickening and sclerosis [1, 12].

Fact Box 3: Radiographs and Stress Fractures

- 28% have a positive X-ray finding on the first imaging study
- A periosteal cortical shaft reaction may be found at around 2–3 weeks from pain onset.
- Only 50% of radiographs are positive at 12 weeks.
- 6% never have a positive X-ray.

Historically, triple-phase scintigraphy (technetium-99 m) was the image modality of choice as it could show fracture-associated changes 3–5 days after symptoms initiation [1].

However, magnetic resonance imaging (MRI) is now considered the gold standard for diagnosis, as it is the most sensitive and specific image study available for stress fractures [1, 13]. MRI provides

information regarding bone integrity and fracture orientation, and it can demonstrate focal tissue damage and edema early in the process [12]. Wright et al. carried out a systematic review assessing the diagnostic accuracy of imaging modalities used to diagnose lower-extremity stress fractures, as presented in Fact Box 4 [13].

Fact Box 4: Reported Sensitivities and Specificities (with 95% Confidence Interval) of Imaging Modalities for Stress Fractures [13]

- Conventional radiography—Sensitivity, 12% (0–29%) to 56% (39–72%); specificity, 88% (55–100%) to 96% (87–100%).
- Bone scan/nuclear scintigraphy—Sensitivity, 50% (23–77%) to 97% (90–100%); specificity, 33% (12–53%) to 98% (93–100%).
- MRI—Sensitivity, 68% (45–90%) to 99% (95–100%); specificity, 4% (0–11%) to 97% (88–100%).
- CT—Sensitivity, 32% (8–57%) to 38% (16–59%); specificity, 88% (55–100%) to 98% (91–100%).
- Ultrasonography—Sensitivity, 43% (26–61%) to 99% (95–100%); specificity, 13% (0–45%) to 79% (61–96%).

Table 44.1 Radiologic grading of stress fractures [2, 14]

Grade	Radiographic finding	Bone scan finding	MRI finding
1	Normal	Poorly defined area	Increased activity on STIR image
2	Normal	More intense	Poor definition on STIR and T2-weighted images
3	Discrete line	Sharp area of uptake	No focal or fusiform cortical break on T1- and T2-weighted images
4	Fracture or periosteal reaction	More intense, localized, transcortical uptake	Fracture line on T1- and T2-weighted images

common among athletes, they have a higher incidence in jumping and leaping sports like basketball [2, 15]. Anterior-cortex tibial stress fractures can be problematic as they have been reported to have the highest risk for nonunion [15]. Tibial stress fractures are typically transverse in nature although longitudinal fractures may rarely occur and have a higher nonunion rate [2].

44.3 Grading for Stress Fractures

Several grading systems for stress fractures based on MRI or scintigraphic findings have been developed to help correlate image findings with fracture severity (Table 44.1) [2, 14].

44.4 Lower Extremity Stress Fractures

44.4.1 Tibial Stress Fractures

Tibial stress fractures in athletes occur in three areas of the tibia; the anterior cortex, the tibial plateau, and the posteromedial cortex. Although stress fractures in the anterior cortex are the least

44.4.2 Presentation and Physical Evaluation

Typically, athletes will complain of leg pain after a period of activity without any history of clear trauma. Initially, pain will increase with prolongation of activity and decrease with rest. However, as the fracture progresses, symptoms may occur even at rest and at night [2, 15].

Tibial stress fractures can be hard to clinically distinguish from tibial periostitis, medial tibial stress syndrome (MTSS), or “shin splints”; however, pain persisting after activity or with daily ambulation is a key feature to differentiate them [4].

Physical exam findings include localized tibial tenderness and pain with direct palpation or percussion [4]. Percussion away from the fracture sight may also cause discomfort. A few specific physical exam maneuvers have been

described. The hop test, where the patient performs a repeated single leg hop $\times 10$, can reproduce the pain. Additionally, the fulcrum test, where a bending moment is placed on the tibia either by the physician's arm, knee, or exam table, will produce severe pain in patients with a tibial stress fracture [15].

Fact Box 5: Differentiating Between MTSS and Tibial Stress Fracture

- The hop test could aid in differentiating between MTSS and tibial stress fractures as MTSS are usually able to complete the test while patients with a tibial stress fracture rarely could.
- Most athletes report a crescendo-type pain with stress fractures as the pain increases through individual workouts and from one workout to the next. Shin splint pain tends to be present at the start of activity in those athletes who are symptomatic.
- Physical examination of an athlete with shin splints should reveal tenderness to palpation over a wide region of the tibia and the tibialis muscle, whereas the pain from stress fractures tends to be localized to a specific area on the tibia.

44.4.3 Imaging

Initial imaging should include anteroposterior (AP), lateral, and oblique plain radiographs of the tibia although these are often normal and have a relatively low sensitivity (10–50%) for stress fractures. Positive findings include thickening of the cortex, pretibial swelling, and a visible fracture line or a “dreaded black line” (Fig. 44.2). If initial radiographs are negative, the recommendation is to repeat the imaging in 2 weeks if symptoms persist [15].

MRI findings include early increased signal in the endosteum on T2-weighted sequences and a possible visible fracture line. MRI may also help rule out any other pathology [4, 16].

44.4.4 Treatment

The majority of tibial stress fractures, especially those involving the posteromedial cortex, can be treated successfully with nonoperative management. Treatment includes a period of rest followed by a gradual return to low impact exercises and then high-impact activity [4, 15, 17]. Other strategies such as pneumatic bracing, low-intensity pulsed ultrasound (LIPUS), and extra-corporeal shockwave therapy (ECST) have demonstrated increased healing and time to union in several studies [15].

Surgical management is reserved for high-risk fractures such as those involving the anterior cortex that have a greater risk of non-union or delayed union [15, 17]. Anterior-cortex stress fractures of the tibia are considered high risk because of the tensile forces across the anterior aspect of the tibia [16]. Additionally, in athletes, surgical management might allow a faster return to play. Several surgical techniques exist including intramedullary nailing and tension band plating. Intramedullary nailing is commonly used and has demonstrated promising results in the literature [18] (Fig. 44.3). The major downside of this technique is the high incidence of anterior knee pain following the procedure. To avoid the sequela of anterior knee pain, a 2.7-mm or 3.5-mm plate applied on the anterior cortex in compression mode can be used. One study demonstrated 100% union in 13 tibial stress fractures at an average of 9.6 weeks treated with tension band plating [19]. The downside of this technique is hardware irritation as these often need for a second surgery to remove the hardware [15].

44.4.5 Return to Play

Tibial stress fractures of the posteromedial cortex treated nonoperatively tend to heal within 4–8 weeks with an average time to return to sport of 8–12 weeks [20]. Higher risk anterior-cortex fractures may take up to 12 months for return to play [2]. As mentioned above, operative treatment of these fractures can expedite return to



Fig. 44.2 AP and lateral radiographs of the tibia demonstrating a mid-shaft anterior-cortex stress fracture (white arrow)

play to around 4 months [18, 19]. However, Khan et al. demonstrated high rates of inability to play (69.2%) among NBA players with either tibial stress fractures or tibial stress reactions [3].

44.5 Metatarsals

Metatarsal stress fractures were originally described in military recruits and termed “march fractures.” Fractures occur commonly in the neck and shaft of the second and third metatarsal [4]. Particularly common in basketball players are

fractures of the fifth metatarsal at the metaphysis/diaphysis junction called a “Jones fracture.” Khan et al. found that among NBA players, a fracture of the fifth metatarsal was the most commonly reported stress fractures (18.4%) [3].

44.5.1 Presentation and Physical Evaluation

Athletes present with pain and tenderness around the affected metatarsal, with occasional swelling [4]. For fifth metatarsal fractures, athletes will

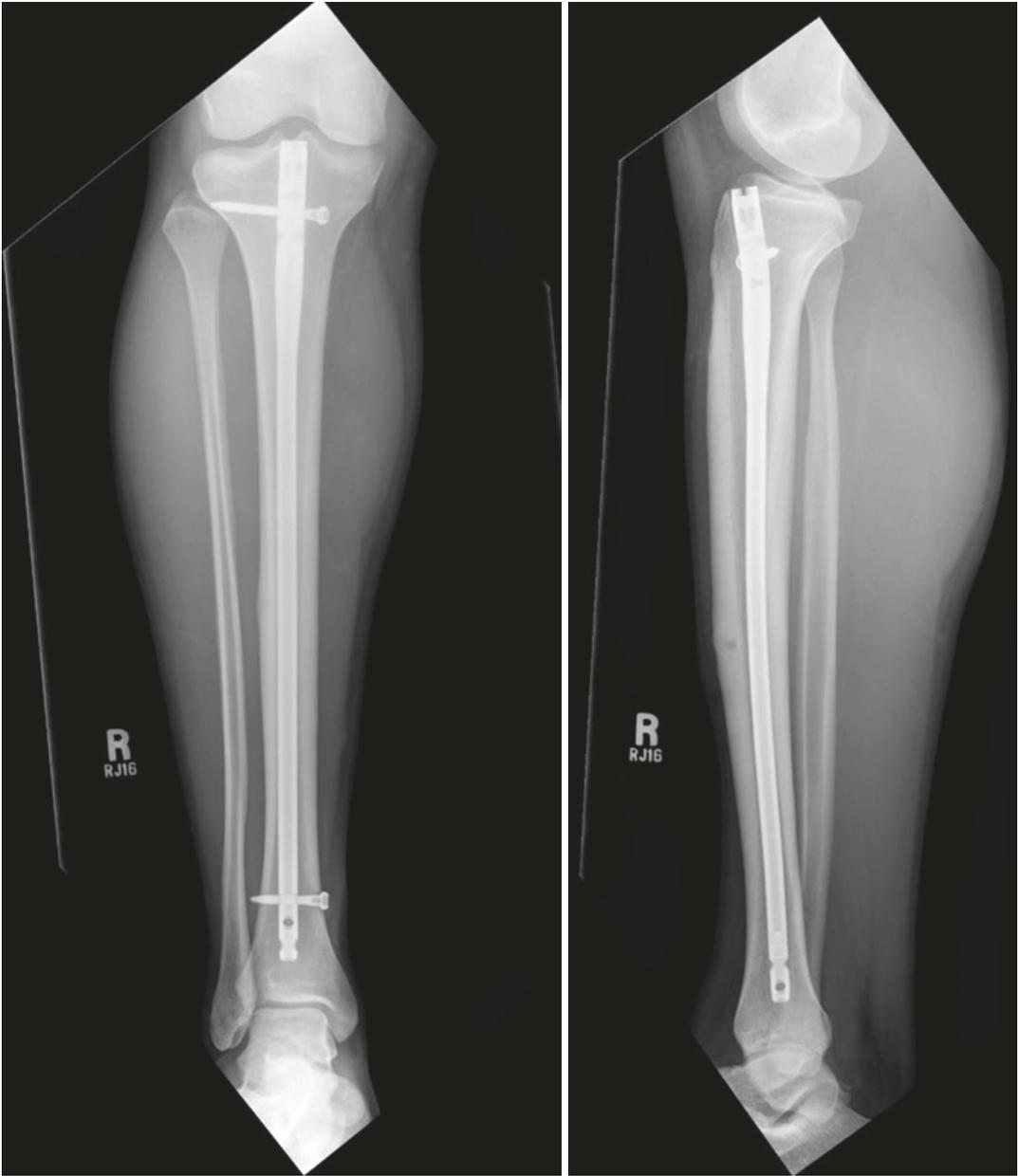


Fig. 44.3 AP and lateral radiographs following intramedullary nail fixation for an anterior-cortex tibia stress fracture

present with lateral foot pain and fifth metatarsal pain exacerbated by inversion [21]. Jones fractures can occur as an acute fracture or as a true stress fracture. Athletes may report pain after a specific event; however, typically the bone has undergone repetitive microtrauma that has predisposed it to injury [4]. Studies have demonstrated that up to 59% of Jones fractures can be classified as true stress injuries [4].

44.5.2 Diagnosis

Initial weight-bearing radiographs may appear normal. Periosteal bone reaction can be visible on plain radiographs 2–3 weeks after injury [4, 20]. Radiographs may also reveal a radiolucent fracture line and intramedullary sclerosis [22] (Fig. 44.4).

MRI will depict bone marrow and periosteum edema and a possible fracture line [20]. Athletes



Fig. 44.4 Oblique radiograph of a right foot demonstrating a fifth metatarsal stress fracture (white arrow)

with fifth metatarsal fractures may have predisposing factors including a protruding fifth metatarsal head, a cavovarus foot, or an increased inter-metatarsal angle between the fourth and fifth metatarsal [21, 23].

44.5.3 Treatment

Most metatarsal stress fractures can be successfully treated with activity modification and return to sport in 4–6 weeks when ambulation pain subsides [20]. Stress fractures of the fifth metatarsal typically occur in a watershed zone between the nutrient artery and metaphyseal artery, at the metaphysis/diaphysis junction [4, 24]. These fractures have a high risk of delayed union, nonunion, or refracture



Fig. 44.5 ORIF of right fifth metatarsal stress fracture

if treated nonoperatively because of the poor blood supply [22]. If the fracture is diagnosed early, prior to radiographic changes, a period of non-weight-bearing and an unloader brace may be curative [22]. Otherwise, most high-level athletes with Jones fracture will undergo operative treatment with ORIF using a compression screw \pm bone grafting [4, 22, 23] (Fig. 44.5). A functional metatarsal brace may be used postoperatively for 1 month to avoid refracture [22]. Khan et al. reported that 100% (14/14) of fifth metatarsal fractures seen in NBA players were treated with ORIF [3].

44.5.4 Return to Play

Return to play following operative treatment is generally around 6–8 weeks [4]. O'Malley

et al. observed the outcomes of 10 NBA players with Jones fractures treated surgically and found that radiographic healing was observed around 7.5 weeks and return to play at 9.8 weeks [23]. Khan et al. found that 42.9% (6/14) of NBA players who suffered a fifth metatarsal fracture from 2005 to 2015 were unable to return to play following injury [3]. This is in contrast to Begly et al. who reported that of 26 NBA players with Jones fractures from 1994 to 2013, 85% returned to their previous level of competition. They also noted that 19% (5) of players in their study had recurrence of their injury despite initial surgical management, stressing the increased strain these athletes put on this fracture, warranting careful postoperative care [25].

44.6 Navicular

Although rare in the general population, navicular stress fractures are more common in elite-level athletes, accounting for 25% of all the diagnosed stress fractures [26]. They are frequently observed in short-distance runners and basketball players due to high-intensity jumping, explosive movement, and rapid cutting that leads to forceful and repetitive loading of the forefoot [26].

The navicular is an important structural link between the midfoot and hindfoot, allowing for forceful push-off during gait. Studies have demonstrated an avascular region of the navicular predisposing it to risk of fracture with repetitive load and delayed healing [26].

44.6.1 Presentation and Physical Evaluation

Patients present with vague midfoot or medial arch pain that is aggravated by weight-bearing activities [22, 27]. Initially, patients may present with pain just at the initiation of activities. They will have tenderness on palpation over the navicular bone, particularly the proximal dorsal area known as the “N-spot” [26, 27]. They may

have limited subtalar motion or dorsiflexion of the ankle [22]. They often will have pain standing on the tip of their toes and pain with a single leg hop [26].

44.6.2 Diagnosis

Initial imaging includes weight-bearing (AP, lateral, and oblique) radiographs. Although plain radiographs have low sensitivity for diagnosing navicular stress fractures, they can be used to rule out other sources of midfoot pain. Navicular stress fractures are seen in the sagittal plane in the central third of the bone, which correlates to the area of maximal shield stress [4, 22]. They began at the proximal dorsal articular surface and propagate plantar. Since they do not often involve the plantar cortex, it makes them hard to distinguish on plain radiographs [26](Fig. 44.5).

Due to often vague symptoms and negative X-rays, diagnosis of navicular stress fractures can be delayed 4–7 months from the onset of symptoms [26].

MRI T2-weighted images will show bone edema and stress reaction early on, even before an actual fracture line is apparent [26].

CT is useful for preoperative planning as it will best demonstrate the orientation of the fracture line. Additionally, it can be used to help determine fracture healing [26] (Fig. 44.6).

44.6.3 Treatment

Early diagnosed partial or complete navicular stress fracture can successfully be managed nonoperatively with a non-weight-bearing cast for 6–8 weeks until the bone is nontender [22, 28]. The other option is to fix the fractures with percutaneous screws in situ followed by a period of 6–8 weeks in a non-weight-bearing cast. This is followed by another 6 weeks of rehabilitation where a semi-rigid custom orthosis may be used for arch support [4]. Adjunctive therapy may include bone stimulator, shock wave therapy, Vitamin-D supplementation, and teraparotide [26]. Complete or Displaced frac-



Fig. 44.6 (a) AP radiograph of a left foot demonstrating increased sclerosis and cortical thickening and subtle fracture line at the midline of the navicular bone. (b) Axial CT image demonstrating midline dorsal navicular stress fracture

tures, delayed union, or non-unions should be treated with open ORIF and bone grafting or bone marrow aspirate [29]. This is done with one or two compression screws across the fracture site [22], followed by a period of non-weight-bearing until healing has occurred [4, 26] (Fig. 44.7).

44.6.4 Return to Play

On average, return to play takes between 4 and 5 months after treatment [4, 26, 30]. Recently, Saxena et al. prospectively analyzed the outcomes of 62 athletes diagnosed with navicular stress fractures treated with either ORIF (47) or nonoperative management (15) [27]. Return to activity was similar for both groups; $4.56 \pm 1/5$ months for the

ORIF group and 3.97 ± 1.9 months for the nonoperative group ($p = 0.28$) [27]. Of the 62 navicular stress fractures, 57 (91.9%) were able to return to play at their previous activity level [27]. Previous studies have typically demonstrated a faster return to play with ORIF [30, 31].

Whalen et al. prospectively followed NFL players who had a navicular stress fractures and found that although the prevalence of the injury was low, those with a history of a navicular stress fracture had a significantly lower chance of getting drafted ($p = 0.001$) and playing >2 years in the NFL compared to controls ($p = 0.2$) [32].

Finally, Khan et al. demonstrated that among NBA players with navicular stress fractures between 2005 and 2015, two-third of players were not able to return to play [3].



Fig. 44.7 (a) Lateral radiograph and (b) axial CT status post ORIF of a left navicular stress fracture

44.7 Great Toe Sesamoids

Sesamoid stress fractures are uncommon but can lead to disability and dysfunction of an athlete if untreated. They comprise around 1–3% of all stress fractures [11]. The injury is seen slightly more in the medial (tibial) sesamoid which lies directly under the head of the first metatarsal [33]. Repetitive dorsiflexion of the great toes such as in sprinting and jumping can cause forces sufficient enough to cause a transverse fracture [21, 22, 34].

44.7.1 Presentation and Physical Evaluation

Athletes present with tenderness directly over the plantar aspect of the first metatarsophalangeal joint and pain with passive dorsiflexion of the first toe [33, 34].

44.7.2 Diagnosis

Initial radiographs include weight-bearing AP, lateral and axial views centered on the sesamoids [11] (Fig. 44.8). Fractures must not be confused

with bipartite sesamoids which is seen in 5–30% of the general population [22].

44.7.3 Treatment

Acute stress fractures are treated conservatively with 2–6 months of activity cessation and modification, often involving up to 6 weeks in a non-weight-bearing cast which extends to the distal end of the great toe to prevent dorsiflexion [22, 33, 34]. Great toe sesamoid stress fractures have a high rate of delayed union or nonunion, often indicating early operative treatment [21]. Surgery typically involves excision of the fractured sesamoid or ORIF, with caution not to disrupt the flexor hallucis longus and brevis [22, 34].

44.7.4 Return to Play

In a meta-analysis of 14 studies, Robertson et al. demonstrated no significant difference in return to play rate with either nonoperative (86%) or operative management (95%). However, return time to sport was significantly faster with surgically managed fractures (11 weeks) than nonoperative treatment (13.9 weeks, $p = 0.017$).



Fig. 44.8 (a) AP radiograph and (b) axial T2-weighted MRI of a fibular sesamoid fracture (white arrow)

Fractures treated with a sesamoidectomy (10.5 weeks) returned faster ($p < 0001$) than those treated with internal fixation (11.8 weeks). However, internal fixation did demonstrate the highest return to sport (100%) and the best return rate to the same level of sport (100%) [34].

44.8 Stress Fracture Prevention

Modification of physical activity duration and appropriate recovery time is imperative in stress fracture prevention. Additionally, ensuring an adequate daily intake of calcium (2000 mg) and Vitamin D (800 IU) may have protective measures [1].

Understanding and correcting poor athlete biomechanics and kinematics may also decrease the risk of stress fractures. Protective biomechanical measures include the use of orthotic devices and shoe inserts [35]. Finestone et al. found a lower incidence of lower-extremity stress fractures in military cadets using semirigid orthoses (15.7%) or soft biomechanical orthoses (10.7%) versus controls (27%). Additionally, the recruits toler-

ated the soft biomechanical orthoses better than they did the semi-rigid orthoses [36]. It is also important to identify players at risk for a stress fracture and provide cautious management of their loads as well as address nutritional deficits.

44.9 General Return to Sport Considerations Following Stress Fractures in Basketball

Injury management in the professional athlete is complex, and many factors may affect various return to sport (RTS) aspects, from time to RTS to return to pre-injury level of play. Several considerations have been highlighted with regard to RTS following stress fractures or stress reaction in basketball players.

Khan et al. have shown that the strongest predictor of overall performance postinjury was player efficiency rating (PER) in the year before the injury [3]. There were no other factors that were significantly associated with performance at 1 year post-

injury. However at 2 years, differences became more apparent, highlighting, unsurprisingly, that players who sustained a stress fracture as opposed to a stress reaction performed significantly worse after 2 years. However, players who were treated surgically had significantly better performance at 2 years than those who were managed conservatively, independent of the type of injury [3].

Take Home Messages

- Stress fractures are an important group of sports injuries seen in high-level athletes due to their high physical demand. Diagnosis of these injuries can be difficult but is key in order to avoid delay in treatment and faster return to play.
- In basketball, the most common types of stress fracture are those of the lower extremity.
- The need for a fast return to play pushes clinicians around sports to look for treatment options that can safely expedite good-quality bone healing with the lowest chance of recurrent injury.
- Particularly in high-level athletes, operative treatment for these fractures is often the best option to ensure the fastest recovery and return to play.
- Biologic therapies such as stem cells and autologous growth factors are becoming more commonly utilized, and, with more scientific evidence, could be a helpful adjunct in the future of stress fracture management.

References

1. Astur DC, Zanatta F, Arliani GG, Moraes ER, Pochini Ade C, Ejnisman B. Stress fractures: definition, diagnosis and treatment. *Rev Bras Ortop.* 2016;51(1):3–10.
2. Behrens SB, Deren ME, Matson A, Fadale PD, Monchik KO. Stress fractures of the pelvis and legs in athletes: a review. *Sports Health.* 2013;5(2):165–74.
3. Khan M, Madden K, Burrus MT, et al. Epidemiology and impact on performance of lower extremity stress injuries in professional basketball players. *Sports Health.* 2018;10(2):169–74.
4. Fredericson M, Jennings F, Beaulieu C, Matheson GO. Stress fractures in athletes. *Top Magn Reson Imaging.* 2006;17(5):309–25.
5. Pepper M, Akuthota V, McCarty EC. The pathophysiology of stress fractures. *Clin Sports Med.* 2006;25(1):1–16. vii
6. Popp KL, Hughes JM, Smock AJ, et al. Bone geometry, strength, and muscle size in runners with a history of stress fracture. *Med Sci Sports Exerc.* 2009;41(12):2145–50.
7. Myburgh KH, Hutchins J, Fataar AB, Hough SF, Noakes TD. Low bone density is an etiologic factor for stress fractures in athletes. *Ann Intern Med.* 1990;113(10):754–9.
8. Nattiv A, Armsey TD Jr. Stress injury to bone in the female athlete. *Clin Sports Med.* 1997;16(2):197–224.
9. Schnackenburg KE, Macdonald HM, Ferber R, Wiley JP, Boyd SK. Bone quality and muscle strength in female athletes with lower limb stress fractures. *Med Sci Sports Exerc.* 2011;43(11):2110–9.
10. Giladi M, Milgrom C, Simkin A, Danon Y. Stress fractures. Identifiable risk factors. *Am J Sports Med.* 1991;19(6):647–52.
11. Rizzone KH, Ackerman KE, Roos KG, Dompier TP, Kerr ZY. The epidemiology of stress fractures in collegiate student-athletes, 2004–2005 through 2013–2014 academic years. *J Athl Train.* 2017;52(10):966–75.
12. Deutsch AL, Coel MN, Mink JH. Imaging of stress injuries to bone. Radiography, scintigraphy, and MR imaging. *Clin Sports Med.* 1997;16(2):275–90.
13. Wright AA, Hegedus EJ, Lenchik L, Kuhn KJ, Santiago L, Smoliga JM. Diagnostic accuracy of various imaging modalities for suspected lower extremity stress fractures: a systematic review with evidence-based recommendations for clinical practice. *Am J Sports Med.* 2016;44(1):255–63.
14. Arendt EA, Griffiths HJ. The use of MR imaging in the assessment and clinical management of stress reactions of bone in high-performance athletes. *Clin Sports Med.* 1997;16(2):291–306.
15. Feldman JJ, Bowman EN, Phillips BB, Weinlein JC. Tibial stress fractures in athletes. *Orthop Clin North Am.* 2016;47(4):733–41.
16. Gmachowska AM, Zabicka M, Pacho R, Pacho S, Majek A, Feldman B. Tibial stress injuries - location, severity, and classification in magnetic resonance imaging examination. *Pol J Radiol.* 2018;83:e471–81.
17. Young AJ, McAllister DR. Evaluation and treatment of tibial stress fractures. *Clin Sports Med.* 2006;25(1):117–28. x
18. Varner KE, Younas SA, Lintner DM, Marymont JV. Chronic anterior midtibial stress fractures in athletes treated with reamed intramedullary nailing. *Am J Sports Med.* 2005;33(7):1071–6.
19. Zbeda RM, Sculco PK, Urch EY, et al. Tension band plating for chronic anterior Tibial stress fractures in high-performance athletes. *Am J Sports Med.* 2015;43(7):1712–8.
20. Liang SY, Whitehouse RW. Lower extremity and pelvic stress fractures in athletes. *Br J Radiol.* 2012;85(1016):1148–56.

21. Welck MJ, Hayes T, Pastides P, Khan W, Rudge B. Stress fractures of the foot and ankle. *Injury*. 2017;48(8):1722–6.
22. Boden BP, Osbahr DC. High-risk stress fractures: evaluation and treatment. *J Am Acad Orthop Surg*. 2000;8(6):344–53.
23. O'Malley M, DeSandis B, Allen A, Levitsky M, O'Malley Q, Williams R. Operative treatment of fifth metatarsal Jones fractures (zones II and III) in the NBA. *Foot Ankle Int*. 2016;37(5):488–500.
24. Afsar T, Razak S, Khan MR, Almajwal A. Acacia hydaspica ethyl acetate extract protects against cisplatin-induced DNA damage, oxidative stress and testicular injuries in adult male rats. *BMC Cancer*. 2017;17(1):883.
25. Begly JP, Guss M, Ramme AJ, Karia R, Meislin RJ. Return to play and performance after Jones fracture in National Basketball Association Athletes. *Sports Health*. 2016;8(4):342–6.
26. Shakked RJ, Walters EE, O'Malley MJ. Tarsal navicular stress fractures. *Curr Rev Musculoskelet Med*. 2017;10(1):122–30.
27. Saxena A, Behan SA, Valerio DL, Frosch DL. Navicular stress fracture outcomes in athletes: analysis of 62 injuries. *J Foot Ankle Surg*. 2017;56(5):943–8.
28. Chen YT, Tenforde AS, Fredericson M. Update on stress fractures in female athletes: epidemiology, treatment, and prevention. *Curr Rev Musculoskelet Med*. 2013;6(2):173–81.
29. Ahlen M, Roshani L, Liden M, Struglics A, Rostgard-Christensen L, Kartus J. Inflammatory cytokines and biomarkers of cartilage metabolism 8 years after anterior cruciate ligament reconstruction: results from operated and contralateral knees. *Am J Sports Med*. 2015;43(6):1460–6.
30. Saxena A, Fullem B. Navicular stress fractures: a prospective study on athletes. *Foot Ankle Int*. 2006;27(11):917–21.
31. Khan KM, Fuller PJ, Brukner PD, Kearney C, Burry HC. Outcome of conservative and surgical management of navicular stress fracture in athletes. Eighty-six cases proven with computerized tomography. *Am J Sports Med*. 1992;20(6):657–66.
32. Vopat B, Beaulieu-Jones BR, Waryasz G, et al. Epidemiology of navicular injury at the NFL combine and their impact on an athlete's prospective NFL career. *Orthop J Sports Med*. 2017;5(8):2325967117723285.
33. Nihal A, Trepman E, Nag D. First ray disorders in athletes. *Sports Med Arthrosc Rev*. 2009;17(3):160–6.
34. Robertson GAJ, Goffin JS, Wood AM. Return to sport following stress fractures of the great toe sesamoids: a systematic review. *Br Med Bull*. 2017;122(1):135–49.
35. Milgrom C, Giladi M, Kashtan H, et al. A prospective study of the effect of a shock-absorbing orthotic device on the incidence of stress fractures in military recruits. *Foot Ankle*. 1985;6(2):101–4.
36. Finestone A, Giladi M, Elad H, et al. Prevention of stress fractures using custom biomechanical shoe orthoses. *Clin Orthop Relat Res*. 1999;360:182–90.



The Role of Orthobiologics in the Management of Tendon and Fascia Injuries in Sports

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45.1 Introduction

Orthobiologics have emerged as a promising treatment modality, seeking to enhance musculo-skeletal regeneration and repair. This overarching term comprises many developing treatments, including isolated growth factors, platelet-rich plasma (PRP), cell-based therapies, and scaffolds. Preclinical studies and initial enthusiasm have resulted in substantial research efforts. Preliminary results of these efforts suggest improved function, decreased pain, and early return to play in several different soft tissue injuries; however, true reproducible soft tissue regeneration has not been demonstrated [1, 2].

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As in all athletes, tendon and fascia injuries are very prevalent in basketball players. Common tendon and fascia injuries in athletes will be addressed in this chapter including patellar tendinopathy, Achilles tendinopathy, Achilles tendon rupture, plantar fasciitis, and rotator cuff pathology. The optimal treatment for soft tissue injuries is under debate, but most can be managed with conservative measures, including rest, icing, physical therapy, and analgesics for symptomatic relief. While these treatments are often effective, there is increasing interest in the role of orthobiologics in promoting healing, reducing pain, and enabling early return to play [3]. This chapter will relay the most current evidence regarding the efficacy of different orthobiologics for the treatment of tendon injuries in athletes [1].

45.2 Orthobiologics

45.2.1 Corticosteroids

Corticosteroids have long been used as anti-inflammatory agents in the treatment of soft tissue injuries in athletes. Corticosteroids can be given intravenously or orally for a systemic effect or injected (intra-muscular, intra-articular, intra-bursal, intra-tendinous, or peritendinous) for a more local effect. Either way, corticosteroids are well known for their ability to induce symptomatic relief.

Corticosteroids decrease leukotrienes, prostaglandins, thromboxane A₂, and prostacyclin, as well as by stabilize lysosomal membranes of inflammatory cells, decreasing vascular permeability, altering neutrophil chemotaxis and function. They also possess the ability to cross cell membranes and influence RNA transcription and subsequent protein production [4, 5].

Several clinical studies have reported improved outcomes with corticosteroids injections for soft tissue injuries in athletes. Levine et al. have reported improved return to play following corticosteroids and anesthetic injection for severe hamstring injuries in 58 National Football League (NFL) players. They have also reported no complications related to the injection [6]. Stahl et al. performed a prospective, randomized, double-blinded study to assess the effect of methylprednisolone on medial epicondylitis. They have reported short-term improvement in symptoms at 6 weeks follow-up in the experimental group; however, later follow-ups did not differ with regard to pain [7]. For the treatment of Achilles tendinopathy, corticosteroids have shown no benefit when compared to nonsteroidal anti-inflammatory drugs (NSAIDs) [8].

The use of corticosteroids should be carefully considered due to its significant side effects. There are several potential reported systemic side effects of corticosteroids including, but not limited to, diabetes, weight gain, hypertension, and psychosis when used systemically. Local injections in high concentrations may also confer systemic side effects, as well as local side effects [5]. Nichols performed a meta-analysis reporting on the complications associated with the use of corticosteroids in the treatment of athletic injuries. He reported local side effects including tendon weakening and rupture, postinjection pain flares, subcutaneous fat atrophy, and skin hypopigmentation. As for specific rates for tendon ruptures, he reported plantar fascia rupture to be the most common (53.7%), quadriceps/patellar tendon rupture rates were 9.5%, Achilles tendon rupture rates were 8.4%, and biceps tendon rupture rates were as high as 8.4% as well [9].

Fact Box

The use of corticosteroids should be carefully considered, mainly due to its significant side effects, which includes significant rates of tendon rupture, muscle weakness, and atrophy. However, in select cases their use is applicable and has shown favorable results.

45.2.2 Platelet-Rich Plasma (PRP)

The use of PRP for the management of soft tissue injuries has become increasingly common over the last decade [10, 11]. This is probably due to its potential benefits including its safety, efficient delivery of growth factors, and proteins that might modify acute and chronic pathology, and the potential for expedited recovery from soft tissue injuries used in isolation or as adjunctive treatment [11].

PRP is created by the process of centrifugation of a patient's own blood to produce small-volume plasma with high platelet concentration. Platelets contain an abundance of growth factors (transforming growth factor [TGF]- β 1, platelet-derived growth factor, basic fibroblast growth factor, vascular endothelial growth factor, epidermal growth factor, insulin-like growth factor [IGF]-1), which may modify the inflammatory response and impact cell differentiation and proliferation [12–14]. Previous literature has defined PRP as any plasma volume with a platelet concentration above baseline [15]. However, recent literature supports defining PRP as a volume of plasma that has a platelet count of over one million platelets per milliliter (mL) [16, 17]. PRP with a platelet concentration above this proposed cutoff is thought to have a clinically significant impact on tissue healing [17, 18]. However, other authors reported that increased platelet concentration beyond the physiologic level did not improve functional graft healing in an anterior cruciate ligament (ACL) [19] and medial collateral ligament (MCL) animal models [20]. More recent studies are aiming to define optimal concentrations to

be used to induce healing according to the specific injured tissue [21–23].

Preparation protocols vary between the many available commercial PRP systems. Generally, blood is drawn from the patient and is mixed with an anticoagulant. Subsequently, a 1- or 2-step centrifugation separates the red blood cells, platelet-poor plasma, and the “buffy-coat”. The buffy-coat which contains the highly concentrated platelets and leukocytes and the plasma is then isolated for a second centrifugation (when a manual system is utilized). Prior to the injection, some commercial systems recommend “activation” of the platelets using thrombin or calcium chloride, in order to induce platelet degranulation and release of growth and differentiation factors [24]. A recent analysis of the reporting of PRP processing for musculoskeletal conditions (105 studies) showed that only 11.5% of studies reported on all necessary variables of PRP processing required to repeat the protocol [25]. Moreover, there was no consensus in the machines to be used to prepare the PRP (manual or automatic), number of spins, speed, and time of centrifugation. Automated commercial systems and manual processing methods are used to minimally manipulate desired blood fractions to concentrate LR-PRP and LP-PRP but have been found to produce product variations in blood cell and growth factor concentrations [26–30]. In this regard, both systems can produce similar results when performed correctly [31].

PRP can further be stratified to leukocyte (neutrophil)-rich PRP (LR-PRP) and leukocyte-poor PRP (LP-PRP) according to the white blood cell concentration. It has been suggested that LR-PRP can produce pro-inflammatory effects by induction of interleukin-1 β (IL-1 β), tumor necrosis factor (TNF)- α , and metalloproteinases, which may adversely affect tissue healing [32–35]. Recent two case series by Hanisch et al. have found no significant difference in effect between LR-PRP and LP-PRP for Achilles tendinopathy [36]. Many variables contribute to the preparation of what is broadly named PRP, thus the discovery of the optimal preparation method for each unique type of patient, tissue, and injury remains elusive.

Fact Box

The World Anti-Doping Agency (WADA) does not prohibit PRP use generally; however, use of independent growth factors (such as PDGF, VEGF, IGF-1, and FGF) is prohibited. Stem-cell-based therapies may or may not be prohibited, depending on how the cellular material is manipulated or modified for use [37].

45.2.3 Cell-Based Therapies

The rationale for use of mesenchymal stem cells (MSC) is the potential to improve symptoms and possibly augment healing of tissues that have relatively poor intrinsic healing ability such as cartilage, muscle, tendon, ligament, meniscus, and soft tissue to bone interfaces. Although pre-clinical studies suggest promising potential for MSC to enhance tissue healing, there is limited clinical data to support the use of MSC for the management of musculoskeletal pathologies. These mostly unsubstantiated therapies are being aggressively marketed directly to athletes, with unproven claims regarding their efficacy on outcomes and early return to play.

45.2.3.1 Bone Marrow Aspirate Concentrate (BMAC)

As one of the few techniques approved by the United States Food and Drug Administration (FDA) for the delivery of stem cells, bone marrow aspirate concentrate (BMAC) has gained popularity in recent years [38]. Aside from progenitor cells, BMAC is reported to contain an abundance of growth factors and cytokines [33, 39, 40]. All together the contents of BMAC are thought to promote neogenesis, tissue regeneration, immunomodulatory, and anti-inflammatory effects [38, 41].

Bone marrow aspirate is usually performed by percutaneous aspiration of trabecular bone of the iliac crest due to the ease of procurement, relatively low donor site morbidity and a high concentration of progenitor cells [42] (Fig. 45.1). Using a small syringe and multiple aspirations at



Fig. 45.1 Bone marrow aspiration from proximal tibia



Fig. 45.2 Bone marrow aspiration centrifugation

different locations have been reported to increase progenitor cells concentration [43]. The bone marrow aspirate is then centrifuged in order to separate the mesenchymal stem cells (MSC) [44, 45] (Fig. 45.2). MSC concentration following centrifugation is still relatively low and is estimated to be around 0.001–0.01% [45]. Moreover, the true number of viable MSC that are actually delivered into the lesion is unknown, regardless of the tissue used to procure the cells. To increase the number of MSC, the cells are to be transferred to a lab and undergo cell isolation and culture expansion. However, such laboratory processing of cell preparations is prohibited in the United States, by the Food and Drug Administration (FDA).

Many preclinical and clinical studies have supported the use of BMAC, mainly for the treatment of cartilaginous and articular pathologies (e.g., meniscal injuries) [46–51]; however, there

is a paucity of studies supporting the use of BMAC for the treatment of soft tissue injuries.

45.2.3.2 Other Cell-Based Therapies

MSC were first discovered in bone marrow; however, later studies revealed their presence in fetal tissue (umbilical cord and placenta), as well as in adult tissue (adipose tissue, periosteum, blood vessels, synovium, endometrium, dermis, and more) [52–54].

Commonly used MSC sources are autologous adipose tissue and allogenic amniotic tissue. Amniotic tissue contains higher concentrations of MSC when compared to the aforementioned BMAC, with concentrates of 0.9–1.5% [55]. Advantages for the use of amniotic stem cells are high plasticity and pluripotency of the cells, low immunogenicity, high capability to differentiate to major cell lineages, and the lack of donor site morbidity [55]. Adipose tissue is also a common source of MSC due to the abundance of MSC in adipose tissue and the relative ease of access and harvesting of adipose tissue. Adipose MSC are autologous and therefore also raise less ethical concerns [56].

Preclinical studies have supported the use of MSC for various applications. Most preclinical studies have concentrated on the effect of MSC in the treatment of articular cartilage pathology and bone healing [57–63]. Although the use of MSC for the treatment of soft tissue pathology has not been studied extensively, several preclinical studies have assessed the efficacy of MSC in rotator cuff pathology, with conflicting results. Gullota et al. and Yokoya et al. have reported that MSC promote healing of the rotator cuff in rat and rabbit models, respectively [64, 65]. Other studies by Gullota et al. and Chen et al. have raised doubts regarding the ability of MSC to improve rotator cuff healing when used in isolation [66–68]. A recent study by Ma et al. has shown potential benefit of human placenta-derived cells in patellar tendon injury in rats [69]. Many more clinical studies are in progress however to date, there is no high-quality evidence to support the use of MSC in the treatment of soft tissue injuries [70, 71].

Due to the exponential growth in cell-based treatments worldwide without standardization

and transparency, an international expert consensus proposed the DOSES tool for describing cell therapies, which was aimed to allow for better assessment and comparison of different treatments and techniques in the future [70]. This is of critical importance as lack of standardization and rigorous protocols may expose the athlete to serious adverse side effects and complications, including severe infections.

Cell-based therapies have an immense potential to improve management of soft tissue injuries in the athlete. However, more research is required to optimize the treatment protocol for each type of injury in regard to preparation techniques, dosing, delivery, and rehabilitation. Future research efforts should define the best indications and applications for biologic therapies in a way that maximizes both the benefit and the safety of the athlete.

45.3 Soft Tissue Injuries: Tendons and Fascia

The use of orthobiologics for tendon and fascia pathologies is less frequently studied compared to bone and cartilage pathologies [72]. Determining the optimal protocol for the treatment of tendon pathology should begin with differentiating acute tears from chronic degenerative and overuse tendinopathy or tendinosis. While in acute tears the goal may be to increase cellular proliferation and promote/enhance healing, in chronic tendinopathy the goal may more likely be to target the inhibition of matrix-degrading proteases and inflammatory mediators and possibly “jump-start” a healing response. Other considerations include the specific tendon involved, the location within the tendon (myotendinous junction, intra-tendon, or avulsion injuries), timing, and dosing [73].

45.3.1 Patellar and Quadriceps Tendinopathy

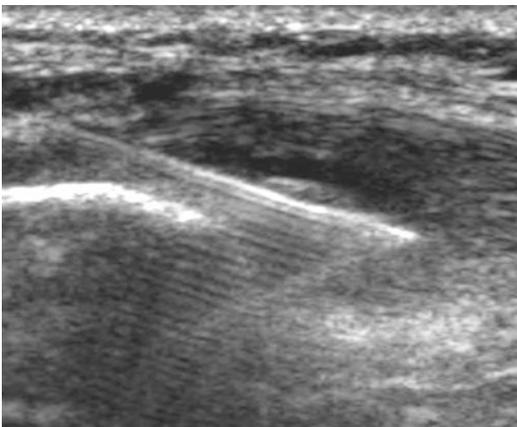
Tendinopathies of the extensor mechanism of the knee are common in both professional and ama-

teur basketball players due to the repetitive jumping and loading subjected to the patellar and quadriceps tendons. The most common tendinopathy location of the extensor mechanism is the patellar tendon origin/proximal insertion (65–70% of the cases), followed by the quadriceps tendon insertion at the superior pole of the patella (20–25%), and the patellar tendon insertion on the tibial tuberosity (5–10%) [74]. Both patellar and quadriceps tendinopathies have been historically called “Jumper’s knee” due to the high prevalence seen in athletes involved in jumping sports. Several classifications exist with regard to the severity of “jumper’s knee” and are based on pain and sports performance [75, 76] and pain intensity [77] (Table 45.1). It has been reported that this injury is prevalent in up to 30% of basketball players. Lian et al. described an overall prevalence of “jumper’s knee” of 14.2% in athletes in their report of 613 athletes with the highest prevalence reported in sports associated with high-impact ballistic/explosive loading of the knee extensor mechanism such as volleyball (44.6%) and basketball (31.9%) [78]. Zwerver et al. described a “jumper’s knee” prevalence of 11.8% in non-elite basketball players in their report of 891 athletes [79].

Several clinical studies have supported the use of PRP injections for the management of patellar tendinopathy (Fig. 45.3). Dragoo et al. performed a randomized controlled trial comparing LR-PRP to dry needling for the management of patellar tendinopathy in 23 patients. At 12 weeks follow-up, the LR-PRP group improved significantly more than the dry needling group in regard to the Victorian Institute of Sports Assessment (VISA) score for patellar tendon (25.4 vs. 5.2 points, respectively, $p = 0.02$). At 26 weeks follow-up, both groups demonstrated a significant improvement, but there was no significant difference between the cohorts ($p = 0.66$). Of note, three patients crossed over from the dry needling group to the LR-PRP group and were excluded from the final >26 weeks analysis. Additionally, there was a between-group statistically significant age difference ($p = 0.04$); while patients in the dry needling group had a mean age of 40, patients in the LR-PRP had a mean age of 28 [80]. Vetrano et al.

Table 45.1 The different existing classifications for “Jumper’s knee”

Stage/ grade	Blazina classification [75]	Ferretti classification [77]	Roels classification [76]
0		No pain	
I	Pain only following activity without functional impairment	Pain only following intense sports activity with no functional impairment	Pain at the infrapatellar or suprapatellar region following training or event
II	Pain during and following activity with satisfactory performance levels	Moderate pain during sports activity with no sports performance restriction	Pain at the beginning of activity, disappearing after warm-up and reappearing after activity completion
III	More prolonged pain during and following activity with progressively increasing difficulty in performing at a satisfactory level	Pain with slight sports performance restriction	Pain during and after activity. The patient is unable to participate in sports
IV		Pain with severe sports performance restriction	Complete rupture of the tendon
V		Pain during daily activity and inability to participate in sport at any level	

**Fig. 45.3** Ultrasound-guided PRP injection for the management of patellar tendinopathy

performed a randomized controlled trial, enrolling 46 consecutive athletes with jumper’s knee, and comparing between two ultrasound-guided injections of PRP (performed within 2 weeks) and three sessions of extracorporeal shock wave therapy (ESWT). Both groups significantly improved in symptoms at all follow-up assessments. At 2-months there were no significant differences between the groups in the VISA-patella, VAS, or Blazina scale scores, but, in 6- and 12-month follow-up, the PRP group showed a significantly greater improvement in all scores

($P < 0.05$ for all) [81]. Along with these randomized controlled studies, several other studies with a lower evidence level have also supported the use of PRP for the treatment of patellar tendinopathy [82–89].

However not all studies have found PRP to be beneficial in patellar tendinopathy; a recent level-I study by Scott et al. compared LR-PRP, LP-PRP, and normal saline injection in athletes with patellar tendinopathy for >6 months. They reported no significant differences in VISA-P, pain, or global rating of change among the three treatment groups at all time points [24]. Notably, this study did not indicate that PRP is ineffective, but rather it was no more effective than saline which in and of itself may have some therapeutic benefits.

Several preclinical and clinical studies involving the management of patellar tendinopathy using cell-based therapies. Ni et al. studied the use of tendon-derived stem cells (TDSC) in rat patellar tendon window defect model. They reported significantly higher ultimate stress and young’s modulus of elasticity in the TDSC group and concluded that the use of TDSC had promoted earlier and better tendon repair in this rat model [90]. Pascual-Garrido et al. reported on eight patients with patellar tendinopathy where BMAC was used. A 5-year follow-up revealed significantly higher Tegner score, international

knee documentation committee (IKDC) score, and symptoms and sports subscales of the knee injury and osteoarthritis outcomes score (KOOS). They also reported that most patients said that they would have the procedure again if they had the same problem in the opposite knee [91]. Clarke et al. conducted a randomized controlled trial to compare skin-derived tenocyte-like collagen-producing cells to autologous plasma for refractory patellar tendinopathy. There was a significantly greater improvement of 8.1 points in the VISA score for patellar tendon in the cell group. Of note, one patient in the cell group had a late rupture and underwent surgery [92] (Table 45.2).

Fact Box

There is evidence to support the use of PRP injections in persistent patellar tendinopathy. Although, one recent level-I study has created doubt regarding this issue. More high-quality research will shed light on the precise indications, timing, dosing, and other relevant parameters in the athlete. Preliminary evidence suggests that cell-based therapies may play a role in the management of patellar tendinopathy.

45.3.2 Achilles Tendon Pathology

45.3.2.1 Achilles Tendinopathy

Achilles tendinopathy in the athlete can present in escalating severity, from a dull pain to a debilitating injury precluding play. Many conservative treatments have been introduced; however, management of Achilles tendinopathy remains a challenge in many athletes. A preclinical study by Solchaga et al. compared the effect of an intratendon delivery of recombinant human platelet-derived growth factor-BB (rhPDGF-BB), PRP, and corticosteroids in a rat Achilles tendinopathy model. Their results demonstrated increased stiffness and load-to-failure in the rhPDGF-BB when compared to the other groups [93].

Several prospective randomized controlled trials of LR-PRP injections for Achilles tendinopathies have failed to support its efficacy [94–96]. De Vos et al. randomized 54 patients, aged 18–70, with chronic Achilles tendinopathy to receive eccentric exercises with either an LR-PRP injection or a saline injection. They concluded that no greater improvement was observed in pain or activity level with the use of LR-PRP. DeJonge et al. performed a follow-up trial on the same patient population demonstrating similar results, including no difference in tendon appearance as viewed with ultrasonography at 1-year follow-up [97]. Similarly, a randomized controlled study performed by Krogh et al. found no improvement in Achilles VISA scores following a PRP injection when compared to a saline injection. They did, however, report a significant increase in tendon thickness in the PRP group [98].

Boesen et al. have also performed a randomized double-blinded prospective trial assessing the efficacy of PRP for the management of Achilles tendinopathy in 60 patients, aged 18–59. They compared the efficacy of eccentric exercises with either (1) a high-volume injection (HVI) of steroids, saline, and local anesthetics, (2) four PRP injections each 14 days apart, or (3) a placebo (a few drops of saline injected under the skin). Both the HVI and PRP group were found to be effective in reducing pain, improving activity level and reducing tendon thickness and vascularity [99]. Of note, these studies have not targeted the professional athlete population and perhaps may not be generalizable to this patient population. Also, for the elite athlete, reduced tendon thickness can potentially result in decreased strength and velocity, as well as harbor a risk for the dreadful Achilles tendon rupture.

There is limited evidence to support the use of cell-based therapy in Achilles tendinopathy. Usuelli et al. conducted a randomized controlled trial comparing PRP to adipose-derived stromal vascular fraction (SVF) for the management of Achilles tendinopathy. They reported significantly better functional and pain scores at 15 and 30 days in the SVF group ($P < 0.05$); however, no significant differences were measured between the groups in later follow-ups [100] (Table 45.3).

Table 45.2 Characteristics of level-I studies of orthobiologics in the management of patellar tendinopathy

Author	Year of publication	Athletes	Mean age	Type of intervention	Sample size		Intervention details		Number of injections	Follow-up (months)	Favorable result	Results
					Tested group	Control group	Tested group	Control group				
Vetrano et al.	2013	Yes	PRP-26.9, control-26.8	PRP	23	23	2 mL PRP	ESWT	2	12	Yes	Significantly better VISA-P, VAS, and modified Blazina scale in the PRP group
Dragoo et al.	2014	No	PRP-28, control-40 ^a	LR-PRP	10	13	3 mL bupivacaine → 6 mL LR-PRP + dry needling	3 mL bupivacaine + dry needling	1	6	Yes	Significantly better VISA-P at for 12 weeks for the PRP group, no difference in 26 weeks. Significantly better Lysholm score for the control group at 26 weeks
Scott et al.	2015	Yes	LR-PRP-32, LP-PRP-33, NS-31	LR-PRPLP-PRP	1919	19	Two groups: 1. 2 mL Lidocaine → 3.5 mL LR-PRP + rehabilitation 2. 2 mL Lidocaine → 3.5 mL LP-PRP + rehabilitation	2 mL Lidocaine → 3.5 mL NS + rehabilitation	1	12	No	No significant change in VISA-P, pain scores or global rating of change among the 3 treatment groups at all time points
Clarke et al.	2011	No	36	Skin-derived tenocyte-like cells	33	27	Skin-derived tenocyte-like cells + autologous plasma	Autologous plasma	1	6	Yes	Significantly better VISA-P scores and faster improvement in the tested group. One patient in cell group had late patellar tendon rupture

Abbreviations: *ESWT* extracorporeal shock wave therapy, *NS* normal saline, *PRP* platelet-rich plasma, *LP-PRP* leukocyte-poor PRP, *LR-PRP* leukocyte-rich PRP, *VAS* visual analog scale, *VISA-P* Victorian Institute of Sport Assessment-Patella

^aSignificant difference in age between groups ($P = 0.04$)

Table 45.3 Characteristics of level-I studies of orthobiologics in the management of Achilles pathologies

Achilles tendinopathy	DeJonge et al.	2011	No	49.7	LR-PRP	27	27	4 mL LR-PRP	4 mL NS	1	12	No significant difference in VISA-A or sonographic parameters
Achilles tendinopathy	Kearney et al.	2013	No	PRP-47.8, control-49.9	PRP	10	10	3–5 mL PRP	Eccentric loading program	1	6	No significant difference in VISA-A
Achilles tendinopathy	Krogh et al.	2016	No	PRP-46.7, NS-51.8	LR-PRP	12	12	10–15 mL Lidocaine → 6 mL LR-PRP	10–15 mL Lidocaine → 6 mL NS	1	3	No significant difference in VISA-A. Significantly increased tendon thickness in PRP group
Achilles tendinopathy	Boensen et al.	2017	No	PRP-43.1, HVI-41.9, placebo-40.9	LP-PRP	20	(1) 20 (2) 20	4 mL LP-PRP + eccentric exercise	Two groups: 1. HVI—10 mL bupivacaine +20 mg methylprednisolone + ~40 mL NS + eccentric exercise 2. A few drops of NS under skin + eccentric exercise	4	6	Yes HVI and PRP more effective in reducing pain (VAS), improving activity level (VISA-A), reducing tendon thickness and intra-tendinous vascularity. HVI may be better than PRP in the short term
Achilles tendinopathy	Uselli et al.	2017	No	SVF-47.3, PRP-46.6	Adipose derived SVF vs. LR-PRP	21	23	4 mL SVF	4 mL LR-PRP	1	6	Yes Better VAS, AOFAS Ankle-Hindfoot score and VISA-A in both groups. Significantly better early (15 and 30 days) improvement in the SVF group. No difference in later follow-up
Achilles tendon rupture	Zou et al.	2016	No	PRP-30.2, control-28.9	LR-PRP	16	20	3–4 mL LR-PRP + Achilles tendon repair	Achilles tendon repair	1	24	Yes Significantly better SF-36, Leppilahti scores and ankle ROM for the LR-PRP group

Abbreviations: AOFAS American Orthopaedic Foot and Ankle Society, HVI high-volume injection, NS normal saline, PRP platelet-rich plasma, LP-PRP leukocyte-poor PRP, LR-PRP leukocyte-rich PRP, SVF stromal vascular fraction, VAS visual analog scale, VISA-A Victorian Institute of Sport Assessment-Achilles

45.3.2.2 Achilles Tendon Rupture

Complete rupture of the Achilles tendon is a devastating injury, with only 61.1–68% of professional National Basketball Association (NBA) players returning to play at a professional level [101, 102]. Most NBA players who do return to play suffer from a decline in performance, games, and minutes played when compared to pre-injury levels [101, 102].

Several preclinical studies have supported the potential benefit of platelets, PDGF, and tendon stem cells in promoting healing of injured Achilles tendon in rats and rabbits [103–111]. However, caution should be taken when trying to translate these results to humans, as differences between the species such as Achilles tendon size and loading can dramatically affect outcome [110, 112, 113]. Moreover, other studies have not shown positive effects in a long-term follow-up [114].

Several clinical studies assessed the efficacy of adding PRP to surgical Achilles tendon repair. Schepull et al. performed a randomized, single-blinded, controlled trial of 30 patients undergoing Achilles tendon repair. Sixteen patients were injected with 10 mL of PRP to the rupture site during primary repair and 14 were not. They found no significant differences in elasticity modulus or heel raise index. They did, however, report significant lower Achilles Tendon Total Rupture Score in the PRP group, suggesting a detrimental effect associated with the use of PRP ($P = 0.014$) [115]. De Carli et al. performed Achilles tendon repair using mini-open technique in 30 patients and tested the addition of two injections of PRP (one during surgery and another 14 days later). They reported no difference in structural and functional outcomes [116].

Sanchez et al. published a small case–control study of 12 athletes, in which 6 athletes were treated with preparation rich in growth factors (PRGF) during primary Achilles tendon repair. They reported earlier recovery of range of motion (ROM) and return to training activity [117]. Zou et al. performed a prospective study ($n = 36$) using LR-PRP as biologic augmentation to Achilles tendon repair with a 2-year follow-up. They reported improved functional outcomes

(ankle ROM, Leppilahti score, and the SF-36 score) in the PRP group in both short- and mid-term follow-ups [118].

Literature reporting on biologic augmentation using cell-based therapy to Achilles tendon repair is scarce. Stein et al. reviewed retrospectively 27 patients (28 tendons) treated with open Achilles repair augmented with BMAC. Ten patients were injured while playing basketball. Mean follow-up was 29.7 months. Twenty-five (92%) patients returned to their sporting activity at an average of 5.9 months postoperatively. Mean Achilles Tendon Rupture Score was 91 [119].

Fact Box

Current best evidence does not support the use of PRP or cell-based therapies for the management of chronic Achilles tendinopathy and for augmentation during primary suture repair of Achilles tendon rupture.

45.3.3 Plantar Fasciitis

The plantar fascia is prone to injury in basketball players due to the ballistic nature of motion required during the game, while jumping, running, cutting, and changing pace [120–123]. More specifically, Pau et al. demonstrated a significant increase in plantar peak pressure in women basketball players while attempting three-point shots and lay-ups [124]. Injuries can present either as a result of an acute injury or a more gradual presentation of chronic symptoms accompanied by acute exacerbations. The first line of treatment for plantar fasciitis in athletes is conservative management with rest, plantar fascia-specific stretching, NSAIDs, foot orthosis, and shock wave therapy [125]. When more conservative measures fail and when early return to play is sought, several local injections can be offered. Several injectables have been studied including Botulinum Toxin Type A (BTX-A) [126], corticosteroids [126], platelet-rich plasma (PRP) [127, 128], and amniotic-derived stem cells [129–132].

45.3.3.1 Corticosteroids

Corticosteroids are still commonly used for plantar fasciitis with 89% of the American Orthopaedic Foot and Ankle Society (AOFAS) surgeons performing an average of 13.9 injections per year [133]. Studies suggest short-term pain relief up to 3 months [134, 135], and better results with lower recurrence rates when ultrasound guidance is utilized [136]. However, high rates of plantar fascia rupture (53.7%) and fat pad atrophy have been reported following corticosteroids injections [9, 137–140]. In an effort to achieve better outcomes and mitigate the concerns regarding the adverse effects of corticosteroids, several injectables have been compared to corticosteroids.

45.3.3.2 Botulinum Toxin Type A (BTX-A)

Elizondo-Rodriguez et al. performed a randomized, double-blinded study, comparing BTX-A injection to the gastrocnemius muscle and a dexamethasone isonicotinate injection to the plantar fascia in 36 patients with plantar fasciitis. They found that the BTX-A group had a more rapid and sustained improvement in the functional scores measured [126].

45.3.3.3 PRP

Several level-I and level-II studies have compared the efficacy of PRP or corticosteroids injection for the treatment of plantar fasciitis [127, 141–153]. Most studies reported favorable outcomes of PRP over corticosteroids [127, 143, 145, 146, 149–153]. This statement was supported by two recent meta-analyses published by Singh et al. and Yang et al. [154, 155] Singh et al. performed a meta-analysis of 10 studies comparing PRP and corticosteroids for plantar fasciopathy. They concluded that PRP injections were associated with improved pain and functional scores at 3-months follow-up ($p = 0.04$ and $p = 0.03$, respectively), but there were no differences at 1-, 6-, and 12 months follow-ups [154]. Yang et al. included nine randomized controlled studies in their meta-analysis. They found greater long-term (24 weeks) pain relief with PRP injections when compared to corticosteroids ($p = 0.03$). However, there was no difference in

pain relief in short (4 weeks) and intermediate (12 weeks) follow-up ($p = 0.51$ and $p = 0.44$, respectively), and also no difference in functional outcomes was observed ($p > 0.05$) [155]. The efficacy of a PRP injection for chronic plantar fasciitis was also compared to a platelet-poor plasma (PPP) injection in a study performed by Malahias et al. They reported significant improvement in pain relief and functional outcomes at 3- and 6-months follow-up in both groups. There was no significant difference between the two treatment modalities [156]. Due to the small sample sizes, relatively short follow-up periods, and the lack of data regarding adverse effects in most of these studies, large-scale high-quality studies are warranted.

45.3.3.4 Amniotic-Derived Products

A few studies investigated the role of amniotic-derived products for the treatment of plantar fasciitis. Cazzell et al. conducted a multicenter single-blinded, randomized, controlled trial to evaluate the safety and efficacy of micronized dehydrated human amniotic/chorionic membrane (mDHACM) for the treatment of plantar fasciitis. Fourteen sites enrolled 145 patients to receive one injection of either mDHACM or saline. At 3-months follow-up, there was a significantly greater decrease in VAS scores in the mDHACM group (76%) compared to the control group (45%) ($p < 0.0001$). Greater reduction in the Foot Function Index—Revised (FFI-R) scores was also observed in the mDHACM group ($p = 0.0004$). There were four serious adverse events that were determined as unrelated to the study procedures. Two patients in the treatment group complained of postinjection pain and one patient reported postinjection itching [132]. Zelen et al. published a randomized, controlled, single-blinded study reporting on 45 patients with chronic plantar fasciitis. Patients were randomized to receive 2 milliliters (mL) of 0.5% marcaine with either saline, 0.5 mL mDHACM, or 1.25 mL mDHACM. At 1-week follow-up, increase in AOFAS hindfoot scores was 2.2 for the control group and 38.7 and 33.7 for the 0.5 mL mDHACM and 1.25 mL mDHACM, respectively. At 8-weeks follow-up,

AOFAS scores were 12.9, 51.6, and 53.3, respectively. The mDHACM groups showed a significantly greater increase when compared to the control ($p < 0.001$ for all), but there was no dose-related difference between the two mDHACM groups [131]. Hanselman et al. performed a randomized, controlled, double-blinded trial comparing one or two injections of cryopreserved human micronized amniotic membrane (c-hAM) versus corticosteroids for patients with plantar fasciitis. Nine patients were randomized to receive c-hAM, and 14 patients were randomized to receive a corticosteroid injection. Patients were offered to undergo a second injection at 6-weeks follow-up and three patients from each group elected to do so. While the majority of outcome measures did not demonstrate a significant difference between the groups of patients who received one injection, patients receiving two injections of c-hAM had a greater improvement in Foot Health Status Questionnaire (FHSQ) pain score at 18 weeks ($p = 0.0113$) [129]. Werber performed a prospective, open-label case series using a cryopreserved micronized amniotic membrane and amniotic fluid product (PalinGen SportFLOW) in 44 patients with plantar fasciitis and/or Achilles tendinopathy. By the fourth-week postinjection, there was a significant decrease in the visual analog scale (VAS) ($p < 0.001$). Pain relief was sustained for the remainder of the study (up to 12 weeks). No adverse events were reported by any of the patients [130]. Early results of amniotic-derived injections for plantar fasciitis are encouraging. Further studies will hopefully allow determining the true role of these novel injectables (Table 45.4).

45.3.4 Rotator Cuff Pathology

Several level-I studies assessed the efficacy of PRP for the management of rotator cuff tendinopathy [157–160]. Rha et al. performed a double-blinded, randomized, controlled study comparing two injections of LR-PRP to two dry needling sessions for rotator cuff; they report better Shoulder

and Disability Index (SPADI) Scores from 6 weeks to 6 months (17.7 vs. 29.5, $p < 0.05$) and reduced pain in the LR-PRP group [159]. Kesikburun et al. compared LR-PRP injection to a saline injection for rotator cuff tendinopathy with a 1-year follow-up. They have found no difference in Western Ontario Rotator Cuff Index [WORC], SPADI, or VAS of shoulder pain with the Neer test at 1-year follow-up ($p = 0.174$, $p = 0.314$ and $p = 0.904$, respectively) [157]. A recent systematic review of randomized controlled trials concluded that PRP may not be beneficial in the short term for rotator cuff disease. They did however state that this interpretation may be confounded by the lack of reporting of the cytology and characteristics of PRP [161] (Table 45.5).

Augmentation of rotator cuff repair with platelet-rich fibrin matrix (PRFM) has also been studied by several high-quality studies [162–182]. Saltzman et al. and Filardo et al. performed meta-analyses that demonstrated no improvement in clinical outcomes or retear rates with PRP augmentation [183, 184].

Hernigou et al. compared outcomes of rotator cuff repair with ($n = 45$) and without ($n = 45$) augmentation of BMAC derived from the iliac crest. At 6 months, 100% of the BMAC group demonstrated a healed rotator cuff on MRI, compared to 67% in the control group. Moreover, at 10-years follow-up, they found less additional ruptures in the BMAC group [71]. Kim et al. studied the effect of a single BMAC-PRP injection ($n = 12$) vs. rotator cuff exercise ($n = 12$) for rotator cuff tear. The BMAC-PRP group had lower VAS in 3 months ($p = 0.039$), but not in 3 weeks ($p = 0.147$). American Shoulder and Elbow Surgeons (ASES) scores increased significantly more in the BMAC-PRP group at 3 months ($p = 0.011$) [185] (Table 45.6).

Take-Home Messages

- There is sufficient evidence to recommend the use of PRP for patellar tendinopathy that is refractory to a first line of conservative treatment and that it is considered a safe treatment option. Although, recent level-I study has raised doubts regarding the benefits of PRP for patellar tendinopathy.

Table 45.4 Characteristics of level-I studies of orthobiologics in the management of plantar fasciitis

Author	Year of publication	Athletes	Mean age	Type of intervention	Sample size		Intervention details		Number of injections	Follow-up (months)	Favorable result	Results
					Tested group	Control group	Tested group	Control group				
Acosta-Olivo et al.	2016	No	44.8	PRP	14	14	Lidocaine +0.45 mL 10% Ca ²⁺ Gluconate +3 mL PRP	2 mL Lidocaine +2 mL dexamethasone	1	4	No	No difference in VAS, AOFAS, and FADI scores.
Jain et al.	2015	No	55.6	LR-PRP	30	30	2.5 mL PRP	40 mg triamcinolone + Levobupivacaine hydrochloride	1	12	Yes	At 12-months the PRP group had better VAS, AOFAS, and roles- Maudsley scores. At 3- and 6-months follow-ups there was no significant differences between the groups
Mahindra et al.	2016	No	PRP-30.7, CS-34.9, NS-35.5	PRP	25	25	2.5-3 mL PRP	2 mL methyl- prednisolone or NS	1	3	Yes	At 3-months follow-up, the PRP group had significantly higher AOFAS scores. Other comparisons between PRP and CS were not statistically significant

(continued)

Table 45.4 (continued)

Author	Year of publication	Athletes	Mean age	Type of intervention	Sample size		Intervention details		Number of injections	Follow-up (months)	Favorable result	Results
					Tested group	Control group	Tested group	Control group				
Monto et al.	2014	No	PRP-51, CS-59	LR-PRP	20	20	6 mL bupivacaine → 3 mL PRP	6 mL bupivacaine → 1 mL methyl-prednisolone	1	24	Yes	Significantly greater post-treatment AOFAS scores. PRP was more effective and durable than CS injection
Sherry et al.	2015	No	PRP-37.5, CS-38.5	LR-PRP	25	25	Mepivacaine → PRP	Mepivacaine → 1 mL triamcinolone	1	3	No	No significant difference in VAS and FHSQ at 3 months.
Tiwari et al.	2013	No	Not reported. Range 30–85	LR-PRP	30	30	5 mL PRP	1 mL Prilocaine +1 mL methyl-prednisolone	1	6	Yes	Significantly decreased VAS scores in the PRP group
Vahdatpour et al.	2016	No	PRP-45.4, CS-47.1	LR-PRP	16	16	3 mL PRP	1 mL Lidocaine +1 mL methyl-prednisolone	1	6	Yes/no	The PRP group had higher VAS scores at 1- and 3-months follow-ups, but lower scores at 6-months. Roles-Maudsley scores was lower in the PRP group at 1- and 3-months, but greater at 6 months.

Zelen et al.	2013	No	1. 51, 2. 56, NS-50	mDHACM	1. 15 2. 15	15	2 mL 0.5% Marcaine + 1. 1.25 mL mDHACM 2. 0.5 mL mDHACM	2 mL 0.5% Marcaine + 1.25 mL NS	1	2	Yes	Greater decrease in VAS and FFI-R scores in the mDHACM groups. No difference between the mDHACM groups.
Hanselman et al.	2014	No	51	c-hAM	9	14	c-hAM ± repeat injection at 6 weeks	DepoMedrol ± repeat injection at 6 weeks	1/2	4.5	Yes	No difference in most outcomes for one injection. Greater FHSQ for patients receiving 2 c-hAM injections.
Cazzel et al.	2018	No	mDHA CM-49CS -53	mDHACM	73	72	1 mL mDHACM	1 mL NS	1	3	Yes	Greater decrease in VAS and FFI-R in the mDHACM group at 3 months.

Abbreviations: AOFAS American Orthopaedic Foot and Ankle Society, c-hAM cryopreserved human amniotic membrane, CS Corticosteroids, ESWT extracorporeal shock wave therapy, FADI Foot and Ankle Disability Index, FFI-R Foot Function Index—Revised, FHSQ Foot Health Status Questionnaire, mDHACM micronized dehydrated human amnion/chorion membrane, NS normal saline, PRP platelet-rich plasma, LP-PRP leukocyte-poor PRP, LR-PRP leukocyte-rich PRP, VAS visual analog scale

Table 45.5 Characteristics of level-I studies of orthobiologics in the management of rotator cuff tendinopathy

Author	Year of publication	Athletes	Mean age	Type of intervention	Sample size		Intervention details		Number of injections	Follow-up (months)	Favorable result	Results
					Tested group	Control group	Tested group	Control group				
Rha et al.	2013	No	PRP—52.2, control—53.9	LR-PRP	20	19	<1 mL Lidocaine → 3 mL LR-PRP	<1 mL lidocaine	2	6	Yes	Better Shoulder and Disability Index Scores and reduced pain in the PRP group
Kesikburun et al.	2013	No	PRP—45.5, NS—51.4	LR-PRP	20	20	1 mL lidocaine → 5 mL LR-PRP	1 mL Lidocaine → 5 mL NS	1	12	No	No difference in WORC, SPADI, or VAS of shoulder pain with the Neer test at 1-year follow-up
Ilhanli et al.	2015	No	PRP—59.2, control—59.7	LR-PRP	30	32	6 mL LR-PRP	Physical therapy	1	12	Y/N	PRP group had significantly better DASH scores; however, the physical therapy group had significantly better ROM and VAS
Nejati et al.	2017	No	PRP—52.5, control—53.9	LR-PRP	22	20	4 mL LR-PRP	Physical therapy	2	6	No	Physical therapy group had superior functionality, ROM, and decreased pain in the first and third months

Abbreviations: *DASH* Disabilities of Arm Shoulder and Hand questionnaire, *NS* normal saline, *PRP* platelet-rich plasma, *LP-PRP* leukocyte-poor PRP, *LR-PRP* leukocyte-rich PRP, *ROM* range of motion, *SPADI* Shoulder and Disability Index, *VAS* visual analog scale, *WORC* Western Ontario Rotator Cuff Index

Table 45.6 Characteristics of level-I studies of orthobiologics in the management of rotator cuff repair

Author	Year of publication	Athletes	Mean age	Type of intervention	Sample size		Intervention details		Number of injections	Follow-up (months)	Favorable result	Results
					Tested group	Control group	Tested group	Control group				
D'Ambrosi et al.	2016	No	PRP—57.9, control—62	PRP	20	20	16 mL PRP + repair	Repair alone	1	6	Yes	PRP leads to a reduction in pain during a short-term follow-up.
Ebert et al.	2017	No	PRP—59.7, control—59.5	LP-PRP	30	30	2–4 mL LP-PRP + repair	Repair alone	2	42	Y/N	Reduced pain and maximal abduction strength at midterm. No additional benefit to tendon integrity.
Holtby et al.	2016	No	PRP—59, control—59	LP-PRP	41	41	7 mL PRP + repair	Repair alone	1	6	No	PRP had short-term effect on pain; however, no significant impact on patient-related outcomes or structural integrity
Malavolta et al.	2014	No	PRP—55.3, control—54	LR-PRP	27	27	20 mL PRP + repair	Repair alone	1	24	No	No beneficial clinical results for PRP
Jo et al.	2015	No	PRP—60, control—60.9	LP-PRP	37	37	3 mL PRP gel + repair	Repair alone	3	12	Yes	PRP group had significantly decreased re-tear rate and increased cross-sectional area of the supraspinatus, but not the speed of healing
Randelli et al.	2011	No	PRP—61.6, control—59.5	LR-PRP	26	27	6 mL PRP + repair	Repair alone	1	24	Yes	Early reduced pain with PRP. Positive effect on healing of rotator cuff at long-term follow-up
Castricini et al.	2011	No	PRP—55.2, control—55.5	PRFM	43	45	PRFM + repair	Repair alone	1	16	No	PRFM did not improve rotator cuff healing

(continued)

Table 45.6 (continued)

Author	Year of publication	Athletes	Mean age	Type of intervention	Sample size		Intervention details		Number of injections	Follow-up (months)	Favorable result	Results
					Tested group	Control group	Tested group	Control group				
Gumina et al.	2012	No	PRP—60, control—63	LR-PRFM	39	37	PRFM + repair	Repair alone	1	12	No	PRFM group had improved integrity of the rotator cuff; however, no improvement in functional outcomes
Weber et al.	2013	No	PRP—59.7, control—64.5	LP-PRFM	30	30	PRFM clot + repair	Repair alone	1	12	No	No significant improvement in perioperative morbidity, clinical outcomes, or structural integrity
Zamstein et al.	2016	No	PRP—65, control—66	LR-PRFM	17	18	PRFM + repair	Repair alone	1	12	No	No beneficial effect in clinical outcome, anatomic healing rate, mean postoperative defect size, and tendon quality

Abbreviations: *PRFM* platelet-rich fibrin matrix, *PRP* platelet-rich plasma, *LP-PRP* leukocyte-poor PRP, *LR-PRP* leukocyte-rich PRP, *PPP* platelet-poor plasma

- High-quality evidence supports the use of PRP injections for plantar fasciitis due to slightly greater efficacy compared to corticosteroids, accompanied by a presumed lower complications rate.
- Current literature is conflicting and heterogeneous regarding the use of PRP for rotator cuff pathology, Achilles tendinopathy, and for biologic augmentation of Achilles tendon repair.
- Studies reporting outcomes of cell-based therapies for the management of soft tissue injuries are limited. Early studies on amniotic-derived injectables for plantar fasciitis are promising but are not sufficient to support formal clinical recommendations at this point in time.
- In general, orthobiologics have yet to be thoroughly studied in specific soft tissue injuries in athletes in general and basketball players in particular; however many studies are on their way, and they will shed light on the future of this sprouting field.

References

1. LaPrade RF, Dragoo JL, Koh JL, Murray IR, Geeslin AG, Chu CR. AAOS research symposium updates and consensus: biologic treatment of orthopaedic injuries. *J Am Acad Orthop Surg*. 2016;24(7):e62.
2. Cole BJ, Frank RM. Orthobiologics in sports medicine: real-time applications are Here, and future developments are promising! *Clin Sports Med*. 2019;38(1):xiii–xiv.
3. Setayesh K, Villarreal A, Gottschalk A, Tokish JM, Choate WS. Treatment of muscle injuries with platelet-rich plasma: a review of the literature. *Curr Rev Musculoskelet Med*. 2018;11(4):635–42.
4. Nepple JJ, Matava MJ. Soft tissue injections in the athlete. *Sports Health*. 2009;1(5):396–404.
5. Baxter JD, Forsham PH. Tissue effects of glucocorticoids. *Am J Med*. 1972;53(5):573–89.
6. Levine WN, Bergfeld JA, Tessendorf W, Moorman CT III. Intramuscular corticosteroid injection for hamstring injuries: a 13-year experience in the National Football League. *Am J Sports Med*. 2000;28(3):297–300.
7. Stahl S, Kaufman T. The efficacy of an injection of steroids for medial epicondylitis. A prospective study of sixty elbows. *JBJS*. 1997;79(11):1648–52.
8. DaCruz D, Geeson M, Allen M, Phair I. Achilles paratendonitis: an evaluation of steroid injection. *Br J Sports Med*. 1988;22(2):64–5.
9. Nichols AW. Complications associated with the use of corticosteroids in the treatment of athletic injuries. *Clin J Sport Med*. 2005;15(5):E370.
10. Hussain ZB, Chahla J, LaPrade RF, Mandelbaum BR. Orthobiologics: today and tomorrow. In: *Cartilage restoration*. Berlin: Springer; 2018. p. 131–42.
11. Mishra A, Woodall J Jr, Vieira A. Treatment of tendon and muscle using platelet-rich plasma. *Clin Sports Med*. 2009;28(1):113–25.
12. Boswell SG, Cole BJ, Sundman EA, Karas V, Fortier LA. Platelet-rich plasma: a milieu of bioactive factors. *Arthroscopy*. 2012;28(3):429–39.
13. Foster TE, Puskas BL, Mandelbaum BR, Gerhardt MB, Rodeo SA. Platelet-rich plasma: from basic science to clinical applications. *Am J Sports Med*. 2009;37(11):2259–72.
14. LaPrade RF, Geeslin AG, Murray IR, Musahl V, Zlotnicki JP, Petrigliano F, et al. Biologic treatments for sports injuries II think tank—current concepts, future research, and barriers to advancement, part 1: biologics overview, ligament injury, tendinopathy. *Am J Sports Med*. 2016;44(12):3270–83.
15. Zhu Y, Yuan M, Meng H, Wang A, Guo Q, Wang Y, et al. Basic science and clinical application of platelet-rich plasma for cartilage defects and osteoarthritis: a review. *Osteoarthr Cartil*. 2013;21(11):1627–37.
16. Dhillon RS, Schwarz EM, Maloney MD. Platelet-rich plasma therapy—future or trend? *Arthritis Res Ther*. 2012;14(4):219.
17. Marx RE. Platelet-rich plasma (PRP): what is PRP and what is not PRP? *Implant Dent*. 2001;10(4):225–8.
18. Rughetti A, Giusti I, D’Ascenzo S, Leocata P, Carta G, Pavan A, et al. Platelet gel-released supernatant modulates the angiogenic capability of human endothelial cells. *Blood Transfus*. 2008;6(1):12.
19. Fleming BC, Proffen BL, Vavken P, Shalvoy MR, Machan JT, Murray MM. Increased platelet concentration does not improve functional graft healing in bio-enhanced ACL reconstruction. *Knee Surg Sports Traumatol Arthrosc*. 2015;23(4):1161–70.
20. LaPrade R, Goodrich L, Philipps J. Use of platelet-rich plasma immediately post-injury to improve ligament healing was not successful in an in vivo animal model. *Am J Sports Med*. 2018;46(3):702–12.
21. Fleming BC, Proffen BL, Vavken P, Shalvoy MR, Machan JT, Murray MM. Increased platelet concentration does not improve functional graft healing in bio-enhanced ACL reconstruction. *Knee Surg Sports Traumatol Arthrosc*. 2015;23(4):1161–70.
22. Weibrich G, Hansen T, Kleis W, Buch R, Hitzler W. Effect of platelet concentration in platelet-rich plasma on peri-implant bone regeneration. *Bone*. 2004;34(4):665–71.

23. Yoshida R, Cheng M, Murray MM. Increasing platelet concentration in platelet-rich plasma inhibits anterior cruciate ligament cell function in three-dimensional culture. *J Orthop Res.* 2014;32(2):291–5.
24. Scott A, LaPrade RF, Harmon KG, Filardo G, Kon E, Della Villa S, et al. Platelet-rich plasma for patellar tendinopathy: a randomized controlled trial of leukocyte-rich PRP or leukocyte-poor PRP versus saline. *Am J Sports Med.* 2019;47(7):1654–61.
25. Chahla J, Cinque ME, Piuze NS, Mannava S, Geeslin AG, Murray IR, et al. A call for standardization in platelet-rich plasma preparation protocols and composition reporting: a systematic review of the clinical orthopaedic literature. *J Bone Joint Surg Am.* 2017;99(20):1769–79.
26. Kawase T. Platelet-rich plasma and its derivatives as promising bioactive materials for regenerative medicine: basic principles and concepts underlying recent advances. *Odontology.* 2015;103(2):126–35.
27. Cassano JMK, Ross KA, Fraser EJ, Goodale MB, Fortier LA. Bone marrow concentrate and platelet-rich plasma differ in cell distribution and interleukin 1 receptor antagonist protein concentration. *Knee Surg Sports Traumatol Arthrosc.* 2016.
28. Castillo TN, Pouliot MA, Kim HJ, Dragoo JL. Comparison of growth factor and platelet concentration from commercial platelet-rich plasma separation systems. *Am J Sports Med.* 2011;39(2):266–71.
29. Amable PR, Carias RB, Teixeira MV, da Cruz PI, Correa do Amaral RJ, Granjeiro JM, et al. Platelet-rich plasma preparation for regenerative medicine: optimization and quantification of cytokines and growth factors. *Stem Cell Res Ther.* 2013;4(3):67.
30. Dhurat R, Sukesh M. Principles and methods of preparation of platelet-rich plasma: a review and Author's perspective. *J Cutan Aesthet Surg.* 2014;7(4):189–97.
31. Fontenot RL, Sink CA, Werre SR, Weinstein NM, Dahlgren LA. Simple tube centrifugation for processing platelet-rich plasma in the horse. *Can Vet J.* 2012;53(12):1266–72.
32. Dragoo JL, Braun HJ, Durham JL, Ridley BA, Odegaard JI, Luong R, et al. Comparison of the acute inflammatory response of two commercial platelet-rich plasma systems in healthy rabbit tendons. *Am J Sports Med.* 2012;40(6):1274–81.
33. McCarrel T, Fortier L. Temporal growth factor release from platelet-rich plasma, trehalose lyophilized platelets, and bone marrow aspirate and their effect on tendon and ligament gene expression. *J Orthop Res.* 2009;27(8):1033–42.
34. Sundman EA, Cole BJ, Fortier LA. Growth factor and catabolic cytokine concentrations are influenced by the cellular composition of platelet-rich plasma. *Am J Sports Med.* 2011;39(10):2135–40.
35. Riboh JC, Saltzman BM, Yanke AB, Fortier L, Cole BJ. Effect of leukocyte concentration on the efficacy of platelet-rich plasma in the treatment of knee osteoarthritis. *Am J Sports Med.* 2016;44(3):792–800.
36. Hanisch K, Wedderkopp N. Platelet-rich plasma (PRP) treatment of noninsertional Achilles tendinopathy in a two case series: no significant difference in effect between leukocyte-rich and leukocyte-poor PRP. *Orthop Res Rev.* 2019;11:55.
37. (WADA) WA-DA. List of prohibited substances and methods; 2019. <https://www.wada-ama.org/en/content/what-is-prohibited>.
38. Chahla J, Dean CS, Moatshe G, Pascual-Garrido C, Serra Cruz R, LaPrade RF. Concentrated bone marrow aspirate for the treatment of chondral injuries and osteoarthritis of the knee: a systematic review of outcomes. *Orthop J Sports Med.* 2016;4(1):2325967115625481.
39. Indrawattana N, Chen G, Tadokoro M, Shann LH, Ohgushi H, Tateishi T, et al. Growth factor combination for chondrogenic induction from human mesenchymal stem cell. *Biochem Biophys Res Commun.* 2004;320(3):914–9.
40. Schnabel LV, Mohammed HO, Miller BJ, McDermott WG, Jacobson MS, Santangelo KS, et al. Platelet rich plasma (PRP) enhances anabolic gene expression patterns in flexor digitorum superficialis tendons. *J Orthop Res.* 2007;25(2):230–40.
41. Sampson S, Bemden ABV, Aufiero D. Autologous bone marrow concentrate: review and application of a novel intra-articular orthobiologic for cartilage disease. *Phys Sportsmed.* 2013;41(3):7–18.
42. Hyer CF, Berlet GC, Bussewitz BW, Hankins T, Ziegler HL, Philbin TM. Quantitative assessment of the yield of osteoblastic connective tissue progenitors in bone marrow aspirate from the iliac crest, tibia, and calcaneus. *JBJS.* 2013;95(14):1312–6.
43. Hernigou P, Honna Y, Lachaniette CHF, Poignard A, Allain J, Chevallier N, et al. Benefits of small volume and small syringe for bone marrow aspirations of mesenchymal stem cells. *Int Orthop.* 2013;37(11):2279–87.
44. Caplan AI. Mesenchymal stem cells. *J Orthop Res.* 1991;9(5):641–50.
45. Pittenger MF, Mackay AM, Beck SC, Jaiswal RK, Douglas R, Mosca JD, et al. Multilineage potential of adult human mesenchymal stem cells. *Science.* 1999;284(5411):143–7.
46. Duygulu F, Demirel M, Atalan G, Kaymaz FF, Kocabey Y, Dulgeroglu TC. Effects of intra-articular administration of autologous bone marrow aspirate on healing of full-thickness meniscal tear: an experimental study on sheep. *Acta Orthop Traumatol Turc.* 2012;46(1):61–7.
47. Fortier LA, Potter HG, Rickey EJ, Schnabel LV, Foo LF, Chong LR, et al. Concentrated bone marrow aspirate improves full-thickness cartilage repair compared with microfracture in the equine model. *JBJS.* 2010;92(10):1927–37.
48. Gobbi A, Karnatzikos G, Sankineani SR. One-step surgery with multipotent stem cells for the treatment of large full-thickness chondral defects of the knee. *Am J Sports Med.* 2014;42(3):648–57.

49. Kim J-D, Lee GW, Jung GH, Kim CK, Kim T, Park JH, et al. Clinical outcome of autologous bone marrow aspirates concentrate (BMAC) injection in degenerative arthritis of the knee. *Eur J Orthop Surg Traumatol*. 2014;24(8):1505–11.
50. Krych AJ, Nawabi DH, Farshad-Amacker NA, Jones KJ, Maak TG, Potter HG, et al. Bone marrow concentrate improves early cartilage phase maturation of a scaffold plug in the knee: a comparative magnetic resonance imaging analysis to platelet-rich plasma and control. *Am J Sports Med*. 2016;44(1):91–8.
51. Saw K-Y, Hussin P, Loke S-C, Azam M, Chen H-C, Tay Y-G, et al. Articular cartilage regeneration with autologous marrow aspirate and hyaluronic acid: an experimental study in a goat model. *Arthroscopy*. 2009;25(12):1391–400.
52. Young HE, Steele TA, Bray RA, Hudson J, Floyd JA, Hawkins K, et al. Human reserve pluripotent mesenchymal stem cells are present in the connective tissues of skeletal muscle and dermis derived from fetal, adult, and geriatric donors. *Anat Rec*. 2001;264(1):51–62.
53. Asatrian G, Pham D, Hardy WR, James AW, Peault B. Stem cell technology for bone regeneration: current status and potential applications. *Stem Cells Cloning*. 2015;8:39.
54. Weber AE, Mlynarek RA, Cole BJ, Bedi A. Overview on small molecule biologic and gene-based treatments in sports medicine. *Oper Tech Orthop*. 2016;26(2):62–7.
55. Pipino C, Pandolfi A. Osteogenic differentiation of amniotic fluid mesenchymal stromal cells and their bone regeneration potential. *World J Stem Cells*. 2015;7(4):681.
56. Miana VV, González EAP. Adipose tissue stem cells in regenerative medicine. *Ecancermedicalscience*. 2018;12:822.
57. Minamide A, Yoshida M, Kawakami M, Yamasaki S, Kojima H, Hashizume H, et al. The use of cultured bone marrow cells in type I collagen gel and porous hydroxyapatite for posterolateral lumbar spine fusion. *Spine*. 2005;30(10):1134–8.
58. Fernandes MBC, Guimarães JAM, Casado PL, dos Santos CA, Gonçalves NN, Ambrósio CE, et al. The effect of bone allografts combined with bone marrow stromal cells on the healing of segmental bone defects in a sheep model. *BMC Vet Res*. 2014;10(1):36.
59. Wakitani S, Goto T, Pineda SJ, Young RG, Mansour JM, Caplan AI, et al. Mesenchymal cell-based repair of large, full-thickness defects of articular cartilage. *J Bone Joint Surg Am*. 1994;76(4):579–92.
60. Drago J, Carlson G, McCormick F, Khan-Farooqi H, Zhu M, Zuk PA, et al. Healing full-thickness cartilage defects using adipose-derived stem cells. *Tissue Eng*. 2007;13(7):1615–21.
61. Chang CH, Kuo TF, Lin FH, Wang JH, Hsu YM, Huang HT, et al. Tissue engineering-based cartilage repair with mesenchymal stem cells in a porcine model. *J Orthop Res*. 2011;29(12):1874–80.
62. Tay LX, Ahmad RE, Dashtdar H, Tay K, Masjuddin T, Ab-Rahim S, et al. Treatment outcomes of alginate-embedded allogenic mesenchymal stem cells versus autologous chondrocytes for the repair of focal articular cartilage defects in a rabbit model. *Am J Sports Med*. 2012;40(1):83–90.
63. Sato M, Uchida K, Nakajima H, Miyazaki T, Guerrero AR, Watanabe S, et al. Direct transplantation of mesenchymal stem cells into the knee joints of Hartley strain Guinea pigs with spontaneous osteoarthritis. *Arthritis Res Ther*. 2012;14(1):R31.
64. Gulotta LV, Kovacevic D, Packer JD, Deng XH, Rodeo SA. Bone marrow-derived mesenchymal stem cells transduced with scleraxis improve rotator cuff healing in a rat model. *Am J Sports Med*. 2011;39(6):1282–9.
65. Yokoya S, Mochizuki Y, Natsu K, Omae H, Nagata Y, Ochi M. Rotator cuff regeneration using a bio-absorbable material with bone marrow-derived mesenchymal stem cells in a rabbit model. *Am J Sports Med*. 2012;40(6):1259–68.
66. Gulotta LV, Kovacevic D, Ehteshami JR, Dagher E, Packer JD, Rodeo SA. Application of bone marrow-derived mesenchymal stem cells in a rotator cuff repair model. *Am J Sports Med*. 2009;37(11):2126–33.
67. Gulotta LV, Kovacevic D, Montgomery S, Ehteshami JR, Packer JD, Rodeo SA. Stem cells genetically modified with the developmental gene MT1-MMP improve regeneration of the supraspinatus tendon-to-bone insertion site. *Am J Sports Med*. 2010;38(7):1429–37.
68. Chen C-H, Chang C-H, Wang K-C, Su C-I, Liu H-T, Yu C-M, et al. Enhancement of rotator cuff tendon-bone healing with injectable periosteum progenitor cells-BMP-2 hydrogel in vivo. *Knee Surg Sports Traumatol Arthrosc*. 2011;19(9):1597–607.
69. Ma R, Schär M, Chen T, Wang H, Wada S, Ju X, et al. Use of human placenta-derived cells in a preclinical model of tendon injury. *JBJS*. 2019;101(13):e61.
70. Murray IR, Chahla J, Safran MR, Krych AJ, Saris DB, Caplan AI, et al. International expert consensus on a cell therapy communication tool: DOSES. *JBJS*. 2019;101(10):904–11.
71. Hermigou P, Lachaniette CHF, Delambre J, Zilber S, Dufflet P, Chevallier N, et al. Biologic augmentation of rotator cuff repair with mesenchymal stem cells during arthroscopy improves healing and prevents further tears: a case-controlled study. *Int Orthop*. 2014;38(9):1811–8.
72. Ho JO, Sawadkar P, Mudera V. A review on the use of cell therapy in the treatment of tendon disease and injuries. *J Tiss Eng*. 2014;5:2041731414549678.
73. Piuze NS, Dominici M, Long M, Pascual-Garrido C, Rodeo S, Huard J, et al. Proceedings of the signature series symposium “cellular therapies for orthopaedics and musculoskeletal disease proven and unproven therapies—promise, facts and fantasy,” international society for cellular therapies,

- Montreal, Canada, may 2, 2018. *Cytherapy*. 2018;20(11):1381–400.
74. King D, Yakubek G, Chughtai M, Khlopa A, Saluan P, Mont MA, et al. Quadriceps tendinopathy: a review, part 2—classification, prognosis, and treatment. *Ann Transl Med*. 2019;7(4).
 75. Blazina M. Jumper's knee. *Orthop Clin North Am*. 1973;4:665–78.
 76. Roels J, Martens M, Mulier J, Burssens A. Patellar tendinitis (jumper's knee). *Am J Sports Med*. 1978;6(6):362–8.
 77. Ferretti A, Conteduca F, Camerucci E, Morelli F. Patellar tendinosis: a follow-up study of surgical treatment. *JBJS*. 2002;84(12):2179–85.
 78. Lian ØB, Engebretsen L, Bahr R. Prevalence of jumper's knee among elite athletes from different sports: a cross-sectional study. *Am J Sports Med*. 2005;33(4):561–7.
 79. Zwerver J, Bredeweg SW, van den Akker-Scheek I. Prevalence of Jumper's knee among nonelite athletes from different sports: a cross-sectional survey. *Am J Sports Med*. 2011;39(9):1984–8.
 80. Drago JL, Wasterlain AS, Braun HJ, Nead KT. Platelet-rich plasma as a treatment for patellar tendinopathy: a double-blind, randomized controlled trial. *Am J Sports Med*. 2014;42(3):610–8.
 81. Vetrano M, Castorina A, Vulpiani MC, Baldini R, Pavan A, Ferretti A. Platelet-rich plasma versus focused shock waves in the treatment of jumper's knee in athletes. *Am J Sports Med*. 2013;41(4):795–803.
 82. Kon E, Filardo G, Delcogliano M, Presti ML, Russo A, Bondi A, et al. Platelet-rich plasma: new clinical application: a pilot study for treatment of jumper's knee. *Injury*. 2009;40(6):598–603.
 83. Filardo G, Kon E, Della Villa S, Vincentelli F, Fornasari PM, Marcacci M. Use of platelet-rich plasma for the treatment of refractory jumper's knee. *Int Orthop*. 2010;34(6):909–15.
 84. Ferrero G, Fabbro E, Orlandi D, Martini C, Lacelli F, Serafini G, et al. Ultrasound-guided injection of platelet-rich plasma in chronic Achilles and patellar tendinopathy. *J Ultrasound*. 2012;15(4):260–6.
 85. Gosens T, Den Oudsten BL, Fievez E, van't Spijker P, Fievez A. Pain and activity levels before and after platelet-rich plasma injection treatment of patellar tendinopathy: a prospective cohort study and the influence of previous treatments. *Int Orthop*. 2012;36(9):1941–6.
 86. van Ark M, Zwerver J, van den Akker-Scheek I. Injection treatments for patellar tendinopathy. *Br J Sports Med*. 2011;45(13):1068–76.
 87. Volpi P, Quaglia A, Schoenhuber H, Melegati G, Corsi M, Banfi G. Growth factors in the management of sport-induced tendinopathies: results after 24 months from treatment. A pilot study. *J Sports Med Phys Fitness*. 2010;50(4):494–500.
 88. Volpi P, Marinoni L, Bait C, De Girolamo L, Schoenhuber H. Treatment of chronic patellar tendinosis with buffered platelet rich plasma: a preliminary study. 2007.
 89. Charousset C, Zaoui A, Bellaiche L, Bouyer B. Are multiple platelet-rich plasma injections useful for treatment of chronic patellar tendinopathy in athletes? A prospective study. *Am J Sports Med*. 2014;42(4):906–11.
 90. Ni M, Lui P, Rui Y, Lee YW, Lee YW, Tan Q, et al. Tendon-derived stem cells (TDSCs) promote tendon repair in a rat patellar tendon window defect model. *J Orthop Res*. 2012;30(4):613–9.
 91. Pascual-Garrido C, Rolón A, Makino A. Treatment of chronic patellar tendinopathy with autologous bone marrow stem cells: a 5-year-followup. *Stem Cells Int*. 2012;2012.
 92. Clarke AW, Alyas F, Morris T, Robertson CJ, Bell J, Connell DA. Skin-derived tenocyte-like cells for the treatment of patellar tendinopathy. *Am J Sports Med*. 2011;39(3):614–23.
 93. Solchaga LA, Bendele A, Shah V, Snel LB, Kestler HK, Dines JS, et al. Comparison of the effect of intra-tendon applications of recombinant human platelet-derived growth factor-BB, platelet-rich plasma, steroids in a rat achilles tendon collagenase model. *J Orthop Res*. 2014;32(1):145–50.
 94. Kearney R, Parsons N, Costa M. Achilles tendinopathy management: a pilot randomised controlled trial comparing platelet-rich plasma injection with an eccentric loading programme. *Bone Joint Res*. 2013;2(10):227–32.
 95. Kearney RS, Parsons N, Metcalfe D, Costa ML. Injection therapies for Achilles tendinopathy. *Cochrane Database Syst Rev*. 2015(5).
 96. Lin M-T, Chiang C-F, Wu C-H, Hsu H-H, Tu Y-K. Meta-analysis comparing autologous blood-derived products (including platelet-rich plasma) injection versus placebo in patients with Achilles tendinopathy. *Arthroscopy*. 2018;34(6):1966–75.e5.
 97. de Jonge S, de Vos RJ, Weir A, van Schie HT, Bierma-Zeinstra SM, Verhaar JA, et al. One-year follow-up of platelet-rich plasma treatment in chronic Achilles tendinopathy: a double-blind randomized placebo-controlled trial. *Am J Sports Med*. 2011;39(8):1623–30.
 98. Krogh TP, Ellingsen T, Christensen R, Jensen P, Fredberg U. Ultrasound-guided injection therapy of Achilles tendinopathy with platelet-rich plasma or saline: a randomized, blinded, placebo-controlled trial. *Am J Sports Med*. 2016;44(8):1990–7.
 99. Boesen AP, Hansen R, Boesen MI, Malliaras P, Langberg H. Effect of high-volume injection, platelet-rich plasma, and sham treatment in chronic midportion Achilles tendinopathy: a randomized double-blinded prospective study. *Am J Sports Med*. 2017;45(9):2034–43.
 100. Usulli FG, Grassi M, Maccario C, Lanfranchi L, Montrasio UA, de Girolamo L. Intratendinous adipose-derived stromal vascular fraction (SVF) injection provides a safe, efficacious treatment for Achilles tendinopathy: results of a randomized con-

- trolled clinical trial at a 6-month follow-up. *Knee Surg Sports Traumatol Arthrosc.* 2018;26(7):2000–10.
101. Trofa DP, Miller JC, Jang ES, Woode DR, Greisberg JK, Vosseller JT. Professional athletes' return to play and performance after operative repair of an Achilles tendon rupture. *Am J Sports Med.* 2017;45(12):2864–71.
 102. Amin NH, Old AB, Tabb LP, Garg R, Toossi N, Cerynik DL. Performance outcomes after repair of complete Achilles tendon ruptures in National Basketball Association players. *Am J Sports Med.* 2013;41(8):1864–8.
 103. Allahverdi A, Sharifi D, Takhtfooladi MA, Hesaraki S, Khansari M, Dorbeh SS. Evaluation of low-level laser therapy, platelet-rich plasma, and their combination on the healing of Achilles tendon in rabbits. *Lasers Med Sci.* 2015;30(4):1305–13.
 104. Chen L, Dong SW, Liu JP, Tao X, Tang KL, Xu JZ. Synergy of tendon stem cells and platelet-rich plasma in tendon healing. *J Orthop Res.* 2012;30(6):991–7.
 105. Cummings SH, Grande DA, Hee CK, Kestler HK, Roden CM, Shah NV, et al. Effect of recombinant human platelet-derived growth factor-BB-coated sutures on Achilles tendon healing in a rat model: a histological and biomechanical study. *J Tiss Eng.* 2012;3(1):2041731412453577.
 106. Kaux JF, Drion PV, Colige A, Pascon F, Libertiaux V, Hoffmann A, et al. Effects of platelet-rich plasma (PRP) on the healing of a chilles tendons of rats. *Wound Repair Regen.* 2012;20(5):748–56.
 107. Kim HJ, Nam H-W, Hur C-Y, Park M, Yang HS, Kim B-S, et al. The effect of platelet rich plasma from bone marrow aspirate with added bone morphogenetic protein-2 on the Achilles tendon-bone junction in rabbits. *Clin Orthop Surg.* 2011;3(4):325–31.
 108. Lyras DN, Kazakos K, Georgiadis G, Mazis G, Middleton R, Richards S, et al. Does a single application of PRP alter the expression of IGF-I in the early phase of tendon healing? *J Foot Ankle Surg.* 2011;50(3):276–82.
 109. Lyras DN, Kazakos K, Tryfonidis M, Agrogiannis G, Botaitis S, Kokka A, et al. Temporal and spatial expression of TGF- β 1 in an Achilles tendon section model after application of platelet-rich plasma. *Foot Ankle Surg.* 2010;16(3):137–41.
 110. Virchenko O, Aspenberg P. How can one platelet injection after tendon injury lead to a stronger tendon after 4 weeks?: interplay between early regeneration and mechanical stimulation. *Acta Orthop.* 2006;77(5):806–12.
 111. Yuksel S, Guleç MA, Gultekin MZ, Adanır O, Caglar A, Beytemur O, et al. Comparison of the early period effects of bone marrow-derived mesenchymal stem cells and platelet-rich plasma on the Achilles tendon ruptures in rats. *Connect Tissue Res.* 2016;57(5):360–73.
 112. Aspenberg P. Platelet concentrates and Achilles tendon healing. *J Orthop Res.* 2013;31(9):1500.
 113. Andersson T, Eliasson P, Hammerman M, Sandberg O, Aspenberg P. Low-level mechanical stimulation is sufficient to improve tendon healing in rats. *J Appl Physiol.* 2012;113(9):1398–402.
 114. Kraus T, Imhoff F, Reinert J, Wexel G, Wolf A, Hirsch D, et al. Stem cells and bFGF in tendon healing: effects of lentiviral gene transfer and long-term follow-up in a rat Achilles tendon defect model. *BMC Musculoskelet Disord.* 2016;17(1):148.
 115. Schepull T, Kvist J, Norrman H, Trinks M, Berlin G, Aspenberg P. Autologous platelets have no effect on the healing of human achilles tendon ruptures: a randomized single-blind study. *Am J Sports Med.* 2011;39(1):38–47.
 116. De Carli A, Lanzetti RM, Ciompi A, Lupariello D, Vadalà A, Argento G, et al. Can platelet-rich plasma have a role in Achilles tendon surgical repair? *Knee Surg Sports Traumatol Arthrosc.* 2016;24(7):2231–7.
 117. Sánchez M, Anitua E, Azofra J, Andía I, Padilla S, Mujika I. Comparison of surgically repaired Achilles tendon tears using platelet-rich fibrin matrices. *Am J Sports Med.* 2007;35(2):245–51.
 118. Zou J, Mo X, Shi Z, Li T, Xue J, Mei G, et al. A prospective study of platelet-rich plasma as biological augmentation for acute Achilles tendon rupture repair. *Biomed Res Int.* 2016;2016.
 119. Stein BE, Stroh DA, Schon LC. Outcomes of acute Achilles tendon rupture repair with bone marrow aspirate concentrate augmentation. *Int Orthop.* 2015;39(5):901–5.
 120. Losito JM. Basketball and volleyball. In: *Athletic footwear and Orthoses in sports medicine.* Berlin: Springer; 2017. p. 381–7.
 121. Daffé M, Kivandat D, Dembélé B. Epidemiological study of Technopaties in amateur basketball players. *Orthop Traumatol Sports Med Int J.* 2018;1:17–20.
 122. Sobhani S, Dekker R, Postema K, Dijkstra PU. Epidemiology of ankle and foot overuse injuries in sports: a systematic review. *Scand J Med Sci Sports.* 2013;23(6):669–86.
 123. Chen Y, Li JX, Hong Y, Wang L. Plantar stress-related injuries in male basketball players: variations on plantar loads during different maximum-effort maneuvers. *Biomed Res Int.* 2018;2018.
 124. Pau M, Ciuti C. Stresses in the plantar region for long-and short-range throws in women basketball players. *Eur J Sport Sci.* 2013;13(5):575–81.
 125. Petraglia F, Ramazzina I, Costantino C. Plantar fasciitis in athletes: diagnostic and treatment strategies. A systematic review. *Muscles Ligaments Tendons J.* 2017;7(1):107.
 126. Elizondo-Rodriguez J, Araujo-Lopez Y, Moreno-Gonzalez JA, Cardenas-Estrada E, Mendoza-Lemus O, Acosta-Olivo C. A comparison of botulinum toxin a and intralesional steroids for the treatment of plantar fasciitis: a randomized, double-blinded study. *Foot Ankle Int.* 2013;34(1):8–14.
 127. Monto RR. Platelet-rich plasma efficacy versus corticosteroid injection treatment for chronic severe plantar fasciitis. *Foot Ankle Int.* 2014;35(4):313–8.

128. Le AD, Enweze L, DeBaun MR, Dragoo JL. Platelet-rich plasma. *Clin Sports Med.* 2019;38(1):17–44.
129. Hanselman AE, Tidwell JE, Santrock RD. Cryopreserved human amniotic membrane injection for plantar fasciitis: a randomized, controlled, double-blind pilot study. *Foot Ankle Int.* 2015;36(2):151–8.
130. Werber B. Amniotic tissues for the treatment of chronic plantar fasciosis and Achilles tendinosis. *J Sports Med.* 2015;2015.
131. Zelen CM, Poka A, Andrews J. Prospective, randomized, blinded, comparative study of injectable micronized dehydrated amniotic/chorionic membrane allograft for plantar fasciitis—a feasibility study. *Foot Ankle Int.* 2013;34(10):1332–9.
132. Cazzell S, Stewart J, Agnew PS, Senatore J, Walters J, Murdoch D, et al. Randomized controlled trial of micronized dehydrated human amnion/chorion membrane (dHACM) injection compared to placebo for the treatment of plantar fasciitis. *Foot Ankle Int.* 2018;39(10):1151–61.
133. Johnson JE, Klein SE, Putnam RM. Corticosteroid injections in the treatment of foot & ankle disorders: an AOFAS survey. *Foot Ankle Int.* 2011;32(4):394–9.
134. de Cesar NC, da Fonseca LF, Nascimento FS, O'Daley AE, Tan EW, Dein EJ, et al. ☆ diagnostic and therapeutic injections of the foot and ankle—an overview. *Foot Ankle Surg.* 2018;24(2):99–106.
135. Schulhofer SD. Short-term benefits of ultrasound-guided corticosteroid injection in plantar fasciitis. *Clin J Sport Med.* 2013;23(1):83–4.
136. Li Z, Xia C, Yu A, Qi B. Ultrasound-versus palpation-guided injection of corticosteroid for plantar fasciitis: a meta-analysis. *PLoS One.* 2014;9(3):e92671.
137. Acevedo JJ, Beskin JL. Complications of plantar fascia rupture associated with corticosteroid injection. *Foot Ankle Int.* 1998;19(2):91–7.
138. Leach R, Jones R, Silva T. Rupture of the plantar fascia in athletes. *JBJS.* 1978;60(4):537–9.
139. Ahstrom JRJP. Spontaneous rupture of the plantar fascia. *Am J Sports Med.* 1988;16(3):306–7.
140. Neufeld SK, Cerrato R. Plantar fasciitis: evaluation and treatment. *JAAOS.* 2008;16(6):338–46.
141. Acosta-Olivo C, Elizondo-Rodríguez J, Lopez-Cavazos R, Vilchez-Cavazos F, Simental-Mendia M, Mendoza-Lemus O. Plantar fasciitis—a comparison of treatment with intralesional steroids versus platelet-rich plasma: a randomized, blinded study. *J Am Podiatr Med Assoc.* 2017;107(6):490–6.
142. Jain K, Murphy PN, Clough TM. Platelet rich plasma versus corticosteroid injection for plantar fasciitis: a comparative study. *Foot.* 2015;25(4):235–7.
143. Mahindra P, Yamin M, Selhi HS, Singla S, Soni A. Chronic plantar fasciitis: effect of platelet-rich plasma, corticosteroid, and placebo. *Orthopedics.* 2016;39(2):e285–e9.
144. Sherpy NA, Hammad MA, Hagrass HA, Samir H, Abu-ElMaaty SE, Mortada MA. Local injection of autologous platelet rich plasma compared to corticosteroid treatment of chronic plantar fasciitis patients: a clinical and ultrasonographic follow-up study. *Egypt Rheumatol.* 2016;38(3):247–52.
145. Tiwari M, Bhargava R. Platelet rich plasma therapy: a comparative effective therapy with promising results in plantar fasciitis. *J Clin Orthop Trauma.* 2013;4(1):31–5.
146. Vahdatpour B, Kianimehr L, Moradi A, Haghghat S. Beneficial effects of platelet-rich plasma on improvement of pain severity and physical disability in patients with plantar fasciitis: a randomized trial. *Adv Biomed Res.* 2016;5.
147. Akşahin E, Doğruyol D, Yüksel HY, Hapa O, Doğan Ö, Çelebi L, et al. The comparison of the effect of corticosteroids and platelet-rich plasma (PRP) for the treatment of plantar fasciitis. *Arch Orthop Trauma Surg.* 2012;132(6):781–5.
148. Jain SK, Suprashant K, Kumar S, Yadav A, Kearns SR. Comparison of plantar fasciitis injected with platelet-rich plasma vs corticosteroids. *Foot Ankle Int.* 2018;39(7):780–6.
149. Omar AS, Ibrahim ME, Ahmed AS, Said M. Local injection of autologous platelet rich plasma and corticosteroid in treatment of lateral epicondylitis and plantar fasciitis: randomized clinical trial. *Egypt Rheumatol.* 2012;34(2):43–9.
150. Shetty VD, Dhillon M, Hegde C, Jagtap P, Shetty S. A study to compare the efficacy of corticosteroid therapy with platelet-rich plasma therapy in recalcitrant plantar fasciitis: a preliminary report. *Foot Ankle Surg.* 2014;20(1):10–3.
151. Say F, Gürler D, Inkaya E, Bülbül M. Comparison of platelet-rich plasma and steroid injection in the treatment of plantar fasciitis. *Acta Orthop Traumatol Turc.* 2014;48(6):667–72.
152. Puri VP, Gaur AK. A comparative study between local injection of autologous platelet rich plasma and injection of corticosteroid in functional improvement of plantar fasciitis. *Int Surg J.* 2019;6(3):653–7.
153. Shetty SH, Dhond A, Arora M, Deore S. Platelet-rich plasma has better Long-term results than corticosteroids or placebo for chronic plantar fasciitis: randomized control trial. *J Foot Ankle Surg.* 2019;58(1):42–6.
154. Singh P, Madanipour S, Bhamra JS, Gill I. A systematic review and meta-analysis of platelet-rich plasma versus corticosteroid injections for plantar fasciopathy. *Int Orthop.* 2017;41(6):1169–81.
155. Yang W-y, Han Y-h, Cao X-w, Pan J-k, Zeng L-f, Lin J-t, et al. Platelet-rich plasma as a treatment for plantar fasciitis: a meta-analysis of randomized controlled trials. *Medicine.* 2017;96(44).
156. Malahias M-A, Mavrogenis AF, Nikolaou VS, Megaloikonomos PD, Kazas ST, Chronopoulos E, et al. Similar effect of ultrasound-guided platelet-rich plasma versus platelet-poor plasma injections for chronic plantar fasciitis. *Foot.* 2019;38:30–3.
157. Kesikburun S, Tan AK, Yılmaz B, Yaşar E, Yazıcıoğlu K. Platelet-rich plasma injections in the treatment of chronic rotator cuff tendinopathy: a randomized con-

- trolled trial with 1-year follow-up. *Am J Sports Med.* 2013;41(11):2609–16.
158. Ilhanli I, Guder N, Gul M. Platelet-rich plasma treatment with physical therapy in chronic partial supraspinatus tears. *Iran Red Crescent Med J.* 2015;17(9).
 159. D-w R, Park G-Y, Kim Y-K, Kim MT, Lee SC. Comparison of the therapeutic effects of ultrasound-guided platelet-rich plasma injection and dry needling in rotator cuff disease: a randomized controlled trial. *Clin Rehabil.* 2013;27(2):113–22.
 160. Nejati P, Ghahremaninia A, Naderi F, Gharibzadeh S, Mazaherinezhad A. Treatment of subacromial impingement syndrome: platelet-rich plasma or exercise therapy? A randomized controlled trial. *Orthop J Sports Med.* 2017;5(5):2325967117702366.
 161. Hurley ET, Hannon CP, Pauzenberger L, Fat DL, Moran CJ, Mullett H. Nonoperative treatment of rotator cuff disease with platelet-rich plasma: a systematic review of randomized controlled trials. *Arthroscopy.* 2019.
 162. Zhang Z, Wang Y, Sun J. The effect of platelet-rich plasma on arthroscopic double-row rotator cuff repair: a clinical study with 12-month follow-up. *Acta Orthop Traumatol Turc.* 2016;50(2):191–7.
 163. Flury M, Rickenbacher D, Schwyzer H-K, Jung C, Schneider MM, Stahnke K, et al. Does pure platelet-rich plasma affect postoperative clinical outcomes after arthroscopic rotator cuff repair? A randomized controlled trial. *Am J Sports Med.* 2016;44(8):2136–46.
 164. Pandey V, Bandi A, Madi S, Agarwal L, Acharya KK, Maddukuri S, et al. Does application of moderately concentrated platelet-rich plasma improve clinical and structural outcome after arthroscopic repair of medium-sized to large rotator cuff tear? A randomized controlled trial. *J Shoulder Elb Surg.* 2016;25(8):1312–22.
 165. Zumstein MA, Rumian A, Th  lu C  , Lesbats V, O'Shea K, Schaer M, et al. SECEC research Grant 2008 II: use of platelet-and leukocyte-rich fibrin (L-PRF) does not affect late rotator cuff tendon healing: a prospective randomized controlled study. *J Shoulder Elb Surg.* 2016;25(1):2–11.
 166. Carr AJ, Murphy R, Dakin SG, Rombach I, Whewey K, Watkins B, et al. Platelet-rich plasma injection with arthroscopic acromioplasty for chronic rotator cuff tendinopathy: a randomized controlled trial. *Am J Sports Med.* 2015;43(12):2891–7.
 167. Wang A, McCann P, Colliver J, Koh E, Ackland T, Joss B, et al. Do postoperative platelet-rich plasma injections accelerate early tendon healing and functional recovery after arthroscopic supraspinatus repair? A randomized controlled trial. *Am J Sports Med.* 2015;43(6):1430–7.
 168. Hak A, Rajaratnam K, Ayeni OR, Moro J, Peterson D, Sprague S, et al. A double-blinded placebo randomized controlled trial evaluating short-term efficacy of platelet-rich plasma in reducing postoperative pain after arthroscopic rotator cuff repair: a pilot study. *Sports Health.* 2015;7(1):58–66.
 169. Werthel J-D, Pelissier A, Massin P, Boyer P, Valenti P. Arthroscopic double row cuff repair with suture-bridging and autologous conditioned plasma injection: functional and structural results. *Int J Shoulder Surg.* 2014;8(4):101.
 170. Malavolta EA, Gracitelli MEC, Ferreira Neto AA, Assun  o JH, Bordalo-Rodrigues M, de Camargo OP. Platelet-rich plasma in rotator cuff repair: a prospective randomized study. *Am J Sports Med.* 2014;42(10):2446–54.
 171. Zumstein MA, Rumian A, Lesbats V, Schaer M, Boileau P. Increased vascularization during early healing after biologic augmentation in repair of chronic rotator cuff tears using autologous leukocyte-and platelet-rich fibrin (L-PRF): a prospective randomized controlled pilot trial. *J Shoulder Elb Surg.* 2014;23(1):3–12.
 172. Jo CH, Shin JS, Lee YG, Shin WH, Kim H, Lee SY, et al. Platelet-rich plasma for arthroscopic repair of large to massive rotator cuff tears: a randomized, single-blind, parallel-group trial. *Am J Sports Med.* 2013;41(10):2240–8.
 173. ANTU  a S, Barco R, Martinez Diez JM, Sanchez Marquez JM. Platelet-rich fibrin in arthroscopic repair of massive rotator cuff tears: a prospective randomized pilot clinical trial. *Acta Orthop Belg.* 2013;79(1):25–30.
 174. Castricini R, Longo UG, De Benedetto M, Panfoli N, Pirani P, Zini R, et al. Platelet-rich plasma augmentation for arthroscopic rotator cuff repair: a randomized controlled trial. *Am J Sports Med.* 2011;39(2):258–65.
 175. Randelli PS, Arrigoni P, Cabitza P, Volpi P, Maffulli N. Autologous platelet rich plasma for arthroscopic rotator cuff repair. A pilot study. *Disabil Rehabil.* 2008;30(20–22):1584–9.
 176. Rodeo SA, Delos D, Williams RJ, Adler RS, Pearle A, Warren RF. The effect of platelet-rich fibrin matrix on rotator cuff tendon healing: a prospective, randomized clinical study. *Am J Sports Med.* 2012;40(6):1234–41.
 177. Gumina S, Campagna V, Ferrazza G, Giannicola G, Fratolocchi F, Milani A, et al. Use of platelet-leukocyte membrane in arthroscopic repair of large rotator cuff tears: a prospective randomized study. *JBJS.* 2012;94(15):1345–52.
 178. Weber SC, Kauffman JI, Parise C, Weber SJ, Katz SD. Platelet-rich fibrin matrix in the management of arthroscopic repair of the rotator cuff: a prospective, randomized, double-blinded study. *Am J Sports Med.* 2013;41(2):263–70.
 179. Holtby R, Christakis M, Maman E, MacDermid JC, Dwyer T, Athwal GS, et al. Impact of platelet-rich plasma on arthroscopic repair of small-to medium-sized rotator cuff tears: a randomized controlled trial. *Orthop J Sports Med.* 2016;4(9):2325967116665595.
 180. Jo CH, Shin JS, Shin WH, Lee SY, Yoon KS, Shin S. Platelet-rich plasma for arthroscopic repair of medium to large rotator cuff tears: a randomized controlled trial. *Am J Sports Med.* 2015;43(9):2102–10.

181. Ebert JR, Wang A, Smith A, Nairn R, Breidahl W, Zheng MH, et al. A midterm evaluation of postoperative platelet-rich plasma injections on arthroscopic supraspinatus repair: a randomized controlled trial. *Am J Sports Med.* 2017;45(13):2965–74.
182. D'Ambrosi R, Palumbo F, Paronzini A, Ragone V, Facchini R. Platelet-rich plasma supplementation in arthroscopic repair of full-thickness rotator cuff tears: a randomized clinical trial. *Musculoskelet Surg.* 2016;100(1):25–32.
183. Saltzman BM, Jain A, Campbell KA, Mascarenhas R, Romeo AA, Verma NN, et al. Does the use of platelet-rich plasma at the time of surgery improve clinical outcomes in arthroscopic rotator cuff repair when compared with control cohorts? A systematic review of meta-analyses. *Arthroscopy.* 2016;32(5):906–18.
184. Filardo G, Di Matteo B, Kon E, Merli G, Marcacci M. Platelet-rich plasma in tendon-related disorders: results and indications. *Knee Surg Sports Traumatol Arthrosc.* 2018;26(7):1984–99.
185. Kim SJ, Kim EK, Kim SJ. Effects of bone marrow aspirate concentrate and platelet-rich plasma on patients with partial tear of the rotator cuff tendon. *J Orthop Surg Res.* 2018;13(1):1.



The Role of Orthobiologics for the Management of Ligament and Muscle Injuries in Sports

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46.1 Introduction

The use of orthobiologics has become increasingly common in the field of sports medicine to enhance healing of soft tissue injuries. Despite some promising outcomes reported and the surge in orthobiologic use in sports medicine, clarity is lacking with regard to the proper indications, optimal orthobiologic product for each indication, preparation method and dosage, as well as application method and protocol [1–3]. These variabilities have led to inconsistencies in both preclinical and clinical reported results. Therefore, establishing an optimal treatment protocol for the various soft tissue sports injuries still presents a challenge due to the number of independent variables and absence of high-

quality evidence [4]. When attempting to optimize orthobiologic treatment for a specific musculoskeletal injury, one has to consider additional important factors which can affect treatment outcomes, such as the local environment as well as tissue biomechanics and load vectors, which may influence optimal adherence, stability, and potency of the orthobiologic agent in the injured tissue [5].

The purpose of this chapter is to review the best current evidence and recommendations on several orthobiologic treatment approaches in the management of common ligament and muscle injuries in basketball. In addition, regulatory aspects will dictate potential utilization based upon geographic considerations (i.e., in and outside the United States).

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46.2 Ligament Injuries

Ligament injuries in basketball represent some of the most severe injuries in terms of time loss from play and are responsible for a substantial burden of injury. Some of these injuries (knee cruciate ligament injuries; ankle ligament injuries) may often require surgical management and could lead to a permanently reduced level of sports performance or even be career ending.

The clinical use of platelet-rich plasma (PRP) for soft tissue injuries is based on previous pre-clinical studies investigating its effect on tendons, muscles, and ligaments. Several basic science studies have supported the application of growth factors to increase collagen synthesis and for healing enhancement in ligament tissue [6, 7]. Early studies of PRP use for ligament tissue in animal models have shown promising healing potential for both medial collateral ligament (MCL) and anterior cruciate ligament (ACL) injuries [8–10]. These basic science studies have assisted in supporting and developing the concepts of PRP use for ligament healing in the clinical setting, as well as provide promising future directions.

46.2.1 Anterior Cruciate Ligament

Anterior cruciate ligament (ACL) tears are among the most common severe sports-related injuries and therefore ACL reconstruction surgery is one of the most frequently performed procedures in the field of sports medicine [11]. ACL surgery relies upon both technical and biologic factors to assure the highest likelihood of success. Various strategies have been attempted over the years to improve ACL reconstruction (ACLR) outcomes such as targeting the biologic healing process to improve the graft's healing capacity and incorporation hoping to shorten return to sports duration and reduce failure rates. For this purpose, bio-regenerative/orthobiologic treatment options have shown potential to improve graft incorporation and strength from gene activation level through microenvironment optimization in order to possibly delay or prevent early progression to osteoarthritis [12].

46.2.1.1 Basic Science and Preclinical Evidence

Important preclinical work in the last two decades has provided the basis to improve bio-regenerative approaches to enhance ACL graft healing. Many of the previous investigated regenerative agents contain various growth factors (GFs) which have demonstrated positive effects on various biological processes necessary for ACL healing such as cell proliferation, cell migration, angiogenesis, and extracellular matrix (ECM) production in both in vivo and in vitro studies [13]. The fibroblast, the primary cell in the ACL, has receptors for many of these GFs, such as transforming GF b-1 (TGF-b1), fibroblast GF-2 (FGF-2), platelet-derived GF (PDGF), insulin-like GF, epidermal GF, and vascular endothelial GF (VEGF). Therefore, these GFs have been the focus of many preclinical studies. For example, TGF-b1, FGF-2, and basic-FGF have been shown to have a role in the repair process of a torn ligament by regulating and improving cellular proliferation and ECM production, as well as affect mesenchymal stem cells (MSCs) differentiation into fibroblasts [6, 14, 15]. PDGF has been shown to stimulate fibroblast growth, cell migration, and a biologic cascade reducing the postoperative release of proinflammatory factors [16], therefore potentially improving graft ligamentization and incorporation potentially reducing graft failure risk [17]. TGF-b1 was reported to potentially stimulate initial and overall healing in both histologically and biomechanically tested partial ACL tears in animal models [18, 19]. Kondo et al. reported significantly improved biomechanical and histologic healing properties of injured ACLs treated with TGF-b1 in a rabbit ACL injury model when compared with controls [18]. Marui et al. reported that TGF-b1 application resulted in up to $\times 1.5$ increase in collagen synthesis compared with controls in both ACL and MCL fibroblasts [7]. VEGF-augmented grafts demonstrated improved vascularization and fibroblast infiltration compared to controls following ACL reconstruction in a sheep model although increased graft laxity was evident at 12 weeks [20]. More recently, blocking VEGF has been reported to reduce angiogenesis, graft maturation, and bio-

mechanical strength following an ACL reconstruction model in rats [21].

Murray et al. investigated the application of clotted PRP in the gap of a transected ACL in a porcine ACL repair model, reporting no beneficial effect for PRP use compared with controls. They concluded that the fibrin clot used was not sufficiently biologically stable and may have prematurely dissolved in the intra-articular environment and synovial fluid containing plasmin [22]. These observations led to the development of scaffolds to hold the PRP at the ACL injury site and protect it from the intra-articular environment and from early degradation. In a later study, the same group added PRP to a collagen hydrogel showing significantly increased cellular metabolic activity, a reduced apoptotic rate, and collagen production stimulation in cells from immature and adolescent animals although less effect was achieved in adult animal cells [23]. In a later study from the same group, Vavken et al. combined a collagen scaffold with autologous platelets, demonstrating significantly improved ACL repair outcomes in a porcine model. They reported primary repair augmented with a collagen-PRP hydrogel resulted in superior tissue mechanical properties compared with suture repair alone [24].

Several preclinical studies have investigated the use of stem cells for the management of ACL tears. A recent systematic review of the available preclinical evidence of adult stem cells as a biological augmentation in the treatment of animal anterior cruciate ligament (ACL) injury was performed by Guo et al. [25]. Thirteen animal studies were included. Six of seven studies using bone marrow-derived mesenchymal stem (stromal) cells (BMSCs) reported a positive enhancement in histology, biomechanics, and biochemistry within 12 weeks postoperatively. Four studies using ACL-derived vascular stem cells showed a promoting effect in histology, biomechanics, and imaging within 8 weeks postoperatively. Two studies focusing on animal tendon-derived stem cells (TDSCs) and human umbilical cord blood-derived mesenchymal stem cells (hUCB-MSCs) reported promotable effects for the early healing in a small animal ACL model. Authors concluded that BMSCs, ACL-derived vascular stem cells,

TDSCs, and hUCB-MSCs were shown to enhance ACL healing during the early phase in small animal models. Oe et al. compared ACL regeneration between groups subjected to intra-articular injection of either fresh whole bone marrow cells (BMCs), cultured MSCs, or saline in partial ACL tears in a rat model [26]. They concluded that administration of fresh whole BMCs is an effective treatment for partial ACL rupture and reported nearly normal ligament healing and strength compared with controls. Similar findings were reported by Kanaya et al. following intra-articular injection of MSCs showing improved ligament healing with superior histologic features and a greater load-to-failure compared with nontreated controls in a rat model [27]. More recently, in another rat model, Lui et al. added tendon-derived stem cell sheets to ACL reconstructions. The treated knees exhibited higher intra-articular graft integrity with lower cellularity, improved cell organization and vascularity, as well as better tunnel-bone mineral density, bone volume, and better graft osteointegration compared to the control group [28]. A recent study by Hur et al. reported that the use of MSCs with ACL reconstruction decreased tunnel widening in rabbit model [29]. Sun et al. investigated the effect of human bone marrow stem cells (hBMSC)-CM on ACLR in a rat model and reported that hBMSC-CM accelerated graft-bone incorporation, midsubstance ligamentization and enhanced fibroblast proliferation, differentiation, and collagen synthesis [30].

46.2.1.2 Clinical Evidence

Clinical studies on the use of orthobiologics in ACL surgery have focused on the following applications: (1) Healing enhancement in partial tears with or without repair; (2) healing enhancement in ACLR graft, focusing on osteoligamentous integration into the tibial and femoral tunnels and maturation of the articular portion of the graft, and (3) graft harvest site healing.

Partial ACL Tears

Management of partial ACL tears presents a significant challenge to clinicians as the natural history of these lesions is poorly understood and due to the limited evidence regarding treatment

options. Although it is generally accepted that spontaneous healing capacity of the ACL following an injury is limited [31, 32], there are reports on spontaneous healing of partial ACL tears [33, 34]. More recently, Nguyen et al. reported an intrinsic healing response in the proximal third of human ACLs with typical spontaneous healing characteristics similar to the MCL, in a histological study investigating spontaneously reattached tibial ACL remnants [35]. This evidence prompted attempts to enhance the ACL's healing potential with or without repair.

ACL Healing Enhancement Without Repair

Seijas et al. reported a high return to sports rate in 19 professional soccer players with a partial ACL tear treated with intra-ligamentous application of platelet-rich growth factors (PRGF-Endoret) into the intact bundle (Fig. 46.3) [36]. Administration of 4 mL of this product, described by Anitua [37], was performed during arthroscopy using a spinal needle in both the proximal origin and the middle portion of the intact bundle (Fig. 46.1). An additional injection of PRGF (6 mL) was administered in the articular space at the end of the surgery,

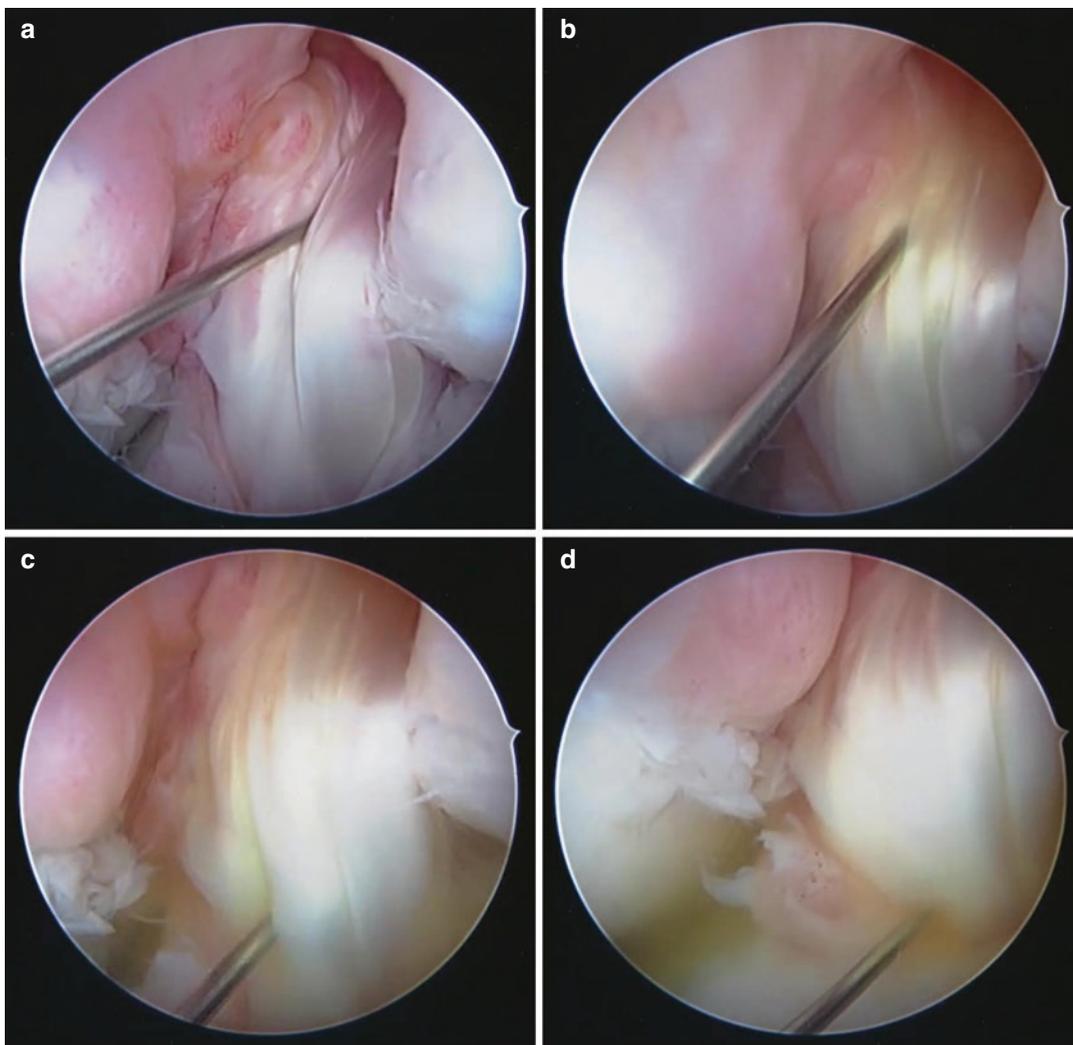


Fig. 46.1 Intraoperative PRP infiltration of a partial ACL tear with a mostly intact sheath into the proximal aspect of the tear (a). The ligament has an “inflated” appearance

following the initial infiltration (b). Mid-substance ACL infiltration (c). Distal ACL aspect infiltration (d)

when the joint had been dried. Average time between injury to surgery in this study was 5.8 weeks. Eighteen of 19 players were able to return to their previous level of play, with 15 players (Tegner level 9) returning to play at an average of 16.20 weeks (1 re-rupture at 7 months), while the 3 patients playing at a higher level (Tegner level 10) returned at an average of 12.33 weeks. One patient was not able to return to sport due to significant cartilage lesions. No notable complications were reported in any of the patients in the study. A postoperative magnetic resonance imaging (MRI) at 1 year from surgery showed complete ligamentization and good anatomic organization of the remnant in all patients. In a recent study, Koch et al. evaluated 42 patients following an intra-ligamentous autologous conditioned plasma (ACP™, Arthrex, Florida, USA) by clinical scoring and functional performance assessment at a mean 33 months follow-up [38]. Failure was recorded in 4 patients (9.5%). Good to excellent results were reported on all outcome scores. Clinical examination showed marked improvements from the preoperative status in terms of the Lachman test, pivot shift phenomenon, and a significant reduction in AP laxity (rolimeter preoperative: 1.9 (SD 1.4); postoperative 0.6 (SD 1.8), $p = 0.001$) in all patients. Functional performance testing showed no significant differences between the injured and healthy side. Return to sport was achieved at a mean of 5.8 months (SD 3.6) in 71.1% of patients with a subjective return to pre-injury sports activity in 85.8% (SD 19%). Notably, the absence of a control group with equivalent pathology, it makes it difficult to conclude that the addition of PRP in either of these studies was responsible for the functional outcome.

Only one study reported the use of bone marrow aspirate concentrate (BMAC) for ACL tears. Centeno et al. published a small case series of 10 patients with ACL tears treated with a fluoroscopically guided intra-ligamentous injection of autologous bone marrow concentrate and PRP [39]. Assessments involved ACL laxity and tear grade on MRI and patients with partial and complete ACL tears with less than 1-cm retraction were included. Pre- and post-injection MRIs were obtained and objectively assessed through

five different types of measurements of ACL pixel intensity for ligament integrity. Seven of ten patients showed improvement in at least four of five of these objective MRI measures. Improvements in mean visual analog scale (VAS) and mean Lower Extremity Functional Scale were documented, as well as mean reported improvement of 86.7%.

ACL Healing Enhancement With Partial Tear Repair

Gobbi et al. assessed the clinical outcomes of adding a PRP injection to ACL suture repair in addition to microfracture of the intercondylar notch in 58 athletes with partial ACL tears, with a 5-year follow-up [40]. They reported that 78% of the patients returned to their sports activities. They also reported a statistically significant decrease in side-to-side difference in anterior translation, from 4.1 mm (SD $\frac{1}{4}$ 1.6) preoperatively to 1.4 mm (SD $\frac{1}{4}$ 0.8) postoperatively at 5 years ($P < 0.05$). Four patients had a re-tear during sporting activity and underwent ACLR within 2 years from the primary repair surgery. The authors concluded that ACL repair + intercondylar notch microfracture + PRP was an effective technique to restore knee stability and function for acute partial ACL tears in young individuals.

ACL Reconstruction

Assessment of orthobiologics use in ACL reconstruction surgery has focused on three main parameters: (1) Maturation of the articular portion of the graft, (2) osteo-ligamentous integration of the graft into the tibial and femoral tunnels, and (3) clinical outcomes [41]. ACL graft maturation is most commonly assessed on MRI, with a low homogeneous intensity signal on T2-weighted and proton density-weighted MRI accepted as likely indicative of a maturing ACL graft. Several studies have shown improved graft maturation with PRP [42–45], while others reported no significant differences [46, 47]. A recent systematic review of 11 controlled trials concluded that PRP likely improves ACL graft maturation by up to 50% [17]. The authors suggested insufficient sample size as a potential explanation for lack of statistical significance

despite MRI improvement in measuring ACL graft maturation.

In the only study where histologic samples were obtained from PRP augmented ACL grafts, Sanchez et al. compared PRGF-assisted ACLRs ($n = 22$) to nonaugmented ACLRs ($n = 15$) in which a second-look arthroscopy was required (for either loose body or hardware removal, meniscal tears treatment, or cyclops lesions resection) at a minimum of 6 months [45]. Histologic analysis showed that newly formed connective tissue enveloping the graft was present in 77.3% of the PRP group compared to 40% in controls.

In a prospective randomized trial, Radice et al. compared 25 patients treated with ACLR in combination with PRP (GPS System, Biomet) to a control group of 25 patients who underwent surgery alone matched for age and gender [43]. They included 15 bone-patellar tendon-bone (BTB) autografts and 10 hamstrings autografts in each group. For the BTB autografts, 5 mL of activated PRP was added to an absorbable gelatin-compressed sponge (Gelfoam; Pfizer, Ixelles, Belgium) and sutured to the femoral plug and intra-articular parts of the graft, acting as a scaffold. For the hamstrings autografts, the same product was placed between the folded tendons and sutured in a similar manner. Monthly MRIs were performed from 3 to 9 months post-op to track the homogenization curve of the intra-articular portion of the graft, showing time to complete homogeneity in the PRP group was 177 days compared to 369 days in the control group. Moreover, in an analysis of the BTB autograft subgroup, the maturation time in the PRP group was 109 days versus 363 days in BTB controls. The authors concluded that PRP use in ACLR accelerates graft maturation by half of the expected time, with an additional reduction in maturation time from 12 months to 3.6 months when used in BTB autograft ACLR.

While several studies focused on the assessment of graft osteo-ligamentous integration (graft-bone tunnel incorporation) reported improved characteristics, there is a lack of sufficient evidence showing its correlation with clinical benefit of PRP use in ACLR [46, 48]. In a randomized, controlled, dou-

ble-blind study, Vogrin et al. investigated the effects of PRP gel application in hamstring autograft ACL reconstruction in 50 patients (25 thrombin-activated PRP-soaked grafts and 25 controls) [47]. They reported MRI evidence of improved vascularization along the ACL graft-bone interface at 3 months in the PRP group, with improved anterior-posterior instrumented knee stability measurements using a KT-2000 (MEDMETRIC; San Diego, CA) arthrometer at 6 months. However, in another double-blind, randomized controlled trial (RCT) involving 100 patients undergoing ACLR with BTB allograft (50 treated with platelet-enriched gel and 50 controls), Nin et al. reported no difference in International Knee Documentation Committee (IKDC) scores and KT-1000 arthrometer measurements (MEDmetric, San Diego, CA). Ventura et al. reported no differences in knee injury and osteoarthritis outcome score (KOOS), Tegner scores, or anterior-posterior instrumented knee stability measurements using a KT-1000 (MEDMETRIC; San Diego, CA) measurements between the PRP-treated group and control subjects at six following ACLR, despite the evidence of significant difference in graft appearance [49]. Orrego et al. similarly found no significant benefit in both Lysholm and International Knee Documentation Committee (IKDC) scores at 6 months post-ACLR, despite a favorable effect of PRP on graft maturation. Other studies have similarly reported limited to no evidence to support the use of PRP to augment ACL graft-bone tunnel incorporation [50–53]. An interesting observation from the existing literature is that nearly all of the studies used (leukocyte-rich) LR-PRP formulations, which have been known to increase local tissue inflammation and thus may delay or alter healing [54].

In a recent retrospective cohort of 151 knees in 143 patients ≤ 21 years of age in which hamstrings autograft ACLRs were augmented with PRP and a porous collagen carrier, Berdis et al. reported a decreased rate of second ACL injury, as well as reduced rates of ACL revision surgery. The patients in this study also show higher return to preinjury level of competition, with 132 returning to competitive sports at a pre-injury level, out of which 39 basketball players, and at an average of 22 weeks postsurgery [55].

ACL Reconstruction + Stem Cells/Cell-Based Therapy

Recently, Alentorn-Geli et al. published clinical outcomes of 20 soccer players undergoing ACL reconstruction using BTB autograft infiltrated with adipose-derived regenerative stem cells (ADRC) at the end of the procedure, with a 12-month follow-up [56]. This cohort was compared to a historical, matched cohort of 19 soccer players undergoing the same procedure without ADRC. They reported no significant differences in outcomes improvement between groups across time ($p > 0.05$). All patients returned to sports after surgery, but 8 (40%) patients in the ADRC and 13 (68.4%) patients in the control group had lower Tegner activity score at 12 months postop.

PRP for ACL Harvest/Donor Site

Another utilization of PRP use in ACLR focused on its influence on donor site (graft harvest site) pain and healing, with several clinical studies presenting promising early results. De Almeida et al. reported that adding PRP to the patellar tendon harvest site resulted in better immediate postoperative pain scores, and less patellar tendon gapping on MRI at 6 months from surgery [57]. In a double-blind RCT, Seijas et al. reported decreased anterior knee pain following PRGF application to BTB harvest site in ACLR, compared to controls [58]. In another study, Cervellin et al. 128 did not find a significant difference in VAS pain scores, but the PRP group had a significantly higher VISA score [59]. A recent double-blinded RCT by Walters et al. in which PRP was applied to the harvest site in BTB ACLR showed less favorable results with similar levels of kneeling pain and patellar defect sizes in both the PRP and control groups [60].

Future Directions

New approaches are continuously being developed in an attempt to harness the advantageous regenerative properties in orthobiologics to enhance the existing healing potential of the ACL. One interesting direction was introduced by Murray et al. following many years of preclinical studies [61]. They introduced a technique

using a collagen scaffold soaked with whole blood to deliver platelets in combination with a novel bio-enhanced primary repair technique using a suture stent, called: bridge-enhanced ACL repair (BEAR technique). Use of this technique in an animal model was reported to result in equivalent biomechanical properties between the repaired ACL equivalent and an ACLR at 3, 6, and 12 months postsurgery [24]. Furthermore, this novel technique of bio-enhanced repair prevented the development of cartilage lesions, which were seen 12 months after untreated ACL transection and ACLR in an animal model. In a recent first clinical trial with the using BEAR technique in humans, ten patients underwent treatment using the BEAR technique compared to ten hamstrings autograft ACLRs [62]. Authors reported that the BEAR group produced similar outcomes to ACLR with a hamstring autograft at 12 and 24 months postsurgery, measured by subjective and objective IKDC scores, stability measures by arthrometer, and in functional hop testing. The BEAR group presented with higher hamstring strength indices.

46.2.2 Medial Collateral Ligament (MCL)

Medial collateral ligament (MCL) injuries are the most common knee ligament injuries. Spontaneous healing and nonoperative management is the usual clinical scenario in the large majority of cases [63]. Thirty-five MCL injuries have been recorded in female collegiate and high-school basketball between 2009–2010 and 2013–2014 seasons, and 33 injuries in males [64]. While the MCL has shown to have good healing potential [31], it has been reported that these injuries can lead to chronic pain, laxity, joint instability, and possibly osteoarthritis [65]. These injuries often present a serious problem to athletes as they can result in significant time away from sports in the competitive context. Several attempts have been made to enhance the healing process of the MCL with orthobiologic therapies and restore the normal ligament functionality as much as possible.

Early preclinical evidence suggested promising properties of PRP use for MCL injury with enhanced healing potential in animal models [8–10]. However, later studies reported less favorable results. Yoshioka et al. observed significantly improved structural properties of rabbit MCLs treated with PRGF (LP-PRP) compared to controls [66]. Conversely, Amar et al. reported no histological or biomechanical differences between PRP-treated MCLs and controls in a rat model [67]. More recently, LaPrade et al. reported that either a single dose of platelet-poor plasma (PPP) or a 2-times dose of PRP at the time of injury did not accelerate ligament healing. Moreover, a 4-times dose of PRP resulted in a significant negative effect on collagen orientation and ligament strength (compared to a sham group) at 6 weeks post injury.

Reports on clinical use of PRP for the treatment of MCL injuries are limited with the majority being in the form of case reports [68–70]. Recently, Lundblad et al. reported the use of PRP injections for MCL injuries in 20 elite-level football players out of a prospective cohort of 130 players over three full seasons (2 players with MRI grade I; 17 players with MRI grade II; and 1 player with MRI grade III MCL injuries) [71]. There were no differences in lay-off times in players treated with PRP or not, in grade II MCL injury grading (n.s.). However, there is no information on which type of PRP was used, how many injections were administered, time from injury to injection, MCL area involved as well as post-injection protocol (variance in brace administration was reported in this study), all factors which substantially limit the quality of any conclusion drawn from this study with regard to the use of PRP for treatment of MCL injuries.

46.2.3 Ankle Sprains

Ankle sprains have been highlighted in most epidemiologic studies as the most common type of injury in basketball across age groups, genders, and all levels of play [72–76]. To date, there are very few high-level studies analyzing the use of PRP injections for ankle or high-ankle sprains,

with two published RCTs (one for ankle sprains and one for high-ankle sprains). In a double-blinded placebo-controlled RCT, Rowden et al. compared ultrasound-guided LR-PRP injections with local anesthetic versus normal saline injection with local anesthetic for acute ankle sprains in 37 patients [77]. Primary outcome measures were VAS pain score and Lower Extremity Functional Sale (LEFS) on day 0 (baseline), day 3, and day 8. This study had various limitations, apart from the small sample size and short follow-up, with lack of documentation of ankle sprain grade. All patients were treated with a posterior splint with non-weight-bearing restrictions for 3 days. Pain medication was given at the physician's discretion with no documentation as well. The investigators found no statistical difference in VAS pain score or LEFS between the two groups. In a recent, small RCT in 21 patients with grade II lateral ankle sprain, 12 ankles were treated with a single PRP injection to the anterior talofibular ligament (ATFL) and rigid immobilization compared to a control group of 11 patients treated with rigid immobilization alone. The PRP group showed better pain reduction and better functional scores than the control group at 8 weeks, but clinical outcomes were similar in both groups at 24 weeks [78].

In another RCT, Laver et al. treated 16 elite athletes diagnosed with high ankle sprains, with either an ultrasound-guided LP-PRP injection to the antero-inferior tibiofibular ligament (AITFL) at initial presentation with a repeat injection 7 days later in conjunction with a rehabilitation program (eight athletes), versus a rehabilitation program alone (eight athletes) [79]. Primary outcomes were measured by return to play (RTP) and dynamic ultrasound studies. All patients followed the same rehabilitation protocol and RTP criteria. They reported the LP-PRP group returned to play in a shorter period of time (40.8 days) compared with the control group (59.6 days, $P < 0.006$). Only one patient had residual pain following RTP in the PRP group, whereas five patients had residual pain in the control group. No significant difference was seen on dynamic ultrasound examination in external rotation between the two groups 6 weeks

post injury. In another study, Samra et al. treated ten Rugby Union players with a single PRP injection into the AITFL within 14 days of an MRI confirmed ankle syndesmosis injury. A historical control group included 11 comparable Rugby Union players [80]. They reported a significantly shortened RTP time ($p = 0.048$). Additionally, athletes in the intervention group showed higher agility ($p = 0.002$) and vertical jump ($p = 0.001$), as well as a lower level of fear avoidance associated with rugby ($p = 0.014$). They concluded that a single PRP injection for high ankle sprains may accelerate a safe and successful return to Rugby Union, with improved functional capacity [80].

Fact Box

Current evidence has not shown PRP to be efficacious in acute ankle sprains; however, evidence suggests that LP-PRP injections may be beneficial in the management of high ankle sprains to reduce return-to-play time and decrease incidence of residual pain in elite athletes. Further high-quality evidence is needed to define the role of PRP and cell-based therapies in ankle and high-ankle sprains.

46.2.4 Ulnar Collateral Ligament of the Elbow Injuries

Ulnar collateral ligament (UCL) tears and subsequent medial elbow instability are highly prevalent and dreadful injuries in athletes participating in overhead throwing sports, particularly baseball pitchers. Additionally, javelin throwers, arm wrestlers, and collegiate wrestlers are also at risk for these types of injuries [81]. Since Jobe et al. performed the first medial ulnar collateral reconstruction in 1974, this once considered career-ending injury has become a surgically treatable pathology in most athletes with moderate to excellent rates (53–90%) of return to play at a professional level (depending on the author) [82, 83]. Although the Tommy John procedure has

become the standard of care for UCL deficiency, orthobiologics may play a role in athletes during mid-season in order to postpone surgery, as an adjunct during surgery to promote healing or as a sole treatment in mild UCL injuries and patients who elect conservative treatment.

Literature supporting the use of orthobiologics in UCL injuries is limited. Dines et al. published a retrospective series of 44 baseball players with partial UCL tears treated with PRP injections and rehabilitation protocol [84]. They reported that 32 patients (73%) had a good to excellent outcome and that 67% of professional players returned to professional play. Podesta et al. treated 34 athletes who failed 2 months of conservative treatment for a partial UCL tear, with a PRP injection under ultrasound. They reported that 88% of patients returned to the same level of play without complaints and the average time to return to play was 12 weeks. Only one patient suffered persistent UCL instability and underwent surgery [85].

A recent study, McQueen et al. performed a comparative analysis of nonoperative treatment of UCL injuries in professional baseball players with or without PRP. The Health and Injury Tracking System (HITS) was reviewed, and the authors of the study found that players who received a PRP injection had longer time before returning to throwing (64 days vs. 51 days, $p < 0.001$); however, they concluded it might be due to a delay between the injury date and PRP injection (mean time from injury date to PRP injection was 14.5 days). There was no significant effect on the likelihood of surgical intervention [86].

More recently, Kato et al. published a series of 30 baseball players with partial or complete UCL injuries (9 grade 1 UCL injury; 13 grade 2; 8 grade 3) who failed 2 months of conservative treatment, and were treated with ultrasound guided trephination and an LP-PRP injection (ACP™, Arthrex, Florida, USA) [87]. They reported that 26 out of the 30 athletes were able to RTS at pre-injury level of play at an average of 12.4 weeks (range: 10–18), while four athletes required surgery (3 grade 2; 1 grade 3; 3 had distal tears; and 1 proximal). Improvements were recorded in visual analog scale (VAS) scores, Disabilities of the Arm,

Shoulder, and Hand (DASH) sports module scores, and sonographic ulno-humeral joint space opening with valgus stress.

To date, limited evidence exists on outcomes of management of UCL injuries using cell-based therapies.

46.2.5 Muscle Injuries

Muscle injuries are very common among athletes in general and basketball players in particular; In a recent prospective follow-up study of 59 male professional European basketball players, muscle injuries were found to have a higher incidence than ankle sprains, accounting for 21.2% of all injuries, with similar return to play (RTP) time of 7.6 ± 7.1 days for muscle injuries compared to 8.4 ± 9.5 days for ankle injuries [88]. This presents a significant problem for basketball team clinicians as management of muscle injuries can often be challenging no less than ankle injuries, as shown in this study [88]. Muscle injuries in the athlete can be classified into intrinsic and extrinsic injuries. Intrinsic muscle injuries occur most commonly at the myotendinous junction during eccentric contraction with tearing of the muscle fibers. Extrinsic muscle injuries in the athlete occur most commonly as a result of a contusion injury [89]. Conservative management has been the mainstay of treatment for most muscle injuries and usually consists of rest, ice, compression and elevation (RICE protocol), physiotherapy, NSAIDs, and time [90]. Aiming to promote early return to play, decrease recurrence rates and minimize fibrosis and subsequent muscle weakness, newer treatment modalities have been introduced into the field, including PRP and cell therapy [89, 91].

Muscle tissue regeneration is commonly limited by scar tissue formation, rather than by the rate of muscle regeneration [92, 93]. While the potential benefit of orthobiologics use for muscle injuries is not only early return to sports but also improved tissue healing with improved structural properties, potentially reducing the risk of recurrence, most clinical studies have only focused on return to sports rates and durations. With this in mind, Terada et al. performed a preclinical study

assessing muscle healing of contusion-injured tibialis anterior muscle in mice with combined treatment of an oral antifibrotic agent (Losartan) and PRP [94]. The study showed increased muscle regeneration and function, along with decreased fibrosis in the experimental group. In vitro work has shown that PRP use can lead to myoblast proliferation, but not to myoblast differentiation, which is important in producing muscle tissue [95]. Furthermore, some growth factors contained in platelets, specifically myostatin and TGF- β 1, have been proven detrimental to muscle regeneration [96, 97]. Several other preclinical studies involving the injection of PRP alone for gastrocnemius muscle injury in mice and rats have shown mixed results regarding the acceleration of tissue healing [98–103] performed controlled laboratory studies which suggested that platelet poor plasma (PPP) and PRP preparations subjected to a second spin to remove platelets led to the induction of myoblast cells into the muscle differentiation pathway [102, 103]. PRP that was not modified with a second spin led to induction into the muscle proliferation pathway. They concluded that these results suggests that PPP and LP-PRP subjected to a second spin to remove platelets could be used to stimulate muscle differentiation and subsequent muscle regeneration [102, 103].

Several studies have reported positive outcomes of autologous conditioned serum (ACS) and PRP injections for the treatment of muscle strains. Wright-Carpenter et al. have reported significantly shorter recovery time in a case-control study of professional sportsmen with various muscle strains who were treated with ACS [104]. Sanchez et al. have reported the use of PRP in muscle injuries of different severities in 21 professional soccer players. Their results suggested the PRP group required half the time to resume normal training activities when compared to matched historical controls [90]. Rossi et al. performed a randomized controlled trial comparing a rehabilitation program alone vs. a rehabilitation program plus a PRP injection for muscle injury (Hamstrings, quadriceps, and gastrocnemius) [105]. They presented significantly earlier full recovery (21 days vs. 2 days) and significantly lower pain scores in the PRP group.

We are unaware of clinical studies supporting the use of BMAC or other cell-based therapies in the management of muscle injuries.

46.2.6 Hamstring Muscle Injuries

Hamstrings injuries are one of the most common injuries in professional athletes and is also common in basketball [106], usually dictating a prolonged rest period and delayed return to play even in mild injuries. Hamid et al. published a randomized controlled trial of 28 patients comparing a rehabilitation program with and without a PRP injection for hamstrings injury [107]. They found shorter time to return to play in the PRP group (26.7 days) when compared to the rehabilitation alone group (42.5 days). Another recent prospective study by Bezuglov et al. reported similar results in 40 soccer players [108].

Other studies on outcomes of PRP injections in hamstrings injuries, however, do not support its use. Reurink et al. performed a double-blind, placebo-controlled, randomized study on 80 professional and recreational athletes with acute hamstrings injuries treated with two intramuscular injections of PRP or isotonic saline [109]. They found no benefit for intramuscular PRP injections for acute hamstrings tears. Several other studies reported the lack of benefit for intramuscular PRP injections for acute hamstrings tears [110–113].

Fact Box

Current literature does not provide sufficient evidence to support the use of PRP for muscle injury; however, many studies are relatively heterogenous regarding injury type and preparation method. Laboratory studies suggest PPP or LP-PRP with platelets removed may induce muscle regeneration. Further high-quality evidence is needed to define the role of PRP and cell-based therapies in muscle injuries.

Our personal approach to orthobiologics use for muscle injuries depends on the extent of muscle injury and would be considered in cases where true and significant muscle fibers disruption is confirmed on imaging studies. In cases where hematomas or seromas are present, they are evacuated under ultrasound guidance to decompress the area of injury; if and once the hematoma is evacuated, a platelet poor fraction (i.e., Fraction 1—F 1 in PRGF) is activated and injected into the injury site and adjacent peripheral healthy muscle (Fig. 46.2). We primarily prefer to use the platelet-poor fraction since it has a reduced concentration of the pro-fibrotic factor TGF β -1, unlike the platelet-rich fraction, which is adjacent to the buffy coat layer or leukocytes sediment; Repeated ultrasound (US) or MRI imaging is used to follow healing progression and assess for excessive fibrosis which may predispose to reinjury. Repeated injections may be applied at a minimum of 1 week intervals and are based on US imaging (to assess muscle tissue damage) and symptoms.

While the majority of muscle injuries are managed conservatively, another potential application of orthobiologics use for muscle injuries is in scenarios requiring surgical management. Such scenarios include: Complete or extensive musculo-tendinous junction (MTJ) avulsion in athletes, chronic symptomatic limiting injuries, and/or symptomatic nerve involvement. In such cases, for example in hamstrings and rectus femoris proximal injuries which are of severe definition in athletes, orthobiologics use could be considered during to surgery, with platelet-poor plasma fraction infiltration into and around the repair site (Fig. 46.3).

46.3 Summary

Orthobiologics have emerged in recent years as a safe and promising treatment option for musculo-skeletal injuries and pathologies. However, evidence of its efficacy has been mixed and highly variable depending on the specific indication. Current evidence presents large heterogeneity in the various orthobiologic products, protocols, and characteristics making interpretation of existing literature a complicated task. Recent litera-

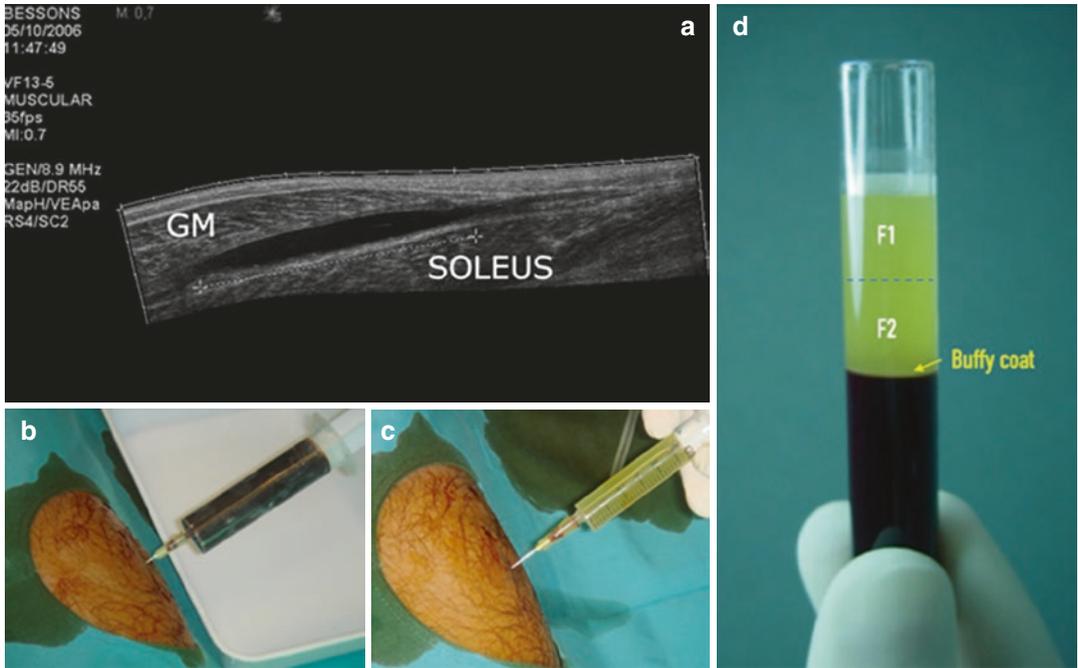


Fig. 46.2 (a) Ultrasound image of an extensive soleus muscle injury and the area of surrounding hematoma (Arrow; black area inside the muscle); (b) Hematoma evacuation using a 10 cc syringe; (c) Injection of platelet-

poor plasma (PRGF F1 fraction, Endoret® System) intramuscular injection using a 10 cc syringe; (d) PRGF fractions distribution following centrifugation



Fig. 46.3 Surgically repaired proximal rectus femoris MTJ injury. With platelet-poor plasma being injected into the repair site

ture has shown orthobiologics to have promising potential in improving tissue regeneration in laboratory and animal studies; however, results in the clinical setting have been mixed and variable in demonstrating consistent efficacy. Future high-quality large clinical trials are necessary to determine the true clinical value of these treatment options.

Take Home Messages

- The best available clinical evidence does not demonstrate efficacy of PRP injections for ACL reconstruction.
- There is currently insufficient high-quality evidence for the recommendation of PRP injections in high ankle sprains, but small clinical trials have shown promising efficacy for LP-PRP injections for high ankle sprains.
- There is sufficient evidence to consider the use of PRP for UCL injuries that are refractory to a first line of conservative treatment; however, there are conflicting results reported and athletes who have significant restrictions on time lines for return to sport must weigh that factor into the decision process.
- Current literature is conflicting and heterogeneous regarding the use of PRP for muscle injuries, while preclinical stud-

ies suggest that PPP may hold promise for muscle injuries. Further high-quality clinical trials are necessary to validate this.

- There is lack of sufficient studies to support cell-based therapies for the management of soft tissue injuries and therefore concrete evidence-based clinical recommendations cannot be made.
- In summary, orthobiologics have yet to be thoroughly studied in specific soft tissue injuries in athletes in general and basketball players in particular. Future high-quality studies in these populations will unveil the true clinical value of this emerging field.

References

1. DeLong JM, Russell RP, Mazzocca AD. Platelet-rich plasma: the PAW classification system. *Arthroscopy*. 2012;28(7):998–1009.
2. Dohan Ehrenfest DMAI, Zumstein MA, Zhang CQ, et al. Classification of platelet concentrates (Platelet-Rich Plasma-PRP, Platelet-Rich Fibrin-PRF) for topical and infiltrative use in orthopedic and sports medicine: current consensus, clinical implications and perspectives. *Muscles Ligaments Tendons J*. 2014;4(1):3–9.
3. Holton J, Imam M, Ward J, et al. The basic science of bone marrow aspirate concentrate in chondral injuries. *Orthop Rev*. 2016;8(3):6659.
4. Sheth U, Simunovic N, Klein G, et al. Efficacy of autologous platelet-rich plasma use for orthopaedic indications: a meta-analysis. *J Bone Joint Surg Am*. 2012;94(4):298–307.
5. Zhang L, Hu J, Athanasiou KA. The role of tissue engineering in articular cartilage repair and regeneration. *Crit Rev Biomed Eng*. 2009;37(1–2):1–57.
6. Madry H, Kohn D, Cucchiari M. Direct FGF-2 gene transfer via recombinant adeno-associated virus vectors stimulates cell proliferation, collagen production, and the repair of experimental lesions in the human ACL. *Am J Sports Med*. 2013;41:194–202.
7. Marui T, Niyibizi C, Georgescu HI, Cao M, Kavalkovich KW, Levine RE, et al. Effect of growth factors on matrix synthesis by ligament fibroblasts. *J Orthop Res*. 1997;15:18–23.
8. Hildebrand KA, Woo SL-Y, Smith DW, Allen CR, Deie M, Taylor BJ, et al. The effects of platelet derived growth factor-BB on healing of the rabbit medial collateral ligament: an in vivo study. *Am J Sports Med*. 1998;26:549–54.23.
9. Batten ML, Hansen JC, Dahners LE, et al. Influence of dosage and timing of application of platelet derived growth factor on early healing of the rat medial collateral ligament. *J Orthop Res*. 1996;14:736–41.
10. Fleming BC, Spindler KP, Palmer MP, Magarian EM, Murray MM. Collagen-platelet composites improve the biomechanical properties of healing anterior cruciate ligament grafts in a porcine model. *Am J Sports Med*. 2009;37:1554–63.
11. Garrett WE Jr, Swiontkowski MF, Weinstein JN, et al. American Board of Orthopaedic Surgery Practice of the Orthopaedic surgeon: part-II, certification examination case mix. *J Bone Joint Surg Am*. 2006;88(3):660–7.
12. Kiapour AM, Murray MM. Basic science of anterior cruciate ligament injury and repair. *Bone Joint Res*. 2014;3(2):20–31.
13. Hutchinson ID, Rodeo SA, Perrone GS, et al. Can platelet-rich plasma enhance anterior cruciate ligament and meniscal repair? *J Knee Surg*. 2015;28(1):19–28.
14. Xie J, Wang C, Huang DY, et al. TGF-beta1 induces the different expressions of lysyl oxidases and matrix metalloproteinases in anterior cruciate ligament and medial collateral ligament fibroblasts after mechanical injury. *J Biomech*. 2013;46(5):890–8.
15. Kobayashi D, Kurosaka M, Yoshiya S, et al. Effect of basic fibroblast growth factor on the healing of defects in the canine anterior cruciate ligament. *Knee Surg Sports Traumatol Arthrosc*. 1997;5(3):189–94.
16. Molloy T, Wang Y, Murrell G. The roles of growth factors in tendon and ligament healing. *Sports Med*. 2003;33(5):381–94.
17. Andriolo L, Di Matteo B, Kon E, et al. PRP augmentation for ACL reconstruction. *Biomed Res Int*. 2015;2015:371746.
18. Kondo E, Yasuda K, Yamanaka M, et al. Effects of administration of exogenous growth factors on biomechanical properties of the elongation-type anterior cruciate ligament injury with partial laceration. *Am J Sports Med*. 2005;33(2):188–96.
19. Spindler KP, Imro AK, Mayes CE, et al. Patellar tendon and anterior cruciate ligament have different mitogenic responses to platelet-derived growth factor and transforming growth factor beta. *J Orthop Res*. 1996;14(4):542–6.
20. Yoshikawa T, Tohyama H, Katsura T, et al. Effects of local administration of vascular endothelial growth factor on mechanical characteristics of the semitendinosus tendon graft after anterior cruciate ligament reconstruction in sheep. *Am J Sports Med*. 2006;34(12):1918–25.
21. Takayama K, Kawakami Y, Mifune Y, et al. The effect of blocking angiogenesis on anterior cruciate ligament healing following stem cell transplantation. *Biomaterials*. 2015;60:9–19.
22. Murray MM, Palmer M, Abreu E, et al. Platelet-rich plasma alone is not sufficient to enhance suture

- repair of the ACL in skeletally immature animals: an in vivo study. *J Orthop Res.* 2009;27(5):639–45.
23. Cheng M, Johnson VM, Murray MM. Effects of age and platelet-rich plasma on ACL cell viability and collagen gene expression. *J Orthop Res.* 2012;30:79–85.
 24. Vavken P, Fleming BC, Mastrangelo AN, Machan JT, Murray MM. Biomechanical outcomes after bio-enhanced anterior cruciate ligament repair and anterior cruciate ligament reconstruction are equal in a porcine model. *Arthroscopy.* 2012;28:672–80.
 25. Guo R, Gao L, Xu B. Current evidence of adult stem cells to enhance anterior cruciate ligament treatment: a systematic review of animal trials. *Arthroscopy.* 2018;34(1):331–340.e2.
 26. Oe K, Kushida T, Okamoto N, et al. New strategies for anterior cruciate ligament partial rupture using bone marrow transplantation in rats. *Stem Cell Dev.* 2011;20(4):671–9.
 27. Kanaya A, Deie M, Adachi N, et al. Intra-articular injection of mesenchymal stromal cells in partially torn anterior cruciate ligaments in a rat model. *Arthroscopy.* 2007;23(6):610–7.
 28. Lui PP, Wong OT, Lee YW. Application of tendon-derived stem cell sheet for the promotion of graft healing in anterior cruciate ligament reconstruction. *Am J Sports Med.* 2014;42(3):681–9.
 29. Hur CI, Ahn HW, Seon JK, Song EK, Kim GE. Mesenchymal stem cells decrease tunnel widening of anterior cruciate ligament reconstruction in rabbit model. *Int J Stem Cells.* 2019;12(1):162–9.
 30. Sun Y, Chen W, Hao Y, Gu X, Liu X, Cai J, Liu S, Chen J, Chen S. Stem cell-conditioned medium promotes graft remodeling of Midsubstance and Intratunnel incorporation after anterior cruciate ligament reconstruction in a rat model. *Am J Sports Med.* 2019;47(10):2327–37.
 31. Woo SL, Vogrin TM, Abramowitch SD. Healing and repair of ligament injuries in the knee. *J Am Acad Orthopaed Surg.* 2000;8(6):364–72.
 32. Vavken P, Murray MM. The potential for primary repair of the ACL. *Sports Med Arthrosc.* 2011;19:44–9.
 33. Costa-Paz M, Ayerza MA, Tanouira I, Astoul J, Muscolo DL. Spontaneous healing in complete ACL ruptures: a clinical and MRI study. *Clin Orthop Relat Res.* 2012;470:979–85.
 34. Ihara H, Miwa M, Deya K, Torisu K. MRI of anterior cruciate ligament healing. *J Comput Assist Tomogr.* 1996;20:317–21.
 35. Nguyen DT, Ramwadhoebe TH, van der Hart CP, Blankevoort L, Tak PP, van Dijk CN. Intrinsic healing response of the human anterior cruciate ligament: an histological study of reattached ACL remnants. *J Orthop Res.* 2014;32:296–301.
 36. Seijas R, Ares O, Cusco X, Alvarez P, Steinbacher G, Cugat R. Partial anterior cruciate ligament tears treated with intraligamentary plasma rich in growth factors. *World J Orthop.* 2014;5:373–8.
 37. Anitua E. Plasma rich in growth factors: preliminary results of use in the preparation of future sites for implants. *Int J Oral Maxillofac Implants.* 1999;14:529–35.
 38. Koch M, Mayr F, Achenbach L, Krutsch W, Lang S, Hilber F, Weber J, Pfeifer CG, Woehl R, Eichhorn J, Zellner J, Nerlich M, Angele P. Partial anterior cruciate ligament ruptures: advantages by intraligament autologous conditioned plasma injection and healing response technique-midterm outcome evaluation. *Biomed Res Int.* 2018;2018:3204869.
 39. Centeno CJ, Pitts J, Al-Sayegh H, Freeman MD. Anterior cruciate ligament tears treated with percutaneous injection of autologous bone marrow nucleated cells: a case series. *J Pain Res.* 2015;8:437–47.
 40. Gobbi A, Karnatzikos G, Sankineani SR, Petreria M. Biological augmentation of ACL refixation in partial lesions in a group of athletes: results at the 5-year follow-up. *Tech Orthop.* 2013;28:180–4.
 41. Di Matteo B, Loibl M, Andriolo L, et al. Biologic agents for anterior cruciate ligament healing: a systematic review. *World J Orthop.* 2016;7(9):592–603.
 42. Orrego M, Larrain C, Rosales J, et al. Effects of platelet concentrate and a bone plug on the healing of hamstring tendons in a bone tunnel. *Arthroscopy.* 2008;24(12):1373–80.
 43. Radice F, Yanez R, Gutierrez V, et al. Comparison of magnetic resonance imaging findings in anterior cruciate ligament grafts with and without autologous platelet-derived growth factors. *Arthroscopy.* 2010a;26(1):50–7.
 44. Seijas R, Ares O, Catala J, et al. Magnetic resonance imaging evaluation of patellar tendon graft remodeling after anterior cruciate ligament reconstruction with or without platelet-rich plasma. *J Orthop Surg (Hong Kong).* 2013;21(1):0–4.
 45. Sanchez M, Anitua E, Azofra J, et al. Ligamentization of tendon grafts treated with an endogenous preparation rich in growth factors: gross morphology and histology. *Arthroscopy.* 2010;26(4):470–80.
 46. Nin JRV, Gasque GM, Azcarate AV, et al. Has platelet-rich plasma any role in anterior cruciate ligament allograft healing? *Arthroscopy.* 2009;25(11):1206–13.
 47. Vogrin M, Ruppreht M, Dinevski D, et al. Effects of a platelet gel on early graft revascularization after anterior cruciate ligament reconstruction: a prospective, randomized, double-blind, clinical trial. *Eur Surg Res.* 2010;45(2):77–85.
 48. Mirzatooleei F, Alamdari MT, Khalkhali HR. The impact of platelet-rich plasma on the prevention of tunnel widening in anterior cruciate ligament reconstruction using quadrupled autologous hamstring tendon. *Bone Joint J.* 2013;95-B(1):65–9.
 49. Ventura A, Terzaghi C, Borgo E, et al. Use of growth factors in ACL surgery: preliminary study. *J Orthop Traumatol.* 2005;6(2):76–9.

50. Ji Q, Yang Y, Chen H, Geng W, Dong H, Yu Q. Clinical evaluations of anterior cruciate ligament reconstruction with platelet rich plasma. *Zhongguo Xiu Fu Chong Jian Wai Ke Za Zhi*. 2017;31(4):410–6.
51. Figueroa D, Melean P, Calvo R, et al. Magnetic resonance imaging evaluation of the integration and maturation of semitendinosus-gracilis graft in anterior cruciate ligament reconstruction using autologous platelet concentrate. *Arthroscopy*. 2010;26(10):1318–25.
52. Sözkese S, Karahan HG, Kurtulmus A, Kayali C, Altay T. PRP on Prevention of tunnel enlargement in ACL Reconstruction. *Ortop Traumatol Rehabil*. 2018;20(4):285–91.
53. Del Torto M, Enea D, Panfoli N, Filardo G, Pace N, Chiusaroli M. Hamstrings anterior cruciate ligament reconstruction with and without platelet rich fibrin matrix. *Knee Surg Sports Traumatol Arthrosc*. 2015;23(12):3614–22.
54. Drago J, Braun HJ, Durham JL, et al. Comparison of the acute inflammatory response of two commercial platelet-rich plasma systems in healthy rabbit tendons. *Am J Sports Med*. 2012;40(6):1274–81.
55. Berdis AS, Veale K, Fleissner PR Jr. Outcomes of anterior cruciate ligament reconstruction using biologic augmentation in patients 21 years of age and younger. *Arthroscopy*. 2019; pii: S0749-8063(19)30495-5
56. Alentorn-Geli E, Seijas R, Martínez-De la Torre A, Cuscó X, Steinbacher G, Álvarez-Díaz P, Barastegui D, Navarro J, Serra-Renom JM, Nishishinya B, Català J, Laiz P, García-Balletbó M, Cugat R. Effects of autologous adipose-derived regenerative stem cells administered at the time of anterior cruciate ligament reconstruction on knee function and graft healing. *J Orthop Surg (Hong Kong)*. 2019;27(3):2309499019867580.
57. de Almeida AM, Demange MK, Sobrado MF, et al. Patellar tendon healing with platelet-rich plasma: a prospective randomized controlled trial. *Am J Sports Med*. 2012;40(6):1282–8.
58. Seijas R, Cusco X, Sallent A, et al. Pain in donor site after BTB-ACL reconstruction with PRGF: a randomized trial. *Arch Orthop Trauma Surg*. 2016;136(6):829–35.
59. Cervellin M, de Girolamo L, Bait C, et al. Autologous platelet-rich plasma gel to reduce donor-site morbidity after patellar tendon graft harvesting for anterior cruciate ligament reconstruction: a randomized, controlled clinical study. *Knee Surg Sports Traumatol Arthrosc*. 2012;20(1):114–20.
60. Walters BL, Porter DA, Hobart SJ, Bedford BB, Hogan DE, McHugh MM, Klein DA, Harousseau K, Nicholas SJ. Effect of Intraoperative Platelet-Rich Plasma Treatment on Postoperative Donor Site Knee Pain in Patellar Tendon Autograft Anterior Cruciate Ligament Reconstruction: A Double-Blind Randomized Controlled Trial. *Am J Sports Med*. 2018;46(8):1827–35.
61. Murray MM, Fleming BC. Use of a bioactive scaffold to stimulate anterior cruciate ligament healing also minimizes posttraumatic osteoarthritis after surgery. *Am J Sports Med*. 2013;41:1762–70.
62. Murray MM, Kalish LA, Fleming BC, Team BEART, Flutie B, Freiburger C, Henderson RN, Perrone GS, Thurber LG, Proffen BL, Ecklund K, Kramer DE, Yen YM, Micheli LJ. Bridge-enhanced anterior cruciate ligament repair: two-year results of a first-in-human study. *Orthop J Sports Med*. 2019;7(3):2325967118824356.
63. Swenson DM, Collins CL, Best TM, Flanigan DC, Fields SK, Comstock RD. Epidemiology of knee injuries among us high school athletes, 2005/06–2010/11. *Med Sci Sports Exerc*. 2012.
64. Stanley LE, Kerr ZY, Dompier TP, Padua DA. Sex differences in the incidence of anterior cruciate ligament, medial collateral ligament, and meniscal injuries in collegiate and high school sports: 2009–2010 through 2013–2014. *Am J Sports Med*. 2016;44(6):1565–72.
65. Felson DT, Lawrence RC, Dieppe PA, Hirsch R, Helmick CG, Jordan JM, Kington RS, Lane NE, Nevitt MC, Zhang Y, Sowers M, McAlindon T, Spector TD, Poole AR, Yanovski SZ, Ateshian G, Sharma L, Buckwalter JA, Brandt KD, Fries JF. Osteoarthritis: new insights. Part 1: the disease and its risk factors. *Ann Intern Med*. 2000;133(8):635–46.
66. Yoshioka T, Kanamori A, Washio T, Aoto K, Uemura K, Sakane M, Ochiai N. The effects of plasma rich in growth factors (PRGF-Endoret) on healing of medial collateral ligament of the knee. *Knee Surg Sports Traumatol Arthrosc*. 2013;21(8):1763–9.
67. Amar E, Snir N, Sher O, Brosh T, Khashan M, Salai M, Dolkart O. Platelet-rich plasma did not improve early healing of medial collateral ligament in rats. *Arch Orthop Trauma Surg*. 2015;135(11):1571–7.
68. Eirale C, Mauri E, Hamilton B. Use of platelet rich plasma in an isolated complete medial collateral ligament lesion in a professional football (soccer) player: a case report. *Asian J Sports Med*. 2013;4(2):158–62.
69. Yoshida M, Marumo K. An autologous leukocyte-reduced platelet-rich plasma therapy for chronic injury of the medial collateral ligament in the knee: a report of 3 successful cases. *Clin J Sport Med*. 2019;29(1):e4–6.
70. Bagwell MS, Wilk KE, Colberg RE, Dugas JR. The use of serial platelet rich plasma injections with early rehabilitation to expedite grade III medial collateral ligament injury in a professional athlete: a case report. *Int J Sports Phys Ther*. 2018;13(3):520–5.
71. Lundblad M, Häggglund M, Thomeé C, Hamrin Senorski E, Ekstrand J, Karlsson J, Waldén M. Medial collateral ligament injuries of the knee in male professional football players: a prospective three-season study of 130 cases from the UEFA

- Elite Club Injury Study. *Knee Surg Sports Traumatol Arthrosc* 2019;4.
72. Zuckerman SL, Wegner AM, Roos KG, Djoko A, Dompier TP, Kerr ZY. Injuries sustained in National Collegiate Athletic Association men's and women's basketball 2009/2010 – 2014/2015. *Br J Sports Med*. 2018;52(4):261–8.
 73. Clifton DR, Hertel J, Onate JA, Currie DW, Pierpoint LA, Wasserman EB, Knowles SB, Dompier TP, Comstock RD, Marshall SW, Kerr ZY. The first decade of web-based sports injury surveillance: descriptive epidemiology of injuries in US high school girls' basketball (2005–2006 through 2013–2014) and National Collegiate Athletic Association women's basketball (2004–2005 through 2013–2014). *J Athl Train*. 2018a;53(11):1037–48.
 74. Meeuwisse WH, Sellmer R, Hagel BE. Rates and risks of injury during intercollegiate basketball. *Am J Sports Med*. 2003;31(3):379–85.
 75. Clifton DR, Onate JA, Hertel J, Pierpoint LA, Currie DW, Wasserman EB, Knowles SB, Dompier TP, Marshall SW, Comstock RD, Kerr ZY. The first decade of web-based sports injury surveillance: descriptive epidemiology of injuries in US high school boys' basketball (2005–2006 through 2013–2014) and National Collegiate Athletic Association men's basketball (2004–2005 through 2013–2014). *J Athl Train*. 2018b;53(11):1037–48.
 76. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the national basketball association: a 17-year overview. *Sports Health*. 2010;2(4):284–90. <https://doi.org/10.1177/1941738109357303>.
 77. Rowden A, Dominici P, D'Orazio J, Manur R, Deitch K, Simpson S, Kowalski MJ, Salzman M, Ngu D. Double-blind, randomized, placebo controlled study evaluating the use of platelet-rich plasma therapy (PRP) for acute ankle sprains in the emergency department. *J Emerg Med*. 2015;49(4):546–51.
 78. Blanco-Rivera J, Elizondo-Rodríguez J, Simental-Mendía M, Vilchez-Cavazos F, Peña-Martínez VM, Acosta-Olivo C. Treatment of lateral ankle sprain with platelet-rich plasma: a randomized clinical study. *Foot Ankle Surg*. 2019;. pii: S1268-7731(18)30140-1
 79. Laver L, Carmont MR, McConkey MO, Palmanovich E, Yaacobi E, Mann G, Nyska M, Kots E, Mei-Dan O. Plasma rich in growth factors (PRGF) as a treatment for high ankle sprain in elite athletes: a randomized control trial. *Knee Surg Sports Traumatol Arthrosc*. 2015;23(11):3383–92.
 80. Samra DJ, Sman AD, Rae K, Linklater J, Refshauge KM, Hiller CE. Effectiveness of a single platelet-rich plasma injection to promote recovery in rugby players with ankle syndesmosis injury. *BMJ Open Sport Exerc Med*. 2015;1(1):e000033.
 81. Azar FM, Andrews JR, Wilk KE, Groh D. Operative treatment of ulnar collateral ligament injuries of the elbow in athletes. *Am J Sports Med*. 2000;28(1):16–23.
 82. Savoie FH III, Morgan C, Yaste J, Hurt J, Field L. Medial ulnar collateral ligament reconstruction using hamstring allograft in overhead throwing athletes. *JBJS*. 2013;95(12):1062–6.
 83. Jobe FW, Stark H, Lombardo S. Reconstruction of the ulnar collateral ligament in athletes. *J Bone Joint Surg Am*. 1986;68(8):1158–63.
 84. Dines J, Williams P, ElAttrache N, Conte S, Tomczyk T, Osbahr D, et al. Platelet-rich plasma can be used to successfully treat elbow ulnar collateral ligament insufficiency in high-level throwers. *Am J Orthop*. 2016;45(5):296–300.
 85. Podesta L, Crow SA, Volkmer D, Bert T, Yocum LA. Treatment of partial ulnar collateral ligament tears in the elbow with platelet-rich plasma. *Am J Sports Med*. 2013;41(7):1689–94.
 86. McQueen PD, Camp CL, Chauhan A, Erickson BJ, Potter HG, D'Angelo J, et al. Comparative analysis of the nonoperative treatment of elbow ulnar collateral ligament injuries in professional baseball players with and without platelet-rich plasma. *Orthop J Sports Med*. 2018;6(7_suppl4):2325967118S00086.
 87. Kato Y, Yamada S, Chavez J. Can platelet-rich plasma therapy save patients with ulnar collateral ligament tears from surgery? *Regen Ther*. 2019;10:123–6.
 88. Rodas G, Bove T, Caparrós T, Langohr K, Medina D, Hamilton B, Sugimoto D, Casals M. Ankle Sprain Versus Muscle Strain Injury in Professional Men's Basketball: A 9-Year Prospective Follow-up Study. *Orthop J Sports Med*. 2019;21;7(6):2325967119849035. <https://doi.org/10.1177/2325967119849035>.
 89. Colio S, McAuliffe M, Uribe Y, Bodor M. Regenerative medicine for muscle and ligament problems: technical aspects and evidence. *Tech Reg Anesth Pain Manag*. 2015;19(1–2):80–4.
 90. Sánchez M, Albillos J, Angulo F, Santisteban J, Andía I. Platelet-rich plasma in muscle and tendon healing. *Oper Tech Orthop*. 2012;22(1):16–24.
 91. Mishra A, Woodall J Jr, Vieira A. Treatment of tendon and muscle using platelet-rich plasma. *Clin Sports Med*. 2009;28(1):113–25.
 92. Piuze NS, Dominici M, Long M, Pascual-Garrido C, Rodeo S, Huard J, et al. Proceedings of the signature series symposium “cellular therapies for orthopaedics and musculoskeletal disease proven and unproven therapies—promise, facts and fantasy,” international society for cellular therapies, Montreal, Canada, may 2, 2018. *Cytherapy*. 2018;20(11):1381–400.
 93. Stilhano RS, Martins L, Ingham SJM, Pesquero JB, Huard J. Gene and cell therapy for muscle regeneration. *Curr Rev Musculoskelet Med*. 2015;8(2):182–7.
 94. Terada S, Ota S, Kobayashi M, Kobayashi T, Mifune Y, Takayama K, et al. Use of an antifibrotic agent improves the effect of platelet-rich plasma on muscle healing after injury. *JBJS*. 2013;95(11):980–8.

95. Li H, Usas A, Poddar M, et al. Platelet-rich plasma promotes the proliferation of human muscle derived progenitor cells and maintains their stemness. *PLoS One*. 2013;8(6):e64923.
96. Artaza JN, Bhasin S, Magee TR, et al. Myostatin inhibits myogenesis and promotes adipogenesis in C3H 10T(1/2) mesenchymal multipotent cells. *Endocrinology*. 2005;146(8):3547–57.
97. Burks TN, Cohn RD. Role of TGF- β signaling in inherited and acquired myopathies. *Skelet Muscle*. 2011;1(1):19.
98. Delos D, Leineweber MJ, Chaudhury S, Alzoobae S, Gao Y, Rodeo SA. The effect of platelet-rich plasma on muscle contusion healing in a rat model. *Am J Sports Med*. 2014;42(9):2067–74.
99. Quarteiro ML, Tognini JRF, ELFd O, Silveira I. The effect of platelet-rich plasma on the repair of muscle injuries in rats. *Rev Bras Ortop*. 2015;50(5):586–95.
100. Wright-Carpenter T, Opolon P, Appell H, Meijer H, Wehling P, Mir L. Treatment of muscle injuries by local administration of autologous conditioned serum: animal experiments using a muscle contusion model. *Int J Sports Med*. 2004a;25(08):582–7.
101. Hammond JW, Hinton RY, Curl LA, Muriel JM, Lovering RM. Use of autologous platelet-rich plasma to treat muscle strain injuries. *Am J Sports Med*. 2009;37(6):1135–42.
102. Dragoo JL. The use of platelet-rich and platelet-poor plasma to enhance differentiation of skeletal myoblasts: implications for the use of autologous blood products for muscle regeneration. *Orthop J Sports Med*. 2016;4(7_suppl4):2325967116S00150.
103. Miroshnychenko O, Chang WT, Dragoo JL. The use of platelet-rich and platelet-poor plasma to enhance differentiation of skeletal myoblasts: implications for the use of autologous blood products for muscle regeneration. *Am J Sports Med*. 2017;45(4):945–53.
104. Wright-Carpenter T, Klein P, Schäferhoff P, Appell H, Mir L, Wehling P. Treatment of muscle injuries by local administration of autologous conditioned serum: a pilot study on sportsmen with muscle strains. *Int J Sports Med*. 2004b;25(08):588–93.
105. Rossi LA, Rómoli ARM, Altieri BAB, Flor JAB, Scordo WE, Elizondo CM. Does platelet-rich plasma decrease time to return to sports in acute muscle tear? A randomized controlled trial. *Knee Surg Sports Traumatol Arthrosc*. 2017;25(10):3319–25.
106. Jackson TJ, Starkey C, McElhiney D, Domb BG. Epidemiology of hip injuries in the National Basketball Association: a 24-year overview. *Orthop J Sports Med*. 2013;1(3):2325967113499130.
107. Hamid MS, Mohamed Ali MR, Yusof A, George J, Lee LPC. Platelet-rich plasma injections for the treatment of hamstring injuries: a randomized controlled trial. *Am J Sports Med*. 2014;42(10):2410–8.
108. Bezuglov E, Maffulli N, Tokareva A, Achkasov E. Platelet-rich plasma in hamstring muscle injuries in professional soccer players. A pilot study. *Muscles Ligaments Tendons J*. 2019;9(1).
109. Reurink G, Goudswaard GJ, Moen MH, Weir A, Verhaar JA, Bierma-Zeinstra SM, et al. Platelet-rich plasma injections in acute muscle injury. *N Engl J Med*. 2014;370(26):2546–7.
110. Zanon G, Combi F, Combi A, Peticarini L, Sammarchi L, Benazzo F. Platelet-rich plasma in the treatment of acute hamstring injuries in professional football players. *Joints*. 2016;4(01):017–23.
111. Rettig AC, Meyer S, Bhadra AK. Platelet-rich plasma in addition to rehabilitation for acute hamstring injuries in NFL players: clinical effects and time to return to play. *Orthop J Sports Med*. 2013;1(1):2325967113494354.
112. Hamilton B, Tol JL, Almusa E, Boukarroum S, Eirale C, Farooq A, et al. Platelet-rich plasma does not enhance return to play in hamstring injuries: a randomised controlled trial. *Br J Sports Med*. 2015;49(14):943–50.
113. Guillodo Y, Madouas G, Simon T, Le Dauphin H, Saraux A. Platelet-rich plasma (PRP) treatment of sports-related severe acute hamstring injuries. *Muscles Ligaments Tendons J*. 2015;5(4):284.



The Role of Orthobiologics in the Management of Cartilage and Meniscal Injuries in Sports

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47.1 Cartilage Injuries

47.1.1 Introduction

Articular cartilage injuries in the athlete represent a significant source of pain and disability, resulting in time lost from play and predisposing athletes to early joint degeneration and shortened athletic careers [1, 2]. Athletes competing in sports requiring repetitive, high-impact loading such as basketball are particularly susceptible to chondral injuries secondary to acute traumatic episodes or repeated loading over time. A systematic review of 11 studies, comprising 931

American basketball, football, and endurance running athletes reported chondral defects to be present in up to 36% of knees [3]. Combined with the rising incidence of athletic participation at all levels of competition, a growing incidence of sports-related injuries to the articular cartilage has been reported [4–6], with a consequential increase in surgical procedures being performed annually for chondral injuries [7–10].

Particularly in basketball players, the integrity of articular cartilage is essential to optimize joint motion and minimize friction while providing support for the mechanical joint stresses placed across the knees during jumping, running, and cutting [11]. However, due to the poor inherent healing capacity of cartilage, secondary to the aneural, avascular nature of chondrocytes, the intrinsic potential for repair is minimal, with injured cartilage substituted by fibrocartilage [12–16]. Moreover, continued loading on injured cartilage has been shown to result in the accumulation of degradative enzymes and cytokines, leading to disruption of the collagen ultrastructure, resulting in further chondral damage [17–19]. As such, articular cartilage injuries represent a therapeutic challenge in the athlete due to the high functional demands placed on the articular surfaces and the athlete's desire to return to the same or higher levels of competition following injury [20].

The goal of returning athletes to pre-injury levels quickly following chondral injuries while minimizing the risk for development of long-term

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chondral degeneration has led to increased interest in the use of minimally invasive treatment options [21–24]. Specifically, increased attention has focused on the use of orthobiologics both as an isolated, nonoperative treatment modality and as an adjunct therapy during operative cartilage restoration procedures to promote cartilage healing and regeneration [6, 20, 22, 24]. Orthobiologics are defined as naturally occurring substances in the human body used to improve healing of injured cartilage, muscle, tendon, ligaments, and fractures [16]. Orthobiologics currently utilized for the treatment of sport-related chondral injuries include platelet-rich plasma (PRP), bone marrow aspirate concentrate (BMAC), and mesenchymal stem cells (MSC). The purpose of this chapter is to review the current literature on the use of orthobiologics in the treatment of articular cartilage injuries in the athlete.

47.2 Platelet-Rich Plasma

The use of PRP in isolation or as an adjunct to surgery for the treatment of sports-related chondral injuries in the athlete has gained significant interest in recent years [25–28]. PRP is defined as harvested autologous biological blood, concentrated via centrifuge to contain 1.5–2 to around a ninefold increase in platelet concentration compared with baseline endogenous serum levels [29, 30] or more than one million platelets per milliliter of serum although these values may not be agreeable by all, and reports of higher concentrations exist as well as commercially available products [31, 32] (Figs. 47.1 and 47.2). Injection of PRP leads to platelet activation, while also stimulating the release of various growth factors and cytokines [22, 30]. These biologic mediators include vascular endothelial growth factor (VEGF), platelet-derived growth factor (PDGF), fibroblast growth factor (FGF), insulin-like growth factor-1 (IGF-1), interleukin-1 β , interleukin-10, and tumor necrosis factor- β [33]. These growth factors and cytokines have been shown to activate biologic pathways, providing anti-inflammatory effects, while stimulating matrix synthesis, endothelial growth, angiogene-



Fig. 47.1 Platelet-rich plasma (PRP) before centrifugation



Fig. 47.2 Platelet-rich plasma (PRP) after centrifugation

sis, collagen synthesis, cell proliferation, and cell differentiation to initiate tissue healing [15, 16, 22, 34–37]. PRP preparations are traditionally divided based on leukocyte concentration, separated into leukocyte-rich PRP (LR-PRP) and leukocyte-poor PRP (LP-PRP) preparations [18, 38]. Prior studies have shown that LR-PRP creates a less ideal environment for chondral repair and detrimental to clinical outcomes through the expression of pro-inflammatory markers and catabolic cytokines [39, 40].

The widespread use of PRP, coupled with the relative ease and safety of obtaining autologous PRP with little risk to the patient, have made the use of PRP an attractive option for the management of cartilage injuries [33, 41]. However, there remains limited evidence on the efficacy of PRP for the treatment of cartilage injuries, particularly in the athlete. As such, while the majority of clinical investigations have examined outcomes associated with PRP use in non-athletes, multiple investigations have reported superior outcomes associated with the use of PRP treatment in younger patients with less severe degenerative chondral changes in the knee, comparable to the athletic population. Kon et al. compared 50 patients with symptomatic knee osteoarthritis (OA) undergoing three PRP injections to 100 patients treated with either high-molecular-weight hyaluronic acid (HA) or low-molecular-weight HA. The authors found that PRP provided improved outcomes and a longer duration of efficacy in reducing pain and symptoms based on visual analog scale (VAS) and International Knee Documentation Committee (IKDC) scores [42]. When analyzed based on patient age and degree of osteoarthritic changes, superior results were reported in younger and more active patients with lower degrees of cartilage degeneration. Cole et al. similarly reported in their double-blind, randomized controlled trial of patients with unilateral knee OA comparing LP-PRP to HA injections that patients with mild OA and lower body mass index experienced significantly better outcomes [43]. Meanwhile, the systematic review by Campbell et al. examining three meta-analyses comparing the use of PRP injection to the knee versus corticosteroids, HA,

oral nonsteroidal anti-inflammatory drugs, or placebo found increased benefit for using PRP with reduced pain, improved range of motion, and quality of life in patients with focal chondral defects and early mild to moderate osteoarthritis [44]. Moreover, assessing athletes at the end of their career with chronic knee pain secondary to degenerative chondral lesions of the knee, Papalia et al. analyzed 48 professional soccer athletes randomized into two groups receiving either three injections of hybrid HA (HHA; $n = 24$ athletes) or three injections of PRP ($n = 23$ athletes) [45]. While athletes in the HHA group demonstrated significantly superior results when compared to the PRP group at 3 and 6 months follow-up, no significant differences in outcomes were reported by 12 months.

Fact Box

Multiple investigations examining outcomes following the use of PRP for the treatment of articular cartilage injuries have reported improved outcomes in younger patients with lower degrees of cartilage degeneration [46–48].

Few studies have examined the impact of PRP for the treatment of chondral lesions about the hip, with limited data in athletic patients. Dallari et al. examined 111 patients aged 18–65 years old, randomized to three groups, receiving three weekly injections of either PRP, PRP + HA, or HA [49]. The authors reported that patients receiving PRP alone had lower VAS pain scores at 2, 6, and 12 months follow-up, along with significantly better Western Ontario and McMaster Universities Arthritis Index (WOMAC) scores at 2 and 6 months. Meanwhile, Battaglia et al. performed a non-blinded, randomized trial comparing PRP versus HA in 100 consecutive patients with hip OA. Harris Hip Score (HHS) and VAS pain scores were found to be significantly improved between 1 month and 3 months follow-up in both groups. However, progressive worsening of symptoms was reported between 6 months

and 12 months follow-up despite scores remaining significantly improved when compared to preoperative values [50]. Moreover, no significant differences were found between the PRP or HA groups at any time point, while a significant association was appreciated between higher Kellgren-Lawrence (K-L) OA grade (Grade IV) and VAS score over the course of the investigation. As such, the use of PRP for chondral lesions affecting the hip of the athlete remains largely unknown and warrants further investigation.

In addition to the lack of high-quality studies examining the efficacy of PRP in athletes with chondral injuries, a major limitation to the use of PRP for the treatment of cartilage defects remains the lack of standardization and variability in PRP preparation techniques. The development of proper terminology to describe and classify the many different available PRP products and the variability in their characteristics is essential, especially when comparing results between various studies and analyzing the benefits of such treatments. The need for improved terminology, categorization, and classification has emerged in recent years with the growing number of reported studies using various PRP products and resulted in several classification systems. The first described and most comprehensive classification system is the Dohan Ehrenfest classification [32], which is based on cell content (mostly leukocytes) and fibrin architecture. Four main families were defined in this classification: Pure platelet-rich plasma (P-PRP)—or leukocyte-poor platelet-rich plasma (LP-PRP)—products are preparations without leukocytes and with a low-density fibrin network after activation; leukocyte- and platelet-rich plasma (L-PRP) products are preparations with leukocytes and with a low-density fibrin network after activation; pure platelet-rich fibrin (P-PRF)—or leukocyte-poor platelet-rich fibrin—are preparations without leukocytes and with a high-density fibrin network; and leukocyte- and platelet-rich fibrin (L-PRF) products are preparations with leukocytes and with a high-density fibrin network. Two other classification systems were proposed in recent years,

which were more directed toward sports medicine applications. These are the Mishra classification and the PAW (platelet, activation, white cells) classification [34, 51]. Mishra et al. [51] proposed a classification that takes into consideration the presence of leukocytes, activation of platelets, and platelets concentration.

This classification established four types of PRP: an L-PRP solution (type 1 PRP), an L-PRP gel—with activation (type 2 PRP), a P-PRP solution (type 3 PRP), and a P-PRP gel—with activation (type 4 PRP). Each type can be described as an A or B subtype, with the A subtype standing for $\geq X5$, the blood concentration of platelets, and the B subtype standing for $< X5$, the blood concentration of platelets. The PAW classification [34] has similarities with the Mishra classification and is based on the absolute number of platelets, the manner in which platelet activation occurs, and the presence or absence of white cells. Different concentrations of platelets, leukocytes, and other growth factors in the final PRP preparation have been reported due to the availability of various commercial PRP preparation systems, resulting in wide variability in the contents of the final PRP product [25, 47, 52]. This inconsistency may account for the variable and oftentimes conflicting results among studies [25, 53, 54]. Moreover, lack of transparency and detail in reporting preparation techniques in many studies has made comparisons between studies difficult [55]. Further questions regarding the number of injections, timing of doses, use of LP-PRP versus LR-PRP, and the volume of injection require further studies to establish a gold standard protocol for the preparation and use of PRP in athletes [27, 33, 54].

Fact Box

There remains substantial discrepancy in PRP preparation techniques, injection contents, and delivery methods, warranting additional studies to better define the optimal treatment protocol for use in the athlete [16, 18, 30, 49, 50, 56].

Many more preparations are being investigated for their efficacy in cartilage injuries such as PRP-conjugate preparations and autologous conditioned serum (ACS). There is recent interest in creating PRP conjugates with other biologics (such as hyaluronic acid) to enhance healing through the properties of both materials [31, 57, 58]. However, there is no sufficient evidence to support its efficacy in athletes. ACS products such as Regenokine (marketed in the US) and Orthokine (marketed in Europe) have shown some success in the management of cartilage pathology. Although, most studies have reported efficacy in osteoarthritis [38, 59, 60], ACS is commonly used in athletes, particularly in Europe.

47.3 Bone Marrow Aspirate Concentrate

The popularity of BMAC has recently increased due to BMAC being one of the few procedures approved by the Federal and Drug Administration (FDA) for intra-articular, single-step delivery of MSCs [48]. In addition to MSCs, BMAC possesses hematopoietic stem cells, endothelial progenitor stem cells, and PDGFs that have been shown to improve tissue healing [61]. Despite only accounting for 0.001–0.01% of nucleated cells in standard BMAC injections [62], MSCs possess strong inherent regenerative properties. BMAC can presumably assist in the treatment of articular cartilage injuries, due to its regenerative potential, and ability to modulate the immune system via enhanced secretion of growth factors and cytokines [63–65]. The contents of BMAC have been shown to signal surrounding tissues to secrete growth factors and cytokines, including VEGF, PDGF, transforming growth factor-beta (TGF- β), bone morphogenetic protein (BMP)-2 and BMP-7, which are present in higher quantities when compared to PRP [22, 66]. These biologic modulators have been linked to chondrocyte proliferation, MSC differentiation, wound healing, as well as the suppression of potentially detrimental pro-inflammatory cytokines [67].

Clinically, BMAC augmentation has been shown to play a role in regenerating more hyaline-like repair tissue, improving patient-reported outcomes, and improving radiographic evidence of healing [68]. However, no current investigation has focused on the use of BMAC specifically in athletes. Chahla et al. reported in their systematic review that despite the lack of high-quality studies examining the use of BMAC for the treatment of early-onset OA, BMAC injection was a safe procedure with few reported adverse effects [66]. However, varying degrees of beneficial results with respect to the effect of BMAC for the treatment of chondral defects and early OA were reported, due to the high number of patients treated with and without an additional procedure. Meanwhile, Kim et al. reported outcomes of BMAC injection with adipose tissue in a case series of 75 knees ($n = 41$ patients) with knee OA (K-L grades I–IV) [69]. While statistical significance was not reported, VAS pain score, IKDC, short-form (SF)-36, Knee Injury and Osteoarthritis Outcome Score (KOOS), and Lysholm scores were found to be increased when compared to preoperative values by 12 months. Moreover, the authors noted a significant association between higher K-L grade and inferior clinical outcomes at final follow-up ($p = 0.02$).

Fact Box

Results following BMAC treatment for articular cartilage injuries have demonstrated improved outcomes in patients with lower grades of knee degeneration [60].

When utilized as a surgical adjunct, Gobbi et al. examined 15 patients with International Cartilage Repair Society (ICRS) grade IV knee chondral lesions (average size, 9.2 cm [4]) undergoing operative transplantation with BMAC covered with a collagen I/III matrix [70]. At 24 months, significant improvements in VAS pain score, IKDC, KOOS, Lysholm, Marx, SF-36, and Tegner scores were reported when compared to preoperative values. The presence of

hyaline-like tissue over the lesions was also reported based on magnetic resonance imaging (MRI) and histologic evaluation. Overall, superior outcomes were reported in patients with solitary cartilage defects and in patients with small lesions. Gobbi et al. further reported in their prospective cohort study of patients with ICRS grade 4 chondral lesions of the knee treated with HA-based scaffolds soaked in BMAC that clinical outcomes were correlated with the size of the chondral lesion treated [13]. Specifically, significantly better subjective IKDC scores and a trend toward a significantly better KOOS pain scores were found in patients with lesions smaller than 8 cm [4] compared to those with lesions larger than 8 cm [4] at final follow-up. For chondral defects of the talus, Giannini et al. compared the use of a single injection of BMAC ($n = 25$ cases) versus open autologous chondrocyte implantation (ACI) ($n = 10$ cases) versus arthroscopic ACI ($n = 46$ cases) in 81 patients with a mean age of 30 ± 8 years [71]. At second look arthroscopy with biopsy at 12 months, no significant difference in change in American Orthopaedic Foot and Ankle Society (AOFAS) score was appreciated between the three groups. However, BMAC was noted to permit a marked reduction in procedure morbidity and costs as a “one-step” procedure.

A recent systematic review by Migliorini et al. reported improved outcomes in patients receiving MSCs injections for knee osteoarthritis with 12 months follow-up. They included 18 studies and 1069 treated knees. Average age of patients was 57 years old. They reported improvement in patient-reported outcomes and a 12.7% local complications rate [15].

Hede et al. evaluated the clinical outcomes of ten patients treated with a one-step procedure using autologous BMAC and PRP on a collagen scaffold for large full-thickness cartilage lesions of the knee. They reported an increase in clinical outcome scores and pain scores at 1 and 2 years postoperatively. However, they also found that MRI and histology (from second-look arthroscopy that was performed in seven patients) have demonstrated repair tissue inferior to native hyaline cartilage [21].

Fact Box

When used as an adjunct during operative management, improved outcomes have been reported in patients with isolated chondral lesions or lesions measuring less than 8cm^2 [2, 32].

Similar to PRP, the current literature examining the utilization of BMAC for athletic injuries remains lacking in high-quality studies focusing on athletes, as there remains limited evidence supporting the efficacy of the BMAC product, while standardized guidelines for preparation remain limited [16]. In addition, the ideal harvest site and technique, carrier for BMAC, number of BMAC treatments, injection timing, and volume remain poorly characterized [35].

47.4 Mesenchymal Stem Cells

MSCs have been shown to possess high plasticity while being immune-suppressive, anti-inflammatory, and capable of self-renewal. MSCs are known to produce proteins conducive to cartilage regeneration, making them perhaps the most promising stem cell option for articular cartilage repair [12, 35]. MSCs are present and can be harvested from various adult tissues, including bone marrow, peripheral blood, adipose tissue, and synovium. Recently, adipose-derived stem cells (ASCs) have gained increased popularity due to the ease of accessibility and harvest from liposuction aspirate or from the infrapatellar fat pad, resulting in minimal morbidity [37, 72]. Moreover, ASCs have been shown to possess up to 300-fold more stem cells per volume when compared to BMAC [9, 73] while maintaining their phenotype better over culture passages when compared with bone-marrow-derived MSCs [63, 72, 74].

There are currently few studies examining the use of MSCs for the treatment of chondral injuries in athletes. However, MSCs have been shown to possess chondro-inductive properties in vitro, capable of inducing chondrocyte

proliferation and extracellular matrix production [75], resulting in encouraging clinical results and pain reduction without significant complications. A systematic review by Chahla et al. identified six studies examining intra-articular injections of stem cells within the knee for the treatment of cartilage injuries [75]. While no studies commented on the use of MSCs for athletes, all studies were noted to report improvement for patients with OA and focal chondral defects without significant adverse events. However, the authors noted that reported improvements were modest and that the presence of a placebo effect could not be ruled out. The retrospective cohort study by Kim et al. examined 20 patients with knee OA treated with MSC injection combined with PRP versus a pair-matched cohort of patients undergoing MSC implantation using a fibrin glue scaffold [23]. At a mean follow-up of 28.6 months, the authors reported significant improvement in IKDC and Tegner activity score in both groups compared to preoperative values, with significantly higher IKDC scores in the implantation group. Moreover, Nejadnik et al. compared outcomes between patients with chondral defects undergoing repair using ACI ($n = 36$) or bone-marrow-derived MSCs ($n = 36$), with outcomes measured at 3, 6, 9, 12, 18, and 24 months following treatment [76]. No significant differences between groups were reported based on Lysholm, IKDC, or Tegner activity scores, highlighting the effectiveness of bone-marrow-derived MSCs for focal cartilage lesions. Meanwhile, arthroscopic implantation of synovial MSCs in 10 patients with single chondral lesions of the femoral condyles was found to improve MRI features score, qualitative histology, and Lysholm score, but no improvement in Tegner activity level was reported [77]. Kyriakidis et al. have recently published a study on 25 patients undergoing treatment with ASCs implantation for focal cartilage defects of the knee with a 3-year follow-up [78]. Patient-reported outcomes significantly improved ($p < 0.05$), including IKDC, KOOS, Tegner, and VAS. Interestingly, histological analysis from two patients who underwent post-operative biopsies demonstrated the presence of

hyaline-like tissue. Bastos et al. performed a randomized, controlled, and double-blinded study assessing the efficacy of culture-expanded MSCs injection with or without PRP in patients with knee osteoarthritis. They enrolled 47 patients in three groups, MSCs ($N = 16$), MSCs + PRP ($N = 14$), and corticosteroids ($N = 17$). They reported an improvement in most KOOS domains and global scores for the three groups in 1 and 12 months ($p < 0.05$). At 12-month follow-up, the corticosteroids group only showed significant improvement in the pain and function sub-scores, while the MSCs and the MSCs+PRP groups showed improvement in all KOOS domains and global scores (except quality of life for the MSCs+PRP group) [46].

Further research on the indication, safety, and efficacy of MSCs, particularly ASCs, for the treatment of athletic chondral-related injuries are warranted. Most current studies lack a control group, while in many other studies, additional therapeutic interventions and orthobiologics have been simultaneously added, preventing an accurate understanding of the contribution of MSCs to chondral healing. Similar to other orthobiologics, additional studies are necessary to better understand optimal harvest location, culture methods, cell concentration, and transplantation method for the treatment of cartilage injuries in athletes [77].

47.5 Meniscal Injuries

There are several inherent factors that create an unfavorable environment for healing of a meniscus tear. These include the avascular nature of the meniscus, the presence of synovial fluid and pro-inflammatory cytokines, and the repetitive load on the meniscus, which is virtually unavoidable. The avascular nature of the meniscus poses a significant challenge for meniscus tear healing. As demonstrated by Arnoczky and Warren, only the peripheral 10–30% of the meniscus is vascularized [79]. Furthermore, the presence of synovial fluid and proinflammatory cytokines has been shown to have a catabolic effect on meniscal healing [80].

In recent years, there has been growing interest in the role of orthobiologics in the treatment of meniscal pathology. There are several modalities for biologic augmentation of meniscal repair, including use of a fibrin clot, cytokines and growth factors, PRP, and cell-based therapies. Although most studies were focused on augmentation of meniscal repair, several studies have also assessed the efficacy of orthobiologics injections as a sole treatment for meniscal tears. Wei et al. hypothesized that PRP can enhance the healing of white on white meniscal tears [81]. Shin et al. have studied the effect of LR-PRP on healing of a horizontal medial meniscus tear in a rabbit model and found no significant differences in meniscal healing between the LR-PRP group and controls [82]. Ishida et al. reported an in-vitro and an in-vivo study in a rabbit model demonstrating increased healing with filling of the meniscal defect with a gelatin hydrogel delivery system for PRP [83]. Betancourt and Murrell presented a case of a 29-year-old woman improving after treatment with LP-PRP injections for a meniscal tear [84]. Blanke et al. reported on the use of percutaneous PRP injections of intrasubstance meniscal tears (grade 2) in ten recreational athletes. The injections were aimed at the affected site with the use of fluoroscopy guidance. Each patient received three sequential injections in a 7-day interval. Six patients (60%) showed improvement in outcomes and increased sports activity [85]. Literature on the effect of other injectables is limited. However, of note, Pak et al. have presented a case of a patient treated successfully with an ASC percutaneous injection [10].

47.6 Augmentation of Meniscal Repair

A recent randomized controlled trial investigated PRP augmentation of repaired vertical tears as compared to isolated suture repair. The results were favorable, with statistically significant functional outcome improvement, lower failure rates, and better healing on second look arthroscopy at 42 months after surgery in PRP-augmented

repairs [14]. Another recent study by Everhart et al. found that PRP augmentation of isolated meniscus repairs resulted in significantly decreased failure rates at 3 years after surgery [86]. However, PRP augmentation of meniscus repairs with concomitant ACL reconstruction was found to have no difference in failure rates when compared to controls at the same time period. A number of smaller studies with lower levels of evidence have produced more mixed results, with some showing modest benefits in functional outcomes while others finding no benefits when compared to placebo [24, 56, 87, 88]. While promising, there remains significant heterogeneity in PRP preparation techniques as well as the type of tear being repaired, and future well-designed studies are needed to corroborate these early findings.

Fibrin clot augmentation has also proven to be an efficacious adjunct to meniscus repairs in several investigations. In an early investigation of five patients with complete radial tears of the posterolateral aspect of the lateral meniscus traditionally treated with meniscectomy, van Trommel et al. completed suture repairs enhanced with a fibrin clot [89]. On MRI assessment of three of the five patients at an average of 71 months after surgery, all menisci were completely healed with no evidence of degenerative change. Recently Ra et al. also utilized fibrin clot augmentation of complete radial tear repairs in 12 patients, which similarly results in excellent rates of healing and functional outcome improvement [90]. Fibrin clots have also been investigated in the repair of horizontal cleavage tears, with significant functional outcome improvement, but only a 70% healing rate on second look arthroscopy at 12 months after repair [36]. As with PRP augmentation, future large and well-designed studies are needed to confirm the preliminary benefits of fibrin clots in the healing of meniscus repairs.

Additionally, the use of BMAC in meniscus repair surgery has shown promise. In a basic science study using a rabbit model with avascular meniscal lesions, Koch et al. found that BMAC augmentation demonstrated macroscopic and histologic evidence of superior healing when

compared to PRP and no augmentation of suture repaired meniscus lesions at 6 and 12 weeks after surgery [91]. Another recent investigation by Piontek et al. utilized bone marrow aspirate injection and collagen wrapping of repaired meniscus lesions, finding favorable functional and radiographic outcome improvements at 2 years after surgery [92]. While preliminary, bone-marrow-derived augmentation techniques are being actively studied, with several ongoing clinical trials currently investigating BMAC and meniscus repair.

47.7 Conclusion

The popularity of orthobiologics for the use of cartilage and meniscal injuries in sports continues to increase. Current treatment guidelines for the use of orthobiologics for cartilage defects and meniscal tears based on type, size, location, and defect severity while accounting for patient's age, activity level, and desire to return to competition remain poorly characterized, requiring further research to define an optimal treatment algorithm. Such algorithm should also differentiate between the efficacy of orthobiologics for focal defects, osteochondritis dissecans (OCD), and osteoarthritis, as these pathologies may defer in many aspects. Despite most investigations reporting orthobiologics to be safe with few serious adverse effects, inconsistent and at times conflicting data has been reported. The discrepancy in outcomes requires standardization for orthobiologic processing while defining the optimal contents of orthobiologic preparations to allow for reliable comparison among studies. By defining a standard procedure, future basic science and clinical research utilizing well-designed randomized controlled trials are warranted to determine the long-term impact of chondral injuries in the knee of the athlete. Meanwhile, establishing short- and medium-term data for injuries within the hip and ankle is necessary to better understand the role of orthobiologics as a minimally invasive individual treatment or adjunct during operative intervention. Such limitations must be weighed against the popularity of ortho-

biologics in their use for the treatment of articular chondral and meniscal injuries in the athlete.

Take-Home Message

Despite the increased popularity of orthobiologics for the treatment of articular cartilage injuries, further investigations standardizing treatment preparation, contents, and protocols are necessary to better understand the efficacy and long-term effects of the use of orthobiologics in the athlete.

The role of orthobiologics in meniscal injuries is less understood, and current evidence does not allow making recommendations regarding the use of orthobiologics in meniscal injuries.

References

1. Drawer S, Fuller CW. Propensity for osteoarthritis and lower limb joint pain in retired professional soccer players. *Br J Sports Med.* 2001;35(6):402–8.
2. Engstrom B, Forssblad M, Johansson C, Tornkvist H. Does a major knee injury definitely sideline an elite soccer player? *Am J Sports Med.* 1990;18(1):101–5.
3. Flanigan DC, Harris JD, Trinh TQ, Siston RA, Brophy RH. Prevalence of chondral defects in athletes' knees: a systematic review. *Med Sci Sports Exerc.* 2010;42(10):1795–801.
4. Arendt E, Dick R. Knee injury patterns among men and women in collegiate basketball and soccer. NCAA data and review of literature. *Am J Sports Med.* 1995;23(6):694–701.
5. Jones SJ, Lyons RA, Sibert J, Evans R, Palmer SR. Changes in sports injuries to children between 1983 and 1998: comparison of case series. *J Public Health Med.* 2001;23(4):268–71.
6. Mithofer K, Peterson L, Mandelbaum BR, Minas T. Articular cartilage repair in soccer players with autologous chondrocyte transplantation: functional outcome and return to competition. *Am J Sports Med.* 2005;33(11):1639–46.
7. Ciccotti MC, Kraeutler MJ, Austin LS, et al. The prevalence of articular cartilage changes in the knee joint in patients undergoing arthroscopy for meniscal pathology. *Arthroscopy.* 2012;28(10):1437–44.
8. McCormick F, Harris JD, Abrams GD, et al. Trends in the surgical treatment of articular cartilage lesions in the United States: an analysis of a large private-payer database over a period of 8 years. *Arthroscopy.* 2014;30(2):222–6.

9. Oedayrajsingh-Varma MJ, van Ham SM, Knippenberg M, et al. Adipose tissue-derived mesenchymal stem cell yield and growth characteristics are affected by the tissue-harvesting procedure. *Cytherapy*. 2006;8(2):166–77.
10. Pak J, Lee JH, Lee SH. Regenerative repair of damaged meniscus with autologous adipose tissue-derived stem cells. *Bio Med Res Int* 2014;2014.
11. Sakata R, Iwakura T, Reddi AH. Regeneration of articular cartilage surface: morphogens, cells, and extracellular matrix scaffolds. *Tissue Eng Part B Rev*. 2015;21(5):461–73.
12. Chang YH, Liu HW, Wu KC, Ding DC. Mesenchymal stem cells and their clinical applications in osteoarthritis. *Cell Transplant*. 2016;25(5):937–50.
13. Gobbi A, Scotti C, Karnatzikos G, Mudhigere A, Castro M, Peretti GM. One-step surgery with multipotent stem cells and Hyaluronan-based scaffold for the treatment of full-thickness chondral defects of the knee in patients older than 45 years. *Knee Surg Sports Traumatol Arthrosc*. 2017;25(8):2494–501.
14. Kaminski R, Kulinski K, Kozar-Kaminska K, et al. A prospective, randomized, double-blind, parallel-group, placebo-controlled study evaluating meniscal healing, clinical outcomes, and safety in patients undergoing meniscal repair of unstable, complete vertical meniscal tears (bucket handle) augmented with platelet-rich plasma. *Biomed Res Int*. 2018;2018:9315815.
15. Migliorini F, Rath B, Colarossi G, et al. Improved outcomes after mesenchymal stem cells injections for knee osteoarthritis: results at 12-months follow-up: a systematic review of the literature. *Arch Orthop Trauma Surg*. 2019;1–16.
16. Moatshe G, Morris ER, Cinque ME, et al. Biological treatment of the knee with platelet-rich plasma or bone marrow aspirate concentrates. *Acta Orthop*. 2017;88(6):670–4.
17. Ding C, Cicuttini F, Scott F, Cooley H, Boon C, Jones G. Natural history of knee cartilage defects and factors affecting change. *Arch Intern Med*. 2006;166(6):651–8.
18. Lohmander LS, Roos H, Dahlberg L, Hoerner LA, Lark MW. Temporal patterns of stromelysin-1, tissue inhibitor, and proteoglycan fragments in human knee joint fluid after injury to the cruciate ligament or meniscus. *J Orthop Res*. 1994;12(1):21–8.
19. Mandelbaum B, Waddell D. Etiology and pathophysiology of osteoarthritis. *Orthopedics*. 2005;28(2 Suppl):s207–14.
20. Di Matteo B, El Araby MM, D'Angelo A, et al. Adipose-derived stem cell treatments and formulations. *Clin Sports Med*. 2019;38(1):61–78.
21. Hede K, Christensen BB, Jensen J, Foldager CB, Lind M. Combined bone marrow aspirate and platelet-rich plasma for cartilage repair: two-year clinical results. *Cartilage*. 2019;1947603519876329.
22. Holton J, Imam M, Ward J, Snow M. The basic science of bone marrow aspirate concentrate in chondral injuries. *Orthop Rev*. 2016;8(3):6659.
23. Kim YS, Kwon OR, Choi YJ, Suh DS, Heo DB, Koh YG. Comparative matched-pair analysis of the injection versus implantation of mesenchymal stem cells for knee osteoarthritis. *Am J Sports Med*. 2015;43(11):2738–46.
24. Pujol N, Salle De Chou E, Boisrenoult P, Beauflis P. Platelet-rich plasma for open meniscal repair in young patients: any benefit? *Knee Surg Sports Traumatol Arthrosc*. 2015;23(1):51–8.
25. Dohan Ehrenfest DM, Andia I, Zumstein MA, Zhang CQ, Pinto NR, Bielecki T. Classification of platelet concentrates (platelet-rich plasma-PRP, platelet-rich fibrin-PRF) for topical and infiltrative use in orthopedic and sports medicine: current consensus, clinical implications and perspectives. *Muscles Ligaments Tendons J*. 2014;4(1):3–9.
26. Redler LH, Thompson SA, Hsu SH, Ahmad CS, Levine WN. Platelet-rich plasma therapy: a systematic literature review and evidence for clinical use. *Phys Sportsmed*. 2011;39(1):42–51.
27. Sanchez M, Anita E, Orive G, Mujika I, Andia I. Platelet-rich therapies in the treatment of orthopaedic sport injuries. *Sports Med*. 2009;39(5):345–54.
28. Xie X, Ulici V, Alexander PG, Jiang Y, Zhang C, Tuan RS. Platelet-rich plasma inhibits mechanically induced injury in chondrocytes. *Arthroscopy*. 2015;31(6):1142–50.
29. Deal JB, Smith E, Heard W, O'Brien MJ, Savoie FH 3rd. Platelet-rich plasma for primary treatment of partial ulnar collateral ligament tears: MRI correlation with results. *Orthop J Sports Med*. 2017;5(11):2325967117738238.
30. Nguyen RT, Borg-Stein J, McInnis K. Applications of platelet-rich plasma in musculoskeletal and sports medicine: an evidence-based approach. *PM R*. 2011;3(3):226–50.
31. Chen W-H, Lo W-C, Hsu W-C, et al. Synergistic anabolic actions of hyaluronic acid and platelet-rich plasma on cartilage regeneration in osteoarthritis therapy. *Biomaterials*. 2014;35(36):9599–607.
32. Ehrenfest DMD, Rasmusson L, Albrektsson T. Classification of platelet concentrates: from pure platelet-rich plasma (P-PRP) to leucocyte-and platelet-rich fibrin (L-PRF). *Trends Biotechnol*. 2009;27(3):158–67.
33. Fice MP, Miller JC, Christian R, et al. The role of platelet-rich plasma in cartilage pathology: an updated systematic review of the basic science evidence. *Arthroscopy*. 2019;35(3):961–76. e963
34. DeLong JM, Russell RP, Mazzocca AD. Platelet-rich plasma: the PAW classification system. *Arthroscopy*. 2012;28(7):998–1009.
35. Dimarino AM, Caplan AI, Bonfield TL. Mesenchymal stem cells in tissue repair. *Front Immunol*. 2013;4:201.
36. Kamimura T, Kimura M. Meniscal repair of degenerative horizontal cleavage tears using fibrin clots: clinical and arthroscopic outcomes in 10 cases. *Orthop J Sports Med*. 2014;2(11):2325967114555678.
37. LaPrade RF, Geeslin AG, Murray IR, et al. Biologic treatments for sports injuries II think tank-current con-

- cepts, future research, and barriers to advancement, part I: biologics overview, ligament injury, Tendinopathy. *Am J Sports Med.* 2016;44(12):3270–83.
38. Baltzer A, Moser C, Jansen S, Krauspe R. Autologous conditioned serum (Orthokine) is an effective treatment for knee osteoarthritis. *Osteoarthr Cartil.* 2009;17(2):152–60.
 39. Braun HJ, Kim HJ, Chu CR, Dragoo JL. The effect of platelet-rich plasma formulations and blood products on human synoviocytes: implications for intra-articular injury and therapy. *Am J Sports Med.* 2014;42(5):1204–10.
 40. Sundman EA, Cole BJ, Fortier LA. Growth factor and catabolic cytokine concentrations are influenced by the cellular composition of platelet-rich plasma. *Am J Sports Med.* 2011;39(10):2135–40.
 41. Middleton KK, Barro V, Muller B, Terada S, Fu FH. Evaluation of the effects of platelet-rich plasma (PRP) therapy involved in the healing of sports-related soft tissue injuries. *Iowa Orthop J.* 2012;32:150–63.
 42. Kon E, Mandelbaum B, Buda R, et al. Platelet-rich plasma intra-articular injection versus hyaluronic acid viscosupplementation as treatments for cartilage pathology: from early degeneration to osteoarthritis. *Arthroscopy.* 2011;27(11):1490–501.
 43. Cole BJ, Karas V, Hussey K, Pilz K, Fortier LA. Hyaluronic acid versus platelet-rich plasma: a prospective, double-blind randomized controlled trial comparing clinical outcomes and effects on intra-articular biology for the treatment of knee osteoarthritis. *Am J Sports Med.* 2017;45(2):339–46.
 44. Campbell KA, Saltzman BM, Mascarenhas R, et al. Does intra-articular platelet-rich plasma injection provide clinically superior outcomes compared with other therapies in the treatment of knee osteoarthritis? A systematic review of overlapping meta-analyses. *Arthroscopy.* 2015;31(11):2213–21.
 45. Papalia R, Zampogna B, Russo F, et al. Comparing hybrid hyaluronic acid with PRP in end career athletes with degenerative cartilage lesions of the knee. *J Biol Regul Homeost Agents.* 2016;30(4 Suppl 1):17–23.
 46. Bastos R, Mathias M, Andrade R, et al. Intra-articular injection of culture-expanded mesenchymal stem cells with or without addition of platelet-rich plasma is effective in decreasing pain and symptoms in knee osteoarthritis: a controlled, double-blind clinical trial. *Knee Surg Sports Traumatol Arthrosc.* 2019:1–11.
 47. Castillo TN, Pouliot MA, Kim HJ, Dragoo JL. Comparison of growth factor and platelet concentration from commercial platelet-rich plasma separation systems. *Am J Sports Med.* 2011;39(2):266–71.
 48. Gobbi A, Chaurasia S, Karnatzikos G, Nakamura N. Matrix-induced autologous chondrocyte implantation versus multipotent stem cells for the treatment of large patellofemoral chondral lesions: a nonrandomized prospective trial. *Cartilage.* 2015;6(2):82–97.
 49. Dallari D, Stagni C, Rani N, et al. Ultrasound-guided injection of platelet-rich plasma and hyaluronic acid, separately and in combination, for hip osteoarthritis: a randomized controlled study. *Am J Sports Med.* 2016;44(3):664–71.
 50. Battaglia M, Guaraldi F, Vannini F, et al. Efficacy of ultrasound-guided intra-articular injections of platelet-rich plasma versus hyaluronic acid for hip osteoarthritis. *Orthopedics.* 2013;36(12):e1501–8.
 51. Mishra A, Harmon K, Woodall J, Vieira A. Sports medicine applications of platelet rich plasma. *Curr Pharm Biotechnol.* 2012;13(7):1185–95.
 52. Wasterlain AS, Braun HJ, Harris AH, Kim HJ, Dragoo JL. The systemic effects of platelet-rich plasma injection. *Am J Sports Med.* 2013;41(1):186–93.
 53. Riboh JC, Saltzman BM, Yanke AB, Fortier L, Cole BJ. Effect of leukocyte concentration on the efficacy of platelet-rich plasma in the treatment of knee osteoarthritis. *Am J Sports Med.* 2016;44(3):792–800.
 54. Southworth TM, Naveen NB, Tauro TM, Leong NL, Cole BJ. The use of platelet-rich plasma in symptomatic knee osteoarthritis. *J Knee Surg.* 2019;32(1):37–45.
 55. Murray IR, Chahla J, Safran MR, et al. International expert consensus on a cell therapy communication tool: DOSES. *JBJS.* 2019;101(10):904–11.
 56. Griffin JW, Hadeed MM, Werner BC, Diduch DR, Carson EW, Miller MD. Platelet-rich plasma in meniscal repair: does augmentation improve surgical outcomes? *Clin Orthop Relat Res.* 2015;473(5):1665–72.
 57. Lee M-I, Kim J-H, Kwak H-H, et al. A placebo-controlled study comparing the efficacy of intra-articular injections of hyaluronic acid and a novel hyaluronic acid-platelet-rich plasma conjugate in a canine model of osteoarthritis. *J Orthop Surg Res.* 2019;14(1):314.
 58. Russo F, D’Este M, Vadalà G, et al. Platelet rich plasma and hyaluronic acid blend for the treatment of osteoarthritis: rheological and biological evaluation. *PLoS One.* 2016;11(6):e0157048.
 59. Arbel R. *Orthokine.* In: *Bio-orthopaedics.* Berlin: Springer; 2017. p. 561–9.
 60. Fox BA, Stephens MM. Treatment of knee osteoarthritis with Orthokine®-derived autologous conditioned serum. *Expert Rev Clin Immunol.* 2010;6(3):335–45.
 61. Yokoya S, Mochizuki Y, Natsu K, Omae H, Nagata Y, Ochi M. Rotator cuff regeneration using a bioabsorbable material with bone marrow-derived mesenchymal stem cells in a rabbit model. *Am J Sports Med.* 2012;40(6):1259–68.
 62. Kasten P, Beyen I, Egermann M, et al. Instant stem cell therapy: characterization and concentration of human mesenchymal stem cells in vitro. *Eur Cell Mater.* 2008;16:47–55.
 63. Chahla J, Mandelbaum BR. Biological treatment for osteoarthritis of the knee: moving from bench to bedside-current practical concepts. *Arthroscopy.* 2018;34(5):1719–29.
 64. Dar A, Goichberg P, Shinder V, et al. Chemokine receptor CXCR4-dependent internalization and resecretion of functional chemokine SDF-1 by bone

- marrow endothelial and stromal cells. *Nat Immunol.* 2005;6(10):1038–46.
65. Simmons PJ, Torok-Storb B. Identification of stromal cell precursors in human bone marrow by a novel monoclonal antibody, STRO-1. *Blood.* 1991;78(1):55–62.
 66. Chahla J, Dean CS, Moatshe G, Pascual-Garrido C, Serra Cruz R, LaPrade RF. Concentrated bone marrow aspirate for the treatment of chondral injuries and osteoarthritis of the knee: a systematic review of outcomes. *Orthop J Sports Med.* 2016;4(1):2325967115625481.
 67. Cotter EJ, Wang KC, Yanke AB, Chubinskaya S. Bone marrow aspirate concentrate for cartilage defects of the knee: from bench to bedside evidence. *Cartilage.* 2018;9(2):161–70.
 68. Southworth TM, Naveen NB, Nwachukwu BU, Cole BJ, Frank RM. Orthobiologics for focal articular cartilage defects. *Clin Sports Med.* 2019;38(1):109–22.
 69. Kim JD, Lee GW, Jung GH, et al. Clinical outcome of autologous bone marrow aspirates concentrate (BMAC) injection in degenerative arthritis of the knee. *Eur J Orthop Surg Traumatol.* 2014;24(8):1505–11.
 70. Gobbi A, Karnatzikos G, Scotti C, Mahajan V, Mazzucco L, Grigolo B. One-step cartilage repair with bone marrow aspirate concentrated cells and collagen matrix in full-thickness knee cartilage lesions: results at 2-year follow-up. *Cartilage.* 2011;2(3):286–99.
 71. Giannini S, Buda R, Cavallo M, et al. Cartilage repair evolution in post-traumatic osteochondral lesions of the talus: from open field autologous chondrocyte to bone-marrow-derived cells transplantation. *Injury.* 2010;41(11):1196–203.
 72. Ruetze M, Richter W. Adipose-derived stromal cells for osteoarticular repair: trophic function versus stem cell activity. *Expert Rev Mol Med.* 2014;16:e9.
 73. Aust L, Devlin B, Foster SJ, et al. Yield of human adipose-derived adult stem cells from liposuction aspirates. *Cytotherapy.* 2004;6(1):7–14.
 74. Strioga M, Viswanathan S, Darinkas A, Slaby O, Michalek J. Same or not the same? Comparison of adipose tissue-derived versus bone marrow-derived mesenchymal stem and stromal cells. *Stem Cells Dev.* 2012;21(14):2724–52.
 75. Bosetti M, Borrone A, Follenzi A, Messaggio F, Tremolada C, Cannas M. Human Lipoaspirate as autologous injectable active scaffold for one-step repair of cartilage defects. *Cell Transplant.* 2016;25(6):1043–56.
 76. Nejadnik H, Hui JH, Feng Choong EP, Tai BC, Lee EH. Autologous bone marrow-derived mesenchymal stem cells versus autologous chondrocyte implantation: an observational cohort study. *Am J Sports Med.* 2010;38(6):1110–6.
 77. Kraeutler MJ, Chahla J, LaPrade RF, Pascual-Garrido C. Biologic options for articular cartilage Wear (platelet-rich plasma, stem cells, bone marrow aspirate concentrate). *Clin Sports Med.* 2017;36(3):457–68.
 78. Kyriakidis T, Iosifidis M, Michalopoulos E, Melas I, Stavropoulos-Giokas C, Verdonk R. Good mid-term outcomes after adipose-derived culture-expanded mesenchymal stem cells implantation in knee focal cartilage defects. *Knee Surg Sports Traumatol Arthrosc.* 2019:1–7.
 79. Arnoczky SP, Warren RF. Microvasculature of the human meniscus. *Am J Sports Med.* 1982;10(2):90–5.
 80. Taylor SA, Rodeo SA. Augmentation techniques for isolated meniscal tears. *Curr Rev Musculoskelet Med.* 2013;6(2):95–101.
 81. Wei L-C, Gao S-G, Xu M, Jiang W, Tian J, Lei G-H. A novel hypothesis: the application of platelet-rich plasma can promote the clinical healing of white-white meniscal tears. *Med Sci Monit.* 2012;18(8):HY47.
 82. Shin KH, Lee H, Kang S, et al. Effect of leukocyte-rich and platelet-rich plasma on healing of a horizontal medial meniscus tear in a rabbit model. *Biomed Res Int* 2015;2015.
 83. Ishida K, Kuroda R, Miwa M, et al. The regenerative effects of platelet-rich plasma on meniscal cells in vitro and its in vivo application with biodegradable gelatin hydrogel. *Tissue Eng.* 2007;13(5):1103–12.
 84. Betancourt J, Murrell W. Leukocyte-poor platelet-rich plasma to treat degenerative meniscal tear: a case report. *J Clin Orthop Traum.* 2016;7:106–9.
 85. Blanke F, Vavken P, Haenle M, von Wehren L, Pagenstert G, Majewski M. Percutaneous injections of platelet rich plasma for treatment of intrasubstance meniscal lesions. *Musc Ligam Tend J.* 2015;5(3):162.
 86. Everhart JS, Cavendish PA, Eikenberry A, Magnussen RA, Kaeding CC, Flanigan DC. Platelet-rich plasma reduces failure risk for isolated meniscal repairs but provides no benefit for meniscal repairs with anterior cruciate ligament reconstruction. *Am J Sports Med.* 2019;47(8):1789–96.
 87. Dai WL, Zhang H, Lin ZM, Shi ZJ, Wang J. Efficacy of platelet-rich plasma in arthroscopic repair for discoid lateral meniscus tears. *BMC Musculoskelet Disord.* 2019;20(1):113.
 88. Kemmochi M, Sasaki S, Takahashi M, Nishimura T, Aizawa C, Kikuchi J. The use of platelet-rich fibrin with platelet-rich plasma support meniscal repair surgery. *J Orthop.* 2018;15(2):711–20.
 89. van Trommel MF, Simonian PT, Potter HG, Wickiewicz TL. Arthroscopic meniscal repair with fibrin clot of complete radial tears of the lateral meniscus in the avascular zone. *Arthroscopy.* 1998;14(4):360–5.
 90. Ra HJ, Ha JK, Jang SH, Lee DW, Kim JG. Arthroscopic inside-out repair of complete radial tears of the meniscus with a fibrin clot. *Knee Surg Sports Traumatol Arthrosc.* 2013;21(9):2126–30.
 91. Koch M, Hammer S, Fuellerer J, et al. Bone marrow aspirate concentrate for the treatment of avascular meniscus tears in a one-step procedure-evaluation of an in vivo model. *Int J Mol Sci.* 2019;20(5):1120.
 92. Piontek T, Ciemniowska-Gorzela K, Naczek J, et al. Complex meniscus tears treated with collagen matrix wrapping and bone marrow blood injection: a 2-year clinical follow-up. *Cartilage.* 2016;7(2):123–39.



In-Season Management of Injuries in Basketball: A Pragmatic Approach

48

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48.1 Introduction

The basketball season, especially at higher levels, can span multiple months demanding sustained physical and mental fitness from players. The medical team is a critical contributor to maintaining individual player health and maximizing performance. Presently, a reactive approach to sports medicine is no longer acceptable at any level of competition. The role of the medical team has extended to evaluation and treatment of injuries during the season along with instituting programs to maintain and boost performance longitudinally. Necessary to achieving such goals requires comprehensive programs that include pre-participation evaluations, in-season checks and balances, and end-of-season/offseason programs for recovery and advancement agendas.

Generally, the team physician is the natural professional leader of the medical team, with supporting members including the athletic trainer, strength coach, massage therapist, physiotherapist, nutritionist, and coaches. Open communication, common goals, and respect are key across the extended multidisciplinary health-care team. Treatment coordination has been linked to

decreased injury rates and more rapid return to play after injury [1, 2].

Fact Box

In-season management of injuries requires constant communication and documentation by the health-care team.

The effective management of basketball injuries has become more important as the incidence of injuries in basketball players has increased over the years as the sport has evolved into a more physical game with significant contact between players. Studies have reported an overall incidence between 1.94 and 9.9 injuries per 1000 player hours (AE) depending on the level of play and setting [3–5]. Injuries to lower limbs account for the majority of injuries, followed by head and neck, upper limb, and spine and pelvis (Table 48.1). Most injuries can be classified as acute or overuse/chronic (Table 48.2).

Recognizing the injury trends across demographics can help the medical team to prepare for, identify, and treat players. At the high school level, injuries are more common in games versus practice (3.27 game versus 1.4 practice injures per 1000 AE). Women have higher rates of injury per 1000 AE (2.08 women versus 1.82 men). The same trend was found for higher rates of injuries in games compared to practice in college basketball.

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Table 48.1 Most common injuries sustained during basketball participation

NCAA injury surveillance data	
Men	Women
Ankle sprain (26.2%)	Ankle sprain (24.6%)
Knee injury (7.4%)	Knee injury (15.9%)
Upper leg contusion (3.9%)	Concussion (6.5%)
Patella injury (3.7%)	Patella injury (2.4%)
Concussion (3.6%)	Nose fracture (1.7%)
Muscle strain lower back (2.0%)	Shoulder subluxation (1.4%)
Muscle strain pelvis/hip (1.9%)	Muscle strain lower back (1.3%)

Table 48.2 Common basketball injuries categorized as acute or chronic/overuse

Common basketball injuries	
Acute	Chronic/overuse
Ankle sprain	Patellar and quadriceps tendinopathy
Muscle strains	Patellofemoral pain
Finger injuries	Achilles tendinopathy
Knee ligament injuries	Rotator cuff tendinopathy
Shoulder instability	Lower back conditions
Contusions	Stress reactions/fractures

48.2 Preseason Preparation

In-season management of injuries begins with preseason evaluation and planning. Studies have shown that the highest rates of injury occur during the first month of the season, likely due a rapid increase in workload often in combination with a lack of adequate rehabilitation from prior injury [6]. Moreover, new players to a team/organization can lead to disruption of previous treatment or rehabilitation. Meticulous evaluation and documentation of each player should be completed prior to participation. While a comprehensive pre-season evaluation is difficult at lower levels of competition, an understanding of cardiac history, surgical history, and concussion history is necessary for each athlete. For higher levels of competition posture, muscle imbalances, core stability, joint mobility/stability, muscular flexibility, range of motion (ROM), strength, functional deficits, and agility require evaluation. Individualized programs directed toward areas of

weakness have successfully decreased injury rates in basketball players as well as athletes in similar dynamic sports [2, 7, 8]. Commercially available products are also available which can help guide initial assessment and recording [9].

Ultimately, the establishment of baseline measurements may help predict as well as possibly identify and treat in-season injuries. Changes from baseline recorded serially over the season can be a warning sign of impending decompensation, warranting intervention or actually identifying an injury. Moreover, baseline values can help frame timeline for return to play, return to play criteria, as well as provide direction for the multidisciplinary team.

48.3 In-season Management

In-season management of injuries can be complex and requires attention to multiple factors. The etiology and classification of the injury, stage of the season, long-term goals of the athlete, as well as risk for future complications should be considered by any medical provider.

Generally, injuries common to basketball participation can be categorized into acute or chronic/overuse injuries (Table 48.2) [10]. Acute injuries generally coincide with a specific event and often occur to previously normal tissue. Overuse or chronic injuries, while certainly can be linked to specific events, often onset insidiously secondary to microtrauma that overloads the capacity of the tissue to repair leading to failure of remodeling. Initial and follow-up management of acute versus chronic/overuse injuries is different, warranting distinct in-season management algorithms. Table 48.2 reviews common acute and chronic/overuse injuries related to basketball.

48.3.1 Acute Injuries

Acute injuries present with swelling, focal pain, and stiffness consistent with an inflammatory state. Secondary to the rapid onset, early diagnosis can be difficult. While an on-court examination can be helpful (before the onset of swelling and

guarding), it is usually not necessary in most cases. However, early evaluation is recommended prior to the onset of the inflammatory state. A typical physical exam can yield a diagnosis in the majority of cases although there should be a low threshold for imaging. A number of guidelines (covered in other chapters) such as the Ottawa ankle rules to help guide decision-making are available [11]. The authors of this chapter consider deformity as strict criteria for imaging, especially if reduction is required.

Fact Box

Early treatment of acute injuries to reduce swelling, bleeding, and pain is critical to decreasing time lost due to injury.

Following initial assessment, the immediate goals of treatment are to limit bleeding, swelling, control pain, and protect the involved extremity. RICE, a common acronym for rest, ice, compression, and elevation should be instituted over the first 24–72 h to reduce the acute physiologic response. The PRICE regimen geared toward lateral ankle sprains, the most common injury in basketball, is based on the same principles with the additional of protecting the involved ankle to avoid further injury [12]. Introduction of anti-inflammatory medication, electrotherapeutic modalities, and early range of motion can also be helpful. In all cases, early dialogue with the health-care team and coaching staff is paramount to provide a comprehensive initial approach as this can reduce time lost to injury.

Specific treatment of acute injuries varies greatly depending on the location and extent of injury following initial management. Specific recommendations for various injuries are outside the scope of this chapter; however, a few injuries common to basketball that are less often considered justify comment, given the catastrophic potential if missed. Fractures (and stress fractures) with poorer healing potential include fractures of the scaphoid [13], talus, navicular [14], and proximal fifth metatarsal fractures (Jones fractures) (Fig. 48.1) [15]. While early recognition and immobilization are paramount for the

above fractures, evidence also supports the role of surgical intervention to hasten return to play with early healing and altered bone mechanics after hardware insertion. Sesamoid stress injuries or fractures constitute another injury that requires increased awareness in basketball players, given the higher rate of occurrence, potential for missed diagnosis, and significant consequence if not treated, or treated incorrectly [16–18]. Additionally, particular orofacial injuries require prompt attention including tooth fractures with nerve root exposed that carry higher infection potential, globe injuries with visual field deficits, as well as nose and jaw fractures that can obstruct the airway [19, 20]. Specific on-court management and treatment algorithms for such injuries are discussed in a separate chapter (On-court Examination in Basketball: What the Clinician Should No't Miss).

Overall, instituting early treatment of acute injuries will significantly decrease the time lost to injury and allow quicker initiation of functional rehabilitation when appropriate. Multimodal approaches to treatment yield best results to reduce the initial inflammatory phase; however, early diagnosis and appropriate workup remain the key for reducing devastating outcomes that can occur in the acute setting.

48.3.2 Chronic Injuries

Management of chronic injuries tends to vary from acute injuries. Unlike acute injuries, there is commonly absence of a clear mechanism. As such, to effectively manage and prevent chronic/overuse injuries, it is important to understand the factors causing or predisposing to injury. Categorizing as either related to intrinsic or extrinsic factors can be helpful.

Intrinsic factors are related to the athlete such as leg length discrepancy, muscle imbalance, poor flexibility, mal-alignment, and reduced physical fitness. Evaluation of the specific athlete including a thorough physical exam and functional testing can help to identify contributing intrinsic factors. For example, tight hamstrings along with weak quadriceps and hip musculature have been linked to anterior knee pain [21].



Fig. 48.1 Fifth metatarsal fracture treated with open reduction internal fixation to allow earlier return to play in a collegiate athlete

Physical therapy has been successful in treating anterior knee pain by addressing such factors among others [22]. Understanding the contributing intrinsic factors can help design a plan to effectively return an athlete back to play in the shortest period.

Fact Box

Identification of intrinsic and extrinsic factors leading to chronic/overuse injuries can help guide treatment and reduce recurrence and severity.

Extrinsic factors include the equipment used by the athlete, playing surface, training volume and intensity, as well as environmental conditions. Often extrinsic factors are modifiable. Variations in extrinsic factors can expose an athletics propensity for a certain injury, influence the intensity, as well as impact the duration. For example, tibia, midfoot and fore-foot stress injuries may be related to a new pair of shoes in conjunction with increased workload, among other factors. Recognizing patterns among both individual players and groups can provide guidance for treatment by addressing the necessary extrinsic factors. Changes for

stress injuries may include vitamin and calcium supplementation, cross-training and load reduction, foot orthotics as well as diet modification [23–26].

Recurrent injuries, a subset of chronic injuries, can be challenging and devastating to the athlete and similarly require consideration of both intrinsic and extrinsic factors. Common to basketball players, recurrent injury to the groin, hamstring, and calf can keep a player from full participation for an extended period. Recognizing related factors remains critical, including muscle imbalance, flexibility, fatigue, and alignment. Additionally, return to play timing can be uneasy, given propensity for recurrence and no clear consensus recommendation [27]. Strategies to reduce recurrence include load management (reduced practice participation, modifying minutes and games played) as well as employing imaging such as ultrasonography to help monitor healing [28, 29]. Nutritional supplements have also shown promise in helping to mitigate soft tissue injury [30–32].

48.3.3 Additional Return to Play Factors

Regardless of the acute or chronic etiology, treatment decisions for in-season injuries must also consider additional factors including timing of season, player goals, and potential for long-term risk. These decisions must be individualized and done with appropriate informed consent of the player. Unfortunately, the paradigm presented to the medical staff faced with weighing the best interests of the player and team can be challenging.

Fact Box

Timing of season, level of play, goals of the athlete, and risk for irreversible damage should be considered when discussing treatment and return to play.

Any medical provider when advising a player should always consider the risk for permanent or



Fig. 48.2 Radiograph of a left tibia demonstrating a stress fracture of the anterior tibial cortex. Evident is the “dreaded black line” suggestive of a tibial cortical non-union, and thus the potential risk of impending fracture

irreversible injury with continued participation. As noted previously, certain mid-foot stress reactions or fractures can have catastrophic or career ending consequences if untreated. Additional high-risk injuries include concussions, ACL tears, locked knees, stress fractures of the anterior tibial cortex (Fig. 48.2), and tension-sided femoral neck fractures. Outside of a few exceptions, players should not return to play until treated and fully rehabilitated. Medium-risk and low-risk injuries must be weighed against other factors.

An important factor to consider for lower risk injuries is the timing of injury in relation to the stage of the season. Injuries that do not carry long-term risk may be handled symptomatically toward the end of the season through workload modulation as well as short-term pain relief techniques. In contrast, when an injury is sustained earlier in the season, it may be prudent to remove a player from participation in order to allow for healing and return to play during more critical portions of the season. For example, low risk stress fractures such as second and third

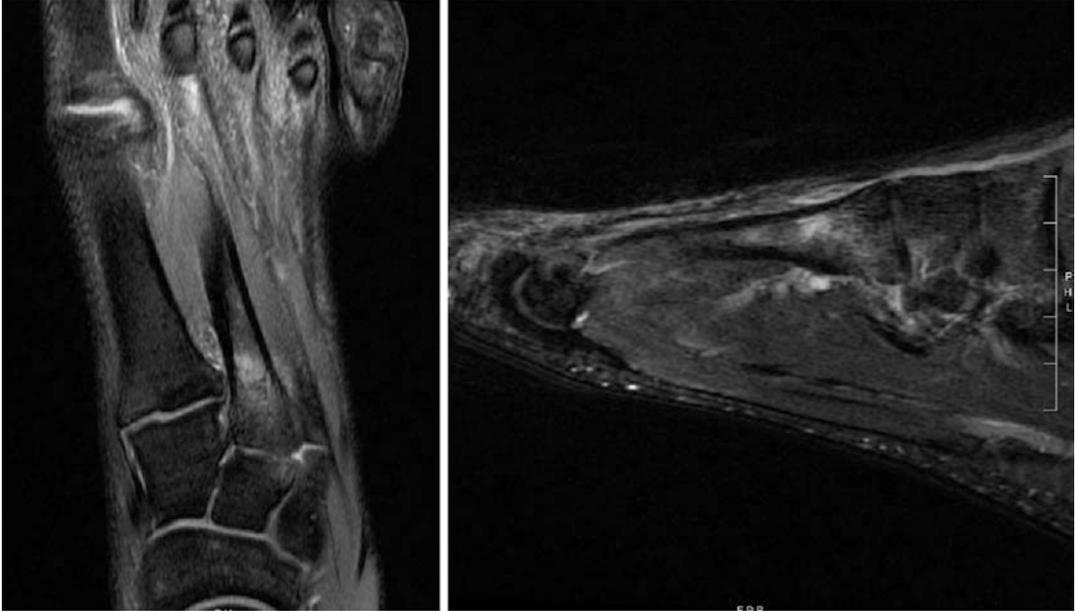


Fig. 48.3 MRI (coronal and sagittal views) demonstrating second metatarsal stress reaction in collegiate basketball player

metatarsal fractures can often be symptomatically treated, but carry a risk for fracture completion (Fig. 48.3). If encountered early in the season, a player may be removed from competition in order to allow adequate healing. However, toward the end of the season or playoffs, a player may be allowed to participate, given the consequence of fracture is not career threatening and treatable with a high chance of returning to play at the same level the following season. Shoulder dislocations and ankle sprains present a similar paradigm with the timing of injury likely dictating return to play recommendations assuming the player has achieved adequate recovery, i.e., full range of motion and strength. Treatment of medium risk injuries such as fifth metatarsal fractures and medial malleolus injuries should be counseled regarding the risk of more substantial sequelae within the context of season and player goals. In all cases, open communication with the player and training staff is paramount to achieving an optimal outcome. Further, documentation of these discussions is also important.

48.4 End-of-Season Evaluation/ Training Programs

Completion of a season for many athletes represents the end of competition, not necessarily the end of athletic activity. Transition to the off-season regularly includes a period of physical and mental rest followed by re-introduction of strength and conditioning along with individual workouts. For the medical staff, the end of the season provides ample opportunity to assess injury patterns from the previous year, address individual injuries to allow healing and recovery, as well as institute preventative measures to reduce future injuries as competition commences the following season. Exit physicals are strongly encouraged to compare pre- and post-season metrics, review injury history, and initiate an off-season programs tailored to the individual athlete [9]. Early incorporation of corrective exercises even before beginning off-season strength and conditioning will prevent strengthening dysfunctional patterns. In some instances, the off-season

may provide the opportunity to surgically address injuries or enforce alterations in training practices, even including complete cessation, to allow for optimal healing. Communication and planning among the entire training staff are again of paramount importance. Regular updates are also critical to ensure benchmarks are met, and changes are implemented when necessary.

Fact Box

Exit physicals are important for injury prevention, treatment, and identification of trends.

48.5 Summary

Management of in-season basketball injuries requires a multidisciplinary approach from the entire health-care team. Open dialogue, regimented documentation, and prospective evaluation are necessary to optimize player recovery. Preseason evaluations provide important metrics for injury prevention, risk stratification, and identification. During the season, labeling injuries as acute or chronic in etiology can help guide treatment, especially immediately following an acute injury. In the case of chronic injuries, recognizing and addressing intrinsic and extrinsic factors can help frame treatment and future injury prevention. Athletes with an injury, where continued play may risk irreversible and permanent damage, are held out in most circumstances. For other injuries, the risk of exacerbation, reinjury, or compensatory injury must be weighed against the timing of the season and level of competition when evaluating a basketball injury. End of the season and subsequent off-season provide an important opportunity to reassess, treat, and implement new practices for caring for the athletes. In-season management of injuries is truly a team effort that does not begin or end with competition.

References

1. Dijkstra HP, Pollock N, Chakraverty R, Alonso JM. Managing the health of the elite athlete: a new integrated performance health management and coaching model. *Br J Sports Med.* 2014;48(7):523–31.
2. Lim BO, Lee YS, Kim JG, An KO, Yoo J, Kwon YH. Effects of sports injury prevention training on the biomechanical risk factors of anterior cruciate ligament injury in high school female basketball players. *Am J Sports Med.* 2009;37(9):1728–34.
3. Borowski LA, Yard EE, Fields SK, Comstock RD. The epidemiology of US high school basketball injuries, 2005-2007. *Am J Sports Med.* 2008;36(12):2328–35.
4. Agel J, Olson DE, Dick R, Arendt EA, Marshall SW, Sikka RS. Descriptive epidemiology of collegiate women's basketball injuries: National Collegiate Athletic Association Injury Surveillance System, 1988-1989 through 2003-2004. *J Athl Train.* 2007;42(2):202–10.
5. Dick R, Hertel J, Agel J, Grossman J, Marshall SW. Descriptive epidemiology of collegiate men's basketball injuries: National Collegiate Athletic Association Injury Surveillance System, 1988-1989 through 2003-2004. *J Athl Train.* 2007;42(2):194–201.
6. Anderson L, Triplett-McBride T, Foster C, Doberstein S, Brice G. Impact of training patterns on incidence of illness and injury during a women's collegiate basketball season. *J Strength Cond Res.* 2003;17(4):734–8.
7. Croisier JL, Ganteaume S, Binet J, Genty M, Ferret JM. Strength imbalances and prevention of hamstring injury in professional soccer players: a prospective study. *Am J Sports Med.* 2008;36(8):1469–75.
8. Grimm NL, Jacobs JC Jr, Kim J, Denney BS, Shea KG. Anterior cruciate ligament and knee injury prevention programs for soccer players: a systematic review and meta-analysis. *Am J Sports Med.* 2015;43(8):2049–56.
9. Duralde X, Jones T, Griffith T. Challenges of medical care delivery in professional sports: lessons from professional baseball. *J Am Acad Orthop Surg.* 2018;26(24):872–80.
10. van Mechelen W, Hlobil H, Kemper HC. Incidence, severity, aetiology and prevention of sports injuries. A review of concepts. *Sports Med.* 1992;14(2):82–99.
11. Bachmann LM, Kolb E, Koller MT, Steurer J, ter Riet G. Accuracy of Ottawa ankle rules to exclude fractures of the ankle and mid-foot: systematic review. *BMJ.* 2003;326(7386):417.
12. Safran MR, Zachazewski JE, Benedetti RS, Bartolozzi AR 3rd, Mandelbaum R. Lateral ankle sprains: a comprehensive review part 2: treatment and rehabilitation with an emphasis on the athlete. *Med Sci Sports Exerc.* 1999;31(7 Suppl):S438–47.

13. Winston MJ, Weiland AJ. Scaphoid fractures in the athlete. *Curr Rev Musculoskelet Med.* 2017;10(1):38–44.
14. Mayer SW, Joyner PW, Almekinders LC, Parekh SG. Stress fractures of the foot and ankle in athletes. *Sports Health.* 2014;6(6):481–91.
15. Porter DA. Fifth metatarsal Jones fractures in the athlete. *Foot Ankle Int.* 2018;39(2):250–8.
16. Robertson GAJ, Goffin JS, Wood AM. Return to sport following stress fractures of the great toe sesamoids: a systematic review. *Br Med Bull.* 2017;122(1):135–49.
17. York PJ, Wydra FB, Hunt KJ. Injuries to the great toe. *Curr Rev Musculoskelet Med.* 2017;10(1):104–12.
18. McCormick JJ, Anderson RB. The great toe: failed turf toe, chronic turf toe, and complicated sesamoid injuries. *Foot Ankle Clin.* 2009;14(2):135–50.
19. Piccininni P, Clough A, Padilla R, Piccininni G. Dental and orofacial injuries. *Clin Sports Med.* 2017;36(2):369–405.
20. Rodriguez JO, Lavina AM, Agarwal A. Prevention and treatment of common eye injuries in sports. *Am Fam Physician.* 2003;67(7):1481–8.
21. Sanchis-Alfonso V. Holistic approach to understanding anterior knee pain. Clinical implications. *Knee Surg Sports Traumatol Arthrosc.* 2014;22(10):2275–85.
22. Werner S. Anterior knee pain: an update of physical therapy. *Knee Surg Sports Traumatol Arthrosc.* 2014;22(10):2286–94.
23. Young AJ, McAllister DR. Evaluation and treatment of tibial stress fractures. *Clin Sports Med.* 2006;25(1):117–28. x
24. Tuan K, Wu S, Sennett B. Stress fractures in athletes: risk factors, diagnosis, and management. *Orthopedics.* 2004;27(6):583–91. quiz 92-3
25. Welck MJ, Hayes T, Pastides P, Khan W, Rudge B. Stress fractures of the foot and ankle. *Injury.* 2017;48(8):1722–6.
26. Close GL, Sale C, Baar K, Bermon S. Nutrition for the prevention and treatment of injuries in track and field athletes. *Int J Sport Nutr Exerc Metab.* 2019;29(2):189–97.
27. Erickson LN, Sherry MA. Rehabilitation and return to sport after hamstring strain injury. *J Sport Health Sci.* 2017;6(3):262–70.
28. Hall MM. Return to play after thigh muscle injury: utility of serial ultrasound in guiding clinical progression. *Curr Sports Med Rep.* 2018;17(9):296–301.
29. Bright JM, Fields KB, Draper R. Ultrasound diagnosis of calf injuries. *Sports Health.* 2017;9(4):352–5.
30. Moon AS, Boudreau S, Mussell E, He JK, Brabston EW, Ponce BA, et al. Current concepts in vitamin D and orthopaedic surgery. *Orthop Traumatol Surg Res.* 2019;105(2):375–82.
31. Rebolledo BJ, Bernard JA, Werner BC, Finlay AK, Nwachukwu BU, Dare DM, et al. The association of vitamin D status in lower extremity muscle strains and core muscle injuries at the national football league combine. *Arthroscopy.* 2018;34(4):1280–5.
32. Chiang CM, Ismaeel A, Griffis RB, Weems S. Effects of vitamin D supplementation on muscle strength in athletes: a systematic review. *J Strength Cond Res.* 2017;31(2):566–74.

Part V

Injury Prevention, Rehabilitation and Back to Field Process in Basketball



Wayne J. Diesel, Ido Dana, and Lior Laver

49.1 Introduction

Basketball is a sport that requires highly skilled and complex coordination of upper and lower body movements. Shoulder and scapular muscles are integral for shooting, dribbling, and defensive work such as blocking and grabbing rebounds [1]. Even minor injuries to shoulder or scapular muscles or elsewhere in the kinetic chain may cause pain and reduction of effective motor patterns controlling movement and could impair player's ability to play [2].

Given the effect that minor shoulder injuries may have on the playing ability of basketball players, it is somewhat surprising that shoulder injuries are relatively uncommon and result in very few games missed. According to league-wide injury analysis data released by the NBA (National Basketball Association) in 2019, the incidence and

impact of shoulder injuries in the NBA over the past six seasons (2013/14 to 2018/19) have been consistently very low. Shoulder injuries that were reported were shoulder instabilities, rotator cuff injury, acromioclavicular sprains, and shoulder strains. The average number of players with a shoulder injury ranged from 1 to 3%; number of players missing at least one game ranged from <1 to 2%; and average recurrence within 1 year ranged from 5 to 15% [3]. Further support for the low incidence of shoulder-related basketball injuries is found in the report by Deitch et al., stating that in the NBA and WNBA (Women's National Basketball Association) rotator cuff injuries and AC joint sprains were the only shoulder injuries reported between 1996 and 2006, accounting for 1.3% and 0.7%, respectively, for all the reported shoulder injuries [4].

Another interesting finding is that unlike significant declines in PER (player efficiency ratings) and number of games played during the season following surgical repair of other basketball injuries, such as ACL or Achilles tendons ruptures, players returning from shoulder stabilization procedures had significantly lower declines in PER and games missed during the season following surgery [5].

The aim of this chapter is to provide an outline of the principles and tests recommended for the most common basketball-related shoulder injuries. Using a battery of tests rather than relying on a single test for making a diagnosis of shoulder pathology is recommended, including symptom altering tests to establish the presence of other contributing factors along the kinetic chain [6].

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627

49.2 Assessment

Despite recent advances in the diagnosis and treatment of shoulder injuries, the shoulder joint remains one of the most challenging areas to assess [7]. The shoulder joint is unique because of its heavy reliance on muscle activity, mediated by the sensorimotor system (SMS), to provide dynamic stability. Pain and tissue pathology following injury can negatively impact the SMS's ability to control proper shoulder movement [8]. Establishing the nature and site of tissue pathology, cause of pain and the resultant SMS (neuromuscular control and proprioception) impact should therefore be a goal of the clinical assessment.

Indirect support linking kinetic chain deficits to shoulder injuries comes from a review on the incidence of shoulder injury in elite wheelchair athletes [9]. In their review, it was reported that wheelchair athletes with better trunk control had fewer overuse shoulder injuries than wheelchair athletes with decreased trunk control. Fairburn and Bliven (2019) hypothesized that an unstable base (deficit in kinetic chain) leads to upper extremity compensations that ultimately lead to overuse injuries to the shoulder [9].

According to Kibler et al., the entire kinetic chain must be assessed when assessing shoulder injuries [10]. They argued that standard diagnostic tests could be used to establish overt causes of a shoulder injury (e.g., tendon injury and instability), but it is the nonovert alterations occurring locally or distant which, unless diagnosed, may delay return to full function. Local nonovert alterations could be a loss of shoulder internal rotation, strength imbalances, and scapular dyskinesia (scapular motion and position). Distant nonovert alterations include lumbar, hip, and/or knee inflexibility [10].

Kibler et al. [10] suggested that nonovert (distant and local) issues that relate/contribute to shoulder issues should be included in the clinical examination of shoulder injuries.

49.2.1 Standing Posture to Evaluate Legs, Lumbar, Thoracic, and Cervical Spines [11]

Young et al. theorized that compromised spinal function could play a role in shoulder pathology

[12]. This led researchers to include evaluation of posture in their shoulder assessments [10]. However, despite this theoretical link between posture and shoulder symptoms, the scientific literature is not as supportive.

Greenfield et al. found significant forward head positioning in subjects with symptomatic shoulders compared to asymptomatic subjects. They argued that normal shoulder function may be altered by postural induced cervical dysfunction [11]. However, they concluded it is unlikely that changes in cervical function, caused by poor posture, are an isolated finding in shoulder overuse injuries.

In a systematic review by Barrett et al., it was reported that despite evidence that reducing thoracic kyphosis could increase shoulder range-of-motion, there is no evidence linking thoracic kyphosis to shoulder pain [13]. Barrett et al. advised that a better approach would be to assess whether or not shoulder symptoms, present during a provocative test, are immediately eased following correction of thoracic kyphosis [13]. Symptom alteration tests (described later in this chapter) for possible links to altered posture should therefore also be incorporated to establish links between an observed postural inconsistency and shoulder symptoms.

49.2.2 Scapular assessment and Scapular Dysfunction/Scapular Diskinesis

McClure et al. referred to the scapula as an essential component of shoulder function and required both stability and mobility. Assessment of the scapula is complex because of its 3D motion during arm elevation involving upward rotation, posterior tilting, and external rotation coupled with clavicle elevation and protraction [14]. Appropriate assessment should involve (a) visual observation, (b) symptom alteration tests, as well as (c) specific muscle strength tests. Establishing whether or not scapula dyskinesia may be linked to shoulder symptoms is the goal of scapula assessment [15].

49.2.2.1 Scapular Visual Assessment

This is best done with the player standing with their shirt off and viewed from behind. First, with the player standing, statically with arms relaxed

by their side, a note should be taken of the presence of any of the most common prominences. These include (1) inferomedial border (anteriorly rotated coracoid and acromion); (2) medial border (classic “winging”); and (3) superior medial border (superior scapula translation).

Valid, reliable, and accurate recording of scapula malpositioning can be measured using the Palpation Meter (PALM—Performance Attainment Associates, St. Paul, MN). The PALM has been shown to be useful in assessing overhead athletes with shoulder “impingement” [16]. Prior to measuring, several landmarks are palpated and marked with a pen. These landmarks (T3 spinous process, scapula spine root, acromion angle, medial scapula border, scapula spine, coracoid process, and fourth intercostal space) are believed to measure scapula upward/downward rotation, protraction/retraction, anterior/posterior tipping, internal/external tipping, elevation/depression, pectoralis minor length, and coracoid height. Two static positions, while holding a weight equivalent to 3% body weight, are used for measurements. The first position is with the arm in the resting position of 0° of flexion and abduction. The second position has the arm held in 90° of abduction, with a 15 s rest between each measurement.

- The mid-line of the spine was found by first marking C7-T1 and then palpating the PSIS to find mid-line of the spine. A flexible ruler was placed down the spine to use as a guide to mark the spine. To obtain scapular position, the selected landmarks were palpated again over the marked landmarks and then the tips of the calipers were placed directly on top of the chosen site.
- The inferior angle was defined as the point on the medial border of the scapula that first begins to travel laterally.
- Mid-line of the spine to inferior angle of the scapula—the caliper was placed on the spinous process of T7 and the inferior angle to measure protraction.
- Mid-line of the spine to the root of the scapula—the caliper was placed on the spinous process of T3 and the root of the scapula to measure protraction/retraction.

- The root of the scapula to the angle of the acromion—the caliper is placed on the root of the scapula and the inferior angle of the acromion to measure the width of the scapula.
- Spinous process of T3 to the inferior angle of the acromion—the caliper was placed on the spinous process of T3 to the inferior angle of the acromion to measure scapular protraction/retraction.
- Coracoid process and the sternal notch—to measure protraction, coracoid height, and coracoid distance, the caliper was placed on the middle of the coracoid process and the sternal notch.
- Pectoralis minor length—calipers were placed on the medial coracoid process of the scapula and the fourth intercostal space at the sternum.”

Pectoralis minor may become shortened in players with forward head and rounded shoulder postures and has been linked to Neurogenic Thoracic Outlet Syndrome (NTOS) [17]. Thoracic Outlet Syndrome (TOS) is an extremely rare, while potentially very limiting condition among professional basketball players. Another clinical shoulder condition that may benefit from measuring pectoralis minor length (Fig. 49.1) is the SICK (Scapula malposition, Inferior medial border prominence, Coracoid pain and malposition, and scapula dyskinesia) Scapula Syndrome. SICK scapula syndrome has been described as an overuse muscle fatigue syndrome causing symptoms of anterior shoulder pain, posterosuperior scapula pain, superior shoulder pain, proximal lateral pain, or a combination of these symptoms. Due to the variety of symptoms, SICK can be misinterpreted as anterior instability (anterior shoulder symptoms), cervical spine referral (posterosuperior symptoms), subacromial impingement (proximal lateral arm pain), acromioclavicular joint pathology (superior shoulder pain) [18].

Once static observations and measurements have been completed, dynamic scapular motion should be viewed. This can be done using the modified scapula dyskinesia test (SDT), described by Uhl et al., where any abnormality in scapular motion is recorded simply as a “yes” and normal scapulothoracic motion is recorded as a “no.” The



Fig. 49.1 Pectoralis Minor Length measurement. The distance from the medial-inferior border of the coracoid process to the sternocostal junction of the inferior aspect of the 4th rib is measured using a caliper or a measuring tape (a) [19]. Another common clinical method to assess pectoralis minor length is to measure the distance from the posterolateral angle of the scapula to the examination table of a supine patient, with distances greater than 2.54 cm (1 in) suggesting pectoralis minor tightness (b) [20]

players performs 3–5 arm elevations in the scapular and sagittal planes [21]. Any abnormal inferior angle, medial border, or superior margin prominences were recorded as a “yes.” The presence of multi-plane asymmetries was observed more frequently using forward flexion motions. As scapula dyskinesia may be found in both symptomatic and asymptomatic individuals, results should be viewed in conjunction with other tests. If a “no” is recorded, then scapula dyskinesia is not linked to the shoulder complaint. However, if “yes” is recorded, then scapula dyskinesia may be a potential contributing factor. Symptom alteration tests of the scapula should then be used to determine a link between observable scapular dyskinesia and the shoulder complaint [21].

49.2.2.2 Scapular Symptom Alteration Tests

Symptom alteration tests are designed to establish the presence of a link between shoulder symptoms and scapula malposition. A significant decrease in pain during the test is indicative that scapula dyskinesia is a contributing factor and needs to be addressed in order to correct the shoulder injury. The scapular assistance test (SAT) and scapular reposition test (SRT) are the two most often used tests that involve manual positioning of the scapula.

The current version of the SAT involves manually assisting upward rotation and posterior tilting of the scapula during elevation of the shoulder.

The scapular retraction test has been modified by Tate et al. and called the scapular reposition test (SRT) [22]. Instead of emphasizing scapula retraction, the SRT assists posterior tilt and external rotation of the scapula. The SRT is performed by the clinician correcting for scapula posterior tilt and external rotation by using their fingers to hold the acromioclavicular joint and use the thenar eminence to apply pressure on the spine of the scapula while using their forearm, obliquely positioned, towards the inferior angle of the scapular. Any positive provocation tests for “impingement” (Neer, Hawkins, Jobe) are then repeated using the SRT. Manual reduction using the SRT test was shown by Tate et al. to decrease pain in a subset of overhead athletes with “impingement” symptoms. If positive, the SRT can be used to identify players that would benefit from interventions targeting scapula malposition such as taping, bracing, or strengthening [22].

Other symptom alteration tests include posture correction involving cervical spine (forward head position), thoracic spine (kyphosis), and lumbar spine (lumbar lordosis).

If scapular dyskinesia is believed to be contributing to shoulder symptoms, the next step is to examine structures that may contribute to the dyskinesia. Examination of muscle strength, motor control, flexibility of the scapular muscles, and general posture is required [15]. In certain persistent cases with substantial scapular winging, nerve conduction studies may be warranted to

asses the long thoracic nerve, spinal accessory nerve or dorsal scapular nerve.

49.2.2.3 Scapular Muscle Strength

The benefits of assessing muscle strength using a hand-held dynamometer (HHD) are the short amount of time required to test, low cost, portability, and results, which are comparable to the gold standard of isokinetic dynamometry. In a series of systematic reviews, Stark et al. (2011) and Schrama et al. (2014) concluded that HHD was a valid tool for assessing shoulder internal and external rotation, flexion, extension, abduction, adduction, as well as elbow extension and flexion [23, 24]. Scapular muscles responsible for stabilization and movement include the serratus anterior, middle, and lower trapezius and should be tested using the HHD.

Liberatori et al. concluded that scapular protraction (primarily serratus anterior) muscle strength is best tested in a seated position, using a HHD but has poor interrater reliability [25, 26]. The player sits on a stool with their back and head against a wall such that feet are flat on the floor and knees are flexed to 90° (Fig. 49.2). The shoulder, to be tested, is flexed to 90° with the elbow held in neutral and fully extended. The clinician places the HHD between their trunk and the player's hand then uses their trunk to manually resist protraction. The player determines the test position by actively finding the midpoint between retraction and protraction. Mid-positioning of the scapula maximizes isometric contraction force by optimizing the length–tension relationship. The

player is then instructed to push against the dynamometer with maximal effort. A “make” test was used to determine peak force.

HHD muscle testing for the lower trapezius muscle has been described and validated by Michener et al. [27]. The mid position of scapula depression and elevation is actively determined by the player and then used as the test position. The player lies prone with the arm to be tested held in 140° of elevation (Fig. 49.3). The clinician applies a superior and lateral force in parallel to the long axis of the humerus with the HHD positioned midway between the root of the spine of the scapula and the acromion process. The player attempts to depress and adduct the scapula.

HHD testing found to have a high inter-rater reliability by Ammar TA might also be used for testing Middle Trapezius muscle strength (Fig. 49.4) [28].



Fig. 49.2 Serratus Anterior HHD Muscle Test

Fig. 49.3 Lower trapezius HHD muscle test. Used with permission [27]





Fig. 49.4 Middle trapezius HHD muscle test. Used with permission [27]

49.3 Common Basketball Shoulder Injuries

Hegedus et al. following an update of their systematic review in 2008 reached two conclusions regarding clinical examination of shoulder injuries [6, 29]. The first was that no single clinical examination test could be relied on to make a pathognomonic diagnosis of a shoulder injury. Secondly, using a cluster of tests proved only marginally better in making a diagnosis of shoulder pathology. These conclusions stress the importance of including other investigations, such as MRIs and ultrasound scans (USS), to assist medical practitioners working in basketball to reduce the likelihood of delaying diagnosis that may result in decreased performance levels and greater time-loss injuries later.

49.3.1 Instability

Shoulder instability, traumatic or atraumatic, is one of the most common severe shoulder complaints seen in basketball players. Instability can be anterior, posterior, inferior, or multidirectional, and their clinical features have been well described by Kibler and Murrell [7].

A recent retrospective analysis identified 50 NBA players who experienced a shoulder instability event between 1999–2018 [30]. Of the 50 players with shoulder instability, 14 players

experienced a subluxation whereas 36 experienced a shoulder dislocation. Twenty-five athletes were treated operatively whereas 25 were treated nonoperatively. Most operated cases were due to dislocations (88%). Recurrence was reported in 16% of cases. Another review of NBA data revealed shoulder stabilization procedures accounted for a significant percentage of orthopedic procedures in the NBA ($n = 46$, 13.2%) from 1984 to 2012 (1.6 per season) [5]. A review of injury history among Women's National Basketball Association athletes between 2000–2008, showed a shoulder instability incidence of 1.25 per season [31].

Anterior instability that is traumatic results from acute trauma causing an anterior subluxation or dislocation. Atraumatic anterior instability may result from an acute episode in a lax shoulder. In traumatic instances, the player will report a reasonably forceful incident, typically involving abduction and external rotation. Symptoms reported by the player may include recurrent subluxations/dislocations, catching shoulder pain and “dead arm” syndrome. The “dead arm” symptoms are brief episodes of arm numbness and weakness resulting from impingement or traction of neurovascular structures [10]. The clinical tests for anterior shoulder instability include Apprehension, Relocation, and Surprise Tests (Figs. 49.5 and 49.6, Table 49.1). These have the highest diagnostic odds ratios (DOR) with 95% confidence intervals, according to Hegedus et al. [6].

Posterior instabilities are more often atraumatic, can be voluntarily subluxated, and are typically part of multidirectional instabilities. Multidirectional instability, defined as a combination of two or three directional instabilities, is most commonly atraumatic. Generalized body laxity is a common finding in players with multidirectional shoulder instability [7].

Laxity and instability are typically clinically assessed by a group of symptom-provoking and relieving instability tests followed by tests to assess for possible underlying laxity. Eshoj et al. found good levels of interpreter reliability when using the Apprehension, Surprise, Load and

Shift, and Gagey tests (Figs. 49.5, 49.6, 49.7, 49.8 and 49.9, Table 49.1) for shoulder instability [36]. Commonly used tests, Relocation and Sulcus Tests, however, require further standardization to achieve acceptable levels of interpreter reliability.

49.3.2 Rotator Cuff Tears

Differentiating between full and partial tears of the rotator cuff is important in deciding the ideal management pathway. Partial-thickness rotator cuff tears (RCT) may benefit from conservative



Fig. 49.5 The Apprehension test



Fig. 49.6 The Relocation test



Fig. 49.7 Load-and-shift: anterior direction



Fig. 49.8 Load-and-shift: posterior direction



Fig. 49.9 The Gagey (Hyperabduction) test

management, whereas full-thickness RCT should be sent for a surgical opinion. Unfortunately, current research suggests that most clinical testing, apart from stronger shoulder abduction strength in partial RCT, may not be sufficient in separating partial from full-thickness RCT [37].

Musculoskeletal Ultrasonography (MSK US) is a relatively cheap, practical, and effective tool that sports medicine practitioners are using to assist in making a quick and accurate diagnosis. MSK US can be used to diagnose full-thickness RCT. However, if the MSK US is negative but there is a high clinical suspicion of full-thickness RCT, it is recommended that an MRI should be performed. MSK US is less reliable for picking up partial-thickness RCT and an MRI is recommended if conservative management fails [38].

The diagnostic accuracy of clinical tests versus MRI in rotator cuff tears has been compared [8]. Clinical testing of supraspinatus and subscapularis had reasonable levels of sensitivity but low specificity implying false-positive tests do occur. The reason for false positives may be because pain is considered a positive sign during

testing, but other shoulder pathologies may be responsible for the pain provocation during testing. Once there is suspected rotator cuff pathology, involving either supraspinatus or subscapularis, imaging can help to confirm the pathogenesis. Infrapinatus testing and MRI both appear not to have good sensitivity or specificity. This implies, in the case of suspected infrapinatus lesions, MRI scans may not add any additional information to what could be gained from appropriately conducted clinical tests. Teres minor lesions are unlikely to be seen on MRI, so clinical examination appears to be the best method for detecting lesions in teres minor [8].

RCT typically have a history of sudden pain onset over the top of the shoulder radiating down to the deltoid area and complaints of pain with overhead activities and at rest. Etiologies of RCT are typically multifactorial with multiple episodes of microtrauma and altered mechanics (fatigue-related). Symptoms are associated with deficits in Range-of-Motion (ROM) and muscle strength [7, 39].

Table 49.1 Shoulder instability and laxity testing procedures and outcomes

Test	Patient position	Examiner	Outcomes
Apprehension	Supine with tested shoulder supported by edge of examination table. Shoulder abducted 90°, elbow 90° flexion	One hand gently resting in front of the tested shoulder, other hand holding the wrist. Examiner’s thigh supports patient’s elbow. Shoulder moved towards maximal external rotation by examiner	Positive if presence of subjective or objective apprehension and/or pain
Surprise	Same position as apprehension test	At the end position of the apprehension test, the examiner gently pushes the head of the humerus posteriorly (relocation) then, without warning, removes their hand	Positive if presence of subjective or objective apprehension and/or pain
Load-and-shift	Supine with scapular supported by the examination table	One hand holds the patient’s olecranon while patient’s hand is supported by their trunk and elbow	Four-level laxity scale is used to record the amount of laxity
– Anterior	Arm in 90° abduction, scapular plane, with elbow flexed	Examiner places hand on back of the shoulder and gently shifts the humerus anteriorly	0 = Minimal to no movement 1 = Humeral head moves to glenoid rim 2 = Humeral travels beyond the rim of glenoid but relocates spontaneously after pressure is released 3 = Humeral head moves beyond the rim of glenoid but does not relocate once pressure is released
– Posterior	Arm in 20° abduction, scapular plane, with elbow flexed	Examiner places hand in front of the shoulder and gently shifts the humerus posteriorly	Positive if >1 cm movement for laxity/instability
Gagey (Hyperabduction)	Seated with shoulder in 90° abduction and elbow flexed. Facing a mirror	Stand behind patient and one hand on top of the acromion preventing elevation of the shoulder. The other hand supporting the elbow passively moves the arm into further abduction	Positive if abduction >105° for laxity/instability

49.3.3 Supraspinatus Tears

The tests for supraspinatus tears were Jobe’s (empty can) test (Fig. 49.10, Table 49.2) and drop arm test (Fig. 49.11, Table 49.2) [8].

49.3.4 Subscapularis Tears

Yoon et al. (2015) found that while the Lift-Off sign was very accurate in diagnosis of full-thickness RCT, other tests were more suitable for partial-thickness/strength deficits of subscapularis [40]. These tests included the internal rotation lag sign, belly-press, and bear-hug tests (Figs. 49.12, 49.13, 49.14 and 49.15, Table 49.3).

49.3.5 Infrapinatus Tears

The drop sign test (Fig. 49.16, Table 49.4) when combined with the resisted external rotation test (RERT) (Fig. 49.17, Table 49.4) [2, 41] has been shown to accurately diagnose tears of the infra-spinatus tendon. However, these tests are not capable of accurately distinguishing between full or partial-thickness RCT [42].

49.3.6 Teres Minor Tears

Clinical examination of teres minor dysfunction is essential because imaging is unreliable. Collin et al. recommend starting posterior rotator cuff

examination with an external rotation lag sign test [43] (Fig. 49.18, Table 49.5). A lag of more than 10 but less than 40° is indicative of infraspinatus weakness. A lag of greater than 40° indicates infraspinatus and teres minor pathology. If there is weakness but no Lag Sign, then the Drop Sign and Patte Maneuver (Figs. 49.19 and 49.20, Table 49.5) are performed to test teres minor in 90° abduction.

49.3.7 Acromioclavicular Joint Sprain

Acute acromioclavicular (AC) sprains often occur when players fall directly onto the shoulder. Pain is normally well localized over the AC joint and a step deformity can be felt in any injury greater than a Type I sprain [44]. Chronic AC pain, resulting from multiple minor AC injuries or after Type II or III



Fig. 49.10 Jobe's (empty can) test

sprains, may be associated with shoulder “impingement” [7].

There are cluster of tests including the cross-arm (Scarf), active compression (O'Brien), and resisted extension test (Figs. 49.21, 49.22 and 49.23, Table 49.6) to assess AC sprains.

Scapula dyskinesia is associated with AC sprains and should be included in clinical examinations of suspected AC sprains. Starting by assessing scapula position after manual reduction of the AC joint followed by tests described earlier (specifically SAT and SRT).

49.3.8 Subacromial Pain Syndrome

Cools and Michener challenged the use of the term Subacromial Impingement Syndrome (SIS), used to describe a host of underlying structural and biomechanical causes of shoulder pain [45]. They supported changing the name to Subacromial Pain Syndrome (SPS). SPS is considered as one of the most common sports-related shoulder pathologies. SPS results from pathology to any of the structures that sit within the subacromial space. Typical conditions such as subacromial bursitis, rotator cuff pathology, shoulder laxity/instability, labral tears, scapular dyskinesia, biceps pathology, muscle imbalances, glenohumeral internal rotation deficit, or simply overuse can fall under the umbrella of SPS [45].

Although not reported as a “common” shoulder injury in professional basketball, it is important for the medical staff to be aware of typical

Table 49.2 Supraspinatus testing procedures and outcomes

Test	Patient position	Examiner	Outcomes
Jobe's (empty can) test	Seated with arm actively held in scapular plane and thumb pointing towards the floor	Examiner stands in front of the patient and attempts to push the arm down.	Positive if pain and weakness compared to opposite shoulder
Drop arm test	Patient seated with arm at their side	Examiner passively abducts the arm to 90°. Instructs patient to slowly lower the arm back down	Positive if unable to slowly lower arm in a controlled manner

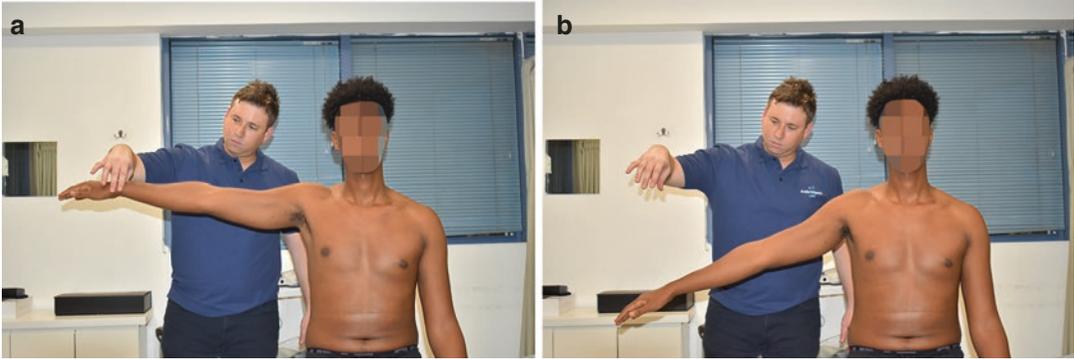


Fig. 49.11 Drop arm test



Fig. 49.12 Lift-off (Gerber's) test. (Description in Table 49.3)



Fig. 49.13 Internal rotation lag sign. (Description in Table 49.3)



Fig. 49.14 Belly-press test: biased towards the upper scapularis. (Description in Table 49.3)



Fig. 49.15 Bear hug test: biased towards the upper scapularis. (Description in Table 49.3)

Table 49.3 Subscapularis testing procedures and outcomes

Test	Patient position	Examiner	Outcomes
Lift-off (Gerber's)	Standing with the back of the hand against their mid-lumbar area	Standing behind the patient. Instructs the patient to lift their hand off their back	Positive if unable to lift their hand off their back. If abnormal scapular movement is detected, then scapular instability may be present
Internal rotation lag sign	Patient standing with back of their hand resting on their mid-lumbar spine	Examiner lifts hand and forearm off the lumbar spine. Instructs patient to hold arm/hand off their back	Positive if unable to maintain forearm/hand position off the lumbar spine
Belly-Press	Standing with palm of hand resting on their abdomen and elbow anterior to the mid-axillary line	Patient instructed to press against their stomach, without moving their elbow	Positive if unable to maintain elbow position. Upper scapularis bias
Bear hug	Patient standing with palm of the hand placed onto the opposite shoulder with elbow parallel to the floor	Examiner instructs the patient to resist an attempt to externally rotate their shoulder	Positive if weakness/pain compared to opposite shoulder. Upper scapularis bias

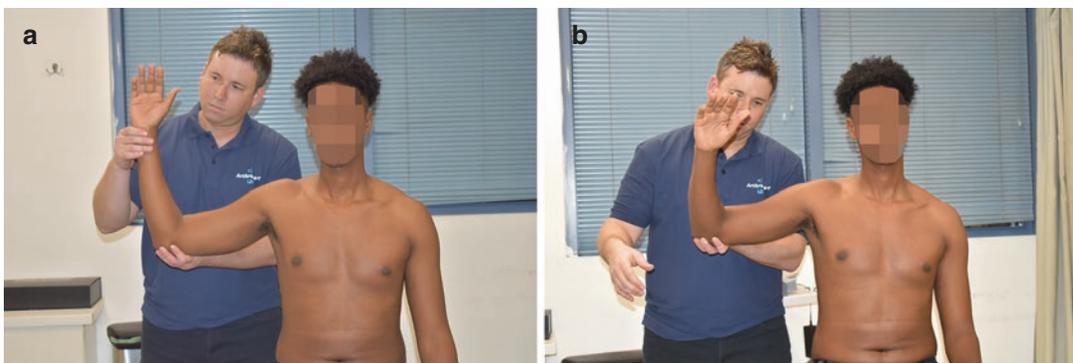


Fig. 49.16 Drop sign test

Table 49.4 Infraspinatus testing procedures and outcomes

Test	Patient position	Examiner	Outcomes
Drop sign	Seated on examination table	Stands behind patient. Supports arm to be tested in the scapular plane with 90° of elevation and elbow flexed to 90°. Patient asked to maintain position after examiner releases the wrist but continues to support the elbow	Positive if arm “drops” or “lags.” Amount of “drop” is measured in degrees.
Resisted external rotation test (RERT)	Patient seated with arm at their side and elbow flexed to 90°	Examiner pushes on the distal forearm to produce a medially directed force, and patient provides resistance	Positive if weakness detected compared to asymptomatic shoulder



Fig. 49.17 Resisted external rotation test (RERT). The test can be performed for each side separately (a) or for both sides simultaneously (b)

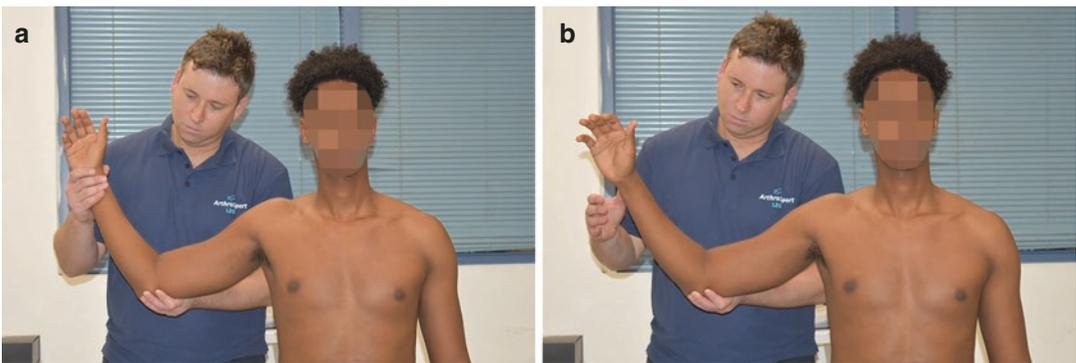


Fig. 49.18 External rotation lag sign

symptoms and how to best assess SPS. As in other shoulder pathologies, the etiology and subjective history help guide the clinician’s choice of tests. Etiology is often the gradual onset of symptoms related to overuse, changing technique, or postural changes caused by injuries to a part of the distant kinetic chain. The main symptoms of SPS include the following:

- Lifting activities provoking an ache or sharp pain in the shoulder or upper arm.
- Pain reproduced moving the arm through a specific range of motion (painful arc).
- Stiffness or weakness whenever the arm is used.
- Lying on the painful side is uncomfortable and may disturb sleep.

Table 49.5 Teres minor testing procedures and outcomes

Test	Patient position	Examiner	Outcomes
External rotation lag sign	Patient seated, elbow flexed to 90° and shoulder elevation 20° in the scapular plane	Examiner passively moves the arm into 5° short of maximal external rotation. Patient instructed to maintain that position.	Positive for infraspinatus weakness if lag >10°. Positive for infraspinatus and teres minor dysfunction if lag >40°.
Drop sign	Patient seated, elbow flexed to 90°, shoulder elevated to 90° of abduction in scapular plane and externally rotated to 90°	Examiner supports the elbow and wrist. Examiner releases hold of the wrist and instructs patient to maintain position against gravity	Positive if unable to resist gravity and shoulder internally rotates.
Patte maneuver	Patient seated, elbow flexed to 90°, shoulder elevated to 90° of abduction in scapular plane and neutral rotation	Examiner supports the forearm on the top of their forearm. Instructs the patient to externally rotate against resistance	Positive if external rotation is less than grade 4 strength (Medical Research Council)

**Fig. 49.19** Patte maneuver**Fig. 49.20** Cross-arm adduction (Scarf) test**Fig. 49.21** The active compression (O'Brien's) test



Fig. 49.22 Resisted extension test



Fig. 49.23 Hawkins-Kennedy impingement sign

Table 49.6 Acromioclavicular joint testing procedures and outcomes

Test	Patient position	Examiner	Outcomes
Cross-arm adduction (scarf)	Patient standing or seated. Elbow flexed to 90° and shoulder at 90° flexion.	Patient instructed to horizontally adduct their arm and place their hand on the opposite shoulder. Examiner then pushes arm into more horizontal adduction	Positive if localized pain on top of the shoulder is provoked
Active compression (O'Brien)	Patient seated or standing. Arm held in 90° shoulder flexion and approx. 15° horizontal adduction, elbow in full extension	Patient instructed to fully internally rotate their shoulder and pronate their forearm. Examiner places their hand on the forearm and instructs the patient to push upwards. The patient then returns their shoulder and forearm to a neutral position and push up against the examiner again	Positive if pain is reproduced in the first position but absent in the second position. Note: If pain is superficial, then acromioclavicular pathology is likely. However, if pain is deep within the joint, then labral pathology should be suspected.
Resisted extension	Patient seated with elbow flexed to 90°, shoulder in 90° flexion, and internal rotation.	Examiner stands behind patient. One hand rests on top of the acromioclavicular joint and the other hand on the back of the elbow. Instructs patient to attempt to horizontally abduct against resistance	Positive if localized pain on top of the shoulder is provoked

Park et al. reviewed eight different clinical tests for diagnostic value in predicting SPS and found the cluster of positive tests (Hawkins-Kennedy impingement sign, the painful arc sign, and the infraspinatus muscle test—Figs. 49.24 and 49.25, Tables 49.5 and 49.7) provided 95% likelihood for SPS [41]. Sports medical staff could also use the closed kinetic chain upper extremity stability test (CKCUES Test) (Table 49.7) as a reliable functional performance test in SPS [46].

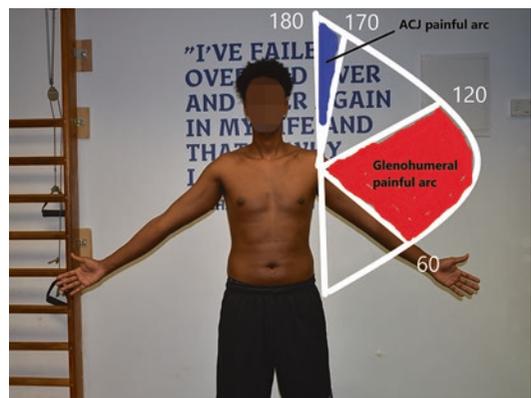


Fig. 49.24 Painful arc sign

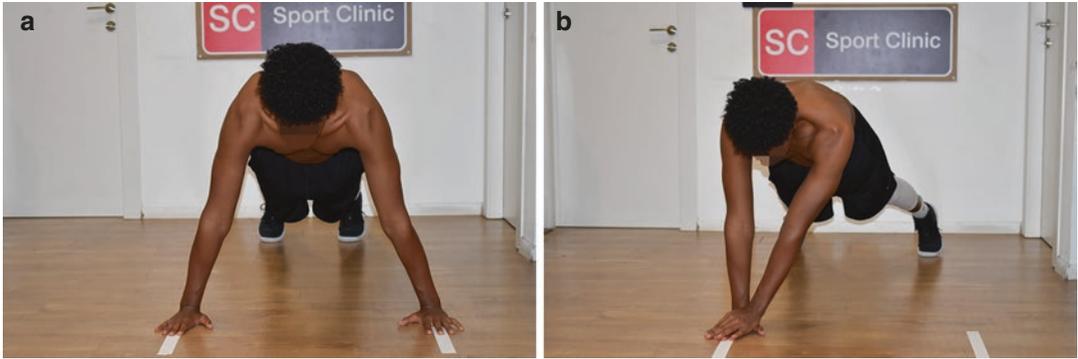


Fig. 49.25 Closed kinetic chain upper extremity stability test

Table 49.7 Subacromial pain syndrome (SPS) testing procedures and outcomes

Test	Patient position	Examiner	Outcomes
Hawkins-Kennedy impingement sign	Patient standing with arms relaxed by their sides	Examiner flexes the patient’s elbow to 90° then forward flexes the arm to 90°. Examiner then places one hand on the patient’s scapular (to assess scapular movement) and with the other hand gently rotates the patient’s arm into internal rotation.	End range of internal rotation is either when patient reports pain or scapular starts to rotate. Test is positive if pain is reported
Painful arc sign	Patient standing with arms relaxed by their sides	Examiner instructs patient to lift arm, in the scapular plane, to full elevation and then lower the arm back to the starting position	Positive if pain or painful click felt between 60 and 120° of elevation
Closed kinetic chain upper extremity stability	Male patients in press-up position. Females in modified (kneeling) press-up position. Back flat, arms perpendicular over hands, hands 36 inches apart (marked with two lines)	To start the test, the examiner instructs the patient to alternate touching the opposite hand and return back to the starting position. Examiner counts how many times the patient touches in 15 s. Rest for 45 s and repeat 3 times	Acceptable test if back remains flat, knees do not touch the ground (males), weight-bearing upper extremity perpendicular to hand, feet stay in starting position. Rating Numeric Rating Scale used if pain reported. Normalized score determined by dividing patient’s height by number of touches. Power Score determined by multiplying the average number of touches by 68% of body weight and divided by 15. A meaningful difference is at least 3 touches

49.4 Summary

Shoulder injuries in professional basketball, tend not to be a common injuries. However, shoulder function is essential in many basketball activities, especially shooting and dribbling, so an injury to

the shoulder is likely to impact the ability to perform. Because sports medicine practitioners, in basketball, do not commonly see shoulder injuries when injuries to the shoulder occur, they may be misdiagnosed or downplayed. Sports medicine practitioners therefore need to remain up to

date with constantly evolving diagnostic tests for shoulder pathology.

The aim of this chapter was to highlight the most frequently seen shoulder pathologies in professional basketball and to provide sports medicine practitioners with appropriate clinical tests for each of the pathologies. Numerous structures within and distant to the shoulder may be responsible for causing the pain or dysfunction. Before subjecting the player to multiple pain-provoking clinical tests, a detailed history should be taken. History taking should include the precise site of pain, nature of the onset of pain/dysfunction, mechanism and shoulder position when injury occurred if acute, aggravating movements/positions in chronic cases, severity of pain, and impact on their ability to perform. Another key take-home point is that alterations to the players' kinetic chain, such as poor posture or scapular dyskinesia, often play a role in the pathogenesis of shoulder problems and must be included in the clinical examination.

References

1. Cavazos M. Muscles used during basketball; N.D. (Jillian.Michaels.com). <https://getfit.jillianmichaels.com/muscles-used-during-basketball-1937.html>
2. Tennent TD, Beach WR, Meyers JF. A review of the special tests associated with shoulder examination. Part I: The Rotator Cuff Tests. *Am J Sports Med.* 2003;31(1):154–60.
3. Mack CD, Herzog MM, Dreyer NA. National Basketball Association: injury reference: frequency, return to competition, and reinjury. *IQVIA Injury Surveillance & Analytics.* NBA Player Health Analytics; 2019.
4. Deitch JR, Starkey C, Walters SL, Moseley JB. Injury risk in professional basketball players: a comparison of Women's National Basketball Association and National Basketball Association athletes. *Am J Sports Med.* 2006;34(7):1077–83.
5. Minhas SV, Kester BS, Larkin KE, Hsu WK. The effect of an orthopaedic surgical procedure in the National Basketball Association. *Am J Sports Med.* 2016;44(4):1056–61.
6. Hegedus EJ, Goode AP, Cook CE, et al. Which physical examination tests provide clinicians with the most value when examining the shoulder? Update of a systematic review with meta-analysis of individual tests. *Br J Sports Med.* 2012;46(14):964–78.
7. Kibler WB, Murrell GAC. Shoulder pain. In: Brukner P, Khan K, editors. *Clinical sports medicine.* 3rd ed. NSW. Chapter 17: McGraw-Hill; 2010. p. 243–88.
8. Modipalli D, Hedge AS, Shetty CB. Diagnostic accuracy in rotator cuff tears: clinical tests vs MRI. *Int J Res Orthop.* 2019;5(4):727–31.
9. Fairbairn JR, Bliven KCH. Incidence of shoulder injury in elite wheelchair athletes differs between sports: a critically appraised topic. *J Sports Rehabil.* 2019;28:294–8.
10. Kibler WB, McMullen J, Uhl T. Shoulder rehabilitation strategies, guidelines, and practice. *Orthop Clin North Am.* 2001;32(3):527–38.
11. Greenfield B, Catlin AP, Coats PW, Green E. Posture in patients with shoulder overuse injuries and healthy individuals. *J Orthop Sports Phys Ther.* 1995;21(5):287–95.
12. Young JL, Herring SA, Press JM, Casazza BA. The influence of the spine on the shoulder in the throwing athlete. *J Back Musculoskelet Rehab.* 1996;7(1):5–17.
13. Barrett E, O'Keefe M, O'Sullivan K, Lewis J, McCreesh K. Is thoracic spine posture associated with shoulder pain, range of motion and function? A systematic review. *Man Ther.* 2016;26:38–46.
14. Kibler WB. The role of the scapula in athletic shoulder function. *AJSM.* 1998;26(2):325–37.
15. McClure P, Greenberg E, Kareha S. Evaluation and management of scapular dysfunction. *Sports Med Arthrosc Rev.* 2012;20(1):39–48.
16. Rondeau MR. The accuracy of the palpation meter (palm) for measuring scapular position in overhead athletes. A thesis submitted to the University of North Carolina at Chapel Hill in partial fulfillment of the requirement of the degree of Master of Arts in the Department of Exercise and Sport Sciences (Athletic Training). Chapel Hill; 2007.
17. Sanders RJ, Rao NM. The forgotten pectoralis minor syndrome: 100 operations for pectoralis minor syndrome alone or accompanied by neurogenic thoracic outlet syndrome. *Ann Vasc Surg.* 2010;24(6):701–8.
18. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology Part III: the SICK scapula, scapular dyskinesia, the kinetic chain, and rehabilitation. *Arthroscopy.* 2003;19(6):641–61.
19. Borstad JD. Measurement of pectoralis minor muscle length: validation and clinical application. *J Orthop Sports Phys Ther.* 2008;38(4):169–74.
20. Sahrman SA. *Diagnosis and Treatment of Movement Impairment Syndromes.* St Louis, MO: Mosby; 2002.
21. Uhl TL, Kibler WB, Gecewich B, Tripp BL. Evaluation of clinical assessment methods for scapular dyskinesia. *Arthroscopy.* 2009;25(11):1240–8.
22. Tate AR, McClure PW, Kareha S, Irwin D. Effect of the Scapula Reposition Test on shoulder impingement symptoms and elevation strength in overhead athletes. *J Orthop Sports Phys Ther.* 2008;38(1):4–11.
23. Schrama PPM, Stenneberg MS, Lucas C, van Trijffe. Intra-examiner reliability of hand-held dynamometry in the upper extremity: a systematic review. *Arch Phys Med Rehab.* 2014;95(12):2444–69.

24. Stark T, Walker B, Phillips JK, Fejer R, Beck R. Hand-held dynamometry correlation with the gold standard isokinetic dynamometry: a systematic review. *P.M. R.* 2011;3(5):472–9.
25. IJspeert J, Kerstens HCJW, Janssen RMJ, et al. Validity and reliability of serratus anterior hand held dynamometry. *BMC Musculoskelet Disord.* 2019;20:360.
26. Liberatori Junior RM, Netto WA, Carvalho GF, et al. (2019) Concurrent validity of handheld dynamometer measurements for scapular protraction strength. *Braz J Phys Ther* 23(3): 228–235.
27. Michener LA, Boardman ND, Pidcoe PE, Frith AM. Scapular Muscle Tests in Subjects With Shoulder Pain and Functional Loss: Reliability and Construct Validity. *Phys Ther.* 2005;85(11):1128–38.
28. Ammar TA. Inter-rater and test-retest reliability of hand held dynamometer in shoulder dysfunction. *Bull Fac Phys Ther.* 2011;16(2):115–20.
29. Hegedus EJ, Goode AP, Campbell S, et al. Physical examination tests of the shoulder: a systematic review with meta-analysis of individual tests. *Br J Sports Med.* 2008;42(2):80–92.
30. Lu Y, Okoroa KR, Patel BH, Nwachukwu BU, Baker JD, Idarraga AJ, Forsythe B. Return to play and performance after shoulder instability in National Basketball Association athletes. *J Shoulder Elbow Surg.* 2020;29(1):50–7.
31. McCarthy MM, Voos JE, Nguyen JT, Callahan L, Hannafin JA. Injury profile in elite female basketball athletes at the Women’s National Basketball Association combine. *Am J Sports Med.* 2013;41:645–51.
32. Rowe CR, Zarins B. Recurrent transient subluxation of the shoulder. *J. Bone Joint Surg. Am.* 1981;63(6):863–72.
33. Lo IK, Nonweiler B, Woolfrey M, Litchfield R, Kirkley A. An evaluation of the apprehension, relocation, and surprise tests for anterior shoulder instability. *Am. J. Sports Med.* 2004;32(2):301–7.
34. Jobe FW, Kvitne RS, Giangarra CE. Shoulder pain in the overhand or throwing athlete. The relationship of anterior instability and rotator cuff impingement. *Orthop. Rev.* 1989;18(9):963–75.
35. Speer KP, Hannafin JA, Altchek DW, Warren RF. An evaluation of the shoulder relocation test. *Am. J. Sports Med.* 1994;22(2):177–83.
36. Eshoj H, Ingwersen KG, Larsen CM, Kjaer BH, Juul-Kristensen B. Intertester reliability of clinical shoulder instability and laxity tests in subjects with and without self-reported shoulder problems. *BMJ Open.* 2018;8(3):e018472.
37. Edwards A, Chepeha J, Jones A, Sheps DM, Beaupre L. Can clinical assessment differentiate partial thickness rotator cuff tears from full thickness rotator cuff tears? A secondary analysis *Disabil Rehabil* 2019. <https://doi.org/10.1080/09638288.2018.1563637>.
38. Okoroa KR, Fidai MS, Tramer JS, Davis KD. Diagnostic accuracy of ultrasound for rotator cuff tears. *Ultrasonograph.* 2019;38(3):215–20.
39. Hudson VJ. Evaluation, diagnosis, and treatment of shoulder injuries in athletes. *Clin Sports Med.* 2010;29:19–32.
40. Yoon JP, Chung SW, Kim SH, Joo H. Diagnostic value of four clinical tests for the evaluation of subscapularis integrity. *J Shoulder Elb Surg.* 2013: 1186–92.
41. Park HB, Yokota A, Gill HS, El RG, McFarland EG. Diagnostic accuracy of clinical tests for the different degrees of subacromial impingement syndrome. *J Bone Joint Surg Am.* 2005;87:1446–55.
42. Sgori M, Loitsch T, Reichel H, Kapper T. Diagnostic value of clinical tests for Infraspinatus tendon tears. *Arthroscopy.* 2019;35(5):1339–47.
43. Collin P, Treseder T, Denard PJ, et al. What is the best clinical test for assessment of the teres minor in massive rotator cuff tears? *Clin Orthop Relat Res.* 2015;473(9):2959–66.
44. Beitzel K, Mazzocca AD, Bak K, et al. ISAKOS Upper Extremity Committee Consensus Statement on the need for diversification of the Rockwood Classification for acromioclavicular Joint Injuries. *Arthroscopy.* 2014;30(2):271–8.
45. Cools AM, Michener LA. Shoulder pain: can one label satisfy everyone and everything? *Br J Sports Med.* 2017;51:416–7.
46. Tucci HT, Martins J, de Carvalho SG, Camarini PMF, de Oliveira AS. Closed kinetic chain upper extremity stability test (CKCUES test): a reliability study in persons with and without shoulder impingement syndrome. *BMC Musculoskelet Disord.* 2014;15:1.



Functional Assessment in Elite Basketball Players

50

Nicholas D. Potter and Jeffrey B. Taylor

50.1 Introduction

Functional movement testing has become an integral and standard athlete assessment procedure at all levels of basketball. While the actual procedures may vary quite significantly, the ultimate goal tends to be the same. Athletes are asked to perform movements that replicate those that occur during their sport, with the goal of identifying subtle or gross movement deviations from what we would consider optimal. Upon identifying these aberrant motions, we may implement corrective interventions in an effort to correct the deviations and restore optimal movement characteristics.

Depending on the resources available and type of data desired, our methods of analyzing functional movement may vary greatly. In this chapter, we will discuss two methods of analysis. The first method, “clinical observation” involves the clinician observing and grading athlete quality of movement as certain functional tasks are performed. The second method is performed in a biomechanical laboratory utilizing built-in force plates and 3D motion analysis to capture both

movement quality and force production information, in a highly detailed manner, while functional movements are performed.

50.2 Clinical Observation: Functional Movement Testing

The most common method of functional movement testing is performed by a clinician observing and grading an athlete’s quality of movement while a certain functional task, or group of tasks, are performed. While expensive equipment is not required often, it is helpful to record the test using a 2D camera (such as a smartphone camera) to allow repetitive reviewing of the test, visual feedback to the athlete, and the ability to archive the movement result for later comparison. There are numerous functional movements that may be used as a test. When deciding which tests to implement, it is most appropriate to choose tests that replicate movements that commonly occur in the athlete’s sport and/or provide information that will improve athlete training for their sport.

Functional movement tests include, but are not limited to: squat, single-leg squat, single- and double-leg hop movements, vertical jump, lunge (varied directions), hurdle step, and balance tests. Clinicians often choose to utilize multiple functional movements to create a battery of tests to establish a comprehensive view of how their ath-

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letes move. In effort to quantify quality of movement, several of these test batteries have been assigned scoring systems with the goal of creating thresholds that might indicate possible injury risk and/or need for corrective interventions [1, 2].

An example of a battery of functional tests is the functional movement screen (FMS). This battery consists of seven individual tests that challenge an athlete's fundamental movement patterns and are scored based on the athlete's performance and motor control [1, 2]. Though commonly used in the clinical setting, this battery is not specific to basketball loading and movement demands. More specific to basketball, the landing error scoring system (LESS) quantifies landing biomechanics using two-dimensional video in the sagittal and frontal planes that can be taken on a tablet or other mobile device [3]. Clinicians observe the video recordings and score the athlete on 17 different criteria, including knee flexion and valgus angles and foot position when landing.

It is important to understand the current evidence and limitations of these tests, especially when it comes to predicting injury and implementing injury prevention programs. The most recent evidence in the literature has found individual tests and battery of tests are unable to reliably predict injury [4–6]. This may be because of the lack of sport specificity. The FMS does not require any jumping and landing, and the LESS only evaluates athletes during a controlled two-

legged landing though basketball injuries are most commonly seen during single-legged jump landings. However, this does not mean the tests do not provide valuable and useful data. If our goal is to train athletes to move as efficiently and effectively on the court as possible, we need to understand how they are currently moving and where they might have deficits. Understanding how the athlete moves will provide the foundation for additional investigation and ultimately the roadmap to implementing interventions to optimize performance. Furthermore, while there is not great evidence to support prediction of injury through testing, there is an abundance of evidence to support various forms of training to prevent injury [7–11].

When choosing functional movement tests to implement in basketball, one must consider the physical demands of the game and component motions that make up those demands. On the court, a basketball player performs repetitive and quick movements including dropping into a defensive stance, lateral sliding, jumping, landing, running, and changing direction. We find the squat (Fig. 50.1), single-leg squat (Fig. 50.2), and single-leg hop (Fig. 50.3) tests provide great insight into these basketball-specific movement demands. The squat test provides information on how an athlete gets into a defensive stance and their movement quality during a double-leg jump. The single-leg squat provides information regard-

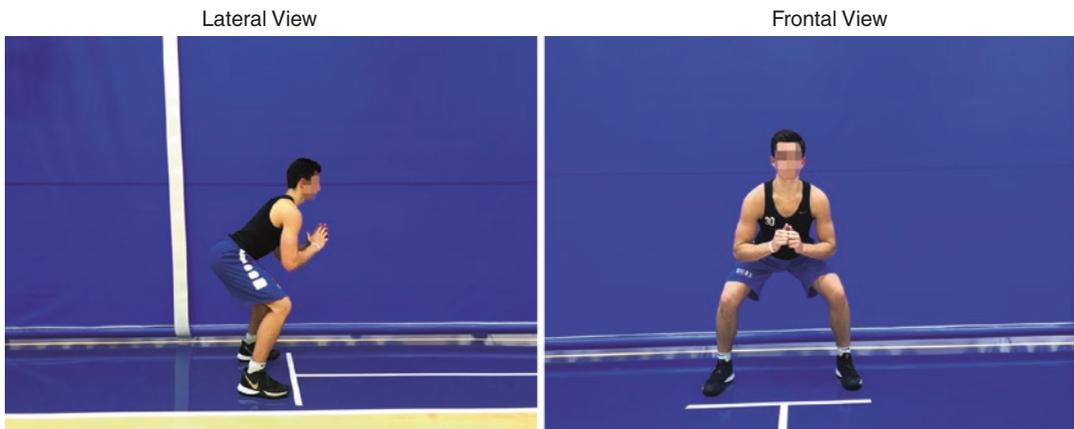


Fig. 50.1 Squat test

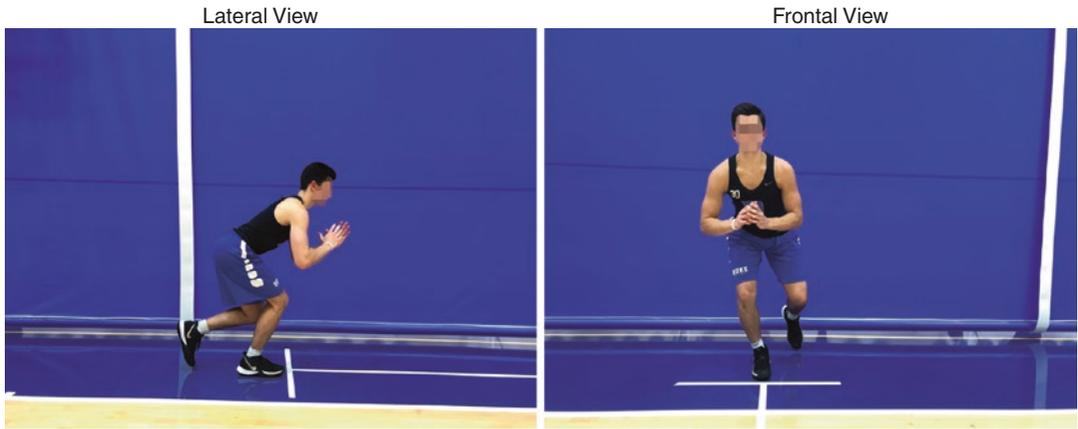


Fig. 50.2 Single-leg squat test

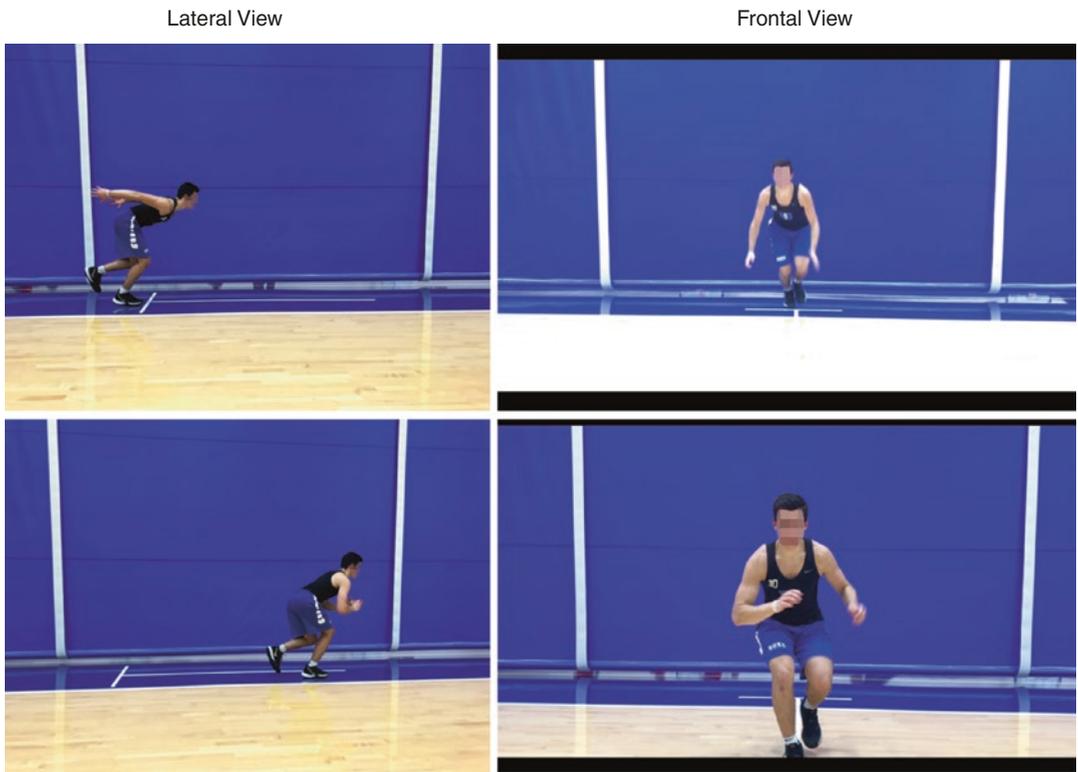


Fig. 50.3 Single-leg hop test

ing how well they can stabilize proximally while bending through the kinematic chain as during running, cutting, and single-leg jumping. The single-leg hop test demonstrates how the athlete may generate force (during take-off), dissipate force (on the landing), and what their actual single-leg jumping ability might be via the distance they can jump. The last aspect, jump distance, actually holds the least worth in our experience as this often does not correlate with jump height.

50.3 Example: Clinical Observation—Functional Movement Testing

While there are many methods and philosophies for assessing functional movement tests, we will describe one that is systematic, practical and provides applicable information. For

each test, we will begin our observation from the side (lateral view) asking the athlete to perform 10 repetitions (1–2 jumps for single-leg hop) of the desired movement. During the first set of repetitions (from the side view), we will quickly but carefully scan through a system of inspection. First, identifying the general trunk angle (in relation to the ground). Second, looking at the spine and pelvis for any deviation from neutral. Third, looking at the lower extremity for depth, knee position, and foot contact with the ground. Following observation from the side, we will move to view the athlete from the front (frontal view) asking them to perform one more set of the same movement. In this position, we are looking for asymmetries in pelvic position and lower extremity alignment (hip-knee-foot).

Boxes 1, 2, and 3 provide an order for investigating each region and associated ideal and common dysfunctional movement qualities.

Box 1

Movement test	View	Region	Ideal movement	Common dysfunctional movements
Squat test	Lateral	Trunk angle	35–55°	Vertical (>60°), hip flexion (<30°)
		Spine	Neutral spine	Thoracic flexion, lumbar flexion, increased Lordosis, posterior pelvic rotation
		Lower extremity	Knee angle 55–90°	Decreased depth (<45° knee), knees FW >2" past toes, heel lift off ground
	Frontal	Lower extremity	Aligned hip-knee-foot	Knees inward collapse (hip IR and ADD)

Box 2

Movement test	View	Region	Ideal movement	Common dysfunctional movements
Single-leg squat test	Lateral	Trunk angle	35–55°	Vertical (>60°), hip flexion (<30°)
		Spine	Neutral spine	Thoracic flexion, lumbar flexion, increased lordosis, posterior pelvic rotation
		Lower extremity	Knee angle 55–90°	Decreased depth (<45° knee), knees FW >2" past toes, heel lift off ground
	Frontal	Lower extremity	Aligned hip-knee-foot	Pelvic drop, knees inward collapse (hip IR and ADD)

Box 3

Movement test	View	Region	Ideal movement	Common dysfunctional movements
Single-leg hop (take off)	Lateral	Trunk angle	35–55°	Vertical (>60°), hip flexion (<30°)
		Spine	Neutral spine	Thoracic flexion, lumbar flexion, increased lordosis
		Lower extremity	Knee angle 55–90°	Decreased depth (<45° knee), hip flexion
	Frontal	Trunk	No rotation	Trunk rotation
Single-leg hop (landing)	Lateral	Lower extremity	Aligned hip-knee-foot	Pelvic drop, knees inward collapse (hip IR and ADD)
		Trunk angle	35–55°	Vertical (>60°), hip flexion (<30°)
		Spine	Neutral spine	Thoracic flexion, lumbar flexion
	Frontal	Lower extremity	Knee angle 55–90°	Decreased depth (<45° knee), straight knee (<15° knee)
Lower extremity		Aligned hip-knee-foot	Pelvic drop, knees inward collapse (hip IR and ADD) trunk rotation	

50.4 Laboratory-Based Movement Assessments

When resources allow, the basketball athlete may have the opportunity for movement assessment in a laboratory setting. These facilities allow for more robust and accurate analysis, but require more time, manpower, and cost. The following sections will detail the instrumentation, type of data, and specific tests that can be used to provide high-level information on a basketball player's movement pattern.

50.4.1 Motion Capture Technology

A laboratory generally utilizes 8–16 motion analysis cameras integrated with 2–3 in-ground force platforms. These cameras can collect data at up to 300 frames per second, and an elaborate software package transforms the two-dimensional coordinate system from each camera into a global three-dimensional coordinate system in the devoted motion analysis capture volume. To collect the data, the athlete must be instrumented with passive (retroreflective) or active (light emitting) markers that are placed to either define an anatomical landmark (joint) or function as a tracking marker for a segment of the body. Once instrumented, the athlete stands in a normal and

neutral standing posture as a reference for which to measure all future joint movement. Alone, the motion capture camera technology can supply angular kinematic data such as peak angles, angular excursions, and joint velocities and accelerations.

Force platforms (or plates) typically use either strain gauge or piezoelectric crystals to quantify forces at sampling rates typically around 1200 times per second. This technology can provide ground reaction forces in three dimensions (vertical, anterior/posterior, medial/lateral). Force platforms sum together all forces sensed on one platform, and in essence provide a center of pressure for those measures. Biomechanists use a method called inverse dynamics to integrate three-dimensional kinematic data with ground reaction vectors to calculate three-dimensional forces at various joints. These estimates of muscle torque production can provide valuable insight to an athlete's movement pattern. Forces can also be measured by portable, flexible, in-shoe force- or pressure-measurement systems that can allow for real-time force and/or pressure measurement during basketball activities like running, jumping, and cutting.

For a more in-depth perspective of three-dimensional motion analysis, please refer to a biomechanics textbook [12], as this section only provides a brief overview of these techniques.

50.4.2 Movement Tests

All of the movement-based tests described previously in the clinical assessment section can also be analyzed using three-dimensional motion analysis. Two-dimensional analysis can adequately identify sagittal and frontal plane kinematic during a double- and single-legged squat. Three-dimensional analysis can provide objective data to a higher level. Kinematically, higher level analyses can provide data on transverse plane motion, such as hip and knee rotations, a common element of dynamic lower extremity valgus. Joint moments can be calculated to identify torque production during the activity. Of particular interest may be the distribution of moments in the sagittal plane to identify hip- (gluteus maximus) or knee- (quadriceps) dominant movement patterns.

Three-dimensional movement analysis can also be used to quantify movement patterns during more dynamic jump landing tasks that can simulate a basketball shot or rebound. The drop vertical jump (DVJ) (Fig. 50.4a) is the gold-standard task to assess jump landing biomechanics. To perform the DVJ, participants stand on a 30-cm box with their feet spaced 35-cm apart. The athlete is instructed to fall forward off the box (without stepping or jumping), land with both feet on separate force platforms and immediately perform a maximal vertical jump. Variables of interest from the DVJ include peak knee and hip flexion angles and moments, sagittal plane angular excursions, peak knee abduction angles and moments, and the distribution of sagittal plane moments. The DVJ has been used to prospectively identify individuals at higher risk of primary [13] and secondary [14, 15] anterior cruciate ligament (ACL) injury.

The single-leg countermovement jump (Fig. 50.4b) has been reported to best elicit asymmetrical lower extremity function [16]. This test is performed with athlete balancing on one limb on a force platform. They are then asked to perform a maximal vertical jump while reaching with the opposite upper extremity for a physical target over their head that was previously set at a

height equal to their maximum double-leg countermovement jump height. Three-dimensional motion analysis of the single-leg countermovement jump can provide accurate analysis of jump height by measuring the vertical displacement of the athlete's center of mass, and similar to the other tasks, an elaborate analysis of the distribution of sagittal plane joint torques.

The single-leg lateral hop (Fig. 50.4c) integrates a number of basketball-specific demands into one task: (1) lateral demands (defending), (2) vertical demands (rebounding, defending, shooting), and (3) single-leg landings (rebounding, cutting). For this task, the athlete stands a distance equal to one half of their height away from the side of the force plate. The athlete is then instructed to balance on the outside leg, jump to the side, and land on their other leg and immediately perform a maximal vertical jump reaching for a target with the hand opposite the jumping leg [17]. Because of the basketball-specific demands, this test best differentiates the mechanics of a basketball player from athletes of other sports [18]. Additionally, this task elicits high levels of knee abduction moments and ankle inversion velocity and moments which have been identified as predictors of future knee [13] and ankle [19, 20] injury, respectively.

Repetitive hopping tests may also provide valuable clinical information. Basketball often demands consecutive vertical hops in order to defend and rebound. A 10-second single-leg hop for height test requires the athlete to jump as quickly as possible for as much height as possible in a controlled manner on one leg. Because of the single-leg nature of this test, it is easy to identify side-to-side asymmetries. Additionally, one can measure the degree of height degradation over time, with the least amount of degradation wanted. The movement quality can also be assessed as athletes often transition from a more preferred hip-dominant strategy for maximum height to a knee- or ankle-dominant strategy for quickness as they become fatigued.

Box 4 provides a summary of the most common and applicable laboratory-based functional movement tests used in the analysis of elite basketball players.



Fig. 50.4 Biomechanical lab testing. (a) Drop vertical jump (DVJ). (b) Single-leg countermovement jump. (c) Single-leg lateral hop

Box 4 Fact Box

Lab-based movement assessment tests can provide valuable information on movement quality, including joint angles and torques. Examples of basketball-specific tests include:

- Drop vertical jump
- Single-leg countermovement jump
- Single-leg lateral hop
- 10-second single-leg hop for height

50.5 Movement Correction/ Biomechanical Enhancement Philosophy

The ultimate goal of testing is to obtain data that will better inform decision-making of how to best train each athlete. Functional movement testing provides valuable information regarding “*how*” an athlete moves. While this is extremely important, it does not indicate “*why*” they are moving that way. When working with an elite athlete, there must be a comprehensive understanding of the “*why*” (meaning their underlying biomechanics) to improve “*how*” they move. Thus, it is essential to combine functional movement testing results with additional testing measures such as range of motion and strength testing in addition to their injury history. With this additional information, when an athlete displays an aberrant motion during a functional movement test, the clinician will better be able to identify if it is impacted by specific areas of tightness, weakness, or poor motor control (possibly learned compensatory movement).

Upon completion and assessment of various tests, there should be a complete picture of the athlete’s biomechanical function. At this point, the program transitions from testing and assessment into implementing the corrective and biomechanical enhancement interventions. As with any physical performance program, it is helpful

to break up the process into phases where different principles may be emphasized. Box 5 below provides a clinical example, the corrective/biomechanical enhancement program progression through 3 phases. Phase-1 involves prescribing specific interventions to correct the underlying isolated variables contributing to “*why*” they have a dysfunctional movement test. Once the isolated variables have been treated there is a progression into Phase-2, functional movement training. In Phase-2, interventions are implemented that specifically address the issues we may have found during the functional movement testing (Example interventions [Fig. 50.5a-c]). With a strong base of functional movement Phase-3 begins, basketball-specific integration. In this final phase, the goal is to optimize biomechanical carryover into high-level basketball activity. Thus, it is essential to train the athlete on the court, with a ball in their hands and moving against an opponent to stimulate environment and unanticipated nature of the game to obtain optimal carryover into game performance (Example interventions [Fig. 50.6a, b]). In recent years, it has been highly advocated that all athletes, not just those with poor movement tests, progress through the final two phases consisting of functional movement training and sports-specific training. When specific interventions enhance movement quality, reduce movement compensation, and improve biomechanical performance, resulting in potential reduced injury risk and improved functional performance on the court, then all athletes should be given them.

50.6 Summary

Functional movement testing is a valuable assessment tool that provides informative data regarding how an athlete moves. While the available resources and technology used to conduct the testing may vary, it is always essential to choose functional movements to test that replicate the



Fig. 50.5 (a) Squat series. (b) Sliders to the back. (c) Step-up drive



Fig. 50.6 (a) Lunge drive to basket. (b) Lunge drive against another player

Box 5

Movement correction/training philosophy			
Phase	Corrective category	Intervention objective	Intervention example
Phase 1	Isolated variable interventions	Joint mobility	Posterior-to-anterior hip mobilization
		Muscle extensibility	Anterior hip stretch
		Muscle strength	Hip extension (prone)
		Muscle co-activation	Hip extension with transverse abdominis stabilization (standing)
Phase 2	Functional movement training	Functional joint mobility	1. Squat series (mini band at knees) [Fig. 50.5a] – Squat – Squat to calf raise – Squat to hop 2. Sider to Back (lightly resisted) [Fig. 50.5b] 3. Step-up drive (resisted) [Fig. 50.5c]
		Functional muscle extensibility	
		Functional strength/stability	
Phase 3	Basketball specific integration	Neuromuscular coordination	1. Lunge drive (resisted) to basket with ball [Fig. 50.6a] 2. Drive against player with ball [Fig. 50.6b]
		Motor control	
		Power	
		Technical skill/awareness	
		Sport vision	

demands of high-level basketball. When considering the most recent evidence in the literature, we have to be careful not to associate testing results with injury prediction; however, it is important to know our various interventions following testing have a profound impact on injury prevention. These interventions will always have the greatest impact on health and performance when progressed into functional movement and basketball-specific training. Lastly, while it is outside the scope of this chapter, when considering the impact of movement quality on athlete performance and well-being, we must appreciate their physical loading history. The greater the loading demands placed upon an athlete, the more important it is for them to perform with optimal quality of functional movement, with minimal compensation, for prolonged success.

References

1. Cook GC, Burton L, Hoogenboom B. Pre-participation screening: the use of fundamental movements as an assessment of function—Part 1. *N Am J Sports Phys Ther.* 2006;1(2):62–72.
2. Plisky PJ, Gorman PP, Butler RJ, Liesel KB, Underwood FB, Elkins B. The reliability of an instrumented device for measuring components of the star excursion balance test. *N Am J Sports Phys Ther.* 2009;4(2):92–9.
3. Padua DA, Marshall SW, Boling MC, Thigpen CA, Garrett WEJR, Beutler AI. The landing error scoring system (LESS) is a valid and reliable clinical assessment tool of jump-landing biomechanics: the JUMP-ACL study. *Am J Sports Med.* 2009;37(10):495–505.
4. Moran RW, Schneiders AG, Mason J, Sullivan SJ. Do functional movement screen (FMS) composite scores predict subsequent injury? A systematic review with meta-analysis. *Br J Sports Med.* 2017;51:1661–9.
5. Wright AA, Dischiavi SL, Smoliga JM, Taylor JB, Hegedus EJ. Association of lower quarter Y-balance test with lower extremity injury in NCAA division I athletes: an independent validation study. *Physiotherapy.* 2017;103:231–6.
6. Hegedus EJ, McDonough S, Bleakley C, Cook CE, Baxter GD. Clinician-friendly lower extremity physical performance measures in athletes: a systemic review of measurement properties and correlation with injury, Part 1. The tests for knee function including the hop test. *Br J Sports Med.* 2014;0:1–8.
7. Lauersen JB, Anderson TE, Anderson LB. Strength training as superior, dose-dependent and safe prevention of acute and overuse sports injuries: a systematic review, qualitative analysis and meta-analysis. *Br J Sports Med.* 2018;52:1557–63.
8. Attar WSA, Soomro N, Sinclair PJ, Pappas E, Sanders RH. Effects of injury prevention programs that include the nordic hamstring exercise on hamstring injury rates in soccer players: a systematic review and meta-analysis. *Sports Med.* 2017;47:907–16.
9. Attar WSA, Soomro N, Pappas E, Sinclair PJ, Sanders RH. How effective are F-MARC injury prevention programs for soccer players? A systematic review and meta-analysis. *Sports Med.* 2016;46:205–17.
10. Lauersen JB, Bertelsen DM, Anderson LB. The effectiveness of exercise interventions to prevent sports injuries: a systematic review and meta-analysis of randomized control trials. *Br J Sports Med.* 2014;48:871–7.
11. Hubscher M, Zech A, Pfeifer K, Hansel F, Vogt L, Banzer W. Neuromuscular training for sports injury prevention: a systematic review. *Med Sci Sports Exerc.* 2010;42:413–21.
12. Roberston DGE, Caldwell GE, Hamill J, et al. *Research methods in biomechanics.* 2nd ed. Champaign, IL: Human Kinetics; 2014.
13. Hewett TE, Myer GD, Ford KR, et al. Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. *Am J Sports Med.* 2005;33(4):492–501.
14. Paterno MV, Huang B, Thomas S, et al. Clinical factors that predict a second acl injury after acl reconstruction and return to sport: preliminary development of a clinical decision algorithm. *Orthop J Sports Med.* 2017;5(12):2325967117745279.
15. Paterno MV, Schmitt LC, Ford KR, et al. Biomechanical measures during landing and postural stability predict second anterior cruciate ligament injury after anterior cruciate ligament reconstruction and return to sport. *Am J Sports Med.* 2010;38(10):1968–78.
16. Fort-Vanmeerhaeghe A, Montalvo AM, Sitja-Rabert M, et al. Neuromuscular asymmetries in the lower limbs of elite female youth basketball players and the application of the skillful limb model of comparison. *Phys Ther Sport.* 2015.
17. Taylor JB, Ford KR, Nguyen AD, et al. Biomechanical comparison of single- and double-leg jump landings in the sagittal and frontal plane. *Orthop J Sports Med.* 2016;4(6):2325967116655158.
18. Taylor JB, Ford KR, Schmitz RJ, et al. Biomechanical differences of multidirectional jump landings among female basketball and soccer players. *J Strength Cond Res.* 2017;31(11):3034–45.
19. Fong DT, Ha SC, Mok KM, et al. Kinematics analysis of ankle inversion ligamentous sprain injuries in sports: five cases from televised tennis competitions. *Am J Sports Med.* 2012;40(11):2627–32.
20. Fong DT, Hong Y, Chan LK, et al. A systematic review on ankle injury and ankle sprain in sports. *Sports Med.* 2007;37(1):73–94.



Injury Prevention in Basketball

51

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51.1 Introduction

Basketball is a non-contact sport that involves fast acceleration and deceleration with cutting manoeuvres and high physical demands that may expose the participant to the risk of injury. Increased knowledge of injury prevention is an important mission, as basketball is one of the most popular sports in the world at all levels, among both men and women and the young and old. To be able to minimise the overall risk of injuries, prevention strategies for both acute and over-use injuries must be implemented and, optimally, also individualised.

In basketball, injuries to the lower extremities are by far the most common. In a newly performed systematic review [1], it was shown that 63.7% of all injuries, irrespective of gender and level, were seen in the lower limbs; 21.9% in the ankle and 17.8% in the knee. Wrist, hand, and finger injuries were most common in the upper extremities, even if only 12–14% of all injuries occurred in the upper limbs. However, the injury rates differed between genders and level of play (Table 51.1).

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Table 51.1 Percentage of men, women, professional players, and children and adolescents who sustained an ankle or knee injury [1]

Injury	Men	Women	Professional	Children and adolescents
Ankle (%) of all injuries	14.6	19.5	17.5	25.6
Knee (%) of all injuries	17.5	20.6	19.5	16.3

For professional athletes playing in the National Basketball Association (NBA), a 17-year follow-up showed that 50% of the injuries were game related and the ankle was the most injured body part [2]. Surprisingly, no correlations were found between the injury rate and the demographics of the players, or their experience of playing in the NBA.

On the other hand, in recreational basketball players, it has been found that defending, weight over 75 kg, increased postural sway, and high vertical ground reaction force are significantly correlated with an increased risk of injury [3].

Fact Box

In basketball, ankle and knee injuries are the most common injuries in both female and male players.

51.2 Risk Factors

When it comes to preventing injuries, one important aspect is being able to identify the risk factors both generally and individually. Injuries can be caused by both trauma and overload. Either way, the injury risk increases when the applied load and the capacity of the player are unbalanced. So, in order to prevent injuries in basketball, it is as important to screen basketball players individually for physical and psychological deficits as it is to take control of the applied load. If the applied load is too high for the players' capacity, there is an increased risk of injury. On the other hand, if the load is too low, the training has no effect.

51.2.1 The Importance of Optimal Load

In 2015, the International Olympic Committee (IOC) held a consensus meeting for scientific evidence in the field of associations between load and healthiness in sport. The IOC defined the concept of load as “the sport and non-sport burden (single or multiple physiological, psychological, or mechanical stressors) as a stimulus that is applied to a human biological system (including subcellular elements, a single cell,

tissues, one or multiple organ systems, or the individual)” [4]. A load can be internal or external and, in order to avoid injuries due to too high load, it is important to monitor both internal and external loads. Examples of internal load include sleep, heart rate, perception of exercise, and psychological well-being. External loads can include training and competition type, frequency and time of training, and competition and neuromuscular function [4, 5]. Putting it all together, in order to increase capacity, the total load applied to the player must be followed by an appropriate time of recovery (Fig. 51.1).

A useful, reliable, and valid tool for monitoring the total training load is the session ratings of perceived exertion (sRPE) [6, 7]. This method includes both internal and external load monitoring. The session RPE is a quota between the external load in terms of time and the internal load in terms of the rate of perceived exertion on the RPE scale (Fig. 51.2) [7]. The sRPE has been endorsed when scheduling training loads for basketball players [8]. One of its qualities is that it is very simple to use. About 15–30 min after training, the coach asks the athlete: “How was your training?” and the individual answers using a numerical score between 1 and 10 according to the RPE scale. It is then possible to see whether the training

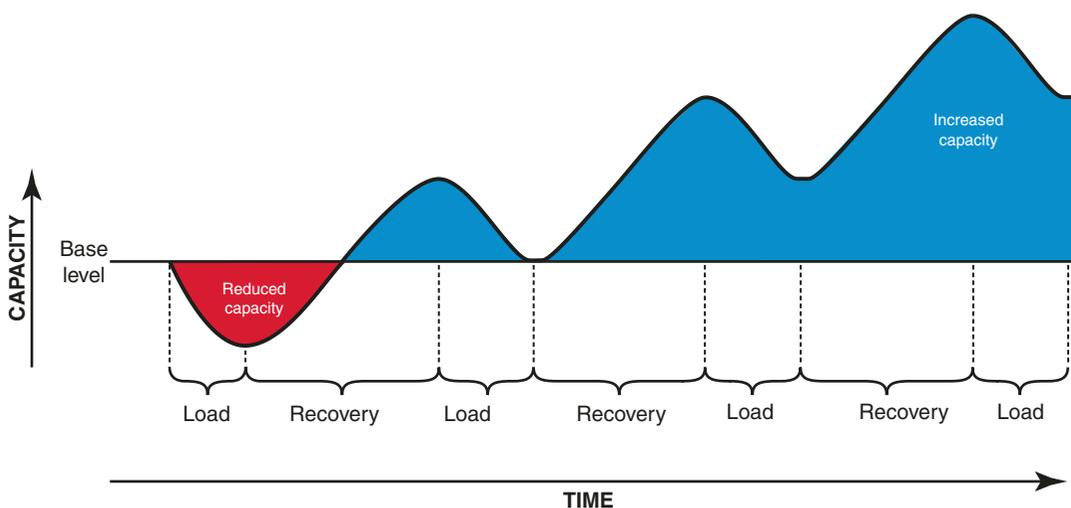


Fig. 51.1 Example of appropriate loads and recovery times resulting in increased capacity

RATING OF PERCEIVED EXERTION (RPE SCALE)	
10	Maximal
9	Really, Really Hard
8	Really Hard
7	
6	Hard
5	Challenging
4	Moderate
3	Easy
2	Really Easy
1	Rest

Fig. 51.2 RPE scale [7]

load is getting too high with regard to the exercises performed [9].

51.2.2 Risk Factors in Professional Athletes

One of the greatest risk factors for injuries is previous injuries. A muscle or a ligament might have healed and be pain free, but it has not acquired the strength needed to return to play [10].

When evaluating injury risk factors resulting in missing or leaving a game in 627 players in the NBA in 2012–2015, the results showed that a higher performance load (frequency of rebounds and field goal attempts), fatigue (accumulated competition minutes and number of days rest), and more years of playing in NBA were identified as risk factors [11]. In this study, it was shown that, for every 96 min of playing, the risk of injury increased by 2.9% and, for each day of

rest, it decreased by 16%. The performance load was related to an increased injury risk. When a player increased the number of rebounds by three above his/her average, the odds of injury also increased by 8.2%. Every three field goal attempts above the player's average increased the odds of injury by 9.9%. Another interesting result was that, for every year played in the NBA, the risk of injury in the first game of the season increased by 3.0%. Height appeared to affect injury risk as well, as similar players with a 6 cm height difference had different odds of sustaining an injury by 10.6% in favour of the taller players [11].

In the National Collegiate Athletic Association (NCAA), the overall injury rate was higher for men than women (7.9 vs. 8.2 per 1000 athletic exposures (AE), respectively). Overall, non-time-loss injuries were more frequent than time-loss injuries. Both in practice and in competition, the rate of time-loss injuries was higher in men than in women (3.2 vs. 2.9 per 1000 AE, respectively). Contact with another player may be a common cause of injury in men, while non-contact and over-use injuries are more typical mechanisms of injury in women [12].

51.2.3 Risk Factors in Young Players

Injury risk for younger participants differs from that in adults. Growth can make the cartilage and the soft tissues more vulnerable in younger people than in adults. During growth, there are anatomical and physiological factors to consider, where growth plates might be affected, as well as muscle strength and flexibility [10]. In general, it has been shown that, in young athletes (between 5 and 17 years), traumatic injuries are more common in boys, while over-use injuries are more common in girls [13, 14].

Biomechanics play an important role when executing specified movements such as jumping/landing, cutting manoeuvres, and acceleration/deceleration. The differences between boys' and girls' performances during these activities may

Fig. 51.3 A list of advice for preventing injuries in young players published by Straccolini et al. 2017 [10]

- Encourage general health maintenance and fitness year-round
- Suggest temporary reductions or restructuring in training load to allow for more adequate rest between training sessions during vulnerable periods of growth
- Educate young women athletes and their parents about neuromuscular training programs to reduce anterior cruciate ligament and other lower extremity injuries
- Encourage participation in neuromuscular training programs aimed at building hip and core strength especially for young prepubescent girl athletes (optimally before signs of injury)
- Encourage proper sleep hygiene including maximizing sleep quantity and quality each night
- Encourage plenty of time for free play, “cross training,” and sport diversity early in childhood (delaying specialization in a single sport until late adolescence)
- Encourage proper sport-specific gear use and fit
- Enforce early recognition of sport-related concussion and removal from competition until medical evaluation, treatment, and clearance for return to sport criteria have been met
- Educate young athletes about the symptoms of concussions and encourage them to report symptoms promptly to coaches, trainers, and parents
- Support and advocate for policies that promote fair play and rule enforcement as well as youth sports schedules that do not compromise sleep

be part of the explanation of why ACL injuries are more common in girls than in boys, comparing the same sport [10]. A useful list of advice for preventing injuries in young players was published by Straccolini et al. in 2017 [10] (Fig. 51.3).

Fact Box

- The injury risk increases when the applied load and the capacity of the player are unbalanced.
- A useful tool for monitoring total training load is the session ratings of perceived exertion (sRPE).
- One of the strongest risk factors for injuries is previous injuries.
- It has to be remembered that the injury risk for young basketball players differs from that for adults.

an athlete is ready to return to play [16]. The optimal way would be to make an assessment at the beginning of the (pre-)season to have a baseline for the athletes’ functional status. Even though screening tests do not appear to be able to identify injury risk, the tool might be valuable for a return to sports/play evaluation [17].

51.3.1 Prevention Programmes for the Lower Extremities

Many neuromuscular prevention programmes have been created for athletes and quite a few have shown good results [15]. One difficulty is often finding time to implement these programmes in the training sessions.

51.3.1.1 11+ Programme

One programme that has been shown to be effective in reducing injury rates in the lower extremities among basketball players is the 11+ (formerly known as the FIFA 11+) [19], originally designed for football (soccer) players [18]. It has subsequently been shown that this programme also functions well for other sports where cutting manoeuvres, jumping, and acceleration/deceleration are prominent. Specifically, the programme has been shown to be effective in preventing injuries in elite male basketball players [19].

51.3 Prevention of Injuries

There is some evidence that preventive actions and programmes tailored to lower leg injuries may prevent general injuries in the lower leg, as well as ankle sprains, but not anterior cruciate ligament (ACL) injuries in basketball players [15]. Screening and functional movement analysis might also be a good way to evaluate whether

Table 51.2 The Warm-up Exercise Program, published by Longo et al. [19], has been shown to be effective in reducing the injury rate among elite basketball players

Exercise	Repetitions
I. Running exercises, 8 min (along the major diameter of the basketball court, about 28 m)	
Running straight ahead	10
Running, hip out	2
Running, hip in	2
Running, circling	2
Running and jumping	2
Running, quick run	2
II. Strength, plyometrics, balance, 15 min	
Bending with both legs	10 × 3
Nordic hamstrings lower	10 × 3
Single-leg balance	
Level 1: holding ball	2 (each leg)
Level 2: throwing ball with partner	3 (each leg)
Level 3: testing partner	3 (each leg)
Squats	
Level 1: with heels raised	2 × 30 s
Level 2: walking lunges	2 × 30 s
Level 3: 1-leg squats	2 × 10 (each leg)
Jumping	
Level 1: vertical jumps	3 × 15 s
Level 2: lateral jumps	Three times along the major diameter of the basketball court
Level 3: box jumps	Three times along the major diameter of the basketball court
III. Running exercises, 1 min and 40 s (along the major diameter of the basketball court, about 28 m)	
Running over pitch	3
Bounding run	3
Running and cutting	3

The programme, which consists of different warm-up exercises, is designed to take 25 min to complete and is recommended to be performed twice a week (Table 51.2) [18].

51.3.1.2 Special Prevention Programmes for Ankle Injuries

For basketball players in the NBA, the overall risk of sustaining an ankle sprain is 26% in every season [20], so the need for prevention actions is high. The prevention of ankle sprains in basketball could take the form of either programmes to improve balance and/or proprioception in the

ankle or external ankle braces or a combination of both.

In a randomised, controlled trial, Eils et al. [21] showed that a 20-min programme comprising six different stations with balance and proprioception exercises performed once a week was able to reduce the risk of an ankle sprain by 35% in 198 basketball players. The exercises were performed barefoot and consisted of single-leg balance exercises on different surfaces, as well as jumping exercises with and without a basketball.

External ankle devices could be braces, taping, or high-top shoes. There is no consensus on whether a brace or taping is superior, but it has been suggested that an ankle brace is preferable to high-top shoes [15].

51.3.1.3 Special Prevention Programmes for Knee Injuries

A severe injury that may be career ending is an ACL injury. The need for prevention programmes for ACL injuries in basketball is highly desirable, as the injury rate for ACL injuries in basketball is rising, especially among female athletes [22]. The prevention programmes for an ACL injury in basketball focus primarily on neuromuscular training, including landing strategies. Unfortunately, these programmes may not be effective enough to prevent ACL injuries in female basketball players compared with female athletes in football (soccer) or handball [23]. The reason for this is not fully understood, but it has been suggested that the prevention programmes need to be more specifically designed for the typical demands in basketball, in terms of biomechanical requirements, and that the same programs are tested across sports to allow for accurate comparison [23, 24]. However, a recent 12-year prospective intervention study reported promising results in preventing ACL injuries in female basketball players [25]. This prevention programme was implemented for 8 years in 448 basketball players who performed the programme three times a week, for 20 min each session. The aim was to improve hip joint function through balance exercises, strength training for hip muscles, and landing strategies after jumping. This programme was shown to reduce the rate of ACL injuries significantly [25].

51.3.2 Prevention Programmes for Concussion

A concussion is a severe head injury that can sometimes even lead to the end of a sports career. A mouthguard has been proposed as a safety instrument in contact sport, and it appears to reduce the risk of injury by up to 19%, as well as reducing the risk of orofacial injury. Changing policies in (youth) basketball might also be of benefit, as has been done in youth ice hockey, where regulations in terms of body checking were changed [26].

51.3.3 Prevention Programmes for Young Players

Young athletes benefit from preventive programmes, but, as in adults, it can be difficult to find the time. A practical solution might be to perform the programmes at school instead of doing them at practice. It has been shown that implementing neuromuscular programmes in schools throughout the season reduces the lower leg injury incidence significantly in young basketball players [27].

Fact Box

- There is evidence that preventive action and programmes tailored to lower leg injuries are effective.
- One effective special prevention programme for lower extremity injury prevention is the 11+ programme (formerly known as the FIFA 11+).
- Balance and/or proprioception exercises and/or external ankle braces are effective in preventing ankle sprains.
- Neuromuscular training exercises may prevent knee injuries in male players, while the effect for female players is not yet significant.
- Mouthguards may reduce the risk of concussion.
- Implementing neuromuscular programmes in schools will reduce lower leg injury incidence significantly for young basketball players.

Take-Home Message

- In order to prevent injuries in basketball, it is as important to screen basketball players individually for physical and psychological deficits as it is to take control of the applied load.
- A useful, reliable, and valid tool for monitoring the total training load is the session ratings of perceived exertion (sRPE).
- Prevention programmes are effective in preventing lower leg injuries in basketball players.

References

1. Andreoli CV, Chiamonti BC, Buriel E, Pochini AC, Ejnisman B, Cohen M. Epidemiology of sports injuries in basketball: integrative systematic review. *BMJ Open Sport Exerc Med.* 2018;4(1):e000468.
2. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the national basketball association: a 17-year overview. *Sports Health.* 2010;2(4):284–90.
3. Kilic O, Van Os V, Kemler E, Barendrecht M, Gouttebauge V. The ‘Sequence of Prevention’ for musculoskeletal injuries among recreational basketballers: a systematic review of the scientific literature. *Phys Sportsmed.* 2018;46(2):197–212.
4. Soligard T, Swellnus M, Alonso JM, Bahr R, Clarsen B, Dijkstra HP, et al. How much is too much? (part 1) International Olympic Committee consensus statement on load in sport and risk of injury. *Br J Sports Med.* 2016;50(17):1030–41.
5. Swellnus M, Soligard T, Alonso JM, Bahr R, Clarsen B, Dijkstra HP, et al. How much is too much? (part 2) International Olympic Committee consensus statement on load in sport and risk of illness. *Br J Sports Med.* 2016;50(17):1043–52.
6. Foster C, Florhaug JA, Franklin J, Gottschall L, Hrovatin LA, Parker S, et al. A new approach to monitoring exercise training. *J Strength Cond Res.* 2001;15(1):109–15.
7. Foster C. Monitoring training in athletes with reference to overtraining syndrome. *Med Sci Sports Exerc.* 1998;30(7):1164–8.
8. Sansone P, Tschan H, Foster C, Tessitore A. Monitoring training load and perceived recovery in female basketball: implications for training design. *J Strength Cond Res* 2018.
9. Turner AN, Bishop C, Marshall G, Read P. How to monitor training load and mode using sRPE. *Prof Strength Cond.* 2015;39:15–20.

10. Straccolini A, Sugimoto D, Howell DR. Injury prevention in youth sports. *Pediatr Ann.* 2017;46(3):e99–e105.
11. Lewis M. It's a hard-knock life: game load, fatigue, and injury risk in the National Basketball Association. *J Athl Train.* 2018;53(5):503–9.
12. Zuckerman SL, Wegner AM, Roos KG, Djoko A, Dompier TP, Kerr ZY. Injuries sustained in National Collegiate Athletic Association men's and women's basketball, 2009/2010-2014/2015. *Br J Sports Med.* 2018;52(4):261–8.
13. Straccolini A, Casciano R, Friedman HL, Meehan WP 3rd, Micheli LJ. A closer look at overuse injuries in the pediatric athlete. *Clin J Sport Med.* 2015;25(1):30–5.
14. Straccolini A, Casciano R, Levey Friedman H, Stein CJ, Meehan WP 3rd, Micheli LJ. Pediatric sports injuries: a comparison of males versus females. *Am J Sports Med.* 2014;42(4):965–72.
15. Taylor JB, Ford KR, Nguyen AD, Terry LN, Hegedus EJ. Prevention of lower extremity injuries in basketball: a systematic review and meta-analysis. *Sports Health.* 2015;7(5):392–8.
16. Bird SP, Markwick WJ. Musculoskeletal screening and functional testing: considerations for basketball athletes. *Int J Sports Phys Ther.* 2016;11(5):784–802.
17. Bond CW, Dorman JC, Odney TO, Roggenbuck SJ, Young SW, Munce TA. Evaluation of the functional movement screen and a novel basketball mobility test as an injury prediction tool for collegiate basketball players. *J Strength Cond Res.* 2019;33(6):1589–600.
18. Soligard T, Myklebust G, Steffen K, Holme I, Silvers H, Bizzini M, et al. Comprehensive warm-up programme to prevent injuries in young female footballers: cluster randomised controlled trial. *BMJ.* 2008;337:a2469.
19. Longo UG, Loppini M, Berton A, Marinozzi A, Maffulli N, Denaro V. The FIFA 11+ program is effective in preventing injuries in elite male basketball players: a cluster randomized controlled trial. *Am J Sports Med.* 2012;40(5):996–1005.
20. Herzog MM, Mack CD, Dreyer NA, Wikstrom EA, Padua DA, Kocher MS, et al. Ankle sprains in the National Basketball Association, 2013-2014 through 2016-2017. *Am J Sports Med.* 2019;47(11):2651–8.
21. Eils E, Schroter R, Schroder M, Gerss J, Rosenbaum D. Multistation proprioceptive exercise program prevents ankle injuries in basketball. *Med Sci Sports Exerc.* 2010;42(11):2098–105.
22. Agel J, Rockwood T, Klossner D. Collegiate ACL injury rates across 15 sports: National Collegiate Athletic Association injury surveillance system data update (2004-2005 through 2012-2013). *Clin J Sport Med.* 2016;26(6):518–23.
23. Michaelidis M, Koumantakis GA. Effects of knee injury primary prevention programs on anterior cruciate ligament injury rates in female athletes in different sports: a systematic review. *Physical therapy in sport: official journal of the Association of Chartered Physiotherapists in Sports Med.* 2014;15(3):200–10.
24. Taylor JB, Ford KR, Schmitz RJ, Ross SE, Ackerman TA, Shultz SJ. Sport-specific biomechanical responses to an ACL injury prevention programme: a randomised controlled trial. *J Sports Sci.* 2018;36(21):2492–501.
25. Omi Y, Sugimoto D, Kuriyama S, Kurihara T, Miyamoto K, Yun S, et al. Effect of hip-focused injury prevention training for anterior cruciate ligament injury reduction in female basketball players: a 12-year prospective intervention study. *Am J Sports Med.* 2018;46(4):852–61.
26. Emery CA, Black AM, Kolstad A, Martinez G, Nettel-Aguirre A, Engebretsen L, et al. What strategies can be used to effectively reduce the risk of concussion in sport? A systematic review. *Br J Sports Med.* 2017;51(12):978–84.
27. Foss KDB, Thomas S, Khoury JC, Myer GD, Hewett TE. A school-based neuromuscular training program and sport-related injury incidence: a prospective randomized controlled clinical trial. *J Athl Train.* 2018;53(1):20–8.

Best Evidence Based References

- Kilic O, Van Os V, Kemler E, Barendrecht M, Goutteborge V. The 'Sequence of Prevention' for musculoskeletal injuries among recreational basketballers: a systematic review of the scientific literature. *Phys Sportsmed.* 2018;46(2):197–212.
- Longo UG, Loppini M, Berton A, Marinozzi A, Maffulli N, Denaro V. The FIFA 11+ program is effective in preventing injuries in elite male basketball players: a cluster randomized controlled trial. *Am J Sports Med.* 2012;40(5):996–1005. <https://doi.org/10.1177/0363546512438761>.
- Soligard T, Schweltnus M, Alonso JM, Bahr R, Clarsen B, Dijkstra HP, et al. How much is too much? (Part 1) International Olympic Committee consensus statement on load in sport and risk of injury. *Br J Sports Med.* 2016;50(17):1030–41. <https://doi.org/10.1136/bjsports-2016-096581>.
- Schweltnus M, Soligard T, Alonso JM, Bahr R, Clarsen B, Dijkstra HP, et al. How much is too much? (Part 2) International Olympic Committee consensus statement on load in sport and risk of illness. *Br J Sports Med.* 2016;50(17):1043–52. <https://doi.org/10.1136/bjsports-2016-096572>.
- Taylor JB, Ford KR, Nguyen AD, Terry LN, Hegedus EJ. Prevention of lower extremity injuries in basketball: a systematic review and meta-analysis. *Sports Health.* 2015;7(5):392–8. <https://doi.org/10.1177/1941738115593441>.

Digging Deep into the Etiology of Basketball Injuries: A Complex Systems Approach for Risk Mitigation

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52.1 Basketball Injury Prevention Situation Room

Basketball is a fast-paced sport involving repeated jumping, landing, pivoting, cutting, running back and forth, and moderate contacts among players; all of which makes participants susceptible to injuries. Injury prevention in basketball is essential for continued player participation, performance, team success, and future health [1–5]. Depending on injury definition and level of play, injury rates are substantial in basketball [6–9]. Studies on the risk factors for basketball injury are currently limited. Female sex, game (vs. practice), offense position (vs. defense), and injury history are currently reported to be associated with injury outcomes [8, 10, 11]. Based on best available evidence, primary targets for injury prevention in basketball are exercise-based neuromuscular training warm-up programs [11–16], workload mediation [17–21] and equipment strategies (e.g., mouthguards) [22, 23]. Screening

as a “first layer” prediction tool for injury prevention (to determine who should have prevention intervention—all players should) in team sports has been recently discouraged in scientific discussions considering the multifaceted nature of sports injury [24]. However, screening for the right reasons, e.g., as a “second layer” monitoring tool appears to be valuable for effective risk management in team clusters and individual players; more like identifying who needs extra attention based on player risk profiles [25, 26].

Sports injuries occur when intrinsic and extrinsic risk factors (e.g., playing environment, equipment) interact with an inciting event (e.g., cumulative tissue overload, collision) in a timely fashion [27, 28]. Irrespective of the number of risk factors a player possesses (intrinsic factors), none is capable of causing an injury until a player is exposed to basketball training (practice or strength sessions) or competition/game [29]. The opportunity for injury, that is, potential inciting events, only comes when players are exposed to basketball load. Recognizing basketball load as a primary exposure is thus expedient to understand the etiology of injury or the development of fitness performance in players [29]. Furthermore, an understanding of the interactions between the impact of basketball load and player capacity (load tolerance), and other intrinsic/extrinsic factors is crucial to avoid injuries and build fitness [29–31]. Surrogate measures for player/tissue capacity

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for load, and load magnitude and distribution have been suggested [32]. Although player capacity and load magnitude and distribution have not been fully studied in traditional regression analyses, they are key elements that contribute to the biologically plausible mechanism of not just the emergence of sports injuries but also performance outcomes [32]. Sports injury is a multidimensional emergent phenomenon and a complex systems approach is crucial to unravel its emergence [33, 34].

First, this chapter describes the characteristics of a complex system and provides a rationale for its application in basketball injury etiology and prevention. Next, a review of current models/frameworks for sports injury etiology and injury prevention is presented and a novel Complex Model for the Etiology of Basketball Injury (CMEB) is proposed. Finally, potential applications of the proposed model in basketball injury prevention research and practice are discussed.

52.2 What Is a Complex System and why Is it Important in Basketball Injury Prevention?

A complex system “attempts to understand the underlying processes along with the overall functioning of a system in relation to its principles, rather than to identify individual causal effects between isolated parameter estimates [33].” Briefly, the characteristics of a complex system include: (1) an open system capable of producing several pathways to similar emerging patterns (i.e., outcomes); (2) non-linearity in which a system may respond in different ways to the same input depending on their state or prevailing context; (3) feedback or recursive loops in which system output is reprocessed to become a new input; (4) self-organization in which there is a spontaneous occurrence of order within the system and interactions among system units, and not prediction from individual units, to produce regularities and emerging patterns; (5) uncertainty due to the complex phe-

nomenon of self-organization [28, 35, 36]. Additionally, a complex system models three key concepts that are applicable in sports: (1) interacting units that constitute the “web of determinants;” (2) regularities or profiles; and (3) emerging patterns [36].

Evidence underpinning the development of most of the injury prevention strategies currently available in sports has relied on linear models of cause and effects [27, 28, 37, 38]. This is analogous to giving a simple answer to a complex question; perhaps one of the reasons why the large protective effects demonstrated in clinical trials for existing injury prevention interventions do not reflect in the real world [39–41]. A focus on “isolated” risk factors, a simple reductionist approach that excludes variables that are not related to, or do confound relationships with injury outcome, is insufficient to explain injury mechanism [32, 33, 36]. Rather, focus should be on recognizing “integrated” risk patterns (i.e., based on interactions between key factors) from conceptually plausible (i.e., theory-informed) injury mechanisms [33, 36, 42, 43]. Consequently, a multimodal intervention comprising discrete evidence-based strategies, rather than a single injury prevention strategy (as mostly practiced), is likely to be more impactful. A complex systems approach to basketball injury prevention provides the platform to advance the evidence base towards the development of more robust interventions targeting the multiple factors precipitating injury.

52.3 Modelling Sports Injury Etiology and Prevention: The Journey So Far

From an epidemiological perspective, musculoskeletal injury prevention in basketball entails an understanding of the burden and etiology of injuries and subsequent development and implementation of context-specific injury countermeasures. The four-step model for injury prevention proposed by van Mechelen et al. has been used by researcher over the last 25 years to address the aforementioned perspective up to the stage of

intervention development and laboratory evaluation [38]. The van Mechelen model however does not address steps to moving injury prevention evidence into practice. To address this limitation, Finch et al. proposed the Translating Research into Injury Prevention Practice Framework—this adds two supplementary steps to direct the translation of research into injury prevention practice, including a description of the intervention context to inform implementation strategies (step five) and evaluation of the intervention in real-world contexts (step 6) [37]. However, the problem of underuse and suboptimal adherence to “proven” injury prevention interventions continues to be a challenge, instigating a crucial need for more rigorous implementation research in the field of sports injury prevention. To advance the field of implementation research in sports injury prevention, Owoeye et al. proposed a four-step model to optimize adherence to sports injury prevention interventions [44]. This model recommends the identification and modification of the determinants of intervention adherence for successful real-world implementation and maintenance.

In addition to injury prevention models, research in sports injury prevention have been guided by injury etiology models [27–29, 36]. Etiological models are fundamental for understanding the mechanism of injury occurrence, a crucial step for developing injury countermeasures. Popular in this field is the earlier model proposed by Meeuwisse et al. [27] which was subsequently updated to the dynamic, recursive model of etiology in sport injury to reflect the dynamic nature of injury etiology due to repeated player exposure [28]. However, successive injury etiology models have indicated additional limitations in the dynamic, recursive model, including its simplistic and reductionist approach [36, 45] and lack of consideration for player workload, a key determinant of injury [29]. Recently, non-linear complex systems approach has been proposed to better understand the etiology of sports injuries [32, 33, 36, 45, 46]. Of note is the Complex Systems Model by Bittencourt et al. describing sports injuries as complex emergent phenomena resulting from dynamic interactions among a web of determi-

nants that are capable of producing regularities in the form of a risk profile and subsequently prompting an emergent pattern—an injury [36]. There is no doubt that current etiological models continue to contribute towards evidence-based sports injury risk mitigation; however, reviewing existing models to fit advancements in knowledge and to be sport specific is imperative to maximize outcomes.

52.4 A Complex Systems Approach for Basketball Injury Risk Mitigation

Predicated on Bittencourt et al.’s Complex Systems Model [36] and current literature [28, 29, 32, 33], the CMEB is proposed to capture the essential elements and process that elucidate the mechanisms involved in the generation of basketball injuries (Fig. 52.1). The CMEB explains that at any given time, a player carries a unique risk profile that determines their susceptibility to injury. This risk profile is strongly dependent on the constituents and characteristics of individual-player web of determinants. The occurrence of an injury in the face of an inciting event (e.g., collision, overuse, tissue overload) is subsequently determined by a player’s current physiological state of either fatigue/maladaptation from negative training effects (e.g., ongoing basketball-related load exceeding player/tissue tolerance in combination with other risk factor interactions) or physical fitness/adaptation by way of supercompensation from positive training effects, including protective effects from injury prevention intervention(s) (e.g., neuromuscular training warm-up exercise programs), if applicable. In reflection of real-world sporting environment of repeated events of training and games, a player re-enters the model in a recursive loop with slight to wide variations in their injury risk profile each time they engage in basketball-related activities. A variation towards increased injury risk is expected when a previously injured player returns to play post-rehabilitation, especially when not fully recovered from injury.

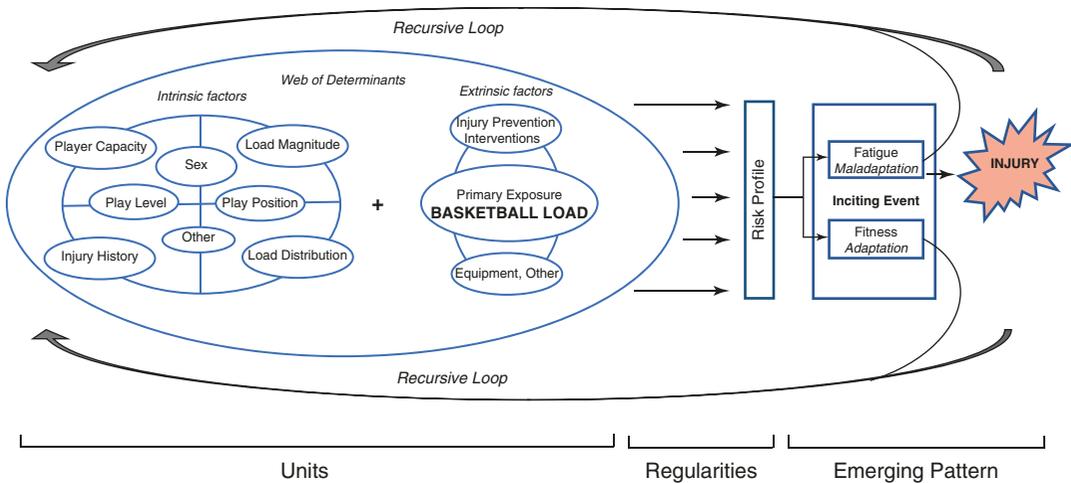


Fig. 52.1 A complex model for the etiology of basketball injury. Variables larger in size potentially have higher interactions and they exert a greater influence on injury outcomes. Figure is based on reference [28, 29, 32, 33, 36]

In the CMEB, key effect modifiers: player capacity for basketball-related load, the potential impact of the magnitude and distribution of load on major joints and other body structures, and history of a previous time-loss injury are considered crucial moderators of the load-injury relationship that contribute to the biologically plausible mechanism of basketball injury emergence or improved performance outcomes. Surrogate measures for these factors may include aerobic fitness, measures of lower extremity strength and wellness (including sleep and mental wellness) for player/tissue capacity, knee and/or ankle range of motion for load distribution and body weight and measures of adiposity for load magnitude [32].

52.5 Perspectives on the Application of the CMEB in Research and Practice

As demonstrated in the CMEB, several factors determine whether a player gets injured each time they are exposed to basketball load and potential inciting events that may precipitate injury. The CMEB supports the novel concept that injury prevention attempts in team sports should be directed towards injury risk profiles/

pattern recognition in players [36, 42, 43] and subsequent injury risk mitigation through risk management approaches implemented not only at the team level but also at individual player level [25]. One of the most important questions that should be paramount to coaches and the medical team, from community basketball to the National Basketball Association, is: “how are my players handling daily basketball load, particularly the training load impacted on them?” It is not enough to simply throw intense training at athletes and assume they will/can handle it. The problem is, in basketball, as in other team sports, most times the overall external load of about the same volume is imposed on a group of players with varying risk profiles; thus, the external load potentially invokes different internal load responses. The CMEB provides the opportunity to advance knowledge towards developing multifactorial injury prevention strategies to keep basketball players in the “low risk” zone essential to keep them fit and resilient in the face of events that may incite injury.

In the CMEB, load is considered the primary exposure that is moderated by other risk factors to predict injury. This CMEB provides the platform for a predictive model to stratify injury risk in individual players. In essence, a player monitoring system will have to be in place to obtain relevant data to inform a “second layer” risk miti-

gation strategy in individual players. For example, a weekly evaluation of load-injury relationship, considering the moderating effects of relevant risk factors (measured at baseline or every two months) in individual players, may inform effective load management programs such as minutes restriction in certain players. Sports injury prevention researchers should consider exploring the CMEB (an adaptation for other team sports may be considered) for a complex modelling of load-injury relationships. Depending on researchers' or practitioners' statistical prowess, data analytics to test/utilize the CMEB, including examining effect modification by modifiable risk factors (i.e., influence of key factors on load-injury relationship) may range from stratified analyses [42, 43], and test of interactions in regression models [11] to more complex statistical models such as machine learning [47, 48].

References

1. Roos EM. Joint injury causes knee osteoarthritis in young adults. *Curr Opin Rheumatol*. 2005;17(2):195–200. <https://doi.org/10.1097/01.bor.0000151406.64393.00>.
2. Valderrabano V, Hintermann B, Horisberger M, Fung TS. Ligamentous posttraumatic ankle osteoarthritis. *Am J Sports Med*. 2006;34(4):612–20. <https://doi.org/10.1177/0363546505281813>.
3. Eirale C, Tol JL, Farooq A, Smiley F, Chalabi H. Low injury rate strongly correlates with team success in Qatari professional football. *Br J Sports Med*. 2013;47(12):807–8. <https://doi.org/10.1136/bjsports-2012-091040>.
4. Whittaker JL, Toomey CM, Nettel-Aguirre A, et al. Health-related outcomes after a youth sport-related knee injury. *Med Sci Sports Exerc*. 2019;51(2) <https://doi.org/10.1249/MSS.0000000000001787>.
5. Williams S, Trewartha G, Kemp SPT, et al. Time loss injuries compromise team success in Elite Rugby Union: a 7-year prospective study. *Br J Sports Med*. 2016;50(11):651–6. <https://doi.org/10.1136/bjsports-2015-094798>.
6. Andreoli CV, Chiaramonti BC, Buriel E, Pochini ADC, Ejnisman B, Cohen M. Epidemiology of sports injuries in basketball: integrative systematic review. *BMJ Open Sport Exerc Med*. 2018; <https://doi.org/10.1136/bmjsem-2018-000468>.
7. Owoeye OB, Akodu A, Oladokun B, Akinbo SR. Incidence and pattern of injuries among adolescent basketball players in Nigeria. *Sports Med Arthrosc Rehabil Ther Technol*. 2012;4(1):15. <https://doi.org/10.1186/1758-2555-4-15>.
8. Pasanen K, Ekola T, Vasankari T, et al. High ankle injury rate in adolescent basketball: a 3-year prospective follow-up study. *Scand J Med Sci Sport*. 2017;27(6):643–9. <https://doi.org/10.1111/sms.12818>.
9. Taylor JB, Ford KR, Nguyen A-D, Terry LN, Hegedus EJ. Prevention of lower extremity injuries in basketball: a systematic review and meta-analysis. *Sports Health*. 2015;7(5):392–8. <https://doi.org/10.1177/1941738115593441>.
10. Cumps E, Verhagen E, Meeusen R. Prospective epidemiological study of basketball injuries during one competitive season: ankle sprains and overuse knee injuries. *J Sport Sci Med*. 2007;6(2):204–11.
11. Owoeye OBA, Palacios-Derflingher LM, Emery CA. Prevention of ankle sprain injuries in youth soccer and basketball. *Clin J Sport Med*. 2017;1 <https://doi.org/10.1097/JSM.0000000000000462>.
12. Emery CA, Rose MS, McAllister JR, Meeuwisse WH. A prevention strategy to reduce the incidence of injury in high school basketball: a cluster randomized controlled trial. *Clin J Sport Med*. 2007;17(1):17–24. <https://doi.org/10.1097/JSM.0b013e31802e9c05>.
13. Van Reijen M, Vriend I, Zuidema V, van Mechelen W, Verhagen EA. Increasing compliance with neuromuscular training to prevent ankle sprain in sport: does the 'strengthen your ankle' mobile App make a difference? A randomised controlled trial. *Br J Sports Med*. 2016;bjsports-2015-095290. <https://doi.org/10.1136/bjsports-2015-095290>.
14. Longo UG, Loppini M, Berton A, Marinozzi A, Maffulli N, Denaro V. The FIFA 11+ program is effective in preventing injuries in elite male basketball players: a cluster randomized controlled trial. *Am J Sports Med*. 2012;40(5):996–1005. <https://doi.org/10.1177/0363546512438761>.
15. Riva D, Bianchi R, Rocca F, Mamo C. Proprioceptive training and injury prevention in a professional men's basketball team: a six-year prospective study. *J Strength Cond Res*. 2016;30(2) <https://doi.org/10.1519/JSC.0000000000001097>.
16. Eils E, Schröter R, Schröder M, Gerss J, Rosenbaum D. Multistation proprioceptive exercise program prevents ankle injuries in basketball. *Med Sci Sports Exerc*. 2010; <https://doi.org/10.1249/MSS.0b013e3181e03667>.
17. Weiss KJ, Allen SV, McGuigan MR, Whatman CS. The relationship between training load and injury in men's professional basketball. *Int J Sports Physiol Perform*. 2017; <https://doi.org/10.1123/ijsp.2016-0726>.
18. Caparrós T, Casals M, Peña J, et al. The use of external workload to quantify injury risk during professional male basketball games. *J Sport Sci Med*. 2017.
19. Gabbett TJ. The training-injury prevention paradox: should athletes be training smarter and harder? *Br J Sports Med*. 2016; <https://doi.org/10.1136/bjsports-2015-095788>.

20. Soligard T, Schwelunus M, Alonso J-M, et al. How much is too much? (Part 1) International Olympic Committee consensus statement on load in sport and risk of injury. *Br J Sports Med.* 2016;50(17):1030–41. <https://doi.org/10.1136/bjsports-2016-096581>.
21. Ghali BM, Owoeye OBA, Stilling C, et al. Internal and external workload in youth basketball players who are symptomatic and asymptomatic for patellar tendinopathy. *J Orthop Sport Phys Ther* 2019. <https://doi.org/10.2519/jospt.2020.9094>.
22. Labella CR, Smith BW, Sigurdsson A. Effect of mouthguards on dental injuries and concussions in college basketball. *Med Sci Sports Exerc.* 2002; <https://doi.org/10.1097/00005768-200201000-00007>.
23. McGuine T, Brooks A, Hetzel S. The effect of a lace-up ankle brace on ankle injury rates in adolescent basketball players. *Br J Sports Med.* 2011;45(4):314. <https://doi.org/10.1136/bjism.2011.084038>.
24. Bahr R. Why screening tests to predict injury do not work—and probably never will: a critical review. *Br J Sports Med.* 2016; <https://doi.org/10.1136/bjsports-2016-096256>.
25. Roe M, Malone S, Blake C, et al. A six stage operational framework for individualising injury risk management in sport. *Inj Epidemiol.* 2017; <https://doi.org/10.1186/s40621-017-0123-x>.
26. Verhagen E, Van Dyk N, Clark N, Shrier I. Do not throw the baby out with the bathwater; screening can identify meaningful risk factors for sports injuries. *Br J Sports Med.* 2018; <https://doi.org/10.1136/bjsports-2017-098547>.
27. Meeuwisse WH. Athletic injury etiology: distinguishing between interaction and confounding. *Clin J Sport Med.* 1994; <https://doi.org/10.1097/00042752-199407000-00005>.
28. Meeuwisse WH, Tyreman H, Hagel B, Emery C. A dynamic model of etiology in sport injury: the recursive nature of risk and causation. *Clin J Sport Med.* 2007;17(3):215–9. <https://doi.org/10.1097/JSM.0b013e3180592a48>.
29. Windt J, Gabbett TJ. How do training and competition workloads relate to injury? The workload-injury aetiology model. *Br J Sports Med.* 2016;bjsports-2016-096040. <https://doi.org/10.1136/bjsports-2016-096040>.
30. Lathlean TJH, Gastin PB, Newstead SV, Finch CF. Absolute and relative load and injury in elite junior Australian football players over 1 season. *Int J Sports Physiol Perform.* 2019; <https://doi.org/10.1123/ijsp.2019-0100>.
31. Lathlean TJH, Gastin PB, Newstead SV, Finch CFA. Prospective cohort study of load and wellness (sleep, fatigue, soreness, stress, and mood) in elite junior Australian football players. *Int J Sports Physiol Perform.* 2018; <https://doi.org/10.1123/ijsp.2018-0372>.
32. Bertelsen ML, Hulme A, Petersen J, et al. A framework for the etiology of running-related injuries. *Scand J Med Sci Sports.* 2017;27(11):1170–80. <https://doi.org/10.1111/sms.12883>.
33. Hulme A, Finch CF. From monocausality to systems thinking: a complementary and alternative conceptual approach for better understanding the development and prevention of sports injury. *Inj Epidemiol.* 2011:1–12. <https://doi.org/10.1186/s40621-015-0064-1>.
34. Bekker S, Clark AM. Bringing complexity to sports injury prevention research: from simplification to explanation. *Br J Sports Med.* 2016; <https://doi.org/10.1136/bjsports-2016-096457>.
35. Rickles D, Hawe P, Shiell A. A simple guide to chaos and complexity. *J Epidemiol Community Health.* 2007; <https://doi.org/10.1136/jech.2006.054254>.
36. Bittencourt NFN, Meeuwisse WH, Mendonça LD, Nettel-Aguirre A, Ocarino JM, Fonseca ST. Complex systems approach for sports injuries: moving from risk factor identification to injury pattern recognition—narrative review and new concept. *Br J Sports Med.* 2016; <https://doi.org/10.1136/bjsports-2015-095850>.
37. Finch C. A new framework for research leading to sports injury prevention. *J Sci Med Sport.* 2006;9(1-2):3–9. <https://doi.org/10.1016/j.jsams.2006.02.009>.
38. van Mechelen W, Hlobil H, Kemper HCG. Incidence, severity, aetiology and prevention of sports injuries: a review of concepts. *Sport Med.* 1992;14(2):82–99. <https://doi.org/10.2165/00007256-199214020-00002>.
39. J O'Brien, Hagglund MB. Implementing injury prevention. The rocky road from RCT to real-world injury reduction. *Aspetar Sport Med J.* 2018.
40. Owoeye OBA, Akinbo SRA, Olawale OA, Tella BA, Ibeabuchi NM. Injury prevention in football: knowledge and behaviour of players and availability of medical care in a Nigerian youth football league. *S Afr J Sport Med.* 2013;25(3):77. <https://doi.org/10.7196/sajsm.471>.
41. Agel J, Rockwood T, Klossner D. Collegiate ACL injury rates across 15 sports: National Collegiate Athletic Association Injury Surveillance System Data Update (2004–2005 through 2012–2013). *Clin J Sport Med.* 2016; <https://doi.org/10.1097/JSM.0000000000000290>.
42. Malisoux L, Nielsen RO, Urhausen A, Theisen D. A step towards understanding the mechanisms of running-related injuries. *J Sci Med Sport.* 2015; <https://doi.org/10.1016/j.jsams.2014.07.014>.
43. Møller M, Nielsen RO, Attermann J, et al. Handball load and shoulder injury rate: a 31-week cohort study of 679 elite youth handball players. *Br J Sports Med.* 2017; <https://doi.org/10.1136/bjsports-2016-096927>.
44. Owoeye OBA, McKay CD, Verhagen EALM, Emery CA. Advancing adherence research in sport injury prevention. *Br J Sports Med.* 2018;bjsports-2017-098272. <https://doi.org/10.1136/bjsports-2017-098272>.
45. Malisoux L, Nielsen RO, Urhausen A, Theisen D. A step towards understanding the mechanisms of running-related injuries. *J Sci Med Sport.* 2014;18(5):523–8. <https://doi.org/10.1016/j.jsams.2014.07.014>.

46. Kakavas G, Malliaropoulos N, Pruna R, Maffulli N. Artificial intelligence. A tool for sports trauma prediction. *Injury*. 2019; <https://doi.org/10.1016/j.injury.2019.08.033>.
47. Ayala F, López-Valenciano A, Gámez Martín JA, et al. A preventive model for hamstring injuries in professional soccer: learning algorithms. *Int J Sports Med*. 2019;40(05):344–53. <https://doi.org/10.1055/a-0826-1955>.
48. Rossi A, Pappalardo L, Cintia P, Iaia FM, Fernández J, Medina D. Effective injury forecasting in soccer with GPS training data and machine learning. *PLoS One*. 2018;13(7):e0201264. <https://doi.org/10.1371/journal.pone.0201264>.



Practical Guidelines for Injury Prevention in Basketball: How to Get it Right

Steve Short

53.1 Introduction

Musculoskeletal injury is one of the major adverse events from physical activity and competitive basketball. Time loss away from the court has the potential to physically and mentally impair athletes and impact numerous stakeholders involved [1, 2]. Additionally, prior injury history has been identified as a risk factor for future injury [3]. With these key considerations, reducing the risk of initial injury or future injury following a rehabilitation is of significant interest to all vested parties. While identifying common injuries and risk factors involved in basketball participation may be of clinical importance, the current evidence-base suggests that a strong focus of injury prevention should be placed on properly dosed exercise strategies.

53.2 Competitive Demands and Common Injury

Basketball has been defined broadly as an anaerobic sport [4]. Despite this label, a strong aerobic baseline is required to perform. International elite basketball competition typically consists of 40 min of playing time, while a National

Basketball Association (NBA) game involves 48 min of playing time. Considering stoppages in play, athletes who play considerable minutes may be required to perform for an “on-foot” volume of up to 75 min. Within this 75 min, an athlete commonly travels 2–3 miles [5, 6]. High speeds may reach up to 18 miles per hour, but high-speed efforts generally consist of less than 7% of the total duration [6]. However, the game is predominately multidirectional as athletes have been documented as performing up to a 1000 differing movements, including significant volumes of cutting, jumping, and sprinting to compose the noted distance [4, 6]. The intensity of this activity is very high as documented heart rate demands range from 85% maximal heart rate and beyond [4]. Despite potential links between these internal and external variables, a linear relationship between the physiologic and mechanical variables imposed on the athletes has not been identified, inferring the multifactorial nature of these stressors [7].

The benefits of physical activity, including competitive sport are well documented elsewhere, but briefly consist of lower risk of chronic disease, reduced risk of mortality, and increased global wellness, among many others. An unfortunate adverse reaction of these noted exercise demands is musculoskeletal injury [8]. In basketball, it has been extensively documented that injuries of the lower extremity and lumbopelvis are most common. In the National Basketball

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Association, lateral ankle sprains are the most common injury, while the knee is the most commonly injured joint [9]. Injuries to the low back are well documented [10]. Hip and groin injuries are becoming more common and may have been historically underdiagnosed [11, 12]. It is not the focus of this chapter to deeply investigate these common injuries, but it is important to note that an understanding of the incidence, pathomechanics, and resultant impairments to proactively combat them.

53.3 Injury Prevention Foundation

A strong understanding of the demands of competition and the most common injuries is necessary to attempt to prevent injury. However, sporting injuries are complex, multifactorial, and our current understanding of why they occur may be best defined within a complex-systems approach [13]. With so many factors in play, complete prevention may not be practical, while a goal of injury risk reduction may be more clinically relevant. Once this context is defined and sporting environment is understood, clinicians may be able to seek out risk factors that may contribute to injury.

Risk factors have typically been defined as modifiable and nonmodifiable, as well as intrinsic and extrinsic. Modifiable risk factors include factors such as aerobic capacity, strength, and exercise volume while nonmodifiable risk factors include age and prior injury history. Intrinsic risk factors are defined as factors that are internal in nature, such as sex or genetics. Extrinsic risk factors can be defined as environmental in nature and may consist of floor conditions, level of competition, opponent contact, etc. [14] Many of these risk factors will fall into multiple categories. Age can be both a nonmodifiable, intrinsic factor while training volume can be a modifiable, extrinsic factor. Within a complex systems approach, a practitioner will consider these interacting factors within a biopsychosocial framework to evaluate and intervene within a constantly evolving and individualized athlete ecosystem [13].

Typically, risk factors are studied to specific populations and specific injuries [14]. For example, anterior cruciate ligament disruption within adolescent females with defined aberrant jump and landing biomechanics [15]. Other examples include limited strength profiles, identified as poor relative hip (adductor to abductor ratio) and maximal adductor strength in football players with hip and groin symptoms [16] and limited ankle dorsiflexion in volleyball players with patellar tendinopathy [17]. Despite efforts to apply the evidence base broadly across sports and more specifically to basketball, there are limited reviews to support this application of risk factors. Additional challenges exist in the application of defined risk factors due to the dominant methodologies in the evidence base [14]. The primary study designs include case-control, cohort, and intervention studies, each with their own respective limitations. A reliance on retrospective, as opposed to prospective data due to the study design may create questions in interpreting results. All of these designs are often limited by low sample sizes. Inconsistent injury recording methods, including a reliance on only time-loss injury, add to the challenges of appropriately interpreting risk factors.

Risk reduction relies on variables that are objectively measurable as well as clinically modifiable. Despite the challenges involved with identifying valid and reliable risk factor models, training individuals to be more robust and resilient against the various prospective variables associated with athletic musculoskeletal injury continues to fit best practice for prevention models [3, 18–22]. Examples of this include sport-specific conditioning to improve aerobic capacity [20], or eccentric hamstring resistance training to develop muscular architecture, both reducing the potential risk of soft tissue injury [23].

53.4 Screening for Risk

A comprehensive global health screen is common practice, often required, and recommended practice prior to sporting activity [24]. Additionally, musculoskeletal injury risk screening is common

practice across all levels of basketball. Despite its commonplace, there is limited evidence to support the predictive and clinical value of musculoskeletal injury risk screening [25]. Many stand alone screening systems have been trialed to be predictive of injury with limited success [25–28]. In theory, if modifiable risk factors can be identified in screening, then a training program may be appropriate at reducing the risk of future injury by training to be resilient against said risk factors. While the face validity of this practice is strong, there is limited supporting evidence [26, 27]. Numerous testing batteries have proposed and consistently shown to have little predictive value [25]. There is even less evidence to support specific training programs once risk factors are identified [29].

Despite the limitations of many injury screening protocols, specific constructs involved in screening tools have been shown to be useful [30]. Constructs that may hold value in screening for musculoskeletal injury include, but may not be limited to, active mobility, motor control, strength, and hip stability [30]. As opposed to relying on a single test measure, a battery of tests and various related algorithms that account for complex systems may give clinicians the best practical solution for injury risk screening [31, 32].

The most common actionable risk factors are those that can be addressed through exercise prescription [22, 33]. Individual variables such as mobility and strength are often addressed through exercise. Despite the construct validity of training mobility, lasting change of various mobility-based training techniques is quite limited [34, 35]. Transient gains in mobility may be of clinically protective value, but have not been rigorously tested as stand alone interventions to reduce injury risk [21, 33]. For example, active warm-up strategies have been shown to be more impactful than static stretching strategies to increase flexibility prior to physical activity. Conversely, progressive neuromuscular training, typically focused on increasing strength and adjunct physical properties of the lower extremity (balance, motor control, etc.) have been consistently shown to reduce the risk of injury [21, 33, 36].

Training load variables and sport-specific training volumes are also modifiable exercise variables that are typically best addressed through planning, objective measuring, athlete monitoring, and communication with all key parties involved in sporting activities of the athlete, including coaches and management [37–39]. As a result of properly prescribed exercise activity, results such as increased aerobic performance may be achieved and result in improved performance while reducing the risk of injury [19]. It is important to note that decreased volumes of sport-specific training in the pre-season period have been identified as an injury risk [40]. Thus, developing the appropriate cardiovascular profile through exercise should be matched to the competitive demands of each individual sport [4, 20]. For basketball, manipulating variables such as competitive training duration, distance traveled, intensity (heart rate and movement speed), work-to-rest ratios, repeat sprint efforts, offensive and defensive jumping and cutting repetitions, among others, are recommended to achieve the appropriate sport-specific training effect [7].

Despite the growing evidence for exercise-based risk reduction programs, adherence to prescribed exercise programs remains poor [21]. An adequate volume of training is necessary to achieve the appropriate protective stimulus [21, 22]. Athlete and coach buy-in, appropriate time allotment, and numerous cultural-based challenges are barriers for such actionable change. Thus, educating athletes and those who influence their participation is necessary in the practical application of injury risk reduction [39]. Providing coaches and athletes opportunities to ask questions and appropriately understand the goals of implementing programs are a key part of adherence. Open conversation and effective communication can lead to mutual understanding and often lead to collaboration and new ideas to achieve a common goal. This often allows an increase in time allowed before, during, or after team events such as practice or competition for implementation, and additional attention and engagement to be applied to the drills imposed by the practitioner.

53.5 Neuromuscular Training Programs

Strong evidence recommends exercise interventions, often defined as neuromuscular training programs, to reduce the risk of injury [33]. Despite these vigorous recommendations, detail in the prescription of said programs remains challenging. Exercise is dose dependent and requires an appropriate stimulus to achieve physiological and functional changes [41]. Consistency of dosage is poor in reviews of neuromuscular training programs [33]. Vague descriptions in exercise programming presents a challenge for clinicians to apply evidence-based principles. Common fundamental principles of exercise training will often accomplish the desired task of achieving an effective clinical dosage. Athletes will adapt to their imposed demands if the stimulus achieves the appropriate volume and intensity via mechano-transduction [41].

Generally, athletes require 15–30 min 3–5 days a week of adjunct, non-sporting exercise volume. These exercises should address functional mobility and tissue extensibility, proximal strength, hip strength, single leg tasks that address strength and balance, and motor control [30, 42]. Motor control drills may include low intensity (Fig. 53.1) drills that focus on decreasing the aberrant movement of the kinetic chain and often center around minimal displacement of the trunk, lumbopelvis, and knee. Progressions of these drills include plyometric tasks that increase the demand on the movement system and require the athlete to attenuate, control, and produce force throughout the kinetic chain [43]. Specific movement pattern training or motor control tasks may be dependent upon the population. For example, training a resistance to knee valgus may be of protective value in female basketball athletes [44]. While the kinematics may or may not change according to the training program, specific and/or nonspecific adaptations to the training stimulus may reduce the athletes' risk to injury [45, 46]. (Fig. 53.2a–d).

A focus on eccentric training should be implemented within training and prevention programs.

The benefits of eccentric training include superior improvements in muscular architectural changes, functional strength, and decreased risk of injury [47–50]. (Fig. 53.3a, b) Exercises that combine concepts of single leg motor control and proximal hip and trunk muscle recruitment are often clinically time efficient and effective as they train key concepts within a single activity (Figs. 53.1, 53.2a–d, 53.4, and 53.5).

Practically, available time to complete the volume of exercise is an important consideration. Efficiently performed dynamic warm-up, such as the 11+ (formerly known as the FIFA 11+), is one method to achieve the dosage and target goals of

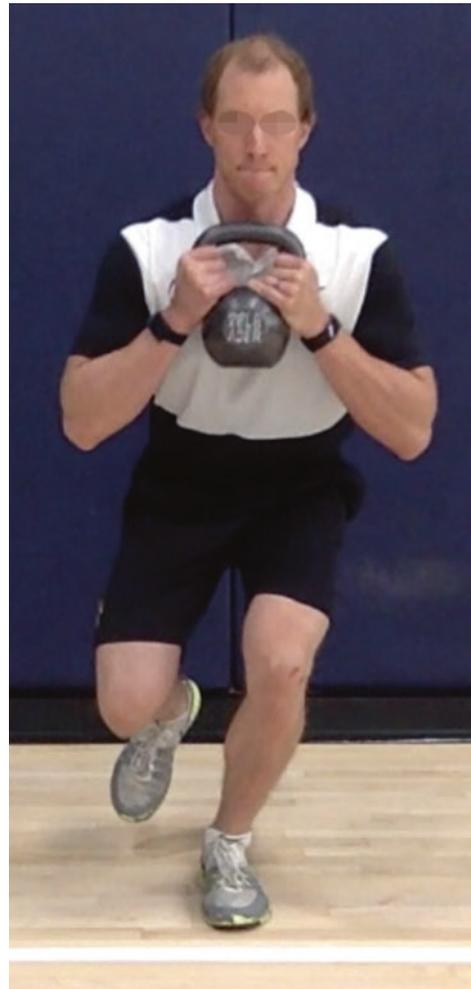


Fig. 53.1 Single-Leg Squat. Exercise focus on lower extremity motor control with the capability of recruiting and strengthening the hip and thigh musculature

exercises defined above. When programmed before or after team practices, these programs have been shown to improve physical qualities while being protective of injury. Like all protocols, individualized details may be left out, and sport-specific considerations must be made to modify these programs to the needs of basketball athletes. Due to the nature of sport, adding an emphasis on adductor training and triplanar movements within the exercise prescription may further reduce the risk of injury [46, 51, 52]. (Figs. 53.5 and 53.6a, b).

The exercises and movements included in these prevention programs are often included in strength and conditioning programs and illustrate

the importance of communication and collaboration in program design, as well as the multifaceted capabilities of exercise. Developing physical qualities such as strength, power, and speed to improve athletic capabilities are often goals of traditional strength and conditioning programs. Differing training loads between the pre-season and in-season training programs must be considered to avoid overtraining, excessive soft tissue trauma, and related soreness [51, 53]. This does not mean that training should cease as an ongoing resistance training program could possibly provide an augmented protective stimulus or prevent the loss of previously developed physical qualities [54, 55].



Fig. 53.2 (a–d) Single-Leg Jump. Dynamic activity that is easily included within a training or dynamic warm-up program that involves lower extremity motor control, deceleration capacity, and power development



Fig. 53.2 (continued)

53.6 Secondary Prevention with Return to Play Considerations

Safely developing sport-specific physical qualities throughout the duration of rehabilitation is necessary to not just return to play, but to return to performance [56] in a capacity that also reduces the risk of future injury [57]. Collaboration is a must between coaches and medical providers to safely implement prevention programs, sporting drills, and exercise as

early and as safely as possible. Multicomponent return to play assessment is preferred in the shared decision-making model as the working relationship between clinicians, athletes, and coaches needs to be maximized to ensure safe and appropriate progression and integration into sport [58]. In situations when an athlete is already injured, the challenge of reducing the risk of future injury is heightened. In addition to the specific concern of the type of tissue affected (muscle vs. tendon vs. ligament vs. bone), status of tissue healing, adequate timeline respective of

Fig. 53.3 (a and b) Nordic Hamstring. Exercise focus on hamstring eccentric capacity and the resultant architectural and attribute-derived protective and performance-related stimulus



the injury and underlying clinical properties of range of motion, strength, power, and specific sport performance, one must consider again the demands of sport.

To achieve each respective return to play criteria, the emphasis remains on properly dosed exercise modalities defined previously. A bolus of work must be performed in order to reach a

Fig. 53.4 Single-Leg Stiff-Legged Deadlift. Exercise provides an eccentric hamstring stimulus while also providing hip and trunk muscle recruitment, in addition to balance and trunk control challenges



Fig. 53.5 Lateral Lunge. Exercise provides a triplanar stimulus that involves muscle recruitment of multiple muscle groups about the hip and thigh in addition to the deceleration capabilities related to the eccentric workload of the quad



stage where testing and return is deemed appropriate. This accentuates the need to continuously obtain objective measures throughout a rehabilitation. Setting and tracking these measurable

goals enables progress and sets the stage for return to play testing to even be possible. If clinicians and coaches are not communicating effectively in regard to the medical status and objective

Fig. 53.6 (a and b)
Hip Adduction Plank.
Exercise focus on
adductor and hip
eccentric capacity and
the resultant
architectural and
attribute-derived
protective and
performance-related
stimulus



tracking, there is a high risk to expose the athlete to inappropriate stress [59] and inadequate training loads [60, 61]. This may come in the form of precautions that need to be observed to protect healing tissue, or the need for the coach to push the athlete to achieve a competition-like stimulus. It has been shown that athletes who are exposed to more training before returning to competition are less likely to sustain injury [62], and this training stimulus can only be provided through properly planned, communicated, and executed training exposure. It is also known in basketball that performance may be impaired upon returning to play [63]; thus, the focus on collaborative preparation of health and performance be maximized to achieve the goals of all vested stakeholders.

Athlete health and availability to perform has been related to individual and team competitive success [64]; thus, all vested parties serve to benefit in optimizing outcomes [65]. Educational seminars and regular group meetings to emphasize the importance of injury prevention programs or properly dosed activity during practices are key to gaining buy-in from these colleagues. Microdosing educational information, such as supplying infographics to athletes and coaches may be another method to efficiently educate and improve retention of desired content [66]. The athlete's goals and desires within a patient-centered care model and effectively communicating their importance within their belief system are necessary to comprehensively reach these desired shared outcomes all while increasing program adherence [67, 68].

The attitude of coaches [69] and the communication between the medical staff and the coaching staff [61] have been shown to impact the health, training and competitive availability of athletes. The competitive and demanding nature of sport requires an emphasis on a return-to-elite performance and not only does the dosage of exercise need to be prescribed to achieve as much, but the planning, communication, trust, and motivation required to achieve adherence to achieve such a volume and intensity of protective exercises is a must to help reduce the risk of future injury.

Take-Home Message

Injury prevention in basketball is a complex and challenging task. An evolving understanding of risk factors and risk screening is necessary to address injury risk. Focusing on properly dosed exercise principles is an essential component to reduce the risk of injury in both healthy populations and in individual return to play cases. An individualized, dynamic, and shared decision-making process is required to reduce the risk of future injury when rehabilitating from an original injury.

References

1. Eirale C, Tol JL, Farooq A, et al. Low injury rate strongly correlates with team success in Qatari professional football. *Br J Sports Med.* 2013;47(12):807–8. <https://doi.org/10.1136/bjsports-2012-091040>. [Published Online First: 2012/08/21].
2. Podlog L, Buhler CF, Pollack H, et al. Time trends for injuries and illness, and their relation to performance in the National Basketball Association. *J Sci Med Sport.* 2015;18(3):278–82. <https://doi.org/10.1016/j.jsams.2014.05.005>. [Published Online First: 2014/06/09].
3. Murphy DF, Connolly DAJ, Beynon BD. Risk factors for lower extremity injury: a review of the literature. *Br J Sports Med.* 2003;37(1):13–29. <https://doi.org/10.1136/bjism.37.1.13>.
4. Schelling J, Torres L. Conditioning for basketball: quality and quantity. *Strength Cond J.* 2013;35(6):89–94.
5. NBA Advanced Stats. <https://stats.nba.com/players/speed-distance/>: National Basketball Association; 2018–19. Accessed 3 Sep 2019.
6. Caparrós T, Casals M, Solana Á, et al. Low external workloads are related to higher injury risk in professional male basketball games. *J Sports Sci Med.* 2018;17(2):289–97.
7. Scanlan AT, Wen N, Tucker PS, et al. The relationships between internal and external training load models during basketball training. *J Strength Cond Res.* 2014;28(9):2397–405. <https://doi.org/10.1519/jsc.0000000000000458>. [Published Online First: 2014/03/26].
8. Hootman JM, Macera CA, Ainsworth BE, et al. Epidemiology of musculoskeletal injuries among sedentary and physically active adults. *Med Sci Sports Exerc.* 2002;34(5):838–44. [Published Online First: 2002/05/02].

9. Drakos MC, Domb B, Starkey C, et al. Injury in the national basketball association: a 17-year overview. *Sports Health*. 2010;2(4):284–90. <https://doi.org/10.1177/1941738109357303>. [Published Online First: 2010/07/01].
10. Minhas SV, Kester BS, Hsu WK. Outcomes after lumbar disc herniation in the National Basketball Association. *Sports Health*. 2016;8(1):43–9. <https://doi.org/10.1177/1941738115608361>. [Published Online First: 2015/10/27].
11. Jackson TJ, Starkey C, McElhiney D, et al. Epidemiology of hip injuries in the National Basketball Association: a 24-year overview. *Orthop J Sports Med*. 2013;1(3):2325967113499130. <https://doi.org/10.1177/2325967113499130>. [Published Online First: 2013/08/01].
12. Ekhtiari S, Khan M, Burrus T, et al. Hip and groin injuries in professional basketball players: impact on playing career and quality of life after retirement. *Sports Health*. 2019;11(3):218–22. <https://doi.org/10.1177/1941738119838274>. [Published Online First: 2019/04/24].
13. Bittencourt NFN, Meeuwisse WH, Mendonca LD, et al. Complex systems approach for sports injuries: moving from risk factor identification to injury pattern recognition-narrative review and new concept. *Br J Sports Med*. 2016;50(21):1309–14. <https://doi.org/10.1136/bjsports-2015-095850>. [Published Online First: 2016/07/23].
14. Bahr R. Risk factors for sports injuries a methodological approach. *Br J Sports Med*. 2003;37:384–92.
15. Lopes TJA, Simic M, Myer GD, et al. The effects of injury prevention programs on the biomechanics of landing tasks: a systematic review with meta-analysis. *Am J Sports Med*. 2018;46(6):1492–9. <https://doi.org/10.1177/0363546517716930>.
16. Mosler AB, Agricola R, Weir A, et al. Which factors differentiate athletes with hip/groin pain from those without? A systematic review with meta-analysis. *Br J Sports Med*. 2015;49(12):810. <https://doi.org/10.1136/bjsports-2015-094602>. [Published Online First: 2015/06/03].
17. Malliaras P, Cook JL, Kent P. Reduced ankle dorsiflexion range may increase the risk of patellar tendon injury among volleyball players. *J Sci Med Sport*. 2006;9(4):304–9. <https://doi.org/10.1016/j.jsams.2006.03.015>. [Published Online First: 2006/05/05].
18. de la Motte SJ, Lisman P, Gribbin TC, et al. Systematic review of the association between physical fitness and musculoskeletal injury risk: part 3-flexibility, power, speed, balance, and agility. *J Strength Cond Res*. 2019;33(6):1723–35. <https://doi.org/10.1519/JSC.0000000000002382>. [Published Online First: 2017/12/15].
19. Gabbett TJ. Debunking the myths about training load, injury and performance: empirical evidence, hot topics and recommendations for practitioners. *Br J Sports Med*. 2018;bjsports-2018-099784. <https://doi.org/10.1136/bjsports-2018-099784>.
20. Lisman PJ, de la Motte SJ, Gribbin TC, et al. A systematic review of the association between physical fitness and musculoskeletal injury risk: part 1-cardiorespiratory endurance. *J Strength Cond Res*. 2017;31(6):1744–57. <https://doi.org/10.1519/jsc.0000000000001855>. [Published Online First: 2017/05/26].
21. Thorborg K, Krommes KK, Esteve E, et al. Effect of specific exercise-based football injury prevention programmes on the overall injury rate in football: a systematic review and meta-analysis of the FIFA 11 and 11+ programmes. *Br J Sports Med*. 2017;51(7):562–71. <https://doi.org/10.1136/bjsports-2016-097066>. [Published Online First: 2017/01/15].
22. Lauenstein JB, Andersen TE, Andersen LB. Strength training as superior, dose-dependent and safe prevention of acute and overuse sports injuries: a systematic review, qualitative analysis and meta-analysis. *Br J Sports Med*. 2018;52(24):1557–63. <https://doi.org/10.1136/bjsports-2018-099078>. [Published Online First: 2018/08/23].
23. Buckthorpe M, Wright S, Bruce-Low S, et al. Recommendations for hamstring injury prevention in elite football: translating research into practice. *Br J Sports Med*. 2019;53(7):449–56. <https://doi.org/10.1136/bjsports-2018-099616>.
24. Bakken A, Targett S, Bere T, et al. Health conditions detected in a comprehensive periodic health evaluation of 558 professional football players. *Br J Sports Med*. 2016;50(18):1142–50. <https://doi.org/10.1136/bjsports-2015-095829>.
25. Whittaker JL, Booyesen N, de la Motte S, et al. Predicting sport and occupational lower extremity injury risk through movement quality screening: a systematic review. *Br J Sports Med*. 2017;51(7):580–5. <https://doi.org/10.1136/bjsports-2016-096760>. [Published Online First: 2016/12/10].
26. Hegedus EJ, McDonough S, Bleakley C, et al. Clinician-friendly lower extremity physical performance measures in athletes: a systematic review of measurement properties and correlation with injury, part 1. The tests for knee function including the hop tests. *Br J Sports Med*. 2015;49(10):642–8. <https://doi.org/10.1136/bjsports-2014-094094>. [Published Online First: 2014/12/17].
27. Hegedus EJ, McDonough SM, Bleakley C, et al. Clinician-friendly lower extremity physical performance tests in athletes: a systematic review of measurement properties and correlation with injury. Part 2--the tests for the hip, thigh, foot and ankle including the star excursion balance test. *Br J Sports Med*. 2015;49(10):649–56. <https://doi.org/10.1136/bjsports-2014-094341>. [Published Online First: 2015/01/24].
28. Taylor JB, Ford KR, Nguyen AD, et al. Prevention of lower extremity injuries in basketball: a systematic review and meta-analysis. *Sports Health*. 2015;7(5):392–8. <https://doi.org/10.1177/1941738115593441>. [Published Online First: 2015/10/27].

29. Kiesel K, Plisky P, Butler R. Functional movement test scores improve following a standardized off-season intervention program in professional football players. *Scand J Med Sci Sports*. 2011;21(2):287–92. <https://doi.org/10.1111/j.1600-0838.2009.01038.x>. [Published Online First: 2009/12/25].
30. Hegedus EJ, McDonough S, Bleakley C, et al. Physical performance tests predict injury in National Collegiate Athletic Association athletes: a three-season prospective cohort study. *Br J Sports Med*. 2016;50(21):1333–7. <https://doi.org/10.1136/bjsports-2015-094885>. [Published Online First: 2016/01/10].
31. Mendonca LD, Ocarino JM, Bittencourt NFN, et al. Association of hip and foot factors with patellar tendinopathy (Jumper's knee) in athletes. *J Orthop Sports Phys Ther*. 2018;48(9):676–84. <https://doi.org/10.2519/jospt.2018.7426>. [Published Online First: 2018/05/25].
32. Bird S, Markwick W. Musculoskeletal screening and functional testing: considerations for basketball athletes. *Int J Sports Phys Ther*. 2016;11(5):784–802.
33. Brunner R, Friesenbichler B, Casartelli NC, et al. Effectiveness of multicomponent lower extremity injury prevention programmes in team-sport athletes: an umbrella review. *Br J Sports Med*. 2019;53(5):282–8. <https://doi.org/10.1136/bjsports-2017-098944>. [Published Online First: 2018/09/12].
34. Weppler CH, Magnusson SP. Increasing muscle extensibility: a matter of increasing length or modifying sensation? *Phys Ther*. 2010;90(3):438–49. <https://doi.org/10.2522/ptj.20090012>. [Published Online First: 2010/01/16].
35. Brusco CM, Blazeovich AJ, Pinto RS. The effects of 6 weeks of constant-angle muscle stretching training on flexibility and muscle function in men with limited hamstrings' flexibility. *Eur J Appl Physiol*. 2019; <https://doi.org/10.1007/s00421-019-04159-w>. [Published Online First: 2019/05/12].
36. Bourne MN, Timmins RG, Opar DA, et al. An evidence-based framework for strengthening exercises to prevent hamstring injury. *Sports Med*. 2018;48(2):251–67. <https://doi.org/10.1007/s40279-017-0796-x>. [Published Online First: 2017/11/09].
37. Impellizzeri FM, Marcora SM, Coutts AJ. Internal and external training load: 15 years on. *Int J Sports Physiol Perform*. 2019;14(2):270–3. <https://doi.org/10.1123/ijsspp.2018-0935>.
38. Malone S, Hughes B, Doran DA, et al. Can the workload-injury relationship be moderated by improved strength, speed and repeated-sprint qualities? *J Sci Med Sport*. 2019;22(1):29–34. <https://doi.org/10.1016/j.jsams.2018.01.010>. [Published Online First: 2018/07/31].
39. McLean BD, Strack D, Russell J, et al. Quantifying physical demands in the National Basketball Association-Challenges around developing best-practice models for athlete care and performance. *Int J Sports Physiol Perform*. 2019;14(4):414–20. <https://doi.org/10.1123/ijsspp.2018-0384>. [Published Online First: 2018/07/25].
40. Whittaker JL, Small C, Maffey L, et al. Risk factors for groin injury in sport: an updated systematic review. *Br J Sports Med*. 2015;49(12):803–9. <https://doi.org/10.1136/bjsports-2014-094287>. [Published Online First: 2015/04/03].
41. Dunn SL, Olmedo ML. Mechanotransduction: relevance to physical therapist practice-understanding our ability to affect genetic expression through mechanical forces. *Phys Ther*. 2016;96(5):712–21. <https://doi.org/10.2522/ptj.20150073>. [Published Online First: 2015/12/25].
42. Gomes Neto M, Conceicao CS, de Lima Brasileiro AJA, et al. Effects of the FIFA 11 training program on injury prevention and performance in football players: a systematic review and meta-analysis. *Clin Rehabil*. 2017;31(5):651–9. <https://doi.org/10.1177/0269215516675906>. [Published Online First: 2016/11/05].
43. Arundale AJH, Bizzini M, Giordano A, et al. Exercise-based knee and anterior cruciate ligament injury prevention. *J Orthop Sports Phys Ther*. 2018;48(9):A1–a42. <https://doi.org/10.2519/jospt.2018.0303>. [Published Online First: 2018/09/02].
44. Myer GD, Ford KR, Brent JL, et al. Differential neuromuscular training effects on ACL injury risk factors in “high-risk” versus “low-risk” athletes. *BMC Musculoskelet Disord*. 2007;8:39. <https://doi.org/10.1186/1471-2474-8-39>. [Published Online First: 2007/05/10].
45. Taylor JB, Ford KR, Schmitz RJ, et al. Sport-specific biomechanical responses to an ACL injury prevention programme: a randomised controlled trial. *J Sports Sci*. 2018;36(21):2492–501. <https://doi.org/10.1080/02640414.2018.1465723>. [Published Online First: 2018/04/20].
46. King E, Franklyn-Miller A, Richter C, et al. Clinical and biomechanical outcomes of rehabilitation targeting intersegmental control in athletic groin pain: prospective cohort of 205 patients. *Br J Sports Med*. 2018;52(16):1054–62. <https://doi.org/10.1136/bjsports-2016-097089>.
47. Goode AP, Reiman MP, Harris L, et al. Eccentric training for prevention of hamstring injuries may depend on intervention compliance: a systematic review and meta-analysis. *Br J Sports Med*. 2015;49(6):349–56. <https://doi.org/10.1136/bjsports-2014-093466>. [Published Online First: 2014/09/18].
48. Sullivan K, McAulliffe S, De Burca N. The effects of eccentric training on lower limb flexibility: a systematic review. *Br J Sports Med*. 2014;48(7):648. <https://doi.org/10.1136/bjsports-2014-093494.234>.
49. Lorenz D, Reiman M. The role and implementation of eccentric training in athletic rehabilitation: tendinopathy, hamstring strains, and acl reconstruction. *Int J Sports Phys Ther*. 2011;6(1):27–44.

50. Douglas J, Pearson S, Ross A, et al. Chronic adaptations to eccentric training: a systematic review. *Sports Med.* 2017;47(5):917–41. <https://doi.org/10.1007/s40279-016-0628-4>. [Published Online First: 2016/09/21].
51. Haroy J, Clarsen B, Wiger EG, et al. The adductor strengthening programme prevents groin problems among male football players: a cluster-randomised controlled trial. *Br J Sports Med.* 2019;53(3):150–7. <https://doi.org/10.1136/bjsports-2017-098937>. [Published Online First: 2018/06/13].
52. Haroy J, Thorborg K, Serner A, et al. Including the Copenhagen adduction exercise in the FIFA 11+ provides missing eccentric hip adduction strength effect in male soccer players: a randomized controlled trial. *Am J Sports Med.* 2017;45(13):3052–9. <https://doi.org/10.1177/0363546517720194>. [Published Online First: 2017/08/15].
53. Lovell R, Whalan M, Marshall PWM, et al. Scheduling of eccentric lower limb injury prevention exercises during the soccer micro-cycle: which day of the week? *Scand J Med Sci Sports.* 2018;28(10):2216–25. <https://doi.org/10.1111/sms.13226>. [Published Online First: 2018/05/26].
54. de la Motte SJ, Gribbin TC, Lisman P, et al. Systematic review of the association between physical fitness and musculoskeletal injury risk: part 2-muscular endurance and muscular strength. *J Strength Cond Res.* 2017;31(11):3218–34. <https://doi.org/10.1519/jsc.0000000000002174>. [Published Online First: 2017/08/11].
55. de Hoyo M, Pozzo M, Sanudo B, et al. Effects of a 10-week in-season eccentric-overload training program on muscle-injury prevention and performance in junior elite soccer players. *Int J Sports Physiol Perform.* 2015;10(1):46–52. <https://doi.org/10.1123/ijsp.2013-0547>. [Published Online First: 2014/06/10].
56. Morrison S, Ward P, du Manoir GR. Energy system development and load management through the rehabilitation and return to play process. *Int J Sports Phys Ther.* 2017;12(4):697–710. [Published Online First: 2017/09/14].
57. Gabbett TJ. The training-injury prevention paradox: should athletes be training smarter and harder? *Br J Sports Med.* 2016;50(5):273–80. <https://doi.org/10.1136/bjsports-2015-095788>. [Published Online First: 2016/01/14].
58. Shrier I. Strategic assessment of risk and risk tolerance (StARRT) framework for return-to-play decision-making. *Br J Sports Med.* 2015;49(20):1311–5. <https://doi.org/10.1136/bjsports-2014-094569>. [Published Online First: 2015/06/04].
59. Caparros T, Alentorn-Geli E, Myer GD, et al. The relationship of practice exposure and injury rate on game performance and season success in professional male basketball. *J Sports Sci Med.* 2016;15(3):397–402. [Published Online First: 2016/11/03].
60. Sporer BC, Windt J. Integrated performance support: facilitating effective and collaborative performance teams. *Br J Sports Med.* 2018;52(16):1014–5. <https://doi.org/10.1136/bjsports-2017-097646>. [Published Online First: 2017/08/23].
61. Ekstrand J, Lundqvist D, Davison M, et al. Communication quality between the medical team and the head coach/manager is associated with injury burden and player availability in elite football clubs. *Br J Sports Med.* 2019;53(5):304–8. <https://doi.org/10.1136/bjsports-2018-099411>.
62. Bengtsson H, Ekstrand J, Waldén M, et al. Few training sessions between return to play and first match appearance are associated with an increased propensity for injury: a prospective cohort study of male professional football players during 16 consecutive seasons. *Br J Sports Med.* 2019;bjsports-2019-100655. <https://doi.org/10.1136/bjsports-2019-100655>.
63. Minhas SV, Kester BS, Larkin KE, et al. The effect of an orthopaedic surgical procedure in the National Basketball Association. *Am J Sports Med.* 2016;44(4):1056–61. <https://doi.org/10.1177/0363546515623028>. [Published Online First: 2016/01/24].
64. Eirale C, Tol JL, Farooq A, et al. Low injury rate strongly correlates with team success in Qatari professional football. *Br J Sports Med.* 2013;47(12):807–8. <https://doi.org/10.1136/bjsports-2012-091040>.
65. Drew MK, Raysmith BP, Charlton PC. Injuries impair the chance of successful performance by sportspeople: a systematic review. *Br J Sports Med.* 2017;51(16):1209–14. <https://doi.org/10.1136/bjsports-2016-096731>.
66. Scott H, Fawkner S, Oliver C, et al. Why healthcare professionals should know a little about infographics. *Br J Sports Med.* 2016;50(18):1104–5. <https://doi.org/10.1136/bjsports-2016-096133>.
67. Owøye OBA, McKay CD, Verhagen EALM, et al. Advancing adherence research in sport injury prevention. *Br J Sports Med.* 2018;52(17):1078–9. <https://doi.org/10.1136/bjsports-2017-098272>.
68. Gluyas H. Patient-centred care: improving healthcare outcomes. *Nurs Stand.* 2015;30(4):50–7; quiz 59. <https://doi.org/10.7748/ns.30.4.50.e10186>. [Published Online First: 2015/09/24].
69. Ekstrand J, Lundqvist D, Lagerback L, et al. Is there a correlation between coaches' leadership styles and injuries in elite football teams? A study of 36 elite teams in 17 countries. *Br J Sports Med.* 2018;52(8):527–31. <https://doi.org/10.1136/bjsports-2017-098001>. [Published Online First: 2017/10/24].



Rehabilitation of Shoulder Injuries in Basketball

54

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54.1 Introduction

Rehabilitation of the shoulder complex is challenging for clinicians due to the vast number of structures in a small area that can undergo extreme ranges of motion and angular velocities during sport. The glenohumeral joint has minimal bony congruency, and therefore heavily relies upon the rotator cuff and surrounding musculature to provide dynamic stabilization to the joint. This highlights the importance of rehabilitation to restore shoulder joint biomechanics and muscular function following shoulder injuries. Evidence for shoulder rehabilitation programs is limited, with the majority of clinical practice guidelines and protocols relying upon expert opinion. This chapter will utilize the Staged

Approach for Rehabilitation of Shoulder Disorders (STAR) [1] as a guiding framework, combined with an objective criterion-based progression, to return athletes to basketball following shoulder injury/surgery.

54.2 Early Phase

The focus of early stage rehabilitation is to correctly identify and mitigate pain irritability of the injured/post-surgical athlete. Irritability refers to the ability of the tissue to handle load in relation to the symptomatic or inflammatory response. The irritability classification utilizes pain intensity, at what point pain occurs during motion (e.g., pain at mid vs. end range), and level of pain-related disability, to classify athletes into low, moderate, or high irritability. Athletes have differential responses to injury, which necessitates the use of classification criteria to identify the current status of the athlete. The focus of rehabilitation interventions changes based on the stage of irritability, with the goal of achieving low irritability as quickly as possible. Matching interventions to the stage of patient irritability is a priority in the STAR approach.

A point guard, for example, presents with acute onset of shoulder pain after competing for a rebound with an opponent, and was unable to return to the game. No frank dislocation was reported, and radiographic/MRI imaging was

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negative. The following day she presented with high pain levels, diminished passive range of motion (PROM) and active range of motion (AROM) capabilities, no apprehension to movement, altered Activities of Daily Living (ADL), and inability to sleep on her shoulder. This presentation is consistent with the high irritability stage [1, 2] of the irritability classification (Table 54.1: Reprint from McClure et al. [1]).

For this level of irritability, priority is placed on pain management to restore pre-injury sleep and functional arm use for activities of daily living (ADL). This may require modification of upper extremity activities which can include immobilization, relative rest from provoking activities, and/or modification of a conditioning/training routine. If direct use of the arm cannot be performed due to surgical restrictions or degree of irritability, then contralateral arm exercise is suggested to minimize disuse as well as improve range of motion [3]. In the context of our example athlete, immobilization is not indicated, but

education in relative rest techniques (e.g., ADL or practice/training modifications) will assist in minimizing early pain levels. In addition to activity modification, pain management can also include modality use as indicated by the clinical presentation as well as pharmacological interventions if appropriate. Electrical stimulation can be effective in reducing acute pain of various soft tissue injuries (Table 54.2: Reprinted with permission from Tara Manal PT DPT OCS SCS FAPTA) [4]. Modality and/or manual therapy techniques can be applied concurrently to assist in the reduction of symptom irritability and help progress the athlete to the next stage of the rehabilitation as appropriate.

If range of motion and joint mobility are impairing function, then gentle range of motion exercises are included with short duration hold times along with low-grade Kalteborn or Maitland (Table 54.3 adapted from Duenas et al. [2]) mobilizations. Graded stress can also be effective in reducing pain and improving func-

Table 54.1 Staging of symptom irritability for shoulder disorders

	Stage of irritability		
	High	Moderate	Low
History & examination findings	<ul style="list-style-type: none"> - High pain ($\geq 7/10$) - Consistent night or rest pain - Pain before end of ROM - AROM < PROM - High disability 	<ul style="list-style-type: none"> - Moderate pain (4–6/10) - Intermittent night or rest pain - AROM \cong PROM - Moderate disability 	<ul style="list-style-type: none"> - Low pain ($\leq 3/10$) - Absent night or rest pain - AROM = PROM - Low disability
Interventions focus	<ul style="list-style-type: none"> - Minimize physical stress - Activity modification - Monitor impairments 	<ul style="list-style-type: none"> - Mild-moderate physical stress - Address impairments - Basic-level functional activity restoration 	<ul style="list-style-type: none"> - Moderate-high physical stress - Address impairments - High-demand functional activity restoration

Table 54.2 Electrical stimulation parameters for treating pain

	Sensory TENS	Motor TENS	Noxious
Clinical utility	General pain management	Soft tissue pain source: Usually muscle	Soft tissue pain source: Usually ligament/tendon
Pulse width	200 us	400 us	400 us
Frequency	10–100 pps depending on pt preference	2 pps	50 pps; increase if pt cannot tolerate
On time	30 s	45 s	12 s
Off time	0 s	15 s	8 s
Ramp	0–2 s	2 s	2–5 s
Treatment time	15	15	10
Additional information	May get motor involvement depending on stim pad locations	Should look like a “jumping” muscle; helps to decrease pain	Use smaller pads to bracket the pinpoint pain

Table 54.3 Joint mobilization guidelines according to irritability classification

	High irritability	Moderate irritability	Low irritability
Joint mobilizations	Grade I–II/ empty feels	Grade II–III/stiff & painful	Grade III–IV/stiff & limited mobility
Passive range of motion/ active assisted range of motion	≤5 s	5 to 15 s	>15 s if ROM limited
Active range of motion	Performed in low to mod irritability arc/ranges	Performed in low to mod irritability arc/ranges	Performed with increased loads and/or volume

tion during this stage. The high irritability of our case athlete's shoulder may not allow for common open chain shoulder exercises due to excessive pain or neuromuscular compensations. Utilizing axial loading, closed chain exercises, or supported arm movements are alternative ways to stress the shoulder tissues until irritability levels allow for progression. Revisiting our case, performing stair railing slides, quadruped weight-shifting, or even wall slides would help elevation movements. As pain and irritability reduces, then graded progression in load or movement at expanded ranges of motion may occur. Overall the goal of this phase is to reduce pain levels and encourage general mobility of the arm within the restrictions of the injury or surgical procedure. Continual reassessment is paramount to detect changes in irritability and response to treatment to maximize rehabilitation.

As irritability reduces to moderate or low levels, rehabilitation intensity will progress accordingly. Priority will shift to resolving acute impairments identified during objective examination. Isometrics are indicated for moderate irritability while patients utilize a pain monitoring model to guide acceptable pain allowance in performance. Utilizing a rate of perceived exertion (RPE) scale with isometrics may also be beneficial to allow the athlete to track their clinical progress. In the case of our point guard, she demonstrates only painful internal rotation and flexion

of the shoulder at 45° in the scapular plane. Keeping with criterion progressions, the goal to progress shoulder isometrics to isotonic or further into elevation is ≤3/10 pain with resisted isometric testing (low irritability). For example, after 2 days of isometric exercise, our case athlete has noticed a change in 5/10 pain with 2/10 RPE to 2/10 pain with 7/10 RPE, and she and the clinician now know that she is likely ready for exercise progression. Additionally isotonic abduction and external rotation exercises would be indicated at lower angles to progressively load the shoulder joint. If advancement to different ranges of motion is limited, manual resistance techniques or proprioceptive neuromuscular facilitation (PNF) techniques would be appropriate while progressing other ranges that are not pain limited. An example of this with our basketball player would be continued limitations in progressing internal rotation and flexion strengthening to higher ranges needed for sport. As a result, manual internal rotation strengthening via alternating isometrics or rhythmic stabilization is applied at different angles with accommodating resistances according to pain irritability (Fig. 54.1). The player's remaining non-painful ranges will continue to be progressed in difficulty or discontinued per objective strengthening testing.

Due to the multi-planar and dynamic nature of the shoulder, active range of motion in various planes or angles around the painful arc is suggested if a movement is limited by pain intensity. This may occur with the ability to perform active motion in the flexion or scapular plane but not abduction. Additionally it may occur in overhead positions but not at lower angles or vice versa (Fig. 54.2). Table 54.4 details some progressions for painful arcs assuming no additional impairments are present limiting performance (Table 54.4 Sample progression for a patient with painful shoulder abduction).

Palpation of soft tissue may identify structures that are symptomatically impeding performance of general function or exercises. Repeated examination can be utilized to determine the effect these structures are having on exercise or functional capabilities. For example, if our point guard presents with tenderness to her infraspinatus



Fig. 54.1 Sample exercise variations for rhythmic stabilization techniques of the shoulder



Fig. 54.2 Example of limiting range of motion to address shoulder abduction while promoting quality movement

Table 54.4 Example of progressing planes of motion

	Option A	Option B	Option C	Option D	Option E
Painful abduction (ABD) plane	ABD above or below painful arc → progress loads or move arc closer to pain	ABD isometrics at painful arc → progress intensity or add perturbation	Eccentrics through painful arc → progress loads	Progress loads in scapular plane → reduce weight and progress toward ABD plane	Perform horizontal ABD plane at angle of pain

tus muscle belly that is limiting progression of external rotation strengthening or overhead reaching, then direct application of Motor TENS or soft tissue techniques to this region could be indicated. If these treatments result in reduced

pain during movement, continued application is suggested.

Ideally, athletes will move with good coordination and neuromuscular control; however, this can be limited by pain, fear, or compensations.

It is important to correctly identify the barrier(s) to progression and determine if/when progression can occur despite these limitations or if remaining at the current level is necessary. Progression into full overhead or sport-specific motions is not recommended if compensations that could impact exercise performance/sport technique are seen or if moderate tissue irrita-

bility is present. Adaptations to typical open kinetic chain neuromuscular control exercises can be utilized in response to pain or compensations. These can include use of support from wall, dowels, tables, or assistance from balls, resistance bands, or hand in hand assist from the therapist (Figs. 54.3, 54.4, 54.5, and 54.6). Variables may be weaned or added to assist in



Fig. 54.3 Active assistive flexion using a dowel. The dowel can decrease the amount of load required to complete the motion



Fig. 54.4 Variation of active assisted shoulder flexion using a ball. The height and incline of the table can be adjusted to alter load



Fig. 54.5 Sample exercise of eccentric lowering. The athlete can use the wall to get into overhead position and then slowly control arm motion as it lowers back down to the athlete’s side



Fig. 54.6 Active assistive exercise for shoulder abduction. The band assists in bringing the arm into abduction. The concentric phase of this exercise can also be used to work on scapulothoracic control

this progression, i.e., weight to wall slides, eccentric phases only of upper extremity motions, gravity minimization using changes in body position (e.g., supine and reclined) for scapular/glenohumeral muscle training. For example, in the case of our point guard and her limited ability to perform internal rotation and flexion motions (still causing >6/10 pain). Pain reduced to low irritability when she performed flexion with bands around her wrists (external rotation bias or concurrent external rotation isometric) as well as reduced pain with internal rotation strengthening with elbow supported on a table (Figs. 54.7 and 54.8). If the previously mentioned flexion and internal rotation limitations were not present, this clinical decision may not be needed. Again the goal of using criterion-based progressions is to identify when someone is appropriate to progress. In this case, continued limitations, and ability to reach criteria, resulted in a clinical treatment decision to progression into the middle phase of the rehab process.

While impairment resolution is key to progressing the rehabilitation plan, it is important to also recognize the conditioning demands to compete in their sport. Immobilization or pro-

tection of injured tissues can result in deconditioning of the athlete. If possible, the athlete's aerobic capacity should be maintained with cross training through ergonomic cycling, pool conditioning, elliptical machine, stair master, or running. This is particularly important due to known changes in shooting technique that occur with the onset of physical fatigue [5]. In the case of our point guard, she was encouraged to immediately perform ergonomic cycling intervals in her high irritability stage with progression to jogging/shuttle interval once she showed reduced irritability on clinical testing and continued reduction to low irritability with aerobic exercise performance.

The following criteria can be used to progress from the early to middle phase of rehabilitation:

- Resisted isometric testing: Low to moderate irritability classification
- Joint mobility: Low irritability or stiff/painful with Grade III to IV mobilizations
- ROM: Low irritability with end ranges; capable of AROM with low irritability
- Soft tissue irritability to palpation: low to absent irritability

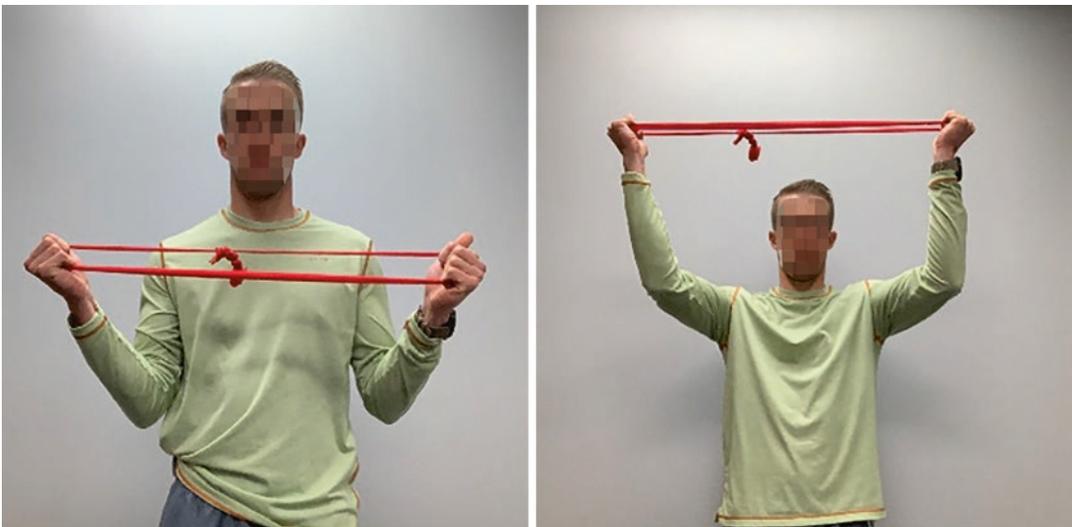


Fig. 54.7 Sample exercise of maintaining an external rotation contraction while moving through should flexion range of motion



Fig. 54.8 Strengthening shoulder rotation in an overhead (90 degrees of abduction) position

54.3 Middle Phase

Once the athlete demonstrates readiness for the middle rehabilitation phase, the treatment objective is to fully restore mobility to the shoulder complex, improve/restore neuromuscular control within the newly restored arc of motion, and prepare for the later phases of rehabilitation. While tissue irritability has likely decreased, pain may persist or intermittently increase requiring management to optimize tissue load tolerance and progressive tissue adaptation. Consequently, ongoing tissue irritability assessment is necessary in order to guide the rehabilitation process and load tissues appropriately.

Fully restoring mobility of the shoulder girdle complex requires unrestricted mobility of the glenohumeral, sternoclavicular, and acromioclavicular joints. Full glenohumeral joint (GHJ) elevation is interdependent upon retraction, elevation and posterior spin of the clavicle at the sternoclavicular joint (SCJ) and upward rotation of the scapula

involving gliding at the acromioclavicular joint (ACJ) [6]. Consequently, joint mobilizations to address end-range restrictions can be used, particularly if end ranges are painful (moderate to low irritability). While the contributions of the SCJ and ACJ accessory motions for full overhead GHJ motion may be minimal, full mobility may be necessary for unrestricted overhead arm elevation to be pain-free. Attention to the SCJ and ACJ is particularly relevant for athletes who have undergone any period of immobilization necessary in the early phases of tissue healing. Once tissue irritability is moderate to low, end-range accessory motion mobilizations with the joint at the end of the available physiological range should be included. A combination of oscillatory mobilizations and low-load, prolonged stretching can be used to improve end range of motion. Of particular importance for athletes, such as the point guard in this example, tolerance of end-range elevation and internal and external rotation are paramount to return to play. Shooting and

Table 54.5 Joint and soft tissue mobilization progression to restore full shoulder complex mobility

Early middle phase	Late middle phase
Patient supine Inferior GHJ glide at end-range flexion, Grade III–IV oscillations for 2 min Posterior GHJ glide at end-range flexion, Grade III–IV oscillations for 2 min	Patient seated Active shoulder flexion, therapist provides arced inferior GHJ glide as patient completes the range, 10 repetitions
Submaximal isometrics, alternating flexion/extension at the point of pain in elevation, 45 second hold, 4 repetitions, patient supine	Alternating flexion–extension–flexion isometric after arced mobilization, 5 s hold in each direction
Sustained end-range flexion stretch, hold 45–60 s, 3 repetitions	End-range flexion stretch in kneeling with both hands on physioball—“Ball rollout,” hold 30 s, 5 repetitions
Thoracic PA mobilizations in prone for 5–10 min, Grade III–IV	Self-thoracic extension mobilizations in hook lying over foam roller, patient with arms crossed over chest or flexed overhead for 2–3 min

rebounding require producing and absorbing forces at the end ranges of three-dimensional shoulder elevation. The addition of three-dimensional or arced mobilizations, also known as mobilizations with movement, has been demonstrated to reduce shoulder pain at end-range elevation (Table 54.5) [7]. In addition, authors have suggested that oscillatory mobilizations to the thoracic spine reduce protective responses or guarding in patients with shoulder pain [8].

Joint mobility is necessary but not sufficient on its own to restore full mobility of the shoulder complex. In order to address soft tissue extensibility, low-load, prolonged stretching, or other PNF techniques such as contract-relax or alternating isometrics (agonist/antagonist) may be beneficial to allow for reduction of protective responses and progressive elongation of muscle as soft tissue adapts to newly gained joint mobility. If the athlete continues to complain of pain at end range, low-load sustained isometrics (70% maximum voluntary contraction at 45–60 second

hold, 4 reps, several times per day) may be helpful for pain reduction [9]. In the case of the point guard, she may continue to experience pain at end-range internal rotation and available flexion as she progresses through the stages of restoring full motion. Instructing her to perform an isometric hold at varying degrees through her elevation and rotation range as well as an isometric hold at the end of her available range will increase her tissue tolerance to load at those progressively increasing angles. Intermittent reports of increased tissue irritability as the athlete progressively loads the shoulder complex are typical and expected. Consequently, the rehabilitation specialist will need to use methods such as pain rating scales, palpation, and the presence of pain at rest or at night to assess irritability level and adjust the treatment plan accordingly by allowing time for rest and recovery or temporarily reducing tissue load. If pain at end ranges persists, the use of TENS at end ranges may be indicated.

During the middle phase of rehabilitation, the athlete should be prescribed increased loading of the musculotendinous tissues, such as 6–12 repetitions at 70 to 85% 1 RM to achieve muscle hypertrophy or <6 repetitions at more than 85% 1 RM to increase muscle strength. Loading progressions have been established to incorporate speed that begin with slow movement and little to no resistance progressing to moderate and fast speed movement in the absence of pain before increasing resistance and repeating the cycle [1]. If the point guard was strengthening at low angles in the early phase of rehabilitation, the middle phase for restoring strength should include a wide repertoire of strengthening modalities, such as open and closed kinetic chain and concentric and eccentric exercises, with an increasing demand on coordinating the scapulothoracic and glenohumeral joints (Table 54.6). As with range of motion restoration, the athlete may experience periods of increased irritability each time the load increases. By utilizing irritability assessment methods previously described, the rehabilitation specialist can objectively judge when progressing or regressing the load is appropriate. As soon as the athlete tolerates the load (resistance, speed, range, repetitions) with evidence of

Table 54.6 Strength progression

Early Middle Phase	Late Middle Phase
Open kinetic chain, single plane, full arc of motion Flexion Abduction External rotation	Multiplane flexion/abduction/external rotation, full arc of motion Concentric/eccentric
Closed kinetic chain Push-up progression → kneeling push-up → full body push-up → push-up plus for serratus anterior	Upper extremity star excursion or Y-balance

Table 54.7 Rules to aid progression/regression of therapeutic exercise based on the athlete’s response to the previous treatment. Soreness refers to the presence of delayed onset muscle soreness

Soreness rules	
Criterion	Action
Soreness during warm-up that continues	2 days off, regress resistance
Soreness during warm-up that goes away	Maintain same resistance
Soreness during warm-up that goes away but redevelops during session	2 days off, regress resistance
Joint soreness	1 day off and reassess
No soreness	Progress resistance

low tissue irritability (Table 54.7), load progression is appropriate. The goal of the middle phase of rehabilitation is to restore strength to within 90% of the uninvolved side.

Strengthening at all points in the available arc of motion of the shoulder complex is a necessary criterion for dynamic neuromuscular control. Given the shoulder’s extensive mobility, the complexity of neuromuscular control increases during this phase of rehabilitation. Increasing neuromuscular complexity would involve advancing from single-plane to multi-plane exercises as outlined in Table 54.6. While early-phase strengthening involves single plane, low-angle resistance training, the middle phase can begin combining planes of movement. For example, the point guard can begin lifting in combined planes of flexion/abduction/external rotation or flexion/adduction/internal rotation to prepare for overhead ball handling.

In order to prepare for return to sport, the middle phase of rehabilitation will need to restore the athlete’s ability to respond to perturbations associ-

Table 54.8 Neuromuscular training progression

Early middle phase	Late middle phase
Rhythmic stabilization × 2–4 min Patient position: Supine → sitting/standing → quadruped	Body blade × 2–4 min Patient position: Standing → standing on unstable surface → ½ kneel
Ball circles on wall 20 reps, clockwise/counterclockwise	Light weight ball toss/catch using a wall or rebounder, 20 reps

ated with ball handling. While the exercises described thus far incorporate anticipatory muscle activation for purposeful movement, the middle phase advances the athlete by incorporating reactive responses to perturbations. Rehabilitation specialists can introduce neuromuscular perturbations initially by using manual techniques such as rhythmic stabilization in a variety of patient positions: supine progressing to sitting or standing with the arm at varying degrees of elevation or quadruped with the therapist introducing perturbations at the shoulders or hips. Moving from a scapula-supported position such as supine to a seated or standing position requires more strength, control, and coordination between the GHJ and STJ. As patient tolerance and control improve, stabilization training can become more dynamic, involving weighted medicine balls or a body blade (Table 54.8). The point guard may enhance her neuromuscular adaptability by increasing the variability of her training conditions. She may stand on an unstable surface, such as a foam pad or ball, to progressively alter the attention demands of the exercise or training task, thereby increasing the pace of the neuromuscular adaptation. By increasing confidence and capability responding to perturbations, the athlete demonstrates a readiness to return to sports-specific tasks such as passing and rebounding. As the athlete’s response to external perturbations improves, apprehension at points in the available range will naturally decrease.

As the point guard begins meeting criteria for later-stage rehabilitation, progressing to sport-specific tasks such as passing or shooting layups and free throws is encouraged. These tasks involve the player and ball only, controlled variables that reduce the likelihood of prematurely exposing the athlete to loads that might be detrimental to return to play. The player may begin shooting activity

with a blocked practice design, standing at a specific chosen location and distance from the basket, shooting at a high number of repetitions. As she advances through this phase, practice conditions should change with location and shooting distance randomly chosen by the rehabilitation specialist. Given that the goal of the middle-phase rehabilitation is to prepare the athlete for sport-specific activity, restoring core and lower extremity strength and power, as well as aerobic capacity, is necessary. The point guard will need to demonstrate full lower extremity strength and power as measured by vertical leap and run vertical jump. Improved aerobic capacity will help her prepare aerobic readiness for full court running, cutting, and agility.

The following criteria enable the athlete to advance from the middle to late phase of rehabilitation:

- Full shoulder complex ROM (equivalent to the uninjured side)
- Involved limb strength $\geq 90\%$ of the uninjured limb
- Little to no pain or apprehension with strengthening, neuromuscular training, and controlled sport-specific activities

54.4 Late Phase

The main focus of late phase rehabilitation is graded return to activity and sports participation. Meeting

milestones of shooting/passing/catching without pain or apprehension is paramount as return to participation progressions will include increased volume, intensity, and unpredictable sport demands. Late-stage rehabilitation can be organized into peak-level sport demands, return to sport scenarios, and return to sport scenario volume.

Peak-level sports demands of the basketball shoulder are largely related to power training and reactive/dynamic stability. This may include exercises to target power, such as medicine ball passing or throwing, which can mimic conditions necessary for cross court passes or game plays that require quick ball movements or change of direction. Overload training is not always required for the purposes of power development, time between touches (time it takes a player to receive and pass the ball to an auditory stimulus) can also simulate plyometric and reactive stability needed for their sport. For our rehabbing point guard, power training and reactive stability with a medicine ball will begin with simple chest passes off a trampoline or with a stationary partner. The weight of the ball can be altered to increase load or a metronome to shorten the amortization required for quick catch/pass scenarios. In the absence of pain/apprehension, progression into overhead movements/postures may occur utilizing the same load/reactive stability principles. This can include bilateral diagonals then progressing to 1 handed throws from overhead positions (Figs. 54.9, 54.10 and 54.11). It is essential

Fig. 54.9 Bilateral ball throw allows the uninvolved limb to assist in throwing and catching the weighted ball

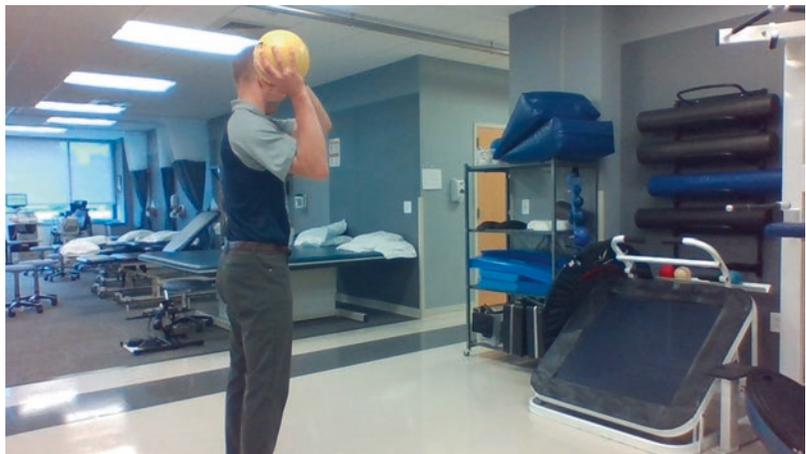


Fig. 54.10 Progression to unilateral ball toss and catch to work on plyometric contractions of the shoulder muscles



Fig. 54.11 Progression of ball toss to further into the overhead range of motion to more closely mimic the demands of basketball on the shoulder



to ensure graded stress of the tissue as well as challenge stability in ranges that could occur in sport to assist in secondary prevention of reinjury. Passing/throwing distance may also have to be increased without transitions, and is a primary skill set of the player that requires graded exposure. If trampoline equipment is not available, medicine ball throwing for distance is an acceptable alternative to work on power, and the use of rapid resistance band movements could simulate reactive stability and the amortization phase.

While power and reactive stability are essential for return to peak sport demands, it might not completely simulate the full demands of their sport. Basketball, particularly defending, requires physical contact and perturbation of the shoulder at high speeds and force. This can be seen in vari-

ous shoulder angles throughout a game including opposed rebounding, blocking a shot, boxing out on a free throw, taking on a pick, or attempting a steal. These scenarios may not be completely predictable, but progression using motor learning concepts and graded tissue exposure, discussed in the middle phase, can assist both the player and clinician in appropriate criterion progression. Once the players can demonstrate plyometric and reactive passing of the basketball without apprehension or pain, then adding an external stimulus or focus is suggested. These unanticipated events can be in the form of a visual cue, verbal cue, or un-cued. The purpose is for the player to not think about their intended shoulder movements and to focus on the game situation. For example with our athlete, her catch/pass plyometrics can

Table 54.9 Variables to stability/perturbation and plyometric exercises

Distance	Shots: Layups Jump Shots → Three pointers Passing: from the block to three-point line and vice versa → half court → full court (if needed)
Volume	Repetitions for power/max strength goal; sets (number of possessions in a game/expected playing time)
Shoulder angles	“Low five” position → “high five” position
Perturbations	Blocked contact → random contact Same intensity of contact → random intensity of contact
Level of competition	1 vs. 1 → live play without contact → live play with contact

be progressed to random targets which are cued by a coach pointing to a target or yelling numbers assigned to targets. Additionally, while defending, the opposing player may start by moving the same direction or performing a predictable move. This can be progressed to random directions or variable dribbling skills that need to be reacted toward. Once a player can perform upper extremity sport movements without internal focus on the task, then additional volume and stress can be placed on the shoulder (Table 54.9).

These variables should be applied and progressed gradually to best determine if one aspect is a greater limiter to a player’s return to performance levels.

The final aspect of the late-stage rehabilitation process is return to sports scenario volume. Sport scenario volume includes the player’s ability to perform all drill, skill, or volume-related tasks that practice or play might require. Restoration of the athlete’s capacity to complete all these scenarios is again to best prepare for unpredictable sport demands. Ignoring the known conditioning, playing, and practice demands of the position in basketball could hamper full return to their prior competitive level. This highlights the importance of continued aerobic and anaerobic conditioning throughout the rehabilitation stages and graded increases of skill volume in the late stages. When all prior peak physical performance and return to play scenarios have been achieved, the athlete then can begin to move into more unrestricted

practice participation. Organized programming is still suggested but should include variation in playing scenarios for the player to best prepare the shoulder for sport demand while still allowing the clinician to progress in a criterion manner. This could include performing skill work while in a fatigued state (shooting, passing, offensive/defensive skill work intermixed with anaerobic conditioning), intermixing skill work and live play (rebounding practice with offense live plays), or intermixing live play and conditioning (shuttle intervals performed after a turnover or change of possession) [10]. The end goal of this variability is to achieve the necessary amount of repetitions, playing time, and conditioning intensity/volume that mimics full sport demands.

Criterion for to move from late stage to return to sport:

- Power: Single Arm Shotput Test: $\geq 100\%$ if dominant arm; or $\geq 90\%$ if nondominant arm
- Strength: Isokinetic or isometric strength testing 90%; $\geq 100\%$ if dominant arm
- Sport demands: No apprehension or pain with full contact practice/conditioning demands
- Sport scenario volume: Unrestricted and symptom-free completion of all sport-specific tasks (e.g., shooting, passing, and rebounding)
- Patient reported outcomes: Disability of the arm shoulder hand sport $<10\%$, Penn Shoulder $>90\%$.

References

1. McClure PW, Michener LA. Staged approach for rehabilitation classification: shoulder disorders (STAR-shoulder). *Phys Ther.* 2015;95(5):791–800. <https://doi.org/10.2522/ptj.20140156>.
2. Duenas L, Balasch-Bernat M, Aguilar-Rodriguez M, Struyf F, Meeus M, Lluch E. A 12-week tailored manual therapy and home stretching program based on level of irritability and range of motion impairments in patients with primary frozen shoulder contracture syndrome: a case series with 9-months follow-up. *J Orthop Sports Phys Ther.* 2019;49(3):192–201.
3. Fermin S, Larkins L, Beene S, Wetzel D. The effect of contralateral exercise on patient pain and range of motion. *J Sport Rehabil.* 2018;27(2):185–8.
4. Shebab D, Adham N. Comparative effectiveness of ultrasound and transcutaneous electrical stimulation

- in treatment of periarticular shoulder pain. *Physiother Canada*. 2000;52(3):208–10.
5. Erculj F, Supej M. Impact of fatigue on the position of the release arm and shoulder girdle over a longer shooting distance for an elite basketball player. *J Strength Cond Res*. 2009;23(3):1029–36.
 6. Dutton M. Dutton's orthopaedic examination evaluation and intervention. 3rd ed. Morita J, Kearns B China: McGraw-Hill; 2012.
 7. Vooght L, de Vries J, Meeus M. Analgesic effects of manual therapy in patients with musculoskeletal pain: a systematic review. *Man Ther*. 2015;20:250–6.
 8. Sueki D, Chaconas E. The effect of thoracic manipulation on shoulder pain: a regional interdependence model. *Phys Ther Rev*. 2011;16(5):399–408.
 9. Rio E, van Ark M, Docking S. Isometric contractions are more analgesic than isotonic contractions for patellar tendon pain: an in-season randomized clinical trial. *Clin J Sport Med*. 2017;27(3):253–9.
 10. Klnç, F. An intensive combined training program modulates physical, physiological, biomotoric, and technical parameters in women basketball players. *The Journal of Strength & Conditioning Research*, 2008;22(6):1769–78.



Rehabilitation of Trunk, Hip and Groin Injuries in Basketball Players

55

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55.1 Introduction

Basketball is a sport that requires consistent, dynamic, high-intensity, multidirectional movement often in single-leg stance position, as well as demonstrations of repetitive explosive power and high-speed changes of direction. Basketball players commonly experience trunk, hip and groin (THG) pathology as a consequence of this compounding mechanical loading pattern, while treatment and management of such injuries are challenging for the rehabilitation professional.

The aim of this chapter is to discuss the landscape and multi-factorial issues that clinicians face when managing THG pathology and provide

a clear structure of a rehabilitation programme. While no authoritative protocol exists, guidelines and shared experience of the authors can serve to assist in the approach to care and further understanding of these types of injuries in basketball.

55.1.1 The Landscape

In order to accurately assess, treat and rehabilitate an injury to the THG, all clinicians must know the anatomical landscape, which is complex due to the overlapping nature of the structures. An accurate diagnosis can be challenging when considering the likelihood of concurrent pathology, episodic recurrence and the number of structures potentially impacted. As Ryan et al. [1] alluded to, the complexity of the diagnosis in hip and groin injuries creates a difficult rehabilitation prospect for clinicians.

THG pain can be caused by many different conditions as outlined in the DOHA consensus [2]; adductor-related, iliopsoas-related, inguinal-related, pubic-related (see Fig. 55.1), hip-related and referred pain from the lumbar spine and sacro-iliac joint.

Additionally, adverse and altered biomechanics can predispose a basketball player of any age to an injury, be it driven from pain or impaired movement control. In order to return to the sport of basketball quicker, safer and

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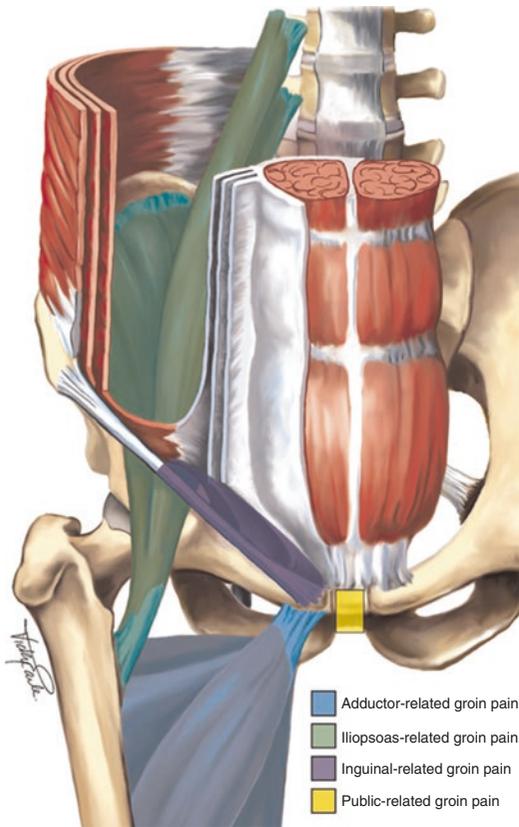


Fig. 55.1 Common sites of groin pain for the basketball player—adductor-related, iliopsoas-related, inguinal-related and pubic-related

with minimal chance of injury recurrence, it is crucial to determine the correct rehabilitation intervention based on an accurate assessment and be both progressive and equanimous throughout the complete programme.

With the increased prevalence of imaging techniques, coupled with a demand for imaging by athletes, there is likely an increased incidence and prevalence of THG diagnoses in clinical practice and the literature [3]. However, it may be possible to propose two main reasons why there is a high reported incidence and prevalence of THG injuries in basketball. Firstly, due to the nature of the multidirectional stop-start movements required in play load the THG region frequently. Secondly, as the game has evolved dramatically over the last decade, there

are increased physical demands on an athlete during a game and practice.

Jackson et al. [4] published a 24-year follow-up study and reported that 14.6% of all athletic-related injuries sustained in the NBA were associated with the THG, specifically the hip joint. Drakos et al. [5] demonstrated over a 17-year injury incidence surveillance study that hip pain and/or injury accounted for 11.5% of all injuries; the most common issues were strains and contusions.

Kerbel et al. [6] looked at collegiate sports with basketball included and reported an overall rate of hip and groin injury in 53.06 per 100,000 athlete-exposures, with non-contact and overuse mechanisms being the most prevalent. At the other end of the career spectrum, Ekhtiari et al. [7] reported that retired NBA athletes are at high risk of hip and groin pain after retirement and are more likely to require total hip arthroplasty compared with the general population.

Injuries to the THG can occur acutely, with movements like slipping or cutting sharply. Injuries can present themselves as an accumulation of chronic load or a spike in acute: chronic workload leading to the onset of pain and pathology (see Fig. 55.2 from [8]).

55.1.2 Differential Diagnoses of Trunk, Hip and Groin Injury

Diagnosing a basketball player who has a THG area injury is not always straightforward. High interregional anatomical crossover, varied nomenclature describing injuries and often misleading imaging results (such as MRI) of asymptomatic versus symptomatic players make establishing a specific diagnosis challenging.

One must not forget that while an acute injury to a specific anatomical site can be treated directly, chronic and cumulative stress injuries often have many factors to consider. Biomechanics and deficits in motor control capacity can predispose a player to pain and injury.

Common sites of pain and dysfunction in the THG region include the following:

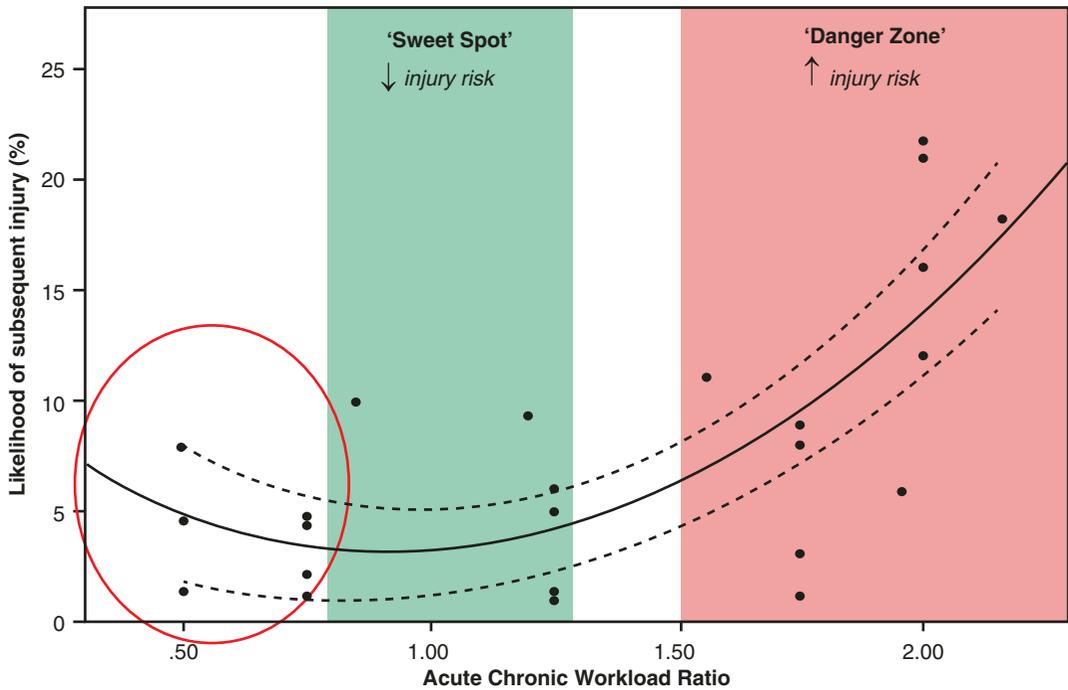


Fig. 55.2 Acute: Chronic workload ratio showing low injury risk area and high injury risk area

- Trunk/abdomen—rectus strain, oblique tear, abdominal hernia, non-specific low back pain [9]
- Hip—hip impingement, labral tear, hip flexor strain, bursitis
- Groin—strain, adductor longus tear, inguinal hernia

A pathoanatomical approach has been proposed as the most effective treatment model for the THG. A focus on individual sites of pain or dysfunction weighs less important than one thinks at first. Consequently, a treatment plan should not simply target identified singular structures and also aim to identify and treat any movement impairments.

55.1.3 Outcome Measures

THG injury presents with a large number of applicable outcome measures; these are very important to incorporate into a rehabilitation programme for initial and regular objective clinical

benchmarking, prior to clearance for return to basketball play.

1. Hip and Groin Outcome Score (HAGOS) is a self-reported questionnaire. It is a reliable, valid and responsive measure of hip and groin disability in athletic populations so would be fit for purpose and applicable for a cohort of basketball players [10].
2. Hip Dysfunction and Osteoarthritis Outcome Score (HOOS) consists of five subscales: pain, other symptoms, function in daily living (ADL), function in sport and recreation and hip-related quality of life (QOL).
3. Hip and Lumbar Range of Motion (ROM): Flexion, adduction, abduction and rotation (hip); flexion, extension, side flexion left, side flexion right—measured with a goniometer, using standard procedures.
4. Hip and Lower Limb Movement Screen (HLLMS) is a tool to identify biomechanical and movement control issues to aid in the rehabilitation programme design. The HLLMS is a battery of seven functional move-

ments developed at the Arthritis Research UK Centre for Sport, Exercise and Osteoarthritis (CSEO) [11] aiming to identify the athlete's ability to control lower limb movement. An inability to control movements or poor quality of movement may contribute to the insidious onset of, chronicity, or recurrence of musculoskeletal-driven pain at the hip and groin.

55.1.4 Rehabilitation and Return-to-Play Protocol

Larry Bird, the Boston Celtics hall-of-famer, once said, 'first master the fundamentals'. This strategy can be adopted with regard to a rehabilitation programme in addition to basketball, focusing on keeping things simple rather than overly complex.

Due to its complexity as previously mentioned, managing THG injuries and pain is tricky from a diagnostic and treatment perspective. A recent systematic review and meta-analysis [12] comparing the outcomes of surgery versus rehabilitation stated that despite the lack of high-quality studies, rehabilitation has significantly quicker return to play (RTP) times for pubic-driven injuries, and similar outcomes for other THG injury groups. The researchers concluded that rehabilitation paying heed to biomechanics should be the primary choice.

It is important to point out that a surgical consult may be considered when pain and range of motion restrictions are not improved, and overall symptoms are persisting without any positive changes. More often than not in these cases in elite basketball, the physiotherapist or physical therapist is the 'primary care' clinician. It is this person who considers onward referral for an orthopaedic consult when issues that cannot be treated conservatively present. There is more art through means of experience-driven clinical reasoning science in these cases.

There are occasions when groin surgery or similar is undergone and quicker RTP, acceptable outcomes and less episodic symptoms occur

rather than when managed conservatively. Following surgical treatment of femoroacetabular impingement (FAI), studies have shown no performance deficits when compared with matched controls in the NBA [13]. Moreover, a study of 24 NBA players showed a 100% return to play and no significant performance decline post hip arthroscopy for FAI [14].

The rehabilitation goals established for a basketball player require the inclusion of knowledge associated with both the physical and psychological demands of basketball and expectation of the athletes and medical team supporting the player. In relation to the physical demands of the game, a clinician needs to understand some key principles. Basketball is a game that requires lots of physical dexterity in that players need to be able to move quickly in many directions, jump repeatedly and sustain energy for long periods of high intensity whilst on court. Psychologically, a player needs to be in tune with his or her demands on the court (i.e. set play running and strategy), the role within the team, tactics for a certain rehearsed play, player or game, etc. Developing an intervention that meets the needs of a basketball player must include consideration of the most efficient method of returning one to basketball with minimal risk of compromising the healing process and minimal risk of re-injury [15].

Following on from a comprehensive subjective and objective assessment (see Fig. 55.3), an individualised and specific programme can be created to help the player with a THG injury (Fig. 55.4). This programme can be used as a template to adhere to for any of the chronic or acute issues a clinician could face in the field of basketball sports medicine and physiotherapy for a THG injury.

The steps include the initial clinical consultation, by the case manager or leading clinician as outlined (Fig. 55.4), phased physiotherapy and rehabilitation, movement analysis, if possible, follow-up with the case manager, on-court conditioning and return to play and game fitness.

Fig. 55.3 Standard evaluation form used in clinic for THG injury

History with CM (Case manager)- how did the injury take place; mechanism of injury?
 Severity- how bad is it? - severe, moderate or mild nature
 Pattern- day/night, worse at rest or with exercise, sharp or dull ache
 Aggravating and easing factors- ice, heat, NSAIDS, rest, movement *etc*
 Previous Injury History- to this area or other?
 Medical/ drug history- anything of note that could impact THG issue mentioned
 Diagnosis: Preliminary, differential, final
 Gaps analysis: nutrition, motivational/ behavioural psychology, sports medicine, physiotherapy (other areas that need addressing along with primary clinical injury)
 Outcome measure used- e.g. HAGOS
 Objective examination- observation, palpation of focused region, range of motion (all planes of movement in relation to the trunk, hip and groin), strength (e.g. using hand held dynamometer, adductor squeeze tests at varying degrees), special tests (Thomas test, FABER, FADIR, *etc*), functional movements (looking for quality and willingness to move)
 Short-term and long-term goals- 3 for each
 Recommended treatment plan (pool/gym/court)- split of pool, gym and court planned

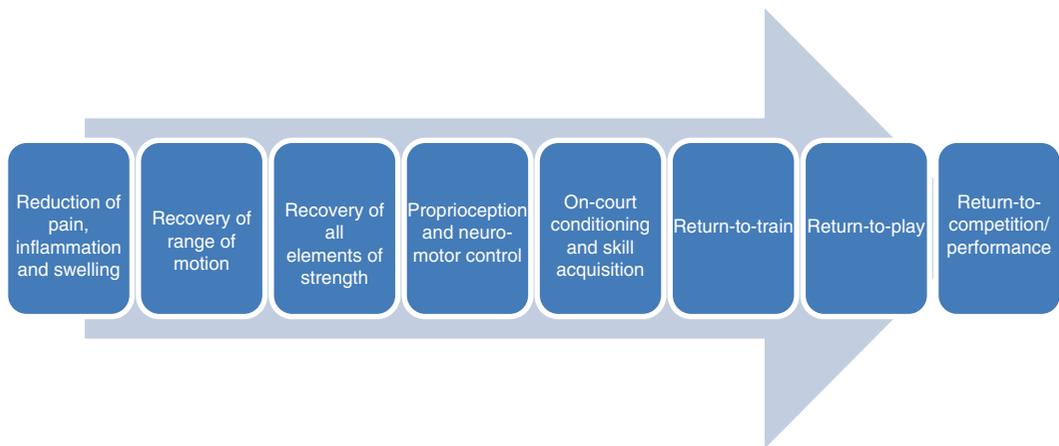


Fig. 55.4 Rehabilitation pathway for THG injury in a basketball player—each phase explained in greater detail below (adapted from model of Isokinetic Medical Group)

55.1.4.1 Reduction of Pain, Inflammation and Swelling

To achieve an optimal outcome, it is crucial to start well in the early stages after an injury, utilising isolated corrective exercises to target specific muscle dysfunction and weakness, such as using the Copenhagen protocol and exercises for adductor-related groin pain. It is elemental to retrain and recondition all exercises without the presence of pain. It makes little sense to further stress a joint or soft tissue structure that is already potentially overloaded. Modalities to reduce pain and inflammation must be utilised, and any swelling must be managed and resolved as soon as possible.

55.1.4.2 Recovery of Range of Motion

It is important to address deficits in range of motion, as examined during the assessment. This will further help create an optimal foundation for a more functional rehabilitation phase. Strategies should be employed to help increase joint and capsular mobility, which would also impact soft tissue in near proximity, such as a hip-based mobility routine or controlled articular rotations.

Furthermore, posture is often overlooked in rehabilitation, and yet it is so important for gait and static positions such as sitting or standing [16]. Ross et al. [17] stated that increased anterior tilting at the pelvis reduces hip flexion, adduction and

internal range of motion, which may lead to early onset of degenerative hip impingement. Thus, it helps to add postural cueing to encourage good pelvic setting.

55.1.4.3 Recovery of All Elements of Strength

Progressions should aim to reduce the focus on isolated strengthening whilst introducing more global and basketball-specific exercises. A key point here is to place a demand on the tissues to tolerate rapid loading capacities at lengthened states. These progressions in strength demands should integrate the previously dysfunctional muscles into motor patterns whilst gradually increasing the degrees of movement to build up movement complexity. Exercises should target the entire kinetic chain and utilise eccentric and concentric contractions, whilst continuing to build hip stability strategies.

At this point, generally following the attribution of adequate local and global muscle strengthening, compound multi-joint movements are used to challenge the ability of the athlete to maintain a neutral lumbopelvic posture. Common exercises for basketball players to target this area would include hip-hinge exercises such as deadlifts, Romanian deadlifts, goblet squats and split squats, as examples [16]. Please refer to Fig. 55.5 for a breakdown of some exercises that can be chosen for the trunk, hip and groin areas in rehabilitation.

Muscle strength testing provides a key role in understanding THG injury severity and for helping guide return to court decision-making. During the physical examination after an injury, testing gives immediate information on which activities the athlete can perform without the presence of pain or symptoms that could jeopardise a timely return to play.

During the testing of a groin injury the practitioner should assess muscle strength (see Fig. 55.6) and endurance (see Fig. 55.7) with some easy to do tests.

55.1.4.4 Proprioception and Neuro-Motor Control

One of the possible explanations for an injury to the THG is related to poor motor patterning due to

underlying muscle imbalances; the local stability system is often inhibited by pain and dysfunction, forcing the body to adopt compensatory patterns. These can in-turn lead to improper pelvic positioning during functional movements, resulting in uneven load distribution and site-specific overload.

The prescription of therapeutic exercises should be based on noted biomechanical factors, while considering current evidence and demands of basketball. A comprehensive approach should involve identifying and correcting imbalances and relative compensatory patterns, retraining movement competency, focusing on neuro-motor control patterning and developing lumbar-pelvic proprioception, stability and dynamic control.

At this point in the phases of rehabilitation, functional tests are crucial as milestones in the rehabilitation pathway and can help inform final decision-making calls with regard to an unrestricted return to the court with low re-injury risk. These are more commonly used towards the end of the gym-based work prior to progressing to on-court rehabilitation.

Here are some examples of common functional tests validated and deemed reliable for THG injury [18]:

Single leg squat
Star excursion balance test
Single leg hop for distance: anterior/medial/lateral
Triple hop
Change of direction tests (<i>t</i> -test, Illinois agility test)

Rehabilitation must follow a continuum from the therapy room to the court; however, it is important to remember that a player can take part in therapy exercises whilst on court, such as shooting from the free throw line on a chair, or balancing on one leg and shooting inside the key. It is useful to think of the end goal and build backgrounds from return to court play to injury [19].

It is important to remember that patience is a virtue and rather than becoming overly focused on individual impairments within rehab, a focus on global neuromuscular training and psychological readiness may provide the greatest risk reduction moving forward. A player returning to play prematurely represents not only an increased injury risk but also poses as a threat to achieving optimal performance.

Exercise	Trunk			Hip			Groin	
	Lat	Flex	Ext	Flex	Ext	Abd	IR	Add
SQUAT								
Back	+	-	++	-	++	++	-	+
Single Leg	++	-	+++	-	+++	++	-	+
Dumbbell	++	-	++	-	++	++	-	+
LUNGE								
Bulgarian	+	-	+	+	++	+	-	-
Barbell	+	-	++	-	++	++	-	-
Dumbbell	++	-	+	-	++	+	-	-
Lateral Slider	-	-	+	-	++	+	++	++
DEADLIFT								
Barbell	++	-	+++	-	+++	++	+	+
Sumo	++	-	+++	-	+++	++	++	++
Romanian	++	-	+++	-	+++	++	+	+
ISO BALL SQUEEZE								
90 - 90	-	-	-	+	-	-	+++	+++
Straight	-	-	-	-	-	-	+++	+++
HIP THRUST								
Barbell	-	+	++	-	+++	++	-	+
Banded	-	+	++	-	+++	++	-	+
PRONE								
Plank	+	++	-	+	-	-	-	-
- Hip Ext	+	++	-	+	++	-	-	-
COPENHAGAN								
ADDUCTION	++	+	-	-	-	+	+++	+++
Conventional								

Fig. 55.5 Table formatted as companion for exercise selection and prescription. These exercises should be chosen alongside sound and justified clinical reasoning and with athlete individualised interventions. +++ *Very effective*,

++ *Effective*, + *Small effect*, - *Negligible to minimal effect or unknown data*. (Scores are based off of clinical experience)

Fig. 55.6 Groin muscle testing—strength parameters examining the maximal strength as measured by a dynamometer or cuff. Squeeze 0°, Squeeze 90° (see Fig. 55.7 below), eccentric hip adduction, eccentric hip abduction, isometric hip flexion at 90°



Fig. 55.7 Groin muscle testing—endurance parameters examining how long the athlete can hold the position with optimal control and form. Side plank, long lever posterior tilt plank, Copenhagen adduction (see Fig. 55.7 below)



55.1.4.5 On-Court Conditioning and Skill Acquisition

The final stages of rehabilitation will be on-court, where adequate and appropriate high-speed and multidirectional movement takes place with a coach helping the performance team.

Basketball-specific conditioning, the restoration of linear running and cutting drills and cognitive training are employed at this point. The load, as measured and monitored by GPS accelerometry, should be gradually progressed to build towards an accumulative appropriate load for training. A player needs to be able to complete a full training scenario session at maximal intensity to build up tolerance and robustness of the tissue and body overall. Additionally, focusing on the re-introduction of adequate court vol-

ume and adequately dosed rehabilitation and strengthening exercises are critical to reducing the risk for re-injury.

55.1.4.6 Return-to-Train

A collaborative decision between rehabilitation professional, player and coaching team must be taken to integrate the player back into team-based on-court training. Involvement in pre-planned drills involving pre-determined movement requirements and skills are the starting platform for return to training for the player. Ongoing assessment of cardiovascular fitness levels specific to basketball, muscular strength and endurance, psychological readiness and overall team play integration are required to monitor injury risk potential associated with a timely return to play.

Chronological and outcome-based increments in performance demands are placed on the player as appropriate including unplanned movement patterning, landing mechanics, change of direction in response to on-court basketball demands.

55.1.4.7 Return-to-Play

The battery of strength, endurance and functional testing that was undertaken earlier on in rehabilitation must now be repeated to ensure an adequate recovery. The resultant decision must be based on hitting the needed parameters and benchmarks to avoid re-injury, return-to-training, return-to-play, competition and ultimately performance. Specific criterion which should be targeted before a return to play are strength deficits of less than 10% on the injured side compared with the uninjured side, particularly relevant in adductor-related groin pain. Clinically strength testing provides the clinician and player with a specific and easily implementable outcome marker as the player can act as their own comparative control.

Following on from these higher demands of open play in training, the athlete must reach not only an adequate level of court fitness but also a sufficient level of tactical knowledge and performance to return to play. Once the player has taken part in a comprehensive and consistent training schedule, the medical staff and coaching staff can agree on and coordinate a return to play.

55.1.4.8 Return-to-Competition/ Performance

An athlete who has utilised the rehabilitation process to return to play represents the best outcome for player, rehabilitation professional and coaching staff. For example, an athlete with a recurrent groin strain who not only focuses on adductor musculature strengthening but also addresses deficits in glute strength, hip mobility and lumbopelvic control may return with higher potential performance outcomes and a reduced injury risk. The real objective is for the basketball player to return to previous levels of performance in comparison to pre-injury levels or perhaps even exceed those levels.

Take-Home Messages and Concluding Remarks

Trunk, hip and groin injuries create a multitude of challenges to those involved in basketball sports medicine and performance science. Rehabilitation must address biomechanical issues that may compound an acute injury or may correlate with increased loading on tissues chronically over time. The clinician must blend principles of anatomy, movement and basketball-specific retraining drills in order to achieve a permanent change in the players' movement patterns and reduce re-injury risk.

Frequent testing of strength, endurance and functional parameters must be part of the rehabilitation plan that is grounded by outcome measures taken during the initial assessment period. Finally, rehabilitation should follow a robust and graduated phased approach from early stages through to completing all pertinent return to play criteria to enable a safe return to full basketball play and competition.

References

1. Ryan J, DeBurca N, McCreesh K. Risk factors for groin/hip injuries in field-based sports: a systematic review. *Br J Sports Med.* 2014;48:1089–96.
2. Weir A, Brukner P, Delahunt E, et al. Doha agreement meeting on terminology and definitions in groin pain in athletes. *Br J Sports Med.* 2015;49:768–74.
3. Griffin DR, Dickenson EJ, O'Donnell J, Agricola R, Awan T, Beck M, Cholis JC, Dijkstra HP, Falvey E, Gimpel M, Hinman RS, Hölmich P, Kassarian A, Martin HD, Martin R, Mather RC, Philippon MJ, Reiman MP, Takla A, Thorborg K, Walker S, Weir A, Bennell KL. The Warwick Agreement on femoroacetabular impingement syndrome (FAI syndrome): an international consensus statement. *Br J Sports Med.* 2016;50:1169–76.
4. Jackson, T. J., Starkey C., McElhiney D., Domb B. (2013) 'Epidemiology of hip injuries in the National Basketball Association: a 24-year overview', *Orthop J Sports Med* 1(3).
5. Drakos M, Domb B, Starkey C, Callahan L, Allen AA. Injury in the National Basketball Association- a 17 year overview. *Sports Health.* 2010;2(4):284–90.

6. Kerbal, Y., Smith C., Nzeogu M., Prodromo J. (2018) 'Epidemiology of hip and groin injuries in collegiate athletes in the United States, *Orthop J Sports Med* 6(5).
7. Ekhiari S., Khan M., Burrus M.T., Madden K. (2019) 'Hip and groin injuries in professional basketball players: impact on playing career and quality of life after retirement' *Sports Health*, 11(3).
8. Blanch P, Gabbett T. Has the athlete trained enough to return to play safely? The acute: chronic workload ratio permits clinicians to quantify a player's risk of subsequent injury. *Br J Sports Med*. 2016;50(8):471–5.
9. Cabri J, Sousa JP, Kots M, Barreiros J. Golf-related injuries: a systematic review. *Eur J Sport Sci*. 2009;9:353–66.
10. Thorborg K, Hölmich P, Christensen R, et al. The Copenhagen Hip and Groin Outcome Score (HAGOS): development and validation according to the COSMIN checklist. *Br J Sports Med*. 2011;45:478–91.
11. Booyesen N, Wilson D, Hawkes R, Dickenson E, Stokes M, Warner M. Characterising movement patterns in elite male professional golfers using an observational hip and lower limb movement screen. *Osteoarthr Cartil*. 2017;25(1):356–9.
12. King E, Ward J, Small L. Athletic groin pain: a systematic review and meta-analysis of surgical versus physical therapy rehabilitation outcomes. *Br J Sports Med*. 2015;49:1447–51.
13. Jack RA, Sochacki KR, Hirase T, Vickery JW, Harris JD. Performance and return to sport after hip arthroscopy for femoroacetabular impingement in professional athletes differs between sports. *Arthroscopy*. 2019;35(5):1422–8.
14. Begly JP, Buckley PS, Utsunomiya H, Briggs KK, Philippon MJ. Femoroacetabular impingement in professional basketball players: return to play, career length, and performance after hip arthroscopy. *Am J Sports Med*. 2018;46(13):3090–6.
15. Konin JG, Nofsinger CC. Physical therapy management of athletic injuries of the hip. *Oper Techn Sports Med*. 2007;15(4):204–16.
16. Joyce D., Lewindon D. Sports injury prevention and rehabilitation. Routledge, 2016;312–4.
17. Ross JR, Nepple JJ, Philippon MJ, Kelly BT, Larson CM, Bedi A. Effect of changes in pelvic tilt on range of motion to impingement and radiographic parameters of acetabular morphologic characteristics. *Am J Sports Med*. 2014;42(10):2402–9.
18. Pruna R, Andersen TE, Clarsen B, McCall A. Muscle injury guide: prevention of and return to play from muscle injuries. Barcelona: Barca Innovation Hub; 2018. p. 61–2.
19. Knowles B. Return to competition strategies for the joint compromised athlete' Isokinetic Conference personal communication, London; 2016.



Rehabilitation of Knee Injuries in Basketball Players

56

Amelia J. H. Arundale

56.1 Introduction

At every level of play, knee injuries are some of the most common, and often most severe, injuries in basketball [1–4]. In the National Basketball Association (NBA), knee injuries (patella and knee joint combined) account for 19.1% of injuries, and result in more missed games than ankle and lumbar spine injuries, the two most common injuries, combined [3]. In the Women’s NBA (WNBA), knee injuries account for 22.5% of all injuries, with a knee injury rate of 4.4/1000 athletic exposures, almost twice the rate of the NBA (2.5/1000 athletic exposures) [2].

Although much of the rehabilitation literature focuses on anterior cruciate ligament (ACL) injuries, other knee injuries common in basketball include meniscal, chondral, patellofemoral or anterior knee pain, patella tendinopathy, medial (MCL) or lateral collateral ligament (LCL), or less commonly posterior cruciate ligament (PCL) injuries [1]. This chapter will focus on relevant information in the rehabilitation of all knee injuries, with notes related to specific injuries. For

more details on treating patella tendinopathy, see Chap. 55.

The goal of knee rehabilitation, regardless of injury or surgical intervention, is to allow an athlete to safely return to activities of daily living and, when desired, to sport. Working with the athlete to identify goals both specific to their injury as well as to basketball and their wider life is important to building a tailored rehabilitation plan. Engaging the athlete in the rehabilitation process, from goal setting to planning and execution, is beneficial both for their engagement and motivation as well as for setting appropriate expectations [5].

56.2 Rehabilitation Content

There are multiple clinical practice guidelines on knee and ACL injury rehabilitation to direct clinicians in building rehabilitation programs aligned with the best available evidence [6, 7]. Although not specific to basketball, the best practices presented in clinical practices guidelines should be followed by clinicians and performance teams.

In the case of an ACL injury, or other injury requiring surgical intervention, there is significant value in the athlete performing a period of rehabilitation prior to surgery. This pre-operative rehabilitation, or prehab, allows the athlete time to regain range of motion, decrease effusion, and

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build strength [8–10]. Building quadriceps strength pre-operatively is related to greater strength post-operatively [8]. Quadriceps strength is not the only benefit for prehab; athletes who participate in pre- and postoperative rehabilitation have better patient-reported outcomes 2 years after ACL reconstruction [8–10]. Prehab is not always possible and it does not replace high-quality postoperative rehabilitation, but it is an important component to knee rehabilitation that should be considered for the long-term outcomes of the athlete.

Rehabilitation progression should be guided by objective milestones and regular assessment along with feedback from the athlete. Milestones should be tailored to the unique needs of the athlete and will help the clinician gauge the athlete's progress as well as identify deficits. Early milestones often include range of motion, strength, and normalizing gait. These are important objective markers because deficits in extension range [11], quadriceps strength [12, 13], and aberrant biomechanics (stiffening gait, dynamic valgus, and unloading the involved knee) [14–16] are associated with the development of osteoarthritis.

Motivation, fear of reinjury, and confidence are also important in the rehabilitation process. Athlete engagement in the rehab planning and assessment can help educate, engage, and motivate an athlete, particularly in longer rehabilitations, such as after ACL reconstruction [5]. Regular assessment of an athlete's self-reported function, through patient-reported outcome measures such as the International Knee Documentation Committee's 2000 Subjective Knee Form (IKDC) or the Knee Injury Osteoarthritis Outcome Score (KOOS), can give clinicians insight into how the athlete views their knee function [17, 18]. The KOOS includes five subscales: symptoms, pain, function during activities of daily living, function during sport and recreational activities, and quality of life, allowing clinicians to hone in on particular areas where an athlete might be struggling [18]. The Anterior Cruciate Ligament Return to Sport after Injury Scale (ACL-RSI) is another patient-reported outcome measure valuable in assessing an athlete's progression through rehabilitation.

The ACL-RSI assesses psychological readiness to return to sport via questions regarding the athlete's emotions, confidence, and risk appraisal related to returning to sport [19].

Strengthening the involved and uninvolved lower extremities is an important part of rehabilitation for the knee of any athlete, but particularly basketball players. Knee effusion is common with many knee injuries, and can inhibit quadriceps muscle function [20]. RICE (rest, ice, compression, elevation) or POLICE (protection, optimal loading, ice, compression, elevation) [21] after acute injury to reduce inflammation can help minimize the effects on quadriceps activation. Neuromuscular electrical stimulation can also facilitate quadriceps activation [7]. Strengthening of the entire lower extremity, not just the quadriceps, is valuable to provide stability to the knee and lower extremity. The hamstrings, quadriceps, glutes, gastrocnemius, and soleus, all play significant roles in running, jumping, and cutting which are integral to basketball, making their strength essential. Baseline measurements, such as preseason measurements, of strength can be helpful markers for clinicians to use as they assess progress through rehabilitation or readiness to return to sport.

Building or rebuilding neuromuscular control is also a key component of rehabilitation. As basketball requires quick accelerations and decelerations, running, cutting, pivoting, and jumping at high speeds, the ability to control the lower extremity during these movements is essential. Knee valgus collapse, or the combination of hip adduction, hip internal rotation, and knee abduction, during jumping, running, and cutting has been linked to knee injury risk [16, 22, 23]. Thus, building single-leg balance, stability, and control to be able to avoid high-risk movement patterns is likely important for secondary knee injury prevention. For example, training neuromuscular control generally with simple exercises such as stationary single-leg balance activities with a basketball, building to slower controlled movements such as footwork patterns, before progressing to higher speeds and more demanding moving tasks.

It is important for clinicians to consider the demands of basketball as they plan their athlete's rehabilitation. Basketball requires cardiovascular fitness and the ability to perform repeated accelerations and decelerations. In addition to horizontal-based movements, basketball also has a significant vertical component with jumping involved in many actions from shooting to rebounding. All of these horizontal and vertical movements place a high demand on the athlete's knee. Thus, progressively building an athlete's load so that the joint can handle the impact and forces, and so that the athlete can handle the overall loads of basketball, is important. Basketball, particularly at higher levels, also involves a large amount of physical contact. Rehabilitation must gradually expose and prepare athletes to return to these physical and physiological demands.

Rehabilitation for a basketball player after a knee injury will also need to be tailored to their position. Guards will spend more time with a ball in their hands, thus dribbling exercises (Fig. 56.1a), endurance in a squat position, as well as building toward cutting/pivoting at high speeds is important. Forwards/wings have some dribbling responsibilities, however are often looked to for shooting. Thus, jumping, rebounding, and shooting are key foci, as well as driving to the basket (Fig. 56.1b). Driving to the basket can involve contact, thus preparing athletes for unexpected

perturbations and agility through tight spaces between people is valuable (with/without contact). Centers traditionally are relied on for rebounding and scoring from under the basket (Fig. 56.1c). Centers must be very physical as they are often relied on to set screens, sometimes resulting in significant contact. Rehabilitation for centers must prepare them to be stable in a squat/screen position with unexpected physical perturbations. The role of a center may vary from team to team, some playing in a role closer to a wing, thus adjusting rehabilitation to the demands that the player will face is crucial to fully prepare them for a safe return to sport.

56.3 Rehabilitation Progression

Progression of exercise during knee rehabilitation should be guided by the athlete's subjective reports and objective clinical measures. Throughout rehabilitation markers such as effusion, joint soreness, and pain can be used to gauge how the athlete's knee is responding. Guidelines for using soreness and effusion to adjust exercise progression have been published (Table 56.1) [24, 25].

Good guidance for all exercise progression is centered around principles of exercise progression: specificity, overload, progression [27]. In

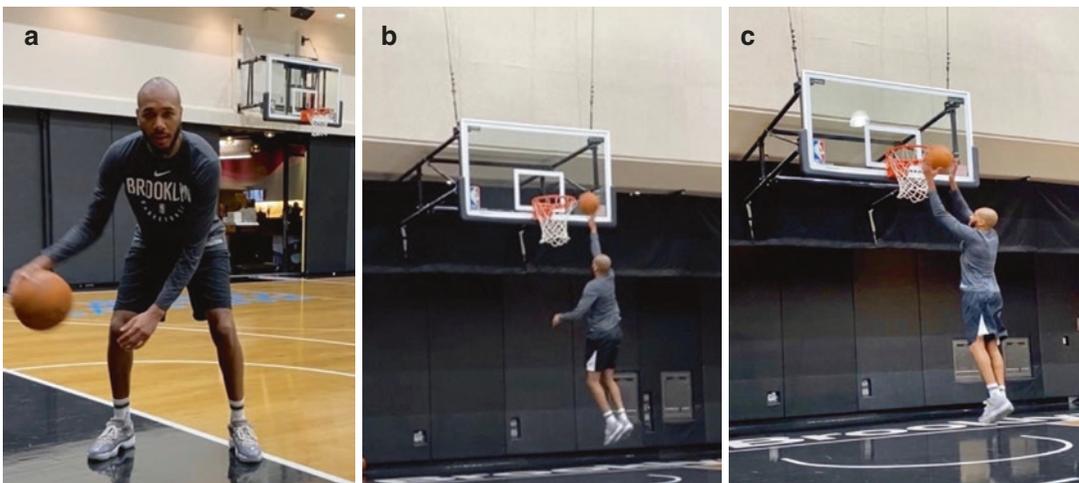


Fig. 56.1 (a) Dribbling. (b) Lay-up. (c) Rebounding

Table 56.1 Soreness and effusion guidelines [24, 25]

No soreness	Advance
Soreness guidelines	
If sore during warm-up but soreness is gone during warm-up	Repeat previous day's session
If sore during warm-up and soreness continues with initiation of exercises	Stop; take 2 days off; and upon return repeat session from 2 days prior
If sore more than 1 h after session or the next day	Take 1 day off; repeat previous day's session
Effusion guidelines	
1+ effusion or less	Complete planned session and advance as appropriate
2+ effusion or larger	Hold session, treat effusion, and review effusion management with athlete At the next session: <ul style="list-style-type: none"> • If trace or less effusion: Advance as appropriate. • If 1+, maintain same level of exercise. • If 2+, continue effusion management.

This table is written as if the clinician is seeing the athlete daily. If the athlete is being seen less frequently, adjust accordingly. Effusion is graded using the sweep test [26]

other words, exercises should target a particular structure (specificity); they should challenge the athlete's current abilities (overload), and be planned so that they continue to challenge the athlete over time (progression). Two models which clinicians may find helpful in building their progressions are Blanchard and Glasgow's [28] model for exercise progression and Taberner et al.'s [29] control to chaos continuum.

Given the emphasis in basketball knee rehabilitation on building good neuromuscular control, it is beneficial to the athlete to use principles of motor control and learning. Gokeler et al. [30] laid out four principles of motor learning that are helpful in guiding a clinician's rehabilitation implementation. The principles are as follows: (1) external focus of feedback, (2) implicit learning, (3) differential learning, and (4) self-controlled learning and contextual interference [30]. These principles are described in more detail in Chap. 60 On-Court Rehabilitation.

The author is of the opinion that a basketball should be integrated into rehabilitation even from the acute stages. This can be from holding a ball during core exercises to shooting while seated in early phases to building on-court rehabilitation in to later phases of rehabilitation. More details on integrating a basketball into rehabilitation are covered in Chap. 60.

Regardless of the knee injury, timelines for healing must be an integral part of rehabilitation planning and exercise progression. After ACL reconstruction, it is possible that graft healing may take 9–24 months, with bone bruises concomitant with ACL injuries taking as many 12–16 months to heal [31]. MCL injuries, depending on the grade, may take a few weeks to months to heal, allowing for scar tissue to build and mature. Chondral injuries, particularly if treated surgically with procedures such as osteochondral allograft transplantation, may require extended periods of time non-weight-bearing. Surgical or surgeon protocols will determine the progression of weight-bearing as well as time before return to sport is allowed. It is important that these timeframes are respected to allow the injured structures to heal, and build up the athlete's exposure to the demands that they will face on-court before they return to play. One caveat is that while time from injury/surgery is needed as one of the objective criteria for return to sport, it cannot be the only criteria [32].

56.4 Rehabilitation Phases

The phases of rehabilitation and duration of each phase will vary based on the injury as well as numerous other factors. Progression from one

phase to another should be based on objective criteria laid out at the beginning of rehabilitation. The potential phases presented here are focused on a rehabilitation perspective and do not include progression of cardiovascular fitness or upper/general lower extremity strengthening that must be advanced simultaneously with rehabilitation.

56.4.1 Prehabilitation (In the Case of Surgical Management, Particularly of ACL Injuries)

Focus: The primary focus of prehabilitation is to “quiet the joint”, reducing inflammation, improving range of motion, and maximizing quadriceps strength in an attempt to maximize postsurgical outcomes [8, 33].

Goals:

- Full range of motion (active and passive)
- Minimal to no effusion
- 90% quadriceps strength limb symmetry [8]

56.4.2 Acute (Post-Injury or Post-Surgery Depending on Treatment Course)

Focus: The acute phase is focused on reducing inflammation and restoring basic function.

Goals:

- Full range of motion (active and passive)
- Minimal to no effusion
- No quadriceps lag with straight leg raise
- Symmetrical walking gait pattern

56.4.3 Intermediate

Focus: The focus of the intermediate phase is to restore activities of daily living, progress strength and neuromuscular control to prepare the athlete for the higher-level demands of sport-related tasks.

Goals:

- Pain-free and unrestricted activities of daily living
- $\geq 80\%$ quadriceps strength limb symmetry
- Control of the knee during dynamic weight-bearing, particularly single-leg, tasks
- Able to control knee under load and in closed kinetic chain positions
- Return to running initiated

56.4.4 Late

Focus: Late phase rehabilitation focuses on more complex and dynamic movements and tasks related to sport. This phase includes return to play progressions on- and off-court.

Goals:

- Identify and expose the athlete to the demands that they will face upon return to basketball
- Meet or exceed all return to sport criteria set out by medical team
- Able to move on- and off-court with good neuromuscular control
- Able to control knee during high-speed cutting, pivoting, and jumping activities

56.4.5 Return to Performance/ Secondary Prevention

Focus: Once an athlete has returned to play, this phase of rehabilitation involves helping the athlete reach or exceed their preinjury level of performance on and off the court. Further, this phase must include ongoing efforts to prevent subsequent injuries, particularly knee injuries.

Goals:

- On-going performance of a knee injury prevention program
- Meet or exceed preinjury performance indicators (performance indicators could be performance-based, such as acceleration/ decelerations per game, or basketball-based, such as points or rebounds per game.)

56.5 Special Considerations for Particular Injuries

Many aspects of basketball knee rehabilitation are similar across injuries. Some injuries do require particular attention. For example, after MCL injuries shooting needs to be monitored closely. Many basketball players collapse into a valgus pattern (hip adduction, hip internal rotation, and knee abduction) when they shoot (Fig. 56.2a). As valgus stresses the MCL, it is important that even low-level shooting drills be done with proper squat form to avoid stress on the healing MCL (Fig. 56.2b). A clinician's emphasis on good form should continue through to athletic tasks, such as cutting and lateral changes in direction which also place high stresses on the MCL.

Clinicians' awareness during rehabilitation of cartilage and meniscal injuries should be around weight-bearing stress and impact. Weight-bearing timelines after chondral or meniscal surgery should be strictly adhered to. Clinicians can use devices such as accelerometers or inertial measurement units to quantify exactly how much load is going through the knee joint. Thus, once weight-bearing is allowed, clinicians should map out a gradual increase in load, building toward a full return to basketball, to avoid over- or under-loading the tissue.

PCL injuries are rare in basketball [3, 4]. Rehabilitation of a PCL injury must heavily emphasize quadriceps strength and avoid hamstring activation early after injury/surgery [34]. The quadriceps act as a stabilizer after PCL injury/surgery, but the hamstrings can pull the tibia posterior potentially stressing healing tissues [34]. Given the importance of both the hamstrings and quadriceps acting both concentrically and eccentrically during running, jumping, cutting, and pivoting, clinicians must help the athlete appropriately build the strength of both muscle groups so that they can stabilize the knee as well as perform the movements needed to play basketball.

56.6 Return to Sport Decision-Making

The decision of when an athlete is ready to return to sport is one of the most difficult that sports medicine professionals have to make. As such, the decision should not be made by one clinician, but rather as a collaboration of those involved in the diagnosis, treatment, rehabilitation, and training. The athlete should play an integral role in decision-making, as well as coaches.

Although time from injury or surgery is an easy criteria, time alone cannot be used to

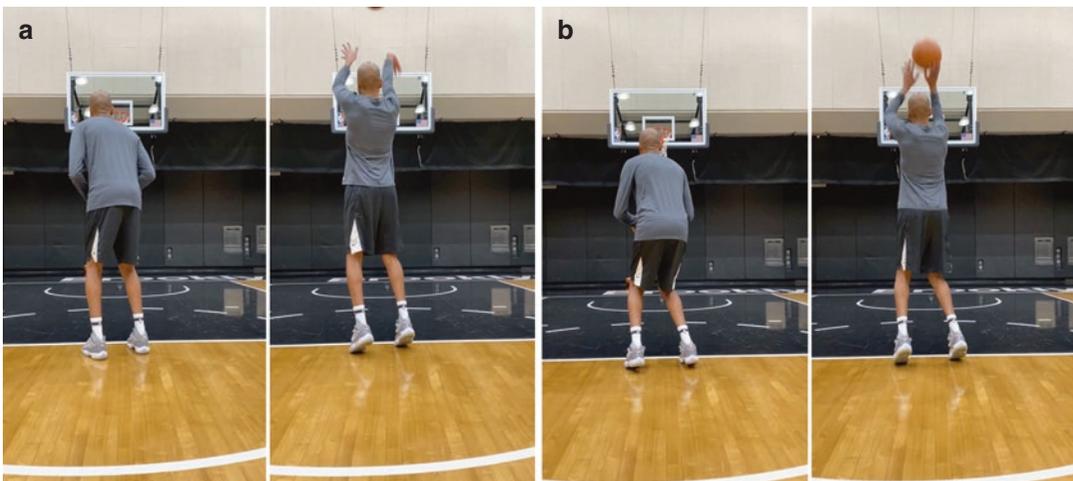


Fig. 56.2 (a) Free throw. (b) Free throw with knee valgus

make a decision on whether or not an athlete is ready to return to basketball after knee injury [32, 35]. Healing timeframes must be adhered to, but objective criteria are necessary to best advise medical teams on when an athlete is safe to return. Currently, there is no gold standard or ideal set of criteria [35–38]. Criteria must be tailored to the individual athlete. Clinicians should use the Strategic Assessment of Risk and Risk Tolerance (StARRT) framework [39] for return-to-play decision-making in planning return to sport criteria. The StARRT framework has clinicians first assess the athlete’s health risk, evaluating factors such as demographics, signs, symptoms, injury and medical history, to evaluate the tissue health. Next, the framework recommends clinicians assess the risks associated with activity, for example, the type of sport, position, competitive level, the ability to protect the injury, as well as the athlete’s psychological readiness, all factors that will affect the tissue stress. Finally, the StARRT framework prescribes assessment of risk tolerance and modifiers of the risk, such as time in the season, external pressures, conflicts of interest, risk/fear of litigation, and pressure from the athlete. By examining from a tissue level up to the external pressures and stakeholders, the StARRT framework guides clinicians toward building specific criteria that will hopefully facilitate the safest return to basketball for the athlete.

Although there is no gold-standard return to sport criteria, the literature indicates that after ACL reconstruction using a battery of return to sport criteria likely decreases the risk of a future knee injury and future ACL injury (Table 56.2) [12, 35, 37, 38, 40].

Return to sport testing should likely also include assessments of an athlete’s cardiovascular capacities as well as their ability to perform basketball-type movements. The YoYo Intermittent Recovery Test is a valid and reliable test [45]. The YoYo test results can be used to estimate VO_{2max} [46] or be compared to preinjury data, for a relative understanding of the athlete’s progress. Recording the fitness and basketball work the athlete has performed, what could be referred to as their chronic workload is also

Table 56.2 Potential return to sport criteria after knee injury

Criteria common in the literature [12, 35, 40]
≥ 90% quadriceps strength limb symmetry (isometric or isokinetic)
≥ 90% on single-legged hop tests (single hop, triple hop, and cross over hop for distance, and the 6 m timed hop)
≥ 90% score on the Knee Outcomes Survey—Activities of Daily Living
≥ 90% global rating of perceived knee function (0–100%, with 100% being preinjury level of function)
≥ 90% score on the Anterior Cruciate Ligament Return to Sport after Injury (ACL-RSI) Questionnaire
Other potential criteria
Quadriceps strength equal or greater than preinjury
≥ 90% preinjury strength of other lower extremity muscles such as hamstrings, gluteus medius, and adductors
Achieved maximum preinjury running speed (both running straight as well as during play)
Preinjury or better scores on cardiovascular test such as the YoYo Intermittent Recovery Test
Preinjury or better scores on tests related to movements involved in basketball such as 10 m acceleration, 505 test [41, 42], and jump height
Achieved preinjury or better score on a reliable movement assessment such as Nae et al. [43]
Performed multiple sessions of high-speed running
Performed on-court progressions involving increasing contact as well as increasing number of players, such as those laid out in Chap. 60
Completed at least one (ideally multiple) full training/practice session with team
Built up chronic work load sufficient to allow for no large spike in workload upon return to play [44]

important to determine how much an athlete can be safely pushed on court without putting them at a high risk for a new injury [44]. Among other techniques, recordings could be made using an athlete’s rate of perceived exertion, heart rate monitor, or accelerometer (see Chaps. 64 and 65 for more information).

Tests such as 10 m acceleration, the 505 test [41, 42], or jump height can help the medical team understand the athlete’s progress with regard to acceleration, deceleration [47], change of direction, and jumping. These tests can help assess the knee’s tolerance to higher forces and stresses that mimic basketball. A basketball can be added to tests, such as sprint dribbling 10 m or

dribbling the 505 test. The time difference between performing the test with and without a basketball can be referred to as the dribble deficit or the extent that dribbling affects the athlete's speed [47].

Further, an athlete's ability to perform basketball-specific movements, particularly with good neuromuscular control, may be important in reducing their future injury risk [16]. There is significant discussion in the literature on whether return to sport testing should be performed in a fatigued or non-fatigued state [48, 49]. However, it may be of value to the clinician to assess the athlete in various states, such as no fatigue, cardiovascular fatigue, and cognitive fatigue as the athlete may perform differently in each state and could help identify deficits that require attention.

56.7 Return to Play, Return to Performance, and Secondary Prevention

It is easy for a clinician to assume that once an athlete has returned to training or to game-play that rehabilitation is done. However, there is a difference between an athlete returning to play and returning to their preinjury level of performance [50]. A return to training could be considered, an athlete returning to sports participation [50]. Participation may be followed by a return to play, where an athlete begins to play games, but that return to play does not necessarily mean that the athlete is performing physically or sporting-wise at the same level as they did prior to their injury. The athlete's return to performance is when they are able to achieve key performance indicators equal to or greater than their preinjury level of play.

Return to performance is discussed in greater detail in Chap. 60 (On-Court Rehabilitation); important factors for clinicians to consider are identifying key performance indicators, both from a physical-performance and basketball-performance point of view. Physical-performance

indicators could include accelerations or decelerations per minute, total distance covered, or total distance covered above a certain speed. Basketball-performance indicators could be points, rebounds, assists, or blockers per game. These performance indicators will help clinicians and coaches fairly judge an athlete's performance as well as identify areas where they may still need work.

In addition to a return to performance, clinicians have a duty of care in assisting an athlete prevent a future injury. After any knee injury, but particularly after ACL injury, athletes are at a higher risk for a new knee injury [51]. Incidence of a second ACL injury, ipsi- or contralateral, is as high as 30% [52], with younger and female athletes being at higher risk.

Secondary prevention, or preventing an injury from worsening or reoccurring, once an athlete returns to play can take numerous forms. Some known modifiable risk factors for future injury include biomechanical risk factors [16] as well as workload [44]. Modifying workloads, with the goal of reducing injury risk, is discussed in more detail in Chaps. 64 and 65. Biomechanical risk factors can be addressed as part of a neuromuscular injury prevention program, or in clinician designed neuromuscular training built as an extension of the athlete's rehabilitation and continuing work on deficits identified and addressed but not eliminated during rehabilitation. Neuromuscular prevention programs, designed for primary prevention, such as the 11+ (previously known as the FIFA 11+) [53, 54] or Knäkontroll [55] programs have demonstrated effectiveness in soccer players in preventing injuries, particularly knee injuries, and may hold benefit for basketball players. There is very little literature on prevention in basketball players; however programs such as the Sportsmetrics program demonstrate potential benefit in women [56]. Common elements to effective programs include ≥ 20 min per prevention session, ≥ 30 min total volume per week, and exercise components that involve strengthening and plyometrics [57].

Take Home Messages

- Planning is essential. From the initial injury, performance/medical teams should work with the athlete and coaches to develop a plan for rehabilitation, including objective criteria for progression and return to sport as well as key performance indicators for assessing return to performance.
- Rehabilitation can be monitored and advanced using soreness and effusion guidelines as well as the principles of exercises progression (specific, overload, progression).
- Rehabilitation should be tailored to the needs of the athlete, such as their position.
- Objective return to sport criteria should be specific to the athlete. Use of the StARRT framework can help guide clinicians in developing appropriate criteria.

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References

1. Arendt E, Dick R. Knee injury patterns among men and women in collegiate basketball and soccer. Ncaa data and review of literature. *Am J Sports Med.* 1995;23(6):694–701.
2. Deitch JR, Starkey C, Walters SL, et al. Injury risk in professional basketball players: a comparison of women's national basketball association and national basketball association athletes. *Am J Sports Med.* 2006;34(7):1077–83.
3. Drakos MC, Domb B, Starkey C, et al. Injury in the national basketball association: a 17-year overview. *Sports Health.* 2010;2(4):284–90.
4. McCarthy MM, Voos JE, Nguyen JT, et al. Injury profile in elite female basketball athletes at the women's national basketball association combine. *Am J Sports Med.* 2013;41(3):645–51.
5. Ardern CL, Taylor NF, Feller JA, et al. A systematic review of the psychological factors associated with returning to sport following injury. *Br J Sports Med.* 2013;47(17):1120–6.
6. van Melick N, van Cingel REH, Brooijmans F, et al. Evidence-based clinical practice update: practice guidelines for anterior cruciate ligament rehabilitation based on a systematic review and multidisciplinary consensus. *Br J Sports Med.* 2016;50(24):1506–15.
7. Logerstedt D, Scalzitti D, Risberg MA, et al. Knee stability and movement coordination impairments: knee ligament sprain revision 2017. *J Orthop Sports Phys Ther.* 2017;47(11):A1–A47.
8. Eitzen I, Holm I, Risberg MA. Preoperative quadriceps strength is a significant predictor of knee function two years after anterior cruciate ligament reconstruction. *Br J Sports Med.* 2009;43(5):371–6.
9. Failla MJ, Logerstedt DS, Grindem H, et al. Does extended preoperative rehabilitation influence outcomes 2 years after ACL reconstruction?: a comparative effectiveness study between the moon and Delaware-Oslo ACL cohorts. *Am J Sports Med.* 2016;44(10):2608–14.
10. Grindem H, Granan LP, Risberg MA, et al. How does a combined preoperative and postoperative rehabilitation programme influence the outcome of ACL reconstruction 2 years after surgery? A comparison between patients in the Delaware-Oslo ACL cohort and the Norwegian National Knee Ligament Registry. *Br J Sports Med.* 2015;49(6):385–9.
11. Shelbourne KD, Benner RW, Gray T. Results of anterior cruciate ligament reconstruction with patellar tendon autografts: objective factors associated with the development of osteoarthritis at 20 to 33 years after surgery. *Am J Sports Med.* 2017;45(12):2730–8.
12. Grindem H, Snyder-Mackler L, Moksnes H, et al. Simple decision rules can reduce reinjury risk by 84% after ACL reconstruction: the Delaware-Oslo ACL cohort study. *Br J Sports Med.* 2016;50(13):804–8.
13. Øiestad B, Juhl C, Eitzen I, et al. Knee extensor muscle weakness is a risk factor for development of knee osteoarthritis. A systematic review and meta-analysis. *Osteoarthr Cartil.* 2015;23(2):171–7.
14. Di Stasi SL, Logerstedt D, Gardinier ES, et al. Gait patterns differ between ACL-reconstructed athletes who pass return-to-sport criteria and those who fail. *Am J Sports Med.* 2013;41(6):1310–8.
15. Wellsandt E, Gardinier ES, Manal K, et al. Decreased knee joint loading associated with early knee osteoarthritis after anterior cruciate ligament injury. *Am J Sports Med.* 2016;44(1):143–51.
16. Paterno M, Schmitt L, Ford K, et al. Biomechanical measures during landing and postural stability predict second anterior cruciate ligament injury after anterior

- cruciate ligament reconstruction and return to sport. *Am J Sports Med.* 2010;38(10):1968–78.
17. Irrgang JJ, Anderson AF, Boland AL, et al. Responsiveness of the international knee documentation committee subjective knee form. *Am J Sports Med.* 2006;34(10):1567–73.
 18. Roos EM, Roos HP, Lohmander LS, et al. Knee injury and osteoarthritis outcome score (koos)—development of a self-administered outcome measure. *J Orthop Sports Phys Ther.* 1998;28(2):88–96.
 19. Webster KE, Feller JA, Lambros C. Development and preliminary validation of a scale to measure the psychological impact of returning to sport following anterior cruciate ligament reconstruction surgery. *Phys Ther Sport.* 2008;9(1):9–15.
 20. Hart JM, Pietrosimone B, Hertel J, et al. Quadriceps activation following knee injuries: a systematic review. *J Athlet Train.* 2010;45(1):87–97.
 21. Bleakley CM, Glasgow P, MacAuley DC. Price needs updating, should we call the police? *Br J Sports Med.* 2012;46(4):220–1.
 22. Hewett T, Myer G, Ford K, et al. Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. *Am J Sports Med.* 2005;33(4):492–501.
 23. Myer GD, Ford KR, Di Stasi SL, et al. High knee abduction moments are common risk factors for patellofemoral pain (PFP) and anterior cruciate ligament (ACL) injury in girls: is PFP itself a predictor for subsequent ACL injury? *Br J Sports Med.* 2015;49(2):118–22.
 24. Adams D, Logerstedt D, Hunter-Giordano A, et al. Current concepts for anterior cruciate ligament reconstruction: a criterion-based rehabilitation progression. *J Orthop Sports Phys Ther.* 2012;42(7):601–14.
 25. White K, Di Stasi S, Smith A, et al. Anterior cruciate ligament—specialized post-operative return-to-sports (ACL -sports) training: a randomized control trial. *BMC Musculo Disord.* 2013;14(1):108.
 26. Sturgill L, Snyder-Mackler L, Manal T, et al. Interrater reliability of a clinical scale to assess knee joint effusion. *J Orthop Sports Phys Ther.* 2009;39(12):845–9.
 27. Haff GG, Triplett NT. *Essentials of strength training and conditioning.* 4th ed. Windsor: Human Kinetics; 2015.
 28. Blanchard S, Glasgow P. A theoretical model to describe progressions and regressions for exercise rehabilitation. *Phys Ther Sport.* 2014;15(3):131–5.
 29. Taberner M, Allen T, Cohen DD. Progressing rehabilitation after injury: consider the ‘control-chaos continuum’. *Br J Sports Med.* 2019;53(18):bjsports-2018-100157.
 30. Gokeler A, Neuhaus D, Benjaminse A, et al. Principles of motor learning to support neuroplasticity after ACL injury: implications for optimizing performance and reducing risk of second ACL injury. *Sports Med.* 2019;49(6):853–65.
 31. Nagelli CV, Hewett TE. Should return to sport be delayed until 2 years after anterior cruciate ligament reconstruction? Biological and functional considerations. *Sports Med.* 2017;47(2):221–32.
 32. Arundale AJH, Kvist J, Häggglund M, et al. Jumping performance based on duration of rehabilitation in female football players after anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc.* 2018;27(2):556–63.
 33. Logerstedt D, Lynch A, Axe MJ, et al. Pre-operative quadriceps strength predicts ikdc2000 scores 6 months after anterior cruciate ligament reconstruction. *Knee.* 2013;20(3):208–12.
 34. Pierce CM, O’Brien L, Griffin LW, et al. Posterior cruciate ligament tears: functional and postoperative rehabilitation. *Knee Surg Sports Traumatol Arthrosc.* 2013;21(5):1071–84.
 35. Burgi CR, Peters S, Ardern CL, et al. Which criteria are used to clear patients to return to sport after primary ACL reconstruction? A scoping review. *Br J Sports Med.* 2019;53(18):1154–61.
 36. Webster KE, Hewett TE. What is the evidence for and validity of return-to-sport testing after anterior cruciate ligament reconstruction surgery? A systematic review and meta-analysis. *Sports Med.* 2019;49(6):917–29.
 37. Capin J, Snyder-Mackler L, Risberg MA, et al. Keep calm and carry on testing—a substantive reanalysis including risk of bias critique of “what is the evidence for and validity of return-to-sport testing after anterior cruciate ligament reconstruction surgery? A systematic review and meta-analysis”. *Br J Sports Med.* 2019;. Epub ahead of print
 38. Capin J, Snyder-Mackler L, Risberg MA, et al. Keep calm and carry on return to sport testing after an ACL injury: clinician-scientists weigh in on knee injury risk. *Br J Sports Med Blog.* 2019. <https://blogs.bmj.com/bjism/2019/05/09/keep-calm-and-carry-on-return-to-sport-testing-after-an-acl-injury-clinician-scientists-weigh-in-on-knee-injury-risk/>
 39. Shrier I. Strategic assessment of risk and risk tolerance (starrt) framework for return-to-play decision-making. *Br J Sports Med.* 2015;49(20):1311–5.
 40. Kyritsis P, Bahr R, Landreau P, et al. Likelihood of ACL graft rupture: not meeting six clinical discharge criteria before return to sport is associated with a four times greater risk of rupture. *Br J Sports Med.* 2016;50(15):946–51.
 41. Barber OR, Thomas C, Jones PA, et al. Reliability of the 505 change-of-direction test in netball players. *Int J Sports Phys Perf.* 2016;11(3):377–80.
 42. Lockie RG, Callaghan SJ, Jeffriess MD. Can the 505 change-of-direction speed test be used to monitor leg function following ankle sprains in team sport athletes. *J Aust Strength Cond.* 2015;23:10–6.
 43. Nae J, Creaby MW, Nilsson G, et al. Measurement properties of a test battery to assess postural orientation during functional tasks in patients undergoing anterior cruciate ligament injury rehabilitation. *J Orthop Sports Phys Ther.* 2017;47(11):863–73.
 44. Gabbett TJ. The training—injury prevention paradox: should athletes be training smarter and harder? *Br J Sports Med.* 2016;50(5):273–80.

45. Castagna C, Impellizzeri FM, Rampinini E, et al. The yo-yo intermittent recovery test in basketball players. *J Sci Med Sport*. 2008;11(2):202–8.
46. Krstrup P, Mohr M, Amstrup T, et al. The yo-yo intermittent recovery test: physiological response, reliability, and validity. *Med Sci Sports Exerc*. 2003;35(4):697–705.
47. Ramirez-Campillo R, Gentil P, Moran J, et al. Dribble deficit enables measurement of dribbling speed independent of sprinting speed in collegiate, male, basketball players. *J Strength Cond Res* 2019.
48. Bourne MN, Webster KE, Hewett TE. Is fatigue a risk factor for anterior cruciate ligament rupture? *Sports Med*. 2019;49(11):1629–35.
49. van Melick N, van Rijn L, Nijhuis-van der Sanden MWG, et al. fatigue affects quality of movement more in ACL-reconstructed soccer players than in healthy soccer players. *Knee Surg Sports Traumatol Arthrosc*. 2019;27(2):549–55.
50. Ardern CL, Glasgow P, Schneiders A, et al. 2016 consensus statement on return to sport from the first world congress in sports physical therapy, Bern. *Br J Sports Med*. 2016;50:853–64.
51. Fältström A, Kvist J, Gauffin H, et al. Female soccer players with anterior cruciate ligament reconstruction have a higher risk of new knee injuries and quit soccer to a higher degree than knee-healthy controls. *Am J Sports Med*. 2019;47(1):31–40.
52. Wiggins AJ, Grandhi RK, Schneider DK, et al. Risk of secondary injury in younger athletes after anterior cruciate ligament reconstruction: a systematic review and meta-analysis. *Am J Sports Med*. 2016;44(7):1861–76.
53. Silvers-Granelli H, Bizzini M, Arundale A, et al. Does the FIFA11+ injury prevention program reduce incidence of ACL injury in male soccer players? *Clin Orthop Rel Res*. 2017;475(10):2447–55.
54. Silvers-Granelli H, Mandelbaum B, Adeniji O, et al. Efficacy of the FIFA 11+ injury prevention program in the collegiate male soccer player. *Am J Sports Med*. 2015;43(11):2628–37.
55. Waldén M, Atroshi I, Magnusson H, et al. Prevention of acute knee injuries in adolescent female football players: cluster randomised controlled trial. *Br Med J*. 2012;344:e3042.
56. Hewett T, Lindencfeld T, Riccobene J, et al. The effect of neuromuscular training on the incidence of knee injury in female athletes. A prospective study. *Am J Sports Med*. 1999;27(6):699–706.
57. Arundale A, Bizzini M, Giordano A, et al. Exercise-based knee and anterior cruciate ligament injury prevention. *J Orthop Sports Phys Ther*. 2018;48(9):A1–A42.



A Biomechanical Perspective on Rehabilitation of ACL Injuries in Basketball

57

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57.1 Introduction

Anterior cruciate ligament (ACL) injuries are one of the most common serious knee conditions affecting the career of many athletes.

Outcomes after ACL rupture and subsequent reconstructive surgery are suboptimal, with one-third of athletes failing to return to play (RTP) [1] and a high second ACL injury rate, particularly in young active athletes (20–30%) [2, 3]. Furthermore, the clinician should be aware about the long-term consequences. The catabolic knee environment, additional joint damage, and dysfunctional loading are potential factors that could contribute to the very high rate of post-traumatic osteoarthritis after ACL reconstruction (ACLR) [4]. Even if conservative treatment is a possible option for patients participating in jumping, cutting, and pivoting sports like basketball, ACLR is the most common (and recommended) choice, thus this chapter will target athletes after ACLR.

Reducing the high secondary ACL injury risk after ACLR is a key priority within the sports medicine field. Optimization of rehabilitation after ACLR is essential to achieve this. RTP rates and second ACL injury risk can be improved through targeting specific modifiable

factors [5, 6]. Among these modifiable factors, movement dysfunction after ACLR is known to influence ACL injury risk, and this key area is an important topic in reducing both primary and secondary ACLR injury risks [7–12]. Thus, the incorporation of targeted neuromuscular training (NMT) programs, which address both neuromuscular performance factors such as strength and power as well as support the optimization of movement quality prior to RTP, is important and recommended after ACLR to improve player outcomes [13].

Basketball is a complex sport, with very tall and athletic players who are exposed to movements which highly challenge the movement system. A recent systematic review does not demonstrate a preventative effect of NMT on primary ACL injury in basketball [14], which is in contrast to other team sports [15]. It is likely that basketball players require more specialized NMT programs which effectively prepare them for the high demands of the sport. Most rehabilitation programs fail to fully restore movement quality prior to RTS. This is in part likely due to insufficient volume of and lower intensities of movement practice during the rehabilitation phases, as well as a lack of sport specificity in the final stages prior to RTP [16–18].

We believe that an increased focus on movement restoration during the rehabilitation process after ACLR can support better player outcomes upon RTP. Movement is complex and influenced

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by an array of neuromuscular, biomechanical, sensorimotor, and neurocognitive factors [19, 20]. In restoring movement quality, it is recommended to have a systematic approach involving (1) addressing the neuromuscular and biomechanical factors, which affect movement quality and motor learning, (2) including a progressive movement retraining approach to re-learn an array of functional tasks optimizing movement quality as previously described and (3) performing the final aspect of rehabilitation and movement training on-parquet, in realistic environments progressively simulating the sporting movement demands and environmental interactions [16, 17, 21]. This complete approach should address the biomechanical factors affecting movement quality, as well as ensure sufficient motor learning employing motor learning principles. The goal is to support RTP, at lower risk of reinjury.

This chapter will share a biomechanical perspective on ACL injuries applied to basketball. It will focus on late stages of rehabilitation after ACLR, when biomechanical/movement quality factors can be more important. We will discuss the balance between RTP and second ACL injury risk, followed by the biomechanics of ACL injuries. Then we discuss stage 2 and 3 of our movement retraining approach (late-stage rehabilitation program) considering the movement pattern assessment and restoration (stage 2, non-sport-specific) and on-parquet movement program (stage 3).

57.2 Return to Play and Second ACL Injury: A Delicate Balance

When dealing with serious knee injuries, such as ACL tears, honest evidence-based counseling to the athlete is a very important topic and should not be overlooked. Regarding the outcomes following ACLR, two primary outcomes should be pursued, and the athlete should be aware about the critical relationship that exists between them.

On one side there is a complete and successful RTP, which is not easy to achieve, and many athletes, even at the professional level, fail to restore

their complete athletic profile [22]. This outcome is more correlated to performance and should be considered a main objective of the athlete. When dealing with high-level athletes, RTP is generally the primary goal.

Prevention of secondary ACL injuries after ACLR is the other primary focus of any rehabilitation, given the high rate of reinjuries reported in young athletes upon RTP (21–35% ACL reinjury rates) and the devastating outcomes of a second ACL injury. Biomechanical and neuromuscular factors are thought to be important in determining player reinjury risk.

When designing a customized treatment program for a basketball player after ACLR, the sports medicine team should integrate elements of primary ACL injury prevention within the RTP strategies. The effectiveness of this approach in reducing second ACL injuries has been demonstrated in a small-scale study [13] but will also depend on if the athlete returns to play or not as exposure is a necessary condition for traumatic reinjury.

Fact Box 1

The main goal of a targeted NMT program after ACLR is the reduction of the second ACL injury rate through the modification of biomechanical factors.

In the authors' vision, RTP is as important as preventing second ACL injuries and the clinical approach should always be customized to an athlete's goals and perceptions.

57.3 Biomechanics of ACL Injuries in the Basketball Player

In order to know which factors should be targeted in prevention and treatment, a thorough understanding of injury mechanism is warranted.

Krosshaug and colleagues studied 39 cases of ACL injuries in basketball players [23]. In most cases, ACL injuries happened while attacking



Fig. 57.1 Landing mechanism of ACL injury in a male basketball player. (a) Player to player contact put the injured athlete out of balance. (b) At initial contact with the ground, the player shows leg dominance and quadri-

ceps dominance. (c) At estimated injury frame, a *knee valgus loading* (ligament dominance) can be clearly seen. The trunk is also displaced (trunk dominance). (d) The player out of balance falls on the parquet

and with the injured player in ball possession. The most common case scenario was landing after jump (Fig. 57.1) [23]. In the authors' experience another common mechanism of ACL injury in basketball is cutting while in ball possession, often triggered by an indirect contact with an opponent (Fig. 57.2). Most of the

injuries in Krosshaug et al.'s study were non-contact in nature (72%), in many cases a player-to-player interaction (including indirect contact) was described and may have played an important role in the ACL injury (Fig. 57.1) [23]. Biomechanically, ACL injuries in basketball tend to happen with consistent inter-segmental body



Fig. 57.2 Cutting mechanism of ACL injury in a male basketball player. (a) While in ball possession the player is perturbed by opponents. (b) At initial contact with the ground, the player shows leg dominance and trunk domi-

nance. (c) At estimated injury frame, an intra-rotated foot and *knee valgus loading* (ligament dominance) can be clearly seen. The trunk is also displaced (trunk dominance). (d) Loss of balance after ACL injury

positioning (Table 57.1). At knee level, shallow knee flexion angles (commonly referred as quadriceps dominance) and knee valgus appearance (ligament dominance) are very common features of such injuries and may also differ depending on gender, with women showing a five times greater risk to display a knee valgus collapse mechanism [23].

Hewett and colleagues described the common movement asymmetries linked to primary ACL injury (listed in Table 57.1), and for each

dysfunctional pattern, described the related neuromuscular training goals thought to address ACL injury risk [7]. These same patterns and treatment approaches should be applied to reduce the risk of secondary ACL injury. In developing our movement retraining approach after ACLR, we considered the concepts from Hewett et al. [7] and integrated a movement retraining approach to update our existing ACL functional recovery framework.

Table 57.1 Underlying NM imbalances, observed movement patterns, and related NMT intervention to be applied afterwards

Neuromuscular imbalances and specific intervention for primary ACL injury prevention based on a movement quality assessment		
Neuromuscular imbalance	Observed movement during ACL injury	Primary prevention intervention
Excessive knee adduction on the frontal plane (ligament dominance)	Valgus appearance on the frontal plane	Train for proper technique Functional strengthening of hip abductors and ER Frontal plane feedback technique
Low knee flexion angle at landing with stiff landing (quadriceps dominance)	Early knee flexion during load acceptance	Plyometric training (easy to advance) aiming to increase knee flexion at landing Lateral plane feedback technique Promote soft landing
Asymmetrical landing with favored lower limb (leg dominance)	All the body weight is on one limb	Train side to side symmetry using also quantitative feedback techniques (e.g., vGFR graph)
Trunk and pelvic imbalances (tilts) with inability to control the center of mass (trunk dominance)	Ipsilateral trunk tilt	Advanced core stability and functional core-strengthening program

Modified from Hewett et al. [7]

Fact Box 2

The comprehension of the sport-specific ACL injury causation is the first step toward a better treatment strategy after ACLR.

57.4 Adopting a Biomechanical and Movement Restoration Approach After ACLR

Considering that biomechanical impairments often present at the time of primary ACL injuries, it is evident that neuromuscular function should be properly optimized after ACLR. The approach to the basketball player after ACLR must consider some key aspects of recovery following sports injuries. The protocol should be *personalized* to the athlete’s needs, *progressive* in using optimal loading strategies, and *supervised* by a rehabilitation team, but moreover *focused on the neuromuscular function*. A careful integration of rehabilitation and conditioning concepts should be considered and applied every day following ACL injuries. The program should adopt a systematic approach and progress from an *isolated* neuromuscular training (focusing largely on addressing muscle strength imbalances) to *targeted* neuromuscular training (focused more on functional neuromuscular training and motor re-patterning) to sport-specific retraining. To achieve we adopt an approach focused on targeted NMT, based on an identified movement profile of the athlete, followed by a structured on-parquet rehabilitation program prior to RTS.

It is crucial to understand when an athlete can effectively begin the targeted NMT program. During the targeted NMT program and subsequent on-parquet programs, there is a gradual increase in both task loading (e.g., peak ground reaction forces during each task, rate of loading, as well as volume of tasks performed) and complexity. It is important to have specific criteria in place to ensure that the athlete is prepared to tolerate the higher loading demands of the program. This should include understanding if they have restored sufficient neuromuscular and movement capacity to be able to tolerate and benefit from the potentially dangerous tasks. To enter the targeted NMT program, we recommend the player have achieved the following:

1. A limb symmetry index >80% on an isokinetic test for knee extensor and flexor strength.

2. Good movement quality (good control of the movement with no presence of excessive dynamic knee valgus, altered motor strategy or trunk and pelvis deviations) [16] during a single leg squat task (60° knee flexion).
3. The ability to jog on a treadmill for at least 10 min at 8 km h⁻¹ with good running gait.

Failure to develop the load tolerance (often referred to as the joints envelope of function, or the joint range of function that the joint can tolerate without aggravation [24, 25]) prior to commencing the program may result in exposing the athlete to tasks which they are not prepared for. In our experience, it is a clinical mistake to commence this targeted NMT program (e.g., late-stage rehabilitation program [16]), with a strength deficit greater than 20% [16, 26]. When the player has met the criteria, a qualitative movement evaluation should ideally be performed to assess specific impairments during high load movements, such as landing, jumping, and cutting.

Fact Box 3

Certain criteria should be met before starting a targeted NMT program.

57.4.1 Common Movement Patterns of the Basketball Player Following ACL Injury

It is important to assess and quantify the athletes' movement quality during an array of bilateral and unilateral sport-type tasks as part of the ACL rehabilitation process. Understanding the athletes' movement profile can support the creation of more targeted NMT programs to address individual movement asymmetries. Basketball players like many other athletes after ACLR display various kinds of qualitative movement impairments, of which are also in many cases associated with increased ACL injury risk. The movement impairments the clinician should be aware of refer to risk factors for dysfunctional joint loading, increasing the stress on the ACL and poten-

tially contributing to higher second ACL injury risk.

The impairments to be discussed, have two main causes, the first being muscle strength deficits. Knee spanning muscles (especially the quadriceps) and non-knee spanning muscles (especially the posterolateral hip muscles) often show clinically relevant strength deficits at the time of RTP [16, 27]. Secondly, movement patterns are developed over the course of an athlete's sporting career sometimes as a result of sports specialization and performance. In the authors experience, movement patterns are as important as muscle strength, as in isolation neither can fully explain the complex dynamic control of the lower limb during jumping and cutting tasks.

Assessment of athletes' movement quality during the performance of sporting type tasks (e.g., running, jumping, landing, and cutting) can reveal various deficits important for a basketball player after ACLR:

- Deficit(s) in coronal/transverse plane dynamic knee stability.
- Deficit(s) in core control (pelvis drops or hikes and trunk tilts).
- Deficit(s) in shock absorption (stiff landing or altered strategy).
- Task-specific deficit(s) typical (but not limited to) in basketball players
 - Internally rotated cutting pattern.

57.4.1.1 Deficit(s) in Coronal/ Transverse and Frontal Plane Dynamic Knee Stability

A common (and important) dysfunctional movement pattern, which is often addressed in research and clinical practice, is the appearance of dynamic knee valgus. Greater angles of dynamic knee valgus during jumping is known to be associated with increased tension on the ACL, as well as prospectively with increased secondary ACL injury risk [9, 12]. Dynamic knee valgus is the result of a combination of hip/femoral and knee/tibial kinematics (Fig. 57.3). The complex interactions of dynamic factors have been recently demonstrated to stretch or tension primarily the



Fig. 57.3 Dynamic knee valgus loading at drop vertical jump in a young basketball player following ACL reconstruction. The external appearance of the overlay of the ground reaction force vector is indicative of dynamic knee valgus loading. The patient is also shifting toward the uninjured side. Ligament and leg dominance

ACL (instead of the MCL) [28]. Addressing a dysfunctional movement pattern with excessive dynamic knee valgus requires both the correction of the biomechanical factors (e.g., altered muscle strength balance and arthrokinetic dysfunction) and focused work on reestablishing an optimal motor pattern, through use of motor re-learning principles. Having a correct neurophysiological progression, utilizing exercises with frontal plane biofeedback, is strongly recommended, in conjunction with a corrective functional strengthening program (with strong focus on the hip abductors and external rotators). Typically in our experience (unpublished findings), and this is true for all the deficits, a program of minimum 10–12 sessions should be initiated.

57.4.1.2 Deficit(s) in Core Control (Pelvis Drops or Hip Hikes and Trunk Tilt)

Alterations in lumbo-pelvic-hip stability are associated with increased risk of sustaining a noncontact knee injury, such as an ACL tear [29]. It is accepted that ipsilateral trunk tilt should be avoided during single leg tasks to reduce knee



Fig. 57.4 Ipsilateral trunk tilt during lateral shuffle task. The patient cannot stabilize the trunk and pelvis and show a core dysfunction, while maintaining the dynamic limb stability

abduction moment, a potential factor in ACL injuries [30]. Ipsilateral trunk tilt may be seen in single leg tasks (such as cutting or lateral shuffle) (Fig. 57.4). The goal of treatment is dynamic stabilization of the trunk, with a program of advanced core stability, starting with non-weight-bearing exercises and progressively moving to more functional/sport-specific exercises, always challenging the patient to maintain the trunk stability in the frontal plane.

57.4.1.3 Deficit(s) in Shock Absorption (Stiff Landing or Altered Strategy)

Another concept biomechanically is that stiff and loud landings or decelerations, with a small knee flexion angle, are associated with ACL injury risk [7, 10]. A reduced knee flexion angle is correlated to an increase in peak vertical ground reaction force (pvGRF) (Fig. 57.5). Athletes should be encouraged and taught to dissipate forces through eccentric muscle action, rather than through the knee joint, after ACLR.

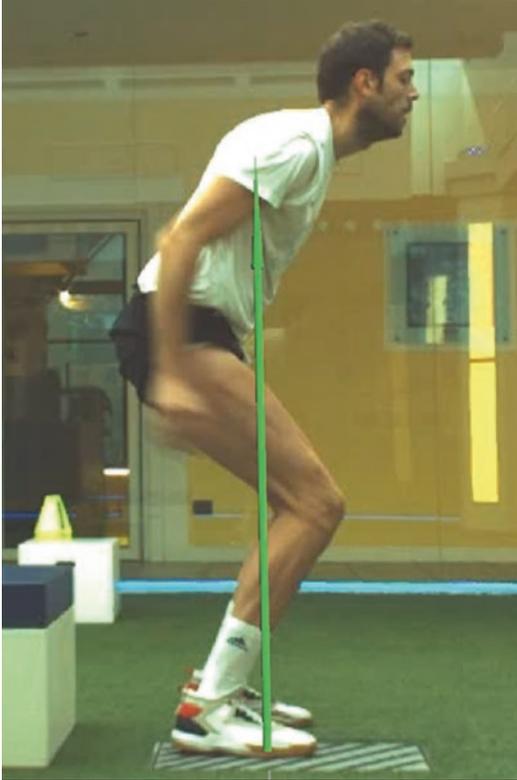


Fig. 57.5 Stiff landing at drop vertical jump in a basketball player following primary ACL reconstruction. The high vertical ground reaction force results from the scarcely flexed pattern (both at knee, hip, and trunk level)

When a stiff landing is noted following ACLR, especially if the deficit is observed in single leg tasks, the clinician should check quadriceps muscle strength. A deficit in quadriceps strength is often correlated to higher pvGRFs, along with increased trunk flexion [31]. If a quadriceps strength deficit is noted, the patient should follow the adequate strengthening program before starting the targeted NMT, as stated above.

If the progression is correct and isolated strength is not a major issue, the athlete should start a plyometric training program (complemented where possible with biofeedback techniques) to increase the knee flexion angles, while obtaining soft landings. Feedback with pvGFR graph may help in the process.

57.4.1.4 Task-Specific Deficit(s) Typical (But Not Limited to) in Basketball Players

There are some task-specific considerations that should be done on the movement evaluation. We will use the example of the side-stepping technique in association to likelihood of high external knee joint loading. The optimal technique adopted during a change of direction task (at various degrees 45°, 60°, 90°) is typically debated, with a specific conflict between movement performance and injury risk [32]. Following ACLR (and also in screening setting), an “internally rotated cutting pattern” may be present (Fig. 57.6). In our experience, this pattern (when evaluated with 2D video) is associated with the following:

1. Internally rotated foot progression angle.
2. Internally rotated hip appearance.



Fig. 57.6 Example of internally rotated cutting pattern, with dynamic knee valgus loading at a 90° sidestepping cut in a young basketball player following ACLR

3. Dynamic valgus appearance of the knee (knee falling medially).
4. Contralateral pelvic drop.
5. Variable trunk position.
6. Generally reduced knee flexion angles.

This internally rotated cutting pattern (Fig. 57.6) is potentially associated with high knee abduction moments (KAM). An internally rotated foot and hip angle are considered predictors of KAM during cutting [33] as well as the knee abduction angle, observed at initial ground contact [34]. As discussed above, an ipsilateral trunk tilt (trunk moving away from intended cut direction) may also increase the KAM and should be avoided [34]. A wide distance between the plant foot and midline pelvis may also increase the external knee load [34, 35]. A laterally planted foot is required to generate medial GRFs and to facilitate the change of direction, but this technique also increases the KAM, highlighting again the potential conflict between movement performance (this movement may aid faster change of direction speeds) and elevated risk of injury (this movement pattern is associated with elevated ACL injury risk).

Considering the evidence, Dempsey et al. recommended a cutting technique to reduce the KAM which is based on (1) a close placement of the stance foot relative to the coronal plane midline of the pelvis, (2) a neutral foot alignment, (3) upright torso posture, and (4) torso facing the direction of travel [35]. Dempsey et al.'s recommendation is in line with our practice that is based on a large volume of clinical activity.

It is important to understand which movement impairments may negatively affect knee loading during sporting tasks and address these as part of the RTP process, adopting a motor learning framework, based on neurophysiological considerations. It is well established that the learning of a new motor sequence follows different stages (cognitive, associative, automatic) [36]. Practically, we recommend the application of feedback techniques (such as instantaneous and delayed visual feedback on the frontal and sagittal plane) to increase athlete awareness of

body position during an array of sporting type tasks to optimize the motor re-learning process.

Once an athlete has achieved good movement quality (defined as the ability to control the limbs and achieve sufficient balance and kinematic alignment during functional activities, not displaying movement asymmetries or risk factors linked to ACL injury) during sporting type tasks (e.g., running, jumping, landing, and cutting), they should progress to sport-specific movement retraining with the intention to prepare for an optimal and safe return to the team environment at lower risk of reinjury.

Fact Box 4

A combination of corrective biomechanics and motor leaning interventions should be used to optimize movement quality following ACLR.

57.5 Integrating Qualitative Movement Patterns on Court: On Parquet Rehabilitation

On-parquet rehabilitation is a key aspect of the RTP continuum. Buckthorpe et al. [18] proposed a model of RTS that provides a detailed transition from rehabilitation to performance. Key progressions of the model include (1) on-parquet rehabilitation, (2) return to training, (3) return to competitive match play, and (4) return to performance. Conceptually, on-parquet rehabilitation constitutes four pillars (1) restoring movement quality, (2) physical conditioning, (3) restoring sport-specific skills, and (4) progressively developing chronic training load, to comprehensively cover the needs of a basketball player in during the progression back to on-parquet activities [17]. The on-parquet rehabilitation program should effectively bridge the gap between the controlled movements performed in the "clinic" and the required sport-specific movements required for RTP.

57.5.1 Key Considerations for On-Parquet Motor Skill Re-training: Bridging the Gap in Movement Performance Between the Clinic and the On-Parquet Performance

The need to restore movement quality after ACLR is nothing new. However, it must be noted that current practices in ACLR rehabilitation typically fail to adequately restore movement quality, given the residual movement impairments identified at the time of RTS in many athletes [11]. Of particular relevance is the lack of sport-specific training prior to RTP seen in many rehabilitation programs, which likely results in an athlete been under-prepared for the demands of the sport. As discussed, the final aspect of a movement-based retraining approach after ACLR should consider sport-specific movement on-parquet. There are many factors, which are different between the typical “in-clinic”-based movement retraining and the demands of sport, which may limit the success of traditional movement quality retraining approaches.

As described, qualitative movement retraining starts before on-parquet rehabilitation, to serve as a foundation for the development of optimal movement quality during moderate and high load movement tasks in a controlled environment. Although this movement training is important and undoubtedly better prepares an athlete for movement retraining on-parquet, the challenge is transferring learned skills within the clinic to realistic basketball scenarios on-parquet. It is essential to effectively prepare a player for the demands of sport, by bridging the gap between sporting type movements undertaken during the targeted NMT and sport-specific movements (e.g., reactive change of direction with the ball to beat a player to the basket). The main differences between the targeted NMT program movements and actual movements during basketball training and match play relate to intensity and specific loading parameters, specificity of movement patterns, and movement volume.

57.5.1.1 Movement Intensity

A key goal of on-parquet rehabilitation is to progressively bridge the gap in movement intensity between the clinic and basketball practice/match play. Basketball involves high-intensity actions, with many accelerations, decelerations, jumps, and change of directions, all done at rapid intensity. Progressively arriving at “maximal intensity” movements is important for injury prevention, neuromuscular conditioning, and optimal motor learning during on-parquet rehabilitation. It is known that knee loading increases as approach velocity increases during change of direction tasks [37]. Furthermore, with increases in movement intensity, there is higher challenge on the neuromuscular system, with increased muscle work as movement intensity increases. Thus it is important to progressively increase the movement/task speeds during the on-parquet program. It is essential to arrive at maximal movement intensity, prior to return to the team.

57.5.1.2 Movement Specificity/Complexity

Throughout the rehabilitation program, there is a progressive increase in tasks’ difficulty in order to train sport-specific patterns gradually challenging movement quality (e.g., maintain low-risk knee positioning) and increasing movement variability. With regard to ACL injuries, we know from previous research that both neurophysiological (distractions) as well as mechanical (indirect body contact) perturbations [38–40] play an important role in injury mechanism. So, there is a need to progressively increase the specificity and complexity of movement. Reactive movements are associated with worse knee biomechanics and higher knee loads than planned movements [41]. As such, we propose a gradual progression from planned to reactive movements [21]. We also recommend a gradual increase in the number of options presented to players during movement and sport-specific tasks to challenge decision-making capacity and the required neurocognition (e.g., “brain training”). Finally, this should include a gradual increase in player contact and number of players,

once the athlete can commence more basketball-specific retraining, to prepare for the contact demands of basketball participation.

57.5.1.3 Movement Volume

It is known in other sports that exposing an athlete to workload demands that they are not prepared for increases in their risk of injury. Blanch and Gabbett [42], as well as Buckthorpe [16], recommend progressively developing an athlete’s chronic training load prior to RTS to reduce a jump in workload demands upon RTS. Gradually building chronic load involves the final periods of on-parquet rehabilitation replicating the workload demands of training allowing for progressive development of chronic training loads to ensure better preparedness for RTS with the team.

57.5.2 Utilizing a Five-Stage on-Parquet Rehabilitation Program to Prepared Players for Return to Team Training

Buckthorpe et al. [17, 21] described on-field rehabilitation for soccer players involving five stages of increasing intensity, complexity, and sport-specificity. These five stages however also have strong relevance for basketball-specific retraining to prepare for RTS and the context of motor pattern retraining. An overview of the program, specific criteria for progressions and aims and content in each stage can be seen in Table 57.2.

57.6 Conclusion

RTP following ACLR in basketball players may be challenging. Detailed attention to the biomechanical factors potentially correlated to the second ACL injury risk is warranted. As such, the focus of the rehabilitation of the basketball player should be predominantly on restoring

Fact Box 5
The functional recovery of a basketball player following ACLR ends with a program of on-parquet rehabilitation.

Table 57.2 Five stages of on-parquet rehabilitation with the overall focus for each stage, the type of activity, and specific examples of content

	The on-parquet rehabilitation program				
	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5
Specific entry criteria	No pain or swelling No subjective instability No positive laxity tests Symmetrical RoM Knee flexor and extensor LSI > 80% Ability to run at 8 km h ⁻¹ for 10 min with sufficiently normalized running mechanics Sufficient movement quality during foundation movements	No pain or swelling Satisfactory progression through Stage 1 on-parquet activity	No pain or swelling Knee flexor and extensor LSI > 90% Optimal movement quality pre-planned sport-type tasks	No pain or swelling Satisfactory progression through Stage 3 on-parquet activity	No pain or swelling Satisfactory progression through Stage 4 on-parquet activity

(continued)

Table 57.2 (continued)

	The on-parquet rehabilitation program				
	Stage 1	Stage 2	Stage 3	Stage 4	Stage 5
Goal of stage	Linear movement training	Multidirectional movement training	Basketball technical and reactive movement training	Basketball-specific movement and skill restoration	Training simulation/re-conditioning
On-parquet movement exercises	Linear running (forward and lateral) Foundation movement tasks (e.g., squatting, lunging, and athletic walks) Deceleration tasks in pre-planned situations of differing velocities Mobility drills	Increased speeds of movements from Stage 1 Multidirectional pre-planned coordination drills (e.g., cutting drills at increasing angles/bended running drills/figure of eight drills/accelerations/decelerations)	Maximum speed pre-planned linear and multidirectional movement drills (change of direction drills, peak running speed exposure, ladder drills) Reactive movement retraining—high speed multidirectional pre-planned speed, acceleration, and deceleration training (closed tasks) and movement practice under external focus with technical based drills	Continued pre-planned and reactive movement training—high speed multidirectional pre-planned and reactive movements; movement in basketball-specific situations; closed basketball-specific fitness drills (e.g., Stage 3 basketball movement drills for conditioning), repeated sprint running; Reactive movement training with perturbations (e.g., ropes, swiss ball, agility circuit with ropes, swiss balls, player contact). Technical drills with pressure, contact to force the player off-balance	Basketball-specific movement training—Basketball-specific plus speed and agility training in pre-planned and reactive tasks with and without fatigue

Modified from Buckthorpe et al. [21]

RoM range of motion, *LSI* limb symmetry index

neuromuscular function. A targeted NMT program should be carried out with a personalization based on a sporting-type movement evaluation, and the functional recovery path should be completed with an on-parquet rehabilitation program.

References

1. Andriolo L, Filardo G, Kon E, Ricci M, Della Villa F, Della Villa S, et al. Revision anterior cruciate ligament reconstruction: clinical outcome and evidence for return to sport. *Knee Surg Sports Traumatol Arthrosc.* 2015;23(10):2825–45.
2. Webster KE, Feller JA. Exploring the high reinjury rate in younger patients undergoing anterior cruciate ligament reconstruction. *Am J Sports Med.* 2016;44(11):2827–32.
3. Wiggins AJ, Granhi RK, Schneider DK, Stanfield D, Webster KE, Myer GD. Risk of secondary injury in younger athletes after anterior cruciate ligament reconstruction: a systematic review and meta-analysis. *Am J Sports Med.* 2016;44(7):1861–76.
4. Cinque ME, Dornan GJ, Chahlija J, Moatshe G, LaPrade RF. High rates of osteoarthritis develop after anterior cruciate ligament surgery: an analysis of 4108 patients. *Am J Sports Med.* 2018;46(8):2011–9.
5. Della Villa F, Andriolo L, Ricci M, Filardo G, Gamberini J, Caminati D. Compliance in post-operative rehabilitation is a key factor for return to

- sport after revision anterior cruciate ligament reconstruction. *Knee Surg Sports Traumatol Arthrosc.* 2019; <https://doi.org/10.1007/s00167-019-05649-2>. [Epub ahead of print]
6. Grindem H, Snyder-Mackler L, Moksnes H, Engebretsen L, Risberg MA. Simple decision rules can reduce reinjury risk by 84% after ACL reconstruction: the Delaware-Oslo ACL cohort study. *Br J Sports Med.* 2016;50:804–8.
 7. Hewett TE, Ford KR, Hoogenboom B, Myer GD. Understanding and preventing acl injuries: current biomechanical and epidemiologic considerations - update 2010. *N Am J Sports Phys Ther.* 2010;5(4):234–51.
 8. Hewett TE, Myer GD, Ford KR, Heidt RS, Colosimo AJ, McLean SG, et al. Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. *Am J Sports Med.* 2005;33(4):492–501.
 9. Krosshaug T, Steffen K, Kristianslund E, Nilstad A, Mok KM, Mykelbust G, et al. The vertical drop jump is a poor screening test for ACL injuries in female elite soccer and handball players: a prospective cohort study of 710 athletes. *Am J Sports Med.* 2016;44(4):874–83.
 10. Leppänen M, Pasanen K, Kujala UM, Vasankari T, Kannus P, Äyrämö S, et al. Stiff landings are associated with increased ACL injury risk in young female basketball and floorball players. *Am J Sports Med.* 2017;45(2):386–93.
 11. Paterno MV, Kiefer AW, Bonnette S, Riley MA, Schmitt LC, Ford KR, et al. Prospectively identified deficits in sagittal plane hip-ankle coordination in female athletes who sustain a second anterior cruciate ligament injury after anterior cruciate ligament reconstruction and return to sport. *Clin Biomech.* 2015;30(10):1094–104.
 12. Paterno MV, Schmitt LC, Ford KR, Rauh MJ, Myer GD, Huang B, et al. Biomechanical measures during landing and postural stability predict second anterior cruciate ligament after anterior cruciate ligament reconstruction and return to sport. *Am J Sports Med.* 2010;38(10):1968–78.
 13. Arundale AJH, Capin JJ, Zarzycki R, Smith AH, Snyder-Mackler L. Two year ACL reinjury rate of 2.5%: outcomes report of the men in a secondary ACL injury prevention program (acl-sports). *Int J Sports Phys Ther.* 2018;13(3):422–31.
 14. Taylor JB, Ford KR, Nguyen AD, Tarry LN, Hegedus EJ. Prevention of lower extremity injuries in basketball: a systematic review and meta-analysis. *Sports Health.* 2015;7(5):392–8.
 15. Silvers-Granelli HJ, Bizzini M, Arundale A, Mandelbaum BR, Snyder-Mackler L. Does the FIFA 11+ Injury Prevention Program reduce the incidence of ACL injury in male soccer players? *Clin Orthop Relat Res.* 2017;475(10):2447–55.
 16. Buckthorpe M. Optimising the late-stage rehabilitation and return-to-sport training and testing process after ACL reconstruction. *Sports Med.* 2019;49(7):1043–58.
 17. Buckthorpe M, Della Villa F, Della Villa S, Roi GS. On-field rehabilitation part 1: 4 pillars of high-quality on-field rehabilitation are restoring movement quality, physical conditioning, restoring sport-specific skills, and progressively developing chronic training load. *J Orthop Sports Phys Ther.* 2019;49(8):565–9.
 18. Buckthorpe M, Frizziero A, Roi GS. Update on functional recovery process for the injured athlete: return to sport continuum redefined. *Br J Sports Med.* 2019;53(5):265–7.
 19. Grooms DR, Page SJ, Nichols-Larsen DS, Chaudhari AM, White SE, Onate JA. Neuroplasticity associated with anterior cruciate ligament reconstruction. *J Orthop Sports Phys Ther.* 2017;47(3):180–9.
 20. Swanik CB. Brains and sprains: the brain's role in noncontact anterior cruciate ligament injuries. *J Athl Train.* 2015;50(10):1100–2.
 21. Buckthorpe M, Della Villa F, Della Villa S, Roi GS. On-field rehabilitation Part 2: a 5-stage program for the soccer player focused on linear movements, multidirectional movements, soccer-specific skills, soccer-specific movements, and modified practice. *J Orthop Sports Phys Ther.* 2019;49(8):570–5.
 22. Almeida AM, Santos Silva PR, Pedrinelli A, Hernandez AJ. Aerobic fitness in professional soccer players after anterior cruciate ligament reconstruction. *PLoS One.* 2018;13(3):e0194432.
 23. Krosshaug T, Nakamae A, Boden BP, Engebretsen L, Smith G, Slauterbeck JR, et al. Mechanisms of anterior cruciate ligament injury in basketball: video analysis of 39 cases. *Am J Sports Med.* 2007;35:359–67.
 24. Dye SF. The pathophysiology of patellofemoral pain: a tissue homeostasis perspective. *Clin Orthop Relat Res.* 2005;436:100–10.
 25. Dye S, Staubli H, Biedert R, Vaupel G. The mosaic of pathophysiology causing patellofemoral pain: therapeutic implications. *Oper Tech Sports Med.* 1999;7:46–54.
 26. Della Villa S, Boldrini L, Ricci M, et al. Clinical outcomes and return-to-sports participation of 50 soccer players after anterior cruciate ligament reconstruction through a sport-specific rehabilitation protocol. *Sports Health.* 2012;4(1):17–24.
 27. Petersen W, Taheri P, Forkel P, Zantop T. Return to play following ACL reconstruction: a systematic review about strength deficits. *Arch Orthop Trauma Surg.* 2014;134:1417–28.
 28. Bates NA, Schilaty ND, Nagelli CV, Krych AJ, Hewett TE. Multiplanar loading of the knee and its influence on anterior cruciate ligament and medial collateral ligament strain during simulated

- landings and noncontact tears. *Am J Sports Med.* 2019;47(8):1844–53.
29. Hewett TE, Myer GD. The mechanistic connection between the trunk, hip, knee, and anterior cruciate ligament injury. *Exerc Sport Sci Rev.* 2011;39(4):161–6.
 30. Myer GD, Ford KR, Di Stasi SL, Foss KD, Micheli LJ, Hewett TE. High knee abduction moments are common risk factors for patellofemoral pain (PFP) and anterior cruciate ligament (ACL) injury in girls: is PFP itself a predictor for subsequent ACL injury? *Br J Sports Med.* 2015;49(2):118–22.
 31. Powers CM. The influence of abnormal hip mechanics on knee injury: a biomechanical perspective. *J Orthop Sports Phys Ther.* 2010;40(2):42–51.
 32. Fox AS. Change-of-direction biomechanics: is what's best for anterior cruciate ligament injury prevention also best for performance? *Sports Med.* 2018;48(8):1799–807.
 33. Sigward SM, Powers CM. Loading characteristics of females exhibiting excessive valgus moments during cutting. *Clin Biomech.* 2007;22(7):827–33.
 34. Jones PA, Herrington LC, Graham-Smith P. Technique determinants of knee joint loads during cutting in female soccer players. *Hum Mov Sci.* 2015;42:203–11.
 35. Dempsey AR, Lloyd DG, Elliot BC, Steele JR, Munro BJ. Changing sidestep cutting technique reduces knee valgus loading. *Am J Sports Med.* 2009;37(11):2194–200.
 36. Doyon J, Benali H. Reorganization and plasticity in the adult brain during learning of motor skills. *Curr Opin Neurobiol.* 2005;15(2):161–7.
 37. Vanrenterghem J, Venables E, Pataky T, Robinson MA. The effect of running speed on knee mechanical loading in females during side cutting. *J Biomech.* 2012;45(14):2444–9.
 38. Brophy RH, Stepan JG, Silvers HJ, et al. Defending puts the anterior cruciate ligament at risk during soccer: a gender-based analysis. *Sports Health.* 2015;7(3):244–9.
 39. Grassi A, Smiley SP, Roberti di Sarsina T, et al. Mechanisms and situations of anterior cruciate ligament injuries in professional male soccer players: a YouTube-based video analysis. *Eur J Orthop Surg Traumatol.* 2017;27(7):967–81.
 40. Waldén M, Krosshaug T, Bjørneboe J, Andersen TE, Faul O, Hägglund M. Three distinct mechanisms predominate in non-contact anterior cruciate ligament injuries in male professional football players: a systematic video analysis of 39 cases. *Br J Sports Med.* 2015;49(22):1452–60.
 41. Besier TF, Lloyd DG, Ackland TR, et al. Anticipatory effects on knee joint loading during running and cutting maneuvers. *Med Sci Sports Exerc.* 2001;33(7):1176–81.
 42. Blanch P, Gabbett TJ. Has the athlete trained enough to return to play safely? The acute:chronic workload ratio permits clinicians to quantify a player's risk of subsequent injury. *Br J Sports Med.* 2016;50:471–5.

Rehabilitation of Foot and Ankle Injuries in Basketball Players

58

Jennifer A. Zellers and Karin Grävare Silbernagel

58.1 Epidemiology of Foot and Ankle Injury in Basketball

The foot and ankle is the most commonly injured body region in high school and collegiate basketball players [1–4] (Fact Box 1), comprising 20–40% of all reported injuries [3, 4]. Further, the foot and ankle is the most commonly injured body region during game play in the National Basketball Association (NBA) and Women’s National Basketball Association (WNBA) [5]. Despite the frequency of injury to the foot and ankle, surgery is more frequent in other body regions [4], making the rehabilitation professional a particularly important part of the health-care team in this body region.

Fact Box 1

The foot and ankle is the most commonly injured body region in high school and collegiate basketball players.

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Lateral ankle sprains are the most common foot and ankle injury sustained by basketball players [5], comprising 14% of injuries in the NBA/WNBA [5]. Foot fractures, tendon rupture, and syndesmotic sprain are much less common than lateral ankle sprains, but present a significant challenge to successfully returning to play. This chapter is an overview of general rehabilitation considerations for the basketball player with more specific information on the treatment and return to play considerations after ligament sprain (lateral and syndesmotic sprains), foot fracture, and Achilles tendon rupture (Fig. 58.1).

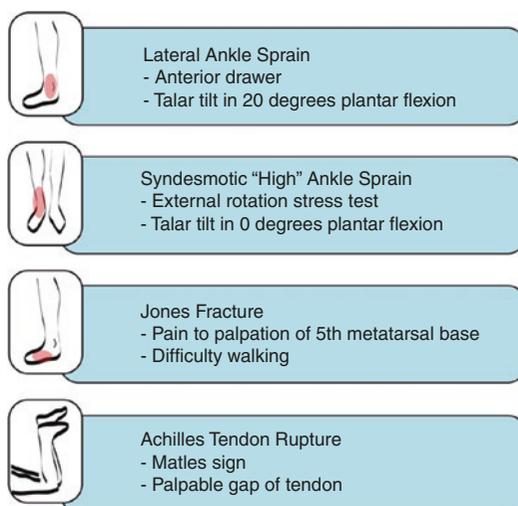


Fig. 58.1 Problematic foot and ankle injuries in basketball players with pain location (red) along with common special tests and clinical signs

58.2 General Rehabilitation Considerations

There are a few general considerations when approaching rehabilitation of the athlete with foot and ankle injury (Fact Box 2). In the early phase post-injury, the immediate concerns are to appropriately protect the tissue, manage inflammation, maintain range of motion, and normalize gait using assistive devices (orthopedic boots, bracing, crutches, etc.) with altered weight-bearing as needed. Even when a player is immobilized, it may be possible to slow deconditioning by strategic conditioning of unaffected structures. For example, in the context of a lateral ankle sprain, it is still possible to strengthen the foot

intrinsic and perform isometric strengthening of the ankle stabilizers with the foot and ankle in a neutral position to avoid placing undue stress on the healing ligament. Throughout all phases of recovery, strengthening should include both the foot intrinsic (Fig. 58.2) as well as the lower leg musculature (Fig. 58.3).

Fact Box 2

Rehabilitation should initially emphasize protection and off-loading of the injured structure, followed by progressively loading the structure and then returning the player to basketball-specific drills and activity.



Fig. 58.2 Example of foot intrinsic muscle strengthening progression. (a) Relaxed foot, (b) foot with raised arch during “short foot” or “doming” exercise. Black dots mark the first metatarsal head, navicular, and medial malleolus. Note the raised position of the navicular in (b).

(c–e) Progression of doming exercise from bilateral standing (c) to unilateral standing with internal perturbation with opposite foot on ball (d) to unilateral standing with large internal perturbation of the trunk as the patient touches the ball to the ground and returns to standing (e)

Fig. 58.3 Example of ankle eversion (peroneal) strengthening progression. (a) Non-weight-bearing ankle eversion with elastic band resistance. (b) Foot eversion against band resistance during side-stepping. (c) Foot eversion against band resistance during side-stepping with a ball toss task



It is also important to educate the player in a general cardiovascular and strength conditioning program that can be performed while protecting the healing tissue. The intention of this program is to avoid general deconditioning and promote the player's psychosocial well-being so that the player will limit risk of injury and avoid needing to rebuild cardiovascular fitness when moving to

later phases of rehabilitation and return to play. For cardiovascular fitness, stationary biking is considered to place low loads on the foot and ankle. There are many other options, though, including body weight supported or pool-based walking or jogging depending on the type and severity of injury (and healing status of surgical wounds in the case of pool-based programs).

Even when a player is immobilized or non-weight-bearing, strengthening of the hip, knee, and trunk can generally be modified using weight machines to condition unaffected structures while maintaining healing precautions.

The player enters the middle phase of rehabilitation as inflammation subsides. This phase emphasizes progressive loading, restoration of muscle control, and optimization of movement patterns and limb symmetry. Specific recommendations depend on the structures affected. For example, in a player with a lateral ankle sprain,

strengthening may progress into inversion ranges. Closed chain, balance and proprioception activities should also be included to assist the player in integrating the neuromotor control needed to return to sport participation. Players can progress from bilateral to unilateral stances, from stable to unstable surfaces, from uniplanar (e.g. forward/backward running) to multiplanar (e.g. shuffling on diagonals with direction changes) agilities, and from low-velocity to high-velocity activity.

In the late phase, return to sport is gradually progressed (Fig. 58.4). It is important to attend to



Fig. 58.4 Example agility and lateral jumping progression. (a) Progression of lateral agilities from uniplanar to multiplanar emphasizing diagonal patterns with dribble.

(b) Progression of lateral jumping from bilateral to unilateral to bilateral with unstable surface landing to unstable surface landing with a ball toss

altered movement patterns that may predispose the player to other injuries or reinjury if the foot and ankle condition is not fully resolved (Fact Box 3).

Specific, standardized return to play criteria are lacking for the majority of foot and ankle conditions common in basketball players. Therefore, clinicians are often left to apply criteria developed for other orthopedic conditions to the foot and ankle when clinically reasonable. Achieving 85–90% limb symmetry during strength and jumping tasks is a common benchmark for return to play, and fluctuations in pain and swelling can provide important guidance for progression of sports-related activity. Consideration for a player’s self-reported function (see Table 58.1 for validated measures) and readiness to return to play are also important considerations in return to play decision-making [6].

Fact Box 3
 Rates of reinjury are high in basketball players—rehabilitative professionals should pay close attention to addressing biomechanical factors and movement patterns that predispose a player to reinjury.

Table 58.1 Selected sport-related foot and ankle outcome measures

Outcome Measure	Description
<i>Performance measures</i>	
Player efficiency rating (PER)	Rating in professional basketball that accounts for per-minute player statistics (free throws, 3-pointers, assists, etc.)
Wearable technology	Metrics including total running distance and running intensity [37]
<i>Self-reported outcomes</i>	
Foot and Ankle Outcome Score [38]	Patient self-reported function questionnaire, includes sports function subscale
Foot and Ankle Ability Measure [39]	Patient self-reported function questionnaire, includes sports function subscale
Patient Specific Functional Scale [40]	Patient self-reported function questionnaire, player can list and rate sport-specific activities

58.3 Problematic Injuries and Specific Rehabilitation Considerations

58.3.1 Lateral Ligament Sprain

Lateral ligament sprains are a common athletic injury affecting around 40–70% of athletes [2, 3]. It is the most common lower extremity injury in basketball players [7]. Ankle sprains are typically a minor injury; however, 20% of people with history of a lateral ankle sprain will go onto develop chronic ankle instability [3, 4, 8]. Due to the rate of players with residual symptoms and recurrent ankle sprains [7, 9], it is important to adequately treat lateral ankle sprains in an effort to decrease the risk of a player developing chronic ankle instability [10, 11].

Ankle sprains occur more frequently during game play than training, in offensive than defensive positions, and in women than men [7]. Most commonly, the player lands on another players foot, resulting in an inversion sprain [7]. Bracing and proprioception training [12, 13] have both been reported to decrease the risk of sustaining an inversion sprain. Both bracing and taping have been suggested for use as prophylaxis in players with a history of ankle sprain, with bracing being a more economic choice [14]. Bracing options have been found to decrease ground reaction force with cutting maneuvers more than taping [15], but bracing has also been found to impair performance with jumping and sprinting tasks compared with taping [16].

Acute management of an ankle sprain follows general inflammatory management guidelines (rest, ice, compression, elevation) along with bracing that allows movement while protecting the healing ligament [17]. There are a few additional specific considerations. Maximizing strength, proprioception, dorsiflexion range of motion, cardiovascular endurance, balance, and neuromuscular control—particularly of the peroneals—are critical rehabilitative targets [17, 18]. Injury or dysfunction of other structures are easily missed after lateral ankle sprain. Lisfranc injury should be triaged, with characteristic bruising on the plantar aspect of the midfoot. Additionally, foot fracture, cuboid syndrome, fibular dysfunction, and peroneal subluxation/

tendinopathy [2] should be screened and addressed as applicable during the course of treatment. With consideration for severity of the sprain, strengthening progresses from isometric to isotonic resistive exercise. Biomechanical factors, particularly limited dorsiflexion range of motion, that may predispose the player to reinjury should be addressed. Joint mobilization should be provided as needed to ensure proper joint mobility [19]. As a player progresses through the early stage of rehabilitation, treatment shifts to weight-bearing proprioception exercises. Return to play training initially begins with straight-plane activities at slower speeds to cutting and pivoting activity at faster speeds [20, 21].

A recent systematic review by Tassignon et al. highlights the lack of developed return to play criteria for athletes following lateral ankle sprain [18]. They identify that time-based criteria for return to play based on grade of the sprain traditionally used in the literature, but underscore the importance of considering function/performance-based criteria to inform the return to sport decision [18]. Given that the predominant mechanism of injury in basketball players is a contact injury that is unpredictable and non-modifiable, there is particular concern for reinjury in this sport. Participating in a proprioception training program and bracing may help to decrease the risk of reinjury [22, 23].

58.3.2 Syndesmotc Sprain

Syndesmotc, or “high ankle,” sprains are less common than lateral ankle sprains; however, they are associated with longer recovery time. The typical mechanism of injury is internal rotation of the leg with external rotation and hyperdorsiflexion of the ankle and foot. The player will typically present with pain at the mortis extending proximally, with increased pain at end range of dorsiflexion. Weight-bearing X-ray will show increased diastasis on the injured side, with a diastasis of 6 mm between tibia and fibular 1 cm proximal to plafond indicating syndesmotc injury [24]. It is important to note that non-weight-bearing and stress X-rays are likely to result in false negatives [24].

Acute syndesmotc sprains with diastasis or concomitant fracture can indicate the need for surgical management [24]; however, syndesmotc injury without fracture or diastasis can be managed nonsurgically. Systematic review-level evidence is available to inform surgical decision-making (screw versus dynamic fixation) [25]; however, there is not randomized controlled trial evidence available to guide rehabilitation decision-making.

Rehabilitation follows a similar approach as lateral ankle sprains; however, rehabilitation tends to progress more slowly, avoiding dorsiflexion and eversion in early stages along with more restrictive weight-bearing. The initial phases of surgical and nonsurgical management include modalities for pain relief and edema management, and assistive device use to encourage normalcy of gait. Immobilization may be required for tissue protection in the initial stages of healing along with pain-free range of motion utilizing caution with dorsiflexion and eversion ranges. Depending on the severity of the sprain, immobilization and protected weight-bearing generally lasts 6–8 weeks [24] and is discontinued when the patient is able to walk and navigate stairs with minimal pain or discomfort [26]. The focus of rehabilitation begins with weaning assistive devices, strengthening in open kinetic chain positions, weight-bearing proprioception exercises (such as weight shifting), and stationary cycling for range of motion and cardiovascular benefit. Rehabilitation then progresses to restoration of normal range of motion, strengthening in closed chain positions, and more advanced proprioception exercises (such as single limb standing activities and balancing on unstable surfaces). Functional testing, including jumping and hopping, can assist in guiding clinical decision-making for when it is appropriate to initiate a gradual return to sports [26]. Dribbling drills, shooting balls, running, and cutting can be introduced in the final phases of rehabilitation, with return to play occurring when the patient is able to perform sport activities at speeds similar to game play with minimal discomfort and adequate movement quality [26].

58.3.3 Fractures

Foot fractures requiring surgical fixation are a particular problem in basketball players. Singh et al. reports that 61% of the foot fractures in the professional American athletic leagues (NBA, NFL, MLB, NHL) occurred in the NBA [27]. NBA players were also the only population to sustain refracture requiring a second operation [27]. Foot fractures tend to occur in younger players, which may partially account for the relatively high rates of return to play and positive performance observed in athletes post foot fracture compared to players recovering from other types of orthopedic injury [28]. Foot fractures most commonly occurs in the fifth metatarsal (“Jones fractures”) in basketball players [27, 29]. In addition to being a common injury (Fig. 58.5), this fracture tends to be managed with surgery [30–32] due to concerns regarding blood supply and non-union. Return to play rates in the NBA range from 57 to 85% [29, 32] after Jones fracture. However, refracture is of concern in these players with rates between 15 and 30% [27, 32, 33] reported in the NBA.

Fifth metatarsal fractures are considered to have an acute and chronic component. Typically there is an acute event on top of a pre-existing bone stress reaction [34], which may result in radiographic changes that occur well after symptom

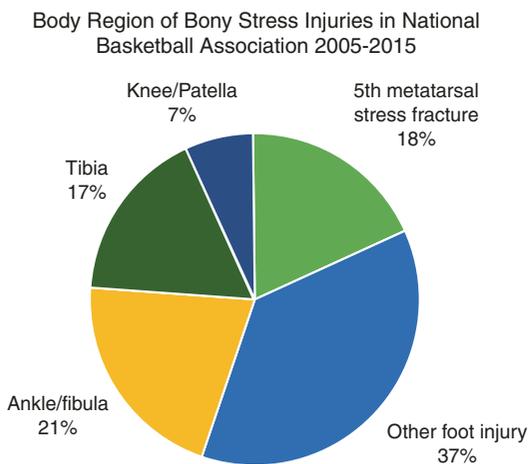


Fig. 58.5 Body region of bony stress injuries (Data from Khan et al. [29])

onset [2]. Particularly considering the accumulation of stress over time, this injury is associated with foot structural characteristics that increase lateral loading of the foot such as varus foot [35] and hindfoot [34] foot type.

Despite the risk of refracture, athletes returning to play after fifth metatarsal fracture have been reported to perform well. A study by Begley et al. [32] reported the majority of players returned to sport with equal amounts of playing time as uninjured controls. There were slight decreases in points per game and assists per 36 min in the individuals returning to sport after Jones fracture. Their findings suggest that pre-injury player efficiency rating was the strongest predictor of post-injury player efficiency rating [32].

Treatment begins with a period of non-weight-bearing through progressively increasing weight-bearing for the first 6 weeks post-injury [33]. It is important that individuals who are managed non-operatively be progressed and observed carefully, given the high rates of failed treatment [36]. Both operative and nonoperative treatment emphasizes preventing or addressing deconditioning from immobilization. The rehabilitation professional should give special attention to identifying biomechanical factors that may be tied with high loads on the lateral aspect of the foot (such as a supinated foot type or use of an orthotic with a medial wedge) as these factors may increase refracture risk. Studies investigating management after Jones fracture have suggested using radiographic evidence of union as a criterion to return to play [33, 36]—with one of these studies being performed in professional basketball players [33].

58.3.4 Achilles Tendon Rupture

Participation in basketball is a common mechanism of injury in Achilles tendon rupture [41, 42]. In a study in the NBA, 39% of players were unable to return to play after Achilles rupture, and players who did return demonstrated decreased performance based on PER [43]. Taken together, this injury is concerning to both recreational and professional basketball players.

While not as common as ankle sprains, Achilles tendon rupture results in significant deficits in plantar flexor structure and function [44, 45] with corresponding deficits in running and jumping performance [46–49] that persist into the long term.

There is likely degeneration of the Achilles tendon prior to rupture [50]; however, it is rarely preceded with pain or discomfort in the tendon. Achilles tendon rupture occurs with forceful eccentric contraction of the plantar flexors such as upon landing a jump or during a cutting maneuver. Anecdotally, this injury is more common in the later portions of the game when the player is fatigued.

Both surgical and nonsurgical management can be used as initial treatment after this injury [51]. There is growing evidence to support using the distance between tendon ends imaged by ultrasound to inform the decision of whether an individual likely requires surgery to limit rerupture risk or risk of poor functional outcomes [52, 53]. With comparable, high-quality rehabilitation, rerupture risk is similar between individuals managed surgically compared to nonsurgically [51]. However, when rehabilitation is not standardized or similar, there is a lower rerupture risk with surgical management [54].

Current literature aimed at optimizing rehabilitation after this injury is investigating timing of rehabilitation onset and what that early intervention should entail as far as dosing of specific interventions. The basic premise of rehabilitation is to expose the tendon to progressively increasing loads to stimulate tendon repair and remodeling [55–57] while protecting the tendon from high loads and avoiding tendon elongation [58–61]. Initially, this is accomplished through progressive weight-bearing with some form of immobilization in plantar flexion. Exercises generally begin with range of motion avoiding dorsiflexion and progress with the addition of plantar flexor strengthening exercises. It is important to remember that players can continue to engage in safe forms of activity throughout the rehabilitation process to avoid substantial deconditioning.

Up to 80% of the general population is able to return to some level of sporting activity after

Achilles tendon rupture [62], though, this rate is much less in elite athletes [43, 63]. On average, return to play occurs around 6 months post-injury [62], but it is important to realize that player performance at this time is substantially affected by the injury. There are a variety of prognostic indicators of long-term function that are being further investigated, including non-modifiable patient characteristics (i.e., age, BMI, sex) [64], distance between tendon ends acutely after rupture [52] and heel-rise height at 1-year post-injury [48].

Standardized return to play criteria are not yet established in this patient population; however, there are common themes across the literature. Heel-rise performance seems to be a key indicator of plantar flexor function and has been used as a return to play criterion [65, 66]. It has been suggested that a running progression can be initiated once the player is able to perform five unilateral heel rises at 90% of the available height on the ruptured side [66]. It is important to note that an individual may never recover heel-rise height comparable to the uninjured side, so maximum available height on the ruptured side should be used to assess this criterion. If the player is unable to meet this criterion by 16 weeks, a running progression can be initiated once the player is able to raise at least 70% of bodyweight in a unilateral heel-rise [66]. Tendon tissue requires at least 36 h [67] to recover after heavy loading activities (such as sprinting and jumping), so basketball players may require additional rest periods between practices or games, consisting of light to moderate activity as they progress back to sport.

Take Home Message

Foot and ankle injury is extremely common in basketball players; however, there are limited diagnosis-specific rehabilitation guidelines to support clinical decision-making. Initial management of these conditions typically involves off-loading the damaged structure for protection and then progressively exposing the structure to increasing loads. There are a lack of strict return to play guidelines, so a combination

of radiographic/imaging indicators of healing, symmetry of functional capacity, pain and swelling with athletic activity, and player perception of injury and ability to return to play should be considered when returning an athlete to play. Basketball tends to be associated with higher rates of reinjury relative to other types of sporting activity, particularly with sprains and fractures, so it is likely beneficial to take time to consider and address biomechanical factors that may predispose a player to reinjury.

References

- Iwamoto J, Ito E, Azuma K, Matsumoto H. Sex-specific differences in injury types among basketball players. *Open Access J Sports Med.* 2015;6:1–6.
- Newman JS, Newberg AH. Basketball Injuries. *Radiol Clin N Am.* 2010;48:1095–111.
- Dane Ş, Can S, Gürsoy R, Ezirmik N. Sport injuries: relations to sex, sport, injured body region. *Percept Mot Skills.* 2004;98:519–24.
- Borowski LA, Yard EE, Fields SK, Comstock RD. The epidemiology of US high school basketball injuries, 2005–2007. *Am J Sports Med.* 2008;36:2328–35.
- Deitch JR, Starkey C, Walters SL, Moseley JB. Injury risk in professional basketball players: a comparison of Women's National Basketball Association and National Basketball Association athletes. *Am J Sports Med.* 2006;34:1077–83.
- Ardern CL, Glasgow P, Schneiders A, et al. 2016 consensus statement on return to sport from the first world congress in sports physical therapy, Bern. *Br J Sports Med.* 2016;50:853–64.
- Cumps E, Verhagen E, Meeusen R. Prospective epidemiological study of basketball injuries during one competitive season: ankle sprains and overuse knee injuries. *J Sports Sci Med.* 2007;6:204–11.
- Colville MR. Surgical treatment of the unstable ankle. *J Am Acad Orthop Surg.* 1998;6:368–77.
- Yeung MS, Chan KM, So CH, Yuan WY. An epidemiological survey on ankle sprain. *Br J Sports Med.* 1994;28:112–6.
- Li X, Killie H, Guerrero P, Busconi BD. Anatomical reconstruction for chronic lateral ankle instability in the high-demand athlete: functional outcomes after the modified Broström repair using suture anchors. *Am J Sports Med.* 2009;37:488–94.
- Gribble PA, Bleakley CM, Caulfield BM, et al. Evidence review for the 2016 International Ankle Consortium consensus statement on the prevalence, impact and long-term consequences of lateral ankle sprains. *Br J Sports Med.* 2016;50:1496–505.
- Cumps E, Verhagen E, Meeusen R. Efficacy of a sports specific balance training programme on the incidence of ankle sprains in basketball. *J Sports Sci Med.* 2007;6:212–9.
- Verhagen EALM, Van Mechelen W, De Vente W. The effect of preventive measures on the incidence of ankle sprains. *Clin J Sport Med.* 2000;10:291–6.
- Olmsted LC, Vela LI, Denegar CR, Hertel J. Prophylactic ankle taping and bracing: a numbers-needed-to-treat and cost-benefit analysis. *J Athl Train.* 2004;39:95–100.
- de Camargo Neves Sacco I, Takahashi HY, Suda EY, Battistella LR, Kavamoto CA, Fernandes Lopes JA, de Vasconcelos JCP. Ground reaction force in basketball cutting maneuvers with and without ankle bracing and taping. *Sao Paulo Med J.* 2006;124:245–52.
- MacKean LC, Bell G, Burnham RS. Prophylactic ankle bracing vs. taping: effects on functional performance in female basketball players. *J Orthop Sports Phys Ther.* 1995;22:77–81.
- Vuurberg G, Hoorntje A, Wink LM, et al. Diagnosis, treatment and prevention of ankle sprains: update of an evidence-based clinical guideline. *Br J Sports Med.* 2018;52:956.
- Tassignon B, Verschueren J, Delahunt E, Smith M, Vicenzino B, Verhagen E, Meeusen R. Criteria-based return to sport decision-making following lateral ankle sprain injury: a systematic review and narrative synthesis. *Sports Med.* 2019;49:601–19.
- Cleland JA, Mintken P, McDevitt A, Bieniek M, Carpenter K, Kulp K, Whitman JM. Manual physical therapy and exercise versus supervised home exercise in the management of patients with inversion ankle sprain: a multicenter randomized clinical trial. *J Orthop Sports Phys Ther.* 2013;43:443–55.
- Hudson Z. Rehabilitation and return to play after foot and ankle injuries in athletes. *Sports Med Arthrosc.* 2009;17:203–7.
- Pearce CJ, Tourné Y, Zellers J, Terrier R, Toschi P, Silbernagel KG. Rehabilitation after anatomical ankle ligament repair or reconstruction. *Knee Surg Sports Traumatol Arthrosc.* 2016;24:1130–9.
- Verhagen E, Van Der Beek A, Twisk J, Bouter L, Bahr R, Van Mechelen W. The effect of a proprioceptive balance board training program for the prevention of ankle sprains: a prospective controlled trial. *Am J Sports Med.* 2004;32:1385–93.
- Janssen KW, Hendriks MRC, Van Mechelen W, Verhagen E. The cost-effectiveness of measures to prevent recurrent ankle sprains: results of a 3-arm randomized controlled trial. *Am J Sports Med.* 2014;42:1534–41.
- Del Buono A, Florio A, Bocconera MS, Maffulli N. Syndesmosis injuries of the ankle. *Curr Rev Musculoskelet Med.* 2013;6:313–9.
- Zhang P, Liang Y, He J, Fang Y, Chen P, Wang J. A systematic review of suture-button versus syndesmotic screw in the treatment of distal tibiofibular syndesmosis injury. *BMC Musculoskelet Disord.* 2017;18:286.

26. Williams GN, Allen EJ. Rehabilitation of syndesmotic (high) ankle sprains. *Sports Health*. 2010;2:460–70.
27. Singh SK, Larkin KE, Kadakia AR, Hsu WK. Risk factors for reoperation and performance-based outcomes after operative fixation of foot fractures in the professional athlete: a cross-sport analysis. *Sports Health*. 2018;10:70–4.
28. Minhas SV, Kester BS, Larkin KE, Hsu WK. The effect of an orthopaedic surgical procedure in the National Basketball Association. *Am J Sports Med*. 2015;44:1056–61.
29. Khan M, Madden K, Burrus MT, Rogowski JP, Stotts J, Samani MJ, Sikka R, Bedi A. Epidemiology and impact on performance of lower extremity stress injuries in professional basketball players. *Sports Health*. 2018;10:169–74.
30. Fernández Fairen M, Guillen J, Busto JM, Roura J. Fractures of the fifth metatarsal in basketball players. *Knee Surg Sports Traumatol Arthrosc*. 1999;7:373–7.
31. Granata JD, Berlet GC, Philbin TM, Jones G, Kaeding CC, Peterson KS. Failed surgical management of acute proximal fifth metatarsal (Jones) fractures: a retrospective case series and literature review. *Foot Ankle Spec*. 2015;8:454–9.
32. Begly JP, Guss M, Ramme AJ, Karia R, Meislin RJ. Return to play and performance after Jones fracture in National Basketball Association athletes. *Sports Health*. 2016;8:342–6.
33. O'Malley M, Desandis B, Allen A, Levitsky M, O'Malley Q, Williams R. Operative treatment of fifth metatarsal Jones fractures (zones II and III) in the NBA. *Foot Ankle Int*. 2015;37:488–500.
34. Raikin SM, Slenker N, Ratigan B. The association of a varus hindfoot and fracture of the fifth metatarsal metaphyseal-diaphyseal junction: the Jones fracture. *Am J Sports Med*. 2008;36:1367–72.
35. Carreira DS, Sandilands SM. Radiographic factors and effect of fifth metatarsal Jones and diaphyseal stress fractures on participation in the NFL. *Foot Ankle Int*. 2013;34:518–22.
36. Mologne TS, Lundeen JM, Clapper MF, O'Brien TJ. Early screw fixation versus casting in the treatment of acute Jones fractures. *Am J Sports Med*. 2005;33:970–5.
37. Fanchini M, Impellizzeri FM, Silbernagel KG, Combi F, Benazzo F, Bizzini M. Return to competition after an Achilles tendon rupture using both on and off the field load monitoring as guidance: a case report of a top-level soccer player. *Phys Ther Sport* 2017.
38. Roos EM, Brandsson S, Karlsson J. Validation of the foot and ankle outcome score for ankle ligament reconstruction. *Foot Ankle Int*. 2001;22:788–94.
39. Martin RL, Irrgang JJ, Burdett RG, Conti SF, Van Swearingen JM. Evidence of validity for the Foot and Ankle Ability Measure (FAAM). *Foot Ankle Int*. 2005;26:968–83.
40. Kowalchuck Horn K, Jennings S, Richardson G, Van Vliet D, Hefford C, Haxby Abbott J. The patient-specific functional scale: psychometrics, clinimetrics, and application as a clinical outcome measure. *J Orthop Sports Phys Ther*. 2011; <https://doi.org/10.2519/jospt.2012.3727>.
41. Hsu AR, Jones CP, Cohen BE, Davis WH, Ellington JK, Anderson RB. Clinical outcomes and complications of percutaneous Achilles repair system versus open technique for acute Achilles tendon ruptures. *Foot Ankle Int*. 2015;36:1279–86.
42. Raikin SM, Garras DN, Krapchev PV. Achilles tendon injuries in a United States population. *Foot Ankle Int*. 2013;34:475–80.
43. Amin NH, Old AB, Tabb LP, Garg R, Toossi N, Cerynik DL. Performance outcomes after repair of complete Achilles tendon ruptures in national basketball association players. *Am J Sports Med*. 2013;41:1864–8.
44. Heikkinen J, Lantto L, Flinkkila T, Ohtonen P, Pajala A, Siira P, Leppilähti J. Augmented compared with nonaugmented surgical repair after total Achilles rupture: results of a prospective randomized trial with thirteen or more years of follow-up. *J Bone Jt Surg*. 2016;98:85–92.
45. Lantto I, Heikkinen J, Flinkkila T, Ohtonen P, Kangas J, Siira P, Leppilähti J. Early functional treatment versus cast immobilization in tension after Achilles rupture repair: results of a prospective randomized trial with 10 or more years of follow-up. *Am J Sports Med*. 2015;43:2302–9.
46. Powell HC, Silbernagel KG, Brorsson A, Tranberg R, Willy RW. Individuals post-Achilles tendon rupture exhibit asymmetrical knee and ankle kinetics and loading rates during a drop countermovement jump. *J Orthop Sports Phys Ther*. 2018;48:34–43.
47. Willy RW, Brorsson A, Powell HC, Willson JD, Tranberg R, Grävare Silbernagel K. Elevated knee joint kinetics and reduced ankle kinetics are present during jogging and hopping after Achilles tendon ruptures. *Am J Sports Med*. 2017;45:1124–33.
48. Brorsson A, Willy RW, Tranberg R, Grävare Silbernagel K. Heel-rise height deficit 1 year after Achilles tendon rupture relates to changes in ankle biomechanics 6 years after injury. *Am J Sports Med*. 2017;45:3060–8.
49. Zellers JA, Marmon AR, Ebrahimi A, Silbernagel KG. Lower extremity work along with triceps surae structure and activation is altered with jumping after Achilles tendon repair. *J Orthop Res*. 2019; <https://doi.org/10.1002/jor.24260>.
50. Józsa L, Kannus P. Histopathological findings in spontaneous tendon ruptures. *Scand J Med Sci Sports*. 1997;7:113–8.
51. Soroceanu A, Sidhwa F, Aarabi S, Kaufman A, Glazebrook M. Surgical versus nonsurgical treatment of acute Achilles tendon rupture: a meta-analysis of randomized trials. *J Bone Jt Surg*. 2012;94:2136–43.
52. Westin O, Nilsson Helander K, Grävare Silbernagel K, Möller M, Kälebo P, Karlsson J. Acute ultrasonography investigation to predict reruptures and

- outcomes in patients with an Achilles tendon rupture. *Orthop J Sports Med.* 2016;4:1–7.
53. Lawrence JE, Nasr P, Fountain DM, Berman L, Robinson AHN. Functional outcomes of conservatively managed acute ruptures of the Achilles tendon. *Bone Joint J.* 2017;99–B:87–93.
 54. Nilsson-Helander K, Silbernagel KG, Thomeé R, Faxén E, Olsson N, Eriksson BI, Karlsson J. Acute Achilles tendon rupture: a randomized, controlled study comparing surgical and nonsurgical treatments using validated outcome measures. *Am J Sports Med.* 2010;38:2186–93.
 55. Schepull T, Aspenberg P. Early controlled tension improves the material properties of healing human Achilles tendons after ruptures: a randomized trial. *Am J Sports Med.* 2013;41:2550–7.
 56. Schepull T, Kvist J, Aspenberg P. Early E-modulus of healing Achilles tendons correlates with late function: similar results with or without surgery. *Scand J Med Sci Sports.* 2012;22:18–23.
 57. Zellers JA, Cortes DH, Pohlig RT, Silbernagel KG. Tendon morphology and mechanical properties assessed by ultrasound show change early in recovery and potential prognostic ability for 6 month outcomes. *Knee Surg Sports Traumatol Arthrosc.* 2018;27(9):2831–9.
 58. Silbernagel KG, Steele R, Manal K. Deficits in heel-rise height and Achilles tendon elongation occur in patients recovering from an Achilles tendon rupture. *Am J Sports Med.* 2012;40:1564–71.
 59. Kangas J, Pajala A, Ohtonen P, Leppilahti J. Achilles tendon elongation after rupture repair: a randomized comparison of 2 postoperative regimens. *Am J Sports Med.* 2007;35:59–64.
 60. Amendola A. Elongation of the Achilles tendon after rupture repair occurred slightly less with postoperative early motion than with postoperative immobilization. *J Bone Joint Surg Am.* 2007;89:1873.
 61. Brumann M, Baumbach SF, Mutschler W, Polzer H. Accelerated rehabilitation following Achilles tendon repair after acute rupture - development of an evidence-based treatment protocol. *Injury.* 2014;45:1782–90.
 62. Zellers JA, Carmont MR, Grävare Silbernagel K. Return to play post-Achilles tendon rupture: a systematic review and meta-analysis of rate and measures of return to play. *Br J Sports Med.* 2016;50:1325–32.
 63. Parekh SG, Wray WH, Brimmo O, Sennett BJ, Wapner KL. Epidemiology and outcomes of Achilles tendon ruptures in the National Football League. *Foot Ankle Spec.* 2009;2:283–6.
 64. Olsson N, Petzold M, Brorsson A, Karlsson J, Eriksson BI, Grävare Silbernagel K. Predictors of clinical outcome after acute Achilles tendon ruptures. *Am J Sports Med.* 2014;42:1448–55.
 65. Saxena A, Ewen B, Maffulli N. Rehabilitation of the operated Achilles tendon: parameters for predicting return to activity. *J Foot Ankle Surg.* 2011;50:37–40.
 66. Silbernagel KG, Brorsson A, Karlsson J. Rehabilitation following Achilles tendon rupture. In: Karlsson J, Calder J, van Dijk CN, Maffulli N, Thermann H, editors. *Achilles tendon disord. A Compr. Overv. Diagnosis treat.* London: DJO Publications; 2014. p. 151–63.
 67. Magnusson SP, Langberg H, Kjaer M. The pathogenesis of tendinopathy: balancing the response to loading. *Nat Rev Rheumatol.* 2010;6:262–8.



Rehabilitation of Tendinopathy in Basketball

59

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59.1 Introduction

Tendinopathy is one of the most common over-use injuries in basketball players, with patellar and Achilles tendinopathy occurring most frequently [1–3]. In athletic populations, these injuries typically result from excessive load (acute or chronic) placed upon the tendon with inadequate time for recovery [4, 5]. The primary symptoms are pain and stiffness; however, the injury is also accompanied by alterations in tendon structure, tendon mechanical properties [6–8], and muscle-tendon function [9, 10]. Tendinopathy poses a unique challenge for rehabilitation since many athletes can initially play through symptoms. Therefore, they often wait to seek treatment until symptoms become debilitating. A variety of non-invasive treatment options are available for tendinopathy, but controlled tendon loading is supported by the highest level of evidence [11, 12]. In this chapter we will describe key tendinopathy rehabilitation principles. Furthermore,

we will describe how these principles are applied to patellar and Achilles tendinopathy.

59.2 Diagnosis

Tendinopathy is a clinical diagnosis characterized by activity-provoked, localized tendon pain and stiffness. In the initial stages of the injury, athletes will complain of stiffness and/or pain that appears after activity, then progress to pain at the onset of activity that reduces following a warm-up [13]. In the later stages, pain will be present throughout activity and at rest, limiting sports participation (Table 59.1) [13, 14]. Stiffness will be most pronounced in the morning and after prolonged rest. In more superficial tendons, clinicians may be able to observe or palpate focal thickening. Ultrasound imaging may be

Table 59.1 Classification system for the effect of pain on athletic performance [14]

Level	Description of pain	Level of sports performance
1	No pain	Normal
2	Pain only with extreme exertion	Normal
3	Pain with extreme exertion and 1–2 h afterwards	Normal or slightly decreased
4	Pain during and after vigorous activities	Somewhat decreased
5	Pain during activity and forcing termination	Markedly decreased
6	Pain during daily activities	Unable to perform

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Fig. 59.1 Single leg decline squat test for patellar tendinopathy [18]

Instructions: The patient begins in the standing position on a 25° decline surface. Instruct the patient to squat down, using only the involved leg, to approximately 50° of knee flexion while keeping the trunk as upright and erect as possible. After returning to the start position, ask the patient to rate their symptoms on numeric pain rating scale (NPRS) (0 = No pain, 10 = Worst pain imaginable) and to identify their symptom location.

Interpretation: Pain isolated to the patellar tendon suggests patellar tendinopathy. Pain ratings during this test can be used to monitor symptom severity throughout treatment.



Royal London Hospital Test

Instructions: Locate and palpate the most symptomatic region of the tendon. Have the patient actively dorsiflex and plantarflex their foot while continuing to palpate the same location.

Interpretation: The test is positive if the patient's symptoms decrease when dorsiflexed or plantarflexed. Indicative of Achilles tendinopathy.

Painful Arc Sign

Instructions: Palpate the tendon for the presence of focal thickening. If a thickened nodule is present, ask the patient to actively dorsiflex and plantarflex their foot while lightly palpating the nodule.

Interpretation: This test is positive if the nodule moves beneath the fingers with plantarflexion and dorsiflexion. Indicative of Achilles tendinopathy.

Fig. 59.2 Royal London Hospital Test and painful arc sign for Achilles tendinopathy [20]

used to assist in the diagnosis of this condition with findings of hypoechoic regions, tendon thickening, loss of collagen fiber organization, and potentially neovascularization [15, 16].

In patellar tendinopathy, pain is usually localized to the inferior pole of the patella, and the athlete will report that pain is provoked with jumping and squatting activities [17]. In Achilles tendinopathy, pain most frequently occurs at the mid-substance of the tendon and is aggravated by running and jumping [1]. However, it is important to be aware that symptoms may present anywhere along the length of the tendon or its insertion sites for both injuries. The single-leg decline squat test, a pain provocation test, may help confirm the diagnosis of patellar tendinopathy (Fig. 59.1) [18, 19]. Similarly, the Royal London Hospital Test and

painful arc sign can be used to confirm the presence of Achilles tendinopathy (Fig. 59.2) [20–22].

59.3 Outcome Measures

Tendinopathies have a notoriously long recovery time compared to other overuse injuries. Therefore, it is important to utilize reliable outcome measures to track progress and readiness to return to sport. The most commonly utilized outcome measure for tendinopathy is pain, either through self-report, palpation, or other pain provocation tests. However, absence of pain does not ensure full recovery of tendon health, and impairments often persist after symptoms have resolved [23]. So it is beneficial to utilize

outcome measures that address additional aspects of tendon injury. Recently, a consortium of patients and health-care professionals released a consensus statement identifying core outcome domains for tendinopathy [24]. The core outcome domains were patient rating of overall condition, participation, pain on loading/activity, function, psychological factors, physical function capacity, disability, quality of life, and pain over a specified period of time. Furthermore, it is important to monitor tendon structure and

mechanical properties, when possible. A summary of suggested outcome measures for patellar and Achilles tendinopathy are presented in Tables 59.2 and 59.3. In a clinical setting, it may not be feasible to address each of these domains for every athlete, so clinicians must use their clinical judgment to determine the most pertinent measures. However, it is advisable to use several measures from different domains to ensure that the impact of the injury is being accurately measured.

Table 59.2 Summary of selected outcome measures for patellar tendinopathy [76]

Outcome measure	Concept/rationale	Method	Measurement properties
Patient reported outcome measures			
Victorian Institute of Sport Assessment—Patella (VISA-P) Questionnaire [77]	Assesses patient-reported symptoms, function, and ability to be physically active.	Eight-item questionnaire. The score ranges from 0 to 100, with a higher score indicating lesser symptoms and higher level of physical activity.	<ul style="list-style-type: none"> • Significant differences in scores between healthy individuals and patients with patellar tendinopathy [77]. • Test–retest reliability: intraclass correlation coefficient (ICC) = 0.91–0.97 [78, 79]. • Minimal detectable change: 12.2 points [78].
Oslo Sports Trauma Research Centre (OSTRC) Overuse injury Questionnaire [80]	Assesses patient-reported function or sports performance limitation due to injury. May be adapted for any region of the body.	Four-item questionnaire. Scores range from 0 to 100, with a higher score indicating greater function or sports limitation.	Patellar tendinopathy [81] <ul style="list-style-type: none"> • Sensitivity: 79% (95% confidence interval [CI]: 65–90%). • Specificity: 98% (CI: 94–100%). • Positive predictive value: 95% (CI: 83–99%). • Negative predictive value: 92% (CI: 86–96%). • Positive likelihood ratio (+LR): 48 (CI: 12–191). • Negative likelihood ratio (–LR): 0.21 (95%: 0.12–0.37).
Pain			
Five single-legged squat test [82]	The primary symptom in patients with patellar tendinopathy is pain at the inferior pole of the patella, which is aggravated by jumping or squatting. Therefore, pain evaluation with activity is a central part of evaluation. Squatting on a decline surface increases loads through the patellar tendon when compared to level ground [52].	Patients are asked to perform five single-legged squats in a row and then rated pain on the visual analog scale (VAS).	Not established.
Single-leg decline squat test [18]		The patient is positioned in standing on a 25° decline board and instructed to perform a single-leg squat to 50° of knee flexion. Additionally, the patient is instructed to keep their trunk as upright as possible. Pain during or after the squat is rated on the numeric pain rating scale (NPRS).	<ul style="list-style-type: none"> • Test–retest reliability: Kappa = 1 [19]. • Ability to discriminate individuals with patellar tendon abnormality on ultrasound imaging: [19]. <ul style="list-style-type: none"> – +LR: 4.2 (CI: 2.3–7.14) – –LR: 0.4 (0.3–0.6)

(continued)

Table 59.2 (continued)

Outcome measure	Concept/rationale	Method	Measurement properties
Structure			
Thickness	Patients with patellar tendinopathy have increased tendon thickness and cross-sectional area compared to an unaffected region or side.	The patient is positioned in supine with the knee flexed 30° and supported by a bolster. Three B-mode ultrasound images are taken 1 cm distal to the inferior pole of the patella, in the longitudinal (long axis) plane. The average thickness at this location is compared to an unaffected region of the tendon or the same location on the opposite side.	<ul style="list-style-type: none"> • Intra-rater reliability: ICC = 0.82–0.91 [83]. • Inter-rater reliability: ICC = 0.85 [83].
Cross-sectional area (CSA)		The patient is positioned in supine with the knee flexed 30° and supported by a bolster. Three B-mode ultrasound images are taken 1 cm distal to the inferior pole of the patella, in the transverse (short axis) plane. The average CSA at this location is compared to an unaffected region of the tendon or the same location on the opposite side.	<ul style="list-style-type: none"> • Intra-rater reliability: ICC = 0.87–0.96 [84]. • Inter-rater reliability: ICC = 0.90–0.92 [84].

Table 59.3 Summary of selected outcome measures for Achilles tendinopathy [76]

Outcome measure	Concept/rationale	Method	Measurement properties
Patient reported outcome measures			
Victorian Institute of Sport Assessment—Achilles (VISA-A) questionnaire [85]	Assesses patient-reported clinical severity of Achilles tendinopathy in two domains: (1) pain/symptoms and (2) physical activity.	Eight-item questionnaire. Scores range from 0 to 100, with 100 indicating no symptoms and full participation in physical activity.	<ul style="list-style-type: none"> • Test–retest reliability: ICC = 0.93 in a group of mixed Achilles tendon injuries [85]. • Minimally clinically important difference: 16 points [86, 87].

Table 59.3 (continued)

Outcome measure	Concept/rationale	Method	Measurement properties
Impairment/endurance			
Heel-Rise Test [88]	The heel-rise test is commonly used to evaluate calf muscle endurance in patients with Achilles tendinopathy.	The patient is positioned in single-leg stance on a 10° inclined surface with two finger tips from each hand placed on the wall for support. The patient is instructed to raise up onto the ball of their foot, as high as they can, as many times as they can while keeping the knee straight. Forward or lateral body sway is discouraged. A metronome can be used to ensure consistent rhythm with a frequency of 30 repetitions per minute. The test is terminated when the patient is no longer able to continue or can no longer maintain proper form. A number of variations of the test have been proposed, which may be more suitable depending on equipment availability.	<ul style="list-style-type: none"> • Test–retest reliability: ICC = 0.78–0.84 [89, 90].
Functional performance tests			
Hopping [88]	Patients with Achilles tendinopathy have been found to have pain and difficulty with jumping activity.	Standing on a single leg, patients are instructed to perform a continuous rhythmical jumping motion, similar to jumping rope. Pain, contact, and flight times are usually recorded.	<ul style="list-style-type: none"> • Test–retest reliability: ICC = 0.83–0.94 [88].
Drop Counter-Movement Jump [88]		The patient is positioned in single-leg stance on a box with the arms behind the back. They are instructed to drop off the box and perform a maximal vertical jump upon landing. Pain and jump height are recorded.	<ul style="list-style-type: none"> • Test–retest reliability: ICC = 0.88–0.92 [88].
Structure			
Thickness	Patients with Achilles tendinopathy have increased tendon thickness and cross-sectional area compared to an unaffected region or side.	The patient is positioned in prone with their foot hanging off the table in a relaxed position. Three B-mode ultrasound images are taken at the location of maximal thickening, in the longitudinal (long axis) plane. The average thickness at this location is compared to an unaffected region of the tendon or the same location on the opposite side.	<ul style="list-style-type: none"> • Intra-rater reliability: ICC = 0.98–0.99 [83]. • Inter-rater reliability: ICC = 0.98 [83].
Cross-sectional area (CSA)		The patient is positioned in prone with their foot hanging off the table in a relaxed position. Three B-mode ultrasound images are taken at the location of maximal thickening, in the transverse (short axis) plane. The average CSA at this location is compared to an unaffected region of the tendon or the same location on the opposite side.	<ul style="list-style-type: none"> • Intra-rater reliability: ICC = 0.89–0.97 [91]. • Inter-rater reliability: ICC = 0.84 [91].

59.4 Principles of Tendon Load Management and Rehabilitation

59.4.1 Mechanotherapy

In order to effectively utilize controlled tendon loading protocols, it is critical to understand the mechanism of action for this intervention. Loads placed upon the tendon result in strain or a relative lengthening of the tendon. Tendons respond to strain through a process called mechanotransduction, “whereby cells convert physiological mechanical stimuli into biochemical responses” [25]. In the context of tendons, loading triggers a cellular cascade, resulting in collagen synthesis and tendon remodeling [25]. In vitro and in vivo studies suggest that there is a dose–response relationship between the magnitude of load applied and the adaptive response [26–30]. Up to a certain point, greater loads result in greater collagen synthesis and remodeling. However, the optimal amount of load remains unclear and loads that exceed this threshold may be detrimental. The tendon’s response also appears to be governed by the duration of loading, with longer duration contractions resulting in greater improvements in tendon structure compared to shorter duration contractions [26, 31, 32]. In summary, optimal loading parameters have not been established, but tendons appear to respond more favorably to higher loads at longer durations compared to shorter duration and lower loads. However, there is likely an upper limit beyond which the load can be detrimental to recovery.

59.4.2 Rehabilitation

Rehabilitation of tendinopathies generally consists of three phases: (1) symptom management and load reduction, (2) controlled tendon loading, and (3) return to sport. The athlete’s progression through these phases is dependent on their symptom severity, response to treatment, and timing in the competitive season. Although the rehabilitation process is broken into three distinct phases, not all athletes will need to begin in phase 1.

Additionally, principles of prior phases may be continued after they progress to the next phase.

Prior to discussing each phase in detail, it is important to note that controlled tendon loading can take two forms during the rehabilitation process: (1) therapeutic loading and (2) sport-specific loading. Therapeutic loading, achieved through strengthening exercises, is designed to promote tendon remodeling. Sport-specific loading is designed to increase the tissue’s tolerance to repetitive activities, such as running and jumping. Therapeutic loading is initiated as soon as treatment begins and can generally be performed daily. Sport-specific loading is initiated in the later phases of treatment and will require careful prescription of recovery days to prevent exacerbation of symptoms.

59.4.2.1 Phase 1: Symptom Management and Load Reduction

The aim of phase 1 is to reduce symptom severity, gradually initiate therapeutic tendon loading, and curtail abusive training. When an athlete is diagnosed with tendinopathy, the athlete is educated on the treatment plan for treating the condition, the timeline for recovery and the benefits of managing loads related to exercise, practices, and games. Load management is discussed with all relevant parties including the player, coaches, managers, team doctors, and training staff since many considerations come into play in this decision-making process. The player and team’s short- and long-term success may rely on accurate and thorough information provided from the medical staff.

For lower extremity tendinopathies, therapeutic tendon loading is typically initiated using bilateral bodyweight resistance exercises, such as heel raises or squatting. In highly symptomatic patients, isometric exercises, constrained range of motion, or partial bodyweight resistance may be necessary to prevent excessive pain. Furthermore, open-chain active range of motion movements may be beneficial to increase blood flow to the region and manage symptoms.

To curtail abusive training, a thorough review of the athlete’s training and competition schedule is necessary, along with identification of provocative activities (Table 59.4). Sport-specific tendon loads can be reduced by altering the frequency, duration, intensity, or types of activities performed outside of treatment. For example, reducing the number of minutes played, limiting the athlete to form shooting rather than jump shots, or providing additional recovery days between practice or games. A complete cessation of physical activity may not be necessary. In athletes with Achilles tendinopathy, continued activity, utilizing a pain-monitoring model to guide activity intensity, was found to not be detrimental to treatment outcomes [33]. In brief, the pain-monitoring model allows patients to continue their usual activity, as long as symptoms do not exceed a 5/10 on the numeric pain rating scale (NPRS) during or immediately after activity (Fig. 59.3). Additionally, symptoms

should return to pre-activity levels by the following morning, and resting symptoms should not increase from week to week. This approach has been successfully used in the treatment of other tendinopathies [34]. Endurance activities that minimally load the tendon may be utilized to maintain physical conditioning, for example, swimming in cases of patellar or Achilles tendinopathy. However, the type of activity will be dependent on the tendon that is injured.

59.4.2.2 Phase 2: Controlled Tendon Loading

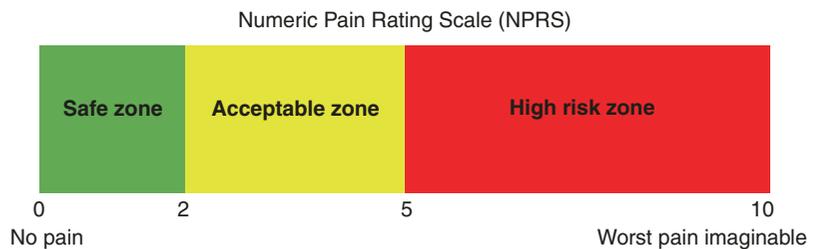
In phase 2, tendon loading activities are advanced by transitioning to unilateral activities and incorporating external resistance. As symptoms improve, exercises are progressed to full range of motion and greater external loads. Often, isotonic loading protocols have emphasized the eccentric component of muscle contraction. However, favorable clinical outcomes have been obtained using isolated eccentric, isolated concentric, and combined protocols for tendinopathy [35]. Furthermore, when the resistance is similar, tendons experience the same magnitude of load and demonstrate similar increases in collagen synthesis and tendon hypertrophy, regardless of contraction type [36, 37].

Therapeutic tendon loading exercises need not to be confined to the clinic or training room. Many of the activities can be completed on the court and incorporated with basketball activities.

Table 59.4 Considerations when reviewing training and competition

Considerations when reviewing training and competition
<ul style="list-style-type: none"> • Frequency and duration of training and competition. • Type and intensity of training (e.g., strength training and jump training). • Recent changes in training and/or competition. • Current phase of the competitive season. • Recovery time between bouts of activity. • Provocative activities. • Pain levels (morning and prior to, during, and after activity).

Fig. 59.3 Pain-monitoring model [33]



1. The pain is allowed to reach 5 on the NPRS during the activity.
2. The pain after completion of the activity is allowed to reach 5 on the NPRS.
3. The pain the morning after the activity should not exceed a 5 on the NPRS.
4. Pain and stiffness is not allowed to increase from week to week.

For example, squats or lunges to load the patellar tendon, or heel raises for the Achilles tendon, can be performed while dribbling or completing passing drills. Since absence from sport due to injury can negatively impact the psychological and emotional health of athletes, use of on-the-court loading activities may be especially beneficial in patients that are unable to participate in team activities [38].

59.4.2.3 Phase 3: Return to Full Sports Participation

Phase 3 focuses on a gradual introduction of plyometric activities, increasing tissue tolerance to sport-specific loading, and a return to full sports participation. During this phase, prior therapeutic tendon loading activities are continued daily, or the athlete is transitioned to heavier loading exercises, performed 3 days a week.

Plyometric activities should mimic the demands placed upon the tendon during basketball participation. Therefore, in lower extremity tendinopathies, jumping, sprinting, and directional changes should be included. The loads placed upon the tendon during plyometric activities can substantially exceed those experienced during isotonic loading exercises. For example, patellar tendon and Achilles tendon forces can reach 6.5- and 8-times bodyweight during running and 6.5- and 5-times bodyweight during jumping, respectively [39–42]. Furthermore, the tendon may require up to 72 h to fully recover after high load activities. Thus, a key component of the return-to-sport phase is ensuring the tendon has adequate rest between heavy bouts of loading.

Unfortunately, there is a lack of specific return-to-sport guidelines for most tendinopathies. However, a return-to-sport algorithm has been developed for the Achilles tendon, which can be adapted to other tendinopathies [43]. In this algorithm, plyometrics are initiated when pain with daily activities does not exceed 2/10 on the NPRS. As with other tendon loading exercises, a graded approach is utilized, beginning with submaximal effort. Training diaries are

Table 59.5 Classification of tendon loading activities and suggested recovery days [43]

	Classification of Activities		
	Light	Medium	High
Pain level during activity, NPRS (0–10)	1–2	2–3	4–5
Pain level after activity (next day), NPRS (0–10)	1–2	3–4	5–6
The athlete’s RPE (with regard to their tendon) (0–10) [46]	0–1	2–4	5–10
Recovery days needed between activities	0 (can be performed daily)	2	3
Examples of activities for patellar tendinopathy	Free throw shooting	Stationary jump shots for 20 min	Half-court scrimmage for 20 min
Examples of activities for Achilles tendinopathy	Straight line dribbling at light effort for 15 min	Dribbling and passing drills with directional changes at moderate effort for 20 min	

typically used to monitor the athlete’s response, and intensity can be progressed by increasing the speed or height of the movements as the tendon adapts to the new demands [44, 45]. Recovery days are prescribed based on the intensity of the activity, which can be categorized by pain during and after activity, and the athlete’s rating of perceived tendon exertion (Table 59.5). The Borg CR10 is used to rate tendon exertion, which is an 11-point scale (0–10) where 0 indicates no exertion and 10 indicates maximal exertion [46]. Light intensity activities can be performed daily, medium intensity activities require 2 days of recovery, and high intensity activities require 3 days of recovery. It is preferable that plyometric activities are completed in a single session, rather than back to back days at lower volumes.

Once the athlete is competing and training at pre-injury levels, it is important to recognize that the tendon may not be fully recovered. Alterations in tendon structure, mechanical properties, or muscle-tendon function may persist for a year or more, predisposing the athlete to reinjury [23, 34]. Therefore, it is critical that tendon loading exercises are continued, and the athlete is monitored closely for recurrence of symptoms.

59.4.3 Selected Patellar Tendon Loading Exercises

Several patellar tendon loading protocols have been utilized in clinical treatment studies with positive outcomes for patellar tendinopathy [34, 47–51]. However, there is insufficient evidence to suggest that one protocol is superior to others. The two most commonly utilized are the Eccentric Decline Squat protocol, first described by Purdam et al., and the Heavy Slow Resistance

protocol, described by Kongsgaard et al. The Eccentric Decline Squat protocol consists of single-leg eccentric squats performed on a 25° decline board. The rationale for using a decline board rather than level ground is based on biomechanical studies demonstrating that a decline board increases quadriceps muscle activity and patellar tendon loads [52, 53]. The Heavy Slow Resistance protocol consists of three isotonic exercises, the leg press, squat and hack squat, performed at relatively heavy loads and slow speeds [34]. Although, the exercises differ in each protocol, they share the common feature of progressive tendon loading. Therefore, we advocate using an individualized approach, based on the principles of tendon rehabilitation described above, the presentation of the athlete, and your clinical judgment.

To aid in designing an individualized patellar tendon loading protocol, example exercises and suggested phases for use are presented in Table 59.6.

Table 59.6 Suggested exercises for loading the patellar and Achilles tendons

Patellar tendon	<ul style="list-style-type: none"> • Wall sits • Spanish squat • Decline squat • Isometric knee extension • Step-ups • Bodyweight squat • Lunge • Leg press • Weighted squat • Isotonic knee extension • Leg press • Weight squat
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(continued)

Table 59.6 (continued)

Achilles tendon	<ul style="list-style-type: none"> • Isometric plantarflexion • Decline heel raise • Bilateral heel raises • Eccentric heel raises • Unilateral heel raises • Seated heel raises • Rebounding heel raise • Step-up with plantarflexion
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59.4.4 Other Considerations for Rehabilitation of Patellar Tendinopathy

While patellar tendon loading programs alone have proven effectiveness, they are not one-size-fits-all and may need to be adapted to optimize clinical outcomes.

59.4.4.1 Risk Factors

Deficits in ankle dorsiflexion range-of-motion, hip extension, external rotation and abduction strength, posterior thigh and quadriceps flexibility, and quadriceps activation have been identified as potential risk factors or impairments associated with patellar tendinopathy [54–60]. Therefore, these items may be of interest to address during the rehabilitation process.

Greater bodyweight has also been identified as a risk factor for the injury [54]. Although higher bodyweight typically reflects a more muscular build in highly athletic populations, weight loss may need to be considered in athletes with poor body composition.

59.4.4.2 Landing Biomechanics

Alterations in landing biomechanics are also common and may need to be addressed during the return-to-sport phase of rehabilitation. Athletes with patellar tendinopathy typically utilize a “stiff” landing pattern, with excessive hip and knee flexion at initial contact [61]. As a result of this flexed position at touch down, their available range of motion to dissipate ground reaction forces is limited and consequently, peak patellar tendon forces and loading rates are increased.

Furthermore, these athletes often demonstrate knee valgus upon landing, increasing strain on the medial portion of the tendon, which is the most common site of pathology within the patellar tendon [62].

59.4.4.3 Alterations to Loading Exercises

Compression of the tendon at the inferior pole of the patella has been hypothesized as an aggravating factor, especially in end-range knee flexion [63]. Although the primary mechanism of injury is likely excessive strain, it may be beneficial to avoid deep knee flexion if bony changes have been observed on imaging [64].

59.4.4.4 Pain Relief

Isometric exercises have recently gained popularity as a tool to manage pain during the competitive season. Two versions of this approach have been proposed, one utilized in a clinical setting and one utilized when on the road (e.g., training or locker room) [57, 65]. However, the results of the original study have not been able to be replicated and more recent studies found no difference between isometric and isotonic exercises in acute pain relief [66, 67]. Tendon loading may assist in pain reduction, but it is unlikely that contraction type matters.

Noxious electrical stimulation may also be used to manage symptoms when applied at the site of pain.

59.4.5 Selected Achilles Tendon Loading Exercises

For a long time, eccentric training was the primary treatment method for Achilles tendinopathy. In an article by Alfredson et al. in 1998, the authors described an eccentric loading program for the treatment of runners with tendon pain [68]. Silbernagel et al. described a more comprehensive treatment protocol that includes progressions from double limb to single limb loading exercises with inclusion of eccentric and concentric contractions in both standing and seated positions [33]. There is no longer a focus on solely eccentric contractions as comprehensive loading

programs that include isometric, concentric, and eccentric contractions have been found to be equally effective.

Tendon loading is typically initiated with standing and seated heel raises. If symptoms are highly irritable, it may be necessary to perform these exercises on a level surface, as opposed to a step or incline, or with less resistance. The athlete is encouraged to perform gentle circulation exercises with ankle pumps throughout the day during team meetings and prolonged periods of inactivity. Standing heel raises include bilateral, unilateral, and eccentric varieties as this will incorporate multiple types of contractions and provide an opportunity for high volume of repetitions. This should be trialed and utilized during training sessions and practices and games if the specific athlete responds positively to such a contraction.

As irritability decreases, repetitions of the loading program may be progressed by performing the heel raises on an incline or off a step to increase the range of motion required of the loading task. Quick rebounding heel raises may also be added at this time to provide the tendon an opportunity to respond to stresses at higher speeds of loading and prepare for plyometric exercises. If the athlete is responding well to the increased demands within the guidelines set forth by the pain-monitoring model, the athlete may begin to incorporate heavier strength training and may start increasing running and jumping activities. As the athlete enters Phase 3, return to sport, it will be important to continue a maintenance program with a focus on primarily single leg and quick rebounding heel raises.

Examples of specific Achilles tendon loading exercises are provided in Table 59.6.

59.4.6 Other Considerations for Rehabilitation of Achilles Tendinopathy

59.4.6.1 Variations Based on Location of Pathology

Interventions for Achilles tendinopathy have primarily been developed for mid-substance pathology; however, some athletes will present with

insertional symptoms and management must be adjusted as standard rehabilitation protocols may aggravate symptoms and irritate the tendon insertion. When there is pathology near the insertion of the tendon onto the calcaneus, dorsiflexion stretching and loading in dorsiflexion ranges should be avoided [69, 70]. As the ankle moves into dorsiflexion, the posterior–superior surface of the calcaneus impinges against the deep side of the tendon insertion [71, 72]. This location of impingement may actually contribute to the pathology, which is more prevalent on the deep side of the Achilles tendon. Heel lifts may have a role in avoiding this impingement position and decrease irritation of the tendon [73]. The avoidance of uphill or incline running may be beneficial as well.

Based on imaging results, it may be found that there is bursal or paratenon irritation in combination with the Achilles tendinopathy. In cases with bursal involvement, the treatment is modified to limit dorsiflexion, as with insertional symptoms, in an effort to reduce compression of the bursa. If paratenon irritation is present, trialing shoe cut-outs at the heel counter is recommended to offload the painful area and reduce external pressure/friction.

59.4.6.2 Risk Factors

There is limited evidence for risk factors relating to Achilles tendinopathy. A recent systematic review reported limited evidence for the following as risk factors, lower leg tendinopathy, use of fluoroquinolone medication, plantar flexion weakness, and abnormal gait patterns [74]. Of interest was that the study did not find support for body weight or body mass index, static foot posture measurements, and physical activity level being risk factors. Altered tendon structure observed with ultrasound imaging has been found to increase the risk of developing symptoms. It is therefore of importance to perform a thorough evaluation of the individual with Achilles tendinopathy and design an individual treatment plan to both address the tendon injury and other possible impairments that could be related to the development of the overuse injury [75].

59.5 Conclusion

Controlled tendon loading is currently the best supported treatment for rehabilitation of tendinopathies. To maximize the effectiveness of this intervention, it is critical to combine individualized tendon loading exercises with activity modification and a progressive return-to-sport phase, which gradually improves the tendon's tolerance to sport-specific demands. Although symptoms at the patellar and Achilles tendons are most common in basketball players, the principles described in this chapter can be applied to tendinopathies at other locations in the upper and lower extremity. In these cases, it is important to consider anatomical and biomechanical factors that may increase compression or require alterations to loading. Furthermore, a subset of patients may not respond to tendon loading exercises alone. Adjunctive treatments, such as shockwave or platelet-rich plasma injections, may need to be considered, and surgery may be utilized as a last resort. In these instances, the athlete will still benefit from tendon loading exercises to restore tendon health and prepare them for a return to sport.

References

1. Lian ØB, Engebretsen L, Bahr R. Prevalence of Jumper's knee among elite athletes from different sports: a cross-sectional study. *Am J Sports Med.* 2005;33:561–7.
2. Zwerver J, Bredeweg SW, van den Akker-Scheek I. Prevalence of Jumper's knee among nonelite athletes from different sports: a cross-sectional survey. *Am J Sports Med.* 2011;39:1984–8.
3. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the national basketball association: a 17-year overview. *Sports Health.* 2010;2:284–90.
4. Gross MT. Chronic tendinitis: pathomechanics of injury, factors affecting the healing response, and treatment. *J Orthop Sports Phys Ther.* 1992;16:248–61.
5. Abate M, Silbernagel KG, Siljeholm C, Di Iorio A, De Amicis D, Salini V, Werner S, Paganelli R. Pathogenesis of tendinopathies: inflammation or degeneration? *Arthritis Res Ther.* 2009;11:235.
6. Arya S, Kulig K. Tendinopathy alters mechanical and material properties of the Achilles tendon. *J Appl Physiol.* 2010;108:670–5.

7. Obst SJ, Heales LJ, Schrader BL, Davis SA, Dodd KA, Holzberger CJ, Beavis LB, Barrett RS. Are the mechanical or material properties of the Achilles and patellar tendons altered in tendinopathy? A systematic review with meta-analysis. *Sports Med.* 2018;48:2179–98.
8. Helland C, Bojsen-Møller J, Raastad T, Seynnes OR, Moltubakk MM, Jakobsen V, Visnes H, Bahr R. Mechanical properties of the patellar tendon in elite volleyball players with and without patellar tendinopathy. *Br J Sports Med.* 2013;47:862–8.
9. Chang Y-J, Kulig K. The neuromechanical adaptations to Achilles tendinosis. *J Physiol.* 2015;593:3373–87.
10. Rio E, Kidgell D, Moseley GL, Cook J. Elevated corticospinal excitability in patellar tendinopathy compared with other anterior knee pain or no pain. *Scand J Med Sci Sports.* 2016;26:1072–9.
11. Larsson MEH, Käll I, Nilsson-Helander K. Treatment of patellar tendinopathy—a systematic review of randomized controlled trials. *Knee Surg Sport Traumatol Arthrosc.* 2012;20:1632–46.
12. Magnusson RA, Dunn WR, Thomson AB. Nonoperative treatment of midportion Achilles tendinopathy: a systematic review. *Clin J Sport Med.* 2009;19:54–64.
13. Nirschl RP, Ashman ES. Elbow tendinopathy: tennis elbow. *Clin Sports Med.* 2003;22:813–36.
14. Stanish WD, Curwin S, Mandell S. *Tendinitis: its etiology and treatment.* New York: Oxford University Press; 2000.
15. Maffulli N, Regine R, Angelillo M, Capasso G, Filice S. Ultrasound diagnosis of Achilles tendon pathology in runners. *Br J Sports Med.* 1987;21:158–62.
16. Fritschy D, de Gautard R. Jumper's knee and ultrasonography. *Am J Sports Med.* 2016;16:637–40.
17. Ferretti A, Ippolito E, Mariani P, Puddu G. Jumper's knee. *Am J Sports Med.* 1983;11:58–62.
18. Purdam CR, Cook JL, Hopper DM, et al. Discriminative ability of functional loading tests for adolescent jumper's knee. *Phys Ther Sport.* 2003;4:3–9.
19. Mendonça LDM, Ocarino JM, Bittencourt NFN, Fernandes LMO, Verhagen E, Fonseca ST, Maria L, Fernandes O. The accuracy of VISA-P questionnaire, single-leg decline squat and tendon pain history to identify patellar tendon abnormalities in adult athletes. *J Orthop Sports Phys Ther.* 2016;46:1–22.
20. Maffulli N, Kenward MG, Testa V, Capasso G, Regine R, King JB. Clinical diagnosis of Achilles tendinopathy with tendinosis. *Clin J Sport Med.* 2003;13:11–5.
21. Maffulli N, Binfield PM, Moore D, King JB. surgical decompression of chronic central core lesions of the Achilles tendon; 1999.
22. Williams JGP. Achilles tendon lesions in sport; 1986.
23. Silbernagel KG, Thomeé R, Eriksson BI, Karlsson J. Full symptomatic recovery does not ensure full recovery of muscle-tendon function in patients with Achilles tendinopathy. *Br J Sports Med.* 2007;41:276–80; discussion 280.
24. Vicenzino B, de Vos R-J, Alfredson H, et al. ICON 2019: International Scientific Tendinopathy Symposium CONsensus on Core Outcome Domains for Tendinopathy (COR-T): A Delphi Study of Patients and Health Care Professional. *Br J Sports Med* 2019.
25. Khan KM, Scott A. Mechanotherapy: how physical therapists' prescription of exercise promotes tissue repair. *Br J Sports Med.* 2009;43:247–52.
26. Arampatzis A, Karamanidis K, Albracht K. Adaptation responses of the human Achilles tendon by modulation of the applied cyclic strain magnitude. *J Exp Biol.* 2007;210:2743–53.
27. Kongsgaard M, Reitelseder S, Pedersen TG, Holm L, Aagaard P, Kjaer M, Magnusson SP. Region specific patellar tendon hypertrophy in humans following resistance training. *Acta Physiol (Oxf).* 2007;191:111–21.
28. Lavagnino M, Arnoczky S, Tian T, Vaupel Z. Effect of amplitude and frequency of cyclic tensile strain on the inhibition of MMP-1 mRNA expression in tendon cells: an in vitro study. *Connect Tissue Res.* 2007;44:181–7.
29. Yang G, Crawford RC, Wang JHC. Proliferation and collagen production of human patellar tendon fibroblasts in response to cyclic uniaxial stretching in serum-free conditions. *J Biomech.* 2004;37:1543–50.
30. Joshi SD, Webb K. Variation of cyclic strain parameters regulates development of elastic modulus in fibroblast/substrate constructs. *J Orthop Res.* 2008;26:1105–13.
31. Kubo K, Kanehisa H, Fukunaga T. Effects of different duration isometric contractions on tendon elasticity in human quadriceps muscles. *J Physiol.* 2001;536:649–55.
32. Arampatzis A, Peper A, Bierbaum S, Albracht K. Plasticity of human Achilles tendon mechanical and morphological properties in response to cyclic strain. *J Biomech.* 2010;43:3073–9.
33. Silbernagel KG, Thomeé R, Eriksson BI, Karlsson J. Continued sports activity, using a pain-monitoring model, during rehabilitation in patients with Achilles tendinopathy: a randomized controlled study. *Am J Sports Med.* 2007;35:897–906.
34. Kongsgaard M, Kovanen V, Aagaard P, Doessing S, Hansen P, Laursen AH, Kaldau NC, Kjaer M, Magnusson SP. Corticosteroid injections, eccentric decline squat training and heavy slow resistance training in patellar tendinopathy. *Scand J Med Sci Sports.* 2009;19:790–802.
35. Couppé C, Svensson RB, Silbernagel KG, Langberg H, Magnusson SP. Eccentric or concentric exercises for the treatment of tendinopathies? *J Orthop Sports Phys Ther.* 2015;45:853–63.

36. Garma T, Kobayashi C, Haddad F, Adams GR, Bodell PW, Baldwin KM. Similar acute molecular responses to equivalent volumes of isometric, lengthening, or shortening mode resistance exercise. *J Appl Physiol.* 2007;102:135–43.
37. Farup J, Rahbek SK, Vendelbo MH, Matzon A, Hindhede J, Bejder A, Ringgard S, Vissing K. Whey protein hydrolysate augments tendon and muscle hypertrophy independent of resistance exercise contraction mode. *Scand J Med Sci Sports.* 2014;24:788–98.
38. Leddy MH, Lambert MJ, Ogles BM. Psychological consequences of athletic injury among high-level competitors. *Res Q Exerc Sport.* 1994;65:347–54.
39. Fukashiro S, Komi PV, Järvinen M, Miyashita M. In vivo Achilles tendon loading' during jumping in humans. *Eur J Appl Physiol Occup Physiol.* 1995;71:453–8.
40. Scott SH, Winter DA. Internal forces of chronic running injury sites. *Med Sci Sports Exerc.* 1990;22:357–69.
41. Edwards S, Steele JR, Cook JL, Purdam CR, McGhee DE, Munro BJ. Characterizing patellar tendon loading during the landing phases of a stop-jump task. *Scand J Med Sci Sports.* 2012;22:2–11.
42. Edwards S, Steele JR, Purdam CR, Cook JL, McGhee DE. Alterations to landing technique and patellar tendon loading in response to fatigue. *Med Sci Sports Exerc.* 2014;46:330–40.
43. Grävare Silbernagel K, Crossley KM. A proposed return-to-sport program for patients with midportion Achilles tendinopathy: rationale and implementation. *J Orthop Sports Phys Ther.* 2015;45:876–86.
44. Janssen I, Brown NATA, Munro BJ, Steele JR. Variations in jump height explain the between-sex difference in patellar tendon loading during landing. *Scand J Med Sci Sports.* 2015;25:265–72.
45. Lai A, Schache AG, Lin Y-C, Pandy MG. Tendon elastic strain energy in the human ankle plantar-flexors and its role with increased running speed. *J Exp Biol.* 2014;217:3159–68.
46. Borg G. Borg's perceived exertion and pain scales. Champaign, IL: Human Kinetics; 1998.
47. Young MA, Cook JL, Purdam CR, Kiss ZS, Alfredson H. Eccentric decline squat protocol offers superior results at 12 months compared with traditional eccentric protocol for patellar tendinopathy in volleyball players. *Br J Sports Med.* 2005;39:102–5.
48. Kongsgaard M, Qvortrup K, Larsen J, Aagaard P, Doessing S, Hansen P, Kjaer M, Magnusson SP. Fibril morphology and tendon mechanical properties in patellar tendinopathy: effects of heavy slow resistance training. *Am J Sports Med.* 2010;38:749–56.
49. Purdam CR, Jonsson P, Alfredson H, Lorentzon R, Cook JL, Khan KM. A pilot study of the eccentric decline squat in the management of painful chronic patellar tendinopathy. *Br J Sports Med.* 2004;38:395–7.
50. Cannell LJ, Taunton JE, Clement DB, Smith C, Khan KM. A randomised clinical trial of the efficacy of drop squats or leg extension/leg curl exercises to treat clinically diagnosed jumper's knee in athletes: pilot study. *Br J Sports Med.* 2001;35:60–4.
51. Frohm A, Saartok T, Halvorsen K, Renström P. Eccentric treatment for patellar tendinopathy: a prospective randomised short-term pilot study of two rehabilitation protocols. *Br J Sports Med.* 2007;41:e7.
52. Zwerver J, Bredeweg SW, Hof AL. Biomechanical analysis of the single-leg decline squat. *Br J Sports Med.* 2007;41:264–8.
53. Kongsgaard M, Aagaard P, Roikjaer S, Olsen D, Jensen M, Langberg H, Magnusson SP. Decline eccentric squats increases patellar tendon loading compared to standard eccentric squats. *Clin Biomech.* 2006;21:748–54.
54. Sprague AL, Smith AH, Knox P, Pohlig RT, Grävare Silbernagel K. Modifiable risk factors for patellar tendinopathy in athletes: a systematic review and meta-analysis. *Br J Sports Med.* 2018;52:1575–85.
55. Zhang ZJ, Lee WC, Ng GYF, Fu SN. Isometric strength of the hip abductors and external rotators in athletes with and without patellar tendinopathy. *Eur J Appl Physiol.* 2018;118:1635–40.
56. Scattone Silva R, Nakagawa TH, Ferreira ALG, et al. Lower limb strength and flexibility in athletes with and without patellar tendinopathy. *Phys Ther Sport.* 2016;20:19–25.
57. Rio E, Kidgell D, Purdam C, Gaida J, Moseley GL, Pearce AJ, Cook J. Isometric exercise induces analgesia and reduces inhibition in patellar tendinopathy. *Br J Sports Med.* 2015;49:1277–83.
58. van der Worp H, van Ark M, Roerink S, Pepping G-JJ, van den Akker-Scheek I, Zwerver J. Risk factors for patellar tendinopathy: a systematic review of the literature. *Br J Sports Med.* 2011;45:446–52.
59. Malliaras P, Cook JL, Kent P. Reduced ankle dorsiflexion range may increase the risk of patellar tendon injury among volleyball players. *J Sci Med Sport.* 2006;9:304–9.
60. Scattone Silva R, Ferreira ALG, Nakagawa TH, Santos JEM, Serrão FV. Rehabilitation of patellar tendinopathy using hip extensor strengthening and landing-strategy modification: case report with 6-month follow-up. *J Orthop Sports Phys Ther.* 2015;45:899–909.
61. Van Der Worp H, de Poel HJ, Diercks RL, Van Den Akker-Scheek I, Zwerver J, der Van WH, de Poel HJ, Diercks RL, Van Den Akker-Scheek I, Zwerver J. Jumper's knee or lander's knee? A systematic review of the relation between jump biomechanics and patellar tendinopathy. *Int J Sports Med.* 2014;35:714–22.
62. Yu JS, Popp JE, Kaeding CC, Lucas J. Correlation of MR imaging and pathologic findings in athletes undergoing surgery for chronic patellar tendinitis. *AJR Am J Roentgenol.* 1995;165:115–8.
63. Hamilton B, Purdam C. Patellar tendinosis as an adaptive process: a new hypothesis. *Br J Sports Med.* 2004;38:758–61.
64. Schmid MR, Hodler J, Cathrein P, Duester S, Jacob HA, Romero J. Is impingement the cause of jumper's knee? Dynamic and static magnetic resonance

- imaging of patellar tendinitis in an open-configuration system. *Am J Sports Med.* 2012;30:388–95.
65. Rio E, Purdam C, Girdwood M, Cook J. Isometric exercise to reduce pain in patellar tendinopathy in-season; is it effective “on the road?”. *Clin J Sport Med.* 2017;0:1.
 66. Holden S, Lyng K, Graven-Nielsen T, Rathleff MS. Isometric versus isotonic exercises for acute analgesia in patellar tendinopathy: A randomised crossover trial. *J Sci Med Sport.* 2020;23(3):208–14.
 67. van Ark M, Cook JL, Docking SI, Zwerver J, Gaida JE, van den Akker-Scheek I, Rio E. Do isometric and isotonic exercise programs reduce pain in athletes with patellar tendinopathy in-season? A randomised clinical trial. *J Sci Med Sports.* 2016;19:702–6.
 68. Alfredson H, Pietilä T, Lorentzon R. Heavy-load eccentric calf muscle training for the treatment of chronic Achilles tendinosis; 1998.
 69. Kearney R, Costa ML. Insertional Achilles tendinopathy management: a systematic review. *Foot Ankle Int.* 2010;31:689–94.
 70. Wiegerinck JI, Kerkhoffs GM, van Sterkenburg MN, Sierrevelt IN, van Dijk CN. Treatment for insertional Achilles tendinopathy: a systematic review. *Knee Surg Sports Traumatol Arthrosc.* 2013;21:1345–55.
 71. Leitze Z, Sella EJ, Aversa JM. Endoscopic decompression of the retrocalcaneal space. *J Bone Jt Surg.* 2003;85:1488–96.
 72. Shibuya N, Thorud JC, Agarwal MR, Jupiter DC. Is calcaneal inclination higher in patients with insertional Achilles tendinosis? A case-controlled, cross-sectional study. *J Foot Ankle Surg.* 2012;51:757–61.
 73. Chimenti RL, Flemister AS, Ketz J, Bucklin M, Buckley MR, Richards MS. Ultrasound strain mapping of Achilles tendon compressive strain patterns during dorsiflexion. *J Biomech.* 2016;49:39–44.
 74. Van Der Vliet AC, Breda SJ, Oei EH, Verhaar JAN, De Vos RJ. Clinical risk factors for Achilles tendinopathy: a systematic review. *Br J Sports Med.* 2019;1–11.
 75. Carcia CR, Martin RL, Wukich DK. Achilles pain, stiffness, and muscle power deficits: Achilles tendinitis. *J Orthop Sports Phys Ther.* 2010;40:A1–A26.
 76. Macdermid JC, Silbernagel KG. Outcome evaluation in tendinopathy: foundations of assessment and a summary of selected measures. *J Orthop Sports Phys Ther.* 2015;45:950–64.
 77. Visentini PJ, Khan KM, Cook JL, Kiss ZS, Harcourt PR, Wark JD. The VISA score: an index of severity of symptoms in patients with jumper’s knee (patellar tendinosis). *J Sci.* 1998;1:22–8.
 78. Wageck BB, de Noronha MA, Lopes AD, da Cunha RA, Takahashi RH, Pena Costa LO. Cross-cultural adaptation and measurement properties of the Brazilian Portuguese version of the Victorian Institute of Sport Assessment-Patella (VISA-P) scale. *J Orthop Sports Phys Ther.* 2013;43:163–71.
 79. Frohm A, Saartok T, Edman G, Renström P. Psychometric properties of a Swedish translation of the VISA-P outcome score for patellar tendinopathy. *BMC Musculoskelet Disord.* 2004;5:49.
 80. Clarsen B, Myklebust G, Bahr R. Development and validation of a new method for the registration of overuse injuries in sports injury epidemiology: the Oslo Sports Trauma Research Centre (OSTRC) overuse injury questionnaire. *Br J Sports Med.* 2013;47:495–502.
 81. Owoeye OBA, Wiley JP, Walker REA, Palacios-Derflingher L, Emery CA. Diagnostic accuracy of a self-report measure of patellar tendinopathy in youth basketball. *J Orthop Sports Phys Ther.* 2018:1–26.
 82. Vetrano M, Castorina A, Vulpiani MC, Baldini R, Pavan A, Ferretti A. Platelet-rich plasma versus focused shock waves in the treatment of Jumper’s knee in athletes. *Am J Sports Med.* 2013;41:795–803.
 83. Del Baño-Aledo ME, Martínez-Payá JJ, Ríos-Díaz J, Mejías-Suárez S, Serrano-Carmona S, de Groot-Ferrando A. Ultrasound measures of tendon thickness: intra-rater, inter-rater and inter-machine reliability. *Musc Ligam Tend J.* 2015;7:192–9.
 84. Gellhorn AC, Carlson MJ. Inter-rater, intra-rater, and inter-machine reliability of quantitative ultrasound measurements of the patellar tendon. *Ultrasound Med Biol.* 2013;39:791–6.
 85. Robinson JM, Cook JL, Purdam C, Visentini PJ, Ross J, Maffulli N, Taunton JE, Khan KM, Victorian Institute Of Sport Tendon Study Group. The VISA-A questionnaire: a valid and reliable index of the clinical severity of Achilles tendinopathy. *Br J Sports Med.* 2001;35:335–41.
 86. Tumilty S, Munn J, Abbott JH, McDonough S, Hurley DA, Baxter GD. Laser therapy in the treatment of Achilles tendinopathy: a pilot study. *Photomed Laser Surg.* 2008;26:25–30.
 87. Tumilty S, McDonough S, Hurley DA, Baxter GD. Clinical effectiveness of low-level laser therapy as an adjunct to eccentric exercise for the treatment of Achilles’ tendinopathy: a randomized controlled trial. *Arch Phys Med Rehabil.* 2012;93:733–9.
 88. Silbernagel KG, Gustavsson A, Thomee R, Karlsson J, Thomeé R. Evaluation of lower leg function in patients with Achilles tendinopathy. *Knee Surg Sports Traumatol Arthrosc.* 2006;14:1207–17.
 89. Svantesson U, Carlsson U, Takahashi H, Thomee R, Grimby G, Svantesson U. Comparison of muscle and tendon stiffness, jumping ability, muscle strength and fatigue in the plantar flexors. *Scand J Med Sci Sports.* 1998;8(5 Pt):1.
 90. Möller M, Lind K, Styf J, Karlsson J. The reliability of isokinetic testing of the ankle joint and a heel-raise test for endurance. *Knee Surg Sports Traumatol Arthrosc.* 2005;13:60–71.
 91. Kruse A, Stafilidis S, Tilp M. Ultrasound and magnetic resonance imaging are not interchangeable to assess the Achilles tendon cross-sectional-area. *Eur J Appl Physiol.* 2017;117:73–82.



On-Court Rehabilitation—From Treatment Table and Return to Play to a Return to Performance

60

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60.1 Introduction

Rehabilitation for basketball players needs to include a basketball as well as basketball-specific movements to properly prepare the athlete for a return to the court. Although the specifics of a rehabilitation program will be tailored to each individual athlete and injury, there are common elements to almost all rehabilitations. In particular, on-court progressions to gradually build and expose the athlete to the demands they will face upon a return to basketball can be similar across injuries. This chapter will focus on rehabilitation after a lower extremity injury and provide ideas for performance teams (medical, rehabilitation, strength and conditioning, sports science, and coaching) to consider in preparing an athlete.

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60.2 Rehabilitation and Return to Performance Planning

Immediately after injury, communication and education with the athlete and all stakeholders are essential. A detailed rehabilitation plan should be compiled, with input from rehabilitation professionals, strength and conditioning specialists, sports scientists, doctors, surgeons, coaches, and the athlete. Rehabilitation plans should include (1) clear objective milestones demarcating the phases of rehabilitation, (2) athlete-tailored return to sport criteria, and (3) key performance indicators (KPIs) defining an athlete's return to preinjury level of performance.

There is a great deal of literature on objective milestones in rehabilitation and return to sport criteria [1–6]. Structures such as the Strategic Assessment of Risk and Risk Tolerance (StARRT) framework [7] for return-to-play decision-making can assist performance teams in planning rehabilitation phases and return to sport criteria. This chapter will not focus on the objective milestones or return to sport criteria, particularly as the other chapters in this book provide recommendations specific to each injury type.

Return to sport is a continuum [8]. An athlete's return to training may be considered their return to participation. A resumption of full training and game play could be considered a return to play

[8]. However, rehabilitation is not over when an athlete returns to play. A point guard who, preinjury, averaged 25 points a game, but upon return is only averaging 10, has returned to play but not returned to their preinjury level of performance. Rehabilitation is not complete until the athlete has returned to performance. Return to performance is defined uniquely for each athlete. Similar to how objective measures and clinical milestones are laid out in the rehabilitation plan, clearly establishing KPIs for return to performance will help the team fairly assess the athlete's progress toward their full return to performance.

KPIs in return to performance should be quantifiable and include both physical-performance as well as basketball-performance measures. Physical-performance indicators could include peak speed, accelerations/decelerations, total distance, or number of jumps. Basketball-performance indicators could include points, rebounds, assists, blocks, or turnovers. There are numerous variables that could be chosen as KPIs, but only those of highest priority for a given player should be selected. For example, points and assists may be more appropriate KPIs for a point guard, whereas blocks or rebounds may be more appropriate for a center.

Deficits identified via KPIs can guide prevention or practice efforts. Clinicians may be tempted to see a deficit in a basketball-performance KPI and associate it with skill or coaching fault; however, it is important to examine all deficits from both a basketball and a rehabilitation standpoint. For example, if an athlete's KPI was driving to the basket equally on the right and left, but in their first few games back after a left ankle sprain the athlete was only driving to the basket on their right, the underlying cause for the deficit could be basketball-related or rehabilitation-related. From a basketball perspective, the reason could be that they could more easily beat their opponents on that side. Or from a rehabilitation perspective, the reason could be that the athlete is fearful of driving off the left ankle or lacks the strength or power to generate enough force to drive off the left ankle. Coaching and performance staff working collaboratively to uncover the reasons and to address

deficits in KPIs is the fundamental basis for return to performance rehabilitation.

Education is important so that the players understand and have some ownership over the injury, objective milestones, approximate time frames for each phase of rehabilitation, return to sport criteria, and KPIs. Trust is also required to ensure the player is open about symptom response, concerns with specific movement patterns, and the influence of external pressures like coaches, general managers, agents, playing contracts, or family members [9]. Interdepartmental collaboration is crucial to ensure all objective milestones are being met within the confines of what is safe in each phase of rehabilitation. In effect, making sure everyone is clear about any limitation that may prohibit strength and conditioning or coaches/player development from performing certain activities with the athlete to facilitate the player's safety. Conversely, clear communication can also allow for a new movement pattern introduced during a rehabilitation exercise to be reinforced during lifting or on-court session.

For an athlete, sport participation and self-worth are often deeply intertwined. Following an injury, lack of full participation may pose a significant psychological barrier. Thus, safe ways to introduce sports-specific drills into rehabilitation as soon as possible may be beneficial. Facilitating ways for the player to stay involved in team activities such as helping or observing at practice, film, and game walk throughs can assist with maintaining skills and cortical motor mapping while injured simply through visualization [10]. Involvement in team activities also helps with motivation, as the player still feels part of the team. Giving the player responsibilities, such as with scouting reports, assisting younger players, or rebounding, can also help players feel as though they are contributing to the team, even though they are not on-court playing.

60.3 Acute Phase of Rehabilitation

The acute phase of rehabilitation is focused on addressing impairments and reestablishing basic levels of function, such as activities of

daily living. Additional priorities include education and maintenance of general strength and conditioning.

Even in early stages of rehabilitation when impairments are significant, a basketball can be integrated into rehabilitation. For example, if an athlete is non- or partial-weight-bearing, they can still shoot a basketball with the leg on a scooter or while sitting in a chair. The focus of these shooting drills should be on form and performed within a small distance from basket. Coaches' input can help prevent poor mechanics from developing or addressing previously existing form habits. A basketball can also be incorporated into stationary drills (such as seated ball handling or static passing drills), core exercises (such as Russian twists or crunches into overhead throws), or hip strengthening (such as isometric adduction or using the ball as a prop for manual perturbation). Further, not all basketball drills have to be part of planned rehabilitation. Independent work on free throw form in supine can be performed while the athlete is icing or elevating.

Regardless of injury, good neuromuscular control and proprioception are important for a favorable prognosis and secondary prevention [6, 11, 12]. Neuromuscular control and proprioceptive exercises can begin when an athlete is non-weight-bearing but become easier to incorporate a basketball when the athlete is able to weight-bear. Progression of balance can be done in a multitude of ways: double leg to single leg, firm surfaces to compliant or unstable surfaces, controlled perturbations to reactive perturbations, eyes open to eyes closed, etc. Within all of these variations, clinicians can add a basketball either to have the athlete simply hold, throw/catch, bounce, dribble, or shoot. For example, the exercise might start by performing single-leg balance on stable surface, then on a foam pad. A basketball might then be added to drill so that as the athlete balances they have to dribble, then progress to dribbling in patterns between hands as well as pounding or alternating rhythms such as bouncing in-and-out, forward/backward, quickly and close to the ground, or high-power dribbles (Fig. 60.1). Other progressions could include throwing and catching at the chest, then in mul-

tle directions, and then adding a weighted basketball.

Once an athlete can weight-bear, stationary dribbling drills can be initiated. Examples include stationary dribbling with one hand or the other, pounding, or crossing over. Patterns that involve dribbling and passing can also challenge the athlete while progressing weight-bearing, for example, dribbling twice with the right hand, crossing over¹ and passing with the left, receiving with the right, and repeating. Ball movements such as these can also facilitate movements that clinicians are trying to promote. Crossovers with the feet shoulder width apart force the athlete to weight shift from leg to leg. Dribbling between the legs forces the athlete to step and weight-bear through both legs, potentially forcing the athlete to weight shift into the involved limb. Progressing dribbling drills to higher-level patterns with successive moves can add a cognitive load to further challenge the physical skill. A common series is receive pass, crossover, crossover; receive pass, crossover, between the legs²; and receive pass, crossover, behind the back.³ The player then changes the starting move while keeping the crossover, between the legs, behind the back sequence without stopping motion. To clarify, the next sequence would be receive pass, between the legs, crossover; receive pass, between the legs, between the legs; and receive pass, between the legs, behind the back. This sequence is repeated then with behind the back as the starting move.

When able to fully bear weight but yet intolerant to high impact activities, shooting from flat foot can be initiated, followed by elevating onto the toes (Fig. 60.2). The focus of these shooting exercises should still be on form shooting from small distances and progressing outward. If force plates are available, having the athlete go through

¹A cross over is when the ball is bounced from one hand to the other in front of the body

²Between the legs means the ball is bounced from one hand to the other through the opening between the athlete's legs

³Behind the back is bouncing the ball from one hand to the other behind the body



Fig. 60.1 (a) Dribbling close to body. (b) Dribbling further out from body or across body. (c) Dribbling around legs. (d) Dribbling between legs. (e) Single-leg balance on foam pad while dribbling

their shooting motion with biofeedback can help them gain insight about their own movement patterns and potentially help them equally distribute their weight.

As the athlete's tolerance to impact continues to increase, stationary jump shooting may take place. Shooting as such can start with very small jumps (1–2 cm from ground) and increase in height and distance from basket. Stepping into shots can also be introduced, based on the injury.

For example, in the case of a lateral ankle sprain, taking one step forward or backwards into a shot can be progressed to stepping laterally into a shot. In contrast, for a hamstring injury, laterally stepping might be introduced first, then forward and then backward steps.

Variables within tissue-specific exercises and basketball rehabilitation drills that can be modified include speed and stationary vs. moving linearly. Linear movements (sagittal plane) can be

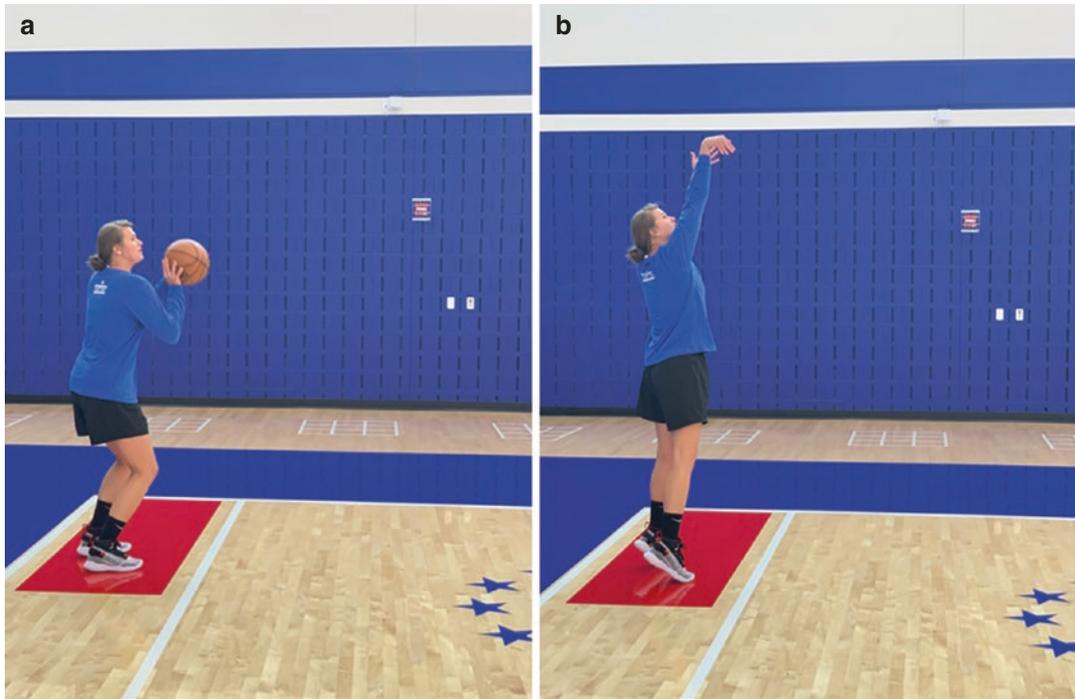


Fig. 60.2 (a) Starting position for shot. (b) Ending position for shot. Weight is transferred to toes but feet do not leave the ground

progressed to lateral (frontal plane) and then to rotational (transverse plane). These sport-specific drills should be in complement to rehabilitation exercises and be done when safely appropriate. With activities in rehabilitation, using the general soreness criterion (see Chap. 56 Rehabilitation of Knee Injuries in Basketball Players) can help assess patient's tolerance to activity. Other subjective and objective measures to monitor throughout the acute phase are stiffness, pain, and swelling. A significant increase in these measures may demonstrate intolerance and necessitate an adjustment to rehabilitation.

60.4 Subacute Phase of Rehabilitation to Return to Participation

60.4.1 Exercise Progression

In the subacute phase of rehabilitation, the performance team can begin to incorporate higher

level training for the athlete, building on the foundational strength and neuromuscular gains from the acute phase. Strengthening, which was focused on building stability in the acute phase, in the subacute phase will begin to be focused more on endurance and progressing to power. Neuromuscular control and proprioception exercises can begin integrating sport-specific movement patterns and begin to enhance control with more dynamic tasks, specifically in single-leg and multiplanar activities. Practitioners may find utilizing a “working backwards” approach, an easy way to help progress an athlete into more dynamic and basketball-specific tasks. “Working backwards” means that practitioner starts with the end task or on-court skill and deconstructs the skill into components. Introducing these components individually and then building them together will help the practitioner plan how to train and then progress the task/skill within a controlled environment (Table 60.1) [13].

Cutting and pivoting are important movements in basketball. The obvious main compo-

Table 60.1 “Working Backwards” to deconstruct and then train movements

Skill	Baseline components to achieve	Methods to progress	High-level adaptations
Cutting	Multidirectional stepping	Submaximal 45/90/180° cutting; Curling	Increase speed and intensity; Add defender
Defensive slides	Controlled side stepping	Resistance with band/cables; Anticipated change of direction	Unanticipated change of direction; Add offensive player
Jumping	Double and single-leg hop	Hopping patterns (multiplanar, over obstacles)	Perturbations; Cognitive load such as catching ball

ment of cutting is a change of direction, but these changes of direction occur at many different angles in a game. Thus, training must include a variety of movement patterns to accommodate differences such as the V cut,⁴ L cut,⁵ flare,⁶ or curl.⁷

Jumping also needs to be gradually progressed through the subacute phase. Given the load through the lower extremity involved in jumping, the height of jump should be gradually progressed based on the injury and athlete’s tolerance. The quantity and intensity of jumping, such as length of time the athlete spends shooting, should be closely monitored to ensure there is a gradual progression rather than a large spike. Shooting often is performed on 2 feet; however, it is important to introduce the athlete to single-leg landings. Double-leg takeoff/landing can be progressed to double-leg takeoff/single-leg landing, to hop-scotch patterns (alternating double and single-leg takeoff/landings), to single-leg takeoff/landings.

Once the athlete exhibits good control of the underlying basic movements, these can be further progressed by manipulating other variables such as controlled vs. open environment, speed,

intensity, perturbations, and cognitive load [14]. Sport-specific movements may initially need to be trained without a ball, but as the athlete gains competency, adding a ball is essential in preparing the athlete for the demands of training/game play.

Maintaining good cardiovascular readiness is also key in the subacute phase of rehabilitation, particularly as fatigue may increase the risk of injury [15]. Cardiovascular fitness can be trained off-court by biking, swimming, arm-biking, rowing, or alternatives such as battle ropes, boxing, or use of a VersaClimber™. On-court examples of cardiovascular training appropriate for the demands of basketball include successive full court transitions, acceleration and deceleration training, cutting while running, and curved line running. Additionally, conditioning drills might involve basketball skills such as having the athlete running diagonally at speed across court, shooting at 3-point shot, and then repeating 4 times. The drill can be repeated until that athlete makes 3 of 4 shots, challenging the athlete’s endurance and skills under fatigue. Another example is accelerating from underneath the basket to the corner, setting up to defend a coach with the ball, then sprinting to the opposite corner to defend another coach.

60.4.2 On-Court Progression

Shooting, ball handling, and team play progressions occur hand-in-hand with rehabilitation skill acquisition. For example, once an athlete can

⁴A V cut is when the athlete starts around the three point line, runs towards the basket and then cuts at an acute angle back out towards the 3 point line to receive a pass

⁵An L cut is when the athlete starts in the paint, running down the court, cutting 90 degrees towards the 3 point line to receive a pass

⁶A flare is when an athlete cuts around a screen and runs away from the basket towards the perimeter

⁷A curl is when an athlete cuts around a screen towards the basket, generally the opposite of a flare

safely perform single-leg jumping tasks, clinicians can add an overhead focus, such as with catching a basketball while landing. Tables 60.2, 60.3, and 60.4 describe progressions for building exercises from simple to more complex (Fig. 60.3).

Transitioning the player back to practice should be a gradual process, particularly in extended rehabilitations. Collaboration with

coaches is essential during this time as they can help provide safe environments for the athlete to play at controlled intensities. Scripting, or the process of going through an offensive or defensive play, provides the player with the opportunity to learn or familiarize themselves with team tactics while also getting on-court loads and basketball-specific movements. Starting at slower speeds in the half court, scripting can be built up to higher speeds in the full court, first with coaches and then with players (Fig. 60.4). Scripting and scrimmaging can be tailored to the load or movement needs of the player. Full court 3v3 and 5v5 provide higher external workloads than 2v2 or 4v4 [16]. Half court 2v2 and 5v5 may provide the highest accelerations per minute [16].

If accelerometer/inertial measurement units are available to help quantify intensity or play or jogging/running speeds, these may be closely prescribed by the performance team to make sure the player’s progression is gradual. If this technology is not available, these variables may have to be estimated but require close attention as

Table 60.2 Ball handling progression



Table 60.3 Shooting progression



Table 60.4 Defensive progression

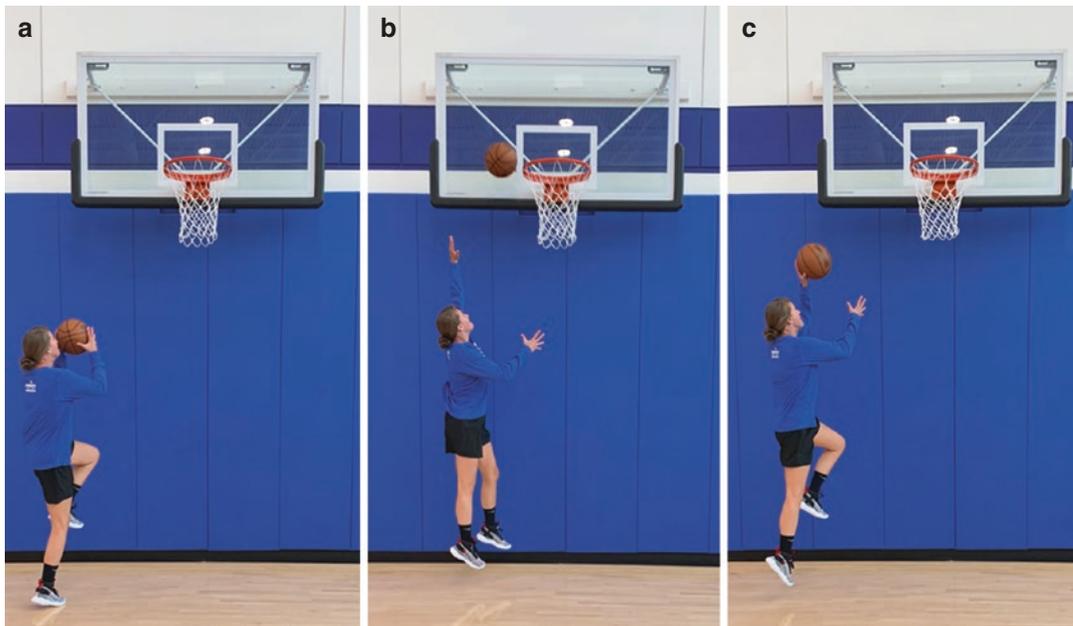
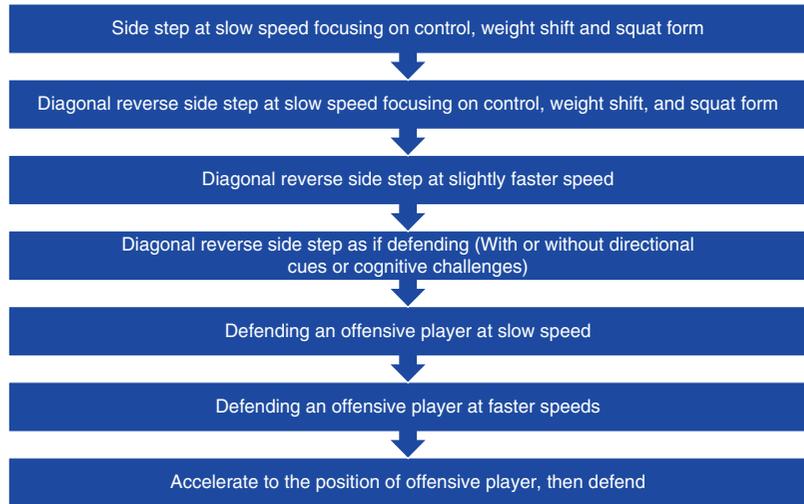


Fig. 60.3 (a) Single-leg shot from a stationary position. Shots can be taken from close under the basket and made further to increase difficulty. (b) Small jump with single-leg shot, beginning from under the basket and progressing

distance. (c) Layup. Progression can start with slow steps into layup and then adding a faster run or drive to the basket and eventually adding an opponent or tackling pad in the path of the run to the basket

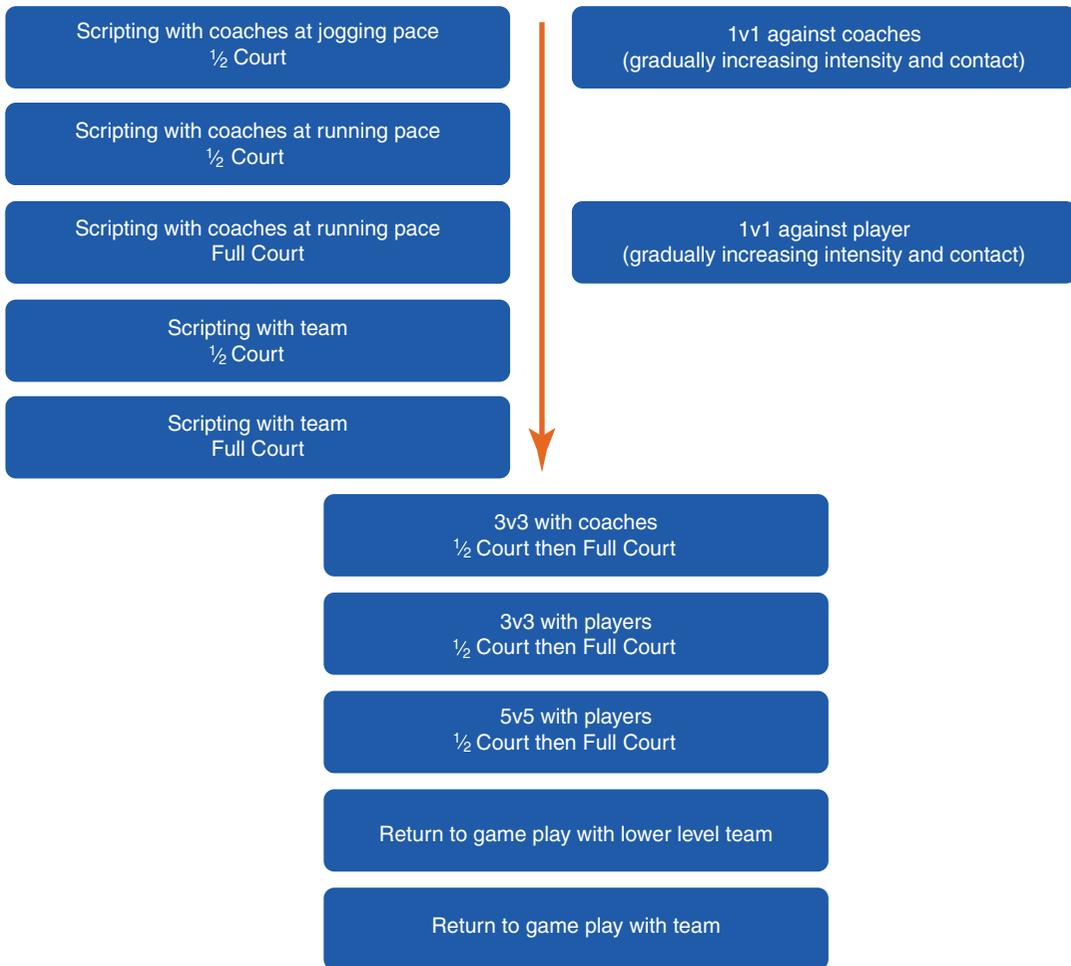


Fig. 60.4 Example of return to practice progression

coaches may not be as good at estimating speeds/intensities.

60.5 From Return to Play to Return to Performance

60.5.1 Training Under Pressure; Decision-Making, and Cognitive Load

Many athletes state that it takes them time after returning to play to get back into the “rhythm” of the game. Part of this difficulty may be because it is hard to replicate the intensity of a game in practice [17]. Even compared to 3 v. 3 or 5 v. 5 situations during practice, playing in a

game still involves greater psychological pressure (both internal and external), decision-making, and speed of play [16, 17]. Thus, a player’s first few games after returning from injury are in essence practice for subsequent games. Where possible, returning to play via lower level teams, such as playing in reserve team games, may help the athlete more quickly adjust back to their preinjury level of play. In addition, it is also important when assessing KPIs and an athlete’s performance to take into consideration changes in tactics or personnel that may have occurred while the athlete was out. These changes could require the player to learn new plays or even a new position/role when they return from injury, impacting their performance upon return.

Techniques that put the athlete under pressure during training could help athletes adjust more quickly to the faster speed of play during games. Three ways to put an athlete under pressure during an exercise include adding decision-making, cognitive load, and perturbations (Table 60.5).

There are many ways to add decision-making into an exercise. Decisions could be as simple as reacting to visual or verbal cues or as complex as anticipating or reacting to an opponent’s movements. Simple cues, such as a hand signal indicating which direction to move, can be made

more complex by reducing the amount of time the athlete has to make the decision. For example, an exercise could start with the athlete dribbling toward the basket and receiving a verbal cue to perform a layup on the right or left. To start, the athlete could be given the verbal cue as early as the free throw line, giving them plenty of time to prepare. As the drill is progressed, the verbal cue could be given later, such as when the athlete crosses the charge circle. Adding another player into an exercise can also add decision-making. Offensively, decision-making with

Table 60.5 Examples of adding decision-making, cognitive load, or perturbations to exercises

Exercise	Decision-making	Cognitive load	Perturbation
Single-leg squat	<ul style="list-style-type: none"> • Hold single-leg stance and only perform a squat when receiving a visual cue. • Watching a game, athlete performs single-leg squat when they anticipate the player they are watching to shoot. 	<ul style="list-style-type: none"> • Player must tell a story while performing squats. • Play I spy (guessing game to find what the other person is looking at). 	<ul style="list-style-type: none"> • Manual perturbation given at random to different body parts. • Move various objects of different colors through the athlete’s peripheral vision.
Free throw	<ul style="list-style-type: none"> • Tossing a tennis ball and basketball back-and-forth, shoots free throw only when received basketball. • Coach describes various end of game scenarios, player only shoots free throw when the scenario calls for a foul or shot to be made. 	<ul style="list-style-type: none"> • Must watch and commentate another game, as soon as passed a ball though they must set up and shoot the free throw. • Tell tongue-twisters while shooting (e.g., Simon sells seas shells on the sea shore). 	<ul style="list-style-type: none"> • Background noise with audience sounds and loud random noises. • Glasses with tape or different opaque covering various aspects of visual field.
Layup	<ul style="list-style-type: none"> • Clinician gives verbal cue for which side the layup is performed. • React to defender on when to start running into the layup. 	<ul style="list-style-type: none"> • Solve simple math problems. • Recite the scouting report on players on an upcoming opposing team. 	<ul style="list-style-type: none"> • Begin with a defender just gently touching the athlete as they go up for the layup, progress to fully contact. • Change takeoff or landing surface.
Defensive closeout (starting with one foot in paint, the player accelerates to 3-point line to defend an opponent)	<ul style="list-style-type: none"> • Player starts with eyes closed, opponent moves to a new spot, player opens their eyes, must find opponent and close out before their opponent can shoot. • As player closes out, they must read their opponent on whether the opponent plans to shoot or drive to the basket and adjust their body accordingly to either “fly-by” the player shooting, or try and block the player from driving to the basket. 	<ul style="list-style-type: none"> • Commentating (third person) the play. • Counting backwards from 100 as performing exercise. 	<ul style="list-style-type: none"> • Push off/contact with opponent. • Use glasses with blinders to eliminate peripheral vision.

another player could be reading the other player's actions to receive a pass or roll off of a pick. Defensively, decision-making includes reacting to the other player's movements to block or obstruct their progression. Addressing the athlete's agility in reacting to an offensive player could be important in secondary prevention of injury, as defending is a high injury risk component of basketball play [18].

Similar to decision-making, there are many, both simple and complex, ways to add cognitive load to an exercise (Table 60.5). Simple cognitive tasks such as counting or reading can be added to exercises. Progression can include making the cognitive task more difficult or making the exercise more difficult. Examples of more difficult cognitive tasks could include commentating a video, carrying on a conversation, or games like I spy or 20 questions. Further, mental fatigue can also be used to train the athlete. Mental fatigue is a psychobiological state caused by prolonged periods of cognitive activity [19]. Performing rehabilitation after taking a test, at the end of a school day, or after studying game film for a long period could all be examples of training when an athlete is mentally fatigued [19].

Perturbations in basketball can be auditory, visual, or kinesthetic. Auditory perturbations that can be used in training include music, unexpected noises, or the sound of a crowd. Visual perturbations can be created through occluding an athlete's eyesight, limiting an athlete's visual field, or creating extraneous visual information in the athlete's peripheral vision. For an example, while an athlete is shooting, the clinician could pass their hand through the athlete's peripheral vision or an object could be thrown past the athlete. Kinesthetic perturbations can include performing exercises on different surfaces such as foam pads or BOSU balls, manual resistance to the athlete, or opposition contact. Contact with another player is the most sport-specific perturbation. Manipulating the amount of contact in an exercise not only allows for physical perturbations but also challenges the athlete's decision-making. At a basic level, contact can be provided via pads or blocking devices and be progressed to contact with another player at higher levels.

60.5.2 On-Court Movement and Motor Learning/ Secondary Prevention On Court

An athlete's movement patterns can put them at risk for a reinjury, particularly when considering knee injuries [11]. High-risk movement patterns may be stiff landings (high ground reaction force, minimal knee and hip flexion) and knee valgus (hip adduction, internal rotation, and knee abduction) [11]. Generally these high-risk movement patterns are key targets of neuromuscular reeducation during rehabilitation; however, deficits may still exist upon return to play. Movement deficits still seen upon return to sport are often in higher level/more complex sport-related tasks and tasks at high speeds. Gokeler et al. [13] provide a good resource on motor learning for clinicians with the focus on changing movement patterns toward secondary prevention. The paper provides four principles:

1. Facilitate an external focus of attention. Clinicians can use external cues (focused on the environment) rather than internal cues (focused on the athlete's body or movement) to help with movement learning and automaticity [13]. For example, rather than an internal cue of "fire your glutes when you extend your leg," a clinician could use the external cue "think about pushing the ground behind you."
2. Encourage implicit learning. Explicit directions regarding movements or exercises require attention and working memory [13]. Athletes need movement patterns to be automatic when they are playing. Implicit learning strategies, such as using analogies in instruction, helps the athlete come up with their own image of what a movement should look like. Internalizing the movement could help the athlete unconsciously integrate proper movement patterns on court, particularly when attentional demands are higher [13]. Example directions to an athlete could be "I want you to think about a column holding up a building. With your shoulder, hip, knee, and foot, I want

you to build a column and keep that stable column throughout the exercise.”

3. Create environments for differential learning. Differential learning involves intentionally experimenting with multiple movement patterns and exploring the numerous ways to achieve a similar outcome [13]. Differential learning tasks could involve giving an athlete a goal and challenging them to come up with different ways to achieve that goal. For example, picking a basketball up off the floor ten different ways, none the same and each with a different starting stance. More simply, differential learning could be facilitated through performing the same task in different ways, such as a single-leg squat on the ground, on a foam pad, with manual perturbations, with or without a basketball.
4. Use self-controlled learning and contextual interference. An abundance of literature demonstrates that athletes engaged in their own rehabilitation process and decision-making tend to be more motivated and have better outcomes [20]. Self-controlled learning takes engagement in the rehab decision-making further, by giving an athlete more control over aspects such as exercise selection, duration, or feedback [13]. Actively engaging the athlete may help promote motivation which in turn may help facilitate motor learning and transfer. An example of self-controlled learning is only providing movement feedback when the athlete asks, as opposed to when the clinician deems it necessary, or letting the athlete determine when they are ready to add a basketball into an exercise. Contextual interference is defined as the interference of performing one task within the context of another [13, 21]. Where blocked practice (performing the same task in repetition) has no contextual interference, variable practice (performing variations or different tasks) has high contextual interference [13]. Although skill acquisition is slower or has poorer initial performance with higher contextual interference, there may be better retention and transfer [13, 22, 23]. For example, having an athlete in a defensive

position reacting to a player dribbling at them could be done with the eyes open, one eye closed, with glasses blocking their peripheral vision, or even with both eyes closed, forcing them to rely purely on sound.

Use of these principles throughout rehabilitation on- and off-court can be valuable for the athlete’s motor learning, but may be of particular value in integrating movement into performance.

Key Points

- A rehabilitation plan with objective milestones and return to sport criteria should be set out at the beginning of rehabilitation. Key performance indicators should also be established in order to help determine when an athlete has returned to their preinjury level of play.
- A basketball can easily be introduced early in rehabilitation exercises and as an athlete progresses, used as a complement and part of rehabilitation on- and off-court.
- Variables such as speed, intensity, cognitive load, decision-making, and perturbation that are generally used in rehabilitation can also be variables manipulated in basketball exercises, starting in a simple manner and then building complexity.
- On-court progressions should be within the confines of what is safe in each phase of the athlete’s recovery, allowing for sport- and position-specific reinforcement of new movement patterns and tissue loading introduced off court. Special attention should be placed on movements and skills that may pose a challenge to the athlete’s particular injury.

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References

- Burgi CR, Peters S, Ardern CL, et al. Which criteria are used to clear patients to return to sport after primary acl reconstruction? A scoping review. *Br J Sports Med.* 2019;53(18):1154–61.
- Barber-Westin SD, Noyes FR. Factors used to determine return to unrestricted sports activities after anterior cruciate ligament reconstruction. *Arthroscopy.* 2011;27(12):1697–705.
- Barber-Westin SD, Noyes FR. Objective criteria for return to athletics after anterior cruciate ligament reconstruction and subsequent reinjury rates: a systematic review. *Phys Sportsmed.* 2011;39(3):100–10.
- Grindem H, Snyder-Mackler L, Moksnes H, et al. Simple decision rules can reduce reinjury risk by 84% after acl reconstruction: the Delaware-Oslo acl cohort study. *Br J Sports Med.* 2016;50(13):804–8.
- Kyritsis P, Bahr R, Landreau P, et al. Likelihood of acl graft rupture: not meeting six clinical discharge criteria before return to sport is associated with a four times greater risk of rupture. *Br J Sports Med.* 2016;50(15):946–51.
- Tassignon B, Verschueren J, Delahunt E, et al. Criteria-based return to sport decision-making following lateral ankle sprain injury: a systematic review and narrative synthesis. *Sports Med.* 2019;49(4):601–19.
- Shrier I. Strategic assessment of risk and risk tolerance (startt) framework for return-to-play decision-making. *Br J Sports Med.* 2015;49(20):1311–5.
- Ardern CL, Glasgow P, Schneiders A, et al. 2016 consensus statement on return to sport from the first world congress in sports physical therapy, Bern. *Br J Sports Med.* 2016;50:853–64.
- Paterno MV, Schmitt LC, Thomas S, et al. Patient and parent perceptions of rehabilitation factors that influence outcomes after anterior cruciate ligament reconstruction and clearance to return to sport in adolescents and young adults: a qualitative investigation. *J Orthop Sports Phys Ther.* 2019;0:1–35.
- Pascual-Leone A, Nguyet D, Cohen LG, et al. Modulation of muscle responses evoked by transcranial magnetic stimulation during the acquisition of new fine motor skills. *J Neurophysiol.* 1995;74(3):1037–45.
- Paterno M, Schmitt L, Ford K, et al. Biomechanical measures during landing and postural stability predict second anterior cruciate ligament injury after anterior cruciate ligament reconstruction and return to sport. *Am J Sports Med.* 2010;38(10):1968–78.
- Vuurberg G, Hoorntje A, Wink LM, et al. Diagnosis, treatment and prevention of ankle sprains: update of an evidence-based clinical guideline. *Br J Sports Med.* 2018;52(15):956.
- Gokeler A, Neuhaus D, Benjaminse A, et al. Principles of motor learning to support neuroplasticity after acl injury: implications for optimizing performance and reducing risk of second acl injury. *Sports Med.* 2019;49(6):853–65.
- Oliveira AS, Silva PB, Lund ME, et al. Balance training enhances motor coordination during a perturbed sidestep cutting task. *J Orthop Sports Phys Ther.* 2017;47(11):853–62.
- Mair SD, Seaber AV, Glisson RR, et al. The role of fatigue in susceptibility to acute muscle strain injury. *Am J Sports Med.* 1996;24(2):137–43.
- Schelling X, Torres L. Accelerometer load profiles for basketball-specific drills in elite players. *J Sport Sci Med.* 2016;15(4):585.
- Montgomery PG, Pyne DB, Minahan CL. The physical and physiological demands of basketball training and competition. *Int J Sports Phys Perf.* 2010;5(1):75–86.
- Monfort SM, Comstock RD, Collins CL, et al. Association between ball-handling versus defending actions and acute noncontact lower extremity injuries in high school basketball and soccer. *Am J Sports Med.* 2015;43(4):802–7.
- Van Cutsem J, Marcora S, De Pauw K, et al. The effects of mental fatigue on physical performance: a systematic review. *Sports Med.* 2017;47(8):1569–88.
- Ardern CL, Taylor NF, Feller JA, et al. A systematic review of the psychological factors associated with returning to sport following injury. *Br J Sports Med.* 2012;47(17):1120–6.
- Magill RA, Hall KG. A review of the contextual interference effect in motor skill acquisition. *Hum Mov Sci.* 1990;9(3):241–89.
- Bjork EL, Bjork RA. Making things hard on yourself, but in a good way: creating desirable difficulties to enhance learning. *Psychology and the real world: Essays illustrating fundamental contributions to society.* 2011;2:59–68.
- Porter JM, Magill RA. Systematically increasing contextual interference is beneficial for learning sport skills. *J Sports Sci.* 2010;28(12):1277–85.



Strength Training for Basketball

61

A Methodological Framework Based on Basketball and Player's Needs

Lorena Torres Ronda and Francesco Cuzzolin

61.1 Introduction

Strength training programs oriented according to specificity for team sports are something relatively new. Traditionally, team sports have extrapolated and adapted knowledge and training procedures from individual sports, with an extensive history in training theories and methodologies. However, nowadays, it is widely accepted that the needs are completely different and specific to each discipline, even within the team sports themselves. Accordingly, the authors of this chapter believe that the main goal of a strength program, and especially at high-performance level, is to provide the optimal conditions to allow the players to achieve, maintain, or enhance the proper performance of functional movements significant for the sport, in this particular case, for basketball.

It is quite common to find methodologies based on the functionality of the muscles (or its

properties), organizing the contents around them (e.g., main body area, muscle adaptations, typology of contraction, and mono- or polyarticular), or based on designing attractive exercises for the executor, instead of programs under a philosophical or methodological framework with the sport characteristics as the core focus from which the rest of contents are developed. Moreover, when deciding on the most appropriate resistance methods, the best equipment or the exercises to perform in the program, it is important to consider some other components, including body positions, movement directions, coordination, or the period of the season. The proposal we present here has the sport of basketball as a focal point, with an integrative methodology based on basketball movements and players' needs.

61.2 Basketball Overview

Basketball is a team sport that requires repeated short high-intensity actions (e.g., accelerations, decelerations, jumps, and alike), where the players either have to outwit opponents, and even teammates, with cuts or side-steps, changing direction rapidly to avoid contact, looking for or responding to unexpected contacts, performing specific basketball movements (e.g., defense actions, screening, blocking, rebounding, dribbling, and post-up). These actions are interposed by periods of low-to-mid intensity activities (e.g., walk, jog, and run), and playing with a ball cre-

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ates impulsive, nonlinear, and unpredictable movements and actions. Also, the ability to repeat these activities determines effective performance, which requires the ability to perform the actions per se and the endurance to do so constantly; thus, the players must not only *be strong*, but fast, balanced, coordinated, agile, perform sport-specific skills, have the capacity to perform high-intensity actions repeatedly, with tactical intelligence, among other qualities.

A competitive season can be very different depending on the league (e.g., EuroLeague, National Basketball Association (NBA), or National Collegiate Athletic Association (NCAA) basketball). In the NCAA teams play 25–35 games in 5 months; for an EuroLeague team the number of games range from 73 to 90 in 8 months; and an NBA team plays 82 games during the regular season (6 months), and this does not include play-off games. Regardless of the competition, it is noticeable that there is a high density of games for those periods of times. In addition, some players can also be part of Summer Leagues, National Teams, or pre-season tournaments, usually conducted during the “off season.” Being able to keep a proper and optimal level of strength for these long periods is a challenging goal for the performance staff and the players. When designing the strength training programs, aspects such as the period of the season (pre-season, in-season, off-season), the density of games per week, or the minutes played should be considered.

61.3 The Basic Concepts of Strength

Despite the emphasis we place on the need to consider the specificity of sport, we wanted to review some general and basic concepts related to strength training. It is beyond the scope of this text to make an exhaustive description of these concepts, or the physiological mechanisms and the metabolic demands of strength training, which are widely described in other manuscripts in the literature, but to refresh some of those that we consider most significant. It is important for the authors of this chapter because we want to stress the need to know *Why* we

make the decisions we make, and it will serve as the basis for future steps when designing the programs.

61.3.1 Strength, Rate of Force Development, and Mechanical Power

Basketball activities require an optimal expression of strength and neuromuscular power. The primary components that contribute to them are as follows:

- Maximal dynamic strength (MDS)
- Rate of force development (RFD)
- High-velocity movements (HV)
- Stretch-shortening cycle capabilities (SSC)
- Neuromuscular skill and coordination (NSC)

These components serve as a reference when planning the strength and neuromuscular goals of the programs.

- *MDS*: is the ability to develop high levels of force. This quality is required at the initiation of the movement to overcome inertia/resistance when the system velocity is zero or slow. For activities such as jumps, changes of direction, or accelerations, even in the absence of external resistance, there is a significant inertial component due to the athlete’s own body mass. Thus, maximum strength relative to body mass is a key element in expression of power for gross motor actions involved in a variety of athletic movements. Moreover, in contact sports the ability to generate neuromuscular power against an external resistance is an important factor, which relies heavily upon high levels of force output and therefore maximum strength.
- *RFD*: is the ability of the neuromuscular system to increase contractile force from a low or resting level, when muscle activation is intended to be performed as quickly as possible ($RFD = \Delta\text{Force}/\Delta\text{Time}$) [1]. RFD is considered important for movements performed rapidly, when force production is short (100–300 ms). It is well accepted that performance of high-intensity activities, like many actions

performed by basketball players, is largely determined by mechanical power. Mechanical power is defined as the rate at which force is developed over a range of motion (d), in a specific period of time ($P = F \times d/t$), or as force multiplied by velocity ($P = F \times V$), which means, power is the product of force and velocity. The velocity reached when moving an external load (or body weight) is a consequence of the force applied; therefore, the way to develop more speed at a certain load (or body weight) is to apply more force to the load [2, 3].

- *HV movements*: is the ability to exert force at high velocities. Increasing maximum strength at slow movement velocity is of limited relevance if the player is unable to express this greater force-generating capacity at the movement velocities encountered during athletic and sport skill movements.
- *SSC*: involves a pre-stretch (eccentric) phase (e.g., countermovement and landing), followed by a concentric/ballistic phase. A great number of movements and activities in sport involve some kind of pre-stretch or countermovement, which serves to increase force and power output for the concentric portion of the movement [3]. Based on the duration of the ground contact or force application, the SSC capabilities can be divided into the following:
 - *Fast SSC*: it involves a preceding flight phase with a very brief (100–200 ms) ground contact phase (e.g., drop jump)
 - *Slow SSC*: it involves a preparatory eccentric phase (lasting about 300–500 ms) performed on a fixed base of support (e.g., countermovement jump)
- *NSC*: is the ability to coordinate the sequential motion of joints and limbs, between the multiple linked segments of the kinetic chain involved in a specific movement. The training stimulus must feature a high degree of specificity (movement pattern) and at competition's velocity. Intermuscular coordination is a critical factor for the motor abilities involved in athletic movements such as running or jumping, and especially for fine or complex motor abilities involved with particular skill movements [3].

On the basis of these components, we propose the following strength goal categories, mainly oriented to achieve, maintain, or foster: (i) maximal dynamic strength (structural or neural), (ii) optimal mechanical power, and (iii) velocity and/or movement speed (exploiting the neuromuscular properties using the eccentric-to-concentric stretch-shortening cycle—SSC, elastic (slow: 300–500 ms) or reactive (fast: 100–200 ms); includes plyometrics). An additional goal, (iv) endurance or maintenance, has neither a force-velocity nor a mechanical power component, but involves correcting or adjusting muscular imbalances, muscle or movement deficiencies, and/or weaknesses, with a more prophylactic and tissue preparation focus for future high-intensity actions.

61.4 A Methodological Framework for Basketball

For some time now, team sports have evolved the theory of training aiming to propose a framework more in line with the needs of these sports [4]. However, the explosion of innovative methodologies has not occurred until more recently, due to the inclusion of the new paradigm of the dynamic systems theories [5], incorporating two fundamental concepts: (i) specific training of sports demands produces greater improvements in performance, and therefore drills and movements in (team) sports should replicate game conditions allowing to enhance maximal transfer to skilled performance, and (ii) the constraints associated with human movement (and skill acquisition) can be classified into three components: the individual, the task, and the environment [6]. Under this vision, Seirul-lo et al. proposed the idea of structured training theory, which begins with the classification of exercises at different levels of specificity, according to the orientation and level of approach to the competition. More recently, Schelling and Torres-Ronda presented a personal adaptation of this philosophy specific for basketball [7].

The proposal presented in this chapter follows the guidelines of the abovementioned works where the program structure is based on the specific characteristics of the sport, levels of specificity, or progressions according to task orientation and player's needs.

61.4.1 Levels of Specificity

The similarity with the actual competition/official games grades the levels of specificity:

- **Game performance—on court—(L5):** this level requires the drills to include the sport tactics, skills, and physical capabilities according to teammates and opponent's reactions in a competitive environment. Level 5 ranges from official games to scrimmages, 5on5, and 4on4 games with modified rules.
- **Basketball-oriented practice—on court—(L4):** this level promotes the development of basketball skills reaching levels of automatization or execution at a stage that allows optimal performance, from no-opposition (e.g., 1on0) to certain level of complexity (e.g., 3on3).
- **Basketball skills—on court—(L3):** this level implies abilities applied with specific components of the game, covering individual skills development, which might include tasks under constraints that favor decision-making.
- **General abilities—on/off court—(L1 and L2):** events combined for a specific goal, which include the following:
 - **Actions:** movements performed in sequence or simultaneously
 - **Movements:** basic movement patterns

61.4.2 Movement Content

Basketball coaches tend to divide basketball movements into two categories, offensive and defensive, each possessing certain particularities (Fig. 61.1). Understanding this categorization in the design of actions or movements will help facilitate the choice of exercises. The actions included in these categories, leaving aside skills such as passing or shooting, can be grouped into three generic movement contents: (i) moves, (ii) jumps, and (iii) contacts. Likewise, movements in basketball can be described starting from a power position, a stagger position, or a transition to one leg.

Figure 61.1 shows the most common abilities (i.e., actions and movements) and actions for offensive and defensive movements. Most abilities involve a combination of actions, such as move and jump, move and contact, or jump and contact, with constant changes of direction, and only rarely requiring linear or unidirectional movements.

61.5 Designing the Program

When designing the program, we suggest following these steps:

- **Assessing players' characteristics, needs, and flags:** the first thing to do is determining the player's characteristics and profile, including, but not limited to, the following: age, anthropometry, body composition, range of motion (ROM), basic movement pattern forms (bi- and unilateral), strength and neuromuscular power profile (and strength training history), cardiovascular and recovery shape, previous injury history, asymmetries, and deficiencies. It is also advisable to consider the specific on-court movement needs, under the direction of coaches, as well as the following characteristics:
 - *Designation or playing position:* small or big, and/or guard, wing, or center
 - *Status:* development player, veteran
 - *Role or rotation:* first (starters), second, or third unit
 - *Minutes played:* individual minutes played, or players clustered by minutes played
- **Moment of the season:** For the purpose of this chapter two periods are discussed: in-season (competitive season) and off-season (recovery from previous season and/or preparing for next season; might include other competitions and/or playing commitments).
- **Levels of specificity, strength needs, and movement content**
 - Once the information required in the previous sections has been collected, we can begin to design the programs and their contents. At this stage, we suggest considering: (i) the lev-

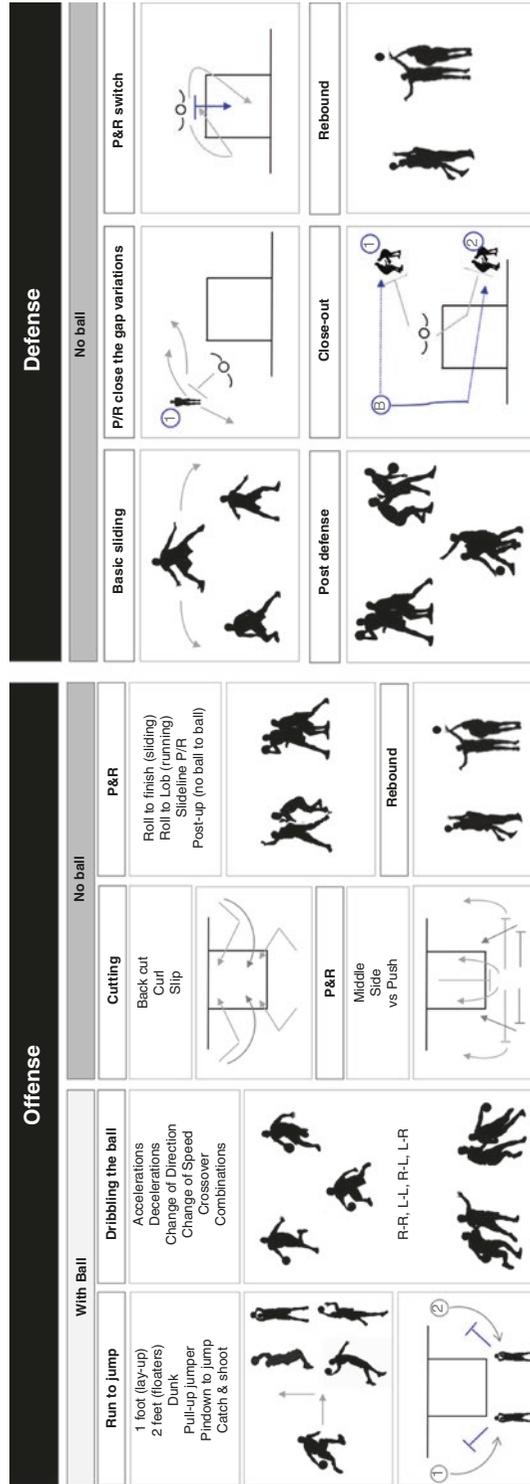


Fig. 61.1 Example of a generic categorization of offense and defense movements in basketball

els of specificity [7], (ii) the strength needs, (iii) the abilities, actions, and movements, and (iv) the typology of exercises and its combinations.

- **L1 and L2:** it is necessary to work toward an optimal activation of the muscles and execution of basic movement patterns. These levels include proprioception and balance, neuromuscular control, dynamic flexibility, ROM, static and dynamic muscle exercises, addressing imbalances and asymmetries. In addition, contents should ensure a proper execution of exercises with regard to ankle–knee–hip–trunk motions in different planes and axes. Once minimums are guaranteed, progress must be made toward actions and abilities closely related to basketball contents and movements at specific (higher) speeds.

The resistance training goals in this level will depend on the players' needs ranging from endurance/maintenance to maximal dynamic strength, from optimal neuromuscular power to velocity and/or movement speed (SSC elastic, reactive). There is also the possibility to combine different strength goals, depending on a specific need for different, abilities, movements, and/or actions.

- **L2 and L3:** the contents are sport-specific. The focus of sport-specific training is “transfer” to sport movements; therefore, the abilities, actions, and movements in these levels should replicate specific movement patterns and skills. Emphasis is placed on speed-agility, quickness, ballistic and plyometric training, coordination, and locomotion. L2 is performed between the weight room and the court, while L3 implies movements on the court, which are basketball-specific (see Fig. 61.1 for offense and defense abilities, movements, and actions). It is recommended not only to perform the perfect movement patterns (proper form), but exploring its limits too, introducing perturbations implied in the specific context of the sport.

The resistance training goals are therefore focused on velocity and/or movement speed (SSC elastic, reactive), and neuromuscular skills. Minimize the impact of potential asymmetries due to the specificity (e.g., drills performed full court, at game pace) and the competition, as well as injury reduction orientation contents should be guaranteed in these levels too.

- **L4 and L5:** the main goal is to improve game performance, based on small-sided games and scrimmage. These stages are usually run by the coaching staff. A priori, the practice is not designed to specifically work on strength, neuromuscular power, speed, and so on; however, the inherent nature of these tasks imply that a neuromuscular load occurs. On the one hand, this will serve to improve or maintain the required levels of these abilities, and on the other hand, it will be critical to monitor the loads to adjust the rest of the physical work contents.

Strength training and injury reduction contents pursue the objective to enhance the quality of athletic movements, especially if specific transfer is expected to occur. Hence, strength and injury reduction training is a must to account for the complexity of the contextual movement patterns. Oversimplification of such patterns in strength training, such as training muscles in isolation or isolated of joint ranges of motion, is unlikely to be of much use [8], unless there is a need to address a specific deficiency during a certain period of time. Injury reduction training can also be directed toward dynamic stabilization and might allow exploration of movement variables related to the specific gesture.

Finally, it is important to remember that what works for one individual may not work for another (individualization), and what works for an individual today may not work in the same way for the same individual a year from now (periodization), particularly in experienced athletes [8].

61.6 Designing the Session

In a multidisciplinary and integrative program, the strength session incorporates elements from different areas of the program, including strength–power–speed goals (based on proper assessments), injury prevention/reduction goals (which might include recommendations coming from the medical staff), or basketball movement needs (ideally discussed with coaches).

Greater levels of potentiation are generally observed with multiple sets of moderate- to high-intensity exercises interposed with low- to moderate-intensity exercises, allowing enough time for recovery from the primary exercise [9], also known as complex training (combination of two or more exercises in supersets). Thus, we propose to organize the contents of the session in sequences of exercises, according to specificity, stimulus requirements, and physiological responses, which at the same time provides a time-efficient workout. These sequences of exercises, or circuits, include two types of exercises: (i) primary and (ii) secondary exercises (compensatory or complementary to the primary exercise) [7]. The ratio between primary and secondary exercises will depend on the desired net balance between fatigue and potentiation. The balance required is based on physical and physiological responses, recovery intervals between primary exercises, or qualities such as post-activation potentiation (PAP) (Fig. 61.2). Sequences with shorter rest intervals (e.g., 30–60 s) produce a greater fatigue than the sequences with longer rest intervals [10]. For this reason, a period from 90 s to 2 min between primary exercises might ensure a proper recover. Although, the ideal rest period between sets for multi-joint and single-joint exercise is still controversial, the main responses from strength training, metabolic and neural, have to be kept clearly separated to better understand the associated phenomena as well as the pursuit of the optimal rest period length to achieve different training goals. Regarding the number of repetitions, there is evidence that the specific number of repetitions (e.g., 3–5 vs. 8–10) or time-under-load (e.g., 30 s vs. 90 s) impacts athlete strength/optimal mechanical power [1, 11].

The sequence of the primary exercises in a circuit can be based on the basketball movement categories (i.e., offense or defense actions), or the generic movement contents (i.e., moves, jumps, and contacts), and a specificity-based progression, from general movement patterns (e.g., L1: squat and lunges) to more specific abilities and actions (e.g., L2: side-steps, jumps, and CoD drills) [7] (Fig. 61.2b).

During the competitive period, the number of strength sessions per week will vary depending on multiple factors, including player's age, role, minutes played, number of games per week, back-to-back games, and others. A strength stimulus performed twice a week may allow for maintaining levels of strength [12], but it is important to consider the factors around a player. For example, even one session a week (every 5–7 days) for a basketball player who is playing high minutes per game in a congested schedule could be enough to prevent decreases in strength levels. Despite a lack of time, to keep the continuity of training and to avoid unnecessary acute fatigue, it is advantageous to design sessions that last 20–30 min. Under this scenario, when only one or two strength sessions per week can be guaranteed, we highly recommend designing a program for strength maintenance that can be performed daily, before practices or games, as a preparation for both motor control activation and psychological purposes.

The strength training workout is rarely a standalone practice, and it is often combined with individual workouts such as shooting, individual skills, or team practice sessions. Strength training can be performed before the basketball practice (skills, shooting, or team practice), where it can favor the nervous stimulus activation to create benefits for technical coordination. Strength training can also be performed after the basketball practice (if the priority is the development of skills on the court or avoid situations of pre-fatigue), or even before or after games. The time of day to perform the strength session might also depend on other factors including the way the basketball organization operates, the culture of the team, or players and/or coaches' preferences.

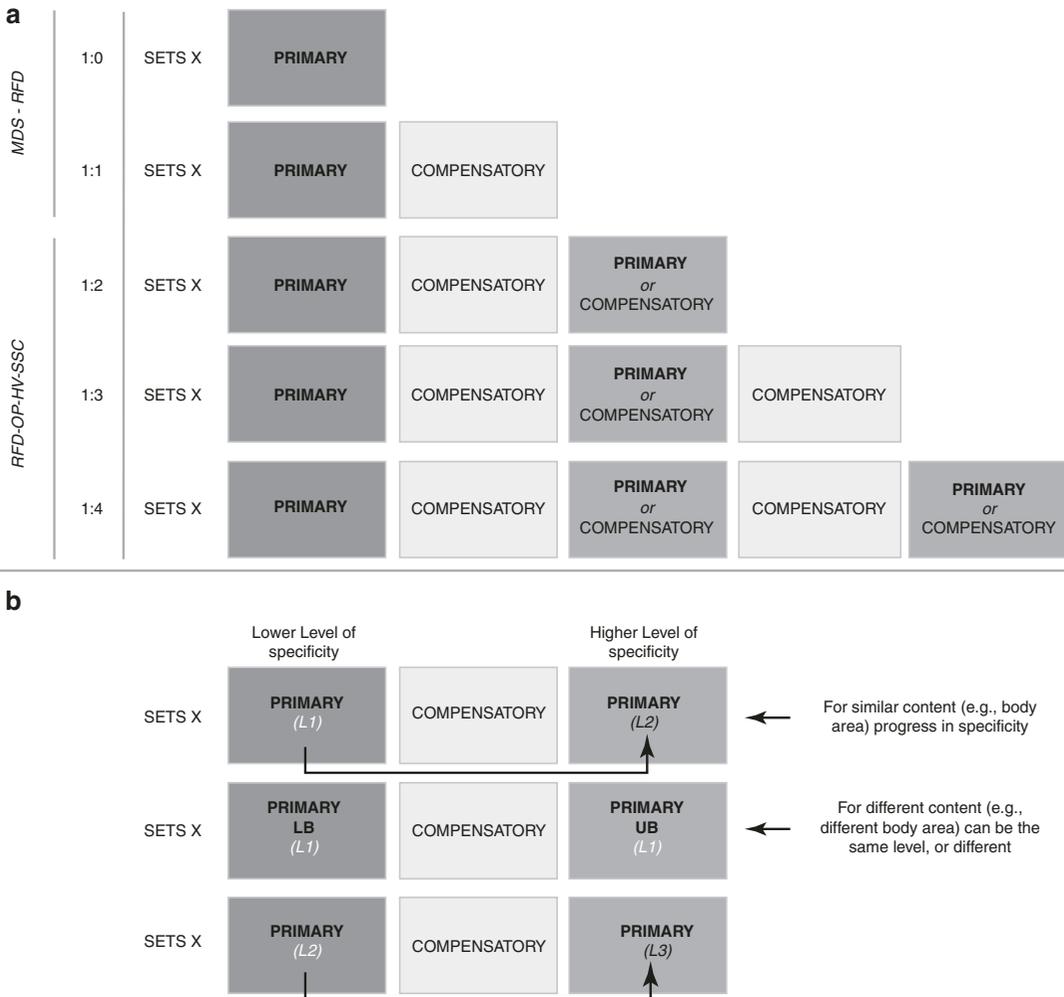


Fig. 61.2 (a) Work-to-rest ratio between potentiation and fatigue when using primary and secondary (complementary/compensatory) exercises, and the predominantly expressions of strength associated to them. (b) Examples of sequences of primary and secondary exer-

cises and progressions of specificity. *MDS* maximal dynamic strength, *RFD* rate of force development, *OP* optimal mechanical power, *HV* high-velocity movements, *SSC* stretch-shortening cycle capabilities, *LB* lower body, *UB* upper body

61.7 Defining the Exercises: Typology, Parameters, and Its Combinations

Most on-court movements (see Fig. 61.1) require players to produce force under different conditions; unilaterally in unpredictable and variable contexts with emphasis on the application of multi-vector forces (anterior/lateral/posterior) and multidirectional movements [13].

When designing the exercises, strength coaches should consider what influences the on-court movements:

- the environment: e.g., the size of resistance, more or less stable, and/or the direction and stability of the surface.
- the movement (task): e.g., movement patterns, body area, laterality, planes and axes, difficulty, type of resistance, and/or enforcement.

- the individual performing the movement: e.g., different levels of fatigue and neuromuscular control.

The authors of this chapter propose to design and/or select the exercises defined by the parameters laid out in Fig. 61.3.

Following the principle of specificity and considering the strength goals and the players' needs, we can design myriads of exercises to reach the optimal movement performance. In addition to the exercises following the conventional paradigm (e.g., classified by the body area, performing repetitive concentric, bilateral, vertical/horizontal, and controlled movements), which will be appropriate and convenient at certain times, the inclusion of exercises containing unilateral stances and movements, executed in different planes and axes, under some degree of uncertainty (i.e., perturbations), with eccentric emphasis [13], would be desired.

It has been proposed that the similarity of the movement with the sporting movements (i.e.,

basketball movements performed during competition), and therefore the value of the specificity of the strength exercise can be determined by analyzing five categories of specificity [8]:

- Similarity in the inner structure of the movement: (i) intramuscular (coordination within a muscle), and (ii) intermuscular (cooperation between different movements)
- Similarity in the outer structure of the movement (similar execution of joints)
- Similarity in the energy production
- Similarity in sensory patterns: (i) when monitoring the environment, and (ii) when monitoring the body (proprioception)
- Similarity in the intention of the movement

Exercises will not always be similar to the sporting movements in every category, but these categories can serve as a reference for progressions when designing or selecting exercises, and for the selection of the most appropriate exercises for each moment and need.

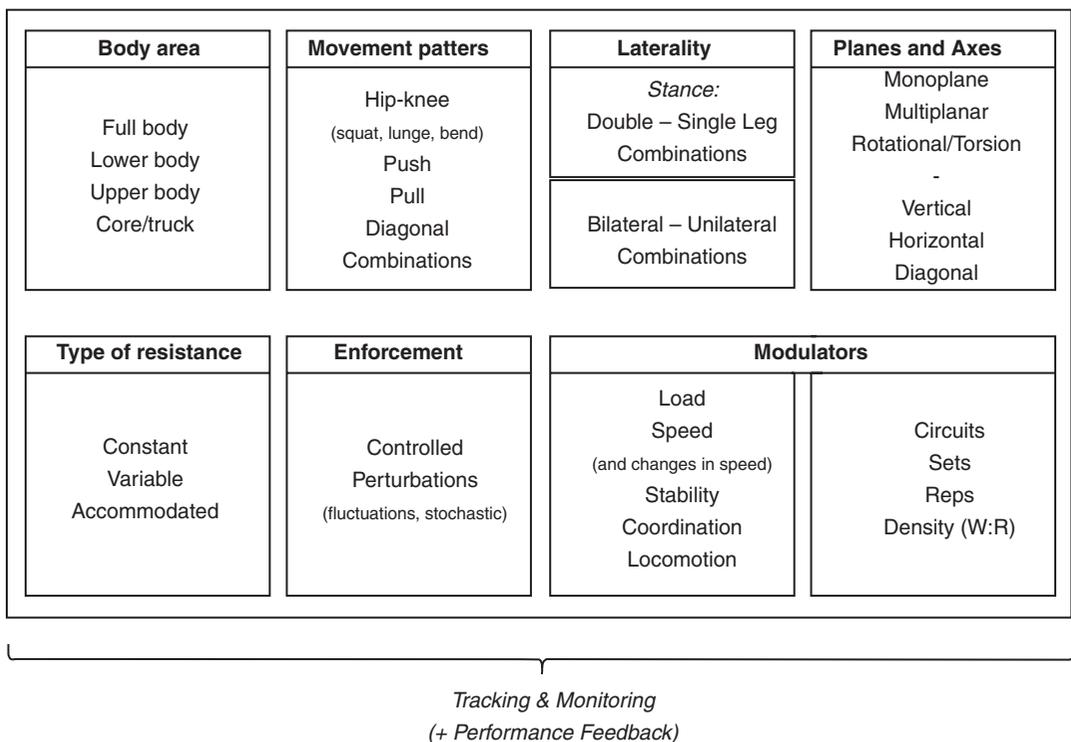


Fig. 61.3 Parameters to consider when classifying, selecting, or designing the exercises

Table 61.1 Examples of fundamental exercises according to the body area, the movement category, and laterality, performed with body weight, bands cables, barbells, and dumbbells

Exercise type	Movement category	Bilateral		Unilateral	
Full body	Hybrid	Snatch, clean, jerk, hang pull	<i>BB</i>	Snatch, clean	<i>DB</i>
Lower body	Knee dominant	Back squat, front squat	<i>DB/BB</i>	Single leg squat, Bulgarians	<i>DB/BB</i>
	Hip dominant	Deadlift, Romanian deadlift, good morning	<i>DB/BB</i>	Step-up, lunge, split squat	<i>DB/BB</i>
Upper body	Vertical push	Standing press	<i>DB/BB/cable</i>	Single arm press	<i>DB/cable</i>
	Vertical pull	Chin-ups, pull-ups		Single arm pulls	<i>DB/cable</i>
	Horizontal push	Bench press flat, bench incline press, push-ups, standing press	<i>DB/BB/cable</i>	Single arm: bench press flat, bench incline press, standing press	<i>DB/cable</i>
	Horizontal pull	Row (kneeling, seated, standing), lying row, reverse/inverted row, pullover, pull-down	<i>DB/BB/cable</i>	Single arm: row (kneeling, seated, standing), one-arm rotational row	<i>DB/cable</i>
Core	Abdominal dominant	Sit-ups, dead bug		Sit-ups	
	Flexion/extension	Planks, sit-ups		Lateral planks	
	Rotational (anti-rotation)	Landmine, plate series		Palof variations, chop variations, landmine	<i>Cable</i>

DB dumbbell, *BB* barbell

Furthermore, we have to contemplate the type of resistance to maximize the objectives of the exercise. Resistance can be classified into the following three different categories [14]:

- Constant: the resistance is mainly influenced by gravity (e.g., free weights)
- Accommodating: the resistance changes depending on the position within the ROM (e.g., rubber bands, pneumatic resistance, or cam lever-based equipment)
- Variable: the resistance is dependent on velocity, force/torque, or acceleration (e.g., isokinetic or robotic engine)

Considering the evident need of neuromuscular power, coordination, and stability, equipment such as dumbbells, barbells, kettlebells, bands, medicine balls, slam balls, suspension training kits, hydro balls and aqua bags, cables and cross cables, jump boxes, and eccentric overload-multidirectional training equipment are important pieces for a complete training experience (Table 61.1).

Fact Boxes

- The main goal of a strength program, and especially at a high-performance level, is to provide the optimal conditions to allow the players to achieve, maintain, or enhance the proper performance of functional movements significant for basketball.
- Basketball coaches tend to divide basketball movements into two categories, offense and defense, each of them possessing certain particularities. Understanding these abilities and actions will facilitate in designing of tasks and the choice of exercises.
- A program that works for one individual might not work for another, and what works for an individual today might not work in the same way a year from now, particularly in experienced athletes.

Take Home Message

- Specific training of sports demands produces greater improvements in performance, and therefore drills and movements in (team) sports should replicate game conditions allowing to enhance maximal transfer to skilled performance.
- A strength program should be designed based on the player's characteristics, profile and needs, as well as the levels of specificity, the strength goals, the abilities-actions-movements required in basketball, and other parameters related to the typology of exercises and its combinations.
- Designing the sessions according to sequences of exercises, or circuits, including and combining primary and secondary exercises (compensatory or complementary to the primary exercise) provides a time-efficient workout.
- When designing the exercises, strength coaches should consider what influences the on-field movements, the constraints associated with human movement (i.e., the task, the environment, and the individual) follow the principle of specificity, consider the strength goals and the players' needs.

References

1. Gonzalez-Badillo JJ, Ribas J. Fundamentos del entrenamiento de fuerza. INDE: Barcelona; 1999.
2. Cormie P, McGuigan MR, Newton RU. Developing maximal neuromuscular power. Part 1—biological basis of maximal power production. *Sports Med.* 2011;41:17–38.
3. Cormie P, McGuigan MR, Newton RU. Developing maximal neuromuscular power. Part 2—training considerations for improving maximal power production. *Sports Med.* 2011;41:125–46.
4. Seirul-lo F. Entrenamiento de la fuerza en balonmano. *Rev Entrenamiento Deportivo.* 1990;IV(6):30–4.
5. Davids K, Araújo D, Shuttleworth R, Button C. Acquiring skill in sport: a constraints led perspective. *Int J Comput Sci Sport.* 2003;2(2):31–9.
6. Newell KM. Change in movement and skill: learning, retention, and transfer. In: Latash M, Turvey M, editors. *Dexterity and its development.* Hillsdale, NJ: Erlbaum; 1996.
7. Schelling X, Torres-Ronda L. An integrative approach to strength and neuromuscular power training for basketball. *Strength Cond J.* 2016;38:72–80.
8. Bosh F. *Strength training and coordination: an integrative approach.* Rotterdam: Ten Brink; 2010 Publishers; 2015.
9. Seitz LB, Haff GG. Factors modulating post-activation potentiation of jump, sprint, throw, and upper-body ballistic performances: a systematic review with meta-analysis. *Sports Med.* 2015;46(2):231–40.
10. Lemos S, et al. Effects of strength training sessions performed with different exercise orders and intervals on blood pressure and heart rate variability. *Int J Exerc Sci.* 2018;11(2):p55.
11. Carpinelli RN, Otto RM, Winett RNA. Critical analysis of the ACSM position stand on resistance training: insufficient evidence to support recommended training protocols. *J Exerc Physiol.* 2004;7(3):1–60.
12. Baechle TR, Earle RW, Wathen D. Resistance training. In: *Essentials of strength training and conditioning.* 2nd ed. Champaign, IL: Human Kinetics; 2000. p. 395–426.
13. Gonzalo-Skok O, Tous-Fajardo J, Valero-Campo C, Berzosa C, Bataller AV, Arjol-Serrano JL, Moras G, Mendez-Villanueva A. Eccentric overload training in team-sports functional performance: constant bilateral vertical vs. variable unilateral multidirectional movements. *Int J Sports Physiol Perform.* 2017;12(7):951–8.
14. Frost DM, Cronin J, Newton RU. A biomechanical evaluation of resistance: fundamental concepts for training and sports performance. *Sports Med.* 2010;40(4):303–26.

Training and Performance Differences Between the NBA and FIBA Rules and Major Competition Aspects (Euro, WC, and Olympics)

Francesco Cuzzolin

62.1 Introduction

Periodization for team sports is somewhat a process of reverse engineering that starts considering different factors such as the following [1]:

1. amount and density of games already planned for the competition;
2. length of the competition, from just few weeks like during national team activities, to 8–9 months, like for some of the main domestic leagues;
3. how much time and the number of practices that can be used before the beginning of the competition, if the number of weeks for the training camp can be freely decided from the coaching staff or there are, like in NBA or NCAA, restrictions;
4. players' trainability;
5. team history; player's experience with each other, experience of the coaching staff, and team's experience in the competition.

These are just the main variables to take into consideration, and the aim of this chapter is not to be exhaustive on this topic but to provide as much information possible to clarify the complexity

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and sometime the amount of compromises that has to be taken from the coaching staff.

62.2 Game Rules

The International Basketball Federation (FIBA) is an association of national organizations which governs the sport of basketball worldwide. FIBA defines the rules of basketball, specifies the equipment and facilities required, organizes international competitions, regulates the transfer of athletes across countries, and controls the appointment of international referees. A total of 213 national federations are now members, organized since 1989 into five zones: Africa, Americas, Asia, Europe, and Oceania. FIBA organizes all the competitions between national teams, including the World Cup and Olympics.

According to the FIBA rules, the game of basketball is played with two teams of 12 players, with a maximum of 5 players of each team on the court at any time. Teams may make as many substitutions as they like. Games are divided into two halves, each half consisting of 10-min quarters, with a 2-min break between the quarters and a 15-min at the half time. The clock runs whenever the ball is in play.

The clock is stopped whenever the ball goes out of bounds, a foul is called, free throws are being shot, and during time outs. When the ball is inbound, the clock starts once a player touches

the ball. In contrast, NBA games consist of four 12-min quarters, making the total game longer by 8 min. NCAA college basketball changed the rules in 2015–2016 from 10-min quarters to 20-min halves [2], and in order to speed up the game and to prevent teams from stalling, a shot clock was added. The shot clock determines how long you have to shoot the ball. If the ball changes possession or hits the rim of the basket, the shot clock starts over. The length of the shot clock is different for different basketball leagues; the NBA has had a 24-s limit since 1954, FIBA switched from 30 to 24 s in 2000. The Women’s National Basketball Association (WNBA) switched from 30 to 24 s in 2006. Only NCAA basketball has retained the 30-s shot clock since the 1970–1971 season for women and the 2015–2016 season for men [3].

62.3 European Leagues

In Europe, there are some countries where teams play domestic leagues, like Spain, France, Italy, Germany, Greek, Israel, and Turkey, and in others where some teams prefer to compete in regional leagues. The two regional leagues are the ABA League (Adriatic Basketball Association) with teams from Slovenia, Croatia, Serbia, Montenegro, Bosnia and Herzegovina, and the VTB United League for mainly Russian teams and few others from Kazakhstan, Estonia, Latvia, Poland, and Belorussia.

The EuroLeague, involving 18 top teams in Europe, can be considered a real European Championship for clubs. There are 11 top teams that have been chosen as licensed clubs, long term licenses; 5 spots are given to associated clubs with an annual licence (for Adriatic, Germany, VTB United League, Spain and Eurocup Champion); 2 for two-years wild cards.

FIBA’s Basketball Champions League, which began in 2016, is a competition at rival level with the EuroLeague. Initially, FIBA had intended to take over the running of the EuroLeague, but this was rejected by the clubs involved, so FIBA began its own rival competition. The Champions League involves 32 teams each year; to date clubs from over 20 countries have taken part.

There are two more competitions in Europe to consider, the Euro Cup and the FIBA Europe Cup, widely regarded as the third- and fourth-tier European professional basketball competition for clubs. Thus, the competition demand for a European team can be very different, considering, not just the amount of games (if a team is participating in EuroLeague in addition to their domestic league), but also the density of games. Further, the schedule can change such as during League Cup or Playoffs. This is why the amount of time spent in traveling, quality of traveling, if the team that is traveling commercial is having exit row seats and priority lanes or better if the team is flying business or with a charter flight, can add or take away a huge amount of time that could be spent in training the teams for a proper and consistent performance or to apply all the most beneficial procedures to improve their quality of recover and probably to reduce the amount of injuries which are afflicting basketball at this level.

Without reaching any final, the team in Table 62.1 has played 80 official games in about 9.5 months, and if we consider that some of their players play also for their national team, this number can easily increase.

Like we can see the number of games per week change considering the different moment of the year and from the results obtained. This is why for a basketball team, a nonlinear training periodization, which consists of varying patterns

Table 62.1 Example of season schedule for a EuroLeague team playing Italian league

Phase	Duration	Games played	Games per week	Average games per week
Preseason	7 weeks	11	2/4 weeks–3/1 week	1.5
Competitive season	8 months	69	1/11 weeks–2/15 weeks–3/8 weeks–4/1 week	1.9
Playoffs	3 weeks	8	1/1 week–3/1 week–4/1 week	2.6
Off-season	2.5 months	–	–	–

of training, involving exercise selection, volume, and intensity, can be considered more appropriate during the competitive season [4]. Training load management and an effective recovery planning in addition to a proper distribution of minutes played between players are fundamental goals for the whole coaching and the physical/medical staff to prepare the teams to be ready for this competitive demand.

62.4 National Basketball Association (NBA)

The NBA is a men's professional basketball league in North America, composed of 30 teams (29 in the United States and 1 in Canada), and it is widely considered the most spectacular professional basketball league in the world. Its organization to cover such a great number of games in spite of the distances between teams and traveling so frequently in different time zones is very impressive. The NBA's regular season runs from October to April, with each team playing 82 games, its playoffs extend into June. The NBA is an active member of USA Basketball (USAB), which is recognized by FIBA as the national governing body for basketball in the United States. The current league organization divides 30 teams into 2 conferences of 3 divisions with 5 teams each. Reflecting the population distribution of the United States and Canada as a whole, most teams are in the eastern half of the country: 13 teams are in the Eastern Time Zone, 9 in the Central, 3 in the Mountain, and 5 in the Pacific.

Comparing the information highlighted in Tables 62.1 and 62.2, the main difference is for sure game density. An average of 3.4 games per week over 6 months from 30 teams is very demanding, and it takes great organization to cover and facilitate all the players' needs to be part of this competition.

Basketball is a team sport, but with different level of participation above all between the starters and backup players. For this reason training load has to be individualized to get the best

Table 62.2 Example of season 2018/2019 schedule for an NBA team

Phase	Duration	Games played	Games per week	Average
Preseason	2 weeks	4	2 weeks/2 games	2
Regular season	6 months	82	4/1–8/2–10/3–8/4	3.4
Play offs/finals	2 months	16–28	3/2–5/3–1/4	3.1
Full season	8 months	Max 110	–	–
Off-season	3–4 months	–	–	–

results. Staff and players have to be participated actively to codesign these procedures.

NBA players are the world's best-paid athletes by average annual salary per player [5], many of them have other professionals working for them and supporting any decision. Players are not just actors, but directors of their own performance, image, and business. This is why, from my personal experience, it is very important to share as much information as possible between the player, his personal staff, and the team staff.

What the player is doing during the off-season, where he is practicing, for how long, with which goal? Is he doing everything possible at the best the quality necessary to be in a good shape for the beginning of the training camp? Which kind of exercises and treatments should he continue during the season? Is there any kind of information that can be useful to manage the player from one staff to another or from one team to another?

NBA Team Staff work now to be proactive, to foresee players' problems not just to solve them. They know how important any marginal gain about their players is, for a such demanding season. Any stop of a player, also for few days, costs many missing games. The pressure of the schedule that is running frantically does not help them to act always in the best way, so anything that can be done before to avoid any kind of player forced stop has to be taken under consideration.

It is firmly impossible to predict any type of injury, but helping players to improve their train-

ability (capable of being trained consistently) and readiness to perform is a goal on which a lot of work can still be done from science, in terms of providing the right indications, and from the team staff, in terms of applying and verifying the effectiveness of new procedures.

Table 62.3 [6] shows the number of games lost and the economic impact due to injuries, without considering how missing a player can impact team results. There is no doubt how important a clear strategy for load monitoring, management, and recovery procedures is. Age, initial training status, training history, typology of training, injuries history, recovery potential, psychology, stress tolerance, just to mention the main ones, can affect a player's trainability and performance.

Now with microtechnology, many information can be provided about players' external and internal load.

Heart rate monitoring have more precise new interpretations, like TRIMP (training impulse) [7] or the SHRZ (summated heart rate zone) [8]. RPE session [9] for the perception of effort can be very useful and easily done. Questionnaires to track sleeping quality, muscular soreness, nutrition, or more objective evaluations of biomarkers to understand physical conditions are now accessible to almost everyone.

New digital platforms are becoming not only repository of all the information but using machine learning to forecast some status, they can provide interesting indications to staff and players than in the past.

For sure, in some fields technology and science have increased the ability to be effective; however, in sport the human factor remains important. How managers, coaches, staff, and players work together reflects the spirit of the team as well as influences performance.

Table 62.3 Cumulative injured list by injury type, top five results from 2018

Reason	Players	Games	Cash earned while injured
Knee	99	1183	\$152,233,091
Ankle	121	870	\$71,846,122
Foot	34	477	\$43,301,272
Groin	26	175	\$27,114,602
Shoulder	21	257	\$26,802,609

62.5 FIBA World Cup

The FIBA World Cup is a 32-team tournament for national basketball teams. The tournament also acts as a qualification for the following Summer Olympics. The top two teams from the Americas and Europe and the top team from Africa, Asia, and Oceania, alongside the host country's team, qualify for the Olympic Games. The tournament is played in three stages. During the first stage, the 32 qualified teams are sorted into 8 groups of 4 (A–H), with each team playing the other 3 teams once. The top two teams from each group advance to the second group stage. In the second group stage, there are four groups of four (I–L) made up of the teams that advanced from the first round, again playing each other once. The top two teams from groups I to L qualify for the final knockout phase. In total, 108 games are played over a total of 16 days [10]. From the first stage in this tournament, the density of games is very high. Teams start playing three games in 5 days and to reach the final game play eight games in 15 days. The competitive demand for a national team tournament like the World Cup and the Olympic Games is the hardest for a basketball team. Preparing a team at this level of performance is not work that can start just a few weeks before the first game. Many federations have built organizations that work year-round to follow their players, to communicate with player's clubs and staff, and to be available any time that any problem appears, because the first goal for a national team player is to finish the club season and be ready for national team duties healthy. Top basketball players play in NBA, in EuroLeague Teams, or in teams with very intense seasons. For them, being able to start the off-season without main issues to take care about and spend some time to regenerate, physically and mentally, it is very important before approaching to an intense summer with their National Team. Most of these elite players prefer to split their individual physical program with some periods of vacation than long periods of stop. There is some evidence from football [11] that injury incidence does not change in

the weeks following national team duties, and that the injury rate is comparable regardless of if players participate on the national team or not. However, it is very difficult and delicate to manage players that come back from this type of effort like the World Cup. Players have to join their teams that are already in training camp or playing preseason tournaments. Players are trying to work hard to prepare for their upcoming season and may feel mentally and physically empty; looking to recharge their batteries as fast as possible. Once again, there is not a crystal ball that can predict injuries, but there is significant value in transparent communication. Sharing all the information that is necessary to help the player rejoin the team will help them to understand how fundamental training is to preserve their health and performance. Many national teams prepare for high levels of competition, like the World Cup, using the following:

1. Staff follow national team players during their season with the club to share information and procedures that can be helpful for both sides;
2. When a player's club season is over, national team staff meet the player for a physical screening and to discuss their status, providing suggestions on how to personalize their off-season activities;
3. National team coaches and trainers support players, when necessary, to optimize their off-season program;
4. Preparation period before the tournament, 5–6 weeks the most, with players that start generally with a good status of physical health and if possible ready to be trained without restrictions.
5. Inside the team's periodization, a strategic schedule of tournaments against other national teams is built. The goal of these games is to get as close as possible to reproducing the same games density that they will find during the World Cup.
6. Verify the response of the players to the competitive demands and how recovery procedures can be applied; from the nutrition plan to treatments and therapies.
7. Training load management is very important in such an intense working period. Load management helps coaches to optimize their practices and helps players arrive at the tournament in the best shape possible.
8. At the end of the tournament help players to rejoin their club teams, providing information to the clubs that can be useful to facilitate their management and prepare for the upcoming season.

62.6 Olympic Games

The Olympic Games are the most prestigious competition to attend for an athlete of any sport. The tournament is very exclusive, in contrast to the FIBA World Cup, only 12 teams qualify [12]. The tournament starts with two groups of six, with each team playing five games. The top 4 teams from each group advance to the knock out stages, where winning teams advance to play a maximum of three more games. Comparing the FIBA World Cup and the Olympic Games, game density is exactly the same, eight games in 15 days of tournament. The difference between the two tournaments is that at the Olympics all of the games are played in the same arena. Thus, to fit all the games in, games may happen at uncommon times for a basketball game, like 9 am or 10.15 pm. The schedule is a variable that has to be taken into consideration when preparing the team to compete. The game schedule will determine the whole day, from the wakeup call, to what kind of breakfast and when, departure to the arena, preparation to the game, the kind of warm-up, the after game meal, treatments, meetings, and all details have to be properly organized to support players to perform and have to be verified ahead of time. Another unusual aspect of the Olympics is that during the tournament players and staff live at the Olympic Village where it is not easy to optimize time and all the procedures decided. In spite of everything, it is an amazing experience.

Fact Boxes

- Training periodization for basketball, to be effective, has to consider all the variables that influence players' performance. The model that can be created could be very specific to the competition demands and not easily replicable in others.
- The number of games that an elite basketball player plays in a competitive season is very impressive and proportional to his/her level of ability. Having a clear strategy to how to manage training, for team or individual needs and recovery, has become necessary to reduce injuries and to maintain the best performance.
- Continuously improving training load management, well-being, and recovery procedures are fundamental factors to sustain a very demanding season like in NBA.
- To prepare a National Team for a FIBA World Cup or the Olympics, it is advisable to start following national team players at least the season before within their club, to optimize all the summer activities they should follow to start the preparation period in the best way possible.

Take Home Message

- Professional Basketball is very demanding not only for the physical intensity or the technical and tactical spectacularity reached during the game but also for the amount and density of games that some competitions are scheduling. This offering is absolutely positive, mainly for business reason, to keep alive the interest of fans and sponsors is bringing for sure many investments and conse-

quently better players. This is why they have to prepare themselves properly to perform at this level of demand.

- Analyzing all the peculiarities of each competition at the highest level of this sport can help to understand which kind of strategies can be useful to players to prepare themselves for long and intense seasons or for short periods, like during the NT tournaments, where teams play almost every single day. A great contribution in terms of procedures to improve team sports planning is coming from sport science and technology. Many team staff are working to transform theory in practice, creating effective working methods to optimize players' performance.
- Being focus on trying to reduce the risk of injuries, and it comes only from a holistic approach. There are many variables that can influence, statistically, this risk, and we have to consider as many as possible. A basketball player, like any human being, has to react to many stressful agents, and just few of them come from playing basketball. Just monitoring players daily with some basic indicators like sleeping quality, morning muscular soreness, fatigue perception, general stress level, or any feedback that could be relevant can define a general status of readiness to practice, helpful to match with all the other information available.
- Helping players to improve and being able to keep a consistent high level of performance that for them mean to foster their career potential are the main goals for any basketball staff at any level. Collecting as many information as possible to influence decisions became easier nowadays, but how we use all this data and how we roll them out depends only on human factors.

References

1. Lyakh V, Mikołajec K, Bujas P, Witkowski Z, Zajac T, Litkowycz R, Banyś D. Periodization in team sport games—a review of current knowledge and modern trends in competitive sports. *J Hum Kinet.* 2016;54:173–80.
2. NCAA Men’s Basketball Direction of the Game Update—Ncaaorg. https://ncaaorg.s3.amazonaws.com/championships/sports/basketball/rules/men/Jan2019PRMBB_DirectionoftheGameMemo.pdf
3. History of the Shot Clock. NBA Media Ventures, LLC. Archived from the original on April 24, 2012. Accessed 8 Apr 2013.
4. Gamble P. Periodization of training for team sports. *Strength Cond J.* 2006;28:56–66.
5. Gaines C. The NBA is the highest-paying sports league in the world. *Business Insider.* Accessed 20 May 2015.
6. NBA Injured List Tracker on Spotrac. <https://www.spotrac.com/nba/injured-reserve/2018/cumulative-reason/>
7. Aoki MS, Ronda LT, Marcelino PR, et al. Monitoring training loads in professional basketball players engaged in a periodized training program. *J Strength Cond Res.* 2017;31:348–58.
8. Scanlan TA, Fox LJ, Poole J, Dalbo VJ. Smaller heart rate zones improves the precision of the summated heart rate zones training load model in basketball players. *J Aust Strength Cond.* 2017;25(6):63.
9. Lupo C, Tessitore A, Gasperi L, Gomez M. Session-RPE for quantifying the load of different youth basketball training sessions. *Biol Sport.* 2017;34(1):11–7.
10. 2017–2021: FIBA New Competition System. FIBA.com. 21 Oct 2015. Accessed 28 Dec 2015.
11. Carling C, McCall A, Le Gall F, Dupont G. The impact of in-season national team soccer play on injury and player availability in a professional club. *J Sports Sci.* 2015;33(17):1751–7.
12. Qualification for Olympic Games. FIBA. Accessed 10 Nov 2017.



Post-Exercise Recovery Strategies in Basketball: Practical Applications Based on Scientific Evidence

Thomas Huyghe, Julio Calleja-Gonzalez, and Nicolás Terrados

Chapter Objectives

1. Describe up-to-date evidence-based methods to optimize player recovery specifically following basketball competition and/or practice(s);
2. Provide primary and secondary post-exercise recovery protocols applicable to an elite basketball setting;
3. Highlight novel post-exercise recovery strategies in elite basketball with suggestions for future research.

level in 213 countries [1]. During the last decade, elite basketball has become more competitive, with increasingly condensed game schedules. Top players must deal with many national and international championships, with on average a game played every 2.5 days [2]. In addition, the new rules introduced in 2000 by Federation International Basketball Association (FIBA) had a profound effect on the game. In particular, the game became more dynamic as the shot clock was reduced from 30 seconds to 24 seconds, and the time allowed to cross half court was reduced from 10 seconds to 8 seconds. Consequently, a greater total time is spent in high-intensity activities and a greater number of actions take place per game [3, 4]. In addition, to successfully cope with ever increasing demands, players regularly train intensively, with not enough time to fully recover between sessions [5]. Therefore, how to recover faster after basketball training and competition becomes a central question [6].

Complete recovery (return to homeostasis) has been shown to result in the restoration of organic and psychological states [7]. Recovery from competition or training is dependent on the exercise, and it is thus essential to understand the specific mechanisms of fatigue and influences from external factors. The demands placed on basketball players during practice and matches are short, and explosive accelerations, decelerations, change of directions, jumps, and contacts among players which could potentially create

63.1 Introduction

Basketball is the second most popular sport in the world with over 450 million athletes regularly playing the game either on a competitive or recreational

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trauma [8]. Players are also typically characterized by a relative tall body and long limb length, which in turn could influence their susceptibility to fatigue compared to smaller athletes [9]. Therefore, it is important to establish procedures to reduce injury risk, aid recovery, and optimally train basketball players [10], as these become central questions in current basketball practice.

In the scientific literature, a considerable number of methods used to enhance recovery have been discussed [11]. Their use depends on the type of activity performed, the time until the next training session or event and equipment, coaching, or medical staff available. The main recovery methods practically used by teams include rest and sleep, nutritional practices (CHO, proteins, fats, vitamins, minerals), hydration practices, ergogenic aids, cool-down strategies, psychological strategies, and manual therapy techniques (Fig. 63.1). However, there is a lack of consensus on the benefits of many of these approaches in the scientific community.

While several reviews about recovery methods have been published in other team sports, such as soccer [12] and rugby [13], to our knowledge, there has been no practical review or report about recovery in basketball. Therefore, this chapter will focus on specific recovery processes in basketball and will attempt to give practical information for coaches, clinicians, and practitioners.

63.2 Monitoring Post-Exercise Fatigue

The recovery process in elite basketball may take up to 48 h during regular practices and games [48] and is challenging to manage considering the multiple contextual factors involved (e.g., travel direction, travel duration, individual chronotype, activity type, playing position, playing style, variation of total playing time, time distribution of playing time, and physical qualities of the player). These variables together determine how players respond to game load and play an important role in their ability to recover from that load. Therefore, monitoring the individual load from games and practices in combination with markers of performance and fatigue (internal response) is crucial in elite basketball.

In order to prescribe recovery strategies customized to each player individually, both objective markers (e.g., blood parameters, salivary biomarkers, heart rate parameters, sleep parameters, performance tests) and subjective markers (e.g., questionnaires, and conversations with players) are used to monitor the time course of recovery from basketball training and games. For instance, the counter movement jump (CMJ) is a commonly used neuromuscular performance

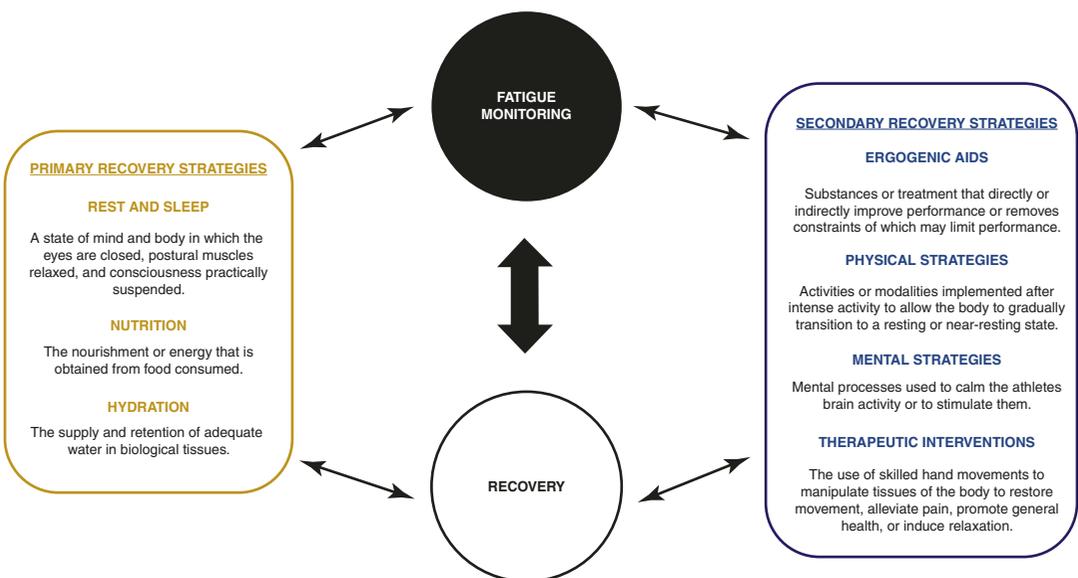


Fig. 63.1 Individualization of post-exercise recovery strategies in elite basketball

test which may serve as an indication of recovery throughout the course of a basketball season. In particular, flight time:contraction time tends to be a more sensitive measure of recovery compared to jump height [49]. Alternatively, a 10-m sprint test may also be used where baseline values have been recorded 48 h after simulated basketball games [50]. However, despite the value behind performance tests, players may return to baseline performance levels while still not being fully recovered. Therefore, performance tests should be analyzed alongside biochemical markers in order to identify precise muscle damage in each player. Common biochemical markers that reflect muscle recovery include creatine kinase (CK) and cortisol (C) which may take up to 72 h to return to baseline levels, despite recovery of physical performance after 48 h [48]. This slow decrease in CK and C suggests that although performance may already be at optimal pregame values, the muscles may need significantly more time to recover. Hence, high-performance practitioners should take this into consideration when preparing players during specific phases of the season (either allowing or avoiding cumulative fatigue at that time).

Although “fatigue monitoring” is not the prime emphasis of this chapter, high-performance practitioners should be aware that the suggestions given regarding recovery within this chapter should always be modified toward the individual needs of each player as well as the situation at hand in order to stimulate optimal adaptations (Fig. 63.1).

63.3 Primary Post-exercise Recovery Strategies in Basketball

63.3.1 Rest and Sleep

The amount of sleep an athlete gets appears to have a large impact on sports performance through a variety of direct and indirect pathways (e.g., immune system, mood, focus, motivation, reaction time, glucose metabolism, muscle growth and repair, etc.) [43]. Mah et al. con-

cluded that improvements in specific measures of sprint time, shooting accuracy, and free throw percentage occurred after sleep extension (minimum of 10 h of sleep with optional daytime napping), thus indicating that optimal sleep (8–10 h of nocturnal sleep) is beneficial in allowing athletes to reach their peak athletic performance [45]. In the last season after the lock-out in the NBA, interviews with players revealed that they were not doing much to compensate for the loss of sleep and instead were feeling the effects of the condensed season. This issue remains in today’s NBA environment, perhaps due to frequent social media usage, as late-night tweeting (between 11:00 PM and 7:00 AM) 1 day prior to the game has demonstrated to impair next-day game performance [51]. In particular, NBA players significantly spent 2 fewer minutes on the court, shot 1.7% less accurate, as well as experienced negative returns on total rebounds, points, fouls, and turnovers following late-night tweeting [51]. An investigation conducted by Steenland and Deddens (1997) analyzed the effect of travel and rest on performance over 8,495 regular season games (each team) in the NBA across eight seasons and found that more time between games significantly improved performance [44]. This finding was consistent throughout the 8-year observation [44]. This suggests that the NBA travel schedule induces misalignments in circadian rhythm that cannot be avoided. In order to resynchronize the circadian rhythms of players, it is recommended to apply blue light exposure in the morning and red light exposure in the evening [52, 53]. Other strategies may include the ingestion of a high-carbohydrate, low-protein meal in the evening, which may enhance serotonin production to promote drowsiness and sleep [52], or the ingestion of a high-protein, low-carbohydrate meal in the morning, which may increase the uptake of tyrosine and its conversion to adrenaline, which elevates arousal and promotes alertness [52] (Table 63.1). Recently, the consumption of tryptophan-rich protein (e.g., milk) has gained popularity as it induces changes in bedtime core body temperature improving sleep quality [54, 55]. Furthermore, tart cherries have also been suggested to enhance post-exercise recovery as

Table 63.1 Practical recommendations to optimize post-exercise recovery in elite basketball players (primary recovery)

Primary post-exercise recovery strategies		
Rest and sleep	Nutrition	Hydration
<p><i>Light exposure</i></p> <ul style="list-style-type: none"> • Avoid blue light exposure (e.g., late-night social media usage) between 11:00 PM and 7:00 AM [51] • Promote blue light exposure in the AM and red light exposure in the PM to facilitate optimal arousal levels and a stable circadian rhythm [52, 53] • Consider body irradiation with red light to players experiencing difficulties falling asleep [46] <p><i>Nutritional interventions</i></p> <ul style="list-style-type: none"> • Consume a high-carbohydrate, low-protein meal in the PM and a high-protein, low-carbohydrate meal in the AM [52] • Promote tryptophan-rich protein (e.g., milk) and melatonin-rich foods (e.g., 2× servings of 30 mL tart cherry concentrate) prior to bedtime [54, 55] • Avoid alcohol, caffeine, or large meal or fluid consumptions prior to bedtime [57] <p><i>Education and Awareness</i></p> <ul style="list-style-type: none"> • Facilitate opportunities for players to learn about topics such as sleep quality and quantity, time zone shifts, travel fatigue, circadian rhythm, caffeine, alcohol, napping, sleep apnea, and acute vs. chronic sleep deprivation <p><i>Lifestyle Habits and Environment</i></p> <ul style="list-style-type: none"> • Maintain a regular sleep–wake cycle/routine [45, 57] • Ensure a comfortable, quiet, dark, and temperature-controlled bedroom [57] • Consider taking a warm bath to lower core body temperature prior to bed [57] • Design a “to-do” list or diary to avoid over-thinking and manage work-related stress [57] • Use relaxation/breathing techniques after high-intensity training sessions or games [57] 	<p><i>Carbohydrates (CHO) and proteins</i></p> <ul style="list-style-type: none"> • Include CHO in rehydration beverages • Include rapidly absorbed CHO and hydrolyzed whey protein in recovery drinks, using 3–4/1 ratio, being 1 g/kg the amount of the recommended CHO [15] • During the season, CHO, maintain protein and leucine intake (doses: 0.3 g/kg of CHO, 0.2 g/kg of protein and 0.01 g/kg of leucine) per day, immediately after training or games [16] • During the season, maintain daily macronutrient ratio of amino acids (14.5%), proteins (12.7%), and CHO (12.7%); however, individual needs and differences should be taken into consideration before any prescriptions [17] <p><i>Individualization</i></p> <ul style="list-style-type: none"> • Use blood or salivary to identify individual vitamin and mineral status and individual needs throughout the season [64] <p><i>Food selection</i></p> <ul style="list-style-type: none"> • Promote foods high in vitamins C, vitamins E, carotenoids, and flavonoids [19, 22] • Promote magnesium-rich foods (e.g., nuts, seeds, fatty fish, legumes, whole grains) • When inadequate Mg levels are reported or when going through intense periods, 400 Mg/day of mg, in the form of Mg lactate, may be considered as ergogenic aid [63] • Promote vitamin D-rich foods (e.g., fatty fish) especially when sunlight exposure is limited [61] • If inadequate vitamin D serum levels are reported, vitamin D supplementation may be considered as ergogenic aid [58–60] <p><i>Nutrient timing</i></p> <ul style="list-style-type: none"> • Refuel and rehydrate within 30 min after exercise to help muscles recover faster [16] <p><i>Education and Awareness</i></p> <ul style="list-style-type: none"> • Facilitate opportunities for players to learn about topics such as food choices, timing of meal consumption, and short-term vs. long-term unhealthy/healthy food choices 	<p><i>Education and Awareness</i></p> <ul style="list-style-type: none"> • Facilitate opportunities for players to learn about euhydration, dehydration, and monitoring hydration status [17] • Ensure consistent beverage availability throughout the day [17]. <p><i>Individualization</i></p> <ul style="list-style-type: none"> • Monitor hydration status in each player throughout the day through their thirst, changes in body mass, and/or their urine color [17] <p><i>Lifestyle Habits and Fluid Selection</i></p> <ul style="list-style-type: none"> • Promote electrolyte-rich foods and fluids including sodium, potassium, chloride, and magnesium [69] • Consider 2.5–3.0 L of high-alkaline water during high-intensity anaerobic exercises [65]

well as sleep due to its anti-inflammatory and antioxidant properties as well as high concentrations of melatonin [54]. In particular, a recent study investigated the effect of tart cherry juice (2×30 mL concentrate) on sleep enhancement, sleep duration, and sleep quality in healthy subjects in which the intervention group significantly increased their total melatonin content, increased time in bed (+24 min), increased total sleep duration (+34 min), improved sleep efficiency (82.3%), and reduced daytime napping (−22%) [56]. Finally, recent research demonstrated the effectiveness of body irradiation with red light in improving the quality of sleep of elite female players and offered a non-pharmacologic and noninvasive therapy to prevent sleep disorders after training [46] (Table 63.1). Nevertheless, the effects of tryptophan-rich protein, tart cherry consumptions, and other promising alternatives (e.g., napping, sleep education, chrononutrition) on sleep and recovery in elite basketball players warrant further investigation, especially considering sleep disturbances may occur due to a player's unique lifestyle, and thus results derived from sleep intervention studies in the general population may be limited in their applicability to elite basketball players [57] (Table 63.1).

63.3.2 Nutritional Strategies

63.3.2.1 Carbohydrates and Proteins

Carbohydrates should be included in rehydration beverages to improve palatability and to aid in the immediate restoration of muscle glycogen stores [11]. From a nutritional point of view, basketball players use carbohydrates (CHO) as the primary source of fuel during exercise, given the type of training they perform and the characteristics of competition [4]. After a training session or match, muscular stores of carbohydrate are depleted, and thus consuming CHO and protein during recovery has been shown to positively affect subsequent exercise performance and could be of benefit for athletes involved in multiple training or competition sessions on the same or consecutive days [14]. The ideal combination will be of rapidly absorbed CHO together with

hydrolyzed whey protein, using 3–4/1 ratio, being 1 g/kg the amount of the recommended CHO [15] (Table 63.1). Eating and drinking the right kind of fuel after exercise is important for restoring energy levels and repairing muscle damage. Refueling with CHO, protein, and fluid within 30 min after exercise will help muscles recover faster. Within this context, it has been established that consumption of macronutrients, particularly CHO and possibly a small amount of proteins and leucine (doses: 0.3 g/kg of CHO, 0.2 g/kg of protein and 0.01 g/kg of leucine), in the early recovery period after practice can enhance muscle glycogen resynthesis [16], per day during the season (Table 63.1). For instance, in an analysis on the daily consumption of dietary supplements on 55 professional basketball players from seven different teams of the First Spanish Basketball League (ACB), 12.7% of players consumed on average 10.3 g protein per day, 14.5% of players consumed 1.9 g amino acids per day, and 12.7% of players consumed 16.2 g carbohydrates per day [17]. However, future studies should analyze the ingestion of CHO in combination with protein in different dosages to determine which dose will enhance recovery specifically for basketball players.

63.3.2.2 Vitamins and Minerals

Oxidative stress occurs when the body does not have enough capacity to defend itself against free radicals. Reactive oxygen species are the main source of oxidative stress and play a major role in the initiation and progression of damage to the muscle fibers after exercise [18]. Several antioxidants have been introduced to protect the cells from free radicals such as vitamins C and E, carotenoids, and flavonoids [19].

Oxidative stress may be involved in the aging process, cell damage, muscular fatigue, and overtraining [20], especially during maximal exercise in basketball. Hence, playing competitive basketball may involve greater utilization of aerobic metabolism than previously expected, with values of VO_2 of 33.4–4.0 and 36.9–2.6 mL/kg/min for females and males, respectively [21]. Within this context, the consumption of vitamins C and E may strengthen the antioxidant defense system

by decreasing reactive oxygen species involved in maximal- or high-intensity exercise [22]. Regarding the nutritional habits of players, it has been shown that multivitamins were the most frequently used supplements among these athletes (50.9%), followed by sport drinks (21.8%) [17].

Vitamin D serum levels has an impact on a player's overall health and ability to train (e.g., bone mass density, overall immune system, exercise-induced inflammation, stress fractures, muscle recovery, and athletic performance) [58–60]. Professional basketball players may be at higher risk for hypovitaminosis D (vitamin D deficiency) after the season due to the amount of time spent indoors and often lack of sunlight exposure [61]. For instance, 57% (12 of 21) of professional Spanish basketball players from the same team were below the optimal standards serum vitamin D levels (<50 nmol/L) after wintertime during two consecutive seasons [61]. Another study confirmed these findings in players at the NBA combine (2009–2013) in which 79.3% (221 players) appeared to be vitamin-D-deficient or insufficient (<32 ng/mL) [60]. Consequently, adequate intake of dietary vitamin D is recommended if elite basketball players are to avoid low serum vitamin D, especially when sunlight exposure is limited. For instance, an adequate consumption of fish at least four times per week promotes the maintenance of a healthy status of vitamin D serum levels (>60 ng/mL) in athletes [61]. If athletes dislike fatty fish (or consume limited amounts), ergogenic supplementation should be considered to avoid vitamin D deficiency [61] (Table 63.1).

Magnesium (Mg) is another vital mineral for the body's function, specifically regarding energy metabolism, transmembrane transport, muscle relaxation and contraction [62]. Mg tends to be of increased demand in elite athletes [63]. Mg concentrations significantly decreased over time in 12 elite basketball players competing in the Asociación de Clubs de Baloncesto (Spanish highest professional basketball league) who were tested four consecutive times throughout the season (8 weeks between each test) [63]. Furthermore, Mg concentrations significantly dropped below the optimal norm at the third test [63]. Consequently, player education and

awareness of magnesium-rich foods (e.g., nuts, seeds, fatty fish, legumes, whole grains) should be encouraged. During extremely intense periods (e.g., tournaments, playoffs), ergogenic supplementation of 400 mg/day of Mg, in the form of Mg lactate, may also be considered [63] (Table 63.1).

63.3.3 Hydration

A frequent issue in elite basketball is the inadequate supply of water or other fluids both during periods of rest or activity. For instance, in a study by Osterberg (2009), 29 NBA players were tested for urine specific fluid and gravity. Approximately half of the players entered the game in a hypo-hydrated state [64]. Unfortunately, fluid intake during the game did not compensate for poor hydration status before games. Additionally, the NBA players who participated in this study also lost more than 2 L of sweat in approximately 20 min of game time [64]. In a study on fluid intake habits of 55 elite basketball players competing in the Spanish Basketball League (ACB), 44% of the players recorded not to drink before getting thirsty [17], which suggests the need for player education and awareness on (re)hydration throughout the day (Table 63.1).

A dehydration of 1–2% may negatively impact focus, cognitive functions [65], reaction time [65], exercise tolerance [65], aerobic endurance [66], and jumping performance [67]. Subsequently, this may progressively deteriorate the ability to complete basketball skills during games such as sprinting, defensive slides, sprinting-defensive slides combination, repetitive jumping, and total number of shots [68]. Therefore, both pregame and in-game hydration strategies (e.g., player education, beverage availability) should be encouraged (Table 63.1).

The hydration status of players can be monitored in a variety of ways such as their thirst, changes in body mass, or their urine color. However, once these variables have changed, dehydration has already occurred, which means it becomes too late to compensate for it. Therefore, it is recommended to approach players' fluid

balance in a proactive manner throughout the day. Furthermore, it is essential to acknowledge that sweat loss includes vital electrolytes such as sodium, potassium, chloride, and magnesium [69]. Recently, high-alkaline water has been suggested to promote faster rehydration as well as delay muscular acidosis during anaerobic exercise, prevent dehydration, and speed up the recovery process [65]. In particular, professional basketball players who consumed high-alkaline water during anaerobic activity showed a positive impact on their acid-base balance with a significant increase in blood and urine pH which in turn may lower concentration of free radicals as well as the improvement of connective tissue condition [65]. More specifically, the daily intake of 2.5–3.0 L of highly alkalized water has been suggested during strenuous basketball games or practices [65] (Table 63.1).

63.4 Secondary Post-exercise Recovery Methods in Basketball

63.4.1 Ergogenic Aids and Supplements

An *ergogenic aid* can be broadly defined as a technique or substance used for the purpose of enhancing performance.

63.4.1.1 Creatine

Since 1992, the interest in creatine (Cr) as a nutritional supplement has dramatically increased. Over the past two decades, the main focus of research has been on the ergogenic value of Cr and its underlying mechanisms (resynthesis of ATP) [23]. Creatine is a naturally occurring nutrient, consumed through food intake and synthesized in the body. In basketball, Shi et al. [24] (2005) concluded that supplementation of CHO and Cr could promote the recovery of physical performance, demonstrating its efficacy in a sport like basketball characterized by high-intensity efforts [24]. In this sense, data from top-level Spanish players showed that low-dose supplementation with Cr monohydrate did not produce laboratory abnormalities for the major-

ity of the health parameters during three competition seasons [25]. Besides the benefits of Cr on physical performance in elite athletes, supplementation of Cr has also demonstrated to have a positive impact on brain energy homeostasis and improved cognitive function in athletes suggesting the need for a holistic approach to future studies on creatine supplementation in elite basketball players [70]. According to a recent systematic review and meta-analysis on elite soccer players, creatine supplementation with a Cr loading dose of 20–30 g/day, divided 3–4 times per day, ingested for 6–7 days, and followed by 5 g/day for 9 weeks or with a low dose of 3 mg/kg/day for 14 days seem to be optimal for improving anaerobic performance [71] (Table 63.2). Furthermore, the International Society of Sports Nutrition (I.S.S.N.) supports a Cr loading dose of approximately 0.3 g per kg per day for at least 3 days [72]. Consequently, although the existing evidence behind the benefits of Cr, future studies in elite basketball players are warranted to confirm optimal Cr dosing strategies.

63.4.1.2 β -Alanine and Sodium Bicarbonate

β -alanine supplementation has become a common practice among different sports. Although the mechanism by which chronic β -alanine supplementation could have an ergogenic effect is widely debated, the popular view is that β -alanine supplementation augments intramuscular carnosine content, leading to an increase in muscle buffer capacity, a delay in the onset of muscular fatigue, and a facilitated recovery during repeated bouts of high-intensity exercise [26].

β -alanine supplementation has been shown to improve high-intensity exercise performance and capacity [27]. However, its effect on recovery is not clear, but some authors indicated that β -alanine supplementation in highly-trained athletes could be of importance [27]. However, nowadays there is no scientific evidence about the ergogenic effect of β -alanine in team sports (including basketball). Among the most recent investigations, the focus has been on the effect of β -alanine supplementation and sodium bicarbonate on high-intensity efforts, but these studies have been performed predominantly on endur-

Table 63.2 Practical recommendations to optimize post-exercise recovery in elite basketball players (secondary recovery)

Secondary post-exercise recovery strategies			
Ergogenic aids	Cooldown strategies	Psychological strategies	Manual therapy
<p><i>Creatine monohydrate</i></p> <ul style="list-style-type: none"> During intense periods, consider consumption of 20–30 g/day of β-alanine (0.3 g per kg body weight), divided 3–4 times per day, for 6–7 days, followed by 5 g/day for 9 weeks or with a low dose of 3 mg/kg/day for 14 days [71] <p><i>Sodium bicarbonate</i></p> <ul style="list-style-type: none"> For highly trained players, consider consumption of 0.2 g kg⁻¹ of sodium bicarbonate (NaHCO₃) 90 and 60 min prior to high-intensity practices or games [73] 	<p><i>Active recovery</i></p> <ul style="list-style-type: none"> Consider 7 min of low-intensity exercise after strenuous exercise [13] <p><i>Stretching + massage</i></p> <ul style="list-style-type: none"> Consider the combination of stretching + massage <2 h after training or games [34] <p><i>Hydrotherapy</i></p> <ul style="list-style-type: none"> Consider hydrotherapy as a recovery modality 24–72 h after exercise; however, account for individual differences in physiological status, body mass and body composition [35] Consider full-body cold-water immersion <5 min after practices or games, with 5 bouts of 2 min immersions of the lower limbs in cold water (11.8 °C), separated by 2 min rest in ambient air (sitting, room temperature of 20.8 °C). Add ice at regular intervals to maintain water temperature at 11 ± 0.78 °C [35, 36] Consider full-body contrast water therapy in which warm water (40–42 °C; 3 min) and cold water (8 ± 1 °C; 1 min) are altered for a 20-min period [76] <p><i>Compression clothing</i></p> <ul style="list-style-type: none"> Wear full-length or lower limb compression garments and/or medical-grade compression socks during travel or rest periods; however, customize garments to individual size/fit and account for differences in sensitivity to blood flow changes [37, 38, 78] <p><i>Cryotherapy</i></p> <ul style="list-style-type: none"> Consider cryotherapy for 2–4 min of extreme cold air (–110° to –140 °C) during strenuous periods of the year; however, take into account the increased sensitivity for athletes with a relative low BMI [74, 79] 	<p><i>Wellness and Recovery Surveys</i></p> <ul style="list-style-type: none"> Collect wellness and RPE scores on a daily basis to gain specific insight into psychological status of each athlete [42] <p><i>Mental recovery breaks</i></p> <ul style="list-style-type: none"> Use short rest breaks between training sessions as an opportunity to cultivate and explore optimal mental recovery routines including: <ul style="list-style-type: none"> Relaxation techniques [83]; Breathing techniques [83]; Mental imagery [83]; Povernaps [83]; Debriefing [83]; Mental detachment [83]; Restorative environments [83]; Music [83]; Caffeine [83] 	<p><i>Acupuncture</i></p> <ul style="list-style-type: none"> Consider acupuncture stimulation after strenuous exercise, however, based on personal preferences [47] <p><i>Dry needling</i></p> <ul style="list-style-type: none"> Consider dry needling of the fibularis muscles in players with a history in lateral ankle sprains, however, based on personal preferences [85] <p><i>Massage</i></p> <ul style="list-style-type: none"> Consider 10 min of full-body effleurage in a room with temperature of 25–26 °C, however, based on personal preferences [87]

ance exercise [28]. Nevertheless, the results of a recent study by Ansdell et al. revealed that the supplementation of 0.2 g kg⁻¹ of sodium bicarbonate (NaHCO₃) 90 and 60 min prior to commencing a simulated basketball game may

promote recovery through slowing down the rate of post-game fatigue occurrence by protecting contractile elements of the muscle fibers [73] (Table 63.2). Acknowledging the role of β -alanine and NaHCO₃, it could be interesting

to further investigate the effects of these supplements in basketball since it is an intermittent sport with a variety of multidirectional movements such as running, dribbling, and shuffling at variable velocities and jumping [29].

63.4.2 Physical Recovery Strategies

Cooldown is a widely accepted practice after training sessions, used to reduce heart rate back to resting rate, stretch muscles, remove lactate concentration, *resynthesize* high-energy phosphates, replenish oxygen in the blood, body fluid, and myoglobin, and support the small energy cost to sustain an elevated circulation and ventilation [30, 31] (Table 63.2). However, despite being considered as essential for optimum performance, there is no investigation that has identified the optimum cooldown process in basketball.

63.4.2.1 Active Recovery

Although the dearth of scientific research, active recovery may be beneficial for athletes through enhanced blood flow in muscle tissue, which facilitates the removal of metabolic waste, and may contribute to a reduction in muscle lesions and pain [74]. Active recovery may have a larger effect on delayed onset muscle soreness (DOMS) than perceived fatigue in athletes [74]. However, the prescribed protocol seems play a role in the impact of active recovery. For instance, after a rugby contest, 1 h of low-intensity aquatic exercise did not alter creatine kinase (CK) concentrations [75], but 7 min of low-intensity exercise enhanced CK clearance [13] (Table 63.2). Considering the practicality of active recovery for athletes [32], future investigations should further analyze the physiological and psychological effect of this cooldown method, particularly in elite basketball players.

63.4.2.2 Stretching

Post-event cooldown strategies relying on stretching techniques should not be done with the goal to drastically improve flexibility. Dynamic stretching has gained popularity, due to a number of studies showing an increase in high-intensity

performance compared to static stretch modalities [33]. However, post-competition, static stretching is not recommended as a way to improve flexibility and reduce adhesions caused by physical activity [11]. In basketball, little research has been reported about this concept, Delextrat et al. 2014 demonstrated that female basketball players benefit slightly more from the combination treatment (massage + stretching) than men, and therefore this type of recovery intervention should be adopted by physiotherapists especially in women's teams within 2 h after training or matches, in particular during tournaments where matches are played daily [34] (Table 63.2). Both recovery procedures improved perceptions of overall fatigue and leg soreness, with greater benefits of the combination on leg soreness [34].

63.4.2.3 Hydrotherapy

One method gaining popularity as a means to enhance post-game or post-training recovery is immersion in cold water. Much of the literature on water immersion as an intent to improve athletic recovery appears to be based on anecdotal information, but it is suggested that this method can improve recovery 24–72 h after exercise [35]. In this study, the cold-water immersion occurred within 5 min of the completion of the match and consisted of five 2 min intermittent immersions of the lower limb (up to the iliac crest) in a cold-water bath (11.8 °C), separated by 2 min rest in ambient air (sitting, room temperature of 20.8 °C). Ice was added to the bath at regular intervals to maintain water temperature at 11 ± 0.78 °C (Table 63.2). In basketball, few articles have analyzed the effect of water immersion on recovery. They demonstrated that it is more useful than massage in the recovery from basketball matches [36]. It has been shown that a tournament elicited small to moderate impairments in physical performance, and that cold-water immersion appears to promote better restoration of physical measures, such as 20-m acceleration, than CHO and stretching routines or compression garments [8].

Contrast water therapy (CWT) or changing from cold to warm water immersion and/or vice versa is another recovery method gaining popularity in recent years. This method has

demonstrated to significantly reduce the perception of pain at 24, 48, and 72 h post-eccentric exercise [74] due to peripheral vasoconstriction and vasodilation as this reduces the formation of edema after exercise, alters acute inflammatory responses, and reduces blood CK concentrations, suggesting less muscle damage [74]. In particular, altering warm water (40–42 °C; 3 min) and cold water (8 ± 1 °C; 1 min) for a 20-min period has demonstrated to improve recovery (specifically hematological and physical components) compared to passive recovery in university-level rugby players [76] (Table 63.2). However, future studies are needed to identify the reliability of this recovery protocol at various times throughout the season in elite basketball players.

63.4.2.4 Compression Garments

Compression garments are articles of clothing such as socks or leggings that provide support. For instance, small-to-moderate benefits of in-flight compression socks have been reported on systolic blood pressure, right calf girth, CMJ height, mean velocity, and relative power in elite volleyball players after long-haul travel, compared with a passive control group [86]. The utilization of compression garments has also been adopted for athletes due to their potential benefits for physical performance and recovery [37]. Compression garments apply mechanical pressure to the body, and they compress and support underlying tissues [38]. The garments can come in varying degrees of compression and therefore enhance recovery. In a recent systematic review (with meta-analysis) conducted by Marqués-Jiménez et al., evidence concluded the benefits of compression garments on exercise-induced muscle damage, specifically in reducing perceived muscle soreness and swelling while improving neuromuscular power and strength [77]. Although controversy in optimal pressure, time of treatment and type of garment to maximize these benefits, Brown et al. suggested the largest benefits resulted from compression garments applied 2–8 h and >24 h after strength training sessions [78] (Table 63.2). Nevertheless, future studies are warranted in basketball players participating in a variety of exercise modalities, dif-

ferent recovery period lengths, length of applied compression, amount of pressure applied, and place of applied compression (upper limbs, lower limbs, or whole body) in order to prescribe more specific protocols to each player. Finally, individual differences in sensitivity to blood flow changes may also impact the rate of recovery when wearing compression garments [74].

63.4.2.5 Cryotherapy

Whole-body cryotherapy is an expensive recovery method which typically includes short exposure (2–4 min) to extreme cold (–110° to –140 °C) induced by air (Table 63.2). Due to the numerous cryostimulation methods used by researchers and practitioners, the impact of cryotherapy on DOMS and fatigue remain inconsistent. However, whole-body cryotherapy (WBC) could reduce DOMS after an exposure of 3 min at –140 and –195 °C in recreational participants [79]. This cooldown method may also improve muscle fatigue, pain, and well-being (DOMS and self-perceived fatigue) [74] although the effects on DOMS seem to occur shortly after exercise (<6 h after exercise), while not after 24 h or later [74], and multiple cryostimulation treatments are required to experience positive benefits on recovery. Furthermore, in elite basketball players competing at the European Championship, 3 min of cold exposure (at –130 °C) significantly improved their mean thermal sensation score during partial-body cryostimulation (PBC) exposure in athletes [80]. However, large inter-individual differences were reported mainly due to differences in body mass index (BMI) [80]. Although future studies are needed on the effects of WBC and PBC on recovery and sensation in elite basketball players, a 3-min exposure seems to be well tolerated by athletes in general, and may be most effective during the heaviest training or competition periods of the year. In physically active men, 3-min WBC in the evening after training has recently demonstrated to improve both objective and subjective sleep quality, thus WBC may be considered for that particular reason as well [81]. However, special attention should be given to female athletes with a low BMI as they seem to be significantly more sensitive to cold air exposure [80].

63.4.3 Mental Recovery Strategies

To ensure that athletes maximize the benefits from demanding training sessions and remain robust enough to cope with multiple performances, it is vital that individual athletes have the ability to recognize when and how they need to recover. Burnout is defined as a state of mental, emotional, and physical exhaustion brought on by persistent devotion to a goal in which its achievement is dramatically opposed to reality [40]. Recently, it was demonstrated that an increase in self-control could reduce negative anxiety effects and improve player's performance under pressure [41]. Furthermore, mental fatigue may play a significant role in technical performance of elite basketball players [82], suggesting the need for a holistic approach to recovery encompassing both physical and mental strategies.

Results from recent studies indicated that session Ratings of Perceived Exertion (sRPE) seems to be a viable tool in monitoring internal load. These responses might help coaches to plan appropriate loads, hence maximizing recovery and performance [42]. Recently, short rest periods during the day (between subsequent training sessions) have been suggested as vital opportunities for mental recovery interventions and may help in assisting players to return to baseline levels of mental abilities (e.g., concentration, vigilance, attention) and restoration of mental energy [83]. For instance, relaxation techniques (e.g. yoga, meditation, and autogenic training), breathing techniques (e.g., pranayama), mental imagery (e.g., focusing on the upcoming performance, imagination of positive and confidence-boosting atmospheres, positive self-talk, and goal-setting), power naps, debriefing (e.g., in-person post-training and post-game evaluations, distancing and reorientation from previous experiences and feelings), mental detachment (e.g., disengaging from work-related topics), restorative environments (e.g., immersion into natural settings such as fascinating landscapes), post-exercise music (e.g., slow and sedative music), and controlled consumption of caffeine have all demonstrated to serve as beneficial psychological recovery strategies in elite athletes [83] (Table 63.2). However,

basketball-specific research on mental recovery strategies and their impact on player mental status is lacking.

63.4.4 Therapeutic Interventions

63.4.4.1 Acupuncture

Acupuncture is a recovery method used in traditional Chinese medicine for many years in which thin needles are inserted into the body. Although the scarcity of scientific research on this method in elite basketball players, one interesting study indicated the positive effects of acupuncture stimulation (beginning 15 min prior to exercise and ending after player exhaustion) on the recovery abilities of university basketball players [47]. In particular, 30 university basketball players were randomly assigned to three groups: experiment group (acupuncture at the Neiguan (PC6) and Zusanli (ST36) acupoints), sham group (acupuncture 1 cm away from the PC6 and ST36 acupoints), and control group (no acupuncture) while heart rate (HR_{max}), oxygen consumption (VO_{2max}), and blood lactate concentration were recorded in each group during the rest period and at 5-, 30- and 60-min after exercise [47]. Significantly lower levels of HR_{max} , VO_{2max} and blood lactate concentration were reported in the experiment group compared to both sham and normal groups at the 30 min post-exercise test period while blood lactate concentration of the experiment group was the lowest at the 60-min post-exercise test period compared to all other groups [47] (Table 63.2). Hence, these findings suggest that acupuncture schemes may enhance the recovery ability in elite basketball athletes [47]. Tandy et al. also demonstrated the positive effects of acupuncture on lactate clearance following high-intensity exercise in elite basketball players [84]. However, it should be noted that external factors (e.g., lifestyle, exercise preferences) were not taken into account and may have influenced these findings. Hence, future studies should aim to include these factors when possible. Moreover, many variations have accumulated over the years, and techniques vary depending on the country in which the acupuncture is performed, thus

future studies are needed to identify best-practice protocols in order to optimize recovery in elite basketball players. For instance, an alternative method described as “myofascial trigger point puncturing” (dry needling) has gained popularity in recent years. In a recent study on participants with a history in lateral ankle sprains, the authors concluded that dry needling the fibularis muscles may induce short-term improvements in strength and unilateral balance [85], though follow-up studies are needed with larger sample sizes and in basketball players specifically to substantiate these findings. Considering the current evidence on acupuncture, this recovery method can be safely applied as a complementary strategy to optimize the recovery of elite basketball players after strenuous exercise by enhancing the clearance of lactate levels in muscles. It is also a relative simple and well-tolerated method with little side effects reported in the literature.

63.4.4.2 Massage

Many athletes consider sports massage as an essential part of their training and recovery routine. These athletes report that a sports massage helps them train more effectively, improve performance, prevent injury, and speed recovery. Massage is effective in alleviating DOMS by approximately 30% and reducing swelling [39]. However, Delextrat and colleagues (2005) demonstrated that massage did not have any effect on repeated sprint ability (RSA) [36]. Although massage alone may not impact RSA, another study found that stretching as an adjunct to massage may improve recovery from official matches in basketball players [34]. This study by Delaxtrat et al. (2005) was the first study to analyze the impact of massage on recovery in basketball [36]. Recently, Kaesaman et al. (2019) indicated that massage (specifically traditional Thai massage) is a more effective strategy than passive rest on recovery [87]. In particular, 16 basketball players were randomly assigned to either experiment group (10 min of traditional Thai massage throughout the body including biceps and triceps, deltoid, latissimus dorsi, thoracolumbar fascia, trapezius, and stretching muscles of hamstrings, rectus femoris, arms, and back, while the room

was controlled at a temperature of 25–26 °C following basketball activity) or control group (10 min of rest following basketball activity) [87] (Table 63.2). Each player was required to perform 20 min of basketball simulation after which they were monitored on HRV and physical fitness, and monitored again after the intervention (massage or rest). The results indicated that traditional Thai massage can increase HRV, reduce sympathetic activity, and increase parasympathetic activity which contributes to the greater recovery rate in basketball players [87]. Nevertheless, more studies are needed to confirm the positive benefits of various massage techniques.

Take-Home Message

Recovery from training is recognized as one of the most important components of training regimen. To maximize year-round player recovery, primary recovery strategies (e.g., sleep, nutrition, and hydration) should be prioritized and encompassed by secondary recovery strategies (e.g., ergogenic aids, cooldown strategies, psychological strategies, manual therapy). Furthermore, daily fatigue monitoring is essential to understand and maximize the applicability, effectiveness, and efficiency of each of these recovery strategies at the individual level.

Consistent high-quality sleep, food, and fluid consumption play a fundamental role in the recovery process following exercise in elite basketball players. Adequate sleep can be promoted and maintained through a variety of strategies, such as nutritional interventions (e.g., high-carbohydrate and melatonin-rich foods prior to bedtime), optimal timing of light exposure (e.g., blue light in the AM vs red light in the PM), and lifestyle habits (alcohol, caffeine, social media usage, pre-bed routines). High muscle glycogen restoration and status of euhydration can be achieved through high-carbohydrate consumption plus leucine and adequate proactive drinking immediately

after exercise. Ergogenic aids may support the recovery process through metabolic alkalosis via bicarbonate ingestion plus beta-alanine. Hydrotherapy (e.g., cold-water immersion and/or contrast bath), cryotherapy, and compression clothes may also aid in player recovery by reducing DOMS and/or self-perceived fatigue 24 h after a game or strenuous training session. Finally, time between training sessions serves as great opportunities for athletes to develop proactive mental recovery habits, suggesting relaxation techniques, breathing techniques, power naps, debriefing, mental detachment, immersion to restorative environments, controlled consumption of caffeine, mental imagery, and/or post-exercise music. However, differences in individual responses to each of these modalities should be taken into account prior to intervention. Although minimal risk is involved with the practical suggestions outlined in this chapter, future research is needed to identify which resources are most effective and to provide individualized recovery strategies in elite basketball players most specifically.

References

- Ziv G, Lidor R. Physical attributes, physiological characteristics, on-court performances and nutritional strategies of female and male basketball players. *Sports Med.* 2009;39:547–68.
- Cormery B, Marcil M, Bouvard M. Rule change incidence on physiological characteristics of elite basketball players: a 10-year-period investigation. *Br J Sports Med.* 2008;42:25–30.
- Ben Abdelkrim N, Chaouachi A, Chamari K, et al. Positional role and competitive-level differences in elite-level men's basketball players. *J Strength Cond Res.* 2010;24:1346–55.
- Ben Abdelkrim N, El Fazaa S, El Ati J. Time-motion analysis and physiological data of elite under-19-year-old basketball players during competition. *Br J Sports Med.* 2007;41:69–75.
- Delextrat A, Trochym E, Calleja-Gonzalez J. Effect of a typical in-season week on strength jump and sprint performances in national-level female basketball players. *J Sports Med Phys Fitness.* 2012;52:128–36.
- Moraska A. Sports massage: a comprehensive review. *J Sports Med Phys Fitness.* 2005;45:370–80.
- Jentjens R, Jeukendrup A. Determinants of post-exercise glycogen synthesis during short-term recovery. *Sports Med.* 2003;33:117–44.
- Montgomery PG, Pyne DB, Hopkins WG, et al. The effect of recovery strategies on physical performance and cumulative fatigue in competitive basketball. *J Sports Sci.* 2008;26:1135–45.
- Banfi G, Colombini A, Lombardi G, et al. Metabolic markers in sports medicine. *Adv Clin Chem.* 2012;56:1–54.
- Alaphilippe A, Mandigout S, Ratel S, et al. Longitudinal follow-up of biochemical markers of fatigue throughout a sporting season in young elite rugby players. *J Strength Cond Res.* 2012;26:3376–84.
- Bishop PA, Jones E, Woods AK. Recovery from training: a brief review. *J Strength Cond Res.* 2008;22:1015–24.
- Nédélec M, McCall A, Carling C, et al. Recovery in soccer: part II—recovery strategies. *Sports Med.* 2013;43:9–22.
- Gill N, Beaven C, Cook C. Effectiveness of post-match recovery strategies in rugby players. *Br J Sports Med.* 2006;40:260–3.
- Beelen M, Burke LM, Gibala MJ, et al. Nutritional strategies to promote postexercise recovery. *Int J Sport Nutr Exerc Metab.* 2010;20:515–32.
- Rodríguez NR, Di Marco NM, Langley S. American college of sports medicine position stand nutrition and athletic performance. *Med Sci Sports Exerc.* 2009;41:709–31.
- Koopman R, Wagenmakers AJ, Manders RJ, et al. Combined ingestion of protein and free leucine with carbohydrate increases postexercise muscle protein synthesis in vivo in male subjects. *Am J Physiol Endocrinol Metab.* 2005;288:E645–53.
- Schroder H, Navarro E, Mora J, et al. The type, amount, frequency and timing of dietary supplement use by elite players in the first Spanish basketball league. *J Sports Sci.* 2002;20:353–8.
- Bloomer RJ, Goldfarb AH, McKenzie MJ, et al. Effects of antioxidant therapy in women exposed to eccentric exercise. *Int J Sport Nutr Exerc Metab.* 2004;14:377–88.
- Packer L. Protective role of vitamin E in biological systems. *Am J Clin Nutr.* 1991;53:1050S–5S.
- Finaud J, Lac G, Filaire E. Oxidative stress: relationship with exercise and training. *Sports Med.* 2006;36:327–58.
- Narazaki K, Berg K, Stergiou N, et al. Physiological demands of competitive basketball. *Scand J Med Sci Sports.* 2009;19:425–32.
- Naziroglu M, Kilinc F, Uguz AC, et al. Oral vitamin C and E combination modulates blood lipid peroxidation and antioxidant vitamin levels in maximal exercising basketball players. *Cell Biochem Funct.* 2010;28:300–5.

23. Gualano B, Roschel H, Lancha-Jr AH, et al. In sickness and in health: the widespread application of creatine supplementation. *Amino Acids*. 2012;43:519–29.
24. Shi D. Oligosaccharide and creatine supplementation on glucose and urea nitrogen in blood and serum creatine kinase in basketball athletes. *J Huazhong Univ Sci Technolog Med Sci*. 2005;25:587–9.
25. Schroder H, Terrados N, Tramullas A. Risk assessment of the potential side effects of long-term creatine supplementation in team sport athletes. *Eur J Nutr*. 2005;44:255–61.
26. Bellinger PM. Beta-alanine supplementation for athletic performance: an update. *J Strength Cond Res*. 2014;28:1751–70.
27. Hoffman JR, Ratamess NA, Faigenbaum AD, et al. Short-duration beta-alanine supplementation increases training volume and reduces subjective feelings of fatigue in college football players. *Nutr Res*. 2008;28:31–5.
28. Sale C, Saunders B, Hudson S, et al. Effect of beta-alanine plus sodium bicarbonate on high-intensity cycling capacity. *Med Sci Sports Exerc*. 2011;43:1972–8.
29. Crisafulli A, Melis F, Tocco F, et al. External mechanical work versus oxidative energy consumption ratio during a basketball field test. *J Sports Med Phys Fitness*. 2002;42:409–17.
30. Saltin B. Metabolic fundamentals in exercise. *Med Sci Sports*. 1973;5:137–46.
31. Banfi G, Lombardi G, Colombini A, et al. Whole-body cryotherapy in athletes. *Sports Med*. 2010;40:509–17.
32. West D, Cunningham D, Bevan H, et al. Influence of active recovery on professional rugby union player's ability to harness postactivation potentiation. *J Sports Med Phys Fitness*. 2013;53:203–8.
33. Fletcher IM. The effect of different dynamic stretch velocities on jump performance. *Eur J Appl Physiol*. 2010;109:491–8.
34. Delextrat A, Hippocrate A, Leddington-Wright S, et al. Including stretches to a massage routine improves recovery from official matches in basketball players. *J Strength Cond Res*. 2014;28:716–27.
35. Leeder J, Gissane C, van Someren K, et al. Cold water immersion and recovery from strenuous exercise: a meta-analysis. *Br J Sports Med*. 2012;46:233–40.
36. Delextrat A, Calleja-González J, Hippocrate A, et al. Effects of sports massage and intermittent cold-water immersion on recovery from matches by basketball players. *J Sports Sci*. 2013;31:11–9.
37. de Glanville KM, Hamlin MJ. Positive effect of lower body compression garments on subsequent 40-km cycling time trial performance. *J Strength Cond Res*. 2012;26:480–6.
38. MacRae MBA, Cotter JD, Laing RM. Compression garments and exercise. *Sports Med*. 2011;41:815–43.
39. Zainuddin Z, Newton M, Sacco P, et al. Effects of massage on delayed-onset muscle soreness, swelling, and recovery of muscle function. *J Athl Train*. 2005;40:174–80.
40. Gustafsson H, Skoog T. The mediational role of perceived stress in the relation between optimism and burnout in competitive athletes. *Anxiety Stress Coping*. 2012;25:183–99.
41. Englert C, Bertrams A. Anxiety, ego depletion, and sports performance. *J Sport Exerc Psychol*. 2012;34:580–99.
42. Moreira A, McGuigan MR, Arruda AF, et al. Monitoring internal load parameters during simulated and official basketball matches. *J Strength Cond Res*. 2012;26:861–6.
43. Fullagar HH, Duffield R, Skorski S, et al. Sleep and recovery in team sport: current sleep-related issues facing professional team-sport athletes. *Int J Sports Physiol Perform*. 2015;10:950–7.
44. Steenland K, Deddens JA. Effect of travel and rest on performance of professional basketball players. *Sleep*. 1997;20:366–9.
45. Mah CD, Mah KE, Kezirian EJ, et al. The effects of sleep extension on the athletic performance of collegiate basketball players. *Sleep*. 2011;34:943–50.
46. Zhao J, Tian Y, Nie J, et al. Red light and the sleep quality and endurance performance of Chinese female basketball players. *J Athl Train*. 2012;47:673–8.
47. Lin Z, Lan LW, He T, et al. Effects of acupuncture stimulation on recovery ability of male elite basketball athletes. *Am J Chin Med*. 2009;37:471–81.
48. Doeven SH, Brink MS, Kosse SJ, Lemmink KA. Postmatch recovery of physical performance and biochemical markers in team ball sports: a systematic review. *BMJ Open Sport Exerc Med*. 2018;4:e000264.
49. Rowell AE, Aughey RJ, Hopkins WG, et al. Identification of sensitive measures of recovery after external load from football match play. *Int J Sports Physiol Perform*. 2017;12:969–76.
50. Pliauga V, Kamandulis S, Dargevičiūtė G, et al. The effect of a simulated basketball game on players' sprint and jump performance, temperature and muscle damage. *J Hum Kinet*. 2015;46:167–75.
51. Jones JJ, Kirschen GW, Kancharla S, Hale L. Association between late-night tweeting and next-day game performance among professional basketball players. *Sleep Health*. 2019;5:68–71.
52. Huyghe T, Scanlan A, Dalbo V, Calleja-González J. The negative influence of air travel on health and performance in the National Basketball Association: a narrative review. *Sports*. 2018;6:89.
53. Czeisler CA, Allan JS, Strogatz SH. Bright light resets the human circadian pacemaker independent of the timing of the sleep-wake cycle. *Science*. 1986;233:667–71.
54. Doherty R, Madigan S, Warrington G, Ellis J. Sleep and nutrition interactions: implications for athletes. *Nutrients*. 2019;11:822.
55. Milagres MP, Minim VP, Minim LA, Simiqueli AA, Moraes LE, Martino HS. Night milking adds value to cow's milk. *J Sci Food Agric*. 2014;94:1688–92.
56. Howatson G, Bell PG, Tallent J, Middleton B, McHugh MP, Ellis J. Effect of tart cherry juice

- (Prunus Cerasus) on melatonin levels and enhanced sleep quality. *Eur J Nutr.* 2012;51:909–16.
57. Bonnar D, Bartel K, Kakoschke N, Lang C. Sleep interventions designed to improve athletic performance and recovery: a systematic review of current approaches. *Sports Med.* 2018;48:683–703.
 58. Cannell JI, Hollis BW, Sorenson MB, Taft TN, Anderson JI. Athletic performance and vitamin D. *Med Sci Sports Exerc.* 2009;41:1102–10.
 59. Willis KS, Peterson NJ, Larson-Meyer DE. Should we be concerned about the vitamin D status of athletes? *Int J Sport Nutr Exerc Metab.* 2008;18:204–24.
 60. Fishman MP, Lombardo SJ, Kharrazi FD. Vitamin D deficiency among professional basketball players. *Orthop J Sports Med.* 2016;4:2325967116655742.
 61. Garcia RB, Guisado FR. Low levels of vitamin D in professional basketball players after wintertime: relationship with dietary intake of vitamin D and calcium. *Nutr Hosp.* 2011;26:945–51.
 62. Bohl CH, Volpe SL. Magnesium and exercise. *Crit Rev Food Sci Nutr.* 2002;42:533–63.
 63. Alfredo C, Diego F, Juan M, Calvo S, Jesús CGA. Effect of magnesium supplementation on muscular damage markers in basketball players during a full season. *Magnes Res.* 2017;30:61–70.
 64. Osterberg KL, Horswill CA, Baker LB. Pregame urine specific gravity and fluid intake by National Basketball Association players during competition. *J Athl Train.* 2009;44:53–7.
 65. Maszczyk A, Anna K, Tomasz Z, Chycki J, Adam M. Anaerobic performance and acid-base balance in basketball players after the consumption of highly alkaline water. *Int J Food Sci Nutr.* 2018;5:134–9.
 66. Sawka MN, Burke LM, Eichner ER, Maughan RJ, Montain SJ, Stachenfeld NS. American College of Sports Medicine position stand. Exercise and fluid replacement. *Med Sci Sports Exerc.* 2007;39:377–90.
 67. Díaz-Castro F, Astudillo S, Calleja-González J, Zbinden-Foncea H, Ramirez-Campillo R, Castro-Sepúlveda M. Change in marker of hydration correspond to decrement in lower body power following basketball match. *Sci Sport.* 2018;33:e123–8.
 68. Baker LB, Dougherty KA, Chow M, Kenney WL. Progressive dehydration causes a progressive decline in basketball skill performance. *Med Sci Sports Exerc.* 2007;39:1114–23.
 69. Shirreffs SM, Maughan RJ. Whole body sweat collection in humans: an improved method with preliminary data on electrolyte content. *J Appl Physiol.* 1997;82:336–41.
 70. Machek SB, Bagley JR. Creatine monohydrate supplementation: considerations for cognitive performance in athletes. *Strength Cond J.* 2018;40:82–93.
 71. Mielgo-Ayuso J, Calleja-Gonzalez J, Marqués-Jiménez D, Caballero-García A, Córdova A, Fernández-Lázaro D. Effects of creatine supplementation on athletic performance in soccer players: a systematic review and meta-analysis. *Nutrients.* 2019;11:757.
 72. Buford TW, Kreider RB, Stout JR. International Society of Sports Nutrition position stand: creatine supplementation and exercise. *J Int Soc Sports Nutr.* 2007;4:6.
 73. Ansdell P, Deckerle J. Sodium bicarbonate supplementation delays neuromuscular fatigue without changes in performance outcomes during a basketball match simulation protocol. *J Strength Cond Res.* 2017; <https://doi.org/10.1519/JSC.0000000000002233>.
 74. Dupuy O, Douzi W, Theurot D, Bosquet L, Dugué B. An evidence-based approach for choosing post-exercise recovery techniques to reduce markers of muscle damage, soreness, fatigue, and inflammation: a systematic review with meta-analysis. *Front Physiol.* 2018;9:403.
 75. Suzuki M, Umeda T, Nakaji S, Shimoyama T, Mashiko T, Sugawar K. Effect of incorporating low intensity exercise into the recovery period after a rugby match. *Br J Sports Med.* 2004;38:436–40.
 76. Broodryk A, Coetzee B, Pienaar C, Spark M. Post-exercise effects of cold water immersion and contrast water therapy-part 2: acute effects of contrast water therapy and passive recovery on the physical and haematological parameters in male university rugby players over a 48-hour recovery period. *Afr J Phys Health.* 2017;23:371–85.
 77. Marqués-Jiménez D, Calleja-González J, Arratibel I, Delextrat A, Terrados N. Are compression garments effective for the recovery of exercise-induced muscle damage? A systematic review with meta-analysis. *Physiol Behav.* 2016;153:133–48.
 78. Brown F, Gissane C, Howatson G, Van Someren K, Pedlar C, Hill J. Compression garments and recovery from exercise: a meta-analysis. *Sports Med.* 2017;47:2245–67.
 79. Fonda B, Sarabon N. Effects of whole-body cryotherapy on recovery after hamstring damaging exercise: a crossover study. *Scand J Med Sci Sports.* 2013;23:e270–8.
 80. Bouzignon R, Ravier G, Dugue B, Grappe F. Thermal sensations during a partial-body cryostimulation exposure in elite basketball players. *J Hum Kinet.* 2018;62:55–63.
 81. Douzi W, Dupuy O, Tanneau M, Boucard G, Bouzignon R, Dugué B. 3-min whole body cryotherapy/cryostimulation after training in the evening improves sleep quality in physically active men. *Eur J Sport Sci.* 2019;19:860–7.
 82. Moreira A, Aoki MS, Franchini E, da Silva Machado DG, Paludo AC, Okano AH. Mental fatigue impairs technical performance and alters neuroendocrine and autonomic responses in elite young basketball players. *Physiol Behav.* 2018;196:112–8.
 83. Loch F, Ferrauti A, Meyer T, Pfeiffer M, Kellmann M. Resting the mind—a novel topic with scarce insights. Considering potential mental recovery strategies for short rest periods in sports. *Perform Enhanc Health.* 2019;6:148–55.

84. Tandya L, Mihardja H, Srilestari A, Kurniarobbi J, Kurniawan A. Effect of acupuncture on decreasing blood lactate levels after exercise in elite basketball athletes. *J Phys Conf Ser.* 2018;1073(6):062050. <https://iopscience.iop.org/article/10.1088/1742-6596/1073/6/062050/meta>. Accessed 9 July 2019
85. Rossi A, Blaustein S, Brown J, Dieffenderfer K, Ervin E, Griffin S, Johanson M, et al. Spinal and peripheral dry needling versus peripheral dry needling alone among individuals with a history of lateral ankle sprain: a randomized controlled trial. *Int J Sports Phys Ther.* 2017;12:1034.
86. Broatch J, Bishop D, Zadow E, Halson S. Effects of sports compression socks on performance, physiological, and hematological alterations after long-haul air travel in elite female volleyballers. *J Strength Cond Res.* 2019;33:492–501.
87. Kaesaman N, Eungpinichpong W. The acute effect of traditional Thai massage on recovery from fatigue in basketball players. *Int J.* 2019;16:53–8.



Tim J. Gabbett

64.1 What Is “Load”?

Imagine you work as part of the Sports Medicine team for a major National Basketball Association (NBA) franchise. This franchise has consistently been the most successful team over the past 5 years. Leading into the play-offs, one of your key players misses a month of games due to a calf injury. The team soldiers on through the play-offs, winning its way through to the NBA finals. However, despite having a star-studded roster, the team finds itself trailing 3-1 in the best of seven series. With the finals’ series on the line, the team decides to roll the dice! The best player has to return. The team wins the fifth game of the series by 1 point. The only problem is that the key player was only able to play 12 min before rupturing his Achilles tendon. Your opponent goes on to win the sixth game of the series and become the 2019 NBA Champions—but a much bigger media storm emerges. Was he “carrying” a pre-existing injury? Was he pressured to play? Did he know the risks? Had he performed enough training to return to play safely?

“Load” has been described in many ways, but in general it can be defined as follows: “the act of per-

forming ‘work’ (either training or competition) in preparation for or carrying out an event.” This “work” is referred to as *external load*. Load is also described in terms of the stress it places on the organism (e.g., human). This “stress” can take the form of physiological, biomechanical, or psychological—and is referred to as *internal load*. Finally, an athlete’s ability to tolerate load is influenced by several factors including (but not necessarily limited to) age, fitness, sleep, nutrition, and external stressors [1].

The balance between load and load capacity (also referred to as tissue capacity) is thought to play a significant role in injury causation [2]. When the load that is applied to a tissue greatly exceeds that tissue’s capacity, injury risk is heightened. Complicating the load-capacity issue is that for a tissue to adapt and load capacity to improve, gradual progressions in load that are slightly greater than current tissue capacity are required. Therefore, for improvements in load capacity to occur, load must be increased—but not so much as to injure the tissue (and ultimately decrease load capacity). A final and often neglected part of the load-capacity puzzle is consideration of the athlete’s health. The load that an athlete can tolerate today could be quite different to tomorrow, simply due to decreases (or increases) in health (Fig. 64.1) [3]. These factors are particularly complex for professional basketball players as they navigate a tortuous 82-game season, such as in the NBA, involving back-to-back games and multiple games in a week, all the while negotiating competition stress, team (and even spectator) expectations, as

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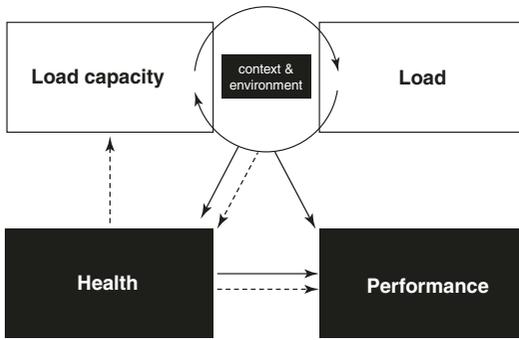


Fig. 64.1 An integrated view on load, load capacity, performance and health in sports. Dotted lines represent negative relationships and solid lines represent positive relationships Redrawn from [3]

well as travel and sleep disturbances associated with changing time zones.

64.2 What Is “Load Management” All About? And Why Is It Important in Basketball?

Anyone who has worked with sports coaches understands they have competing demands. Not only are they required to devise an overall strategy for the team to execute, but they need to liaise with administrators (e.g., the owner and General Manager), sponsors, and media, manage rosters, assistant coaches, and individual athletes (and their unique personalities), and play a key role in the day-to-day planning of technical, tactical (and sometimes) strength, and conditioning sessions. There is no doubt that the principles of load management are important for coaches to understand, but *coaches are generally are time poor!* With all of the athlete monitoring and workload management resources that are available, it is not surprising when coaches state “*Just tell me what I need to know!*”

64.3 How Do I Capture Load?

With the variety of skills and physical activities, players are required to perform throughout a basketball game; it is unlikely that one single variable (also commonly referred to as a “*metric*”) will capture basketball-specific load. Equally, for various reasons, both external and internal load have proven

difficult to capture in both youth and professional basketball. First, youth teams rarely have access to wearable technology (e.g., inertial measurement sensors) to measure external load—the technology is expensive and typically requires skilled practitioners to interpret and apply the information. Even though professional players often have access to this technology and are encouraged to wear them in practice, their use is not permitted in NBA games. While some external load data (e.g., number of accelerations/decelerations performed, and distances covered) can be captured by Second-Spectrum (company that provides (video-based tracking of players in NBA games), other important activities (e.g., changes of direction, jumping, and blocking) cannot. Equally, the two technologies (i.e., video and inertial sensors) will provide *similar but not identical data*, meaning that the data used to quantify training load may be different from the data used to quantify game load. Second, if the above challenges surrounding the capture of external load prove insurmountable, then practitioners can measure the internal load of athletes. Heart rate monitors (another wearable sensor) have been used extensively to quantify internal load, although perhaps the most common form of internal load monitoring is through the use of a session rating of perceived exertion (s-RPE) [4].

First described by Foster et al. [5], the s-RPE requires players to provide a subjective estimate of intensity using a 0 (rest) to 10 (maximal effort) scale (Table 64.1). This intensity score is multiplied by

Table 64.1 Session-rating of perceived exertion scale [6]

Rating	Descriptor ^a
0	Rest
1	Very, very easy
2	Easy
3	Moderate
4	Somewhat hard
5	Hard
6	–
7	Very hard
8	–
9	–
10	Maximal

^aThe verbal anchors have been changed slightly to reflect American English (e.g., light becomes easy; strong or severe becomes hard), Briefly, the subject is shown the scale approximately 30 min following the conclusion of the training bout and asked, “How was your workout?”

Table 64.2 Calculation of training load, monotony, and strain over a training week for a basketball player

Session	Mon	Tue	Wed	Thu	Fri	Sat	Sun
Session 1	Practice	Shoot around	Off	Weights	Practice	Shoot around	Off
<i>Intensity/duration</i>	6 × 90	2 × 60	0 × 0	7 × 45	5 × 75	2 × 50	0 × 0
Load	540	120	0	315	375	100	0
Session 2	Game	Game	Practice	Shoot Around	Game	Game	Off
<i>Intensity/duration</i>	9 × 48	9 × 48	6 × 90	2 × 60	7 × 42	9 × 48	0 × 0
Load	432	432	540	120	294	432	0
Daily load	972	552	540	435	669	532	0
Total sessions	11						
Total duration	656						
Weekly load	3700	Weekly load = sum of 7 day load					
Monotony	1.82	Monotony = average weekly load/standard deviation of weekly load					
Strain	6740	Strain = weekly load × monotony					

the duration of practice or competition to provide “Load” for that session. “Monotony” and “Strain” can also be calculated to provide an indication of the amount of variation within a training week. The higher the monotony and strain scores, the less variation (and likely recovery) within the training week. Load is a function of frequency, volume, and intensity. As such, increases or decreases of load can be made by manipulating either frequency, volume, and/or intensity. An example of weekly training load for a professional basketball player and the calculations for Training *Load*, *Monotony*, and *Strain* are shown in Table 64.2. Although not without limitations, the s-RPE allows coaches and sports medicine staff to quantify internal load with minimal resources.

64.4 Load Can Have Positive and Negative Effects

Acute training load represents the short-term “fatigue” that arises from training, while chronic training load is analogous to “fitness.” As stated above, injury occurs when load exceeds the ability of the tissue to adapt (i.e., when load is greater than load capacity). The size of the short-term training load (anywhere from one session to 1 week) (termed *acute training load*) in relation to longer term training load (termed *chronic training load*) is used to calculate the “*acute:chronic workload ratio*” (ACWR). Consequently, for many years it was believed that injuries occurred as a result of high training loads. However, a large number of studies have recently shown that high chronic loads are associated with lower injury risk [7]. These findings have been confirmed across

multiple sports and research groups. Of equal interest is the influence of high chronic load on performance. In the mid-1990s, Foster et al. [5] studied the performance of runners, cyclists, and speed skaters, and demonstrated that performance was closely linked to training load; athletes with higher training loads had faster time-trial performances. Team sport performance can also be explained, at least in part by training load [8, 9]. It appears that appropriately high chronic training loads reduce injury risk and enhances athlete performance in several ways. Firstly, *exposure to load helps athletes withstand subsequent load*. Secondly, appropriately prescribed training develops physical qualities that not only protects against injury but also allows athletes to perform the high-intensity tasks required of competition.

64.5 How Can Practitioners Safely Progress Workload?

Studies of team sport athletes have shown that when the ACWR was within the range of 0.8–1.3 (i.e., the acute training load was approximately equal to the chronic training load), the risk of injury was relatively low. However, when the ACWR was ≥ 1.5 (i.e., the acute training load [or “fatigue”] was much greater than chronic training load [or “fitness”]), the risk of injury increased markedly [7]. Because spikes in workload that result in a large ACWR increase injury risk, strength coaches, physical therapists, athletic trainers, and even sport coaches have embraced measurements of the ACWR. The ACWR uses the common training principle of *progressive over-*

load. It allows practitioners working with patients and athletes to safely progress and regress training loads, based on the individual's load capacity.

64.6 Spikes in Load Increase Injury Risk

Some of the first publications that examined the relationship between training load and injury were performed in cricket, rugby league, and Australian football (all very popular Australian sports). These studies all found similar results—(1) *high training loads* were associated with *lower injury risk*, but (2) *rapid increases* (i.e., *spikes*) in training loads were associated with *greater injury risk* (Fig. 64.2) [7]. The obvious question that arose from these studies was whether these findings could be replicated in other sports. For example, how well do the findings from a collision sport like rugby league transfer to a completely different sport such as basketball? In a recent study, Caparros et al. [10] demonstrated that low external workloads were associated with higher injury risk in professional male basketball players. Presumably, the low chronic loads leave players underprepared for the

high physical demands associated with competition. These findings of low chronic loads and spikes in load increasing injury risk have subsequently been replicated by over 20 different research groups from a wide range of sports, including basketball [11, 12].

The NBA season spans 82 games; basketball players need to be well-conditioned to tolerate the running, jumping, and change of direction loads required during the season. However, over the off-season many players completely break from training—having a 3-month period where they do not train at all. When the off-season concludes, it is not uncommon for these players to immediately re-engage with their in-season levels of training, drastically spiking their on-court loads. Injury rates are substantially higher when players spike their load in this way following the off-season. Better practice would result in (1) maintaining a minimal standard of training load (and hence fitness) over the off-season, and (2) gradually increasing the training load following the off-season. In this respect, we are not discussing “overuse injuries,” but rather we are referring to injuries that occur as a result of *under-preparation*.

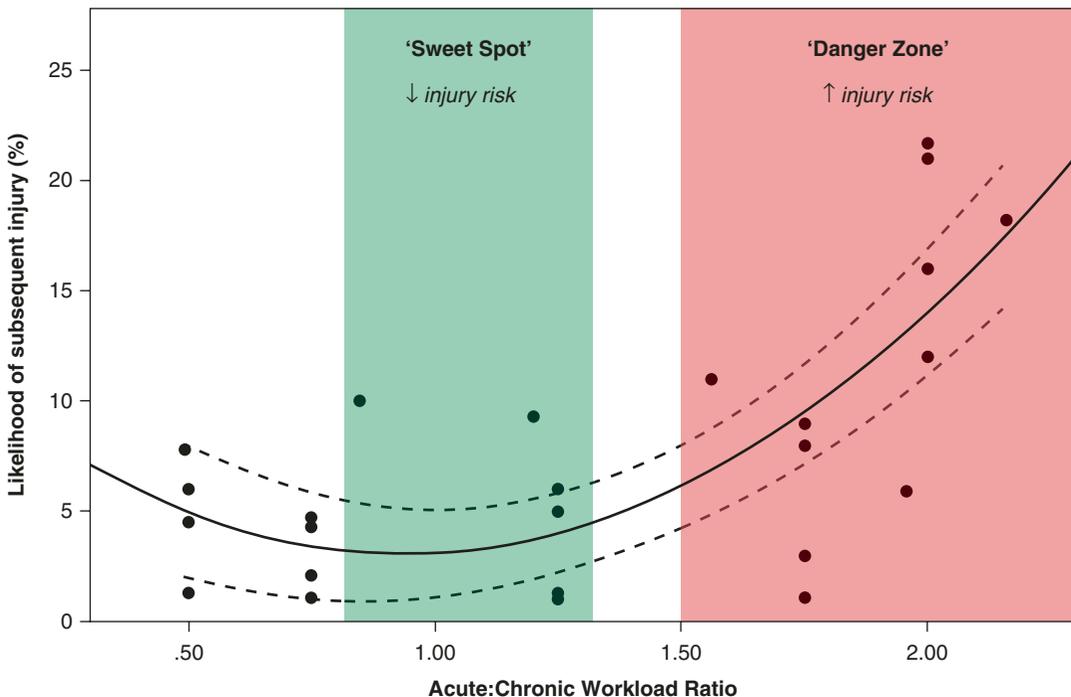


Fig. 64.2 The acute:chronic workload ratio and injury risk in athletes [7]

64.7 Prepare for the “Worst Case Scenario”

The “worst case scenario” refers to the most demanding passage of match play. These passages of play occur at critical moments of competition (e.g., shots on goal, goals scored or conceded), and are often termed “repeated-sprint” (e.g., football and field hockey) or “repeated high-intensity effort” (e.g., basketball, rugby, and ice hockey) bouts. The ability (or inability) to perform these repeated high-intensity efforts often proves critical to the outcome of competition. Indeed, in other team sports it has been shown that a large percentage of points scored in competition occurred in close proximity to a repeated high-intensity effort bout [13]. *These demanding passages of play should therefore be viewed as an “opportunity” to gain ascendancy over an opponent.* Exposing athletes to the most demanding passages of play in training on a regular basis (with adequate recovery between bouts and sessions) improves their ability to tolerate those demands in competition. After all, if training programs focus on the average demands of competition and neglect to target

the most demanding passages of play, then at best, athletes can only ever be prepared for 50% of competition (Fig. 64.3). When developing a “load management” plan, practitioners are advised to remember the *training* aspect of “training monitoring”—*athletes need to load in order to withstand load!*

64.8 The Risk-Reward Conundrum—“Is He Ready to Play or Not?!”

Returning to the example, we can see that based on the evidence, the sports medicine staff faced a significant dilemma. Even if the athlete was able to perform some training during rehabilitation, having been injured for 1 month severely limited his game time, resulting in a gradual reduction in chronic load (i.e., the player was likely detraining). This likely resulted in a double dilemma—(1) the higher chronic load that previously “protected” the athlete had diminished, and (2) any subsequent load that was applied represented a “spike” relative to his current capacity.

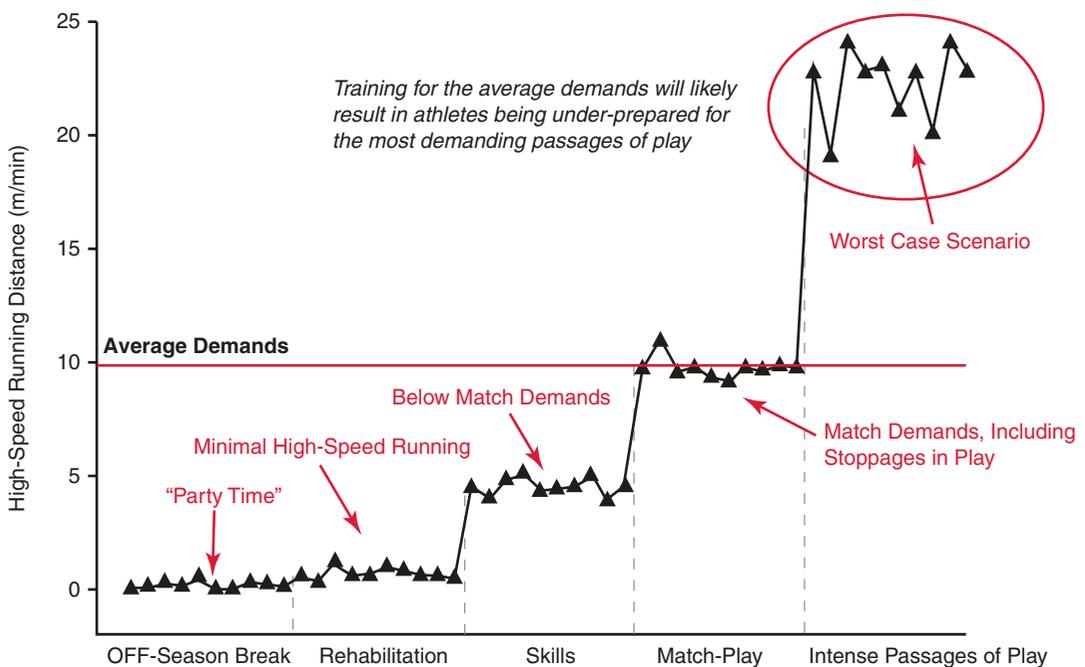


Fig. 64.3 An example of the most demanding passage of play (or “worst case scenario”) for a team sport athlete [14]

64.8.1 Return-to-Play Decisions Require Ethics but They also Require Context

Apart from making return-to-play decisions within an ethical and medically sound framework, what contextual factors *might* influence decision-making when returning an athlete to competition? First, the quality of the athlete will impact decision-making—if the player is a potential match-winner, then it changes the decision! Teams might be willing to risk a key athlete under some circumstances, and not in others. Second, the timing of the injury will also influence decision-making. If the injury was sustained in the first game of the season, then the sports medicine team may not have faced the same level of pressure and scrutiny that they faced in a potential “finals deciding” game. Clearly, return-to-play decisions encompass the “risk-reward” question. What are the potential risks? And what is the potential reward?

64.8.2 Do You Want the Athlete Back Quickly or “Permanently”?

If loading strategies can influence injury risk, a second and equally important consideration for sports medicine staff is whether they return the athlete to sport “quickly” or “permanently.” This is a critical consideration as the answer will influence the loading strategies that are employed. For example, Australian football research has shown that accruing lower chronic loads can hasten return-to-play; however, the lower chronic load is also associated with a greater risk of subsequent injury [15]. Higher chronic loads are associated with lower subsequent injury risk, but the return-to-play time is delayed, as it takes longer to develop the high chronic loads to protect against subsequent injury! [15] Clearly, when making return-to-play decisions, sports medicine staff must balance the risks associated with early return against the potential benefits of having that player on the court.

64.9 How Can We Develop a Robust Athlete?

As stated above, in contrast to early views, there is a growing body of evidence demonstrating that low chronic workloads increase injury risk [16]. These findings suggest that *progressing to higher chronic loads* would be a preferable approach to improving athlete availability than reducing training loads. But sport performance and medicine staff are not only responsible for keeping athletes available—those athletes must be ready to perform when required. In line with load-injury studies, recent evidence has shown that athletes with higher chronic loads and fewer “spikes” in load also demonstrate *greater readiness to perform* [17]. These findings highlight the performance-enhancing capabilities of an effective load management program. While it is recognized that a myriad of factors other than workload can impact both injury risk [18] and performance [17], the absence of an effective loading program is akin to shooting at a target while blindfolded.

How can sports medicine staff avoid spikes in workload if they have no information on the athlete’s training history?

How can an athletic trainer, physical therapist, or strength and conditioning coach progress to higher workloads if they do not measure workload?

Given the relationship among training load, athlete availability, and readiness to perform, how can an athlete have the best chance of achieving their performance potential and remaining injury-free if appropriate loading strategies do not underpin their training program?

64.10 What About “Fitness” and Tolerance to Workload?

Although spikes in workload may contribute to injuries, “load” does not explain all injuries. Furthermore, some players are more resilient to spikes in workload, while others are more vul-

nerable. In some cases, spikes in workload are unavoidable (e.g., a key player returning rapidly from injury, multiple double overtime games in the play-offs). If spikes in workload are unavoidable, what practical steps can performance and medical staff take to mitigate injury risk and give players the best chance of competitive success?

First, if medical staff know that loads are likely to increase in the near future (e.g., schedule changing from one game to multiple games in a week), then raising chronic load in the weeks prior to that change in schedule will likely improve load tolerance. This *pre-loading* raises chronic load, thereby reducing the gap between the “floor” and the “ceiling”—it is much harder to spike loads from the “ceiling” than the “floor” because when chronic load is low the only way is up! Second, well-developed physical qualities (e.g., eccentric hamstring strength) have been shown to independently decrease injury risk. Third, specific physical qualities (e.g., lower body strength, speed, and aerobic fitness) *moderate* the workload–injury relationship—players with better developed aerobic fitness, speed, and lower-body strength are more resilient to spikes in workload than players with poorly developed physical qualities [19, 20] (Fig. 64.4).

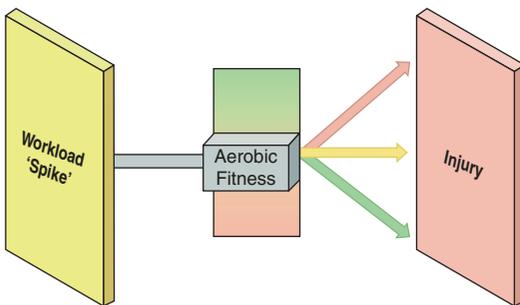


Fig. 64.4 An example of how physical qualities (in this case, aerobic fitness) moderate the workload–injury relationship [20]. The workload–injury relationship is also moderated by speed, repeated-sprint ability, and lower body strength [19]

Table 64.3 Potential practical applications for two different athletes with similar workloads

Moderator	When exposed to a “spike” in workload	
Age/training age	Very young/very old	Middle of career
Injury history	Extensive	Limited
Training history	Poor, inconsistent	Good, consistent
Speed	Slow	Moderate-fast
Aerobic fitness	Unfit	Fit
Lower-body strength	Weak	Strong
Practical application	Monitor recovery and modify training if required	Monitor recovery and continue to train and play

NB “Modifying the training program” does not necessarily mean “providing more rest.”

If absolute load (either the “floor” or “ceiling”) cannot be modified in the short term, then performance and medical staff can use physical testing results to stratify players into well-defined risk groups based on particular loading patterns. An example of different *moderators* of the workload–injury relationship and suggestions to practically manage *two different athletes with similar workloads* is provided in Table 64.3.

Take Home Message

With the myriad of factors that can influence injury risk and performance, at this stage, the best we can offer is an educated guess. Although these incidents will always divide opinion, most practitioners agree that successful training programs are underpinned by effective loading strategies which consider external stressors that may impact load tolerance. A well-structured, systematic training program that gradually exposes athletes to higher loads while considering the moderators of load tolerance represents a best-practice approach to developing load capacity.

References

1. Soligard T, Schwelunus M, Alonso J, Bahr R, Clarsen B, Dijkstra P, et al. International Olympic Committee consensus statement on load in sport and risk of injury: how much is too much? *Br J Sports Med.* 2016;50:1030–41.
2. Kibler WB, Chandler TJ, Stracener ES. Musculoskeletal adaptations and injuries due to overtraining. *Exerc Sport Sci Rev.* 1992;20:99–126.
3. Verhagen E, Gabbett T. Load, capacity and health: critical pieces of the holistic performance puzzle. *Br J Sports Med.* 2019;53:5–6.
4. Foster C. Monitoring training in athletes with special reference to overtraining syndrome. *Med Sci Sports Exerc.* 1998;30:1164–8.
5. Foster C, Daines E, Hector L, Snyder AC, Welsh R. Athletic performance in relation to training load. *Wis Med J.* 1996;95:370–4.
6. Sweet TW, Foster C, McGuigan MR, Brice G. Quantitation of resistance training using the session rating of perceived exertion method. *J Strength Cond Res.* 2004;18:796–802.
7. Gabbett TJ. The training—injury prevention paradox: should athletes be training smarter and harder? *Br J Sports Med.* 2016;50:273–80.
8. Aughey RJ, Elias GP, Esmaili A, Lazarus B, Stewart AM. Does the recent internal load and strain on players affect match outcome in elite Australian football? *J Sci Med Sport.* 2016;19:182–6.
9. Lazarus BH, Stewart AM, White KM, Rowell AE, Esmaili A, Hopkins WG, et al. Proposal of a global training load measure predicting match performance in elite sport. *Front Physiol.* 2017;8:930. <https://doi.org/10.3389/fphys.2017.00930>. eCollection
10. Caparros T, Casals M, Solana A, Pena J. Low external workloads are related to higher injury risk in professional male basketball games. *J Sports Sci Med.* 2018;17:289–97.
11. Anderson L, Triplett-McBride T, Foster C, Doberstein S, Brice G. Impact of training patterns on incidence of illness and injury during a women's collegiate basketball season. *J Strength Cond Res.* 2003;17:734–8.
12. Weiss KJ, Allen SV, McGuigan MR, Whatman CS. The relationship between training load and injury in men's professional basketball players. *Int J Sports Physiol Perform.* 2017;12:1238–42.
13. Gabbett TJ, Gahan CW. Repeated high-intensity effort activity in relation to tries scored and conceded during rugby league match-play. *Int J Sports Physiol Perform.* 2016;11:530–4.
14. Gabbett TJ, Kennelly S, Sheehan J, Hawkins R, Milsom J, King E, et al. If overuse injury is a “training load error” should undertraining be viewed the same way? *Br J Sports Med.* 2016;50:1017–8.
15. Stares J, Dawson B, Peeling P, Drew M, Heasman J, Rogalski B, et al. How much is enough in rehabilitation? High running workloads following lower limb muscle injury delay return to play but protect against subsequent injury. *J Sci Med Sport.* 2018;21:1019–24.
16. Gabbett TJ. Debunking the myths about training load, injury and performance: empirical evidence, hot topics and recommendations for practitioners. *Br J Sports Med.* 2020;54:58–66. <https://doi.org/10.1136/bjsports-2018-099784>.
17. Hulin BT, Gabbett TJ, Pickworth NJ, Johnston RD, Jenkins DG. Relationships Among PlayerLoad, High-Intensity Intermittent Running Ability, and Injury Risk in Professional Rugby League Players. *Int J Sports Physiol Perform.* 2019;14:1–7. <https://doi.org/10.1123/ijsp.2019-0139>. [Epub ahead of print].
18. Bittencourt NF, Meeuwisse WH, Mendonca LD, Nettel-Aguirre A, Ocarino JM, Fonseca ST. Complex systems approach for sports injuries: moving from risk factor identification to injury pattern recognition—narrative review and new concept. *Br J Sports Med.* 2016;50:1309–14.
19. Malone S, Hughes B, Doran DA, Collins K, Gabbett TJ. Can the workload-injury relationship be moderated by improved strength, speed and repeated-sprint qualities? *J Sci Med Sport.* 2019;22:29–34.
20. Windt J, Zumbo BD, Sporer B, MacDonald K, Gabbett TJ. Why do workload spikes cause injuries, and which athletes are at higher risk? Mediators and moderators in workload—injury investigations. *Br J Sports Med.* 2017;51:993–4.



Practical Considerations for Workload Measurement in Basketball

65

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65.1 Monitoring Player Workloads in Basketball

Monitoring is important to quantify the demands encountered by basketball players during training and games. Specifically, monitoring workloads across pre- and in-season phases is encouraged to thoroughly understand the demands placed on players across the season [1, 2]. However, basketball coaches and performance staff must also consider the validity and reliability of the available workload metrics, in addition to the practical utility and limitations associated with collecting and analysing player workload data using each method.

Workload metrics can be classified as either external or internal. External workload refers to

the training and game stimuli imposed, while internal workload represents the physiological and perceptual reactions of players to the external demands [3]. Internal workload will ultimately determine training responses and physiological adaptations, directly impacting performance-related outcomes; however, external workloads must be manipulated by basketball coaches and performance staff to bring about the desired responses from players [2]. Consequently, workload data can be used most effectively where external and internal methods are applied and considered together [4].

65.2 Monitoring External Workload

In basketball, external workload has historically been assessed via video-based time–motion analysis and reported as speed, distance and movement frequencies. While time–motion data demonstrate acceptable validity [5] and reliability [6], data analysis is time-consuming. Therefore, video-based time–motion analysis is typically not frequently utilised in practice to quantify player workload in basketball as many teams do not have access to the necessary performance staff and resources needed to efficiently process data [1]. Consequently, external workloads are typically monitored in applied environments using microsensors. In basketball, microsensors

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refer to devices containing accelerometers and various inertial sensors such as gyroscopes and magnetometers. Tri-axial accelerometers are the primary components utilised in microsensors and measure accumulated load based on the instantaneous rate of change in acceleration across the x , y and z axes. Inertial sensors on the other hand provide information relating to orientation and direction of travel to calculate movement frequencies, which are estimated using proprietary algorithms.

Microsensors are the most commonly used devices for objectively measuring external workload in basketball. Microsensors demonstrate acceptable validity and reliability [7]. In addition, microsensors present a non-invasive method of monitoring players, while also providing data that can be quickly analysed using proprietary software for routine implementation. However, a primary limitation with using microsensors is key workload metrics derived from the accelerometer component are typically reported in arbitrary units. Accelerometer-derived external workload can therefore be difficult to interpret compared to established metrics such as speed and distance. Therefore, combining accelerometer-derived workload metrics with movement counts captured via inertial sensors may optimise interpretation of microsensor-derived external workloads. Specifically, inertial sensors can quantify the frequency of movements performed including jumps, accelerations, decelerations and changes in direction, which are typically physically demanding activities in basketball. From a practical perspective, accelerometers and inertial sensors are typically combined in the same device to be collected simultaneously and subsequently analysed together using the same proprietary software.

While microsensors are the most commonly utilised approach to monitor external workload in basketball, local positioning systems are an emerging technology available to use. Local positioning systems utilise a combination of indoor nodes placed around the training or game venue, and microsensors worn by players. Indoor nodes receive player tracking data from the microsensors and determine the time taken for each node

to receive the signals. These data are used to estimate position and displacement of players, which is reported via kinematic parameters such as speed, distance and movement frequencies. Data processing and analysis using proprietary software with local positioning systems is also efficient. Furthermore, the acquisition of kinematic data with recognisable units from local positioning systems may enhance interpretability by basketball coaches and performance staff compared to microsensors. However, the validity and reliability of local positioning systems for monitoring external workload data in basketball have not been assessed. In addition, local positioning systems are in their infancy and as such are typically more expensive than other microsensor monitoring systems, which may reduce their accessibility until they become more affordable for a wider range of teams. Accordingly, local positioning systems represent a promising future direction but should not yet replace conventional microsensors.

65.3 Monitoring Internal Workload

Internal workload can be monitored via objective or subjective metrics, each with respective advantages and limitations. Objective internal workload is most frequently quantified using heart rate monitoring, which is non-invasive and efficient in terms of data processing using proprietary software or customised spreadsheets [1]. However, heart rate reported simply as beats·min⁻¹, or % HR_{max} may underestimate the intensity of the training session or game in basketball [8]. Therefore, heart rate responses inputted into workload models may counter this limitation and are therefore typically implemented in basketball. Use of heart rate-derived workload models offers practical and convenient data collection and processing procedures and provides internal workload metrics, which capture the responses of players to the external stimuli imposed.

A foundation heart rate workload model is Banister's training impulse (TRIMP), which incorporates individualised resting and maximum

heart rate responses, along with the average heart rate response across the session. Using Banister's TRIMP, internal workload is then calculated using a pre-determined exponential relationship between heart rate and blood lactate concentration during incremental exercise, with higher intensities given larger weightings [9]. While Banister's TRIMP only requires heart rate data, the relationship between heart rate and blood lactate concentration across exercise intensities varies between individuals, which is not accounted for in Banister's model. To overcome this limitation, Lucia's TRIMP [10] has been applied in basketball [11] and incorporates three individualised heart rate zones corresponding to blood lactate concentrations at $<2.5 \text{ mmol L}^{-1}$, $2.5\text{--}4 \text{ mmol L}^{-1}$ and $>4 \text{ mmol L}^{-1}$. However, Lucia's TRIMP requires specialised expertise to conduct additional exercise testing, typically in laboratory settings, involving invasive blood sampling to determine lactate concentrations [1]. In addition, the relationship between heart rate and blood lactate may change with fluctuations in anaerobic and aerobic fitness, therefore requiring repeated testing across the season. Based on these considerations, Lucia's TRIMP is not widely implemented to monitor internal workload in basketball due to the practical restrictions involved.

Given the aforementioned limitations of Banister's and Lucia's TRIMP, the most common heart rate workload model applied in basketball is the Summated-Heart-Rate-Zones (SHRZ) model [12]. In the SHRZ model, exercise duration (in minutes) is combined with exercise intensity using weighted heart rate zones. Specifically, each heart rate response is placed into pre-defined zones that incrementally increase by $10\% \text{ HR}_{\text{max}}$, starting at $50\% \text{ HR}_{\text{max}}$. Time spent in each zone is then multiplied by corresponding weightings from 1 to 5, with the sum of the accumulated weightings taken as the SHRZ workload. The SHRZ model can detect periodised increases in workload in basketball players [13]. Furthermore, the data used in the SHRZ model are simple to collect and can be efficiently processed and analysed using proprietary software or customised spreadsheets. Consequently, the

sensitivity and practical advantages of the SHRZ model support its use for routine player monitoring in basketball [1]. The primary limitation of SHRZ however is that heart rate responses are categorised rather than being treated as continuous. In this regard, heart rate responses that differ by only $1 \text{ beat}\cdot\text{min}^{-1}$ may be assigned different weightings in the SHRZ model. While the use of smaller zones (e.g. 2.5%) with less pronounced jumps in weighting has been explored to negate this limitation [14], more work may be needed to identify an optimal approach to calculate the SHRZ workload in basketball.

Subjective internal workload is typically assessed via the session rating of perceived exertion (sRPE) method. sRPE is a product of session duration and self-reported rating of perceived exertion, generally using the 0–10 Category-Ratio scale [15, 16]. Unlike objective measures that capture physiological responses to exercise, sRPE is sensitive to a range of stimuli including external and internal workloads, as well as psychological inputs such as stress, arousal and fatigue [3]. Given the multifaceted nature of sRPE, using this method in isolation can make it difficult for basketball coaches and performance staff to differentiate between physiological and psychological factors influencing player workloads [1]. As such, a combination of external workload metrics, as well as objective and subjective internal workload metrics, is recommended to attain a complete picture of training and game demands in basketball [17] (Table 65.1).

65.4 Integrating External and Internal Workloads in Basketball

When using player monitoring data to inform the prescription of player workloads, quantifying and interpreting individualised relationships between external and internal workloads are particularly important. Specifically, internal workload relative to the external workload prescribed should be considered in deciding whether a stimulus elicits favourable or maladaptive responses. The relationship between

Table 65.1 A comparison of common workload metrics in basketball with reference to data quality and practical considerations

	Non-invasive to players?	Valid?	Reliable?	Hardware cost	Software cost	Data processing time	Ease of interpretation
<i>External workload</i>							
Time-motion analysis	✓	✓	✓	\$\$-\$\$\$	\$-\$\$\$	⌚⌚⌚	✓✓✓
Accelerometers	✓	✓	✓	\$\$-\$\$\$	\$\$-\$\$\$	⌚⌚	✓
Inertial sensors	✓	–	✓	\$\$-\$\$\$	\$\$-\$\$\$	⌚⌚	✓✓
Local positioning systems	✓	–	–	\$\$\$	\$\$-\$\$\$	⌚⌚	✓✓✓
<i>Internal workload</i>							
Heart rate	✓	✓	✓	\$-\$\$	\$-\$\$	⌚	✓✓
Banister’s training impulse	✓	✓	✓	\$-\$\$	\$	⌚	✓
Lucia’s training impulse	✗	✓	✓	\$-\$\$\$	\$-\$\$	⌚⌚	✓
SHRZ	✓	✓	✓	\$-\$\$	\$-\$\$	⌚	✓
sRPE	✓	✓	✓	\$	\$	⌚	✓

‘–’ indicates that validity/reliability has not been assessed; reliable is defined as coefficient of variation ≤10% (moderate); hardware cost, software cost, data processing time, and ease of interpretation rated on a 1–3 scale with 1 indicating the lowest rating and 3 indicating the highest rating; *SHRZ* Summated-Heart-Rate-Zones, *sRPE* session rating of perceived exertion

external and internal workloads fluctuates based on the phase of the season and type of training completed [4]. Therefore, basketball coaches and performance staff should seek to develop normative internal workloads for each player relative to a given external workload across multiple sessions in training and games across different phases of the periodisation programme (e.g. overloading and tapering). Understanding typical player responses to training and competition stimuli across a range of scenarios can then be used to support decision-making regarding management of player workloads by understanding what constitutes a normal response and identifying maladaptive reactions to the workloads prescribed in players.

65.5 Quantifying Workload Intensity and Duration in Basketball

Most microsensor, heart rate and perceptual workload metrics used in basketball represent workload volume—that is, they indicate the accu-

lated external or internal demands imposed on players across specified monitoring periods. In the popularised external [18] and internal [9, 12, 16] workload metrics discussed earlier in this chapter, workload volume is dependent on the intensity and duration of the stimuli imposed. While the overall loading imposed on players is important to quantify, it is equally important to be able to measure the intensity and duration of training and games. In this regard, basketball coaches and performance staff should be aware of existing approaches used to quantify session intensity and duration while also recognising the advantages and limitations of each approach.

Intensity is an important aspect of training and games to consider when periodising programmes to promote positive changes in performance variables [19], while also minimising the risk of maladaptive responses, such as injury [20], in players. However, the intensities reached during training and games can be masked when using overall workload volumes [1]. In this regard, a player may achieve similar workload volumes despite working at vastly different intensities. For example, in Fig. 65.1, a player is undergo-

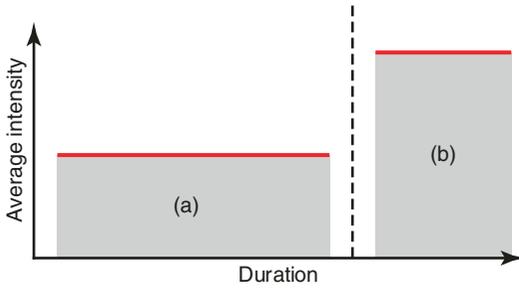


Fig. 65.1 An example of identical workload volumes (shaded grey) being attained in training scenarios performing at a (a) low average intensity for a long duration and (b) high average intensity for a short duration. *Note:* Red line indicates average intensity for each session

ing the same volume of loading when working at a lower average intensity for a long duration compared to working at a high average intensity for a shorter period. Consequently, similar workload volumes can be achieved with very different stimuli, resulting in varied adaptive responses in players. Therefore, basketball coaches and performance staff need to have methods available to quantify session intensity to better understand the stimuli players are exposed to.

A relatively straightforward method to quantify workload intensity involves calculating data relative to the session duration (per minute). Specifically, relative intensity data have been reported for accumulated external (e.g. PlayerLoad™) and internal (e.g. SHRZ) workload metrics, as well as the frequency of various demanding activities such as decelerations, accelerations, jumping and changes in direction in basketball [4, 21, 22]. It should be noted that, in the SRPE model, the relative intensity is simply represented by the RPE given by players rather than workload per minute. While approaches quantifying relative intensity are useful to indicate the average intensity attained across the entire session relative to time, they do not identify the proportion of training or games spent working at specific intensities. For example, in Fig. 65.2, a player is working at the same average intensity across separate sessions (per minute) by performing frequent high-intensity bouts interspersed with low-intensity recoveries in one session and

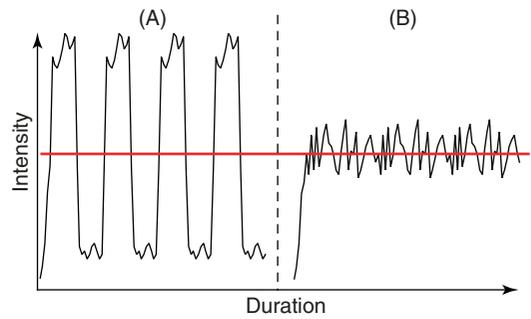


Fig. 65.2 An example of similar average workload intensities (red line) being attained in training scenarios performing (a) frequent high-intensity bouts interspersed with low-intensity recoveries and (b) consistent moderate-intensity activity

more consistently at moderate intensities in the other session. In this regard, activities performed at higher intensities are important to quantify and have been readily measured in basketball [23, 24], given they represent the most stressful loading experienced during training and games.

Measuring the time spent in different intensity zones provides more detailed insight into the workload intensities reached during basketball training and gameplay. In particular, this approach has been readily used to measure internal workload intensities via reporting of absolute (min) or proportional (%) time spent in different heart rate zones. Heart rate zones have typically been determined relative to HR_{max} using various cut-points to demarcate different intensity bands [22, 24, 25]. The variation in methodologies to demarcate heart rate intensity zones should be recognised when interpreting the literature, with a need for greater consistency across future studies to allow for effective comparisons and normative data to be established in different player populations. Furthermore, when reporting relative heart rate responses, the approach to determine HR_{max} should also be considered. Laboratory tests [24], field tests [25], age-predicted equations [26] and peak values obtained during training or gameplay [27] have been used to determine HR_{max} in basketball settings. It is recommended that maximal laboratory exercise testing with metabolic measurement should be used to confirm HR_{max} [27]. However, in the absence of laboratory facilities

and metabolic equipment, maximal field testing should initially be applied to identify peak heart rate with peak responses taken during training and games substituted if higher than the testing response [27].

Less attention has been dedicated to quantify external workload intensity using intensity zones than internal workload intensity. Specifically, accelerometer-derived external workload using fixed arbitrary cut-points provided by software manufacturers and individualised cut-points relative to the peak workload attained during training have been scarcely reported in basketball players [28]. This research showed that less time was measured in higher external workload intensity zones when using individualised compared to fixed cut-points [28]. Given individualised approaches may account for variations in movement and performance capacities between players [29], it has been recommended that individualised approaches be used to best prescribe workload intensities when calculating accelerometer-derived metrics [28]. However, further work is needed to identify suitable cut-points capable of accurately detecting high-intensity activity during basketball training and gameplay using accelerometer-derived workloads. It should also be noted that although video-based time–motion analyses might possess some notable limitations for implementation in practice and local positioning systems require more evidence confirming their validity and reliability in basketball, these methods have also measured intensity relative to time (m min^{-1}) [23, 30] and using different intensity zones (based on fixed speed cut-points) [23, 31] to quantify external workload intensities in basketball. A notable limitation of reporting time spent in intensity zones is the most intense periods of sessions are not identified in isolation. These data represent the upper workload thresholds encountered across sessions in each player and may assist basketball coaches and performance staff in prescribing training to meet the most demanding passages. However, further research is needed applying suitable methods, such as moving workload averages across relevant durations, to quantify the peak demands encountered in basketball.

In addition to workload intensity, determination of session duration is equally important and inconsistencies in approaches are apparent in the literature. Specifically, variations in approaches to define the session period representative of the workload encountered have been adopted when monitoring workloads in basketball players. Approaches to measure training and game duration have either trimmed inactive periods of sessions (e.g. substitutions, inter-drill or inter-period breaks, time-outs) [24, 30] or included the entire session [2, 32]. While removing monitoring periods where players are inactive isolates portions of samples where players are engaged in training drills and gameplay, it excludes important rest periods administered to players. In this way, when data are trimmed, similar workloads will be detected but across shorter durations compared to unedited data, inflating the average intensity of the session [1]. Quantifying workload across entire sessions encompasses all active and rest periods and better represents the average intensity encountered. However, when seeking to measure the demands of specific drills during training (e.g. 5 vs. 5 compared to 3 vs. 3 games) or periods of play during games (e.g. comparison across quarters), it is necessary for irrelevant periods to be trimmed for accurate comparisons to be made.

65.6 Monitoring Well-Being in Conjunction with Player Workloads

When monitoring the workloads encountered by players across the season, it may be equally important to consider their well-being status. These data together may be fundamental to minimise the risk of maladaptive responses from occurring, such as fatigued states requiring extended recovery (non-functional overreaching) and non-contact injuries [33]. In this regard, determining the week-to-week changes in workload and well-being is considered essential to prescribe adequate training stimuli according to player readiness [34, 35]. It has

been theorised that spikes in training load above 15% might increase the injury risk in team sport athletes [33] although no precise information is available in basketball. Research quantifying the percent change in workload each week using the sRPE method demonstrated fluctuations considerably higher than previous prescription recommendations with spikes up to 226% observed in male, college basketball players during the in-season phase (10 weeks) [35]. High spikes in weekly workloads have also been reported for elite, female basketball players monitored across the entire basketball in-season phase (24 weeks) with weekly fluctuations up to 120% [34]. These data suggest that basketball players may be placed at higher injury risk at specific points across the season, given injury risk has been shown to increase by 21–49% in team sport athletes when weekly workload changes by >15% [33]. However, given no data exist regarding the effect of workload spikes on non-contact injuries specifically in basketball players, more research is encouraged on this topic to understand the consequences of high workload fluctuations across the season.

The monitoring of player well-being status is being increasingly reported in basketball research [35, 36]. Many well-being questionnaires in which players report several parameters (e.g. fatigue, sleep quality and mood) have been used in basketball research [35, 37, 38]. Well-being questionnaires offer a useful and inexpensive method of monitoring player responses to a given workload. In this way, despite players undergoing large weekly changes in sRPE workload (up to 226%), constant well-being scores measured via 5-point Likert scales encompassing fatigue, sleep quality, mood, stress levels and general muscle soreness were evident with the highest spike reaching 7% [35]. Therefore, given the different trends in fluctuations across the season between workload and well-being responses, the combination of these two monitoring methods might be more useful for basketball coaches and performance staff to make informed decisions on the management of players rather than either in isolation.

65.7 Consideration of Playing Time When Monitoring Players

Playing time is considered a fundamental aspect to consider when designing training plans [34, 35]. Players usually experience variations in playing time during the in-season due to the number of scheduled weekly games or their differing playing statuses (starting vs. bench players). It has been suggested that the management of player workloads is dependent on the timing of the in-season phase [34]. In this way, basketball coaches might consider adopting strategies to manage the playing time of specific players (e.g. restricting playing time and resting players) at the end of the in-season period to possibly increase their preparedness for the post-season phase (playoffs) rather than at the beginning of the in-season period. In support of this idea, research assessing player workloads in weeks characterised by different match schedules (1-game vs. 2-game weeks) showed a substantially higher weekly sRPE workload during training sessions only and during both training sessions and games combined, in 1-game weeks compared to 2-game weeks in male, collegiate players monitored during the first 10 weeks of the in-season [35]. Similarly, other data showed 1-game weeks elicited moderately greater sRPE workloads than weeks characterised by 2 or more games in professional, male basketball players across the entire in-season [38]. However, it was also reported in this study that players perceived higher fatigue and lower sleep quality during 2-game weeks than 1-game weeks, indicating game factors other than workload may impact perceptual indicators of well-being [38]. Furthermore, a separate study revealed no significant difference in player sRPE workloads between 1-game and 2-game weeks in professional, male players during the last 12 weeks of the in-season [39]. Despite the limited evidence available, initial findings suggested that basketball coaches and performance staff should optimise player workloads and well-being based on the number of weekly games held and the timing of the in-season phase.

The effect of playing time on workload and well-being through considering playing status (starting vs. bench players) has also been recently investigated [35]. This research reported starting players with high playing times and bench players with low playing times belonging to the same male, collegiate basketball team were similar when only considering training sessions [35]. However, it was also observed that a substantially higher weekly workload was evident in starting players when considering training and games combined [35]. These results highlight that starting and bench players experienced similar weekly training demands across the in-season, but the additional playing time during games increases the overall demands in starting players [35]. Conversely, a previous study assessing the effect of playing time on workload during the entire in-season in elite, female basketball players documented significant differences in weekly workloads between players with high and low playing time in games only, and trivial to small differences in weekly workload when considering training sessions (in isolation or in combination with games) [34]. In this instance, increased training stimuli for players with low playing time were likely added to their weekly plans. Nevertheless, the collective trends in these data highlight the relative homogeneity of training workloads accomplished across entire basketball teams; however, the disparity in game workloads between players receiving high playing time and players receiving low playing time should be considered when managing weekly workloads across the team.

Future Directions

- Local positioning systems are a potentially useful means for monitoring external workload in basketball, but their validity and reliability in basketball first need to be confirmed.
- Individualised thresholds to accurately delineate between accelerometer-derived external workload intensities should be developed.

- When quantifying internal workload intensity using heart rate zones, consistent cut-points to differentiate between intensities should be adopted across studies.
- The peak training and game workloads encountered by basketball players should be identified using suitable methods.
- Well-being questionnaires should be implemented in more workload monitoring basketball studies, given they offer an inexpensive and non-invasive method to quantify perceptual parameters such as fatigue, sleep quality, mood, stress and soreness.
- The relationships between fluctuations in player workloads and well-being across the season with performance and injury measures are warranted to identify optimal undulations in workload prescription.

Fact Boxes

- Microsensors encompassing accelerometers and inertial sensors are currently the most popular and appropriate method for monitoring external workloads in basketball.
- The SHRZ model is the most appropriate for objectively monitoring internal workloads in basketball due to its supported validity and reliability, as well as the practical advantages of monitoring procedures and data processing involved.
- Measurement of subjective, perceptual internal workload in combination with objective measures of external and internal workloads provides the most comprehensive insights into player demands during training and games.
- The integration of external and internal workloads may be useful to assess the

appropriateness of training dosages in promoting favourable responses in players.

- In addition to quantifying workload volume, it is important to incorporate approaches that specifically quantify workload intensity.
- Inactive periods during training and games should only be trimmed from analyses when seeking to identify the specific demands of drills or game periods.
- Training workloads appear to be homogenous across all players in a basketball team; however, the disparity in game workloads between players receiving high and low playing times should be considered when prescribing individualised training plans across the season.

References

1. Fox JL, Scanlan AT, Stanton R. A review of player monitoring approaches in basketball: current trends and future directions. *J Strength Cond Res.* 2017;31(7):2021–9. <https://doi.org/10.1519/jsc.0000000000001964>.
2. Fox JL, Stanton R, Sargent C, Wintour S-A, Scanlan AT. The association between training load and performance in team sports: a systematic review. *Sports Med.* 2018;48(12):2743–74. <https://doi.org/10.1007/s40279-018-0982-5>.
3. Impellizzeri FM, Marcora SM, Coutts AJ. Internal and external training load: 15 years on. *Int J Sports Physiol Perform.* 2019;00:1–4.
4. Fox JL, Stanton R, Scanlan AT. A comparison of training and competition demands in semiprofessional male basketball players. *Res Q Exerc Sport.* 2018;89(1):103–11. <https://doi.org/10.1080/02701367.2017.1410693>.
5. McInnes SE, Carlson JS, Jones CJ, McKenna MJ. The physiological load imposed on basketball players during competition. *J Sports Sci.* 1995;13(5):387–97.
6. Hulka K, Cuberek R, Svoboda Z. Time–motion analysis of basketball players: a reliability assessment of video manual motion tracker 1.0 software. *J Sports Sci.* 2013;32(1):53–9. <https://doi.org/10.1080/02640414.2013.805237>.
7. Barrett S, Midgley A, Lovell R. PlayerLoad™: reliability, convergent validity, and influence of unit position during treadmill running. *Int J Sports Physiol Perform.* 2014;9(6):945–52.
8. Narazaki K, Berg K, Stergiou N, Chen B. Physiological demands of competitive basketball. *Scand J Med Sci Sports.* 2009;19(3):425–32. <https://doi.org/10.1111/j.1600-0838.2008.00789.x>.
9. Banister EW, Calvert TW, Savage MV, Bach T. A systems model of training for athletic performance. *Aust J Sci Med.* 1975;7(3):57–61.
10. Lucia A, Hoyos J, Santalla A, Earnest C, Chicharro JL. Tour de France versus Vuelta a Espana: which is harder? *Med Sci Sports Exerc.* 2003;35(5):872–8.
11. Scanlan AT, Fox JL, Borges NR, Dascombe BJ, Dalbo VJ. Cumulative training dose's effects on interrelationships between common training load models during basketball activity. *Int J Sports Physiol Perform.* 2016;12(2):168–74.
12. Edwards S. *The heart rate monitor book.* Sacramento: CA Fleet Feet Press; 1993.
13. Scanlan AT, Wen N, Tucker PS, Dalbo VJ. The relationships between internal and external training load models during basketball training. *J Strength Cond Res.* 2014;28(9):2397–405. <https://doi.org/10.1519/jsc.0000000000000458>.
14. Scanlan AT, Fox JL, Poole JL, et al. A comparison of traditional and modified summated-heart-rate-zones models to measure internal training load in basketball players. *Meas Phys Educ Exerc Sci.* 2018; 22(4):303–9.
15. Borg G, Hassmén P, Lagerström M. Perceived exertion related to heart rate and blood lactate during arm and leg exercise. *Eur J Appl Physiol Occup Physiol.* 1987;56(6):679–85.
16. Foster C, Florhaug J, Franklin J, et al. A new approach to monitoring exercise training. *J Strength Cond Res.* 2001;15(1):109–15.
17. Bourdon PC, Cardinale M, Murray A, et al. Monitoring athlete training loads: consensus statement. *Int J Sports Physiol Perform.* 2017;12(Suppl 2):161–70. <https://doi.org/10.1123/ijspp.2017-0208>.
18. Montgomery P, Pyne D, Minahan C. The physical and physiological demands of basketball training and competition. *Int J Sports Physiol Perform.* 2010;5(1):75–86.
19. Vetter R, Yu H, Foose A. Effects of moderators on physical training programs: a Bayesian approach. *J Strength Cond Res.* 2017;31(7):1868–78. <https://doi.org/10.1519/jsc.0000000000001585>.
20. Vetter R, Symonds M. Correlations between injury, training intensity, and physical and mental exhaustion among college athletes. *J Strength Cond Res.* 2010;24(3):587–96.
21. Scanlan A, Stanton R, Sargent C, O'Grady C, Lastella M, Fox J. Working overtime: the effects of overtime periods on game demands in basketball players. *Int J Sports Physiol Perform.* 2019;14(10):1331–7. <https://doi.org/10.1123/ijspp.2018-0906>.
22. Reina Román M, García-Rubio J, Feu S, Ibáñez S. Training and competition load monitoring and

- analysis of women's amateur basketball by playing position: approach study. *Front Psychol.* 2019;9:2689. <https://doi.org/10.3389/fpsyg.2018.02689>.
23. Vazquez-Guerrero J, Reche X, Cos F, Casamichana D, Sampaio J. Changes in external load when modifying rules of 5-on-5 scrimmage situations in elite basketball. *J Strength Cond Res.* 2018; <https://doi.org/10.1519/jsc.0000000000002761>.
 24. Sanders G, Boos B, Rhodes J, Kollock R, Peacock C. Competition-based heart rate, training load, and time played above 85% peak heart rate in NCAA Division I women's basketball. *J Strength Cond Res.* 2018; <https://doi.org/10.1519/jsc.0000000000002876>.
 25. Delextrat A, Kraiem S. Heart-rate responses by playing position during ball drills in basketball. *Int J Sports Physiol Perform.* 2013;8(4):410–8.
 26. Abad C, Pereira L, Kobal R, et al. Heart rate and heart rate variability of Yo-Yo IR1 and simulated match in young female basketball athletes: a comparative study. *Int J Perform Anal Sport.* 2016;16(3):776–91. <https://doi.org/10.1080/24748668.2016.11868927>.
 27. Berkelmans D, Dalbo V, Fox J, et al. Influence of different methods to determine maximum heart rate on training load outcomes in basketball players. *J Strength Cond Res.* 2018;32(11):3177–85. <https://doi.org/10.1519/jsc.0000000000002291>.
 28. Scanlan A, Fox J, Milanovic Z, Stojanovic E, Stanton R, Dalbo V. Individualized and fixed thresholds to demarcate PlayerLoad intensity zones produce different outcomes. *J Strength Cond Res.* 2019; <https://doi.org/10.1519/jsc.0000000000003001>.
 29. Nakamura F, Pereira L, Loturco I, Rosseti M, Moura F, Bradley P. Repeated-sprint sequences during female soccer matches using fixed and individual speed thresholds. *J Strength Cond Res.* 2017;31(7):1802–10. <https://doi.org/10.1519/jsc.0000000000001659>.
 30. Scanlan A, Dascombe B, Kidcaff A, Peucker J, Dalbo V. The gender-specific activity demands experienced during semi-professional basketball game-play. *Int J Sports Physiol Perform.* 2015;10(5):618–25.
 31. Scanlan A, Dascombe B, Reaburn P. A comparison of the activity demands of elite and sub-elite Australian men's basketball competition. *J Sports Sci.* 2011;29(11):1153–60. <https://doi.org/10.1080/02640414.2011.582509>.
 32. Manzi V, D'Ottavio S, Impellizzeri F, Chaouachi A, Chamari K, Castagna C. Profile of weekly training load in elite male professional basketball players. *J Strength Cond Res.* 2010;24(5):1399–406.
 33. Gabbett T. The training-injury prevention paradox: should athletes be training smarter and harder? *Br J Sports Med.* 2016;50(5):273–80.
 34. Paulauskas H, Kreivyte R, Scanlan A, Moreira A, Siupsinskas L, Conte D. Monitoring workload in elite female basketball players during the in-season phase: weekly fluctuations and effect of playing time. *Int J Sports Physiol Perform.* 2019;14(7):941–8.
 35. Conte D, Kolb N, Scanlan A, Santolamazza F. Monitoring training load and well-being during the in-season phase in National Collegiate Athletic Association Division I Men's Basketball. *Int J Sports Physiol Perform.* 2018;13(8):1067–74.
 36. Sansone P, Tschan H, Foster C, Tessitore A. Monitoring training load and perceived recovery in female basketball: implications for training design. *J Strength Cond Res.* 2019; <https://doi.org/10.1519/JSC.0000000000002971>.
 37. Antonio Nunes J, Moreira A, Crewther B, Nosaka K, Viveiros de Castro LE, Aoki M. Monitoring training load, recovery-stress state, immune-endocrine responses, and physical performance in elite female basketball players during a periodized training program. *J Strength Cond Res.* 2014;28:2973–80. <https://doi.org/10.1519/jsc.0000000000000499>.
 38. Clemente F, Mendes B, Teles G, et al. Perceived training load, muscle soreness, stress, fatigue, and sleep quality in professional basketball: a full season study. *J Hum Kinet.* 2019;67:199–207.
 39. Manzi V, D'Ottavio S, Impellizzeri F, Chaouachi A, Chamari K, Castagna C. Profile of weekly training load in elite male professional basketball players. *J Strength Cond Res.* 2010;24:1399–406.

Part VI

Special Considerations in Basketball



The Female Basketball Player

66

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66.1 Introduction

The number of female athletes, including female basketball players, at all levels of competition has steadily grown over the past several decades. Basketball remains the third most popular high school level sport for females [1]. Since the creation of the WNBA in 1996, the number of collegiate women's basketball teams has grown. While management of many injuries and medical conditions is similar for both male and female athletes, there are important anatomical and

physiological differences between the sexes. These differences influence the risk of injury as well as athletic performance. Also unique to female athletes, pregnancy during training and competition may occur.

This chapter will review the basics of the menstrual cycle as well as hormonal contraceptive options. It also summarizes the present knowledge on the influence of female hormones on muscle, tendon, and ligament properties. There will also be a focus on biomechanical and neuromuscular differences in the female sex and their relationship to a female athlete's higher risk of sustaining an anterior cruciate ligament (ACL) injury. This chapter will highlight some of the most important aspects of the female athlete triad/relative energy deficiency in sport (RED-S) with a focus on bone health and stress fractures. Lastly, this chapter will provide insight into current recommendations regarding participation in basketball and physical activity in general during pregnancy and the postpartum period.

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66.2 Hormonal Fluctuations During the Menstrual Cycle

The menstrual cycle involves a complex interconnection involving the hypothalamus, pituitary, and ovarian hormones. It is traditionally divided into three main phases (follicular, ovulatory, and luteal) based on ovarian function. The

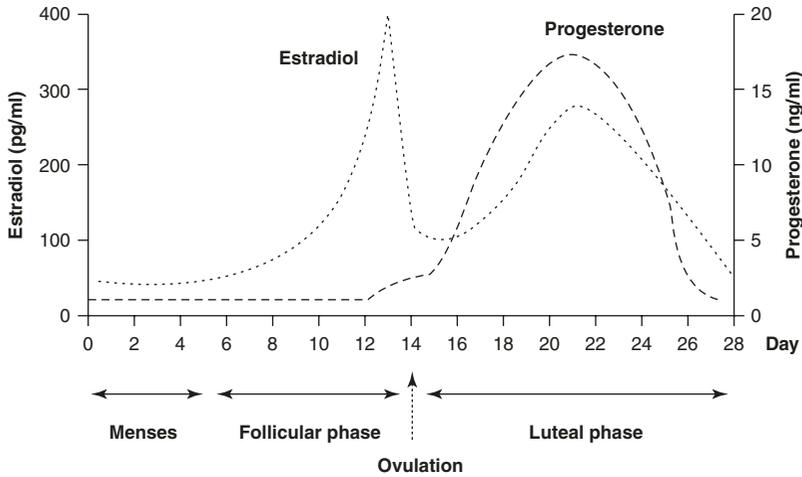


Fig. 66.1 Hormonal changes during the menstrual cycle. Overview over the hormonal changes in estradiol and progesterone during the menstrual cycle in young women who do not use oral contraceptives. (From: Hansen M, Dalggaard

LB, Zebis MK, Gliemann L, Melin A, and Torstveit MK (2018) The Female Handball Player. In: Laver L, Landreau P, Seil R, Popovic N (Hrsg) Handball Sports Medicine. Springer, Berlin, Heidelberg, S 553–569)

three phases of the cycle are differentiated by varying estrogen and progesterone levels: (1) low estrogen and low progesterone levels during the follicular phase, (2) high estrogen and low progesterone levels during the ovulatory phase, and (3) high estrogen and high progesterone levels during the luteal phase as illustrated in Fig. 66.1 [2]. Natural variability in cycle-phase length, each with different hormone ratios, is seen commonly in females.

66.3 Hormonal Contraceptive Options

Hormonal contraception plays a valuable role in hormonal modification of the menstrual cycle and may be useful for female athletes. These medications are commonly used for cycle control and/or for pregnancy prevention. They are also commonly used for females with prolonged menstrual dysfunction (amenorrhea, oligomenorrhea), premenstrual symptoms (fatigue, fluid retention), polycystic ovarian syndrome (PCOS), premenstrual dysphoric disorder, chronic migraines, and dysmenorrhea.

Oral contraceptive pills (OCPs) are available in various combinations and dosages. Monophasic

combination pills consist of estrogen and progestin in fixed doses (constant levels of estrogen and progestin), whereas biphasic and triphasic preparations attempt to more closely mimic the conditions of a natural menstrual cycle by varying the hormonal concentrations. Overall, monophasic pills are easier to manipulate for travel and athletic competitions and have lesser hormonal adverse reactions. Progestin-only “minipills” are also available and typically used in those with relative and absolute contraindications to estrogen-containing OCPs. There is also an extended-cycle OCP which allows women the option of decreasing the number of withdrawal bleeds, theoretically inducing menstrual bleeding only four times per year. OCPs can also reduce endometrial cancer, ovarian cancer, physiological ovarian cysts, and benign breast disorders [2].

Long-acting reversible contraception, also known as LARC, is a good option for those who have barriers to taking a pill daily. This category of contraception includes hormonal (progestin-containing) and nonhormonal (copper-containing) intrauterine devices (IUDs). A progestin-only implant and injection are also available and highly effective options. Progestins vary in terms of pharmacologic properties. Some have antiandrogenic or antimineralocorticoid

activity, which can offer favorable effects in terms of skin, hair, and water retention, especially for those females with troublesome acne and hair thinning. It is important to note that unpredictable menstrual bleeding is a common side effect that is expected in the first few months after starting any progestin-only agent.

The choice of contraception must be individualized after thoroughly assessing a female's medical history, menstrual cycles, menstrual concerns, health and performance goals. Engaging the athlete in her health-care decision-making is warranted to address potential health advantages and disadvantages in initiating and continuing hormonal contraception.

66.4 Differences in Tissue Properties

As the number of females participating in athletics has grown, the research surrounding estrogen and the effects it has on the musculoskeletal system has simultaneously expanded. Historically, a majority of the studies in this realm focused on the effects estrogen has on bone, but more recently there have been increasing efforts to determine how the presence of increased estrogen impacts muscles, tendons, and ligaments. Though there is still much to learn in this space, it has become increasingly clear that estrogen has significant effects on all of these tissues, some of which appear to be advantageous, while others result in females being more susceptible to certain injuries.

66.4.1 Muscle

Animal models have shown that estrogen is beneficial for building muscle mass and strength. A deficiency in this hormone results in a decrease in both strength and muscle fiber cross-sectional area [3, 4]. Given this and the previously discussed fluctuations of hormones throughout the menstrual cycle, it has been hypothesized that muscle strength and therefore performance may be enhanced during certain points in the men-

strual cycle. Most human studies have been unable to find differences in muscle strength during different menstrual cycle phases, but a small amount of research suggests that there may be an increase in muscle strength around the time of ovulation [5, 6]. During this time in the cycle, estrogen levels are high, while progesterone levels are low.

There is evidence to suggest that estradiol plays an important role in diminishing muscle damage and preserving muscle function after intense non-weight-bearing, eccentric exercise [7]. In a study by Savage, after performing eccentric exercise, normally menstruating females had lower serum levels of muscle injury markers than both male subjects and female subjects on OCPs. Also of note in this study, all groups demonstrated an expected ~10% loss of strength 24 h after exercise, but normally menstruating women observed no further loss, while the other two groups continued to demonstrate significant losses in leg strength up to 48 h post exercise. Other studies have reported similar results, suggesting that estrogen levels in normally menstruating females play a role in reducing muscle recovery time [8]. Though these findings have not been demonstrated in association with weight-bearing activity, this information is useful in planning strength training sessions for athletes.

66.4.2 Tendons and Ligaments

Estrogen may also decrease the stiffness of both ligaments and tendons, suggested by the changes in knee laxity in association with estrogen levels during the menstrual cycle [9]. In a study by Shultz, knee laxity gradually increased in women as estradiol levels began to rise around the time of ovulation. In vivo measurement of tendon stiffness is also lower in women compared to men during maximum isometric loading. Tendon laxity may be responsible for the decreased level of exercise-associated muscle damage described in the previous section. Tendon laxity may dampen the impact of contraction on muscle tissue [10]. Overall, female athletes suffer more

ligament tears and fewer muscle injuries than their male counterparts [11]. A direct relationship has been described between ACL laxity and rupture [12]. Therefore, it is thought that one of the reasons female athletes suffer more ACL injuries is because of increased ligamentous laxity.

66.5 Biomechanical Differences

During puberty, female hips widen in anticipation of childbirth [13]. The wider pelvis results in an increased inward slant of the femur. The knee joint also then sits medial to the hip joint after this widening occurs. The relationship between the hip joint and the knee can be objectively characterized by measuring a quadriceps angle (Q angle). The female Q angle is about 3° greater than males (17° vs. 14°), leading to an increased predisposition to a variety of knee pathologies.

Non-modifiable anatomical risk factors that have been associated with a higher risk of ACL injury in females are generalized joint laxity (as discussed in the previous section), ACL notch size and shape, and posterior tibial slope [14]. The PPE can be used to screen for biomechanical risk factors associated with higher rates of ACL injury, especially for female athletes participating in pivoting and cutting sports such as basketball.

The single leg landing test is one of the best ways to evaluate for biomechanical deficiencies, such as decreased knee, hip, and trunk flexion. The maximum knee valgus not to be exceeded is different between male and female at 5° and 10°, respectively [15–17]. Excessive knee valgus and/or the presence of a heavy landing pattern involving very little knee, hip, and trunk flexion may increase the risk of future knee injury or reinjury, such as noncontact ACL injuries [18].

66.6 Neuromuscular Differences

Neuromuscular factors such as muscle imbalances, core strength, neuromuscular control, physical fitness, muscle fatigue, and sport-skill level have been found to be risk factors contributing to ACL injury in female athletes [19, 20].

During 3D motion analysis, female athletes had a greater knee abduction angle when preparing to perform a directional change compared with male athletes [17]. Studies on side-cutting show that women produce smaller knee flexion angles and greater valgus moments during jumping and side-cutting drills. Studies on sex differences in muscular activation during jumping or cutting have shown a tendency to higher activation of quadriceps and lower activation of hamstrings in females compared to males. The lower neuromuscular pre-activity of the hamstrings among females supports the notion that female athletes display different neuromuscular strategy in situations where ACL injuries occur. The Landing Error Scoring System (LESS) is a neuromuscular screening test in which landing posture is observed after a drop landing from a 30-cm-high platform [21, 22]. From this observation, a risk score is calculated—a higher score reflecting higher risk movement patterns. The most recent PPE monograph recommends consideration of the LESS screening test for female athletes.

66.7 Anterior Cruciate Ligament Injury

ACL tears in female athletes occur at a rate of 0.29 per 1000 athlete exposure compared to only 0.07 per 1000 athlete exposure for men [23]. Similarly, in women's collegiate basketball, ACL injury has been reported at a rate of 0.22 per 1000 athlete exposures compared to the men's rate of 0.08 per 1000 exposures [24]. A relatively high rate of noncontact ACL injuries in female athletes has long been reported, as high as 70% of all ACL tears sustained by female athletes. Female athletes have five times the rate of noncontact ACL tears than men [25]. Both intrinsic and extrinsic factors, many of which have been discussed earlier in this chapter, have been studied to determine why the incidence of ACL tears is so high. As a result of some of the studies exploring biomechanical differences, there has been an emphasis on hamstring strengthening for female athletes and neuromuscular training to improve the biomechanics of female athletes [26–29].

Plyometric training has been shown to increase the change of direction ability, but it is unclear whether this will lead to decreased injury rates [30]. In one study, the rate of noncontact ACL injury decreased 72% in athletes who underwent preseason neuromuscular training [31, 32].

In those who have had the ACL reconstructed, sex has not been determined definitively as a risk factor for ACL graft rupture, and in fact, males, particularly younger males, have been shown to be at greater risk [33–35]. However, females may be at higher risk for subsequent contralateral ACL injury [17, 36]. Before returning to basketball after ACL reconstruction, many experts would recommend assessments of jumping/landing, change of direction, reactive agility, sport-specific activity, and psychological readiness. Currently, however, there are few studies examining criteria-based return to sport (RTS) decisions following ACL reconstruction, and it is not clear that RTS programs reduce the risk of a subsequent anterior cruciate ligament injury [37].

66.7.1 Estrogen Levels and ACL Injury Risk

As touched on earlier in this chapter, there is interest in better understanding the relationship between the menstrual cycle and risk of sustaining an ACL injury. In several studies, the ACL injury risk was lowest during the luteal phase and highest late in the follicular phase around the time of ovulation [38]. As discussed in an earlier section in this chapter, around the time of ovulation is when knee laxity has also been reported to be increased [9]. This time also correlates with the time of highest estradiol concentration.

A systematic review has also examined the association between oral contraceptive use and ACL injury risk. The authors concluded that the use of an oral contraceptive pill might reduce injury risk by as much as 20%. This may be secondary to the absence of a peak in estradiol. It has also been noted that those on oral contraception have a lower tendon collagen turnover rate, which may also improve tendon and ligament stiffness [38].

66.8 Female Athlete Triad/RED-S

While the prevalence is higher in endurance, aesthetic, and weight class sports, the female athlete triad also affects female basketball players. The female athlete triad involves three interrelated components: (1) low energy availability (EA) with or without disordered eating, (2) menstrual dysfunction, and (3) low bone mineral density (BMD). The triad is illustrated in Fig. 66.2. Energy availability is the energy remaining for normal metabolic processes after the energy cost of training has been subtracted. The other two components happen as a result of low energy availability. Studies have shown that a sustained EA less than 30 kcal/kgFFM/day can lead to suppressed hypothalamic–pituitary axis hormones. A goal EA for most athletes should be closer to 45 kcal/kg FFM/day.

Low energy availability can happen inadvertently, can be from disordered eating, and sometimes is a result of a clinical eating disorder. A recent study by Silva et al. calculated daily energy needs for elite female basketball players to average approximately 3500 kcal/day which can be challenging to obtain for some athletes. Risk factors for low EA include restrictive diets, exercising for extended periods of time (endurance sports), and a personal history of abuse, low self-esteem, or family issues. It is important to note that athletes may stay at a stable weight but also be in a state of low EA at the same time. This may happen as a result of suppression of other physiologic functions.

As part of the triad, females with low EA may experience functional hypothalamic amenorrhea (FHA). In FHA, the loss of menstrual cycle is presumed to be from functional disruption of pulsatile hypothalamic gonadotropin-releasing hormone (GnRH) secretion. This abnormal GnRH secretion leads to decreased pulses of gonadotropins, an absent midcycle surge in luteinizing hormone (LH) secretion, absence of normal follicular development, anovulation, and low serum estradiol (E2) concentrations. This can manifest as either primary amenorrhea (no onset of menarche by age 15) or secondary amenorrhea (absence of menstrual cycle for 3 consecu-

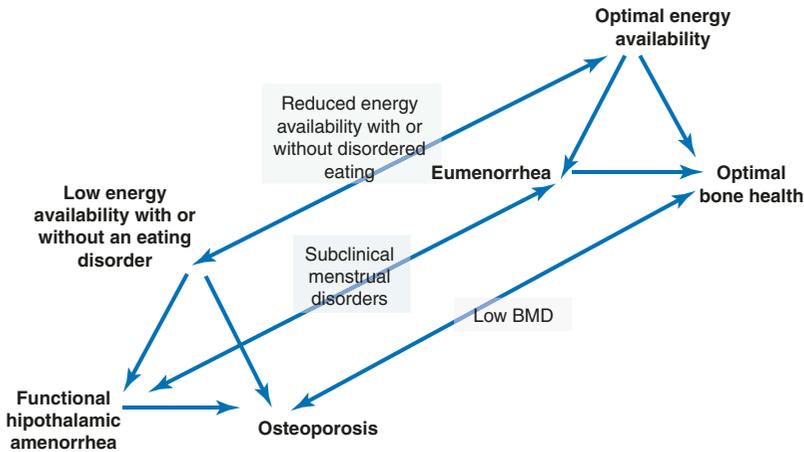


Fig. 66.2 The Female Athlete Triad. The spectrum of energy availability, menstrual function, and bone mineral density along which female athletes are distributed (long arrows). An athlete's condition can change in one direction or the other, according to her diet and exercise habits. Energy availability, defined as dietary energy intake minus exercise energy expenditure, affects bone mineral density

both directly via metabolic hormones and indirectly via effects on menstrual function and thereby estrogen (short arrows). (Used with permission from: Muradas K, Costa L, Seijas R, Álvarez-Díaz P, Ares O, Sallent A, Cugat R. Female Athlete Triad Syndrome: Prevention. *Int Journal of Orthopaedics* 2015 February 23 2(1): 205–209)

tive months). However, many athletes may not experience amenorrhea but instead may experience more difficult to diagnose subclinical menstrual disorders.

The last component of the female athlete triad is low bone mineral density. When an energy deficient state is partnered with a hypoestrogenic state, markers of bone formation are suppressed, and markers of resorption are elevated. Mild to moderate low BMD has even been found in athletes with oligomenorrhea and subclinical menstrual disorders. The most severe clinical end point of this component is osteoporosis. Z-scores from a DXA scan, comparing bone density to age-matched controls, are used to diagnose low BMD or osteoporosis [39]. A delay in menarche, oligomenorrhea/amenorrhea, and/or low bone mineral density are significant risk factors for bone stress injuries in athletes [40], which will be discussed later.

Early detection of athletes at risk is pivotal and an expert panel on the triad recommends including screening for components of the triad as part of the standard preparticipation physical. This screening is most often performed at the collegiate level but would likely be beneficial at even the high school level (95% of peak bone

mass is reached by age 20). Any concern for one component of the triad should prompt a more thorough investigation for the others as well. While many standard PPE forms do contain questions that screen for the triad, the consensus panel offers a standardized set of questions which can be found in their 2014 paper. The Low Energy Availability in Females (LEAF) Questionnaire can also be used as a screening tool.

In regard to treating the triad, increased caloric intake (often associated with weight gain) that leads to restoration of menstrual function has been linked to improvement of BMD. Therefore, initial treatment should be increasing energy availability by either increasing caloric intake or decreasing energy expenditure during exercise. Treatment should also focus on identifying and correcting the underlying causes of low energy availability. Assistance from behavioral health/psychology may be helpful in athletes with disordered eating and eating disorders. Managing an athlete with the triad may be challenging as components may be hard to identify, especially if athletes do not provide accurate information. Athletes may also be resistant to engaging in treatment given that it often mandates them having to gain weight. Having a

multidisciplinary team on board, including a physician, dietician, and a behavioral health expert, is optimal [39].

Relative energy deficiency in sport (RED-S) is a relatively new term, introduced to emphasize that effects of sustained low EA can extend beyond bone health and menstrual function. The term RED-S is also used to broaden this concept to male athletes [41].

66.9 Stress Fractures

Women have higher rates of stress fracture than men. In a study of collegiate athletes, women were more likely to sustain a stress fracture than their male counterparts [42]. Stress fractures of the foot followed by tibia were the most common in women. Over one-fifth of stress fractures in collegiate athletes were found to be recurrent [43]. Apart from low bone mineral density, often seen as part of the female athlete triad (discussed earlier in this chapter), women may be at a higher risk for stress fractures because of their relatively lower amount of muscle mass. Muscle can both act to disperse and to absorb the forces that lead to stress fractures [44]. Interestingly, participation in ball sports during youth (basketball) may reduce the future risk for stress fractures in runners and military [45].

The location of the stress fracture often dictates treatment [46]. For low risk fractures such as the fibula, lateral malleolus, calcaneus, cuboid, cuneiform and distal metatarsal, treatment consists of rest followed by graduated return to play over several weeks as pain resolves. Higher risk stress fractures, such as femoral neck, femoral shaft, and tibial, may require operative intervention to stabilize the bone to promote healing, especially after a trial of limited or no weight-bearing [46, 47].

For athletes managed with nonoperative treatment, location and severity of the stress injury determined by imaging should help guide timing of return to sport [47]. If surgery is required, imaging of postoperative healing guides RTS as well as recovery of strength, agility, endurance, and the ability to perform sport-specific tasks pain-free.

66.10 Pregnancy

As more women participate in sports, the rate of pregnancy at all levels of play has increased. In general, exercise is recommended during uncomplicated singleton pregnancies. Physical activity during pregnancy is associated with fewer newborn complications and significant maternal benefits including decreased risk of preeclampsia and gestational diabetes. Exercise during pregnancy is not associated with preterm labor, miscarriage, or birth defects [48].

The risks associated with playing basketball during pregnancy can be divided into physiologic risk and risk of trauma/collision. The American College of Gynecology (ACOG) recommends avoiding contact sports, which includes basketball, during pregnancy as participation in these activities could increase the risk of abdominal and therefore fetal trauma. There is an opacity of data regarding physiologic risk of high intensity aerobic activity in elite athletes during pregnancy. However, the current recommendation is that women who habitually engage in vigorous intensity aerobic exercise can continue physical activity during pregnancy if they stay healthy and follow up closely with their provider [49]. Resistance training is also recommended during pregnancy, and there is no evidence that suggests that weightlifting increases risk of miscarriage [50]. A provider may want to follow up more closely with an elite athlete who is continuing to train in order to ensure adequate caloric intake. In general, pregnant female basketball players may continue to participate in noncontact aerobic conditioning as well as strength training.

During pregnancy, several physiologic changes such as increased heart rate (at rest and during activity), increased cardiac output, increased minute ventilation, and increased oxygen consumption occur as early as the first couple of weeks. This increase in oxygen consumption may cause some women to experience a decline in exercise tolerance. A small number of studies have measured VO_2 max during pregnancy. A few of these studies have shown that well-conditioned athletes who maintain a high level of fitness during pregnancy may experience an increase in VO_2 max following pregnancy [51].

Increased ligamentous laxity occurs during pregnancy and is thought to be secondary to the hormone relaxin as well as increased estrogen. Pregnant female athletes may want to consider avoiding ballistic activities and unnecessary loads on joints. Pregnant athletes, especially basketball players who may experience increased load on the pelvic floor during jumps and other high-impact exercises, may also want to consider participation in pelvic floor training to reduce the risk of urinary incontinence and other pelvic floor complications. Lastly, during pregnancy there is also an increase in weight and a shift in center of gravity, increasing the risk of falls [48].

66.11 Postpartum and Breastfeeding

The postpartum period has been typically defined as the first 6 weeks following delivery. However, recent effort has been made to dispel this arbitrary definition. Some groups define the postpartum period as the first 12 weeks following delivery, some, the first year, and others argue that once a female is postpartum, she is always postpartum. It is important to recognize that time of recovery may vary tremendously. Postpartum return to sport should be an individualized process. There is very limited research on athletes returning to activity and sport after childbirth. Some studies on physically fit soldiers have found that the amount of time needed for postpartum soldiers to return to prepregnancy fitness levels ranged from 2 to 24 months, with a mean of 11 months. Early return to heavy physical activity after delivery may increase the risk of urinary incontinence and pelvic prolapse in the general population, but this has not been investigated in female athletes. It may be advisable that athletes whose delivery involved increased pelvic floor stress (large baby, operative vaginal delivery) minimize activities that increase intra-abdominal pressure or result in high impact for the first several months postpartum while they retrain/strengthen their pelvic floor. Some females may also experience diastasis recti during pregnancy. This generally resolves during the early postpartum period but may persist in some, resulting in

core weakness, hip pain, and back pain. At this time, there is insufficient evidence as to the optimal postpartum rehab program for this condition. Those who have undergone cesarean birth should be cleared by obstetric provider before returning to exercise. It is important to note that the abdominal fascia regains less than 60% of its original tensile strength in the first 6 weeks following surgery. In general, postpartum women should return to exercise gradually and progress their time, frequency, and intensity as tolerated by their body.

The World Health Organization recommends exclusively breastfeeding for the first 6 months and if possible, continuing to breastfeed for the first year or longer. There is no evidence that intense exercise impairs milk production as long as caloric needs are met and hydration is maintained. In fact, high-volume aerobic activity may result in greater quantity and quality of breast milk [49]. It is important to note that breastfeeding may contribute to increased resorption of bone and decreased bone mineral density, which hypothetically could increase the risk of stress fracture. For most, this loss in BMD is restored in the months following cessation of breastfeeding [50].

Take-Home Messages

As the number of females participating in athletics in general and basketball in particular has grown, the research surrounding female hormones, biomechanics, and neuromuscular differences and their relationship with female athlete performance and injury risk has increased. Furthermore, discussion and knowledge surrounding the female athlete triad has significantly grown. Lastly, physical activity is now promoted during pregnancy and breastfeeding and sport participation are no longer seen as mutually exclusive.

Current data suggests the following:

1. Gains in strength training may be enhanced when intense training is performed late in the follicular phase, around the time of ovulation.

2. Exposure to high estrogen levels is associated with increased tendon and ligament laxity.
3. ACL injury may be increased around the time of ovulation and use of oral contraceptives may reduce this risk.
4. Preseason neuromuscular training may decrease the risk of ACL injury.
5. Female athletes are at risk for the Female Athlete Triad and should be screened for this condition to aid in early detection and intervention.
6. Location and severity of stress fractures should guide management and return to play decisions. Some high-risk stress fractures may require surgical treatment.
7. Female basketball athletes can continue to participate in noncontact physical activity during pregnancy and return to sport postpartum should be individualized.

While great advances have been made in the care of female athletes, there is still substantial room for further research and advancement.

References

1. [Nfhs.org](https://www.nfhs.org/articles/high-school-sports-participation-increases-for-29th-consecutive-year/). High School Sports Participation Increases for 29th Consecutive Year. 2018. <https://www.nfhs.org/articles/high-school-sports-participation-increases-for-29th-consecutive-year/>. Accessed 1 Nov 2019.
2. Constantini NW, et al. The menstrual cycle and sport performance. *Clin Sports Med*. 2005;24(2):e51–82. <https://doi.org/10.1016/j.csm.2005.01.003>.
3. McClung JM, Davis JM, Wilson MA, Goldsmith EC, Carson JA. Estrogen status and skeletal muscle recovery from disuse atrophy. *J Appl Physiol*. 2006;100:2012–23. <https://doi.org/10.1152/jappphysiol.01583.2005>.
4. Kitajima Y, Ono Y. Estrogens maintain skeletal muscle and satellite cell functions. *J Endocrinol*. 2016;229:267–75. <https://doi.org/10.1530/JOE-15-0476>.
5. Sarwar R, Niclos BB, Rutherford OM. Changes in muscle strength, relaxation rate and fatigability during the human menstrual cycle. *J Physiol*. 1996;493(1):267–72.
6. Bambaiechi E, Reilly T, Cable NT, Giacomoni M. The isolated and combined effects of menstrual cycle phase and time-of-day on muscle strength of eumenorrhic females. *Chronobiol Int*. 2004;21(4–5):645–60.
7. Minahan C, Joyce S, Bulmer AC, Cronin N, Sabapathy S. The influence of estradiol on muscle damage and leg strength after intense eccentric exercise. *Eur J Appl Physiol*. 2015;115(7):1493–500.
8. Savage K, Clarkson P. Oral contraceptive use and exercise induced muscle damage and recovery. *Contraception*. 2002;66:67–714.
9. Shultz SJ, Sander TC, Kirk SE, Perrin DH. Sex differences in knee joint laxity change across the female menstrual cycle. *J Sports Med Phys Fitness*. 2005;45:594–603. <https://doi.org/10.1249/00005768-200405001-0071>.
10. Leblanc DR, Schneider M, Angele P, Vollmer G, Docheva D. The effect of estrogen on tendon and ligament metabolism and function. *J Steroid Biochem Mol Biol*. 2017;172:106–16. <https://doi.org/10.1016/j.jsbmb.2017.06.008>.
11. Hägglund M, Markus W, Ekstrand J. Injuries among male and female elite football players. *Scand J Med Sci Sports*. 2009;19:819–27. <https://doi.org/10.1111/j.1600-0838.2008.00861>.
12. Myer GD, Ford KR, Paterno MV, Nick TG, Hewett TE. The effects of generalized joint laxity on risk of anterior cruciate ligament injury in young female athletes. *Am J Sports Med*. 2008;36:1073–80. <https://doi.org/10.1177/036354650731357>.
13. Warrener AG, Lewton KI, Pontzer H, et al. A wider pelvis does not increase locomotor cost in humans, with implications for the evolution of childbirth. *PLoS One*. 2015;10(3):e0118903.
14. Giffin JR, Vogrin TM, Zantop T, Woo SL, Harner CD. Effects of increasing tibial slope on the biomechanics of the knee. *Am J Sports Med*. 2004;32(2):376–82.
15. Hewett TE, Myer GD, Ford KR. Biomechanical measures of neuromuscular control and valgus loading of the knee predict anterior cruciate ligament injury risk in female athletes: a prospective study. *Am J Sports Med*. 2005;33:492–501.
16. Ford KR, Myer GD, Hewett TE. Valgus knee motion during landing in high school female and male basketball players. *Med Sci Sports Exerc*. 2003;35(10):1745–50.
17. Ford KR, Myer GD, Toms HE, Hewett TE. Gender differences in the kinematics of unanticipated cutting in young athletes. *Med Sci Sports Exerc*. 2005;37:124–9.
18. Renstrom P, Ljungqvist A, Arendt E, Beynonn B, Fukubayashi T, Garrett W, Georgoulis T, Hewett TE, Johnson R, Krosshaug T, Mandelbaum B, Micheli L, Myklebust G, Roos E, Roos H, Schamash P, Shultz S, Werner S, Wojtys E, Engebretsen L. Non-contact ACL injuries in female athletes: an International Olympic Committee current concepts statement. *Br J Sports Med*. 2008;42(6):394–412.
19. Price MJ, Tuca M, Cordasco FA, Green DW. Nonmodifiable risk factors for anterior cruciate ligament injury. *Curr Opin Pediatr*. 2017;29(1):55–64.

20. Smith HC, Vacek P, Johnson RJ, et al. Risk factors for anterior cruciate ligament injury: a review of the literature—part 1: neuromuscular and anatomic risk. *Sports Health*. 2012;4(1):69–78.
21. Fox AS, Bonacci J, McLean SG, Spittle M, Saunders N. A systematic evaluation of field-based screening methods for the assessment of anterior cruciate ligament (ACL) injury risk. *Sports Med*. 2016;46(5):715–35.
22. Padua DA, DiStefano LJ, Beutler AI, de la Motte SJ, DiStefano MJ, Marshall SW. The landing error scoring system as a screening tool for an anterior cruciate ligament injury-prevention program in elite-youth soccer athletes. *J Athl Train*. 2015;50(6):589–95.
23. Agel J, Arendt EA, Bershadsky B. Anterior cruciate ligament injury in national collegiate athletic association basketball and soccer: a 13-year review. *Am J Sports Med*. 2005;33(4):524–31.
24. Agel J, Rockwood T, Klossner D. Collegiate ACL injury rates across 15 sports. *Clin J Sport Med*. 2016;26(6):518–23.
25. Arendt E, Dick R. Knee injury patterns among men and women in collegiate basketball and soccer: NCAA data and review of literature. *Am J Sports Med*. 1995;23(6):694–701.
26. Buchanan PA, Vardaxis VG. Lower-extremity strength profiles and gender-based classification of basketball players ages 9–22 years. *J Strength Cond Res*. 2009;23(2):406–19.
27. Myer GD, Ford KR, Palumbo OP, Hewett TE. Neuromuscular training improves performance and lower-extremity biomechanics in female athletes. *J Strength Cond Res*. 2005;19(1):51–60.
28. Urabe Y, Kobayashi R, Sumida S, Tanaka K, Yoshida N, Nishiwaki GA, et al. Electromyographic analysis of the knee during jump landing in male and female athletes. *Knee*. 2005;12(2):129–34.
29. Herman K, Barton C, Malliaras P, Morrissey D. The effectiveness of neuromuscular warm-up strategies, that require no additional equipment, for preventing lower limb injuries during sports participation: a systematic review. *BMC Med*. 2012;10(1):75.
30. Asadi A, Arazi H, Young WB, de Villarreal ES. The effects of plyometric training on change-of-direction ability: a meta-analysis. *Int J Sports Physiol Perform*. 2016;11(5):563–73.
31. Hewett TE, Ford KR, Myer GD. Anterior cruciate ligament injuries in female athletes: part 2, a meta-analysis of neuromuscular interventions aimed at injury prevention. *Am J Sports Med*. 2006;34(3):490–8.
32. Hewett TE, Stroupe AL, Nance TA, Noyes FR. Plyometric training in female athletes: decreased impact forces and increased hamstring torques. *Am J Sports Med*. 1996;24(6):765–73.
33. Webster KE, Feller J. Exploring the high reinjury rate in younger patients undergoing anterior cruciate ligament reconstruction. *Am J Sports Med*. 2016;44(11):2827–32.
34. Bourke H, Gordon D, Salmon L, Waller A, Linklater J, Pinczewski L. The outcome at 15 years of endoscopic anterior cruciate ligament reconstruction using hamstring tendon autograft for ‘isolated’ anterior cruciate ligament rupture. *J Bone Joint Surg*. 2012;94(5):630–7.
35. Shelbourne KD, Gray T, Haro M. Incidence of subsequent injury to either knee within 5 years after anterior cruciate ligament reconstruction with patellar tendon autograft. *Am J Sports Med*. 2009;37(2):246–51.
36. Brophy RH, Schmitz L, Wright RW, Dunn WR, Parker RD, Andrish JT, et al. Return to play and future ACL injury risk after ACL reconstruction in soccer athletes from the Multicenter Orthopaedic Outcomes Network (MOON) group. *Am J Sports Med*. 2012;40(11):2517–22.
37. Losciale JM, Zdeb RM, Ledbetter L, Reiman MP, Sell TC. The association between passing Return-to-Sport criteria and second anterior cruciate ligament injury risk: a systematic review with meta-analysis. *J Orthop Sports Phys Ther*. 2019;49(2):43–54.
38. Herzberg S, Motu’apuaka M, Lambert W, Fu R, Brady J, Guise J. The effect of menstrual cycle and contraceptives on ACL injuries and laxity: a systematic review and meta-analysis. *Orthop J Sports Med*. 2017;5(7):232596711771878.
39. Joy E, De Souza M, Nattiv A, Misra M, Williams N, Mallinson R, Gibbs J, Olmsted M, Goolsby M, Matheson G, Barrack M, Burke L, Drinkwater B, Lebrun C, Loucks A, Mountjoy M, Nichols J, Borgen J. 2014 female athlete triad coalition consensus statement on treatment and return to play of the female athlete triad. *Curr Sports Med Rep*. 2014;13(4):219–32.
40. De Souza M, West S, Jamal S, Hawker G, Gundberg C, Williams N. The presence of both an energy deficiency and estrogen deficiency exacerbate alterations of bone metabolism in exercising women. *Bone*. 2008;43(1):140–8.
41. Mountjoy M, Sundgot-Borgen J, Burke L, Carter S, Constantini N, Lebrun C, Meyer N, Sherman R, Steffen K, Budgett R, Ljungqvist A. The IOC consensus statement: beyond the female athlete triad—relative energy deficiency in sport (RED-S). *Br J Sports Med*. 2014;48(7):491–7.
42. Hame SL, LaFemina JM, McAllister DR, Schaadt GW, Dorey FJ. Fractures in the collegiate athlete. *Am J Sports Med*. 2004;32(2):446–51.
43. Rizzone KH, Ackerman KE, Roos KG, Dompier TP, Kerr ZY. The epidemiology of stress fractures in collegiate student-athletes, 2004–2005 through 2013–2014 academic years. *J Athl Train*. 2017;52(10):966–75.
44. Markey K. Stress fractures. *Clin Sports Med*. 1987;6(2):405–25.
45. Tenforde A, Carlson J, Sainani K, Chang A, Kim J, Golden N, Frederickson M. Sport and triad risk factors influence bone mineral density in collegiate athletes. *Med Sci Sports Exerc*. 2018;50(12):2536–43.
46. Chen Y-T, Tenforde AS, Frederickson M. Update on stress fractures in female athletes: epidemiology, treatment, and prevention. *Curr Rev Musculoskelet Med*. 2013;6(2):173–81.
47. Dobrindt O, Hoffmeyer B, Ruf J, Seidensticker M, Steffen IG, Fischbach F, et al. Estimation of return-

- to-sports-time for athletes with stress fracture—an approach combining risk level of fracture site with severity based on imaging. *BMC Musculoskeletal Disord.* 2012;13(1):139.
48. Mottola M, Davenport M, Ruchat S, Davies G, Poitras V, Gray C, Jaramillo Garcia A, Barrowman N, Adamo K, Duggan M, Barakat R, Chilibeck P, Fleming K, Forte M, Korolnek J, Nagpal T, Slater L, Stirling D, Zehr L. 2019 Canadian guideline for physical activity throughout pregnancy. *Br J Sports Med.* 2018;52(21):1339–46.
 49. Committee opinion no. 650: physical activity during pregnancy and the postpartum period. *Obstet Gynecol.* 2015;126(6):e135–42.
 50. Bø K, Artal R, Barakat R, Brown W, Dooley M, Evenson K, Haakstad L, Larsen K, Kayser B, Kinnunen T, Mottola M, Nygaard I, van Poppel M, Stuge B, Davies G. Exercise and pregnancy in recreational and elite athletes: 2016 evidence summary from the IOC expert group meeting, Lausanne. Part 2—the effect of exercise on the fetus, labour and birth. *Br J Sports Med.* 2016;50(21):1297–305.
 51. Bø K, Artal R, Barakat R, Brown W, Davies G, Dooley M, Evenson K, Haakstad L, Kayser B, Kinnunen T, Larsén K, Mottola M, Nygaard I, van Poppel M, Stuge B, Khan K. Exercise and pregnancy in recreational and elite athletes: 2016/17 evidence summary from the IOC expert group meeting, Lausanne. Part 3—exercise in the postpartum period. *Br J Sports Med.* 2017;51(21):1516–25.



The Young/Adolescent Basketball Player

67

Alex B. Diamond and Andrew W. Kuhn

67.1 Introduction

Basketball continues to be a popular sport among youth and adolescent athletes. Recent national survey data estimate that in the United States, 970,983 high school athletes and 4,225,321 children aged 6–12 participate in basketball on a regular basis, annually [1, 2]. While the benefits of youth sports participation are both expansive and innumerable, [3–9] there underlies an inherent risk of injury, given the training and athletic maneuverability required in basketball, such as frequent pivoting and jumping. In high school basketball, the ankle and foot have been consistently identified as most commonly injured, with diagnoses of ligament and muscle/tendon strains made most frequently [10, 11]. Similar injury patterns have been found in school-aged children and adolescent basketball players, where the most common injury diagnosed over an 11-year period was a strain or sprain to the lower extremity,

especially the ankle [12]. Although similar injury patterns exist also at both the collegiate and professional levels [13, 14], youth and adolescent athletes are a unique population as these athletes also have unique musculoskeletal and social considerations that need to be taken into account.

67.2 Development of the Youth Basketball Player

The Long-Term Athlete Development (LTAD) Model is “the planned, systematic, and progressive development of individual athletes ... and answers one fundamental question: *What needs to be done at each stage of human development to give every child the best chance of engaging in lifelong, health-enhancing physical activity; and for those with the drive and talent, the best chance of athletic success?*” [15]. Despite its generic (rather than individualized) approach and a lack of empirical evidence upon which the LTAD model is built, many national governing bodies have endorsed it as a first step toward athletic development [16]. US Basketball has developed its own “Player Development Curriculum” based on the LTAD Model in order to provide a practical, functional, and sequential development model to properly impart the game to a player

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Stage 1: Active Start (Approximate Age 0–6)

Opportunities for children to be physically active each day should be provided within a safe, fun environment.

Activity should incorporate fundamental movement skills in water, on the ground, in the air (gymnastics), and on ice and snow (skating and snowboarding) to maximize a child's physical potential.

Stage 2: Fundamentals (Approximate Age 6–9)

Children should be learning all fundamental movement skills (building overall motor skills). Again, it is stressed that participation in basketball should be limited to once or twice a week, with daily participation in other sports for further excellence.

Stage 3: Learning to Train (Approximate Age 8–12)

Incorporates learning all fundamental and basic basketball-specific skills. A 70:30 training to competition ratio is recommended.

Stage 4: Training to Train (Approximate Age 12–15)

Includes building the aerobic base, building strength towards the end of the stage and further developing basketball skills. A 60:40 training to competition ratio is recommended.

Stage 5: Training to Compete (Approximate Age 14–17)

Adolescents optimize fitness preparation as well as basketball, individual, and position-specific skills. The training to competition ratio shifts to 50:50.

Stage 6: Training to Win (Approximate Age 17+)

Includes maximizing fitness preparation as well as basketball, individual, and position-specific skills. Training to competition ratio changes to 25:75.

The Introductory Level

Introduces movement skills and builds overall motor skills. Importantly, participation in basketball once or twice per week is recommended, but daily participation in other sporting activities is stressed. Group skills competitions and introduction to team principles/concepts are also introduced at this level.

The Foundational Level

Athletes develop all fundamental and basic basketball-specific skills. The majority of time is spent on individual training. Position concepts are taught, but positions are not yet assigned.

The Advanced Level

Athletes develop the aerobic base, and build strength at the end of this level and further develop overall basketball skills. More time is spent in competition including 5x5 play, special games, as well as team oriented practices.

The Performance Level

Focuses on maximizing fitness and competition preparation as well as individual and position specific skills. Competition, including team oriented practices and other competition-specific preparations outweighs individual training.

Fig. 67.1 USA Basketball's Player Development Curriculum. (Not Shown is Stage 7: *Basketball for Life*, where athletes are retained for recreational play, coaching, administration, officiating, and other basketball-related activities)

[17]. The “Player Development Curriculum” consists of four levels and seven stages summarized in Fig. 67.1.

67.2.1 The Importance of Avoiding Early Single Sport Specialization in LTAD

Athletes who play multiple sports during early development have been shown to demonstrate improved physical and gross motor skills such as greater dynamic balance and landing techniques [18–20]. Compared to youth athletes who play multiple sports, the potential consequences of early single sport specialization include both an increased risk of emotional issues (burnout, social isolation, overdependence) as well as overuse injuries [21, 22]. In basketball, the benefits of playing multiple sports in high school have been previously demonstrated at the highest level. Rugg et al. [23] examined 237 first-round National Basketball Association (NBA) draft picks and found that while only 15% were multisport athletes in high school, the multisport cohort played in significantly more games and

were less likely to sustain a major injury during their NBA careers. While USA Basketball certainly mentions the importance of playing multiple sports during early development and training in the LTAD model, the NBA supports the concept of sport sampling and delaying single sport specialization as well, but for even longer—up until mid-adolescence to late-adolescence [24]. Research on the risks of single sport specialization in youth athletes is increasing, but more questions than answers exist including the following: the need to identify the age range at which single sport specialization is clearly detrimental for each sport and to determine the risk of improper, repetitive form on soft tissue or bony overload in a specific sport all in the setting of changing musculoskeletal tissue properties during adolescence [25]. Some of the more common overuse injuries that can result from single sport specialization in youth athletes, including basketball, include apophyseal injuries, injuries to the developing joint surfaces (osteochondral lesions), and stress reactions and fractures [21]. Ligamentous injuries and sport-related concussion are also injuries that warrant discussion.

- The Long-Term Athlete Development Model (LTAD) was developed to provide a framework for the development of fundamental skills allowing for the creation of both lifelong exercisers and elite-level athletes.
- Playing multiple sports (i.e., “sport sampling”) early in development has been shown to lead to improved physical and gross motor skills.
- Early single sport specialization may lead to psychological concerns as well as overuse injuries.

67.3 Injuries Common or Unique in the Youth Basketball Player

67.3.1 Epiphyseal Plate and Apophyseal Injuries

Children are skeletally immature with open growth plates, making the physis and epiphysis important considerations in youth and adolescent sporting injuries. There are two types of epiphyses: pressure and traction (apophyses), the latter of which is a normal bony outgrowth that arises from a separate ossification center, fuses with bone overtime, and is the attachment site of a tendon or ligament [26]. The epiphyseal plate is 2–5× weaker than the surrounding fibrous structures (ligaments, tendons, joint capsule) in children when compared to adults, so a force that would cause a ligament or tendinous tear in adults is more likely to cause an epiphyseal plate or apophyseal injury in a growing child [27–31].

Epiphyseal plate injuries, also known as Salter–Harris fractures, have been classified as follows: Type I is through the growth plate, Type II through the growth plate and metaphysis, Type III through the growth plate and epiphysis, Type IV through the growth plate, epiphysis, and metaphysis, and Type V is a crush or compression injury of the growth plate [32].

Type II fractures are the most common (75%), followed by types III (10%), IV (10%), I (5%),

and type V which is very rare [33]. Patients report a traumatic event resulting in joint pain and the inability to bear weight with limited range of motion. They may have swelling and tenderness over the physis, and it is important to note that symptoms may mimic ligamentous injury, which may lead to misdiagnosis [33]. If suspicious for a Salter–Harris fracture, radiographs are first-line imaging; CT can provide a more detailed analysis of the physis, as well as fracture extent and alignment; and MRI can offer additional information about chronic injuries, potential avascular necrosis, and more detail regarding surrounding soft tissues [34]. Type I and II fractures could be managed nonoperatively with closed reduction and casting or splinting, whereas Type III and IV fractures usually require open reduction and internal fixation [33]. Complications, including further injury and growth arrest, can arise if these fractures are not detected, which becomes important in the setting of Type V injuries as these fractures can be easily missed, involve the germinal matrix, and have a high potential for growth arrest [33].

The apophysis can be injured either acutely or chronically. Additionally, apophyseal injuries can range in severity from inflammation (apophysitis) to frank avulsion of the secondary growth center. Acute injuries and avulsion fractures are typically due to forceful, violent eccentric contractions and inherent weakness across the unfused growth plate, whereas chronic injuries are seen in athletes undergoing long periods of intensive training [26]. Data regarding acute apophyseal injuries (including avulsion fractures) and chronic apophyseal injuries in youth sports have been primarily published as case series or case reports, and in basketball specifically, injuries to the tibial tubercle (Osgood–Schlatter’s), inferior pole of the patella (Sinding Larsen Johansson) calcaneus (Sever’s), fifth metatarsal tuberosity (Iselin’s), and medial malleolus have been documented [26, 35–41].

The incidence of tibial tuberosity apophysitis (Osgood–Schlatter’s disease) is thought to be as high as 9.8% in adolescents aged 12–15, affecting boys and those who play sports such as basketball more [42–45]. Patients with Osgood–Schlatter’s disease usually present with atraumatic anterior knee pain worsened by activities, such as jumping. Modifiable risk factors such as athletic shoes

worn and playing surface used should be inquired about [46]. Patients are tender to palpation at the patellar tendon insertion site at the tibial tuberosity. Radiographs may be normal or reveal fragmentation with overlying soft tissue swelling [47] (Fig. 67.2).



Fig. 67.2 Lateral knee radiograph demonstrating Osgood–Schlatter’s disease

MR will show enlargement of the apophysis with T2 hyper intense edema and enhancement in the bone, tendon, physis, and surrounding soft tissue, specifically edema in the infrapatellar tendon, tibial tuberosity apophysis, and infrapatellar fat pad in Osgood–Schlatter’s disease [47] (Fig. 67.3a and b).

Osgood–Schlatter’s disease is self-limiting but can persist for years until the apophysis closes [44]. Conservative therapy including ice, NSAIDs, and stretching can help reduce pain levels and a knee brace or patella strap can be worn. Activity modification should be based on the level of pain the athlete is experiencing.

In the more traumatic setting, athletes with tibial tuberosity avulsion fractures will report an acute event that caused the injury often accompanied by a “pop” and inability to continue in their activity. Plain films and CT can help characterize the pattern of injury and displacement. Displacement of one or more fragments of the tuberosity anterosuperiorly and extension of the fracture through the proximal tibial ossification center into the joint with disruption of the articular cartilage are indications for surgery and can be managed with closed reduction and percutaneous pinning, ORIF with

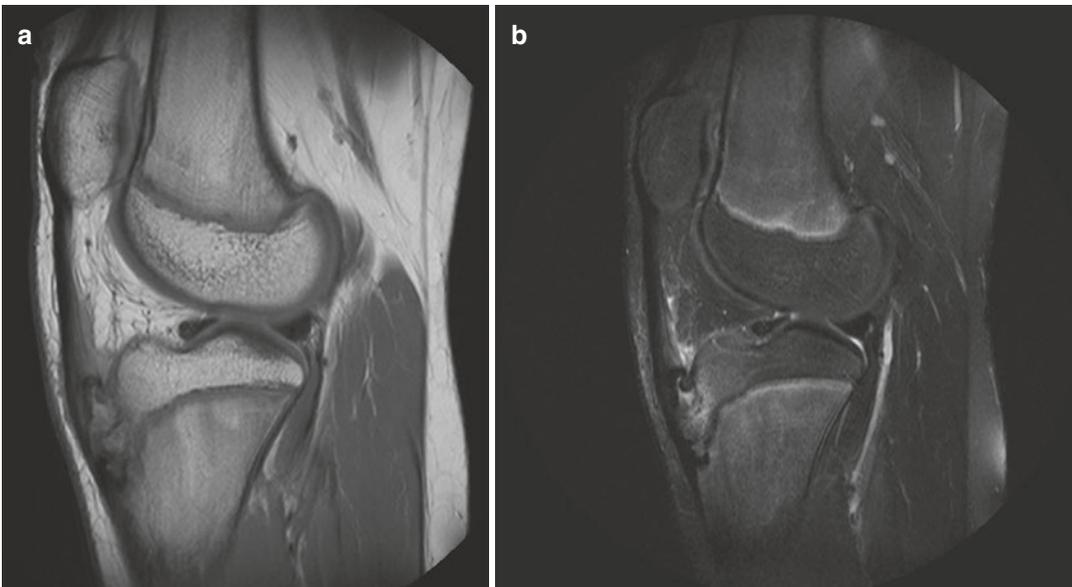


Fig. 67.3 (a and b) Sagittal T1 proton density weighted (a) and T2 fat-saturated MRI sequences demonstrating Osgood–Schlatter’s disease

compression screws, or with supplemental buttress plating [48, 49].

Calcaneal apophysitis (Sever's disease) is a common cause of heel pain in adolescent athletes, but reported incidence and prevalence rates are limited [50]. A recent study recorded incidence estimates of 3.7 per 1000 persons aged 6–17, affecting boys more often [51]. Risk factors include higher BMI as well as differences in foot posture (pronated foot while weight-bearing) and ankle joint range of motion [52]. Patients complain of heel pain and difficulty with athletic activities such as jumping and sprinting, and often limp or walk on their toes to avoid putting weight on their heels [53]. Patients are tender at the insertion of the Achilles tendon at the posterior aspect of the calcaneus. A diagnosis of Sever's disease does not require imaging but should be utilized if there is concern for other causes of heel pain including fractures, dislocations, bone cysts, an osteoid osteoma, or conditions of the tarsal bones [47, 53, 54].

Treatment includes a combination of conservative management including activity restriction/modification, stretching of plantar flexors and the Achilles tendon, NSAIDs, and heel cushioning or orthotic inserts [53]. There is limited evidence to make recommendations about the management of calcaneal avulsion fractures, and these injuries are best managed on a case-by-case basis [49].

Apophysitis of the fifth metatarsal tuberosity (Iselin's disease) is a known cause of lateral foot pain in children engaging in sports [55]. While it can result from repetitive overuse, it can also arise from a simple inversion injury [56]. Unfortunately most of the literature documenting Iselin's disease are case reports or case series [57]. Patients present with lateral foot pain when weight-bearing and/or with activity, and may also demonstrate swelling and tenderness at the base of the fifth metatarsal. Engaging the peroneus brevis with resisted eversion and ranging the ankle may recreate pain. Oblique view plain films are most useful and may show a fleck of bone oriented longitudinally to the long axis of the metatarsal [55]. Radiographs and the clinical history are useful in ruling out other variants or pathology affecting the base of the fifth metatar-

sal including a Jones fracture, avulsion fracture, stress fracture, and Os Vesalianum [58]. Treatment for Iselin's disease often involves conservative management including rest, activity modification, and pain control; however in some cases immobilization with a boot or short-leg cast may be necessary [57, 59].

- Youth athletes are more likely to injure their physis or epiphysis rather than their soft tissues.
- Osgood–Schlatter's, Sever's, and Iselin's disease can be causes of pain in youth basketball players.
- These injuries can be managed conservatively with rest, activity modification, physical therapy, and pain control, whereas some avulsion fractures may require surgical fixation.

67.3.2 Osteochondral Injury

Osteochondral injury, most notably juvenile osteochondritis dissecans (JOCD), has been identified as a frequent cause of joint, especially knee, pain in adolescent athletes and also in basketball players [60–62]. The incidence of JOCD is estimated to be between 11 and 29 per 100,000 individuals under the age of 19 [63, 64]. The true etiology of JOCD is unknown but is thought to be multifactorial, including focal repetitive trauma at sites with unique vascular architecture, triggering ischemic events that cause subchondral bone to separate with cracking of the overlying articular cartilage and loosening of the piece further in later stages [65, 66]. Kessler et al. [67] found that JOCD in the knee has a higher incidence in those aged 12–19 compared to other age groups, and more so in boys than in girls. The authors also found that the medial femoral condyle was the most common location (63.6%) for JOCD, followed by the lateral femoral condyle (32.5%) and patella (1.5%) [67].

In the early stages of JOCD, athletes will present with nonspecific knee pain, stiffness with or

after activities, and occasional effusions, whereas in the later stages, mechanical symptoms such as grinding, locking, and catching are more common when loose or detached lesions are present [68–72]. Wilson described a test to identify the presence of JOCD in the medial femoral condyle, [73] which involves internally rotating the tibia during 30–90° of knee flexion to recreate pain, and then externally rotating the tibia to abate symptoms; however, the test has proven to be of limited diagnostic value and there are no pathognomic symptoms or signs of JOCD [74–76].

JOCD is often seen on plain radiographs and appear as a circumscribed area of subchondral bone separated by a crescent-shaped sclerotic radiolucent outline [77]. One of most commonly utilized classification systems for OCD on plain films is that of Berndt and Hardy [78]. MRI has a high degree of sensitivity and is useful for the diagnosis, judging stability, and follow-up of OCD lesions due to its ability to assess surrounding cartilage and subchondral bone, which are unable to be evaluated fully on plain films [76] (Fig. 67.4a, b).

Factors such as a high T2-signal intensity line surrounding the lesion, a fluid-filled cartilage defect, the presence of subchondral cysts, and a high T2-signal fracture line in the articular cartilage have been shown to be useful in characterizing OCD lesion instability in adults (which directs management) [79]; however, MR and these characteristics do not reliably predict instability well in skeletally immature patients with great certainty [77, 80–84].

A large majority (50–67%) of JOCD lesions heal in 6–12 months with nonoperative management [83, 85–88] and thus initial treatment includes immobilization, limited weight-bearing, and/or activity restriction for at least 3–6 months [76]. Diagnostic arthroscopy and surgical intervention are recommended for stable lesions refractory to nonoperative management and for unstable lesions, including those with an intact articular surface but with an atypical location, large size, mechanical symptoms, or closing physis, as well as for lesions with clinical and imaging signs suggestive of instability [76]. One of the first diagnostic arthroscopic classification systems developed

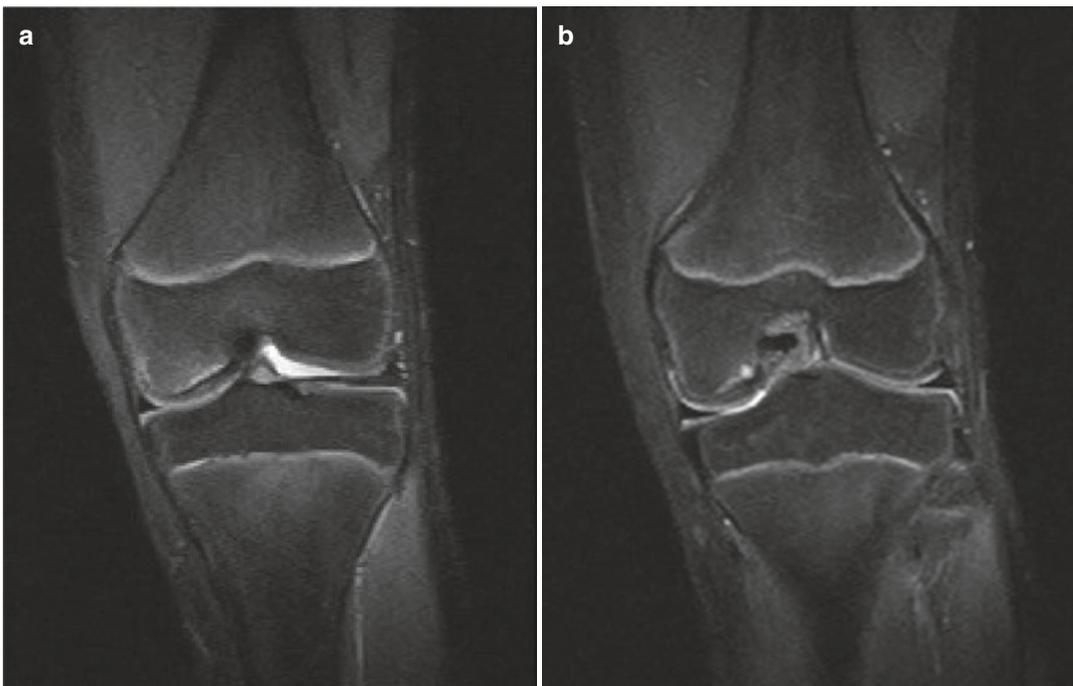


Fig. 67.4 (a and b) Coronal T2 MRI sequences demonstrating an osteochondral lesion about the medial femoral condyle (MRI)

was Guhl's [89]. The Research in Osteochondritis of the Knee (ROCK) group has since recently developed a diagnostic arthroscopic classification system with high interrater reliability that first separates lesions based on whether or not they are immobile or mobile and then based on the severity of cartilage damage [90]. Surgical options for these lesions include retroarticular drilling, with/without in-situ internal fixation; debridement, reduction, and internal fixation with/without bone graft; or if needed osteochondral autograft (OATS), fresh osteochondral allograft, autologous chondrocyte implantation (ACI), or matrix-induced autologous chondrocyte implantation (MACI), all depending on displacement and whether or not the lesion is salvageable [76].

- JOCD can be a cause of knee pain in adolescent athletes and basketball players.
- It has a predilection for older boys at the medial femoral condyle and is thought to have a multifactorial etiology.
- Plain films and MRI may help characterize these lesions.
- Most lesions can successfully be treated nonoperatively, but diagnostic arthroscopy and operative intervention may be needed if the lesion is unstable or if the patient's symptoms are refractory to conservative management.

67.3.3 Stress Fractures

Stress fractures in youth or adolescent athletes are typically due to abnormal chronic repetitive stress on bone, leading to bone remodeling with resorption outpacing replacement, ultimately causing a stress reaction and eventual fracture [91]. In a large epidemiologic study evaluating stress fractures in high school athletes, fractures were most common in track and field, cross country, and gymnastics; however, girls' basketball was also considered a "high-risk sport" (injury rate: 2.71 per 100,000 AEs) [92]. These

data are similar to a study by Field et al. [93] who found that in 267 preadolescent and adolescent girls who sustained stress fractures, the sport of basketball was also considered a risk factor. Importantly, the female sex is a known risk factor for stress fractures, as younger nulliparous women with lower BMIs who train frequently are likely to have menstrual disturbances resulting in lowered estrogen levels and thus lower bone mineral density, accelerated bone remodeling, and negative calcium balance [94]. The most common anatomical locations of stress fractures were found to be the lower leg and foot in both boys' and girls' high school basketball [92]. Similarly, in a case series of 196 adolescent and older athletes (mean age 20.1 years old) who sustained stress fractures, basketball players typically injured their tibial shaft and medial malleolus or metatarsal bones [95].

On presentation and exam [96, 97], athletes with stress fractures report an atraumatic insidious onset of pain after an increase in intensity or volume of training. They report that the pain occurs with activity and subsides with rest. The athlete demonstrates tenderness to bony palpation at the area of the suspected stress fracture, and for lower extremity stress fractures, a "fulcrum test" (for femoral stress fractures) or a "single leg hop test" (for tibial stress fractures) can reproduce the pain and raise the suspicion for stress fractures versus other conditions, such as medial tibial stress syndrome. Plain films of the affected extremity should be taken first if suspicion is high. Radiographs may be negative in the setting of early stress fractures but when present or healing, it may demonstrate subtle linear sclerosis, focal endosteal reaction, and a fracture through one cortex with superimposed periosteal reaction [94] (Fig. 67.5a and b).

MR imaging, which has very high sensitivity (100%) and specificity (85%) for stress fractures, is a second-line imaging modality that can be obtained when radiographs are normal, pain is of unknown etiology, or in athletes requiring a definitive diagnosis [91]. MR with fat suppression has shown promise in grading the progressive stages of stress fracture severity [98] (Fig. 67.6a–c).

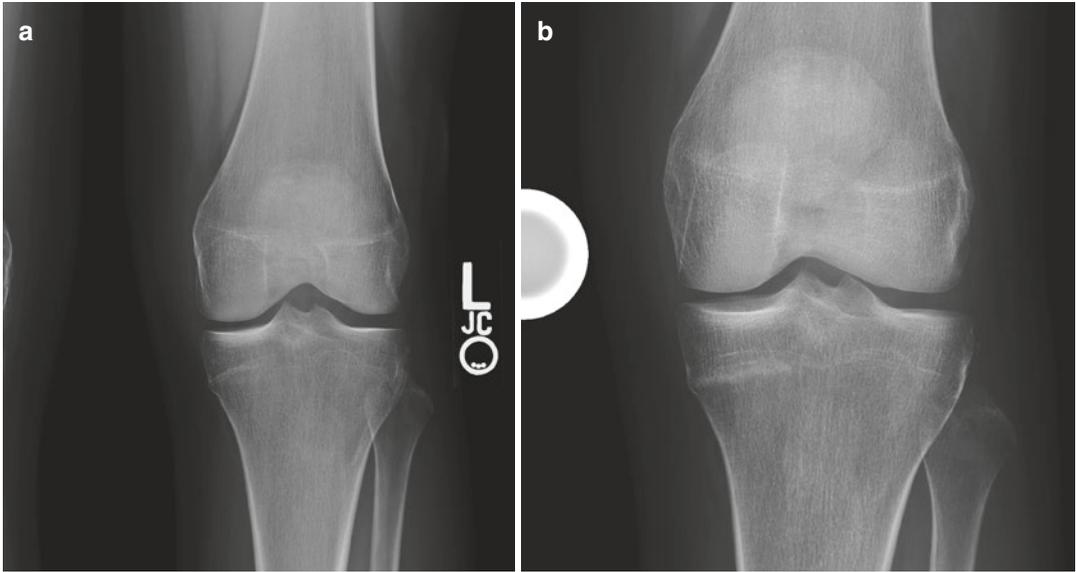


Fig. 67.5 (a and b) AP plain films of the left knee showing an occult fracture on presentation (a) and 3 weeks later a healing stress fracture in the medial proximal tibia (b)

CT can be useful in identifying longitudinal fracture lines and stress fractures of the spine, but can miss transverse fractures without reformations, and nuclear medicine scintigraphy affords the opportunity to see tracer uptake in all three phases, but has a low specificity for stress fractures, potentially resulting in a high rate of false positives [91].

In general, those with low-grade stress injuries have been shown to return to activity significantly faster than those with more severe ones (grade I: 3.3 weeks, grade II: 5.5 weeks, grade III: 11.4 weeks, and grade IV: 14.3 weeks) [99]. However, management depends not only on the grade of the fracture but also on whether or not they are in “low-” or “high-risk” areas. “High-risk” stress fractures have a higher risk of protracted recovery, progression to complete fracture, delayed union, nonunion, and chronic pain because these specific locations (tension sided femoral neck, patella, anterior tibia, medial malleolus, talus, tarsal navicular, proximal fifth metatarsal, and the great toe sesamoids) have a region of maximal tensile load in a zone of diminished blood flow that is vulnerable to stress injury with suboptimal healing [100]. Low-grade stress

injuries at “high-risk” sites should be managed with absolute rest before returning to play; however, these and other fractures in “high-risk” locations may eventually require operative management to (1) speed fracture healing and allow earlier return to play (tibial shaft “dreaded black line” with intramedullary nailing), (2) reduce the risk of refracture (internal fixation of a proximal fifth metatarsal fracture), and (3) to prevent a catastrophic fracture progression (internal fixation of a femoral neck stress fracture) [101]. On the contrary, “low-risk” fractures (femoral shaft, tibial shaft, fibula, calcaneus, metatarsal shaft) can be managed conservatively, typically with immobilization or activity and risk modification depending on the athlete’s symptoms, goals, and understanding of risks and benefits. For instance, athletes who are in-season that have pain with no functional limitations should be able to continue participation using their pain to guide their activities, whereas those who have pain with functional limitations should rest and modify their activities to reach a functional level of pain [101]. As mentioned, “low-risk” stress fractures usually heal when an athlete is limited to pain-free activity for 2–12 weeks [102].

- Basketball is a risk factor for stress fractures, particularly in adolescent girls.
- The tibial shaft, medial malleolus, and metatarsal bones most commonly affected in basketball.
- Plain films are first line for imaging, followed by MRI if radiographs are negative and there is a high suspicion for a stress fracture.
- Management depends not only on the grade but also on the anatomical location and whether or not the stress fracture is considered “low” or “high risk.”

67.3.4 Ligamentous Injuries

In a study of U18 adolescent basketball teams in Finland over three consecutive seasons, the ankle was the most commonly injured (48%) body region, with the majority of injuries involving a joint or ligament (67%), with the most commonly injured ligament structure being the lateral complex of the ankle joint [103]. A study of US high school athletes reported that girls’ and boys’ basketball accounted for the highest incidence of ankle sprains across nine different team sports, accounting for nearly a quarter (23.8%) of all ankle sprains recorded [104]. The study reported



Fig. 67.6 (a–c) Sagittal T1 proton density weighted (a), and coronal (b) and sagittal (c) T2 MRI sequences demonstrating a stress fracture in the medial proximal tibia

an incidence rate of 7.74 injuries per 10,000 AEs. Finally, in a study of children and adolescent basketball injuries treated in emergency departments across the United States between 1997 and 2007, strains and sprains (44.8%) of the ankle (23.8%) were the most commonly made diagnosis.

Most lateral ankle sprains occur during landing after jumping while either rebounding or shooting the ball [104]. The mechanism of injury in basketball players has been shown to involve sudden inversion with or without internal rotation, with low levels of plantar flexion [105]. Some risk factors identified for lateral ankle sprains include a higher BMI, slower eccentric inversion strength, fast concentric plantar flexion strength, passive inversion joint position sense, and the reaction time of the peroneus brevis [106].

Typically patients will present with bruising, swelling, and tenderness over the anterolateral ankle. They may demonstrate tenderness at the lateral ankle ligament complex, including the anterior talofibular ligament (ATFL), calcaneofibular ligament (CFL), and posterior talofibular ligament (PTFL). Lateral ankle sprains have been graded based on the severity of the strain, with grade II and III injuries demonstrating partial or complete tearing of the ligament, consistent with anterior drawer test laxity with and without firm end points, respectively [107]. The Ottawa Ankle Rules have been previously validated in children with ankle injuries, and therefore plain films can be taken if suspicion for a fracture is high [108]. Initial treatment should focus on rest, ice, compression, and elevation immediately after the injury, as well as early range of motion to help reduce swelling [107]. Crutches or a walking boot can be utilized intermittently if unable to bear weight fully initially. Later on, protection of inversion with controlled stress on the ligament through motion, stretching, and strengthening should be undertaken to restore normal function and reduce the risk of recurrent injury [109]. Functional bracing or taping can help provide additional stability by compensating for ligamentous laxity. Generally, the higher the grade of sprain, the longer the recovery, but once an athlete can demonstrate normal heel-to-toe gait without

pain or a limp and can progress through functional testing relevant for the sport they are returning to, such as jumping without limitations for basketball, the athlete can return to play [107]. In cases of severe functional and mechanical instability with recurrent sprains refractory to nonsurgical management, operative repair may be necessary [110]. Medial (deltoid ligament; eversion mechanism) and high ankle (anterior and/or posterior inferior talofibular ligament, interosseous ligament, and syndesmosis; excessive external rotation of the ankle mechanism) sprains can be more significant injuries and involve the syndesmosis, leading to ankle instability that may require operative management [107].

- Ankle sprains are one of the most common injuries in basketball.
- They occur most often when landing on an inverted ankle after rebounding or jumping.
- Most injuries can be managed nonoperatively, and functional bracing can be used to provide additional ligamentous stability.
- Surgical management may be necessary in cases of severe functional and mechanical instability with recurrent sprains, refractory to nonoperative management.

Injury to the anterior cruciate ligament (ACL) of the knee also requires discussion, especially as it relates to youth basketball. In middle school and high school Japanese athletes, female high school basketball players had the highest incidence of ACL injury [111]. In the USA, girls' basketball had the third highest rate of ACL injury (10.3 per 10,000 AEs), only behind boys' football and girls' soccer [112]. The authors found that noncontact mechanisms made up the majority of ACL injuries in both boys' (50.8%) and girls' (46.0%) basketball [112]. Another study utilizing video analysis also found that most injuries were occurred during landing and

without contact. But those movement and landing patterns were likely affected by opponent before the injury [113]. The likely conditions for a noncontact injury are an anterior tibial shear force, knee abduction, and tibial internal rotation moments [114]. In a recent systematic review, girls were at a 3.80 times relative risk for ACL injury compared to boys in the high school basketball population [115]. Along with female sex, other non-modifiable (generalized joint laxity, knee recurvatum, increased lateral tibial slope, decreased size of intercondylar notch, structural lower extremity valgus, femoral anteversion, limb length discrepancy, family history, contralateral knee ACL injury) and modifiable (neuromuscular control patterns, gross biomechanical movement patterns, environmental factors, hamstring and quadriceps strength, core strength and proprioception, knee flexion angle during jumping/landing, dynamic valgus, and sports played) have all been previously studied and discussed [116].

Most patients will present with a traumatic effusion. A hemarthrosis combined with a positive Lachman's or anterior drawer test coupled with a suggestive history raises the positive likelihood of an ACL tear considerably [117, 118]. MR is the preferred imaging modality for ACL injuries [119] and has been shown to be 95% sensitive and 88% specific in diagnosing ACL tears in the pediatric population [120]. Skeletal age should also be determined for children and adolescents with open physis in cases where operative management is required [121]. Partial ACL tears, where there is not a complete disruption of all the ACL fibers, occur more frequently in children than in adults, and nonoperative treatment has been successful in select patients [121]. For patients with complete tears, ACL reconstruction is needed. Given that children have open physis, a variety of reconstruction options have been developed including extraphyseal, all-epiphyseal, partial transphyseal, and transphyseal [121]. It is also worth noting that patients who are skeletally immature can fracture their tibial eminence at the insertion of the ACL rather than tearing or rupturing their ACL itself. The mechanisms, presentation, and

workup are similar to that of an ACL injury. Plain radiographs can be utilized to visualize the fracture. CT and MRI can be used to better characterize the fracture or to assess for concomitant intraarticular injuries (Fig. 67.7a–c).

Meyers and McKeever developed a classification system for tibial spine fractures, with Type I being non-displaced, Type II displaced anteriorly with an intact posterior cortical hinge, and Type III being completely displaced [122]. Type I injuries can be managed nonoperatively with immobilization in a cast, whereas Type III and, frequently, Type II fractures can be managed operatively by a variety of reported techniques [123].

- ACL injuries are common in basketball, especially in girls and are usually due to a noncontact mechanism.
- Most patients present with a traumatic knee effusion and ligamentous laxity.
- MRI can help characterize the injury and any concomitant injuries, and radiographs should be taken to determine skeletal age.
- Partial tears can be managed nonoperatively, whereas complete ruptures will require reconstruction.
- Skeletally immature athletes can fracture their tibial eminence instead of rupturing their ACL.

67.3.5 Sport-Related Concussion

While there is no single agreed upon definition for sport-related concussion (SRC), it has been described as the rapid onset of short-lived neurological impairment often due to a direct blow to the head, face, neck, or elsewhere on the body that ultimately resolves spontaneously [124]. In basketball, most concussions are due to player–player or player–surface contact while defending, chasing a loose ball, rebounding, or during general play [125, 126]. In a recent systematic review and meta-analysis, the incidence of con-

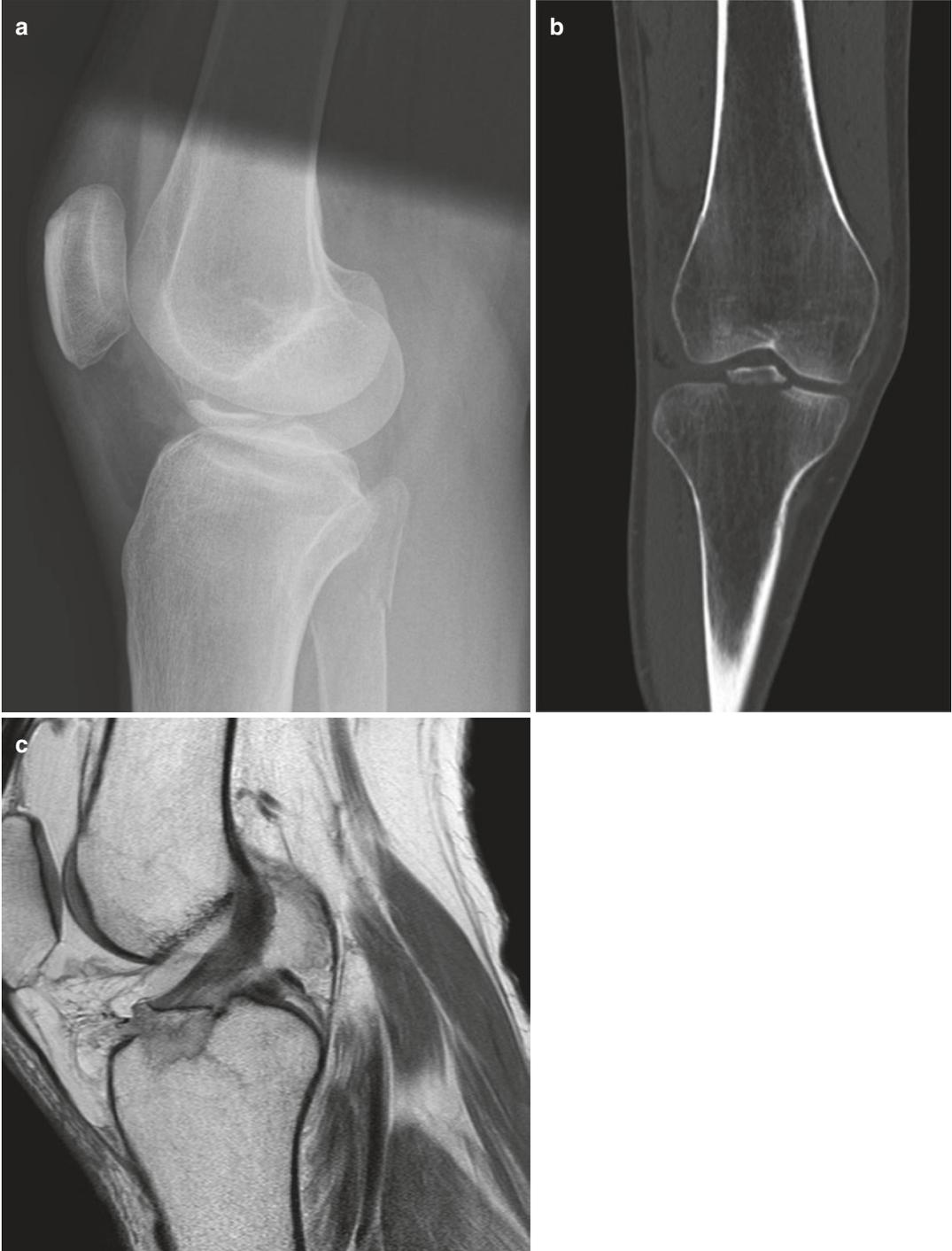


Fig. 67.7 (a–c) ACL tibial spine fracture demonstrated on a lateral plain film (a), coronal CT (b), and T1 MRI sequence (c)

cussion in youth basketball was found to be 0.13 per 1000 athlete exposures (AE), which was the sixth highest incidence rate of SRC across 12 different sports in athletes under the age of 18 [127].

A diagnosis of SRC is clinical, which can be challenging, given that nonspecific symptoms such as headaches, mood changes, dizziness, and visual changes can also be due to other causes. The American Medical Society for Sports Medicine (AMSSM) [128] recommends that when a provider becomes aware of a potential SRC, an event history should be taken from the athlete as well as someone who witnessed the event. A brief exam including how the athlete responds to elements of orientation, memory, concentration, and balance should be conducted as well as evaluating their cervical spine. If suspicion is still high for SRC after the brief assessment, more thorough and specific concussion assessments can be administered. Sideline concussion tests, including the SCAT5 and Child SCAT5, offer a standardized approach to sideline evaluation, including the assessment of neurological functioning, a symptom checklist, a brief cognitive assessment, and a balance test [129, 130]. All tests should be interpreted in combination with relevant clinical information.

Most child and adolescent athletes with SRC will recover spontaneously within 2–4 weeks; however, a small subset will go on to develop prolonged symptoms, known as “post-concussive syndrome” (PCS) [131]. Risk factors for PCS include a previous history of concussion, preinjury mood disorders, family history of mood disorders, and delayed symptom onset [132], factors which should be documented and taken note of as part of the initial workup.

SRC used to be managed with rest, strictly, until symptoms were completely resolved; however, new data are emerging demonstrating that sub-symptom exacerbation levels of light activity and placing athletes in stimulating environments may actually be more beneficial during recovery from SRC [133–137]. Most athletes return to sport based on a graduated return to play protocol [138], moving from rest to symptom-limited activity, light aerobic exercise, sport-specific exercise, noncontact drill training drill, and full

contact practice before returning to normal gameplay. Athletes can progress through the stages so long as their symptoms do not recur. In youth and adolescent athletes, return to learn is also an important consideration. Students who sustain an SRC may need academic accommodations and a multidisciplinary team to minimize worsening of their symptoms at school, including but not limited to, shortened school days or classes, allowance for breaks, a reduction in workload or assignments, preferential classroom seating, extra time to complete assignments, and quiet rooms or the ability to avoid noisy areas [139, 140]. Additionally, some athletes who have prolonged symptoms may require more involved plans, such as a 504 or, while unlikely, an individualized educational plan (IEP) [141]. Students should be performing at their academic baseline before returning to sports [141–143].

- Most SRCs in basketball result from player–player or player–surface contact while defending, chasing a loose ball, rebounding, or during general play.
- Standardized SRC tests should not be used in lieu of a clinical exam.
- Most athletes will return to play after their symptoms resolve spontaneously in 2–4 weeks and after advancing through a graduated return to play protocol.
- Athletes may require academic accommodations to ensure an adequate return to learn.

67.4 Social, Other Considerations, and Conclusions

Youth sports are unique because it requires effective communication, coordination, and maintenance of relationships among multiple adults, including coaches, parents, and practitioners all of whom are vested stakeholders in an athlete’s sport experience [144]. Social influence has been shown to be moderated by age such that younger athletes may be more influenced by their parents,

whereas older athletes by their peers [145]. Athletes who participate in athletics in positive social climates and with a positive coaching relationship report higher levels of enjoyment, effort, and have greater intentions to continue playing sports [145, 146]. On the contrary, coaches can cause perfectionist desires and concerns in youth athletes [147]. Padaki et al. surveyed 235 athletes between the ages of 7 and 18 and found that approximately one-third of the players were told not to participate in other sports, with specialized athletes reporting this significantly more often [148]. As previously mentioned, single sport specialization can lead to emotional issues including burnout and overuse injuries. Additionally and unfortunately, coaches as well as parents, peers, and even medical professionals have been reported as culprits of all forms of child abuse in sport including sexual, physical, and psychological [149]. In addition to understanding the musculoskeletal injuries unique to youth athletes and basketball players, health practitioners also have a duty to recognize and prevent abuse, as well as educate parents and their families, and advocate for programmatic and cultural changes that protect and keep youth athletes healthy [149].

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References

1. The Aspen Institute. State of play 2018: trends and developments. Washington, DC: The Aspen Institute; 2018.
2. The National Federation of State High School Associations. (2015) 2014-15 High School Athletics Participation Survey.
3. Reverdito RS, Galatti LR, Carvalho HM, et al. Developmental benefits of extracurricular sports participation among Brazilian youth. *Percept Mot Skills*. 2017;124(5):946–60.
4. Bruner MW, Balish SM, Forrest C, et al. Ties that bond: youth sport as a vehicle for social identity and positive youth development. *Res Q Exerc Sport*. 2017;88(2):209–14.
5. Oliveira A, Monteiro A, Jacome C, Afreixo V, Marques A. Effects of group sports on health-related physical fitness of overweight youth: a systematic review and meta-analysis. *Scand J Med Sci Sports*. 2017;27(6):604–11.
6. Howie EK, McVeigh JA, Smith AJ, Straker LM. Organized sport trajectories from childhood to adolescence and health associations. *Med Sci Sports Exerc*. 2016;48(7):1331–9.
7. McCabe KO, Modecki KL, Barber BL. Participation in organized activities protects against adolescents' risky substance use, even beyond development in conscientiousness. *J Youth Adolesc*. 2016;45(11):2292–306.
8. Diamond AB. The cognitive benefits of exercise in youth. *Curr Sports Med Rep*. 2015;14(4):320–6.
9. Taliaferro LA, Eisenberg ME, Johnson KE, Nelson TF, Neumark-Sztainer D. Sport participation during adolescence and suicide ideation and attempts. *Int J Adolesc Med Health*. 2011;23(1):3–10.
10. Borowski LA, Yard EE, Fields SK, Comstock RD. The epidemiology of US high school basketball injuries, 2005–2007. *Am J Sports Med*. 2008;36(12):2328–35.
11. Clifton DR, Hertel J, Onate JA, et al. The first decade of web-based sports injury surveillance: descriptive epidemiology of injuries in US high school Girls' basketball (2005–2006 through 2013–2014) and National Collegiate Athletic Association Women's Basketball (2004–2005 through 2013–2014). *J Athl Train*. 2018;53(11):1037–48.
12. Randazzo C, Nelson NG, McKenzie LB. Basketball-related injuries in school-aged children and adolescents in 1997–2007. *Pediatrics*. 2010;126(4):727–33.
13. Drakos MC, Domb B, Starkey C, Callahan L, Allen AA. Injury in the national basketball association: a 17-year overview. *Sports Health*. 2010;2(4):284–90.
14. Zuckerman SL, Wegner AM, Roos KG, et al. Injuries sustained in National Collegiate Athletic Association men's and women's basketball, 2009/2010–2014/2015. *Br J Sports Med*. 2018;52(4):261–8.
15. Balyi I, Way R, Higgs C. Long-term athlete development. Chelsea: Sheridan Books; 2018.
16. Ford P, De Ste Croix M, Lloyd R, et al. The long-term athlete development model: physiological evidence and application. *J Sports Sci*. 2011;29(4):389–402.
17. USA Basketball. USA basketball youth development guidebook. Colorado Springs, CO: USA Basketball; 2014.
18. Fransen J, Pion J, Vandendriessche J, et al. Differences in physical fitness and gross motor coordination in boys aged 6–12 years specializing in one versus sampling more than one sport. *J Sports Sci*. 2012;30(4):379–86.
19. Miller MM, Trapp JL, Post EG, et al. The effects of specialization and sex on anterior Y-balance performance in high school athletes. *Sports Health*. 2017;9(4):375–82.
20. DiStefano LJ, Beltz EM, Root HJ, et al. Sport sampling is associated with improved landing technique in youth athletes. *Sports Health*. 2018;10(2):160–8.
21. Smucny M, Parikh SN, Pandya NK. Consequences of single sport specialization in the pediatric and adolescent athlete. *Orthop Clin North Am*. 2015;46(2):249–58.

22. Bell DR, Post EG, Biese K, Bay C, Valovich McLeod T. Sport specialization and risk of overuse injuries: a systematic review with meta-analysis. *Pediatrics*. 2018;142(3)
23. Rugg C, Kadoor A, Feeley BT, Pandya NK. The effects of playing multiple high school sports on National Basketball Association Players' propensity for injury and athletic performance. *Am J Sports Med*. 2018;46(2):402–8.
24. DiFiori JP, Brenner JS, Comstock D, et al. Debunking early single sport specialization and reshaping the youth sport experience: an NBA perspective. *Br J Sports Med*. 2017;51(3):142–3.
25. Feeley BT, Agel J, LaPrade RF. When is it too early for single sport specialization? *Am J Sports Med*. 2016;44(1):234–41.
26. Longo UG, Ciuffreda M, Locher J, Maffulli N, Denaro V. Apophyseal injuries in children's and youth sports. *Br Med Bull*. 2016;120(1):139–59.
27. Caine D, DiFiori J, Maffulli N. Physeal injuries in children's and youth sports: reasons for concern? *Br J Sports Med*. 2006;40(9):749–60.
28. Schwab SA. Epiphyseal injuries in the growing athlete. *Can Med Assoc J*. 1977;117(6):626–30.
29. Rossi F, Dragoni S. Acute avulsion fractures of the pelvis in adolescent competitive athletes: prevalence, location and sports distribution of 203 cases collected. *Skelet Radiol*. 2001;30(3):127–31.
30. Micheli LJ. Overuse injuries in children's sports: the growth factor. *Orthop Clin North Am*. 1983;14(2):337–60.
31. Arnaiz J, Piedra T, de Lucas EM, et al. Imaging findings of lower limb apophysitis. *AJR Am J Roentgenol*. 2011;196(3):W316–25.
32. Brown JH, DeLuca SA. Growth plate injuries: Salter-Harris classification. *Am Fam Physician*. 1992;46(4):1180–4.
33. Levine RH, Foris LA, Nezwek TA, Waseem M. Salter-Harris fractures. *Treasure Island (FL): StatPearls*; 2019.
34. Jawetz ST, Shah PH, Potter HG. Imaging of physical injury: overuse. *Sports Health*. 2015;7(2):142–53.
35. Canale ST, Williams KD. Iselin's disease. *J Pediatr Orthop*. 1992;12(1):90–3.
36. Ozer H, Turanli S, Baltaci G, Tekdemir I. Avulsion of the tibial tuberosity with a lateral plateau rim fracture: case report. *Knee Surg Sports Traumatol Arthrosc*. 2002;10(5):310–2.
37. Ergun M, Taskiran E, Ozgurbuz C. Simultaneous bilateral tibial tubercle avulsion fracture in a basketball player. *Knee Surg Sports Traumatol Arthrosc*. 2003;11(3):163–6.
38. Crosby LA, McMullen ST. Heel pain in an active adolescent? *Phys Sportsmed*. 1993;21(4):89–91.
39. Ishii T, Miyagawa S, Hayashi K. Traction apophysitis of the medial malleolus. *J Bone Joint Surg Br*. 1994;76(5):802–6.
40. Lokiec F, Wientroub S. Calcaneal osteochondritis: a new overuse injury. *J Pediatr Orthop B*. 1998;7(3):243–5.
41. Nakano E. (2006) Sever's disease. *Radiology cases in pediatric emergency medicine*. 1(Case 20).
42. Indiran V, Jagannathan D. Osgood-Schlatter Disease. *N Engl J Med*. 2018;378(11):e15.
43. Nkaoui M, El Alouani EM. Osgood-Schlatter disease: risk of a disease deemed banal. *Pan Afr Med J*. 2017;28:56.
44. Smith JM, Varacallo M. Osgood Schlatter's disease (Tibial tubercle Apophysitis). *Treasure Island (FL): StatPearls*; 2019.
45. Kujala UM, Kvist M, Heinonen O. Osgood-Schlatter's disease in adolescent athletes. Retrospective study of incidence and duration. *Am J Sports Med*. 1985;13(4):236–41.
46. Launay F. Sports-related overuse injuries in children. *Orthop Traumatol Surg Res*. 2015;101(1 Suppl):S139–47.
47. O'Dell MC, Jaramillo D, Bancroft L, Varich L, Logsdon G, Servaes S. Imaging of sports-related injuries of the lower extremity in pediatric patients. *Radiographics*. 2016;36(6):1807–27.
48. Ogden JA, Tross RB, Murphy MJ. Fractures of the tibial tuberosity in adolescents. *J Bone Joint Surg Am*. 1980;62(2):205–15.
49. Schiller J, DeFroda S, Blood T. Lower extremity avulsion fractures in the pediatric and adolescent athlete. *J Am Acad Orthop Surg*. 2017;25(4):251–9.
50. Scharfbillig RW, Jones S, Scutter SD. Sever's disease: what does the literature really tell us? *J Am Podiatr Med Assoc*. 2008;98(3):212–23.
51. Wiegerinck JI, Yntema C, Brouwer HJ, Struijs PA. Incidence of calcaneal apophysitis in the general population. *Eur J Pediatr*. 2014;173(5):677–9.
52. James AM, Williams CM, Luscombe M, Hunter R, Haines TP. Factors associated with pain severity in children with calcaneal apophysitis (sever disease). *J Pediatr*. 2015;167(2):455–9.
53. Ramponi DR, Baker C. Sever's disease (Calcaneal Apophysitis). *Adv Emerg Nurs J*. 2019;41(1):10–4.
54. Rachel JN, Williams JB, Sawyer JR, Warner WC, Kelly DM. Is radiographic evaluation necessary in children with a clinical diagnosis of calcaneal apophysitis (sever disease)? *J Pediatr Orthop*. 2011;31(5):548–50.
55. Gillespie H. Osteochondroses and apophyseal injuries of the foot in the young athlete. *Curr Sports Med Rep*. 2010;9(5):265–8.
56. Chauvin NA, Jaimes C, Khwaja A. Ankle and foot injuries in the young athlete. *Semin Musculoskelet Radiol*. 2018;22(1):104–17.
57. Forrester RA, Eyre-Brook AI, Mannan K. Iselin's disease: a systematic review. *J Foot Ankle Surg*. 2017;56(5):1065–9.
58. Kishan TV, Mekala A, Bonala N, Sri Pavani B. Iselin's disease: traction apophysitis of the fifth metatarsal base, a rare cause of lateral foot pain. *Med J Armed Forces India*. 2016;72(3):299–301.
59. Deniz G, Kose O, Guneri B, Duygun F. Traction apophysitis of the fifth metatarsal base in a child: Iselin's disease. *BMJ Case Rep*. 2014;2014

60. Price MJ, Tuca M, Nguyen J, et al. Juvenile osteochondritis dissecans of the trochlea: a cohort study of 34 trochlear lesions associated with sporting activities that load the patellofemoral joint. *J Pediatr Orthop*. 2018;
61. Filho JS, Garms E, Sayum J, et al. Bilateral osteochondritis dissecans of the knee in a basketball player: case report. *Rev Bras Ortop*. 2012;47(2):257–9.
62. Takahashi Y, Nawata K, Hashiguchi H, et al. Bilateral osteochondritis dissecans of the lateral trochlea of the femur: a case report. *Arch Orthop Trauma Surg*. 2008;128(5):469–72.
63. Kocher MS, Tucker R, Ganley TJ, Flynn JM. Management of osteochondritis dissecans of the knee: current concepts review. *Am J Sports Med*. 2006;34(7):1181–91.
64. Ananthaharan A, Randsborg PH. Epidemiology and patient-reported outcome after juvenile osteochondritis dissecans in the knee. *Knee*. 2018;25(4):595–601.
65. Martel G, Kiss S, Gilbert G, et al. Differences in the vascular tree of the femoral trochlear growth cartilage at osteochondrosis-susceptible sites in foals revealed by SWI 3T MRI. *J Orthop Res*. 2016;34(9):1539–46.
66. Bruns J, Werner M, Habermann C. Osteochondritis dissecans: etiology, pathology, and imaging with a special focus on the knee joint. *Cartilage*. 2018;9(4):346–62.
67. Kessler JI, Nikizad H, Shea KG, et al. The demographics and epidemiology of osteochondritis dissecans of the knee in children and adolescents. *Am J Sports Med*. 2014;42(2):320–6.
68. Williams JS Jr, Bush-Joseph CA, Bach BR Jr. Osteochondritis dissecans of the knee. *Am J Knee Surg*. 1998;11(4):221–32.
69. Cahill BR, Ahten SM. The three critical components in the conservative treatment of juvenile osteochondritis dissecans (JOCD). Physician, parent, and child. *Clin Sports Med*. 2001;20(2):287–98.
70. Glancy GL. Juvenile osteochondritis dissecans. *Am J Knee Surg*. 1999;12(2):120–4.
71. Cain EL, Clancy WG. Treatment algorithm for osteochondral injuries of the knee. *Clin Sports Med*. 2001;20(2):321–42.
72. Robertson W, Kelly BT, Green DW. Osteochondritis dissecans of the knee in children. *Curr Opin Pediatr*. 2003;15(1):38–44.
73. Wilson JN. A diagnostic sign in osteochondritis dissecans of the knee. *J Bone Joint Surg Am*. 1967;49(3):477–80.
74. Conrad JM, Stanitski CL. Osteochondritis dissecans: Wilson's sign revisited. *Am J Sports Med*. 2003;31(5):777–8.
75. Zaremski JL, Herman DC, Vincent KR. Clinical utility of Wilson test for Osteochondral lesions at the knee. *Curr Sports Med Rep*. 2015;14(6):430.
76. Masquijo J, Kothari A. Juvenile osteochondritis dissecans (JOCD) of the knee: current concepts review. *EFORT Open Rev*. 2019;4(5):201–12.
77. Uppstrom TJ, Gausden EB, Green DW. Classification and assessment of juvenile osteochondritis dissecans knee lesions. *Curr Opin Pediatr*. 2016;28(1):60–7.
78. Berndt AL, Harty M. Transchondral fractures (osteochondritis dissecans) of the talus. *J Bone Joint Surg Am*. 1959;41-A:988–1020.
79. De Smet AA, Fisher DR, Graf BK, Lange RH. Osteochondritis dissecans of the knee: value of MR imaging in determining lesion stability and the presence of articular cartilage defects. *AJR Am J Roentgenol*. 1990;155(3):549–53.
80. Yoshida S, Ikata T, Takai H, et al. Osteochondritis dissecans of the femoral condyle in the growth stage. *Clin Orthop Relat Res*. 1998;346:162–70.
81. Kijowski R, Blankenbaker DG, Shinki K, et al. Juvenile versus adult osteochondritis dissecans of the knee: appropriate MR imaging criteria for instability. *Radiology*. 2008;248(2):571–8.
82. Heywood CS, Benke MT, Brindle K, Fine KM. Correlation of magnetic resonance imaging to arthroscopic findings of stability in juvenile osteochondritis dissecans. *Arthroscopy*. 2011;27(2):194–9.
83. Samora WP, Chevillet J, Adler B, Young GS, Klingele KE. Juvenile osteochondritis dissecans of the knee: predictors of lesion stability. *J Pediatr Orthop*. 2012;32(1):1–4.
84. Rossbach BP, Paulus AC, Niethammer TR, et al. Discrepancy between morphological findings in juvenile osteochondritis dissecans (OCD): a comparison of magnetic resonance imaging (MRI) and arthroscopy. *Knee Surg Sports Traumatol Arthrosc*. 2016;24(4):1259–64.
85. Krause M, Hapfelmeier A, Moller M, Amling M, Bohndorf K, Meenen NM. Healing predictors of stable juvenile osteochondritis dissecans knee lesions after 6 and 12 months of nonoperative treatment. *Am J Sports Med*. 2013;41(10):2384–91.
86. Pill SG, Ganley TJ, Milam RA, et al. Role of magnetic resonance imaging and clinical criteria in predicting successful nonoperative treatment of osteochondritis dissecans in children. *J Pediatr Orthop*. 2003;23(1):102–8.
87. Cahill BR, Phillips MR, Navarro R. The results of conservative management of juvenile osteochondritis dissecans using joint scintigraphy. A prospective study. *Am J Sports Med*. 1989;17(5):601–5.
88. Wall EJ, Heyworth BE, Shea KG, et al. Trochlear groove osteochondritis dissecans of the knee patellofemoral joint. *J Pediatr Orthop*. 2014;34(6):625–30.
89. Guhl JF. Arthroscopic treatment of osteochondritis dissecans. *Clin Orthop Relat Res*. 1982;167:65–74.
90. Carey JL, Wall EJ, Grimm NL, et al. Novel arthroscopic classification of Osteochondritis Dissecans of the knee: a multicenter reliability study. *Am J Sports Med*. 2016;44(7):1694–8.
91. Matcuk GR Jr, Mahanty SR, Skalski MR, et al. Stress fractures: pathophysiology, clinical presentation, imaging features, and treatment options. *Emerg Radiol*. 2016;23(4):365–75.

92. Changstrom BG, Brou L, Khodae M, Braund C, Comstock RD. Epidemiology of stress fracture injuries among US high school athletes, 2005-2006 through 2012-2013. *Am J Sports Med.* 2015;43(1):26-33.
93. Field AE, Gordon CM, Pierce LM, Ramappa A, Kocher MS. Prospective study of physical activity and risk of developing a stress fracture among preadolescent and adolescent girls. *Arch Pediatr Adolesc Med.* 2011;165(8):723-8.
94. Bennell K, Matheson G, Meeuwisse W, Brukner P. Risk factors for stress fractures. *Sports Med.* 1999;28(2):91-122.
95. Iwamoto J, Takeda T. Stress fractures in athletes: review of 196 cases. *J Orthop Sci.* 2003;8(3):273-8.
96. Kiel J, Kaiser K. Stress reaction and fractures. Treasure Island (FL): StatPearls; 2019.
97. Denay KL. Stress fractures. *Curr Sports Med Rep.* 2017;16(1):7-8.
98. Fredericson M, Jennings F, Beaulieu C, Matheson GO. Stress fractures in athletes. *Top Magn Reson Imaging.* 2006;17(5):309-25.
99. Arendt EA, Griffiths HJ. The use of MR imaging in the assessment and clinical management of stress reactions of bone in high-performance athletes. *Clin Sports Med.* 1997;16(2):291-306.
100. McInnis KC, Ramey LN. High-risk stress fractures: diagnosis and management. *PM R.* 2016;8(3 Suppl):S113-24.
101. Diehl JJ, Best TM, Kaeding CC. Classification and return-to-play considerations for stress fractures. *Clin Sports Med.* 2006;25(1):17-28.
102. Kahanov L, Eberman LE, Games KE, Wasik M. Diagnosis, treatment, and rehabilitation of stress fractures in the lower extremity in runners. *Open Access J Sports Med.* 2015;6:87-95.
103. Pasanen K, Ekola T, Vasankari T, et al. High ankle injury rate in adolescent basketball: a 3-year prospective follow-up study. *Scand J Med Sci Sports.* 2017;27(6):643-9.
104. Nelson AJ, Collins CL, Yard EE, Fields SK, Comstock RD. Ankle injuries among United States high school sports athletes, 2005-2006. *J Athl Train.* 2007;42(3):381-7.
105. Panagiotakis E, Mok KM, Fong DT, Bull AMJ. Biomechanical analysis of ankle ligamentous sprain injury cases from televised basketball games: understanding when, how and why ligament failure occurs. *J Sci Med Sport.* 2017;20(12):1057-61.
106. Kobayashi T, Tanaka M, Shida M. Intrinsic risk factors of lateral ankle sprain: a systematic review and meta-analysis. *Sports Health.* 2016;8(2):190-3.
107. Halstead ME. Pediatric ankle sprains and their imitators. *Pediatr Ann.* 2014;43(12):e291-6.
108. Plint AC, Bulloch B, Osmond MH, et al. Validation of the Ottawa ankle rules in children with ankle injuries. *Acad Emerg Med.* 1999;6(10):1005-9.
109. van den Bekerom MP, Kerkhoffs GM, McCollum GA, Calder JD, van Dijk CN. Management of acute lateral ankle ligament injury in the athlete. *Knee Surg Sports Traumatol Arthrosc.* 2013;21(6):1390-5.
110. Gruskay JA, Brusalis CM, Heath MR, Fabricant PD. Pediatric and adolescent ankle instability: diagnosis and treatment options. *Curr Opin Pediatr.* 2019;31(1):69-78.
111. Takahashi S, Okuwaki T. Epidemiological survey of anterior cruciate ligament injury in Japanese junior high school and high school athletes: cross-sectional study. *Res Sports Med.* 2017;25(3):266-76.
112. Joseph AM, Collins CL, Henke NM, et al. A multisport epidemiologic comparison of anterior cruciate ligament injuries in high school athletics. *J Athl Train.* 2013;48(6):810-7.
113. Krosshaug T, Nakamae A, Boden BP, et al. Mechanisms of anterior cruciate ligament injury in basketball: video analysis of 39 cases. *Am J Sports Med.* 2007;35(3):359-67.
114. Dordevic M, Hirschmann MT. Injury mechanisms of ACL tear. In: Siebold R, Dejour D, Zaffagnini S, editors. *Anterior cruciate ligament reconstruction.* Berlin, Heidelberg: Springer; 2014. p. 44-53.
115. Gornitzky AL, Lott A, Yellin JL, et al. Sport-specific yearly risk and incidence of anterior cruciate ligament tears in high school athletes: a systematic review and meta-analysis. *Am J Sports Med.* 2016;44(10):2716-23.
116. Price MJ, Tuca M, Cordasco FA, Green DW. Nonmodifiable risk factors for anterior cruciate ligament injury. *Curr Opin Pediatr.* 2017;29(1):55-64.
117. Wagemakers HP, Luijsterburg PA, Boks SS, et al. Diagnostic accuracy of history taking and physical examination for assessing anterior cruciate ligament lesions of the knee in primary care. *Arch Phys Med Rehabil.* 2010;91(9):1452-9.
118. Noyes FR, Bassett RW, Grood ES, Butler DL. Arthroscopy in acute traumatic hemarthrosis of the knee. Incidence of anterior cruciate tears and other injuries. *J Bone Joint Surg Am.* 1980;62(5):687-695, 757.
119. Remer EM, Fitzgerald SW, Friedman H, et al. Anterior cruciate ligament injury: MR imaging diagnosis and patterns of injury. *Radiographics.* 1992;12(5):901-15.
120. Lee K, Siegel MJ, Lau DM, Hildebolt CF, Matava MJ. Anterior cruciate ligament tears: MR imaging-based diagnosis in a pediatric population. *Radiology.* 1999;213(3):697-704.
121. Fabricant PD, Kocher MS. Management of ACL injuries in children and adolescents. *J Bone Joint Surg Am.* 2017;99(7):600-12.
122. Meyers MH, McKeever FM. Fracture of the intercondylar eminence of the tibia. *J Bone Joint Surg Am.* 1970;52(8):1677-84.
123. Adams AJ, Talathi NS, Gandhi JS, Patel NM, Ganley TJ. Tibial spine fractures in children: evaluation, management, and future directions. *J Knee Surg.* 2018;31(5):374-81.

124. McCrory P, Feddermann-Demont N, Dvorak J, et al. What is the definition of sports-related concussion: a systematic review. *Br J Sports Med.* 2017;51(11):877–87.
125. Zuckerman SL, Kerr ZY, Yengo-Kahn A, et al. Epidemiology of sports-related concussion in NCAA athletes from 2009–2010 to 2013–2014: incidence, Recurrence, and mechanisms. *Am J Sports Med.* 2015;43(11):2654–62.
126. Zuckerman SL, Totten DJ, Rubel KE, et al. Mechanisms of injury as a diagnostic predictor of sport-related concussion severity in football, basketball, and soccer: results from a regional concussion registry. *Neurosurgery.* 2016;63(Suppl 1):102–12.
127. Pfister T, Pfister K, Hagel B, Ghali WA, Ronskley PE. The incidence of concussion in youth sports: a systematic review and meta-analysis. *Br J Sports Med.* 2016;50(5):292–7.
128. Harmon KG, Clugston JR, Dec K, et al. American medical Society for Sports Medicine position statement on concussion in sport. *Br J Sports Med.* 2019;53(4):213–25.
129. Davis GA, Purcell L, Schneider KJ, et al. The child sport concussion assessment tool 5th edition (child SCAT5): background and rationale. *Br J Sports Med.* 2017;51(11):859–61.
130. Echemendia RJ, Meeuwisse W, McCrory P, et al. The sport concussion assessment tool 5th edition (SCAT5): background and rationale. *Br J Sports Med.* 2017;51(11):848–50.
131. Zuckerman SL, Brett BL, Jeckell AS, Yengo-Kahn AM, Solomon GS. Prognostic factors in pediatric sport-related concussion. *Curr Neurol Neurosci Rep.* 2018;18(12):104.
132. Morgan CD, Zuckerman SL, Lee YM, et al. Predictors of postconcussion syndrome after sports-related concussion in young athletes: a matched case-control study. *J Neurosurg Pediatr.* 2015;15(6):589–98.
133. Majerske CW, Mihalik JP, Ren D, et al. Concussion in sports: postconcussive activity levels, symptoms, and neurocognitive performance. *J Athl Train.* 2008;43(3):265–74.
134. Wiebe DJ, Nance ML, Houseknecht E, et al. Ecologic momentary assessment to accomplish real-time capture of symptom progression and the physical and cognitive activities of patients daily following concussion. *JAMA Pediatr.* 2016;170(11):1108–10.
135. Leddy JJ, Kozlowski K, Donnelly JP, Pendergast DR, Epstein LH, Willer B. A preliminary study of subsymptom threshold exercise training for refractory post-concussion syndrome. *Clin J Sport Med.* 2010;20(1):21–7.
136. Thomas DG, Apps JN, Hoffmann RG, McCrea M, Hammeke T. Benefits of strict rest after acute concussion: a randomized controlled trial. *Pediatrics.* 2015;135(2):213–23.
137. Kuemmerle K, Meehan WP 3rd. Treatment of pediatric concussion. *Semin Pediatr Neurol.* 2019;30:60–7.
138. McCrory P, Meeuwisse W, Dvorak J, et al. Consensus statement on concussion in sport—the 5(th) international conference on concussion in sport held in Berlin, October 2016. *Br J Sports Med.* 2017;51(11):838–47.
139. Santiago S. Adolescent concussion and return-to-learn. *Pediatr Ann.* 2016;45(3):e73–5.
140. Grady MF, Master CL. Return to school and learning after concussion: tips for pediatricians. *Pediatr Ann.* 2017;46(3):e93–8.
141. Halstead ME, McAvoy K, Devore CD, et al. Returning to learning following a concussion. *Pediatrics.* 2013;132(5):948–57.
142. McCrory P, Meeuwisse W, Aubry M, et al. Consensus statement on concussion in sport—the 4th international conference on concussion in sport held in Zurich, November 2012. *Clin J Sport Med.* 2013;23(2):89–117.
143. McLeod TC, Lewis JH, Whelihan K, Bacon CE. Rest and return to activity after sport-related concussion: a systematic review of the literature. *J Athl Train.* 2017;52(3):262–87.
144. Blom LC, Visek AJ, Harris BS. Triangulation in youth sport: healthy partnerships among parents, coaches, and practitioners. *J Sport Psychol Action.* 2013;4(2):86–96.
145. Chan DK, Lonsdale C, Fung HH. Influences of coaches, parents, and peers on the motivational patterns of child and adolescent athletes. *Scand J Med Sci Sports.* 2012;22(4):558–68.
146. Gardner LA, Magee CA, Vella SA. Social climate profiles in adolescent sports: associations with enjoyment and intention to continue. *J Adolesc.* 2016;52:112–23.
147. Madigan DJ, Curran T, Stoeber J, Hill AP, Smith MM, Passfield L. Development of perfectionism in junior athletes: a three-sample study of coach and parental pressure. *J Sport Exerc Psychol.* 2019;41(3):167–75.
148. Padaki AS, Popkin CA, Hodgins JL, Kovacevic D, Lynch TS, Ahmad CS. Factors that drive youth specialization. *Sports Health.* 2017;9(6):532–6.
149. Lamb M, Kuhn AW, LaBotz M, Diamond AB. Safeguarding the child and adolescent athlete. *Curr Sports Med Rep.* 2018;17(12):419–24.



Management of the Sickle Cell Spectrum in Basketball Players

68

Kimberly G. Harmon

68.1 Epidemiology

It is estimated by the CDC that 1 in 13 (8%) of black or African-American individuals in the United States has SCT and 1 out of every 365 (0.2%) Black or African-American persons has SCD (CDC website). Between 0.01% and 0.07% of the remaining population also has SCT, primarily those of Arab, Southeast Asian, Hispanic, or Mediterranean descent [1]. SCT affects an estimated 3,000,000 persons in the United States and SCD about 100,000 [2]. SCD is the most common inherited disease in the world and has persisted because of a protective effect of SCT against malaria. The gene for sickle hemoglobin mutation is distributed throughout the world with higher prevalence in those areas where malaria is prevalent: sub-Saharan Africa, Mediterranean countries (especially Greece), the Middle East, and parts of India. While SCT is considered relatively benign, SCD has many variants with different clinical and health implications. We discuss the most common variants of SCD: sickle cell anemia, sickle β -thalassemia, and sickle SC disease.

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Fact Box

- 1 in 13 (8%) of black/African-American individuals has SCT.
- An estimated 3,000,000 people in the United States have SCT.
- Athletes of other ethnicities/races can also be affected by SCT.

68.2 Pathogenesis

Normal adult hemoglobin (Hgb) is composed of two α -chains and two β -chains which come together to form the tetramer HgbA. HgbA is highly soluble in both the oxygenated and deoxygenated state. HgbS results from the substitution of a valine for glutamic acid at the sixth amino acid of the β -globin chain. HgbS forms a tetramer that is poorly soluble when deoxygenated, polymerizes into chain, and can cause the red blood cell to form a sickle shape. In addition to red blood cell sickling, endothelial adhesion, intravascular hemolysis, increased oxidative stress, and inflammation contribute to the pathophysiology of SCD [3]. Vaso-occlusive phenomena and hemolytic anemia are clinical manifestations of SCD which can lead to recurrent painful episodes and end-organ complications. Although the clinical manifestations may be typically absent or not as severe, many of these same mechanisms account for adverse outcomes in those with SCT.

Table 68.1 Hemoglobin in normal, sickle cell trait, and sickle cell disease

	α -Globin chain	α -Globin chain	β -Globin chain	β -Globin chain	Genotype	% HgbA	% HgbS	% HgbC
Normal	Normal	Normal	HgbA	HgbA	AA	95–98	0	0
Sickle cell trait	Normal	Normal	HgbA	HgbS	AS	50–60	35–40	0
Sickle cell disease	Normal	Normal						
Sickle cell anemia	Normal	Normal	HgbS	HgbS	SS	0	85–95	0
Sickle β^0 -thalassemia	Normal	Normal	HgbS	Hgb β	S β^0	0	80–92	0
Sickle β^+ -thalassemia	Normal	Normal	HgbS	Hgb β	S β^+	3–30	65–90	0
Hemoglobin SC	Normal	Normal	HgbS	HgbC	SC	0	45–50	45–50

SCT occurs when a gene for HgbS is inherited from one parent for one β -globin allele and a HgbA β -globin allele from the other parent. This results in the production of both HgbA and HgbS in the red blood cell. Because there is a significant amount of soluble HgbA within the cell in SCT, the cell is less likely to sickle and lead to the sequelae associated with SCD.

SCD refers to any one of the syndromes where HgbS is co-inherited with a mutation at the other β -globin allele that reduces or abolishes normal HgbA production. These conditions include sickle cell anemia (Hgb SS), sickle β -thalassemia, hemoglobin SC disease, and others (Table 68.1). There is significant clinical variation among the different SCD conditions. SCD is often thought of as incompatible with exercise; however, there is variation in the clinical manifestations between the SCD conditions and in individuals within genotypes.

68.3 Screening for Sickle Cell Trait and Disease

In the late 1970s and 1980s statewide programs screening for sickle cell disease were implemented in New York, Colorado, Texas, and Georgia which demonstrated decreased morbidity and mortality in those identified with disease [4]. This led to a push for nationwide screening and there has been mandated testing of all newborns in the United States, Puerto Rico, and the Virgin Islands for SCT and SCD at birth since 2006. Early identification of SCD leads to improved outcomes in children primarily by

reducing the risk of life-threatening infections with *Streptococcus pneumoniae* and *Haemophilus influenzae* by the use of routine prophylaxis with penicillin and pneumococcal vaccines [4]. While the benefit of early recognition of SCD is clear, the value of SCT status knowledge is primarily seen in terms of preconception counseling. However, there are highly variable reporting practices around the country and many parents of children with sickle cell trait are unaware of their newborn screening results [5].

Testing for SCT and disease in the newborn setting is done as part of a blood spot from a heel stick which is used to screen for several conditions. If the screening test is positive as a newborn, confirmatory testing is done at around 6 weeks of age. The other setting in which screening for sickle cell status is considered is when entering certain branches of the military or before participating in college athletics at a National Collegiate Athletics Association (NCAA) school. Screening practices in the military vary by branch. Currently the Navy and Air Force screen recruits with differing restrictions and limitation on service in those who are SCT positive. The Army, as of 2019, does not screen but this is currently under evaluation.

68.4 Athletes with Sickle Cell Trait

68.4.1 Medical Issues in SCT

SCT is usually considered a benign condition and often time athletes are unaware of their status. There is no decrease in life expectancy associated

with SCT and epidemiological and laboratory studies have demonstrated that SCT status does not appear to limit participation in athletics, even at the highest levels of performance [6, 7]. There may be some individuals, however, that are more affected than others. Sudden exertional death has been the primary concern in athletes with SCT; however, it is also associated with hyposthenuria (an inability to concentrate urine), hematuria, chronic kidney disease, splenic sequestration and infarction, complications from hyphema, and an increased risk of venous thromboembolism and pulmonary embolism. We will review these concerns in detail.

Fact Box

Medical issues associated with SCT include:

- Inability to concentrate urine
- Hematuria
- Splenic sequestration and infarction (especially at altitude)
- Increased risk of pulmonary embolism
- Increased risk of exertional rhabdomyolysis
- Increased risk of exertional sudden death

68.4.2 Exertional Collapse and Death

SCT in the strenuously exercising individual first became a concern in the 1970s when a disproportionate number of deaths in military recruits training at moderate altitude were noted in individuals with SCT [8]. A study in 1987 expanded on these findings reported that the risk for sudden death in black/African-American recruits with SCT was 28 times that of recruits without SCT [9]. Other studies confirmed an increased risk of sudden death in military recruits with SCT compared to those without the trait [10, 11]. It was hypothesized that these deaths occurred in settings where individuals pushed themselves past their physiologic capacity, a circumstance which also occurs in athletics.

In 1974, after the death of a college football athlete who lived in Florida but went to school in Denver, SCT as a potential risk for exertional death became a concern in athletes. The NCAA addressed this concern with a guideline for the first time in 1975. In that guideline, they noted that SCT was not a barrier to achieving the highest levels of athletic performance and that most athletes with SCT would not experience complications [6]. Over the last 56 years, however, intermittent deaths in NCAA athletes have continued with a total of 23 deaths related to SCT in NCAA athletes [12]. All but two of those deaths have occurred in Division I football athletes. The football deaths were all associated with conditioning, either in practice or in dedicated conditioning sessions. No documented deaths have occurred during a football game, presumably because there is a built-in rest/recovery period between football plays.

The NCAA SCT guideline was updated in 1982, 1992, 2002, 2008, and 2009. In 2007 the National Athletic Trainer's Association published a consensus statement in order to raise awareness of SCT and provide measures to reduce the risk of exertional collapse associated with SCT (ECAST) [13]. They advised confirming an athletes' SCT status at the time of the preparticipation examination and further recommended that if SCT status was unknown each institution carefully weigh the decision to screen or not to screen for SCT [13]. The NATA guideline also recommended steps to mitigate risk in athletes with SCT that still serve as the basis for current recommendations and included a slow buildup of conditioning activities, preseason preparation, exclusion of athletes with SCT from performance tests, and cessation of activity with the onset of symptoms, allowing the athletes to set their own pace, modifications for environmental stressors, and education of athletes and coaches. At the time, the NCAA allowed institutions to set their own policies regarding screening athletes for sickle cell trait and focused on education. Although screening for SCT was not required by the NCAA, a 2006 survey indicated that 64% of responding Division IA institutions (the largest institutions, with the most sports and scholarship aid) screened football athletes for SCT [14].

In 2010, the NCAA adopted a policy requiring Division I institutions to perform testing for SCT on all incoming student-athletes. This requirement was part of a settlement of a lawsuit alleging wrongful death of a college football player in 2006 during a conditioning session at a school that did not screen for SCT and whose parents (and presumably the athlete) were unaware of his SCT status. The legislation further provided that if an athlete refuses SCT screening they must sign a waiver documenting their decision. A 2012 study examined the rate of exertional death in Division I football players over a 5-year period and found that football athletes with SCT had a 37 times higher risk than those without SCT [15]. Division II began requiring screening for SCT in August of 2012 and Division III followed suit in August of 2014.

The American Society for Hematology, the Sickle Cell Disease Association of American, and others decried the NCAA policy citing concerns of loss of privacy, stigmatization, and discrimination as well as a lack of evidence that screening prevents exertional death [16]. The American Society of Hematology also pointed to evidence, from a non-peer-reviewed report, of a decrease in deaths associated with sickle cell trait after “universal precautions” were implemented in the

Army. Despite the opposition, the SCT screening policy has remained in effect and there is initial evidence that the rate of death due to SCT has significantly decreased since legislation [14]. Whether the decrease in death rate is due to screening, education, or both is unknown.

Most recently, in 2019, the NCAA produced the Interassociation Recommendation Preventing Catastrophic Injury and Death in Collegiate Athletes which requires annual education for many health conditions which can lead to death including ECAST [17]. Educational material for providers, athletes and coaches is provided by the NCAA and available on a website which is periodically updated (<http://www.ncaa.org/sport-science-institute/sickle-cell-trait>; Fig. 68.1).

Death in those with SCT is less well documented in sports outside of football. There are two non-football deaths in the NCAA: one in a track athlete who was trying out for the team and one in a Division II basketball player who was playing pickup. One may think that basketball would be particularly affected by issues related to SCT as 78% of Division I of male basketball athletes are black/African-American compared to 48% of Division I football athletes [19]; however, because there are many more football players overall, the absolute number of SCT-affected ath-

The NCAA recommends the following principles:

- 1) Athletes should slowly build up their training intensity
- 2) Fitness tests should be scheduled later in the training program once athletes are acclimated
- 3) Athletes should be allowed to set their own pace when conditioning
- 4) Implement a slow and gradual preseason conditioning regimen that prepares them for the rigors of their sport
- 5) Athletes should be provided adequate rest and recovery between repetitions
- 6) Athletes with SCT should be given alternatives for performance testing rather than serial sprints or timed mile runs
- 7) An athlete should stop activity immediately upon struggling or experiencing symptoms such as muscle pain, abnormal weakness, undue fatigue or breathlessness
- 8) Athletes with SCT should stay well hydrated at all times, especially in hot and humid conditions
- 9) Athletes should refrain from consuming high caffeine energy drinks and supplements, or other stimulants as they may contribute to dehydration.
- 10) Athletes should maintain proper asthma management
- 11) Athletes with SCT should refrain from extreme exercise during acute illness, if feeling ill, or while experiencing fever.
- 12) Beware when adjusting to a change in altitude (a rise in altitude as little as 2,000 feet). Modify training and have supplemental oxygen available.
- 13) Seek prompt medical care when experiencing unusual physical distress.

Fig. 68.1 NCAA fact sheet for coaches [18]

letes is higher in football: an estimated 1125 Division I football players with SCT per year compared to an estimated 250 Division I male basketball athletes. Despite the differential in participation, however, it is still unclear why football athletes seem to be primarily affected. Some have suggested that the increased rate of death in football players may be due to equipment; however, the vast majority of deaths have occurred during conditioning when football equipment is not worn.

Basketball deaths associated with SCT are sparsely reported in the literature. A registry of athlete sudden deaths from 1980 to 2010 was reviewed in 2012; there were 23 deaths attributed to SCT with 3 of those being in basketball [20]. Interestingly, 2 of the 3 deaths were in female basketball athletes (one college, one high school). Likewise, deaths in high school athletes with SCT are not well recorded. There is no mandatory reporting of deaths or common mandatory registry to track deaths. This is further complicated by variability in autopsy quality and reported cause of death. There is speculation that some deaths associated with sickle cell trait may be miscategorized as heat stroke or sudden cardiac death.

Although many consider SCT to be a risk factor for ECAST and sudden death, a recent study of 47,944 black/African-American active duty soldiers from 2010 to 2014 compared death in those with and without SCT and did not find an increased risk of death in soldiers with SCT [1]. There are several possible explanations for this. First, most military studies suggesting an increased risk of exertional death in those with SCT examine at basic training, not active duty military personnel, where recruits may either be insufficiently conditioned for the required training or not feel able to stop when pushed past their physiologic limit. Second, this study looked at all-cause death and there were no deaths in the cohort-attributed ECAST; thus the study was likely not powered to detect a difference in the rate of ECAST death. A 2018 meta-analysis which included two studies in the military also concluded that there was no evidence of increased risk of sudden death in those with SCT [7]. Again,

this may be more related to the rarity of SCT-associated death than an absence of risk. More studies in larger cohorts need to be done in order to answer this question definitively.

The pathogenesis of ECAST is unknown but has been postulated to occur during high-intensity exercise when sickling occurs in the microvasculature of the exercising muscle due to dehydration, thermal strain, hyperosmolality, acidosis, and hypoxia [21–23]. The proposed mechanism of death is arrhythmia caused by hyperkalemia from muscle cell necrosis and altered ion channel permeability. Others, however, debate the proposed pathophysiology because it is dissimilar to issues seen in those with SCD which include splenic, pulmonary, and bony infarcts.

The clinical presentation of ECAST is distinct from collapse caused by cardiac arrest or heat stroke. In ECAST there is typically intense pain of muscles, usually the legs but sometime the back, that is often described as cramping. Although the pain is described as cramping, the muscles are not tense and may or may not be tender. Those with ECAST are conscious when they collapse, but, if the condition progresses, will lose consciousness after collapsing. The collapse is typically after bouts of intense sustained exercise without adequate rest. In contrast, collapse associated with sudden cardiac arrest is an unconscious, abrupt collapse secondary to ventricular fibrillation. There may be tonic-clonic seizure-like movement or agonal breathing after collapse from sudden cardiac arrest. Finally, collapse from heat stroke is associated with elevated core temperature and mental status changes. Although an elevated core temperature may occur with ECAST, muscle pain is the primary feature and mental status is intact.

Any athlete that is struggling with conditioning drills beyond their capacity should be removed from the activity, but this is critically important in those with SCT. Treatment of an athlete with SCT involves discontinuing activity immediately if there are symptoms of exertional sickling, typically pain or weakness in the legs or sometimes the back. If an athlete discontinues the offending activity, the symptoms most often go away quite quickly and they can return to activity

immediately. If there is suspicion for heat illness, obtain the athlete's temperature with a rectal thermometer and cool if temperature is $>104^{\circ}\text{F}$. If symptoms of muscle pain and weakness do not subside quickly, it is recommended to start high-flow oxygen if available. If there is not a rapid return to baseline then the emergency action system should be activated. When transporting the athlete it is important to communicate with the emergency room physicians. Many physicians are not familiar with ECAST and consider SCT benign. Tell them to be prepared for explosive rhabdomyolysis and metabolic abnormalities. A member of the medical team should accompany the athlete in order to facilitate rapid and effective treatment. Death and collapse in athletes with SCT are preventable. ECAST occurs only when athletes are pushed beyond their physiologic capacity.

Return to play after an ECAST event which is survived is based on expert consensus (Fig. 68.2). A detailed medical history should be done to identify comorbidities (asthma, illness), medication or supplement use, environmental factors (altitude, heat, level of conditioning), and family history. The athlete should be asymptomatic and have normal end-organ function before return to play and a gradual, medically supervised return to activity is recommended. Education of the athlete, coaching staff, and medical staff is critical, and should stress the need to stop immediately when symptomatic, stay hydrated and mitigate risk factors when possible. Most athletes with SCT will never have an ECAST event and there are likely cofactors other than SCT associated with risk; however, once an athlete has had an ECAST event they are more likely to have another one and should be monitored closely with a low threshold for removal from activity and treatment [24].

68.4.3 Exertional Rhabdomyolysis

Rhabdomyolysis is the breakdown of striated muscle fibers due to mechanical and metabolic insults that result in the release of muscle contents into the circulation. Exertional rhabdomyo-

lysis typically occurs in response to prolonged, repetitive, or excessive exercise. Athletes with SCT are at increased risk of rhabdomyolysis. It is hypothesized that red blood cell sickling in the microvasculature of the muscle causes ischemia and increased muscle breakdown compared to those without SCT. In the 2016 study of 47,944 soldiers, those with SCT had a 1.54 relative risk of rhabdomyolysis compared to those that did not [1]. Being male and older as well as smoking and statin use also showed increased risk [1]. In a meta-analysis which included two military studies it was also concluded that SCT was a risk factor for rhabdomyolysis [7]. Exertional rhabdomyolysis in those with SCT is likely a precursor to ECAST that is aborted prior to collapse or does not progress enough to cause massive potassium release from cells and arrhythmic death.

Return to play after exertional rhabdomyolysis in athletes with SCT should follow a similar algorithm as those without SCT; they should be rest and rehydrate prior to return, their creatinine kinase should be approaching normal, and there should be no myoglobin in their urine [24]. In addition, education regarding exertional rhabdomyolysis and ECAST should be reviewed and any mitigating risk factors addressed. Return to play should involve a gradual reintroduction of aerobic and sport-specific activity.

68.4.4 Urologic Manifestations of SCT

Hematuria and hyposthenuria are the most common urologic manifestations of SCT. The physiologic environment of the kidney includes low oxygen tension, hyperosmolarity, and low pH which promote polymerization of HgbS and sickling of the red blood cells. It is hypothesized that repetitive episodes of sickling lead to ischemic injury and microinfarction which result in loss of the vascular architecture of the renal medulla where concentrating ability is affected and leads to hyposthenuria and hematuria [25]. In athletes, this may be amplified due to strenuous workouts which increase acidosis and decrease renal blood

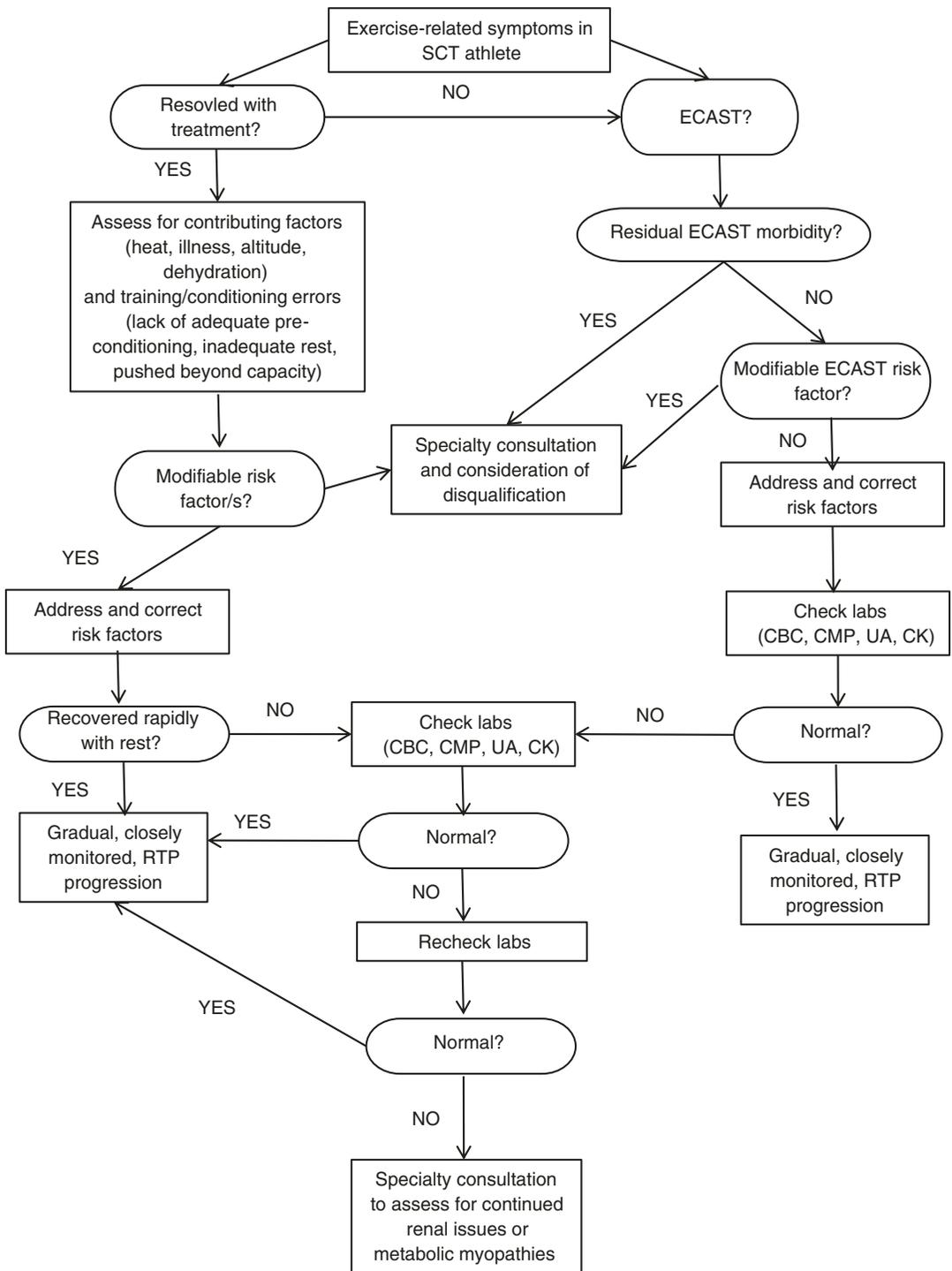


Fig. 68.2 Return to play after exercise related symptoms associated with SCT

flow. It is also speculated that an inability to concentrate urine may enhance dehydration which in

turn promotes sickling creating a viscous cycle. Hyposthenuria can be seen as early as 10 years of

age in those with SCT. Athletes with SCT need to drink more fluid than is required in non-SCT athletes in order to stay hydrated. Ongoing hemolysis, vaso-occlusive injury, and other factors contribute to continuing renal injury. Repeated insults to the kidney can lead to chronic kidney disease in later life. Those with SCT have a 1.5–2-fold increased risk of chronic kidney disease compared to those without SCD [25].

68.4.5 Splenic Issues

Splenic syndrome is a rare, but well-documented complication of SCT that presents in the presence of low oxygen tension associated with major changes in altitude, such as an unpressurized air flight or when mountain climbing or skiing [26]. Splenic syndrome presents with left upper quadrant pain and may be accompanied by vomiting, fever, a palpable spleen, or low oxygen tension [27]. Symptoms generally occur rapidly, within the first 24 h of ascent and are more common in individuals who normally reside at sea level. Ultrasound or CT showing splenomegaly in an individual with SCT and left upper quadrant pain is generally enough to confirm the diagnosis. The pathophysiology of splenic syndrome is thought to be vaso-occlusion of the red pulp of the spleen by sickled red blood cells, leading to infarction. Splenic syndrome often resolves with transfer to a lower altitude and supportive treatment, however, may require splenectomy [28].

Spleen issues as a consequence of SCT in athletes made the news in 2009 when Pittsburgh Steeler safety Ryan Clark developed symptoms in 2005 while playing in Denver (5260 ft. above sea level) and again when playing at Denver in 2007; which eventually led to complications and splenectomy. He skipped the game played in Denver in 2009 [29]. Likewise, splenic syndrome was diagnosed in a college basketball athlete from Alabama playing in Wyoming who was treated conservatively before flying home with supplemental oxygen. Although splenic syndrome appears to be rare, athletes at altitude should take extra care to remain hydrated and if

left upper quadrant pain develops, splenic syndrome should be suspected.

68.4.6 Hyphema

Hyphemia, or accumulation of blood in the anterior chamber of the eye, typically after trauma, is a higher risk injury in athletes with SCT compared to those without. The low-oxygen environment in the aqueous humor of the eye promotes sickling and sickled red blood cells are hypothesized to clog the trabecular meshwork which leads to increased intraocular pressure [30]. SCT athletes with a hyphema are at increased risk for complications including increased intraocular pressure, optic atrophy, and secondary hemorrhage [30]. Any hyphema should be referred for ophthalmologic consultation and followed closely.

68.4.7 Venous Thromboembolism

Venous thromboembolism includes both deep venous thrombosis (DVT) and pulmonary embolus (PE). It is hypothesized that the altered structure of sickled red cells leads to hemolysis and release of procoagulant substances which cause activation of platelets and coagulation cascade resulting in hypercoagulability, endothelial dysfunction, and blood stasis (Virchow's triad) and ultimately an increased risk of venous thromboembolism [31]. A recent meta-analysis, however, showed that there is likely increased risk of PE in those with SCT but not DVT [7]. Nonetheless, it is not unwise to remind athletes who fly to remain hydrated and move their calves/legs on flights.

68.5 Sickle Cell Disease

The benefits of exercise are well described; however, there is concern that exercise in those with SCD will precipitate pain and other complications. Ability to exercise strenuously with sickle

cell disease will vary tremendously depending on which subtype is being considered. Those with only one HgbS allele and a variant allele (β -thalassemia or HgbC) may be able to tolerate higher levels of exercise. This is illustrated by the case of Billy Garrett, Jr., a basketball athlete who played college ball at DePaul and the NBA with SCD. His story details episodes of painful sickle cell crisis as well as his strenuous training [32]. Although the type of SCD is not specifically stated, it is likely that he had hemoglobin SC disease, which represents the less severe end of the spectrum, rather than sickle cell anemia (HgbSS).

For those with sickle cell anemia, the risks and benefits of exercise need to be carefully weighed and tested. Acute exercise results in an increase in inflammation, increased metabolic acidosis, increased oxidative stress, and increased endothelial activation. These effects are up-modulated in those with SCD [33]. However, in animal models of SCD, habitual physical activity blunts oxidative stress, and inflammation and endothelial activation are decreased. Thus, for most, mild-to-moderate exercise with appropriate attention to hydration and temperature is safe and may confer many of the benefits seen with exercise, including improved cardiovascular outcomes, improved mortality, and improved health-related quality of life [34].

68.6 Conclusion

SCT and SCD are common. Understanding the risks and benefits of exercise in these populations is critical. For athletes with SCT, especially basketball athletes with SCT, the risk of complications from the condition is small. Athletes should be aware of the potential for death if they push themselves past their physiologic limit, and should take extra care to acclimate, condition gradually, and remain hydrated. Coaches need to be aware of the potential issues associated with SCT and employ prudent training practices. Medical personnel should be familiar with potential issues associated with SCT and include it in the differential of any collapsed athlete. Exercise is beneficial for long-term health and this is true across the sickle cell spectrum.

References

1. Nelson DA, Deuster PA, Carter R 3rd, et al. Sickle cell trait, rhabdomyolysis, and mortality among U.S. Army soldiers. *N Engl J Med.* 2016;375(5):435–42. <https://doi.org/10.1056/NEJMoa1516257>. [Published Online First: 2016/08/16].
2. Data and statistics on sickle cell disease: center for disease control and prevention [updated October 21, 2019]. <https://www.cdc.gov/ncbddd/sicklecell/data.html>. Accessed 24 Oct 2019.
3. Martin C, Pialoux V, Faes C, et al. Does physical activity increase or decrease the risk of sickle cell disease complications? *Br J Sports Med.* 2018;52(4):214–18. <https://doi.org/10.1136/bjsports-2015-095317>. [Published Online First: 2015/12/25].
4. Yusuf HR, Lloyd-Puryear MA, Grant AM, et al. Sickle cell disease: the need for a public health agenda. *Am J Prev Med.* 2011;41(6 Suppl 4):S376–83. <https://doi.org/10.1016/j.amepre.2011.09.007>. [Published Online First: 2011/12/07].
5. Taylor C, Kavanagh P, Zuckerman B. Sickle cell trait--neglected opportunities in the era of genomic medicine. *JAMA.* 2014;311(15):1495–6. <https://doi.org/10.1001/jama.2014.2157>. [Published Online First: 2014/03/08].
6. O'Connor FG, Bergeron MF, Cantrell J, et al. ACSM and CHAMP summit on sickle cell trait: mitigating risks for warfighters and athletes. *Med Sci Sports Exerc.* 2012;44(11):2045–56. <https://doi.org/10.1249/MSS.0b013e31826851c2>. [Published Online First: 2012/07/20].
7. Naik RP, Smith-Whitley K, Hassell KL, et al. Clinical outcomes associated with sickle cell trait: a systematic review. *Ann Intern Med.* 2018;169(9):619–27. <https://doi.org/10.7326/M18-1161>. [Published Online First: 2018/11/02].
8. Jones SR, Binder RA, Donowho EM Jr. Sudden death in sickle-cell trait. *N Engl J Med.* 1970;282(6):323–5. <https://doi.org/10.1056/NEJM197002052820607>. [Published Online First: 1970/02/05].
9. Kark JA, Posey DM, Schumacher HR, et al. Sickle-cell trait as a risk factor for sudden death in physical training. *N Engl J Med.* 1987;317(13):781–7. <https://doi.org/10.1056/NEJM198709243171301>. [Published Online First: 1987/09/24].
10. Scoville SL, Gardner JW, Magill AJ, et al. Nontraumatic deaths during U.S. Armed Forces basic training, 1977–2001. *Am J Prev Med.* 2004;26(3):205–12. <https://doi.org/10.1016/j.amepre.2003.12.003>. [Published Online First: 2004/03/18].
11. Eckart RE, Scoville SL, Campbell CL, et al. Sudden death in young adults: a 25-year review of autopsies in military recruits. *Ann Intern Med.* 2004;141(11):829–34. [Published Online First: 2004/12/08].
12. D. K. Student Athlete Deaths Related to Sickle Cell Trait. <http://www.ncaa.org/health-and-safety>. Accessed 2 Mar 2012.

13. Ellis MJ, Ritchie LJ, Koltek M, et al. Psychiatric outcomes after pediatric sports-related concussion. *J Neurosurg Pediatr.* 2015;16(6):709–18. <https://doi.org/10.3171/2015.5.PEDS15220>.
14. Harmon KG, Zigman ML, Siebert DM, et al. Sudden death associated with sickle cell trait before and after mandatory screening. *Clin J Sport Med.* 2017;27(2):192.
15. Harmon KG, Drezner JA, Klossner D, et al. Sickle cell trait associated with a RR of death of 37 times in National Collegiate Athletic Association football athletes: a database with 2 million athlete-years as the denominator. *Br J Sports Med.* 2012;46(5):325–30. <https://doi.org/10.1136/bjsports-2011-090896>.
16. Thompson AA. Sickle cell trait testing and athletic participation: a solution in search of a problem? *Hematology Am Soc Hematol Educ Program.* 2013;2013:632–7. <https://doi.org/10.1182/asheducation-2013.1.632>.
17. Interassociation Recommendations Presenting Catastrophic Injury and Death in Collegiate Athletes July 2019. https://ncaaorg.s3.amazonaws.com/ssi/injury_prev/SSI_PreventingCatastrophicInjuryBooklet.pdf, Accessed 8 Jan 2020.
18. A fact sheet for coaches. <http://www.ncaa.org/sport-science-institute/sickle-cell-trait>, Accessed 28 Oct 2019.
19. NCAA Sports Sponsorship and Participation Rates Database. <http://www.ncaa.org/about/resources/research/ncaa-sports-sponsorship-and-participation-rates-database>.
20. Harris KM, Haas TS, Eichner ER, et al. Sickle cell trait associated with sudden death in competitive athletes. *Am J Cardiol.* 2012;110(8):1185–8. <https://doi.org/10.1016/j.amjcard.2012.06.004>. [Published Online First: 2012/07/20].
21. Loosemore M, Walsh SB, Morris E, et al. Sudden exertional death in sickle cell trait. *Br J Sports Med.* 2011; <https://doi.org/10.1136/bjsports-2011-090521>. [Published Online First: 2011/10/04].
22. Eichner ER. Sickle cell trait in sports. *Curr Sports Med Rep.* 2010;9(6):347–51. <https://doi.org/10.1249/JSR.0b013e3181fc73d7>. [Published Online First: 2010/11/12].
23. Eichner ER. Sickle cell considerations in athletes. *Clin Sports Med.* 2011;30(3):537–49. <https://doi.org/10.1016/j.csm.2011.03.004>.
24. Asplund CA, O'Connor FG. Challenging return to play decisions: heat stroke, Exertional Rhabdomyolysis, and Exertional collapse associated with sickle cell trait. *Sports Health.* 2016;8(2):117–25. <https://doi.org/10.1177/1941738115617453>. [Published Online First: 2016/02/21].
25. Naik RP, Derebail VK. The spectrum of sickle hemoglobin-related nephropathy: from sickle cell disease to sickle trait. *Expert Rev Hematol.* 2017;10(12):1087–94. <https://doi.org/10.1080/17474086.2017.1395279>. [Published Online First: 2017/10/20].
26. Murano T, Fox AD, Anjaria D. Acute splenic syndrome in an African-American male with sickle cell trait on a commercial airplane flight. *J Emerg Med.* 2013;45(5):e161–5. <https://doi.org/10.1016/j.jemermed.2013.05.009>. [Published Online First: 2013/07/03].
27. Goodman J, Hassell K, Irwin D, et al. The splenic syndrome in individuals with sickle cell trait. *High Alt Med Biol.* 2014;15(4):468–71. <https://doi.org/10.1089/ham.2014.1034>. [Published Online First: 2014/11/02].
28. Nuss R, Feyerabend AJ, Lear JL, et al. Splenic function in persons with sickle cell trait at moderately high altitude. *Am J Hematol.* 1991;37(2):130–2. <https://doi.org/10.1002/ajh.2830370214>. [Published Online First: 1991/06/01].
29. Eichner ER. On spleens and genes. *Curr Sports Med Rep.* 2017;16(4):214–5. <https://doi.org/10.1249/JSR.0000000000000377>. [Published Online First: 2017/07/12].
30. Bansal S, Gunasekaran DV, Ang B, et al. Controversies in the pathophysiology and management of hyphema. *Surv Ophthalmol.* 2016;61(3):297–308. <https://doi.org/10.1016/j.survophthal.2015.11.005>. [Published Online First: 2015/12/04].
31. Noubiap JJ, Temgoua MN, Tankeu R, et al. Sickle cell disease, sickle trait and the risk for venous thromboembolism: a systematic review and meta-analysis. *Thromb J.* 2018;16:27. <https://doi.org/10.1186/s12959-018-0179-z>. [Published Online First: 2018/10/12].
32. Johnson M. Playing through sickle cell the undefeated. 2018.
33. Martin C, Pialoux V, Faes C, et al. Does physical activity increase or decrease the risk of sickle cell disease complications? *Br J Sports Med.* 2018;52(4):214–8. <https://doi.org/10.1136/bjsports-2015-095317>. [Published Online First: 2015/12/25].
34. Liem RI. Balancing exercise risk and benefits: lessons learned from sickle cell trait and sickle cell anemia. *Hematology Am Soc Hematol Educ Program.* 2018;2018(1):418–25. <https://doi.org/10.1182/asheducation-2018.1.418>. [Published Online First: 2018/12/07].



Playing Basketball with a Cardiac Condition: Recommendations and Guidelines

69

David S. Owens and Jonathan A. Drezner

69.1 Introduction

Sudden cardiac death (SCD) among athletes is always tragic event and often high-profile event, as exemplified by the shocking deaths of basketball players Hank Gathers and Reggie Lewis in the 1990s. While SCD among athletes is relatively infrequent, it is usually due to an undiagnosed genetic or congenital heart abnormality. Developed societies have generally agreed that screening athletes for these underlying heart disorders is an important public health measure but have differing recommendations with regard to the intensity and methods of screening. Currently, most international and professional sports require cardiovascular screening that includes electrocardiography in addition to history and physical examination.

Routine vigorous exercise training can result in exercise-induced cardiac adaptations in which the heart changes in size, thickness, and autonomic tone. In some cases, this process can resemble a pathologic disorder, leading to “gray

zones” of uncertainty between health and disease. Athletes with known or suspected cardiovascular disorders should undergo evaluation and risk stratification by sports cardiologists or disease experts prior to participating in sports competition.

The present chapter will review the current knowledge pertaining to participation recommendations and guidelines for athlete screening and for management of athletes with known or suspected cardiovascular conditions.

69.2 Sudden Cardiac Death in Athletes

Sudden cardiac death (SCD), occurring without preceding symptoms and seemingly striking at random, is a tragic occurrence under any circumstance. When it occurs in youth, adolescent, or competitive athletes who are the epitome of health, these events are especially shocking. SCD in well-known athletes often capture headlines, and these harrowing events are on the minds of care providers whenever they deal with heart conditions in athletes.

The epidemiology of SCD is largely age-dependent. SCD in individuals aged >35 years is most often caused by acquired heart disease and coronary atherosclerosis in particular, whereas SCD among those <35 years is more often due to inherited or congenital heart disorders and

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Table 69.1 Causes of sudden cardiac death in athletes

<i>Cardiomyopathies</i>
Hypertrophic cardiomyopathy
Arrhythmogenic right ventricular cardiomyopathy
Dilated cardiomyopathy
Left ventricular non-compaction
<i>Aortopathies</i>
Marfan syndrome
Loeys–Deitz syndrome
Ehler–Danlos disorder (vascular subtype)
Thoracic aortic aneurysm and dissection
<i>Channelopathies</i>
Long QT syndrome
Short QT syndrome
Catecholaminergic polymorphic ventricular tachycardia
Brugada syndrome
Wolff–Parkinson–White syndrome
<i>Congenital abnormalities</i>
Coronary artery anomalies
Bicuspid aortic valve with aortopathy
<i>Acquired disorders</i>
Myocarditis
Atherosclerotic coronary artery disease

primary arrhythmia syndromes. A partial list of disorders capable of causing SCD in athletes is shown in Table 69.1.

The incidence rate of SCD among those <35 years is estimated at 0.7–3.0 cases per 100,000 person-years [1]. The exact incidence has been challenging to determine, largely due to differences in populations being studied and incomplete case capture from media reports or insurance claims. The rate of SCD was observed to be 13.0 per 100,000 person-years (1:9000) among military recruits during initial training [2], but only 1.2 per 100,000 person-years over a 10-year period of observation [3].

Fact Box

SCD is the sudden, unexpected death caused by loss of heart function, most often due to arrhythmias or ineffective pulse generation. It is commonly due to the presence of underlying heart disease with a superimposed trigger. Among individuals <35 years of age, inherited or congenital heart disorders and primary arrhythmia syndromes are the most common causes of death.

Observational studies have shown the rate of SCD to be higher among specific subgroups. Male athletes, compared to female athletes, have an increased risk of SCD both in the general population and in athletes [4–7]. Whereas male athletes have an estimated 1.3-fold relative risk in the general population [8, 9] this may be as high as 3.2–5.6-fold among athletes [4, 10]. The reason for this higher risk among athletes is unclear, but may be related to biologic or societal factors and more frequent exposure to intense exercise. Individuals of African descent have an increased risk of SCD compared to Caucasians, both in the general population [11] and among athletes [6, 7], possibly related to an increased rate of underlying cardiomyopathy.

There are regional variations in the reported distributions of causes of SCD. Whereas definite or possible hypertrophic cardiomyopathy (HCM) appears to be the most common cause of SCD in the US and UK [12, 13], arrhythmogenic right ventricular cardiomyopathy (ARVC) is the most common cause of SCD in Italy (Figure 69.1) [4]. This may be due to genetic variation in population subgroups, societal differences in healthcare and screening, or differences in the medical examiners' approach to postmortem diagnosis. Sudden unexplained death (SUD), in which the autopsy reveals a structurally normal heart and in which primary arrhythmia syndromes are suspected, is present in 15–40% of cases [3, 13, 14].

Although exercise has myriad benefits on health, longevity, and overall well-being, exercise has also been shown to increase the risk of SCD. Data from the Physicians Health Study estimated a 16.9 (95% CI: 10.5–27.0)-fold increased risk of SCD during or within 30 min of exercise [15]. This risk was highest among sedentary individuals who exercised intermittently [15]. A prospective study of Italian youth (age 12–35 years) comprising 29 million person-years of observation suggested that the relative risk of SCD was 2.8 (95% CI: 1.9–3.7) times higher among athletes compared to non-athletes, with 89% of athlete deaths and 9% of non-athlete deaths occurring in the setting of acute exercise [4].

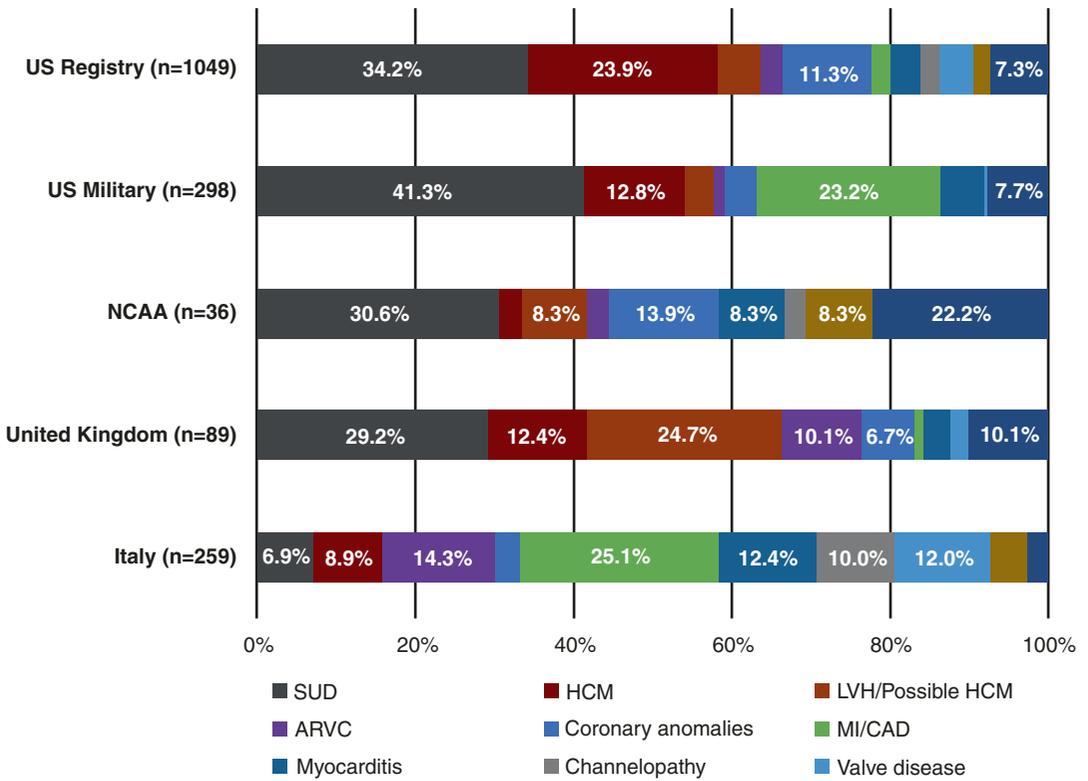


Fig. 69.1 Comparison of etiologies of sudden cardiac death among youth and athletes from (a) U.S. Registry [12] (age ≤ 39 years); (b) United Kingdom [13] (age ≤ 35 years); (c) U.S. military [3] (age < 35 years); (d) NCAA athletes [6] (age 17–26 years); and (e) Italy [4] (age 12–35 years)

In both young and old alike, SCD is thought to arise from an acute trigger superimposed on an underlying susceptible substrate. Exercise can serve as an arrhythmogenic trigger by increasing catecholamine levels, causing dehydration and electrolyte imbalance, increasing blood pressure and shear stress on the aorta, and inducing myocardial ischemia in susceptible individuals [16]. It has been observed that burst sports (e.g., basketball, soccer, football) have a higher rate of SCD than other sporting disciplines [6, 16, 17]. The exact reasons for this are unclear, but may be related to abrupt changes in heart rate and/or greater periods of “supra-maximal” exercise. Alternatively, patients with underlying heart disorders may be underrepresented in sporting disciplines that require high aerobic conditioning due to an inability to maintain high aerobic levels [18].

A study by Harmon et al. showed the incidence of SCD among NCAA athletes (age 17–23 years) over a 10-year period (2003–2013) to be 1:53,703 athlete/years, and observed differences based on sex, ethnicity, sporting discipline, and sporting intensity [10]. Higher risk was seen among male athletes (incidence risk ratio (IRR) 3.22), black athletes (IRR 3.18 versus Caucasians), and in Division 1 and 2 athletes (IRR 1.98 and 2.05, respectively, versus Division 3). Among sporting disciplines, basketball was observed to have the highest risk of SCD (1 in 8978 athlete-years), with black male Division 1 basketball players having a 1 in 1095 risk of SCD over their 4 year eligibility period. Because of this, basketball players have been identified as a particular focus of cardiovascular screening.

Fact Box

Among athletes with SCD, basketball is the most frequent sporting discipline implicated [7, 10]. The incidence of SCD in NCAA Division 1 male basketball players is estimated at 1:5200 per year, corresponding to an average 1:1300 risk over a 4-year collegiate eligibility period [10].

increase in cardiac chamber size. In addition to the mode, duration, and intensity of exercise, age, sex, genetics, and other biologic factors influence this remodeling process [19]. The resultant “athlete’s heart” phenotype can mimic pathologic disease states such as hypertrophic, dilated, and arrhythmogenic cardiomyopathies.

69.3 Exercise-Induced Cardiac Remodeling

Individuals engaging in moderate to vigorous exercise training over months to years undergo a process called “exercise-induced cardiac remodeling” (EICR) in which the heart increases in size and thickness [19, 20]. The resultant physiologic changes—commonly referred to as “athlete’s heart”—increase stroke volume and heart rate reserves, enabling higher cardiac outputs at peak exercise. This process is akin to skeletal muscle adaptation to training.

Although EICR is a physiologic process, it may lead to “abnormalities” (classically defined) on medical testing, such as the appearance of voltage criteria for left ventricular (LV) hypertrophy (LVH) on an electrocardiogram (ECG) or cardiac chambers appearing thickened and/or enlarged on echocardiography. In some instances, it becomes challenging to differentiate EICR from a pathologic condition capable of causing sudden death, such as HCM or ARVC [21, 22]. These “gray zones” of uncertainty can be the source of considerable consternation for medical professionals, which can lead to unnecessary and expensive diagnostic testing or to excluding an athlete from sporting competition unnecessarily. It is therefore essential for clinicians who care for or evaluate athletes to have a fundamental understanding of the ways in which athletic training can affect the cardiovascular system.

Fact Box

In a process called exercise-induced cardiac remodeling, routine vigorous exercise can result in cardiac hypertrophy and an

The “Morganroth Hypothesis” postulates that the manner of cardiac remodeling is determined by the type of exercise performed [23, 24]. Athletes who participate in high dynamic sports (e.g., running, cycling, rowing) experience a repetitive increase in cardiac output with high flow states and lesser increases in afterload. In response, cardiac chambers undergo balanced eccentric dilation and hypertrophy. This results in enlargement of all four cardiac chambers, though this effect is seen earliest in the more compliant chambers (atria and right ventricle). In contrast, athletes who participate in high resistance sports (e.g., weight lifting) experience a repetitive increase in blood pressure and afterload, with less increase in cardiac output, and can develop concentric LVH, though contribution from hypertension needs to be excluded.

In addition to cardiac structural remodeling, exercise also induces changes in cardiac autonomic tone [25]. Endurance training generally results in an increase in vagal parasympathetic innervation, and as a result, bradycardia, junctional escape rhythms, and Mobitz I second-degree AV block (i.e., Wenckebach) are commonly seen on an ECG or rhythm monitoring in athletes and should not be confused with disease.

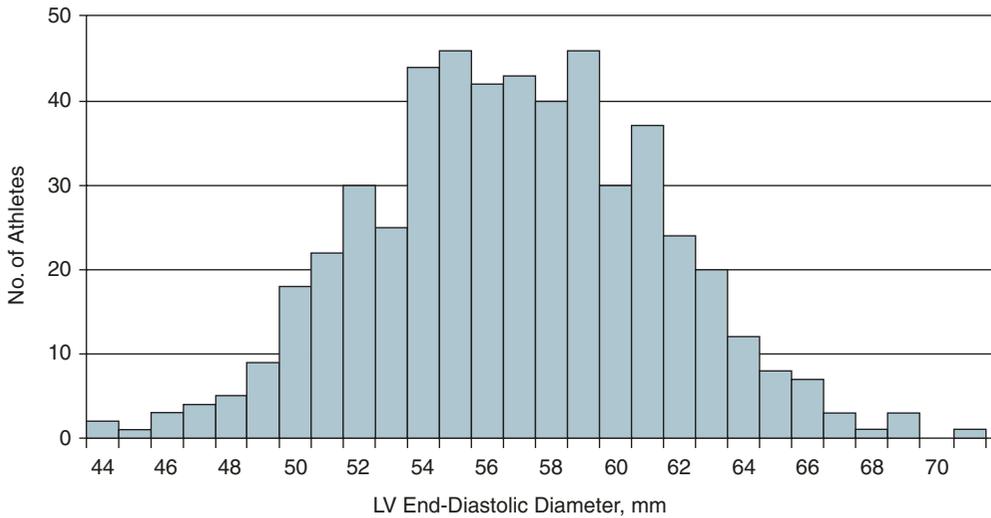
Due to greater degrees of hypertrophy and apical trabeculations, black athletes in particular are prone to “gray zone” conundrums. Black hypertensive patients have greater degrees of LVH compared to white patients with similar age, gender, and blood pressures [26]. Similarly, black athletes have greater LV wall thickness and LV mass index compared to white athletes [27–29], and are more likely to demonstrate LV hypertrabeculation [30] and ECG abnormalities [31]. LV wall thickness >13 mm was seen in 13% of black

male athletes vs. only 4% of white male athletes [32]; similarly, 3% of black female athletes and 0% of white female athletes had LV wall thickness >12 mm [29]. The maximal wall thickness for all athletes in these studies was 16 mm in male and 13 mm in female athletes.

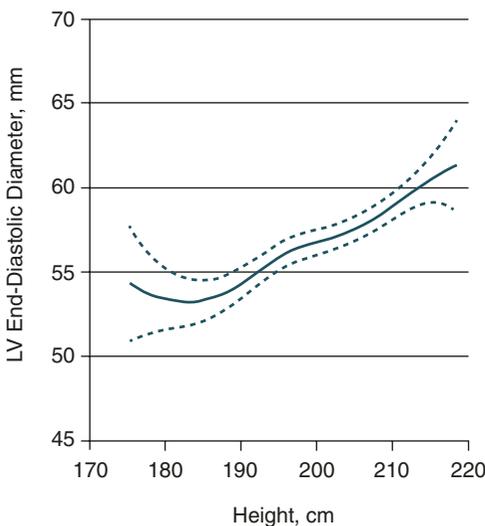
Among National Basketball Association (NBA) basketball players ($n = 526$, 77% African

American), LV dilation (LV end diastolic diameter (LVEDD), ≥ 59 mm) was present in 192 athletes (36.5%), whereas 23 (4.4%) had substantial LV dilation (LVEDD, ≥ 65 mm) [33]. LV chamber size was largely influenced by body size, with increased LVEDD seen with increasing height and body surface area (BSA) (Figure 69.2). Additionally, 27% of NBA athletes had evidence

a Distribution of LVEDD



b Association of LVEDD with height



c Association of LVEDD with BSA

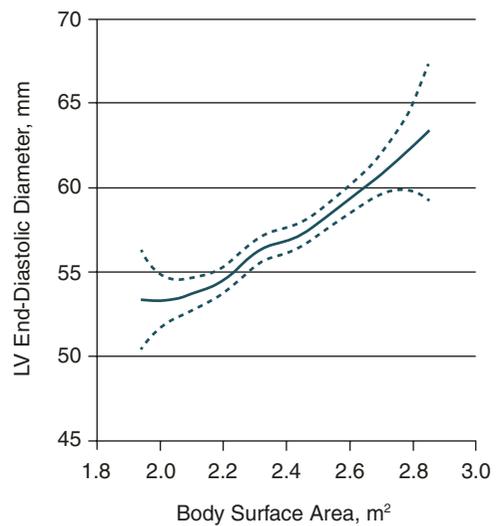


Fig. 69.2 Left ventricular (LV) end-diastolic diameters (LVEDD) in National Basketball Association Athletes ($n = 526$). (a) Overall distribution of LVEDD; (b) mean

LVEDD by athlete height; (c) mean LVEDD by body surface area (BSA). (Reproduced with permission from *JAMA Cardiol.* 2016;1(1):80–87)

of LVH on echocardiography, with LV wall thicknesses ranging from 8 to 15 mm (Figure 69.3).

EICR can also affect the vasculature and the aorta in particular, though this effect is usually small. Aortic dilation is of clinical concern because it results in increased vascular wall stress and increased risk of aortic dissection, an acute, highly morbid condition that does not infrequently result in SCD [34]. Adjusting for age, sex, body size, and blood pressure, a meta-analysis of aortic size among athletes and non-athletes suggested an average increase of 3 mm at the level of the sinuses of Valsalva [35]. Although

current recommendations are for age- and body-size-adjusted normative values, the effect of body size on aortic dimension appears to plateau at anthropomorphic extremes. Among NBA basketball players (mean height 200 cm, weight 101 kg, BSA 2.38), aortic root enlargement >40 mm was seen in 4.6% of the athletes and the maximal aortic root size among all NBA athletes was 42 mm (Figure 69.4). Thus athletes with greater aortic enlargement should be assessed for pathologic conditions, such as the presence of a bicuspid aortic valve or an underlying aortic aneurysm syndrome (e.g., Marfan syndrome).

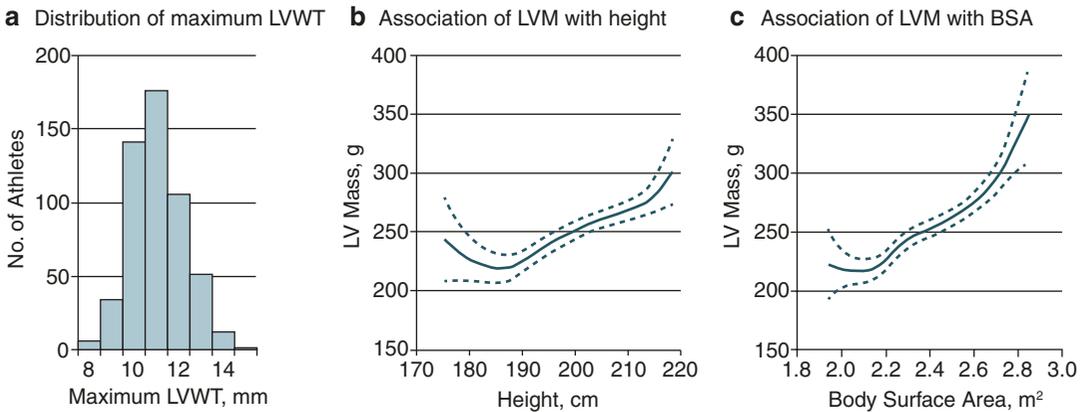


Fig. 69.3 Maximal left ventricular wall thickness (LVWT) and LV mass (LVM) among National Basketball Association Athletes ($n = 526$). (a) Overall distribution of LVWT; (b) mean LVM by athlete height; (c) mean LVM by body surface area (BSA). (Reproduced with permission from *JAMA Cardiol.* 2016;1(1):80–87)

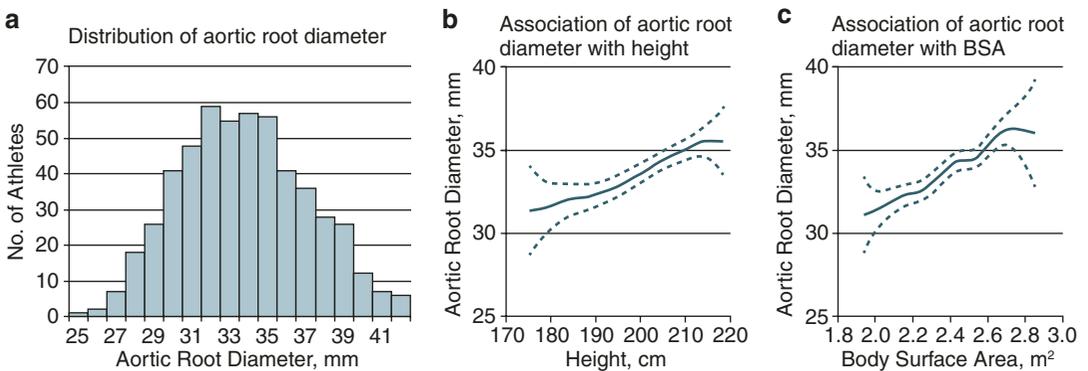


Fig. 69.4 Aortic root diameter among National Basketball Association Athletes ($n = 526$). (a) Overall distribution of aortic root diameters; (b) mean aortic root diameters by athlete height; (c) mean aortic root diameters by body surface area (BSA). (Reproduced with permission from *JAMA Cardiol.* 2016;1(1):80–87)

Fact Box

Among 526 NBA athletes undergoing echocardiographic evaluation, the LV end diastolic diameter ranged from 44 to 71 (mean, 56.8; 95% CI, 56.4–57.2) mm, maximal LV wall thickness ranged from 8 to 15 (mean, 11.0; 95% CI, 10.9–11.1) mm, and aortic diameter ranged from 25 to 42 (mean, 33.7; 95% CI, 33.4–34.0) mm [33].

69.4 Cardiovascular Screening

Most developed countries have adopted public health policies recommending cardiovascular screening prior to organized sports participation. The purpose of pre-participation screening is to identify underlying cardiovascular conditions that predispose to SCD and enhance the safety of sports participation. Indeed, the American Heart Association (AHA) views pre-participation screening as an important public health issue that is “*justifiable, necessary, and compelling* on the basis of ethical, legal, and medical grounds” [36].

Despite broad agreement on the merits for pre-participation screening, there is considerable disagreement regarding the appropriate screening methodology. At the core of this disagreement are tradeoffs between the intensity of screening methods, sensitivity and specificity of testing, cost-effectiveness, infrastructural concerns, and the impact of false-positive results.

Basic screening programs, such as those advocated by the AHA [36] and the Pre-participation Physical Evaluation (PPE) Monograph (fourth edition) [37], consist of a history (including symptom questionnaire and family history assessment) and physical examination with additional evaluation as indicated based on physician judgment. Other health organizations, including the European Society of Cardiology (ESC), recommend the addition of ECG in pre-participation screening [38]. Proponents of ECG screening point to its ability to identify subclinical disease and electrophysiological disorders such as Wolff Parkinson White (WPW) or Long QT syndromes

that are silent on physical examination. Some screening programs, including the NBA, have taken the additional step of including routine echocardiography in the screening process.

Screening tests are generally imperfect, resulting in the need for more expensive, downstream testing to differentiate false-positive results from true disease. Athlete-specific ECG criteria that take EICR into account have been developed, with the International Consensus Criteria being the most recent recommendation [39]. These criteria have been shown to reduce the false-positive rate compared to traditional ECG criteria without compromising the sensitivity to identify conditions associated with SCD [40–42]. In a cohort of 5258 college athletes from the United States, application of the International Criteria lowered the false-positive rate compared to the Seattle Criteria from 2.8% to just 1.3% [41]. However, one study of 409 NBA players (78.8% African-American) still found a relatively high rate of ECG abnormalities (15.6%) when applying the International Criteria [43].

69.5 Cardiovascular Disorders in Athletes and Recommendations for Sports Participation

Exercise has many proven cardiovascular benefits, including a reduction in blood pressure, cholesterol, coronary artery disease, and overall cardiovascular mortality, and should be encouraged for nearly all individuals. However, for individuals with underlying cardiovascular disorders, exercise poses specific challenges and concerns. This is especially true for individuals with disorders that are either capable of causing SCD, or in whom exercise may worsen or accelerate the natural history of the disease.

In 2015, the AHA and American College of Cardiology (ACC) provided updated joint recommendations for sports eligibility and disqualification recommendations for competitive athletes with underlying cardiovascular disease [44]. In 2019, the ESC provided similar, updated recommendations for athletes with

cardiomyopathies [45]. These guidelines have distinguished competitive from recreational sports participation due to the intensity and frequency of training and the external motivation presented and provide separate recommendations regarding recreational sports participation [16]. Current recommendations for the more common cardiovascular conditions found in young athletes are discussed below.

69.5.1 Hypertrophic Cardiomyopathy (HCM)

HCM is defined as unexplained LVH (wall thickness >15 mm) in the absence of other conditions (e.g., hypertension) capable of causing the observed degree of hypertrophy and is present in about 1:200 to 1:500 adults [46]. HCM is caused by gene mutations with proteins of the cardiac sarcomere, usually manifests as asymmetric septal hypertrophy (but can have other morphologies), and can be associated with LV outflow obstruction. Additionally, HCM carries an increased risk of SCD due to ventricular dysrhythmias and is among the most common causes of SCD in youth and athletes [7, 10]. A number of markers (extreme LVH, syncope, family history of SCD, and scarring) infer greater risk of SCD. For patients deemed at high risk of SCD, primary prevention with an implanted cardioverter defibrillators (ICD) should be considered.

The current AHA/ACC recommendation (Class III) is that athletes with a probable or unequivocal diagnosis of HCM should not participate in most competitive sports, with the possible exception of low-intensity activities (e.g., billiards, bowling, archery) [47]. This recommendation applies to all patients with HCM, regardless of symptoms, disease severity, presence of LV outflow obstruction, or perceived SCD risk. In contrast, the ESC recommends thorough risk stratification with disqualification of high-risk athletes, but adopts a shared decision-making model for other, lower-risk athletes [45]. Importantly, an ICD should not be

placed in athletes for the sole purpose of allowing sports participation, and standard criteria for ICD implantation should apply to athletes and non-athletes alike (Class III).

69.5.2 Left Ventricular Non-compaction (LVNC)

LVNC is an uncommon cardiac condition in which there is incomplete myocardial development, resulting in a hypertrabeculated, spongiform appearance of the LV apex and thinning of the compact layer of myocardium [48]. LVNC can progress to a dilated cardiomyopathy, in which LV systolic function decreases. Diagnostic criteria for LVNC based on imaging findings (ratio of trabeculated vs. non-trabeculated criteria) have been proposed. However, differentiation of LVNC from simple hypertrabeculation (normal variant) remains challenging, particularly among black individuals and athletes, in whom hypertrabeculation is more common [49].

Because there is not as much information about the natural history of LVNC and the diagnostic criteria remain challenging, the current AHA/ACC and ESC recommendations are that asymptomatic athletes with hypertrabeculation or possible LVNC and no other concerning features (i.e., preserved ventricular function, no prior syncope, and no arrhythmias on monitoring) may be considered for sports participation [45, 47]. Athletes with LVNC and depressed ventricular function, and those with prior syncope or documented arrhythmias should not participate in competitive sports (Class III).

69.5.3 Arrhythmogenic Right Ventricular Cardiomyopathy (ARVC)

ARVC is a rare disorder caused by genetic mutations in proteins of the cardiac desmosome, an intercellular adhesion complex [50]. This disorder can result in progressive right ventricular enlargement and dysfunction due to fatty infil-

tration and has been associated with ventricular arrhythmias, SCD, and right-sided heart failure. ARVC is the most common cause of SCD in youth and athletes in the Italian registry, but has been less frequently invoked in US registries. The diagnosis of ARVC has been traditionally based on a Task Force diagnostic algorithm, and initial diagnostic evaluation can be extensive [51]. Right ventricular enlargement due to EICR and congenital abnormalities with left to right shunting can mimic ARVC. Importantly, there is growing evidence for an interaction between exercise and ARVC such that competitive sports and/or high levels of endurance exercise accelerate the natural history of the disease, with earlier expression of arrhythmias and RV dysfunction [52–54].

Because of the concern that exercise can worsen the ARVC phenotype, the 2015 AHA/ACC and 2019 ESC recommendations are more conservative than for other cardiomyopathies, and all athletes with a definite, borderline, or possible diagnosis of ARVC are advised not to participate in most competitive sports [45, 47].

69.5.4 Dilated Cardiomyopathy (DCM)

DCM is the most common cardiomyopathy phenotype in the general population but a less common cause of SCD in athletes. DCM results in LV enlargement, and a reduction in LV contractile force can be acquired or genetic. Because EICR results in cardiac chamber dilation with a reduced contractile function at rest in some athletes, the exercise-remodeled heart can resemble DCM. However, a key to differentiating these entities is assessment of peak exercise capacity, as EICR will have ample cardiac reserves with supra-normal exercise capacity and increases in ejection fraction (>10–15%) on stress echocardiography while DCM will have reduced reserves with below average exercise capacity.

The current AHA/ACC recommendations for athletes identified with definite DCM are not to participate in competitive sports [47], whereas the ESC permits some participation if the athlete

is asymptomatic, the ejection fraction is >40%, and no arrhythmias or prior syncope [45].

69.5.5 Wolff Parkinson White (WPW)

WPW is a congenital abnormality in which there is an accessory electrical pathway connecting the atrium to the ventricle, bypassing the atrioventricular (AV) node and potentially enabling rapid electrical conduction. This results in ventricular pre-excitation on ECG with slurred upstroke of the QRS complex called a “delta wave”. WPW is present in about 0.1–0.3% of the population and is often asymptomatic. WPW predisposes to atrial fibrillation, which can be a cause of SCD because of the very rapid conduction and subsequent ventricular rates that can occur. However, the overall contribution of WPW to SCD in athletes has not been well defined due to the challenges in identifying WPW postmortem.

Symptomatic patients with WPW, and all individuals with WPW and atrial fibrillation, should undergo invasive risk stratification with ablation of the accessory pathway when feasible, which is often curative and will eliminate the pre-excitation seen on ECG [55, 56]. Management of the asymptomatic athlete is less clear, though all athletes should undergo either an exercise ECG or invasive EP study for risk stratification. It is then reasonable (class IIa) to ablate asymptomatic but high-risk accessory pathways with refractory periods of <250 ms (corresponding to a heart rate of >240 bpm) [55]. After recovery from a successful ablation, athletes can return to sports competition without restrictions.

69.5.6 Channelopathies

A number of genetic conditions can affect the electrical properties of the heart, predisposing to arrhythmias, syncope, and SCD. Examples of channelopathies include Long QT syndrome (LQTS), Brugada syndrome, and catecholaminergic polymorphic ventricular tachycardia

(CPVT) [57]. These channelopathies are present in about 0.1% of the population and are believed to make up a large proportion of the sudden unexplained deaths seen in athlete SCD registries. LQTS delays repolarization of the myocardium, with resultant prolongation of the QT interval on ECG (corrected for heart rate) to >470 ms in male athletes or >480 ms in female athletes. While there is overlap between QT interval durations in LQTS and the general population, corrected QT intervals >500 ms are generally considered unequivocal LQTS. Brugada syndrome presents with characteristic ECG changes and can be diagnosed on surface ECG, while the diagnosis of CPVT requires ECG monitoring during exercise.

It is recommended (Class I) that all athletes with suspected channelopathies be referred to heart rhythm specialists and that symptomatic athletes refrain from competition until further evaluation [58]. However, there is observational data that asymptomatic and previously symptomatic athletes with channelopathies, when appropriately risk stratified and treated (including beta-blocker therapy and ICD when appropriate), can safely resume sports competition [59–61]. The AHA/ACC guidelines state that competitive sports participation may be considered (Class IIb recommendation) for these athletes after appropriate evaluation and treatment and after 3 months free of symptoms [58].

69.5.7 Bicuspid Aortic Valve

Bicuspid aortic valve (BAV) is the most common congenital heart disorder, with an overall prevalence of 1–2% in the general population [62]. With only two (versus normal three) leaflets, BAVs are prone to progressive stenosis and/or regurgitation. Valve stenosis creates a pressure gradient that increases LV pressures, and this gradient increases with exercise. Valve regurgitation creates a volume load on the LV, but regurgitation may decrease with exercise and higher heart rates. Additionally, there is an association between BAVs and dilation of the ascending aorta. The current AHA/ACC guidelines for BAVs base their recommendations on the sever-

ity of valve dysfunction, placing restrictions on athletes with moderate to severe aortic stenosis (symptomatic or asymptomatic) and on those with severe aortic regurgitation and signs of LV dysfunction [63, 64]. Because valve dysfunction can progress, annual clinical and echocardiographic monitoring is recommended for athletes with BAVs, with periodic exercise stress testing to evaluate the hemodynamic effects of the BAV during exercise.

69.5.8 Anomalous Coronary Arteries

Congenital coronary anomalies are among the most common causes of SCD in athletes [7, 10, 65, 66]. Of particular concern are coronary ostia that arise from the incorrect sinus of Valsalva and course inter-arterially between the pulmonary artery and aorta. Although anomalous right coronary ostia arising from the left sinus are more common in the population, anomalous left coronary ostia are more common in cases of SCD. Acutely angled takeoffs from the aorta and an intramural course are thought to be additional risk markers. Anomalous coronary arteries may present with symptoms of exertional chest pain and occasionally are associated with an abnormal ECG. Coronary or CT angiography is the gold standard for diagnosis, while surgery is the only therapy for correcting these conditions. The ACC/AHA guidelines recommend that all symptomatic athletes with anomalous coronary arteries, and athletes with anomalous left coronary arteries be restricted from sports competition [63]. Athletes may return to sports 3 months after surgical correction. Management of athletes with anomalous right coronary ostia is less clear, though all athletes should be risk stratified by use of stress testing.

69.5.9 Aortic Enlargement

Aortic dissection can be a cause of SCD in athletes but is most commonly seen in the setting of a BAV or an underlying genetic aortopathy

condition such as Marfan syndrome. Although aortic enlargement can occur as part of EICR, aortic root dimensions greater than 40–42 mm in men and 36–38 mm in women rarely occur, even among large body size athletes [33, 35, 67]. Marfan syndrome is caused by pathogenic variation in the Fibrillin-1 (*FBN-1*) gene, leading to an abnormal connective tissue substrate [68]. Multiple organ systems can be involved, and characteristic physical findings for Marfan syndrome may be present, including facial features, pectus abnormalities, pes planus (flat feet), and increased arm wingspan. Aortic aneurysm and dissection are the major causes of morbidity and mortality in Marfan syndrome, and dissections occur at smaller aortic diameters compared to other causes of aortic enlargement (e.g., hypertension). For all athletes with aortic aneurysms, there is concern that exercise, and strength training in particular, will result in increased aortic stress and a faster rate of aneurysm growth and that contact sports will increase the likelihood of dissection. It is recommended that athletes with aortic enlargement undergo routine assessment of aortic size every 6–12 months [67].

The AHA/ACC guidelines provide nuanced recommendations for sports participation based on the size of the aorta and the presence of an underlying aneurysm syndrome [67]. Basketball (a class IIC, moderate static, high dynamic sport) is not recommended for athletes with Marfan syndrome or other genetic aortopathy of any aortic size, or for athletes with BAV and mild-moderate aortic enlargement (>42 mm in men, >39 mm in women).

69.5.10 Myocarditis

Myocarditis is an acquired disorder of myocardial inflammation, usually caused by viral infections. Myocarditis usually presents with chest pain, shortness of breath, or dysrhythmias, often has concurrent ECG changes or troponin elevations, and is a serious condition that can result in permanent cardiac dysfunction and scarring. Cardiac MRI plays an important role in the diagnosis of myocardial inflammation and scar-

ring. For athletes in the acute phase of myocarditis, there is a strong recommendation against sports participation, regardless of the severity of inflammation or the presence of LV dysfunction [47]. After 3–6 months, and prior to returning to sports competition, athletes should then undergo a comprehensive evaluation including an echocardiogram, ambulatory rhythm monitoring, and exercise stress testing. Athletes with normal LV function and no evidence of active myocarditis or clinically significant ectopy or arrhythmias are allowed to return to competition.

69.5.11 Athletes with Implantable Cardioverter Defibrillators (ICDs)

ICDs can play an important role in preventing SCD in individuals deemed at high risk of ventricular dysrhythmias. ICDs are commonly used for the prevention of SCD in HCM, DCM, ARVC, LQTS, and other channelopathies. However, there have been uncertainties regarding the effectiveness of the ICD to detect and treat arrhythmias amid the exercise milieu (tachycardia, dehydration, possible electrolyte imbalance), and concern about inappropriate shocks and damage to the leads and generator amid repetitive exercise and contact sports. To help inform this decision-making, the Sport-ICD Registry tracked athletes with ICDs and found them to be largely effective [69]. Among the 372 registry participants, 13% and 11% had appropriate and inappropriate shocks, with more shocks occurring during exercise than at rest (16% vs. 6%, $p < 0.0001$). However, of the two deaths that occurred during the 31-month follow-up period, neither occurred during or after exercise. In recognition of this data, the AHA/ACC guidelines are more lenient regarding high-intensity sports participation if an athlete has an ICD and has been free of arrhythmias or ICD shocks in recent months [47]. However, standard guidelines regarding ICD implantation apply to athletes and non-athletes alike, and ICDs should not be implanted for the sole purpose of permitting sports participation.

69.6 Shared Decision-Making

The above AHA/ACC recommendations for sports eligibility and disqualification are based on expert consensus, taking into account existing data and important clinical experience. A particular challenge for the guideline committee is the relative paucity of safety data for sports participation for distinct cardiovascular conditions and the tremendous uncertainty that exists when assessing individual athlete risk. Absent this safety data, the current guidelines generally favor risk avoidance, and the overall effect of the guidelines has been criticized for favoring a binary yes-no approach to challenging and nuanced sports eligibility situations.

There has been a general trend in medical practice away from paternalistic recommendations in favor of more patient-centric, shared decision-making, and this approach is gaining increasing acceptance in the sports cardiology community [70] and plays a central role in the ESC sports participation recommendations [45]. With this approach, sports cardiology providers play a fundamental role in informing athletes of the risks involved with sports participation and discussing these risks in the context of the athlete's overall life goals and values. Throughout this process, sports cardiologists and other care providers must continue to place the highest priority on athlete health and wellness.

There are practical challenges to shared decision-making when applied to sports participation questions. First, the risks involved are often unknown and/or unquantifiable, making the overall framework of the discussion less concrete. Second, athletes presented with these dilemmas often have longstanding hopes and dreams tied to sports success, and sports participation is often fundamental to their personal identity. In this regard, the decisions surrounding sports participation are different from most other medical situations. Third, competitive athletes may have external pressures or motivations, including sizeable financial implications in some situations. Fourth, the young athletes who are faced with these questions often tend toward risk-taking behavior and may not accurately appreciate the

risks involved and their long-term implications. Finally, there may be third-party stakeholders such as a school or team who err toward athlete safety or have legal considerations. On the other hand, disqualification from sport can have unintended consequences, such as short- or long-term psychological distress and restricted access to college scholarships or the financial earnings of professional sport.

It is important to understand that the principles of shared decision-making, while attractive to the patient–clinician discourse, are not absolute. For instance, sports cardiologists would uniformly agree that a competitive athlete diagnosed with ARVC should not compete in high-intensity sports. The potential to mitigate risk through medical interventions is also an important element to guide the decision process. Outcome data present for optimally managed LQTS and CPVT is very limited for other diseases, specifically the cardiomyopathies. Thus, while eligibility decisions are particularly difficult in athletes with pathologic conditions associated with SCD, a cautious approach may be prudent until additional safety data emerges.

69.7 Return to Play Considerations

The number of athletes with known cardiovascular conditions participating in sports competition is increasing due to several temporal trends. Contributors include increased disease detection through athlete screening programs, increased recognition of a shared decision-making approach to sports participation determination, and recent observational data regarding the safety of sports participation in athletes with channelopathies, ICDs, and other conditions.

Athletes with confirmed cardiovascular conditions who are deemed eligible to return to play should have risk stratification and treatment optimization prior to resumption of sports competition. A plan for ongoing follow-up and monitoring should be established at that time, and athletes should be serially evaluated on a minimum annual basis while they are actively participating in sports.

Additionally, team physicians, athletic trainers, and coaches should be aware of the athlete's condition, with an emergency action plan in place to be able to respond appropriately should an adverse event occur. For conditions that predispose to SCD, this includes ensuring the availability of an automatic external defibrillator (AED) and educating team personnel in cardiopulmonary resuscitation and AED use.

Take Home Points

1. SCD in athletes is a rare but harrowing event that is usually caused by underlying congenital or genetic heart conditions. Vigorous exercise can increase the risk of SCD among athletes with a pathological cardiac disorder.
2. Athletes participating in competitive sports should undergo cardiovascular screening. ECG screening increases the detection of conditions at risk for SCD and should be considered when appropriate interpretation and cardiology expertise are available.
3. Vigorous exercise training results in physiologic cardiac adaptations, with characteristic enlargement and hypertrophy of cardiac chambers. This process of exercise-induced cardiac remodeling (EICR) can resemble disease states, leading to "gray zones" between health and disease.
4. Athletes with known or suspected cardiovascular conditions should be referred to sports cardiologists or disease experts for further evaluation and risk stratification.
5. The decision to permit sports participation for athletes with cardiovascular conditions should be based on an individual risk assessment, the potential of medical interventions to mitigate risk, and consideration of not only the risk of SCD but also the potential of accelerating or worsening the disease course.

6. For athletes allowed to return to sports competition, there must be good communication between sports cardiologists, team physicians, athletic trainers, and coaches, and an emergency action plan established including access to an AED.

References

1. Harmon KG, Drezner JA, Wilson MG, Sharma S. Incidence of sudden cardiac death in athletes: a state-of-the-art review. *Heart*. 2014;100(16):1227–34.
2. Eckart RE, Scoville SL, Campbell CL, et al. Sudden death in young adults: a 25-year review of autopsies in military recruits. *Ann Intern Med*. 2004;141(11):829–34.
3. Eckart RE, Shry EA, Burke AP, et al. Sudden death in young adults: an autopsy-based series of a population undergoing active surveillance. *J Am Coll Cardiol*. 2011;58(12):1254–61.
4. Corrado D, Basso C, Rizzoli G, Schiavon M, Thiene G. Does sports activity enhance the risk of sudden death in adolescents and young adults? *J Am Coll Cardiol*. 2003;42(11):1959–63.
5. Van Camp SP, Bloor CM, Mueller FO, Cantu RC, Olson HG. Nontraumatic sports death in high school and college athletes. *Med Sci Sports Exerc*. 1995;27(5):641–7.
6. Harmon KG, Asif IM, Klossner D, Drezner JA. Incidence of sudden cardiac death in National Collegiate Athletic Association athletes. *Circulation*. 2011;123(15):1594–600.
7. Maron BJ, Haas TS, Ahluwalia A, Murphy CJ, Garberich RF. Demographics and epidemiology of sudden deaths in young competitive athletes: from the United States National Registry. *Am J Med*. 2016;129(11):1170–7.
8. Kong MH, Fonarow GC, Peterson ED, et al. Systematic review of the incidence of sudden cardiac death in the United States. *J Am Coll Cardiol*. 2011;57(7):794–801.
9. Stecker EC, Reinier K, Marijon E, et al. Public health burden of sudden cardiac death in the United States. *Circ Arrhythm Electrophysiol*. 2014;7(2):212–7.
10. Harmon KG, Asif IM, Maleszewski JJ, et al. Incidence, cause, and comparative frequency of sudden cardiac death in National Collegiate Athletic Association Athletes: a decade in review. *Circulation*. 2015;132(1):10–9.
11. Galea S, Blaney S, Nandi A, et al. Explaining racial disparities in incidence of and survival from out-of-hospital cardiac arrest. *Am J Epidemiol*. 2007;166(5):534–43.

12. Maron BJ, Doerer JJ, Haas TS, Tierney DM, Mueller FO. Sudden deaths in young competitive athletes: analysis of 1866 deaths in the United States, 1980–2006. *Circulation*. 2009;119(8):1085–92.
13. de Noronha SV, Sharma S, Papadakis M, Desai S, Whyte G, Sheppard MN. Aetiology of sudden cardiac death in athletes in the United Kingdom: a pathological study. *Heart*. 2009;95(17):1409–14.
14. Harmon KG, Drezner JA, Maleszewski JJ, et al. Etiologies of sudden cardiac death in National Collegiate Athletic Association Athletes. *Circ Arrhythm Electrophysiol*. 2014;7(2):198–204.
15. Siscovick DS, Weiss NS, Fletcher RH, Lasky T. The incidence of primary cardiac arrest during vigorous exercise. *N Engl J Med*. 1984;311(14):874–7.
16. Maron BJ, Chaitman BR, Ackerman MJ, et al. Recommendations for physical activity and recreational sports participation for young patients with genetic cardiovascular diseases. *Circulation*. 2004;109(22):2807–16.
17. Maron BJ, Shirani J, Poliac LC, Mathenge R, Roberts WC, Mueller FO. Sudden death in young competitive athletes. Clinical, demographic, and pathological profiles. *JAMA*. 1996;276(3):199–204.
18. Basavarajaiah S, Wilson M, Whyte G, Shah A, McKenna W, Sharma S. Prevalence of hypertrophic cardiomyopathy in highly trained athletes: relevance to pre-participation screening. *J Am Coll Cardiol*. 2008;51(10):1033–9.
19. Baggish AL, Wood MJ. Athlete's heart and cardiovascular care of the athlete: scientific and clinical update. *Circulation*. 2011;123(23):2723–35.
20. Dawes TJ, Corden B, Cotter S, et al. Moderate physical activity in healthy adults is associated with cardiac remodeling. *Circ Cardiovasc Imaging*. 2016;9(8):e004712.
21. Chandra N, Bastiaenen R, Papadakis M, Sharma S. Sudden cardiac death in young athletes: practical challenges and diagnostic dilemmas. *J Am Coll Cardiol*. 2013;61(10):1027–40.
22. Maron BJ. Distinguishing hypertrophic cardiomyopathy from athlete's heart physiological remodeling: clinical significance, diagnostic strategies and implications for preparticipation screening. *Br J Sports Med*. 2009;43(9):649–56.
23. Morganroth J, Maron BJ, Henry WL, Epstein SE. Comparative left ventricular dimensions in trained athletes. *Ann Intern Med*. 1975;82(4):521–4.
24. Naylor LH, George K, O'Driscoll G, Green DJ. The athlete's heart: a contemporary appraisal of the 'Morganroth hypothesis'. *Sports Med*. 2008;38(1):69–90.
25. Drezner JA, Fischbach P, Froelicher V, et al. Normal electrocardiographic findings: recognising physiological adaptations in athletes. *Br J Sports Med*. 2013;47(3):125–36.
26. Mayet J, Shahi M, Foale RA, Poulter NR, Sever PS, Mc GTSA. Racial differences in cardiac structure and function in essential hypertension. *BMJ*. 1994;308(6935):1011–4.
27. Sheikh N, Papadakis M, Carre F, et al. Cardiac adaptation to exercise in adolescent athletes of African ethnicity: an emergent elite athletic population. *Br J Sports Med*. 2013;47(9):585–92.
28. Chandra N, Papadakis M, Sharma S. Cardiac adaptation in athletes of black ethnicity: differentiating pathology from physiology. *Heart*. 2012;98(16):1194–200.
29. Rawlins J, Carre F, Kervio G, et al. Ethnic differences in physiological cardiac adaptation to intense physical exercise in highly trained female athletes. *Circulation*. 2010;121(9):1078–85.
30. Luijckx T, Cramer MJ, Zaidi A, et al. Ethnic differences in ventricular hypertrabeculation on cardiac MRI in elite football players. *Neth Heart J*. 2012;20(10):389–95.
31. Sheikh N, Papadakis M, Ghani S, et al. Comparison of electrocardiographic criteria for the detection of cardiac abnormalities in elite black and white athletes. *Circulation*. 2014;129(16):1637–49.
32. Basavarajaiah S, Boraita A, Whyte G, et al. Ethnic differences in left ventricular remodeling in highly-trained athletes relevance to differentiating physiologic left ventricular hypertrophy from hypertrophic cardiomyopathy. *J Am Coll Cardiol*. 2008;51(23):2256–62.
33. Engel DJ, Schwartz A, Homma S. Athletic cardiac remodeling in US professional basketball players. *JAMA Cardiol*. 2016;1(1):80–7.
34. Iskandar A, Thompson PD. Diseases of the aorta in elite athletes. *Clin Sports Med*. 2015;34(3):461–72.
35. Iskandar A, Thompson PD. A meta-analysis of aortic root size in elite athletes. *Circulation*. 2013;127(7):791–8.
36. Maron BJ, Thompson PD, Ackerman MJ, et al. Recommendations and considerations related to preparticipation screening for cardiovascular abnormalities in competitive athletes: 2007 update: a scientific statement from the American Heart Association Council on nutrition, physical activity, and metabolism: endorsed by the American College of Cardiology Foundation. *Circulation*. 2007;115(12):1643–455.
37. Bernhardt DT, Roberts WO. Preparticipation physical evaluation. Elk Grove Village, IL: American Academy of Pediatrics; 2010.
38. Corrado D, Pelliccia A, Bjornstad HH, et al. Cardiovascular pre-participation screening of young competitive athletes for prevention of sudden death: proposal for a common European protocol. Consensus statement of the study Group of Sport Cardiology of the Working Group of Cardiac Rehabilitation and Exercise Physiology and the working Group of Myocardial and Pericardial Diseases of the European Society of Cardiology. *Eur Heart J*. 2005;26(5):516–24.
39. Drezner JA, Sharma S, Baggish A, et al. International criteria for electrocardiographic interpretation in athletes: consensus statement. *Br J Sports Med*. 2017;51(9):704–31.

40. Malhotra A, Dhutia H, Yeo TJ, et al. Accuracy of the 2017 international recommendations for clinicians who interpret adolescent athletes' ECGs: a cohort study of 11 168 British white and black soccer players. *Br J Sports Med.* 2019; <https://doi.org/10.1136/bjsports-2017-098528>.
41. Hyde N, Prutkin JM, Drezner JA. Electrocardiogram interpretation in NCAA athletes: comparison of the 'Seattle' and 'International' criteria. *J Electrocardiol.* 2019;56:81–4.
42. Zorzi A, Calore C, Vio R, Pelliccia A, Corrado D. Accuracy of the ECG for differential diagnosis between hypertrophic cardiomyopathy and athlete's heart: comparison between the European Society of Cardiology (2010) and international (2017) criteria. *Br J Sports Med.* 2018;52(10):667–73.
43. Waase MP, Mutharasan RK, Whang W, et al. Electrocardiographic findings in National Basketball Association Athletes. *JAMA Cardiol.* 2018;3(1):69–74.
44. Maron BJ, Zipes DP, Kovacs RJ, et al. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: preamble, principles, and general considerations: a scientific statement from the American Heart Association and American College of Cardiology. *Circulation.* 2015;132(22):e256–61.
45. Pelliccia A, Solberg EE, Papadakis M, et al. Recommendations for participation in competitive and leisure time sport in athletes with cardiomyopathies, myocarditis, and pericarditis: position statement of the sport cardiology section of the European Association of Preventive Cardiology (EAPC). *Eur Heart J.* 2019;40(1):19–33.
46. Maron BJ. Clinical course and Management of Hypertrophic Cardiomyopathy. *N Engl J Med.* 2018;379(7):655–68.
47. Maron BJ, Udelson JE, Bonow RO, et al. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: task force 3: hypertrophic cardiomyopathy, arrhythmogenic right ventricular cardiomyopathy and other cardiomyopathies, and myocarditis: a scientific statement from the American Heart Association and American College of Cardiology. *Circulation.* 2015;132(22):e273–80.
48. Hussein A, Karimianpour A, Collier P, Krasuski RA. Isolated noncompaction of the left ventricle in adults. *J Am Coll Cardiol.* 2015;66(5):578–85.
49. Gati S, Chandra N, Bennett RL, et al. Increased left ventricular trabeculation in highly trained athletes: do we need more stringent criteria for the diagnosis of left ventricular non-compaction in athletes? *Heart.* 2013;99(6):401–8.
50. Gandjbakhch E, Redheuil A, Pousset F, Charron P, Frank R. Clinical diagnosis, imaging, and genetics of Arrhythmogenic right ventricular cardiomyopathy/dysplasia: JACC state-of-the-art review. *J Am Coll Cardiol.* 2018;72(7):784–804.
51. Marcus FI, McKenna WJ, Sherrill D, et al. Diagnosis of arrhythmogenic right ventricular cardiomyopathy/dysplasia: proposed modification of the task force criteria. *Circulation.* 2010;121(13):1533–41.
52. Ruwald AC, Marcus F, Estes NA 3rd, et al. Association of competitive and recreational sport participation with cardiac events in patients with arrhythmogenic right ventricular cardiomyopathy: results from the north American multidisciplinary study of arrhythmogenic right ventricular cardiomyopathy. *Eur Heart J.* 2015;36(27):1735–43.
53. Ruiz Salas A, Barrera Cordero A, Navarro-Arce I, et al. Impact of dynamic physical exercise on high-risk definite arrhythmogenic right ventricular cardiomyopathy. *J Cardiovasc Electrophysiol.* 2018;29(11):1523–9.
54. La Gerche A, Rakhit DJ, Claessen G. Exercise and the right ventricle: a potential Achilles' heel. *Cardiovasc Res.* 2017;113(12):1499–508.
55. Al-Khatib SM, Arshad A, Balk EM, et al. Risk stratification for arrhythmic events in patients with asymptomatic pre-excitation: a systematic review for the 2015 ACC/AHA/HRS guideline for the Management of Adult Patients with Supraventricular Tachycardia: a report of the American College of Cardiology/American Heart Association Task Force on Clinical Practice Guidelines and the Heart Rhythm Society. *Circulation.* 2016;133(14):e575–86.
56. Zipes DP, Link MS, Ackerman MJ, et al. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: task force 9: arrhythmias and conduction defects: a scientific statement from the American Heart Association and American College of Cardiology. *Circulation.* 2015;132(22):e315–25.
57. Priori SG, Wilde AA, Horie M, et al. Executive summary: HRS/EHRA/APHR expert consensus statement on the diagnosis and management of patients with inherited primary arrhythmia syndromes. *Heart Rhythm.* 2013;10(12):e85–108.
58. Ackerman MJ, Zipes DP, Kovacs RJ, et al. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: task force 10: the cardiac Channelopathies: a scientific statement from the American Heart Association and American College of Cardiology. *Circulation.* 2015;132(22):e326–9.
59. Johnson JN, Ackerman MJ. Competitive sports participation in athletes with congenital long QT syndrome. *JAMA.* 2012;308(8):764–5.
60. Johnson JN, Ackerman MJ. Return to play? Athletes with congenital long QT syndrome. *Br J Sports Med.* 2013;47(1):28–33.
61. Aziz PF, Sweeten T, Vogel RL, et al. Sports participation in genotype positive children with long QT syndrome. *JACC Clin Electrophysiol.* 2015;1(1-2):62–70.
62. Nishimura RA, Otto CM, Bonow RO, et al. AHA/ACC guideline for the Management of Patients with Valvular Heart Disease: a report of the American College of Cardiology/American Heart Association task force on practice guidelines. *Circulation.* 2014;129(23):e521–643.

63. Van Hare GF, Ackerman MJ, Evangelista JA, et al. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: task force 4: congenital heart disease: a scientific statement from the American Heart Association and American College of Cardiology. *Circulation*. 2015;132(22):e281–91.
64. Bonow RO, Nishimura RA, Thompson PD, et al. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: task force 5: Valvular heart disease: a scientific statement from the American Heart Association and American College of Cardiology. *Circulation*. 2015;132(22):e292–7.
65. Cheezum MK, Ghoshhajra B, Bittencourt MS, et al. Anomalous origin of the coronary artery arising from the opposite sinus: prevalence and outcomes in patients undergoing coronary CTA. *Eur Heart J Cardiovasc Imaging*. 2017;18(2):224–35.
66. Harmon KG, Asif IM, Maleszewski JJ, et al. Incidence and etiology of sudden cardiac arrest and death in high school athletes in the United States. *Mayo Clin Proc*. 2016;91(11):1493–502.
67. Braverman AC, Harris KM, Kovacs RJ, et al. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: task force 7: aortic diseases, including Marfan syndrome: a scientific statement from the American Heart Association and American College of Cardiology. *Circulation*. 2015;132(22):e303–9.
68. Radke RM, Baumgartner H. Diagnosis and treatment of Marfan syndrome: an update. *Heart*. 2014;100(17):1382–91.
69. Lampert R, Olshansky B, Heidebuchel H, et al. Safety of sports for athletes with implantable cardioverter-defibrillators: long-term results of a prospective multinational registry. *Circulation*. 2017;135(23):2310–2.
70. Baggish AL, Ackerman MJ, Putukian M, Lampert R. Shared decision making for athletes with cardiovascular disease: practical considerations. *Curr Sports Med Rep*. 2019;18(3):76–81.



Inherited Aortic Disease and Sports Participation

70

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70.1 Introduction

In recent years, there have been multiple athletes who have succumbed to aortic dissection or rupture due to inherited thoracic aortic disease. Many may remember Flo Hyman, a professional volleyball player who collapsed during a volleyball match in Japan in 1986 and was found to have Marfan syndrome (MFS) on autopsy [1]. Chris Patton, a collegiate basketball player, had a similar story 10 years earlier when he collapsed during a recreational basketball game and was found to have MFS on autopsy [1]. More recently, Isaiah Austin was to become the first legally blind athlete to play in the National Basketball Association (NBA); however, after his pre-draft diagnosis with MFS, he has been banned from the NBA [2]. These are just three examples of the cases which have attracted attention in sports medicine to the importance of aortic root disease, including Marfan syndrome, in athletes.

The aortic root is a composite of structures from the aortic valve to the sinotubular junction, just proximal to the beginning of the ascending aorta [3]. The aortic valve has three semilunar leaflets that attach to the ventricular and aortic walls and the anterior mitral leaflet. Above the valve are three aortic sinuses (sinuses of Valsalva). A primary concern in athletes is aortic root dilation (Fig. 70.1) which can result in life-threatening sequelae. Increased wall forces in the area of dilation can lead to dissection—a tear in the wall of the aorta—which can extend retrograde into the coronary vessels or antegrade in the ascending and descending aorta (Fig. 70.2) and ultimately aortic rupture and death [4]. During exercise the body must increase oxygen uptake, which is accompanied by an increase in cardiac output and an increase in blood pressure. The increase in blood pressure, especially during static exercise, adds significant strain on the aorta which can be detrimental in patients with dilated aortas and MFS [5].

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70.2 Normative Values for Aortic Dimensions

The normal aortic root diameter is dependent on multiple factors including age, sex, body size, location of measurement, and the type of imaging modality. Aortic diameters in men are 1–3 mm larger than in women [6]. Variability in the measured aortic diameter may also result from the

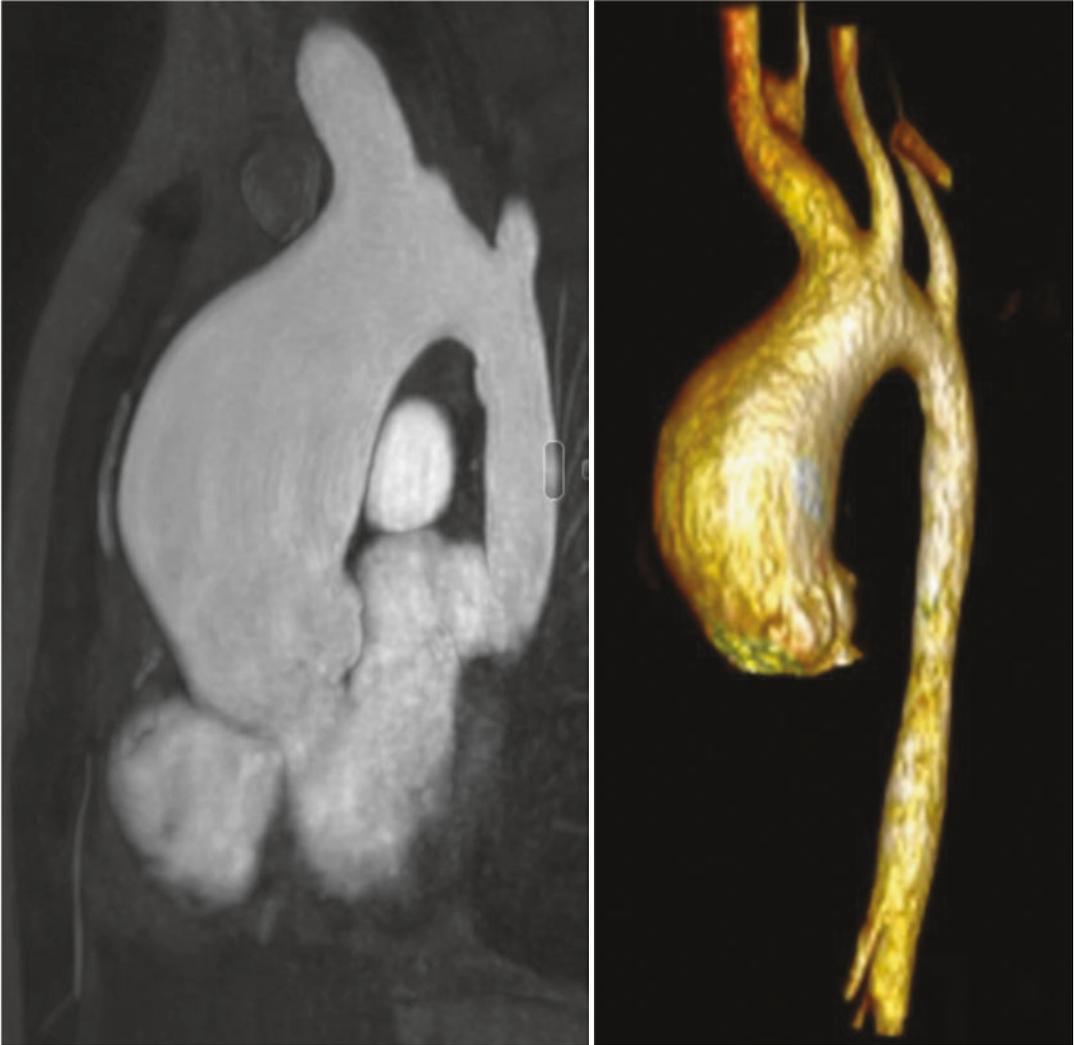


Fig. 70.1 Magnetic resonance imaging of a dilated aortic root, measuring 5.8 cm

type of imaging modality used, whether contrast was administered, and whether internal or external aortic diameters were measured. The upper limit of normal for aortic root diameter in the general population is 39.1 mm in men and 37.2 mm in women [7]. More recently, particularly in non-adults, z scores that incorporate age, sex, height, and weight are preferred for the determination of normal aortic diameter compared with a single aortic dimension [6]. The z score describes the standard deviations above or below the mean a given measurement lies. Aortic dilation is recognized when z score is >2.0 (98th percentile of the general population). Mild, moderate, and severe

aortic dilation are defined by z scores of 2–3, 3–4, and >4 , respectively [6]. The risk of dissection increases with increased aortic diameters, and consequently, accurate measurement and serial determination of aortic dilation is critical.

70.3 Thoracic Aortic Disease

70.3.1 Incidence

The incidence of thoracic aortic disease (TAD) such as thoracic aortic aneurysms and dissection is estimated to be 9.1–16.3/100,000 person-

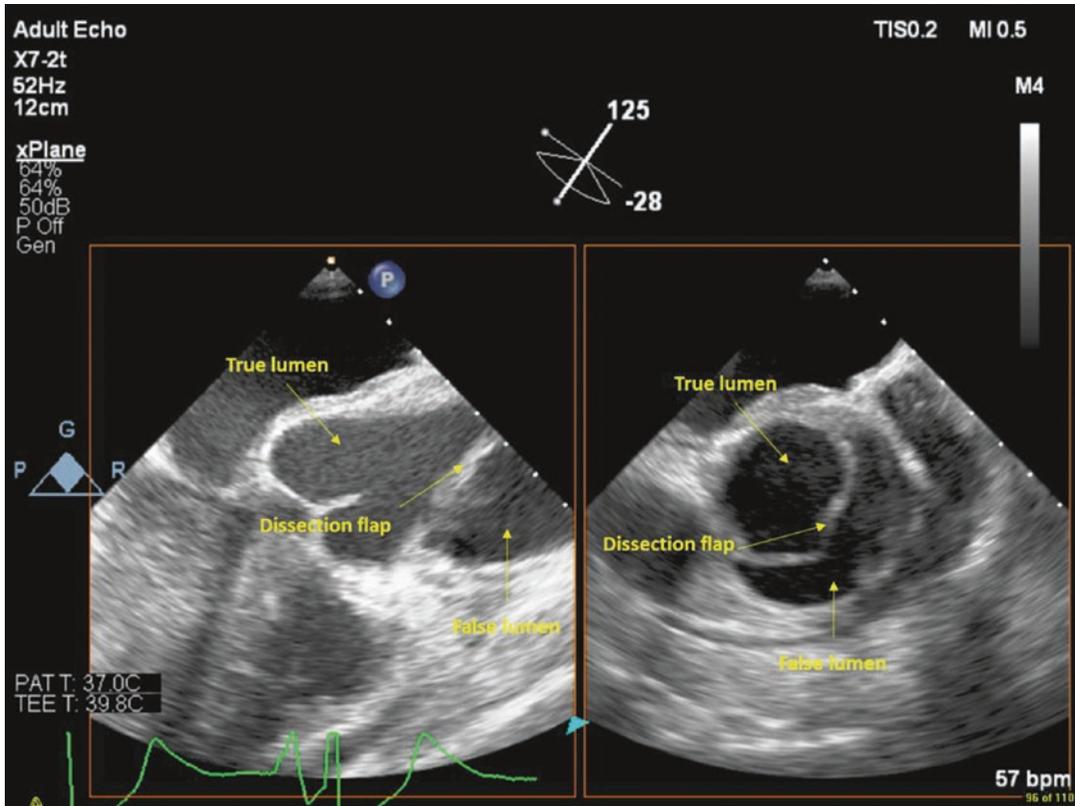


Fig. 70.2 Echocardiogram of an aortic dissection. Arrows marking the dissection flap and true and false lumen

years [8]. However due to the asymptomatic nature of aortic aneurysms, the prevalence is likely underestimated. About 20% of patients with TAD have a family history of aortic disease, either bicuspid aortic valve (BAV) or a genetic connective tissue disorder such as MFS, Loey-Dietz syndrome (LDS), or familial thoracic aortic aneurysm and dissection syndrome (FTAAD) [9]. BAV is of particular interest because it is not uncommon with a prevalence of 1–2% in the general population; however, these individuals seem to be at a lower risk for dissection [10]. MFS has a lower prevalence, about 1 in 5000–10,000, but a higher risk of acute aortic dissection [11]. The prevalence of MFS has been speculated to be higher in basketball and volleyball players due to the tall stature and long limbs associated with the syndrome.

70.3.2 Risk of Dissection

The catastrophic consequence of aortic root dilatation is acute dissection and rupture. The risk of dissection in the general population is due to many factors, but the main risk factor is the severity of the aortic dilation. However, there are some patients with acute aortic dissection who do not have markedly dilated aortas at the time of dissection. For patients with sporadic thoracic aortic aneurysms (TAA) without genetically mediated TAD, TAAs are routinely repaired once they are greater than 5.5 cm, based on the rate of rupture. A 2002 study by Davies et al. included 304 patients with mostly ascending TAA (72%), overall rates of dissection or rupture were 2, 3, and 7% per year for TAA less than 5.0 cm, 5.0–5.9 cm, and ≥ 6.0 cm, respectively. The combined

Table 70.1 Guideline recommendations for surgical intervention [7, 57, 58].

Inherited aortic disease type	American Heart Association 2010, 2014	European Society of Cardiology 2014
Marfan syndrome	>50 mm ^a	≥50
	>40 mm if contemplating pregnancy	≥45 ^e
Loeys–Dietz syndrome thoracic aortic aneurysm/dissection	≥40–42 mm (echocardiogram) ≥44–46 mm (CT or MRI)	Treat patients with marfanoid manifestations as per Marfan syndrome thresholds
Turner syndrome	4.5–5 cm (aorta >2.5 cm/m ²)	>27.5 mm/m ^{2f}
Ehlers–Danlos syndrome (any type)	No specific data ^b	No specific data ^b
Non-syndromic aortopathy	No specific data ^b	No specific data ^b
Bicuspid aortic valve	>55 mm	≥55 mm
	>50 mm ^c	≥50 mm ^g
	>45 mm ^d	
General population	≥55 mm	≥55 mm

^aRepair if family history of aortic dissection at <50 mm, severe aortic regurgitation or rapid growth >5 mm/year

^bPublished data suggests treatment should be based on family history

^cIf any of the following risk factors: family history of dissection, growth ≥5 mm/year

^dIf having surgery for severe aortic stenosis or regurgitation or AR

^eIf any of the following risk factors: family history of dissection, size increase >3 mm/year, severe aortic regurgitation (AR) or mitral regurgitation, desire for pregnancy

^fOther data suggests a cutoff of >25 mm/m² should be utilized

^gIf any of the following risk factors: family history of dissection, hypertension, coarctation of the aorta, size increase >3 mm/year

endpoint of rupture, dissection, or death overall occurred at a rate of 16% per year among patients with TAA ≥6.0 cm [12]. Even though the risk of dissection increases with aortic diameter, most patients who present with dissection have smaller-diameter aneurysms. In a review of 591 patients with type A dissection enrolled in the International Registry of Acute Aortic Dissection (IRAD), the mean diameter was 5.3 cm [13]; however, 40% had an aortic diameter <5.0 cm.

For many patients with genetic causes for their thoracic aneurysms, there is higher risk of dissection than the general population. Specific criteria for elective repair in patients with a genetic cause for their aneurysm are described in Table 70.1.

70.4 Genetic Etiologies for Thoracic Aortic Disease

Genetic/inherited diseases that pose increased risk of aneurysm and dissection include BAV, syndromic conditions such as MFS and LDS, as well

as non-syndromic genetic etiologies known as familial thoracic aortic aneurysm and dissection.

70.4.1 Bicuspid Aortic Valve

The bicuspid aortic valve is the most common congenital heart disorder affecting approximately 1–2% of the population with a 2–3:1 male:female ratio [14]. BAV is a heritable trait. Family studies report the prevalence in the first-degree relatives of an individual with a BAV to be 9–10% [15, 16]. The inheritance of BAV is consistent with an autosomal dominant pattern with reduced penetrance [15]. The genetic causes for BAV remain elusive. Mutations in *NOTCH1* are associated with familial and non-familial BAV and may result in aortic aneurysms and early aortic calcification [14]. Mutations in other single genes such as *ACTA2*, *TGFB2*, *GATA5*, *NKX2-5*, and *SMAD6* have been associated with BAV [17]. Polymorphisms in other genes may associate with BAV risk [18]. In most patients, no specific genetic variant is discovered. BAV is also associated with genetic condi-

tions such as DiGeorge syndrome, LDS, Anderson syndrome (*KCNJ2* mutation), Turner syndrome as well as complex congenital heart disease such as Shone's complex and hypoplastic left heart syndrome [17, 19].

The cardiovascular sequelae associated with BAV include aortic regurgitation (AR), aortic stenosis (AS), as well as aortic pathology; frequently aortic dilation and less commonly aortic coarctation. Over 50% with BAV will have aortic enlargement >2 standard deviations above the norm by 30 years of age. The rate of aortic growth among patients with BAV range from 0.2 to 2.3 mm per year and varies based on age, underlying valve disease (AR versus AS), location of the dilatation, baseline aortic diameter, family history, and leaflet morphology [20–22]. The risk of aortic dissection among BAV populations is unclear. In IRAD, a BAV was present in 9% of dissection patients <40 years old, but only in 1% of those >40 years [23]. In historic necropsy studies, dissections were found in 6–15% of BAV subjects [24]. More contemporary studies have shown the absolute lifetime risk of aortic dissection for the BAV patient followed with routine imaging studies is quite low, with an incidence of 3.1 per 10,000 patient-years [25–28]. Interestingly some studies have also shown individuals with BAV tend to dissect at larger dimensions than their tricuspid aortic valve counterpart (62 mm vs. 53 mm) [29].

70.4.2 Marfan Syndrome

Marfan syndrome is the most common heritable connective tissue disorder with an incidence of 1 in 5000–10,000. MFS is inherited in an autosomal dominant manner with 25% of patients having sporadic, new mutations without a family history [30]. The causative gene mutation involves *FBNI* which encodes the microfibrillar protein fibrillin-1. Alteration in the microfibrillar protein structure results in the classic stigmata of ocular, skeletal, and cardiovascular abnormalities, such as mitral valve prolapse and aortic root dilatation [31]. The diagnosis is based upon the Ghent nosology which was revised by an international expert panel in 2010. The revised criteria places increased impor-

tance on the cardinal clinical features of aortic root dilation/dissection and ectopia lentis (lens dislocation), as well as genetic testing [32]. Molecular genetic testing of the fibrillin 1 (*FBNI*) gene (locus 15q21.1) reveals a causative mutation in ~70–90% of individuals fulfilling the clinical diagnostic criteria for MFS [33]. See Table 70.2 for the clinical criteria to diagnosis MFS.

The most feared consequence of MFS is aortic dissection/rupture. Approximately 60–80% of adult patients with MFS will develop aortic root dilation, with a higher prevalence in males [34]. The mean rate of growth in the ascending aorta/root has been reported to be 0.26 cm ± 0.05 cm/year with annual rates up to 0.46 cm in individuals with aneurysm >6.0 cm. In MFS, the yearly risk of

Table 70.2 Diagnosis of Marfan syndrome based on the Ghent criteria

<i>Diagnosis of Marfan syndrome based on the 2010 Revised Ghent Criteria</i>	
<i>Absence of family history</i>	
1. Aortic root dilation: z score ≥ 2.0 and Ectopia lentis	
2. Aortic root dilation: z score ≥ 2.0 and <i>FBNI</i> mutation	
3. Aortic root dilation: z score ≥ 2.0 and systemic score ≥ 7.0 points	
4. Ectopia lentis and <i>FBNI</i> mutation known to be associated with aortic root dilation	
<i>Presence of family history</i>	
1. Ectopia lentis and family history	
2. Systemic score ≥ 7 points and family history	
3. Aortic root dilation: z score ≥ 2.0 above 20 years old or ≥ 3.0 below 20 years old and family history	
<i>Systemic score</i>	<i>Points</i>
Wrist AND thumb sign	3
Wrist OR thumb sign	1
Pectus carinatum deformity	2
Pectus excavatum or chest asymmetry	1
Hindfoot deformity	2
Plain flat foot	1
Dural ectasia	2
Protocio acetabulae	2
Scoliosis or thoracolumbar kyphosis	2
Reduced elbow extension	1
3 of 5 facial features	1
Skin striae	1
Severe myopia	1
Mitral valve prolapse	1
Reduced upper segment/lower Segment & Increased arm span/height	1

aortic complications (rupture/dissection/death) increases from 8% for aortic root diameter <4.0 cm to over 18% for diameters >6.0 cm [35].

70.4.3 Loey–Dietz Syndrome

Loeys–Dietz syndrome is an autosomal dominant connective tissue disease associated with arterial aneurysms and dissections as well as other systemic involvement [36, 37]. LDS is characterized by the clinical triad of arterial tortuosity and aneurysms, hypertelorism, and bifid uvula or cleft palate. Aneurysms occur in the aortic root, but unlike MFS, are also commonly found in other vascular beds including aortic side branches. Arterial tortuosity commonly affects the head and neck vessels, and cerebral hemorrhage is a common cause of death in individuals with LDS. Importantly, aortic dissection can occur at diameters smaller than those observed in MFS [37, 38]. Abdominal hernias, thin translucent skin, atrophic scars, and poor wound healing are also commonly present in LDS, but not seen in MFS [39]. LDS is associated with deregulation in transforming growth factor-beta (TGF- β) signaling and is now divided into six subtypes based on the pathologic gene variant [40], all of which encode proteins involved in the TGF- β signaling pathway. There is considerable overlap between the subtypes and are considered part of the same clinical continuum.

Specific diagnostic criteria for LDS have not been established. Typically, the diagnosis is rendered when a patient has a vascular aneurysm in addition to identification of a disease-causing mutation on genetic testing. Consideration should be given for genetic testing in families with multiple members with aortic or other arterial dilation. In LDS, dissection can occur at aortic dimensions that are not considered high risk in MFS (<5.0 cm).

70.4.4 Familial Non-syndromic Thoracic Aortic Aneurysm and Dissection

Familial non-syndromic thoracic aortic aneurysm and dissection are characterized by defects of the aorta with few outward physical manifestations.

The diagnosis of FTAAD is based upon the following criteria: progressive enlargement of the ascending aorta, positive family history of thoracic aortic aneurysm and dissection (TAAD), and exclusion of syndromic causes of TAAD such as MFS and LDS. Recent work has identified pathogenic mutations in genes involved in the TGF- β pathway; *TGFBR1*, *TGFBR2*, *TGFB2* ligand, *TGFB3* ligand, and *SMAD3*; and in genes involved in smooth muscle cell function, smooth muscle cell-specific myosin heavy chain 11 (*MYH11*), smooth muscle-specific alpha actin (*ACTA2*), myosin light chain kinase (*MYLK*), and cGMP-dependent protein kinase 1 (*PRKG1*) [41]. However, the genetic etiology is substantially heterogeneous and approximately 80% of all cases of familial non-syndromic TAADs cannot be explained by pathogenic variants in any of these genes [42]. Unlike MFS and even LDS, the natural history and clinical events of non-syndromic TAADs has not been well established. However, like LDS, there has been reports of dissection/rupture at smaller aortic dimensions <5.0 cm.

70.5 Treatment

70.5.1 Medical Management

The majority of currently available recommendations on medical therapy of patients with inherited diseases of the aorta are informed predominantly from MFS studies, as studies on patients with other aortic diseases are scarce. Regardless of the cause and the type of aortic disease, the main goals of medical therapy are to reduce the structural changes within the aortic wall and to delay aortic dilatation and subsequent dissection or rupture. Lowering systemic blood pressure below 120 mmHg and decreasing cardiac contractility are thought to be beneficial strategies in delaying progressive aortic dilatation [7]. Risk factors, particularly smoking and hypertension, should be aggressively addressed in all patients who have aortic disease.

The two main medication classes used for the treatment of inherited aortic diseases are β -blockers and angiotensin receptor blockers (ARB). Although both these classes effectively

reduce blood pressure, each one pertains additional effects by which it reduces the risk of arterial morbidity and mortality. β -Blockers effectively reduce myocardial contractility and the amplitude of aortic wave reflections [43], whereas ARBs affect the TGF- β signaling pathway, which is altered in MFS as well as in other inherited aortic diseases, such as Loeys–Dietz syndrome or some types of FTAAD. There is no specific aortic diameter cutoff value above which initiation of medical therapy is recommended. Rather, data suggests that medical therapy should be initiated at a very young age, potentially during childhood years, in order to effectively modify vascular disease and delay aortic diameter growth [44]. Importantly, no clinical trial to date has demonstrated that medical therapy alone decreases mortality in patients with MFS or other forms of inherited aortic diseases.

70.5.1.1 β -Blockers

For many years, β -blockers were considered the mainstay medical treatment for individuals with inherited aortic diseases. Although these agents were not shown to significantly change the central aortic pressures, a negative inotropic effect of β -blockers may reduce the amplitude of aortic wave reflections [43], and a negative chronotropic effect may reduce the number of contractions per minute and decrease the forces impacting the aorta. Atenolol, a β -blocker that has been extensively studied in MFS, was shown to improve elastic aortic properties and to decrease aortic wall stiffness, a parameter that strongly correlates with aortic-root growth and clinical outcomes [45]. Despite the shortage of data, the American College of Cardiology/American Heart Association/American Association for Thoracic Surgery guidelines recommend that β -blockers should be administered to all patients with MFS and aortic aneurysms unless contraindicated [7].

70.5.1.2 Angiotensin Receptor Blockers and Angiotensin-Converting Enzyme Inhibitors

The deficiency of fibrillin-1 in MFS is accompanied by excessive TGF- β activation and signal-

ing. ARBs possess the capability of modifying this signaling pathway, which provides the rationale behind using these regimens with an aim to slow aortic growth rate. This was first demonstrated in mouse models in 2006, when Habashi et al. showed that the ARB losartan was able to delay aortic aneurysm progression in a mouse model of MFS [46]. Evidence of the beneficial effects of losartan in humans was soon to follow. In their groundbreaking publication from 2008, Brooke et al. reported that in 18 young patients with MFS treated with losartan, the rate of aortic root enlargement decreased considerably as compared to the time period prior to the initiation of this therapy [47]. Despite the small and non-randomized design nature of that study, its results led physicians worldwide to prescribe losartan in an off-label manner to their MFS patients. Fortunately, randomized clinical trials confirmed these favorable findings: in the Dutch COMPARE trial, losartan significantly and specifically reduced dilation of the aortic root when compared with usual therapies, including β -blockers [48]. Furthermore, in a large trial published in 2014 by the Pediatric Heart Network Investigators, atenolol and losartan were shown to almost equally slow aortic diameter growth rates during a 3-year period [44]. An important observation made by the authors of both randomized studies was that the beneficial effects of both ARBs and β -blockers were more prominent in younger patients, indicating that starting medical therapy at a younger age may be warranted. Given the different mechanisms of action, combining ARBs and β -blockers may provide additional protective benefit against aortic growth and dissection, yet data on the efficacy of such combination is limited [49]. Similar to β -blocker therapy, the effects of ARBs have not been evaluated prospectively in patients with other forms of inherited aortic diseases, and the use and the potential vascular protective effects are inferred from the data obtained from MFS patients.

Angiotensin-converting enzyme inhibitors (ACEIs) inhibit the production of angiotensin II and thus reduce the signaling that occurs through both angiotensin II receptors (angiotensin receptors 1 and 2). When comparing the results of

observational studies, losartan appears to be superior to ACEI in protecting the structure of the aortic wall and preventing aortic root dilation in MFS, likely due to its TGF- β blocking capabilities [50].

70.5.1.3 Statins

Statins (3-hydroxy-3-methylglutaryl coenzyme A reductase inhibitors) are primarily used to reduce the progression of atherosclerosis through their lipid-lowering and anti-inflammatory effects. Although interest in incorporating statins into the medical treatment of patients with inherited aortic diseases rose with the appreciation that therapy may reduce the expression of matrix metalloproteinases (MMP) [51], clinical data remains limited. In an MFS mouse model, mice were treated daily from the age of 6 weeks with pravastatin or losartan and both treatments resulted in a significant reduction in aortic root dilatation [52]. Human data primarily exists for individuals with BAV, in which the aortic size was reported lower in statin-treated patients compared to controls [53].

70.5.1.4 Calcium Channel Blockers

Although calcium channel blockers (CCB) have the potential to slow aortic growth rate by reducing central blood pressure, there is some data to suggest that treatment with these agents may in fact lead to exacerbation of aortic disease, as CCB therapy was found to cause premature death in MFS mice due to increased extracellular signal-regulated kinase activation [54]. Therefore, CCBs should be used with caution in patients with MFS and other inherited aortic diseases. Furthermore, when compared to CCBs, atenolol therapy was able to slow the heart rate and delay aortic wave progression more effectively in a randomized clinical trial [55].

70.5.1.5 Fluoroquinolones

In a recently published large case-crossover study, patients with an enlarged aorta or aneurysm were shown to have an overall increased

risk of aortic aneurysm progression of dissection within 60 days of taking fluoroquinolone antibiotics [56]. The likely underlying mechanism for this unwarranted effect is MMP activity induced by fluoroquinolones. Following this and other similar reports, the US Food and Drug Administration published a warning statement, stating that unless there are no other treatment options available, patients with any form of inherited aortic disease, aortic aneurysm, or connective tissue disorder should not be administered fluoroquinolones.

70.5.2 Surgical Management

Medical therapy alone is ineffective in halting aortic growth and preventing aortic dissection or rupture in inherited aortic disease patients. The standard approach to preventing ascending aortic dissection in these patients remains the use of early surgical repair of aortic aneurysms.

Numerical diameter thresholds for such preventive surgical intervention vary by disease type, as aortic dissections or ruptures can occur at different dimensions in different pathologies. Table 70.1 summarizes the current recommendations of aortic diameter thresholds for prophylactic surgery published by the European Society of Cardiology [57] and the American Heart Association [7, 58]. Apart from the actual size of the aorta, the rate of aortic growth, the presence of a concomitant aortic valve disease, and the presence of a positive family history of aortic dissection are also used to risk stratify the patients and for decision-making when considering prophylactic surgery.

70.6 Sports Participation

When contemplating sports participation in patients with aortopathies, the complex effects of exercise on the aortic wall, including (1) the effect of progression of the aortic wall dilatation

and (2) the associated risk for aortic dissection or rupture should be carefully considered. The type (i.e., dynamic or static), intensity, and frequency of exercise can cause distinctive changes in vascular flow dynamics and pressure buildup within the aorta. Thus, conversations with the athlete and the coaching team need to be comprehensive, with an aim to gain as much understanding as possible about the type of the desired sports and the training routine required to become successful in it.

70.6.1 Physiology of Exercise and the Effects on the Aorta

Dynamic exercise causes a mild increase in systolic blood pressure (SBP), a mild decrease in the diastolic blood pressure (DBP), and a concomitant decrease in the peripheral vascular resistance with an overall slight decrease in the mean BP. Maximal efforts do not typically increase SBP to values above 210 mm Hg, whereas submaximal exercise rarely leads to systolic blood pressures higher than 180 mm Hg [59]. Static exercises induce a different physiological response: during intense isometric activities such as heavy weightlifting, there is reflex vasoconstriction to the non-contracting muscles that is aimed to increase perfusion to the contractile muscles. This leads to a significant and disproportionate rise in SBP, DBP, mean BP, and vascular resistance [60]. In one study, healthy volunteers who were evaluated in the catheterization lab with indwelling arterial catheters during exercise were shown to have BP values as high as 480/350 mm Hg during a single bench press, and even higher values with the Valsalva maneuver [61]. Placing these values into context, experimental aortic modeling data and data derived from wall stretch measurements of aortic wall tissues obtained from individuals with aortic diseases indicate that repeat surges of blood pressures exceeding 200–220 mm Hg, especially when accompanied by

the Valsalva maneuver, can cause tearing of the aortic wall and should be avoided in patients with inherited aortic diseases, especially if the aorta is already dilated [62, 63].

Apart from a handful of case reports that described acute aortic dissections during weightlifting in individuals with MFS or other aortopathies, little data exists as to the actual incidence of aortic dissection and rupture during or after exercise in this patient population. One study that showed the potentially higher risk of dissections was published by Hatzaras et al., who reported that among patients who suffered from a dissection at the time of heavy weightlifting, the majority had at least a mildly dilated aorta based on imaging at the time of the dissection (mean 4.6 cm; range 3.0–7.8 cm) [64].

Although isometric exercise causes significant surges in SPB as described above, these surges are relatively short-lived, whereas the more modest but persistent increases in BP associated with dynamic exercise may impact the aortic wall for more prolonged periods. This is the likely underlying mechanism behind the observations that endurance athletes have larger aortas as compared to the general population and even to strength-trained athletes [65, 66]. This provides the rationale for the current recommendation to avoid prolonged dynamic exercise in athletes with inherited aortic diseases, even though like static exercise, the effects have not been directly evaluated in this population.

70.6.2 Recommendations for Physical Activity

In 2015, the American Heart Association/American College of Cardiology published a joint scientific statement on sports participation for *competitive athletes* with aortic diseases, including MFS [67]. As shown in Fig. 70.3, exercise types are divided into static and dynamic components and further sub-categorized into low,

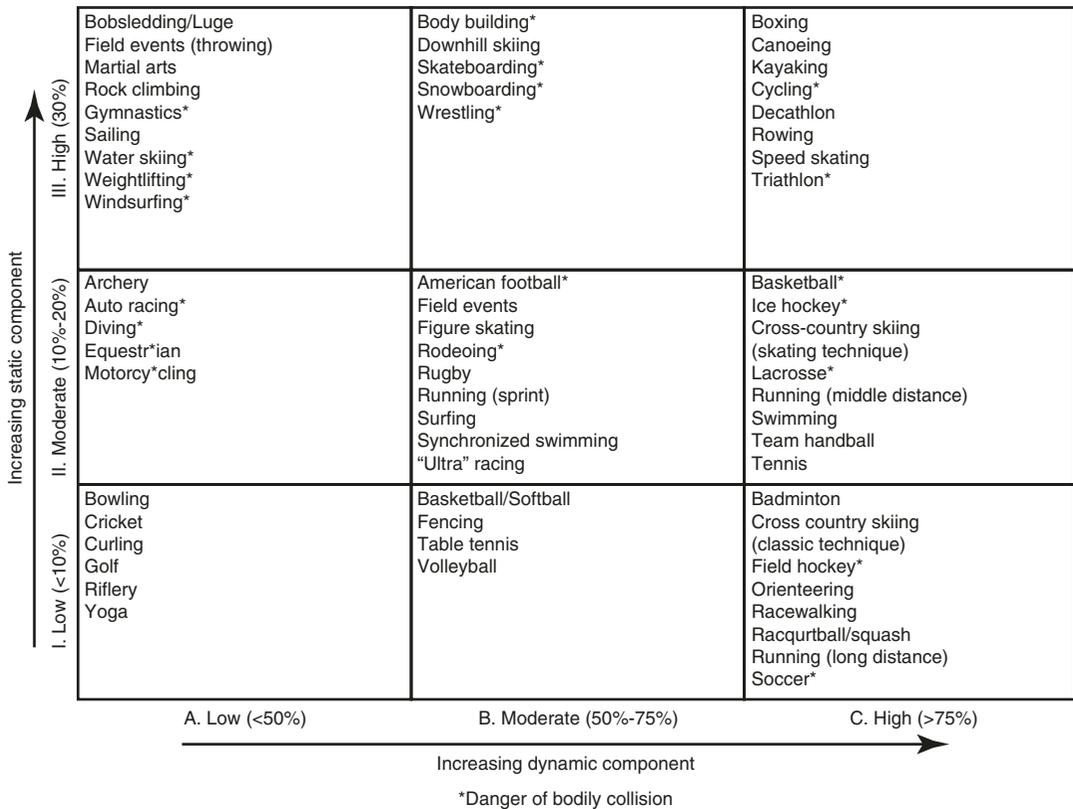


Fig. 70.3 Professional sports classification by dynamic and static components. Increasing static components are based on estimated percent of maximal voluntary contrac-

tion during exercise. Increasing dynamic components are based on maximal oxygen uptake during exercise. (Adapted from Ref. 68)

medium, and high intensity [68]. The recommendations, which are summarized in Fig. 70.4, are provided for each type of aortic disease. Similar to the recommendations on prophylactic surgical intervention, in addition to aortic size, the recommendations for sports participation take into account the presence of positive family history of dissections, left ventricular ejection fraction, the presence of valvular dysfunction, and the presence of disease in arterial vessels other than the aorta.

In certain circumstances, the recommendations permit athletes with MFS and other inherited aortic diseases to participate in low-and-moderate static and low dynamic competitive sports only, yet with the exception of a BAV without aortic dilatation, basketball playing is considered contraindicated in these individuals. The dynamic nature of most inherited aortic

diseases mandates that if participation in competitive sports is continued, close aortic surveillance (i.e., every 6–12 months) with echocardiography or magnetic resonance angiography is warranted. Disqualification from competition should result if the aorta continues to enlarge. Notably, long-term aortic surveillance is recommended even after engagement in the competitive athletic lifestyle has terminated. Athletes with inherited aortic diseases who undergo surgical aortic correction for aneurysm or dissection and who do not have evidence of residual aortic enlargement may participate in only low static, low dynamic sports (class IA, Fig. 70.1) that do not include the potential for bodily collision. Thus, even following aortic surgery, professional basketball playing remains contraindicated.

When considering the basis of this scientific statement, the paucity of published data should

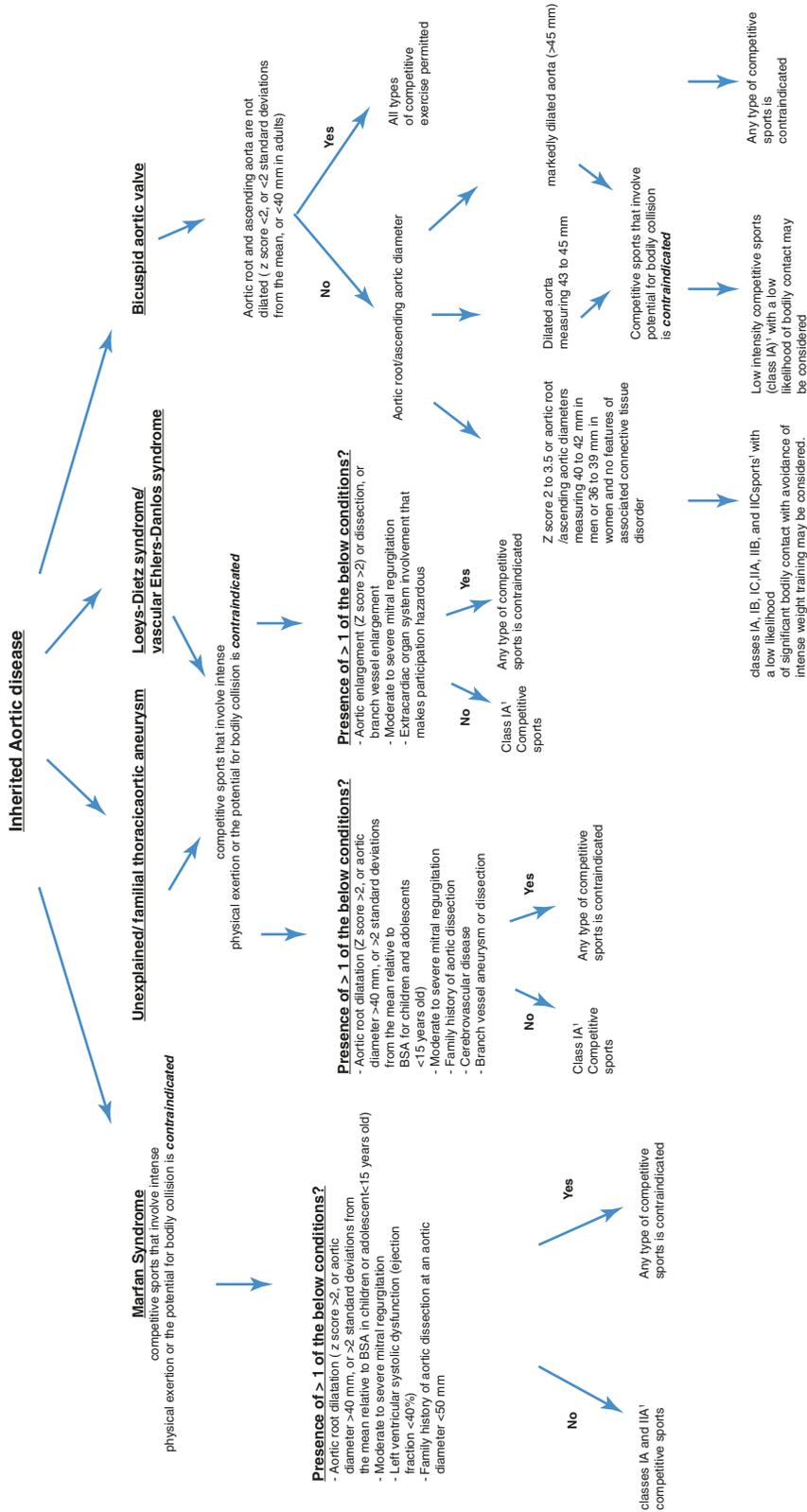


Fig. 70.4 Eligibility and disqualification recommendations for competitive athletes with inherited aortic disease (adapted from Ref. 67). See Fig. 70.3 for relevant classification

be recognized; the recommendations rely on expert opinions and/or small-scale clinical evidence. In a systemic review published by Cheng and Owens, the authors found no outcome studies that evaluated the impact of exercise on the Marfan population or on patients with existing aortic dilation or aneurysms concerning aortic health, aortic enlargement, or adverse outcomes [5]. Thus, we recommend that upon discussing these recommendations with the athlete and/or the coaching staff, full transparency should be practiced.

Take Home Message

MFS and inherited aortic diseases are important to consider in competitive athletes to prevent sudden death from acute aortic dissection or rupture. Despite limited prospective clinical data, the current recommendations permit athletes with MFS and other inherited aortic diseases to participate in low-and-moderate static and low dynamic competitive sports only. With the exception of a BAV without aortic dilatation, participation in competitive basketball is contraindicated in these individuals.

Fact Boxes

1. The incidence of thoracic aortic disease such as thoracic aortic aneurysms and dissection is estimated to be 9.1–16.3/100,000 person-years [8]. About 20% of patients have a family history of aortic disease including Marfan syndrome [9]. The prevalence of Marfan syndrome has been speculated to be higher in basketball and volleyball players due to the tall stature and long limbs associated with the syndrome.
2. No clinical trials to date have demonstrated that medical therapy alone decreases mortality in patients with Marfan syndrome or other forms of inherited aortic diseases. The standard approach to preventing ascending aortic

dissection in these patients remains the use of early surgical repair of aortic aneurysms. Numerical diameter thresholds for preventive surgical intervention vary by disease type, as aortic dissections or ruptures can occur at different dimensions in different pathologies [7, 57, 58].

3. In 2015, the American Heart Association/American College of Cardiology published a joint scientific statement on sports participation for competitive athletes with aortic diseases, including Marfan syndrome. The recommendations permit athletes with Marfan syndrome and other inherited aortic diseases to participate in low-and-moderate static and low dynamic competitive sports only, yet with the exception of a bicuspid aortic valve without aortic dilatation, basketball playing is contraindicated [67].

References

1. Demak R. Marfan Syndrome: A Silent Killer. Vault. <https://www.si.com/vault/1986/02/17/638281/marfan-syndrome-a-silent-killer>. Accessed 8 June 2019.
2. Isaiah Austin daring to dream again after NBA hopes dashed. *SI.com*. <https://www.si.com/nba/2014/11/04/isaiah-austin-marfan-syndrome-nba-baylor>. Accessed 8 June 2019
3. Ho SY. Structure and anatomy of the aortic root. *Eur J Echocardiogr*. 2009;10(1):i3–10.
4. Roman MJ, Rosen SE, Kramer-Fox R, Devereux RB. Prognostic significance of the pattern of aortic root dilation in the Marfan syndrome. *J Am Coll Cardiol*. 1993;22(5):1470–6.
5. Cheng A, Owens D. Marfan syndrome, inherited aortopathies and exercise: what is the right answer? *Heart*. 2015;101(10):752–7.
6. Devereux RB, de Simone G, Arnett DK, Best LG, Boerwinkle E, Howard BV, et al. Normal limits in relation to age, body size and gender of two-dimensional echocardiographic aortic root dimensions in persons ≥ 15 years of age. *Am J Cardiol*. 2012;110(8):1189–94.
7. Hiratzka LF, Bakris GL, Beckman JA, Bersin RM, Carr VF, Casey DE, et al. ACCF/AHA/AATS/ACR/ASA/SCA/SCAI/SIR/STS/SVM guidelines for the diagnosis and management of patients with Thoracic Aortic Disease: a report of the American College of

- Cardiology Foundation/American Heart Association Task Force on Practice Guidelines, American Association for Thoracic Surgery, American College of Radiology, American Stroke Association, Society of Cardiovascular Anesthesiologists, Society for Cardiovascular Angiography and Interventions, Society of Interventional Radiology, Society of Thoracic Surgeons, and Society for Vascular Medicine. *Circulation*. 2010;121(13):e266–369.
8. Christian O, Stefan T, Elisabeth S, Anders E, Fredrik G. Thoracic aortic aneurysm and dissection. *Circulation*. 2006;114(24):2611–8.
 9. Albornoz G, Coady MA, Roberts M, Davies RR, Tranquilli M, Rizzo JA, et al. Familial thoracic aortic aneurysms and dissections—incidence, modes of inheritance, and phenotypic patterns. *Ann Thorac Surg*. 2006;82(4):1400–5.
 10. Girdauskas E, Disha K, Borger MA, Kuntze T. Risk of proximal aortic dissection in patients with bicuspid aortic valve: how to address this controversy? *Interact Cardiovasc Thorac Surg*. 2014;18(3):355–9.
 11. Dietz HC, Pyeritz RE. Mutations in the human gene for fibrillin-1 (FBN1) in the Marfan syndrome and related disorders. *Hum Mol Genet*. 1995;4(suppl_1):1799–809.
 12. Davies RR, Goldstein LJ, Coady MA, Tittle SL, Rizzo JA, Kopf GS, et al. Yearly rupture or dissection rates for thoracic aortic aneurysms: simple prediction based on size. *Ann Thorac Surg*. 2002;73(1):17–27; discussion 27–28
 13. Pape LA, Tsai TT, Isselbacher EM, Oh JK, O’gara PT, Evangelista A, et al. Aortic diameter \geq 5.5 cm is not a good predictor of type a aortic dissection: observations from the international registry of acute aortic dissection (IRAD). *Circulation*. 2007;116(10):1120–7.
 14. Braverman AC, Güven H, Beardslee MA, Makan M, Kates AM, Moon MR. The bicuspid aortic valve. *Curr Probl Cardiol*. 2005;30(9):470–522.
 15. Cripe L, Andelfinger G, Martin LJ, Shoener K, Benson DW. Bicuspid aortic valve is heritable. *J Am Coll Cardiol*. 2004;44(1):138–43.
 16. R Hales A, T Mahle W. Echocardiography screening of siblings of children with bicuspid aortic valve. *Pediatrics*. 2014;133:e1212–7.
 17. Andreassi MG, Della Corte A. Genetics of bicuspid aortic valve aortopathy. *Curr Opin Cardiol*. 2016;31(6):585–92.
 18. Dargis N, Lamontagne M, Gaudreault N, Sbarra L, Henry C, Pibarot P, et al. Identification of gender-specific genetic variants in patients with bicuspid aortic valve. *Am J Cardiol*. 2016;117(3):420–6.
 19. Prakash SK, Bossé Y, Muehlschlegel JD, Michelena HI, Limongelli G, Della Corte A, et al. A roadmap to investigate the genetic basis of bicuspid aortic valve and its complications: insights from the international BAVCon (bicuspid aortic valve consortium). *J Am Coll Cardiol*. 2014;64(8):832–9.
 20. Adamo L, Braverman AC. Surgical threshold for bicuspid aortic valve aneurysm: a case for individual decision-making. *Heart*. 2015;101(17):1361–7.
 21. Avadhani SA, Martin-Doyle W, Shaikh AY, Pape LA. Predictors of ascending aortic dilation in bicuspid aortic valve disease: a five-year prospective study. *Am J Med*. 2015;128(6):647–52.
 22. Verma S, Siu SC. Aortic dilatation in patients with bicuspid aortic valve. *N Engl J Med*. 2014;370(20):1920–9.
 23. Januzzi JL, Isselbacher EM, Fattori R, Cooper JV, Smith DE, Fang J, et al. Characterizing the young patient with aortic dissection: results from the international registry of aortic dissection (IRAD). *J Am Coll Cardiol*. 2004;43(4):665–9.
 24. Larson EW, Edwards WD. Risk factors for aortic dissection: a necropsy study of 161 cases. *Am J Cardiol*. 1984;53(6):849–55.
 25. Tzemos N, Therrien J, Yip J, Thanassoulis G, Tremblay S, Jamorski MT, et al. Outcomes in adults with bicuspid aortic valves. *JAMA*. 2008;300(11):1317–25.
 26. Michelena HI, Desjardins VA, Avierinos J-F, Russo A, Nkomo VT, Sundt TM, et al. Natural history of asymptomatic patients with normally functioning or minimally dysfunctional bicuspid aortic valve in the community. *Circulation*. 2008;117(21):2776–84.
 27. Michelena HI, Khanna AD, Mahoney D, Margaryan E, Topilsky Y, Suri RM, et al. Incidence of aortic complications in patients with bicuspid aortic valves. *JAMA*. 2011;306(10):1104–12.
 28. Kim JB, Spotnitz M, Lindsay ME, MacGillivray TE, Isselbacher EM, Sundt TM. Risk of aortic dissection in the moderately dilated ascending aorta. *J Am Coll Cardiol*. 2016;68(11):1209–19.
 29. Etz CD, von Aspern K, Hoyer A, Girkbach FF, Leontyev S, Bakhtiary F, et al. Acute type a aortic dissection: characteristics and outcomes comparing patients with bicuspid versus tricuspid aortic valve. *Eur J Cardiothorac Surg*. 2015;48(1):142–50.
 30. Gray JR, Bridges AB, Faed MJ, Pringle T, Baines P, Dean J, et al. Ascertainment and severity of Marfan syndrome in a Scottish population. *J Med Genet*. 1994;31(1):51–4.
 31. Pyeritz RE, McKusick VA. The Marfan Syndrome: diagnosis and management. *N Engl J Med*. 1979;300(14):772–7.
 32. Loeys BL, Dietz HC, Braverman AC, Callewaert BL, De Backer J, Devereux RB, et al. The revised Ghent nosology for the Marfan syndrome. *J Med Genet*. 2010;47(7):476–85.
 33. Dietz H. Marfan Syndrome. In: Adam MP, Ardinger HH, Pagon RA, Wallace SE, Bean LJ, Stephens K, et al., editors. *GeneReviews®*. Seattle (WA): University of Washington, Seattle; 1993. <http://www.ncbi.nlm.nih.gov/books/NBK1335/>. Accessed 25 June 2019.
 34. Come PC, Fortuin NJ, White RI, McKusick VA. Echocardiographic assessment of cardiovascular abnormalities in the Marfan syndrome. Comparison with clinical findings and with roentgenographic estimation of aortic root size. *Am J Med*. 1983;74(3):465–74.
 35. Saeyeldin A, Zafar MA, Velasquez CA, Ip K, Gryaznov A, Brownstein AJ, et al. Natural history

- of aortic root aneurysms in Marfan syndrome. *Ann Cardiothorac Surg*. 2017;6(6):625–32.
36. Loeys BL, Chen J, Neptune ER, Judge DP, Podowski M, Holm T, et al. A syndrome of altered cardiovascular, craniofacial, neurocognitive and skeletal development caused by mutations in TGFBR1 or TGFBR2. *Nat Genet*. 2005;37(3):275–81.
 37. Loeys BL, Schwarze U, Holm T, Callewaert BL, Thomas GH, Pannu H, et al. Aneurysm syndromes caused by mutations in the TGF-beta receptor. *N Engl J Med*. 2006;355(8):788–98.
 38. Williams JA, Loeys BL, Nwakanma LU, Dietz HC, Spevak PJ, Patel ND, et al. Early surgical experience with Loeys-Dietz: a new syndrome of aggressive thoracic aortic aneurysm disease. *Ann Thorac Surg*. 2007;83(2):S757–63; discussion S785–790
 39. Meester JAN, Verstraeten A, Schepers D, Alaerts M, Van Laer L, Loeys BL. Differences in manifestations of Marfan syndrome, Ehlers-Danlos syndrome, and Loeys-Dietz syndrome. *Ann Cardiothorac Surg*. 2017;6(6):582–94.
 40. MacCarrick G, Black JH, Bowdin S, El-Hamamsy I, Frischmeyer-Guerrero PA, Guerrero AL, et al. Loeys-Dietz syndrome: a primer for diagnosis and management. *Genet Med*. 2014;16(8):576–87.
 41. Braverman AC. Heritable thoracic aortic aneurysm disease: recognizing phenotypes, exploring genotypes*. *J Am Coll Cardiol*. 2015;65(13):1337–9.
 42. Takeda N, Komuro I. Genetic basis of hereditary thoracic aortic aneurysms and dissections. *J Cardiol*. 2019;74(2):136–43.
 43. Ohte N, Narita H, Sugawara M, Niki K, Okada T, Harada A, et al. Clinical usefulness of carotid arterial wave intensity in assessing left ventricular systolic and early diastolic performance. *Heart Vessel*. 2003;18(3):107–11.
 44. Lacro RV, Dietz HC, Sleeper LA, Yetman AT, Bradley TJ, Colan SD, et al. Atenolol versus losartan in children and young adults with Marfan's Syndrome. *N Engl J Med*. 2014;371(22):2061–71.
 45. Selamet Tierney ES, Levine JC, Sleeper LA, Roman MJ, Bradley TJ, Colan SD, et al. Influence of aortic stiffness on aortic-root growth rate and outcome in patients with the Marfan Syndrome. *Am J Cardiol*. 2018;121(9):1094–101.
 46. Habashi JP, Judge DP, Holm TM, Cohn RD, Loeys BL, Cooper TK, et al. Losartan, an AT1 antagonist, prevents aortic aneurysm in a mouse model of Marfan syndrome. *Science*. 2006;312(5770):117–21.
 47. Brooke BS, Habashi JP, Judge DP, Patel N, Loeys B, Dietz HC. Angiotensin II blockade and aortic-root dilation in Marfan's syndrome. *N Engl J Med*. 2008;358(26):2787–95.
 48. Groenink M, den Hartog AW, Franken R, Radonic T, de Waard V, Timmermans J, et al. Losartan reduces aortic dilatation rate in adults with Marfan syndrome: a randomized controlled trial. *Eur Heart J*. 2013;34(45):3491–500.
 49. Chiu H-H, Wu M-H, Wang J-K, Lu C-W, Chiu S-N, Chen C-A, et al. Losartan added to β -blockade therapy for aortic root dilation in Marfan syndrome: a randomized, open-label pilot study. *Mayo Clin Proc*. 2013;88(3):271–6.
 50. Hackam DG, Thiruchelvam D, Redelmeier DA. Angiotensin-converting enzyme inhibitors and aortic rupture: a population-based case-control study. *Lancet*. 2006;368(9536):659–65.
 51. Nagashima H, Aoka Y, Sakomura Y, Sakuta A, Aomi S, Ishizuka N, et al. A 3-hydroxy-3-methylglutaryl coenzyme a reductase inhibitor, cerivastatin, suppresses production of matrix metalloproteinase-9 in human abdominal aortic aneurysm wall. *J Vasc Surg*. 2002;36(1):158–63.
 52. McLoughlin D, McGuinness J, Byrne J, Terzo E, Huuskonen V, McAllister H, et al. Pravastatin reduces Marfan aortic dilation. *Circulation*. 2011;124(11 Suppl):S168–73.
 53. Goel SS, Tuzcu EM, Agarwal S, Aksoy O, Krishnaswamy A, Griffin BP, et al. Comparison of ascending aortic size in patients with severe bicuspid aortic valve stenosis treated with versus without a statin drug. *Am J Cardiol*. 2011;108(10):1458–62.
 54. Doyle JJ, Doyle AJ, Wilson NK, Habashi JP, Bedja D, Whitworth RE, et al. A deleterious gene-by-environment interaction imposed by calcium channel blockers in Marfan syndrome. *Elife*. 2015;27:4.
 55. Williams A, Kenny D, Wilson D, Fagenello G, Nelson M, Dunstan F, et al. Effects of atenolol, perindopril and verapamil on haemodynamic and vascular function in Marfan syndrome—a randomised, double-blind, crossover trial. *Eur J Clin Investig*. 2012;42(8):891–9.
 56. Lee C-C, Lee M-TG, Hsieh R, Porta L, Lee W-C, Lee S-H, et al. Oral Fluoroquinolone and the risk of aortic dissection. *J Am Coll Cardiol*. 2018;72(12):1369–78.
 57. Erbel R, Aboyans V, Boileau C, Bossone E, Bartolomeo RD, Eggebrecht H, et al. ESC Guidelines on the diagnosis and treatment of aortic diseases: Document covering acute and chronic aortic diseases of the thoracic and abdominal aorta of the adult. The Task Force for the Diagnosis and Treatment of Aortic Diseases of the European Society of Cardiology (ESC). *Eur Heart J*. 2014;35(41):2873–926.
 58. Nishimura RA, Otto CM, Bonow RO, Carabello BA, Erwin JP, Guyton RA, et al. 2014 AHA/ACC guideline for the Management of Patients with Valvular Heart Disease: executive summary: a report of the American College of Cardiology/American Heart Association task force on practice guidelines. *Circulation*. 2014;129(23):2440–92.
 59. Douard H, Vuillemin C, Bordier P, Tourtoulou V, Gosse P, Broustet JP. Normal blood pressure profiles during exercise according to age, sex and protocols. *Arch Mal Coeur Vaiss*. 1994;87(3):311–8.
 60. Williams Mark A, Haskell William L, Ades Philip A, Amsterdam Ezra A, Vera B, Franklin Barry A, et al. Resistance exercise in individuals with and without cardiovascular disease: 2007 update. *Circulation*. 2007;116(5):572–84.
 61. MacDougall JD, Tuxen D, Sale DG, Moroz JR, Sutton JR. Arterial blood pressure response to heavy resistance exercise. *J Appl Physiol*. 1985;58(3):785–90.

62. Koullias G, Modak R, Tranquilli M, Korkolis DP, Barash P, Eleftheriades JA. Mechanical deterioration underlies malignant behavior of aneurysmal human ascending aorta. *J Thorac Cardiovasc Surg.* 2005;130(3):677–83.
63. Vorp DA, Schiro BJ, Ehrlich MP, Juvonen TS, Ergin MA, Griffith BP. Effect of aneurysm on the tensile strength and biomechanical behavior of the ascending thoracic aorta. *Ann Thorac Surg.* 2003;75(4):1210–4.
64. Hatzaras I, Tranquilli M, Coady M, Barrett PM, Bible J, Eleftheriades JA. Weight lifting and aortic dissection: more evidence for a connection. *Cardiology.* 2007;107(2):103–6.
65. Iskandar A, Thompson PD. A meta-analysis of aortic root size in elite athletes. *Circulation.* 2013;127(7):791–8.
66. Pelliccia A, Di Paolo FM, Quattrini FM. Aortic root dilatation in athletic population. *Prog Cardiovasc Dis.* 2012;54(5):432–7.
67. Braverman AC, Harris KM, Kovacs RJ, Maron BJ. American Heart Association electrocardiography and arrhythmias Committee of Council on clinical cardiology, council on cardiovascular disease in young, council on cardiovascular and stroke nursing, council on functional genomics and translational biology, and American College of Cardiology. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: task force 7: aortic diseases, including Marfan Syndrome: a scientific statement from the American Heart Association and American College of Cardiology. *Circulation.* 2015;132(22):e303–9.
68. Levine Benjamin D, Baggish Aaron L, Kovacs Richard J, Link Mark S, Maron Martin S, Mitchell Jere H. Eligibility and disqualification recommendations for competitive athletes with cardiovascular abnormalities: task force 1: classification of sports: dynamic, static, and Impact. *Circulation.* 2015;132(22):e262–6.



Selected Medical Issues in Basketball: Diabetes, Allergies, Asthma, and Dermatologic Issues

71

Christopher M. Miles and Laura Lintner

71.1 Diabetes Mellitus

- Basketball athletes with type 1 diabetes mellitus perform at all levels of competition.
- It is important to check pre-exercise glucose levels to guide play, using the recommended algorithm.
- One must watch for acute hypoglycemia or late-onset post-exercise hypoglycemia (LOPEH).

71.1.1 Introduction

Diabetes mellitus can impact basketball athletes at all levels of competition. Even athletes at the highest level of American professional basketball have competed with this condition. Players such as Gary Forbes, Chris Dudley, and Adam Morrison [1, 2] all have played in the National Basketball Association (NBA) despite their diagnosis of type 1 diabetes mellitus. It is important to be able to recognize the signs and symptoms

of this condition and management options in the basketball athlete.

71.1.2 Epidemiology

Diabetes mellitus (type 1 and 2) is a global phenomenon with a heavy burden of disease. It is estimated that across the globe in 2015, there were 415 million adults with diabetes and that by 2040 this number would rise to 642 million patients with the disease [3]. Type 2 diabetes mellitus is the predominant subtype among adults, while type 1 is predominant among pediatric and adolescent patients. In the United States, type 2 diabetes accounted for 91.2% of diabetes diagnoses in adult patients [4, 5]. Though the incidence of type 2 diabetes is increasing in pediatric and adolescent patients, the majority of diabetes in this age group is still type 1 [6]. There is currently no specific epidemiologic data on the number of basketball athletes with diabetes mellitus.

71.1.3 Pathophysiology

Diabetes mellitus (DM) is typically divided into insulin production and insulin usage disorders. In type 1 diabetes, beta cells of the pancreas are unable to produce sufficient insulin because they are destroyed by an autoimmune cause. This leads to a systemic insulin deficiency causing

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hyperglycemia. Type 2 diabetes usually results from an insulin resistance at the receptors of the peripheral cells. This may ultimately lead to a progressive decline in beta cells' ability to produce insulin. Differentiating the type of diabetes mellitus through the use of autoantibody studies and waist circumference has helped guide management of the condition in adolescent patients [6]. Additional causes of diabetes such as gestational diabetes, pancreatitis, or drug induced diabetes can all lead to hyperglycemia [4].

71.1.4 Presentation

Hyperglycemia may be identified on routine lab screening, or a basketball athlete may present with the symptomatic findings of hyperglycemia. Most commonly a patient with hyperglycemia presents with polyuria, polydipsia, fatigue, weight loss, or vision changes [7]. In severe hyperglycemia, a patient may progress to an acidosis or hyperosmolar state, which can present as lethargy, abdominal pain, nausea and vomiting, dehydration, and abnormal breathing (Kussmaul respirations) [8]. Diabetes mellitus can present as neurologic (neuropathy) or musculoskeletal complaints, though these generally occur later in the disease process. Patients with diabetes mellitus are at an increased risk for conditions like adhesive capsulitis, Dupuytren's contracture, carpal tunnel syndrome, and flexor tenosynovitis (trigger finger). In addition, diabetic amyotrophy can be seen in longer standing disease [9].

71.1.5 Diagnosis

The diagnosis of diabetes mellitus can be confirmed through laboratory testing. There are multiple laboratory tests that can be used to confirm the diagnosis (see Table 71.1). Fasting glucose (greater than 8 h fasting) 126 mg/dL (7.0 mmol/L) or higher is sufficient for the diagnosis. In addition, hemoglobin A1c greater than 6.5% can be used for diagnosis. In any patient with symptoms of hyperglycemia and random plasma glucose of over 200 mg/dL, a diagnosis of diabetes can be made. Finally, a 2-h oral glucose tolerance test

Table 71.1 Criteria for diagnosis of diabetes (any one of the following is sufficient for diagnosis)

In a patient with symptoms of hyperglycemia, a random plasma glucose ≥ 200 mg/dL (11.1 mmol/L)
FPG ≥ 126 mg/dL (7.0 mmol/L). Fasting defined as no caloric intake for at least 8 h ^a
2 h plasma glucose ≥ 200 mg/dL (11.1 mmol/L) during an OGTT The test should have a glucose load containing the equivalent of 75 g anhydrous glucose dissolved in water ^a
A1c $\geq 6.5\%$ ^a

Adapted from Ref. 7

FPG fasting plasma glucose, OGTT oral glucose tolerance test

^aConfirmatory testing may be needed for patients without unequivocal hyperglycemia

that demonstrates plasma glucose above 200 mg/dL (11.1 mmol/L) is considered positive for diabetes mellitus [7] (Table 71.1).

71.1.6 Evaluation and Treatment

Exercise is an important component of glucose regulation in both type 1 and type 2 DM as skeletal muscle is critical to control plasma glucose levels. Skeletal muscle serves as the main site of glucose utilization during insulin-simulated conditions. Basketball is considered a high-intensity exercise and basketball athletes use a combination of aerobic and anaerobic energy systems to fuel the muscles. Glucose, derived from either muscle or hepatic gluconeogenesis, is required for the high-intensity exertion [10, 11].

Current recommendations for the management of basketball athletes with DM include pre-participation blood glucose testing. If blood glucose readings are between 100 and 250 mg/dL, it is generally safe to initiate exercise. Many athletes strive for pre-exercise glucose of 180 mg/dL. Blood glucose should be monitored closely during exercise by checking levels every 30 min or if any symptoms of hypoglycemia exist. Signs of hypoglycemia such as hunger, anxiety, sweating, tremor, palpitations, or feeling of impending doom [10] may be hard to identify as they may mimic normal response to increased exercise [10]. Advanced symptoms of weakness or fatigue, speech changes, lack of coordination, vision changes, odd behavior or confusion may

be the first signs that are recognized by the athlete, teammates, or medical staff [10].

For athletes with pre-exertion blood glucose levels above 250 mg/dL, urine should be screened for the presence of ketones. If ketones are absent, it is generally safe to participate, even if the blood glucose is above 300 mg/dL, extra caution should be used, including glucose monitoring every 15 min [10, 11]. If ketones are present in the urine, exercise should be avoided and

increased hydration should ensue. Once ketosis is resolved, exercise can be initiated, but close monitoring of both glucose and ketones is important [11]. See Fig. 71.1 for pre-exercise glucose testing algorithm.

The mainstay of treatment for type 1 DM is exogenous insulin. There are several different insulin types and delivery methods that basketball athletes may use. Table 71.2 lists insulin types and their general length of action. Adjusting

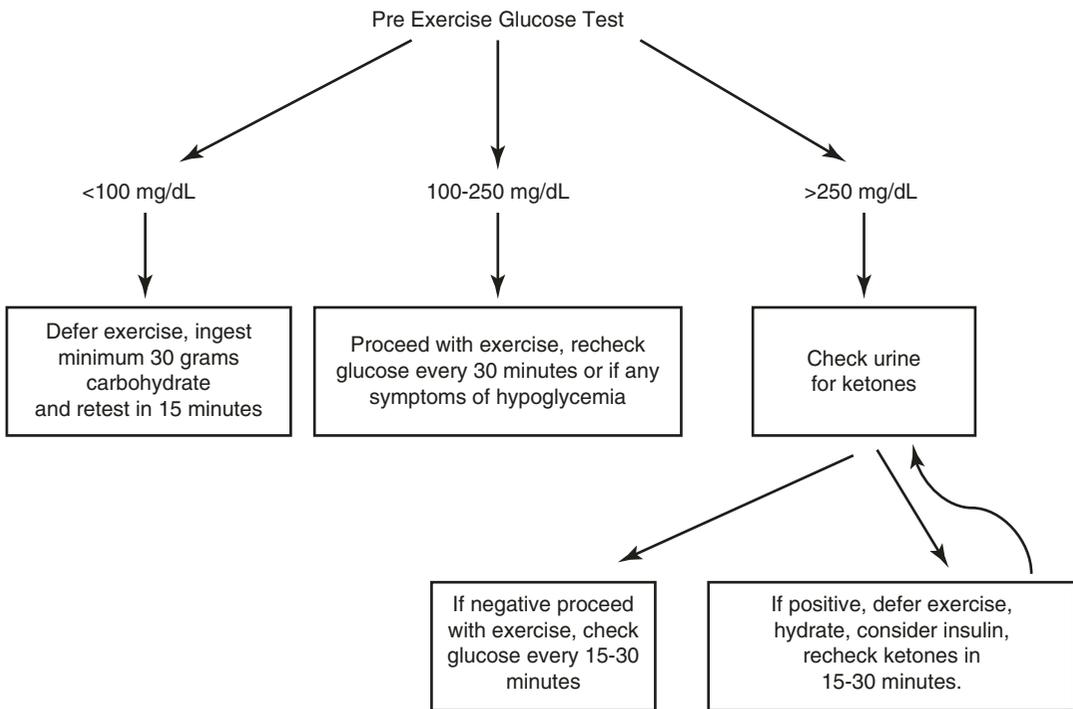


Fig. 71.1 Pre-exercise testing and intervention algorithm

Table 71.2 Insulin types and their general length of action

Insulin type	Approximate onset of action (h)	Approximate peak of action (h)	Approximate duration of action (h)
Ultra-rapid (faster aspart)	<0.1–0.2	1–3	3–5
Rapid acting (aspart, glulisine, lispro)	0.15–0.35	1–3	3–5
Short acting (regular)	0.5–1	2–4	5–8
Intermediate acting (NPH)	2–4	4–10	10–24
Long acting	2–4	4–7	12–24
Detemir	2–4	Varies (minimal peak)	22–24
Glargine	2–6	Varies (minimal peak)	30–36
Glargine U300	0.5–1.5	Varies (minimal peak)	>42
Degludec			
Mix insulin (dual action) (lispro protamine/lispro, aspart protamine/aspartate, NPH/regular)	0.15–1 (varies on individual ingredient)	1–10 (varies on individual ingredient)	10–24 h

insulin doses, both basal and bolus, is important to offset changes in exertion throughout the season. These alterations must be individualized for each athlete, but a decrease of 30–50% of the short acting with the meal preceding completion may be needed. The amount of decrease may also change as an athlete's fitness changes [11]. A key component of insulin administration in the basketball athlete is the location of the injection. It is important to avoid injecting insulin into the site of exercising muscles or overlying subcutaneous tissue as there may be increased absorption leading to a risk of hypoglycemia [10]. In the basketball athlete, that would mean avoiding injection into the legs or arms, preferentially using the abdomen or low back. Some athletes prefer to use insulin pumps for their DM management. The technology for pumps and continuous glucose monitoring is rapidly advancing. There are no specific rules regarding insulin pumps and continuing monitoring devices, though athletes often disconnect their pump and use as needed insulin injections during practice and competition [10].

Exercise, including basketball activities, is the most important treatment for type 2 DM. Some athletes may be able to treat their type 2 DM with diet and exercise alone. For those on oral or injectable non-insulin medications, a dose reduction of their medication may be needed on days of intense exercise to help decrease the risk of hypoglycemia.

Acute hypoglycemia and late-onset post-exercise hypoglycemia (LOPEH) are specific and real risks for basketball athletes with diabetes. LOPEH can present 6–12 h (and up to 28 h) after the completion of sport or exercise. LOPEH occurs because of both increased serum glucose utilization to replenish liver and muscle glycogen stores and due to increased insulin sensitivity [11]. This phenomenon can be dangerous if it occurs while the athlete is sleeping. This condition can be associated with seizures, arrhythmias, and even death [10]. Post-exercise calorie intake is important to prevent hypoglycemia. Combining carbohydrates with fat and protein can help prevent LOPEH [11]. Treating hypoglycemia early is important to prevent an emergency situation.

If an athlete should become unresponsive due to hypoglycemia, the use of parenteral glucagon or intravenous dextrose should be employed [10]. Any athletic trainer or team physician caring for a basketball athlete who uses exogenous insulin should have access to these potentially life-saving medications.

71.1.7 Conclusion

The burden of diabetes across the globe is increasing, and basketball athletes are at risk for the condition. It is important that the diabetic athlete and the healthcare providers for the athlete understand the individualized response to exertion and nutrition. Knowing the pre-exertion glucose levels and warning signs for hypoglycemia is key to preventing dangerous situations.

71.2 Allergies

- Up to 50% of high-level athletes are sensitive to inhalant allergens.
- Food allergies and exercise-induced anaphylaxis are less common, but providers must be aware of the potential for these conditions.
- Avoid exposures is the most important treatment, but several medications are available to treat the symptoms of allergen exposure.

71.2.1 Introduction

Basketball athletes are susceptible to the same potential for exposure to allergens as the general public. With the addition of travel for sport, basketball athletes may be exposed to more or different allergens than those who have less frequent travel. The symptoms that go along with allergen exposure may adversely impact the basketball athlete's ability to perform their tasks.

71.2.2 Epidemiology

Allergic responses are fairly common in the general population. Up to 30% of individuals in Europe and the United States suffer from allergic rhinitis [12]. Studies of Olympic athletes have shown rates of positive skin prick testing as high as 41% [13]. Another study of Olympic athletes showed that as many as 49% had sensitization to inhalant allergens and that allergies to food, drugs, and venom were also present in approximately 7%, 5%, and 2%, respectively [14].

71.2.3 Pathophysiology

Though there are several mechanism that can lead to an allergic response, the most common is immunoglobulin E (IgE)-mediated histamine and leukotriene release from mast cells and basophiles. There are early and late phase components of an allergic response, each of which can impact a basketball athlete's well-being [15]. More severe allergic reactions can lead to anaphylaxis through similar IgE-mediated release of histamine, though IgE-independent anaphylaxis also exists [16].

71.2.4 Presentation

The presentation of someone with an allergic reaction depends on the type of allergy. The most common allergy presentation is allergic rhinitis. This presents as nasal congestion, runny nose, and possibly ocular involvement. It may present however as decreased energy, breathing abnormalities, or poor athletic performance [13]. This may also lead to breathing disturbances such as asthma (discussed elsewhere in this chapter) and other allergic symptoms such as itching, flushing, or cough. Severe allergic reactions can lead to an athlete having angioedema, stridor, hypotension, cardiac manifestations, or gastrointestinal symptoms, all of which are possible symptoms of anaphylaxis [16].

71.2.5 Diagnosis

A validated screening tool for allergies in athletes was developed and called the AQUA: Allergy

Questionnaire for Athletes. This tool was found to be highly specific in the athletic population and is available in at least ten European languages [17]. This questionnaire can be used to screen basketball athletes for allergens that may impact their play. The investigation of allergic rhinitis may involve a nasal allergen provocation test. If this is positive, the gold standard skin prick test or a serum-specific IgE test can be used to confirm a diagnosis. A subset of allergic rhinitis may be diagnosed with nasal-specific IgE or with a basophil activation test [12]. For other (non-rhinitis) allergic conditions, skin prick testing or blood testing for specific IgE is the test of choice [18].

71.2.6 Treatment Options

The most important intervention for the treatment of allergic reactions in basketball athletes is avoidance of triggers. This can be exceedingly challenging with travel, the multiple venues that athletes may play or practice in, and the ubiquity of allergens in the environment. Knowing what triggers an athlete helps guide the practice of avoidance. If avoiding these triggers is insufficient for symptom control, then medications are needed and available [13]. Medications that target the various physiologic pathways are available. Table 71.3 describes classes of medication available. In

Table 71.3 Medication options for allergies and allergic reactions

Class	Delivery routes	WADA regulations
Anti-histamine	PO, IN, Opt	Permitted
Decongestant	PO, IN	Varies, some need TUE
Anticholinergics	IN	Permitted
Leukotriene inhibitors	PO	Permitted
Mast cell stabilizers	IN, Opt, PO	Permitted
Corticosteroid	PO, IN, IV, IM, Opt	Requires TUE
Allergen immunotherapy	PO, IM, SC	Permitted
Epinephrine	IM	Requires TUE

the case of allergic rhinitis, decongestants may be used. In addition, local and systemic steroid may be used if symptoms are severe enough. Prescribers should be aware that systemic steroids and some decongestants are found on the therapeutic use exemption (TUE) list for the World Anti-doping Agency (WADA) [13]. Providers should review league-specific TUE lists. If a basketball athlete has an anaphylaxis-inducing allergy, it is important that epinephrine be available for administration if an exposure occurs.

71.2.7 Special Circumstances

Food allergies seem to be increasing in prevalence, and since they are seen in people of all ages, ethnicities and heritages, food allergies are likely to be seen in some basketball athletes [19]. This may be a challenge due to travel and the inconsistent dining options. It is important for the basketball athlete to be aware of ingredients in order to avoid exposure to allergens. Desensitization and food oral immunotherapy are treatment options to decrease the body's response to allergens and allow for consumption of foods [19]. Consultation with an allergy specialist is recommended if this is considered.

Exercise-induced anaphylaxis (EIA) is a rare condition that results in an anaphylactic reaction as a result of exertion. This can present after years of previous exertion with no symptoms. A subtype of EIA is food-dependent exercise-induced anaphylaxis (FDEIA), which occurs when an athlete exerts within hours of eating. The diagnosis of EIA is usually made clinically and after ruling out conditions such as mastocytosis. An exercise challenge test may be used to assist in diagnosis. Given the potential danger with this condition, athletes with a history of this should have access to epinephrine and should have a detailed action plan when resuming exercise [20].

71.3 Asthma

- Exercise-induced bronchospasm is a common condition in athletes both with and without asthma.
- It is important to mitigate environmental triggers as well as utilize proper pharmacological tools to treat EIB.
- Inhaled short-acting beta agonist is the standard treatment for EIB, but once daily use is necessary, then Inhaled corticosteroid is preferred.

71.3.1 Introduction

Shortness of breath, wheezing, and cough are commonly experienced during exercise. Among athletes who suffer from these conditions, it is important to distinguish when the symptoms meet pathologic criteria. Asthma includes several phenotypic disorders that share a common pathway characterized by airway inflammation, bronchial hyperactivity, and reversible bronchoconstriction [21]. In this chapter, we are going to focus on the transient airway narrowing that occurs as a result of exercise, termed exercise-induced bronchoconstriction (EIB). This has most recently been delineated into EIB with asthma (EIBA), referring to exercise-induced bronchoconstriction with clinical symptoms of asthma and EIB without asthma (EIBwa), referring to acute bronchoconstriction without asthmatic changes in the airway [22].

71.3.2 Etiology

The etiology of EIB has not been established with certainty but is thought to relate to airway cooling and airway dehydration resulting in augmented osmolarity of the airway-lining fluid [23]. Mediators are released from airway inflamma-

tory cells, which lead to smooth-muscle contraction and airway edema [24]. It is also thought that mechanical and osmotic stress over time can lead to remodeling, therefore putting even the well-trained athlete at risk for EIB. Environmental factors such as air temperature, moisture, and quality play a role in the incidence of EIB. Nordic and ice rink athletes have the highest prevalence of asthma due to the cold, dry air, and emission of pollutants from ice resurfacing machines. When basketball teams share facilities with ice hockey, the players would likely be exposed to similar elements.

71.3.3 Prevalence

The prevalence of EIB in the general population ranges from 5% to 20% and has been reported in up to 90% of those with a current diagnosis of asthma (EIBA). EIB is more likely to occur in those whose asthma is severe and uncontrolled, those with recent respiratory infections, and those with allergic rhinitis [25]. The prevalence of EIB in athletes has shown to be even greater at 23–55% [24]. Endurance athletes are the most likely to experience EIB. However, a study done through the Temple Sports Asthma Research Program looked at asthma deaths over the course of 7 years, and the results showed basketball and track to be the two most frequent activities performed at the time of fatal event [26].

71.3.4 Clinical Presentation

Athletes with EIB often report wheeze, cough, dyspnea, chest tightness, and fatigue with exercise. Symptoms are worse 5–30 min after initiating exercise and will often resolve 30–60 min after stopping exercise. These symptoms can also occur with other conditions such as exercise-induced laryngeal obstruction (EILO), cardiovascular causes, and anxiety. It is important to differentiate these diagnoses as many not only

have a negative effect on performance but can also be life-threatening.

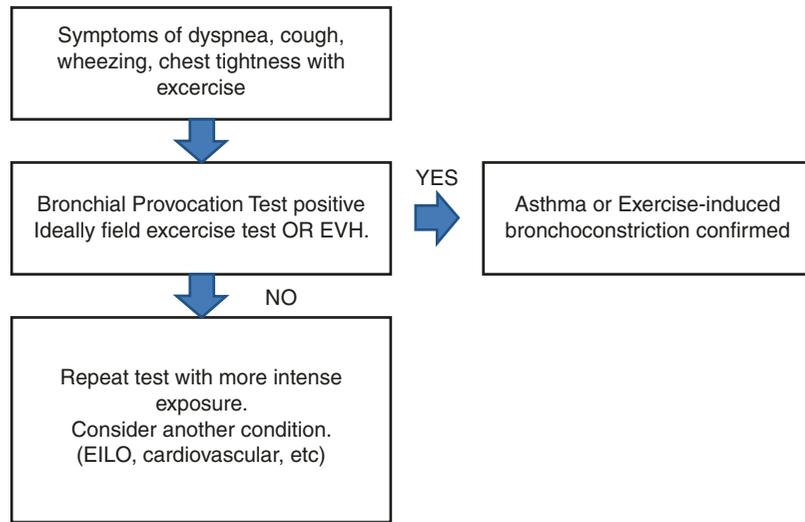
71.3.5 Diagnosis

When an athlete shows signs of EIBwA or EIBA, the diagnosis can be confirmed with bronchial provocation tests (BPT). BPTs use either a direct method (a nebulized drug to stimulate smooth muscle) or an indirect method (attempts to dehydrate the mucosa) to cause a bronchial challenge. Spirometry is measured at baseline and then typically at 5, 10, 15, and 20 min after the stimulus. A drop in the forced expiratory volume in 1 s (FEV1) of >10–15% with associated dyspnea is considered diagnostic for EIB [27]. The preferred methods for diagnosing EIB include exercise, inhaled mannitol, and eucapnic voluntary hyperpnoea (EVH). If an athlete tests negative, but there is a high suspicion of EIB, it is most appropriate to repeat or administer an alternative test [28]. Figure 71.2 shows a suggested algorithm for evaluation of symptomatic athletes. Studies have shown that patient report of symptoms alone is not accurate to diagnose EIB. Though it is a common practice to attempt a trial of B-adrenergic agonist for improvement of symptoms during exercise, this is not acceptable for the diagnosis of EIBA or EIBwA to obtain a therapeutic use exemption (TUE) in national and international competitions [21]. If the BPTs are negative, it is important to consider other possible diagnoses through cardiovascular testing or continuous laryngeal endoscopy.

71.3.6 Management

The management of EIB highlights the variability of the condition. It is important to consider each individual, their triggers, severity of disease, and chronic management. When treating EIB, it is necessary to first assess the severity of the condition. If the player has signs and symptoms of a

Fig. 71.2 Suggested diagnosis pathway of asthma or EIB in an athlete



moderate to severe asthma exacerbation or acute hyperreactive airway, then an emergency action plan should be implemented to transport them to the nearest hospital. If the athlete is experiencing or is known to experience EIB with mild symptoms, then it is important to utilize both non-pharmacologic and pharmacologic measures. One of the most important steps in preventing EIB is a warm up. There have been studies on varied warm-up styles, and though the evidence on specifics is inconclusive, there is typically a 1–3 h refractory period after a 15 min warm-up. During this time, the airways respond with relatively normal reactivity [29]. When considering environmental triggers, it is best to keep the air warm, moist, and void of pollutants as much as possible. Some current areas of research include the use of fish oil and vitamins to limit airway hyperreactivity, but currently, there is no strong evidence for their use [24]. Many pharmacological approaches can be used in treating EIB. The key to prevention of EIBA in the asthmatic population is to maintain control of their asthma utilizing the most recent guidelines [30]. The most commonly used medication to treat EIB is an inhaled short-acting B₂-agonist (SABA) taken 20–30 min prior to activity. However, with frequent use, SABAs have decreasing effectiveness. Once a daily medication is required for management, it is best to use an inhaled glucocorticoid. Leukotriene-receptor antagonists are also an

option for maintenance treatment. If symptoms remain uncontrolled, an inhaled long-acting beta-agonist is the next step [24]. If an athlete is requiring these medications for EIB, then it is likely that he or she has uncontrolled asthma. A major consideration in treating elite basketball players with these medications is World Anti-Doping Agency compliance. Currently, all B₂-agonists are prohibited except salbutamol, formeterol, and salmeterol in limited doses and in accordance with the relevant section of the international standard for therapeutic use exemption (TUE) [31]. Ultimately, it is best for each player who has known EIBwA or EIBA to have an action plan in place and the appropriate rescue medications close at hand to maintain respiratory control.

71.4 Dermatologic Issues

- Basketball players are susceptible to infectious as well as traumatic skin conditions.
- It is important to treat CA-MRSA concerning skin conditions with drainage and appropriate antibiotics as indicated.
- Consider further investigation into sanitizing procedures for basketball shoes and equipment to prevent colonization.

71.4.1 Introduction

There are multiple dermatologic issues to consider when evaluating and caring for a basketball player. Since basketball is a contact sport, it is important to be aware of infectious skin conditions. This includes not only the bacterial skin infections but also the fungal infections. The quick start-stop action on the basketball court predisposes its players to traumatic skin conditions. Finally, though less common, the equipment worn in basketball has the potential to cause skin issues. This section will address a variety of skin conditions as they are considered in the above categories.

71.4.2 Infectious Skin Conditions

When evaluating bacterial skin infections in athletes, it is important to consider the causative organism. The most common infectious agents are *Staphylococcus aureus* and group A streptococcus (GAS) [32]. However, athletes are at high risk for community-acquired methicillin-resistant *Staphylococcus aureus* (CA-MRSA) due to their close contact and sharing of equipment. A systematic review done by Karanika et al. showed that 6% of asymptomatic athletes were colonized with MRSA. Colonization with MRSA means that a person carries the bacteria, and it has been shown that a carrier has a sevenfold increase in incidence of MRSA infection. Among collegiate athletes, basketball was in the top 3 sports for MRSA colonized athletes [33]. If there is concern for a bacterial skin infection, it is ideal to obtain a culture and sensitivity in order to direct antibiotic selection when necessary. However, if there is no access to obtain this objective information, a clinical judgment based on severity of infection and likelihood of contact could direct treatment toward CA-MRSA coverage. A furuncle, or abscess, is a common skin condition in athletes (Picture 71.1). This often presents with the athlete complaining of a pimple that is painful, enlarging, and possibly draining. The treatment for this condition is drainage, so if warm compresses have not provided adequate drain-



Picture 71.1 Furuncle [34]

age, then a physician must lance it. Antibiotics are necessary if there is surrounding induration and erythema concerning for cellulitis, the athlete is immunocompromised, or the location of the furuncle is in a concerning area such as the face. If a culture is obtained and CA-MRSA is a concern, then the antibiotics should provide adequate coverage. At this time, there is no specific decontamination protocol for the athlete or equipment other than to wash equipment separately from the rest of the teams [21]. Contact should be avoided with other players and equipment. The athlete can return to play when the lesion is dry with no discharge [35]. Fungal infections are most common in areas of friction leading to a warm, moist environment. Basketball players are at certain risk for these given the often-daily workouts, shared locker room, and shower equipment. Tinea can occur in a variety of locations. It is the medical term used to describe these infections caused by dermatophytes. It can occur in a variety of locations such as on the scalp (tinea capitis), on the body (tinea corporis), in the groin (tinea cruris), or on the feet [32]. Tinea pedis, or athlete's foot, is commonly seen in basketball players and presents with gradual onset of pain, itch, and rash on the feet (Picture 71.2). The evaluation will reveal erythematous, scaling eruptions on the plantar surface of the foot and between the toes. Skin KOH scrapings or fungal cultures can be used to diagnose the condition, but clinical presentation is often enough to reach a diagnosis. The treatment is typically topical with butenafine or terbinafine. These preparations must be used twice



Picture 71.2 Tinea Pedis [34]



Picture 71.3 Pitted Keratolysis [34]

daily for, typically, 3 weeks or until 1 week after the rash has resolved [16]. It is important to keep footwear clean and dry. Though there is no standard recommendation for footwear sanitization, a study was done with the 2012 Sana Men's basketball team by Messina et al. which showed a sanitizing process of the players' shoes significantly reduced bacterial presence [36].

Pitted keratolysis (PK) is a bacterial infection of the stratum corneum on the plantar surface of the foot (Picture 71.3). This is typically caused by a gram-positive organism [37]. This condition presents with foul-smelling feet and depressions on the sole of the foot that has been present for months to years. There has yet to be good evidence for a treatment regimen. Topical treatment with 20% aluminum chloride or benzoyl peroxide twice daily may be useful [35]. The most widely

studied treatment is with topical antibiotics such as clindamycin and mupirocin [37]. Ultimately it is most important to keep the feet clean and dry. Athletes may return to play without risk of infecting others.

71.4.3 Traumatic Skin Conditions

It is no surprise that basketball players can experience traumatic injury to the skin of their feet. Yan Cong et al. looked at the in-shoe tri-axial stress profiles during maximum effort cutting maneuvers. Their results showed increased stress from cutting and shuffling to occur over the first and second metatarsal heads. This information could be utilized to help appropriate cushioning mechanisms in basketball footwear [38].

Piezogenic pedal papules are small herniation of subcutaneous tissue into the mediolateral surface of the midfoot (Picture 71.4). These can occur because of increased pressure and shifting on the foot. These dome-shaped papules appear when pressure is applied to the sole of the foot. They can be asymptomatic or painful enough to preclude activity. Treatment consists of behavioral medication such as limited pressure on the feet, orthotics, compression socks, and heel cups. Intralesional steroid injections and surgical resection are treatment options for recalcitrant cases [40, 41].

Talon noir, or black heel, is caused by petechial on the heel from lateral shearing forces of



Picture 71.4 Piezogenic Pedal Papule [39]

quick starting and stopping. These small hemorrhages are typically not painful. They can resemble acral melanoma, so this should be considered in the differential diagnosis and may be ruled out with dermatoscopic evaluation. Treatment for this ‘black dot syndrome’ is to ensure properly fitted shoes and use of heel pads or a thick sock. The athlete can return to play without restriction [35, 41].

Blisters are very common in basketball players due to the shearing forces on the feet. These blisters will often present as painful, fluid-filled bullae formed at the site of friction. If not cared for properly, blisters run a risk of becoming infected, leading to a more serious issue. There is some evidence to drain the blister in a sterile manner, leaving the roof of the blister in place as this may reduce the risk of infection as well as provide the most relief for the athlete. There are multiple remedies to assist in treatment of blisters including protective socks, Vaseline, and mole skin. Antibiotic ointment should only be used if the lesion is open [35, 42].

Take Home Message

Basketball athletes can experience the same medical issues that impact the general population. It is important to keep a high index of suspicion for medical issues if a player’s wellness or performance should suffer. An athlete diagnosed with diabetes mellitus can still participate at a high level with appropriate pre-exercise screening and treatment. Basketball athletes with allergies should avoid offending allergens and, when unable to do so, can use medications to decrease symptoms. It is important to consult WADA or local therapeutic use exemption lists when prescribing a medication. Appropriate testing for and treatment of asthma and exercise induced bronchospasm can allow basketball athletes to perform at their highest level. Finally, early recognition and treatment of dermatologic conditions can allow basketball athletes to continue without missing time from their sport.

References

1. D’Arrigo T. Hot shot. NBA rookie Adam Morrison scores with control. *Diabetes Forecast*. 2007;60(2):42–4.
2. Neithercott T. Forward motion the NBA’s Gary Forbes scores on his dreams. *Diabetes Forecast*. 2012;65(3):40–3.
3. Ogurtsova K, da Rocha Fernandes JD, Huang Y, Linnenkamp U, Guariguata L, Cho NH, et al. IDF diabetes atlas: global estimates for the prevalence of diabetes for 2015 and 2040. *Diabetes Res Clin Pract*. 2017;128:40–50. <https://doi.org/10.1016/j.diabres.2017.03.024>.
4. American Diabetes Association. Classification and diagnosis of diabetes: standards of medical Care in Diabetes—2018. *Diabetes Care*. 2018;41(Suppl 1):S13–27. <https://doi.org/10.2337/dc18-S002>.
5. Xu G, Liu B, Sun Y, Du Y, Snetselaar LG, Hu FB, et al. Prevalence of diagnosed type 1 and 2 diabetes among US adults in 2016 and 2017: population based study. *BMJ*. 2018;362:k1497. <https://doi.org/10.1136/bmj.k1497>.
6. Hamman RF, Bell RA, Dabelea D, D’Agostino RB Jr, Dolan L, Imperatore G, et al. The SEARCH for diabetes in youth study: rationale, findings and future directions. *Diabetes Care*. 2014;37(12):3336–44. <https://doi.org/10.2337/dc140574>.
7. American Diabetes Association. Diagnosis and classification of diabetes mellitus. *Diabetes Care*. 2012;36(Suppl 1):S67–74. <https://doi.org/10.2337/dc13-S06>.
8. Umpierrez G, Korytkowski M. Diabetic emergencies—ketoacidosis, hyperglycaemic, hyperosmolar state and hypoglycaemia. *Nat Rev Endocrinol*. 2016;12(4):222–32. <https://doi.org/10.1038/nrendo.2016.15>.
9. Smith LL, Burnet SP, McNeil JD. Musculoskeletal manifestations of diabetes mellitus. *Br J Sports Med*. 2003;37(1):30–5. Review
10. Harris GD, White RD. Diabetes in the competitive athlete. *Curr Sports Med Rep*. 2012;11(6):309–15. <https://doi.org/10.1249/JSR.0b013e3182751007>.
11. Horton WB, Subauste JS. Care of the Athlete with Type 1 diabetes mellitus: a clinical review. *Int J Endocrinol Metab*. 2016;14(2):e36091. <https://doi.org/10.5812/ijem.36091>.
12. Campo P, Eguiluz-Gracia I, Bogas G, Salas M, Plaza Serón C, Pérez N, et al. Local allergic rhinitis: implications for management. *Clin Exp Allergy*. 2019;49(1):6–16. <https://doi.org/10.1111/cea.13192>.
13. Steelant B, Hox V, Hellings PW, Bullens DM, Seys SF. Exercise and Sinonasal disease. *Immunol Allergy Clin N Am*. 2018;38(2):259–69. <https://doi.org/10.1016/j.iac.2018.01.014>.
14. Bonini M, Gramiccioni C, Fioretti D, Ruckert B, Rinaldi M, Akdis C, et al. Asthma, allergy and the Olympics: a 12-year survey in elite athletes. *Curr Opin Allergy Clin Immunol*. 2015;15(2):184–92. <https://doi.org/10.1097/ACI.000000000000149>.

15. Bjermer L, Westman M, Holmström M, Wickman MC. The complex pathophysiology of allergic rhinitis: scientific rationale for the development of an alternative treatment option. *Allergy Asthma Clin Immunol.* 2019;15:24. <https://doi.org/10.1186/s13223-018-0314-1>.
16. Reber LL, Hernandez JD, Galli SJ. The pathophysiology of anaphylaxis. *J Allergy Clin Immunol.* 2017;140(2):335–48. <https://doi.org/10.1016/j.jaci.2017.06.003>.
17. Bonini M, Braido F, Baiardini I, Del Giacco S, Gramiccioni C, Manara M, et al. AQUA: allergy questionnaire for athletes. Development and validation. *Med Sci Sports Exerc.* 2009;41(5):1034–41. <https://doi.org/10.1249/MSS.0b013e318193c663>.
18. Önell A, Whiteman A, Nordlund B, Baldracchini F, Mazzoleni G, Hedlin G, et al. Allergy testing in children with persistent asthma: comparison of four diagnostic methods. *Allergy.* 2017;72(4):590–7. <https://doi.org/10.1111/all.13047>.
19. Grief SN. Food Allergies. *Prim Care.* 2016;43(3):375–91. <https://doi.org/10.1016/j.pop.2016.04.008>.
20. Feldweg AM. Exercise-induced anaphylaxis. *Immunol Allergy Clin N Am.* 2015;35(2):261–75. <https://doi.org/10.1016/j.jac.2015.01.005>. Epub 2015 Feb 28
21. Preparticipation physical evaluation (2019). 5th edn. Itasca, IL: American Academy of Pediatrics.
22. Del Giacco SR, et al. Exercise and asthma: an overview. *Eur Clin Respir J.* 2015;2:27984.
23. Anderson SD, Kippelen P. Exercise-induced bronchoconstriction: pathogenesis. *Curr Allergy Asthma Rep.* 2005;5(2):116–22.
24. Boulet LP, O'Byrne PM. Asthma and exercise-induced bronchoconstriction in athletes. *N Engl J Med.* 2015;372(7):641–8.
25. Bonini M, Silvers W. Exercise-induced bronchoconstriction: background, prevalence, and sport considerations. *Immunol Allergy Clin N Am.* 2018;38(2):205–14.
26. Becker JM, et al. Asthma deaths during sports: report of a 7-year experience. *J Allergy Clin Immunol.* 2004;113(2):264–7.
27. Smoliga JM, Weiss P, Rundell KW. Exercise induced bronchoconstriction in adults: evidence based diagnosis and management. *BMJ.* 2016;352:h6951.
28. Anderson SD, et al. Reproducibility of the airway response to an exercise protocol standardized for intensity, duration, and inspired air conditions, in subjects with symptoms suggestive of asthma. *Respir Res.* 2010;11:120.
29. Elkins MR, Brannan JD. Warm-up exercise can reduce exercise-induced bronchoconstriction. *Br J Sports Med.* 2013;47(10):657–8.
30. Global Initiative for Asthma: Asthma Management and Prevention (2019). <https://ginasthma.org/wp-content/uploads/2019/04/GINA-2019-main-Pocket-Guide-wms.pdf>.
31. Fitch KD, et al. Asthma and the elite athlete: summary of the International Olympic Committee's consensus conference, Lausanne, Switzerland, January 22–24, 2008. *J Allergy Clin Immunol.* 2008;122(2):254–60, 260.e1–7
32. Pecci M, Comeau D, Chawla V. Skin conditions in the athlete. *Am J Sports Med.* 2009;37(2):406–18.
33. Karanika S, et al. Colonization with methicillin-resistant *Staphylococcus aureus* and risk for infection among asymptomatic athletes: a systematic review and Metaanalysis. *Clin Infect Dis.* 2016;63(2):195–204.
34. Images Courtesy of the Wake Forest University School of Medicine Graham Library.
35. Madden CC. Netter's sports medicine. In: Netter clinical science. Philadelphia: Saunders/Elsevier; 2010. p. 732.
36. Messina G, et al. Is it possible to sanitize Athletes' shoes? *J Athl Train.* 2015;50(2):126–32.
37. Bristow IR, Lee YL. Pitted keratolysis: a clinical review. *J Am Podiatr Med Assoc.* 2014;104(2):177–82.
38. Cong Y, et al. In-shoe plantar tri-axial stress profiles during maximum-effort cutting maneuvers. *J Biomech.* 2014;47(16):3799–806.
39. Image Courtesy of Dr. Neil Sparks, DO.
40. Brown F, Cook C. Piezogenic Pedal Papule. Treasure Island (FL): StatPearls; 2019.
41. De Luca JF, Adams BB, Yosipovitch G. Skin manifestations of athletes competing in the summer Olympics what a sports medicine physician should know. *Sports Med.* 2012;42(5):399–413.
42. Trojian TH, et al. Basketball injuries: caring for a basketball team. *Curr Sports Med Rep.* 2013;12(5):321–8.



Is Overload an Issue in Young Basketball Players?

72

Andrew Gregory

72.1 Introduction

It is well accepted that practice and training are necessary for proficiency in sport just like in other activities such as music. The 10,000 hour threshold that was found in a study of musicians is a number that often gets applied to athletes, but we really do not know that the same amount of training is necessary for sports. So, if some training is necessary but too much training can lead to injury, then what we really need to know is how much training is sufficient without putting young athletes at risk for injury.

72.2 Load

In physiologic terms, the internal load is defined as the body's response to an external load that is applied to it [1]. External load can be broken down into multiple components including sets, repetitions, resistance, speed, range of motion, rest, and frequency [2]. A progression of loads refers to increasing external loads in order to

increase internal loads, which then leads to adaptations [3]. Three common strategies used to increase external load are increasing volume, intensity, and/or density. Complexity has recently been introduced and used as an additional strategy to increase external load [4].

72.3 Overload

The term overload has multiple meanings in sports medicine. In terms of physical training, overload refers to the progression of external loads to obtain the wanted adaptation and so is considered an important part of training. However, in sports medicine it also refers to a condition of too much training that can lead to overuse injury, overtraining syndrome, and burnout.

Overuse injuries refer to injuries that occur gradually over time as a result of the body not being able to withstand the repetitive stress of training. About 50% of all injuries in young athletes are overuse injuries. The real challenge is trying to determine how much stress the body can withstand without sustaining injury. There are overuse injuries that are common in adult basketball players such as tendinopathy and those that are unique to young basketball players such as apophysitis. Theoretically, all overuse injuries could be prevented by avoiding overload.

Overtraining syndrome is a vague condition that can occur when intense training leads to a

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prolonged performance decrement as well as severe physiologic and neuroendocrine symptoms [5]. Overtraining syndrome can occur in young athletes just like adults but is even more challenging to diagnose. The exact pathophysiology of overtraining syndrome is unknown and should be considered a diagnosis of exclusion. Burnout refers to when the athlete stops participating in a previously enjoyable activity because of chronic stress and lack of enjoyment [6].

72.4 Recovery

Recovery is the time and process after a given load in which the body responds to that load. Inadequate recovery does not allow the body the time to adapt to the load and can result in impaired performance, injury, and illness [7]. Important parts of recovery include treatment of muscle soreness, rest, hydration, nutrition, and sleep. Adequate recovery is important for all athletes, but young athletes may require more, like sleep for instance, than their adult counterparts. Children of 6–12 years of age should sleep 9–12 h per 24 h, and teenagers of 13–18 years of age should sleep 8–10 h per 24 h on a regular basis to promote optimal health [8].

Fact Box 1

Younger basketball players need more sleep than older ones.

72.5 Specialization

Clinicians have long suspected that early specialization puts young athletes at risk for overuse injuries, overtraining, and burnout. Evidence now exists to support that assertion although it has yet to be studied in the sports of basketball [9]. Specialization refers to the concept of focusing intensely on a single sport year-round at the exclusion of others. The

Table 72.1 Practical recommendations for prevention of overuse injuries in young basketball players

Do not specialize until puberty
Limit number of hours/week to less than age and not more than 16 h
Take 1 day off per week
Take 2 months off per year
Do not play on multiple teams at the same time
Do not do extra training after practice/games
Monitor training workload
Stop training if pain develops and seek medical attention

younger the age that an athlete specializes will increase the exposure to the training specific to that sport and exclude other types of training that may be protective. Certain sports like gymnastics, ice skating, swimming, and diving may require athletes to specialize early for success, but most experts recommend waiting to specialize until puberty [10]. Others have recommended limiting the hours of training per week to less than the age of the athlete and not more than 16 h, i.e., a 12-year-old should practice less than 12 h per week [11]. There are no specific training limit recommendations for youth basketball like there are pitch counts for youth baseball however some basic limitations can be recommended (Table 72.1). Jump counts can be monitored and should be studied in this population.

Fact Box 2

Early specialization is not necessary for success in basketball and may increase risk for overuse injury.

72.6 Overuse Injuries Unique to Young Athletes

Because of the presence of growth plates, young athletes are at risk for different overuse injuries than adults. Where patellar tendinopathy is a common problem in adult basketball

Table 72.2 Common sites for apophysitis in young basketball players

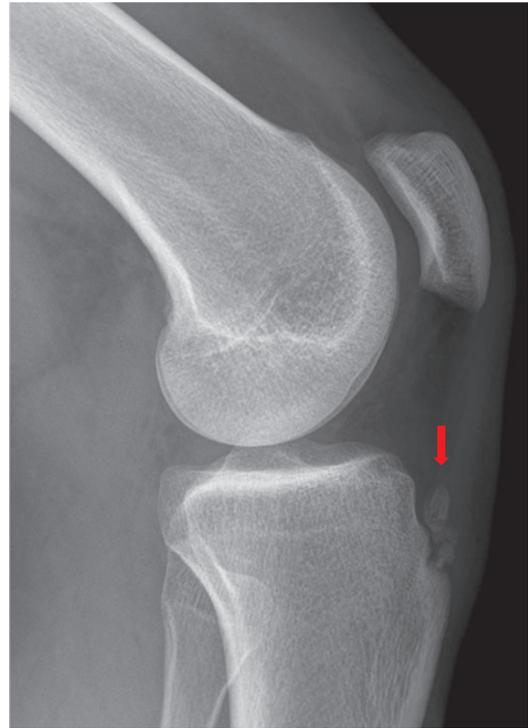
Pelvic rim (iliac crest, anterior-superior, or inferior iliac spine), ischial tuberosity	Yet Unnamed disease
Patella (superior or inferior)	Sinding–Larsen–Johansson’s disease
Tibial tubercle	Osgood–Schlatter’s disease
Calcaneus	Sever’s disease
Fifth metatarsal	Iselin’s disease

players, tibial tubercle apophysitis (Osgood–Schlatter’s disease) is more common in young basketball players. The growth plates are at risk for injury as they are made of cartilage which is weaker in strength than the bone or tendon. Common sites for apophysitis in young basketball players include the pelvic rim, the superior or inferior pole of the patella, the tibial tubercle, the calcaneus, and the fifth metatarsal (Table 72.2).

Fact Box 3

Young basketball players are at risk for unique injuries like apophysitis that older ones are not.

Apophysitis is usually treatable using conservative measures including ice, anti-inflammatory medications, stretches, braces or pads, and relative rest. However, some cases can be severe and significantly limit participation. Once the apophyses fuse at skeletal maturity, the problem usually resolves completely. There does not seem to be an association between apophysitis and avulsion fractures, but there is an association with unfused ossicles that can continue to be symptomatic at skeletal maturity. If the ossicle is symptomatic, then surgical excision is often required for symptom resolution (Picture 72.1: tibial tubercle ossicles).

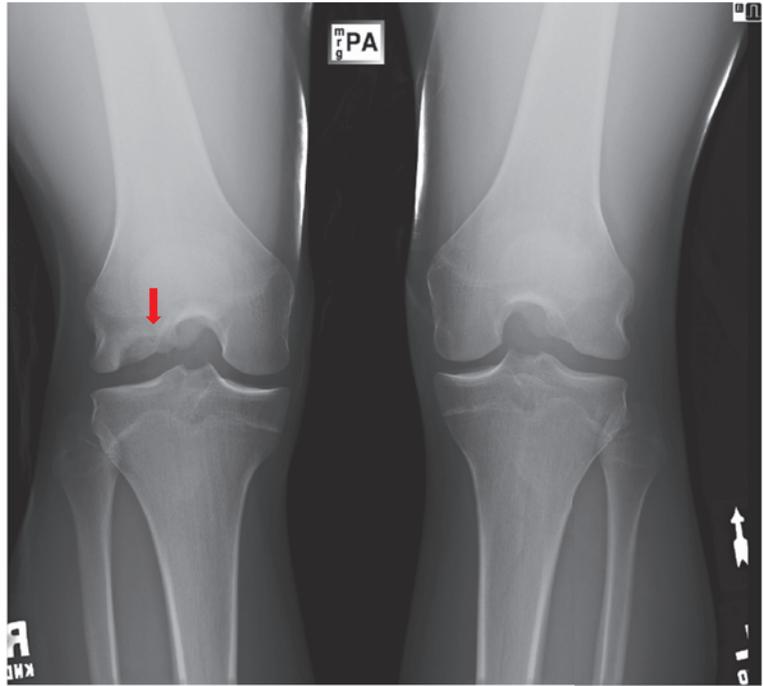
**Picture 72.1** Lateral X-ray: Multiple tibial tubercle ossicles with closed physes

Other injuries unique to young athletes include osteochondritis dessicans, discoid lateral meniscus, tarsal coalition, Os naviculare, spondylolysis, and spondylolisthesis (Pictures 72.2, 72.3, 72.4, 72.5, and 72.6). Several of these conditions were reviewed previously in Chap. 63. None of the conditions are specific to basketball but because of the running and jumping involved may become symptomatic. It is important to be aware of these conditions when treating young athletes as if left untreated can lead to chronic problems such as pain, arthritis or degenerative disc disease.

Fact Box 4

Early detection and proper treatment of overuse injuries can prevent chronic problems.

Picture 72.2 Lateral X-ray—osteochondritis dissecans of the lateral femoral condyle



Picture 72.3 Sagittal T1 MRI—discoid lateral meniscus



Picture 72.4 Oblique X-ray—tarsal (calcaneo-navicular) coalition



Picture 72.5 AP X-ray—Os naviculare (accessory navicular) with closed physes



Picture 72.6 MRI—pars stress fracture (spondylolysis)

72.7 When do Overuse Injuries in Adults Start?

Some overuse injuries that occur in adults may get their start in adolescence. Patellar tendinopathy or jumper's knee may be arguable the most common overuse injury in basketball players. Tendinopathy is a degenerative process that appears to begin as early as adolescence. Mersmann studied the patellar tendons of 16 adolescent elite basketball athletes (14–15 years of age) to determine the mechanical properties with magnetic resonance and ultrasound imaging. There was a significant inverse relationship between tendon strain and peak spatial frequency (PSF) in the proximal tendon region ($r = -0.652$, $p = 0.006$), indicating locally disorganized collagen fascicles in tendons that are subjected to high strain [12].

72.8 Training in Young Basketball Players

We do have some guidance on what amount and type of training works for this specific population. Santos studied the effects of a 10-week resistance training program on explosive strength development in 25 young basketball players aged 14–15 years old. They showed an increased vertical jump and medicine ball throw performance compared with the control group [13]. Asadi studied 16 young basketball players who performed periodized (from 117 to 183 jumps per session) plyometric training for 8 weeks. They showed improved vertical jump (effect size [ES] 2.8), broad jump (ES = 2.4), agility *T*-test (ES = 2.2), Illinois agility test (ES = 1.4), maximal strength (ES = 1.8), and 60-m sprint (ES = 1.6) ($P < 0.05$) compared to the control group [14].

72.9 Summary

It seems apparent that overload issues in young Basketball players maybe even more problematic than in adult basketball players. They are at risk for unique injuries like apophysitis that adults are not. If these injuries are unrecognized or left untreated, they can led to chronic problems. Burnout is another significant concern in this younger age group.

Take Home Message

Load monitoring, setting training limits, ensuring recovery, and seeking appropriate medical attention when indicated are all important measures for injury prevention in this young population.

References

1. Halson SL. Monitoring training load to understand fatigue in athletes. *Sports Med.* 2014;44(Suppl 2):139–47.
2. American College of Sports Medicine Position Stand. Progression models in resistance training for healthy adults. *Med Sci Sports Exerc.* 2009;41(3):687–708.
3. Kraemer WJ, Ratamess NA. Fundamentals of resistance training: progression and exercise prescription. *MSSE.* 2004;36(4):674–88.
4. La Scala Teixeira CV, Evangelista AL, Pereira PEA, Da Silva-Grigoletto ME, Bocalini DS, Behm DG. Complexity: a novel load progression strategy in strength training. *Front Physiol.* 2019;10:839.
5. Kreher JB, Schwartz JB. Overtraining syndrome. A practical guide. *Sports Health.* 2012;4(2):128–38.
6. DiFiori JP, Benjamin HJ, Brenner J, Gregoire AJ, Jayanthi N, Landry GL, Luke A. Overuse injuries and burnout in youth sports: a position statement from the American Medical Society for Sports Medicine. *Clin J Sport Med.* 2014;24(1):3–20.
7. ACSM Position Stand. Load, overload, and recovery in the athlete: select issues for the team physician—a consensus statement. *Med Sci Sports Exerc.* 2019;51(4):821–8.
8. Shalini Paruthi S, Brooks LJ, D'Ambrosio C, Hall WA, Kotagal S, Lloyd RM, Malow BA, Maski K, Nichols C, Quan SF, Rosen CL, Troester MM, Wise MS. Recommended amount of sleep for pediatric populations: a consensus statement of the American Academy of Sleep Medicine. *J Clin Sleep Med.* 2016;12(6):785–6.
9. Bell DR, Post EG, Biese K, Bay C, Valovich McLeod T. Sport specialization and risk of overuse injuries: a systematic review with meta-analysis. *Pediatrics.* 2018;142(3):e20180657.
10. Jayanthi N, Pinkham C, Dugas L, Patrick B, Labella C. Sports specialization in young athletes, evidence-based recommendations. *Sports Health.* 2013;5(3):251–7.
11. Myer GD, Jayanthi N, Difiori JP, Faigenbaum AD, Kiefer AW, Logerstedt D, Micheli LJ. Sport specialization, part I. Does early sports specialization increase negative outcomes and reduce the opportunity for success in young athletes? *Sports Health.* 2015;7(5):437–42.
12. Mersmann F, Pentidis N, Tsai MS, Schroll A, Arampatzis A. Patellar tendon strain associates to tendon structural abnormalities in adolescent athletes. *Front Physiol.* 2019;10:963.
13. Santos EJ, Janeira MA. The effects of resistance training on explosive strength indicators in adolescent basketball players. *J Strength Cond Res.* 2012;26(10):2641–7.
14. Asadi A, Amirez-Campillo R, Meylan C, Nakamura FY, Canas-Jamett R, Izquierdo M. Effects of volume-based overload plyometric training on maximal-intensity exercise adaptations in young basketball players. *J Sports Med Phys Fitness.* 2017;57(12):1557–63.



Doping and Nutrition Supplementation in Basketball

73

Kai Fehske and Christoph Lukas

73.1 Introduction

So far doping has not been a major issue in basketball. Endurance sports such as cross-country skiing, or cycling have been influenced tremendously by doping and the artificial improvement of physical performance. Since the 1980s, FIBA (Fédération Internationale de Basketball) has regularly and in increasing numbers performed doping controls at its championships. FIBA has signed the WADA (World Anti-Doping Code) in 2004 and is strongly committed to the fight against doping in basketball. Between 2010 and 2016 FIBA organized more than 500 tests in competition with an average of 60 out-of-competition doping positives per year leading to 49 anti-doping rule violations resulting from adverse analytical findings [1].

The anti-doping database registered 135 doping cases in basketball until 2018. Most of the cases involved substances classified as cannabi-

noids. There is an increase of positive controls due to supplements [2].

Positive testing for performance-enhancing doping is rare in basketball. Suspicious activity of certain teams before and during competitions as well as positive cases involving steroids (anabolic agents) and amphetamines suggest putting basketball under continuous anti-doping surveillance.

73.2 Effects of Doping in Basketball

As in other ball sports, basketball is a very complex entity with several types of movement categories. On the field the player has moderate to high demands on intermittent endurance running capacity [3]. Even though the physical demands differ between positions, the sport itself is highly demanding on the players' constitution. With at least 82 games per season as a player of an NBA competitor, the performance-enhancing effects of amphetamines and anabolic substances could be tempting. Complex skills such as the ability to jump high to get the rebound or score a basket, to react quickly to different game settings, or just to be a good team player are difficult to manipulate with doping. Teams that have taller players with higher fat-free mass are more successful. Physical recovery as well as increase of fat-free muscle mass can be achieved with the use

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of anabolic substances such as steroid hormones and growth hormones. Amphetamine-like substances could be used to raise the alertness of the player and improve performance by helping the player assess match play situations and overcome fatigue [4].

73.3 Testing the Basketball Player

Professional basketball players in Europe are tested randomly. The current anti-doping regulations of the FIBA are implemented and supervised by the FIBA Anti-Doping Unit. They fulfill the WADA code. The players are part of two different test pools: members of a national team and players of the national leagues/European leagues. National team players are within the registered test pool. Those athletes have to reveal where they will be reachable between 6 in the morning and 11 at night for the upcoming 3 months. The so-called whereabouts program is criticized by the players because it highly interacts with their privacy. If a player is not available for testing at the previewed location, it is a doping offense. Three missed tests are classified as a positive test result, and the player is withdrawn from practice and competition, which has happened in the past to national team players. The Adams app should make it more comfortable for the test pool athletes to inform the NADA about changes in their schedule. There are no specific regulations for competitions on how many players per team have to be tested. The FIBA regulations are quite broad. Also there is no certain time to take urine or blood samples.

The professional basketball league in Germany (easyCredit BBL) implemented for the upcoming 2019/2020 season that every player of each team has to be tested at least once for performance-enhancing drugs per season. Other than that the National Anti-Doping Agency (NADA) test on an irregular basis. Usually, eight players of two teams are tested right after competition per game day.

Players of the National Basketball Association (NBA), which is considered to be

the best league in the world, do not face random testing once they have completed their rookie year. The players are only tested once per season during their 1-month training camp. Besides that the test policy also used to fail to cover a vast number of drugs such as human growth hormone or EPO. The NBA keeps much of their testing program confidential out of concern about their members' privacy. Compared to other sports, the NBA seems to be very timid with its testing policy.

Anabolic substances aimed at increasing power and muscle mass, and improving recovery will most likely be used in preparation of major events since they can be detected for a prolonged time in urine samples. Substances such as growth hormones have a shorter detection window. Anti-doping experts suggest implementing a testing scheme with targeted urine tests approximately 2–4 weeks before the start of major competitions. To cover modern doping substances which are difficult to detect, additional blood samples are also being recommended [4].

73.4 The Athlete Biological Passport

The Athlete Biological Passport is a system implemented by the WADA directed toward enhancing the identification of those athletes accountable for the misuse of performance-enhancing substances [5]. Doping leaves a biological fingerprint in the athlete's body. The Athlete Biological Passport is the paradigm testing that aims to detect this biological fingerprint [6]. Biomarkers of doping measured or inferred from blood and urine samples are used for that purpose, in the same way that biomarkers of disease are used in medicine as indicators of the presence or severity of a disease [7]. The blood passport aims to detect any modification of erythropoiesis, whether by blood transfusion or the use of erythropoiesis-stimulating agents, such as recombinant erythropoietin [8]. The doping substance itself is not detected but rather its effects on the organism.

After exclusion of any possible pathology, specific variation from the individual norms will be considered as a potential misuse of hormones or other modulators to enhance performance [9]. Recently, a new development of the Athlete Biological Passport has been introduced which is aimed at detecting the abuse of anabolic hormones which could also be used in basketball [4]. FIBA has not implemented the Athlete Biological Passport at the time of this writing (2019).

73.5 THC/Cannabis

Positive doping tests due to the use of cannabis are a growing problem in the world of basketball as more states in the USA are legalizing the use of cannabis. This makes recreational use in the USA more prevalent since its legalization in many states. This is compared to European professional leagues where this has not been a common problem. Since cannabis does not enhance performance, many question whether it should be tested for at all, especially if it is a legal substance where the player resides.

73.6 Misuse of Non-doping Listed Medication

Another big issue is the widespread use of non-listed (not forbidden) general medication. It is known that many professional athletes including basketball players suffer from chronic or subacute overuse injuries of their extremities (shoulder, ankle, etc.) and therefore use painkillers such as non-steroidal anti-inflammatory drugs (NSAIDs) during competition. This is not a direct performance enhancement through typical doping substances that improve alertness, body composition, endurance, and power, but those drugs can provide an indirect performance enhancement. Some players consume those substances in high doses which could lead to typical side effects including gastrointestinal bleeding, and renal injury or failure in severe cases. There

have been reported cases of severe organ damage, in one case ending in kidney transplantation (<http://www.spiegel.de/panorama/leute/ivan-klasnic-klage-gegen-werder-bremen-aerzte-vor-entscheidung-a-1139873.html>). This player later sued his doctors for giving him the medication.

73.7 Nutrition Supplementation

Many athletes use nutritional supplements such as creatine, amino acids, or simply vitamins to support their muscle growth or to recover quicker even though the efficacy is not certain [7]. One of the leading anti-doping biochemists Hans Geyer from Cologne, Germany, states that several recent studies have shown evidence of some nutritional supplements containing prohibited anabolic androgenic steroids, so-called prohormones, which were not declared on the label [3]. Within the last 2 years, 21 basketball players worldwide have been tested positive for prohibited substances after the consumption of nutritional supplements. In the blood and urine sample, there have been traces of the pro-hormone higenamine, which has anabolic potential. The positive test led in all cases to a suspension from practice and competition. The stimulant higanamine is found in a variety of plants and is included in many weight loss and sports supplements [10]. In 2016 and 2017, it led to 113 adverse analytic findings worldwide for all sports.

73.8 Doping Prevention

The main aspect of prevention of doping is awareness and knowledge. The athletes need to be aware of prohibited substances. In most European sport leagues, it is mandatory to educate the players. Prevention and education should start at a young age. Since most positive tests have been from cannabinoids, especially the young player has to be aware that lifestyle drugs are also prohibited.

Similar to nutritional supplements, many other products or medication can lead to positive

testing. For instance, consumption of overnight cold medication (stimulants) and even poppy-seed cake (opiates) could already lead to a positive result.

Education on doping should be a team effort. The personnel in charge of medical teams, whether at the national or team level, should be responsible and make efforts to provide such education or make sure it is provided. Consequences can often apply to medical personnel and may affect their careers as well as the players' careers.

Overall, it is important to note that players are not required to memorize every single substance on the prohibited list. They should be aware and have a responsibility to inquire about any drug/medication or substance offered to them and also be aware of the risks in consuming even "innocent" substances as nutritional supplements and certain foods.

The WADA website (<https://wada-ama.org/>) contains further information on rules, regulations, education, and prevention.

73.9 Conclusion

Until now, doping is fortunately not a major issue in basketball. The use of performance-enhancing substances does not seem to be common. Measures have been taken to beware the cleanness of the sport. The main focus should be on prevention through education especially of the young players. Multi-layer testing should

be established in each federation based on the WADA code to detect doping offence and to protect the honest athlete.

References

1. Database A-D. Doping in Basketball 2018 [updated 19.07.2019]. <https://www.dopinglist.com>.
2. Geyer H, Parr MK, Mareck U, et al. Analysis of non-hormonal nutritional supplements for anabolic-androgenic steroids—results of an international study. *Int J Sports Med.* 2004;25(2):124–9.
3. Stojanovic E, Stojiljkovic N, Scanlan AT. The activity demands and physiological responses encountered during basketball match-play: a systematic review. *Sports Med.* 2018;48(1):111–35.
4. Schumacher YO. Doping in handball—conceptual thoughts for the future of the sport. *Aspetar Sports Med J.* 2014;3:228–31.
5. Bucknall V, Rehman H, Bassindale T, Clement RG. The athlete biological passport: ticket to a fair Commonwealth Games. *Scott Med J.* 2014;59(3):143–8.
6. Robinson N, Sottas PE, Schumacher YO. The athlete biological passport: how to personalize anti-doping testing across an athlete's career? *Med Sport Sci.* 2017;62:107–18.
7. Butts J, Jacobs B, Silvis M. Creatine use in sports. *Sports Health.* 2017;10(1):31–4.
8. Robinson N, Saugy M, Vernec A, Pierre-Edouard S. The athlete biological passport: an effective tool in the fight against doping. *Clin Chem.* 2011;57(6):830–2.
9. Saugy M, Lundby C, Robinson N. Monitoring of biological markers indicative of doping: the athlete biological passport. *Br J Sports Med.* 2014;48(10):827–32.
10. Zhang N, Lian Z, Peng X, Li Z, Zhu H. Applications of higenamine in pharmacology and medicine. *J Ethnopharmacol.* 2017;196:242–52.

Long-Distance Traveling in Basketball: Practical Applications Based on Scientific Evidence

Thomas Huyghe and Julio Calleja-Gonzalez

74.1 Introduction

The National Basketball Association (NBA) is the premier basketball league in the world [1, 2] and in recent years a greater emphasis has been placed on player safety [3, 4]. In regard to player safety, there has been increased attention in the areas of training load [3, 5] as well as schedule and travel requirements [5]. In an attempt to reduce the training load and schedule requirements of players, the NBA has modified the preseason schedule. Prior to 2017, NBA teams played eight preseason games across 3–4 weeks in preparation for the regular season [6, 7]. Since the 2017–2018 season, the NBA season has consisted of four to six preseason games played across 3–4 weeks followed by an 82-game regular season played across 26 weeks (177 days). During the regular season, each team plays two to five games per week (~3.2 games per week), with games lasting an average duration of 2 h and 15 min. NBA teams rarely practice during the season, and practices that occur are typically less than 1 h [1, 2].

In response to teams resting players during back-to-back (two games within a 2-day span) games [8], the league extended the duration of the regular season by 7 days with the purpose of scheduling fewer back-to-back games [6]. During the 2017–2018 season, NBA teams played an average of 14.4 ± 0.9 back-to-back games, which was the lowest on record compared to any previous season in the NBA [2]. Furthermore, the 2017–2018 NBA season marked the first season in NBA history in which no team played four games in five nights [6]. Despite adjustments to the NBA schedule, air travel demands remain high due to the geographical span of teams across four time zones (eastern, central, mountain, and western). In this regard, NBA players spend more time above 30,000 ft than athletes competing in all other team sports in the United States of America (USA) [7]. Consequently, air travel requirements are a concern for NBA coaches, players, and owners, as research has demonstrated that short-haul flights (e.g., domestic ≤ 6 h flights) increase injury risk [2, 9–13] and impede performance [9, 14–20] with more regular or longer periods of travel (e.g., ≥ 6 h international transfers) more likely to result in negative responses [21, 22]. In this chapter, we aim to provide a comprehensive summary of scientific evidence about air traveling on five critical aspects: (1) fatigue, (2) athletic performance, (3) sleep, (4) health and injuries, and (5) mood state alongside suggestions for future research

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and real-world applications in professional basketball, particularly in the NBA.

74.2 The Influence of Traveling on Fatigue

74.2.1 Scientific Evidence

Frequent air travel can negatively affect hydration status, nutritional behaviors, sleep quality, and sleep quantity, thus extending the time for sufficient recovery between games and/or training in athletes [15]. As a result, air travel should be considered as an additional stressor imposed on NBA players in conjunction with competition and training schedules [15], especially when less than 72 h of rest is experienced between games [23, 24].

Depending on the magnitude of air travel exposure, traveling can result into a variety of

negative consequences (Fig. 74.1). One of the main consequences associated with frequent air travel exposure is “travel fatigue.” While jet lag refers to the physiologic adaptations after a single trip across one or more time zones, travel fatigue is a more complex summation of physiological and psychological consequences accumulating over a longer period of time (e.g., competitive season) which reduces the player’s ability to recover and perform [18]. Hence, travel fatigue is characterized by feelings of disorientation, light-headedness, gastrointestinal disruption, impatience, lack of energy, loss of motivation, recurrent illness, changes in behavior, and general discomfort following travel across time zones [13, 18], while jet lag is mainly characterized by gastrointestinal disturbance such as heartburn, indigestion, diarrhea, sleep disturbance, intermittent fatigue, loss of appetite, impaired concentration, and disruption of situational awareness [18, 25] (Fig. 74.2).

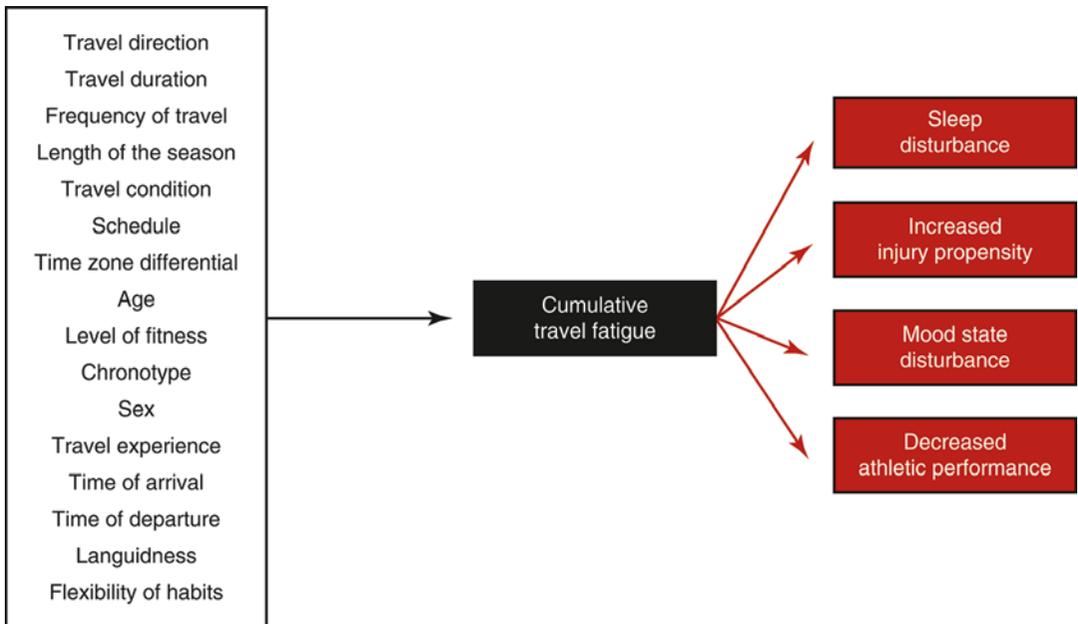


Fig. 74.1 Characteristics of travel fatigue (chronic) versus jet lag (acute)

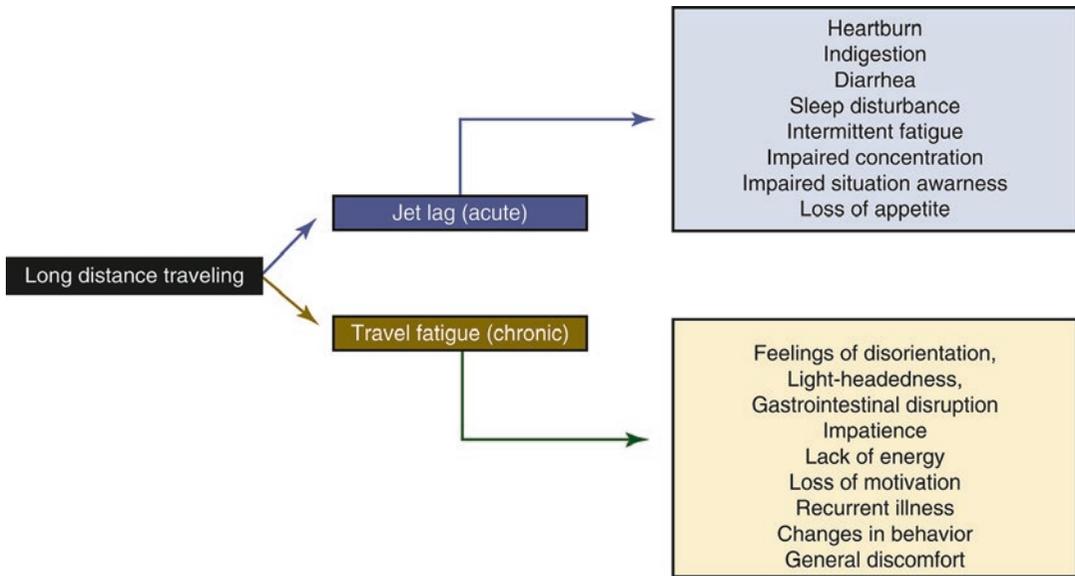


Fig. 74.2 Potential underlying factors and negative consequences of long-distance traveling in NBA basketball players

The magnitude of travel fatigue depends on many factors such as regularity, duration, and conditions of travel [13, 26] (Fig. 74.1). Specific causes of air-related travel fatigue include the following:

- Prolonged exposure to mild hypoxia [16, 27, 28].
- Difficulties in standing, walking, and moving around due to limited room inside the air cabin.
- Reduced air quality in the cabin, which may impair immune function [12].
- Dry cabin air and low hypobaric pressure potentially causing dehydration [29].
- Prolonged sitting in a cramped position reducing mobility and flexibility [10, 16].
- Disruption of routines (e.g., eating and sleeping) [30].
- Noise of plane and cabin (e.g., sleep disturbance) [16].
- Formalities of air travel may induce negative mood states [30].

A primary issue regarding air travel occurs as a result of significant reductions in oxygen

saturation, which has been found to decrease significantly from 97% at ground level to 93% at cruising altitude ($p < 0.05$) [28]. This finding is significant as oxygen saturation levels of 94% or less may prompt physicians to administer supplemental oxygen in hospital patients and thus would slow muscle recovery [28]. Furthermore, researchers examined markers of skeletal muscle damage following trans-American jet travel, but since a non-exercise control was not included in this investigation, meaningful interpretations of the data cannot be determined [32].

The regularity, duration, and direction of air travel, combined with in-cabin conditions, likely predispose NBA players to one or more symptoms of travel fatigue [13]. In turn, travel fatigue can have deleterious effects on player recovery and subsequent performance, particularly when scheduled soon after practices or games. When flying across two or more time zones, symptoms of travel fatigue can remain up to 2–3 days after arrival [13]. Consequently, it is recommended that recovery and practices administered before and after air travel are modified to account for

travel fatigue, especially considering the travel direction and flight duration experienced traveling in NBA basketball players.

74.2.2 Practical Applications

Considering that skeletal muscle and connective tissues become shortened during flights and may stiffen, it is recommended for players to avoid sitting the entire trip, and instead, walk around the cabin every hour, unless they are asleep or advised not to do so by flight staff [33]. With a tentative agreement between the NBA and Delta Airlines (Atlanta, GA, USA) charters, walking inside the air cabin should be attainable, as most NBA teams (27 out of 30 teams) fly with private jets of Delta Airlines (including Airbus 319s, and Boeing 757-200s) with almost 50% more cabin space than standard planes [34]. This cabin space allows most NBA players, who possess an average stature of 6 feet and 7 in, to have more freedom to stand erect during air travel [34]. Additionally, simple stretching exercises can be applied while in the seat or in the cabin, which could help relax muscles while increasing blood flow and delivering oxygen and other nutrients to muscles [31, 33]. As a result, stretching may reduce the negative effects of air travel on flexibility and skeletal muscle recovery. Consequently, future studies are encouraged to examine the efficacy of these in-flight travel strategies in NBA players.

74.2.3 Future Research

Despite recent schedule modifications and an increased awareness of the potential negative consequences of air travel on the health and performance of NBA players, there is still a need to implement effective strategies to address issues with travel fatigue to promote greater equity across western and eastern teams. Future research exploring various aspects of regularity, duration, directions, and conditions of air travel [13] in one or multiple NBA seasons can help identify origins of fatigue in players. Consequently, a

holistic approach to future research (e.g., applied chronobiology and jet lag research) is recommended, with some potential topics of interest encompassing descriptive and intervention-style studies.

74.3 The Influence of Traveling on Athletic Performance

74.3.1 Scientific Evidence

74.3.1.1 Circadian Rhythm and Performance

Shifting a player's circadian rhythm (body clock) relative to real time in the destination has demonstrated to be a major factor in sports performance. Although peak performance times throughout the day may vary among players [33], for most players, peak performance tends to appear between 4:00 and 10:00 pm [33]. Proper planning of physical activities has the ability to support resynchronization of the player's circadian rhythm, which in turn can lead into an increase of up to 10% in endurance, physical strength, and mental function [19, 33]. Consequently, circadian variations should be taken into account when scheduling practices and physical activities before and after air travel. In particular, strength and endurance training tend to be optimally planned in the afternoon or early evening while motor skill training in the morning [35]. However, future research studies are needed to validate these findings in the NBA as well as other professional basketball leagues.

74.3.1.2 Travel Direction and Performance

The direction of air travel should be considered by NBA teams as traveling westward exacerbates reductions in performance [14, 23]. In a sample of 8495 NBA games between 1987 and 1995, west coast teams scored four more points per game ($p < 0.05$) when traveling to the east coast than east coast teams scored when traveling to the west coast [23]. Furthermore, NBA teams traveling eastward had a winning percentage of

45.4% compared with 36.2% for teams traveling westward ($p < 0.001$) between 2010 and 2015 [14]. The increased difficulty of traveling westward across the USA to compete has also been reported in the National Football League and the National Hockey League [14]. Westward travel is likely more difficult since performance tends to peak in the late afternoon, and players traveling from west to east tend to play games closer to their circadian peaks, given most NBA games are played at night.

74.3.1.3 Competition Schedule and Performance

The home court advantage during competition has been reported in multiple team sports [14, 16, 19, 36] including the NBA [37, 38], suggesting either a negative effect of travel or a circadian advantage [39]. However, the magnitude of the home court advantage may be mitigated or enhanced depending on other external factors such as the travel direction, tip-off time, opponents' playing style, game/practice volume and intensity, and travel duration. Therefore, a holistic approach to scheduling and travel management is recommended.

Another factor that plays a role into the home court advantage is the quantity of rest NBA teams attain prior to games [37]. In particular, a consistent advantage was recorded when a team had more than 1 day of rest between games (the home team's score increased by 1.1 points per game and the away team's score increased by 1.6 points per game) in a sample of 8495 regular season NBA games between 1987 and 1995 [23]. Moreover, average total scores (home and away teams) were highest when 3 days of rest were encountered between games with data collected from the 1987 to 1995 seasons [23]. Consequently, the negative influence of air travel during an NBA season may be mitigated by incorporating supplemental days to recover from games.

An optimal recovery window of 72 h following games and practices is needed for an athlete or team to return to optimal levels of performance [24]. Nevertheless, the NBA schedule dictates condensed game schedules that necessitate com-

pressed training schedules, which may inhibit access to active rest days to fully recover from accumulated physical and psychological stress induced by NBA games and practices. In this way, NBA teams are often obligated to intervene with various ergogenic practices in an attempt to speed up the recovery process, such as whole-body cryotherapy, compression tights, cold water immersion, contrast water therapy, and soft tissue massage [40].

74.3.2 Practical Applications

Although age and level of fitness can influence the severity of symptoms associated with long-distance traveling [41], air travel should be recognized and respected as an additional stressor when planning physical activities before, during, and after a travel period. Consequently, practices and workout sessions should be planned on a chronobiological basis to ensure that performance is not impaired during the trip and after returning home [25]. In particular, it is recommended to reduce the overall volume, intensity, and frequency of endurance training sessions, while strength training sessions (neuromuscular work) tend to be less susceptible to the negative consequences of long-distance traveling [18]. However, the benefits should be weighed with the potential risks. Hence, high-volume, high-intensity, and even traditional strength routines should not be assumed to be indispensable [18]. Additionally, the timing of practices and physical activities should not be planned during the circadian nadir (2–4 PM and 2–4 AM of the departure time zone) until the players are fully adapted to the new time zone [18].

74.3.3 Future Research

While these commonly employed recovery practices, including compression tights, [36] cold water immersion, [42] and massage, [43] have been investigated in various samples of basketball players, no data are available specifically in

NBA players. Therefore, more research is needed to ascertain if these recovery practices benefit NBA team and player performance, particularly around intensive and extensive travel periods.

Furthermore, most of the studies that investigate performance decrements related to air travel in team sport athletes have methodological flaws [33, 44] such as the absence of preflight assessments compared to postflight assessments. Therefore, it is recommended that future studies take these issues into account when constructing their research methodology.

74.4 The Influence of Traveling on Sleep

74.4.1 Scientific Evidence

74.4.1.1 Desynchronization of the Circadian Rhythm

The physiological and perceptual stressors associated with flying across one or more time zones may alter sleep patterns in athletes [12], especially when accumulated over an entire season [18, 45]. In particular, short-haul air travel has been reported to impair athletic performance due to the development of an inefficient internally-driven circadian rhythm (i.e., sleep deprivation or disorientation between the circadian system and the environment) [44]. Hence, the circadian rhythm plays a critical role in sports performance [13, 19, 46, 47]. When an athlete's circadian rhythm is synchronized with the environment, the athlete should achieve optimal performance during late afternoons and early evenings [19]. Contrary, a desynchronized circadian rhythm impedes the ability for muscles to accumulate protein synthesis (the ability of skeletal muscle to adapt and repair), which consequently may limit training adaptations [48, 49]. This may be concerning during the preseason period, given sleep disturbances are present during higher training volumes [50]. Since sleep loss can also affect vigor, mood, and perceptual awareness [30, 51], early training sessions could cause reductions in motivation

and consequently reduce optimal training performance and subsequent adaptations [52].

74.4.1.2 Time Zone Differential and Sleep

Considering air travel can cause an athlete's circadian rhythm to become unsynchronized with the environment, air travel may contribute to the home court advantage in the NBA [53, 54] as the body's core temperature (an endogenous measure of circadian rhythm) takes approximately 1 day for each time zone crossed to adapt completely to the new time zone [13, 39]. Consequently, the number of time zones traveled plays a critical role in the magnitude of potential disruptions in the circadian rhythm of NBA players [13]. Hence, the greater the number of time zones traveled, the more difficult it is for an athlete to adapt to a new time zone. For example, a 2-h time zone shift may cause marginal disruption to the circadian rhythm, but a 3-h time zone shift (e.g., NBA players traveling coast to coast within the USA) can cause a significant desynchronization of circadian rhythm [13]. Therefore, it is recommended that NBA players focus on physical activity, eating, and social contact during daylight in their new time zone in order to resynchronize their circadian rhythm, especially when traveling from coast to coast [13].

74.4.1.3 Travel Direction and Sleep

One study examined the effects of air travel from the east coast to the west coast of the USA on physiological performance measures, sleep quality, and hormonal alterations [32]. However, it is important to note the following: participants used in this investigation were not athletes, a simulated sporting event most closely related to demands experienced during soccer was administered, and a non-exercise (control) group was absent. However, air travel-induced jet lag symptoms, which resulted in decreased sleep quality and was paired with significantly increased melatonin levels on flight days (travel from east to west coast and travel from west to east coast) [32].

Finally, flying eastwards requires a phase delay of the player's circadian rhythm, while

traveling westwards requires a phase advance of the players' circadian rhythm. Typically, the body adjusts more efficiently to a phase delay compared to a phase advance because the standard duration of circadian rhythm is approximately 24.5 h (in sync with environmental factors such as light, temperature, food intake, physical and social factors) [25]. Hence, it is easier to fall asleep when days are becoming longer rather than days becoming shorter.

74.4.2 Practical Applications

Implementing a brief data collection procedure pertaining to each player's sleep history during the initial medical screening process as well as a month's worth of sleep logs could help identify which player is more susceptible to jet lag or travel fatigue before the official start of the season. In particular, it is recommended that sleep quality in team sport athletes is monitored through actigraphy as well as the Pittsburgh Sleep Quality Index, Likert scale, Liverpool Jet-Lag Questionnaire, and RESTQ [55]. However, more studies are needed to verify their efficacy in professional basketball teams.

NBA schedule-makers and teams may succeed in mitigating the negative effects of air travel from coast to coast on sleep by implementing up-to-date, evidence-based strategies applied in other professional sports such as the ingestion of more frequent and smaller meals [18]. In particular, a high-carbohydrate, low-protein meal in the evening is recommended to enhance serotonin production and subsequently promote drowsiness and sleep [19, 33], while the ingestion of a high-protein, low-carbohydrate meal is recommended in the morning to induce the uptake of tyrosine and its conversion to adrenaline, which elevates arousal and promotes alertness [33, 56]. However, future studies are required to evaluate the efficacy of the abovementioned strategies in NBA players.

Besides the implementation of specific travel management strategies, basic education and awareness programs on behavioral management

for staff and players to recognize the causes, symptoms, and solutions of cumulative travel fatigue and sleep disturbances in professional basketball players is strongly recommended [18, 47]. For example, prior to long-distance flights (>6 h), high-performance support staff should advise their teams to adopt the timing of training sessions to the destination time zone to help synchronizing the circadian rhythm of their players prior to arrival [18]. Furthermore, an evening flight is recommended for eastward travel (e.g., United States to Europe) [18]. During the flight, eyeshades and earplugs are recommended to help relaxation and mitigate the noise and overstimulation. Furthermore, on-board meals should be consumed according to the destination time [18]. Perhaps, having the players bring their own meals may be helpful in this regard. Additionally, players should also adapt their watch to the destination time as soon as boarding the plane, maintain proper hydration, and sleep according to the destination time [18]. Moreover, to aid with a rapid circadian adaptation to the new destination immediately after a long-distance flight, high-performance support staff and their players are recommended to strategically plan their activities according to the new time zone. Strategic use of naps, caffeine, melatonin, light exposure, light avoidance, and physical activity may all help with accelerating (or decelerating) the adaptation process, especially for those who tend to be more susceptible to travel fatigue compared to others [18, 57–59]. Finally, in the current age of "social media" consumption and late-night tweeting in the NBA, players should also be advised to minimize tablet usage or any other form of blue light exposure before heading to bed as this has demonstrated to sabotage sleep quantity and next-day game performance [60].

74.4.3 Future Research

Considering frequent time zone transitions often disrupt the circadian rhythm in athletes [15, 16, 19, 30, 61, 62], future studies may focus on monitoring both exogenous and endogenous

parameters, such as the measurement of salivary melatonin onset, adrenaline concentrations, and body temperature as these are critical biomarkers of circadian rhythm [19, 63]. Measurement of these biomarkers would provide insight into how each player individually adapts to air travel throughout the NBA season. Consequently, NBA performance support staff may then apply individualized approaches to training and game preparation to combat the negative impact of air travel.

Furthermore, examination of various ergogenic aids will provide a better understanding of practices that may enhance physiological and perceptual responses to air travel in NBA players. For instance, nutrition and hydration are fundamental aspects underpinning circadian rhythm [64]. Therefore, analyzing and comparing the hormonal responses of NBA players adopting different diets may provide NBA coaches and support staff with further insight into beneficial nutritional strategies for coping with air travel in the NBA.

Light therapy, light avoidance, and pharmacological techniques have demonstrated to help shift the circadian phase to optimal time zones before, during, and after flights [58]. However, practical recommendations pertaining to this type of intervention remain difficult due to interindividual and intraindividual differences in response to these interventions alongside the lack of research on light therapy in team sport environments in general. Future studies have an opportunity to validate the impact of light avoidance (e.g., light-blocking glasses), blue light exposure, chronobiotics (circadian phase shifters), and chronohypnotics (sleep inducers) on the sleep quantity and quality of NBA players during one or multiple periods of the season.

Finally, more studies are needed to establish a definition of parameters inferred by actigraphy as well as consistency in sleep quality algorithms, and the validation of self-perceived sleep questionnaires, particularly in team sport athletes [55].

74.5 The Influence of Traveling on Player Health and Injury Propensity

74.5.1 Scientific Evidence

74.5.1.1 Competition Schedule and Injuries

Competing in away games has been reported to significantly increase regular season injury risk in a sample of 1443 NBA players between 2012 and 2015 [9]. Specifically, 54% of regular season injuries occurred in players playing games away from home, which was significantly greater than the expected injury rate for away games of 50% ($p < 0.05$) [9].

74.5.1.2 Playing Time and Injuries

Another factor to consider in reducing injury risk in the NBA is the total amount of in-game minutes accrued by each player. While coaches have presumed withdrawing high-minute players from entire games may reduce injury risk and enhance performance, a tactic which is often seen nearing the conclusion of the regular season, data to support this approach is lacking. In fact, existing data revealed the average minutes played per game did not influence injury risk ($p < 0.001$) in 811 NBA players competing between 2000 and 2015 [8, 9]. However, it should be noted these data are not reflective of performance and injury risk in players who were rested for entire games but rather are indicative of players completing reduced game minutes. Subsequently, future studies are needed to examine the consequences and confirm the efficacy of resting high-minute players for entire games in the NBA.

74.5.1.3 Air Travel and Immune System Suppression

One of the main concerns with long-distance air traveling (e.g., transcontinental flights) is the disturbance of sleep. Subsequently, this may cause a suppression of the immune system of the athlete when sleeping 1 h less than usual

[65]. This can be exacerbated by dry pressurized air cabin conditions during the flight due to fluid loss, abnormalities in breathing, dry nose and throat, similar to responses on acute exposure to altitude [25]. Consequently, diuretics such as caffeine and alcohol should be avoided during long-distance flights to mitigate these symptoms [48].

74.5.1.4 Air Travel and Deep-Vein Thrombosis

A potential negative consequence of prolonged sitting within the cabin of the airplane is “venous thromboembolism” [66] (e.g., career-ending diagnosis of NBA-champion Chris Bosh), often referred to as “travelers thromboses,” which is a condition that occurs when a blood clot (thrombus) forms in one or more of the deep veins in the body, usually in the lower limbs. Deep vein thrombosis can cause leg pain or swelling, but might occur without any symptoms. On a large scale, this condition has been reported as a major health problem with at least 201,000 cases reported each year in the United States in which 25% of these patients die within 7 days [67]. The risk for venous thromboembolism can remain up to 2 weeks after a long-haul flight. Additionally, the annual risk of being diagnosed with this condition increases with 12% if one long-haul flight is taken yearly [66]. Although no studies have been published on this regard within the NBA ecosystem, NBA physicians and high-performance support staff should be well-aware of the potential risks associated with long-distance traveling (e.g., during preseason transcontinental NBA global games) and advise players and staff with customized travel management strategies as well as prophylaxis based on other individual risk factors associated with deep-vein thrombosis [68]. For example, FlightFit is an iOS application which introduces basic stretching techniques to air travelers in an interactive way [69], which may help blood circulation, oxygen, and nutrient supply to the muscles, bones, and connective tissues, and ultimately mitigate the risk for travelers’ thromboses.

74.5.2 Practical Applications

In order to mitigate the negative consequences on players’ health and injury propensity of air traveling, it is recommended for players to drink more than usual during flights, approximately 200 ml extra in 12-h flight [25]. Furthermore, to mitigate the risk of travelers’ thromboses, it is recommended to implement a comprehensive assessment prior to the competition period to identify potential high-risk profile players. Additionally, keeping the body active through in-flight flexibility exercises may also help in reducing joint stiffness and pooling of blood in the legs [25]. For example, through the mobile application “FlightFit,” players can stretch different muscle groups by following an interactive infographic system and instructions visually and acoustically as well as customizing their own stretching plans based on their individual characteristics and needs [69]. FlightFit also includes the possibility to implement notification reminders and in-flight tips which may help with educating and cultivating healthy travel behaviors. Another educational opportunity might be considered through the form of a meeting or workshop with the team or players individually prior to traveling. In this case, keeping the information brief and to-the-point (e.g., infographic or summary table) would help players to comprehend and apply the given recommendations. Each player should also be advised on an individual basis as a difference exists in peak core body temperature and ability to fall asleep or arise between morning type, intermediate type, and evening type of players [25].

Consequently, the player’s chronotype should be taken into consideration when providing guidelines. Finally, high-performance support staff who travel with the team should also consider their age as a subsequent factor in the ability to shift in circadian rhythms because a phase advance (tendency toward morning-type behavior) might be favored once reaching the age of 47 years [25]. Finally, in order to mitigate the suppression of the immune system, special attention should also be given toward proper food hygiene

(e.g., avoiding uncooked foods) and ensuring at least 8 h of high-quality sleep before, during, and after trips [65].

74.5.3 Future Research

In order to mitigate the potential consequences of air travel on NBA players' health state and injury risk, each player's external and internal workload should be monitored and carefully managed throughout the season. However, future research should investigate new metrics or biomarkers that reflect a player's immune system and injury risk profile. For example, researchers may focus on salivary cortisol and salivary immunoglobulin A (SIgA) as demonstrated in a previous study on monitoring stress tolerance in basketball players [70] or vitamin D status, cortisol, testosterone, pro-inflammatory cytokines (e.g., TNF- α and INF- γ), and anti-inflammatory cytokines (e.g., IL-4 and IL-10) [71, 72]. Additionally, future studies should approach a holistic mindset to gain a complete picture about each player. For example, ratings of perceived exertion (training load) and psychometric questionnaires can be included and compared with other insightful metrics as well. Once this information has been collected over a longer period of time (e.g., 4-week period), multiple microcycle stereotypes could then be identified based upon the NBA season schedule (e.g., home week, road week, split week, 3-game week, 2-game week). Subsequently, this data would allow researchers to identify the impact of air traveling on player's immune system and injury risk and identify which microcycle might be at higher risk compared to others. Consequently, this would support more efficient and effective travel management strategies for NBA players and high-performance support staff.

74.6 The Influence of Traveling on Mood State

74.6.1 Scientific Evidence

In addition to sleep disturbances, fatigue, detrimental performance, and increased risk of injury,

traveling can result in an impaired mood state [18, 73, 74] and loss of motivation all of which can affect recovery [18]. If a player's motivation declines, their training intensity and volume during subsequent practices may decline equivalently, [30] and consequently their performance as well [74]. In particular, westward travel across six time zones demonstrated altered mood states for up to 24 h post-travel [74]. Furthermore, sleep disruption and desynchronized circadian rhythm induced by long-distance traveling may further exacerbate these fluctuations in mood states [44]. However, these findings often remain inconsistent in the literature [35, 74] and have yet to be determined in professional basketball players in particular.

74.6.2 Practical Applications

Although it can be difficult for NBA coaches to make the most appropriate decisions toward their training and recovery protocols of the competition schedule for the entire team, especially the day after a game, it is important to consider the type of treatments, timing of practices and activities, and recovery interventions as all these factors can either induce or reduce mood disturbances caused by frequent traveling.

In order to mitigate the negative impact of air travel on mood state, it is recommended that each player's psychological and psychosociological reactions to air travel should be monitored during the season. For instance, comprehensive psychometric questionnaires such as the Acute Recovery and Stress Scale (ARSS) [75] and the REST-Q Sport [76] have been established as logical, practical, and versatile tools to measure self-perceived travel fatigue in professional team sports [75, 76]. Considering the time constraints in the NBA, shorter customized versions of these questionnaires can be completed on a daily basis [77], which have been reported to be valid and reliable in elite Australian Rules Football [78]. However, further research is necessary to provide normative standards, especially with a

focus on individual interpretations, recommendations, and compliance in NBA players.

Furthermore, a systematic exercise regime (even away from home) should be considered as physical activities have demonstrated to improve mood state in athletes [79] and immediately after arriving to the athletes' new destination [41]. However, the timing of these exercise regimes should be carefully planned by high-performance support staff with consideration to the changes in the athletes' body temperature as a symptom of adapting to their new time zone [41].

Unnecessary psychological stress should be avoided by approaching travel with a proactive mindset. For example, players should be advised to adjust their watch and phone to the local time of their upcoming destination as soon as boarding the aircraft [33]. Additionally, if the budget allows, travel managers should incorporate 1 day of adjustment time for each time zone crossed [33]. Moreover, family and friends should be informed about the time differences during each travel period to avoid phone calls at night or during times that may interfere with sleep [33].

Finally, extending sleep over a period of 3 weeks with a mean addition of 110 min per night has demonstrated to improve shooting accuracy, energy, mood state, and mental preparedness for competition in basketball players [80]. However, benefits only occurred in non-sleep deprived players, which means that sleep extension should only be considered when players suffer from significant less total sleep time [80]. Nevertheless, more research is needed pertaining to this particular strategy.

74.6.3 Future Research

More research is needed on the acute and chronic effects of cumulative travel (e.g., over a season) on psychological and physiological recovery parameters of professional team-sport athletes. Future research has the opportunity to validate proactive interventions (e.g., light therapy, daytime nap scheduling) and reactive interventions (e.g., oxygenation therapy).

Take-Home Message

The NBA travel schedule induces misalignments in circadian rhythm that cannot be avoided. Hence, air travel across three time zones has been reported to induce susceptibility to travel fatigue [18, 44, 56, 61, 62], increase injury risk [13, 44, 81], disrupt sleep patterns [12, 18], disturb an optimal mood state [18, 74], and reduce game performance [13, 14, 17, 44, 53].

First, it is important to understand the impact of air travel on NBA players at an individual level, given that each NBA player adapts to the demands of long-distance travel differently. Therefore, a well-structured travel management plan, taking into account both travel conditions and players' individual needs, is the first step in establishing an effective approach to mitigating the negative consequences of frequent air travel. Therefore, adopting a preflight, in-flight, and postflight model [47] and incorporating a travel fatigue monitoring system would help NBA high-performance support personnel to identify problems, limit negative symptoms induced by traveling, and subsequently improve players' health and performance. Nevertheless, future studies in collaboration with multiple NBA stakeholders are needed to identify the impact of current travel management practices and approaches implemented by NBA teams across one or more NBA seasons.

The following recommendations (Table 74.1) are based on the literature reviewed within this chapter as well as empirical findings from research on air travel in elite team sports. It should be noted that there remains a scarcity in research in long-distance traveling in basketball. Nonetheless, there tends to be little risk involved and much (potential) benefit in applying these recommendations. Most importantly, these recommendations should be tailored toward individual differences between players.

Table 74.1 Practical recommendations to mitigate the negative consequences with long-distance traveling for basketball players, coaches, and high-performance support staff

<i>Long distance traveling in basketball</i>			
	Scientific evidence	Practical applications	Future research
Fatigue	<ul style="list-style-type: none"> • Flying reduces oxygen saturation which slows down muscle recovery • Air travel likely predisposes NBA players to one or more symptoms of travel fatigue, which in turn disrupts player recovery and performance, particularly when scheduled soon after practices or games • When flying across two or more time zones, symptoms of travel fatigue can remain up to 2–3 days after arrival 	<ul style="list-style-type: none"> • Incorporate educational sessions with players and staff about the potential increments in stiffness and self-perceived fatigue after flying, and how they can mitigate these negative consequences before, during, and after trips • Maintain extra hydration before, during, and after flights • Avoid sitting the entire trip, but instead, walk around the cabin every hour and perform simple seated or standing stretches (e.g., FlightFit application), unless you are asleep or advised not stand up by the on-board flight crew • If traveling across 3 ≥ time zones in eastward direction or traveling across 4 ≥ time zones in westward direction, consider the following: <ul style="list-style-type: none"> – Ultra short-acting as well as medium-acting and medium half-life hypnotic; – Intake smaller, more frequent, and recovery content meals according to the destination schedule; • If traveling across 3 ≥ time zones in eastward direction or 4 ≥ zones in westward direction, consider the following: <ul style="list-style-type: none"> – Reduce training 2–4 days postflight; – Intake 3–5 mg melatonin 30 min before bed 	<ul style="list-style-type: none"> • Effective strategies to address issues with travel fatigue to promote greater equity across western and eastern teams • The regularity, duration, directions, and conditions of air travel across one or multiple NBA seasons to identify the origins of “travel fatigue” in players • A holistic approach to future research is recommended, with some potential topics of interest (e.g., applied chronobiology) encompassing both descriptive and intervention-style studies

Table 74.1 (continued)

<i>Long distance traveling in basketball</i>			
	Scientific evidence	Practical applications	Future research
Athletic performance	<ul style="list-style-type: none"> • Traveling westward reduces performance, particularly winning percentage and points per game • NBA players who travel from west to east tend to play games closer to their circadian peaks • NBA teams perform significantly worse in away games compared to home games • An optimal recovery window of 72 h following games and practices is needed for an athlete or team to return to optimal levels of performance • For most athletes, peak performance tends to appear between 4:00 and 10:00 pm • Proper planning of physical activities during travel can improve endurance, physical strength, and mental function in athletes up to 10% • Neuromuscular training tend to be less susceptible to negative consequences of long distance traveling compared to high-volume work 	<ul style="list-style-type: none"> • Maintain extra hydration before, during, and after flights • Reduce training frequency, volume, and/or intensity 2–4 days postflight if traveling across 3 \geq time zones in eastward direction or 4 \geq time zones in westward direction • If possible, plan motor skill training sessions in the morning, and strength and endurance sessions in the evening • Consider ergogenic modalities such as cryotherapy, compression tights, cold water immersion, contrast water therapy, and soft tissue massage • If possible, plan individual practices or workout sessions based upon the player's chronobiological profile • If arriving to a new time zone, avoid planning practices or workout sessions during the circadian nadir (2–4 PM and 2–4 AM at the departure time) until players are fully adapted to the new time zone 	<ul style="list-style-type: none"> • Validity and reliability of recovery practices during traveling in NBA players across one or multiple seasons, encompassing both behavioral and pharmacological strategies • Longitudinal studies on travel fatigue in basketball encompassing preflight, in-flight and postflight assessments

(continued)

Table 74.1 (continued)

<i>Long distance traveling in basketball</i>			
	Scientific evidence	Practical applications	Future research
Sleep	<ul style="list-style-type: none"> • Frequent air traveling may induce sleep deprivation or disorientation between the circadian system and the environment • A desynchronized circadian rhythm impedes the ability for muscles to accumulate protein synthesis, and thus the ability of skeletal muscle to adapt and repair • The body’s core temperature takes approximately 1 day for each time zone crossed to adapt completely to the new time zone • The greater the number of time zones traveled, the more difficult it is for an athlete to adapt to a new time zone • Air travel increases melatonin levels on flight days in non-athletes regardless of the travel direction • High-volume training may exacerbate sleep disturbance (e.g., preseason period) • It tends to be easier to adjust to a circadian phase delay (e.g., eastwards travel) than a phase advance (e.g., westwards travel) as the standard duration of the circadian rhythm is approximately 24.5 h • Late-night social media use negatively impacts next-day performance in NBA players 	<ul style="list-style-type: none"> • Include sleep history and a month’s worth of sleep logs in the preseason test battery as well as a month’s worth of sleep logs to identify players who are at high risk for sleep issues • Monitor sleep through actigraphy as well as valid surveys such as the Pittsburgh Sleep Quality Index, Likert scale, Liverpool Jet-Lag Questionnaire or RESTQ • Prior to long-distance travel, educational sessions should be considered to communicate effective strategies moving forward <ul style="list-style-type: none"> – For example, the coaching staff should be advised to adopt the timing of their practice sessions to the time zone of the prospective destination. Additionally, an evening flight should be advised when traveling in eastward direction • Consider the following in-flight strategies to promote relaxation: <ul style="list-style-type: none"> – Eyeshades and earplugs; – On-board meal intake according to the destination time; – Bring custom meals; – Modify the time on personal watch or phone to the destination time as soon as boarding the plane; • Consume a high-carbohydrate, low-protein meal in the evening • Consume of a high-protein, low-carbohydrate meal in the morning • If traveling seems to disturb your sleep, consider taking 20–30 min naps during the circadian nadir of the departure time • If traveling across 3 ≥ time zones in eastward direction or traveling across 4 ≥ time zones in westward direction, seek or avoid light according to a jet leg calculator • Focus on physical activity, eating, and social contact during daylight time in the new time zone • Avoid late-night tablet use or any form of blue light exposure before bed 	<ul style="list-style-type: none"> • Include exogenous and endogenous parameters to measure the circadian rhythms in NBA players, such as salivary melatonin, adrenaline, light exposure, and body temperature • Standardized protocols for the usage of wearable devices and progressive strategies to help with sleep disturbances induced by traveling, such as light-blocking glasses, exercise regimes, caffeine consumption, food intake, melatonin prescriptions, as well as the use of other chronobiotics and chronohypnotics • Definition of parameters inferred by actigraphy, polysomnography, as well as consistency in sleep-quality algorithms and self-perceived sleep questionnaires

Table 74.1 (continued)

<i>Long distance traveling in basketball</i>			
	Scientific evidence	Practical applications	Future research
Injury propensity	<ul style="list-style-type: none"> • Competing in away games increases regular season injury risk in the NBA • Reducing minutes played per game does not influence risk of injury in NBA players • When sleeping <8 h, the immune system becomes suppressed • Dry pressurized air cabin conditions during the flight or acute exposure to altitude may exacerbate a suppressed immune system • Prolonged sitting increases the risk for venous thromboembolism • The annual risk of deep vein thromboembolism in non-athletes increases with 12% if one long-haul flight is taken yearly. This increased risk may remain up to 2 weeks after a long-haul flight 	<ul style="list-style-type: none"> • Avoid caffeine and alcohol during the flight to mitigate fluid loss and the suppression of the immune system. Instead, drink other beverages, approximately 200 ml extra in a 12-h flight • Include a baseline assessment during the preseason medical screening to gain insight into the risk for deep vein thromboembolism in each player and staff member • Perform flexibility exercises (e.g., FitFlight) during flights (when awake) • Advise players on an individual basis according to their chronotype, age, and level of fitness • Ensure 8 h of quality sleep before, during, and after trips • Ensure proper food hygiene, and avoid uncooked foods 	<ul style="list-style-type: none"> • Explore which travel-related biomarkers indicate an increased risk for injury or health problems in basketball. For example: <ul style="list-style-type: none"> – Internal load variables: Psychometric questionnaires, salivary immunoglobulin, vitamin D, cortisol, testosterone, pro-inflammatory, and anti-inflammatory cytokines – External variables: Type of schedule, travel duration, travel direction, and altitude • The magnitude of influence by the NBA ecosystem on the risk of its athletes and employees to being diagnosed with venous thromboembolism • The analysis of preflight, in-flight, and postflight interventions (e.g., FlightFit) and how these interventions play a role on the immune system, hydration status, and blood circulation of NBA players and employees who travel with the team

(continued)

Table 74.1 (continued)

<i>Long distance traveling in basketball</i>			
	Scientific evidence	Practical applications	Future research
Mood state	<ul style="list-style-type: none"> • Traveling tends to impair mood and loss of motivation all of which can affect recovery • Altered mood states after flying may remain up to 24 h after the flight • Sleep disruption may further exacerbate any mood fluctuations • Physical activity has demonstrated to improve mood state in athletes immediately after flying • Extending sleep over a 3-week period may improve energy, mood state, and mental preparedness, especially in sleep-deprived basketball players 	<ul style="list-style-type: none"> • Monitor subjective loads during travel periods based on evidence-based surveys such as the Acute Recovery and Stress Scale (ARSS) and the REST-Q Sport. • If traveling across $3 \leq$ time zones in eastward direction or $4 \leq$ time zones in westward direction, consider intaking 50–200 mg of caffeine as required • If traveling across $3 \geq$ time zones in eastward direction, or traveling across $4 \geq$ time zones in westward direction, consider intaking 50–200 mg of caffeine in late afternoons and in the minutes before a nap • Avoid unnecessary psychological stress by applying a proactive mindset, such as adjusting your watch as soon as boarding, and if the budget allows, travel managers should incorporate 1 day of adjustment period for each time zone crossed. Additionally, inform family and friends about the time difference to avoid calls during inappropriate times 	<ul style="list-style-type: none"> • Validity and reliability of psychometric surveys specifically in professional basketball and during travel periods • The effectiveness of intervention strategies on mood state disturbances in NBA players induced by traveling, such as napping and sleep extension

References

1. Sampaio J, McGarry T, Calleja-González J, Sáiz SJ, Alcázar XS, Balciunas M. Exploring game performance in the National Basketball Association using player tracking data. *PLoS One*. 2015;10:e0132894.
2. Official NBA Statistics and Advanced Analytics. 2018. <https://www.stats.nba.com>. Accessed 15 Aug 2018.
3. McLean BD, Strack D, Russell J, Coutts AJ. Quantifying physical demands in the National Basketball Association (NBA): challenges in developing best-practice models for athlete care and performance. *Int J Sports Physiol Perform*. 2019;4:414–20.
4. Wilke J, Niederer D, Vogt L, Banzer W. Head coaches' attitudes towards injury prevention and use of related methods in professional basketball: a survey. *Phys Ther Sport*. 2018;32:133–9.
5. Lewis M. It's a hard-knock life: game load, fatigue, and injury risk in the National Basketball Association. *J Athl Train*. 2018;53:503–9.
6. The Official Site of the NBA. 2018. <https://www.nba.com>. Accessed 15 Aug 2018.
7. NBA Advanced Stats and Analytics. 2018. <https://www.nbasavant.com>. Accessed 15 Aug 2018.
8. Belk JW, Marshall HA, McCarty EC, Kraeutler MJ. The effect of regular-season rest on play-off performance among players in the National Basketball Association. *Orthop J Sports Med*. 2017;5:2325967117729798.
9. Teramoto M, Cross C, Cushman D, et al. Game injuries in relation to game schedules in the National Basketball Association. *J Sci Med Sport*. 2017;20:230–5.
10. Philbrick JT, Shumate R, Siadaty MS, Becker DM. Air travel and venous thromboembolism: a systematic review. *J Gen Intern Med*. 2007;22:107–14.
11. Drakos MC, Domb B, Starkey C, Callahan L, Allen A. Injury in the National Basketball Association: a 17-year overview. *Sports Health*. 2010;2:284–90.
12. Coste O, Van Beers P, Touitou Y. Hypoxia-induced changes in recovery sleep, core body temperature, urinary 6-sulphatoxymelatonin and free cortisol after a simulated long-duration flight. *J Sleep Res*. 2009;18:454–65.
13. Reilly T. Ergonomics in sport and physical activity: enhancing performance and improving safety. 1st ed. Champaign, IL: Human Kinetics; 2010. p. 75–95.
14. Roy J, Forest G. Greater circadian disadvantage during evening games for the National Basketball

- Association (NBA), National Hockey League (NHL) and National Football League (NFL) teams travelling westward. *J Sleep Res*. 2017;27:86–9.
15. Leatherwood WE, Dragoo JL. Effect of airline travel on performance: a review of the literature. *Br J Sports Med*. 2013;47:561–7.
 16. Forbes-Robertson S, Dudley E, Vadgama P, et al. Circadian disruption and remedial interventions. *Sports Med*. 2012;42:185–208.
 17. Bishop D. The effects of travel on team performance in the Australian national netball competition. *J Sci Med Sport*. 2004;7:118–22.
 18. Samuels CH. Jet lag and travel fatigue: a comprehensive management plan for sport medicine physicians and high-performance support teams. *Clin J Sport Med*. 2012;22:268–73.
 19. Manfredini R, Manfredini F, Fersini C, Conconi F. Circadian rhythms, athletic performance, and jet lag. *Br J Sports Med*. 1998;32:101–6.
 20. Moore S, Scott J. Beware thin air: altitude's influence on NBA game outcomes. *JUR*. 2013;4:11–7.
 21. Fowler PM, McCall A, Jones M, Duffield R. Effects of long-haul transmeridian travel on player preparedness: case study of a national team at the 2014 FIFA World Cup. *J Sci Med Sport*. 2017;20:322–7.
 22. Fowler P, Duffield R, Vaile J. Effects of simulated domestic and international air travel on sleep, performance, and recovery for team sports. *Scand J Med Sci Sports*. 2015;25:441–51.
 23. Steenland K, Deddens JA. Effect of travel and rest on performance of professional basketball players. *Sleep*. 1997;20:366–9.
 24. Nédélec M, McCall A, Carling C, et al. Recovery in soccer. *Sports Med*. 2013;43:9–22.
 25. Reilly T. How can travelling athletes deal with jet-lag? *Kinesiology*. 2009;41:128–35.
 26. Waterhouse JA, Edwards B, Nevill A, et al. Identifying some determinants of “jet lag” and its symptoms: a study of athletes and other travellers. *Br J Sports Med*. 2002;36:54–60.
 27. Palmer BF. Physiology and pathophysiology with ascent to altitude. *Am J Med Sci*. 2010;340:69–77.
 28. Humphreys S, Deyerdmond R, Bali I, Stevenson M, Fee JP. The effect of high altitude commercial air travel on oxygen saturation. *Anaesthesia*. 2005;60:458–60.
 29. Lindgren T. Cabin air quality in commercial aircraft (PhD thesis). Uppsala, Sweden: Uppsala University; 2003.
 30. Reilly T, Edwards B. Altered sleep–wake cycles and physical performance in athletes. *Physiol Behav*. 2007;90:274–84.
 31. Hoffman JR, Im J, Rundell KW, et al. Effect of muscle oxygenation during resistance exercise on anabolic hormone response. *Med Sci Sports Exerc*. 2003;35:1929–34.
 32. Kraemer WJ, Hooper DR, Kupchak BR, et al. The effects of a roundtrip trans-American jet travel on physiological stress, neuromuscular performance, and recovery. *J Appl Physiol*. 2016;121:438–48.
 33. Meir R. Managing transmeridian travel: guidelines for minimizing the negative impact of international travel on performance. *Strength Cond J*. 2002;24:28–34.
 34. Sasso M. 2015. <https://www.bloomberg.com/news/articles/2015-07-06/nba-players-get-roomier-chartered-jets-as-delta-air-adds-teams>. Accessed 28 June 2018.
 35. Atkinson G, Reilly T. Circadian variation in sports performance. *Sports Med*. 1996;21:292–312.
 36. Montgomery PG, Pyne DB, Hopkins WG, et al. The effect of recovery strategies on physical performance and cumulative fatigue in competitive basketball. *J Sports Sci*. 2008;26:1135–45.
 37. Entine OA, Small DS. The role of rest in the NBA home-court advantage. *J Quant Anal Sports*. 2008;4:6.
 38. Jones MB. Home advantage in the NBA as a game-long process. *J Quant Anal Sports*. 2007;3:2.
 39. Sack RL. Clinical practice. Jet lag. *N Engl J Med*. 2010;362:440–7.
 40. The Gatorade Sports Science Institute. 2018. <https://www.gssiweb.org>. Accessed 15 Aug 2018.
 41. Reilly T. Travel and jet lag. Melbourne, Australia: First Australian Science in Football Congress; 1994.
 42. Delextrat A, Calleja-González J, Hippocrate A, Clarke ND. Effects of sports massage and intermittent cold-water immersion on recovery from matches by basketball players. *J Sports Sci*. 2013;31:11–9.
 43. Delextrat A, Hippocrate A, Leddington-Wright S, Clarke ND. Including stretches to a massage routine improves recovery from official matches in basketball players. *J Strength Cond Res*. 2014;28:716–27.
 44. Youngstedt SD, O'connor PJ. The influence of air travel on athletic performance. *Sports Med*. 1999;28:197–207.
 45. Fullagar HH, Duffield R, Skorski S, et al. Sleep and recovery in team sport: current sleep-related issues facing professional team-sport athletes. *Int J Sport Physiol*. 2015;10:950–7.
 46. Reilly T, Waterhouse J. Sports performance: is there evidence that the body clock plays a role? *Eur J Appl Physiol*. 2009;106:321–32.
 47. Reilly T, Waterhouse J, Edwards B. Jet lag and air travel: implications for performance. *Clin Sports Med*. 2005;24:367–80.
 48. Halson SL. Nutrition, sleep and recovery. *Eur J Sport Sci*. 2008;8:119–26.
 49. Samuels C. Sleep, recovery, and performance: the new frontier in high-performance athletics. *Neurol Clin*. 2008;26:169–80.
 50. Taylor SR, Rogers GG, Driver HS. Effects of training volume on sleep, psychological, and selected physiological profiles of elite female swimmers. *Med Sci Sport Exer*. 1997;29:688–93.
 51. Skein M, Duffield R, Minett G, et al. The effect of overnight sleep deprivation after competitive rugby league matches on postmatch physiological and

- perceptual recovery. *Int J Sport Physiol Perform.* 2013;8:556–64.
52. Sargent C, Halson S, Roach GD. Sleep or swim? Early-morning training severely restricts the amount of sleep obtained by elite swimmers. *Eur J Sport Sci.* 2014;14:310–5.
 53. Pollard R, Gómez MA. Components of home advantage in 157 national soccer leagues worldwide. *Int J Sport Exerc Psychol.* 2014;12:218–33.
 54. Goumas C. Home advantage in Australian soccer. *J Sci Med Sport.* 2014;17:119–23.
 55. Claudino J, Gabbet T, de Sá SH, et al. Which parameters to use for sleep quality monitoring in team sport athletes? A systematic review and meta-analysis. *BMJ Open Sport Exerc Med.* 2019;5:e000475.
 56. Leathwood P. Circadian rhythms of plasma amino acids, brain neurotransmitters and behaviour. In: Arendt J, Minors D, Waterhouse J, editors. *Biological rhythms in clinical practice.* 1st ed. London: Butterworths; 1989. p. 136–59.
 57. Czeisler CA, Allan JS, Strogatz SH. Bright light resets the human circadian pacemaker independent of the timing of the sleep-wake cycle. *Science.* 1986;233:667–71.
 58. Waterhouse J, Reilly T. Managing jet lag. *Sleep Med Rev.* 2009;13:247–8.
 59. Mednick S, Cai D, Kanady J, Drummond S. Comparing the benefits of caffeine, naps and placebo on verbal, motor and perceptual memory. *Behav Brain Res.* 2008;193:79–86.
 60. Jones J, Kirschen G, Kancharla S, Hale L. Association between late-night tweeting and next-day game performance among professional basketball players. *Sleep Health.* 2019;5:68–71.
 61. Fowler PM, Knez W, Crowcroft S, Mendham AE, Miller J, et al. Greater effect of east vs. west travel on jet-lag, sleep and team-sport performance. *Med Sci Sports Exerc.* 2017;49:2548–61.
 62. Thornton HR, Miller J, Taylor L, et al. Impact of short-compared to long-haul international travel on the sleep and wellbeing of national wheelchair basketball athletes. *J Sports Sci.* 2017;36:1476–84.
 63. Roach GD, Rogers M, Dawson D. Circadian adaptation of aircrew to transmeridian flight. *Aviat Space Environ Med.* 2002;73:1153–60.
 64. Halson SL. Sleep in elite athletes and nutritional interventions to enhance sleep. *Sports Med.* 2014;44:13–23.
 65. Cohen S, Doyle WJ, Alper CM, Janicki-Deverts D, Turner RB. Sleep habits and susceptibility to the common cold. *Arch Intern Med.* 2009;169:62–7.
 66. Kelman CW, Kortt MA, Becker NG, et al. Deep vein thrombosis and air travel: record linkage study. *Br Med J.* 2003;327:1072.
 67. Heit JA, Silverstein MD, Mohr DN, et al. Risk factors for deep vein thrombosis and pulmonary embolism: a population-based case-control study. *Arch Intern Med.* 2000;160:809–15.
 68. Geerts WH, Heit JA, Clagett GP, et al. Prevention of venous thromboembolism. *Chest.* 2001;119:132S–75S.
 69. Jiang N. 2018. FlightFit: an application enabling air travelers to do stretches onboard. <https://scholarworks.rit.edu/cgi/viewcontent.cgi?article=11070&context=theses>. Accessed July 2019.
 70. Moreira A, Arsati F, de Oliveira Lima-Arsati YB, Simões AC, de Araújo VC. Monitoring stress tolerance and occurrences of upper respiratory illness in basketball players by means of psychometric tools and salivary biomarkers. *Stress Health.* 2001;27:e166–72.
 71. Papacosta E, Nassis GP. Saliva as a tool for monitoring steroid, peptide and immune markers in sport and exercise science. *J Sci Med Sport.* 2011;14:424–34.
 72. Willis KS. Vitamin D status & immune system biomarkers in athletes. Ann Arbor, Michigan: University of Wyoming, ProQuest Dissertations Publishing; 2008.
 73. Srinivasan V, Singh J, Pandi-Perumal SR, et al. Jet lag, circadian rhythm sleep disturbances, and depression: the role of melatonin and its analogs. *Adv Ther.* 2010;27:796–813.
 74. O'Connor PJ, Morgan WP. Athletic performance following rapid traversal of multiple time zones. *Sports Med.* 1990;10:20–30.
 75. Kölling S, Hitzschke B, Holst T, et al. Validity of the acute recovery and stress scale: training monitoring of the German junior national field hockey team. *Int J Sports Sci Coach.* 2015;10:529–42.
 76. Bresciani G, Cuevas MJ, Garatachea N, Molinero O, Almar M, et al. Monitoring biological and psychological measures throughout an entire season in male handball players. *Eur J Sports Sci.* 2010;10:377–84.
 77. Gastin PB, Meyer D, Robinson D. Perceptions of wellness to monitor adaptive responses to training and competition in elite Australian football. *J Strength Cond Res.* 2013;27:2518–26.
 78. Taylor K, Chapman D, Cronin J, Newton MJ, Gill N. Fatigue monitoring in high performance sport: a survey of current trends. *J Aust Strength Cond.* 2012;20:12–23.
 79. Casper RC. Exercise and mood. *World Rev Nutr Diet.* 1993;71:115–43.
 80. Mah CD, Mak KE, Kezirian EJ, et al. The effects of sleep extension on the athletic performance of collegiate basketball players. *Sleep.* 2011;34:943–50.
 81. Fuller CW, Taylor AE, Raftery M. Does long-distance air travel associated with the sevens world series increase players' risk of injury? *Br J Sports Med.* 2015;49:458–64.



General Considerations in Basketball: Court Type, Shoes, and Protective Gear

75

Priscilla Tu and John Travis Nelson

75.1 Introduction

More than 300 million people enjoy playing and watching the game of basketball worldwide. While the rules of the game have remained the same, many aspects surrounding the game have changed since the sport was invented in the 1890s. Its popularity has led businessmen and researchers alike to explore innovative developments surrounding the different features of the sport's environment. From the court materials to clothing and shoes to protective gear, technological advances have been applied to try and improve the game of basketball. The following chapter discusses the latest research regarding these aspects of basketball, noting which trends have merit and which are more of a fad.

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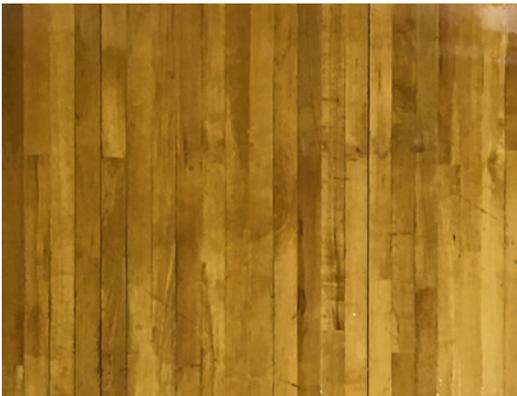
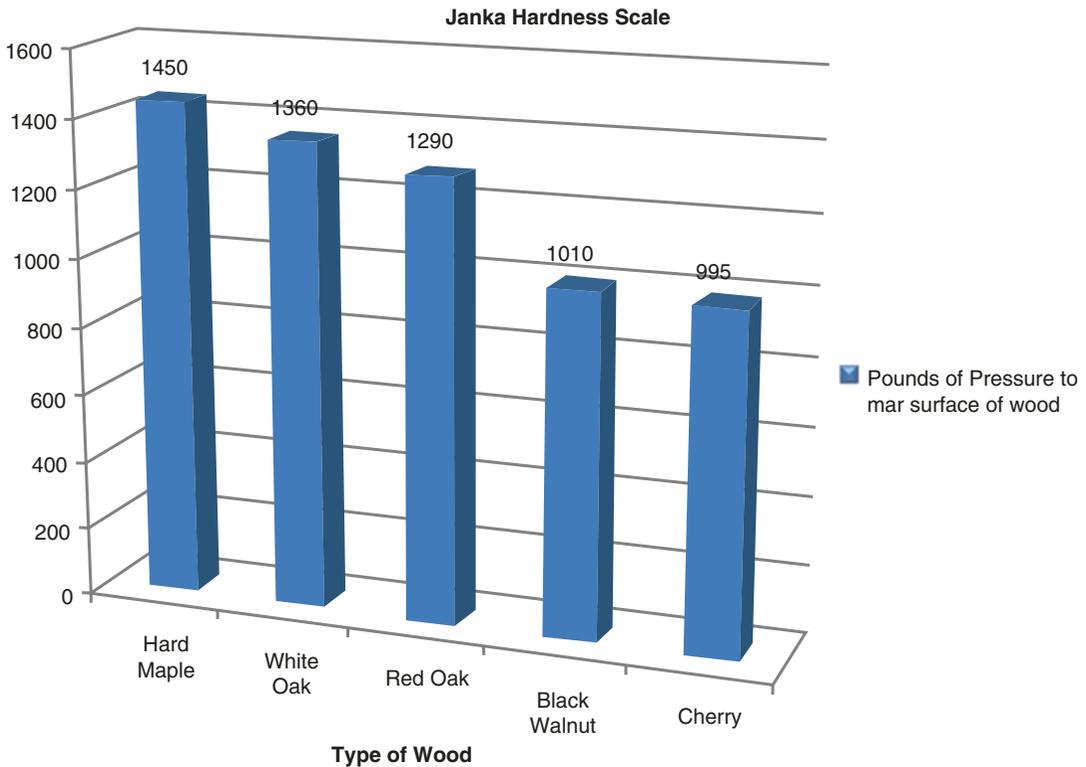
75.2 Court

When Dr. James Naismith wrote down the first rules of basketball in 1892, he wished that the new game could be played in any environment—indoors or outdoors and on any surface whether uneven or smooth. The gymnasium where Naismith played the first game was in the 5-year-old Christian Workers Building. The gymnasium had a 49 feet × 41 feet floor that was equipped with all kinds of equipment including climbing ropes, dumbbells, parallel bars, and flying rings.

75.2.1 Early Courts

The Gymnasium floor was made of hard or rock maple [1]. Maple flooring, at that time, was popular because this type of wood was plentiful and therefore inexpensive. It was also more durable and stable. Maple flooring is harder than oak, walnut, and cherry based on the Janka Hardness Scale [2] (Table 75.1), which measures the hardness of species of hardwood. The scale is based on the pounds of pressure required to mar the wood's surface. It also has a tighter grain that makes it easier to clean, refurbish, and maintain.

The ideal basketball court must be solid and consistent throughout to ensure that the basketball will bounce the exact same way without recoil or dampening. The smallest difference can impact the game. The actual area of courts used

Table 75.1 Hardness classification of the wood court**Picture 75.1** Maple hardwood planks

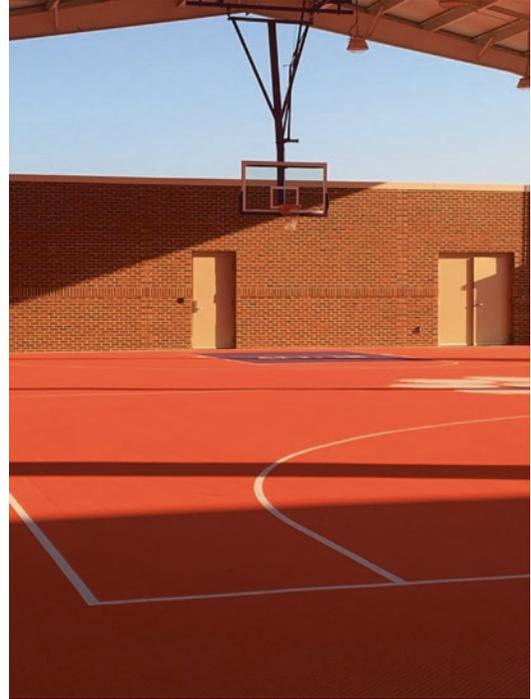
by the NCAA and NBA measures 94 feet by 50 feet [3]. However, most courts incorporate a larger perimeter measuring 140 feet by 70 feet. The hard rock maple planks measure thicker than $\frac{3}{4}$ of an inch thick (Picture 75.1). Therefore, it requires 80–100 trees to construct a single hardwood court.

75.2.2 Polyurethane

There have been experiments with other types of surfaces to play the sport, including canvas and plastic [1]. The development of synthetic surfaces led to the development of rubberized polyurethane flooring. The flooring had somewhat of a grayish coloration. This became popular in the 1970s with incorporation into many NCAA university arenas. The NBA Houston Rockets were the only team to use the material from 1971 to 1975. A small study determined that the hardness of this playing surface is associated with higher levels of bone mineral density and content in growing girls playing basketball [4]. However, the material is much more unforgiving on the knees and elbows, causing it to lose popularity. It is important to note that many high school and recreational gymnasiums still use this flooring, which can affect injuries that can occur. None of this synthetic material is currently being used in the NCAA or NBA at this time.



Picture 75.2 Maple hardwood court



Picture 75.3 Outdoor polyurethane court

75.2.3 Maple Hardwood

As the sport developed, it became evident that maple floors are ideal for the game of basketball [1, 2, 5]. It provides a stable surface for dribbling. The light color of the wood provides a good contrast to the orange basketball as well. Due to being a tighter grained product, the wood is less likely to expand and contract with changes in temperatures and humidity, and it is largely resistant to splintering (Picture 75.2). Maple also provides shock-absorbing qualities to protect players when jumping. This minimizes fatigue, damage to joints, and potential injury. However, there are not any research studies to confirm these benefits. Currently, 29 of the 30 NBA teams play on hard maple (or *Acer saccharum*) floors (the Boston Celtics is made of red oak and the wood color is darker than other surfaces) [5].

As the sport developed, it became evident that maple floors are ideal for the game of basketball.

75.2.4 Mobility

New technology has allowed maple floor courts to be delivered in sections and assembled quickly. This has allowed courts to be constructed in larger arenas for larger tournament crowds. It has also opened the opportunity to play basketball in new environments as the flooring did not need to be maintained within that environment. The Carrier Classic was a college basketball game played on November 11, 2011, between Michigan State and North Carolina on the deck of a US Navy aircraft carrier. The first outdoor game was played between the Phoenix Suns and the Milwaukee Bucks on September 24, 1972, in the Hiram Bithorn Baseball Stadium in San Juan, Puerto Rico. The WNBA had one game held at Arthur Ashe Stadium in Flushing, New York, in 2008. However, weather caused difficulty with these games as debris, obstructions, and dampness on the court are dangerous to the athletes [6], which led to rescheduling these outdoor games (Picture 75.3).

75.2.5 Maintenance

Court maintenance is important in increasing the longevity of the flooring. Ideally, floors should be wet mopped every evening with dry mopping between each activity. Most basketball courts should be buffed, screened, and recoated yearly to ensure the flooring performs as it should. However, if utilized for activities other than sports, it may need this more often [2]. Floor maintenance is important as poorly maintained or uneven surfaces can cause an athlete injury [6]. The National Basketball Association (NBA) requires teams to replace their floor every 10 years [5].

75.3 Clothing and Protective Gear

The official National Basketball Association rule book [3] addresses the topic of clothing and equipment that may be used by players during the game. Based on the 2018–2019 rule book, players are not permitted to wear any type of jewelry. Officials should “not permit any player to wear equipment which, in their judgment, is dangerous to other players. Any equipment that is hard substance (cast, splints, guards, and braces) must be padded or foam covered and have no exposed sharp or cutting edge.” All equipment must be appropriate for basketball. Unnatural equipment designed to increase a player’s height, reach, or advantage is prohibited.

Other than these topics, the discussion of additional gear and athletic shoes is not included. With the advances in technology and research, athletic clothing companies advertise new articles of clothing to players with the hopes of improving their performance with using their products. Limited research is available to determine if these shoes, braces, compression clothing, or other gear improves an athlete’s athletic performance or protects them from injury in the game of basketball.

75.4 Basketball Shoes

75.4.1 Weight

Though hypothesized that the weight of the shoe could influence performance, not much research

has been conducted on the hypothesis. One study noted that performance in lighter shoes was significantly increased by 2% (vertical jump 2%, $P < 0.001$; shuffle cut 2.1%, $P < 0.001$) compared with individuals wearing heavier shoes [7]. However, this difference was only noted in the participants who were aware of the heaviness of the shoe they were wearing. In the blinded group, participants were unable to perceive the shoe-weight variation between conditions, and there were no significant differences in vertical-jump and shuffle-cut performance across shoes. Therefore, the differences in performance of the participants who were aware of the weight of the shoes were most likely due to psychological effects surrounding their expectancies toward the difference in weight of the shoes used. Another study found shoe mass to have no effect on performance [8].

75.4.2 Collar Height

Ankle sprains are common injuries for basketball athletes; hence experiments in shoe designs that attempt to provide more stability, particularly high collar height shoes or high-tops, have emerged. One study examined the effect of varying collar heights (Pictures 75.4 and 75.5) and heel counter-stiffness on ankle stability (using sidestep cutting) and athletic performance (using jumping and agility tasks). It concluded that collar height may play a role in lateral stability of the ankle, but neither collar height nor heel counter-stiffness affected



Picture 75.4 High top shoe



Picture 75.5 Low top shoe

performance [9]. Another study corroborated the findings, noting a smaller ankle range of motion, particularly in ankle inversion and eversion, with no effect on the athlete's cutting performance [10]. A third study also noted that a higher shoe collar height can help to provide ankle stability but suggested that decreased range of motion meant less force exerted at the ankle which could be advantageous with jumping performance [11].

A higher collared shoe, or high-top, can be beneficial in those athletes requiring more ankle stability in their shoe.

A different study evaluated collar height effects on ankle inversion kinematics and pre-landing EMG activation of ankle evertor muscles during landing on a tilted surface. This study determined no decreased inversion with high collar shoes and perhaps a detrimental effect in establishing and maintaining ankle joint stability [12]. While no clear recommendation can be made regarding the use of high collar shoes to prevent ankle sprains or improve athletic performance, it does appear a higher collared shoe would be beneficial in those requiring more ankle stability in their shoe.

75.4.3 Air Cells

Another shoe aspect found to affect ankle injuries were shoes with air cells. One study noted that

players wearing shoes with air cells in the heel were 4.3 times more likely to injure an ankle than those without air cells [13]. The study also noted that athletes with a history of ankle sprain were almost five times more likely to sustain another sprain and those who did not stretch were 2.6 times more likely to injure an ankle [13].

75.4.4 Shoe Stiffness

Shoe stiffness is another aspect of shoe design being researched with regard to athletic performance and injury. One study noted that increased forefoot stiffness demonstrated 2.9% higher jump height while not significantly changing sprint times [14]. Another study demonstrated that a higher forefoot bending stiffness had a moderate benefit on sprinting and cutting performance [15]. However, when researching forefoot stiffness in relation to tendon strains, the least stiff shoe was found to have a 5.3% reduction in Achilles tendon strains during landing when compared to the stiffest shoe [15]. Shoe stiffness did not affect jump height or patellar tendon strain injury rates in this study. It appears that while shoes with a stiffer forefoot may have some effect in improving aspects of athletic performance, they may also alter the rate of Achilles tendon strains by altering the center of pressure location.

A study investigating the effect of midsole stiffness on performance and perception of stability found no difference in performance on any of the tested movements between the varying stiffness of midsoles [16]. However, a majority of participants preferred the softer midsole, perceiving more stability with those shoes. Another study found similar findings of no change in performance with varying midsole stiffness, regardless of the weight of the athlete [17]. However, participants in this study also preferred the softer midsole shoes.

75.4.5 Cushioning

Tibia stress fractures are common in basketball players and are associated with high tibial

shock and impact loading. Shoe cushioning properties can affect the amount of tibial shock, impact loading, and knee kinematics in basketball players. One study [18] examined different cushioning and the effects of landing from different heights. Results showed no significant interactions between landing height and shoe conditions on tibial shock, impact peak, mean loading rate, maximum knee flexion angle, and total ankle range of motion. However, there was found to be greater tibial shock, impact peak, mean loading rates, and total ankle range of motion with higher landing heights. The regular-cushioned shoes had significantly greater tibial shock than the better and best-cushioned shoes. Similarly, another study [19] found the regular-cushioned shoe to have greater tibial shock and impact loading. However, the best-cushioning shoe also had higher shock and impact than the better-cushioned shoe. Comfort rating of the shoe can also help to estimate the level of impact loading. One study [20] suggested that a highly cushioned shoe is more helpful in decreasing peak impact and muscle activation in landings from unexpected situations of impact.

75.4.6 Outsole Traction

In one study [8], the amount of outsole traction was evaluated using three different amounts of traction, varying by 20% each. Outsole traction was found to have the largest influence on performance in all categories, maximal effort sprints, vertical jumps, and most significantly with a cutting drill performance. Participants performed significantly better with increasing amounts of traction.

Outsole traction had the largest influence on performance in all categories (maximal effort sprints, vertical jumps, and cutting drill) of performance.



Picture 75.6 Knee and ankle brace

75.5 Braces

75.5.1 Ankle

Ankle injuries are particularly prevalent in female basketball players. One study [21] compared the effects of a lace-up brace (ASO), a hinged brace (Active T2), and no bracing. The hinged ankle brace significantly restricted peak ankle inversion compared to the ASO or no bracing. It also reduced ankle and knee joint compressive forces at the time of peak ankle dorsiflexion and reduced knee anterior shear forces in the deceleration phase and at peak ankle dorsiflexion. This suggests that the hinged ankle brace may be a better prophylactic ankle support for female basketball players (Picture 75.6). Another study [22] noted that during a 16-week basketball prophylactic ankle-brace intervention, participants showed significantly improved dynamic postural control and single-limb functional tests over time.

The hinged ankle brace may be a better choice of prophylactic ankle support for female basketball players from a biomechanical perspective.

Following an ankle injury, athletes sometimes brace the injured ankle in hopes of minimizing the likelihood of suffering a recurring injury. Therefore, a study [23] examined the effects of wearing an Ankle Stabilizing Orthosis (ASO) ankle brace unilaterally on the dominant side on bilateral ankle joint kinetics and kinematics and peroneus longus EMG activity during rebounding. It demonstrated that wearing an ankle brace on the dominant side reduced ankle and foot inversion and increased ankle inversion moments, with no difference noted in peroneus longus activity. No differences were observed on the non-braced limb. This suggests that wearing a brace unilaterally can protect the braced ankle without posing a risk to the unbraced leg. Another study [24] reproduced these results utilizing different braces, showing that any braced condition shows the benefit.

75.5.2 Foot

Foot orthoses are often used to support those with flat feet to help improve normal foot function. One study [25] examined its effect on jump performance in basketball players without flat feet, measuring countermovement jump and standing broad jump performance as well as lower limb biomechanics. The study demonstrated that the foot orthoses affected biomechanics of take-off, but did not actually affect jumping performance.

75.6 Mouth Guards

Mouth guards have been shown to be effective, inexpensive, easy to use, and readily available. However, it is vastly underutilized in sports such as basketball. One study [26] noted that while 98% of coaches believe mouth guards prevent

dental injuries, only 47% of them suggest use. Even in players who had previously sustained a dental injury while playing, only 6.3% used a mouth guard though 95% of them believed them to help prevent dental injuries. Common reasons cited for not using one were discomfort (37.7%), difficulty breathing (7.3%), and difficulty talking (6.4%). Another study [27] noted that the primary reasons high school athletes were not wearing them was that they were not required (65.3%) and that they could not breathe or talk while wearing one (61.5%). Efforts should be made to improve athletes' use of mouthguards to help prevent sports-related dental injuries.

Efforts should be made to improve athletes' use of mouthguards to help prevent sports-related dental injuries.

75.7 Compression Clothing

While compression garments are often advertised to improve performance, recovery, or comfort, little research is available regarding their benefits. One study [28] analyzed hip and knee kinematics and kinetics during a drop vertical jump while wearing KNEE-Tec compression leggings. The results demonstrated significant reduction of hip abduction range of motion but no differences in peak hip abduction, knee valgus, peak knee valgus, peak hip abduction moment, or peak knee valgus moment. While it appears that the compression garment does alter movement patterns minimally and potentially aid in knee injury prevention by altering jumping mechanics, further research is still needed.

Another study [29] examined lower body compression garments in collegiate basketball players. The results suggested that these garments enhanced mean power output, anaerobic capacity, and total work across successive anaerobic tests (WAnT) while also noting a lower perceived exertion rating. It showed no effect on peak power, anaerobic power, or fatigue index. Previous evidence has noted that performance on

the specific anaerobic tests done in the study may predict performance in basketball-specific skills, so one can postulate improvement in basketball performance with lower body compression garments. However, further studies are needed, focusing on physiological variables, muscle activation patterns, and adaptations over time.

75.8 Conclusion

As has been proven in the preceding chapter, newer does not always mean better. Though there continues to be new court floors available, such as the Eco-Friendly court, the maple hardwood has withstood the test of time and remains the court flooring of choice. Mouthguards have been available since basketball was invented and has been proven to prevent against sports-related dental injuries. However, few athletes use them, and efforts should be made to increase their use. As new technologies for shoes and clothing continue to develop, it is necessary to understand their purpose and to explore how they can affect an athlete's performance and more importantly, their impact on injury. For shoes, the comfort of the shoe to the athlete, outsole traction, and if needed, higher collars are important. Compression garments are showing some promise for injury prevention, but further studies are still needed. With regard to orthotics, a hinged ankle brace can be considered for prophylactic ankle support, particularly in female athletes, but foot orthoses have not been found to be very beneficial.

References

1. Caldwell D. Hard maple: why basketball's perfect surface has lasted more than a century. April 2017. <https://www.theguardian.com/sport/2017/apr/05/hard-maple-basketball-surface-nba>.
2. Bryant C. What makes sports flooring different? <https://home.howstuffworks.com/home-improvement/home-diy/flooring/what-makes-sports-flooring-different3.htm>.
3. <https://official.nba.com/rulebook>.
4. Ubago-Guisado E, Garcia-Unanue J, Lopez-Fernandez J, et al. Association of different types of playing surfaces with bone mass in growing girls. *J Sports Sci.* 2017;35(15):1484–92.
5. Newcomb T. Facts about floors: Detailing the process behind NBA hardwood courts. *Sports Illustrated.* 2015. <https://www.si.com/nba/2015/12/02/nba-hardwood-floors-basketball-court-celtics-nets-magic-nuggets-hornets>.
6. Basketball Injuries and Prevention in Claude T Moorman III (Ed.). Praeger handbook of sports medicine and athlete health volume two. Santa Barbara, CA: Praeger; 2011. p. 361–72.
7. Mohr M, Trudeau MB, Nigg SR, et al. Increased athletic performance in lighter basketball shoes: shoe or psychology effect? *Int J Sports Physiol Perform.* 2016;11(1):74–9.
8. Worobets J, Wannop JW. Influence of basketball shoe mass, outsole traction, and forefoot bending stiffness on three athletic movements. *Sports Biomech.* 2015;14(3):351–60.
9. Liu H, Wu Z, Lam WK. Collar height and heel counter-stiffness for ankle stability and athletic performance in basketball. *Res Sports Med.* 2017;25(2):209–18.
10. Weijie F, Ying F, Yu L, et al. The effect of high-top and low-top shoes on ankle inversion kinematics and muscle activation in landing on a tilted surface. *J Foot Ankle Res.* 2014;7:14.
11. Yang Y, Fang Y, Zhang X, et al. Does shoe collar height influence ankle joint kinematics and kinetics in sagittal plane maneuvers? *J Sports Sci Med.* 2017;16(4):543–50.
12. Lam GW, Park EF, Lee KK, et al. Shoe collar height effect on athletic performance, ankle joint kinematics and kinetics during unanticipated maximum-effort side-cutting performance. *J Sports Sci.* 2015;33(16):1738–49.
13. McKay GD, Goldie PA, Payne WR, et al. Ankle injuries in basketball: injury rate and risk factors. *Br J Sports.* 2001;35(2):103–8.
14. Lam WK, Lee WC, Lee WM, et al. Segmented forefoot plate in basketball footwear: does it influence performance and foot joint kinematics and kinetics? *J Appl Biomech.* 2018;34(1):31–8.
15. Firminger CR, Bruce OL, Wannop JW, et al. Effect of shoe and surface stiffness on lower limb tendon strain in jumping. *Med Sci Sports Exerc.* 2019;51(9):1.
16. Leong HF, Lam WK, Ng WX, et al. Center of pressure and perceived stability in basketball shoes with soft and hard midsoles. *J Appl Biomech.* 2018;34(4):284–90.
17. Nin DZ, Lam WK, Kong PW. Effect of body mass and midsole hardness on kinetic and perceptual variables during basketball landing manoeuvres. *J Sports Sci.* 2016;34(8):756–65.
18. Wei Q, Wang Z, Woo J, et al. Kinetics and perception of basketball landing in various heights and footwear cushioning. *PLoS One.* 2018;13(8):e0201758.
19. Lam WK, Liebenberg J, Woo J, et al. Do running speed and shoe cushioning influence impact loading and tibial shock in basketball players? *PeerJ.* 2018;6:e4753.
20. Fu W, Fang Y, Gu Y, et al. Shoe cushioning reduces impact and muscle activation during landings from

- unexpected, but not self-initiated, drops. *J Sci Med Sport*. 2017;20(10):915–20.
21. Klem NR, Wild CY, Williams SA, et al. Effect of external ankle support on ankle and knee biomechanics during the cutting maneuver in basketball players. *Am J Sports Med*. 2017;45(3):685–91.
 22. Crockett NJ, Sandrey MA. Effect of prophylactic ankle-brace use during a high school competitive basketball season on dynamic postural control. *J Sport Rehabil*. 2015;24(3):252–60.
 23. Dewar RA, Arnold GP, Wang W, et al. The effects of wearing an Ankle Stabilizing Orthosis (ASO) Ankle Brace on ankle joints kinetics and kinematics during a basketball rebounding task. *Foot*. 2019;40:34–8.
 24. Dewar RA, Arnold GP, Wang W, et al. Comparison of 3 ankle braces in reducing ankle inversion in a basketball rebounding task. *Foot*. 2019;39:129–35.
 25. Ho M, Kong PW, Chong LJ, et al. Foot orthoses alter lower limb biomechanics but not jump performance in basketball players with and without flat feet. *J Foot Ankle Res*. 2019;12:24.
 26. Tiryaki M, Saygi G, Yildiz SO, et al. Prevalence of dental injuries and awareness regarding mouthguards among basketball players and coaches. *J Sports Med Phys Fitness*. 2017;57(11):1541–7.
 27. Collins CL, McKenzie LB, Roberts KJ, et al. Mouthguard BITES (behavior, impulsivity, theory evaluation study): what drives mouthguard use among high school basketball and baseball/softball athletes. *J Prim Prev*. 2015;36(5):323–34.
 28. Zamporri J, Aguinaldo A. The effects of a compression garment on lower body kinematics and kinetics during a drop vertical jump in female collegiate athletes. *Orthop J Sports Med*. 2018;6:2325967118789955.
 29. Ballmann C, Hotchkiss H, Marshall M, et al. The effect of wearing a lower body compression garment on anaerobic exercise performance in division I NCAA basketball players. *Sports (Basel)*. 2019;7(6):pii: E144.



Ethical and Medico-Legal Issues in Injury Management and Return to Sport in Basketball

76

Jordan Rawlings and Blake Boggess

76.1 Introduction

The standards of care for providing medical services in sports medicine are rapidly evolving and expanding with each year. New position statements, updates of earlier position statements, new technologies, and new methods and modalities of treatment are introduced frequently. A variety of healthcare professionals are involved in sports medicine. These professionals include family physicians, orthopedic surgeons, internists, physiatrists, pediatricians, neurologists, physical therapists, athletic trainers, chiropractors, psychologists, dentists, nutritionists, dieticians, and physiologists. Although sports medicine services have been growing in recent years, so has the unprecedented growth of claims and lawsuits arising out of the practice of sports medicine. It behooves all members of the sports medicine team to become familiar with their potential legal and professional liabilities.

Since 1990, there has been a significant increase in sports medicine-related litigation [1, 2]. Because of the increasing economic benefits of playing sports, such as college schol-

arships or multimillion-dollar professional contracts, injured athletes have a strong incentive to seek compensation for harm caused by negligent sports medicine care rendered by team physicians, athletic trainers, and others.

76.2 Definition of Sports Medicine

Many consider the term sports medicine to apply to the healthcare professionals who provide professional services to those who participate in athletic activities. Sports medicine, a recognized medical subspecialty of the American Board of Medical Specialties, also includes prescribing exercise and recommending exercise equipment. The American Osteopathic Academy of Sports Medicine (AOASM) defines sports medicine as “that branch of the healing arts profession that uses a holistic, comprehensive team approach to the prevention, diagnosis, and adequate management (including medical, surgical and rehabilitative techniques) of sports and exercise-related injuries, disorders, dysfunctions, and exercise-related disease processes.”

The sports medicine team is usually, and should be, composed of several members from a variety of backgrounds and training. Given the broad range of people involved, it is important to develop an organized structure of responsibilities and duties of each member. Each member should

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also know their limitations, the standard of care, and the limitations to their practice.

76.3 The Standard of Care

Team physicians have a legal duty to conform to the standard of care corresponding to their specialty training. For example, an orthopedic surgeon should be held to the standard of an orthopedist certified in sports medicine if they are providing care for high-level athletes. A physician certified by the American Boards of Emergency Medicine, Internal Medicine, Family Practice, Pediatrics, or Physical Medicine and Rehabilitation may earn a certificate of added qualification in sports medicine by passing a written examination and is held to the standards conferred by this certification. Certification is often tied to their primary board certification.

In malpractice suits involving a medical specialist, the trend is to apply a national standard of care because national specialty certification boards exist to ensure standardized training and certification procedures. A national standard of care for team physicians is preferable because appropriate sports medicine care and treatment should not vary depending on the area of practice. The applicable legal standard of physician conduct is good medical practice within the physician's type of practice. What physicians in the same specialty commonly do generally serves as the standard by which a physician's actions are measured. What should have been done under the circumstances, not what is commonly done, determines applicable standard. Physicians have a legal obligation to keep up to date on new guidelines and advances in sports medicine, and they may be liable for using outdated treatment methods that no longer have a sound medical basis or that do not currently constitute appropriate care [3].

To determine acceptable medical practice, a medical expert may use their own education, training, and experience, as well as any relevant medical literature and medical association guidelines.

The standard of care may be difficult to establish, because of the many medical and allied

health professionals caring for athletes. The standard at any time is influenced by various sources, including published statements from professional associations, government policies, and state and national government regulations [4]. The amount of research and development of new treatment and techniques makes it challenging for the sports medicine provider to stay up to date. Without knowledge of the most current standards and the incorporation of these into operating procedures and protocols, the sports medicine team may be at risk for legal issues coming from personal injury lawsuits.

76.4 Role of a Team Physician

The National Collegiate Athletic Association (NCAA) Sports Medicine Handbook outlines the role of the team physician as follows: "The team physician and athletic health care team should assume responsibility for developing an appropriate injury prevention program and providing quality sports medicine care to injured student-athletes" [5]. The team physician is responsible for minimizing athletic injuries by thorough preparticipation screening, exemplary medical care using state-of-the-art technology, and careful evaluation of athletes before return to play is allowed.

The team physician is required to provide high-quality sports medicine care and to minimize personal and institutional liability. Specifically, the team physician's duties include determining: (1) the athlete's medical eligibility for participation; (2) the athlete's medical eligibility to resume athletic participation after injury; (3) the availability of medical services; (4) the supervision of personnel providing athletic healthcare services; (5) the selection of training practices with health implications; and (6) protection against legal liability [6] (Table 76.1).

Expert testimony in sports medicine litigation usually focuses on whether the practitioner adhered to the proper professional standard of care and whether their care or advice was below the standard of care and thereby negligently led to injury.

Table 76.1 The Duties of the Team Physician [5]

1.	Determine the athlete's medical eligibility for participation
2.	Decide the athlete's medical eligibility and when to resume athletic participation after injury
3.	Organize the availability of medical services
4.	Supervise personnel providing athletic healthcare services
5.	Advise on the selection of training practices with health implications
6.	Protect against legal liability

Professional teams and colleges usually hire a physician or a group of physicians to provide medical care to their athletes. Many high schools also select a physician to provide pre-participation physical examinations and emergency medical care to athletes participating in athletics. A team physician provides medical services to athletes that are arranged, or paid for, at least in part, by an institution or entity other than the patient or their parent or guardian [7]. Team physicians may or may not receive monetary compensation for their time. The team physician's primary responsibility is to provide care for the athlete and be mindful of the best interest of the athlete. Specific responsibilities may include providing preseason physical examinations; providing medical clearance for an athlete to play the sport; and diagnosing, treating, and rehabilitating athletic injuries. The team physician may also be responsible for overseeing all sports medicine services provided to a team's athletes and for the supervision of physician assistants, athletic trainers, student assistants, physical therapists, and nurses providing medical care to athletes.

Although one of the team physician's responsibilities is to avoid the unnecessary restriction of sports participation, their foremost duty should be to protect the athlete's health. Team physicians may face pressure from parents, coaches, team management, fans, or the athletes to provide medical clearance to participate or treatment enabling immediate return to play. However, the team physician's judgment should be governed by current medical practice, instead of the team's need for the services of the player or the athlete's desire to play.

76.5 Ethical Issues

Although ethics and the law are typically considered to be one and the same, it is important to note that they can and often do differ. The International Federation of Sports Medicine notes "Although it is desirable that the law be grounded in moral principles and that matters of moral importance should be given legal backing in many instances, not everything that is illegal is immoral and similarly not every immoral behavior is against the law. Thus when speaking of ethics in sports medicine, one is not concerned with etiquette or law, but with basic morality" [8]. When reflecting on what is the ethical thing to do, keep three principles in mind: (1) make the health of the athlete a priority, (2) never do harm, and (3) always allow for patient autonomy [8]. In general, sports medicine physicians face two main ethical dilemmas: confidentiality and conflicts of interest.

76.5.1 Confidentiality

Due to the responsibility sports medicine physicians have to the player and the team, confidentiality can be difficult to maintain. Team physicians often have contractual duties that require the sharing of player health with team management [9]. It is natural that coaches and owners within a sports organization want to know health concerns regarding their athletes. An athlete's health status impacts coaches' game plans and impacts organizations' future plans. Although athletes often sign paperwork regarding this issue, it may not be fully comprehended among all the other forms and contracts they are rushed to sign [9]. Both the athlete and team physician should have an understanding of the contractual obligations before any personal health information is discussed. Even with the contracts and forms that help with times where medical information is shared, there will be inevitable obscure situations. For example, when should sensitive personal health information like blood-borne infections, psychiatric conditions, and substance abuse disorders be shared? [9].

Additionally, athlete's personal health information often come under the scrutiny of the media, especially when injuries happen. The sports medicine physician must put the athlete's right to privacy first. In conjunction with the athlete, the physician can decide what information can be released for public distribution [8].

76.5.2 Conflicts of Interest

Because of the competitive environment, athletes often feel immense pressure to perform and succeed, even more so after sustaining an injury. Imagine the commonly referenced scenario where an athlete tears his/her medial meniscus. Although the tear is repairable, it presents two conflicting choices to the athlete. They could either (1) opt for a quick arthroscopic meniscectomy allowing them to return to play more rapidly albeit with a greater likelihood of developing osteoarthritis in that joint or (2) a meniscal repair resulting in a much longer recovery process but a better long-term outcome [10]. Athletes due to their unique situation because of contracts feel both mental and social pressure to perform and get better, so they can help the team and maintain themselves as a valuable member. Similarly, the physician may face outside pressures such as the coach or team owner extolling for one medical procedure or option. Helping the athlete understand the pros and cons of each medical option in both the short and long term is one of the most important ways physicians can ethically work within these conflicts of interest. Understanding the patient's right autonomy is the key to making a clear and ethical decision in a situation such as this [11]. In contrast to a normal clinical setting, informed consent becomes even more significant due to the external pressures and influences.

76.6 Practice and Game Coverage: Medico-Legal Obligations

The medico-legal aspects of sport participation dictate appropriate game and practice coverage

regarding the care of the athlete. The sports medicine team must be prepared to quickly take care of any injury or emergency that could compromise an athlete's health. All personnel who are associated with medical coverage of practices, competitions, skill instruction, and strength and conditioning should be at least minimally qualified through certification in cardiopulmonary resuscitation, first-aid techniques, and the prevention of disease transmission [12].

According to the 2014–2015 NCAA Sports Medicine Handbook, an emergency action plan should be in place for each venue and should “incorporate roles and responsibilities of coaching staff, medical staff, spectators and others during injury evaluation/response on the field, to ensure appropriate first response and medical evaluation. Institutions should have on file, and annually update, an emergency action plan for each athletics venue to respond to student-athlete catastrophic injuries and illnesses, including but not limited to, concussions, heat illness, spine injury, cardiac arrest, respiratory distress (e.g., asthma), and sickle cell trait (SCT) collapses. All athletics health care providers and coaches, including strength and conditioning coaches, sport coaches and all athletics personnel conducting activities with student-athletes, should review and practice the plan at least annually” [5].

Although it may not be possible for a physician to be present at all workouts and practices, properly trained personnel, such as a certified athletic trainer, should provide prompt evaluation and emergency care when warranted. Oversight of these duties is the responsibility of the team physician [13]. The NCAA Sports Medicine Handbook specifically recommends that each scheduled practice or contest of an institution-sponsored intercollegiate athletics event, and all out of season practices and skills sessions, should include the presence of a person qualified and delegated to render emergency care to a stricken participant, and the presence of or planned access to a physician for prompt medical evaluation of the situation, when warranted.

An example of an improperly implemented emergency plan is shown in the case of Mogabgab

v Orleans Parish School Board [14]. In this case, a high-school player sustained prolonged heat stress during practice, which was not noted or treated by the coaches in a timely fashion and which led to his death. The facts reported in court were dramatic, in that no medical professionals were notified until an hour and 20 min after the athlete became unconscious. Medical treatment did not commence until approximately 2 h after the athlete became symptomatic. This incident shows that all members of the sports medicine team, including coaches, must be prepared to deal promptly with health emergencies. Institutions sponsoring athletic events must ensure that they have a written emergency action plan, and the sports medicine team must be able to implement it in the event of an emergency.

76.6.1 Risk Management Recommendations

Risk management is the key for preventing lawsuits in sports medicine. Risk management is a method intended to prevent financial, physical, property, and time loss for a group or organization. Factors proved to aid in risk management and liability reduction include maintaining clinical competence, keeping accurate medical records, and appropriate communication among sports medicine team members [15]. A well-designed risk management program should cover four essential elements: compassion, communication, competence, and charting [16]. Maintaining clinical competence and keeping accurate medical records are other important means to avoid liability for malpractice. Studies show that 70% of the medical litigations are caused by poor communication and attitude problems presented by physicians or trainers [16].

76.6.2 Policies and Procedures

All sports medicine facilities and settings must have written policies and procedures that are reviewed and practiced regularly. These plans

should correlate with the type of potential risk from the athletic event and at the particular location. Each program should have policies and procedures that provide a blueprint for actions that need to be taken by any member of the sports medicine team. The policies and procedures documents should be written in conjunction with legal council and appropriate medical and professional standards. The documents should include not only a hierarchical system for evaluation and treatment but also staffing and emergency response protocols and procedures [5].

76.6.3 Informed Consent

Informed consent implies that the provider discloses the diagnosis, the nature and purpose of any proposed treatment, the risks and benefits of the treatment, any reasonably treatment alternatives, and the prognosis if the proposed treatment is not performed [17]. The landmark Canterbury case, and many of the cases decided in the 1970s regarding informed consent, called for disclosure of information beyond what physicians traditionally had provided for patients and what the law had required previously [18]. Despite the physician's required explanation of prognosis, a detailed statistical discussion of prognosis is not required, and the courts have not required physicians to provide detailed statistical information on a patient's prognosis or other aspects of the treatment that was performed [19]. The team physician is best protected by documenting the following points when performing an informed consent with a patient: (1) an oral review of all probable risks and documentation signed by the patient indicating that these risks and benefits of the treatment were discussed; and (2) a catch-all provision to the informed consent that documents, in general terms, the additional or other potential risks or injuries that might occur and any reasonable treatment alternatives [20]. When proper informed consent is obtained with proper documentation, it generally protects the institution and physician from successful litigation.

Informed Consent

The team physician is best protected by documenting the following points when performing an informed consent with a patient:

1. An oral review of all probable risks and documentation signed by the patient indicating that these risks and benefits of the treatment were discussed.
2. A catch-all provision to the informed consent that documents, in general terms, the additional or other potential risks or injuries that might occur and any reasonable treatment alternatives [20].

Consent may be implied under certain circumstances, such as when an athlete is unconscious from a concussion and needs emergency medical treatment [21]. In these cases, the law generally assumes that if the injured athlete had not been injured, they would have authorized the treatment.

76.6.4 Preparticipation Examinations

The sports medicine practitioner traditionally conducts preparticipation physical examinations (PPEs) of athletes seeking medical clearance before participating in sports. The primary purpose of the PPE is to identify an athlete who may be at risk of injury to themselves or others before participation. The PPE has multiple objectives, including collecting basic medical data, determining athletic eligibility, serving as a primary examination for medically underserved populations, and enabling the detection of medical conditions that may not preclude the athlete's participation in sports but still require attention [22].

The right to disqualify an athlete from athletic participation during a PPE has been upheld in the courts. This was the case in *Knapp v Northwestern University* [23]. In this case, Nicholas Knapp, a high-school senior, collapsed in cardiac arrest

at the end of a pickup basketball game. He had documented ventricular fibrillation and returned to sinus rhythm after electrical defibrillation. An implantable cardioverter-defibrillator was placed 10 days after the cardiac arrest. Before the cardiac arrest, Knapp had accepted a basketball scholarship at Northwestern University. The university's team physicians disqualified him from participation shortly after Knapp enrolled in the autumn of 1995. The physicians determined that participation in college basketball would produce an unacceptable risk of sudden death and would be contrary to the 26th Bethesda Conference guidelines for participation with cardiovascular abnormalities. He was allowed to keep his full athletic scholarship for 4 years. Knapp filed suit that Northwestern University had violated the Rehabilitation Act of 1973, which prohibits discrimination against an athlete who is disabled yet has the capabilities to play a competitive sport. The court ruled in favor of Northwestern University and emphasized that it is proper for the team physician to rely on medical guidelines and recommendations such as the Bethesda guidelines in clearing an athlete for participation.

76.6.5 The Medicolegal Aspects of Returning to Play

After an athlete is injured, it is within the team physician's medical judgment to decide whether the athlete can return to athletic participation safely. The team physician has the primary duty to protect the athlete and is charged with this responsibility because of their relationship with the athlete and association with the representative institution [20].

Litigation may arise when a team physician releases an athlete to return to play and subsequently the athlete sustains an additional injury. The athlete may hold the physician responsible, contending that they should have been withheld from athletic participation [24].

In their 1990 lawsuit, Hank Gathers' heirs alleged that the team physician and consulting specialists improperly cleared him to resume playing college basketball with a serious heart

condition. Plaintiffs also alleged not only that the defendant physicians negligently diagnosed and treated Gathers but also that the defendants conspired to intentionally fail to inform Gathers of the seriousness of his heart condition and of the dangers of continuing to play competitive basketball.

Some athletes have sued physicians for refusing to provide them with medical clearance to play a sport. In *Penny v Sands*, Anthony Penny claimed that a cardiologist was negligent for withholding his medical clearance to play college basketball with a potentially life-threatening heart condition [25]. The defendant cardiologist diagnosed Penny as having hypertrophic cardiomyopathy and disqualified him from participation in college basketball. Two other cardiologists were consulted and agreed with the opinion. Central Connecticut State University refused to allow Penny participate in its basketball program for 2 years. Penny eventually got clearance to play basketball from two other cardiologists. Penny claimed economic harm to his potential professional basketball career because he missed out from two seasons of college basketball. Penny subsequently collapsed and died suddenly while playing in a 1990 professional basketball game in England. Although the malpractice suit was voluntarily dismissed after his death, it is unlikely that a court would have awarded Penny's survivors compensation for economic loss after team officials accepted a physician's prudent recommendation that an athlete with established cardiac disease should be disqualified from competitive sports to reduce the risk of sudden death [26].

76.6.6 Dispensing Medications to Athletes

When prescribing drugs to athletes, team physicians should comply with all laws regarding dispensation and record keeping and should follow accepted medical practices regarding the appropriate type and dosage of pharmaceutical treatment [27]. Team physicians may be liable for negligently prescribing anesthetics or painkillers to athletes to accelerate return to play [15].

76.6.7 Confidentiality

The Health Insurance Portability and Accountability Act (HIPAA) regulates the way the team physicians and members of the health-care team communicate and handle patient medical information. Two categories in the final rules of the HIPAA statutes that most likely affect the sports medicine practitioner are consent for treatment and authorization to release information. It is imperative that the team physician becomes familiar with these, and all, regulations. Any final interpretations of this act should be left to the institution's legal counsel and reviewed under applicable state law [21].

Unauthorized disclosure of information about an athlete's medical condition to third parties such as the media violates a physician's ethical obligation to maintain patient confidences [28]. In addition, such unauthorized disclosure may expose the physician to legal liability for invasion of privacy and for defamation or intentional infliction of emotional distress if the information is false [29].

Physician's Role in Confidentiality

Unauthorized disclosure of information about an athlete's medical condition violates a physician's ethical obligation to maintain patient confidences [28].

Unauthorized disclosure may expose the physician to legal liability for invasion of privacy and for defamation or intentional infliction of emotional distress [29].

In *Chuy v Philadelphia Eagles Football Club*, a football player alleged that the team physician defamed him by falsely informing the media that he had a potentially fatal blood disease and also caused him to suffer severe emotional distress [29].

At the beginning of each season, many institutions require athletes to sign a waiver to seek permission to communicate the medical management of an athlete's injury between the athletic trainers, coaching staff, team physician, and the

rest of the sports medicine team. This waiver does not allow for discussion with individuals who are not involved in the management of the athlete's health care such as media, sports information departments, and so forth.

76.7 Summary

Legal issues in sports medicine are rapidly developing and establishing important body of jurisprudence that define the legal rights and duties of all those involved with protecting the health and safety of athletes. The law makes important distinctions between the relevant duty of care owed to high school, college, and professional athletes, because of the differing legal relationships that arise out of athletic participation at different levels of competition. It is important to acknowledge and know your contractual obligations to work within a legal and ethical framework.

Sports Medicine Treatment Algorithm

Sports medicine legal cases usually focus on two things:

1. Did the practitioner know the proper professional standards when providing medical care (i.e., is the physician CPR certified)?
2. Did the practitioner perform the standard of care (i.e., can the physician perform CPR in the event of an emergency)?

References

1. Dunn SR, George MS, Churchill L, et al. Ethics in sports medicine. *Dig Am J Sports Med.* 2007;35(5):840–4. <https://doi.org/10.1177/0363546506295177>.
2. Isaacs CA. Comment: Conflicts of interest for team physicians: a retrospective in light of Gathers v Loyola Marymount University. *Albany Law J Sci Technol.* 1992;2:147–63.
3. *Nowatske v Osterloh.* NW2d. 1996;265:271–4.
4. AOSSM: ACSM, AMSSM, AAOS, AAFP, AOASM. Team Physician Consensus Statement. From the project-based alliance for the advancement of clinical sports medicine, comprised of the American Academy of Family Physicians, the American Academy of Orthopedic Surgeons, the American College of Sports Medicine, the American Medical Society for Sports Medicine, the American Orthopaedic Society for Sports Medicine, and the American Osteopathic Academy of Sports Medicine. 2013. <https://www.sportsmed.org/AOSSMIMIS/members/downloads/education/ConsensusStatements/TeamPhysician.pdf>. Accessed 7 July 2019.
5. NCAA 2014–2015 sports medicine handbook. 25th ed. 2014. <http://www.ncaapublications.com/product-downloads/MD15.pdf>. Accessed 7 July 2019.
6. Bottomley M. Athletes at an overseas venue: the role of the team doctor. In: Payne S, editor. *Medicine, sport and the law.* Oxford: Blackwell Scientific Publications; 1990. p. 158–65.
7. King JH. The duty and standard of care for team physicians. *Houst Law Rev.* 1981;18(4):657–705.
8. International Federation of Sports Medicine. <https://www.fims.org/about/code-ethics/> (2019). Accessed 30 June 2019.
9. Malcom D. Confidentiality in sports medicine. *Clin Sports Med.* 2016;35(2):205–15. <https://doi.org/10.1016/j.csm.2015.10.006>.
10. Devitt B. Fundamental ethical principles in sports medicine. *Clin Sports Med.* 2016;35(2):195–204. <https://doi.org/10.1016/j.csm.2015.10.004>.
11. Tucker A. Conflicts of interest in sports medicine. *Clin Sports Med.* 2016;35(2):217–26. <https://doi.org/10.1016/j.csm.2015.10.010>.
12. Anderson J, Courson R, Kleiner D, et al. National athletic trainers association position statement: emergency planning in athletes. *J Athl Train.* 2002;37:99–104.
13. Herbert D. Medico-legal concerns and risk management suggestions for medical directors of exercise rehabilitation and maintenance programs. *Exerc Stand Malprac Rep.* 1989;3:44–8.
14. *Mogabgab v Orleans Parish School Board.* So2d. 1970. p. 456.
15. Herbert DL. *Legal aspects of sports medicine.* 2nd ed. London: PRC Publishing; 1995.
16. Gallup EM. *Law and the team physician.* Champaign, IL: Human Kinetics; 1995.
17. Rosoff A. Informed consent in the electronic age. *Am J Law Med.* 1999;25:367–86.
18. *Canterbury v Spence.* F2nd. 1972. p. 772, 780n 15.
19. Appenzeller H. *Managing sports and risk management strategies.* Durham, NC: Carolina Academic Press; 1993. p. 99–110.
20. Pearsall AWIV, Kovalski JE, Madanagopal SG. Medico legal issues affecting sports medicine practitioners. *Clin Orthop Relat Res.* 2005;433:50–7.
21. US Department of Health and Human Services. Health information privacy. The Health Insurance Portability and Accountability Act of 1996 (HIPAA)

- privacy and security rules. <http://www.hhs.gov/ocr/privacy/>. Accessed 12 Dec 2012.
22. Glover DW, Maron BJ. Profile of preparticipation cardiovascular screening for high school athletes. *JAMA*. 1998;279:1817–9.
 23. Knapp v Northwestern University. F3d. 1996. p. 473.
 24. Herbert DL. The death of Hank Gathers: an examination of the legal issues. *Sports Med Stand Malprac Rep*. 1991;2(3):41–6.
 25. Penny v Sands (D Conn Filed May 3, 1989) (No. H89–280).
 26. Paterick TE, Paterick TJ, Fletcher GF, et al. Medical and legal issues in the cardio-vascular evaluation of competitive athletes. *JAMA*. 2005;294(23):3011–8.
 27. Wallace v Broyles. SW2d. 1998. p. 712, 719.
 28. Home v Patton. So2d. 1973. p. 824.
 29. Chuy v Phila Eagles Football Club. F2d. 1979. p. 1265, 1273–81.

Part VII

Psychological Aspects in Basketball



The Importance of Sport Psychology in Basketball

77

Vanessa M. LaBode-Richman and Paul Groenewal

77.1 Mental Health Among Elite Athletes

Basketball is one of the most popular sports in the United States [1]. As such, media outlets regularly broadcast the lives and decisions of elite basketball players, which in many ways portrays them as being very different from the general population. From incredible salaries to constant jet-setting, the impression the American public often makes is that elite athletes are different. A common misconception is that if one is financially stable and living out their dream, they must somehow be immune to mental health issues.

This idea that elite athletes have “nothing to worry about” has also permeated the culture of elite sports, as mental health was not always an active consideration when evaluating a player’s well-being [2]. Kevin Love, Center and Power-Forward for the Cleveland Cavaliers, wrote about his experience with anxiety, admitting that “in the NBA, you have trained professionals to fine-tune your life in so many areas...but none of those people could help in the way I needed when I was lying on the floor struggling to breathe [during a panic attack in November of 2017].” Even the commissioner of the NBA, Adam Silver, who is known to have honest conversations about

mental health/wellness with elite basketball players discussed his concern with the mental well-being of players. He is quoted saying, “We are living in a time of anxiety... A lot of players are unhappy.” Many elite athletes, including elite basketball players, in the past decade have spoken publicly about their mental health struggles, dispelling the myth that they experience life’s difficulties differently from lay people. As such, more research has been conducted to understand the prevalence rates of mental health conditions within elite sports.

A review of multiple studies demonstrates that elite athletes share a comparable risk to high-prevalence mental health conditions when compared to those rates reported for the general population [3–8]. For example, the prevalence of depression and anxiety symptoms are similar between elite athletes and non-athletes [9–12]. Unfortunately, however, elite athletes are less likely to seek/attain help from a mental health professional [13, 14]. In fact, many elite athletes’ first interaction with mental health professionals does not occur until they reach the elite level. It would be inaccurate to assume that this first encounter reflects a lack of symptoms prior to that first interaction. While that may be true for some athletes, there is often historical evidence of symptoms that impact players both on and off the court [15].

While research studies and anecdotal disclosure demonstrate that elite athletes similarly experience mood and coping difficulties, athletes

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are dissimilar in ways that negatively impact their mental health [6]. For example, athletes are often under intense physical and mental demands required for training and competition [8]. Elite basketball players have high expectations to perform on demand, to adhere to a grueling travel schedule, and to play well in a considerable number of games, which ultimately leads to few days off throughout the season in most professional leagues. The public is aware of how many games are played, but is often naive to the volume of training and recovery required to stay in elite shape. Furthermore, most basketball games are held at night. While this choice increases viewership (e.g., television), this schedule can also have a considerable impact on athletes' circadian rhythm. Exercising so vigorously at night, traveling immediately following games, and the scheduling logistics of late-night activity (e.g., eating pre- and post-games) can often times lead to poor sleep hygiene.

While typical life stressors (e.g., family responsibility, relationship issues) are often sufficient to take a toll on one's mental health, the combination of common life stressors in conjunction with those specific sport-related pressures may actually increase the risk of mental health issues over the course of an elite athlete's career [16, 17]. Consider elite basketball players who have not yet joined any of the top leagues around the world. Many of these players still experience some of the same pressures that a more "successful" player might; however, they do so without the significant advantage and benefit associated with the wealth most popular, elite basketball players have. Now consider that elite athletes are less likely to seek help when needing to practice healthy coping or stress management. Without the professional help often needed to appropriately manage significant stress and pressure, individuals are left coping in the best way they can, which may not always be sustainable or healthy in the long term.

In support of this idea, research demonstrates that there is an increased prevalence of eating disorders and disordered eating in elite athletes, as compared to non-athletes [18]. Disordered eating

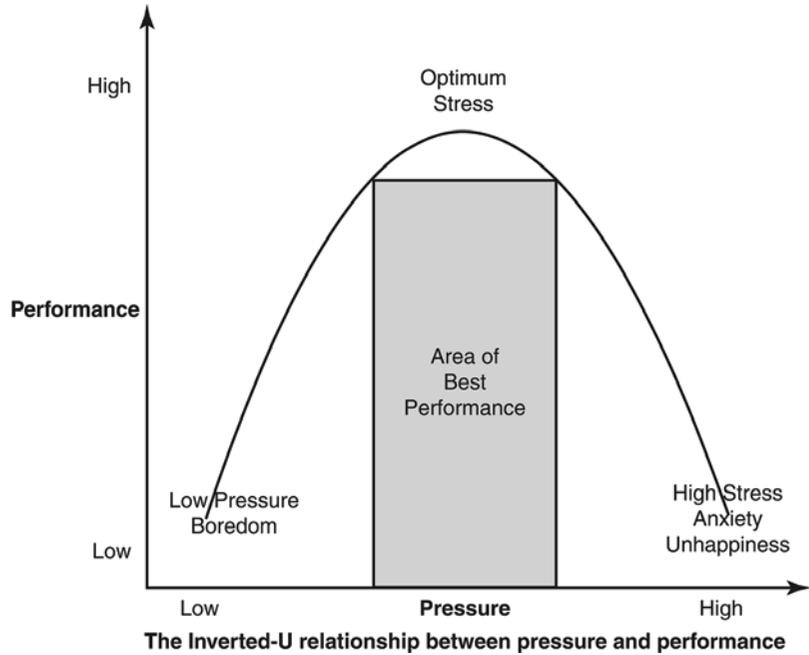
is typically understood to be a maladaptive coping strategy often utilized during moments of significant stress [19]. This is not only a health issue but also a performance issue being that proper nutrition is critical to attain peak performance. Without mental health professionals' added support, psychoeducation relating to mental pressure/stress, and implementation of successful, healthy coping strategies, elite-athletes often employ unhealthy strategies that neither provide adequate relief or successful ways to manage chronic, unrelenting professional demands.

Furthermore, research shows that higher levels of anxiety and depression are associated with negative performance outcomes and skill errors for elite athletes [20–24]. Without proper ways to manage mood symptoms and pressure-related issues, it is possible—and likely—that an athlete's performance suffers, which may only add to the already occurring mood and coping difficulties. In this way, those trained in sport psychology (e.g., sport psychologists) are particularly equipped and trained to help athletes circumvent this process by helping athletes understand the relationship between mental health and performance, while also offering them tools for how to best combat pressure, stress, and performance-related anxiety [25].

77.2 Mental Health, Coping Skills, and Their Impact on Performance Among Athletes

While poorly managed stress hinders performance, appropriate levels of anxiety and pressure are necessary elements of competition [26, 27]. Anxiety is the physiological state that keeps an individual engaged in a particular task, while pressure influences his/her stress response [26]. Research shows that minimal anxiety and pressure lead to boredom and apathy. This suggests that pressure and anxiety—albeit when appropriately managed—improve performance (Fig. 77.1). While the simplicity of this theory has been criticized, a multitude of research sup-

Fig. 77.1 The inverted-U relationship is depicted in the graph above. Low levels of pressure yield low performance and boredom, whereas as high pressure yields low performance, as well as high stress, anxiety, and unhappiness. The inverted-U theory relationship thus posits that there is an area of best performance between low and high pressure [27]



ports its claims [26]. Furthermore, this theory is often a great starting point in helping athletes build self-awareness. The ability to use mental skills always starts with the ability to self-assess.

When an athlete's anxiety is outside of the optimal range of performance, their cognitive skills can suffer. In basketball, visual search is a skill whereby an athlete is able to predict the direction of the ball [28]. Anxiety regulation is particularly important for this skill, because so much of visual search is related to how quickly one can process information in the moment [28]. Processing speed, how quickly one can synthesize information, is a neurocognitive construct consistently shown in research to be negatively affected by anxiety [28]. If an elite basketball player is slow to predict placement of the ball or positioning of other players, it is fair to assume personal performance, as well as the ability to assist teammates, will suffer. While anxiety will always be part of competitive sports, effective coping skills allow players to perform at a high level despite the stress they experience.

Generally, in order for athletes to better manage their stress and anxiety level, they must learn

to exercise psychological flexibility, so they can adapt to the demands of a specific situation, while still holding true to values they personally identify with [29]. In other words, it is important that athletes remember that life does not occur in a vacuum. Professional demands will occasionally confront personal beliefs they hold [29]; however, by maintaining a healthy perspective, elite athletes can learn to reconcile those competing ideas and cope with them in ways that do not impact their athletic performance or overall anxiety level.

Process-focused approaches in conceptualizing improvement also tend to be better for managing stress as compared to outcome-focused approaches [30]. Athletes with an outcome-focused approach are concerned with their statistics (e.g., how many points they scored, how many rebounds they had). While statistics can be utilized to determine the success of a game, they do not always reflect an athlete's impact on the game. In fact, increased focus on statistics can thwart progress and improvement, as it can lead to development of a performance-based identity—the belief that a

person's value is determined by the "stat lines." By focusing solely on outcome, tracking metrics tend to be binary (e.g., win or lose, faster or slower), which tends to oversimplify the process of improvement.

Conversely, by focusing on the process of improvement, multiple metrics and how they all relate can be used to track progress, which allows for appreciation of dynamic and complex interrelated variables. This allows the athletes to separate their identity from their performance/behavior. In other words, athletes' identity remains intact even in the face of poor performance, which allows them to remain highly motivated to identify and work on skills contributing to poor performance. For instance, after a game in which athletes do not meet their performance expectations (e.g., a player who typically averages 20 points/game only scores 2 points in a particular game), the athletes evaluate their process and considers what they can do differently in order to improve their performance. While reviewing film with a coach, they realize they were hesitating and settling for low percentage shots. Their goal for the next game is to focus on taking shots from three high percentage places on the court and using mental skills that remind him of the permission to shoot. Throughout this process, the players' identity remains intact because their value is not based solely on their stats.

Finally, self-compassion is integral toward building resilience [31]. Resilience, the ability to adapt and cope with disappointment, failure, and/or frustration, is the variable that allows an individual to improve performance even while experiencing distress [32, 33]. It is important that athletes practice self-compassion, particularly when conceptualizing failures and frustrations, as seeking perfection is not a healthy motivator for athletes or their support team. Building self-compassion can begin with conceptualizing and preparing for mistakes. Even pointing out that an athlete will need to prepare for mistakes can be a start. Planning for "when" they make a mistake and not "if" they do helps prepare them for that moment, so that they can

effectively manage the feelings and thoughts associated with making errors without it impacting too much of their performance afterward. Perfection is not realistic and does not define even those athletes we consider to be the "greatest of all time." Resilience can be taught and practiced [34], so that no setback ever feels insurmountable. In order to encourage athletes to practice resilience and combat competitive anxiety, appropriate mental skills must be learned and implemented.

77.2.1 Goal Setting

Goal setting requires athletes to create a clear understanding of their goals and the process they need to commit to in order to reach their goals. Goal setting helps to improve motivation and accountability [35–38]. Players who want to improve their drives to the left can identify specific skills as their objectives (e.g., dribbling with their left hand, finishing with their left hand) and can assess growth by measuring the percentage of shots made after driving to the left under pressure. This strategy allows the athlete to gain a clear understanding of whether they are making progress. It also allows them to change their process when progress is not being made. It is important to always highlight that lack of success does not reflect deficient skills or ability, as lack of success may instead be due to ineffective process.

77.2.2 Relaxation

High levels of anxiety increase arousal, which can negatively impact performance. Heightened anxiety can occur due to the pressure of intense competition and performance demands, but also due to pressures off the court. In order to lower arousal and stimulate the parasympathetic nervous system, relaxation strategies are used to reduce feelings of excessive pressure or somatic anxiety (e.g., gastrointestinal distress, headache) [39–41]. It is difficult to remain focused on

demands that may be changing moment-to-moment if anxiety is elevated. For example, deep breathing in moments of stress is a common relaxation technique utilized. Relaxation skills can help players calm their body and mind and allow them to focus all of the effort on the play at hand.

77.2.3 Activation

Mood symptoms can sometimes yield moments of apathy, lethargy, or low energy. As such, activation skills are utilized when athletes need to increase their arousal or simulate their sympathetic nervous system [39, 40, 42]. Being below the optimal range of performance can leave a player looking slow and disengaged. This can also lead to sporadic performance after stoppages or breaks. For instance, players with difficulty maintaining effective levels of energy while sitting on the bench can benefit from a fast-paced jog when heading to the scorer's table or by taking a few intentionally hurried breaths.

77.2.4 Imagery

Imagery is an important psychological skill that can be helpful in conjunction with physical training, but also independently, particularly if an athlete is injured and unable to train/play. Research consistently shows that this skill can be practiced and improved [37, 43–45]. Players can utilize imagery between games to mentally practice skills as a game preparation strategy, while on the bench, and as a post-game strategy in an effort to quickly move on from mistakes. Imagery can also be coupled with physical training for the purpose of skill acquisition, as well as when they need to remain off-legs, for example, on recovery days or while recovering from injury. For example, players can picture themselves taking a shot successfully and then also envision themselves successfully taking the same shot with an “obstacle” (e.g., having their

view obstructed by another players' hand in their face, being off-balance). Envisioning success in ideal situations, as well as those situations that have real-world challenges, can allow a player to feel more prepared during real-time games and training.

77.2.5 Self-Talk

Self-talk is using positive, reality-based dialogue with oneself in a direct, assertive fashion. Self-talk has been shown to impact thoughts, attitudes, and behavior. Research supports the idea that effective self-talk has a positive impact on performance and optimal levels of arousal [46–48]. Players often intensely focus on their offensive statistics and are often eager to utilize self-talk in relation to missed field goals. After a missed shot they might say to themselves, “next one's going in” in order to let go of the miss and move on to the next play.

77.2.6 Attentional Control

Attention is the crux of focus and mindfulness. Attentional training is done to allow an athlete to stay mentally on-task, particularly in times of high stress. The ability to maintain attention on a preferred stimulus, rather than allowing attention to be dictated by potentially discouraging thinking patterns and increased arousal level is important. Research supports that attentional control is a skill that often translates to more effective performance [49–52]. Attentional training is not only for on-the-court demands. Players require a great deal of focus in training and everyday life. They benefit greatly from learning how to minimize distractions and direct their focus where it is most needed. There are many apps and devices that can be helpful for athletes working on their attentional control, which include (but is not limited to) biofeedback devices (see appropriate section below for more information).

77.2.7 Emotional Control

Emotional control skills foster the ability to effectively manage frustration and other negative emotions. This is imperative for professionals who need to perform at high levels in the face of extreme stress. By exercising emotional control, arousal management is practiced, which is needed for athletes to remain in the optimal range of performance [51, 53–55]. Strategies for managing emotional control can be helpful for the player who frequently gets angry with others. For instance, these players might have difficulty making foul shots if they lose focus when angry. Emotional control skills are also important for the players whose anger turns inwards—who becomes angry or disappointed in themselves after a missed shot or when they are not performing at their best. While the player in the latter example may have fewer outward displays of emotion, emotional control skills can still help to remedy the issue, so long as the interplay between emotionality and performance is identified.

77.2.8 Automaticity

The skill of automaticity can be understood as being what allows us to perform activities without

deliberately planning out each step [56]. An everyday example is driving a car. While first learning to drive, one must remember “gas is on the right, brake is on the left” in addition to many other logistical steps needed for successful driving; however, after 5 years of driving, many of us can remember pulling into our parking spot at home without much memory of the many steps it must have taken to get there. For athletes, it is important for them to learn how to maintain a high level of performance without consciously thinking about the logistics of each sub-task that is being performed, as thinking can sometimes disrupt the flow of movement and skill [37, 55, 56].

Practicing other mental skills can also aid in developing automaticity. For instance, mental imagery can alter the mind similarly to actual physical practice in some ways, which can allow an athlete to reach automaticity sooner. For example, a player trying to find high percentage shots in a new offensive system will benefit greatly from actually practicing within that system in addition to utilizing mental imagery to mentally run through the plays on days when physical run through is not possible. Becoming more automatic with these newer plays reduces the cognitive demand and allows the players to allocate more of their mental energy on finding their best shot.

Fact Box 1 Mental Skills

Mental skill	Definition
Goal setting	Athletes create short-term and long-term goals to work toward
Relaxation	Techniques are used to reduce feelings of stress and anxiety
Activation	Activating one’s energy level in order to reach the optimal state of performance
Imagery	A preparation strategy that helps an athlete imagine the barriers and successes of competitive performance
Self-talk	A positive, reality-based dialogue with oneself in a direct, assertive fashion
Attentional control	The ability for an athlete to stay on-task and focused during times of high stress
Emotional control	The ability for an athlete to regulate their emotions and effectively manage feelings of frustration or negative emotions
Automaticity	An instinct that allows athletes to perform skills without deliberately planning each step

Fact Box 2 Pressures Affecting Elite Basketball Players

Pressures experienced by elite basketball players

Physical	Intense physical conditioning, weight-lifting, practices, games, experience of physical injuries, and rehabilitation process
Mental	Being in the public eye, increased scrutiny and judgment, the effects of social media, limited social support, the potential for injuries

77.2.9 Coping Skills and Technology

Biofeedback is a technique utilized to teach individuals how to control some of their body's functions and reactions, such as heart rate [57]. By being hooked up to electrical sensors, individuals can receive information about their bodies noninvasively. This feedback allows for subtle changes in the body to be made by practicing skills, such as relaxation, imagery, and emotional control. Over time, practicing coping skills in conjunction with monitoring physiological arousal can help athletes learn what the optimal level of anxiety or pressure feels like, while also learning how to objectively measure the progress of their coping skills training [57]. Research shows that heart rate variability biofeedback can lower anxiety, which is related to performance optimization in basketball [57]. Players can utilize biofeedback devices, such as heart rate variability monitors, as a mindfulness habit or even as a pre-game strategy in order to focus their attention on the game at hand.

77.3 Mental Health and Overcoming Injury

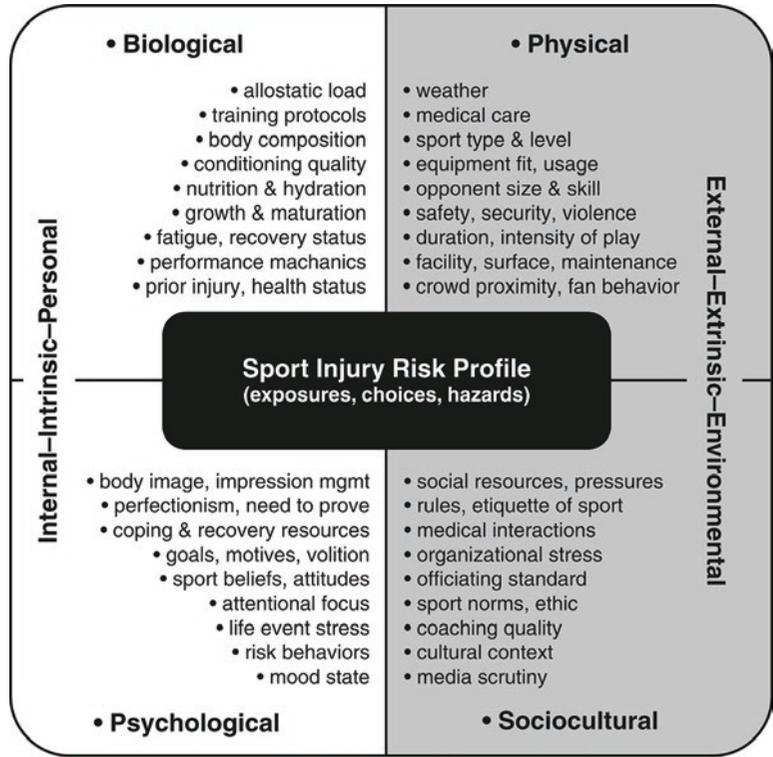
With the opportunity to play sports at the elite level comes the risk of significant injury [58]. While ongoing training and conditioning aim to minimize strain and damage done by intense physical demands, injuries are unfortunately sometimes unavoidable [58]. There is significant physical rehabilitation required of an athlete following an injury to be able to return to play; how-

ever, physical rehabilitation is only part of what is required so that an athlete can return to competition and perform as well as they had. Injury, while often orthopedic, also has a psychological and cognitive component that athletes must also overcome [59, 60]. To further understand the many factors impacting an athlete's sport injury risk, refer to Fig. 77.2. While studies are minimal in demonstrating this relationship among elite athletes, preliminary research does support this theory [61].

Following injury, athletes tend to experience both cognitive and emotional responses [62, 63]. Cognitive responses relate to how thinking is changed by the injury. Emotional responses are the mood symptoms that often interfere with normal functioning. Typical cognitive responses to injury often include concerns about re-injury, doubts about competency, low self-efficacy (the idea that they can overcome the challenge), loss of identity, and concerns about the competency of those providers treating his/her injury [64, 65]. Emotional responses to an injury can include sadness, depression, suicidal thinking, anxiety, amotivation, anger, irritation, frustration, isolation, changes in appetite and/or sleep, low vigor, disengagement, and burn-out [58, 66]. The stress associated with injury recovery is more than just the pain and discomfort associated with the injury and the rehabilitation process [62, 63, 67]. Often times the distress experienced following an injury is most related to the concern of whether a player will fully recover to reach their full performance potential. Generally, players who have not yet made the professional leagues begin thinking about whether teams will see them as "prone to injury," whereas players already in the professional leagues are concerned about their next contract.

In support of the theory that injuries are both physical and psychological, research demonstrates that physical rehabilitation in conjunction with psychological intervention yields the best outcomes for returning to play [58, 68]. For example, concussions predicted to improve with rest alone can also lead to postconcussive (e.g., fatigue, somatic pain) and mood symptoms (e.g., anxiety, depression), which most often are related to new or previously untreated mental

Fig. 77.2 Sport injuries and the risk associated is related to what athletes are exposed to, hazards they face, and choices they make. Internal (i.e., biological and psychological) and external factors (i.e., physical and sociocultural) contribute to the overall risk of injury that an athlete faces [84]



health issues, rather than to the physical injury itself [58, 68–70]. With time, these individuals do not fully recover following what is medically deemed appropriate treatment, and symptoms will remain unresolved until the individual participates in psychological intervention [71].

There are many factors associated with poor injury outcomes, should adequate psychological support and resources not be provided. Those factors include anxiety, worry, low mood, hypervigilance, poor body image, low self-esteem, perfectionism, limited coping strategies, stressful live events, and history of risk-taking behavior, or trauma [58, 68, 72]. Additionally, sport teams and organizations can also impact outcomes post-injury, as psychological effects of injury are also associated with limited social resources, social pressure, organizational stress, negative self-appraisal of athletic performance, coaching quality, and the culture of the sport and related teams [58, 68].

Screening for mental health disorders is always recommended following traumatic musculoskeletal injury [73], as traumatic injuries—such as

ACL tears—can often lead to manifestation of significant behavioral and emotional difficulties. For example, traumatic injuries can sometimes lead to kinesiophobia, the irrational and debilitating fear of physical movement and activity stemming from a feeling of vulnerability and/or susceptibility to pain, injury, or re-injury [59]. Due to the level of avoidance that often accompanies this type of fear, it is important to (1) teach athletes the purpose of pain, as well as the types of pain they may experience during successful healing, (2) practice strategies for pain management, (3) help them reduce the proclivity to catastrophize, and (4) encourage them to work through their fear. Providing them with the support and resources needed to promote trust between athletes and their support team will allow them to recover in a way where they are both prepared physically and mentally to return to play. For professional athletes in particular, trust can be difficult to build. Generally, the individuals employed to rehabilitate players are the same individuals offering opinions regarding the long-term physical health and durability of the athlete. This makes

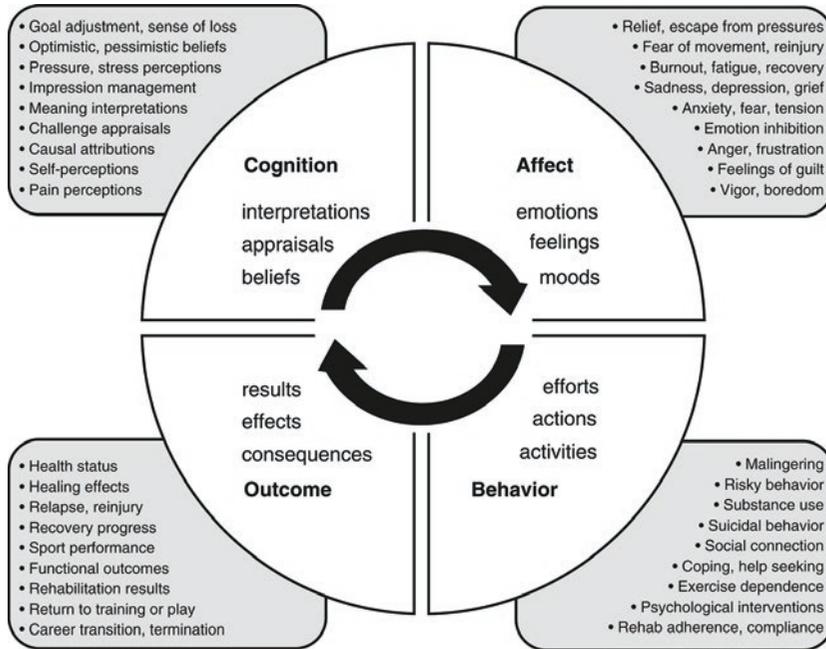


Fig. 77.3 The biopsychosocial pathways of injury response and recovery are the dynamic interplay between cognition, affect, outcome, and behavior. Within each domain is a variety of factors that inform how an athlete

acts and perceives the world. Only by understanding each factor's impact can professionals fully appreciate the potential response to injury an athlete may have [58]

having a good working relationship before the injury imperative. It also points to the need for a clear plan and honest conversations throughout the rehabilitation process.

Although there are several risk factors for recovery outcomes, there are also various protective factors associated with improved recovery following an injury. Examples of these factors include higher levels of optimism, self-efficacy, and lower levels of depression and stress [60, 74–80]. As such, mental health professionals trained in sports psychology often implement and practice strategies with athletes that ultimately provide players with some of these protective factors relating to healthy recovery outcomes. For example, professionals can help reduce re-injury anxieties using modeling techniques. This particular strategy can be executed by watching videos of formerly injured players discussing how they overcame re-injury anxieties or by pairing an injured athlete with an athlete proficient in rehabilitative exercises [79, 81, 82]. Additional strategies that help promote healthy recovery following an injury include fostering independence,

building confidence through goal-setting, building and encouraging the use of social support, and encouraging to remain mentally active with the sport (while also discouraging premature return to play) [79, 81, 82]. Refer to Fig. 77.3 to further review the dynamic pathways of injury response and recovery.

While microcounseling skills such as attending, active listening, empathy, and reflection can be effective when utilized by all professionals, they can be particularly helpful for physicians. These skills are proven to be effective and help enhance the psychological well-being of injured athletes during the rehabilitation process [83]. But as with many treatment approaches, this work requires a collaborative, team approach. Surgeons, physical therapists, athletic trainers, sport psychologists, and coaches can partner with the athlete to create an interdisciplinary plan that allows the player to feel empowered and encouraged. For the athlete, this level of support will help to facilitate better trust and confidence that returning to their pre-injury level of performance is attainable.

77.4 Final Thoughts

Physical training and performance have been touted as the necessary components to elite athleticism; however, more recently, mental health and resilience have been highlighted as areas imperative for athletic success. Basketball players, like all athletes, are susceptible to mental health disorders and general mental health issues. These issues may be long-standing or acute, related to off-the-court pressures, performance pressures, or a complex combination of both. Regardless of where these pressures originate, these mental health issues are likely to have a negative impact on performance. Fortunately, the same techniques that are effective in performance training strongly overlap with therapeutic techniques. It is important that teams and players connect with appropriately trained mental health experts to ensure that players are effectively managing the mental demands of their careers and lives. While the demands of an elite basketball player will always be great, developing and practicing mental skills and coping strategies allow players to successfully manage these pressures, while also help them to remain motivated and optimistic through training, competition, and injury recovery.

Fact Box 3 Sleep Hygiene Strategies Recommended for Elite Athletes

- Remove the bedroom clock from the room
- Avoid drinking coffee or alcohol
- Try to go to bed at the same time every night
- After 15 min of not being able to fall asleep, attempt a different strategy
- Be aware of the quality of food intake and the timing of meals
- Take naps that do not interfere with maintaining good sleep hygiene (e.g., 45 min or less and avoid taking naps in the afternoon)

References

1. Grundy P, Nelson M, Dyreson M. The emergence of basketball as an American national pastime: from a popular participant sport to a spectacle of nationhood. *Int J Hist Sport*. 2014;31(1–2):134–55.
2. Bauman NJ. The stigma of mental health in athletes: are mental toughness and mental health seen as contradictory in elite sport? *Br J Sports Med*. 2016;50:135–6.
3. Gouttebauge V, Kerkhoffs G. A prospective cohort study on symptoms of common mental disorders among current and retired professional ice hockey players. *Phys Sports Med*. 2017;45(3):252–8.
4. Gouttebauge V, Aoki H, Kerkhoffs G. Symptoms of common mental disorders and adverse health behaviours in male professional soccer players. *J Hum Kinet*. 2015;49:277–86.
5. Gouttebauge V, Castaldelli-Maia JM, Gorczynski P, Hainline B, Hitchcock ME, Kerkhoffs GM, Rice SM, Reardon CL. Occurrence of mental health symptoms and disorders in current and former elite athletes: a systematic review and meta-analysis. *Br J Sports Med*. 2019;53(11):700–6.
6. Gouttebauge V, Jonkers R, Moen M, Verhagen E, Wylleman P, Kerkhoffs G. Prevalence and risk indicators of symptoms of common mental disorders among Dutch Olympic athletes. *Br J Sports Med*. 2017;51(4):324.
7. Rice SM, Gwyther K, Santesteban-Echarri O, Baron D, Gorczynski P, Gouttebauge V, Reardon CL, Hitchcock ME, Hainline B, Purcell R. Determinants of anxiety in elite athletes: a systematic review and meta-analysis. *Br J Sports Med*. 2019;53(11):722–30.
8. Rice SM, Purcell R, De Silva S, Mawren D, McGorry PD, Parker AG. The mental health of elite athletes: a narrative systematic review. *Sports Med*. 2016;46(9):1333–53.
9. Bandelow B, Michaelis S. Epidemiology of anxiety disorders in the 21st century. *Dialogues Clin Neurosci*. 2015;17(3):327.
10. Du Preez EJ, Graham KS, Gan TY, Moses B, Ball C, Kuah DE. Depression, anxiety, and alcohol use in elite rugby league players over a competitive season. *Clin J Sport Med*. 2017;27(6):530–5.
11. Goodman WK, Grice DE, Lapidus KA, Coffey BJ. Obsessive-compulsive disorder. *Psychiatr Clin*. 2014;37(3):257–67.
12. Gorczynski PF, Coyle M, Gibson K. Depressive symptoms in high-performance athletes and non-athletes: a comparative meta-analysis. *Br J Sports Med*. 2017;51(18):1348–54.
13. Coyle M, Gorczynski P, Gibson K. “You have to be mental to jump off a board any way”: elite divers’ conceptualizations and perceptions of mental health. *Psychol Sport Exerc*. 2017;29:10–8.
14. Gouttebauge V, Hopley P, Kerkhoffs G, Verhagen E, Viljoen W, Wylleman P, Lambert M. A 12-month prospective cohort study of symptoms of common mental disorders among professional rugby players. *Eur J Sport Sci*. 2018;18(7):1004–12.

15. Castaldelli-Maia JM, de Mello e Gallinaro JG, Falcão RS, Gouttebarga V, Hitchcock ME, Hainline B, et al. Mental health symptoms and disorders in elite athletes: a systematic review on cultural influencers and barriers to athletes seeking treatment. *Br J Sports Med.* 2019;53(11):707–21.
16. Arnold R, Fletcher D. A research synthesis and taxonomic classification of the organizational stressors encountered by sport performers. *J Sport Exerc Psychol.* 2012;34(3):397–429.
17. Engel GL. The need for a new medical model: a challenge for biomedicine. *Science.* 1977;196(4286):129–36.
18. Sundgot-Borgen J, Torstveit MK. Prevalence of eating disorders in elite athletes is higher than in the general population. *Clin J Sport Med.* 2004;14(1):25–32.
19. Hughes L, Leavey G. Setting the bar: athletes and vulnerability to mental illness. *Br J Psychiatry.* 2012;200(2):95–6.
20. Halvari H, Gjesme T. Trait and state anxiety before and after competitive performance. *Percept Mot Skills.* 1995;81(3 suppl):1059–74.
21. Maloney EA, Sattizahn JR, Beilock SL. Anxiety and cognition. *Wiley Interdiscip Rev Cogn Sci.* 2014;5(4):403–11.
22. Morgan WP, O'Connor PJ, Ellickson KA, Bradley PW. Personality structure, mood states, and performance in elite male distance runners. *Int J Sport Psychol.* 1988;19:247–63.
23. Turner PE, Raglin JS. Variability in precompetition anxiety and performance in college track and field athletes. *Med Sci Sports Exerc.* 1996;28(3):378–85.
24. Wolanin A, Gross M, Hong E. Depression in athletes: prevalence and risk factors. *Curr Sports Med Rep.* 2015;14(1):56–60.
25. Gee CJ. How does sport psychology actually improve athletic performance? A framework to facilitate athletes' and coaches' understanding. *Behav Modif.* 2010;34(5):386–402.
26. Bali A. Psychological factors affecting sports performance. *Int J Phys Educ Sports Health.* 2015;1(6):92–5.
27. Yerkes RM, Dodson JD. The relation of strength of stimulus to rapidity of habit-formation. *J Comp Neurol Psychol.* 1908;18(5):459–82.
28. Liu S, Zhou W. The effect of anxiety state on the visual search efficiency of athletes. *Open J Soc Sci.* 2015;3(06):80.
29. Kashdan TB, Rottenberg J. Psychological flexibility as a fundamental aspect of health. *Clin Psychol Rev.* 2010;30(7):865–78.
30. Duda JL, Balaguer I. Coach-created motivational climate. In: Jowette S, Lavallee D, editors. *Social psychology in sport.* Human Kinetics: Champaign, IL; 2007. p. 117–30.
31. Neff KD, McGehee P. Self-compassion and psychological resilience among adolescents and young adults. *Self Identity.* 2010;9(3):225–40.
32. Fletcher D, Sarkar M. A grounded theory of psychological resilience in Olympic champions. *Psychol Sport Exerc.* 2012;13(5):669–78.
33. Mummery WK, Schofield G, Perry C. Bouncing back: the role of coping style, social support and self-concept in resilience of sport performance. *Athletic Insight.* 2004;6(3):1–15.
34. Wagstaff CR, Sarkar M, Davidson CL, Fletcher D. Resilience in sport: a critical review of psychological processes, sociocultural influences, and organizational dynamics. In: *The organizational psychology of sport.* New York: Routledge; 2016. p. 138–68.
35. Burton D. The Jekyll/Hyde nature of goals: reconceptualizing goal setting in sport. In: Horn TS, editor. *Advances in sport psychology.* Human Kinetics: Champaign, IL; 1992. p. 267–97.
36. Gould D, Murphy S, Tammen V, May J. An examination of US Olympic sport psychology consultants and the services they provide. *Sport Psychol.* 1989;3(4):300–12.
37. Hardy L, Jones JG, Gould D. *Understanding psychological preparation for sport: theory and practice of elite performers.* Chichester: John Wiley & Sons Inc; 1996.
38. Healy L, Tincknell-Smith A, Ntoumanis N. Goal setting in sport and performance. In: *Oxford research encyclopedia of psychology.* Oxford: Oxford University Press; 2018.
39. Hardy L. A catastrophe model of performance in sport. In: Jones JG, Hardy L, editors. *Stress and performance in sport,* Wiley series in human performance and cognition. Chichester: John Wiley & Sons; 1990. p. 81–106.
40. Hardy L, Parfitt G. A catastrophe model of anxiety and performance. *Br J Psychol.* 1991;82(2):163–78.
41. Kaufman K, Glass CR, Pineau TR. Mindful sport performance enhancement (MSPE): development and applications. In: Baltzell AL, editor. *Current perspectives in social and behavioral sciences. Mindfulness and performance.* New York: Cambridge University Press; 2016. p. 153–85.
42. van der Lei H, Tenenbaum G, Land WM. Individual arousal-related performance zones effect on temporal and behavioral patterns in golf routines. *Psychol Sport Exerc.* 2016;26:52–60.
43. George L. Mental imagery enhancement training in behavior therapy: current status and future prospects. *Psychother Theory, Res Pract Train.* 1986;23(1):81.
44. Murphy SM, Jowdy DP. Imagery and mental practice. In: Horn TS, editor. *Advances in sport psychology.* Champaign, IL: Human Kinetics Publishers; 1992. p. 221–50.
45. Thomas PR, Fogarty GJ. Psychological skills training in golf: the role of individual differences in cognitive preferences. *Sport Psychol.* 1997;11(1):86–106.
46. Hardy J, Zourbanos N. Self-talk in sport: where are we now? In: *Routledge international handbook of sport psychology.* Oxfordshire: Routledge; 2016. p. 479–90.
47. Hatzigeorgiadis A, Zourbanos N, Galanis E, Theodorakis Y. Self-talk and sports performance: a meta-analysis. *Perspect Psychol Sci.* 2011;6(4):348–56.

48. Van Raalte JL, Brewer BW, Rivera PM, Petitpas AJ. The relationship between observable self-talk and competitive junior tennis players' match performances. *J Sport Exerc Psychol.* 1994;16(4):400–15.
49. Boucher SH. Attention and athletic performance: an integrated approach. In: Horn TS, editor. *Advances in sport psychology.* Champaign, IL: Human Kinetics; 1992. p. 251–66.
50. Ducrocq E, Wilson M, Vine S, Derakhshan N. Training attentional control improves cognitive and motor task performance. *J Sport Exerc Psychol.* 2016;38(5):521–33.
51. Jones J, Hardy LE. *Stress and performance in sport.* Chichester: John Wiley & Sons; 1990.
52. Nideffer RM, Sagal M-S. Concentration and attention control training. In: Williams JM, editor. *Applied sport psychology: personal growth to peak performance.* 2nd ed. Mountain View, CA: Mayfield; 1993. p. 243–61.
53. Josefsson T, Ivarsson A, Lindwall M, Gustafsson H, Stenling A, Böröy J, et al. Mindfulness mechanisms in sports: mediating effects of rumination and emotion regulation on sport-specific coping. *Mindfulness.* 2017;8(5):1354–63.
54. Smith RE, Christensen DS. Psychological skills as predictors of performance and survival in professional baseball. *J Sport Exerc Psychol.* 1995;17(4):399–415.
55. Thomas PR, Over R. Psychological and psychomotor skills associated with performance in golf. *Sport Psychol.* 1994;8(1):73–86.
56. Gray R. Movement automaticity in sport. In: Baker J, Farrow D, editors. *Routledge handbook of sport expertise.* New York: Routledge; 2015. p. 100–9.
57. Paul M, Garg K. The effect of heart rate variability biofeedback on performance psychology of basketball players. *Appl Psychophysiol Biofeedback.* 2012;37(2):131–44.
58. Wiese-Bjornstal DM. Psychology and socioculture affect injury risk, response, and recovery in high-intensity athletes: a consensus statement. *Scand J Med Sci Sports.* 2010;20:103–11.
59. Arden CL, Webster KE, Taylor NF, Feller JA. Return to sport following anterior cruciate ligament reconstruction surgery: a systematic review and meta-analysis of the state of play. *Br J Sports Med.* 2011;45(7):596–606.
60. Everhart JS, Best TM, Flanigan DC. Psychological predictors of anterior cruciate ligament reconstruction outcomes: a systematic review. *Knee Surg Sports Traumatol Arthrosc.* 2015;23(3):752–62.
61. Reardon CL, Hainline B, Aron CM, Baron D, Baum AL, Bindra A, et al. Mental health in elite athletes: International Olympic Committee consensus statement (2019). *Br J Sports Med.* 2019;53(11):667–99.
62. Tjong VK, Cogan CJ, Riederman BD, Terry MA. A qualitative assessment of return to sport after hip arthroscopy for femoroacetabular impingement. *Orthop J Sports Med.* 2016;4(11):1–6.
63. Tjong VK, Murnaghan LM, Nyhof-Young JM, Ogilvie-Harris DJ. A qualitative investigation of the decision to return to sport after anterior cruciate ligament reconstruction: to play or not to play. *Am J Sports Med.* 2014;42(2):336–42.
64. Burland JP, Toonstra J, Werner JL, Mattacola CG, Howell DM, Howard JS. Decision to return to sport after anterior cruciate ligament reconstruction, part I: a qualitative investigation of psychosocial factors. *J Athl Train.* 2018;53(5):452–63.
65. Hainline B, Derman W, Vernec A, Budgett R, Deie M, Dvořák J, et al. International Olympic Committee consensus statement on pain management in elite athletes. *Br J Sports Med.* 2017a;51(17):1245–58.
66. Kiliç Ö, Aoki H, Goedhart E, Hägglund M, Kerkhoffs G, Kuijer P, et al. Severe musculoskeletal time-loss injuries and symptoms of common mental disorders in professional soccer: a longitudinal analysis of 12-month follow-up data. *Knee Surg Sports Traumatol Arthrosc.* 2018;26(3):946–54.
67. Tjong VK, Devitt BM, Murnaghan ML, Ogilvie-Harris DJ, Theodoropoulos JS. A qualitative investigation of return to sport after arthroscopic Bankart repair: beyond stability. *Am J Sports Med.* 2015;43(8):2005–11.
68. Wiese-Bjornstal DM, Smith AM, Shaffer SM, Morrey MA. An integrated model of response to sport injury: psychological and sociological dynamics. *J Appl Sport Psychol.* 1998;10(1):46–69.
69. Alves W, Macciocchi SN, Barth JT. Postconcussive symptoms after uncomplicated mild head injury. *J Head Trauma Rehabil.* 1993;8:48–59.
70. Kontos AP, Covassin T, Elbin R, Parker T. Depression and neurocognitive performance after concussion among male and female high school and collegiate athletes. *Arch Phys Med Rehabil.* 2012;93(10):1751–6.
71. Nelson LD, Janecek JK, McCrea MA. Acute clinical recovery from sport-related concussion. *Neuropsychol Rev.* 2013;23(4):285–99.
72. Timpka T, Janson S, Jacobsson J, Dahlström Ö, Spreco A, Kowalski J, et al. Lifetime history of sexual and physical abuse among competitive athletics (track and field) athletes: cross sectional study of associations with sports and non-sports injury. *Br J Sports Med.* 2018;53:1412–7.
73. Wiseman T, Foster K, Curtis K. Mental health following traumatic physical injury: an integrative literature review. *Injury.* 2013;44(11):1383–90.
74. Arden CL. Anterior cruciate ligament reconstruction—not exactly a one-way ticket back to the preinjury level: a review of contextual factors affecting return to sport after surgery. *Sports Health.* 2015;7(3):224–30.
75. Arden CL, Taylor NF, Feller JA, Webster KE. A systematic review of the psychological factors associated with returning to sport following injury. *Br J Sports Med.* 2013;47(17):1120–6.
76. Czuppon S, Racette BA, Klein SE, Harris-Hayes M. Variables associated with return to sport following anterior cruciate ligament reconstruction: a systematic review. *Br J Sports Med.* 2014;48(5):356–64.

77. Gignac MA, Cao X, Ramanathan S, White LM, Hurtig M, Kunz M, Marks PH. Perceived personal importance of exercise and fears of re-injury: a longitudinal study of psychological factors related to activity after anterior cruciate ligament reconstruction. *BMC Sports Sci Med Rehabil.* 2015;7(1):4.
78. Glazer DD. Development and preliminary validation of the injury-psychological readiness to return to sport (I-PRRS) scale. *J Athl Train.* 2009;44(2):185–9.
79. Podlog L, Banham SM, Wadey R, Hannon JC. Psychological readiness to return to competitive sport following injury: a qualitative study. *Sport Psychol.* 2015;29(1):1–14.
80. Tripp DA, Stanish W, Ebel-Lam A, Brewer BW, Birchard J. Fear of reinjury, negative affect, and catastrophizing predicting return to sport in recreational athletes with anterior cruciate ligament injuries at 1 year postsurgery. *Rehabil Psychol.* 2007;52(1):74.
81. Hainline B, Turner JA, Caneiro J, Stewart M, Moseley GL. Pain in elite athletes—neurophysiological, biomechanical and psychosocial considerations: a narrative review. *Br J Sports Med.* 2017b;51(17):1259–64.
82. Podlog L, Dimmock J, Miller J. A review of return to sport concerns following injury rehabilitation: practitioner strategies for enhancing recovery outcomes. *Phys Ther Sport.* 2011;12(1):36–42.
83. Reese LMS, Pittsinger R, Yang J. Effectiveness of psychological intervention following sport injury. *J Sport Health Sci.* 2012;1(2):71–9.
84. Wiese-Bjornstal DM. Sport injury and college athlete health across the lifespan. *J Intercollegiate Sport.* 2009;2(1):64–80.



Sport Psychology in Basketball: Performance Under Pressure

78

Luca Sighinolfi

78.1 Sport Psychology in Basketball: Performance Under Pressure

Phil Jackson, one of the all time greatest coaches in the game of basketball, once said: “Basketball is a complex dance that requires shifting from one objective to another at lightning speed. To excel, you need to act with a clear mind and be totally focused on what everyone is doing” [1]. The attention span (and the performance as a result) of a basketball player fluctuates throughout the development of a game: the length of the game, the essential cerebral activities, the variety of involved areas, and the different types of analyzed information make the synchronization complicated.

The first part of this chapter focuses on the importance of perception behavioral control and its connection to the attentive system [2] to analyze basketball performance and explains how players’ shooting percentage is influenced by gaze behavior and quiet eye period. The relationship between attentive processes and anxiety will be argued by the theory of Eysenck’s careful control [3] to understand how it affects performance. When anxiety is perceived during the pursuit of a goal, it causes the individual to shift his or her

attention to the search for the cause, which is a source of anxiety, and alerts him in an attempt to adopt a resolution strategy [4].

The second part of the chapter focuses on the relationship between stressful situations, coping strategies and basketball performance. Regardless of how technically and physically trained a player is, stress can affect and alter the shooting mechanism, and thus, the probability that he or she will score is reduced considerably. In all stages of play, there are difficult and potentially stressful situations [5].

The third part of the chapter analyzes and focuses on different coping strategies adopted during basketball games and their potential efficiency in sports performances. Coping strategies are constant cognitive and behavioral changes used to try to handle specific or excessive demands on the person’s resources as the attention is focused on attempting to tackle or avoid the problem. To understand when and why players should adopt engagement coping strategies or disengagement coping strategies. Engagement coping refers to managing a situation through active coping strategies and coping with the problem, and disengagement coping involves coping strategies of distance or distancing one’s self from the situation. This chapter explains several variables that influence coping strategy preferences of basketball players in play-by-play

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situations, managing stressful factors during games.

78.2 Perception Behavioral Control, Attentive Systems, and Gaze Behavior on the Basketball Court

“Basketball is a complex dance that requires shifting from one objective to another at lightning speed. To excel, you need to act with a clear mind and be totally focused on what everyone on the court is doing. Some athletes describe this quality as “the cocoon of concentration [1].” According to Phil Jackson, one of the game’s all time greatest coaches, players need to become more aware of what’s happening ‘right now’, this very moment. The secret is not thinking and calming the continuous overlap of thoughts in such a way that the body instinctively does what it is practiced to do without the mind getting in the way”. All of us have had flashes of this sense of uniqueness when we are completely immersed in the moment, in symbiosis with what we are doing” (Phil Jackson) [1].

Antonio Damasio’s publications on neuroscience argue how to defuse a dangerous stereotype on how our brain works regarding the perception of events [2]. The nervous system processes a wide variety of sensations: sounds, images, flavors, scents, etc. However, these sensations are processed in several different cerebral structures, and not just in one designated area. “The usual metaphor has something to do with a large CinemaScope screen equipped for glorious Technicolor projection, stereophonic sound, and perhaps a track for smell too” (Antonio Damasio) [2].

This idea of a coherent cinematographic projection on one unique film is a false intuition. Damasio and others claim that we do not have an area in our brain capable of processing all the perceptions that hit us at the same time from the various senses. Our experiences (full of sounds, movements, shapes, and colors) are the result of the integration of several neural activities, which are perceived as coherent and simultaneous. Any

form of integration of different systems must take into consideration time as a factor, or, in other words, the timing in which such integration occurs.

If it takes a very short time frame to integrate all the cerebral activities in different areas of the brain, it may be possible to obtain a coherent and integrated “script” unique to us. It is a matter of timing, which is essential for an efficient integration of these cerebral activities. “When the brain integrates separate processes into meaningful combinations by means of time, it is a sensible and economical solution but not one without risks and problems. The main risk is mistiming. Any malfunction of the timing mechanism would likely create spurious integration or disintegration” (Antonio Damasio).

Fact Box 1

“The attention span of a player fluctuates throughout the development of a game.”

One example in basketball is in man-to-man defending situations. The defending player, uses multiple channels of collection of sensory inputs in order to know how to position himself defensively in the most effective way. He uses, for example, both visual inputs (to see where the opponent and the ball are) and tactile inputs (touching the opponent to understand the positioning and/or timing of a possible cut to the basket) in an integrated manner to detect the position of the player he is defending against.

If the opponent is in one side and the ball is on the opposite side, the defender is forced to a continuous rotation of the head in order to maintain as much visual contact as possible with the opponent as well as the ball and other players’ movement.

The speed of analysis and integration between (1) the visual information that the defender has collected about opponent player positioning before the cut to the basket, (2) the visual information collected during the cut to the basket, and (3) the tactile information collected during physical contact with the opponent who is going to the

basket require processing and integration of this information. The brain integrates separate processes into meaningful combinations by means of time. The timing and effectiveness of processing and integration of this information considerably conditions the locus of control of the player in that specific game situation.

Thus, it is understood why the attention span (and the performance as a result) of a player fluctuates throughout the development of a game: the length of the game, the essential cerebral activities, the variety of involved areas, and the different types of analyzed information make the synchronization complicated. Therefore, one of the main variables that influence our experiences in relation to the situation we are in is the perceived behavioral control. The perceived behavioral control is linked to the perception of self-efficacy, which is extremely useful to any performance.

Ajzen introduced the construct 'perceived behavioral control' into his theory of planned behavior as a determinant of both behavioral intention and of the behavior itself [6]. On a conceptual basis, perceived behavioral control is similar to self-efficacy—both constructs refer to the person's belief that the behavior in question is under his or her control—but, operationally, perceived behavioral control is often assessed by the ease or difficulty of the behavior (e.g., 'I find it difficult to exercise three times a week'), while self-efficacy is operationalized by the individual's confidence in being able to carry out the behavior in the face of extenuating circumstances (e.g., 'I am confident that I can exercise three times a week even when I am away on vacation').

The lack of order and the lack of the perception of behavioral control increase our level of anxiety and affects our perception of self-efficacy [7, 8]: First, it reduces the ability to process and absorb information from the memory's executive center. During performance under pressure, motivation can decrease, and physical fatigue frequently increases to demand auxiliary strategies such as running less or start thinking "how can I save energy for the fourth quarter?" [9]. This effort of compensation aims to maintain our performance-

oriented resources by reducing or eliminating apprehension of alarming thoughts of the negative consequences of a poor performance [10].

Basketball performance and shooting percentage are directly correlated. As argued by Joan N. Vickers [11], gaze behavior is determinant to analyze players' free throw percentage. Gaze behavior of elite basketball athletes was determined as they performed 10 accurate and 10 inaccurate free throws (FTs) to a regulation basket wearing an eye tracker that permitted normal accuracy. Experts (mean FT = 78%) differed significantly from near experts (mean FT = 56%) in having a longer fixation on the target combined with an earlier fixation offset during the shooting action. In this study the gaze behavior of elite basketball subjects was analyzed as they performed successful and unsuccessful free throws. The free throw is a complex targeting skill that requires the integration of visual information gained through overt shifts of gaze with effector movements that carry out the aiming movement. Ramat, Schmid and Zambarbieri [12] defined gaze as the absolute position of the eyes in space and depends on both eye position in orbit and head position in space. According to their model, sensory information is received from the environment, with a voluntary generating of commands that direct the gaze according to the goals and intentions of the individual. At the lower levels, the head, body, and oculomotor control systems act as one closely coupled system [13]. When an individual orients his or her gaze to a far target such as a basketball hoop, the most common movement of the eyes is a sequence of events in which the eyes move prior to the head. Gauthier et al. [14] explained that the head follows because of its greater inertia, with the eyes localizing the target first and visual discrimination beginning immediately and maintained on the target even though the head is moving. The movement of the eyes and head is normally smooth, with the processing of information occurring as soon as the eyes stabilize on the target. Gaze behavior in the basketball free throw was therefore defined as the manner in which individuals moved their heads and eyes to take in available information while preparing

and executing the free throw. Gaze behavior has been specified by the frequency, duration, onset, and offset of gaze behaviors in the targeting environment, across phases of the aiming movement [11].

The efficiency with which athletes process information when they are anxious is, however, reduced, and the risk of poor performance is not excluded [15–17]. In an experiment, ten players were asked to perform several free throws in two different experimental conditions structured to manipulate the level of anxiety experienced during execution [9]. An ASL Mobile Eye Tracker measured the direction at which the players were looking and their point of focus while shooting at different situations with different levels of anxiety. It turned out that in shooting situations where the level of anxiety is greater, there is a significant reduction in the duration of the quiet eye period and the percentage of marked pots. Quiet eye period (QE) was defined as that portion of the final fixation from onset to the first observable movement of the hands into the shooting action (preshot). QE is an objective measure of the location, onset, offset, and duration of the gaze recorded while the participant performs a motor skill [11]. During QE, fixation or tracking is maintained on a specific location or object in space [18].

Players' shooting percentage in basketball, as well as performance in other sports such as golf, are influenced by quiet eye period [19, 20]. "There may be a number of mechanisms through which the QE impacts upon putting performance, consisting of both visuomotor control and psychological control elements. First, as highlighted for simple reaching and grasping tasks; well-learned visually guided tasks; and sport skills [18], the motor system tends to be more accurate when provided with timely information about targets from the gaze system. In effect, by holding a ball-focused QE throughout the putting stroke and through impact, golfers are able to ensure a more accurate contact with the sweet spot of the putter, ensuring more consistent ball strike. Second, information about the location of the hole may be more effectively stored in a visual memory buffer by maintaining

a steady gaze on the ball, as this strategy reduces potential distractions from other visual sources and therefore allows this information to guide accurate putting performance [19]. Moreover, the movement parameters (e.g., direction, distance, and force), as well as the timing and coordination of the arms, are fine-tuned in this crucial period of cognitive pre-programming leading to a more effective putting technique and successful performance [20]. Third, the QE may provide the "external focus of attention" described by Wulf (2007) [21] or the "external cue" described in Singer's (2002) five-step pre-performance routine [22]. Singer advocates focusing on an external cue to prevent athletes from focusing on internal or external distracters, negative thoughts, or the mechanics of skill execution [23]. As the stimulus-driven attentional system is more active when performers are anxious [24], such internal and external distracters are more likely to influence pressure putts. The QE may therefore also help provide a focus on what is controllable (ball strike) rather than what is not (a successful outcome) when golfers are under pressure. Fourth, the QE may simply help produce a general quiescence of the psychomotor system. Previous research has demonstrated that superior visuomotor performance is reflected in increased psychomotor and neural efficiency [20]."

Fact Box 2

"The lack of order and the lack of the perception of control increase our level of anxiety and affects our perception of self-efficiency."

Players' shooting percentage is influenced by quiet eye period because anxiety reduces the visual control needed for the individual to select a specific point of visual focus and keep it fixed. Instead, when the player is conditioned by anxiety-provoking stimuli, he or she tends to look at multiple visual targets, staring for a very short duration.

The theory of Eysenck's careful control [24] argues that the effect of anxiety on the attentive processes is crucial to understanding how it affects performance. When anxiety is perceived during the pursuit of a goal, it causes the individual to shift his or her attention to the search for the cause, which is a source of anxiety, and alerts him in an attempt to adopt a resolution strategy [4]. Consequently, cognitive resources are directed toward anxiety-inducing stimuli and not those relevant to the objective to be pursued, whether these stimuli come from outside such as environmental distraction factors (i.e., the public)—or from the inside, such as negative thoughts like, "I hope I don't make a mistake" [24]. Reduction in attentive control leads to discontinuity in the balancing of two attentive systems: a focused top-down system and a bottom-up stimulation-driven attentive system. Generally, anxiety is associated with an increase in the influence of the attention-focused system on stimulation and by a decrease of the influence of the attention system toward the goal [24].

In conclusion, this study [9] demonstrates why a basketball player who is experiencing performance anxiety will probably have a poor performance due to a dysfunctional attentive variation: in an ideal situation, the player manages to maintain his gaze fixed on a single eye (quiet eye period), while a player who is experiencing a lot of performance anxiety will shoot and look at multiple points of the target (i.e., around the basketball rim) for short fractions of the second. The basic lesson of "watching" is not only functional to the tactical component of the game but also integrated with the psychological one.

78.3 Stressful Situations and Coping Strategies in Basketball

In basketball, the diameter of the rim is about 45 cm, and the diameter of the ball is about 24 cm. Regardless of how technically and physically trained a player is, stress can affect and alter the shooting mechanism, and thus, the probab-

ity that he or she will score is reduced considerably. In all stages of play, there are difficult and potentially stressful situations. Stressful situations in sports can be grouped into five macro categories [5].

1. The first category is for high-pressure situations with the expectation of high-level performance, or national/international standards. High-pressure situations with the expectation of high-level performance can be promoted by technical staff, management, third parties, or by the athletes themselves, and they put the player at risk of playing to reward his or her imaginative (and pretended) performance.
2. The second category of stressful situations are related to personal interactions and problems with the coach, teammates, or other people.
3. The third category relates to conditions set by the sports organization to the player: (A) if the practice/match is too demanding with respect to the athlete's interests; (B) if the commitment and sacrifice requested are too high in relation to the athlete's availability; (C) if the player is not financially compensated or experiences economic problems; (D) potentially stressful situations in relation to media-derived pressure and the relationship with the media.
4. The fourth category relates to situations in which players are asked for specific psychological abilities in order to face situations of high professional competitive stress and performance anxiety.
5. The fifth category relates to physical demands:
 - (a) Body performance
 - (b) Recovery, not only from and due to injuries
 - (c) Weight and/or dietary regimen

Why focus on these five categories of stressful situations in sports? First, because they are fundamental to evaluate (and recruit) a professional player. Second, because player abilities to play under pressure, in different stressful situations, influence his/her performance during games and competitions [25].

Fact Box 3

“Even if a player is technically and physically trained, his basketball performance are constantly influenced by adopted coping strategies, while playing under pressure.”

Hence, mental abilities and psychological resources must also be evaluated when assessing a player. The fact that predicting the course of the season in advance is impossible does not mean that it is not worth gathering as much information as possible about how to deal with or avoid stressful situations for the player: analyze coping strategies [26]. Coach Phil Jackson summarized some of his views regarding players’ assessment and developmental process: “My biggest concern about recruiting players right out of high school has always been the temptations of NBA life. Many young players get so seduced by the money and fame that they never develop into mature young men or live up to their promise as athletes. In my view, the key to becoming a successful NBA player is not learning the coolest, highlight-reel moves. It’s learning how to control your emotions and keep your mind focused on the game, how to play through pain, how to carve out your role on the team and perform it consistently, how to stay cool under pressure and maintain your equanimity after crushing losses or ecstatic wins. In Chicago we had a phrase for this, going from a basketball player to a ‘professional’ NBA player” [27].

The meaning and appreciation that we give to a stressful situation shape our perception of the situation. Our emotions and thoughts are strongly influenced by these meanings and these assessments; they are based on our perception of the situation that categorizes it as stimulating, problematic or threatening [28]. Depending on the perception of the situation, we use different coping strategies. Coping strategies have characteristics that we do not know unless we analyze them individually, but they concern us personally and strongly affect our expectations. Coping strategies are “constant cognitive and behavioral changes [used] to try to handle specific or excessive demands on people’s resources” [29].

Coping strategies are a multi-dimensional construct:

- At the deepest and most fundamental level, there are cognitive and emotional pieces of information that enable us to structure our perception of the situation. Thoughts and emotions help us decide whether to choose one coping strategy rather than another. From the study of the interactions of these two types of information (cognitive and emotional), Folkman and Lazarus [29] identified coping strategies focused on emotion-centered coping and other coping strategies. According to Lazarus, the coping process is often centered on the problem as much as the emotion in an ever-present interaction [29].
- At the intermediate level, three components are identified by Richard H. Cox [30]: motivations, behaviors, and attitudes. The functional behaviors of these components derive from adaptation, perceptions of control, and avoiding or solving a realistic problem. Often though, the motivations, behaviors, and attitudes we adopt prove to be dysfunctional or ineffective, so the coping strategies we adopt do not reduce the types of psychological stress we live in. As a result, the situation remains problematic and our performance suffers.
- At the highest level, there is a boundary between automation and consciousness. With this quote, R.E. Smith [31] explains the need to distinguish between automatic and conscious coping skills. Of the automated coping strategies, we adopt and we can acquire awareness afterward only if someone or something makes us aware of the consequences of our actions. Therefore, it is important to help athletes understand coping strategies by analyzing whether they are automated or consciously adopted. Psychological stress lies neither in the individual nor in the situation, but depends on how the person evaluates the event and how he tries to adapt.

Cognitive appraisal is a process through which the person evaluates whether a particular encounter with the environment is relevant to his or her well-being, and if so, in what ways. In primary

appraisal, the person evaluates whether he or she has anything at stake in this encounter. For example, Is there potential harm or benefit with respect to commitments, values, or goals? Is the health or well-being of a loved one at risk? Is there potential harm or benefit to self-esteem? In secondary appraisal, the person evaluates what if anything can be done to overcome or prevent harm or to improve the prospects for benefit. Various coping options are evaluated, such as altering the situation, accepting it, seeking more information, or holding back from acting impulsively and in a counterproductive way. Primary and secondary appraisals converge to determine whether the person-environment transaction is regarded as significant for well-being, and if so, whether it is primarily threatening (containing the possibility of harm or loss), or challenging (holding the possibility of mastery or benefit) [32].

The player asks, “What is the stake?” in his primary appraisal and “what resources do I have?” in his secondary appraisal [32]. Identifying the athlete’s primary and secondary appraisals helps you better understand the coping strategies adopted by that player and then allows you to work on it. For example, a player who is entering the game believes that the priority in his half-court defense is to “not let the opponent I’m covering score or else we’ll lose the game” (first evaluation) and believes he can succeed “because I have a lot more grit and aggressiveness than he does” (secondary evaluation). Depending on the coping strategies he has adopted, positive or negative for his performance, the player could work hard with defensive slides and rebounding (positive coping strategies) or commit more fouls in a few minutes (negative coping strategies). Although thoughts and emotions (when entering the court) are similar, the coping strategies adopted would affect the player’s performance on a mental level. However, coping strategies must not be confused with results! If an athlete fails during a specific play such as losing the ball, it does not mean that he or she is not adopting potentially effective coping strategies; however, it is possible that the adopted strategies were dysfunctional in that specific game situation or that

the technical skills or athletics of the player were not enough [33].

There are three key aspects of defining coping strategies:

1. Coping is related to context (the situation) rather than to stable personality characteristics.
2. Coping does not have to be “successfully completed” and can even be merely an attempt to cope with the problem. Attention is focused on the attempt rather than on the positivity of the outcome.
3. Coping is a process that changes over time as the situation changes. At the base of a coping strategy, there is an assessment of the situation: if the situation changes, it needs to be reinvented. Then, the coping strategy could also be changed or eventually re-formalized.

One of the most commonly used tests by the scientific community to identify coping strategies is termed COPE (Coping Orientation to the Problems Experienced) [31] which classifies coping strategies into the following categories:

- Activity: take some sort of action to eliminate stress or affect its effects.
- Planning: reflect, plan, and develop strategies to overcome the problem.
- Abolition of competitive activities: set aside any other activity and avoid distraction to deal more effectively with the problem.
- Containment: wait for the opportunity to face stress to avoid reacting impulsively.
- Search for information: ask for advice, assistance, and information.
- Understanding: get moral support, reassurance, and understanding.
- Emotional outburst: express emotions and express feelings.
- Positive reinterpretation and growth: elaborate critical experience in positive terms or in terms of growth.
- Acceptance: acceptance of the situation and/or acknowledge your own inability to deal with it.

- Engage in religion: seek help or comfort in religion.
- Humor: make light of the situation and get through it.
- Denial: refuse the existence of a critical situation and try to act as if the stress does not exist.
- Behavioral detachment: it is a condition of helplessness. Efforts are made to deal with the critical situation, abandoning attempts to resolve it. This is the typical behavior of those who think that they will get poor results from their own attempts to counteract it.
- Mental detachment: it involves the suppression of competitive activities. It involves distracting, daydreaming, sleeping longer, “immerse yourself” in television, etc.
- Use of drugs or alcohol: use alcohol or drugs to tolerate stress.

78.4 Efficient Coping Strategies in Basketball

In the context of basketball performance, it is important to distinguish between the use of engagement coping strategies and the use of disengagement coping strategies. *Engagement coping* refers to managing a situation through active coping strategies and coping with the problem, for example, when a basketball player on offense get the physical contact with an opponent player trying to give to his teammate an open passing line; or when a player argues with referee after an offensive foul has been called against his team [34]. *Disengagement coping* involves coping strategies of distance or distancing one’s self from the situation, for example, when a player on offense avoids physical contact with an opponent player which could give an open passing line to his teammate; or when a player calls at the referee after an offensive foul has been called against his team [34].

The study conducted by Anshel and Wells [35] on a sample of 147 basketball players between the ages of 17 and 48 years showed which coping strategies players use in four typically stressful situations in a game: “miss an

easy basket”; “get fouled by an opponent”; “to lose the ball”; and “receive a perceived unjustified warning by a referee.” The reported outcomes revealed that players use engagement coping strategies in three of the four stressful situations analyzed: “to get fouled” (70%), “to miss an easy basket” (76%), and “lose a ball” (78%). While in the scenario of a “perceived unjustified warning from a referee,” the majority (63%) of the players adopted disengagement strategies. The results highlight athletes’ decisions to use engagement strategies in more controllable situations. By contrast, in the face of arbitration or actions beyond the perception of behavioral control, players often adopt strategies to avoid the problem. The results of this study show marked differences in the cognitive assessment of each event. For example, players report more motivation in the game after having been fouled (as it is seen as a situation that can be effectively tackled, for example, by playing more physically) in response to a wrong recall by the referee (low perception of situational control as the referee decided and whistled).

Even the perception of danger is also different from one stressor (stress factor) to the other [36]. Missing an easy basket is perceived as a more serious threat than a foul as well as a warning from the referee. The differences in assessment that lead players to use various coping strategies are conditioned by both personal and situational variables [37, 38]. For example, in a competitive situation where the player has a high perception of controllability of the situation, even his or her percentage of coping strategies dealing with the problem is high. Otherwise, the frequent use of disengagement coping strategies occurs for those competitive plays where the player has a low level of controllability, as in the case of an incorrect call by a referee (according to the player). The results of this study [35] confirm the potential costs and benefits of choosing each coping strategy.

Engagement coping strategies are preferable when:

- (a) The situation is controllable.
- (b) The cause of stress is known.

- (c) The individual has a high awareness of the condition.
- (d) The effect that stressors have in the long term such as injury is due to physical contact or due to serious errors during performance.

Disengagement coping strategies may be more appropriate in accordance with the authors' analysis when either of the following occurs: the situation is less controllable such as when a warning by the referee is received; the cause of stress is unclear; the level of awareness of the person is rather low; and the effects are immediate such as a turnover during the game.

It also considers that every assessment of the situation and the level of controllability considers the perception of self-efficacy that the person has of his resources [39–41]. If a player has a low level of self-perceived efficiency, his or her level of controllability of the situation will decrease, and the player will tend to use strategies to avoid the problem. For example, if a coach during a timeout assigns a player an offensive role that the player does not believe he/she is able to do (low perception of self-efficacy), the player will put greater importance on what his or her teammates or opponents know over what he/she can do (low controllability). Hence, this could lead to situations where players sometimes “hide” behind their defender rather than create openings for passes or are not trying to take the initiative; it's not just a matter of technical-tactical or athletic skills.

Research in basketball players has shown that engagement strategies are used more frequently than disengagement strategies [35], as opposed to a study conducted on table tennis players [42].

Why does this difference exist between sports? The answer may stem from the fact that different sports include different stressors; therefore, different coping strategies are required. Table tennis is a continuous game. Cognitive requests are continuous and marked by very short intervals. Distractions from the task could inhibit performance, and strategy variations should be thought out and applied while playing. In basketball, there is often more time and more opportunities to confront or more

stressful events, to think about both for the scoreboard and the bench. Therefore, mistakes during performance in a sport such as table tennis are less controllable, and the athlete cannot afford to re-elaborate them during the playing phase by “taking a timeout”; instead, in basketball, the athlete may make changes to his or her playing style due to mistakes in the same game. The level of controllability is one of the situational variables that could predict the use of coping strategies in relation to performance: high controllability conditions should encourage the subject to use the approaches to deal with the problem, whereas low controllability conditions should instead induce the use of strategies to avoid the problem [43].

Although other studies have shown that coping strategies of dealing with a problem may sometimes be more effective than avoidance, the results of the study by Anshel and Kassids [44] argue that avoidance strategies are associated with a reduced perception of stress in high-pressure situations such as in basketball competitions.

Fact Box 4

“Engagement coping strategies are preferable when: the situation is controllable; the cause of stress is known; the individual has a high awareness of the condition; the effect that stressors have in the long term such as injury is due to physical contact or due to serious errors during performance.”

Coping research helps identify who can recover after stressful events and who is effective at coping [39]. In general, it is accepted that:

- (a) Flexibility is an important quality associated with resistance, stress, and effective coping. Examples of flexibility regarding basketball performances include to be able to play with different teammates; play in different courts; play against different teams; collaborate with different coaches; adapt to different strategies on offense/defense.

- (b) Individuals who have effective coping styles and strive to dominate their environment do not seek to provide excuses for their failures (like commenting that they made a bad pass because “someone else didn’t cut to the basket”).
- (c) Individuals who are considered incapable of reacting often attribute the blame for their failures in life to others [26].

78.5 Summary

Anxiety variables pay particular attention to performance analysis in any sport. Social anxiety, especially in games, or the sense of apprehension and concern over the judgments of others, are dysfunctional variables for performance [9]. Placing too much importance on the opinion of others or overcoming this situation causes the athlete to become easily distracted by the primary objectives of performance, or excessively fragile towards the possibility and fear of making mistakes. Players then react to the latter if they occur. Such arguments could explain the statistically significant difference in the adoption of strategies of denial and avoidance by high-level players. The role of the sports psychologist on the playing field includes the identification of effective mental variables for performance and cooperation with the trainer to develop tactical-technical skills to be integrated with the strategies of face-to-face coping.

Coping strategies are constant cognitive and behavioral changes used to try to handle specific or excessive demands on the person’s resources as the attention is focused on attempting to tackle or avoid the problem. Flexibility is an important quality associated with strength, stress, and effective coping. Individuals who have an effective coping style react and strive to master their environment and do not seek an excuse to justify their failures. Coping strategies are a multidimensional construct: at the lowest level, there is cognitive and emotional information that enables us to structure our perception of the situation. Thoughts and emotions are the basis for choosing a coping strategy that focuses on the problem or the

emotion. Next, at the intermediate level, there are motivations, behaviors, and attitudes that lead to functional behaviors for adaptation, perceptions of control, as well as the probability of realistically solving a problem. Finally, at the highest level, there is a boundary between automation and conscience, between automatic coping skills and conscious coping skills.

As basketball is a sport with many variables that affect the game, including roles [27] and tasks [45], the player needs a good ability to analyze the situation in a very short period of time (fractions of the second). To analyze the play, the athlete must focus only on the primary stimuli to the goals. The adoption of avoidance strategies could, however, favor this analysis because denial and/or isolation can be used concerning the public’s comments or to the voice of the commentator (for example). Even in team sports such as basketball, it is important to adopt strategies that allow you to evaluate only some of the receptive stimuli during the performance [46]. Coping strategies are preferable when the situation is controllable, the cause of stress is known, and the individual has a high awareness of the condition.

References

1. Jackson P. *Basket e Zen*. Milan: Libreria dello sport; 1998. p. 108.
2. Damasio AR. *Descartes’ error*. Emotion, reason and the human brain. New York: Avon Books; 1994. p. 94–95.
3. Eysenck MW, Calvo MG. Anxiety and performance: the processing efficiency theory. *Cognition & emotion*. 1992;6:409–34.
4. Power M, Dalgleish T. *Cognition and emotion from order to disorder*. Hove: Psychology Press; 1997.
5. Gould D, Weinberg RS. *Foundations of sport and exercise psychology*. Champaign III: Human Kinetics; 2011.
6. Smelser NJ, Baltes PB. *International Encyclopedia of Social & Behavioral Sciences*. Amsterdam: Elsevier; 2001. p. 2724.
7. Bandura A. *Self-efficacy: The exercise of control*. New York: Freeman; 1997.
8. Bandura A. Self-efficacy: toward a unifying theory of behavioral change. *Psychol Rev*. 1977;84:191–215.
9. Wilson M, Vine S, Wood G. The influence of anxiety on visual attentional control in basketball free throw shooting. *J Sport Exerc Psychol*. 2009;31:152–68.

10. Eysenck MW, Calvo MG. Anxiety and performance: the processing efficiency theory. *Cognit Emot.* 1992;6:409–34. <https://doi.org/10.1080/02699939208409696>.
11. Vickers JN. Control of visual attention during the basketball free throw. *American Psychological Association, Inc. J Exp Psychol.* 1996;2:342. <https://doi.org/10.1177/036354659602406S25>.
12. Ramat S, Schmid R, Zambarbieri D. Eye-head Coordination in Darkness: Formulation and Testing of a Mathematical Model. *Journal of Vestibular Research.* 2003; 13:79–91.
13. Guitton D. Control of eye-head coordination during orienting gaze shifts. *Trends in Neurosciences.* 1992; 15:174–9.
14. Vercher JL, Magenes G, Prablanc C, Gauthier GM. Eye-head-hand coordination in pointing at visual targets: spatial and temporal analysis. *Experimental Brain Research.* 1994;99:507–23.
15. Calvo MG, Alamo L. Test anxiety and motor performance the role of muscular and attentional demands. *Int J Psychol.* 1987;22:165–78. <https://doi.org/10.1080/00207598708246775>.
16. Raglin JS. Anxiety and sport performance. *Exerc Sport Sci Rev.* 1992;20:243–74. <https://doi.org/10.1249/00003677-199200200-00009>.
17. Spielberg CD. Stress and anxiety in sport. In: Hackfort D, Spielberg CD, editors. *Anxiety in sport: an international perspectives.* New York: Hemisphere; 1989.
18. Harle SK, Vickers JN. Training quiet eye improves accuracy in the basketball free throw. *Human Kinetics Publishers, Inc. Sport Psychol.* 2001;15:290. <https://doi.org/10.1123/tsp.15.3.289>.
19. Lebeau JC, Liu S, Sáenz-Moncaleano C, Sanduvete-Chaves S, Chacón-Moscoso S, Becker BJ, Tenenbaum G. Quiet eye and performance in sport: a meta-analysis. *Human Kinetics Publishers, Inc. J Sport Exerc Psychol.* 2016;38(5):441–57. <https://doi.org/10.1123/jsep.2015-0123>.
20. Vine SJ, Moore LJ, Wilson MR. Quiet eye training facilitates competitive putting performance in elite golfers. *Front Psychol.* 2011;2:1–9. <https://doi.org/10.3389/fpsyg.2011.00008>.
21. Wulf G, Su J. An external focus of attention enhances golf shot accuracy in beginners and experts. *Taylor & Francis. Research quarterly for exercise and sport.* 2007;4:384–9.
22. Singer RN. Preperformance state, routines, and automaticity: What does it take to realize expertise in self-paced events? *J Sport Exerc Psychol.* 2002;24(4):359–75. <https://doi.org/10.1123/jsep.24.4.359>.
23. Singer RN, Murphey M, Tennant LK. *Handbook of research on sport psychology.* New York: Macmillan; 1993.
24. Eysenck MW, Derakshan N, Santos R, Calvo MG. Anxiety and cognitive performance: Attentional control theory. *Emotion.* 2007;7:336–53. <https://doi.org/10.1037/1528-3542.7.2.336>.
25. Coudeville GR, Gernigon C, Ginis KAM. Self-esteem, self-confidence, anxiety and claimed self-handicapping: a mediational analysis. *Psychol Sport Exerc.* 2011;12:670–5. <https://doi.org/10.1016/j.psychsport.2011.05.008>.
26. Burton D, Raedeke TD. *Sport psychology for coaches.* Champaign, IL: Human Kinetics; 2008.
27. Jackson P. *Eleven rings. The soul of success.* New York: Penguin Books; 2014.
28. Folkman S. Dynamics of a stressful encounter: Cognitive appraisal, coping, and encounter outcomes. *J Pers Soc Psychol.* 1986;50(5):992–1003.
29. Lazarus RS, Averill JR. Emotion and cognition: with special reference to anxiety. In: Spielberg CD, editor. *Anxiety: current trends in theory and research.* New York: Academic Press; 1972.
30. Cox RH. *Sport psychology: concepts and applications.* Madison, WI: Brown & Benchmark; 1994.
31. Smith RE, Smoll FL, Schultz RW. Measurement and correlates of sport-specific cognitive and somatic trait anxiety: The sport anxiety scale. *Anxiety Research.* 1990;2:265–80.
32. Folkman S, Lazarus RS, Dunkel-Schetter C, DeLongis A, Gruen RJ. Dynamics of a stressful encounter: cognitive appraisal, coping, and encounter outcomes. *American Psychological Association, Inc. J Pers Soc Psychol.* 1986;50(5):992–1003. <https://doi.org/10.1037//0022-3514.50.5.992>.
33. Carver CS, Scheier MF, Weintraub JK. Assessing coping strategies: a theoretically based approach. *J Pers Soc Psychol.* 1989;56:267–83. <https://doi.org/10.1037//0022-3514.56.2.267>.
34. Crocker P, Kowalski K, Graham T. *Contemporary Advances in Sport Psychology. A Review.* Taylor & Francis. *Measurement of Coping Strategies in Sport.* 2015; 8: 149–161.
35. Anshel MH, Wells B. Personal and situational variables that describe coping with acute stress in competitive sport. *J Soc Psychol.* 2000;140:434–50. <https://doi.org/10.1080/00224540009600483>.
36. Martens R. *Sport competition anxiety test.* Champaign, IL: Human Kinetics; 1977.
37. Martens R. *Coaches guide to sport psychology.* Champaign, IL: Human Kinetics; 1987.
38. Martens R, Vealey RS, Burton D. *Competitive anxiety in sport.* Champaign, IL: Human Kinetics; 1990.
39. Tenenbaum GER. *Handbook of sport psychology.* 3rd ed. Hoboken, NJ: Wiley; 2007.
40. Vealey R. Personality and sport: a comprehensive view. In: Horn TH, editor. *Advances in sport psychology.* Champaign, IL: Human Kinetics; 1992.
41. Weinberg RS. *Foundations of sport and exercise psychology.* Champaign, IL: Human Kinetics; 1995.
42. Krohne HW, Hindel C. Trait anxiety, state anxiety, and coping behavior as predictors of athletic performance. *Anxiety Research.* 1988;1: 25–34. <https://doi.org/10.1080/08917778808248721>.
43. Lazarus RS. *Psychological stress and the coping process.* New York: McGraw-Hill; 1966.

44. Anshel MH, Kaissidis AN. Coping style and situational appraisals as predictors of coping strategies following stressful events in sport as a function of gender and skill level. *British Journal of Psychology*. 1997; 88: 263–276. <https://doi.org/10.1111/j.2044-8295.1997.tb02634.x>.
45. Sampaio J, Ibanez S, Gomez M, Lorenzo A, Ortega E. Game location influences basketball players' performance across playing positions. *Int J Sport Psychol*. 2008;39:205–16.
46. Tauer J, Guenther C, Rozek C. Is there a home choke in decisive playoff basketball games? *J Appl Sport Psychol*. 2009;21:148–62. <https://doi.org/10.1080/10413200902795331>.



Perceptual-Cognitive Processes in Basketball—Individual and Team Aspects

79

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79.1 Introduction: Psychological Factors in Basketball

Basketball is a fast-paced team sport that is dynamic and high scoring, and requires players to develop diverse skills. Players need to master a self-paced, individual, and stable task such as the free throw shot and be able to work together as a team in a dynamic and complex task such as a half-court play involving the entire team. Additionally, players must perform these diverse tasks consistently during high-pressure situations. The ability to ride a “hot streak,” display clutch performance, and make team-based decisions are just several of the unique skills required to perform skillfully in basketball. This chapter presents research findings related to these various unique situations in basketball and provides rec-

ommendations to players and coaches on how to increase the chances of mastering the perceptual-cognitive skills desired to perform consistently at the highest level.

79.2 Free Throw Shooting: A Gaze Behavior Approach

The basketball free throw line is placed 4.6 m (15 ft.) from the basket; there is no defensive pressure and the clock is stopped. The free throw is an instance in basketball where points are given almost for “free” since the thrower has no opposition to consider. Some great shooters take advantage of the free throw and make over 90% of their attempts. On the other hand, there are several notorious cases of outstanding players who have struggled to pass the 50% conversion rate. Thus, without any physical constraints such as the opposition players, time, and distance from the basket why do highly skilled players struggle to convert the free throw?

Sport psychology is a domain which aims at exploring the factors interfering with motor performance such as the free throw. As a far aiming task, a basketball free throw requires a high level of attention to the task at hand and avoidance of distractions from nonrelevant cues, either external (e.g., distracting objects waved by opposing fans) or internal (e.g., anxiety stemming from

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self-doubt) [1]. The visual attention strategy (i.e., gaze behaviors) plays a crucial role in maintaining and enhancing attention levels that are crucial for performance. Gaze behaviors indicate the degree to which a person is devoting covert attention to a specific object or scene [2]. Moreover, it has been suggested that it is difficult to shift the point of gaze without shifting attention [3]; hence, examining basketball players' gaze behaviors provides valuable information on acquiring, processing, and using visual information to make a free throw shot.

To perform this type of research, high-technology glasses called eye trackers have been used (see Fig. 79.1). Modern eye-trackers look like regular eye-glasses and incorporate at least two high-frequency cameras. One of the cameras is mounted onto the frame's anterior side (or on top of the frame) and enables to record

the player's point of view. The other camera (or cameras) is placed so that it is looking toward the player's eye and records pupil movement. With the use of specialized software, both images are processed, and fixations (location, duration and order), saccades (i.e., rapid movements to change the eyes' location) and eye blinks are identified (see Fig. 79.1).

In sports, gaze behaviors have been examined extensively to identify visual search strategies. One of the most commonly used research methods has been the differentiation between skilled and less-skilled athletes (see review [4]). The findings repeatedly indicated that experts demonstrate fewer fixations in comparison to their lower-skilled counterparts. However, those fixations are longer in duration [4]. Another well-studied phenomenon identified in athletes has been a fixation called the quiet-eye (QE), which is defined as the final fixation before movement initiation; that is, the amount of time a basketball player gazes at the rim (or whatever they are fixating on) before initiating the throwing movement. Numerous studies have demonstrated that longer QE periods are indicative of superior performance in sports [5].

One of the seminal studies in this field was conducted by Vickers [6]. In this study, eight experts (free throw statistics over a full season above 75%) and eight near-expert (<65%) basketball players performed free throws while using an eye-tracker. Results showed that expert players maintained their eyes fixated on one single location (most often the rim's front) for a longer period of time prior and during shooting the ball (1058 ms in comparison to 773 ms by near-experts). Similarly, the QE duration was longer during experts' hits (mean 972 ms) than misses (806 ms), and both durations were significantly longer than the near-experts' hits or misses (a mean of less than 400 ms). These results clearly indicate the imperative role of holding a steady fixation until the shooting action begins. An explanation of the findings suggests that during the QE period task-relevant information is being processed, and motor programs are retrieved and coordinated to successfully perform the action. Once this motor

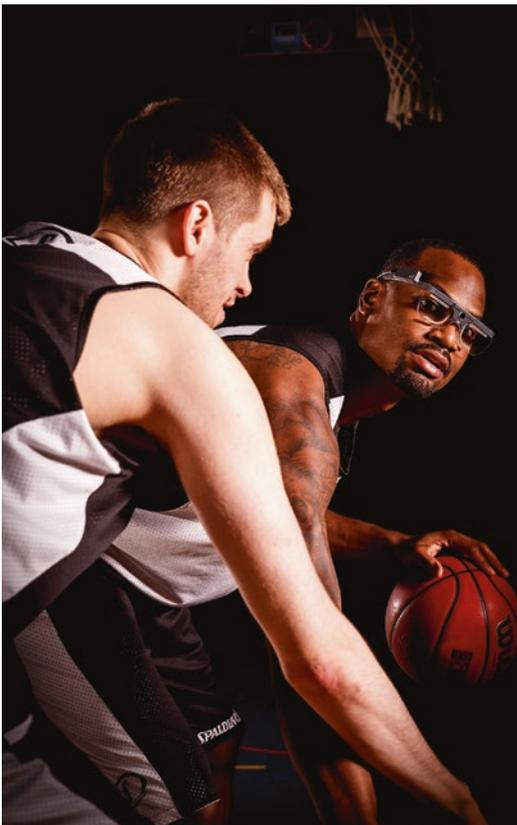


Fig. 79.1 Tobii Glasses 2 being used by a basketball player

programming period is interrupted by distractions or is too short, performance is affected and deteriorated [6].

A more recent study by Oliveira et al. [7] examined six experienced basketball players (four men and two women) performing free throws. However, in this study the researchers took into account the shooter's prevailing shooting style: low-style shooter, who occlude the target with their hands during the final elbow extension, and high-style shooters, to whom the target is always visible until ball release. Results indicated that high-style shooters fixated approximately 780 ms and similar to Vickers' [6] findings, low-style free throwers fixated for over 1 s (1213 ms), but also players fixated their eyes on the rim even after the ball entered the player's field of view. Oliveira et al. [7] suggested that basketball free throw (and jump-shot) shooting is largely controlled by online vision. Visual information is acquired and processed prior, during and after movement execution (and is not only relevant prior to movement initiation to preprogram the movements). Furthermore, they suggest that the specifics of the timing of the acquisition of visual information are dependent on the prevailing shooting style [7].

Notwithstanding the debate on the mechanisms underlying the QE-performance relation, the literature has shown that maintaining a longer fixation before and during the shooting seems to play an important role in performance. Therefore, gaze behaviors and QE training protocols have been proposed to enhance athletic performance [8, 9]. In short, to improve free throws, it has been suggested that players follow these steps: (1) Assume the stance, ensure that the gaze is directed on a preferred spot and bounce the ball as many times as you are used to; (2) Hold the ball in the shooting stance and direct the eyes on a single preferred location for about 1.5 s. Regardless if it is on the front rim, back rim, or the backboard, lock your eyes on that direction without moving them; (3) Shoot and maintain the fixation on the preferred location for around half a second after the ball is released.

Fact Box 1

Gaze behaviors and QE training protocols have been proposed to enhance athletic performance. In order to improve free throws, it has been suggested that players need to:

1. Assume the stance, ensure that the gaze is directed on a preferred spot and bounce the ball as many times as you are used to;
2. Hold the ball in the shooting stance and direct the eyes on a single preferred location for about 1.5 s. Regardless if it is on the front rim, back rim, or the backboard, lock your eyes on that direction without moving them;
3. Shoot and maintain the fixation on the preferred location for around half a second after the ball is released.

79.3 “The Hot Hand”: Increasing the Chances of “Being Hot”

On January 23, 2015, Klay Thompson (from the Golden State Warriors) had the best shooting quarter of any player in the National Basketball Association (NBA) [10]. He made 13 shots in a row without missing even one shot and ended the 3rd quarter with 37 points. His teammate, Draymond Green, was interviewed after the game and was asked what he thought of Klay Thompson's “hot hand” and that some people believe it is a fallacy. He replied by saying: “That's garbage. If we didn't believe it, we believe it now” [11]. On the other hand, Tim Hardaway Sr. missed all 17 shots he took in a game (December 27, 1991), going 0 for 17 [12]. Noteworthy, both players have similar career field goal shooting percentages (around 45%).

Hot (and cold) streaks in sports, especially in basketball, are perceived by players, fans, and coaches as meaningful and real [13]. However, since Gilovich et al.'s [13] seminal study, the majority of studies supported the notion that these streaks are statistically random, meaning

that the chances are not better (or worse) of making a shot after making a few shots compared to missing a few (Avugos et al. [14] and Bar-Eli et al. [15]). In the past 5 years, a few researchers challenged this notion by introducing innovative approaches (e.g., taking into account shot difficulty) [16] and advanced statistical methods [17]. However, even in these studies the “hot hand” phenomenon, if exists, is relatively small and is not as prevalent as believed by players, coaches, and fans.

It is thus inferred that from a statistical perspective, the majority of research indicates that the “hot hand” is a fallacy and an illusion (or if it exists it is much weaker than perceived). However, from a perceptual-cognitive and applied approach, when someone is perceived to be hot or cold, the decision-making process changes. For example, Klay Thompson attempted 13 shots in the “hot” quarter, when his career average is 16 per game. Would he have attempted so many shots if he had made 30% of his shots in that quarter? Indeed, research indicates that when a player is perceived to be on a hot streak, the decision-making process (of the player, teammates, opponents, and coaches) changes such that a player (a) increases the likelihood of taking more shots, (b) takes shots that are more difficult (e.g., further distance and jump shots), (c) has less chance to be replaced by a coach, and (d), from a defensive perspective, is guarded tighter [16, 18]. Whether these decisions are effective or not is debatable [19, 20]; nonetheless, in real-world situations players and coaches react to a hot shooter, and the decision-making process changes accordingly.

Even more important than the decision-making process *during* a “hot streak” is the understanding of the mechanisms *leading* to (and sustaining) a “hot streak.” Research exploring similar (and related) theories to the “hot hand” phenomenon such as momentum [21, 22], zone of optimal functioning [23, 24], and flow [25, 26] provide plausible explanations of the processes that increase the likelihood of a “hot streak” occurring. The theories differ in their theoretic

cal underpinning and the mechanisms that lead to successful performance but are similar in their predictions; that there are instances where players perform better than their typical performance (see also clutch performance in the section below) [27].

For example, taking the *zone of optimal functioning* perspective, which takes a probabilistic approach to performance, being in the zone increases the chances of performing optimally. The zone is idiosyncratic and depends on various psychophysiological factors, such as (a) emotions—ranging on a 2×2 continuum of hedonic tone (i.e., positive/negative) and intensity, (b) arousal level—physiological and emotional, (c) efficacy—the belief in the ability to perform well on a task, and (d) cognitive appraisal—interpreting the situation as a challenge or a threat [24]. From a momentum perspective, the notion is that success breeds success, and thus the emphasis is on maintaining and prolonging the positive momentum [22]. For example, during positive momentum periods, anxiety levels decrease, and efficacy levels increase, thus allowing the positive momentum to continue [21]. From a flow perspective, when performing optimally, everything seems to be working well, and there is complete attention to and absorption in the task [25]. Factors that contribute to the occurrence of a flow state include feeling prepared, receiving positive feedback, being fully concentrated (no or minimal distractions) and in control of thoughts and emotions [26].

Thus, to increase the probability of hot streaks, coaches and players must focus on the processes that can help perform at the optimal level in a consistent manner. This can be conducted by systematically measuring related variables (e.g., arousal and efficacy) before and during every game. This procedure allows the player and coach to capture the reasons and circumstances which increase or decrease the probability of the “hot streak” occurrence, so that in future instances they can increase the chances of optimal performances (and maintaining them) and consequently increase the likelihood of a “hot streak.”

Fact Box 2

Research indicates that when a player is perceived to be on a hot streak, the decision-making process (of the player, teammates, opponents, and coaches) changes such that a player (a) increases the likelihood of taking more shots, (b) takes shots that are more difficult (e.g., further distance and jump shots), (c) has less chance to be replaced by a coach, and (d), from a defensive perspective, is guarded tighter.

79.4 Performing Under Pressure: Last Minute “Clutch” and “Choking” Performances

Imagine this: it is the NBA finals, and your team is down by 8 points with 60 s left in the game. If you lose this game, you are eliminated and can no longer fight for the championship title; on the other hand, if you rally to win, then your team is put in a position to contest for the title. How do you handle the situation (are you even capable)? Does your performance excel or breakdown? What do you do to get yourself in a position to succeed? This section introduces and discusses the processes of “choking” and “clutch” performance in basketball and the psychological skills that can be used to cope with pressure situations.

“Choking” is a psychophysiological disruption that occurs during stressful instances (e.g., pressure situations) in which an athlete’s performance declines unexpectedly with respect to what is expected [28]. There are two main competing theories that are used to explain the processes that lead to the phenomenon: *distraction* and *self-focus theories*. Distraction theorists posit that during a heightened anxiety state, an athlete’s attention shifts from task-relevant cues (e.g., opponents, basket, and teammates) to task-irrelevant cues (e.g., crowd, music, and opponent’s bench), thus resulting in “choking” [29]. On the other hand, self-focus theorists suggest that “choking” occurs when the athlete is

highly self-aware and as a result, reverses from an autonomous to a step-by-step processing during moments of high anxiety (e.g., taking two free throw shots in the final seconds of the game when the score is tied), essentially regressing to a novice-type processing mechanism leading to poor performance [30].

Tenenbaum’s [31] sport-related decision-making model provides an illustration on the process athletes undergo while performing (see Fig. 79.2). Distraction and self-focus processes affect performance by influencing the efficiency of the athlete’s sport-related decision-making process. The first stage of the decision-making process is identifying the relevant environmental cues necessary to maintain optimal performance on the task and to allocate visual attention accordingly. For example, when taking a free throw, it is pertinent to attend to the relevant cues (e.g., the basket) and eliminate the irrelevant cues (e.g., the crowd behind the basket trying to visually distract the athlete taking the shot) for more efficient information processing. It is in this section where an athlete’s inability to cope with the heightened anxiety situation causes a disruption to the decision-making process, thus resulting in poor performance and “choking.”

Inversely, when an athlete is put in a high anxiety situation and has the necessary resources to cope with the situation, then “clutch” performance occurs. “Clutch” performance is defined as any heightened performance that occurs under high pressure situations [32]. To be in a “clutch state,” the athlete must be aware of the situational pressure, have the ability to function within the stressful state, perceive the outcome of the task as important, and succeed in large part through effort [33]. Swann and colleagues [32] conducted a study evaluating “clutch state” and the underlying mechanisms within three primary areas: the performance context, process of occurrence, and outcomes. The performance context is the first step of the model, where “clutch” is identified as an important moment with the outcome on the line. Once the stressful situation is identified (i.e., high pressure), the athlete appraises the challenge, identifies what needs to be done, and

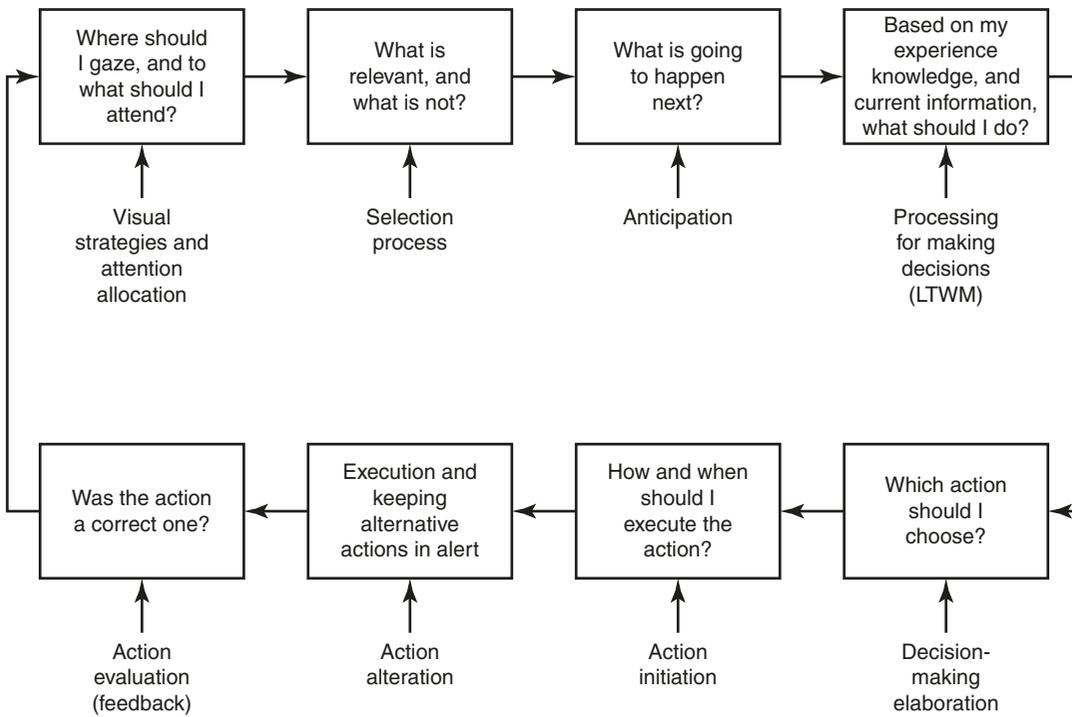


Fig. 79.2 Sport-related decision-making model ([31], p. 195)

makes a decision to increase the effort and intensity of task execution. The authors found that athletes who experienced clutch performance had heightened awareness, deliberate attention allocation, an absence of negative thoughts, and task execution was autonomous [32]. The combination of factors in the performance context and process of occurrence resulted in the athletes feeling a sense of exhaustion, achievement, and satisfaction.

An additional concern is related to how basketball players cope with pressure situations (e.g., when the score is tied, and there are 20 s left in the game) and increase the likelihood of “clutch” performances. Based on the self-focus approach, attention allocation training can be used [30]. The primary goal is to divert attention externally. That is, revert the attentional shift from internal (e.g., attending to the step-by-step technical shooting process) to external (e.g., attending to the rim). The shift back to external attention could be achieved through different modalities. For example, Mesagno et al. [34] used music to alter the athletes’ attention allocation. When pre-

sented with a free throw task, athletes were asked to focus their attention to the lyrics of the song while shooting the ball. This method alleviated the occurrence of “choking” as it allowed the athlete to divert their attention away from a step-by-step processing and shift to automatic mode.

Alternatively, if a distraction theory approach is being implemented to actions, then players can use preperformance routines to attend to the relevant cues. A preperformance routine creates a systematic structure for the athlete to rely on when experiencing a high demand situation [35]. Thus, in a high-pressured free throw situation, attention allocation shifts internally and to a worrying thought (e.g., the importance of the shots and what will happen if missed) [36]. A preperformance routine can be implemented to refocus and eliminate any irrelevant thoughts [37]. One of the first steps in developing a preperformance routine is to create awareness to previous actions and schemas stored in long-term memory [38]. Once the athlete becomes aware of what he/she currently do, then they can begin to understand the behavior, construct a planned routine, modify

that routine and control (i.e., practice) the routine in order to generalize it for game time situations [38]. An example of a preperformance routine for a free throw shot follows these steps: (1) receive the ball from referee; (2) once the ball is caught, position yourself at the spot on the free throw line you are comfortable at; (3) take two deep breaths; (4) bounce the ball three times; (5) take one more breath and imagine the ball leaving your hand and following a perfect arch, moving smoothly through the net, making a “swoosh” sound; (6) put the ball at eye level; (7) look at the basket for 1.5 s; (8) shoot the ball. When developing a pre-performance routine, it may seem cumbersome; however, with practice, it will become automatic and part of the free throw shot process.

Fact Box 3

Once the stressful situation is identified (i.e., high pressure), the athlete appraises the challenge, identifies what needs to be done, and makes a decision to increase the effort and intensity of task execution. The authors found that athletes who experienced clutch performance had heightened awareness, deliberate attention allocation, an absence of negative thoughts, and task execution was autonomous.

79.5 Team Decision-Making: Shared Mental Models and Related Team Dynamics

One of the most fascinating phenomena in basketball is watching an offense that is in complete coordination. The players’ moves look slick, fluid, and synchronized and their plays usually end with a score as a product of perfectly timed or a “mind-reading” pass that leaves the most refined defense completely confused. How does a team achieve those levels of coordination and decision-making that seem to be simply magical? This is a question that not only coaches and athletes marvel about but also sport psychologists. In the last decades, a social-cognitive theoretic

cal approach called “*shared mental models*” has been proposed as an explanation of how superior coordination is achieved in team sports [39].

Individual expert performance in sport embodies an athlete’s superior ability to acknowledge and recall structured information while performing in a specific domain. This is a result of advanced knowledge structures developed through years of practice, which allow the transformation of small bits of information into bigger and more meaningful components [40]. The ability to recognize and recall cues that are specific for sport performance is fundamental to memory structures in which previously acquired information is encoded into short-term memory and stored in a retrievable mode in long-term memory [41, 42]. In short, athletes construct their mental models of a sport through practice (and experience) and are able to update them during competition in order to perform successfully and continue learning and mastering their basketball knowledge.

Unsurprisingly, these individual-level processes do not seem to fully explain what happens at a group level since when more people are involved, processes become more complex. For instance, team sport performance is dependent on a cohesive interaction between team members, which is achieved by having group training in addition to individual practice [43]. The idea is that through training, teams reach a psychological state in which each team member has a shared understanding (i.e., models) of the situation and how its constituent members will perform the task, and all the members can draw from the same knowledge [44].

In order to achieve team coordination, two types of shared knowledge must be established. First, shared knowledge needs to be established prior to competition. During practice sessions, the coaching staff designs training sessions that help players develop shared knowledge of situational probabilities in basketball in general and against a team in particular by placing players in prototypical gaming situations that require them to develop interpersonal synergies. In order to do this, a classic work by Hayes-Roth and Hayes-Roth [45] established four levels in the planning and decision-making training that vary in

abstraction. The more general level, called the *Intention*, describes the desired outcome of the team (e.g., winning the game by 10 points). This level is followed by the *Scheme* level in which the team's general behavior is established (i.e., up-tempo fast break). The third level is related to the *Strategy* which encompasses the specific procedures the team will maintain during the game (i.e., double team defense). Lastly, the most specific level is the *Tactic* in which the specific operations will be planned (i.e., how to execute a specific play when the game is about to end). If the coaching staff are able to communicate these levels properly, each basketball player should know (a) when to use each play and which coach/team leader signal indicates them, (b) what action those players involved need to execute, and (c) how his/her actions coordinate with the actions of a teammate [46].

Since basketball is a very dynamic game, shared knowledge must be established and updated during the game. One of the biggest challenges with this dynamic knowledge is that team members must acknowledge that it has changed, and thus, the team must update the shared knowledge during a game [47]. The game adjustment can occur by two means, either *incidentally* or *deliberately*. Via the former mean, one or two players start adopting strategies different to the ones established prior to the game as a response to game situations. For example, players may start defending man to man after recognizing that the zone defense they were using is ineffective with the opponent team. When other teammates notice changes in their teammates' strategy, they may realize that the predetermined strategy has been abandoned and that an adjusted strategy is being implemented. The second way of updating is via deliberate means, either through verbal (e.g., shouting) or non-verbal communication (e.g., pointing) [46]. In basketball, this can be seen very frequently during time-outs when coaches draw new plays on the whiteboard or explain new playing styles players should adopt, as well as during active play, when coaches often verbally communicate and use predetermined hand signs to pass instructions to players.

Excellent performance in basketball depends on how coordinated a group of five individuals performs on the court, and this coordination has its roots in the shared mental models that the team has created in order to always be "on the same page." Coaches and athletes must reach these levels of collective understanding prior to the game through the development of training plans that allow each member to understand how their game is going to play out. In addition, during games, successful teams are able to update their shared knowledge as the dynamic situations in basketball constantly change and posit new challenges for the team. Enhancing communication in basketball teams is paramount since the way in which strategies are communicated and learned by team members plays an important role in the development of shared knowledge.

79.6 Summary, Limitations, and Future Directions

Unique situations in basketball require specific psychological skills. The diverse skills a player must maintain are as follows: (a) prolong the quiet-eye period during free throws, (b) control emotions and thoughts (i.e., be in the zone) to increase the chances of a hot streak, (c) perceive pressure situations as challenging (as opposed to threatening) to achieve optimal and clutch performance, and (d) share knowledge among teammates to perform as an expert team (and not as individuals). Mastering these skills enable individual players and the team to perform at the highest level.

Mastering these skills takes time, practice, and guidance. Most of the focus in training is on developing the physical, technical, and tactical skills. Unfortunately, psychological skills are only addressed when problems arise, and not a sufficient amount of time is dedicated to the psychological aspect of the game in general. Thus, players and teams do not benefit from a consistent and systematic psychological training program (similar to strength and conditioning), and often the perceptual-cognitive skills are not fully developed. A systematic training plan must be

implemented within every team (and from youth to professional level) to enhance the effectiveness of these psychological processes.

Finally, the advancement of technology in sport science, specifically in the sport psychology domain, enables to measure and train perceptual-cognitive skills. The eye-tracker is one such technology that helps monitor gaze behaviors of players during various tasks (e.g., free throws, passing, and defending). However, a lack of technology that can measure team processes, such as shared mental models, is apparent. More research and applied efforts must be geared to the team unit of analysis (as compared to the individual) so that teams can perform in unity with high levels of cohesion, chemistry, communication, and coordination.

References

1. Wilson MR, Wood G, Vine SJ. Anxiety, attentional control, and performance impairment in penalty kicks. *J Sport Exerc Psychol.* 2009;31:761–75. <https://doi.org/10.1123/jsep.31.6.761>.
2. Posner MI, Raichle ME. *Images of mind.* New York, NY: Scientific American Library; 1997.
3. Henderson JM. Human gaze control during real-world scene perception. *Trends Cogn Sci.* 2003;7:498–504. <https://doi.org/10.1016/j.tics.2003.09.006>.
4. Mann DT, Williams AM, Ward P, Janelle CM. Perceptual-cognitive expertise in sport: a meta-analysis. *J Sport Exerc Psychol.* 2007;29:457–78. <https://doi.org/10.1123/jsep.29.4.457>.
5. Lebeau JC, Liu S, Sáenz-Moncaleano C, Sanduverte-Chaves S, Chacón-Moscoso S, Becker BJ, Tenenbaum G. Quiet eye and performance in sport: a meta-analysis. *J Sport Exerc Psychol.* 2016;38:441–57. <https://doi.org/10.1123/jsep.2015-0123>.
6. Vickers JN. Control of visual attention during the basketball free throw. *Am J Sports Med.* 1996;24(suppl):S93–7. <https://doi.org/10.1177/036354659602406S25>.
7. de Oliveira RF, Oudejans RR, Beek PJ. Gaze behavior in basketball shooting: further evidence for online visual control. *Res Q Exerc Sport.* 2008;79:399–404. <https://doi.org/10.5641/193250308X13086832906193>.
8. Harle SK, Vickers JN. Training quiet eye improves accuracy in the basketball free throw. *Sport Psychol.* 2001;15:289–305. <https://doi.org/10.1123/tsp.15.3.289>.
9. Vine SJ, Moore L, Wilson MR. Quiet eye training facilitates competitive putting performance in elite golfers. *Front Psychol.* 2011;2:8. <https://doi.org/10.3389/fpsyg.2011.00008>.
10. Case J. Legendary moments in NBA history: Klay Thompson's 37-point quarter in 2015 NBA History. 2018. <https://www.nba.com/article/2018/01/23/week-history-klay-thompson-37-point-quarter-vs-sacramento-kings>. Accessed 20 Mar 2019.
11. Pollakoff B, Draymond Green, amazed by Klay Thompson's performance, gives outstanding post-game interview. NBC Sports: Pro Basketball Talk. 2015. <https://nba.nbcsports.com/2015/01/24/draymond-green-amazed-by-klay-thompsons-performance-gives-outstanding-postgame-interview-video/>. Accessed 20 Mar 2019.
12. Turner G. 15 of the worst single-game performances in NBA history. *Complex* 2015. <https://www.complex.com/sports/2014/03/worst-single-game-performances-nba-history/>. Accessed 21 Mar 2019.
13. Gilovich T, Vallone R, Tversky A. The hot hand in basketball: on the misperception of random sequences. *Cogn Psychol.* 1985;17:295–314. [https://doi.org/10.1016/0010-0285\(85\)90010-6](https://doi.org/10.1016/0010-0285(85)90010-6).
14. Avugos S, Köppen J, Czienskowski U, Raab M, Bar-Eli M. The “hot hand” reconsidered: a meta-analytic approach. *Psychol Sport Exerc.* 2013;14:21–7. <https://doi.org/10.1016/j.psychsport.2012.07.005>.
15. Bar-Eli M, Avugos S, Raab M. Twenty years of “hot hand” research: review and critique. *Psychol Sport Exerc.* 2006;7:525–53. <https://doi.org/10.1016/j.psychsport.2006.03.001>.
16. Bocskocsky A, Ezekowitz J, Stein C. The hot hand: a new approach to an old “fallacy”. 2014. Paper presented at the MIT Sloan Sports Analytics Conference, Boston.
17. Miller JB, Sanjurjo A. Surprised by the hot hand fallacy? A truth in the law of small numbers. *Econometrica.* 2018;86(6):2019–47. <https://doi.org/10.3982/ECTA14943>.
18. Attali Y. Perceived hotness affects behavior of basketball players and coaches. *Psychol Sci.* 2013;24(7):1151–6.
19. Spencer Ingels J, Rhodius AA. Novel approach to investigating basketball experts' perceptions of the hot hand. *J Sport Behav.* 2016;39(2):807–19.
20. Tversky A, Gilovich T. The cold facts about the “hot hand” in basketball. *Chance.* 1989;2(1):16–21.
21. Briki W, Den Hartigh R, Bakker C, Gernigon C. The dynamics of psychological momentum: a quantitative study in natural sport situations. *Int J Perform Anal Sport.* 2012;12(3):573–92.
22. Cornelius A, Silva J, Conroy D, Petersen G. The projected performance model: relating cognitive and performance antecedents of psychological momentum. *Percept Mot Skills.* 1997;84(2):475–85.
23. Jokela M, Hanin Y. Does the individual zones of optimal functioning model discriminate between successful and less successful athletes? A meta-analysis. *J Sports Sci.* 1999;17(11):873–87.
24. Tenenbaum G, Basevitch I, Gershgoren L, Filho E. Emotions–decision-making in sport: theoretical

- conceptualization and experimental evidence. *Int J Sport Exer Psychol.* 2013;11(2):151–68.
25. Csikszentmihalyi M. *Flow and the foundations of positive psychology.* New York: Springer; 2016.
 26. Swann C, Keegan R, Piggott D, Crust L. A systematic review of the experience, occurrence, and controllability of flow states in elite sport. *Psychol Sport Exerc.* 2012;13(6):807–19.
 27. Koehler J, Conley C. The “hot hand” myth in professional basketball. *J Sport Exerc Psychol.* 2003;25(2):253–9.
 28. Baumeister R, Showers C. A review of paradoxical performance effects: choking under pressure in sports and mental tests. *Eur J Soc Psychol.* 1986;16(4):361–83.
 29. Hardy L, Mullen R, Martin N. Effect of task-relevant cues and state anxiety on motor performance. *Percept Mot Skills.* 2001;92(3):943–6.
 30. Baumeister R. Choking under pressure: self-consciousness and paradoxical effects of incentives on skillful performance. *J Pers Soc Psychol.* 1984;46(3):610–20.
 31. Tenenbaum G. An integrated approach to decision making. In: Starkes JL, Ericsson KA, editors. *Expert performance in sport: advances in research on sport expertise.* Champaign, IL: Human Kinetics; 2003. p. 191–218.
 32. Swann C, Crust L, Jackman P, Vella S, Allen M, Keegan R. Performing under pressure: exploring the psychological state underlying clutch performance in sport. *J Sports Sci.* 2016;35(23):2272–80.
 33. Hibbs D. A conceptual analysis of clutch performances in competitive sports. *J Philos Sport.* 2010;37(1):47–59.
 34. Mesagno C, Marchant D, Morris T. Alleviating choking: the sounds of distraction. *J Appl Sport Psychol.* 2009;21(2):131–47.
 35. Jackson RC. Preperformance routines. In: Eklund RC, Tenenbaum G, editors. *Encyclopedia of sport and exercise psychology.* Thousand Oaks, CA: Sage Publications, Inc; 2014. p. 550–3.
 36. Beilock SL, Gray R. Why do athletes choke under pressure? In: Tenenbaum G, Eklund RC, editors. *Handbook of sport psychology.* 3rd ed. Hoboken, NJ: John Wiley & Sons Inc.; 2007. p. 425–44.
 37. Cotterill ST, Sanders R, Collins D. Developing effective pre-performance routines in golf: why don't we ask the golfer? *J Appl Sport Psychol.* 2010;22(1):51–64.
 38. Cotterill ST. Experiences of developing pre-performance routines with elite cricket players. *J Sport Psychol Action.* 2011;2(2):81–91.
 39. Eccles DW, Tenenbaum G. Why an expert team is more than a team of experts: a social-cognitive conceptualization of team coordination and communication in sport. *J Sport Exerc Psychol.* 2004;26:542–60.
 40. Ericsson KA, Delaney PF. Long-term working memory as an alternative to capacity models of working memory in everyday skilled performance. In: Miyake A, Shah P, editors. *Models of working memory.* Cambridge, MA: Cambridge University Press; 1999. p. 257–97.
 41. Tenenbaum G, Land WM. Mental representations as an underlying mechanism for human performance. In: Raab M, Johnson JG, Heekeren HR, editors. *Progress in brain research: mind and motion—the bidirectional link between thought and action.* New York, NY: Elsevier Publication; 2009. p. 251–66.
 42. Woo Sohn Y, Doane SM. Roles of working memory capacity and long-term working memory skill in complex task performance. *Mem Cogn.* 2003;31:458–66.
 43. Baker J, Cote J, Abernethy B. Sport-specific practice and the development of expert decision-making in team ball sports. *J Appl Sport Psychol.* 2003;15(1):12–25.
 44. Lee BPH. Mutual knowledge, background knowledge and shared beliefs: their roles in establishing common ground. *J Pragmat.* 2001;33(1):21–44.
 45. Hayes-Roth B, Hayes-Roth F. A cognitive model of planning. *Cogn Sci.* 1979;3(4):275–310.
 46. Eccles DW, Turner KB. Coordination in sports teams. In: Beauchamp MR, Eys MA, editors. *Group dynamics in exercise and sport psychology.* 2nd ed. New York, NY: Routledge; 2014. p. 240–56.
 47. Silva P, Garganta J, Araújo D, Davids K, Aguiar P. Shared knowledge or shared affordances? Insights from an ecological dynamics approach to team coordination in sports. *Sports Med.* 2013;43(9):765–72.



Psychological Aspects in Return to Sport Following ACL Reconstruction

80

Ryan Zarzycki and Clare Ardern

80.1 Part 1: Introduction

Despite advances in surgical techniques and the development of criterion-based postoperative rehabilitation protocols, outcomes following anterior cruciate ligament reconstruction (ACLR) are not optimal. Only 65% of athletes that undergo ACLR return to their previous level of sport [1]. Young individuals that do return to sport (RTS) are at high risk of sustaining an ACL injury [2–4]. Additionally, self-reported functional deficits, strength deficits, and movement asymmetries persist for years after ACLR [5–7]. Psychological factors, such as fear or reinjury and lack of confidence, contribute to these suboptimal outcomes.

An athlete's psychological response to injury commonly follows a “U”-shaped curve [8–10]. Early after injury, negative emotions, such as anger and depression predominate. The severity of these negative emotions is related to the athlete's appraisal of the injury (i.e., the severity of the injury and the time taken to return to sport). Generally, during the initial stages of rehabilitation, these negative emotions improve [11]. However, frustration appears to be the most common negative emotional reaction during rehabili-

tation and is influenced by the difficulty of the rehabilitation program and the athlete's perceived value of the rehabilitation program [8]. As the athlete transitions to the return to sport phase of rehabilitation, negative emotions related to fear of reinjury and lack of confidence may become more pronounced [12]. Maladaptive psychological responses are therefore present at multiple time points post-injury, affect progression through rehabilitation, and have long-term implications on outcomes.

In order to optimize outcomes following ACLR, clinicians must consider psychological factors and look beyond physical impairments. This chapter will review the current evidence regarding the relationship between psychological factors and outcomes after ACLR, discuss the relationship between psychological factors and movement after ACLR, discuss psychological outcome measures and potential interventions, and finally propose recommendations for future research in this area.

80.2 Part 2: Psychological Factors and Outcomes

80.2.1 Return to Sport/Activity Outcomes

There is strong evidence linking psychological factors to activity-related outcomes in patients after ACLR, and fear of reinjury is one of the most

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commonly reported reasons for not returning to sport after ACLR [11, 13]. Cross-sectional studies have examined fear of reinjury and other psychological factors to determine if differences exist between individuals who returned to sport and individuals who did not return to sport at different time points after ACLR. Ardern and colleagues examined psychological factors in 164 individuals 1–7 years after ACLR [14]. Psychological readiness to return to sport, measured via the Anterior Cruciate Ligament Return to Sport after Injury scale (ACL-RSI), was the strongest variable associated with preinjury activity level. Athletes who had returned to preinjury level of sport demonstrated higher scores on the ACL-RSI, lower kinesiophobia (measured with the Tampa Scale of Kinesiophobia shortened version, TSK-11), and higher knee self-efficacy. Lentz and colleagues examined demographic, physical impairments, and psychological factors in 94 individuals 1 year after ACLR [15]. Individuals who had returned to preinjury level of sport had less knee effusion, fewer episodes of knee instability, lower pain rating, greater quadriceps strength, higher self-reported function, and lower levels of kinesiophobia. The significant group difference in TSK-11 score exceeded the minimal clinically important difference.

Observational studies have also investigated the association between psychological factors and RTS. Ardern and colleagues examined fear of reinjury, psychological readiness, and sport locus of control preoperatively and 4 months after ACLR [16]. These psychological factors measured preoperatively and at 4 months after ACLR predicted return to preinjury level of sport 1 year after ACLR. Psychological readiness at 4 months after ACLR was the variable with the best discriminatory capability, and patients with a score of less than 56 on the ACL-RSI indicated an increased risk of not returning to sport by 1 year. A second study by Ardern and colleagues assessed a cohort of individuals who had not returned to sport at 1 year after ACLR [17]. Individuals in this cohort that did not return to preinjury sport level by 2 years demonstrated poorer hop test symmetry, poorer self-reported knee function, and more negative psychological

responses compared to individuals that did return to preinjury sport level. Another large cohort study found that individuals who had returned to sport at 8 or 12 months after ACLR had higher self-efficacy and psychological readiness scores compared to individuals who had not returned to sport at each time point, respectively [18]. Lentz and colleagues examined kinesiophobia, muscle strength, self-reported function, and pain in patients 6 months after ACLR [19]. They surveyed these same patients 1 year after ACLR to determine return to sport (RTS) status and why they had not returned to sport if indeed they had not. Of the 73 patients included in this study, 46 had RTS, 13 had not RTS, and 14 had not returned to sport due to fear of reinjury and/or lack of confidence. The group of patients that had not RTS due to fear of reinjury/lack of confidence had greater kinesiophobia, lower quadriceps strength, and lower self-reported function at the 6-month time point.

80.2.2 Relationship Between Psychological Factors and Self-Reported Function/Knee Impairments

There is evidence linking psychological factors and self-reported knee function early after ACLR. There is an association between a change in psychological factors (kinesiophobia and self-efficacy) and change in self-reported function from 4 to 12 weeks after ACLR [11]. Self-reported knee function is also associated with kinesiophobia in athletes greater than 6 months after ACLR [20].

There is also a relationship between psychological factors and knee impairments early after ACLR. Greater pain catastrophizing and lower knee self-efficacy early after ACLR (1 and 4 weeks) is associated with poorer ROM 12 weeks after ACLR and increases the odd of not meeting advanced rehabilitation criteria (Pain ≤ 2 , symmetrical ROM, and quadriceps index $\geq 60\%$) [21]. Additionally, self-efficacy measured 4 months after ACLR is associated with strength 1 year after ACLR.

The evidence linking psychological factors, self-reported function, and impairment resolution emphasizes the importance of “good” therapy early after ACLR. Restoring full ROM, improving quadriceps strength, and improving knee function early after ACLR may lead to better a better psychological outlook (i.e., better self-efficacy, lower kinesiophobia, and pain catastrophizing) and set the stage for later phases of rehabilitation and return to sport.

80.2.3 Relationship Between Psychological Factors and Reinjury

Recent evidence indicates that psychological factors, specifically kinesiophobia and psychological readiness, may be related to second ACL injury. Athletes who experienced an ipsilateral graft rupture within 1 year from surgery reported higher kinesiophobia at the time of return to sport clearance (mean 7.6 months after ACLR). [22] Studies examining psychological readiness support the premise that negative psychological factors are associated with second ACL injury. Younger athletes (≤ 20 years old) who sustained a second ACL injury (graft rupture or contralateral ACL rupture) within 2 years of ACLR reported lower ACL-RSI scores at 1 year after ACLR [23]. Athletes >20 years old who sustained a second ACL injury did not demonstrate differences in ACL-RSI scores. Sensitivity analysis revealed a score less than 76.7 identified patients at risk of a second ACL injury with a sensitivity of 90%. Evidence also suggests that a change in psychological readiness may be related to second ACL injury. Athletes that sustained a second ACL injury within 2 years from surgery did not demonstrate an improvement in psychological readiness from presurgery to 1 year after surgery, while the patients that did not sustain a second injury did show an improvement.

Taken together, the three previously mentioned studies indicate that greater fear, more negative emotions, and less confidence may put a patient at greater risk of sustaining a second ACL injury. In contradiction to the three previous

study’s findings, one published study indicates that high confidence (rather than low confidence) may be related to second ACL injury. Paterno and colleagues evaluated whether clinical measures could be used to predict second ACL injury and found that athletes who sustained a second ACL injury fell into one of the two high-risk profiles [24]. One of the high-risk profiles included high knee-related confidence as a significant variable (along with age <19 , triple hop distance >1.3 times body weight, female sex, and hop distance LSI $>98.5\%$). The authors theorized that an athlete with good performance on functional tests and high confidence may return to sport sooner, putting them more at risk for a reinjury.

80.3 Part 3: Psychological Factors and Movement

Individuals after ACLR demonstrate altered movement patterns during low-level tasks such as walking and sit to stand transfers, and high-level tasks such as drop vertical jumps and single-legged hop landings [6, 25–27]. Recent evidence suggests that psychological factors may be related to altered movement patterns [28, 29]. Therefore, psychological factors should be considered when addressing movement asymmetry prior to return to sport after ACLR.

Two recent studies have investigated the relationship between psychological factors and walking biomechanics. Zarzycki and colleagues examined the relationship between knee kinematic and kinetic variables during the stance phase of over ground walking, and psychological readiness to return to sport (i.e., ACL-RSI) in athletes at a mean of 5.4 months after ACLR [29]. The authors found a significant relationship between psychological readiness and two knee kinematic variables (peak knee flexion angle symmetry, and knee flexion angle at initial contract symmetry). Athletes after ACLR with lower ACL-RSI scores (i.e., a more negative psychological outlook) demonstrated less knee flexion at IC and less peak knee flexion angles in the surgical limb compared to the nonsurgical limb. A second study by Luc-Harkey and colleagues

examined the relationship between kinesiophobia (i.e., TSK-11) and gait characteristics during treadmill walking in athletes at a mean of 49 months after ACLR [30]. The authors of this study found no relationships between kinesiophobia and kinematics or kinetics. Differences in time from surgery to testing, the psychological instrument administered, and type of gait assessment (over ground vs. treadmill) may explain the differences in results between these two studies.

Only one published study has examined psychological factors and movement during a higher-level task. Trigsted and colleagues examined the relationship between kinesiophobia and lower extremity kinematics and muscle activation during a drop vertical jump task in female individuals at a mean of 26 months after ACLR [28]. Participants with greater kinesiophobia had a more stiffened movement pattern in the sagittal plane characterized by less knee and hip flexion in the surgical limb, and less trunk flexion. Additionally, the participants with greater kinesiophobia demonstrated greater hip adduction and greater gluteus maximus pre-activation.

Research has just started to explore the relationship between psychological factors and movement in individuals after ACLR. While clinical recommendations cannot be made based on the current evidence, clinicians may consider psychological factors, especially kinesiophobia, and psychological readiness as contributors to potential movement alterations after ACLR. Future research should elucidate the relationship between psychological factors and movement after ACLR, and determine if interventions specifically addressing psychological factors lead to a change in movement.

80.4 Part 4: Monitoring Athletes' Psychological Responses

The biopsychosocial model of care encompasses all aspects affecting a person's health in contrast to the traditional biomedical approach. While the biomedical model has led to major advances in medicine, it does not specifically take into account the psychological and social influences that affect function and disability after injury.

The integration of the biopsychosocial model into clinical practice has been termed "psychologically informed practice" (PIP) [31]. PIP is an approach by which clinicians evaluate and address psychological barriers to optimize outcomes after injury. This approach has been studied primarily in patients with low back pain and chronic musculoskeletal pain [32–34]. Fear of movement and reinjury are highly prevalent in these populations and are related to poor outcomes. Addressing these maladaptive beliefs and behaviors can be accomplished with various interventions including pain education, cognitive-behavioral treatment, graded exercise, and graded exposure. Evidence using these strategies in athletes after ACLR is scarce; however, these strategies can be applied to athletes who demonstrate similar issues with fear, confidence, etc. The next session will discuss tools used for screening psychological factors that could affect outcomes in athletes, followed by a section reviewing strategies to address psychological factors.

80.4.1 Psychological Measures for Athletes After ACLR

Table 80.1 lists the psychological construct, scale, minimal detectable change value (MDC), minimal clinically important difference value (MCID), and the established cut-off scores for each psychological measure below.

80.4.2 ACL-RSI

The ACL-RSI was specifically designed to evaluate psychological factors affecting RTS [35]. It includes 12 questions that measure three psychological constructs: emotions, confidence, and risk appraisal. It is scored on a scale from 0 to 100 with higher scores indicating a more positive psychological outlook (i.e., more positive emotions and greater confidence). The authors recommend using the ACL-RSI as an outcome tool infrequently during the course of rehabilitation (i.e., once early, once in the middle, and once toward the end of rehabilitation). Upon completion of the ACL-RSI early, the clinician should inspect each

Table 80.1 Description and psychometric properties of the ACL-RSI, ACL-RSI—Short form, TSK-11, and K-SES

	ACL-RSI	ACL-RSI—Short form	TSK-11	K-SES
Psychological construct	Psychological readiness to RTS consisting of questions related to emotions, confidence, and risk appraisal	Psychological readiness to RTS consisting of questions related to emotions, confidence, and risk appraisal (six questions only)	Kinesiophobia (i.e., fear of movement/reinjury)	Self-efficacy related to tasks involving knee function
Scale	0–100 with higher values indicating a more positive psychological outlook	0–100 with higher values indicating a more positive psychological outlook	11–44 with higher values indicating a greater fear of movement/reinjury	0–10 with higher values indicating greater certainty in performing activities related to knee function
MDC	– 19 intervention of one individual [48] – 3 changes in group mean [48]	No established MDC	3.0 [47]	No established MDC
MCID	No established MCID	No established MCID	3.7 [47]	No established MCID
Cut off scores	– 56 points at 4 months [51] – 62 points at 6 months [45] – 51 points at 6 months [49]	– 60 points at 6 months [51]	Patients with scores >18 at RTS were 13× more likely to experience second injury within 2 years/p ACLR [50]	≥7 equals acceptable level of self-efficacy [46]

question to determine if the athlete reports low scores with a specific construct. After determining the construct with the lowest score, clinicians should consider using one simple question more frequently during the course of rehabilitation. For example, an athlete that scores low on the questions referring to confidence can be asked: “on a scale for 0 to 10 (with 0 being not confident at all and 10 being fully confident) how confident are you about returning to your primary sport?” This can be used frequently as a quick measure of progress during rehabilitation. When progress is not being made with the simple question or with the ACL-RSI, clinicians may consider psychological interventions. A short version of the ACL-RSI, consisting of only six questions, has recently been developed [36].

80.4.3 Other Psychological Measures (TSK-11, K-SES)

The TSK-11 was originally developed to examine kinesiophobia in patients with chronic pain. The TSK-11 includes 11 questions related to

three different constructs: fear of pain, fear of reinjury, and somatic focus [37]. Scores on the TSK-11 range from 11 to 44 with higher scores indicating greater kinesiophobia.

Self-efficacy is a measure of a person’s confidence in performing a task and is task/situation-specific [14]. The K-SES thus assesses a person’s confidence regarding tasks requiring knee function. It includes 22 items broken into four sections: daily activities, sports and leisure activities, physical activities, and you knee function in the future. The person is asked to rate how certain they are about the activities listed within each section on a scale from 0 (not at all certain) to 10 (very certain).

80.5 Part 5: Interventions

80.5.1 Evidence for Psychosocial Interventions After ACLR

A recent systematic review evaluated the effectiveness of psychosocial interventions on outcomes following ACLR [38]. The authors of this

review concluded that there is insufficient evidence supporting the effectiveness of psychosocial interventions on outcomes after ACLR. However, the review only identified four studies using different interventions (i.e., guided imagery, relaxation, coping, and modeling). These interventions were associated with improvements in self-reported knee function, reductions in fear/anxiety, and resolution of knee impairments. No studies to date have examined the effect of cognitive-behavioral interventions on RTS. A current clinical trial is examining the effect of a smartphone application, delivering cognitive-behavioral therapy on RTS outcomes (clinicaltrials.gov, NCT03959215).

80.5.2 Graded Exercise and Graded Exposure

Graded exercise and graded exposure are effective in patients with low back pain [34]. Graded exercise involves a quota-based system in which exercise quotas are increased over time. Positive reinforcement is given to the patient when the quota is met at each session. Graded exposure is based specifically on a movement or task that a patient avoids due to fear. First, the activity/movement is identified, either through a specific question on a questionnaire or through interview. Then, the fearful activity/movement is incorporated in the rehabilitation program. Progressively the level of fear is increased over time until the fear declines.

Graded exercise, in the form of plyometric exercises, has been examined in patients after ACLR. Chmielewski et al. compared low-intensity and high-intensity plyometric exercise in athletes that met clinical milestones allowing them to participate in advanced rehabilitation [39]. Following the 8-week intervention, both groups (low-intensity group and high-intensity group) displayed significant improvements in knee activity self-efficacy, in addition to improved self-reported function, vertical jump height, knee strength, and pain ratings. Another study using a group training model focused on progressive plyometric exercise coupled with agility training and

strengthening exercises [40]. Psychological readiness and performance on all four single-legged hop tests improved significantly from the pre-training time point to post-training. Additionally, the proportion of patients categorized as psychologically ready (based on previous established cut-off scores of the ACL-RSI) significantly increased from pre- to post-training. The aforementioned studies indicate that graded exercise, incorporating plyometrics, and agilities may have potential in improving psychological factors. However, the effect of these interventions on RTS remains unknown.

80.5.3 Other Cognitive Behavioral Interventions

80.5.3.1 Mindfulness Meditation

Mindfulness meditation (MM) involves refocusing the mind on present awareness of one's inner feelings, body sensations, and surrounding environment. MM has been used as a treatment for multiple chronic pain conditions including fibromyalgia, chronic LBP, and musculoskeletal pain. Recent systemic reviews examining indicate that MM is associated with improvements in pain ratings; however, some of these effects were small, leading the authors to conclude that more high-quality studies are needed to determine the effectiveness of MM on various pain conditions [41–43]. In terms of psychological health, MM is effective in increasing subjective well-being, reducing psychological symptoms such as fear responses and emotional reactivity [44]. Therefore, the use of MM may be useful with athletes who display negative psychological factors or persistent pain. More research is needed to determine if MM can be used to improve RTS outcomes.

80.6 Part 6: Summary and Recommendations

There is strong evidence linking psychological factors and activity-related and functional outcomes. Clinicians should consider monitoring

psychological factors that can affect outcomes using validated tools, such as the ACL-RSI and the TSK-11. The established cut-off scores can help clinicians identify patients at risk for suboptimal outcomes due to psychological factors. Once identified, negative psychological factors may be addressed through various techniques such as graded exercise/exposure, MM, goal setting, imagery, and modeling. However, there is no evidence linking these interventions to improved RTS outcomes.

There is a subset of patients that require consultation with a mental health-care provider. Three proposed contraindications for the use of psychological interventions by a physical therapist in patients with musculoskeletal pain are as follows: (1) identification of a current mental disorder, (2) the patient is unable to participate meaningfully in treatment, and (3) the patient is unwilling to participate in treatment [31]. Referral to a mental health professional is indicated when a patient demonstrates any of these three contraindications.

Future research should continue to examine the relationship between psychological factors and outcomes in athletes after ACLR. In addition, examination of the relationships between psychological factors and reinjury, and psychological factors and movement is needed to build on the small body of current evidence. Finally, studies implementing interventions directed specifically at negative psychological factors are needed to determine if negative psychological factors are the driving force behind less than optimal function or the product of less than optimal function.

References

1. Tegner H, Frederiksen P, Esbensen BA, Juhl C. Neurophysiological pain education for patients with chronic low Back pain. *Clin J Pain*. 2018;34(8):778–86. <https://doi.org/10.1097/AJP.0000000000000594>.
2. Paterno MV, Rauh MJ, Schmitt LC, Ford KR, Hewett TE. Incidence of second ACL injuries 2 years after primary ACL reconstruction and return to sport. *Am J Sports Med*. 2014;42(7):1567–73. <https://doi.org/10.1177/0363546514530088>.
3. Webster KE, Feller JA, Leigh WB, Richmond AK. Younger patients are at increased risk for graft rupture and contralateral injury after anterior cruciate ligament reconstruction. *Am J Sports Med*. 2014;42(3):641–7. <https://doi.org/10.1177/0363546513517540>.
4. Wiggins AJ, Grandhi RK, Schneider DK, Stanfield D, Webster KE, Myer GD. Risk of secondary injury in younger athletes after anterior cruciate ligament reconstruction: a systematic review and meta-analysis. *Am J Sports Med*. 2016;44:1861–76. <https://doi.org/10.1177/0363546515621554>.
5. Ithurburn MP, Altenburger AR, Thomas S, Hewett TE, Paterno MV, Schmitt LC. Young athletes after ACL reconstruction with quadriceps strength asymmetry at the time of return-to-sport demonstrate decreased knee function 1 year later. *Knee Surg Sports Traumatol Arthrosc*. 2018;26:426–33. <https://doi.org/10.1007/s00167-017-4678-4>.
6. Kaur M, Ribeiro DC, Theis J-C, Webster KE, Sole G. Movement patterns of the knee during gait following ACL reconstruction: a systematic review and meta-analysis. *Sports Med*. 2016;46:1869–95. <https://doi.org/10.1007/s40279-016-0510-4>.
7. Lisee C, Lepley AS, Birchmeier T, O'Hagan K, Kuenze C. Quadriceps strength and volitional activation after anterior cruciate ligament reconstruction: a systematic review and meta-analysis. *Sports Health*. 2019;11(2):163–79. <https://doi.org/10.1177/1941738118822739>.
8. Clement D, Arvinen-Barrow M, Fetty T. Psychosocial responses during different phases of sport-injury rehabilitation: a qualitative study. *J Athl Train*. 2015;50(1):95–104. <https://doi.org/10.4085/1062-6050-49.3.52>.
9. Hsu C-J, Meierbachtol A, George SZ, Chmielewski TL. Fear of reinjury in athletes: implications for rehabilitation. *Sports Health*. 2016;9:162–7. <https://doi.org/10.1177/1941738116666813>.
10. Morrey MA, Stuart MJ, Smith AM, Wiese-Bjornstal DM. A longitudinal examination of athletes' emotional and cognitive responses to anterior cruciate ligament injury. *Clin J Sport Med*. 1999;9(2):63–9.
11. Chmielewski TL, Zeppieri G Jr, Lentz TA, et al. Longitudinal changes in psychosocial factors and their association with knee pain and function after anterior cruciate ligament reconstruction. *Phys Ther*. 2011;91(9):1355–66. <https://doi.org/10.2522/ptj.20100277>.
12. Ardern CL, Taylor NF, Feller JA, Webster KE. A systematic review of the psychological factors associated with returning to sport following injury. *Br J Sports Med*. 2013;47(17):1120–6. <https://doi.org/10.1136/bjsports-2012-091203>.
13. Ardern CL. Anterior cruciate ligament reconstruction—not exactly a one-way ticket back to the preinjury level: a review of contextual factors affecting return to sport after surgery. *Sports Health*. 2015;7(3):224–30. <https://doi.org/10.1177/1941738115578131>.
14. Ardern CL, Osterberg A, Tagesson S, Gauffin H, Webster KE, Kvist J. The impact of psychological readiness to return to sport and recreational activi-

- ties after anterior cruciate ligament reconstruction. *Br J Sports Med.* 2014;48(22):1613–9. <https://doi.org/10.1136/bjsports-2014-093842>.
15. Lentz TA, Aeppieri G Jr, Tillman SM, et al. Return to preinjury sports participation following anterior cruciate ligament reconstruction: contributions of demographic, knee impairment, and self-report measures. *J Orthop Sports Phys Ther.* 2012;42(11):893–901. <https://doi.org/10.2519/jospt.2012.4077>.
 16. Ardern CL, Taylor NF, Feller JA, Whitehead TS, Webster KE. Psychological responses matter in returning to preinjury level of sport after anterior cruciate ligament reconstruction surgery. *Am J Sports Med.* 2013;41(7):1549–58. <https://doi.org/10.1177/0363546513489284>.
 17. Ardern CL, Taylor NF, Feller JA, Whitehead TS, Webster KE. Sports participation 2 years after anterior cruciate ligament reconstruction in athletes who had not returned to sport at 1 year: a prospective follow-up of physical function and psychological factors in 122 athletes. *Am J Sports Med.* 2015;43:848–56. <https://doi.org/10.1177/0363546514563282>.
 18. Beischer S, Hamrin Senorski E, Thomeé C, Samuelsson K, Thomeé R. How is psychological outcome related to knee function and return to sport among adolescent athletes after anterior cruciate ligament reconstruction? *Am J Sports Med.* 2019;47:1567. <https://doi.org/10.1177/0363546514559707>.
 19. T a L, Zeppieri G, George SZ, et al. Comparison of physical impairment, functional, and psychosocial measures based on fear of reinjury/lack of confidence and return-to-sport status after ACL reconstruction. *Am J Sports Med.* 2015;43(2):345–53. <https://doi.org/10.1177/0363546514559707>.
 20. Chmielewski TL, Jones D, Day T, Tillman SM, Lentz TA, George SZ. The association of pain and fear of movement/reinjury with function during anterior cruciate ligament reconstruction rehabilitation. *J Orthop Sports Phys Ther.* 2008;38(12):746–53. <https://doi.org/10.2519/jospt.2008.2887>.
 21. Chmielewski TL, George SZ. Fear avoidance and self-efficacy at 4 weeks after ACL reconstruction are associated with early impairment resolution and readiness for advanced rehabilitation. *Knee Surg Sport Traumatol Arthrosc.* 2019;27(2):397–404. <https://doi.org/10.1007/s00167-018-5048-6>.
 22. Paterno MV, Flynn K, Thomas S, Schmitt LC. Self-reported fear predicts functional after ACL reconstruction and return to sport : a pilot study. *Sports Health.* 2018;10(3):228–33. <https://doi.org/10.1177/1941738117745806>.
 23. McPherson AL, Feller JA, Hewett TE, Webster KE. Psychological readiness to return to sport is associated with second anterior cruciate ligament injuries. *Am J Sports Med.* 2019;47:857–62. <https://doi.org/10.1177/0363546518825258>.
 24. Paterno MV, Huang B, Thomas S, Hewett TE, Schmitt LC. Clinical factors that predict a second ACL injury after ACL reconstruction and return to sport: preliminary development of a clinical decision algorithm. *Orthop J Sport Med.* 2017;5(12):1–7. <https://doi.org/10.1177/2325967117745279>.
 25. Capin JJ, Zarzycki R, Arundale A, Cummer K, Snyder-Mackler L. Report of the primary outcomes for gait mechanics in men of the ACL-SPORTS trial: secondary prevention with and without perturbation training does not restore gait symmetry in men 1 or 2 years after ACL reconstruction. *Clin Orthop Relat Res.* 2017;475:2513. <https://doi.org/10.1007/s11999-017-5279-8>.
 26. Decker MJ, Torry MR, Noonan TJ, Sterett WI, Steadman JR. Gait retraining after anterior cruciate ligament reconstruction. *Arch Phys Med Rehabil.* 2004;85(5):848–56. <https://doi.org/10.1016/j.apmr.2003.07.014>.
 27. Knoll Z, Kiss RM, Kocsis L. Gait adaptation in ACL deficient patients before and after anterior cruciate ligament reconstruction surgery. *J Electromyogr Kinesiol.* 2004;14(3):287–94. <https://doi.org/10.1016/j.jelekin.2003.12.005>.
 28. Trigsted SM, Cook DB, Pickett KA, Cadmus-Bertram L, Dunn WR, Bell DR. Greater fear of reinjury is related to stiffened jump-landing biomechanics and muscle activation in women after ACL reconstruction. *Knee Surg Sport Traumatol Arthrosc.* 2018;26(12):1–8. <https://doi.org/10.1007/s00167-018-4950-2>.
 29. Zarzycki R, Failla M, Capin J, Snyder-Mackler L. Psychological readiness to return to sport is associated with knee kinematic asymmetry during gait following ACL reconstruction. *J Orthop Sport Phys Ther.* 2018;48(12):1–21. <https://doi.org/10.2519/jospt.2018.8084>.
 30. Luc-Harkey BA, Franz JR, Losina E, Pietrosimone B. Association between kinesiphobia and walking gait characteristics in physically active individuals with anterior cruciate ligament reconstruction. *Gait Posture.* 2018;64:220–5. <https://doi.org/10.1016/j.gaitpost.2018.06.029>.
 31. Keefe FJ, Main CJ, George SZ. Advancing psychologically informed practice for patients with persistent musculoskeletal pain: promise, pitfalls, and solutions. *Phys Ther.* 2018;98(5):398–407. <https://doi.org/10.1093/ptj/pzy024>.
 32. Main CJ, George SZ. Psychologically informed practice for management of low back pain: future directions in practice and research. *Phys Ther.* 2011;91(5):820–4. <https://doi.org/10.2522/ptj.20110060>.
 33. Main CJ, George SZ. Psychosocial influences on low back pain: why should you care? *Phys Ther.* 2011;91(5):609–13. <https://doi.org/10.2522/ptj.2011.91.5.609>.
 34. Nicholas MK, George SZ. Interventions for low back pain: an update for physical therapists. 2011;91(5):765–76.
 35. Webster KE, Feller JA, Lambros C. Development and preliminary validation of a scale to measure the psychological impact of returning to sport following anterior cruciate ligament reconstruction surgery. *Phys Ther Sport.* 2008;9(1):9–15. <https://doi.org/10.1016/j.ptsp.2007.09.003>.

36. Webster KE, Feller JA. Development and validation of a short version of the Anterior Cruciate Ligament Return to Sport after Injury (ACL-RSI) scale. *Orthop J Sport Med.* 2018;6(4):1–7. <https://doi.org/10.1177/2325967118763763>.
37. Steven Z, George PP, Trevor A, Lentz M, Giorgio Zeppieri M Jr, Derek Lee S, Terese L, Chmielewski PTP. Analysis of shortened versions of the Tampa Scale for Kinesiophobia and Pain Catastrophizing Scale for patients following anterior cruciate ligament reconstruction. *Clin J Pain.* 2012;28(1):73–80. <https://doi.org/10.1097/AJP.0b013e31822363f4>.
38. Coronado RA, Bird ML, Van HEE, Huston LJ, Spindler KP, Archer KR. Do psychosocial interventions improve rehabilitation outcomes after anterior cruciate ligament reconstruction ? A systematic review. *Clin Rehabil.* 2018;32:287–98. <https://doi.org/10.1177/0269215517728562>.
39. Chmielewski TL, George SZ, Tillman SM, et al. Low-versus high-intensity plyometric exercise during rehabilitation after anterior cruciate ligament reconstruction. *Am J Sports Med.* 2016;44:609. <https://doi.org/10.1177/0363546515620583>.
40. Meierbachtol A, Yungtum W, Paur E, Bottoms J, Chmielewski TL. Psychological and functional readiness for sport following advanced group training in patients with anterior cruciate ligament reconstruction. *J Orthop Sports Phys Ther.* 2018;48(11):1–33. <https://doi.org/10.2519/jospt.2018.8041>.
41. Cramer H, Haller H, Lauche R, Dobos G. Mindfulness-based stress reduction for low back pain. A systematic review. *BMC Complement Altern Med.* 2012;12(1):1. <https://doi.org/10.1186/1472-6882-12-162>.
42. Hilton L, Hempel S, Ewing BA, et al. Mindfulness meditation for chronic pain : systematic review and meta-analysis. *Ann Behav Med.* 2017;51:199–213. <https://doi.org/10.1007/s12160-016-9844-2>.
43. Mcclintock AS, Mccarrick SM, Garland EL, Zeidan F, Zgierska AE. Brief mindfulness-based interventions for acute and chronic pain: a systematic review. *J Altern Complement Med.* 2019;25(3):265–78. <https://doi.org/10.1089/acm.2018.0351>.
44. Keng S-L, Smoski MJ, Robins CJ. Effects of mindfulness on psychological health. *Clin Psychol Rev.* 2011;31(6):1041–56. <https://doi.org/10.1016/j.cpr.2011.04.006>.
45. Webster KE, Feller JA. Development and Validation of a Short Version of the Anterior Cruciate Ligament Return to Sport After Injury (ACL-RSI) Scale. *Orthop J Sports Med.* 2018;6(4):2325967118763763.
46. Beischer S, Senorski EH, Thomeé C, Samuelsson K, Thomeé R. How is psychological outcome related to knee function and return to sport among adolescent athletes after anterior cruciate ligament reconstruction? *Am J Sports Med.* 2019;47(7):1567–75.
47. Chmielewski TL, Jones D, Day T, Tillman SM, Lentz TA, George SZ. The association of pain and fear of movement/reinjury with function during anterior cruciate ligament reconstruction rehabilitation. *J Orthop Sports Phys Ther.* 2008;38(12):746–53.
48. Kvist J, Österberg A, Gauffin H, Tagesson S, Webster K, Ardern C. Translation and measurement properties of the Swedish version of ACL-Return to Sports after Injury questionnaire. *Scand J Med Sci Sports.* 2012.
49. Müller U, Krüger-Franke M, Schmidt M, Rosemeyer B. Predictive parameters for return to pre-injury level of sport 6 months following anterior cruciate ligament reconstruction surgery. *Knee Surg Sports Traumatol Arthrosc.* 2015;23(12):3623–31.
50. Paterno MV, Flynn K, Thomas S, Schmitt LC. Self-Reported fear predicts functional performance and second ACL injury after acl reconstruction and return to sport: A pilot study. *sports health: a multidisciplinary approach.* 2018;10(3):228–233.
51. Ardern CL, Taylor NF, Feller JA, Whitehead TS, Webster KE. Psychological responses matter in returning to preinjury level of sport after anterior cruciate ligament reconstruction surgery. *Am J Sports Med.* 2013;41(7):1549–58.



The Retired Professional Basketball Player—Psychological Aspects

81

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Retirement, a turning point in everyone's life, is a period that is extremely hard to manage. Retirement from elite sports can be highly distressing for athletes, and many report experiencing depression and anxiety in adjusting to this transition [1]. When retirement is seen as part of the normal life cycle and not as a crucial turning point in life, anxiety, stress, and obscurity can be avoided. The most important step in doing so is the mental preparation process. Mental preparation in order to be ready for the retirement period, just like how athletes prepare mentally for the season aside from their physical training, will help in making the transition to retirement to be as smooth and flawless as possible.

After basketball players retire, they might experience psychological problems such as depression and anxiety, physical problems involving joints and muscles, or sleeping problems. Sudden changes in lifestyle, public position, and daily habits can create serious conditions such as identity problems, intense sorrow and even depression, and a decrease in life satisfaction. Aside from these psychological problems, retired athletes might experience health problems due to their inactivity.

After being involved in years of active sports life, pro-athletes stagger as the regular practices, workouts, and games no longer exist in their rou-

time. The best way to address this phase is to prepare both mentally and physically for the retirement period by planning the future career. Applying the self-discipline and consistency, athletes get used to during their professional careers to the retirement period will make it easier for them to adapt to this new phase. In addition, the continuation of these habits gained through their athletic career will increase and maintain their personal motivation and pave the way for success in various other fields of life.

One of the most important aspects to consider when pro-athletes plan their retirements is to be well-organized and possess realistic goals as they attempt to take solid steps ahead. It is helpful to accurately predefine their designated future career, i.e., in sports management, coaching, or a completely different field of work, and make sure this path will provide them with an appropriate challenge and is in accordance with their values and will leave them satisfied (Fig. 81.1). Doing so is key to overall satisfaction and happiness in their future lives. In both their athletic careers and their retirement journeys, adapting themselves to the order and life standards of their environment and focusing on the positive contributions of these adaptations will provide athletes with tools to easily overcome experienced difficulties in the transition process to retirement.

Discipline possessed in the professional life of an athlete is a value highly required after retirement. Maintaining dietary routines, sleep-

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Fig. 81.1 It is common for professional basketball players to continue their carrier with sports management, coaching, and administrative role



ing patterns, and relations with their family organized is a necessity in retirement as it was during their athletic careers. No longer maintaining this discipline and moving onto a less or unorganized life pattern after retirement may lead to negative psychological effects.

Routine practices and consistency in lifestyle are the biggest sources of motivation in the life of a pro-athlete. Steps to create precise short- and long-term goals that will increase motivation and help retired athletes maintain the same consistency after retiring should be taken. Overall success in life is the result of being satisfied with whatever you do and satisfaction, and content are often driven by the sense of having a purpose and goal in life. Having precise and clear goals and working toward them are just as important for maintaining a postretirement career as it is important for being a top tier athlete.

Even though basketball is a team sport, decisions concerning an athlete's career, the way of managing these decisions, and the goals and the actions necessary to reach them are determined by the individual choices of each athlete. Being determined to work consistently to individually prepare themselves for the highest level is another key point in an athletic career (Fig. 81.2). Determination and self-organization are "must-haves" in retirement just like they are in the active career of an athlete.



Fig. 81.2 Professional basketball players need to prepare for retirement just as they prepared themselves for the highest level of play

Professional basketball players are familiar with the feeling of winning as well as loosing.

Those who strive for better even following a win and maintain a positive mindset following a loss become top athletes. This applies to retirement as well. One builds a successful future when one has the ability obtain positive results from negative situations.

Multiple factors may shape the career and enhance the performance of an athlete. These can be a variation of many factors such as success, appreciation, high income, and passion. One of the most valuable among these factors is passion. Some of the best basketball players acquired their success by loving what they do. A basketball player can retain close ties to basketball after retirement. Professional basketball players can retain such ties with the game by becoming referees, trainers, coaches, or members of management.

An athlete needs to be able to cope with stress and to control emotions on the court. These self-control mechanisms may aid the athlete after he/she retires. Being able to control one's personal emotional state could help prevent depression and anxiety-related problems in the future. Getting professional mental support, maintaining a life of discipline, connecting with family and friends, and replacing your daily routines with new activities will provide the athlete with a solid foundation for a stable and satisfactory life after retirement.

One of many ways of obtaining mentally toughness is mindfulness. Mindfulness is the psychological process of purposely bringing one's attention to experiences occurring in the present moment without judgment. In the simplest sense, mindfulness is a state of mind that allows a person to stay focused on the present moment, to observe and accept self-emotions, thoughts and surroundings gently, without judgment. Mindfulness has been introduced as a construct that may increase flow experience as it enables individuals to practice maintaining attention in the present moment [2]. To achieve this practice, meditation and other mindfulness techniques are necessary. Focusing on the past and the future may have a negative effect on an athlete's performance. Being mindful enables the athlete to stay in their "zone", focus on what they

are doing, avoid distractions, and enhance their sports performance. Many successful basketball players have different ways of getting into their "zone." Mindfulness techniques are known to be one of the most efficient ways to achieve this. These involve breathing methods, body scan, guided imagery, the observer meditation, and other practices to enhance self-awareness and relax the body as well as the mind. This state of mind also takes effect in players' life after retirement, by eliminating stress and anxiety. Being able to control thoughts and emotions through mindful awareness techniques help players to cope and adapt to changes occurring in their life after retirement.

An athlete who can control the stress and anxiety caused by competition is equipped to be able to control the stress and anxiety that might arise during his/her postretirement period. Since high amounts of adrenaline and cortisone affect the decision-making process of the brain, immediate reactions cannot be completely controlled. These hormones, released in high concentrations during an active sports career, have been shown to be one of the biggest reasons for depression during retirement. Positive thoughts cause the release of dopamine and can lead to a less stressful and happier life.

Especially for top-tier athletes, after getting used to a particular lifestyle and a public status derived from their athletic success, retiring and having no goals in life can create a strong feeling of emptiness. Similarly, shortsighted sudden decisions and failures following these decisions may affect their psychology in a negative manner. Making long-term investments according to a roadmap based on designated goals and allowing you to have sufficient time to rest and digest the change after retiring could be critical in maintaining high standards of life.

Athletes who are not prepared for retirement may experience a significant downfall after. Supporting themselves with mental exercises and getting professional psychological support when necessary will help athletes avoid this situation. Recent research has shown that if the reasons for retirement were the result of struggles with coaches, or problems and issues with a sport

federation; athletes experienced difficulties during retirement, missing the social aspect of the sport, lack of self-esteem, and keeping feelings to themselves [3]. Reasons for retirement could be placed in two main categories: the expected retirement, which comes naturally with aging; and unexpected retirement that is caused by an injury or a burnout syndrome.

Early retirement ages of basketball players are often displayed as a disadvantage. An athlete might be forced to retire at a period when his/her mental productivity is peaking. The best time for an athlete to retire is when the body starts signaling for it and thoughts of retiring are being considered both from a physical stand point and psychologically. Athletes can extend the length of their career by taking care of their bodies and their minds. Joining special coaching courses tailored for retired or veteran basketball players is another option for athletes to prepare for their future lives as civilians. Careful planning of the course of their careers after retirement and establishing an investment plan for their savings is crucial.

Serious injuries or a burnout syndrome caused by inability to manage long-term stress are the main reasons for an unexpected retirement. Athletes going through related experiences suffer from fear of injury along with anxiety, worry, and stress. At this point, it is extremely important to stay in the moment instead of worrying about the future through the use of mental exercises that are proven to have positive outcomes. Modern improvements in medicine help determine the physical needs of the athletes, which significantly reduce the risk of injury. The recovery process following injuries is relatively shorter too. Being physically conditioned and consistent strength and agility exercises protect from injuries while preparing the athletes in the best way possible for the games. Athletes getting used to this pace of training should keep exercising at a moderate pace. Having decent body strength contributes positively to the decision-making process of the brain.

Financial problems that are another possible challenge which could be experienced during retirement and may also have adverse effects on an athlete's psychology and mental strength.

Seeking advice from financial and investment professionals as well as the opinions and support of trusted people around the athlete are extremely important in making the right decisions and investments and also give the athlete the peace of mind in planning his/her postretirement career.

Living in the moment instead of constantly thinking about the past or the future can prove highly beneficial for athletes in every period of their lives. An athlete who cannot stop thinking about the future is at a high risk of experiencing anxiety during his/her retirement. Correct and quality planning for the future will result in positive outcomes along the way. That is why being ready both physically and mentally is as important to athletes in retirement as it is in their active professional careers. Continuing to play basketball for fun after retiring can help athletes stay fit and psychologically steady. Another potentially beneficial option for athletes is to take up a new hobby, keep working out, socialize, maintain relations with friends and family, and stay active in every aspect of life after retiring.

81.1 Summary

Retirement could be a stressful event for competitive basketball players at all levels. Preparing for retirement could be a challenging and complex task. Many of the mental qualities which come to use during their playing career could aid players cope and succeed in their postretirement life. Choosing a path of interest and setting clear goals are important factors to achieve and maintain a satisfying post-retirement career.

References

1. Aherne C, Moran A, Lonsdale C. The effect of mindfulness training on athletes' flow: an initial investigation. *Sport Psychol.* 2011;25(2):177–89.
2. Cosh S, Crabb S, Lecouteur A. Elite athletes and retirement: identity, choice and agency. *Aust J Psychol.* 2013;65(2):88–97.
3. Hatamleh MR. The life transitions of high performance athletes retirement from sport. *Eur Sci J.* 2013;9(11):336–53.