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Specific Sports-Related Injuries

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International Society of Arthroscopy, Knee Surgery and Orthopaedic Sports Medicine

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Preface

Most of the times, a defeat may work as the turning point in an athlete's career, a challenging moment but one to learn and analyze the weaknesses and strengths, define new strategies, and become physically and mentally stronger to overcome new challenges.

In sports, conquests are designed and built by teamwork, and come true by the athlete's dedication, persistence, and demanding work on sports abilities, such as in judo, sumo, wrestling, tennis, baseball, American football, soccer, rugby, swimming, skiing, bobsleigh, gymnastics, volleyball, basketball, fencing, cycling, and marathon. Each modality of sports has a unique DNA that is manifested by a particular field of play, rules, and physical and mental demands that identify and make each sport so special and fascinating to watch and practice. However, each sport also has a "dark side"—specific sports-related injuries.

The Specific Sports-Related Injuries book approaches different modalities of sports, and each one of the 36 chapters was structured and designed to start with a brief presentation of the modality, going through the analysis of its dynamics, athlete's physical demands in the field, common sports-related injuries, biomechanics of injuries, treatment, and injury prevention. Moreover, the book analyzes the accuracies related to the individual and team sports in winter and summer sports.

Like in the Sports Medicine Physician book, this book involved fantastic teamwork. Therefore, I would like to thank Phillipe Neyret, Mark R. Hutchinson, Joao Espregueira-Mendes, Moises Cohen, Mark Safran, David Parker, Hélder Pereira, Nicola Maffulli, Daniel Miranda Ferreira, ISAKOS Board members, SBOT/São Paulo Board members, Magda S. Kimoto, and all health professionals that have accepted to share their academic knowledge and expertise in sports medicine.

I would also like to thank my wife Ana Karina Piedade for her kindness and patience, supporting me every single day of my life.

"*One dream, one soul, one prize, one goal. One golden glance of what should be*" **Athletes are driven by Challenges**. In sports, passions, dreams, and nightmares build an athlete's career. Some of them may become "*heroes, but just for one day*," while a few will be forever true sports legends.

Campinas, São Paulo, Brazil — Sérgio Rocha Piedade

Contents

Part II Individual Sports

Part I

Team Sports

1 American Football

Jonathan D. Hughes, Christopher M. Gibbs, Neel K. Patel, Dale G. Thornton, Aaron V. Mares, and Volker Musahl

1.1 Introduction

American football is popular among all age groups in the United States and is played at various levels, including Pop Warner league, junior high school, high school, collegiate, and professional. Athletes that play this high-speed, high-impact collision sport are susceptible to various injuries ranging from muscle strains to career-ending fractures and ligamentous injuries. Common injuries sustained in American football include concussions, anterior cruciate ligament and medial collateral ligament tears, glenohumeral shoulder instability, acromioclavicular joint injuries and clavicle fractures, and ankle sprains and syndesmotic injuries. This chapter will review the epidemiology of injuries incurred during American football, as well as diagnosis, on-field and in-season management, and prevention of the most common injuries seen in American football.

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1.2 Epidemiology

American football is one of the most popular sports in the United States with over 60,000 collegiate, over 1 million high school athletes, and over 225,000 sandlot and professional athletes participating each season [1, 2]. American football is a high-speed, high-impact collision sport that has potential for serious injury to participants. Athletes partake in drills throughout the week that include player-to-player contact and player-to-surface contact, which places the athlete at risk for career-ending fractures, joint dislocations, and ligamentous ruptures. These drills are frequently performed at full-speed, and therefore the athlete is susceptible to noncontact ligament tears and muscle strains, as well as overuse/repetitive injuries. The athletes are required to give full effort throughout the week, then expected to play full-contact games at the end of the week. As the season progresses, risk of injury increases due to continued collisions, fatigue, and repetitive nature of the sport. Offensive and defensive lineman are the most susceptible to injury, as they are involved in contact activities every play [3]. Additionally, the type of playing surface, including artificial turf and natural grass, can have an effect on injury risk. Prior studies have demonstrated artificial turf has an increased risk of injury compared to natural grass, as does poorly maintained playing surfaces [4–6].

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Playing American football puts unique demands on the body, and as a result the injury rate is nearly twice that of other popular sports such as basketball. Among 15 collegiate sports, American football had the highest rate of injury during competition at 35.9 injuries per 1000 athlete-exposures (AE) [7]. All other sports except wrestling (26.4 injuries per 1000 AE) had less than 20 injuries per 1000 AEs [7]. An epidemiologic study found that approximately 500,000 high school American football-related injuries occurred annually. Additionally, the injury rate was much higher during actual competition compared to practice for middle school, high school, and collegiate athletes [2, 8]. However, the overall number of injuries was still higher during regular practice, accounting for nearly 56% of all injuries, than during competition (approximately 40%) in collegiate athletes due to the significantly higher athlete-exposure during practice [9]. One study evaluating the effect of age on American football-related injuries showed that fractures were the most common injury regardless of age, but the rate of fractures was lower in athletes 15–18 years of age compared to younger athletes [10]. Additionally, the rate of closed head injuries, including concussions, was highest in athletes younger than 8 years of age [10]. Epidemiological studies such as the ones discussed in this section are important as they highlight important patterns and potentially modifiable risk factors that can help injury prevention efforts that will be discussed later in this chapter.

1.3 Percentage of Injuries and Their Anatomic Locations

American football-related injuries can involve any part of the musculoskeletal system including the upper and lower extremities as well as the head and cervical spine. One study investigating the distribution of injuries by anatomic location found that 43.2% of injuries were upper extremity injuries and 32.6% of injuries were lower extremity injuries [10]. Other studies have cited that approximately 20% of American football-related injuries involve the knee and about 17% involve the foot and ankle.

The most common American football-related knee injuries are medial collateral ligament injury (MCL), meniscal injury, and anterior cruciate ligament (ACL) injury with 25% of knee injuries requiring surgical intervention [11]. The incidence of ACL injuries in the National Football League (NFL) is 0.7 per 1000 players resulting in approximately 53 ACL injuries annually in the NFL [12, 13]. Running backs and linebackers are at highest risk for ACL injuries with 9.7% and 8.9% of players, respectively, having a history of ACL injury at the NFL combine [14].

With regard to foot and ankle injuries, they occur at a rate of 15 per 10,000 AEs [15]. Lateral ankle ligament sprains, syndesmotic (high ankle) sprains, medial ankle ligament sprains, midfoot injuries, and first metatarsophalangeal joint injuries account for nearly 80% of all foot and ankle injuries related to playing American football [15]. In terms of median time loss from play, the most severe foot and ankle injury was an ankle dislocation, which resulted in a median time loss of 100 days, followed by metatarsal fractures (38 days) and isolated malleolus fracture (31 days) [15]. Additionally, foot injuries such as Lisfranc injuries, which are present in 1.8% of athletes at the NFL combine, can significantly affect draft position, performance, and longevity of an American football career [16].

Shoulder injuries are among the most common American football-related upper extremity injuries. One epidemiological study showed that nearly 50% of athletes at the NFL combine had a history of a shoulder injury with 36% of these injured athletes requiring surgical intervention [17]. The most common shoulder injuries were acromioclavicular (AC) injuries (41%), anterior instability (20%), and rotator cuff injury (12%) [17, 18]. The highest incidence of AC injury occurred in quarterbacks at a rate of 20.9 injuries per 100 players [19]. The most common surgeries performed for shoulder injuries were anterior instability reconstruction (48%), Mumford/Weaver-Dunn surgery (15%), posterior instability reconstruction (10%), and rotator cuff repair

(10%) [17]. Overall, these injuries can result in a significant loss of playing time even in cases of AC injuries, which typically do not require surgical intervention [19].

Cervical spine injuries are less common American football-related injuries, but are still important to highlight. The cervical spine injury rate is 2.91 per 10,000 AEs with stingers being the most common injury at a rate of 1.87 per 10,000 AEs [20]. Cervical spine injuries were most common in linebackers followed by defensive linemen. Overall, one study found that most athletes were able to return to play (RTP) within 24 h of their initial cervical spine injury (64.4%), while only 2.8% remained out of play for >21 days [20]. Once again, information regarding the positions affected and the relative time loss by particular injuries is important in order to implement appropriate and unique prevention measures, which will be discussed later in this chapter.

1.4 General Evaluation of Extremity Injuries

The team physician needs to perform a detailed history and physical on all the athletes before the season starts, and remember all aspects of the history and physical as the season progresses. Therefore, when it comes to on-field injuries, the team physician can obtain a detailed and focused history of injury mechanism and perform a specific evaluation for the suspected injury, followed by formulating a rapid plan of attack (Fig. 1.1).

Initial evaluation of a suspected extremity injury consists of a thorough history to elicit the specific nature and location of their complaint, mechanism or injury, severity of pain, whether they are able to bear weight or move an injured extremity, presence of functional deficits such as mechanical block to motion or neurologic deficit, and history of previous injury or surgery to the injured and contralateral extremities [21].

Fig. 1.1 A focused, detailed history and physical exam conducted on the field to formulate a quick treatment plan

Following this, an examination should be performed of the entire injured extremity and compared to the contralateral side. Examination should begin with inspection of the skin and soft tissue for gross deformity, ecchymosis, effusion, or open wounds. Range of motion of the surrounding joints should be attempted actively and/or passively as tolerated and compared with the contralateral extremity. Crepitus, pain, swelling, or deformity may be detected using palpation. Adjacent ligaments should be stressed and compared to the uninjured extremity. Strength should be evaluated if possible, however full effort may be limited due to pain. A careful neurovascular examination should be performed. Finally, special tests to evaluate for particular injuries based on the initial history and examination should be performed.

1.5 Five Common American Football-Related Injuries

The mechanism of injury, on-field management, and in-season treatment of five common injuries will be discussed. Based upon the senior author's experience and prior epidemiological studies, this chapter will discuss concussion, knee ligament injuries including injuries to the ACL and MCL, anterior and posterior labral tears associated with glenohumeral instability, acromioclavicular (AC) injuries and clavicle fractures, and ankle sprains including syndesmotic injury.

1.5.1 Concussion

1.5.1.1 Mechanism of Injury

Concussion is defined as a transient, functional traumatic brain injury induced by biomechanical forces [22]. Among youth, high school, and college American football players, concussions are most commonly caused by player contact, accounting for 83% of concussions [23]. At the collegiate level, a larger proportion of concussions are caused by player contact (88% vs. 80% and 81% at the youth and high school levels, respectively), while youth athletes are nearly twice as likely to sustain a concussion due to surface contact than high school and college athletes. Youth athletes are more likely to sustain a concussion while being tackled (42% vs. 23% for both high school and college athletes), while college athletes are more likely to sustain a concussion while being blocked (16% vs. 10% and 6% for high school and youth athletes, respectively). At the professional level, the mechanism of sustaining a concussion was studied using video analysis of National Football League (NFL) games [24]. Concussions were found to occur most often during a passing play, accounting for 50% of cases. In 41% of cases, a concussion was sustained during tackling, most commonly from a helmet-to-body impact. The authors demonstrated the mechanism of injury varied by position: cornerbacks were found to be injured most commonly by a helmet-to-body impact while wide receivers and quarterbacks were found to be more commonly injured by a helmet-to-ground or helmet-to-helmet impact.

1.5.1.2 On-Field Management

An athlete suspected of having sustained a concussion after a forceful direct or indirect impact to the head with any symptoms, signs, or concern by any trained observer should be immediately removed from play and evaluated [25]. The athlete should not RTP until an appropriate evaluation has been completed by a qualified healthcare professional. The 2017 Berlin Concussion in Sport Group Consensus Statement described several mandatory and discretionary symptoms which require further evaluation [25]. Mandatory symptoms include loss of consciousness, lying motionless for more than 5 seconds, confusion, disorientation, amnesia, vacant look, motor incoordination, tonic posturing, impact seizure, and ataxia; the presence of one or more of these signs mandates removal from play according to NFL rules [25, 26]. Discretionary signs include clutching the head, being slow to get up, suspected facial fracture, possible ataxia, and behavior change; the presence of the discretionary signs require a dedicated concussion evaluation. Following resolution of the discretionary signs, the athlete may RTP if the diagnosis of concussion is excluded and the discretionary signs can be attributed to another cause.

Initial management of a player suspected to have sustained a concussion should include consideration of intracranial, maxillofacial, cervical spine, and airway injuries, with implementation of appropriate management such as cervical spine immobilization as indicated. If the on-field or sideline evaluation is suggestive of concussion, the athlete should be moved to a distraction-free environment for a more thorough evaluation. A concussion evaluation should include a history and physical exam to elicit symptoms of concussion including confusion, headache, loss of consciousness, post-traumatic amnesia, retrograde amnesia, balance problems, dizziness, visual problems, personality changes, fatigue, sensitivity to light or noise, tinnitus, numbness, and vomiting [22]. The presence of dizziness reported by the athlete on the field or sideline is especially important to identify as this symptom is associated with a six times likelihood of having a protracted recovery of more than 21 days [27]. A thorough neurologic exam including administration of Maddock's questions, evaluation of the cervical spine, speech, gait, and eyes as well as a balance assessment should be performed [22, 27]. Additional tests such as the Sports Concussion Assessment Tool (SCAT5) and King-Devick tests have also been described as aids for diagnosing concussion. The SCAT5 consists of evaluating for "red flag" symptoms and signs of concussion as described above, Maddock's questions, Glasgow coma scale scoring, and cervical spine assessment [28]. The King-Devick test consists of reading numbers from left to right on cards and takes less than 2 min to perform [26]. Finally, athletes diagnosed with concussion should be serially evaluated for 48 hours to detect delayed symptom onset [22, 25].

1.5.1.3 In-Season Treatment

For athletes with concussion, RTP is permitted after concussion-related symptom scores have returned to baseline levels at rest and with competition-intensity exercise, the neurologic examination is normal, and cognitive testing has returned to baseline or age-appropriate levels [25]. A graded RTP protocol is recommended, which typically takes 7 days. Elite athletes with early resolution of symptoms may return to play earlier than 7 days following intensive specialist-directed evaluation in an advanced care setting specializing in concussion management while younger athletes are typically managed more conservatively [25]. In the NFL, the team physician, in consultation with an independent consultant, is responsible for RTP decisions [22, 25]. At all levels, no athlete should RTP until cleared by a qualified healthcare professional.

1.5.1.4 Prevention of Injury

Concussion identification and prevention has gained international attention over the past few years and led to significant changes and improvements to sporting equipment and the game rules. The most important aspect of concussion prevention is educating players, trainers, coaches, and match officials on early recognition, immediate management, and appropriate RTP criteria for players of all ages [29, 30]. Although recent strides have been made, further education is warranted, especially regarding the misconceptions of concussions and less-commonly recognized symptoms [29].

Improvements and implementation of specific sporting equipment, including customized mouth guards and helmets, have led to a decrease in self-reported concussions and traumatic brain injuries [31–34]. Rule changes, including video review and in-arena spotters to identify signs of concussion, have improved diagnosis of in-game concussions [35–37]. Over the past few years, the NFL has made specific changes to kickoff and targeting rules to enhance player safety. However, there is a paucity of literature regarding the effectiveness of these kickoff and targeting rule changes.

1.5.2 ACL and MCL Injury

1.5.2.1 Mechanism of Injury

The typical mechanism of ACL injury during American football is a noncontact injury usually sustained during a lateral movement such as pivoting or cutting resulting in a dynamic valgus moment on the knee while the knee is positioned in near extension with the foot externally rotated [38]. MCL injuries typically occur when a valgus force is directly applied to the knee or results from cutting maneuvers with the foot planted [39–41]. Given the similar mechanism of injury, ACL and MCL injuries commonly occur simultaneously, with American football players sustaining a concomitant ACL and MCL injury at a rate 2.7 times higher than athletes in other sports [42].

1.5.2.2 On-Field Management

Evaluation for ACL injury consists of the anterior drawer and Lachman tests in which an anterior force is applied to the tibia with the knee at 90° and 30°, respectively, with excessive anterior translation indicative of ACL injury [43]. The pivot shift test is useful to assess for rotatory instability and is performed by applying a combined valgus moment to the knee and internal rotation force on the tibia with the knee initially extended followed by flexion of the knee and observation of spontaneous posterior reduction of the tibia [43]. Diagnosis of MCL injury is typically facilitated by palpation over the ligament to elicit tenderness as well as assessment of knee laxity upon application of a valgus load. Evaluation of the MCL to test for ligamentous laxity is performed by applying a valgus force to the knee flexed to 30° [39]. Medial compartment opening of 0–5 mm corresponds to a grade I injury in which the ligament is stretched without ligamentous disruption, 5–10 mm corresponds to a grade II injury with partial ligament tearing, and >10 mm opening corresponds to a grade III injury with complete tear of all MCL fibers [39, 40].

Following initial on-field evaluation, a standard radiographic evaluation should be performed to assess for dislocation or osseous injury followed by magnetic resonance imaging (MRI) to evaluate for ACL or MCL injury and concomitant injuries to the meniscus, articular cartilage, and cruciate or collateral ligaments (Fig. 1.2) [26, 43].

1.5.2.3 In-Season Treatment

Although nonoperative management, consisting of physical therapy, anti-inflammatory medications, increasing range of motion and strength, and progressive RTP, can be considered for recreational athletes, for the majority of American football players, ACL reconstruction is recommended within 5 months of injury to prevent the development of additional injuries such as menis-

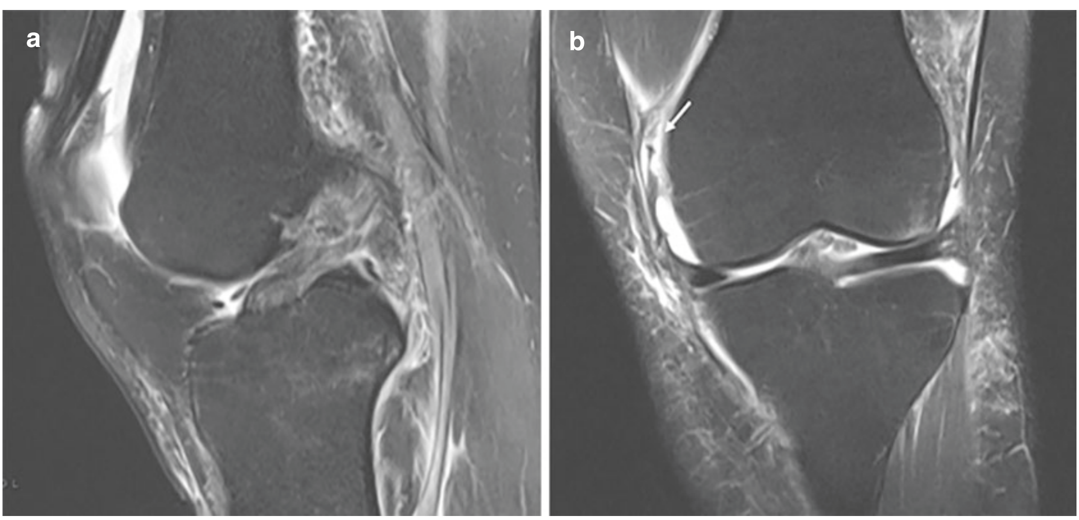

Fig. 1.2 Magnetic Resonance Imaging (MRI) of a left knee with anterior cruciate ligament (ACL) and medial collateral ligament (MCL) tears. Image (**a**) is a T2-weighted sagittal MRI demonstrating a complete midsubstance ACL tear. Image (**b**) is a T2-weighted coronal MRI demonstrating a full thickness MCL tear off its femoral origin (white arrow)

cal tears [43]. When performing an ACL reconstruction in an American football player, bone-patellar tendon-bone autograft is preferred by most surgeons due to high rates of failure with allograft and hamstring tendon autograft in young athletes [44–48].

In a 2016 case series of NFL players who had undergone an orthopedic procedure, 82% returned to play following ACL reconstruction; however, athletes played for an average of 1.6 years following ACL reconstruction, and for those who did return, decreased number of games played and performance can be expected until postoperative seasons 2 and 3 [49]. At the high school and college level, approximately 2/3 of athletes will RTP following an ACL injury [50]. At the professional level, athletes have been shown to RTP on average 10.8 months after primary ACL reconstruction while RTP occurred at 12.6 months following revision ACL reconstruction [51, 52]. In general, athletes should RTP at a minimum of 9 months postoperatively to allow for graft healing once the player is able to meet postoperative RTP protocols such as demonstration of symmetric quadriceps strength and performance in hop testing, as well as the ability to safely perform sport-specific movements [43]. Given the length required for RTP, an ACL injury can considered to be season-ending in most cases.

Treatment of isolated MCL injuries is dictated by the grade of injury. Nonoperative management consisting of initial rest, ice, compression, and elevation of the injured extremity followed by weight-bearing in a hinged knee brace and strengthening exercises, is recommended for isolated grade I–II injuries [39, 40]. Isolated grade III injuries may be treated nonoperatively or operatively, with operative intervention indicated in isolated grade III tears with severe valgus alignment, MCL entrapment of the pes anserinus, or avulsion injuries [41]. Concomitant ACL and grade I or II MCL injuries are typically managed with initial nonoperative management to allow MCL healing followed by reconstruction of the ACL [40]. Management of ACL injuries with concomitant grade III MCL injury is controversial. Nonoperative management of the MCL injury followed by delayed ACL reconstruction as well as early ACL reconstruction with nonoperative or operative management of the MCL tear have been proposed with no clear superiority of any approach [26].

Following nonoperative treatment, athletes with a grade I MCL injury can expect to RTP 11 days post-injury, while those with grade II injuries RTP 20 days post-injury on average [53]. Grade III injuries require longer rehabilitation times, particularly following surgical intervention. Athletes with nonoperatively treated grade III injuries may RTP as early as 5–7 weeks after injury while those treated with surgery may require 6–9 months of rehabilitation [54]. In-season management of MCL tears should be determined after discussion of the risks and benefits of each treatment modality including the average time until RTP with the athlete and his or her family.

1.5.2.4 Prevention of Injury

In order to help prevent ligamentous injury to American football athletes, prevention programs should be incorporated into training regimens. The prevention programs should incorporate neuromuscular training including proprioceptive and muscle-activation exercises, single-leg training, game-time decision-making for unanticipated conditions, and proper foot positioning in dynamic movements [55].

Although bracing is common among American football athletes, there has been conflicting evidence on the efficacy of bracing in American football athletes. Prior studies have demonstrated a protective effect of bracing on MCL injury [56, 57], especially in the high-risk positions of offensive and defensive line, linebacker, and tight end [58]. However, other studies have questioned the effectiveness of bracing and no difference or an increased risk of knee injuries with bracing has been shown [58–60]. There is no conclusive evidence that bracing prevents ACL tears, or is protective of an ACL-reconstructed knee [61].

1.5.3 Anterior and Posterior Labral Tears Associated with Glenohumeral Instability

1.5.3.1 Mechanism of Injury

Anterior and posterior labral tears resulting from acute glenohumeral instability have distinct mechanisms of injury and presentation [62]. Glenohumeral instability events are typically described as either a subluxation, in which symptomatic instability occurs without separation of the articular surfaces requiring reduction, or dislocation, with complete disruption of the articular surfaces requiring reduction [18]. Anterior instability typically results from an acute anterior dislocation event with the arm in the abducted and externally rotated position, while posterior labral injuries typically present in a more insidious manner resulting from high-intensity, dynamic posterior loading to a forward flexed, internally rotated, and adducted arm causing a shearing force on the posterior labrum [62–65]. Anterior instability commonly causes avulsion of the anterior labrum and capsular attachments at the glenoid rim, known as a Bankart lesion [64]. In American football, skilled position players (i.e., running back, quarterback, defensive back, and linebackers) are more likely to sustain an anterior instability injury while linemen are at particular risk for posterior instability due to the nature of blocking and the propensity for bench pressing heavy weight [17, 63].

1.5.3.2 On-Field Management

An acute anterior shoulder dislocation is generally seen with the arm held in abduction and external rotation with a palpable prominence over the anterior shoulder accompanied by a defect or sulcus on the posterior shoulder. An acute posterior shoulder dislocation typically presents with the shoulder held in an adducted, internally rotated position [63]. Reduction may be attempted prior to the onset of muscle spasm, with successful reduction followed by a repeat neurovascular examination. If the glenohumeral joint cannot be reduced, transfer to a medical center for closed or open reduction is necessary.

For athletes presenting without frank dislocation events, physical examination for anterior instability consists of the apprehension sign, or feeling of apprehension induced with the arm in abduction and external rotation [64]. This can be combined with the Jobe relocation test which involves application of a posterior force on the anterior shoulder; this test is positive when this maneuver alleviates the feeling of apprehension. In addition, the anterior load-and-shift test can be used to evaluate the amount of anterior humeral translation present. A sulcus sign may be seen with inferior traction on the arm, particularly with inferior capsular laxity or multidirectional instability.

In cases of suspected posterior subluxation, patients may complain of pain or weakness rather than instability; these symptoms may be more prominent at the end of a game or workout when the surrounding musculature is fatigued and unable to compensate for loss of structural integrity [63]. Physical examination should evaluate for associated atrophy, scapular winging or dyskinesis, and generalized ligamentous laxity. The posterior load-and-shift, jerk, and posterior apprehension tests are also useful for detecting posterior instability [63]. The Kim test is particularly useful for evaluation of posteroinferior labral tears. This test is performed by applying an axial load with the arm abducted to 90° and forward flexed 45° in the sagittal plane [18]. The test is positive if this maneuver recreates the patient's pain.

Radiographic analysis should be performed to assess for associated osseous abnormalities such as fracture and evaluate for possible glenoid dysplasia or bone loss [63]. MRI, often with contrast (magnetic resonance arthrogram), is useful for evaluating the intra-articular structures including the anterior and posterior capsule and labrum (Fig. 1.3) [18, 63]. If there is concern for significant osseous deformity or bone loss, computed tomography scan with three-dimensional reconstruction is useful [63].

1.5.3.3 In-Season Treatment

Following first-time instability events without associated osseous defects, nonsurgical manage-

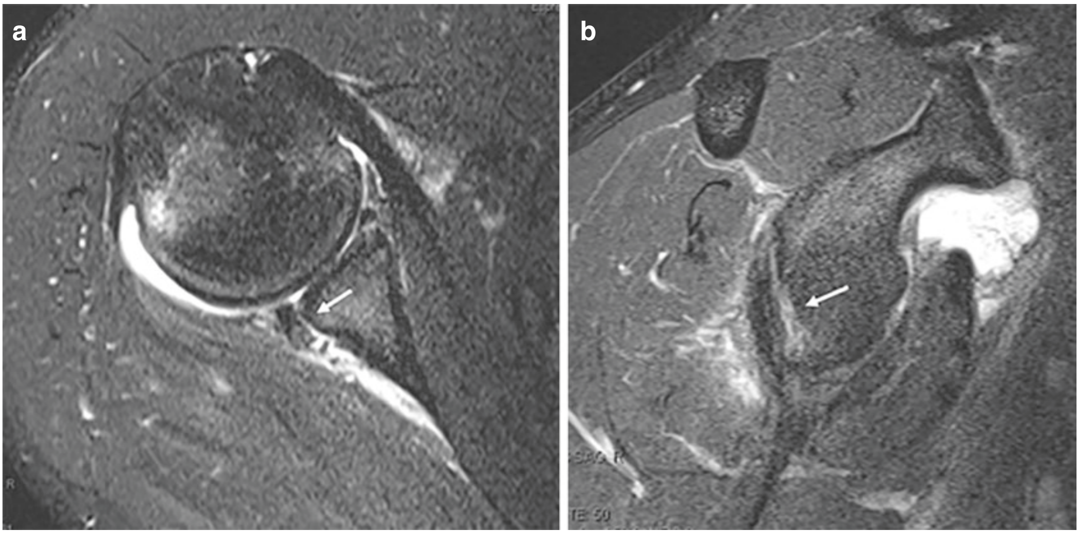

Fig. 1.3 Magnetic Resonance Imaging (MRI) of a left shoulder with a posterior labral tear. Image (**a**) is a T2-weighted axial MRI demonstrating a posterior labral tear (white arrow). Image (**b**) is a T2-weighted sagittal MRI demonstrating the posterior labral tear (white arrow)

ment is often the treatment of choice with surgical management reserved for those who fail initial nonoperative management [18]. Initial nonoperative management is especially attractive to many athletes as it allows earlier RTP than surgical intervention, which is often a season-ending event. Most athletes are able to return to sport after a brief period of immobilization followed by range of motion and strengthening exercises for 3 weeks, provided the athlete has symmetric pain-free range of motion and strength, is able to perform sport-specific skills, and there is no evidence of instability [66]. However, athletes should be counseled that 59–90% will experience a recurrent anterior instability event without surgical treatment [64, 66, 67]. With each instability event, the athlete is at risk of more severe injury, which may necessitate more complex surgical intervention with worse outcomes [66]. Following failed initial nonoperative management or multiple dislocations, concomitant injury such as a rotator cuff tear or periarticular fracture, or in the setting of significant osseous defects of the humeral head or glenoid, surgical intervention is recommended to reduce the likelihood of recurrent episodes of instability [64, 68].

The treatment outcomes for anterior and posterior labral tears as a result of glenohumeral instability are different. Significant improvements in clinical outcome scores have been demonstrated following both anterior and posterior labral repair; however, patients with anterior instability have been shown to have better postoperative clinical outcome scores than those with posterior instability [62]. Ultimately, the management of the in-season instability event requires a discussion with the athlete and his or her family regarding the risks and benefits of each treatment option, and a decision made based on their individual values, preferences, and goals.

1.5.3.4 Prevention of Injury

Periscapular and rotator cuff muscles assist with shoulder stabilization and glenohumeral joint kinematics. A periscapular and rotator cuff strengthening program should be incorporated into daily workouts to prevent glenohumeral joint dislocations.

Shoulder braces are routinely used by athletes with a history of shoulder subluxations or dislocations, as the brace has been shown to reduce shoulder range of motion and add stability to the glenohumeral joint [69, 70]. However, there is a paucity of evidence regarding the efficacy of shoulder bracing to reduce the incidence of shoulder injury in athletes.

1.5.4 AC Injury and Clavicle Fracture

1.5.4.1 Mechanism of Injury

Injuries to the AC joint and clavicle most commonly occur from a direct blow to the top of the shoulder, which may occur when tackling or diving or via an indirect injury such as a fall on an outstretched arm [18, 71–73]. AC joint injuries most frequently affect defensive backs, wide receivers, and special team players, but the incidence of injury is greatest in quarterbacks, special team players, and wide receivers [19].

1.5.4.2 On-Field Management

In the evaluation of an AC joint injury or clavicle fracture, a deformity may be seen and tenderness present at the site of injury. The athlete may report pain with movement of the arm, particularly with cross-body adduction.

Radiographic evaluation should be performed to evaluate for associated fractures in the setting of AC joint injury or to confirm the diagnosis of a clavicle fracture. The use of the Zanca view, or an antero-posterior (AP) radiograph with 15° cephalic tilt, can be particularly helpful in visualizing the AC joint [18]. MRI may be useful for more severe AC joint injuries to evaluate for the presence of concomitant injury; edema in the AC joint is a hallmark of AC joint injury [18, 72].

1.5.4.3 In-Season Treatment

AC joint injuries are typically treated according to the type of injury [72]. Type I and II injuries, which involve sprains of the AC and coracoclavicular (CC) ligaments, respectively, are typically treated nonoperatively in a sling with rest, pain control, cryotherapy, and physical therapy [18, 72, 74]. Type I or II injuries are often also treated with injection of local anesthetic to allow athletes to continue to play [75]. Treatment of type III injuries, with disruption of the AC and CC ligaments and a CC distance increased by 25–100% compared to the contralateral side, is controversial; however, 70% of NCAA team physicians preferred nonoperative management in a 2016 survey (Fig. 1.4) [75]. Type IV–VI, which involve significant displacement of the clavicle relative to the acromion, are typically managed operatively [72, 74].

The majority of AC joint injuries in American football players are Type I and II injuries and can be managed nonoperatively, with surgery required in only 1.4% of injuries [19]. The average loss of time for AC joint injuries was 10 days; however, AC joint dislocations and type III AC sprains had an average of 78 and 26 days lost to injury, respectively [19].

Nondisplaced midshaft clavicle fractures can typically be managed nonoperatively with rest

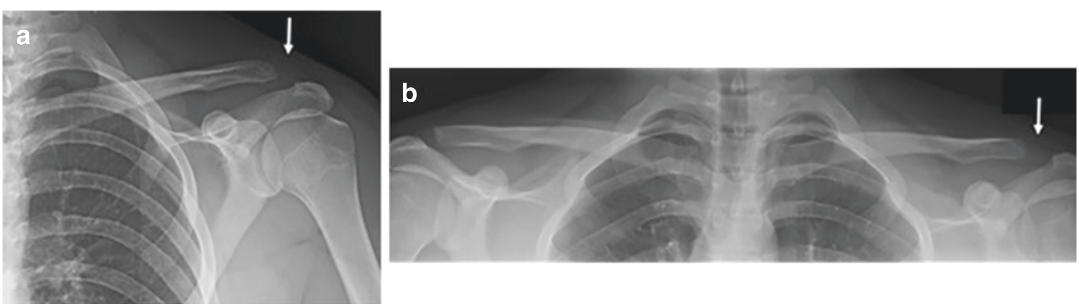

Fig. 1.4 Standing radiographs of a type III left shoulder acromioclavicular joint (AC) injury. Image (**a**) is a left shoulder antero-posterior radiograph demonstrating an AC joint injury (white arrow). Image (**b**) is a bilateral shoulder radiograph demonstrating asymmetric AC joint on the left shoulder (white arrow) as compared to the contralateral side

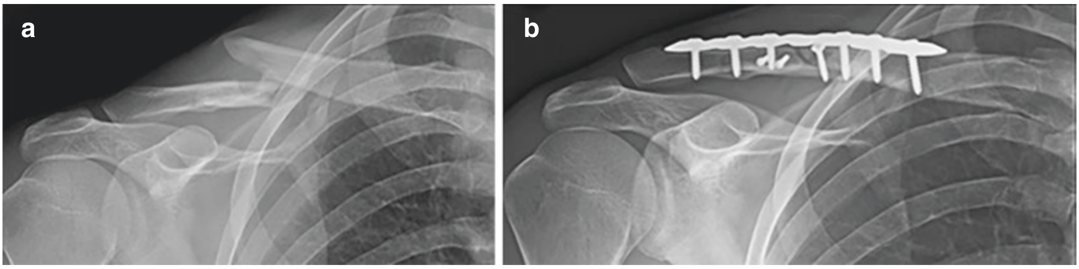

Fig. 1.5 Standing radiographs of a displaced right clavicle fracture. Image (**a**) is a right shoulder antero-posterior radiograph demonstrating a displaced clavicle fracture. Image (**b**) is a postoperative radiograph of the right shoulder after open reduction internal fixation of the fracture

and initial sling immobilization followed by increasing range of motion and strength [76]. Open reduction internal fixation (ORIF) is typically used for treatment of displaced fractures (Fig. 1.5). Distal clavicle fractures are managed based on the relationship of the fracture to the CC ligaments. Fractures distal to the CC ligaments are considered stable and can be managed nonoperatively while those that involve or are medial to the CC ligaments require ORIF [71]. Methods of fixation include hook pate fixation, plate fixation with ligament reconstruction, open suture fixation, arthroscopic Endobutton fixation, CC screw fixation, cerclage wire fixation, and tension band wiring [76].

Athletes can expect a recovery period of 10–12 weeks following midshaft clavicle fractures [76, 77]. Following a distal clavicle fracture, RTP typically occurs between 14 and 20 weeks post-injury [72, 76, 77].

1.5.4.4 Prevention of Injury

Over the past 20 years, the incidence of AC joint injuries have decreased, most notably due to changes in practice regimens and decreased physical contact in practice [19]. There is currently no protective equipment for the prevention of AC joint and clavicle fractures. American football athletes wear shoulder pads that decrease the risk of shoulder injury during routine plays, but a high-energy hit to the shoulder or contact with the playing surface may still lead to an AC joint or clavicle injury.

1.5.5 Ankle Sprains and Syndesmotic Injury

1.5.5.1 Mechanism of Injury

Lateral ligament sprains, or low ankle sprains, typically occur with inversion of the foot accompanied by external rotation of the ankle joint [78, 79]. Syndesmosis injury, also known as a high ankle sprain, typically occurs when the foot is dorsiflexed, everted, and externally rotated in relation to the tibia which typically occurs during a collision causing the athlete to fall forward while the foot is plantarflexed and externally rotated [80].

1.5.5.2 On-Field Management

Evaluation of an acute ankle sprain typically reveals pain, swelling, and ecchymosis located anterolaterally and posteromedially at the level of the ankle joint (Fig. 1.6) [19, 80]. The anterior drawer test, performed by applying an anterior force on the posterior hindfoot with the foot in neutral position, is useful for testing the integrity of the anterior tibiofibular ligament [79]. The calcaneofibular ligament can be tested by inverting the hindfoot with the ankle in neutral dorsiflexion [78, 79]. Several provocative tests are useful in evaluating for syndesmosis injuries. Pain when applying a compressive force between the proximal fibula and tibia with pain (squeeze test) or with external rotation of the foot and ankle relative to the tibia is suggestive of syndesmotic injury [80].

Fig. 1.6 On-field evaluation of a low ankle injury. This evaluation typically reveals pain and swelling about the anterolateral and posteromedial ankle

Radiographic evaluation of low ankle sprains should be dictated by the Ottawa ankle rules for evaluating for ankle fractures [79]. In cases of suspected high ankle sprain, weight-bearing and external rotation stress radiographs are useful to identify associated fractures or tibiofibular diastasis [78, 80, 81]. Full-length radiographs of the tibia and fibula should be obtained to evaluate for possible proximal fibular fracture, also known as a Maisonneuve injury [78]. MRI evaluation is also useful to show the extent of ligamentous injury as well as the presence of nondisplaced fractures.

1.5.5.3 In-Season Treatment

Treatment of low ankle sprains begins with nonoperative management with compression, ice, pain control with anti-inflammatory medications, and elevation. Injuries without complete tear of the lateral ligaments should be treated with immediate functional rehabilitation while those with complete ligamentous tears treated with a short period of immobilization followed by rehabilitation [78]. Functional rehabilitation consists of early mobilization with external support followed by range of motion, strengthening, and proprioceptive exercises, and return to sport-specific activity [79]. Surgical intervention, consisting of repair or reconstruction of the lateral ligaments, can be considered for those who fail initial nonoperative management or for elite athletes [79].

Syndesmotic injury without frank diastasis or dynamic instability on weight-bearing or stress radiographs can be managed nonsurgically [80]. Nonsurgical management consists of rest, ice, and immobilization for 3–5 days to allow inflammation to resolve. Weight-bearing is allowed in a boot with initiation of active and passive range of motion after 3–5 days. Once pain is resolved in the boot, a stabilizing brace is used and strengthening and functional exercises begin, followed by running and initiation of sport-specific activities. The ability to hop on the injured leg ten times is useful to help indicate that the athlete is ready to initiate sport-specific activities [80].

Unstable injuries necessitate surgical intervention, typically with ORIF of the syndesmosis with a screw or suture button construct [80]. Following fixation with syndesmotic screws, the patient is typically made nonweight-bearing for 4–6 weeks, and athletes may RTP between 10 and 12 weeks postoperatively if there is no symptoms of hardware irritation necessitating removal [80]. Stabilization with a suture button construct may allow more physiologic motion between the tibia and fibula and does not require hardware removal [80]. Following treatment of high and low ankle sprains, athletes are allowed to RTP when range of motion and strength has returned to baseline or at least 90% of the contralateral, uninjured extremity [78, 81].

1.5.5.4 Prevention of Injury

Prophylactic ankle taping has been the mainstay of ankle injury prevention, as it reduces ankle plantarflexion and inversion and improves proprioception [82–84]. However, it has been shown that ankle tape loosens with physical activity and may have a significant reduction in support within 30 min of initiation of exercise [85, 86]. Additionally, there is an added cost to the team for taping materials. Therefore, ankle orthoses or braces have been advocated due to `low cost and ability to reuse during the season [87, 88]. A recent randomized control trial compared taping with a commercially available ankle brace. The authors found no difference in ankle sprains sustained throughout the season between taping and bracing and concluded bracing was more economical and time-saving [89].

Summary/Take Home Messages

American football is a violent collision sport that places the athlete at risk for significant injuries including career-ending ligamentous knee injuries and fractures. Although a wide variety of injuries are sustained, five common injuries include concussions, ACL and MCL tears, glenohumeral shoulder instability, AC joint injuries and clavicle fractures, and ankle sprains and syndesmotic injuries. Concussions can have significant physical and mental sequela, and the key to prevention of concussions is recognition of symptoms, timely treatment, and knowledge of RTP guidelines. The majority of extremity injuries cannot be avoided, but can be limited with proper physical and mental training. Knee bracing can be effective for limiting MCL tears, while ankle taping and bracing is effective for limiting ankle sprains. Shoulder bracing has not been shown to prevent shoulder injuries and knee bracing has not been shown to prevent ACL tears. However, further research is needed to better understand the role of sports performance programs in injury prevention for athletes.

References

1. Mueller FO, Colgate B. Annual survey of football injury research, 1931–2009. Prepared for: The American Football Coaches Association, The National Collegiate Athletic Association and the National Federation of State High School Associations; 2010.
2. Shankar PR, Fields SK, Collins CL, Dick RW, Comstock RD. Epidemiology of high school and collegiate football injuries in the United States, 2005–2006. Am J Sports Med. 2007;35:1295–303.
3. Whiteside JA, Fleagle SB, Kalenak A, Weller HW. Manpower loss in football: a 12-year study at the Pennsylvania State University. Phys Sportsmed. 1985;13:102–14.
4. Dragoo JL, Braun HJ, Harris AH. The effect of playing surface on the incidence of ACL injuries in National Collegiate Athletic Association American Football. Knee. 2013;20:191–5.
5. Iacovelli JN, Yang J, Thomas G, Wu H, Schiltz T, Foster DT. The effect of field condition and shoe type on lower extremity injuries in American Football. Br J Sports Med. 2013;47:789–93.
6. Loughran GJ, Vulpis CT, Murphy JP, Weiner DA, Svoboda SJ, Hinton RY, et al. Incidence of knee injuries on artificial turf versus natural grass in National Collegiate Athletic Association American football: 2004–2005 through 2013–2014 seasons. Am J Sports Med. 2019;47:1294–301.
7. 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.

8. Kerr ZY, Cortes N, Ambegaonkar JP, Caswell AM, Prebble M, Romm K, et al. The epidemiology of injuries in middle school football, 2015–2017: the advancing healthcare initiatives for underserved students project. Am J Sports Med. 2019;47:933–41.
9. Kerr ZY, Simon JE, Grooms DR, Roos KG, Cohen RP, Dompier TP. Epidemiology of football injuries in the National Collegiate Athletic Association, 2004–2005 to 2008–2009. Orthop J Sports Med. 2016;4:2325967116664500.
10. Smith PJ, Hollins AM, Sawyer JR, Spence DD, Outlaw S, Kelly DM. Characterization of American Football injuries in children and adolescents. J Pediatr Orthop. 2018;38:e57–60.
11. Bradley J, Honkamp NJ, Jost P, West R, Norwig J, Kaplan LD. Incidence and variance of knee injuries in elite college football players. Am J Orthop (Belle Mead NJ). 2008;37:310–4.
12. 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;10:495–9.
13. Bradley JP, Klimkiewicz JJ, Rytel MJ, Powell JW. Anterior cruciate ligament injuries in the National Football League: epidemiology and current treatment trends among team physicians. Arthroscopy. 2002;18:502–9.
14. Brophy RH, Lyman S, Chehab EL, Barnes RP, Rodeo SA, Warren RF. Predictive value of prior injury on career in professional American football is affected by player position. Am J Sports Med. 2009;37:768–75.
15. Lievers WB, Adamic PF. Incidence and severity of foot and ankle injuries in men's collegiate American Football. Orthop J Sports Med. 2015;3:2325967115581593.
16. McHale KJ, Vopat BG, Beaulieu-Jones BR, Sanchez G, Whalen JM, McDonald LS, et al. Epidemiology and outcomes of Lisfranc injuries identified at the National Football League Scouting Combine. Am J Sports Med. 2017;45:1901–8.
17. Kaplan LD, Flanigan DC, Norwig J, Jost P, Bradley J. Prevalence and variance of shoulder injuries in elite collegiate football players. Am J Sports Med. 2005;33:1142–6.
18. Gibbs DB, Lynch TS, Nuber ED, Nuber GW. Common shoulder injuries in American Football athletes. Curr Sports Med Rep. 2015;14:413–9.
19. Lynch TS, Saltzman MD, Ghodasra JH, Bilimoria KY, Bowen MK, Nuber GW. Acromioclavicular joint injuries in the National Football League: epidemiology and management. Am J Sports Med. 2013;41:2904–8.
20. Chung AS, Makovicka JL, Hassebrock JD, Patel KA, Tummala SV, Deckey DG, et al. Epidemiology of cervical injuries in NCAA Football players. Spine (Phila Pa 1976). 2019;44:848–54.
21. Wascher DC, Bulthuis L. Extremity trauma: field management of sports injuries. Curr Rev Musculoskelet Med. 2014;7:387–93.
22. Ellenbogen RG, Batjer H, Cardenas J, Berger M, Bailes J, Pieroth E, et al. National Football League Head, Neck and Spine Committee's concussion diagnosis and management protocol: 2017–18 season. Br J Sports Med. 2018;52:894–902.
23. Lynall RC, Campbell KR, Wasserman EB, Dompier TP, Kerr ZY. Concussion mechanisms and activities in youth, high school, and college football. J Neurotrauma. 2017;34:2684–90.
24. Lessley DJ, Kent RW, Funk JR, Sherwood CP, Cormier JM, Crandall JR, et al. Video analysis of reported concussion events in the National Football League during the 2015–2016 and 2016–2017 seasons. Am J Sports Med. 2018;46:3502–10.
25. 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;52:635–41.
26. Leong DF, Balcer LJ, Galetta SL, Evans G, Gimre M, Watt D. The King–Devick test for sideline concussion screening in collegiate football. J Opt. 2015;8:131–9.
27. Lau BC, Kontos AP, Collins MW, Mucha A, Lovell MR. Which on-field signs/symptoms predict protracted recovery from sport-related concussion among high school football players? Am J Sports Med. 2011;39:2311–8.
28. 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;51:848–50.
29. Yeo PC, Yeo EQ, Probert J, Sim SH, Sirisena D. A systematic review and qualitative analysis of concussion knowledge amongst sports coaches and match officials. J Sports Sci Med. 2020;19:65–77.
30. Zhang AL, Sing DC, Rugg CM, Feeley BT, Senter C. The rise of concussions in the adolescent population. Orthop J Sports Med. 2016;4:2325967116662458.
31. Cantu RC, Mueller FO. Brain injury-related fatalities in American Football, 1945–1999. Neurosurgery. 2003;52:846–53.
32. Daneshvar DH, Baugh CM, Nowinski CJ, McKee AC, Stern RA, Cantu RC. Helmets and mouth guards: the role of personal equipment in preventing sport-related concussions. Clin Sports Med. 2011;30:145–63.
33. Dave Singh G, Maher GJ, Padilla RR. Customized mandibular orthotics in the prevention of concussion/mild traumatic brain injury in football players: a preliminary study. Dent Traumatol. 2009;25:515–21.
34. Hollis SJ, Stevenson MR, McIntosh AS, Shores EA, Collins MW, Taylor CB. Incidence, risk, and protective factors of mild traumatic brain injury in a cohort of Australian nonprofessional male rugby players. Am J Sports Med. 2009;37:2328–33.
35. Mack C, Myers E, Barnes R, Solomon G, Sills A. Engaging athletic trainers in concussion detection: overview of the National Football League ATC Spotter Program, 2011–2017. J Athl Train. 2019;54:852–7.
36. Mack C, Sendor RR, Solomon G, Ellenbogen RG, Myers E, Berger M, et al. Enhancing concussion man-

agement in the National Football League: evolution and initial results of the Unaffiliated Neurotrauma Consultants Program, 2012–2017. Neurosurgery. 2019;87:312. https://doi.org/10.1093/neuros/nyz481.
37. Zuckerman SL, Elbin RJ, Sills AK, Crandall JR, Lessley DJ, Moran CM, et al. (2020) Concussions in the National Football League: the evolution of video review for assessing the frequency and reliability of visible signs. Phys Sportsmed; https://doi.org/10.1080/00913847.2020.17313791-6
38. Johnston JT, Mandelbaum BR, Schub D, Rodeo SA, Matava MJ, Silvers-Granelli HJ, et al. Video analysis of anterior cruciate ligament tears in professional American Football athletes. Am J Sports Med. 2018;46:862–8.
39. Chen L, Kim PD, Ahmad CS, Levine WN. Medial collateral ligament injuries of the knee: current treatment concepts. Curr Rev Musculoskelet Med. 2008;1:108–13.
40. Elkin JL, Zamora E, Gallo RA. Combined anterior cruciate ligament and medial collateral ligament knee injuries: anatomy, diagnosis, management recommendations, and return to sport. Curr Rev Musculoskelet Med. 2019;12:239–44.
41. Encinas-Ullán CA, Rodríguez-Merchán EC. Isolated medial collateral ligament tears: an update on management. EFORT Open Rev. 2018;3:398–407.
42. Granan L-P, Inacio MCS, Maletis GB, Funahashi TT, Engebretsen L. Sport-specific injury pattern recorded during anterior cruciate ligament reconstruction. Am J Sports Med. 2013;41:2814–8.
43. Musahl V, Karlsson J. Anterior cruciate ligament tear. N Engl J Med. 2019;380:2341–8.
44. Erickson BJ, Harris JD, Fillingham YA, Frank RM, Bush-Joseph CA, Bach BR, et al. Anterior cruciate ligament reconstruction practice patterns by NFL and NCAA Football team physicians. Arthroscopy. 2014;30:731–8.
45. Gifstad T, Foss OA, Engebretsen L, Lind M, Forssblad M, Albrektsen G, et al. Lower risk of revision with patellar tendon autografts compared with hamstring autografts. Am J Sports Med. 2014;42:2319–28.
46. Kane PW, Wascher J, Dodson CC, Hammoud S, Cohen SB, Ciccotti MG. Anterior cruciate ligament reconstruction with bone-patellar tendon-bone autograft versus allograft in skeletally mature patients aged 25 years or younger. Knee Surg Sports Traumatol Arthrosc. 2016;24:3627–33.
47. Kraeutler MJ, Bravman JT, McCarty EC. Bone-patellar tendon-bone autograft versus allograft in outcomes of anterior cruciate ligament reconstruction. Am J Sports Med. 2013;41:2439–48.
48. Schrock JB, Carver TJ, Kraeutler MJ, McCarty EC. Evolving treatment patterns of NFL players by orthopaedic team physicians over the past decade, 2008–2016. Sports Health. 2018;10:453–61.
49. Mai HT, Alvarez AP, Freshman RD, Chun DS, Minhas SV, Patel AA, et al. The NFL Orthopaedic Surgery Outcomes Database (NO-SOD). Am J Sports Med. 2016;44:2255–62.
50. McCullough KA, Phelps KD, Spindler KP, Matava MJ, Dunn WR, Parker RD, et al. Return to high school- and college-level football after anterior cruciate ligament reconstruction. Am J Sports Med. 2012;40:2523–9.
51. Okoroha KR, Kadri O, Keller RA, Marshall N, Cizmic Z, Moutzouros V. Return to play after revision anterior cruciate ligament reconstruction in National Football League players. Orthop J Sports Med. 2017;5:232596711769878.
52. Shah VM, Andrews JR, Fleisig GS, McMichael CS, Lemak LJ. Return to play after anterior cruciate ligament reconstruction in National Football League Athletes. Am J Sports Med. 2010;38:2233–9.
53. Derscheid GL, Garrick JG. Medial collateral ligament injuries in football. Am J Sports Med. 1981;9:365–8.
54. Kim C, Chasse PM, Taylor DC. Return to play after medial collateral ligament injury. Clin Sports Med. 2016;35:679–96.
55. Sugimoto D, Alentorn-Geli E, Mendiguchía J, Samuelsson K, Karlsson J, Myer GD. Biomechanical and neuromuscular characteristics of male athletes: implications for the development of anterior cruciate ligament injury prevention programs. Sports Med. 2015;45:809–22.
56. Albright JP, Powell JW, Smith W, Martindale A, Crowley E, Monroe J, et al. Medial collateral ligament knee sprains in college football: effectiveness of preventive braces. Am J Sports Med. 1994;22:12–8.
57. Sitler M, Ryan CJ, Hopkinson LW, Wheeler LJ, Santomier J, Kolb LR, et al. The efficacy of a prophylactic knee brace to reduce knee injuries in football: a prospective, randomized study at West Point. Am J Sports Med. 1990;18:310–5.
58. Salata MJ, Gibbs AE, Sekiya JK. The effectiveness of prophylactic knee bracing in American Football: a systematic review. Sports Health. 2010;2:375–9.
59. Hewson GF Jr, Mendini RA, Wang JB. Prophylactic knee bracing in college football. Am J Sports Med. 1986;14:262–6.
60. Rovere GD, Haupt HA, Yates CS. Prophylactic knee bracing in college football. Am J Sports Med. 1987;15:111–6.
61. Wright RW, Fetzer GB. Bracing after ACL reconstruction: a systematic review. Clin Orthop Relat Res. 2007;455:162–8.
62. Bernhardson AS, Murphy CP, Aman ZS, Laprade RF, Provencher MT. A prospective analysis of patients with anterior versus posterior shoulder instability: a matched cohort examination and surgical outcome analysis of 200 patients. Am J Sports Med. 2019;47:682–7.
63. Antosh IJ, Tokish JM, Owens BD. Posterior shoulder instability. Sports Health. 2016;8:520–6.
64. Gil JA, Defroda S, Owens BD. Current concepts in the diagnosis and management of traumatic, anterior glenohumeral subluxations. Orthop J Sports Med. 2017;5:232596711769433.

65. Mair SD, Zarzour RH, Speer KP. Posterior labral injury in contact athletes. Am J Sports Med. 1998;26:753–8.
66. Owens BD, Dickens JF, Kilcoyne KG, Rue J-PH. Management of mid-season traumatic anterior shoulder instability in athletes. J Am Acad Orthop Surg. 2012;20:518–26.
67. Watson S, Allen B, Grant JA. A clinical review of return-to-play considerations after anterior shoulder dislocation. Sports Health. 2016;8:336–41.
68. Delong JM, Jiang K, Bradley JP. Posterior instability of the shoulder. Am J Sports Med. 2015;43:1805–17.
69. Conti M, Garofalo R, Castagna A, Massazza G, Ceccarelli E. Dynamic brace is a good option to treat first anterior shoulder dislocation in season. Musculoskelet Surg. 2017;101:169–73.
70. Dellabiancia F, Parel I, Filippi MV, Porcellini G, Merolla G. Glenohumeral and scapulohumeral kinematic analysis of patients with traumatic anterior instability wearing a shoulder brace: a prospective laboratory study. Musculoskelet Surg. 2017;101:159–67.
71. Kirsch JM, Tyrrell Burrus M, Bedi A. Common injuries in professional football quarterbacks. Curr Rev Musculoskelet Med. 2018;11:6–11.
72. Li X, Ma R, Bedi A, Dines DM, Altchek DW, Dines JS. Management of Acromioclavicular Joint Injuries. J Bone Joint Surg Am. 2014;96:73–84.
73. Stanley D, Trowbridge EA, Norris SH. The mechanism of clavicular fracture. A clinical and biomechanical analysis. J Bone Joint Surg Br. 1988;70:461–4.
74. Allemann F, Halvachizadeh S, Waldburger M, Schaefer F, Pothmann C, Pape HC, et al. Different treatment strategies for acromioclavicular dislocation injuries: a nationwide survey on open/minimally invasive and arthroscopic concepts. Eur J Med Res. 2019;24:18.
75. Carver TJ, Schrock JB, Kraeutler MJ, McCarty EC. The evolving treatment patterns of NCAA division I football players by orthopaedic team physicians over the past decade, 2008–2016. Sports Health. 2018;10:234–43.
76. Robertson GAJ, Wood AM. Return to sport following clavicle fractures: a systematic review. Br Med Bull. 2016;119:111–28.
77. Burnier M, Barlow JD, Sanchez-Sotelo J. Shoulder and elbow fractures in athletes. Curr Rev Musculoskelet Med. 2019;12:13–23.
78. Gill LA, Klingele KE. Management of foot and ankle injuries in pediatric and adolescent athletes: a narrative review. Orthop Res Rev. 2018;10:19–30.
79. Maffulli N, Ferran NA. Management of acute and chronic ankle instability. J Am Acad Orthop Surg. 2008;16:608–15.
80. Hunt KJ, Phisitkul P, Pirolo J, Amendola A. High ankle sprains and syndesmotic injuries in athletes. J Am Acad Orthop Surg. 2015;23:661–73.
81. Van Dijk CN, Longo UG, Loppini M, Florio P, Maltese L, Ciuffreda M, et al. Classification and diagnosis of acute isolated syndesmotic injuries: ESSKA-AFAS consensus and guidelines. Knee Surg Sports Traumatol Arthrosc. 2016;24:1200–16.
82. Garrick J, Requa R. Role of external support in the prevention of ankle sprains. Med Sci Sports. 1973;5:200–3.
83. Laughman R, Carr T, Chao E, Youdas J, Sim F. Three-dimensional kinematics of the taped ankle before and after exercise. Am J Sports Med. 1980;8:425–31.
84. Robbins S, Waked E, Rappel R. Ankle taping improves proprioception before and after exercise in young men. Br J Sports Med. 1995;29:242–7.
85. Fumich RM, Ellison AE, Guerin GJ, Grace PD. The measured effect of taping on combined foot and ankle motion before and after exercise. Am J Sports Med. 1981;9:165–70.
86. Myburgh KH, Vaughan CL, Isaacs SK. The effects of ankle guards and taping on joint motion before, during, and after a squash match. Am J Sports Med. 1984;12:441–6.
87. 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.
88. Rovere GD, Clarke TJ, Yates CS, Burley K. Retrospective comparison of taping and ankle stabilizers in preventing ankle injuries. Am J Sports Med. 1988;16:228–33.
89. Mickel TJ, Bottoni CR, Tsuji G, Chang K, Baum L, Tokushige KAS. Prophylactic bracing versus taping for the prevention of ankle sprains in high school athletes: a prospective, randomized trial. J Foot Ankle Surg. 2006;45:360–5.

Baseball, Softball, Cricket

2

Sports-Specific Injuries and Unique Mechanisms in Baseball, Softball, and Cricket

Adam Miller, Garrett Schwarzman, and Mark R. Hutchinson

2.1 Introduction to Baseball, Softball, and Cricket

This chapter focuses on baseball, softball, and cricket as both the physiology and mechanism of injury are similar among all three sports. These sports all fall under the category of "bat and ball" games in which teams take turns batting and fielding. The unique mechanics involved in pitching, or bowling in the case of cricket, provides the mechanism for the majority of injuries involved.

Considered America's pastime, baseball was first described in 1839 by Abner Doubleday [1]. Since that time the game has evolved considerably, with the most significant innovation being the introduction of overhead throwing for pitchers in the 1880s. All plays start with the pitcher, who throws the ball toward the batter up to 150 times per game. The incredible amount of stresses placed on the shoulder and elbow during pitching, compiled with the high amounts of repetition leads to the potential for overuse injuries. Similarly, in batting there is a large amount of power produced by trunk rotation, which can lead to injury. However, as the repetition involved in batting is much less than that seen with pitching, the overall risk for overuse injury is lower.

Softball is very similar to baseball in terms of rules, player positions, protective equipment, and the shape of the field on which the game is played. It was first played in Chicago in 1887 as an indoor variant of baseball [1] and has since become one of the most popular sports in the United States. A unique aspect of softball is the large number of adults who participate in the sport. As a result of this popularity and sheer numbers of participants at a given time, it is estimated that there is one person seen in an emergency department every 4 min in the United States for a softball-related injury [2]. Over the years, softball has evolved to become distinct from baseball in terms of biomechanical techniques and injuries sustained during play. The most notable difference is the underhanded pitching technique used in softball, which provides its own mechanism of injury. At the collegiate level, softball is more commonly played by women and baseball by men.

The sport of cricket has a known history beginning in the late sixteenth century. Having originated in south-east England, it became the country's national sport in the eighteenth century and has developed globally in the nineteenth and twentieth centuries. Cricket is the world's second most popular spectator sport after soccer. Although cricket is a non-contact sport, there is a wide variety of causes of injuries. It is a multi-

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dimensional sport in which players engage in a wide range of diverse activities, namely batting, bowling, fielding, and wicket keeping. Injuries on the cricket field tend to fall into one of the two categories: repetitive/overuse and impact [3] (Table 2.1).

Table 2.1 A comparison of injury patterns by body part between baseball, softball, and cricket

Body part	Baseball	Softball	Cricket
Foot/ankle (all)	2.9–9%	–	12.20%
Foot/ankle (ligament sprain)	–	19.80%	4.20%
Foot/ankle (stress fracture)	–	–	3%
Knee (all)	3.7–6%	21.40%	5.78%
Knee (internal derangement)	–	14.10%	3.10%
Knee (patella/patella tendon injury)	–	5.70%	–
Knee (contusion)	–	1.60%	–
Hip (muscle/tendon sprain)	1.3–3%	4.20%	–
Hamstring strain	–	–	13.60%
Other lower extremity (all)	9.4–12%	21.20%	11.90%
Other lower extremity (muscle/tendon strain)		14.60%	9.10%
Other trunk injuries	–	–	0.63%
Chest/abdomen impact injury	–	–	1.40%
Other lower extremity (contusion)		6.60%	–
Hernia, abdomen	0.8–3%	–	–
Side/abdominal strain	–	–	9.50%
Groin (all)	–	–	7.00%
Chest	2.3–4%	–	
Back/spine (all)	7.4–8%	1.50%	11.70%
Back/spine (muscle/tendon strain)	–	1.50%	–
Back/spine (stress fractures)	–	–	5.00%
Head (all)	0.6–5%	9.80%	2.70%
Head (concussion)	–	8.80%	1.40%
Head (nose/facial fracture)	–	1%	0.94%
Hand/wrist (all)	4.2–14%	13.40%	11.60%
Hand/wrist (fracture)	–	7.50%	7.30%
Hand/wrist (contusion)	–	3%	–
Hand/wrist (ligament sprain)	–	4.80%	–
Elbow (all)	9–26.3%	3.60%	1.70%
Elbow (contusion)	–	1%	–
Elbow (tendonitis)	–	1.60%	–
Elbow (muscle/tendon strain)	–	1%	–
Shoulder (all)	15–30.7%	17.10%	5.80%
Shoulder (muscle/tendon strain)	–	8.20%	2.00%
Shoulder (tendonitis)	–	5.90%	–
Shoulder (subluxation/instability)	–	3.00%	2.30%
Other upper extremity	5.4–6%	–	0.16%
Muscle strain/tear/rupture/cramps	30%	–	–
Contusion/hematoma	20%	–	–
Sprain/ligament injuries	12%	–	–
Tendinopathy/bursitis	8%	–	–
Fracture	3%	–	–
Concussion/brain injury	2%	–	–
Lesion of meniscus/cartilage/disc	2%	–	–
Abrasion	1%	–	–
Laceration	1%	–	–
Nerve injury	1%	–	–
Dislocation/subluxation	1%	–	–
Other injuries	19%	–	–

2.2 Sports-Specific Mechanics and Injury Risks in Baseball, Softball, and Cricket

The majority of injuries unique to these sports revolve around the pitcher/bowler and the mechanics of throwing the ball. These players are able to transfer large amounts of energy to the ball and release it with great velocity. The repetitiveness of the motion combined with the torque and power involved in each throw results in the potential for non-traumatic overuse injuries.

A baseball pitcher's throwing mechanics can be divided into six phases: the windup, stride, cocking, acceleration, deceleration, and follow through [1, 4] (Fig. 2.1). Compressive forces at the glenohumeral joint, internal rotation torque, horizontal abduction torque, and elbow valgus torque have all been identified as possible sources of overuse injury to the shoulder and elbow in pitchers. The windup phase begins with the ball in the pitcher's glove, the arms drop and the body rotates 90°, the stride leg is elevated and flexed [1, 4]. This phase varies between pitchers and provides a rhythm. This can be considered a loading phase and the injury risk is minimal.

The stride phase consists of the stride leg moving toward the plate while the trunk remains back [1, 4]. Injury risk is related to variances in the stride itself. If the stride is too long, there will be inadequate rotation of the hips, resulting in a loss of velocity. This break in the kinetic chain is known as "arm throwing," and the athlete may compensate by overloading the shoulder, resulting in a possible rotator cuff strain [1, 4]. If the stride is off line contralateral to the dominant arm, the torso will be ahead of the shoulder, resulting in "opening" too soon and stressing the anterior capsule; this may result in shoulder instability [1, 4]. If the stride is off line ipsilateral to the dominant arm, the pitcher will "throw across his body" with possible labral shearing [1, 4].

Fig. 2.1 Pitching in baseball can be broken down into phases including: windup, cocking, acceleration, and follow through. Cocking and acceleration have been associated with anterior shoulder instability and UCL injuries of the elbow. (© Mark R. Hutchinson, 2021. All Rights Reserved)

The cocking phase begins when the stride foot makes contact with the mound, hip rotation begins, followed by the trunk, and the shoulder reaches maximum external rotation and abduction [1, 4]. Injury risk in this phase is related to the extremes of range of motion experienced in the shoulder. Internal impingement may occur, wherein the undersurface of the superior rotator cuff impinges against the posterior/superior labrum [1, 4]. This may result in undersurface tearing of the rotator cuff and posterior/superior labrum fraying [1, 4]. Anterior capsule stretching may occur over time and result in increased anterior/inferior laxity and possible glenohumeral instability [1, 4].

The acceleration phase begins with internal rotation of the shoulder and ends with release of the ball [1, 4]. Injury risk involves the primary movers and stabilizers of the shoulder joint. Rotator cuff, latissimus dorsi, and teres major strains, as well as labral impingement/tears, may occur [1, 4]. Additionally, the elbow is placed under significant valgus stress as the ulnar collateral ligament (UCL) is under tension while radiocapitellar articulation is under compression [1, 4]. Thus, the UCL is at a risk of tearing and radiocapitellar articulation at a risk of osteochondral injury. Valgus extension overload may also occur, as the elbow extends under valgus load, the posteromedial aspect of the olecranon may impinge against the olecranon fossa, resulting in posteromedial elbow pain [1, 4]. Over time, spurring and degenerative changes may occur at the posteromedial ulnohumeral articulation as a result [1, 4].

The deceleration phase, where peak distraction force occurs in the shoulder, begins at ball release and ends with final elbow extension occurs and when internal rotation velocity reaches zero [1, 4]. Injury risk includes superior labrum anterior to posterior (SLAP) lesions, which are traction injuries to the superior labrum at insertion of the long head of biceps tendon. Additionally, Bennett lesions may occur, which are traction osteophytes of the posterior glenoid lip with thickening of posterior labrum and capsular attachment due to repetitive traction [1, 4].

The follow-through phase allows for dissipation of energy. It begins at the end of shoulder internal rotation and ends when the trailing leg touches the ground [1, 4]. Injury risk is low in this phase; in fact, a proper follow-through technique may help to minimize injury by implementing a gradual reduction in kinetic energy [1].

The main difference between softball and baseball is noted with pitching. A softball pitch, in contrast to baseball, is delivered in an underhanded fashion with the pitcher utilizing a windmill motion of the arm that requires up to 485° of total shoulder circumduction to complete [5]. Arm motion around the glenohumeral joint rotates the arm through an axis that is relatively perpendicular to the humeral diaphysis [6]. A common misconception regarding the softball pitch is that it produces less stress on the shoulder than the overhead baseball pitch, however, that hypothesis has since been found to be false. In fact, the windmill softball pitch generates similar forces about the shoulder as those seen in overhand pitching [5, 7, 8].

The softball pitch can be divided into four phases: the windup, stride, delivery, and follow through [5] (Fig. 2.2). The windup phase is defined from initial movement until the lead foot toe-off [5]. The stride phase begins with lead foot-toe off and ends with lead foot contact with the ground [5]. The delivery phase, which is where the largest amount of force and torque are seen in the shoulder and elbow, starts with lead foot contact and ends with release of the ball [5]. The follow-through phase begins from ball release and proceeds until the end of motion in throwing arm [5].

Given the circular trajectory the arm goes through during a windmill style pitch, arm motion can be broken down into its own phases and described as positions on a clock [9]. During the windup and early stride, the upward motion of the pitching arm from the 6 o'clock to the 3 o'clock position sees high activity in the supraspinatus, infraspinatus, teres minor, and deltoid muscle [10]. Injury risk is low in these early phases of the pitch as they produce the lowest forces generated during the pitching motion [5, 7, 8]. The humerus is primarily in an internally

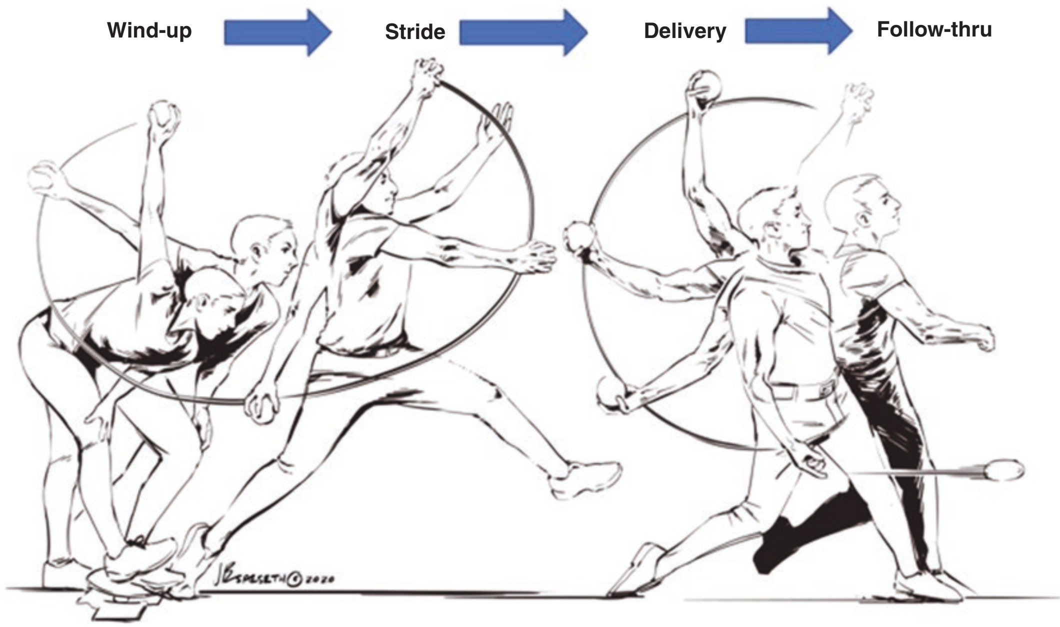

Fig. 2.2 Pitching in softball can be broken into phases including: windup, stride, delivery, and follow through with the round house underhand movement occurring during the stride and delivery phase. (© Mark R. Hutchinson, 2021. All Rights Reserved)

rotated position because it is flexed at the shoulder [9]. The trunk and pelvis see the stride leg moving forward at this stage, with increased activity in the contralateral gluteal muscles to stabilize the pelvis and generate momentum toward home plate [11].

There is an acceleration as the arm moves upward from the 3 o'clock to 12 o'clock position. During this phase, supraspinatus activity decreases, while infraspinatus and teres minor, along with anterior deltoid, remain active [9]. At this time, the pitcher may make ground contact with the stride foot before the top of the backswing or the stride foot ground contact may occur after the top of backswing [12].

As the arm moves from 12 o'clock to 6 o'clock, momentum is built, and the pace of the humeral rotation around the glenoid increases [5, 7, 8]. The greatest magnitude of forces begins during this time and carry through to delivery [5]. The pectoralis major and subscapularis show great activity, as the humerus is internally rotated and flexed simultaneously [5]. The serratus anterior is also very active as it works to stabilize the scapula during this portion of the motion [5]. This also is the portion of the motion with the greatest biceps brachii activity [13]. As the rotational forces and speeds of the humerus around the glenoid are greatest during this phase, it is likely a contributor to the common complaint of anterior shoulder pain in softball pitchers [5, 10]. Once foot contact has occurred, the velocities increase as the arm, torso, and pelvis accelerate forward toward delivery of the ball [5]. The gluteus medius must maintain elevation of the pelvis on the opposite side to transfer energy from one foot to the other [5, 11].

The final stage of the windmill pitch is characterized by deceleration as movement from ball release to the completion of the follow-through motion occurs [9]. There is disagreement as to the level of muscle activity at this point. Some say the muscles involved in the upper extremity all decrease their activity level, with the teres minor remaining the most active [9]. Others found that high levels of distraction forces are placed on the shoulder at this phase of release, similar to that seen in baseball pitchers [7].

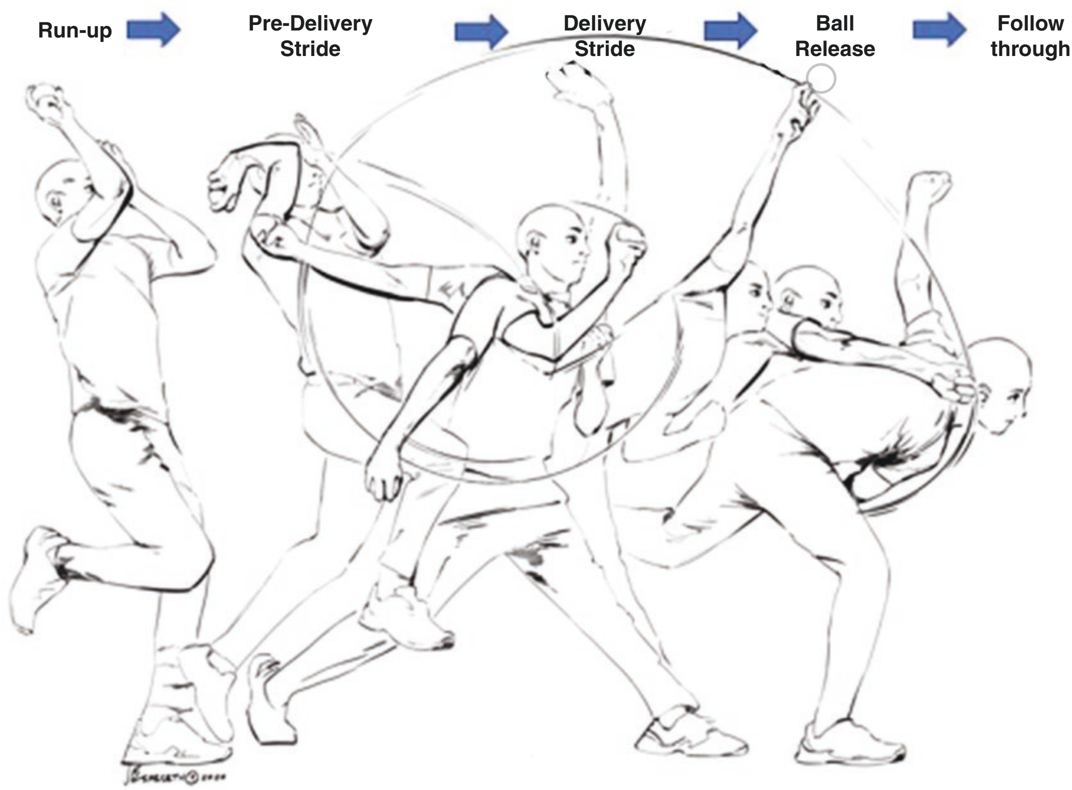

Fig. 2.3 Cricket bowling can be broken down into phases including: run-up, pre-delivery stride, delivery stride, ball release, and follow through. The round house overhead movement maintains a more extended elbow when compared to a baseball pitcher. (© Mark R. Hutchinson, 2021. All Rights Reserved)

The cricket equivalent of pitching is called bowling. There are two major bowling types in cricket, fast, also referred to as "pace," bowling and spin bowling [14]. Players generally specialize in one style or the other. Similar to pitchers in baseball, fast bowlers in cricket are the players most prone to injury [15, 16] which is generally of a non-contact ("overuse") nature. The phases of fast bowling include the run-up, pre-delivery stride, delivery stride (back foot and front foot contact), ball release (inswing/outswing), and the follow through (Fig. 2.3).

In contrast to baseball, the fast bowling motion involves an overhead hurling, not throwing, motion that is characterized by a straight elbow [14]. Consequently, the fast bowling injury profile includes lumbar spine, lower limb, and shoulder injuries, but relatively few elbow injuries [3, 15, 17]. Spin bowlers have fewer injuries than fast bowlers as their run-up is a few slow steps, compared with actual running for fast bowlers, and the ball is delivered with far less momentum [3, 15]. Perhaps because the bowling motion spreads load more evenly around the body and relatively protects the elbow, fast bowlers in cricket have a slightly greater tolerance of match workloads than baseball pitchers [15, 16].

The run-up phase of bowling is defined as the period between the start of the bowler's approach run to the moment of take-off for the pre-delivery stride. The pre-delivery stride, also known as the bound in the cricket coaching literature, is defined as the phase between the end of the run-up and the moment of back foot impact at the start of the delivery stride. The delivery stride has typically been defined as the phase between back foot impact and ball release. As the bowler accelerates their arm in this phase, the external rotators are contracted eccentrically in order to decelerate and control the arm; thus, any external shoulder rotation weakness can contribute to impingement syndrome [18]. The greater relative strength of the internal rotators compared with the external rotators results in decreased deceleration of internal rotation resulting in migration of the humeral head. This leads to a decrease in the subacromial space, which in turn can cause impingement of the rotator cuff tendon [19]. The follow-through has no bearing on ball release speed although it may have implications for injury in the case of decelerating too abruptly [20].

With a good bowling action, the shoulder is not subjected to forces that would lead to instability; however, labral tears and superior labral anterior posterior (SLAP) lesions can occur [21]. Overuse injuries can weaken the rotator cuff, allowing increased translational movements of the humeral head to occur, resulting in shoulder pain. With increased translation of the humerus, the long head of the biceps is recruited to help stabilize the joint. This can in turn lead to traction on the long head of the biceps tendon, predisposing the shoulder to tendonitis and SLAP lesions [18]. Additionally, it has been indicated that a combination of contralateral side flexion and ipsilateral axial rotation of the lumbar spine is likely to be instrumental in the development of lumbar bone stress injury and intervertebral disc derangement [17, 22–24].

As with pitching, batting is common and unique to baseball, softball, and cricket. It too can be broken down into phases in order to better understand the mechanics. In baseball and softball, these phases are stance, stride, drive, bat acceleration, and follow-through [4]. Although not exactly the same, batting in cricket employs similar mechanics. During the stance phase, the batter shifts their weight onto the back foot [4]. Foot and trunk positions in this phase is varied among batters. The stance phase ends when the front foot is lifted off the ground [4]. The stride phase is from when the front foot is lifted until it touches down [4]. Trunk axial rotation during the stance and stride phases is minimal, and thus so is the injury risk [4]. The drive phase is from foot contact to maximum bat lag (i.e., minimum angle between the bat's long axis and an imaginary line from the mid chest to hands) [4]. During the drive phase, the pelvis and then the upper trunk rotate to face the batter; the lag between these rotations creates trunk axial rotation and maximum angular acceleration [4]. The time from maximum bat lag to bat–ball contact is the bat acceleration phase [4]. During this phase, the pelvis and the upper trunk rotate to face the pitcher [4]. Trunk axial rotation decreases as the upper trunk rotated above the pelvis [4]. Trunk axial rotation is minimal near the time of bat–ball contact [4]. After bat–ball contact, trunk axial rotation increases during the follow-through phase [4]. Maximum trunk axial rotation occurs during follow-through [4]. Trunk axial acceleration peaks again during the follow-through phase [4]. The forceful rotation involved in batting leads to a large amount of torque being placed on the trunk, moreover due to the asymmetric nature of batting, a player may develop asymmetric trunk strength or flexibility [25]. This asymmetry leads to a predisposition of the side contralateral to the dominant arm to abdominal strains [26]. In cricket, improper batting techniques as well as inappropriate equipment, such as bats that are too heavy, put unnecessary strain on the elbow, causing tennis elbow or lateral epicondylitis [19].

Also common to all three sports is running between bases or, in the case of cricket, ends. In baseball, this is the top activity associated with a hamstring strain in both the major and minor leagues [27]. The majority of base-running hamstring strains in both leagues were associated with running to first base [27]. Biomechanical studies of sprint mechanics suggest that the posterior thigh is most susceptible to such strains near the end of the swing phase, when the hamstrings reach maximal length and undergo eccentric contraction just before heel strike [28–30]. The acceleration from a stationary, but rotating swinging action required for running to first base, often with no planned deceleration leading into the base, is likely the most complex and frequent acceleration baseball activity compared with running other bases or fielding, catching, or pitching [27].

Abdominal core muscle injuries are also common in baseball, softball, and cricket due to the importance of these muscles in both stabilization and generation of power in batting and pitching/bowling. The core muscles, which include the thoracolumbar, abdominal, pelvic, and hip musculature, are where power is transferred from lower to upper extremities [25, 26, 31]. Activities such as throwing and swinging are unique because they place a dual demand on the core muscles through both trunk stabilization and the creation of side-specific axial torque [25, 26, 31]. Injuries to core musculature typically occur on the contralateral side to the dominant arm or batting side [25, 26, 31].

Fig. 2.4 The cocking/acceleration phase of throwing places significant tension loads over the medial elbow and compression loads over the lateral elbow. (© Mark R. Hutchinson, 2021. All Rights Reserved)

2.3 Mechanisms of Injuries Common and Unique to Baseball, Softball, and Cricket

Perhaps the most infamous injury in baseball is the ulnar collateral ligament (UCL) injury. Commonly known as the "Tommy John" ligament, the UCL, and more specifically its anterior oblique bundle, is the primary stabilizer to valgus stress on the medial elbow [32]. This stress occurs during the late cocking and acceleration phases of the overhead throwing motion (Fig. 2.4). The anterior bundle of the UCL is subject to high tensile stress during the acceleration phase of throwing that may eventually lead to ligament attenuation or failure [32]. High compression loads on the lateral aspect of the elbow secondary to valgus extension overload can lead to radial head hypertrophy and osteochondritis dissecans of the capitellum (Fig. 2.5). Ulnar collateral ligament insufficiency is found most commonly in baseball pitchers, but it may be seen in other overhead throwing athletes as well [32]. A complete injury of the UCL will likely require surgical reconstruction and a 12- to 18-month rehabilitation to be able to return to play. Partial injuries may be treated rest or platelet-rich plasma injections but have an elevated risk of progression to complete tears over time.

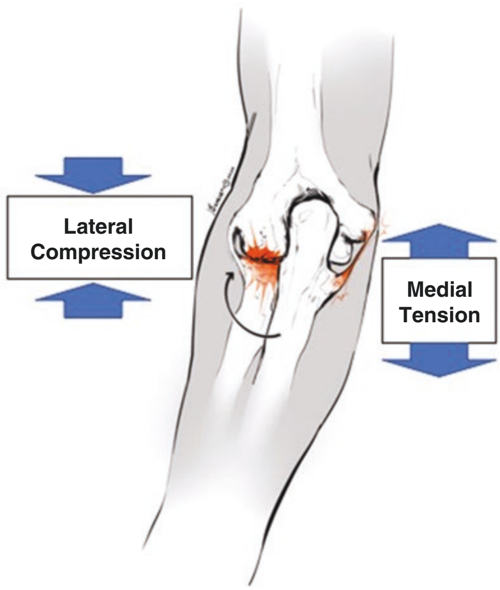

Fig. 2.5 Valgus extension overload of the elbow create tension loads medial potential injuring the UCL, ulnar nerve, or flexor muscle attachment. It also creates compression loads on the lateral aspect of the elbow potential leading to cartilage injury or osteochondritis dissecans of the capitellum. (© Mark R. Hutchinson, 2021. All Rights Reserved)

Injury to the medial flexor-pronator tendon mass of the forearm also occur commonly in baseball, and due to their location of attachment near the UCL, must be distinguished from UCL injuries [33]. These flexor muscles provide dynamic support to the UCL in the stabilization of the elbow against the significant valgus stress experienced in pitching. The fibers of the flexor carpi ulnaris are intimately attached to the origin of the medial epicondyle and optimally positioned to provide support directly in line with the UCL [33]. The contractile forces of this muscle group resist valgus stress during early arm acceleration and help facilitate wrist flexion during ball release [33]. As with UCL injuries, the high amount of tensile forces seen in the elbow during pitching can lead to injury of the medial flexor-pronator tendon mass of the forearm. In the skeletally immature thrower, this may present as an avulsion of the medial epicondyle.

The muscles of the rotator cuff must stabilize the humeral head within the glenoid by offsetting the high-energy forces imparted on the shoulder during pitching. This action places tremendous tensile stresses on the rotator cuff, especially during the deceleration phase of throwing when the rotator cuff muscles must resist distraction, horizontal adduction, and internal rotation [34]. Repetitive performance of the throwing motion, as occurs with pitching, leads to fatigue of the cuff muscles and tensile failure and microtrauma over time, usually involving the undersurface of the posterior half of the supraspinatus and superior half of the infraspinatus [34]. With repeated injury, articular-sided partial-thickness tears and intratendinous tears can develop in the rotator cuff. If any underlying instability exists in the pitcher's shoulder, the risk of injury to the rotator cuff is higher, as the cuff must work harder to stabilize the glenohumeral joint [34]. In addition to these tensile mechanisms of failure, the cuff may also be subjected to compressive loads. In particular, internal impingement, the impingement of the posterior supraspinatus and superior infraspinatus against the posterior superior glenoid rim and labrum, has been recognized as a cause of rotator cuff tendinopathy [34]. Baseball pitchers are thus constantly subjecting the rotator cuff to supraphysiologic loads that commonly lead to rotator cuff tendinitis, partial-thickness rotator cuff tears, and possibly, full-thickness rotator cuff tears [34]. The primary focus of treatment of rotator cuff pathology in throwers must be prevention since the return to play after full-thickness rotator cuff repairs in notoriously poor.

Glenoid labrum tears are named based on the location of the tear within the labrum. Superior labral anterior-posterior tears were first described in 1985 and were later classified and coined "SLAP" tears [35]. During the cocking phase of throwing where the abducted arm is externally rotated, a posterior shear force is created at the superior labrum due to tension of the long head of the biceps [35]. It is believed that SLAP tears may occur due to this shear force which occurs as part of a "peel-back" mechanism [35]. The arm internally rotates during the acceleration phase, and biceps force is produced to both resist shoulder distraction and decelerate elbow extension [35]. The biceps-labrum complex must be able to withstand this tensile force, which is produced by the biceps tendon near the time of ball release and can lead to SLAP tear. It has also been proposed that the typical mechanism of SLAP tear may be a combination of both the "peel-back" mechanism during arm cocking and the tension created at ball release [35]. In elite level baseball pitchers, the outcomes and return to play of surgical interventions to treat a SLAP tear such as SLAP repair or biceps tenodesis have been less than satisfactory leading once again on the emphasis on early recognition of risk and prevention.

Although injuries to the superior and anterior labrum have historically received more attention, posterior labral injury and posterior instability may also occur [36, 37]. Injuries to the posterior labrum in the baseball population commonly result from throwing, diving on the outstretched arm, or posterior subluxation of the lead shoulder during batting (the so-called batter's shoulder) [36, 37]. The high rotational velocities involved in batting, coupled with the weight of the bat, leads to a tremendous amount of force at the shoulders. Repetitive exposure to these forces can result in batter's shoulder [37]. Although posterior labral tears can occur from an acute event, onset is commonly more insidious and related to repetitive microtrauma and capsular contracture, which can lead to failure of the posterior capsulolabral structures [36].

The role of abdominal core muscles in trunk rotation in baseball-specific activities of hitting and pitching has been well studied [26]. In pitching, side-specific activation of the oblique and rectus abdominis muscles is seen throughout all phases of a pitch, with the muscles on the lead side, contralateral to the dominant arm, showing greater activation than the ipsilateral, or trailing-side, muscles [26]. The greatest side-to-side increase in activation of any muscle group was seen in the oblique muscles during the cocking phase. In batters, both the leading and trailing abdominal obliques demonstrated maximal activity throughout the swing, suggesting that they have an important role in trunk stabilization during batting [26]. This maximal activation of the oblique muscles indicates that they play a significant role in swinging, along with the other trunk muscles [26]. The high levels of muscle activation, coupled with the power generation and repetition involved in pitching and batting, is what eventually leads to injury to the core muscles.

As in baseball, many of the injuries common in softball are related to pitching. During the windmill pitch used in softball, there is a much higher degree of biceps brachii activation than is seen in the overhead technique used in baseball [38]. The highest biceps brachii activity is appreciated when the ball moves from the 9 o'clock position until its release, which occurs to control elbow extension and to resist the great distraction forces experienced during windmill pitching [38]. This high level of muscle activation puts great stress on the biceps tendon, which can lead to injury.

In addition to the injuries to the biceps tendon, injuries to the glenoid labrum also occur frequently since the biceps tendon is attached to the superior labrum. When the biceps contract to control elbow extension and resist distractive forces on the shoulder, there is a stress placed on the labrum at this attachment site. Additionally, as mentioned before the amount of biceps activity during a softball pitch is greater than that seen in baseball [38]. This demand on the biceps-labrum complex to both resist glenohumeral distraction and produce elbow flexion torque makes this structure susceptible to overuse injury.

The repetitive motion involved in the windmill softball pitch can cause inflammation and pain resulting in rotator cuff tendinitis much in the same way as baseball [38]. The forceful deceleration of the arm immediately prior to or at the time of ball release puts great stress on the rotator cuff, particularly the infraspinatus and teres minor [38]. Repeating this motion many times over the course of a game/practice can lead to rotator muscle fatigue and eventually tendinopathy or a full tear of the rotator cuff.

The forceful pronation demonstrated in the softball windmill pitch causes a great load to be placed on the mid-diaphysis of the ulna [38]. Fatigue fracture of the ulna occurs when the forearm is stressed during full supination with the elbow flexed to nearly a right angle and the weight is taken approximately at right angles to the ulnar shaft [38]. Repeated stress in this manner, which is what occurs in a softball pitch, can lead to fracture. Pain in the ulna is localized and presents as a dull ache after practices and games [38]. The pain always resolves with rest but commonly reoccurs with pitching activity [38]. Treatment is an appropriate period of rest followed by a gradually progressive return to play.

Although the valgus stress experienced at the elbow in softball pitchers is less than that seen in baseball pitchers, there is still the risk for UCL injury. However, more common in softball than baseball is irritation of the ulnar nerve causing ulnar neuritis [39]. There are two theories as to why this occurs. One posits that the stresses placed at the medial elbow during pitching cause the ulnar neuritis [39]. The other speculates that this occurs because of contact of the medial elbow with the ipsilateral lateral thigh nearly simultaneous to the point of release of the pitched ball to decelerate the arm [39]. It is also possible that a combination of these factors is what plays a role in the development of ulnar neuritis.

In cricket, the most commonly injured player is the bowler, with the vast majority of these injuries being due to overuse [19, 20]. With a good bowling action, the shoulder is not subjected to the same type of forces seen in baseball and softball that would lead to instability. However, labral tears and superior labral anterior posterior (SLAP) lesions can occur [19, 20]. The overuse that occurs during repetitive bowling can weaken the rotator cuff, allowing increased translational movements of the humeral head to occur, resulting in shoulder pain. With increased translation of the humerus, the long head of the biceps is recruited to help stabilize the joint [19–21]. This can in turn lead to traction on the long head of the biceps tendon, predisposing the shoulder to tendonitis and SLAP lesions. When utilizing proper form, the arm does not get into a position of apprehension (abduction and forced external rotation) during bowling and as such is not subject to forces on the capsular ligaments that threaten instability [19–21]. Shoulder injuries in cricketers tend to result from throwing, which is done by fielders, but can be aggravated by bowling.

During the bowling action's acceleration phase, the external shoulder rotators are contracted eccentrically in order to decelerate and control the arm and any external shoulder rotation weakness could contribute to impingement syndrome [21]. The greater relative strength of the internal shoulder rotators compared with the external rotators, would indicate a decreased deceleration of internal rotation resulting in migration of the humeral head and thus a decrease in the subacromial space causing impingement of the rotator cuff tendon [21]. Over time this impingement may lead to weakness or tears within the tendon.

Injuries to the lateral trunk muscles occur frequently in bowlers and are relatively unique to the position [22]. Injury typically occurs on the bowler's nondominant side as a result of the contralateral arm being pulled down from a position of maximum elevation with some lateral trunk flexion during the final delivery action [22]. The tips of the lowest ribs can enlarge and rub against the pelvis during the delivery stride, or the soft tissue can get pinched between the two structures [22]. In some instances, the bone and cartilage tips of the lower ribs can even break off [22]. Occasionally, these injuries are true "side strains," in which the muscle between the ribs tears. In all cases, pain occurs in roughly the mid-axillary line over one or more of the lowest four ribs [22].

Fast bowlers also have a high incidence of serious lumbar spine injuries, such as lesions in the pars interarticularis [17, 19]. Pars lesions occur most often at the L4 and L5 levels and present as symptomatic unilateral lesions on the non-bowling side (in right-arm bowlers the stress lesions occur through the left L4 pars) [17, 19]. These injuries develop as a result of asymmetric loading of the lumbar spine that is associated with specific motions such as lateral flexion and/or axial rotation of the trunk that occur during fast bowling [17, 19]. This predisposes the L4 pars on the side contralateral to the dominant bowling arm to injury in bowlers. Spondylolysis, which is a stress fracture occurring at the pars interarticularis, is a result of continued stress to the pars interarticularis [17, 23]. The repetitive lateral flexion of the trunk involved in bowling, combined with the hyperlordotic posture during the delivery action, further increases the possibility of developing a stress fracture in the lumbar region [17, 23]. Even in athletes with adequate abdominal and paraspinal muscle strength, injuries may still occur as a result of the repetitive action of adopting a hyperlordotic posture when delivering the ball [17, 23].

2.4 Epidemiology of Injuries in Baseball, Softball, and Cricket

Every year, approximately three million children play baseball in the United States [40]. Many of these kids continue to play through their adolescent and high school years. Approximately 25,000 athletes compete each year in National Collegiate Association of America (NCAA) baseball at the collegiate level [40]. A select few of these end up playing minor or major league baseball. Five percent of youth pitchers will suffer a serious elbow or shoulder injury within 10 years, and pitching volume is the strongest known predictor of injury [40]. More than 43% of pitchers who suffered such an injury pitched on consecutive days, 31% pitched on multiple teams with overlapping seasons, and 19% pitched multiple games per day, resulting in an increased risk of pitching-related arm pain [40]. Arm pain related to pitching is indicative of overuse, and these pitchers are likely at increased risk of real injury to the elbow and shoulder [40].

Injuries in high school baseball players occur at approximately 1 injury per 1000 practices or competitions [40]. Shoulder injuries occur around 1.39–1.72 per 10,000 athlete-exposures (AEs), with muscle strains being most common [40] (Fig. 2.6). Juniors and seniors sustain a majority (69%) of shoulder injuries compared to freshmen and sophomores, as they are participating in a higher level of competition and playing in more games [40]. Pitchers account for the largest proportion of shoulder injuries (38%) [40]. Approximately 10% of shoulder injuries require surgery and three-quarters of them are among pitchers [40]. Elbow injuries are less frequent at 0.86 per 10,000 AEs and most often involve ligamentous sprains (42.7%) [40]. The rate of elbow operations in young athletes has significantly risen over the last couple of decades with approximately 13% of ulnar collateral ligament reconstructions being performed on high school players [40]. Fourteen percent of high school baseball injuries result in time loss from participation (sitting out greater than 1 day) [40]. Although most athletes get back to playing in less than a week, approximately 10% will miss an entire season due to their injury [40]. Catastrophic injuries such as commotio cordis, a lethal arrhythmia caused by blunt trauma to the chest, and traumatic brain are fortunately very rare in the sport of baseball. Only 128 cases of commotio cordis reported in the United States over a 20-year period, and of the major high school sports, baseball has the lowest risk of traumatic brain injury at 0.05 per 1000 AEs [40].

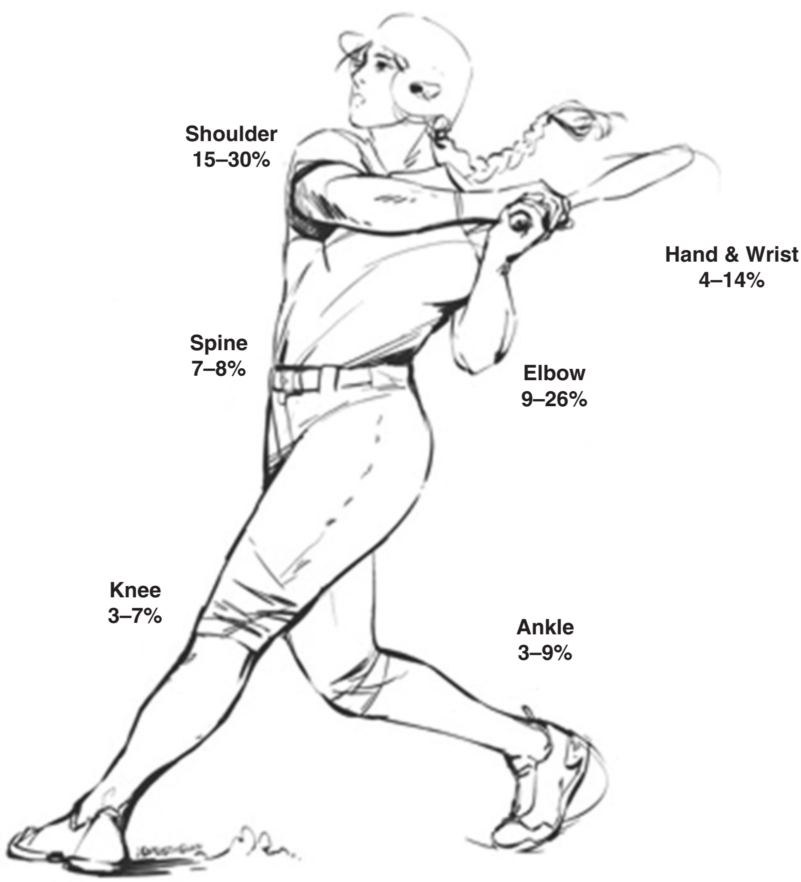

Fig. 2.6 Injuries in baseball by anatomic site. (© Mark R. Hutchinson, 2021. All Rights Reserved)

In college, the overall incidence of injuries is 5.83 per 1000 AEs [40]. It was found that upper extremity injuries accounted for 58% of all injuries [40]. Twenty-five percent of all injuries were considered severe, resulting in 21 or more days of lost participation [40]. The most common injuries were characterized as strains (23%), sprains (19%), and contusions (17%) [40]. The most common player positions to incur injury were base runners (23.7%), pitchers (20.9%), and batters (15.3%) [40]. As expected, upper extremity injuries accounted for the majority of reported injuries. The majority of injuries incurred during games were the result of contact, whereas 63.9% of practice injuries were non-contact [40]. Although rare, clinicians must also be aware of the potential for fatal and non-fatal catastrophic injuries in collegiate baseball. The reported catastrophic injury rate to be 1.7 per 100,000 baseball players and the fatal catastrophic injury rate to be 0.86 per 100,000 baseball players during a 21-year period [40].

In professional baseball, the incidence of injury resulting in placement on the disabled list is 3.61 per 1000 AEs [40]. This is rather high considering it does not include injuries that did not require an assignment to the disabled list, which is common for injuries resulting in less than 15 days out of play. At the professional level, injuries to pitchers continue to increase significantly more than other positions with a 34% higher injury rate among pitchers compared to fielders [40]. In total, 39.1% of injuries in Major League Baseball occur in pitchers [41]. Overall, upper extremity injuries account for 39–51.4% of all injuries, while lower extremity injuries account for 30.6% [40, 41]. Injuries to the spine and core musculature account for 11.7%, while other injuries and illnesses form 6.3% of the total disabled list entries [40, 41]. Lower extremity injuries were much more common in fielders than pitchers, with the most common lower extremity injury being hamstring strains [40, 41]. The injuries most likely to end a player's season are UCL injuries (60% season ending) and SLAP tears (50.9% season ending) [41].

In women's collegiate softball, sliding injuries account for 23% of all game injuries, and the injury rate for sliding is 0.89 injuries per 1000 AEs for games [42] (Fig. 2.7). Softball players employ an average of 3.30 feet-first slides per

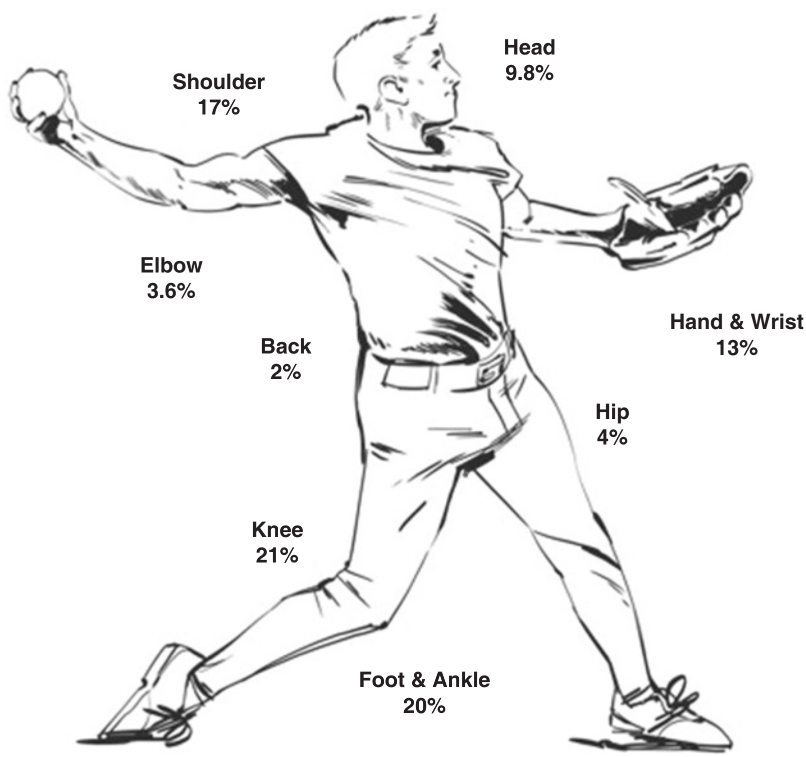

Fig. 2.7 Injuries in softball by anatomic site. (© Mark R. Hutchinson, 2021. All Rights Reserved)

game and 1.34 head-first slides per game [42]. Most softball injuries due to sliding are contusions (33%) and ankle ligament sprains (19%) [42]. Knee internal derangement accounts for 8.7% of game injuries, 31% of which were anterior cruciate ligament (ACL) injuries [42]. Upper extremity injuries represent approximately 33% of injuries of these 27% involve overuse [42]. Concussion rates are estimated to be between 0.14 and 0.26 per 1000 AEs [43].

At the professional level, position players are most likely to injure their lower extremities (55%), while pitchers are equally likely to injure upper and lower extremities [44]. Pitchers are more likely to sustain an injury with an increase in pitch counts [44]. Positional players suffer a majority of their injuries acutely (59%), while the majority of pitching injuries are either overuse, or related to previous injuries/conditions (62%) [44]. The large majority of injuries are minor and do not require a long period away from activity with 70% of all injuries resolving, or at least improving enough to return to action within 1 week of the injury [44].

In cricket, bowling, fielding, and wicket keeping account for most injuries (Fig. 2.8). Acute injuries are most common (64–76%), with the rest being acute-on-chronic (16–22.8%) and chronic

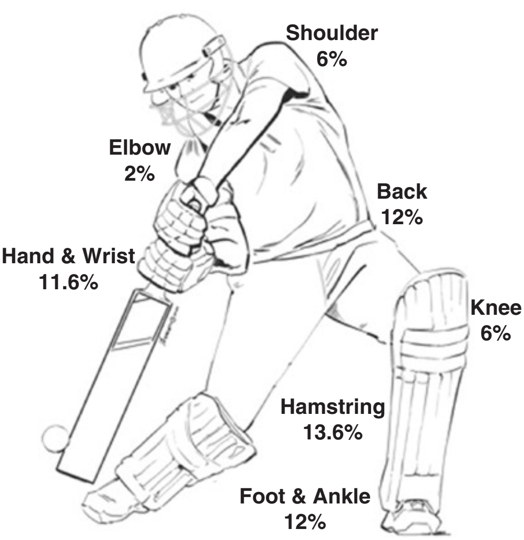

Fig. 2.8 Injuries in cricket by anatomic site. (© Mark R. Hutchinson, 2021. All Rights Reserved)

(8–22%) [45, 46]. Younger players (<24 years old) sustain more overuse and bowling injuries than older players [45]. Lower limb injuries form nearly 49.8% of injuries, followed by back injuries (22.8%), upper limb injuries (23.3%), and neck injuries (4.1%) [45, 46]. Hamstring and quadriceps strains formed the majority of lower limb injuries sustained primarily during bowling and fielding [45]. Injuries to fingers primarily during batting and fielding predominate upper limb injuries (35.4%), and shoulder injuries (21.7%) occurred during throwing and bowling [45, 46].

2.5 Unique Prevention Plans to Avoid and Reduce the Incidence of Injury

As the majority of injuries unique to these sports are a result of overuse, the simplest way to avoid them is to implement mandatory rest periods. Selective rest from inciting activities is critical to a successful outcome, with the amount of rest necessary for recovery varying with the duration of symptoms and severity of injury [47]. The focus of treatment in overuse injuries is to reduce pain, reverse muscle imbalances, restore proper mechanics, and promote tendon healing [47]. Once adequate, pain-free motion and strength have been achieved, a graduated exercise program emphasizing proper mechanics can begin and be progressed [47]. Return to competition is allowed when motion and strength have been restored and the athlete completes the throwing program without further symptoms [47].

An interesting approach to the understanding and management of injuries, especially overuse injuries, utilizes the concept of the kinetic chain, which was originally an engineering concept that has been adapted to human biomechanics. The kinetic chain describes the interrelated groups of body segments, connecting joints, and muscles working together to perform movements [4, 38, 48, 49]. Therefore, the act of throwing a ball or batting all involve the activation of a chain of muscles and joints working together toward one coordinated movement. Using this framework, injury prevention begins with maintenance of the "kinetic chain" that coordinates transmission of force used in a particular action [4, 38, 48, 49]. To help conceptualize this, the kinetic chain may be thought of as a metal chain link, where one weak link leads to failure of the system as a whole. Translated to human movements, one weak muscle or joint can lead to injury.

When analyzing shoulder injuries in particular, the kinetic chain involved coordinates force transmission from the legs and trunk to the upper extremity [50]. Imbalances in this kinetic chain are common in shoulder impingement, rotator cuff tears, and instability [50]. Throwers with labral tears commonly have back inflexibility, reduction in their total arc of glenohumeral motion, infraspinatus and teres minor weakness, and core weakness [50]. Injuries to the foot and ankle, tightness of the muscles crossing the hip and knee, weakness of hip abductors and trunk stabilizers, and conditions altering spine alignment all influence kinetic chain transmission [50]. Utilizing this model, injury prevention should focus not only on rest and strengthening the area of injury itself, but also all the muscles involved in the kinetic chain. Routine screening and re-assessment is key to identify when an in season athlete develops core or flexibility imbalances which in turn indicate early intervention.

Another way to avoid overuse injuries is to train athletes in the proper mechanics of their respective activity in their sport. Improper technique is a likely cause of injury in any athlete. One way to evaluate for this is through a biomechanical video analysis and to undergo training with a certified coach or trainer to correct any mechanical issues that may lead to injury down the road.

References

1. Madden CC, Netter FH. Netter's sports medicine. Amsterdam: Elsevier; 2018.
2. Birchak JC, et al. Softball injuries treated in US EDs, 1994 to 2010. Am J Emerg Med. 2013;31(6):900–5. https://doi.org/10.1016/j.ajem.2013.02.039.
3. Stretch R. Cricket injuries - a longitudinal study of the nature of injuries to South African cricketers. South Afr J Sports Med. 2018;17(3). https://doi.org/10.17159/5122.
4. Fleisig GS, et al. Trunk axial rotation in baseball pitching and batting. Sports Biomech. 2013;12(4):324–33. https://doi.org/10.1080/14763141.2013.838693.
5. Barrentine SW, et al. Biomechanics of windmill softball pitching with implications about injury mechanisms at the shoulder and elbow. J Orthop Sports Phys Ther. 1998;28(6):405–14. https://doi.org/10.2519/jospt.1998.28.6.405.
6. Bogenschutz ED, et al. Midhumerus adaptation in fast-pitch softballers and the effect of throwing mechanics. Med Sci Sports Exerc. 2011;43(9):1698–706. https://doi.org/10.1249/mss.0b013e3182134e4f.
7. Werner SL, et al. Biomechanics of youth windmill softball pitching. Am J Sports Med. 2005;33(4):552–60. https://doi.org/10.1177/0363546504269253.
8. Werner SL, et al. Kinematics and kinetics of elite windmill softball pitching. Am J Sports Med. 2006;34(4):597–603. https://doi.org/10.1177/0363546505281796.
9. Maffet MW, et al. Shoulder muscle firing patterns during the windmill softball pitch. Am J Sports Med. 1997;25(3):369–74. https://doi.org/10.1177/036354659702500317.
10. Lear A, Patel N. Softball pitching and injury. Curr Sports Med Rep. 2016;15(5):336–41. https://doi.org/10.1249/jsr.0000000000000293.
11. Oliver GD, et al. Muscle activation patterns of the upper and lower extremity during the windmill softball pitch. J Strength Cond Res. 2011;25(6):1653–8. https://doi.org/10.1519/jsc.0b013e3181db9d4f.
12. Oliver GD, et al. Kinematic motion of the windmill softball pitch in prepubescent and pubescent girls. J Strength Cond Res. 2010;24(9):2400–7. https://doi.org/10.1519/jsc.0b013e3181dc43af.
13. Rojas IL, et al. Biceps activity during windmill softball pitching. Am J Sports Med. 2009;37(3):558–65. https://doi.org/10.1177/0363546508328105.
14. Orchard JW, et al. Fast bowlers in cricket demonstrate up to 3- to 4-week delay between high workloads and increased risk of injury. Am J Sports Med. 2009;37(6):1186–92. https://doi.org/10.1177/0363546509332430.
15. Orchard J. Injuries in Australian cricket at first class level 1995/1996 to 2000/2001* commentary. Br J Sports Med. 2002;36(4):270–4. https://doi.org/10.1136/bjsm.36.4.270.
16. Orchard JW, et al. Injuries to elite male cricketers in Australia over a 10-year period. J Sci Med Sport. 2006;9(6):459–67. https://doi.org/10.1016/j.jsams.2006.05.001.
17. Foster D, et al. Back injuries to fast bowlers in cricket: a prospective study. Br J Sports Med. 1989;23(3):150–4. https://doi.org/10.1136/bjsm.23.3.150.
18. Hackney RG. Advances in the understanding of throwing injuries of the shoulder. Br J Sports Med. 1996;30(4):282–8. https://doi.org/10.1136/bjsm.30.4.282.
19. Shafi M. Cricket injuries: an orthopaedist's perspective. Orthop Surg. 2014;6(2):90–4. https://doi.org/10.1111/os.12104.

20. Glazier PS, Wheat JS. An integrated approach to the biomechanics and motor control of cricket fast bowling techniques. Sports Med. 2013;44(1):25–36. https://doi.org/10.1007/s40279-013-0098-x.
21. Ranson C, Gregory PL. Shoulder injury in professional cricketers. Phys Ther Sport. 2008;9(1):34–9. https://doi.org/10.1016/j.ptsp.2007.08.001.
22. Ranson CA, et al. The relationship between bowling action classification and three-dimensional lower trunk motion in fast bowlers in cricket. J Sports Sci. 2008;26(3):267–76. https://doi.org/10.1080/02640410701501671.
23. Glazier PS. Is the 'crunch factor' an important consideration in the aetiology of lumbar spine pathology in cricket fast bowlers? Sports Med. 2010;40(10):809–15. https://doi.org/10.2165/11536590-000000000-00000.
24. Stuelcken MC, et al. Three-dimensional trunk kinematics and low back pain in elite female fast bowlers. J Appl Biomech. 2010;26(1):52–61. https://doi.org/10.1123/jab.26.1.52.
25. Watkins RG, et al. Dynamic EMG analysis of torque transfer in professional baseball pitchers. Spine. 1989;14(4):404–8. https://doi.org/10.1097/00007632-198904000-00011.
26. Conte SA, et al. Abdominal muscle strains in professional baseball. Am J Sports Med. 2012;40(3):650–6. https://doi.org/10.1177/0363546511433030.
27. Ahmad CS, et al. Major and minor league baseball hamstring injuries. Am J Sports Med. 2014;42(6):1464–70. https://doi.org/10.1177/0363546514529083.
28. Heiderscheit BC, et al. Identifying the time of occurrence of a hamstring strain injury during treadmill running: a case study. Clin Biomech. 2005;20(10):1072–8. https://doi.org/10.1016/j.clinbiomech.2005.07.005.
29. Schache AG, et al. Biomechanical response to hamstring muscle strain injury. Gait Posture. 2009;29(2):332–8. https://doi.org/10.1016/j.gaitpost.2008.10.054.
30. Yu B, et al. Hamstring muscle kinematics and activation during overground sprinting. J Biomech. 2008;41(15):3121–6. https://doi.org/10.1016/j.jbiomech.2008.09.005.
31. Fleisig GS, et al. Kinetics of baseball pitching with implications about injury mechanisms. Am J Sports Med. 1995;23(2):233–9. https://doi.org/10.1177/036354659502300218.
32. Cantu RC. Ulnar collateral ligament reconstruction in major league baseball pitchers. Yearbook Sports Med. 2008;2008:42–3. https://doi.org/10.1016/s0162-0908(08)79110-4.
33. Hodgins JL, et al. Forearm flexor injuries among major league baseball players: epidemiology, performance, and associated injuries. Am J Sports Med. 2018;46(9):2154–60. https://doi.org/10.1177/0363546518778252.
34. Morrey BF. Repair of full-thickness rotator cuff tears in professional baseball players. Yearbook Orthoped. 2008;2008:211–2. https://doi.org/10.1016/s0276-1092(08)79199-5.
35. Gilliam BD, et al. Return to play and outcomes in baseball players after superior labral anterior-posterior repairs. Am J Sports Med. 2017;46(1):109–15. https://doi.org/10.1177/0363546517728256.
36. Kercher JS, et al. Posterior labral repairs of the shoulder among baseball players: results and outcomes with minimum 2-year follow-up. Am J Sports Med. 2019;47(7):1687–93. https://doi.org/10.1177/0363546519843070.
37. Kang RW, et al. Posterior instability caused by Batter's shoulder. Clin Sports Med. 2013;32(4):797–802. https://doi.org/10.1016/j.csm.2013.07.012.
38. Briskin SM. Injuries and medical issues in softball. Curr Sports Med Rep. 2012;11(5):265–71. https://doi.org/10.1249/jsr.0b013e3182699489.
39. Smith AM, et al. Ulnar neuropathy and medial elbow pain in women's fastpitch softball pitchers: a report of 6 cases. J Shoulder Elb Surg. 2017;26(12):2220–5. https://doi.org/10.1016/j.jse.2017.08.013.
40. Melugin HP, et al. Injury prevention in baseball: from youth to the pros. Curr Rev Musculoskelet Med. 2018;11(1):26–34. https://doi.org/10.1007/s12178-018-9456-5.
41. Posner M, et al. Epidemiology of major league baseball injuries. Am J Sports Med. 2011;39(8):1675–91. https://doi.org/10.1177/0363546511411700.
42. Morrey BF. Descriptive epidemiology of collegiate women's softball injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. Yearbook Orthoped. 2008;2008:152–3. https://doi.org/10.1016/s0276-1092(08)79204-6.
43. Cusimano MD, Zhu A. Systematic review of traumatic brain injuries in baseball and softball: a framework for prevention. Front Neurol. 2017;8:492. https://doi.org/10.3389/fneur.2017.00492.
44. Patel N, et al. Professional women's softball injuries. Clin J Sport Med. 2019; https://doi.org/10.1097/jsm.0000000000000698.
45. Orchard JW, et al. Incidence and prevalence of elite male cricket injuries using updated consensus definitions. Open Access J Sports Med. 2016;7:187–94. https://doi.org/10.2147/oajsm.s117497.
46. Pardiwala DN, et al. Injuries in cricket. Sports Health. 2017;10(3):217–22. https://doi.org/10.1177/1941738117732318.
47. Rizio L, Uribe JW. Overuse injuries of the upper extremity in baseball. Clin Sports Med. 2001;20(3):453–68. https://doi.org/10.1016/s0278-5919(05)70262-3.
48. Camp CL, et al. Summative report on time out of play for major and minor league baseball: an analysis of 49,955 injuries from 2011 through 2016. Am J Sports Med. 2018;46(7):1727–32. https://doi.org/10.1177/0363546518765158.
49. Wang Q. Baseball and softball injuries. Curr Sports Med Rep. 2006;5(3):115–9. https://doi.org/10.1097/01.csmr.0000306299.95448.cd.
50. Greiwe RM, Ahmad CS. Management of the throwing shoulder: cuff, labrum and internal impingement. Orthop Clin N Am. 2010;41(3):309–23. https://doi.org/10.1016/j.ocl.2010.03.001.

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3 Basketball

Sport-Specific Injuries and Unique Mechanisms in Basketball

Kevin Machino, Marshall Haden, and Ankur Verma

3.1 Introduction

Basketball is the only major sport strictly of United States origin. The sport was first introduced by Dr. James Naismith in 1891 at the International Young Men's Christian Association (YMCA) Training School in Springfield, Massachusetts [1]. The first game was played using a soccer style ball and two peach baskets, which gave the sport its name. Basketball has since evolved to become one of the most popular sports played worldwide, competitively and recreationally, with over 450 million people playing the sport globally [2]. The original rules of basketball were designed to be a noncontact sport. Today, it has evolved into an increasingly high-speed, physical sport, which has led to more opportunities for injuries. Basketball has one of the highest injury risks in team sports with injury rates up to ten injuries per 1000 athletic exposures [3].

The unique body mechanics involved when playing basketball result in injury patterns specific to this sport. The joints and muscles most frequently utilized become the most susceptible to injury. The lower limbs are most commonly injured with ankle and knee injuries being the most prevalent [4]. Players are constantly performing complex lower body movements including running with sudden acceleration or deceleration, abrupt multidirectional changes, and repetitive jumps. This leads to ankle sprains, ligamentous injuries at the knee, and muscle strains. Players are also susceptible to upper limb injuries as they rely on upper body mechanics for dribbling, passing, and shooting. This can lead to mallet finger or wrist sprains.

This chapter will focus on the specific movements required by an athlete throughout the course of a basketball game that differentiate the mechanism of injuries and risk factors from other sports. The most common injuries, incidence and prevalence, and injury prevention strategies will also be covered. This section will conclude with a discussion on the body mechanics, common injury patterns, and special clinical considerations in wheelchair basketball athletes.

3.2 Unique Mechanics and Injury Risks in Basketball

Basketball requires an athlete to perform frequent high-intensity movements including jumping, sprinting, accelerations, decelerations, and quick multidirectional changes [5]. Players repeatedly shoot, rebound, defend, dribble, and run throughout the course of the game. The specific body mechanics involved in these actions primarily involve the lower extremities. Therefore, the

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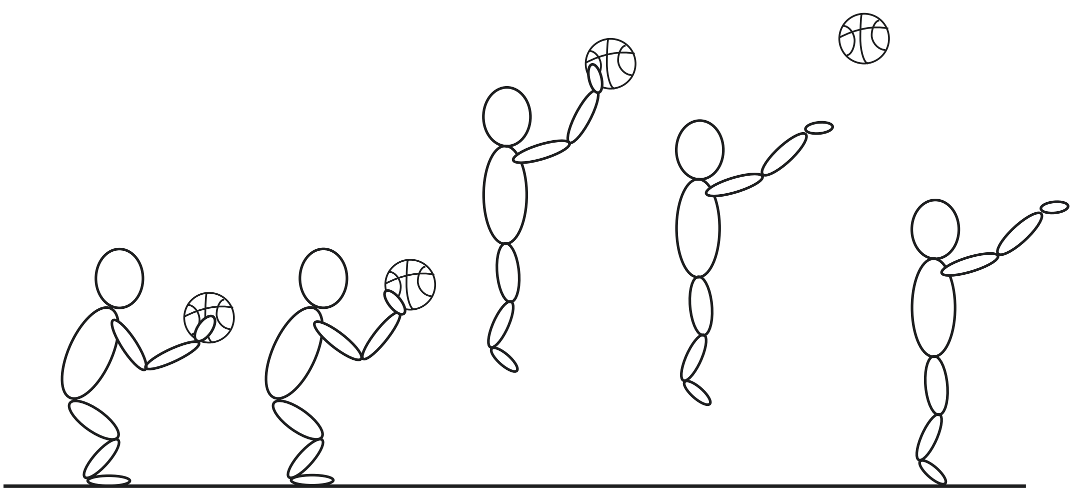
muscles, tendons, ligaments, and joints of the lower extremities are vulnerable to injury and more commonly affected compared to the upper extremities. Due to common basketball maneuvers generating excessive force, the majority of injuries in basketball are related to overuse injuries that manifest as sprains, strains, tendinitis, and stress fractures [6].

A jump shot can be divided into five distinct phases (Fig. 3.1): (1) jump preparation, (2) ball elevation with impulse for the jump, (3) ball stability with upward flight, (4) ball release with descending flight, and (5) follow through with landing [7, 8]. The two most injury-prone moments during the phases of shooting occurs during the take-off and landing. The take-off occurs between phases 2 and 3. The landing occurs between phases 4 and 5. Ground reaction forces (GRFs) reflect the duration and intensity of stress the body endures during contact with the ground. The vertical GRF generated during the take-off can be up to three times the body weight, whereas the GRF during landing can be up to five to six times the body weight [6, 9]. Landing can be classified as stiff or soft landing. A stiff landing is characterized by a more extended final knee position. A soft landing favors knee flexion for the final position [10]. The combination of high impact ratios and landing forces seen in stiff landing may lead to excessive load on the lower limbs which can exceed the body weight, resulting in overload injuries of the muscles, tendons, and ligaments. To prevent such impact injuries, it is important for the player to absorb the shock by landing with flexed lower limbs and avoid extended legs [9]. The primary mechanism by which the body attenuates the shock during landing is distal to proximal, starting with ankle dorsiflexion, followed by knee flexion, and lastly hip flexion [11].

Overall, the ankle is the most injury-prone part of the body during basketball. The ankle is most susceptible to injury because the lateral ankle ligament becomes more relaxed during movements [12]. An ankle injury can occur when twisting and turning during cutting and changing directions. More commonly, there are many instances in a basketball game when a player is airborne, increasing the risk of sustaining an ankle injury during the landing. This can be seen when a player shoots a basketball, soars for a layup or dunk, jumps for a rebound, or attempts



Phase 1	Phase 2	Phase 3	Phase 4	Phase 5
	Ball elevation	Ball stability	Ball release	Follow through
Jump preparation	Impulse for jump	Upward flight	Descending flight	Landing

Fig. 3.1 Five phases of a jump shot

to block an opponent's shot. Any incorrect footing on the landing can lead to an ankle ligament injury. The athlete can land on another player's foot or have an off-balance contact with the court surface. There is high risk of lateral ankle injury due to the vulnerable position of the ankle during the descent and landing. The ankle is in a plantarflexed position with an applied inversion load to the foot [13]. Therefore, a common mechanism of an ankle sprain is due to an applied inversion stress during ankle plantarflexion. This can lead to damage to the anterior talofibular ligament (ATFL) and calcaneofibular ligament (CFL) [14]. A sudden inversion and internal rotation with minimal plantarflexion tend to result in ATFL and CFL sprains, whereas a sudden inversion in minimal plantarflexion without internal rotation tend to result in isolated CFL injury [15].

Abrupt jump stops or pivoting on a planted foot when dribbling and driving as well as rapid deceleration or sudden stop-and-go when running and cutting can lead to knee injuries. The two most common knee injuries from such movements are patellofemoral pain syndrome and anterior cruciate ligament (ACL) injuries. Both injuries more commonly occur during noncontact situations. When a player performs these actions on the court, there are certain high-risk knee positions that result in injury. When there is an increased knee valgus moment coupled with small flexion angles at the knee, the medial collateral ligament, medial patellofemoral ligament, and ACL are trying to prevent excess knee movement towards midline and therefore all three ligaments become strained and susceptible to damage [16, 17]. Another possible site of injury during similar dynamic movements is the Achilles tendon. Achilles tendon injuries occur when large forces are translated through an improperly aligned tendon with a dorsiflexed foot and extended knee [18]. This motion can be seen when basketball players attempt to abruptly take an explosive first step to move or dribble past an opponent. This forceful eccentric contraction places significant stress on the tendons, placing these athletes at increased risk for Achilles tendon injuries. It is possible that players recovering from Achilles injuries experience quantifiable changes in their ability to score layups and dunks, basketball actions that require lower-body explosiveness [19].

Although less common, the upper extremities may also be susceptible to injury when passing, catching, shooting, and defending. Hand and arm injuries are more common than shoulder or elbow injuries [20]. Basketball players are particularly at increased risk for injuries to the digits. They are susceptible to proximal interphalangeal and metacarpophalangeal joint sprains and dislocations as well as mallet finger resulting from direct axial load from the ball to the fingers [21]. A mallet finger can occur when an actively extended distal interphalangeal (DIP) joint sustains forced flexion, disrupting the extensor mechanism at the DIP. This injury is most commonly sustained when a ball jams the player's fingertip when defending or catching a pass [20, 22].

3.3 Mechanism of Common Basketball Injuries

3.3.1 Ankle Ligament Sprains

Ankle ligament injuries are the most commonly encountered injuries in basketball, accounting for anywhere from 14.7% to 35.9% of all injuries [23–27]. The vast majority of these injuries involve the lateral ankle ligament complex (80–90%), with the remainder involving the syndesmosis (5–10%) or the deltoid ligament complex (5–10%) [28, 29]. The lateral, syndesmotic, and deltoid ligaments are most commonly injured by inversion, external rotation, and eversion stresses, respectively. Due to the articular congruity of the ankle joint, an axially loaded ankle has inherent rotational and translational stability [30]. Given this, the ankle ligaments are primarily subject to injury only when loading or unloading the ankle. This commonly occurs with jumping, cutting, or pivoting, all of which occur frequently when playing basketball. There are multiple mechanisms of ankle sprain in basketball. Injury often occurs when landing from a jump, such as with rebounding (30–34% of injuries) or shooting (10–11% of injuries). The remainder of ankle sprains occur from cutting or twisting with activities such as dribbling, defending, competing for a loose ball, or any quick change of direction in general gameplay. Fifty to 57% of ankle injuries involve contact with another player. This can either be from landing/stepping on another player, or another player landing/stepping on the foot or ankle, causing injury [28]. Given the large volume of motions which place the ankle at risk, it is no surprise that ankle sprains are the most commonly encountered injury in basketball.

3.3.2 Knee Sprains

Perhaps the most debilitating injuries encountered in basketball are those to the knee. Jumping, cutting, and pivoting motions can cause high stress across the knee joint and potential injury to the soft tissue structures. Injuries can involve the anterior or posterior cruciate ligaments, medial or lateral collateral ligaments, medial or lateral meniscus, or a combination of these structures. The most frequently encountered of these is an injury to the anterior cruciate ligament (ACL). Even though knee sprains are relatively infrequent compared to other injuries, they cause a disproportionate amount of time lost from sport. For instance, one study demonstrated knee sprains account for 6–7% of all basketball injuries, but 18–21% of all injuries requiring >10 days off from sport [31]. Another study of high school females noted that of all injuries requiring surgery, 47% were injuries of the ACL [25]. The ACL is responsible for preventing anterior translation of the tibia relative to the femur. It is susceptible to both noncontact and contact injuries, with the majority being noncontact [31]. Noncontact injuries generally occur when landing from a jump or with rapid deceleration and/or pivoting to quickly change direction. ACL rupture most often occurs when landing or cutting with the foot planted, knee in extension and valgus, and tibia externally rotated. This subjects the knee to anterior translational and valgus forces, causing ACL rupture and buckling of the knee [32]. In contact injuries, the knee is forced into hyperextension and/or valgus during collision with another player, causing rupture of the ACL.

It has been well established that females are at higher risk of sustaining ACL injuries than male athletes in the same sport. Extensive study has been completed into various possible explanations for this discrepancy, such as BMI, general ligamentous laxity, Q-angle, notch width/ACL size, posterior tibial slope, hormonal differences, and neuromuscular/biomechanical differences. While many of these factors may play some role in risk of ACL injury, the most consistently demonstrated risk factor for ACL injury is poor neuromuscular control and subsequent poor biomechanics [32].

3.3.3 Contusions

While the original rules of basketball prohibit player contact, over time basketball has become an increasingly physical sport. Even though forcible contact between opposing players constitutes a foul, this still occurs frequently throughout gameplay. High energy contact, particularly from bony prominences such as knees or elbows, can cause muscle or soft tissue contusions. In addition to player contact, players can also suffer contusions from contact with the playing surface, such as from an uncontrolled fall, or from other objects outside the boundaries of play. While the boundaries of play are standardized, there is significant variation in the layout of the area outside the boundary. In many cases there may be spectators, photographers, chairs, scorekeeping equipment, or other equipment nearby the playing surface. If a player leaves the boundaries of the court while attempting to keep a ball in play, they can collide with these people or objects, potentially causing harm. A minority of contusions are related to such collisions, but awareness of hazards just off the court is critical when discussing basketball injuries. Players can sustain contusions in essentially any body part, but the majority are isolated to the lower extremities, particularly the thigh region [31]. Not all contusions require time off from play, but in cases where pain is severe enough to limit performance, cessation of sport is recommended until symptoms resolve.

3.3.4 Concussion/Head Trauma

Due to the high speed, physical nature of basketball, players are not infrequently subject to head trauma. In recent years this has become a focal point of rule changes given widespread public concern over long-term effects of concussions. However, in addition to concussion, players may also sustain contusions, face or scalp lacerations, facial fractures, or dental injuries secondary to head trauma. Head trauma is often due to player contact. This can either be head to head contact, contact with an elbow when battling for space, or contact with a knee/foot when diving to the floor for a loose ball. Head trauma can also occur from contact with the floor or other equipment. This happens due to uncontrolled falls after shooting or rebounding, or when diving after a loose ball.

3.3.5 Finger/Hand Injuries

Basketball players are subject to a variety of hand and finger injuries during play. These can be simple contusions or sprains, finger dislocations, mallet injuries, or even finger/wrist fractures. These injuries often occur when mishandling the ball while dribbling, passing, or competing for a loose ball. Hand injuries can also occur due to uncontrolled falls when landing on an outstretched hand.

3.3.6 Patellar Tendinopathy

Patellar tendinopathy, or "jumper's knee", is highly prevalent in basketball players. This is a degenerative tendinosis which occurs when increased loading of the tendon leads to mucoid degeneration, most commonly in the posterior portion of the tendon at its insertion on the inferior pole of the patella. Studies have shown 21–32% of elite basketball players have symptoms consistent with patellar tendinopathy [33, 34]. It has also been shown that 33–76% of elite basketball players have some degree of patellar tendon abnormality on ultrasound imaging, suggesting patellar tendinosis is often asymptomatic [35]. Patellar tendon degeneration occurs secondary to repetitive loading of the tendon. Given this, patellar tendinopathy is not generally an acute injury, but rather an overuse injury associated with higher volume of training and competition [36]. While the high frequency of jumping required during basketball gameplay places players at increased risk, there are also multiple intrinsic factors which may predispose to development of patellar tendinopathy. These include overall limb malalignment, patella alta, increased patellar laxity, or muscle tightness [34].

In adolescent basketball players, patellar tendon overload often leads to apophysitis rather than degenerative tendinosis. Osgood-Schlatter syndrome is a traction apophysitis of the insertion of the patellar tendon on the tibial tubercle. Like patellar tendinopathy, patellar tendon overload is a risk factor. However, in rapidly growing adolescent athletes, the point of the extensor mechanism most susceptible to inflammation is the distal insertion of the patellar tendon [37]. Another consideration in pediatric athletes is the risk of tibial tubercle fracture. While the overall incidence of these fractures is rare, basketball is the most common sport. Fractures occur predominantly in adolescent male athletes with an open proximal tibial physis. Proposed mechanisms include strong quadriceps contraction during jumping and eccentric contraction of the quadriceps while landing [38].

3.3.7 Muscle Strains

Due to a high volume of explosive movements, muscle or tendon strain frequently occurs in basketball. Strains generally occur with activities such as jumping, twisting, and rapid changes in speed or direction. Studies have demonstrated strains account for roughly 7.9–21.8% of all reported injuries in basketball [23–25]. The vast majority of these strains do not progress to full-thickness tears; however, they still can cause significant disability requiring time off of sport. Strains most commonly occur in the lower extremities as well as the core musculature. For instance, a study of professional players identified the most commonly occurring muscle strains as lumbar, hamstring, adductor, and gastrocsoleus injuries, respectively [23].

3.3.8 Achilles Tendinopathies/Tears

Due to the high frequency of jumping and rapid acceleration, basketball players are subject to Achilles tendon pathology. This can either be an incomplete strain or a full-thickness rupture. Injury generally occurs due to an eccentric load to the tendon, such as with jumping or initiation of sprinting. The classic example is a middle-aged male "weekend warrior" who feels a pop when jumping or running. This age group is at higher risk secondary to decreased blood flow and increased stiffness in the tendon associated with aging [39]. While this is traditionally thought of as being most common in older athletes, elite basketball players are also susceptible to Achilles rupture. A study of NBA players identified that all Achilles ruptures were noncontact, and most commonly occurred when taking off from a stopped position [40].

3.3.9 Sudden Cardiac Arrest

While exceedingly rare, exertional sudden cardiac arrest is an important injury to discuss in relation to basketball. This can occur in any aerobic sport or activity, but it most commonly occurs in basketball. It has been shown that males are at higher risk than females, and that African-Americans are at higher risk than white players [41]. The most common underlying etiology leading to sudden cardiac arrest is hypertrophic obstructive cardiomyopathy, an inherited structural abnormality leading to left ventricular septal hypertrophy, predisposing to obstruction of flow in states of high cardiac output. Other causes of sudden cardiac arrest include other structural abnormalities of the heart (e.g., anomalous coronary arteries, ductal ectasia from Marfan syndrome), arrhythmias (Wolff-Parkinson-White, congenital long QT syndrome), or acquired abnormalities (myocarditis, commotio cordis, drug-related, hypo/hyperthermia) [42].

3.4 Epidemiology of Basketball Injuries

There have been multiple studies done to assess the epidemiology of basketball injuries. Many of these studies focus on a specific level of competition, e.g., high school, collegiate, or professional players. Several also include subset analysis by gender. However, due to inconsistent reporting methods there is notable variation in the frequency of common injuries. For instance, some studies report any complaint requiring assessment by training staff or medical personnel as an injury, whereas other studies only report injuries which require time off from sport to recover. Also, there is variation in how injuries are grouped between studies. Some studies group injury by anatomic location, some group by type (sprain, strain, fracture, etc.) and some give an in-depth report detailing each specific diagnosis. Given this variation, it is difficult to draw formal comparisons of injury frequencies between age and competition levels. However, there are multiple trends which remain consistent throughout multiple studies. In all competition levels the majority of injuries occur in the lower extremities. Ankle sprains are the most commonly occurring injuries overall. Females consistently have higher rates of ACL ruptures and concussions than male players. Finally, injuries occur more frequently in competition than in practice.

3.4.1 High School Basketball

Basketball is one of the most popular high school sports, with over 900,000 competitors annually [43]. Injuries requiring time off from participation occur at a rate of 1.53–2.08/1000 athletic exposures (AE) [24–27, 41]. Roughly 40.6–51.3% of these injuries require less than 1 week of missed participation [25–27]. Additionally, non-time loss injuries occur at a rate of 5.75–6.57/1000 AE [24]. A majority of injuries involved the lower extremity. In boys, the ankle was by far the most commonly injured body part, followed by the head/face and the knee [26]. In girls, the most commonly injured sites were the ankle, knee, and head/face respectively [27]. Girls are more likely to sustain knee injuries, concussions, or overuse injuries than boys, and boys are more likely to sustain ankle, hip/groin, or trunk injuries than girls [24, 25]. In both boys and girls, injuries occurred more frequently in competitions than in practices.

3.4.2 Collegiate Basketball

In 2015–2016, over 35,000 student athletes competed in basketball in the NCAA [44]. Injuries occur at a rate of 4.96–5.30/1000 AE, and roughly 53.9–62.2% required less than 1 week off of participation. The majority of injuries occur in the lower extremity. In boys, the most commonly injured sites are the ankle (24.3–25%), head/face (13.2–14.3%), hip/thigh (13%), and knee (12.4–18.6%). In girls the most commonly injured sites are the ankle (21.2–21.7%), knee (17.8–22.1%), and head/face (14.2–19.2%). In both males and females, the most common injury type is ligament sprain (30.2–39.3%), followed by muscle/tendon strain (15.1–16%) and concussion (12.4–14.3%). Injuries occur at higher rates in competition than practice, 9.9/1000 AE vs. 4.3/1000 AE. Notably concussions were as high as three times more likely to occur in competition than practice, whereas muscle/tendon strain was more common in practice than competition [26, 27, 31].

3.4.3 Professional Basketball

Due to the longer seasons and higher number of games, professional basketball players are often prone to injury. A long-term study of NBA players demonstrated an injury rate of 19.1/1000 AE. The most commonly injured sites are the ankle (14.7%), lumbar spine (10.2%), patella (10.1%), and knee (9.0%). The most frequent injury types were ligament sprain (27.8%), muscle/tendon strain (21.8%), inflammatory (21.8%), contusion (15.3%), skin injury (4.2%), fracture (4.1%), and concussion (2.0%) [23]. Evaluation of WNBA players demonstrated an injury rate of 5.97/1000 AE. The most frequently injured sites were the knee (29%), ankle (22%), and head (10%). The most frequent injuries were ankle sprain (20%), concussion (10%), ACL tear (9%), and patellar tendinopathy (8%) [45].

3.4.4 Children

It is difficult to quantify injury rates in pediatric basketball players as there is no governing organization which records and reports injuries. What little data is available comes from review of emergency department visits. While adolescent athletes have a similar injury profile to that seen in high school, there are some differences noted in children ages 5–10. In general, this younger subset of patients had a lower injury rate than their adolescent counterparts. However, young children were noted to have a higher incidence of injury to the upper extremity, particularly finger fractures, as well as a higher incidence of traumatic brain injury. Boys were also more commonly injured than girls. Boys were also more likely to sustain fractures or lacerations than girls [46].

3.4.5 Adults

While competitive basketball consists of five players on the court per team, the game can easily be adjusted to accommodate fewer players. Because the game can still be played even if there are not a full ten players available, basketball is particularly popular in recreational adult athletes. Similar to children, it is difficult to quantify the rate at which adult recreational athletes get injured. However, basketball-related injuries have been reviewed in the emergency department and ambulatory care settings. It is estimated that over 500,000 basketball injuries in adults are evaluated annually. The majority of basketball injuries among adults occurred in males (88%). Much like with competitive basketball the most frequent injury is ankle sprain. However, it is worth noting that 12.2% of injuries were fractures, with 40% of these involving the wrist, hand, or fingers [47]. While basketball is an excellent avenue to maintain physical fitness in recreational adults, further investigation is necessary to quantify the injuries these adults are susceptible to.

3.5 Prevention Plans to Avoid and Reduce Basketball Injuries

Basketball has one of the highest injury rates of any major sport. Given this, there is significant interest in developing prevention strategies to lower injury incidence. Multiple avenues for injury prevention exist, including neuromuscular training, external supports, adequate player rest, rule changes, and preparticipation screening of at-risk players.

3.5.1 Neuromuscular Training

There has been extensive study in the effect of neuromuscular training on injury prevention across multiple sports. A multitude of programs have been developed and studied. In general, these neuromuscular training regimens involve a series of exercises which are often integrated into pre-practice warmups or team conditioning drills. They involve stretching, balancing, power, and agility exercises with the aim of optimizing neuromuscular control around a joint, thereby limiting risk of injury to that joint [48]. Ideally, sport-specific exercises are incorporated into the regimen to simulate high-risk motions encountered during play. For basketball, this involves rebounding, cutting, and rapid side-to-side movements. There is evidence supporting the benefit of neuromuscular training to reduce ankle injuries as well as general lower-extremity injuries in basketball. However, the effect of neuromuscular training on reducing ACL injuries in basketball players is not conclusive [3].

In regard to ankle sprains, the goal of neuromuscular training is to increase postural control and strengthen the dynamic stabilizers of the ankle joint. The peroneal muscles are of particular importance given they are the primary dynamic stabilizers preventing rapid inversion of the ankle. Multiple studies have demonstrated reduced risk of ankle sprain with neuromuscular training. This holds true for prevention of both primary and recurrent ankle sprains [3, 48–51]. Regimens directed exclusively toward the ankle as well as general lower-extremity proprioception regimens have demonstrated decreased risk of ankle sprain, but perhaps the greatest risk reduction comes with regimens that incorporate single leg stance with either perturbation boards and/or sport-specific tasks [48].

In regard to ACL injuries, neuromuscular training is focused toward avoiding positions that put high mechanical stress on the ACL. Since ACL injuries often occur due to valgus collapse when landing or cutting on a leg with knee and hip extension, hip adduction, and hip internal rotation, prevention programs focus on avoiding this position. Focus is on landing with greater knee and hip flexion as well as strengthening of hip abductors and external rotators. These exercises are generally combined with sport-specific tasks to best simulate situations that may be encountered in gameplay. ACL prevention programs have been most extensively studied in female soccer players but results in basketball players have also been evaluated. While multiple studies have demonstrated that ACL prevention programs can reduce ACL injury risk [52–55], some larger meta-analyses have shown no clear benefit of neuromuscular training compared to control groups for both basketball [3] and soccer [56]. Further study is needed to better clarify what interventions are most beneficial in reducing injury risk.

3.5.2 External Supports

Another method for prevention of ankle injuries in basketball players is the use of external support to provide additional ankle stability. This can be through the use of high-top shoes, prophylactic ankle taping, or use of an ankle brace. These modalities provide a rigid support to prevent excessive inversion of the ankle. While evidence suggests that there is no effect of high-top shoes on ankle injury rates [57], both ankle taping and bracing have been shown to decrease ankle sprains. There is some evidence to suggest that use of a lace up ankle brace may have superior results to taping and could be more cost effective in the long term [48].

3.5.3 Rest

It has been established across multiple sports that as training and competition frequency and intensity increase, players are more susceptible to developing injuries, particularly overuse injuries [58, 59]. In the National Basketball Association, it has been shown that games on back-to-back days can increase injury risk [60] and that injury risk decreases following rest days [61]. Due to concern regarding workload, there has been effort in recent years to limit back-to-back gamedays or stretches of four games in 5 days. Excessive workload is also a concern in youth athletes. Players often play a large volume of games during weekend tournaments, play year-round, and potentially play on multiple teams concurrently. This single sport specialization in pediatric athletes has been shown to lead to increased injury rates [62, 63]. While multiple medical societies have developed recommendations regarding single sport specialization in pediatric athletes, such as no more than 8 months per year in one sport or limiting hours per week to fewer than the child's age, there is no regulation to enforce this [62]. Given the high prevalence of overuse injuries in basketball across all competition levels, focus should be placed on providing adequate rest throughout the season to reduce risk of injury. While there has been some effort to quantify workload in order to reduce injury risk [64] there is no clear answer as to the optimal workload in basketball players. Further study is needed to establish evidence-based recommendations regarding appropriate workload to minimize injury risk.

3.5.4 Rule Changes

Basketball is a constantly evolving sport. Rules of gameplay are frequently evaluated and adjusted by each league's respective governing body. Some of these rule changes are implemented to increase the safety of the game by limiting injury risk. For instance, with recent focus on the severity of the sequelae of concussions, emphasis has been placed on reducing head injuries in basketball. This includes emphasis on limiting play with elbows above shoulder level and implementation of video review for plays involving contact to the head to assess for flagrant fouls. Attempts have been made to limit lower-extremity injuries by declaring it a foul when a ball handler is tripped, even if contact is incidental, or by incorporating a restricted area under the rim to limit contact at the basket. Also, in the NBA, injuries which occurred due to barriers and photographers at the baseline led to changes providing more space around the basket and limiting number of other personnel at the baseline to avoid these injuries from occurring outside the playing boundaries. It is unclear the impact these rule changes have had on injury incidence but adjusting the game to avoid aspects contributing to increased risk of injury is an important preventative measure.

3.5.5 Player Tracking

Since the 2013–2014 season, all NBA teams have employed player tracking in their arenas via different providers to detect movements and plays during competition [65]. Categories such as speed and distance an individual player traveled per game have been collected. Average speed, distance traveled, and paint touches could be predictors of soft tissue injuries [66]. Models have been developed incorporating player tracking, among other variables such as team schedules and player workload, to predict injury risk [67].

3.5.6 Screening

One other preventative measure that can be taken to prevent basketball injuries is to screen those at high risk. Identification of previously sustained injuries might prompt intervention to prevent recurrence. For example, individuals who have previously sustained an ankle sprain are at high risk of recurrent sprain [48]. Such players may benefit from prophylactic bracing/taping or from balance and proprioceptive exercises. Patients with history of patellar tendinopathy may be monitored for early signs of recurrence to minimize further injury. Also, attempt should be made to identify players with poor biomechanics. It has been shown that females who land with increased knee extension and valgus are at higher risk of sustaining ACL injury [68]. Players with these high-risk biomechanics should be identified as they may benefit from neuromuscular training to address their landing mechanics. Finally, medical or cardiac conditions should be screened for on preparticipation physicals to minimize risk of medical complications such as sudden cardiac arrest. Although this is rare, the outcome is devastating so care should be taken to identify individuals at risk.

3.6 Paralympics: Wheelchair Basketball

Wheelchair basketball was first played in 1945 by American World War II veterans in the United States. It was one of the sports included in the inaugural Rome 1960 Paralympic Games [69]. Globally, wheelchair basketball has become one of the most widely played sports among people with disabilities, with up over 100,000 participants from 80 different countries competing worldwide [70]. Due to the physical disability of the athletes and the nature of the biomechanics involved in wheelchair sports, wheelchair basketball players are more commonly at risk for injuries to the upper body and present with challenges in managing unique medical symptoms.

Similar to basketball for able-bodied athletes, wheelchair basketball players are required to perform repeated high intensity movements on the court including rapid acceleration and deceleration with dynamic positional changes [71]. However, wheelchair athletes use the upper body to execute these maneuvers by navigating the wheelchair, which is unique to the sport. For this reason, in contrast to the injury-prone lower extremities for ambulatory athletes, the most common injuries sustained during wheelchair basketball are the upper extremities [72]. The lower limbs may be less vulnerable to injury due to athlete's fixation of the lower body in the wheelchair [73]. The increased risk of upper limb injures is related to wheelchair propulsion and repetitive strain injuries.

The most common site of injury for wheelchair athletes is the shoulder. The incidence of shoulder pain and injury among wheelchair users can be up to 85% [74]. Trunk stabilization has also been found to be correlated with the risk of shoulder injury. Athletes with poor trunk control can have twice as much shoulder pain compared to those with good trunk control. These findings can be attributed to the increased load placed on the shoulder joint and differences in sitting posture. Players without pelvic control have to rely more on the upper extremities, increasing stress and overloading the shoulder joint in particular. Athletes with poor trunk control are also found to sit with a more posteriorly tilted pelvis, which promotes inefficient scapulothoracic and glenohumeral positioning, motion, and torque-producing capacity, increasing the risk of shoulder injuries [75].

One of the common shoulder injuries in wheelchair athletes is subacromial impingement. Subacromial impingement syndrome can occur from overuse, improper warmup, glenohumeral or scapulothoracic dyskinesia, poor lumbo-pelvic postural control, poor shoulder flexibility, repetitive overhead arm positioning, and fatigue [75]. Other shoulder pathology can include rotator cuff injury, acromioclavicular pathology, bicep tendon pathology, subacromial and subdeltoid effusion, and shoulder strain [76]. Wheelchair propulsion can also result in repetitive strain injuries to the wrist. Propelling the wheelchair requires highly repetitive wrist flexion and extension, which ultimately intensifies carpal tunnel pressure resulting in carpal tunnel syndrome [77].

As with any sport, proper training, technique, equipment, and nutrition are vital components for injury prevention. It has been found that poor internal and external shoulder rotation range of motion (ROM) is a risk factor for shoulder pain and injury. There have been shoulder injury prevention programs that focus on flexibility and strength that have shown to improve shoulder ROM, which can be considered by the athlete to reduce the risk of injury [78]. Training programs should be directed at strengthening the available trunk muscles, rotator cuff, and scapular stabilizing muscles. Another frequently seen injury in wheelchair athletes is exercise-induced muscle spasms. Possible strategies to prevent such spasms include adequate rehydration, electrolyte supplementation, and stretching [73].

Special consideration is required when evaluating and treating wheelchair basketball players with a spinal cord injury (SCI). These individuals may present with unique clinical features which include autonomic dysreflexia and impaired thermoregulation. Autonomic dysreflexia is a medical emergency and is defined as an acute syndrome of excessive uncontrolled sympathetic output, more common in SCI at or above the sixth thoracic neurologic level. Any noxious stimuli below the level of injury triggers a peripheral sympathetic surge resulting in vasoconstriction and hypertension. However, the compensatory descending inhibitory parasympathetic signals are blocked at the level of SCI [79, 80]. This results in an unopposed sympathetic rush manifesting as headaches, flushing, and diaphoresis above the level of injury. Common triggers include bladder or bowel distension, pressure sores, fractures, or deep vein thrombosis. Management includes removing the noxious stimuli and symptomatic treatment. "Boosting" is the intentional self-induction of autonomic dysreflexia with the goal of enhancing performance [81]. Boosting is a form of doping used by wheelchair athletes and is banned by the International Paralympic Committee.

The two main thermoregulatory effectors of the body are sweating and blood redistribution. Both responses may be impaired in wheelchair athletes with SCI. Thermoregulatory impairment occurs due to the loss of feedback from insensate body regions and inability to make vasomotor adjustments because of the reduction in sweating capacity below the level of SCI. This results in difficulty adjusting to hot and cold environments. The inability to dissipate heat at the rate it is produced internally results in elevated core temperatures and places the athlete at risk for heat-related illnesses [82]. Heat injury may be prevented with implantation of cooling strategies. Pre-exercise cooling and cooling during exercise have been shown to lower body temperature in athletes with SCI [83, 84].

3.7 Summary

Complex lower body movements in basketball, such as running with sudden acceleration or deceleration, abrupt multidirectional changes, and repetitive jumping and landing result in different types of lower-extremity injuries, specifically at the ankle and knee. Upper body injuries, specifically joint sprains and dislocations and mallet finger, can be sustained due to dribbling, passing, and shooting. Head trauma and cardiac issues are also of concern in basketball players. There are subtle differences in injury rate when comparing male and female basketball athletes at the youth, high school, collegiate, and professional levels. Professional basketball players tend to have a higher injury rate compared to their collegiate and high school counterparts. Neuromuscular training has been shown to reduce ankle injuries, while the effect on ACL injuries is unclear. Managing workload helps reduce overuse injuries. Schedule and rule changes in the NBA have had a role in trying to prevent injuries. Player tracking could have a role as well. Finally, high-risk players can be identified through preparticipation screening in order to develop individualized injury prevention plans.

References

1. Mokray WG, et al. Basketball. In: Encyclopaedia Britannica. Encyclopaedia Britannica, Inc. 2020. https://www.britannica.com/sports/basketball. Accessed 15 June 2020.
2. International Basketball Migration Report. CIES Sports Observatory. 2019. www.fiba.basketball/documents/ibmr2019.pdf. Accessed 15 June 2020.
3. Taylor J, 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.
4. Andreoli C, 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.
5. Edwards T, et al. Monitoring and managing fatigue in basketball. Sports (Basel). 2018;6(1):19. https://doi.org/10.3390/sports6010019.
6. McClay IS, et al. A profile of ground reaction forces in professional basketball. J Appl Biomech. 1994;10(3):222–36. https://doi.org/10.1123/jab.10.3.222.
7. Okazaki VH, et al. A review on the basketball jump shot. Sports Biomech. 2015;14(2):190–205. https://doi.org/10.1080/14763141.2015.1052541.
8. Hussain I, et al. Investigation of bio-kinematic elements of three point shoot in basketball. Int J Sports Sci. 2017;7(4):163–9. https://doi.org/10.5923/j.sports.20170704.02.
9. Struzik A, et al. Biomechanical analysis of the jump shot in basketball. J Hum Kinet. 2014;42:73–9. https://doi.org/10.2478/hukin-2014-0062.
10. DeVita P, et al. Effect of landing stiffness on joint kinetics and energetics in the lower extremity. Med Sci Sports Exerc. 1992;24(1):108–15.
11. Gross TS, et al. The shock attenuation role of the ankle during landing from a vertical jump. Med Sci Sports Exerc. 1988;20(5):506–14.
12. Hu H. Common types and countermeasures of ankle ligament injury caused by intense basketball movement. Niger J Clin Pract. 2017;20(8):1036–9. https://doi.org/10.4103/njcp.njcp_145_16.
13. Cordova ML, et al. Influence of external ankle support on lower extremity joint mechanics during drop landings. J Sport Rehabil. 2010;19(2):136–48. https://doi.org/10.1123/jsr.19.2.136.
14. McCaw ST, et al. Prophylactic ankle stabilizers affect ankle joint kinematics during drop landings. Med Sci Sports Exerc. 1999;31(5):702–7. https://doi.org/10.1097/00005768-199905000-00013.
15. Panagiotakis E, et al. 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. https://doi.org/10.1016/j.jsams.2017.05.006.
16. Teng PSP, et al. Effects of foot rotation positions on knee valgus during single-leg drop landing: implications for ACL injury risk reduction. Knee. 2017;24(3):547–54. https://doi.org/10.1016/j.knee.2017.01.014.
17. Weiss K, et al. Biomechanics associated with patellofemoral pain and ACL injuries in sports. Sports Med. 2015;45(9):1325–37. https://doi.org/10.1007/s40279-015-0353-4.
18. Amin NH, et al. Performance outcomes after repair of complete Achilles tendon ruptures in National Basketball Association Players. Am J Sports Med. 2013;41(8):1864–8. https://doi.org/10.1177/0363546513490659.
19. Falk B. Stat of the week: Isaiah Thomas' Finishing. Cleaning the glass. 19 March 2018. https://cleaningtheglass.com/stat-of-the-week-isaiah-thomas-finishing/. Accessed 28 June 2020.
20. Bigoni M, et al. Basketball: epidemiology and injury mechanism. In: Arthroscopy and sport injuries: applications in high-level athletes. Cham: Springer; 2016. p. 33–9. https://doi.org/10.1007/978-3-319-14815-1_5.
21. Gaston RG, et al. Sports-specific injuries of the hand and wrist. Clin Sports Med. 2015;34(1):1–10. https://doi.org/10.1016/j.csm.2014.09.003.
22. Thomas T, et al. Basketball injuries: caring for a basketball team. Curr Sports Med Rep. 2013;12(5):321–8. https://doi.org/10.1097/01.CSMR.0000434055.36042.cd.
23. Drakos MC, 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.
24. Allen AN, et al. Epidemiology of secondary school boys' and girls' basketball injuries: National Athletic Treatment, Injury and Outcomes Network. J Athl Train. 2019;54(11):1179–86. https://doi.org/10.4085/1062-6050-330-18.
25. Borowski LA, et al. 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.
26. Clifton DR, 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. https://doi.org/10.4085/1062-6050-148-17.
27. Clifton DR, 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. https://doi.org/10.4085/1062-6050-150-17.
28. Tummala SV, et al. 10-Year epidemiology of ankle injuries in men's and women's collegiate basketball. Orthop J Sports Med. 2018;6(11):2325967118805400. https://doi.org/10.1177/2325967118805400.
29. Herzog MM, et al. Epidemiology of ankle sprains and chronic ankle instability. J Athl Train. 2019;54(6):603–10. https://doi.org/10.4085/1062-6050-447-17.
30. Watanabe K, et al. The role of ankle ligaments and articular geometry in stabilizing the ankle. Clin Biomech. 2012;27(2):189–95. https://doi.org/10.1016/j.clinbiomech.2011.08.01.
31. Dick R, et al. 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.
32. Alentorn-Geli E, et al. Prevention of non-contact anterior cruciate ligament injuries in soccer players. Part 1: Mechanisms of injury and underlying risk factors. Knee Surg Sports Traumatol Arthrosc. 2009;17:705–29. https://doi.org/10.1007/s00167-009-0813-1.
33. Hutchison MK, et al. Prevalence of patellar tendinopathy and patellar tendon abnormality in male collegiate basketball players: a cross-sectional study. J Athl Train. 2019;54(9):953–8. https://doi.org/10.4085/1062-6050-70-18.
34. Figueroa D, et al. Patellar Tendinopathy: Diagnosis and Treatment. J Am Acad Orthop Surg. 2016;24(12):e184–92. https://doi.org/10.5435/JAAOS-D-15-00703.
35. Benítez-Martínez JC, et al. Lower limb dominance, morphology, and sonographic abnormalities of the patellar tendon in elite basketball players: a cross-sectional study. J Athl Train. 2019;54(12):1280–6. https://doi.org/10.4085/1062-6050-285-17.
36. Peña J, et al. Patellar tendinopathy in team sports. Strength Cond J. 2017;39(3):20–30. https://doi.org/10.1519/SSC.0000000000000303.
37. Ladenhauf H, et al. Osgood-Schlatter disease: a 2020 update of a common knee condition in children. Curr Opin Pediatr. 2020;32(1):107–12. https://doi.org/10.1097/MOP.0000000000000842.
38. Pretell-Mazzini J, 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.
39. Hess GW. Achilles tendon rupture: a review of etiology, population, anatomy, risk factors, and injury prevention. Foot Ankle Spec. 2010;3(1):29–32. https://doi.org/10.1177/1938640009355191.
40. Lemme NJ, et al. Epidemiology and video analysis of Achilles tendon ruptures in the National Basketball Association. Am J Sports Med. 2019;47(10):2360–6. https://doi.org/10.1177/0363546519858609.
41. Trojian TH, et al. Basketball injuries: caring for a basketball team. Curr Sports Med Rep. 2013;12(5):321–8. https://doi.org/10.1097/01.CSMR.0000434055.36042.cd.
42. Emery MS, et al. Sudden cardiac death in athletes. JACC Heart Fail. 2018;6(1):30–40. https://doi.org/10.1016/j.jchf.2017.07.014.
43. National Federation of State High School Associations. 2018–2019 High school athletics participation survey. Indianapolis, IN: National Federation of State High School Associations. https://www.nfhs.org/media/1020406/2018-19-participation-survey.pdf. 23p.
44. Irick E. Student-athlete participation 1981/2–2015/16: NCAA sports sponsorship and participation rates report. National Collegiate Athletic Association: Indianapolis (IN); 2016. https://www.ncaapublications.com/p-4445-2015-16-ncaa-sports-sponsorship-and-participation-rates-report.aspx. 301p.
45. Baker H, et al. Injury in the Women's National Basketball Association (WNBA) from 2015 to 2019. Arthrosc Sports Med Rehabil. 2020;2(3):e213–7. https://doi.org/10.1016/j.asmr.2020.02.003.
46. Randazzo C, et al. Basketball-related injuries in school-aged children and adolescents in 1997–2007. Pediatrics. 2010;126(4):727–33. https://doi.org/10.1542/peds.2009-2497.
47. Hammig BJ, et al. 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.
48. Kaminski TW, et al. Prevention of lateral ankle sprains. J Athl Train. 2019;54(6):650–61. https://doi.org/10.4085/1062-6050-487-17.
49. Schiftan GS, et al. The effectiveness of proprioceptive training in preventing ankle sprains in sporting populations: a systematic review and meta-analysis. J Sci Med Sport. 2015;18(3):238–44. https://doi.org/10.1016/j.jsams.2014.04.00.
50. Eils E, et al. Multistation proprioceptive exercise program prevents ankle injuries in basketball [published correction appears in Med Sci Sports Exerc. 2011 Apr;43(4):741]. Med Sci Sports Exerc. 2010;42(11):2098–105. https://doi.org/10.1249/MSS.0b013e3181e03667.
51. McGuine TA, et al. 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. https://doi.org/10.1177/0363546505284191.
52. Dargo L, et al. Prevention of knee and anterior cruciate ligament injuries through the use of neuromuscular and proprioceptive training: an evidence-based review. J Athl Train. 2017;52(12):1171–2. https://doi.org/10.4085/1062-6050-52.12.21.
53. LaBella CR, et al. Effect of neuromuscular warm-up on injuries in female soccer and basketball athletes in urban public high schools: cluster randomized controlled trial. Arch Pediatr Adolesc Med. 2011;165(11):1033–40. https://doi.org/10.1001/archpediatrics.2011.168.
54. Donnell-Fink LA, et al. Effectiveness of knee injury and anterior cruciate ligament tear pre-

vention programs: a meta-analysis. PLoS One. 2015;10(12):e0144063. https://doi.org/10.1371/journal.pone.0144063.
55. Omi Y, 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. https://doi.org/10.1177/0363546517749474.
56. Grimm NL, et al. 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. https://doi.org/10.1177/0363546514556737.
57. Curtis CK, et al. The role of shoe design in ankle sprain rates among collegiate basketball players. J Athl Train. 2008;43(3):230–3. https://doi.org/10.4085/1062-6050-43.3.230.
58. Eckard TG, et al. The relationship between training load and injury in athletes: a systematic review. Sports Med. 2018;48:1929–61. https://doi.org/10.1007/s40279-018-0951-z.
59. Drew MK, et al. The relationship between training load and injury, illness and soreness: a systematic and literature review. Sports Med. 2016;46(6):861–83. https://doi.org/10.1007/s40279-015-0459-8.
60. Teramoto M, et al. Game injuries in relation to game schedules in the National Basketball Association. J Sci Med Sport. 2017;20(3):230–5. https://doi.org/10.1016/j.jsams.2016.08.020.
61. 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. https://doi.org/10.4085/1062-6050-243-17.
62. Post EG, 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. https://doi.org/10.1177/0363546517690848.
63. Bell DR, et al. Sport specialization and risk of overuse injuries: a systematic review with meta-analysis. Pediatrics. 2018;142(3):e20180657. https://doi.org/10.1542/peds.2018-0657.
64. Weiss KJ, et al. The relationship between training load and injury in men's professional basketball. Int J Sports Physiol Perform. 2017;12(9):1238–42. https://doi.org/10.1123/ijspp.2016-0726.
65. Fingas J. New NBA stats deal will put motion tracking cameras in every arena. Engadget. 7 September 2013. https://www.engadget.com/2013-09-07-nba-stats-llc-motion-tracking-sportvu.html. Accessed 28 June 2020.
66. Arede J, Vaz R, Nuno L. Could non-contact in-game injuries in NBA be predicted using tracking data?. Poster presented at the 6th NSCA International Conference, 26–29 Sept 2018 Sep, Madrid, Spain. https://www.researchgate.net/publication/327968623_Could_non-contact_in-game_injuries_in_NBA_be_predicted_using_tracking_data.
67. Talukder H, et al. Preventing in-game injuries for NBA players. In: MIT Sloan Sports Analytics Conference, 11–12 Mar 2016, Boston, USA. http://www.sloansportsconference.com/wp-content/uploads/2016/02/1590-Preventing-in-game-injuries-for-NBA-players.pdf.
68. Hewett TE, 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. https://doi.org/10.1177/0363546504269591.
69. International Wheelchair Basketball Federation. https://iwbf.org/the-game/history-wheelchair-basketball/. Accessed 16 June 2020.
70. Hutzler Y, et al. Reverse integration in wheelchair basketball: a serious leisure perspective. J Sport Soc Issues. 2016;40(4):338–60. https://doi.org/10.1177/019372351663204.
71. Yanci J, et al. Sprint, agility, strength and endurance capacity in wheelchair basketball players. Biol Sport. 2015;32(1):71–8. https://doi.org/10.5604/20831862.1127285.
72. Fagher K, et al. Sports-related injuries in athletes with disabilities. Scand J Med Sci Sports. 2014;24(5):e320–31. https://doi.org/10.1111/sms.12175.
73. Hollander K, et al. Epidemiology of injuries during the Wheelchair Basketball World Championships 2018: a prospective cohort study. Scand J Med Sci Sports. 2020;30(1):199–207. https://doi.org/10.1111/sms.13558.
74. Yıldırım NU, et al. Developing a shoulder pain scale for wheelchair basketball players. J Back Musculoskelet Rehabil. 2019;32(3):479–85. https://doi.org/10.3233/BMR-181192.
75. Yildirim NU, et al. Shoulder pain: a comparison of wheelchair basketball players with trunk control and without trunk control. J Back Musculoskelet Rehabil. 2010;23(2):55–61. https://doi.org/10.3233/BMR-2010-0250.
76. Heyward OW, et al. Shoulder complaints in wheelchair athletes: a systematic review. PLoS One. 2017;12(11):e0188410. https://doi.org/10.1371/journal.pone.0188410.
77. Kim DK. Electrophysiologic and ultrasonographic assessment of carpal tunnel syndrome in wheelchair basketball athletes. Ann Rehabil Med. 2017;41(1):58–65. https://doi.org/10.5535/arm.2017.41.1.58.
78. Wilroy J, et al. Evaluation of a shoulder injury prevention program in wheelchair basketball. J Sport Rehabil. 2018;27(6):554–9. https://doi.org/10.1123/jsr.2017-0011.
79. Krassioukov A, et al. The role of autonomic function on sport performance in athletes with spinal cord injury. PM R. 2014;6(8 Suppl):S58–65. https://doi.org/10.1016/j.pmrj.2014.05.023.
80. Blackmer J. Rehabilitation medicine: 1. Autonomic dysreflexia. CMAJ. 2003;169(9):931–5.
81. Mazzeo F, et al. "Boosting" in Paralympic athletes with spinal cord injury: doping without drugs. Funct Neurol. 2015;30(2):91–8. https://doi.org/10.11138/fneur/2015.30.2.091.

82. Price MJ. Thermoregulation during exercise in individuals with spinal cord injuries. Sports Med. 2006;36(10):863–79. https://doi.org/10.2165/00007256-200636100-00005.
83. Forsyth P, et al. Physiological and perceptual effects of precooling in wheelchair basketball athletes. J Spinal Cord Med. 2016;39(6):671–8. https://doi.org/10.1080/10790268.2016.1180098.
84. Webborn N, et al. Cooling strategies improve intermittent sprint performance in the heat of athletes with tetraplegia. Br J Sports Med. 2010;44(6):455–60. https://doi.org/10.1136/bjsm.2007.043687.

Field Hockey

4

Maria Tuca, Pía Franz, and Matías Sepulveda

4.1 Sports Modality

Field Hockey is called after the word "hocquet" (shepherd's crook in French) in reference to the shape of the players' stick. Historical evidence suggests that ancient variations of field hockey were first played 4000 years ago in Egypt, Ethiopia (1000 BC) and Irán (2000 BC). Modern hockey emerged in England in the mid-nineteenth century, spreading in popularity among the Commonwealth nations. Nowadays, field hockey has more than 30 million players worldwide, and 137 National Field Hockey Associations. It is an Olympic Sport since Amsterdam 1928 for men, and Moscow 1980 for women.

Hockey can be played on grass, synthetic or watered turf, as well as on indoor surfaces. Hockey field is rectangular (55 × 91.4 m), with an "area" comprising the first 23 m from the backline. The team is constituted by ten field players and one goalkeeper. All players use a stick, which can be made out of wood, carbon fiber, fiberglass, or a combination of these, to hit a hard-plastic round ball. The International Hockey Federation (FIH) represents and organizes hockey players around the world and is responsible for elaboration and register of the game rules that are constantly adapted in favor of the players' safety and sports show [1].

A cardinal game rule is that players are only allowed to hit the ball with the flat portion of the stick, whereas only goalkeepers can use any part of the stick and their whole-body surface. A game match is divided into four periods called "quarters," each of 15 min. The team that scores more goals during these periods wins the game. Penalties are granted when players break the rules. If a rule violation happens in the 23 m designated area, a "penalty hit" or a "penalty corner" will be granted to the opposing team. Most of the rules dictated by the FIH are designed for player safety, considering the risk that implies that hockey has the highest ball speed of swing sports (more than golf or baseball), with up to 103 mph. Thus, field players are allowed and encouraged to use hands, mouth, and shin protections during the match, and face masks or goggles during penalty corners (Figs. 4.1 and 4.2). Goalkeepers must wear protective equipment at all times, consisting at least of headgear, leg guards, and kickers. Players are not allowed to elevate the stick above the head height of their opponents and cannot intentionally raise the ball, with the exception of goal shooting or a flick. Players can raise the

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Fig. 4.1 Chile's National Female Field Hockey Team defending a penalty corner. Defenders are allowed to use face googles only during the penalty corner and must wear them off immediately after it ends. Goalkeeper is wearing specialized protective equipment designed to cover most of the body. (Courtesy of the Chilean Field Hockey Federation)

Fig. 4.2 Former captain of Chile's Female Field Hockey Team wearing hand, mouth and shin protections during a match. (Courtesy of the Chilean Field Hockey Federation)

ball with a *flick* provided it is not dangerous, meaning keeping 5 m distance or more from the opponent [1].

Field hockey is an intense, physically demanding sport, that occurs at high speed, requiring multiple skills. This makes a sport unique, exciting for everyone who practices or watches it. The Olympic Games and the World Cup, each held every 4 years, are the most important field hockey events around the globe.

4.2 Introduction

Field hockey is a high speed, intense continuous game. Because of this, the risk of fatigue injuries is high and requires for proper substitutions, which are unlimited. The absence of an "offside rule" leads to the presence of many players around the goal area, probably increasing the injury risk. However, most injuries are minor without consequences for the players [2]. Injury rates in field hockey are comparable to those in basketball [3], lacrosse [3], and softball [4], and lower compared to soccer and rugby [3–5].

The average injury rate per 1000 player hours varies depending on the competition level, 15 for amateurs [6], 33 for elite players [6], and <1 for children <12 years old [6, 7].

While field hockey is essentially a non-contact sport, players can suffer injuries from non-contact and direct contact mechanisms. It has been reported that field hockey players suffered 64% non-contact mechanism injuries and 36% direct contact injuries during training [8]. Among direct contact injuries, 14% are from contact with the ball, 10% with a stick, 7% with the field surface, and 5% with another player [8]. The same study reported that players are twice as likely to suffer an injury during season games than practice [8]. During games, 28% are non-contact injuries and 72% direct contact injuries. Among these, 29% are from contact with the ball, 18% with the stick, 14% with another player, and 9% with surface [8].

The injury incidence varies according to the player's position, with goalkeepers presenting the lowest rate (4–16%), probably for the fact of using more protective equipment, then the forwards and midfielders (22–37%), mainly due to high balls and defenders (24–36%), associated with penalty corners [6] (Fig. 4.3).

In relation to the location in the body, most injuries occur in the lower extremities (54%), upper extremities (13%), spine and pelvis (12%), face and eyes (9%), and concussions (7%) [6].

The most frequent injuries among field hockey players are ankle sprains, knee injuries, head and

Fig. 4.3 Goalkeeper wearing the protective equipment and catching a shot at goal. (Courtesy of the Chilean Field Hockey Federation)

face injuries, fractures of the hand and fingers and back pain. These top-five sports-related injuries of Field Hockey will be reviewed in detail throughout this chapter.

4.3 Ankle Sprains

Ankle ligamentous injuries are the most common sport-related injury [9]. Field hockey is not the exception, associated to cutting and pivoting maneuvers, and the risk of ankle-forced inversion when accidentally stepping over the ball or the opponent's stick.

Ankle sprains are classified as medial or lateral, high (syndesmal injury), or low (distal syndesmotic injury). The most common ankle sprain is lateral and low, affecting the anterior tibiofibular ligament and calcaneofibular ligament due to inversion mechanism on a plantar-flexed foot [10].

The traditional classification system for ankle sprains includes three degrees: grade I, microscopic injury without ligament stretch at the macroscopic level, minimal bruising and swelling, no weight-bearing pain; grade II, stretching without rupture, moderate bruising and swelling, mild pain with weight-bearing; grade III, complete rupture, severe bruising and swelling, severe pain with weight-bearing [11]. Although its use is controversial, this classification is useful operationally.

Hootman [3] reported injuries for 15 collegiate sports (including field hockey) over a 16-year period, where ankle sprains accounted for 15% of all reported injuries [3].

Ankle sprain was the most common single injury (39.7%) in a cohort of female field hockey players [11]. First-time ankle injury has a rate of 0.9 injuries/1000 person-day, as reported by Beynnon et al. [12] A review from the National Collegiate Athletic Association (NCAA) Injury Surveillance Data for a period of 15 years shows that for women's field hockey there is twice the risk of sustaining an ankle sprain in a season game than during practice [8]. The ankle sprain accounts for 9.1% of severe injuries (at least 10 days loss of activity for hockey players) [8].

The mechanism of injury correlates to the sudden sprints and rapid direction changes that take place in field hockey [13]. A study proposed that poor peak dorsiflexor torque at the ankle was associated with increased risk for ankle sprains [14]. This is explained by the fact that an increased dorsiflexion strength can help the to prevent ankle inversion associated with lateral sprains [14]. When the ankle is in an inverted and plantar-flexed position, the everter and dorsiflexor muscles act eccentrically [14]. Weak dorsiflexor muscles allow excessive movement, putting additional stress on the lateral ligaments of the ankle joints [14].

General treatment for ankle sprains is rest, ice, compression, and elevation of the extremity. For grade I and II, initial immobilization is required for a short period (<1 week) in a walking boot. After swelling and pain have subsided, immobilization should be changed to a functional brace that limits inversion and eversion. Grade III sprains may benefit from 7 to 10 days of immobilization and non-weight-bearing [10].

Treatment strategy for ankle sprains in athletes has two key points: (1) early onset of rehabilitation to reduce the risk of new injuries and fast sport return [15]; (2) prevention of recurrent injury, by balance and neuromuscular training programs (3 months period usually), focusing on proprioceptive rehabilitation and prophylactic bracing for risk activities [16–18]. Evidence supports that neuromuscular and balance training programs and prophylactic bracing reduce the incidence of injury in up to 50% [19, 20].

It is important to prevent these injuries, avoiding injury recurrence and chronic ankle instability due to its long-term morbidity and disability since it represents a potential burden for athletes. Prevention strategies from other sports that point to different types of injuries are effective in preventing ankle injuries. The "Knee Injury Prevention Program," a neuromuscular warm-up program designed for prevention of anterior cruciate ligament injury, is a good example of a program reported to reduce both ankle and knee sprains, and gradual onset injuries of the lower extremities in female football and basketball players [21]. Other study proposed that the use of the proprioceptive balance board program aimed at volleyball players was effective in preventing the recurrence of ankle sprains [22]. Since ankle sprain is the most frequent injury in field hockey, with a loss of an average of 4 days per lesion [23], prevention is of great importance [18].

Sports return depends on the grade of sprain, associated injuries, and compliance with rehabilitation programs. After a grade I–II ankle sprain, return to sports occurs on average 1–2 weeks after the injury, and after a grade III, up to 4 weeks. Complications such as pain and instability are described in up to 30% of cases, associated with non-diagnosed fractures, osteochondral lesions, impingement syndrome among others [10].

Considering the evidence backing up prophylactic bracing, balance and neuromuscular programs, there should be an ongoing support towards implementing these actions in field hockey teams. Acute injuries should be identified and treated early to reduce the risk of recurrence and chronic instabilities. To achieve this, players, coaches, and physical therapists should know the basic care of ankle sprains and follow strictly the recovery period, avoiding premature return to sports.

Ankle sprains should not be underestimated, and players should be evaluated by a specialist to obtain a precise diagnosis after a thorough study, ruling out associated injuries and initiating early rehabilitation for a prompt safe sports return, thus avoiding chronic instability and pain and decreasing the risk of re-injury.

4.4 Knee Injuries: ACL Tears

After ankle sprains, injuries around the knee are the second most frequent lesions among field hockey players, accounting for 18% of all injuries [8].

Panagodage [24] published a systematic review of injuries in female players competing in sports with stick use, and knee injuries were among the most common [24].

In relation to the severity of knee injuries, a prospective study that analyzed all injuries throughout the German Field Hockey Tournament season, reported that severe injuries (defined as absence of practice and games for more than 21 days) were 31.5% of the lesions, 17.6% corresponding to knee injuries [25]. Other study that accounted for injuries among college field hockey players through 15 years showed that 15% of the lesions were severe in-game injuries, with 23% of severe those being knee injuries, all of them related to a non-contact injury mechanism [8].

Considering gender differences, women are more likely to suffer knee injuries, with an increased risk of four times to have an anterior cruciate ligament (ACL) injury, compared to men [26].

ACL injuries in up to 78% of the cases occur after a non-contact mechanism [27]. Injuries are caused by abrupt movements when cutting or pivoting, sudden deceleration and external rotation of the tibia, associated with valgus and semiflexion of the knee.

Women's increased predisposition to injure the anterior cruciate ligament is multifactorial. This includes differences in conditioning level, femoral notch size, ACL dimensions, laxity, and Q angle, among others. However, landing biomechanics after jumping and neuromuscular activation patterns (quadriceps dominant) play the main role. Women tend to land from a jump in a more vertical position than men due to an insufficient knee and hip flexion, increasing the stress in the ACL [28]. Also, their movements involve more internal rotation of the hip along with external rotation at the tibia compared with men, leading to increased knee valgus [29].

Adequate muscular strength, along with appropriate muscle recruitment and timing, are key aspects of knee stability [30].

Factors that have been studied and incorporated into ACL prevention programs include muscle strengthening and recruitment patterns, landing and decelerating patterns, proprioception, and plyometrics [31].

There are no ACL prevention programs in literature specific for field hockey, but there are programs from other sports, mainly soccer, that have proven to be effective.

Caraffa [32] reported a significant reduction in ACL injuries with a proprioceptive training program; however, subsequent studies have not shown a significant difference in injury reduction [32]. Balance and proprioceptive training may be useful to include in an ACL prevention programs, but they are not sufficient on their own. Neuromuscular and biomechanical adaptations need to be addressed [31].

In 2005, Mandelbaum [33] studied the effects of implementing the "Prevent Injury and Enhance Performance Program" (PEEP program) in a female soccer club. The intervention was a 20-min-specific exercises regimen, performed 2–3 times a week during 12 weeks of the season. The athletes watched an educational video on safe and unsafe landing patterns, and participated in team workouts for stretching, strengthening, plyometrics, and soccer-specific agility drills, which replaced the team's regular warm-up during soccer practice. They reported an 88% overall reduction for ACL injury rate in the intervention group [33].

In 2011, LaBella [21] studied the effects of a neuromuscular warm-up program on ACL injury rates in high school female soccer and basketball players. Intervention coaches went to a 2-h training session prior to the season and learned how to implement a 20-min warm-up program designed to reduce ACL injuries. The warm-up involved plyometrics, balance, progressive strengthening, and agility exercises, as well as instruction on how to avoid dynamic knee valgus and how to land from a jump with flexion of hips and knees. At the end of the season, there was a 56% reduction in total non-contact lower extremity injuries in the intervention group compared with the control group [21].

In 2018, Webster and Hewett [34] published a meta-analysis of ACL Prevention programs and concluded that ACL injury reduction programs decrease the risk of all ACL injuries by 50% and non-contact ACL injuries by 75% in female athletes, but there is insufficient data regarding effectiveness of ACL prevention programs in male athletes [34].

To prevent ACL injury in field hockey, it is important to consider the results of these interventions and train coaches to include these routines in their training programs.

The prevention of this injury is essential since affects the sports career of the player, considering the long postoperative rehabilitation period, associated with late sport return, usually with decreased performance.

4.5 Head and Face Injuries

Sports with stick use as field hockey present an increased risk of injuries because of the high-speed movement required to hit the ball with the stick [7], which can reach up to 80 km [1] (Fig. 4.4).

Field hockey is an intense, fast-paced sport, that has become even faster in the last decades. The elimination of the off-side rule (REF), increased dedication to physical conditioning, technique progression towards more powerful shots, better technology in sticks, and games being played more often in artificial turfs, has risen the game speed over the last few years. These changes make this sport more exciting, but also potential for high risk of injuries. Athletes that participate in sports with stick use, such as field hockey, ice hockey, and lacrosse, have an increased risk of trauma because of the high-speed stick movement required to hit a puck or a ball [35]. Field hockey players had a higher proportion of facial injuries (25% for males and 20% for females) compared with ice hockey (10% males and 18% females), and lacrosse (10% boys and 20% girls) [7, 35].

Women presented more head/face injuries than male players, according to an international field hockey injury study [36], and had the highest incidence of concussion [37]. Male players present more risk for orodental injuries compared to female players (OR: 1.4) [38].

Sport-related head and face injuries can be serious and are often underdiagnosed. Injuries resulting in neurological compromise or airway obstruction may be life threatening, but fortunately are extraordinary [39]. The National Center for Catastrophic Sport Injury Research reported two skull fractures in field hockey players from 1982 to 2006 [40].

Fig. 4.4 Chile's Female Field Hockey Team player performing a "slap hit," wearing hand and shin protections. (Courtesy of the Chilean Field Hockey Federation)

According to the fifth Concussion Consensus Conference (2016), the definition of "Sports Related Concussion" is "a traumatic brain injury that is defined as a complex pathophysiological process affecting the brain, induced by biomechanical forces with several common features that help define its nature" [41]. This definition is imminently clinical and there's not a standard to assess their diagnosis.

The main concern in relation to sports-concussions is that it may increase the likelihood of incurring a subsequent head or musculoskeletal injury, where repeated concussions have been associated with long-term consequences such as neurodegenerative disorders, depression, or persistent post-concussive symptoms [37]. This is specially relevant for field hockey since concussions account for 5% of severe game injuries, meaning 10 or more days lost to injury [8].

In one series, head and face injuries account for 34% in field hockey [11]. Gardner [39] reported 75.3% of head and face injuries, other than mouth, nose, and eyes, among US collegiate women's field hockey players [39]. The most common specific injury was concussion, with an incidence of 0.40 per 1000 athlete-exposures [39]. Dick [8] reported an incidence of 9.4% for concussions and concluded that a player had six times more risk of sustaining a concussion in a game than in practice [8]. This might be due to difference in the intensity of play during a game compared to practice, specifically, more frequent direct contact among players during games [8]. Field congestion, within 25 yd of the goal, has been implicated as a potential contributor to the frequent head and face injuries [11]. Also, midfielders and attacking forward players present more concussions than other positions [37].

The "red flags" for concussions are: decreased level of consciousness, absence of movement for more than 5 s, dizziness, nausea, amnesia, blank look, and clutching the head [42]. When a sport-related concussion is suspected, the athlete should be removed from the field immediately and assessed by a physician or other licensed health care professional next to the field and keep the player under observation. In case of presenting any of the red flags, the player must be transferred to an emergency service and evaluated by a specialist. The physician who evaluates the player will carry out multiple serial evaluations, assess the need for neuroimaging, and define the sports return possibilities [42].

The most common post-concussion symptoms are headache and difficulties concentrating [37]. Depending on the severity of the concussion, the sports return varies between 48 h and 1-month [37].

It is important that the player, his family, and coach respect the resting time because of the risk of the second trauma syndrome. This syndrome takes place when the player suffers a new concussion before the previous one has resolved, producing a catastrophic brain edema, leading to loss of brain self-regulation [43]. This condition is known as traumatic encephalopathy syndrome which is a progressive neurodegenerative disease with a 50% mortality rate and disability at long term [44].

For dentofacial injuries, 56.5% are caused by hockey ball hit, 37.7% related to a hit with the hockey stick [8], with similar percentages were presented in numerous studies [2].

To prevent head and face injuries, face masks are permissible since 2007, but are not routinely worn by players [39]. Moreover, this protective gear is not allowed in international tournaments unless medically required [37]. For international competitions, face shields are only allowed for defensive players during penalty corners, but they must remove them once the short corner is over [1]. Perhaps a widespread use of face masks as part of the regulations of field hockey, could reduce the rate of craniofacial injuries, promoting their use from young ages to favor their routine use.

To prevent orodental injuries, mouth guards are effective. The use of mouth guards has increased from 27–36% in 1980s [45] to 77–91% in the late 2000s [46]. Injuries are less severe in athletes who were mouth guards than those who don't (OR = 2.1) [38]. Women had greater odds for regular wear of mouth guard during a match than males, and players who had experienced a dentofacial injury previously also had significantly higher odds to regularly wear a mouth guard [35]. There is a 55% of players who considered the mouth guard unnecessary despite the evidence available [35].

There are different mouth guards. Stock mouth guards provide inadequate protection and can cause breathing and talking problems. Mouth-moulded mouth guards provide better protection but can be too thin in prominent teeth exposing them to injury. Custom-made by dental technicians offer the better protection and aren't related to breathing or speaking problems, also last longer [35].

In a recent study regarding the use of mouth guards in field hockey in Holland, players complained less from custom-made than mouth-moulded mouth guards, and also reported that the use of mouth guards are related to lip cuts [38].

Other important aspect of the game is that the only player that wears a helmet is the goalkeeper because it is mandatory [1]. Some experts have suggested that helmets should become standard equipment for field hockey, but detractors consider that it would change the nature of the game, possibly increasing player-to-player contact, which could ultimately increase the injury rate [8, 11].

It is important to promote and regulate the use of mouth guards in hockey as it should be for all games since their mandatory use in other sports such as rugby and ice hockey have decreased orofacial injuries. More studies are needed to clarify the possible effects of the helmet in the game to recommend its widespread use.

4.6 Fractures of the Hand and Fingers

Murthaugh [11] described 14% of upper limb injuries among female field hockey players, placed in the third place of most prevalent injuries [11]. A recent study showed that upper extremities injuries account for 19.4% of all injuries, being the hand/finger the most affected location for contact injuries, also the most severe injuries [25].

During major international field hockey tournaments, the rate of upper limb injuries was 0.2 injuries per match, and 7.5 injuries per 1000 player match hours in men's tournaments [36].

Bowers [47] showed that collegiate field hockey players have significantly higher odds ratio of sustaining an upper limb injury. Hockey field players presented an injury rate of 0.48/1000 exposures, compared to 0.26/1000 in ice hockey players, 0.27/1000 men's lacrosse players, and 0.11/1000 in women's lacrosse players [47]. The odds of a hand injury (OR = 2.12), hand fracture (OR = 1.93), phalangeal injury (OR = 4.19), or phalangeal fracture (OR = 4.04) occurring in field hockey players were significantly higher than for gloved players [47] (Fig. 4.5).

The hand injuries are common due to the grasping position of the hand on the stick and the proximity of the stick and hands to the ground. The standard field hockey stick is 35–37 in. in length, with a short-curved toe that is flat on the striking surface and rounded on the backside. All field hockey sticks are designed to be held with the left hand at the top of it and the right hand distal to the left hand, typically halfway down to stick, closer to the ground, to effect fine control over the ball. As a result, the fingers and thumb, particularly of the right and frequently dominant hand, are quite vulnerable to injuries from contact with the ball or the opponent's stick [47].

According to a national surveillance of collegiate women's field hockey, finger and hand injuries accounted for 10% of all game injuries and for almost 15% of severe game injuries [8]. Most of these were caused by contact with the ball or the stick [8]. They also report that 68% of the injuries occurred when the player was near the goal or within the 25-yd line. The position associated with most injuries are back fielders (39% hand injuries) [8].

In field hockey, the bent-over posture is used for dribbling and shooting. This posture may place the player's hands closer to the ground, which may make it easier to sustain trauma from other players' sticks or cleats, or a ball during play. In areas of player congestion, such as in front or near the goal, players may easily have their hands crushed between two sticks, a player

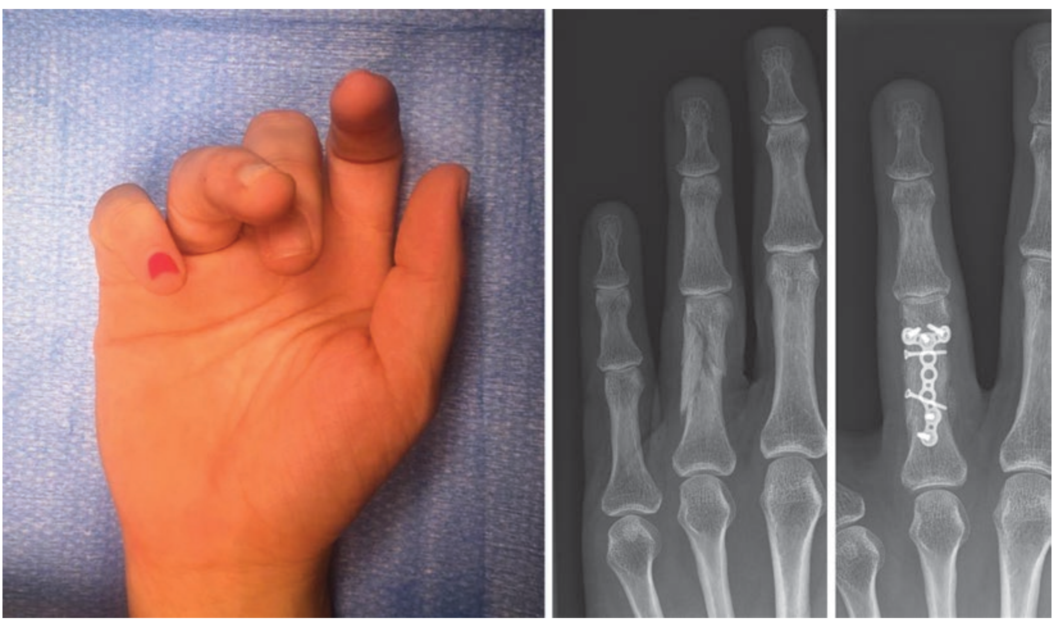

Fig. 4.5 Twenty-three-year-old amateur female hockey player that suffered an attrition of her ring finger between the ball and the stick at high speed, resulting in a comminuted fracture of the proximal phalanx that required open reduction and internal fixation with plate and screws. She was not wearing any hand protections at the moment of the injury. (Courtesy of the Chilean Field Hockey Federation)

and a stick, or a player and the ground. Before 2003, game rules required that during a penalty corner, the ball came to a complete stop outside the circle before a shot on goal, which led players to use the stick to "trap" the ball against the ground to stop it. This technique may still be used in other parts of the game and puts the hands and fingers at considerable potential for injury [8].

Field hockey-related injuries to the upper extremity can also be non-traumatic, but secondary to overuse or repetitive maneuvers. Broekstra [48] presented a cohort of 169 male field hockey players over 60 years old, matched with men from the general population of the same age. They observed Dupuytren's disease in 51.7% of field hockey players with an OR of 9.42. This is explained by the exposure of field hockey players to hand-arm vibration [48].

According to the current regulations of field hockey, the only player who needs to wear hand protection as mandatory is the goalkeeper [1]. It is important not to underestimate the injuries in terms of severity and potential sequelae. The burden of hand injuries is permanent loss of motion and function, osteoarthritis, among others [49]. Prevention of hand and finger injuries is of great importance for its short- and long-term implications. The use of protective gloves should be mandatory and guaranteed for all field hockey players, in all positions.

4.7 Back Pain

Prevalence of back pain among young elite athletes is 3–5 times higher compared to the general population [50]. The prevalence of back pain in athletes goes from 1% to 30% according to literature, and 10–15% of all sport injuries are low back injuries [51]. Serious pathology is present in less than 5% of the cases of low back pain [52]. According to literature [53], being too active might increase the risk of low back pain according to the U-shaped exposure response curve between physical activity and low back pain.

Even though back pain is not necessarily related to an acute trauma, it appeared to be a common complaint among hockey field players.

Murtaugh [11] conducted a questionnaire among high school, university, and national level female field hockey players. It found that 59% of athletes have experienced back pain at some point, and 50% reported that back pain affected them during the field hockey season [11]. The pain was serious enough to cause 12% of athletes to miss a field hockey game, time at school or work. In the same study, traumatic back injuries were only 1% [11].

Among collegiate women field hockey players, the back/trunk injuries account for 16.2% of the injuries during practice, in second place just below lower limb injuries [8].

The prevalence of lumbar flexion posture that accompany participation in field hockey renders the athletes susceptible to injuries of the lumbar spine at a rate significantly greater than general population [54]. The crouched playing position in hockey combined with side flexion and rotation may be contributing factors in the high incidence of low back problems [55].

Non-ergonomic position during field hockey practice has been related to the appearance of degenerative changes in the lumbar spine, according to Ogurkowska [56]. The excessive lumbar lordosis causes a higher load of the gravity force moment at the posterior aspect of the vertebral bodies, transforming them into a wedge shape [56]. Also, there are important changes in the intervertebral discs among field hockey players due to excessive overload. Repetition of a maximal forward bending over the years causes micro ruptures in the posterior part of the annulus fibrosus, which leads to a higher chance of having a hernia of nucleus pulposus in the lumbar segments [56].

In a prospective cohort study of elite field hockey players, 55% of all injuries were due to overuse, and lumbar pain accounted for 8% of this injuries [57].

The players exposed to increased risk of overuse injuries are the drag-flickers (OR: 1.564, compared to non-drag-flickers) [58]. The drag flick is the preferred method of scoring during a penalty corner in field hockey. During a drag flick, the player performs a run up, followed by a powerful "dragging" of the ball before releasing the ball towards the goal with a flicking motion [58]. This may be related to the volume of skill-specific training required to develop and maintain expertise in drag flicking [58].

The key to avoid back pain among field hockey players is the implementation of preventive measures and early detection of symptoms for prompt treatment. Players should undergo prophylactic core strengthening [2] and muscle stretching to reduce the compression on posterior parts of vertebral bodies as well as exercises stabilizing lumbar spine [56]. Factors associated with a lower occurrence of low back pain are satisfaction with their own performance and with the coaching staff [50].

References

1. International Hockey Federation. Rules of hockey. http://www.fih.ch/inside-fih/our-official-documents/rules-of-hockey/. Accessed 1 Apr 2020.
2. Murtaugh K. Field hockey injuries. Curr Sports Med Rep. 2009;8(5):267–72.
3. 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.
4. Rauh MJ, Macera CA, Ji M, Wiksten DL. Subsequent injury patterns in girls' high school sports. J Athl Train. 2007;42(4):486–94.
5. Powell JW, Barber-Foss KD. Injury patterns in selected high school sports: a review of the 1995–1997 seasons. J Athl Train. 1999;34(3):277–84.
6. Barboza SD, Joseph C, Nauta J, van Mechelen W, Verhagen E. Injuries in field hockey players: a systematic review. Sports Med. 2018;48(4):849–66.
7. Yard EE, Comstock RD. Injuries sustained by pediatric ice hockey, lacrosse, and field hockey athletes presenting to United States emergency departments, 1990–2003. J Athl Train. 2006;41(4):441–9.
8. Dick R, Hootman JM, Agel J, Vela L, Marshall SW, Messina R. Descriptive epidemiology of collegiate women's field hockey injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2002–2003. J Athl Train. 2007;42(2):211–20.
9. Fong DT, Hong Y, Chan LK, Yung PS, Chan KM. A systematic review on ankle injury and ankle sprain in sports. Sports Med. 2007;37(1):73–94.
10. Scillia AJ, Pierce TP, Issa K, et al. Low ankle sprains: a current review of diagnosis and treatment. Surg Technol Int. 2017;30:411–4.
11. Murtaugh K. Injury patterns among female field hockey players. Med Sci Sports Exerc. 2001;33(2):201–7.
12. Beynnon BD, Vacek PM, Murphy D, Alosa D, Paller D. First-time inversion ankle ligament trauma: the effects of sex, level of competition, and sport on the incidence of injury. Am J Sports Med. 2005;33(10):1485–91.
13. 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.
14. Naicker M, McLean M, Esterhuizen TM, Peters-Futre EM. Poor peak dorsiflexor torque associated with incidence of ankle injury in elite field female hockey players. J Sci Med Sport. 2007;10(6):363–71.
15. Chinn L, Hertel J. Rehabilitation of ankle and foot injuries in athletes. Clin Sports Med. 2010;29(1):157–67. https://doi.org/10.1016/j.csm.2009.09.006.
16. 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(1):95–100.
17. Gross MT, Liu HY. The role of ankle bracing for prevention of ankle sprain injuries. J Orthop Sports Phys Ther. 2003;33(10):572–7.
18. Janssen KW, van Mechelen W, Verhagen EA. Bracing superior to neuromuscular training for the prevention of self-reported recurrent ankle sprains: a three-arm randomised controlled trial. Br J Sports Med. 2014;48(16):1235–9.
19. Handoll HH, Rowe BH, Quinn KM, de Bie R. Interventions for preventing ankle ligament injuries. Cochrane Database Syst Rev. 2001;(3):CD000018.
20. Kim E, Choi H, Cha JH, Park JC, Kim T. Effects of neuromuscular training on the rear-foot angle kinematics in elite women field hockey players with chronic ankle instability. J Sports Sci Med. 2017;16(1):137–46.
21. LaBella CR, Huxford MR, Grissom J, Kim KY, Peng J, Christoffel KK. Effect of neuromuscular warm-up on injuries in female soccer and basketball athletes in urban public high schools: cluster randomized controlled trial. Arch Pediatr Adolesc Med. 2011;165(11):1033–40.
22. 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(6):1385–93.
23. 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.
24. Panagodage Perera NK, Joseph C, Kemp JL, Finch CF. Epidemiology of injuries in women playing competitive team bat-or-stick sports: a systematic review and a meta-analysis. Sports Med. 2018;48(3):617–40.
25. Hollander K, Wellmann K, Eulenburg CZ, Braumann KM, Junge A, Zech A. Epidemiology of injuries in outdoor and indoor hockey players over one sea-

son: a prospective cohort study. Br J Sports Med. 2018;52(17):1091–6.
26. Ireland ML. The female ACL: why is it more prone to injury? Orthop Clin North Am. 2002;33(4):637–51.
27. Boden BP, Griffin LY, Garrett WE Jr. Etiology and prevention of noncontact ACL injury. Phys Sportsmed. 2000;28(4):53–60.
28. Barber-Westin SD, Noyes FR, Smith ST, Campbell TM. Reducing the risk of noncontact anterior cruciate ligament injuries in the female athlete. Phys Sportsmed. 2009;37(3):49–61.
29. Huston LJ, Wojtys EM. Neuromuscular performance characteristics in elite female athletes. Am J Sports Med. 1996;24(4):427–36.
30. Renstrom P, Ljungqvist A, Arendt E, Beynnon 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.
31. Voskanian N. ACL injury prevention in female athletes: review of the literature and practical considerations in implementing an ACL prevention program. Curr Rev Musculoskelet Med. 2013;6(2):158–63.
32. Caraffa A, Cerulli G, Projetti M, Aisa G, Rizzo A. Prevention of anterior cruciate ligament injuries in soccer. A prospective controlled study of proprioceptive training. Knee Surg Sports Traumatol Arthrosc. 1996;4(1):19.
33. Mandelbaum BR, Silvers HJ, Watanabe DS, Knarr JF, Thomas ST, Griffin LY, et al. Effectiveness of a neuromuscular and proprioceptive training program in preventing anterior cruciate ligament injuries in female athletes: 2-year follow-up. Am J Sports Med. 2005;33(7):1003–10.
34. Webster KE, Hewett TE. Meta-analysis of meta-analyses of anterior cruciate ligament injury reduction training programs. J Orthop Res. 2018;36(10):2696–708.
35. Vucic S, Drost RW, Ongkosuwito EM, Wolvius EB. Dentofacial trauma and players' attitude towards mouthguard use in field hockey: a systematic review and meta-analysis. Br J Sports Med. 2016;50(5):298–304.
36. Theilen TM, Mueller-Eising W, Wefers Bettink P, Rolle U. Injury data of major international field hockey tournaments. Br J Sports Med. 2016;50(11):657–60.
37. Rossiter M, Challis M. Concussion in field hockey: a retrospective analysis into the incidence rates, mechanisms, symptoms and recovery of concussive injuries sustained by elite field hockey players. BMJ Open Sport Exerc Med. 2017;3(1):e000260.
38. Vucic S, Drost RW, van Wijk AJ, Wesselink PR, Wolvius EB. Patterns of orodental injury and mouthguard use in Dutch field hockey. Br J Sports Med. 2016;50(11):661–8.
39. Gardner EC. Head, face, and eye injuries in collegiate women's field hockey. Am J Sports Med. 2015;43(8):2027–34.
40. National Center for Catastrophic Sport Injury Research. 24th annual report: fall 1982–spring 2006. http://nccsir.unc.edu/files/2014/05/AllSport.pdf.
41. McCrory P, Feddermann-Demont N, Dvořák J, Cassidy JD, McIntosh A, Vos PE, et al. What is the definition of sports-related concussion: a systematic review. Br J Sports Med. 2017;51(11):877–87.
42. Muth CC. Sport-related concussion. JAMA. 2018;319(8):840.
43. Graham R, Rivara FP, Ford MA, Spicer CM, Committee on Sports-Related Concussions in Youth; Board on Children, Youth, and Families; Institute of Medicine; Sports-Related Concussions in Youth. Improving the science, changing the culture. Washington, DC: National Academies Press; 2014.
44. Reams N, Eckner JT, Almeida AA, Aagesen AL, Giordani B, Paulson H, et al. A clinical approach to the diagnosis of traumatic encephalopathy syndrome: a review. JAMA Neurol. 2016;73(6):743–9.
45. Bolhuis JH, Leurs JM, Flögel GE. Dental and facial injuries in international field hockey. Br J Sports Med. 1987;21(4):174–7.
46. Hendrick K, Farrelly P, Jagger R. Oro-facial injuries and mouthguard use in elite female field hockey players. Dent Traumatol. 2008;24(2):189–92.
47. Bowers AL, Baldwin KD, Sennett BJ. Athletic hand injuries in intercollegiate field hockey players. Med Sci Sports Exerc. 2008;40(12):2022–6.
48. Broekstra DC, van den Heuvel ER, Lanting R, Harder T, Smits I, Werker PMN. Dupuytren disease is highly prevalent in male field hockey players aged over 60 years. Br J Sports Med. 2018;52(20):1327–31.
49. Rosberg HE, Carlsson KS, Dahlin LB. Prospective study of patients with injuries to the hand and forearm: costs, function, and general health. Scand J Plast Reconstr Surg Hand Surg. 2005;39(6):360–9.
50. Van Hilst J, Hilgersom NF, Kuilman MC, Kuijer PP, Frings-Dresen MH. Low back pain in young elite field hockey players, football players and speed skaters: prevalence and risk factors. J Back Musculoskelet Rehabil. 2015;28(1):67–73.
51. Mortazavi J, Zebardast J, Mirzashahi B. Low back pain in athletes. Asian J Sports Med. 2015;6(2):e24718.
52. Refshauge KM, Maher CG. Low back pain investigations and prognosis: a review. Br J Sports Med. 2006;40(6):494–8.
53. Heneweer H, Vanhees L, Picavet HS. Physical activity and low back pain: a U-shaped relation? Pain. 2009;143(1–2):21–5.
54. Haydt R, Pheasant S, Lawrence K. The incidence of low back pain in NCAA Division III female field hockey players. Int J Sports Phys Ther. 2012;7(3):296–305.
55. Sherker S, Cassell E. A review of field hockey injuries and countermeasures for prevention. Report No 143. Monash University Accident Research Centre. 2002. 11–27.2.

56. Ogurkowska M, Kawałek K. Pathological changes in the lumbar intervertebral discs among professional field hockey players. J Sports Med Phys Fitness. 2016;56(1–2):85–91.
57. Barboza SD, Nauta J, van der Pols MJ, van Mechelen W, Verhagen EALM. Injuries in Dutch elite field hockey players: a prospective cohort study. Scand J Med Sci Sports. 2018;28(6):1708–14.
58. Ng L, Sherry D, Loh WB, et al. The prevalence and severity of injuries in field hockey drag flickers: a retrospective cross-sectional study. J Sports Sci. 2016;34(18):1746–51.

5 Gymnastics (Artistic, Rhythmic, Trampoline)

Sports-Specific Injures and Unique Mechanisms in Artistic and Rhythmic Gymnastics and Trampoline

Samantha Tayne, Lorena Bejarano-Pineda, and Mark R. Hutchinson

5.1 Introduction

Gymnastics has a long history as a competitive sport, becoming part of the Olympic Games in 1896, and continuing as a very popular sport internationally into today. The International Gymnastics Federation has 148 affiliated or associated federations, with an estimated 50 million people of all ages and around the world participating in a gymnastics club [1]. In particular, gymnastics is one of the most popular youth sports [2]. The sport includes artistic or rhythmic gymnastics which are composed of different elements. In addition, acrobatic sports such as trampoline fall under the organizational auspices of gymnastic governing bodies. In artistic gymnastics, female athletes compete in four elements—the uneven or asymmetric bars, the balance beam, the vault, and the floor. Male artistic gymnasts compete in six elements including the vault and the floor which mirror their female counterparts but also the parallel bars, the high bar, the rings, and the pommel horse which have very different loads and mechanics than the balance beam and uneven bars. Rhythmic gymnasts compete as individuals or in groups on a padded floor mat using one of four different pieces of equipment rope, hoop, ball, clubs, ribbon, or in the case of group competitions, combinations of apparatus. Rhythmic gymnasts focus on balance, grace, coordination, and flexibility while simultaneously using one of the above apparatus. In trampoline, athletes launch themselves up to 30 ft in the air while performing twists and flips. Miss-landing on the trampoline surface or completely leaving the field of play can lead to significant or catastrophic injury. While many injuries and problems overlap across each category of gymnastics, each also introduces specific injury patterns secondary to their unique demands.

Most forms of gymnastics involve training that begins at a very young age, typically around 5 or 6 years old. The junior elite gymnasts train on average 21–37 h per week, while the senior elite train up to 40 h per week [1, 3]. Skeletally immature patients have an increased risk of injury during times of rapid growth. Gymnastics requires power, coordination, balance, and flexibility in order to excel at the various skills. These athletes often train year-round without any dedicated rest period, advancing through various competition levels as they train. Due to the format of gymnastics competition, many gymnasts may begin

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training to advance in one set of skills while competing with another set of skills [4]. If they have not mastered the fundamentals of one skill before advancing to the next, then they are at increased risk of falls or injuries. The most common injuries are usually secondary to repetitive overuse which occur when the athlete is not given enough down time to recover from minor strains and sprains. Continuing to compete and train when injured, places the athlete at an increased risk of even more severe injuries and longer time away from sport to recover. When caring for gymnasts, it is important to navigate the balance of their need to intensely train and their risk of injury.

5.2 Unique Injury Risks and Mechanics of Gymnastics

Artistic gymnastics involves a combination of explosive, balance, and artistic skills [1]. Large impact loads and repetitive high velocity muscle contractions are required both to take off and land in a controlled fashion, leading to a high risk of lower extremity injuries [3, 4]. In addition, the compressive, distractive, and rotational forces of various joints may also increase the risk of injury during skills other than landing [3, 4]. For example, giant swings, a complete 360° around the bar, or catch and release skills, require a significant amount of torque and power from the shoulder joint, leading to increased risk of injury to the labrum, rotator cuff, or ligaments of the shoulder [3]. Many skills require extreme range of motion of the spine, shoulders, and hips, which may predispose to pathology in those anatomic areas, as well [3–5]. For artistic gymnasts, a key factor to consider is the height at which they perform. Potential energy and risk of more severe injury is directly related to height and is directly correlated with the amount of kinetic energy leading to injury during a fall. The high bar for male gymnasts is 9 ft above the ground and the athletes fly 2–3 ft above that. The upper of the uneven bars for women is 8 ft off the ground, and the balance beam is 4 ft off the ground.

Injuries vary depending on the sex, age, and competitive level of the athlete. Younger athletes with open growth plates have risk of growth plate injuries due to repetitive trauma, particularly at the wrist. Lower extremity injuries are more common in female athletes and upper extremity injuries are more common in male athletes, likely due to upper body strength and weight-bearing requirements [6–8]. Surface contact, particularly with the floor apparatus, is the most common injury mechanism [6, 8]. The beam is also commonly associated with injury for female athletes, and the high bar for males [6, 8]. Landings have the highest rates of injury (49–76%), followed by falls and collisions (27.8%), which goes along with the risk of surface contact, as well [6]. Athletes suffer from higher risk of injury during competition though the type of injury also varies depending on whether it occurs during training or competition [8]. Training typically involves chronic, overuse type injuries, while acute, instability type injuries occur during competition [3, 8]. This makes intuitive sense given the psychological component of competition.

While lower extremity injuries are the most common, artistic gymnastics involves a significant amount of upper extremity weight-bearing, which is unique to the sport. A large proportion of training time is spent performing maneuvers on the upper limbs whether during static or dynamic movements. Injury rates have been described up to 50%, especially in male gymnasts [9]. The most common location of injuries of the upper extremity in male and female gymnasts is the shoulder and wrist, respectively. Several studies suggest that more than 80% of the athletes will experience some wrist and elbow pain during the season [10, 11]. Additionally, Grapton et al. showed that forearm injuries were independently related to trampoline skills [12].

For rhythmic gymnasts, the mechanics of injury are more commonly related to repetitive overuse and the sport's demand for extremes of flexibility. The athletes will toss, catch, attain a specific posture, and repeat elements hundreds of times to perfect their skills. If not balanced or inadequate recovery time is allowed, then the risk of overuse injuries rises. Rhythmic gymnastics, and to some degree women's artistic gymnastics are esthetic sport which awards visual presentation and favors thin athletes. This introduces pressures for the athlete to control weight which in turn places them at risk of Relative Energy Deficit Syndrome or the Female Athlete Triad.

For trampoline and acrobatic gymnasts, skill level plays a significant role in injury patterns. Attempting to perform advanced level skills before a proper, sequential skill development increases the risk of catastrophic injury. Injuries are most commonly related to overuse; however, the incidence of catastrophic injuries is likely higher in trampoline and acrobatic sport [12].

5.3 Epidemiology

Gymnastics is a sport with high participation rates leading proportionately to increased incident of injury. More than 86,000 gymnastic injuries require medical treatment every year [7]. Participation at the Olympic Games includes artistic, rhythmic, and trampoline gymnastics, with approximately 320 gymnasts registered to compete at each Olympic Games, constituting about 3% of all registered athletes [1, 13]. Edouard et al. reported the incidence of injuries in 963 registered gymnasts during three consecutive Olympic Games with 81 injuries per 1000 gymnasts at the highest level of competition. Artistic gymnastics accounted for most of the injuries (60.4%), followed by rhythmic gymnastics (29.7%), and lastly trampoline (9.9%) [1]. In the American collegiate sports system, women's artistic gymnastics was reported to have the second highest prevalence of injuries after American football [14]. The majority of provided epidemiological data refers to female artistic gymnasts, much less is known regarding injury incidence in male gymnastics or rhythmic and trampoline gymnastics.

In artistic gymnastics, injury rates range from 1.6 to 4.1 per 1000 h of training with elite-level athletes presenting more severe injuries [15, 16]. The 1-year incidence of injury is higher in females than males with 3.7 vs. 1.0 injuries per 1000 h, respectively [6]. Conversely, the 1-year prevalence reported for female and male gymnasts are equal at 2.0 injuries per 1000 h of exposure [17]. The injury risk for rhythmic gymnasts was noted to be 1.08 per 1000 h of training [18]. Lower extremity injuries are more common in female athletes and upper extremity injuries are more common in male athletes, likely due to upper body strength and weight-bearing requirements [6–8]. The most frequent reported injury location was the ankle followed by the lumbar spine and knee [19]. In a 10-year observational study by Westermann et al. men's gymnasts were at increased risk of hand and wrist injuries followed by ankle and foot, knee, shoulder, back, and elbow respectfully [20] (Fig. 5.1). Head and neck injuries accounted for 10–15% of injuries. For women gymnasts, the most common anatomic site of injury was foot and ankle followed by knee, low back, shoulder, elbow, hip, and groin respectfully (Fig. 5.2). When comparing men's and women's artistic gymnasts, the men tended to have more upper extremity and head and neck injuries. The type of injuries among gymnasts are usually sprains (34%), followed by articular pathology (17%), contusions (9%), and fractures (7%) [1]. Similarly in rhythmic gymnastics, the most common site of injury was ankle or foot (26–39%) followed by low back (22–25%) and knee (15–20%) [18, 21] (Fig. 5.3). Leg pain accounted for an additional 15–20% of complaints and injuries followed by hip/thigh and wrist/hand. Shoulder/elbow and head/neck were relatively rare in rhythmic gymnasts. Compared to other acrobatic sports, the frequency of knee and forearm injuries are significantly higher in trampoline [1, 12].

The risk of injury during competition is about two times higher than during practice sessions causing a higher number of time-loss from sport injuries [22]. Most of these injuries are acute in nature. Campbell et al. reported the incidence of acute injuries ranged from 55% to 83%, whereas overuse injuries varied from 26% to 41%, respectively. The incidence of recurrent injuries were lower but considerable ranging from 8% to 32% [6, 19].

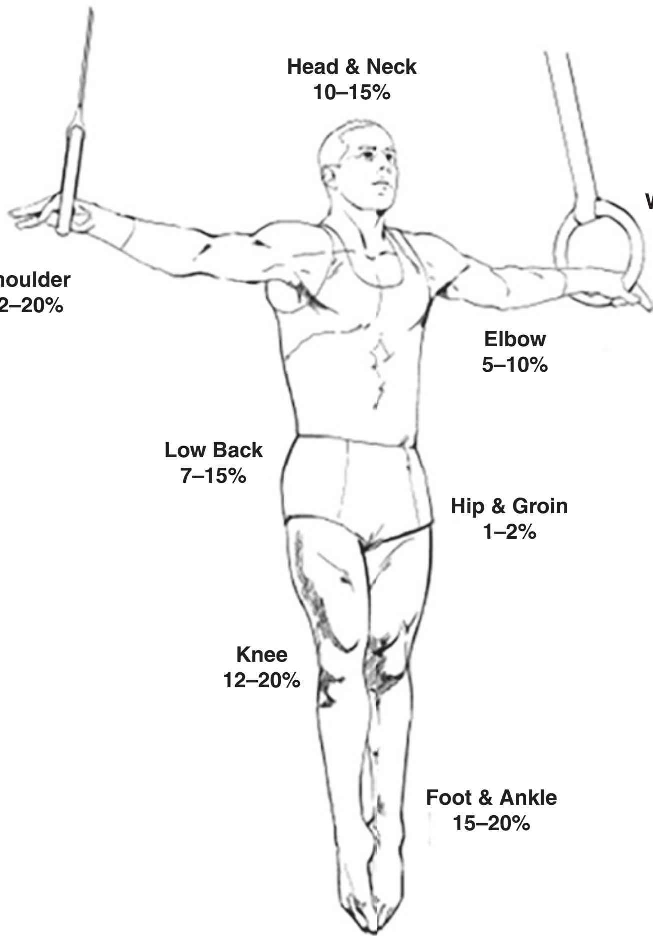

Fig. 5.1 Distribution of injuries by body part in men gymnasts. (© Mark R. Hutchinson, 2021. All Rights Reserved)

5.4 Top Five Injuries Associated with Gymnastics

5.4.1 Ankle Sprains and Strains

The most common reported injury overall in artistic and rhythmic gymnastics is to the lower extremity, specifically sprains of the ankle followed by injuries of the knee [4, 8]. While ankle sprains are the most common injury, injuries to the knee are the most common severe injury requiring surgery [7, 8]. Trampoline skills has been shown to be highly associated with knee injuries, including bony injuries [12]. The mechanism of injury for ankle sprains or knee injuries often stems from incorrect landing, which may occur during a tumbling skill on the floor, skills on the trampoline, a landing from the vault, or from a dismount from the beam or bar [7]. If not stabilized appropriately by the core, or if not absorbing the shock correctly through the knees and hips, injury may occur. Less common injuries of the lower extremity include osteochondral defects of the talus, Osgood Schlatter's, patellofemoral syndrome, or injuries to the hip, such as chondrolabral pathology [5, 8].

The common injuries to the lower extremity, the strains and sprains, particularly of the ankle are treated conservatively with rest, physical therapy, and a guided return to sport. Our recommended treatment and return to sport algorithm is listed in Fig. 5.4. If at any point, the athlete is not progressing as expected, the athlete may need further workup and imaging to rule out fracture, chondral injury, peroneal tendon injury, tarsal coalition, or more severe ligamentous or syndes-

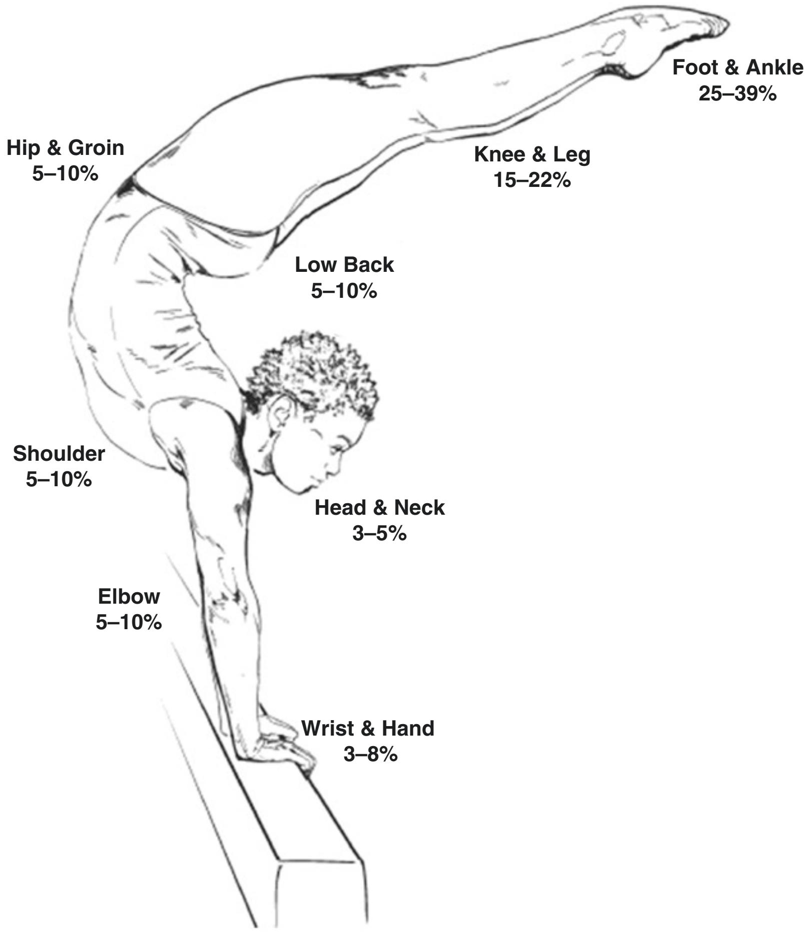

Fig. 5.2 Distribution of injuries by body part in women gymnasts. (© Mark R. Hutchinson, 2021. All Rights Reserved)

motic injury [23–25]. If further workup is negative, the athlete may simply spend more time at the previous step in the algorithm before proceeding. In the acute phase, the athlete will need to rest the extremity and may require limited weight-bearing depending on the severity of the injury. Compressive wraps or stirrup braces may help advance the athlete's progression [23]. The athlete can then progress through physical therapy beginning with range of motion exercises as the swelling decreases, especially since early range of motion has been shown to help with functional outcomes [23]. Therapy should then progress to strengthening and proprioception training, as well as endurance, coordination, and motor control [23, 24]. Ultimately the athlete will begin returning to landing exercises. Once the athlete is prepared to return to gymnastics, he or she should begin with already mastered skills before moving on to more complex movements.

The majority of ankle sprains are treated effectively with conservatively, however up to 20% may develop chronic ankle instability [25]. Surgical treatment is typically not necessary but may be considered in the setting of chronic instability, with a multitude of procedural options depending on the patient and specific injury [23, 25].

5.4.2 Wrist

The wrist is very vulnerable to injuries in male and female gymnasts. Pommel horse and floor exercise are common events causing wrist pain in male gymnasts, whereas floor exercise and vault

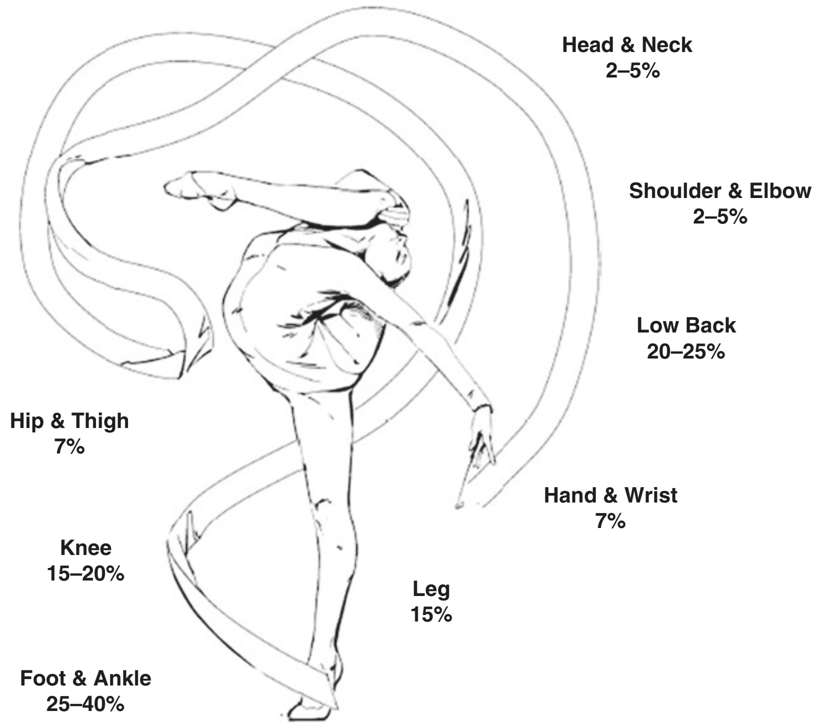

Fig. 5.3 Distribution of injuries by body part in rhythmic gymnastics.

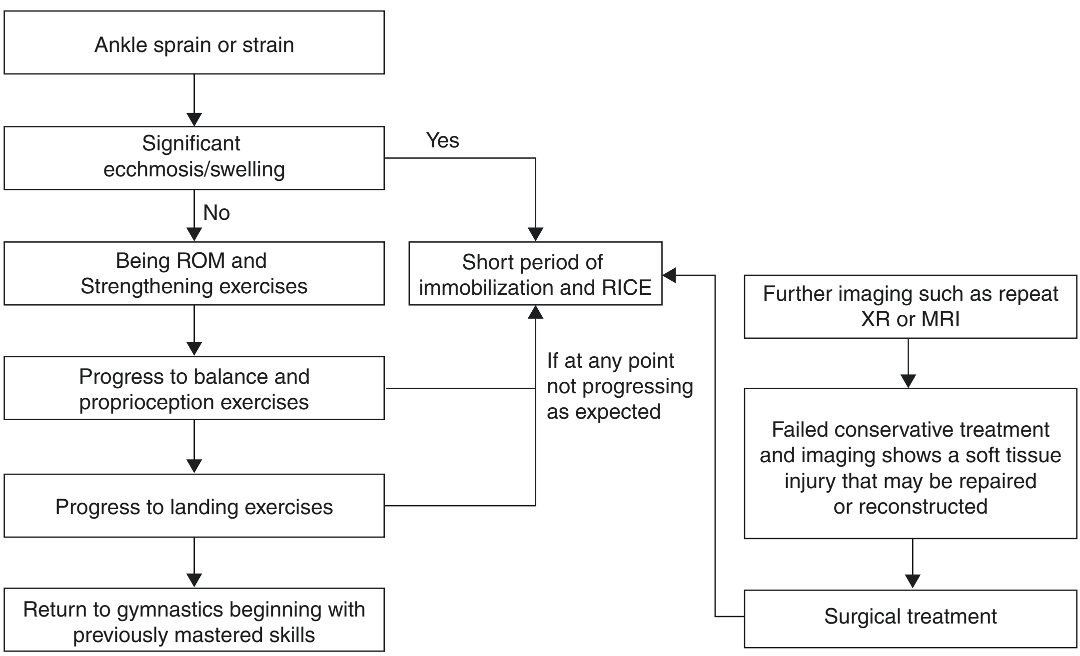

Fig. 5.4 Management of ankle sprain or strain injuries (confirmed no fracture on radiographs).

are the most common in female gymnasts. Rhythmic gymnasts can develop overuse tendinitis secondary to repetitive spirals with the ribbon or when tumbling on the mat. While performing certain skills in gymnastics, the joint experiences forces up to 16 times the body weight making it a common site for injures [11]. The prevalence of wrist pain has been reported as high as 88% in artistic gymnasts [26]. Wrist injuries in gymnasts include distal radial physeal injury, scaphoid stress fracture, triangular fibrocartilage complex (TFCC) tear, grip-lock injuries, ulnar impaction syndrome, and scapholunate dissociation [27].

Distal radial physeal and grip-lock injuries are almost exclusive to artistic gymnastics. Distal radial physis injury occurs in youth athletes with open epiphyseal plates. It results from repetitive compressive loading and shearing forces on the wrist in extension. The distal radius bears about 80% of the axial load during upper extremity weight-bearing activities. In skeletally immature gymnasts, the axial load on the distal radius is higher due to the characteristic negative ulnar variance in this age group [28, 29]. Patients present with chronic dull pain in the dorsal aspect of the wrist. Sport-related activities as floor routines, vaulting or pommel horse precipitates the symptoms. Pain usually is relieved with rest. Chronicity of symptoms should be determined during the assessment, with pain at rest as a sign of severity [27].

Grade of severity is determined by radiographic changes in three stages: stage 1 has an absence of radiographic changes; stage 2 shows cystic changes of the metaphysis and widening of the physis; and in stage 3, there are additional changes in the ulnar variance [7]. Caine et al. described the presence of radiographic abnormalities in 10–85% of gymnastics with distal physeal injury [9]. Magnetic resonance imaging is recommended in the absence of radiographic changes to rule out other causes. The treatment algorithm is shown in Fig. 5.5. For stage 1 and 2 injuries,

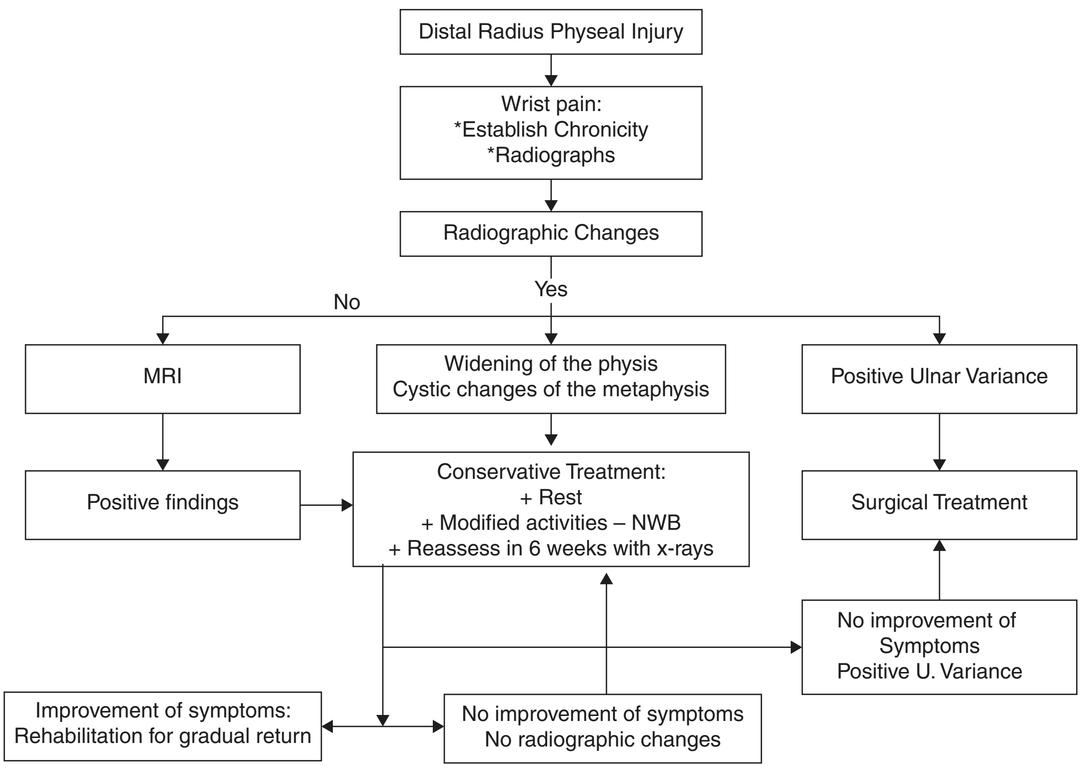

Fig. 5.5 Management of distal radius physeal injury. (© Mark R. Hutchinson, 2021. All Rights Reserved)

treatment is conservative with rest and physical therapy. Rehabilitation should address both upper extremities and focused on strengthening and proprioception. Immobilization using a Gibson brace may help in very symptomatic patients. Reassessment including new radiographs is advised after 6 weeks of treatment to consider progressive increase of training load. Although radiographic changes may take longer to resolve, it is important to monitor patients whose initial radiographs showed significant changes to the physis [2]. Surgery is indicated in advanced cases with stage 3 injuries and positive ulnar variance. Growth arrest is a serious complication in this patient population. Therefore, it is recommended to monitor these patients for at least 1-year post-injury with serial radiographs.

Gymnasts use leather grips or grips with plastic or wooden dowels to protect from friction and increase grip strength. The use of these grips has resulted in a wrist injury called grip-lock injury. This injury occurs when the grip or dowel encircles the bar or a portion of the grip gets caught between the palm and the bar [30]. The gymnast's hand stops rotating, and the body continues moving around the bar, resulting in sprains, tendon injuries, or fractures. Treatment depends on the resulting injury and may require rest and conservative treatment or surgery. Nearly all the patients have reported to return to competition after treatment [27].

Scaphoid stress fractures are commonly reported in elite-level gymnasts, especially in those who rapidly increase of their level of training [31]. Forced hyperextension, radial deviation, and rotation of the wrist generates a significant load over the scaphoid. The majority of these injuries occur at the scaphoid waist, which is the point of greatest bending moment [32]. Scaphoid stress fractures can present in the acute or chronic setting. Patient presents pain over the anatomical snuffbox that is aggravated by extension. The gold standard for diagnosis is MRI. While most injuries can be treated conservatively with good results, with the wrist immobilized in a thumb spica or short arm cast for 8–12 weeks [31], percutaneous fixation may be considered in high-level athletes for earlier rehabilitation and return to sport [33].

5.4.3 Elbow

The high-impact loading of the upper extremity produces high valgus and varus stresses of the elbow during exercises such as bars, tumbling on the floor or trampoline, and landing. Repetitive valgus and varus stress of the elbow results in tension and compression forces over the medial and lateral structures, respectively [7]. The most common elbow injuries are osteochondritis dissecans (OCD) of radiocapitellar joint, ulnar collateral ligament sprains and tears, medial epicondyle apophysitis, or avulsion fractures [34, 35].

Osteochondritis dissecans in the artistic gymnast often involves the capitellum and typically presents between 10 and 14 years of age [36]. Patients present with insidious lateral elbow pain. In more advanced cases, symptoms as catching, locking, or decreased range of motion may be reported. Patients may present with crepitus with range of motion and positive radiocapitellar compression test during the physical exam. Timely diagnosis of capitellar OCD is paramount to avoid premature degenerative changes of the joint [37]. Initial assessment should include three-view plain radiographs including an AP in extension, as well as an AP and lateral in 45° of flexion [38]. Positive findings include lucency of the capitellum, fragmentation and, in advance stages, intra-articular loose bodies. Magnetic resonance imaging is the best modality to evaluate OCD, especially in early-stage lesions with negative radiographs. Treatment is determined by the stability of the lesion, which is shown in the treatment algorithm in Fig. 5.6. Stable lesions have localized flattening, an open physis and adequate range of motion. Conservative treatment is indicated for stable lesions and includes rest and activity modification for 6 months. Outcomes are encouraging with success rates of up to 80% [39]. Unstable lesions consist of intra-articular loose bodies and surgical treatment is indicated in these cases. Athlete may return after achieving full painless range of motion and baseline strength [40].

Ulnar collateral ligament (UCL) injuries are described in artistic gymnasts especially those

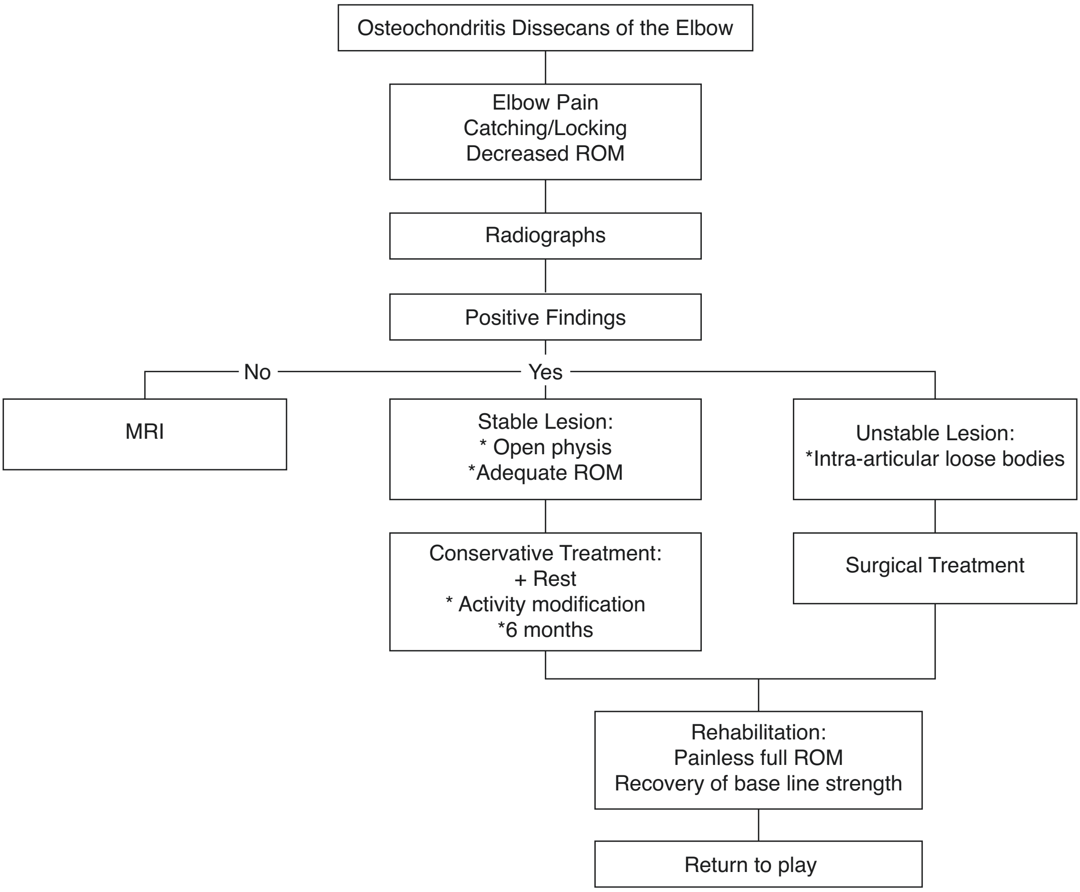

Fig. 5.6 Management of osteochondritis dissecans of the elbow. (© Mark R. Hutchinson, 2021. All Rights Reserved)

with high carrying angles. Repetitive medial tensile stress causes UCL insufficiency or tear. Initial imaging should include AP, lateral, internal/external oblique views, and an oblique axial view with the elbow in 110° of flexion [7]. If medial instability is suspected, MR arthrogram is indicated to evaluate the integrity of the UCL. Partial tears can be treated conservatively with at least 2 months of rest and a progressive strengthening program. Surgical treatment is recommended for complete UCL tear or partial tears that have failed conservative management. The athlete typically cannot return to sport until about 1 year after the procedure, once the athlete has regained full painless range of motion and strength [41].

5.4.4 Shoulder

Shoulder injuries are commonly seen in male's gymnastics and are directly related to the power and repetitive loading demands involved in parallel bars, high bar, pommel horse, and rings. Shoulder injuries include rotator cuff injuries, usually impingement or partial tears, and instability, both chronic and acute [42, 43]. Multidirectional shoulder instability is a unique overuse injury with a high incidence among gymnasts [43]. The hyperlaxity of the athlete and the extreme positions and forces across the shoulder in gymnastics leads to continued microtrauma and plastic deformation of the ligaments. When examining athletes with shoulder pain, signs of generalized laxity, such as the Beighton score, should be assessed. Other specific shoulder tests include apprehension, relocation, load and shift, and Jerk and Kim tests [42]. Initial treatment for many shoulder symptoms consists of periods of rest and restriction from overhead activities. The athlete should have full painless range of motion and full strength before returning to training or competition [44].

5.4.5 Low Back Injuries

Low back pain has been reported in up to 50% of artistic gymnasts and up to 86% of rhythmic gymnasts [21, 45]. Low back pain is the cited as either the most common or second most common injury in rhythmic gymnastics, with one prospective study showing that elite rhythmic gymnasts have an increased relative risk of low back pain compared to other athletes [46]. Young gymnasts have a high incidence of structural pathology related to low back pain, as rapid growth of bone outpaces the growth of soft tissues, including the musculature and ligaments, which may lead to injury at vulnerable areas of growth cartilage and secondary ossification centers [21]. Both artistic and rhythmic gymnasts are at risk for spondylosis and spondylolisthesis due to the repetitive extreme motion, particularly hyperextension, and due to the stresses placed on the lumbar spine, with the spine seeing 6.5–9.2 times their body weight [7, 21, 47]. Though less common, other causes for back pain in gymnasts may include muscle strain, injury to a vertebral body, injury to an intervertebral disc, injury to articular processes, spinous processes, interspinous ligament, or ring apophysis [47].

Differentiating between spondylosis/spondylolisthesis and the other various causes of low back pain begins with physical exam, including pain with single stance extension and point tenderness, but can be confirmed radiographically. The radiographic incidence of spondylosis has previously been found to be as high as 11–16% [7]. Low back pain, spondylosis, and low grade spondylolisthesis are usually treated conservatively with rest for 3–6 months, bracing, and physical therapy [45, 47, 48]. Therapy should address strengthening lumbar extensors, abdominal muscles, as well as range of motion of the lumbar spine and lower extremities [45, 49]. Young patients may also need to followed radiographically to confirm that there is no progression of the slip [45, 47]. The treatment algorithm for low back pain is presented in Fig. 5.7.

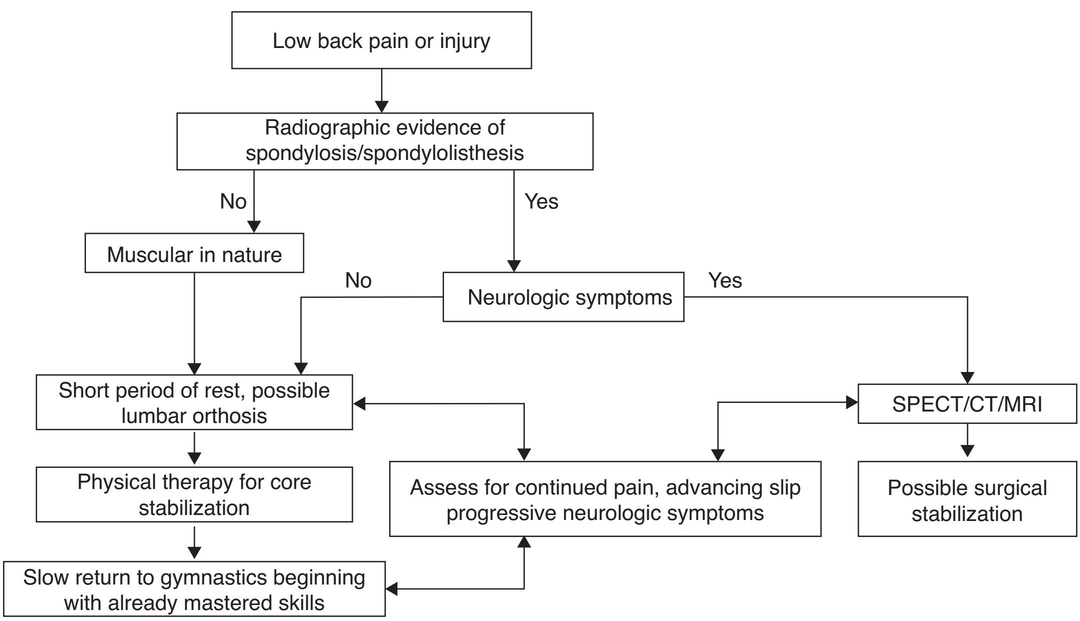

Fig. 5.7 Management of low back pain or injuries. (© Mark R. Hutchinson, 2021. All Rights Reserved)

The algorithm differentiates immediately between low back pain with and without neurologic symptoms. If neurologic symptoms are present, or become present at any time, advanced imaging such as SPECT, CT, or MRI should be obtained. SPECT is best for early diagnosis of spondylosis, while CT is best to assess healing. The athlete should not return to training and competition until completely without symptoms. Additionally, psychosocial factors should be considered in return to play, especially for athletes with back pain and given the high demands placed on young gymnasts [47–49].

Rarely, if pain continues, a slip advances, or neurologic symptoms occur, surgical intervention may be required [7]. Spinal fusion for discogenic back pain is incredibly rare in athletes and is not typically well tolerated; therefore, there is little data on return to play following surgical treatment [45, 47]. Surgical treatment for spondylosis or spondylolisthesis is in situ fusion with autogenous bone graft, and a large majority are able to return to sport with extensive post-operative therapy [48].

More severe or catastrophic injuries of the low back, cervical spine, or head are relatively rare; nonetheless, every safety intervention regarding prevention must be made to avoid them. Collegiate gymnastics and cheerleading account for many of the catastrophic injuries in female sports. The risk is highest when learning a new skill, especially if with inadequate supervision, however is mitigated during training by the use of foam pits, harnesses, and spotters, especially when learning new techniques [8, 47]. If there is concern for an acute head or neck injury, the athlete should immediately be removed from training or competition. Evaluation with imaging or concussion testing should be performed, and further testing determined by those results and the athlete's symptoms. Concussions are the most common injury of the head and neck in gymnastics, which will require removal from training and competition as the athlete completes a concussion-specific return to sport protocol [7]. Athletes must undergo full rehabilitation of balance and any vestibular-ocular deficits before returning training, especially with the twisting and inverting aspects of the sport [7].

5.5 Prevention

Targeting injury prevention in artistic and rhythmic gymnastics as well as trampoline should follow a logical, stepwise approach that is evidenced-based and balances the need for athletes to compete at their highest level with the risk of time-loss from injury. Convincing coaches that injury prevention is important, and often hinges on their understanding of their risk of losing the athlete from performance at the most critical time of competition. In 1992, Willem van Mechelen outlined a logical model that continues to guide prevention strategies nearly 30 years later [50]. First, the extent, incidence, and severity of the injury problem must be established. For gymnastics, this is outlined above in discussing both epidemiology and injury patterns. Next, a fundamental understanding of the etiology and mechanism of injury is essential for creating prevent protocol. For both artistic and rhythmic gymnastics, an important risk factor is the number of repetitions associated with training and competition as well as the timing and intensity of that loading. Another key risk factor is related to acute traumatic events due to falls, landings, and loss of body control while in awkward positions. For trampoline athletes, trained spotters should always be present using a deceleration mat or being able to catch an athlete who leaves the playing field. The third step of van Mechelen's model of injury prevention is the introduction of a targeted intervention that is based on the knowledge gained from steps one and two. The final step is an assessment of the impact of each intervention. Avoiding time-loss from sport and competition, minimizing the expense of injury treatment, and limiting the risk of long-term disability compels the need to invest in preventive measures [50].

Gymnastic equipment has been continuously updated and refined. However, this process has taken place very slowly due to the significant expense and the worldwide nature of the sport. Female gymnasts compete on four apparatuses in artistic gymnastics: floor exercise, vault, balance beam, and uneven bars. The floor exercise is the event where most injuries occur. The floor itself is made of fiberglass or wooden panels placed over springs. The panels are covered with about 5 cm of foam padding. Thinner mats, gaps between landing mats, and hard tumbling surfaces are predisposing factors to injury. The vault horse has also undergone modifications including more padding and a height raise to provide a more optimum platform for the upper extremities. In the past, the rails of the uneven bars were made of solid wood and would break relatively frequently. The newer rails are composed of a layer of wood laminated on fiberglass tubes, which is sturdier and more elastic [51]. Trampolines need to be routinely evaluated for safety and security. Poorly equipped or designed training facilities have been associated with a higher incidence of injuries [52, 53]. Training aids such as foam pits, trampolines, spotting belts, and bungee devices have been added to reduce the risk of injury and enhance the mastering of new skills.

Elite gymnasts practice from 30 to 40 h per week and up to all 12 months per year. National level gymnasts perform between 700 and 1300 elements per day, corresponding to 220,000 to 400,000 elements per year [51]. Gymnastics training is performed in systematic phases consisting of: general preparatory, specific preparatory, precompetitive, and competitive. Higher incidence of injuries have been reported during the onset of training after an enforced break, during competition, or during the initial preparatory phase [54]. This is likely the result of sudden increase in the training load or intensity that predisposes the athlete to injury. Pettrone et al. reported fatigue as a major contributing factor to injury in clubs with training schedules that exceeded more than 20 h a week [55]. To reduce the risk of injuries related to overuse and loading, gymnasts should be provided with adequate opportunity for cross training and rest. Baseline fitness and training intensity should be carefully monitored on a weekly basis. Rapid increases in training intensity should be avoided.

Another potential injury risk for gymnasts occurs when they attempt new or advanced skills which they might not be fully prepared for. If the athlete has underlying motor imbalances, poor coordination, prior incompletely rehabilitated injuries, or simply inadequate skill levels to attempt a new element, they are at increased risk of injury. To decrease the frequency of injuries, it is recommended that each gymnast is prescreened to identify subtle motor skills or balance dysfunction that is either their baseline, related to training or related to prior injuries. When recognized, the abnormalities should be treated with targeted rehabilitation. Once the imbalance or dysfunction is corrected, then the athlete should be allowed to gradually return to play that implements training strategies and a gradual acquisition of skills that is guided by both the coach in communication with the sports medicine professional.

Landing from dismounts, tumbling runs on the floor or trampoline, and vaults place elevated loads on each segment of the lower extremity kinetic chain. With a perfect, well-aligned landing, sequential load-sharing usually allows a successful landing without injury. However, when the athlete lands awkwardly, severe loads can occur at the ankle, knee, or hip leading to a fall and injury. Injury prevention should not only include a gradual and sequential skill acquisition, but also assure that single limb balance and neuromuscular and proprioception training are optimized [23]. The incidence of the common ankle sprain can be minimized by including proprioceptive training into the warm-up program as well as early recognition and treatment of event mild sprains. Since learning new trampoline skills has a high risk of injury, guidelines for safe practice and injury prevention includes dedicated instruction by a qualified instructor prior to attempt at a new skill, all new skill attempts should be performed under direct supervision of a qualified instructor, and athletes should perform skills one at a time [56, 57]. Good control of the core and lower extremity flexibility play a crucial role in injury prevention of both the lower extremity and lumbar spine [7]. Low back pain can be addressed by not only optimizing core strength and balance but also by working with coaches to reduce the total number lumbar hyperextension elements when possible or gradually introducing hyperextension elements into the routine while controlling the number of repetitions when necessary.

In addition, the risk of overuse and more significant injuries can also be minimized by optimizing energy balance for gymnasts as well as early identification of relative energy deficits appropriate intervention and treatment. Energy balance should be routinely self-assessed in all gymnasts assuring appropriate energy intake for their caloric demand and preventing the occurrence zzzzz musculoskeletal injuries such as stress fractures which have been correlated with relative energy deficit and the female athlete triad.

A baseline and continued reassessment of fitness is an important tool in injury prevention. Optimizing general fitness in combination with skill training helps to prepare the gymnast for the high-volume training involved in gymnastics. The gymnastics Functional Measurement Tool (GFMT) is a series of ten sport-specific tests that evaluates the physical fitness of female artistic gymnasts. The tests include rope climb, vertical jump, agility sprint time, and handstand hold time, among others [58, 59]. Ling et al. performed a study in NCAA division I women's artistic gymnastics to determine if the total score on the GFMT can identify gymnasts who are at risk of developing injury. A higher score on the vertical jump test was associated with less trunk injuries. A one-unit increase in vertical jump score was associated with a 30% decrease in trunk injuries [58]. Similarly, in rhythmic gymnastics a program used a jump training program in elite rhythmic gymnasts to improve their leaping ability [60]. In this study, those that underwent the training program that included water training and Pilates improved their leaping ability in terms of height, ground reaction time, and explosive force without the occurrence of injury [60].

Further research including gymnastics-specific testing during the preseason and subsequent injury is recommended to identify risk factors and prevention strategies in the sport. Ultimately, performance optimization programs can be created to improve or build upon gymnastics skills that simultaneously address injury prevent injury. Additional programs or training programs should be created and tested for various skills to improve gymnast's abilities while maintaining safety.

Conclusion

Gymnastics is an exciting but incredibly demanding sport with large popularity worldwide. Artistic gymnastics features explosive power, strength, and torque, combined with extremes of flexibility and balance. Rhythmic gymnastics features grace, balance, coordination, and flexibility. Trampoline demonstrates exciting acrobatics and great heights. Each category of gymnastics has its own unique injury patterns with repetitive overuse as a mechanism pervasive across all categories. Both artistic and rhythmic gymnasts have a high degree of lower extremity injuries, such as sprains and low back injuries. Artistic gymnastics, particularly among male gymnasts, has an increased risk of upper extremity injuries compared to other sports. The injury patterns in trampoline mirror those in artistic gymnastics but appear to have an increased risk of forearm injuries or catastrophic injuries due to the potential energy related to the height the athlete achieves. The relative young age of many gymnasts also increases risk of growth plate injuries. Injury prevention programs should be logical, evidenced-based, and effectively target the most common injury patterns or time-loss injuries in a given sport. For all gymnasts, this should include: optimized and complete rehabilitation of prior injuries; a well-maintained, safe environment of training and competition; attention to loading, training intensity, and fatigue to avoid overuse; and targeted programs to common injuries including landing mechanics and proprioceptive training to avoid ankle and lower extremity injuries as well as core strength and flexibility to avoid lumbar spine injuries.

References

1. Edouard P, Steffen K, Junge A, Leglise M, Soligard T, Engebretsen L. Gymnastics injury incidence during the 2008, 2012 and 2016 Olympic Games: analysis of prospectively collected surveillance data from 963 registered gymnasts during Olympic Games. Br J Sports Med. 2018;52(7):475–81.
2. Benjamin HJ, Engel SC, Chudzik D. Wrist pain in gymnasts: a review of common overuse wrist pathology in the gymnastics athlete. Curr Sports Med Rep. 2017;16(5):322–9.
3. Hinds N, Angioi M, Birn-Jeffery A, Twycross-Lewis R. A systematic review of shoulder injury prevalence, proportion, rate, type, onset, severity, mechanism and risk factors in female artistic gymnasts. Phys Ther Sport. 2019;35:106–15.
4. Sweeney EA, Howell DR, James DA, Potter MN, Provance AJ. Returning to sport after gymnastics injuries. Curr Sports Med Rep. 2018;17(11):376–90.
5. Bolia I, Utsunomiya H, Locks R, Briggs K, Philippon MJ. Twenty-year systematic review of the hip pathology, risk factors, treatment, and clinical outcomes in artistic athletes-dancers, figure skaters, and gymnasts. Clin J Sport Med. 2018;28(1):82–90.
6. Campbell RA, Bradshaw EJ, Ball NB, Pease DL, Spratford W. Injury epidemiology and risk factors in competitive artistic gymnasts: a systematic review. Br J Sports Med. 2019;53(17):1056–69.
7. Desai N, Vance DD, Rosenwasser MP, Ahmad CS. Artistic gymnastics injuries; epidemiology, evaluation, and treatment. J Am Acad Orthop Surg. 2019;27(13):459–67.
8. Hart E, Meehan WP, Bae DS, d'Hemecourt P, Stracciolini A. The young injured gymnast: a literature review and discussion. Curr Sports Med Rep. 2018;17(11):366–75.
9. Caine DJ, Nassar L. Gymnastics injuries. Med Sport Sci. 2005;48:18–58.
10. Caine D, Roy S, Singer KM, Broekhoff J. Stress changes of the distal radial growth plate. A radiographic survey and review of the literature. Am J Sports Med. 1992;20(3):290–8.
11. DiFiori JP, Puffer JC, Aish B, Dorey F. Wrist pain, distal radial physeal injury, and ulnar variance in young gymnasts: does a relationship exist? Am J Sports Med. 2002;30(6):879–85.
12. Grapton X, Lion A, Gauchard GC, Barrault D, Perrin PP. Specific injuries induced by the practice of trampoline, tumbling and acrobatic gymnastics. Knee Surg Sports Traumatol Arthrosc. 2013;21(2):494–9.
13. Kolt GS, Caine DJ. Epidemiology of injury in Olympic sports. 1st ed. Wiley-Blackwell: Oxford, UK; 2009. 536 p.
14. 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.
15. Kolt GS, Kirkby RJ. Epidemiology of injury in elite and subelite female gymnasts: a comparison of retrospective and prospective findings. Br J Sports Med. 1999;33(5):312–8.
16. Caine D, Cochrane B, Caine C, Zemper E. An epidemiologic investigation of injuries affecting young competitive female gymnasts. Am J Sports Med. 1989;17(6):811–20.
17. Dixon M, Fricker P. Injuries to elite gymnasts over 10 yr. Med Sci Sports Exerc. 1993;25(12):1322–9.
18. Cupisti A, D'Alessandro C, Evangelisti I, Umbri C, Rossi M, Galetta F, et al. Injury survey in competitive sub-elite rhythmic gymnasts: results from a prospective controlled study. J Sports Med Phys Fitness. 2007;47(2):203–7.
19. Bak K, Kalms SB, Olesen S, Jargensen U. Epidemiology of injuries in gymnastics. Scand J Med Sci Sports. 1994;4(2):148–54.
20. Westermann RW, Giblin M, Vaske A, Grosso K, Wolf BR. Evaluation of men's and women's gymnastics injuries. Sports Health. 2015;7(2):161–5.
21. Purcell L, Micheli L. Low back pain in young athletes. Sports Health Multidiscip Approach. 2009;1(3):212–22.
22. Marshall SW, Covassin T, Dick R, Nassar LG, Agel J. Descriptive epidemiology of collegiate women's gymnastics injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. J Athl Train. 2007;42(2):234–40.
23. Shawen SB, Dworak T, Anderson RB. Return to play following ankle sprain and lateral ligament reconstruction. Clin Sports Med. 2016;35(4):697–709.
24. Vancolen SY, Nadeem I, Horner NS, Johal H, Alolabi B, Khan M. Return to sport after ankle syndesmotic injury: a systematic review. Sports Health Multidiscip Approach. 2019;11(2):116–22.
25. Giannini S, Ruffilli A, Pagliazzi G, Mazzotti A, Evangelisti G, Buda R, et al. Treatment algorithm for chronic lateral ankle instability. Muscles Ligaments Tendons J. 2015;4(4):455–60.
26. Webb BG, Rettig LA. Gymnastic wrist injuries. Curr Sports Med Rep. 2008;7(5):289–95.
27. Wolf MR, Avery D, Wolf JM. Upper extremity injuries in gymnasts. Hand Clin. 2017;33(1):187–97.
28. Hafner R, Poznanski AK, Donovan JM. Ulnar variance in children—standard measurements for evaluation of ulnar shortening in juvenile rheumatoid arthritis, hereditary multiple exostosis and other bone or joint disorders in childhood. Skelet Radiol. 1989;18(7):513–6.
29. Palmer AK, Werner FW. Biomechanics of the distal radioulnar joint. Clin Orthop. 1984;(187):26–35.
30. Samuelson M, Reider B, Weiss D. Grip lock injuries to the forearm in male gymnasts. Am J Sports Med. 1996;24(1):15–8.
31. Matzkin E, Singer DI. Scaphoid stress fracture in a 13-year-old gymnast: a case report. J Hand Surg. 2000;25(4):710–3.

32. Weber ER, Chao EY. An experimental approach to the mechanism of scaphoid waist fractures. J Hand Surg. 1978;3(2):142–8.
33. Nakamoto JC, Saito M, Medina G, Schor B. Scaphoid stress fracture in high-level gymnast: a case report. Case Rep Orthop [Internet]. 2011 [cited 2020 Jan 20];2011. Available from: https://www.ncbi.nlm.nih.gov/pmc/articles/PMC3504270/.
34. Saluan P, Styron J, Ackley JF, Prinzbach A, Billow D. Injury types and incidence rates in precollegiate female gymnasts: a 21-year experience at a single training facility. Orthop J Sports Med. 2015;3(4):2325967115577596.
35. Overlin AJF, Chima B, Erickson S. Update on artistic gymnastics. Curr Sports Med Rep. 2011;10(5):304–9.
36. Dexel J, Marschner K, Beck H, Platzek I, Wasnik S, Schuler M, et al. Comparative study of elbow disorders in young high-performance gymnasts. Int J Sports Med. 2014;35(11):960–5.
37. Bauer M, Jonsson K, Josefsson PO, Lindén B. Osteochondritis dissecans of the elbow. A long-term follow-up study. Clin Orthop. 1992;(284):156–60.
38. Churchill RW, Munoz J, Ahmad CS. Osteochondritis dissecans of the elbow. Curr Rev Musculoskelet Med. 2016;9(2):232–9.
39. Mihara K, Tsutsui H, Nishinaka N, Yamaguchi K. Nonoperative treatment for osteochondritis dissecans of the capitellum. Am J Sports Med. 2009;37(2):298–304.
40. Baker CL, Romeo AA, Baker CL. Osteochondritis dissecans of the capitellum. Am J Sports Med. 2010;38(9):1917–28.
41. Dugas J, Chronister J, Cain EL, Andrews JR. Ulnar collateral ligament in the overhead athlete: a current review. Sports Med Arthrosc Rev. 2014;22(3):169–82.
42. Levine WN, Prickett WD, Prymka M, Yamaguchi K. Treatment of the athlete with multidirectional shoulder instability. Orthop Clin North Am. 2001;32(3):475–84.
43. Caplan J, Julien TP, Michelson J, Neviaser RJ. Multidirectional instability of the shoulder in elite female gymnasts. Am J Orthop Belle Mead NJ. 2007;36(12):660–5.
44. Gaskill TR, Taylor DC, Millett PJ. Management of multidirectional instability of the shoulder. J Am Acad Orthop Surg. 2011;19(12):758–67.
45. Lawrence JP, Greene HS, Grauer JN. Back pain in athletes. J Am Acad Orthop Surg. 2006;14(13):726–35.
46. Hutchinson MR. Low back pain in elite rhythmic gymnasts. Med Sci Sports Exerc. 1999;31(11):1686–8.
47. Kruse D, Lemmen B. Spine injuries in the sport of gymnastics. Curr Sports Med Rep. 2009;8(1):20–8.
48. Haus BM, Micheli LJ. Back pain in the pediatric and adolescent athlete. Clin Sports Med. 2012;31(3):423–40.
49. Standaert CJ, Herring SA, Pratt TW. Rehabilitation of the athlete with low back pain. Curr Sports Med Rep. 2004;3(1):35–40.
50. van Mechelen W, Hlobil H, Kemper HC. Incidence, severity, aetiology and prevention of sports injuries. A review of concepts. Sports Med Auckl NZ. 1992;14(2):82–99.
51. Sands WA. Injury prevention in women's gymnastics. Sports Med Auckl NZ. 2000;30(5):359–73.
52. Lowry CB, Leveau BF. A retrospective study of gymnastics injuries to competitors and noncompetitors in private clubs. Am J Sports Med. 1982;10(4):237–9.
53. Bale P, Goodway J. Performance variables associated with the competitive gymnast. Sports Med Auckl NZ. 1990;10(3):139–45.
54. Sands WA, Shultz BB, Newman AP. Women's gymnastics injuries. A 5-year study. Am J Sports Med. 1993;21(2):271–6.
55. Pettrone FA, Ricciardelli E. Gymnastic injuries: the Virginia experience 1982–1983. Am J Sports Med. 1987;15(1):59–62.
56. Esposito PW, Esposito LM. The reemergence of the trampoline as a recreational activity and competitive sport. Curr Sports Med Rep. 2009;8(5):273–7.
57. Trampolines at home, school, and recreational centers. American Academy of Pediatrics. Committee on Injury and Poison Prevention and Committee on Sports Medicine and Fitness. Pediatrics. 1999;103(5 Pt 1):1053–6.
58. Ling D, Sleeper M, Casey E. Identification of risk factors for injury in women's collegiate gymnastics with the gymnastics functional measurement tool. PM R. 2020;12(1):43–8.
59. Sleeper MD, Kenyon LK, Casey E. Measuring fitness in female gymnasts: the gymnastics functional measurement tool. Int J Sports Phys Ther. 2012;7(2):124–38.
60. Hutchinson MR, Tremain L, Christiansen J, Beitzel JE. Improving leaping ability in elite rhythmic gymnasts. Med Sci Sports Exerc. 1998;30:1543.

6 Handball

Philippe Landreau, Lior Laver, Romain Seil, and Nebojsa Popovic

6.1 Introduction

Handball, also known as team handball, is a team sport in which two teams of seven players each pass a ball using their hands for the purpose of throwing it into the other team's goal. A standard match consists of two 30-min periods and the team that scores the most goals wins. It has been an Olympic sport since 1972. This sport, which was initially codified in Denmark is most popular in Europe but enjoys popularity in East Asia, North Africa, and parts of South America as well.

The field play formation consists of seven players in four different positions: three back players, two wing players, one lineman, and one goalkeeper. It is a pivot contact and throwing sport that demands physical power and speed.

The game is about scoring as many goals as possible by dribbling, passing, and throwing while opposing players try to prevent these actions. These players therefore need special physical characteristics to generate power and speed.

6.2 Physical Demands and Mechanisms of Injuries

Handball players are tall. Among men, although height differs according to ethnicity and nationality, the average goes from 180 cm to over 195 cm. For women, sizes generally range from 165 to 180 cm. Those who play at a high level are often taller which indicates that a taller handball player is more likely to play at a high level [1]. Body mass varies from 78 kg to over 100 kg in male players, from 60 to 75 kg in female players. In both populations, line players are heavier than wing players, back players, and goalkeepers.

Handball is a high intensity team sport with a large number of actions requiring skill, impulsiveness but also endurance. The game demands an intermittent endurance running ability, interspersed with frequent and short periods of high intensity running coupled with a large amount of very intense game action.

Activities include powerful upper body movements such as high speed ball throwing, tackling as well as lower limb muscle actions

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during vertical jump, side and backward running, acceleration and deceleration, sprinting forward and rapid changes of direction interspersed with intermittent rapid pauses. These actions occur on an adherent floor allowing rapid stops and starts. Players must catch, bounce, and throw a 475 g (male) and 375 g (female) ball, while performing jumps, landings, and rotations, often on one leg, undergoing contact and tackling from the opponent.

Physical demands vary depending on the player's position. Pivots run the shortest distance on the court, but they have a high and intense activity due to the many contacts they undergo. Wing players are the ones who perform the highest intensity runs. They have less contact than the pivots and have lower physiological demands. The physical demands of the back players fall in between but they pass and shoot with high frequency [2].

Handball is therefore a high intensity team sport with a lot of contact. These modalities explain the high risk of trauma existing in this sport. Many mechanisms are possible due to the mode of play, but there are three main high-risk mechanisms for the handball player: throwing, contact, and landing.

These mechanisms can lead to acute injuries or cause repetitive microtraumas with overuse injuries.

6.2.1 Throwing

It is estimated that a professional handball player makes at least 45,000 shots each season [3]. These repetitive movements affect the soft tissues and bone and joint structures of the shoulder and elbow of the dominant limb. These consequences can be physiological and pathological, symptomatic or not. Unlike some sports, the throw is variable depending on the game, throwing upwards, downwards, penalty, etc. and is done extremely quickly with a very short preparation except perhaps for the penalty. The three phases of the throw, wind-up/arm cocking phase, arm acceleration phase, and deceleration phase can be responsible for overuse injuries in the shoulder and elbow due to extreme movements, rapid concentric mechanical forces, and eccentrics imposed on anatomical structures, especially if there are risk factors such as muscle weakness, limited mobility, or scapular dyskinesia. In addition to these throwing movements, the repeated hyperextension forces of the elbow are occurring in goalkeepers.

6.2.2 Player Contact

Handball is a contact sport which means that the contacts, under certain rules and restrictions, are allowed. Players on one team attack and move towards the opposite goal while the other team defends. During these actions, even if the players are used to this kind of situation, excessive contacts can still occur. These contacts either lead to direct trauma (e.g., contusions, head and face direct traumas) or generate a situation that can create an imbalance leading to an indirect trauma (e.g., imbalance leading to non-controlled landing which can cause an ankle sprain or a knee ACL injury).

6.2.3 Landing

Landing is an essential part of the game of handball as a lot of actions, especially when attacking and shooting, requires jumping skills. The landing can be part of the shooting technique or it can be a more or less controlled fall during an attack or defense action.

In handball, players, especially attacking players, shoot with a flight which allows them to increase the power of their shot, break through the opposing defense and get closer to the goal. This flight is followed by a landing which can be done with more or less rotation of the body (with or without a dive). The possibilities of trauma during these actions are multiple, direct, or indirect.

Another particularly well-known situation is reception on one leg. It is therefore a non-contact injury but often this reception is disturbed by a contact or a prior action, with losing balance. The result is a reception during which, if muscle and proprioceptive controls are poor, rupture of the ACL or ankle sprain can occur. These injuries can also occur during a side-step cutting during a game action.

> **Fact Box**
> *There are three main high-risk mechanisms for the handball player: throwing, contact, and landing.*

6.3 Epidemiology of Handball Injuries

One of the challenges of handball epidemiology is to agree on the definition of injury. It is important not to consider only time-loss injuries because many injuries are chronic, do not necessarily result in stopping the sport but affecting the performance of the player. At the same time, it is also important to consider the severity of these lesions and the time-loss is a good means of this. Reading the epidemiology articles on handball injuries should therefore pay attention to these concepts in order to properly assess and compare studies.

6.3.1 Risk of Injuries in Handball

Handball is one of the Olympic sports with the highest risk of injuries (13–17% of all athletes in Beijing, London, and Rio). In the 2012 Olympics, handball had the fourth highest injury score (22%) after taekwondo (39%), football (35%), and BMX (31%) and was ahead of other team sports like basketball (11%) and volleyball (6.9%) [4]. For some it would even be the highest risk ball sport [5].

The risk of injuries is usually represented by the number of injuries occurring during a training or competitive match reported per 1000 exposure hours. The results in the literature are variable probably because some report only lesions causing time-loss and others also include overuse. In a prospective study collecting injuries among high-level handball players over six major tournaments, it was shown that the incidence of time-loss injuries was as high as 40 injuries per 1000 match hours for men and 36 injuries per 1000 match hours for women but the overall incidence of injury (not only time-loss injuries) was on average 108 injuries per 1000 player hours [6]. These high rates has been confirmed recently in a large study in German league (750 players) [7]. During one season, the incidence was around 80/1000 h. Eighty percent of the players sustained an injury during the season, which represented 52 injuries per team.

Injuries occur more during matches than during training, especially during high-level competitions. For example, ACL ruptures occur much more frequently during games than during training. During matches, injuries occur more often in attacks with possession of the ball, offensive part of the game. There is no consensus as to exactly when injuries occur during the match, first half or second half [8].

Line players seem to have the highest risk of injury, which can be explained by the intensity of the play of these players with many changes of direction and contacts and counterattacks [8]. Other studies report that back players are the most at risk, confirming that direct confrontations and contacts are a risk of injury [9].

In an epidemiological study performed during the last World Championship in 2017, 45.3% of all injuries resulted from contact between players, 10.8% were as a result of non-contact trauma (sudden onset) and 26.9% were overuse injuries (gradual onset) [9]. In the same study, older players were at higher risk of injury and players who received a 2-min suspension were also at higher risk, which points to the importance of game aggressiveness in the risk of injury.

6.3.2 Injury Severity

Handball is therefore a provider of a high rate of injuries but also of severe injuries. Usually, the severity of injuries is categorized by the length of absence caused by the trauma: minor injury

(1–7 days absence), moderate injury (8–21 days absence), and major injury (>21 days). The incidence of major injuries ranges from 5% to almost 50% in the literature [6, 10] and re-injuries are frequent. The knee, ankle, and head injuries are those that cause the longest absences from the field.

6.3.3 Anatomical Locations of Handball Injuries

There is no consensus on the accurate percentage of traumatic injuries per body part as the numbers depend on how they are collected; training and match or only competition, one or several seasons or during a tournament, etc. Nevertheless, the authors generally agree on the proportions (Fig. 6.1).

In the literature, the most common types of acute injuries in handball are contusions, strains, and sprains. Fractures and dislocations are less common. The knee and ankle are the most common sites for acute injuries, while overuse problems primarily affect the knee, lower leg, and shoulder [11].

The majority of acute traumatic injuries occur to the lower limb, ankle injuries being the most common, knee injuries being the most severe, particularly anterior cruciate ligament (ACL) ruptures. In fact, the majority of ankle injuries, often sprains, require a few days, or even 2 or 3 weeks to stop playing. In contrast, a rupture of the ACL takes 6–12 months for a return to sport and competition. In the upper limb, acute lesions mainly affect the hand and the shoulder.

Chronic injuries represent 20–35% of all handball injuries [12]. These figures are undoubtedly underestimated because the majority of studies collect injuries resulting in time-loss. Lower leg pain (shin-splints) seem to be the most frequent chronic lesions [12]. The jumper's knee is a common pathology in handball players, especially males (30% against 10 in females) [13]. In

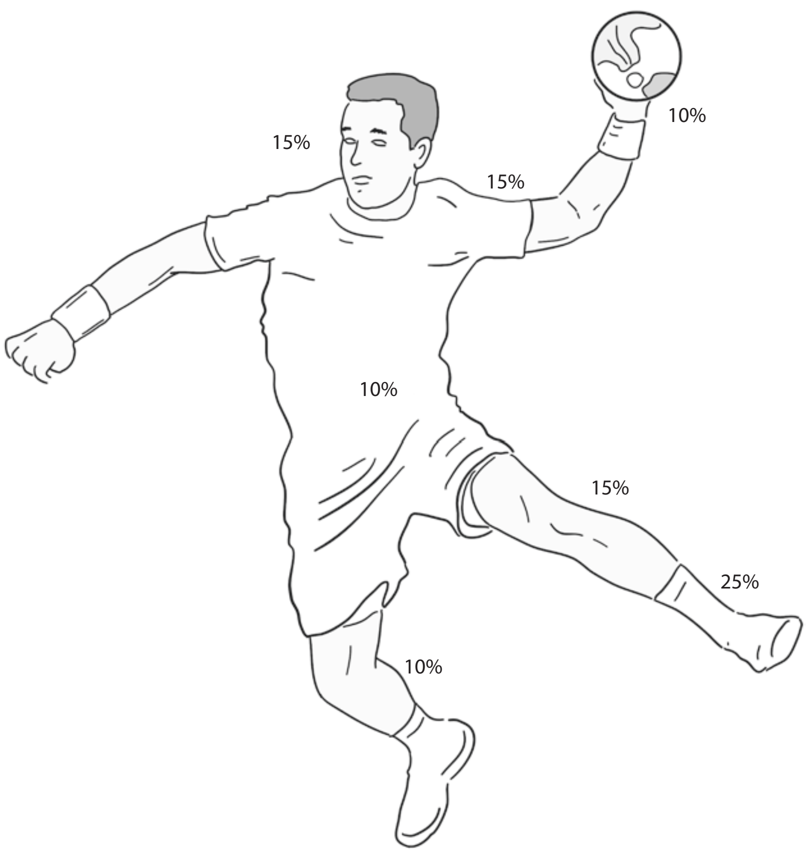

Fig. 6.1 Injury frequency in handball. These percentages are variable from one study to another one but the majority of epidemiologic publications are reporting the same proportion

the upper limb, shoulder injuries are the most frequent chronic injuries [14] followed by elbow injuries, in both field players and goalkeepers [15, 16].

Another interesting question is the risk of re-injuries and the risk of medium and long-term sequelae for handball players. There are fewer studies than football on this subject. Nevertheless, some papers have shown an increased risk of re-injuries after two or more injuries [12]. The risk of osteoarthritis of the knee is greater after ACL reconstruction, which is known, but this risk also seems to exist, even in the absence of a specific lesion, on certain joints such as the hip [17].

6.4 Common and Specific Handball-Related Injuries

Multiple injuries, acute or chronic, can occur in handball.

The foot and ankle represent the most frequent localization in the majority of epidemiological series. Besides dermatological and nail problems related to footwear, Turf toes (first ray metatarsophalangeal sprain), metatarsal fractures and dislocations, Lisfranc injuries, stress fractures, plantar fasciitis, sesamoid dysfunctions, and Hallux rigidus can occur. They are not particularly specific and the majority do not generate significant time-loss. The quality of the shoe is extremely important in handball and has to take into account all these handball-related foot problems, acute or chronic.

The hand and wrist are also frequent locations for injuries. In handball, the hand must catch, hold, and throw a 19 cm (male) and 17.5 cm (female) ball in the conditions of a contact and violent sport. Thus, fractures (scaphoid or triquetrum), ligament injuries (triangular fibrocartilage complex ligament (TFCC), and ulnar collateral ligament of the metacarpophalangeal joint of the thumb), joint injuries (capsuloligamentous structures and volar plates), and tendons injuries (flexor and extensor tendons) can occur.

In this chapter, we have specially developed the knee injuries and the shoulder and elbow injuries. The first ones because they are the most severe injuries in handball, the two others because they represent specific lesions and distinct mechanisms in handball particularly as overuse injuries.

6.4.1 Knee Injuries

Knee injuries are frequent in handball and they represent the most severe injuries, both because of the frequently prolonged time to return to sport and the potential consequences in the long term.

A few studies have compared knee injuries in handball and other sports. Majewski et al. [18] documented 17,397 patients with 19,530 sports injuries in 26 different sports over a 10-year period. They reported that 39.8% of the injuries were related to the knee joint. When considering specific knee structures they reported, the highest risk for a structural lesion was seen in handball and volleyball for the anterior cruciate ligament (ACL) and in handball for the posterior cruciate ligament (PCL).

Different acute injuries can occur in handball practice including ACL and PCL tears. The medial collateral ligament tears and the acute lesions of the lateral collateral ligament and posterolateral structures can happen alone or frequently in combination with a cruciate ligament tear. Meniscus injuries are frequent. When there are concomitant with an ACL tear, they can be repaired and they usually don’t modify the delay to return to sport. When they are isolated, they should be repaired if possible in this population of young patients in order to avoid compromising the long-term sport career. The outcomes are longer than for a simple meniscectomy, and they can affect the availability of the player during a season. Patellar tendon ruptures and patella instability can also affect the handball activity. Patellar tendon rupture can be the ultimate consequence in the spectrum of chronic patellar tendinopathy.

Knee overuse injuries are frequent, and they are usually in the second position after shoulder overuse injuries [10, 11]. The quadriceps and patella tendinopathies (“jumper’s knee”) are frequent, up to 30% in male players [13]. Excentric and intrinsic factors can play a role in the incidence of quadriceps and patellar tendinopathy. It appears that the field type and a player's higher explosive strength can be risk factors in jumper's knee [19].

Anterior cruciate ligament (ACL) rupture needs a special consideration as it is one of the main and most severe injuries [20]. The statistics in literature are influenced by the high number of anterior cruciate ligament tears and the time for recovery after ACL reconstruction is between 6 and 12 months.

Injured players report that injuries often occur while performing a cutting movement or on landing from a jump without direct body contact. Studies, particularly those which have analyzed videos of mechanisms of injury observe that ACL injuries in team handball occurred mainly during two mechanisms: a non-contact plant-and-cut movement or when landing from a jump shot [21]. It is usually a plant-and-cut faking movement (to change direction to pass an opponent, for example) or a one-legged landing from a jump shot. In both cases, the mechanism of injury appears to be the same. A consistent pattern is a forceful valgus-rotation with the knee set close to extension. During this valgus collapse with forced internal rotation of the lower leg, the lateral femoral condyle slides posteriorly and leads to the ACL rupture. This very short movement is followed by a reflex-like external rotation of the lower leg which reduces the bones in anatomical position (Fig. 6.2) [22]. It appears that tearing of the ACL occurs at the time when the foot is planted and firmly fixed to the floor. The injured player usually reports that most of the injury occurs in a move performed numerous times previously, but some perturbation by opponent interaction may help to explain the injury. An inadequate preparation, with an unfavorable lower extremity alignment combined with a poor neuromuscular control can contribute to the injury.

In all these situations, the injuries occurred when the foot was firmly fixed to the floor—it can be assumed that the friction between the shoe and the floor surface can also contribute to the mechanism of injury. It has been shown that the risk of an ACL injury in handball is higher on artificial floors (generally having higher friction) than on wooden floors [23].

Fact Box
ACL injuries often occur while performing a cutting movement or on landing from a jump without direct body contact.

There is a gender difference in ACL injury incidence in handball, with female players suffering four to six times more often than the male athletes [14]. The reasons for this gender difference are multifactorial and may include anatomical factors such as valgus and decreased notch width index, hormonal differences and altered neuromuscular, and biomechanical patterns that help create increased anterior and valgus moments around the knee.

In 70% of cases, several other anatomical structures of the knee can be injured in the same time than the ACL (i.e., meniscus, cartilage, posterior cruciate ligament, medial collateral ligament, lateral collateral ligament and posterolateral structures, anterolateral ligament or iliotibial tract). These combined injuries must be treated in the same time.

Important rotational laxity after an ACL tear must be accurately addressed particularly in the setting of a highly demanding and pivoting sport like handball. The risk of imperfect control of the rotational laxity and the risk of re-rupture brought more attention during the last decade particularly in such a sport like handball. Even if there is still no evidence about exact and accurate indications for the lateral procedures (anterolateral ligament reconstruction or lateral tenodesis), one should consider the utility of the lateral extra-articular procedure in some selected cases [24] like high-grade rotation laxity (pivot shift grade 3) or generalized ligamentous laxity.

Surgery in acute phase is rarely indicated as it can increase the risk of arthrofibrosis. However, in some situations, there is no doubt that the surgical treatment cannot be delayed when combined injuries must be treated as soon as possible. These lesions include: dislocated meniscal bucket

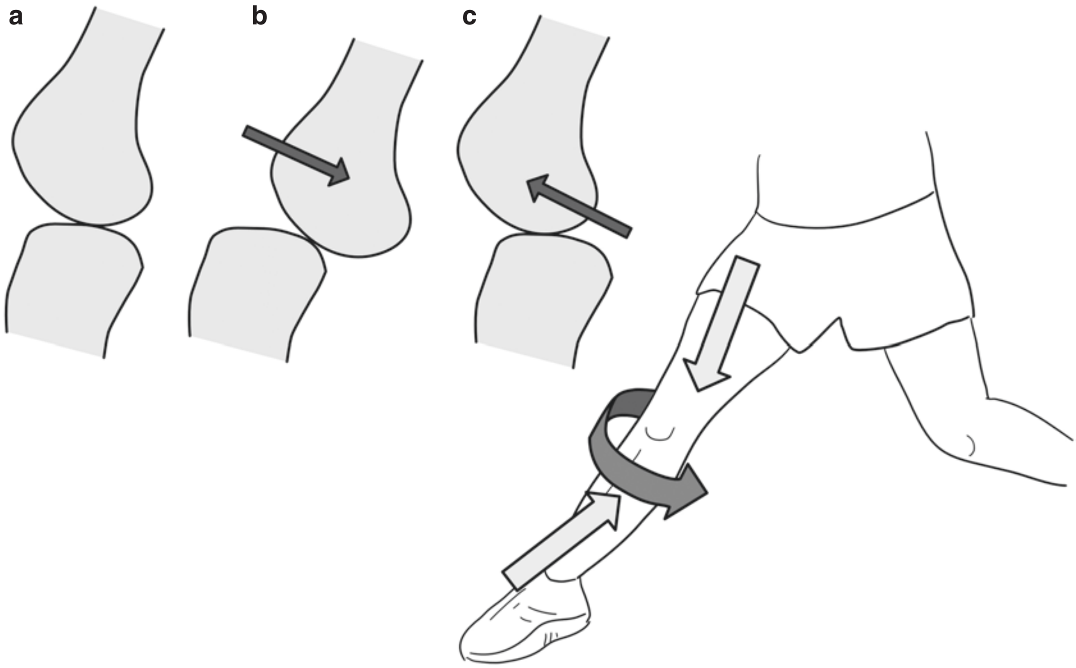

Fig. 6.2 During the valgus collapse with forced internal rotation of the lower leg, the convex lateral femoral condyle slides posteriorly on the convex lateral tibial plateau and leads to the ACL rupture (**b**). This very short movement is followed by a reflex-like external rotation of the lower leg which reduces the bones in anatomical position (**c**). (Adapted from [22])

handle, posterolateral and multiligament injuries, or osteochondral flake fracture.

The surgical treatment is usually indicated in handball population as this sport has a high demand in terms of pivoting and contact and the conservative treatment has a high risk of further instability with meniscus and cartilage lesion. ACL reconstruction with autograft is the usual treatment. Different grafts are possible, hamstrings tendons, bone-patellar tendon-bone, and quadriceps tendon being the most frequently used.

6.4.2 Shoulder Injuries

A professional handball player performs around 50,000 ball-throwing actions in a season, at a speed up to 130 km/h and an angular velocity of 7000°/s, which means 20 turns/min and 150–170 km/h of the throwing arm [25]. It has been shown that the forces applied to the shoulder during the throw can represent up to 1.5 body weight [26]. It is therefore not surprising that shoulder injuries are common in the practice of handball. The lesions can be acute, affecting the dominant or non-dominant shoulder, but most of the time, the overuse due to repetition of throwing movements on the dominant side generates progressive damages of the anatomical structures. In order to throw the ball with maximum speed, the shoulder must reach extreme positions with maximum muscle contraction while maintaining a humeral head in front of the glenoid which achieves a compromise combining mobility and stability, it is the “thrower’s paradox”. These violent movements cause repetitive stress on the anatomical structures. There are still uncertainties about the exact biomechanics of the handball player’s shoulder, but recent studies have brought some information allowing improvement in the treatment and the prevention of shoulder injuries.

From an epidemiological standpoint, it is important to note that to assess handball shoulder injuries; it is not enough to collect the time-loss injuries because some players continue to play despite the pain, with a decrease in performance which influences their participation. Shoulder injuries account for 4–27% of traumatic handball injuries. This disparity is linked to the fact that some studies only collect acute lesions [10, 27]. It is known that around half of handball players complain of chronic shoulder problems with varying degrees of influence on their game and performance [28].

Fact Box
Shoulder injuries are common in the practice of handball. They are caused by repetitive overhead activities leading to overuse injuries rather than by single traumatic mechanism.

Unlike the baseball pitcher, a chronic shoulder model that is probably the most studied in the literature, the handball shoulder is unique in its biomechanical model because it combines repetitive throws in different positions (wide variety of overarm and underarm techniques), combined with direct contact, including blocking. The different phases of the throw remain the same: the wind-up, the early cocking phase, the late cocking phase, the acceleration phase, the deceleration phase, and the follow-through. The first three phases last about 1.5 s (Fig. 6.3). Even though the acceleration phase is very short, 0.05 s, it is during this phase that the greatest speed and change in rotation occur. Anatomical injuries can occur at any time during the throw, but more particularly during acceleration. The throwing movement is actually a complex gesture of the whole body where the energy is transmitted through the "kinetic chain" from the lower limbs to the ball, passing through the trunk, the scapula, the shoulder, the elbow, and finally the hand.

A complete analysis of this "kinetic chain" is essential to understand and treat injuries of the handball player's shoulder [29]. The scapula has a key role in being a link between the trunk and the upper limb. The throwing movement in hand-

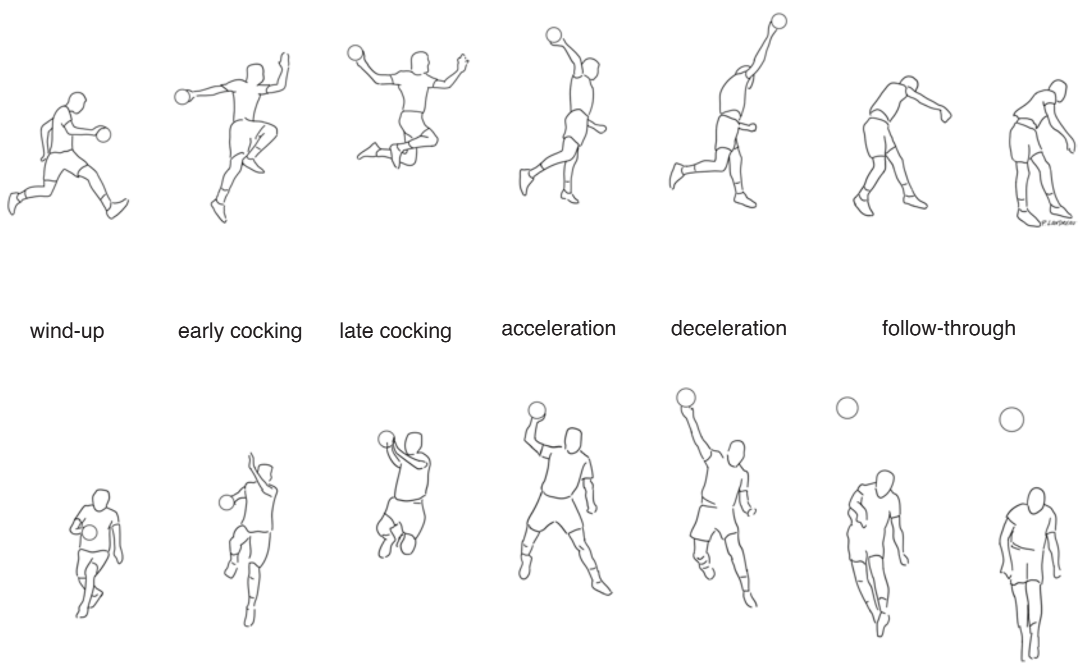

Fig. 6.3 The different phases of the throw in handball: the wind-up, the early cocking phase, the late cocking phase, the acceleration phase, the deceleration phase, and the follow-through. (Adapted from P. Kaczmarek and P. Lubiatowski)

ball is quick and short, the preparation time is less than in baseball as it often happens during the fast actions of the game. The last phase is also shorter for the same reason and to avoid contact with other players. The blocking is also a specificity of handball and can be done with the arm raised, in a horizontal movement, or even at the level of the hip depending on the type of action. The throw in handball is therefore fast, short, variable, and often less predictable than in baseball. The anatomical lesions induced can consequently be variable.

The repeated movements of the shoulder as well as the significant forces applied to this joint can lead to anatomical adaptations. These changes can affect soft tissue but also bone structures. The arc of motion (defined as the angle from maximum internal to maximum external rotation of the abducted arm) is usually shifted posteriorly onto the dominant shoulders of professional handball players, like other throwing athletes, with increased external rotation and decrease internal rotation in abduction. This is the GIRD (glenohumeral internal rotation deficit) [30] (Fig. 6.4). The shoulder laxity is also increased with an overall increase in the arc of motion. The increase in external rotation could also be due to an adaptive increase in humeral retroversion [31], the restriction in internal rotation would be due in this case to soft tissue adaptation. Muscle forces are also modified with sometimes a loss of power of the external rotators with simultaneous increase in force in internal rotation and adduction [32].

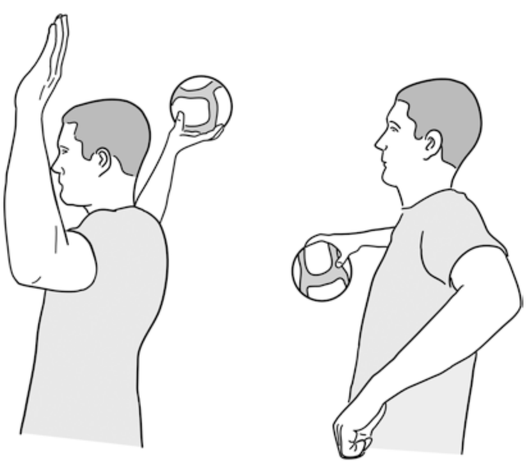

Fig. 6.4 The arc of motion is usually shifted posteriorly onto the dominant shoulders of professional handball players, with increased external rotation and decrease internal rotation in abduction. (*GIRD* glenohumeral internal rotation deficit)

6.4.2.1 The Main Pathologies of the Handball Player's Dominant Shoulder

The internal impingement [33], or postero-superior impingement [34] is frequently observed on the dominant shoulder of the handball player. It is described as a conflict between the articular surface of the cuff and the postero-superior portion of the glenoid. This results in deep lesions of the cuff (Partial Articular Supraspinatus Tendon Avulsion, PASTA) and of the adjacent humeral head as well as lesions of the postero-superior labrum. The mechanism and the cause are debated. For some, it could be due to micro-instability or the consequence of a GIRD secondary to posteroinferior capsular contracture which creates a shift of the glenohumeral center of rotation leading to the internal impingement. This posteroinferior capsular contracture could be the consequence of the repetitive throwing deceleration phase [35]. The treatment of postero-superior impingement is mainly conservative based on rest and physiotherapy techniques. Stretching techniques ("sleeper stretch" and "cross-body stretch") improve the restricted motion such as GIRD. Muscle strengthening, performed after pain and inflammation reduction and after motion improvement, is focused on rotator muscles balance. The return to train and then to practice without limitation is done gradually according to the pain and progression.

It is notable that recurrent anterior dislocations of the shoulder are not frequent on the dominant shoulder of the handball player. This fact is surprising if we consider the repeated movements in ABER (abduction, external rotation). The exact explanation for this clinical challenge is still imprecise and could be linked to the anatomical capsular changes linked to the repetition of throwing gestures. Nonetheless, it is likely that some laxity will develop during repetitive throws, and it may be an underlying cause for throwing kinetic chain alterations leading to pain and decreased performance.

The cuff can present tendinopathies without rupture, related to the overuse, consequence of repetitive throws. Rotator cuff tears can be the result of a chronic internal impingement and are in this case often located at the supraspinatus-infraspinatus junction, but they can be the result of a tensile repetitive overload and micro trauma in eccentric activation [36]. They can affect the subscapularis as well. These rotator tears are usually articular surface lesions, rarely full-thickness. Treatment depends on multiple factors including size, depth, location of the tear as well as the patient's profile. Many partial injuries can benefit from non-operative treatment with resumption of sport. It has been shown that the dominant shoulders of high-level handball players can present variable lesions on MRI (partial cuff tears, bone bruise, SLAP) without necessarily being symptomatic [37]. Care should therefore be taken not to base surgical indications solely on MRI lesions. Surgery is indicated in the event of failure of conservative treatment in cases of partial lesion of the cuff with repair most of the time, simple debridement having shown disappointing results. Total ruptures must be repaired but are of poor prognosis [38].

SLAP lesions are a common pathology among the thrower in general and the handball player particularly. The repetitive external rotation could be a cause for a SLAP lesion with a "peel-back mechanism" that produces the SLAP lesion in the overhead athlete [36]. The isolated surgical treatment of SLAP 2 is more and more debated because the treatment of the cause must be privileged. In addition, it is controversial whether it is better to repair SLAP or to perform biceps tenodesis which is always questionable in a young throwing athlete [39]. SLAP type III and IV in overhead athletes, where there is a "bucket-handle" tear and extension of the lesion into the biceps, requires surgical management.

The long head of biceps can be a source of pain and loss of strength when throwing, especially when there is rotator interval pathology, anterior portion of the supraspinatus, or superior portion of the subscapularis. It can become unstable and develop chronic lesions such as synovitis or tendinopathy. Functional treatment including physiotherapy more or less associated with steroid injections is the first therapeutic option. Severe and recalcitrant lesions can lead to tenodesis.

Scapular dyskinesis is common in handball players. Strictly speaking, it is not a pathology, but its presence increases the risk of shoulder injuries [28]. It is therefore important to detect and prevent it in handball players. There is some controversy over whether scapular dyskinesis is the cause or consequence of other conditions. First described in tennis players, it has been the subject of several publications including the description of SICK scapula syndrome (scapula malposition, inferior medial border prominence, coracoid pain and malposition, dyskinesia of scapular movement) [36]. Different muscle dysfunction can explain this phenomenon with pain on palpation of the pectoralis minor tendon on its insertion on the coracoid. Since the scapula is an essential link during the throw between the thorax and the upper limb, its dysfunction can have significant consequences on the function of the thrower's shoulder [29]. Preventive measures are essential and are now part of the routine preparations for handball players.

6.4.3 Elbow Injuries

Due to the nature of the game of handball, combining speed and contact, the elbow can be the site of acute traumatic injuries as in many sports. However, chronic pathology is common in this joint. The "Handball Elbow" brings together different pathologies developed during repetitive throws and injuries by medial tension, lateral compression, extension overload, and tendinopathies.

As with the shoulder, epidemiological studies only reporting injuries responsible for missed playing time may underestimate the pathology of the elbow in handball players. The prevalence of past and present elbow problems can be as high as 51% in goalkeepers and 32% in field players

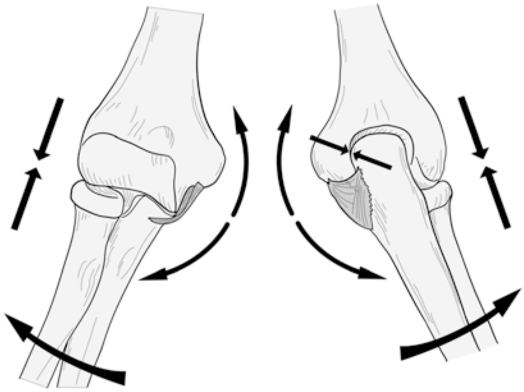

Fig. 6.5 The repeated valgus stress of the elbow results in a significant tension on the medial structures associated with lateral compression phenomena and posteromedial impingement of the olecranon in the fossa

[15, 40]. Two patterns of elbow injuries are described: repetitive throwing for field players and repetitive hyperextension for goalkeeper (Fig. 6.5).

6.4.3.1 Elbow Injuries in Field Player

During each throwing movement, the shoulder transmits to the elbow a large rotational torque, causing valgus stress of the elbow which results in a significant tension on the medial structures associated with lateral compression phenomena especially on the radiocapitellar joint. These phenomena are particularly clear during the late cocking phase and the early acceleration phase. The medial collateral ligament (MCL) is stressed with progressive lesions which can lead to chronic instability, especially if the epicondylar muscles are also damaged with tendinopathy and weakness. Repetitive compressive forces on the lateral compartment, especially in cases of medial deficiency, can lead to progressive cartilage damage causing cartilage or osteocartilaginous foreign bodies and later osteoarthritis. Avascular necrosis or osteochondritis dissecans can also occur.

For the same reasons associated with repeated valgus stresses, the olecranon results in posteromedial compressive forces in the olecranon fossa with impingement. It may develop local inflammation and synovitis, foreign bodies, eventually chondromalacia, osteophytes, and degenerative cartilage lesions. The development of these pathologies can lead to limitation of the extension with pain.

These lesions of the field player can also be aggravated by a violent trauma such as during a blocking, with acute lesion of the MCL and worsen the situation.

6.4.3.2 The Goalkeeper Elbow

The weight of a handball ball is 475 g in male handball (375 g in female). When the goalkeeper makes a stop, the impact is made with a speed of 100–130 km/h. Seventy-five percent of handball goalkeepers have had an elbow problem in their career [41]. The lesions can be acute or chronic. In the majority of the cases, these are hyperextended trauma during blocking shots. The impact of the ball causes stress in valgus with particularly stress on anterior bundles of the MCL at anterior capsule. Micro-ruptures occur in the soft tissues around the elbow (transversal and longitudinal rupture of the flexor-pronator origin and lesion of anterior part of the capsule) with posterior compression lesions on the olecranon and the olecranon fossa including spurs, osteophytes, loose bodies, and calcifications. These lesions are observed on the dominant side but also on the non-dominant side.

The treatment of chronic lesions of the elbow is in the great majority of the cases conservative with adapted rest, physiotherapy, and progressive return to play. Surgical treatment of medial laxity is rarely considered in the event of failure of conservative treatment with the inability to resume exercise.

6.5 Injury Prevention in Handball

Both acute and overuse injuries are a major concern in handball. Two areas in particular have been the subject of recent studies in the prevention of handball injuries, ACL ruptures, and chronic shoulder injuries.

6.5.1 Prevention of ACL Ruptures in Handball

Different factors have been recognized as risk for ACL rupture. The knee valgus (high knee abduction moments) is an important risk factor for ACL tear [42]. For this reason, prevention techniques include a focus on toe landings, a knee-over-toe position and narrow cuts (Fig. 6.6). A key element is the ability of the player to recruit his hamstrings, especially medial as they are a factor in controlling the anterior translation of the tibia. Another important factor is the friction between handball shoes and the playing surface. There are two types of surface: parquet (wooden floor) and artificial floors. Although these have evolved and improved in recent years, artificial floors have been shown to have an increased coefficient of friction and that the risk of ACL injury could be 2.4 times greater when competing on artificial floors in comparison with wooden floors [23]. It is therefore recommended that players have at least two types of shoes, one more "slippery" pair suitable for high-friction floors and another one with more adhesion for slippery floors.

There is a gender difference with female having a higher risk of ACL rupture. The age is also a risk factor with a higher risk in late teens and early 20s. The prevention must be particularly developed in these groups of players with improvement of the neuromuscular function especially in adolescent females [43].

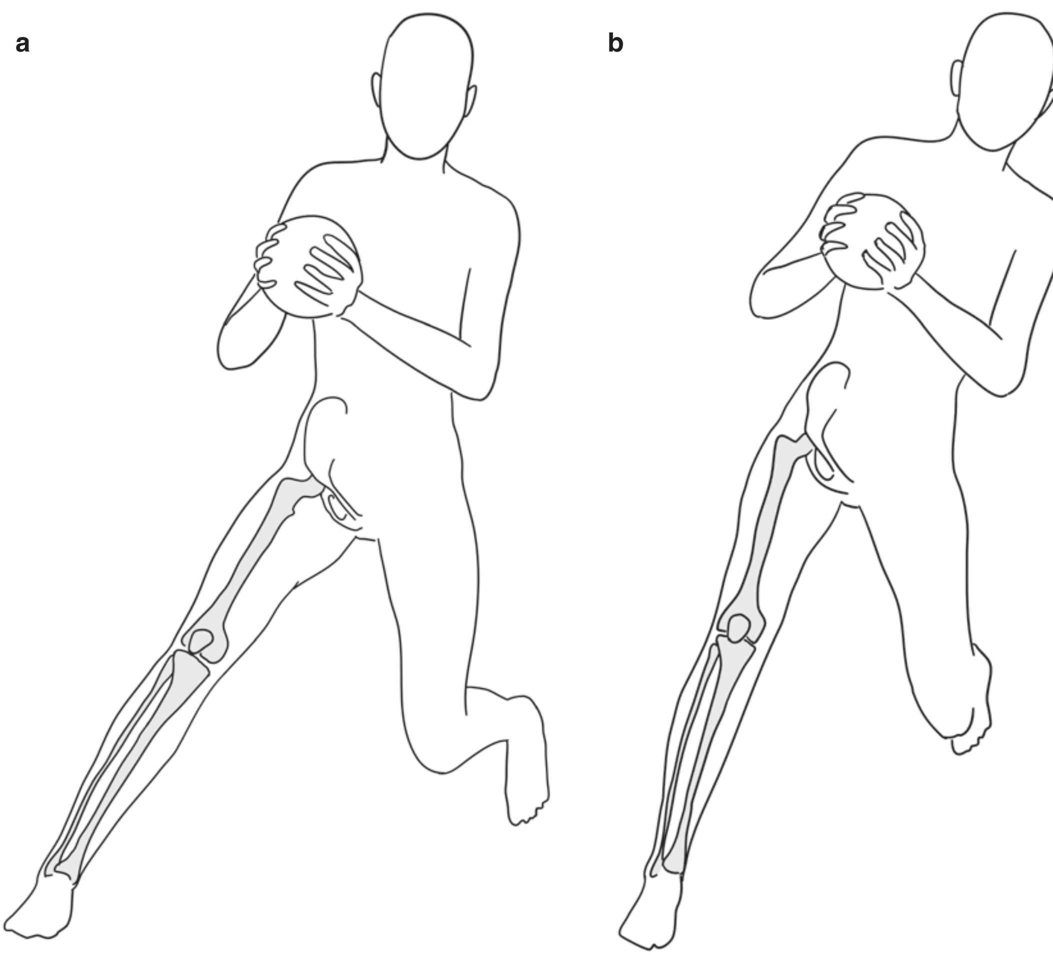

Fig. 6.6 A wide cutting technique is at high risk for ACL rupture (**a**). Prevention include a focus on toe landing, a knee-over-toe position and narrow cutting technique (**b**). (Adapted from Kristianslund et al. [42])

Prevention programs have been shown to be effective in reducing ruptures up to 50% [43]. The goal of these neuromuscular training programs is to increase the muscle strength and to improve the postural balance control and the muscle coordination during high-risk movement for non-contact ACL injury. They includes, under supervision, multiple exercise modalities such as core exercises, balance/coordination, jump training, and muscle strengthening.

6.5.2 Prevention of Shoulder Overuse Injuries

Chronic shoulder injuries are extremely common in handball. It has been shown that over 90% of the throwing shoulders had abnormal magnetic resonance imaging findings (and 37% were symptomatic) [37, 44].

At the elite level, 50% of players experience a shoulder problem during a season, and in general, between 23% and 28% struggle with a shoulder problem affecting their performance and participation [28, 45].

The reduction of internal rotation (GIRD) and increase of external rotation is usual in handball dominant shoulder and this is considered as soft tissue and bony adaptation. However, it has been shown that reduced IR and total ROM (range of motion) can be correlated to shoulder problems [28, 46]. The glenohumeral rotation strength is considered as a risk factor as well [47]. The knowledge about correlation between scapular dyskinesis and shoulder injuries is controversial but it seems that in some groups of players, the scapular dyskinesis can lead to overuse injuries of the shoulder in handball. Beside these factors, the handball load, particularly if it is a rapid sport load can lead to shoulder injuries [47].

Handball shoulder injury prevention programs [48] include exercises aiming at increasing glenohumeral internal rotation (sleeper and cross-body stretch), external rotation strength, and scapular muscle strength.

In general, these prevention programs, neuromuscular training and structured warm-up programs, must be implemented in the youngest in association with information and education that allows players to better adhere to these programs and to avoid injuries with longer term chronic lesions. These programs must be particularly implemented before and during the periods of high participation like championships and other tournaments. In addition, prevention must be specific to the position of each player in the team as the physical demands are significantly different [2].

Take-Home Messages

Handball is a pivot contact and throwing sport that demands physical power and speed. It is a high intensity team sport with a large number of actions requiring skill, impulsiveness but also endurance. It is one of the Olympic sports with the highest risk of injuries. The knee and ankle are the most common locations for acute injuries, while overuse problems primarily affect the knee, lower leg, and shoulder. The majority of acute traumatic injuries occur to the lower limb, ankle injuries being the most common, knee injuries being the most severe, particularly anterior cruciate ligament (ACL) ruptures. ACL injuries often occur while performing a cutting movement or on landing from a jump without direct body contact. Shoulder injuries are common in the practice of handball, and they are most frequently overuse injuries. The repeated movements of the shoulder as well as the significant forces applied to this joint can lead to anatomical adaptations with modification of the total range of motion and pathologies which do not always correlate with the symptoms. The elbow is frequently affected as well with overuse lesions results of repetitive valgus in field players and hyperextension stress in goalkeeper.

The prevention of injuries in handball, particularly in the field of ACL ruptures and chronic shoulder injuries, has grown considerably in recent years and has already shown its effectiveness by implementing neuromuscular training and structured warm-up programs. With the increasing load and intensity in handball, better conditioning and training programs in the future are needed to improve the prevention of these injuries.

References

1. Ziv G, Lidor R. Physical characteristics, physiological attributes, and on-court performances of handball players: a review. Eur J Sport Sci. 2009;9(6):375–86.
2. Karcher C, Buchheit M. On-court demands of elite handball, with special reference to playing positions. Sports Med. 2014;44(6):797–814.
3. Popovic N. Sportske povrede u rukometu Beograd. Sportska Knjiga. 1986;92–101.
4. Engebretsen L, Soligard T, Steffen K, et al. Sports injuries and illnesses during the London Summer Olympic Games 2012. Br J Sports Med. 2013;47:407–14.
5. Åman M, Forssblad M, Henriksson-Larsén K. Incidence and severity of reported acute sports injuries in 35 sports using insurance registry data. Scand J Med Sci Sports. 2016;26(4):451–62.
6. Langevoort G, Myklebust G, Dvorak J, Junge A. Handball injuries during major international tournaments. Scand J Med Sci Sports. 2007;17:400–7.
7. Luig P, Bloch H, Burkhardt K, Klein C, Kühn N. VBG-Sportreport 2017—Analyse des Unfallgeschehens in den zwei höchsten Ligen der Männer: Basketball, Eishockey, Fußball und Handball. Hamburg: VBG; 2017.
8. Bere T, Alonso JM, Wangensteen A, et al. Injury and illness surveillance during the 24th Men's Handball World Championship 2015 in Qatar. Br J Sports Med. 2015;49(17):1151–6.
9. Tabben M, Landreau P, Chamari K, et al. Age, player position and 2 min suspensions were associated with match injuries during the 2017 Men's Handball World Championship (France). Br J Sports Med. 2019;53(7):436–41.
10. Seil R, Rupp S, Tempelhof S, Kohn D. Sports injuries in team handball. A one year prospective study of sixteen men's senior teams of superior non-professional level. Am J Sports Med. 1988;28:681–71.
11. Clarsen B, Bahr R, Heymans MW, et al. The prevalence and impact of overuse injuries in five Norwegian sports: application of a new surveillance method. Scand J Med Sci Sports. 2015;25:323–30.
12. Møller M, Nielsen RO, Attermann J, Wedderkopp N, Lind M, Sorensen H, et al. Handball load and shoulder injury rate: a 31-week cohort study of 679 elite youth handball players. Br J Sports Med. 2017;51(4):231–7.
13. 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. 2005;33:561–7.
14. Myklebust G. Team handball (handball). In: Caine D, Harmer PA, Schiff MA, editors. Epidemiology of injury in Olympic sports, vol. 16. Oxford: Wiley-Blackwell; 2009. p. 260–76.
15. Popovic N, Ferrara MA, Daenen B, Georis P, Lemaire R. Imaging overuse injury of the elbow in professional team handball players: a bilateral comparison using plain films, stress radiography, ultrasound and magnetic imaging. Int J Sports Med. 2001;22:60–7.
16. Tyrdal S, Bahr R. High prevalence of elbow problems among goalkeepers in European team handball—'handball goalie's elbow. Scand J Med Sci Sports. 1996;6:297–302.
17. L'Hermette M, Polle G, Tourny-Chollet C, Dujardin F. Hip passive range of motion and frequency of radiographic hip osteoarthritis in former elite handball players. Br J Sports Med. 2006;40:45–9.
18. Majewski M, Susanne H, Klaus S. Epidemiology of athletic knee injuries: a 10-year study. Knee. 2006;13:184–8.
19. Visnes H, Aandahl HÅ, Bahr R. Jumper's knee paradox—jumping ability is a risk factor for developing jumper's knee: a 5-year prospective study. Br J Sports Med. 2013;47:503–7.
20. Laver L, Myklebust G. Epidemiology and injury characterization. In: Doral MN, Karlsson J, editors. Sports injuries: prevention, diagnosis, treatment and rehabilitation. Berlin: Springer; 2014. p. 1–27.
21. 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;38:2218–25.
22. Seil R, Nührenbörger C, Lion A, Gerich T, Hoffmann A, Pape D. Knee injuries in handball. Sports Orthop Traumatol. 2016;32(2):154–64.
23. Olsen OE, Myklebust G, Engebretsen L, Holme I, Bahr R. Relationship between floor type and risk of ACL injury in team handball. Scand J Med Sci Sports. 2003;13:299–304.
24. Sonnery-Cottet B, Thaunat M, Freychet B, Pupim BH, Murphy CG, Claes S. Outcome of a combined anterior cruciate ligament and anterolateral ligament reconstruction technique with a minimum 2-year follow-up. Am J Sports Med. 2015;43(7):1598–605.
25. Fieseler G, Hermassi S, Hoffmeyer B, Schulze S, Irlenbusch L, Bartels T, Delank KS, Laudner KG, Schwesig R. Differences in anthropometric characteristics in relation to throwing velocity and competitive level in professional male team handball: a tool for talent profiling. J Sports Med Phys Fitness. 2017;57(7–8):985–92.
26. Michalsik LB, Aagaard P. Physical demands in elite team handball: comparisons between male and female players. J Sports Med Phys Fitness. 2015;55(9):878–91.

27. Giroto N, Hespanhol Junior LC, Gomes MR, Lopes AD. Incidence and risk factors of injuries in Brazilian elite handball players: a prospective cohort study. Scand J Med Sci Sports. 2017;27:195–202.
28. Clarsen B, Bahr R, Andersson SH, Munk R, Myklebust G. Reduced glenohumeral rotation, external rotation weakness and scapular dyskinesis are risk factors for shoulder injuries among elite male handball players: a prospective cohort study. Br J Sports Med. 2014;48:1327–33.
29. Kibler WB, Kuhn JE, Wilk K, Sciascia A, Moore S, Laudner K, Ellenbecker T, Thigpen C, Uhl T. The disabled throwing shoulder: spectrum of pathology-10-year update. Arthroscopy. 2013;29:141–61.
30. Reagan KM, Meister K, Horodyski MB, Werner DW, Carruthers C, Wilk K. Humeral retroversion and its relationship to glenohumeral rotation in the shoulder of college baseball players. Am J Sports Med. 2002;30:354–60.
31. Pieper HG. Humeral torsion in the throwing arm of handball players. Am J Sports Med. 1998;26:247–53.
32. Wilk KE, Meister K, Andrews JR. Current concepts in the rehabilitation of the overhead throwing athlete. Am J Sports Med. 2002;30:136–51.
33. 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;18:963–75.
34. Walch G, Boileau P, Noel E, Donell ST. 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.
35. Burkhart SS, Morgan CD. The peel-back mechanism: its role in producing and extending posterior type II SLAP lesions and its effect on SLAP repair rehabilitation. Arthroscopy. 1998;14:637–40.
36. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology. Part III: The SICK scapula, scapular dyskinesis, the kinetic chain, and rehabilitation. Arthroscopy. 2003;19:641–61.
37. Jost B, Zumstein M, Pfirrmann CW, Zanetti M, Gerber C. MRI findings in throwing shoulders: abnormalities in professional handball players. Clin Orthop Relat Res. 2005;434:130–7.
38. Payne LZ, Altchek DW, Craig EV, Warren RF. Arthroscopic treatment of partial rotator cuff tears in young athletes. A preliminary report. Am J Sports Med. 1997;25:299–305.
39. Schrøder CP, Skare Ø, Reikerås O, Mowinckel P, Brox JI. Sham surgery versus labral repair or biceps tenodesis for type II SLAP lesions of the shoulder: a three-armed randomised clinical trial. Br J Sports Med. 2017;51(24):1759–66. https://doi.org/10.1136/bjsports-2016-097098.
40. Popovic N, Lemaire R. Hyperextension trauma to the elbow: radiological and ultrasonographic evaluation in handball goalkeepers. Br J Sports Med. 2002;36:452–6.
41. Tyrdal S, Olsen BS. Hyperextension of the elbow joint: pathoanatomy and kinematic of ligament injuries. J Shoulder Elb Surg. 1998;7:272–83.
42. Kristianslund E, Faul O, Bahr R, Myklebust G, Krosshaug T. Sidestep cutting technique and knee abduction loading: implications for ACL prevention exercises. Br J Sports Med. 2014;48(9):779–83.
43. Myer GD, Sugimoto D, Thomas S, Hewett TE. The influence of age on the effectiveness of neuromuscular training to reduce anterior cruciate ligament injury in female athletes: a meta-analysis. Am J Sports Med. 2013;41(1):203–15.
44. Myklebust G, Hasslan L, Bahr R, Steffen K. High prevalence of shoulder pain among elite Norwegian female handball players. Scand J Med Sci Sports. 2013;23:288–94.
45. Andersson SH, Bahr R, Clarsen B, Myklebust G. Risk factors for overuse shoulder injuries in a mixed-sex cohort of 329 elite handball players: previous findings could not be confirmed. Br J Sports Med. 2017;51:286.3.
46. Lubiatowski P, Kaczmarek P, Cisowski P, et al. Rotational glenohumeral adaptations are associated with shoulder pathology in professional male handball players. Knee Surg Sports Traumatol Arthrosc. 2018;26(1):67–75.
47. Moller M, Attermann J, Myklebust G, Wedderkopp N. Injury risk in Danish youth and senior elite handball using a new SMS text messages approach. Br J Sports Med. 2012;46:531–7.
48. 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. 2016;51(14):1073–80.

7 Ice Hockey

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7.1 Introduction

Ice hockey is currently played in over 76 countries, with a continued growth in popularity. In the United States, more than 12,590 players younger than 19 years of age seek medical attention at emergency departments annually for ice hockey-related injuries [1]. From 1990 to 2006, the number of injuries in athletes aged 9–14 years increased by 163%, and these numbers continue to rise [2]. This escalation is likely explained by increased exposure from amplified youth hockey participation.

Injuries are seen at an even higher frequency at the elite levels [3]. Differences in equipment regulations, such as the lack of full facial protection, lead to increased facial and dental trauma [4]. More lenient rules relating to body checking and fighting result in more physical play, and a higher risk of injury [5, 6]. Last, increased player size, skating speed, and puck velocity leads to a more aggressive nature of play, putting players at an elevated risk of injury.

Hockey is a contact sport, causing a large spectrum of injuries, including serious musculoskeletal trauma and even catastrophic events. An increased risk of injury has been shown to correlate with contact sports where the rules allow and encourage significant contact [7]. Ice hockey-related injuries continue to be a significant concern given the growing popularity and intrinsically physical nature of the sport.

Ice hockey is known for its skillful and dynamic fast-paced play, with ten skaters wearing knife-like skate blades and moving at high speeds on ice, approaching 30 miles per hour (mph) [8]. Players use composite material sticks to propel a frozen rubber puck at velocities of over 100 mph. Body contact, body checking, and falls result in high-speed collisions into hard plastic composite boards and glass which encloses the playing surface. Fighting still occurs, and is even encouraged, in some junior leagues and at the professional level. For these reasons, it comes as no surprise that ice hockey has one of the highest injury rates in competitive sports [1–3, 8–10]. See Fig. 7.1, which demonstrates the playing environment where these athletes compete.

Epidemiology studies defining common injuries in ice hockey athletes remain paramount. Team physicians can utilize this information to be better prepared for common injuries.

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Fig. 7.1 In-game photograph of ice hockey athletes competing

Furthermore, this information helps guide preventative strategies in the areas of education, coaching, rule enforcement and modification, equipment improvement, and sportsmanship. In this chapter, an overview of the epidemiology, diagnosis, and management of common ice hockey injuries is reviewed.

7.2 Mechanism and Sport-Specific Risk of Injury

An understanding of the common mechanisms and unique risks of injury experienced by ice hockey players is vital to allow for targeted interventions and risk reduction. While a majority of injury mechanisms are similar across all levels of play, some unique differences exist due to variations in equipment regulations, rules of play, and level of competition. Multiple studies have found collisions with another player to be the leading mechanism of injury across all age groups, while contact with the boards, hockey stick, and puck represent other common injury mechanisms [9, 11–13]. Sports performance specialists aim to produce bigger, faster, stronger, and more robust athletes, resulting in competitors who are able to skate faster, hit harder, and shoot the puck at a higher velocity, raising the risk and severity of injury.

Fact Box
A collision with another player is the leading mechanism of injury across all age groups; however, contact with the boards, hockey stick, and puck also contribute to a significant number of injuries [9, 11–13].

7.2.1 Youth and High School Ice Hockey

At the youth and high school level, body checking has been identified as the leading mechanism of injury [3, 5, 6, 14, 15]. Matic et al. reported body checking to be the cause of 46.0% of injuries during high school ice hockey participation [14]. In a similar study, Bartley et al. found checking or being checked contributed to 41.1% of injuries in high school athletes [15]. The resulting injuries were most commonly sustained to the head, face, and shoulder [15].

Emery et al. demonstrated a significant increased risk of injury, and three times increased risk of concussion, in youth athletes playing in a league where body checking was allowed compared to a league where it was not [6]. Similar observations have been made in women's ice hockey, which does not allow checking at any level of play. Decloe et al. found that female youth athletes sustained fewer injuries than male athletes of the same age group, which to some extent can be attributed to the elimination of checking [16].

In a study of 33,233 youth hockey athletes, Forward et al. found the second most common injury mechanism in both male and female athletes was a fall (14.8% and 25.3%, respectively) [10]. While falls represent a more infrequent injury mechanism at higher levels of play, these findings are not surprising given the comparative lack of coordination and agility in younger age groups. On the contrary, contact with a stick or puck, and fighting are a less frequent injury mechanism, as expected due to regulations that ban fighting and also require full facial protection, mitigating eye, face, and dental trauma [3].

7.2.2 Junior Ice Hockey

Junior ice hockey typically includes 16–21-year-old athletes at various levels of competition based on experience and skill. Similar to youth hockey, collisions with another player and/or the boards represent the most common injury mechanisms [11, 17]. With more inconsistent requirements for full facial protection, and a faster speed of play, stick and puck-related injuries are more prevalent [11, 17]. Furthermore, some junior hockey leagues allow fighting, introducing a new injury mechanism [17].

Tuominen et al. reported 633 injuries prospectively collected over a 9-year period at the International Ice Hockey Federation (IIHF) World Junior Under-20 and Under-18 Championships [11]. Body checking was the most common injury mechanism, attributing to 32% of all injuries. Checking to the head and body was the most common mechanism for concussion. Interestingly, 63% of concussions were caused by illegal hits, with a penalty called in only 53% of these cases. While contact with the boards was a less common injury mechanism, it contributed to 30% of shoulder injuries and 18% of head injuries. Contact with a stick (13%) or puck (13%) represented the next most common injury mechanism. Puck (31%) and stick (18%) contact was the most common mechanism for finger, hand, and wrist injuries. Furthermore, in the World Junior Under-20 tournaments, stick contact caused 20% of the injuries, a majority of which were to the head and face (78%), accounting for almost half of the diagnosed facial lacerations.

Similar results were reported by Stuart et al., who published a 3-year prospective study of 142 injuries in a US Junior A league [17]. The most common mechanism of injury was a collision, resulting in 51% of all injuries. Collisions with another player (24%) and with the boards (22%) were a more prevalent injury mechanism than collisions with the ice (4%) or the goal (1%). Contact with a stick caused 14% of all injuries, representing a more common mechanism than being struck by the puck (11%), or a skate (3%). Fighting, which is introduced in some leagues, was responsible for 3% of all injuries. Furthermore, it should be noted that 6% of the reported injuries were secondary to an overuse mechanism. As the level of competition increases, overuse injuries become more common due to single sport participation and year round play.

7.2.3 College Ice Hockey

Similar to other levels of play, collision with another player or the boards remains the most common injury mechanism [8, 13, 18–21]. Differences at this level include slightly higher rates of injury as a result of overuse and fighting compared to younger athletes [8, 19]. Furthermore, stick-related injuries are less common, likely due to the regulations now requiring full facial protection [8]. Interestingly, a high percentage of injuries sustained during practice were found to result from a non-contact mechanism [13].

Agel et al. reported a descriptive epidemiology study of collegiate men's ice hockey injuries from the National Collegiate Athletic Association Injury Surveillance System (NCAA ISS) between 1988 and 2004 [13]. Player contact was associated with 47.7% of all reported in-game injuries, while 21.6% of injuries resulted from contact with the boards or glass. Contact with a puck (7.0%) or stick (6.4%) were less common injury mechanisms. Player contact (60.2%) and contact with the boards or glass (26.3%) was the most common injury mechanism for concussion. A non-contact mechanism of injury was significantly more common in practice (32.0%) than during a game (9.7%).

In a similar study focusing on women's ice hockey, Agel et al. reported data from the NCAA ISS between 2000 and 2004 [22]. Comparable to male athletes, player contact (46.8%) and contact with the boards or glass (17.1%) were the most common injury mechanisms. Only 6.5% of injuries were the result of contact with a stick, and 3.0% from contact with a puck. As with male athletes, the most common mechanism for concussion was contact with another player (42.1%); however, the second most common mechanism was contact with the ice (28.1%). Similar to males, a non-contact mechanism of injury was significantly more common in practice (41.0%) than during a game (11.0%).

Flik et al. reported 113 injuries in American collegiate men's ice hockey athletes, finding collisions with an opponent (32.8%) and the boards (18.6%) to be the most common injury mechanism [8]. Interestingly, overuse (8.0%) was the next most common. Contact with the puck (6.2%) and stick (1.8%) were less prevalent. In a study of Canadian intercollegiate injuries over a 6-year period, Pelletier et al. reported 6.5% of injuries were from a fighting mechanism [19]. This is not surprising given the higher prevalence of fighting in the Canadian leagues.

7.2.4 Professional Ice Hockey

Injuries at the most elite level are frequent, likely due to the high-speed aggressive nature of play, and differences in equipment regulations and rules. While body checking remains the most common injury mechanism, stick and puck-related injuries are seen at a higher frequency. At the professional level, players are not required to wear full facial protection. Furthermore, pucks are shot at a higher velocity, resulting in frequent injuries.

Tuominen et al. reported on 528 injuries over a 7-year period at the IIHF Adult World Championship tournaments and Winter Olympic Games [23]. The three most common injury mechanisms were body checking (27.2%), stick contact (21.1%), and puck contact (12.3%). Molsa et al. reported similar findings in a cohort from Finland, with 29.7% of injuries from checking, 14.6% from stick contact, and 7.9% from puck contact [24]. Lorentzon et al. reported on injuries in the Swedish elite team over a 3-year period, and found the most common injury mechanisms were checking (32.9%), player contact (25.0%), puck contact (14.5%), and stick contact (11.8%) [25].

Pettersson et al. published a 4-year prospective study on 376 injuries sustained by the Swedish elite team [26]. In this cohort, contact with the stick was the most common injury mechanism (26.1%), contributing to 56.9% of injuries to the head and face. Player contact was the second most common injury mechanism (23.9%), contributing to 22.9% of injuries to the head and face. This was followed by puck contact, which resulted in 16.0% of all injuries, and 14.7% of injuries to the head and face.

7.3 Common Ice Hockey-Related Injuries

Clinicians and trainers should be familiar with common ice hockey injuries to allow for skillful evaluation and management. Acromioclavicular joint injuries are extremely common from collisions [18, 27, 28]. Femoroacetabular impingement and core muscle injury are two of the most common overuse injuries, especially at the elite level [29–34]. The medial collateral ligament is the most commonly injured knee structure [8, 13, 18, 20, 35]. Quadriceps contusions, which are frequently seen, require early recognition and urgent treatment to limit time lost [10, 36, 37]. The epidemiology, diagnosis, treatment, and return to play for these injuries are detailed in this section.

7.3.1 Acromioclavicular (AC) Joint

Acromioclavicular (AC) joint injuries are common among ice hockey athletes [18, 27, 28]. These injuries typically occur from contact with another player or the boards [18, 27]. A direct blow to the player's shoulder drives the acromion inferiorly leading to AC joint capsule and/or coracoclavicular (CC) ligament injury [38, 39].

Examination of a suspected AC joint injury requires removal of the shoulder pads to expose both the injured and contralateral shoulder for comparison. The AC joint should be palpated for tenderness and inspected for deformity. Shoulder range of motion (ROM) should be examined and may be limited in more severe cases. The cross body shoulder exam is typically positive. A complete examination of the shoulder should be performed to rule out concomitant injuries and neurovascular compromise.

AC injuries are graded according to the Rockwood Classification as follows: Type 1—AC ligament sprain, normal X-ray (Fig. 7.2a, b), Type 2—AC ligaments ruptured, CC ligaments intact, CC distance elevated <25% (Fig. 7.2c), Type 3—AC and CC ligaments disrupted, CC distance elevated 25–100% (Fig. 7.2d), Type 4—AC and CC ligaments disrupted, posterior displacement into trapezius muscle, Type 5—AC and CC ligaments disrupted, disruption of delto-trapezial fascia, CC distance elevated 100–300%, Type 6—AC and CC ligaments disrupted, inferior displacement.

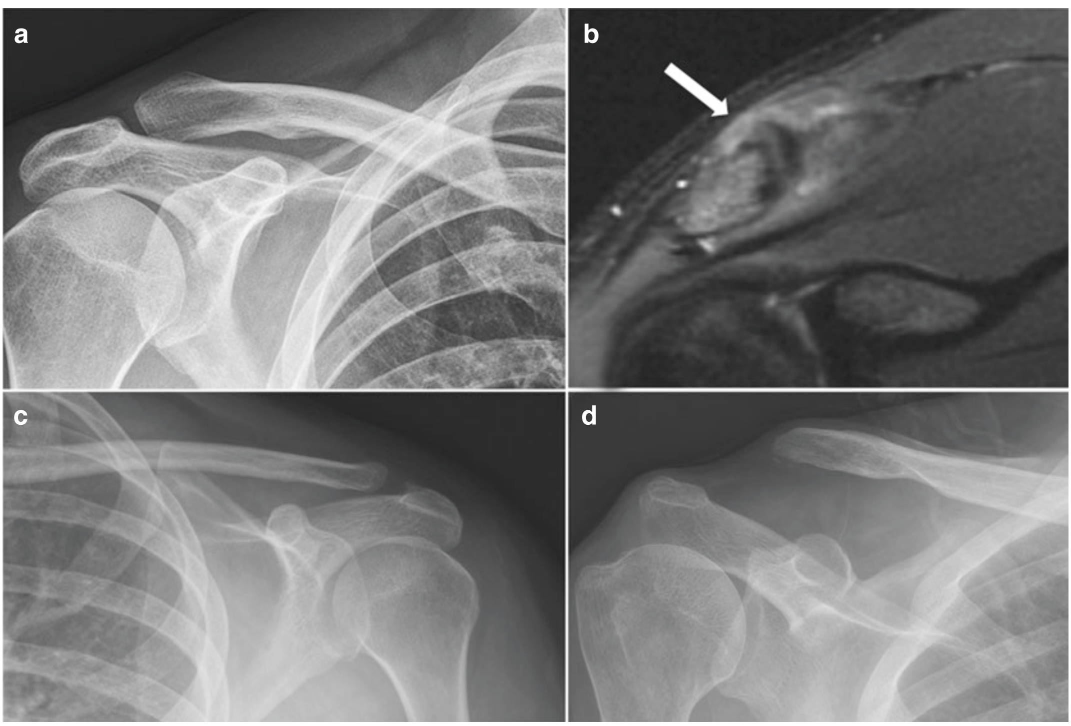

Fig. 7.2 AC joint injuries. (**a**) Normal appearing AP radiograph of the right AC joint, (**b**) with coronal inversion-recovery MRI demonstrating capsular edema (arrow) without widening of the joint, consistent with a type I injury. (**c**) AP radiograph of the left AC joint demonstrating widening without superior displacement, consistent with a type II injury. (**d**) AP radiograph of the right AC joint demonstrating superior displacement of the clavicle, consistent with a type III injury

X-rays of the shoulder and AC joint should be obtained to rule out fracture and assess the severity of the injury. Radiographs should include an AP view of the bilateral AC joints to assess degree of displacement compared to the contralateral side, and an axillary lateral view, which is required to diagnose type IV injuries. Magnetic Resonance Imaging (MRI) is reserved for athletes with a type IV–VI injury, allowing for assessment of disruption through the deltotrapezial fascia and concomitant injuries. The majority of AC joint injuries in ice hockey athletes are types I–III [28].

Treatment and return to play vary based on the severity of injury. Type I and II injuries do well with conservative treatment consisting of a sling, cryotherapy, and early shoulder ROM [18, 38, 40]. While treatment of type III injuries remains controversial in elite athletes, recent studies have shown successful outcomes following non-operative management in hockey players [41, 42]. Type IV–VI injuries require surgical intervention with a CC ligament reconstruction [41].

Return to play varies significantly, with decisions made on a case-by-case basis. For athletes suspected of having a low-grade injury, who report the ability to affectively resume play, immediate return can be considered with caution. Return to play criteria includes return of full pain-free shoulder ROM, normal strength, and the ability to perform sport-specific skills without limitation. In general, elite athletes return to sport after 0–4 weeks for type I and II injuries, 4–8 weeks for type III injuries, and 4–6 months post-operatively for type IV–VI injuries [27].

7.3.2 Femoroacetabular Impingement (FAI)

Hip-related injuries, both acute and chronic, represent a large proportion of ice hockey injuries [13, 20, 43–45]. Ice hockey has the second highest rate of hip and groin injuries in NCAA athletics [29]. Players are at increased risk for the development of femoroacetabular impingement (FAI), especially goaltenders due to abnormal joint positioning during the butterfly technique [30, 31]. Furthermore, in a sprint start, skaters place their hip in external rotation and abduction during the push-off phase, and internal rotation through increasing hip flexion during the recovery phase, placing the player in an “at-risk” hip position [32]. Youth ice hockey athletes have significantly higher alpha angles compared to an age-matched control of youth skiers [31]. FAI is caused by developmental abnormalities, including loss of femoral head-neck offset (cam lesion), focal or global acetabular over coverage (pincer lesion), or a combination of the two. Abnormal contact between the proximal femur and acetabulum, and repetitive entry of a cam lesion into the hip joint, typically with hip flexion and internal rotation, causes labral degeneration/tearing and shearing of the transitional zone and adjacent cartilage [46, 47].

FAI typically presents as an overuse injury; however, in rare instances can present following an acute injury. Athletes typically report pain in the anterior hip or groin and may demonstrate the location through cupping their hands around their groin demonstrating the “C-sign” [48]. Examination should involve both the injured and contralateral hip to use as a control. Pertinent findings include limited hip ROM, specifically in flexion and internal rotation. The FADIR test involves taking the hip through flexion, adduction, and internal rotation with reproduction of symptoms. An extensive exam of the hip, groin, and abdomen is required as multiple diagnoses can mimic FAI and/or are common secondary injuries (Fig. 7.3).

Since FAI typically presents with a gradual insidious onset, in-game imaging is rarely required, except in the case of an acute injury. Radiographs should include an AP pelvis and AP and lateral views of the hip to assess for morphologic characteristics of FAI (Fig. 7.4a). Computed tomography (CT) is useful to confirm bony morphology (Fig. 7.4b). An MRI of the affected hip should be obtained to assess the labrum, cartilage, and evaluate associated soft tissue pathology (Fig. 7.4c, d).

Initial treatment should consist of conservative management. Non-operative treatment includes, physical therapy, deep tissue massage, avoidance of aggravating activity when possible, and NSAIDs [43, 49]. Intra-articular corticosteroid

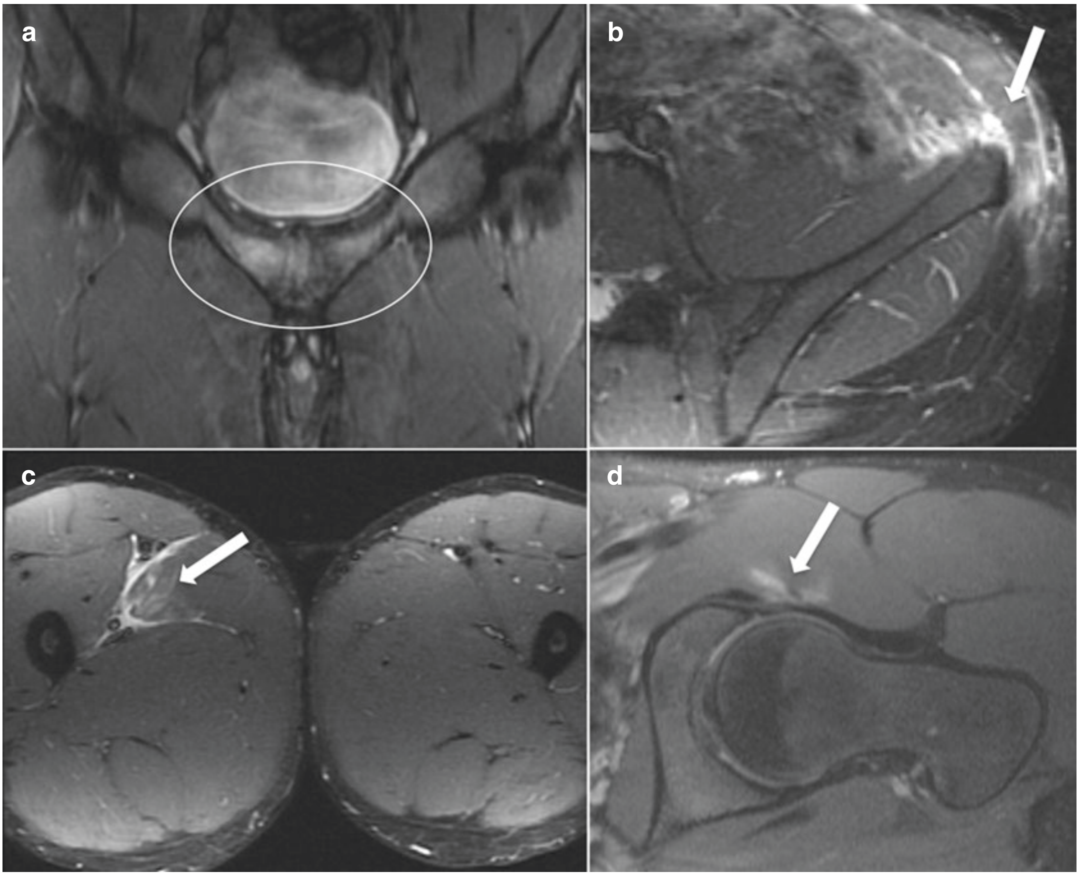

Fig. 7.3 FAI-associated injuries and differential diagnoses. (**a**) Oblique-coronal inversion-recovery MRI demonstrating edema and subchondral sclerosis at the pubic symphysis (circle), consistent with osteitis pubis. (**b**) Axial inversion-recovery MRI of the abdomen demonstrating extensive edema within the left flank (arrow), consistent with a hip pointer. (**c**) Axial inversion-recovery MRI demonstrating edema within the right adductor muscle (arrow), consistent with an adductor muscle injury. (**d**) Axial-oblique T2-fat-suppressed MRI of the left hip demonstrating edema around the iliopsoas tendon (arrow), consistent with iliopsoas tendonitis

injections can be used in athletes with an insufficient response to conservative measures, or for athletes with more severe symptoms, especially while in-season [50]. Operative management is generally recommended for patients with persistent symptoms at the end of the season to address symptoms, and for hip preservation purposes [51, 52].

Return to play depends on an athlete's clinical situation. For athletes with an overuse type of presentation, restriction of play is dependent on the athlete's level of symptoms and the ability to play affectively. For acute injuries, radiographs should be obtained to rule out a fracture or avulsion, with return to play based on negative radiographs, no concerning exam findings, and the players ability to return. For athletes who undergo operative intervention, return to on-ice drills is allowed around 4 months post-operatively [51].

7.3.3 Medial Collateral Ligament (MCL)

The medial collateral ligament (MCL) is the most commonly injured knee structure during ice hockey participation [8, 13, 18, 20, 35]. MCL injuries are the second most common injury behind concussions in NCAA athletes [20]. These injuries are common from on-ice collisions, causing a twisting valgus force to the knee [18, 20, 53]. Furthermore, they can result from a

Fig. 7.4 Cam-type FAI. (**a**) Elongated neck lateral radiograph of a right hip demonstrating a large anterior cam (arrow). (**b**) Radial reformatted axial CT of a right hip demonstrating an anterior cam lesion with intra-osseous ganglion cyst formation (arrow). (**c**) Sagittal proton density MRI of a left hip demonstrating a full-thickness anterosuperior labral tear (arrow). (**d**) Coronal proton density MRI demonstrating delamination at the chondrolabral junction (arrow)

skater catching their edge on the ice [18]. MCL injuries have been reported to represent 9.2% of all injuries in elite hockey players [8, 25, 35, 54].

Examination requires exposure of both the injured and contralateral knee for comparison. Palpation proximally and distally over the origin and insertion of the MCL may elicit pain. Isolated MCL injuries typically result in localized pain and soft tissue swelling [55]. A valgus stress exam should be performed in full extension and at 30° of flexion, using the contralateral knee as a control. Injuries are graded according to the American Medical Association Classification base on the amount of excessive medial joint gapping compared to the contralateral knee as follows: Grade I (0–5 mm), Grade II (5–10 mm), and Grade III (>10 mm). A complete exam of the knee, including an inclusive ligamentous evaluation should be completed to rule out concomitant injuries.

Imaging includes AP, lateral, and oblique radiographs of the knee to rule out fracture. Valgus stress X-rays may be used, with medial compartment gapping greater than 3.2 mm compared to the contralateral knee at 20° of flexion suggestive of a grade III MCL injuries [56]. For athletes with more severe injuries, MRI may be used to assess extent of the injury and to rule out concomitant injuries. A majority of MCL injuries in ice hockey athletes are grade I or II (Fig. 7.5a). Grade III injuries and distal MCL pathology are associated with a worse prognosis (Fig. 7.5b–d).

Fig. 7.5 MCL injuries. (**a**) Coronal proton density MRI demonstrating a left knee proximal grade I MCL injury (arrow). (**b**) Coronal proton density (arrow) and (**c**) sagittal inversion-recovery MRI sequences demonstrating a right knee proximal grade III MCL injury. (**d**) Proton density coronal MRI sequence demonstrating a right knee distal MCL injury (arrow)

Isolated grade I and II MCL injuries typically respond well to non-operative management [27, 53]. Initial management consists of cryotherapy, compression, and elevation to reduce soft tissue swelling. Early rehabilitation should focus on initiation of active knee ROM to prevent stiffness, strengthening exercises to restore quadriceps function, and avoidance of side-to-side activity, with sport-specific drills as symptoms allow [55]. A hinged knee brace is recommended, especially for grade II and III injuries, but may be discontinued after the competitive season is completed [27, 57]. Grade III and distal MCL injuries have a worse prognosis with non-operative management (Fig. 7.5b–d) [27, 57–59]. Athletes with chronic valgus instability or rotatory instability affecting play may require operative management. The use and efficacy of platelet-rich plasma for high-grade MCL injuries remains controversial [60, 61].

Return to play varies significantly depending on the grade of injury. For athletes suspected of having a low-grade injury, who report the ability to effectively resume play, return to the game can be considered with caution. Return to play criteria includes return of full ROM and strength, no swelling, and evidence of healing of the MCL on physical exam. In general, elite athletes return to sport after 0–2 weeks for grade I injuries, 2–4 weeks for grade II injuries, and 4–8 weeks for grade 3 injuries [27, 62].

7.3.4 Core Muscle Injury

Studies have shown a high prevalence of hip and groin injuries in ice hockey athletes, especially at the professional level [33, 34]. Core muscle injuries have only become well recognized in the sports community over the past decade, and are therefore frequently misdiagnosed. They occur in ice hockey athletes due to the requirement for repetitive sudden change in direction and intense twisting movements. They typically present as overuse injuries; however, in rare instances can present following an acute injury.

Athletes often complain of difficulty twisting and turning towards the effective side. Athletes typically have tenderness to palpation in the lower abdomen about the pubis. Reproduction of pain while performing a resisted crunch should be assessed, as pain with this maneuver is the most common finding in athletes with a core muscle injury. Additionally, attempting to do a resisted single-leg raise on the effected side typically reproduces symptoms. Examination of the hip, groin, and abdomen is required to rule out common pathologies that mimic core muscle injury symptoms (Figs. 7.3 and 7.4).

Since core muscle injuries typically present with a gradual onset, in-game imaging is rarely required. Imaging should start with radiographs of the hip and pelvis, largely to rule out injuries that can mimic a core muscle injury. MRI is sensitive and specific in diagnosing causes for groin pain in athletes and can assess the severity of rectus abdominis/adductor longus aponeurosis injury while identifying other pathology within the pelvis (Fig. 7.6) [63].

Core muscle injuries can be nagging injuries that are difficult to manage in-season. Conservative management consists of activity modification, NSAIDs, and strengthening of the core and contralateral lower extremity. Corticosteroid injections are a controversial treatment option that may be tried in more severe cases, where an athlete wishes to delay surgery and finish the season. For cases not responding to conservative management, surgical repair is required.

Return to play depends on the athlete's clinical situation. For athletes with an overuse presentation, restriction of play is dependent on the athlete's level of symptoms and the ability to play affectively. When an acute injury is reported, radiographs should be obtained to rule of fracture or avulsion, with return to play based on negative radiographs, no concerning exam findings, and the players ability to return. For athletes who undergo surgery, return to on-ice drills is allowed around 3 months post-operatively.

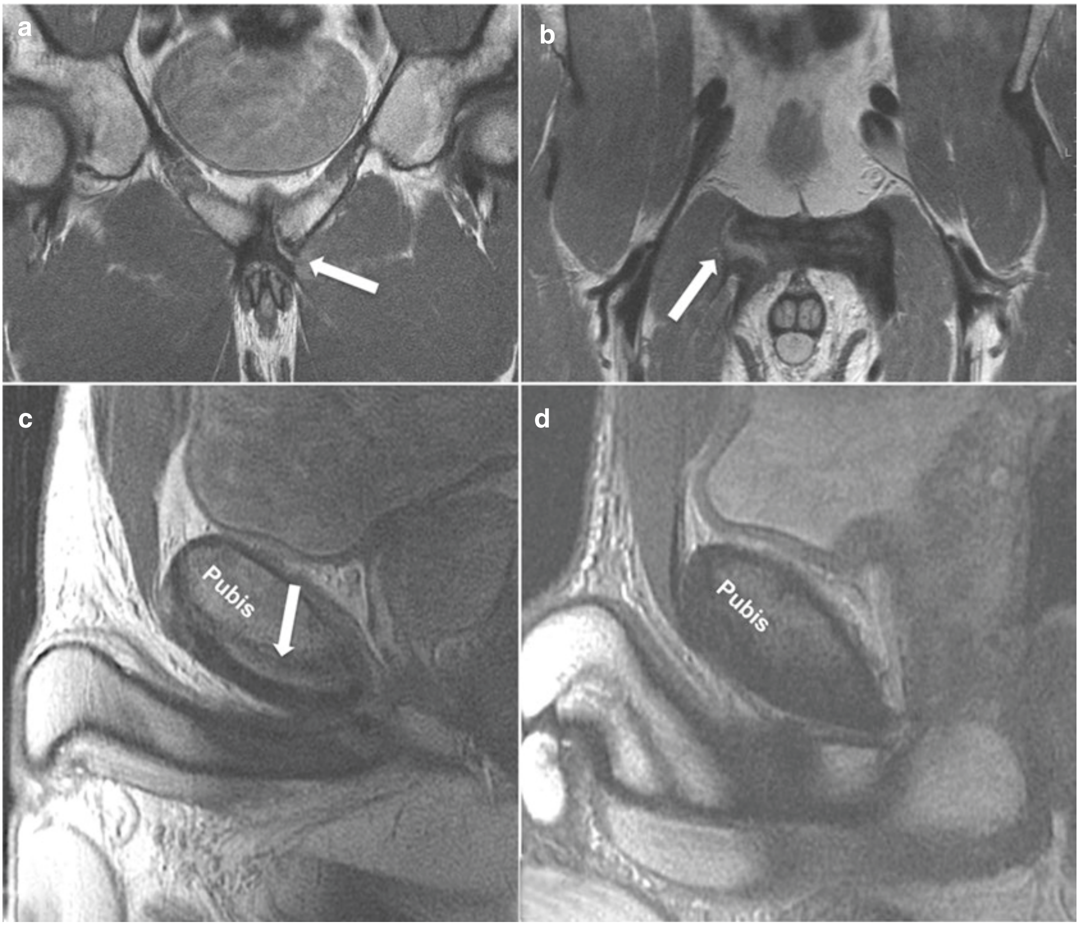

Fig. 7.6 Core muscle injury. (**a**) Coronal-oblique proton density MRI demonstrating a superior cleft sign (arrows) indicating a tear of the adductor aponeurosis, consistent with a low-grade core muscle injury. (**b**) Coronal-oblique proton density MRI demonstrating a high-grade tear of the aponeurosis (arrow) propagating into the adductor tendinous origin. (**c**) Sagittal proton density MRI shows high-grade posterior stripping of the aponeurotic plate from the pubis (arrow). (**d**) Sagittal MRI of the normal contralateral side demonstrating "black to bone" indicating an intact adductor aponeurosis

7.3.5 Quadriceps Contusion

Quadriceps contusions are common, with soft tissue injuries identified as the most common type of injury in both male and female youth athletes [10]. Engebretsen et al. reported injuries during the Winter Olympic Games in 2010, finding contusions to be the most common injury type, with the thigh being the second most common overall injury location [36]. Defensemen are prone to this injury, as the typical mechanism is being struck in the anterior thigh by a puck when a skater drops down to block a shot. Significant bleeding into the muscle can occur in a short period of time, resulting in immediate pain and potential loss of knee ROM secondary to hematoma formation [37].

Examination should include removing the athlete's pads to allow for inspection and palpation of the thigh. Athletes typically have a localized area of tenderness with varying degrees of swelling, and in severe cases a palpable hematoma [37]. Active and passive knee ROM should be assessed. Active knee flexion has been

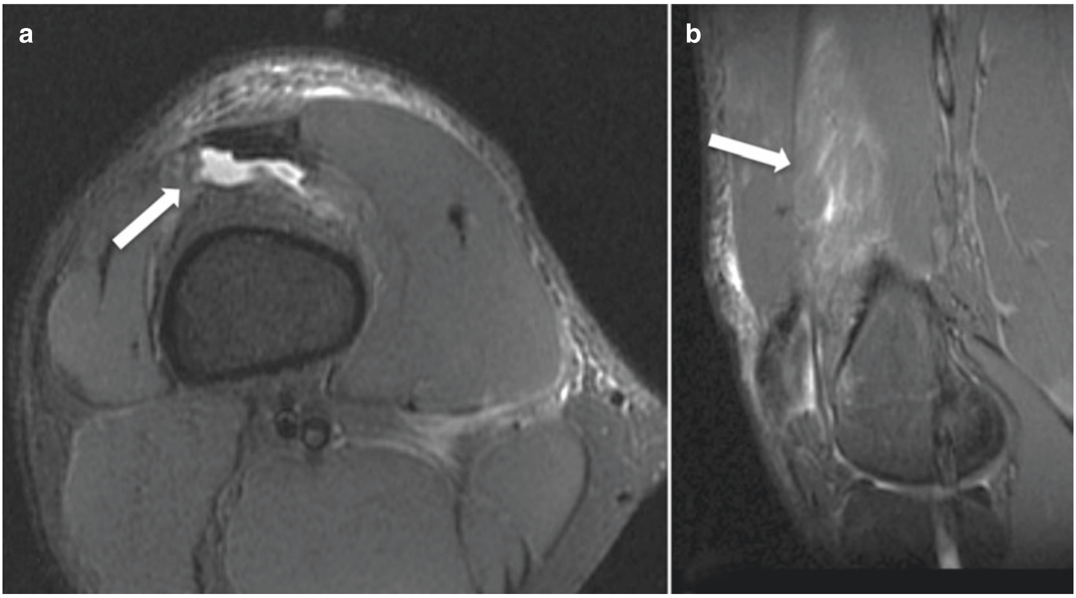

Fig. 7.7 Quadriceps contusion. (**a**) Axial inversion-recovery MRI of the thigh demonstrating a hematoma posterior to the rectus femoris (arrow). (**b**) Sagittal inversion-recovery MRI of the thigh with perifascial edema within the vastus medialis (arrow)

shown to be a prognostic indicator in quadriceps contusions as follows: >90° (mild), 45°–90° (moderate), <45° (severe) [64]. In severe cases, monitoring for compartment syndrome may be necessary.

Radiographs of the femur may be required to rule out fracture in more severe cases. Advanced imaging is rarely required, but can demonstrate the extent of injury and hematoma in severe cases (Fig. 7.7). Follow-up radiographs may be considered for evaluation of heterotopic ossification, which can occur with this injury.

Treatment of quadriceps contusions focuses on decreasing bleeding into the muscle and minimizing knee stiffness. Immediate compression and ice help to limit bleeding and swelling in the thigh. Some advocate for the use of a hinged knee brace with the knee locked at 120° to help limit hematoma formation in severe cases. After 24 h, athletes should start to work on knee ROM, stretching and strengthening of the quadriceps muscle, and low-impact exercise such as jogging in a pool or use of a stationary bike [37].

Return to play is allowed once a skater's quadriceps strength and knee ROM have returned to normal. Ryan et al. reported the average time of disability for contusions: mild—13 days, moderate—19 days, and severe—21 days [64]. In general, ice hockey athletes with access to immediate treatment are often able to return sooner than this time frame, and as soon as a few days in mild and moderate cases. Athletes who struggle to regain their strength and ROM within the first week may require further investigation with advanced imaging.

7.4 Epidemiology of Injury

An understanding of the epidemiology of injuries in ice hockey athletes is important to guide preventative strategies. While the rates of injuries share similarities across all levels of play, some variation is seen due to differences in equipment, rules of play, and level of play. Ice hockey has one of the largest differences in the proportion of player-to-player contact injuries sustained during competition compared to practice at all age levels, with the prevalence of injury increasing at each successive level of competition [15].

7.4.1 Youth and High School Ice Hockey

Even within the youth population, the incidence of injury has been shown to increase with each successive level of participation. Stuart et al. reported athletes 8 years of age and younger sustained 0.8 injuries per 1000 player-hours, compared to 9.3 in high school athletes [65]. This observation is likely due to increased player size, strength, speed, and intensity of competition. Furthermore, injury risk is affected by the different rules at each level and across various leagues. Emery et al. reported a three-times increased risk of concussion and significantly increased risk of overall injury in youth athletes competing in a league where body checking was allowed compared to a league where it was not [6].

The risk of in-game injuries is higher than during practice. Stuart et al. found an injury rate of 10.9/1000 player-hours in games compared to 2.5 during practices in Bantam athletes [65]. Similarly, the Council on Sports Medicine and Fitness et al. found that injury rates for male high school athletes from 2008 to 2012 ranged from 2.03 to 2.56/1000 athlete exposures, compared to a game-related injury rate of 4.18–6.08 [66]. Bartley et al. reported data from the High School Reporting Information Online database for high school athletes from 2005 to 2016, demonstrating 5.35 injuries per 1000 athlete exposures during competition, compared to 0.65 during practice [15].

7.4.2 Junior Ice Hockey

At the Junior level, the increased size and physicality, combined with more inconsistent requirements for full facial protection, relaxed fighting regulations, and a faster speed of play, results in injury rates elevated over those at the youth level [11, 17]. Stuart et al. reported a 3-year prospective study on injury rates in a US league [17]. The authors reported an overall injury rate of 9.4/1000 player-hours. The in-game injury rate was 96.1/1000 player-game hours, 25 times higher than during practice (3.9/1000 player-practice hours). Defensemen had a lower injury rate at 87/1000 player-game hours, compared to forwards with an injury rate of 134/1000 player-game hours. The majority of injuries were rated as mild (58%), while 36% were moderate, and 6% were severe.

Tuominen et al. conducted a 9-year prospective study of injuries at the IIHF Under-18 and Under-20 World Championships [11]. The overall injury rate for male athletes was 11.0/1000 player-games and 39.8/1000 player-game hours. The most common injury was a concussion at the Under-18 World Championships, with an incidence of 4.2 concussions per 1000 player-game hours. Checking to the head and body was the most common mechanism for a concussion, with 63% of concussions caused by illegal hits. Injuries to the shoulder/clavicle were seen at a rate of 6.7/1000 player-game hours, knee injuries at 3.1/1000 player-game hours, and thigh injuries at 2.1/1000 player-game hours.

7.4.3 College Ice Hockey

The ice hockey in-game injury rate is one of the highest in college athletics, while the in-practice injury rate is among the lowest. Flik et al. reported on 113 injuries in American collegiate male athletes [8]. The overall rate of injury was 4.9/1000 athlete exposures, with game injuries 6.3 times more common than practice injuries (13.8/1000 player-games versus 2.2/1000 player-practices). The authors found no significant difference in the injury rates between positions, or between periods of play. The rate of game injuries was slightly higher in the first half of the season (57%) compared to the second half (43%), but this difference was not significant.

Similar studies have reported slightly higher in-game injury rates ranging from 16.27 to 18.69/1000 player-games [13, 18, 20]. Agel and Harvey reported a higher in-game injury rate in men's (18.69/1000 player-games) compared to women's collegiate hockey (12.10/1000 player-games) [20]. The incidence of concussion was 0.72/1000 athlete exposures among men and 0.82/1000 athlete exposures among women. Agel et al. found a similar rate of injury in women's collegiate ice hockey using the NCAA ISS, with an in-game injury rate (12.6/1000 player-games), five times higher than the rate during practice (2.5/1000 player-practices) [22].

7.4.4 Professional Ice Hockey

Injuries at the most elite level are common, in part due to equipment and rule differences and high-speed aggressive nature of play. Ice hockey had the highest rate of injury at the 2010 Winter Olympic Games, with 23% of female and 16% of male athletes reporting an injury [36]. Tuominen et al. reported injuries at the men's IIHF adult World Championship tournaments between 2006 and 2013, finding an injury rate of 52.1/1000 player-game hours [23]. Arenas with flexible boards and glass reduced the risk of injury by 29%.

Other studies report injury rates ranging from 53 to 84/1000 player-game hours [12, 24, 25]. Molsa et al. stratified injuries by severity, reporting 5% of injuries were considered major [24]. The risk of injury in women's international competition has been reported to be much lower at 20/1000 player-game hours [67]. This discrepancy reflects numerous differences between men's and women's games, including player physicality, and variations in rules on body checking and facial protection.

7.5 Injury by Anatomic Location

Although the anatomic distributions of injuries across all ages are similar, subtle differences exist due to equipment and rule variations, as well as differences in physicality and level of play. Understanding the distribution of injuries is vital to allow for targeted interventions and risk reduction. For instance, studies have found the head/face to be the most common location injured, with body checking being the most common injury mechanism. In response, leagues have started implementing rules to limit body checking, fighting, and the requirement for half or full facial protection.

Fact Box
The head/face, shoulder/clavicle, knee, and hip/groin/thigh represent the most common anatomic locations of injuries sustained in ice hockey athletes [5, 6, 11, 13, 15, 17, 19, 21–23, 68, 69].

7.5.1 Youth and High School Ice Hockey

In youth hockey, injuries are most common to the head/face, shoulder/clavicle, knee, and hip/groin/thigh [5, 6]. Emery et al. reported the anatomic distribution of injuries in 2154 youth athletes from two different leagues during a single season [6]. In the league where body checking was allowed, the locations with the highest number of injuries per 1000 player-game hours were the head/face (1.59), knee (0.62), shoulder/clavicle (0.44), and hip/groin/upper leg (0.42). A slightly different distribution existed in the league where body checking was not allowed: head/face (0.41), knee (0.39), hip/groin/upper leg (0.16), and lower leg/ankle/foot (0.14). They concluded that playing in a league where body checking was permitted was associated with a threefold increased risk of in-game injuries.

Bartley et al. reported data from the High School Reporting Information Online database from 2005 to 2016 [15]. The anatomic locations making up the highest percentage of overall injuries in leagues allowing checking included the head/face (37.1%), shoulder (20.6%), hand/wrist (7.4%), and clavicle (6.9%). Hammer et al. reported similar findings in a study of injuries sustained in male high school athletes from 2008 to 2013 [70]. They report the most common anatomic locations for ice hockey injuries were the head/face (32.2%), shoulder/clavicle (20.0%), hip/leg (11.4%), and knee (8.8%). Furthermore, 19.4% of injuries were classified as severe

(≥21 days of missed participation, medical disqualification for the season/career, fatality, or paralysis), and commonly sustained to the shoulder/clavicle (30.7%), head/face (20.0%), hand/wrist (15.7%), and knee (11.4%).

7.5.2 Junior Ice Hockey

Stuart et al. reported a 3-year prospective study with 142 injuries in a US hockey league [17]. The region most frequently injured was the face (26.1%), with a significantly higher incidence in games (38.0/1000 player-game hours) compared to practice (0.6/1000 player-practice hours). The second most common region injured was the shoulder (20.4%), followed by the hip (10.6%). Injuries to the lumbar spine (6.3%), knee (5.6%), hand (4.9%), foot (4.9%), and thigh (4.2%) were less common.

Tuominen et al. conducted a prospective study of injuries at the IIHF Under-18 and Under-20 World Championships between 2006 and 2015 [11]. The most common regions injured reported as a rate per 1000 player-game hours were the face (8.9), shoulder/clavicle (6.7), head (4.9), and knee (3.1). In the World Junior Under-20 Championships, facial trauma comprised 76% of the head/face injuries with an injury rate of 4.4/1000 player-games, 80% of which consisted of lacerations (3.6/1000 player-games). In the World Junior Under-18 Championships, concussions comprised 46% of the head/face injuries with an injury rate of 1.2/1000 player-games, and lacerations accounted for 44% of these injuries (1.1/1000 player-games).

7.5.3 College Ice Hockey

Agel et al. reported on collegiate men's ice hockey injuries from the NCAA ISS from 1988 to 2004 [13]. They assessed the most common anatomic regions and injury types as an injury rate per 1000 player-games as follows: knee—internal derangement (2.2), head—concussion (1.5), shoulder—acromioclavicular joint (1.5), and upper leg—contusion (1.0). In a similar study on collegiate women's ice hockey injuries from the NCAA ISS from 2000 to 2004, Agel et al. reported the following injury rates per 1000 player-games: head—concussion (2.7), knee—internal derangement (1.6), shoulder—acromioclavicular joint (0.9), and ankle—ligament sprain (0.5) [22].

Studies agree that injuries in collegiate ice hockey athletes are most common to the head, face, shoulder, and knee [19, 21]. McKnight et al. reported injury rates per 1000 athlete exposures with the most common regions injured including the shoulder (1.9), knee (1.6), upper extremity (1.2), and head/face/neck (1.13) [21]. Pelletier et al. assessed the distribution of 188 injuries, with 18.6% of the injuries sustained to the knee, 17.6% to the face, 14.9% to the shoulder/clavicle, and 10.6% to the head/neck [19].

7.5.4 Professional Ice Hockey

Tuominen et al. reported a 7-year study of injuries in men's international ice hockey at the IIHF adult World Championship tournaments and Winter Olympic Games [23]. The most common regions injured reported as a rate per 1000 player-game hours included the face (12.7), knee (7.5), shoulder/clavicle (5.8), and head (5.7). In a similar study, Tuominen et al. reported an 8-year study of injuries in women's international ice hockey at the IIHF adult World Championship tournaments and Olympic Winter Games [67]. The most common regions injured reported as a rate per 1000 player-game hours included the knee (4.6), head (3.8), ankle/leg (2.7), and neck/upper back/lower back (2.0). Discrepancies between men's and women's ice hockey injuries can in part be attributed to differences in rules, as body checking is not permitted in women's ice hockey at any level.

McKay et al. reported on injuries in 1685 National Hockey League (NHL) athletes over 6 seasons [68]. The most common regions injured included the head (17%), thigh (14%), knee (13%), and shoulder (12%). Nordstrom et al.

reported injuries in 225 Norwegian Professional League athletes during the 2017–2018 season, categorizing injuries by severity and region [69]. For mild injuries (1–7 days loss of time), the most common regions injured were the head/face, shoulder/clavicle, knee, ankle, and wrist. For severe injuries (>28 days loss of time) the most common regions injured were the shoulder/clavicle, ankle, head/face, and knee.

7.6 Injury Prevention

A multifaceted approach to injury prevention in ice hockey athletes is necessary, with initiatives driven by studies highlighting the epidemiology of injuries in this injury-prone sport. Initial efforts placed emphasis on catastrophic injuries including spine trauma, concussions, eye injuries, and permanent disability. Increased awareness of common injury mechanisms resulted in leagues monitoring and eliminating dangerous infractions such as charging, boarding, checking from behind, head hits, and fighting. Quality education and coaching, improved protective equipment, and enforcement and modification of existing rules are key to injury prevention, and the safety of athletes.

The head and face represent the most common region injured across all age groups, reinforcing the importance of quality helmets and full facial protection. The use of full facial protection has been shown to reduce the risk of facial and dental trauma [4]. Stuart et al. found that in Junior A ice hockey athletes, full facial protection completely eliminated injuries to the eye, while athletes with no facial protection had a 4.7 times greater risk of eye injury than athletes with partial facial protection [71]. Furthermore, facial injuries decreased with added facial protection: no facial protection (158.9/1000 player-game hours), partial facial protection (73.5/1000 player-game hours), and full facial protection with a cage or shield (23.2/1000 player-game hours). Leagues have started implementing stricter visor rules, even at the professional level.

Body checking is the most common mechanism of injury in ice hockey athletes. In response, USA Hockey has sanctioned a "Heads Up, Don't Duck" initiative. Athletes are instructed how to make contact with the boards and to lift their head if head contact is unavoidable to prevent axial loading. These efforts in conjunction with rule and equipment modifications have led to a continued decline in the number of catastrophic and potentially catastrophic injuries among USA Hockey athletes.

Mandatory facial protection and delayed body checking in games until age 13 are proven strategies to reduce the risk of facial injuries and concussions. Emery et al. demonstrated a significant increase in overall risk of injury and three-times increased risk of concussion in youth athletes competing in a league that allowed body checking compared to a league that did not [6]. Interestingly, in a follow-up study, they found the risk of an injury resulting in more than 7 days of lost time was reduced by 33% among Bantam players in a league where body checking was allowed 2 years earlier compared to a league where it was newly introduced [5]. This data highlights that despite the increased risk of overall injury and concussion associated with body checking, policy regarding the appropriate age to introduce body checking requires further investigation.

The prevention of concussions has been one of the largest collaborative efforts in ice hockey, with implementation of prioritized action items by USA Hockey [72–75]. Measures have been taken to instruct athletes on safer body checking, delaying body checking until age 13, and stricter penalties for head hits [72]. Furthermore, work has been done to develop improved equipment, including more effective helmets to prevent head injuries [76]. Newer helmet designs use technology to help mitigate forces, with improved outer shells and energy-absorbing liners that dissipate both linear and rotational acceleration to the brain. Additional strategies to reduce the risk of concussion involve coaching on-ice awareness, body control skills, improving neck muscle strength and proprioception, and expanding Fair Play rules in youth hockey, an initiative to award teams that demonstrate safe play and good sportsmanship [77]. Furthermore, energy-absorbing boards and glass have also shown potential to reduce the risk of concussions and shoulder injuries [23, 36]. Last, current rules in professional and some junior leagues allow for fighting. While rule changes have resulted in stricter penalties for such action, some advocate for rules to limit or prevent fighting across all leagues.

Take Home Message

Ice hockey is a common international sport, with a high incidence of injury. Contact with another player or the boards represent the most common mechanism of injury. Injuries to the head/face, shoulder/clavicle, knee, and hip/groin/thigh occur frequently, especially at the elite levels. Injury prevention has been a focus by many hockey leagues, resulting in recent rule modifications and equipment regulations to help keep athletes safe.

References

1. Yard EE, Comstock RD. Injuries sustained by pediatric ice hockey, lacrosse, and field hockey athletes presenting to United States emergency departments, 1990–2003. J Athl Train. 2006;41:441–9.
2. Deits J, Yard EE, Collins CL, Fields SK, Comstock RD. Patients with ice hockey injuries presenting to US emergency departments, 1990–2006. J Athl Train. 2010;45:467–74.
3. Anderson GR, Melugin HP, Stuart MJ. Epidemiology of injuries in ice hockey. Sports Health. 2019;11:514–9.
4. Benson BW, Mohtadi NG, Rose MS, Meeuwisse WH. Head and neck injuries among ice hockey players wearing full face shields vs half face shields. JAMA. 1999;282:2328–32.
5. Emery C, Kang J, Shrier I, Goulet C, Hagel B, Benson B, Nettel-Aguirre A, McAllister J, Meeuwisse W. Risk of injury associated with bodychecking experience among youth hockey players. CMAJ. 2011;183:1249–56.
6. Emery CA, Kang J, Shrier I, Goulet C, Hagel BE, Benson BW, Nettel-Aguirre A, McAllister JR, Hamilton GM, Meeuwisse WH. Risk of injury associated with body checking among youth ice hockey players. JAMA. 2010;303:2265–72.
7. Latinis B. Frequent sports injuries of children: etiology, treatment, and prevention. Issues Compr Pediatr Nurs. 1983;6:167–78.
8. Flik K, Lyman S, Marx RG. American collegiate men's ice hockey: an analysis of injuries. Am J Sports Med. 2005;33:183–7.
9. Donskov AS, Humphreys D, Dickey JP. What is injury in ice hockey: an integrative literature review on injury rates, injury definition, and athlete exposure in men's elite ice hockey. Sports (Basel). 2019;7(11):227.
10. Forward KE, Seabrook JA, Lynch T, Lim R, Poonai N, Sangha GS. A comparison of the epidemiology of ice hockey injuries between male and female youth in Canada. Paediatr Child Health. 2014;19:418–22.
11. Tuominen M, Stuart MJ, Aubry M, Kannus P, Parkkari J. Injuries in world junior ice hockey championships between 2006 and 2015. Br J Sports Med. 2017;51:36–43.
12. Molsa J, Kujala U, Nasman O, Lehtipuu TP, Airaksinen O. Injury profile in ice hockey from the 1970s through the 1990s in Finland. Am J Sports Med. 2000;28:322–7.
13. Agel J, Dompier TP, Dick R, Marshall SW. Descriptive epidemiology of collegiate men's ice hockey injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. J Athl Train. 2007;42:241–8.
14. Matic GT, Sommerfeldt MF, Best TM, Collins CL, Comstock RD, Flanigan DC. Ice hockey injuries among United States high school athletes from 2008/2009–2012/2013. Phys Sportsmed. 2015;43:119–25.
15. Bartley JH, Murray MF, Kraeutler MJ, Pierpoint LA, Welton KL, McCarty EC, Comstock RD. Epidemiology of injuries sustained as a result of intentional player contact in high school football, ice hockey, and lacrosse: 2005–2006 through 2015–2016. Orthop J Sports Med. 2017;5:2325967117740887.
16. Decloe MD, Meeuwisse WH, Hagel BE, Emery CA. Injury rates, types, mechanisms and risk factors in female youth ice hockey. Br J Sports Med. 2014;48:51–6.
17. Stuart MJ, Smith A. Injuries in Junior A ice hockey. A three-year prospective study. Am J Sports Med. 1995;23:458–61.
18. LaPrade RF, Wijdicks CA, Griffith CJ. Division I intercollegiate ice hockey team coverage. Br J Sports Med. 2009;43:1000–5.
19. Pelletier RL, Montelpare WJ, Stark RM. Intercollegiate ice hockey injuries. A case for uniform definitions and reports. Am J Sports Med. 1993;21:78–81.
20. Agel J, Harvey EJ. A 7-year review of men's and women's ice hockey injuries in the NCAA. Can J Surg. 2010;53:319–23.
21. McKnight CM, Ferrara MS, Czerwinska JM. Intercollegiate ice hockey injuries: a three-year analysis. J Athl Train. 1992;27:338–43.
22. Agel J, Dick R, Nelson B, Marshall SW, Dompier TP. Descriptive epidemiology of collegiate women's ice hockey injuries: National Collegiate Athletic Association Injury Surveillance System, 2000–2001 through 2003–2004. J Athl Train. 2007;42:249–54.

23. Tuominen M, Stuart MJ, Aubry M, Kannus P, Parkkari J. Injuries in men's international ice hockey: a 7-year study of the International Ice Hockey Federation Adult World Championship Tournaments and Olympic Winter Games. Br J Sports Med. 2015;49:30–6.
24. Molsa J, Airaksinen O, Nasman O, Torstila I. Ice hockey injuries in Finland. A prospective epidemiologic study. Am J Sports Med. 1997;25:495–9.
25. Lorentzon R, Wedren H, Pietila T. Incidence, nature, and causes of ice hockey injuries. A three-year prospective study of a Swedish elite ice hockey team. Am J Sports Med. 1988;16:392–6.
26. Pettersson M, Lorentzon R. Ice hockey injuries: a 4-year prospective study of a Swedish elite ice hockey team. Br J Sports Med. 1993;27:251–4.
27. Laprade RF, Surowiec RK, Sochanska AN, Hentkowski BS, Martin BM, Engebretsen L, Wijdicks CA. Epidemiology, identification, treatment and return to play of musculoskeletal-based ice hockey injuries. Br J Sports Med. 2014;48:4–10.
28. Hibberd EE, Kerr ZY, Roos KG, Djoko A, Dompier TP. Epidemiology of acromioclavicular joint sprains in 25 National Collegiate Athletic Association Sports: 2009–2010 to 2014–2015 academic years. Am J Sports Med. 2016;44:2667–74.
29. Cruz CA, Kerbel Y, Smith CM, Prodromo J, Trojan JD, Mulcahey MK. A sport-specific analysis of the epidemiology of hip injuries in National Collegiate Athletic Association Athletes from 2009 to 2014. Arthroscopy. 2019;35:2724–32.
30. Pierce CM, Laprade RF, Wahoff M, O'Brien L, Philippon MJ. Ice hockey goaltender rehabilitation, including on-ice progression, after arthroscopic hip surgery for femoroacetabular impingement. J Orthop Sports Phys Ther. 2013;43:129–41.
31. Philippon MJ, Ho CP, Briggs KK, Stull J, LaPrade RF. Prevalence of increased alpha angles as a measure of cam-type femoroacetabular impingement in youth ice hockey players. Am J Sports Med. 2013;41:1357–62.
32. Stull JD, Philippon MJ, LaPrade RF. "At-risk" positioning and hip biomechanics of the Peewee ice hockey sprint start. Am J Sports Med. 2011;39(Suppl):29S–35S.
33. Worner T, Thorborg K, Eek F. High prevalence of hip and groin problems in professional ice hockey players, regardless of playing position. Knee Surg Sports Traumatol Arthrosc. 2019;28:2302–8.
34. Gallo RA, Silvis ML, Smetana B, Stuck D, Lynch SA, Mosher TJ, Black KP. Asymptomatic hip/groin pathology identified on magnetic resonance imaging of professional hockey players: outcomes and playing status at 4 years' follow-up. Arthroscopy. 2014;30:1222–8.
35. Tegner Y, Lorentzon R. Ice hockey injuries: incidence, nature and causes. Br J Sports Med. 1991;25:87–9.
36. Engebretsen L, Steffen K, Alonso JM, Aubry M, Dvorak J, Junge A, Meeuwisse W, Mountjoy M, Renstrom P, Wilkinson M. Sports injuries and illnesses during the Winter Olympic Games 2010. Br J Sports Med. 2010;44:772–80.
37. Kary JM. Diagnosis and management of quadriceps strains and contusions. Curr Rev Musculoskelet Med. 2010;3:26–31.
38. Reid D, Polson K, Johnson L. Acromioclavicular joint separations grades I–III: a review of the literature and development of best practice guidelines. Sports Med. 2012;42:681–96.
39. Molsa J, Kujala U, Myllynen P, Torstila I, Airaksinen O. Injuries to the upper extremity in ice hockey: analysis of a series of 760 injuries. Am J Sports Med. 2003;31:751–7.
40. Hudson VJ. Evaluation, diagnosis, and treatment of shoulder injuries in athletes. Clin Sports Med. 2010;29:19–32, table of contents.
41. Willimon SC, Gaskill TR, Millett PJ. Acromioclavicular joint injuries: anatomy, diagnosis, and treatment. Phys Sportsmed. 2011;39:116–22.
42. Chillemi C, Franceschini V, Dei Giudici L, Alibardi A, Salate Santone F, Ramos Alday LJ, Osimani M. Epidemiology of isolated acromioclavicular joint dislocation. Emerg Med Int. 2013;2013:171609.
43. Kuhn AW, Noonan BC, Kelly BT, Larson CM, Bedi A. The hip in ice hockey: a current concepts review. Arthroscopy. 2016;32:1928–38.
44. Donaldson L, Li B, Cusimano MD. Economic burden of time lost due to injury in NHL hockey players. Inj Prev. 2014;20:347–9.
45. Ayeni OR, Kowalczuk M, Farag J, Farrokhyar F, Chu R, Bedi A, Willits K, Bhandari M. Trends in reporting of mechanisms and incidence of hip injuries in males playing minor ice hockey in Canada: a cross-sectional study. Open Access J Sports Med. 2014;5:143–9.
46. Bedi A, Kelly BT. Femoroacetabular impingement. J Bone Joint Surg Am. 2013;95:82–92.
47. Bedi A, Dolan M, Leunig M, Kelly BT. Static and dynamic mechanical causes of hip pain. Arthroscopy. 2011;27:235–51.
48. Yen YM, Kocher MS. Clinical and radiographic diagnosis of femoroacetabular impingement. J Pediatr Orthop. 2013;33(Suppl 1):S112–20.
49. Wall PD, Fernandez M, Griffin DR, Foster NE. Nonoperative treatment for femoroacetabular impingement: a systematic review of the literature. PM R. 2013;5:418–26.
50. Krych AJ, Griffith TB, Hudgens JL, Kuzma SA, Sierra RJ, Levy BA. Limited therapeutic benefits of intra-articular cortisone injection for patients with femoro-acetabular impingement and labral tear. Knee Surg Sports Traumatol Arthrosc. 2014;22:750–5.
51. Philippon MJ, Weiss DR, Kuppersmith DA, Briggs KK, Hay CJ. Arthroscopic labral repair and treatment of femoroacetabular impingement in professional hockey players. Am J Sports Med. 2010;38:99–104.
52. Nawabi DH, Bedi A, Tibor LM, Magennis E, Kelly BT. The demographic characteristics of high-level and recreational athletes undergoing hip arthroscopy for femoroacetabular impingement: a sports-specific analysis. Arthroscopy. 2014;30:398–405.
53. Grant JA, Bedi A, Kurz J, Bancroft R, Miller BS. Incidence and injury characteristics of medial

collateral ligament injuries in male collegiate ice hockey players. Sports Health. 2013;5:270–2.
54. Tegner Y, Lorentzon R. Evaluation of knee braces in Swedish ice hockey players. Br J Sports Med. 1991;25:159–61.
55. Laprade RF, Wijdicks CA. The management of injuries to the medial side of the knee. J Orthop Sports Phys Ther. 2012;42:221–33.
56. 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;38:330–8.
57. 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.
58. Borden PS, Kantaras AT, Caborn DN. Medial collateral ligament reconstruction with allograft using a double-bundle technique. Arthroscopy. 2002;18:E19.
59. White K, Di Stasi SL, Smith AH, Snyder-Mackler L. Anterior cruciate ligament-specialized post-operative return-to-sports (ACL-SPORTS) training: a randomized control trial. BMC Musculoskelet Disord. 2013;14:108.
60. LaPrade RF, Goodrich LR, Phillips J, Dornan GJ, Turnbull TL, Hawes ML, Dahl KD, Coggins AN, Kisiday J, Frisbie D, Chahla J. Use of platelet-rich plasma immediately after an injury did not improve ligament healing, and increasing platelet concentrations was detrimental in an in vivo animal model. Am J Sports Med. 2018;46:702–12.
61. 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:e4–6.
62. Chen L, Kim PD, Ahmad CS, Levine WN. Medial collateral ligament injuries of the knee: current treatment concepts. Curr Rev Musculoskelet Med. 2008;1:108–13.
63. Khan W, Zoga AC, Meyers WC. Magnetic resonance imaging of athletic pubalgia and the sports hernia: current understanding and practice. Magn Reson Imaging Clin N Am. 2013;21:97–110.
64. Ryan JB, Wheeler JH, Hopkinson WJ, Arciero RA, Kolakowski KR. Quadriceps contusions. West Point update. Am J Sports Med. 1991;19:299–304.
65. Stuart MJ, Smith AM, Nieva JJ, Rock MG. Injuries in youth ice hockey: a pilot surveillance strategy. Mayo Clin Proc. 1995;70:350–6.
66. Council on Sports Medicine and Fitness, Brooks A, Loud KJ, Brenner JS, Demorest RA, Halstead ME, Kelly AK, Koutures CG, LaBella CR, et al. Reducing injury risk from body checking in boys' youth ice hockey. Pediatrics. 2014;133:1151–7.
67. Tuominen M, Stuart MJ, Aubry M, Kannus P, Tokola K, Parkkari J. Injuries in women's international ice hockey: an 8-year study of the World Championship tournaments and Olympic Winter Games. Br J Sports Med. 2016;50:1406–12.
68. McKay CD, Tufts RJ, Shaffer B, Meeuwisse WH. The epidemiology of professional ice hockey injuries: a prospective report of six NHL seasons. Br J Sports Med. 2014;48:57–62.
69. Nordstrom A, Bahr R, Talsnes O, Clarsen B. Prevalence and burden of health problems in male elite ice hockey players: a prospective study in the Norwegian professional league. Orthop J Sports Med. 2020;8:2325967120902407.
70. Hammer E, Brooks MA, Hetzel S, Arakkal A, Comstock RD. Epidemiology of injuries sustained in boys' high school contact and collision sports, 2008–2009 through 2012–2013. Orthop J Sports Med. 2020;8:2325967120903699.
71. Stuart MJ, Smith AM, Malo-Ortiguera SA, Fischer TL, Larson DR. A comparison of facial protection and the incidence of head, neck, and facial injuries in Junior A hockey players. A function of individual playing time. Am J Sports Med. 2002;30:39–44.
72. Smith AM, Stuart MJ, Dodick DW, Roberts WO, Alford PW, Ashare AB, Aubrey M, Benson BW, Burke CJ, Dick R, et al. Ice Hockey Summit II: zero tolerance for head hits and fighting. Clin J Sport Med. 2015;25:78–87.
73. Smith AM, Stuart MJ, Gaz DV, Twardowski CP, Stuart MB, Margeneau D, Tearse H, Roberts WO. Behavioral modification to reduce concussion in collision sports: ice hockey. Curr Sports Med Rep. 2013;12:356–9.
74. Smith AM, Stuart MJ, Greenwald RM, Benson BW, Dodick DW, Emery CA, Finnoff JT, Mihalik JP, Roberts WO, Sullivan CA, Meeuwisse WH. Proceedings from the ice hockey summit on concussion: a call to action. Clin J Sport Med. 2011;21:281–7.
75. Smith AM, Stuart MJ, Roberts WO, Dodick DW, Finnoff JT, Jorgensen JK, Krause DA. Concussion in ice hockey: current gaps and future directions in an objective diagnosis. Clin J Sport Med. 2017;27:503–9.
76. Rowson B, Rowson S, Duma SM. Hockey STAR: a methodology for assessing the biomechanical performance of hockey helmets. Ann Biomed Eng. 2015;43:2429–43.
77. Smith AM, Gaz DV, Larson D, Jorgensen JK, Eickhoff C, Krause DA, Fenske BM, Aney K, Hansen AA, Nanos SM, Stuart MJ. Does fair play reduce concussions? A prospective, comparative analysis of competitive youth hockey tournaments. BMJ Open Sport Exerc Med. 2016;2:e000074.

8 Rugby

Rugby Injuries

David Anthony Parker
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8.1 Introduction

Rugby is a full contact team sport which has worldwide participation ranging from school games to elite professional tournaments. It is a sport in which individual player fitness including anthropometric and physiological characteristics are vital to the success of the team [1, 2]. It is one of the only contact-collision sports where the rules for women and men are the same [3].

A detailed description of the history and evolution of rugby is a vast topic and beyond the scope of this chapter. However, knowledge of some of the key historical events in this sport should help the reader develop a better understanding of the different formats of the current game and the associated injuries.

8.2 History

The origins of this sport date back to 1823 in Rugby, a town in the midlands area of England. The legendary story of William Webb Ellis, a schoolboy who picked up the ball in a soccer game and ran with it thereby creating an entirely new sport is widely reported [4, 5].

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The sport of Rugby Football League originated in the north of England in the mid-1890s, when players of rugby football union demanded expenses in compensation for wages lost when playing [6]. In 1895, following the refusal of their demand by Rugby Football Union, 21 northern clubs broke away to establish their own version of the game. The rules of the new sport were changed radically from that of rugby union, including the abolition of the lineout, a reduction from 15 to 13 players, and the introduction of the immediate 'play-the-ball' after a tackle. These early modifications still form the basis of the modern-day sport of Rugby Football League. In the early 1900s, the sport was introduced to New Zealand, followed shortly by Australia. A dispute similar to the one in England over professionalism gave rise to Rugby League in France and, more recently, the sport has developed in numerous countries worldwide, including Russia, the islands of the South Pacific and South Africa [7].

8.3 Popular Formats

The sport of rugby is played in two major formats (codes), namely Rugby Union (RU) with 15 players per team and Rugby League (RL) with 13 players per team. World Rugby (WR) and International Rugby League (IRL) are the highest governing bodies of RU and RL, respectively. In their 2018 report, WR noted participation of 9.6

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million players in 123 countries [8] whereas IRL noted that the league version is played in over 50 nations, thereby making rugby a global mega sport with growing popularity [9]. A 'sevens' version of rugby with seven team members is also played widely [10, 11].

8.4 Team Composition

8.4.1 Rugby Union (RU): Fifteen Players per Team

Forward positions consist of the following: (1) Hooker (number 2), (2) Loose and tight head props (number 1 and 3), (3) Locks (number 4 and 5), (4) Flankers (number 6 and 7), (5) Eightman (number 8).

The hooker along with the props (loose head and tight head) constitute the front-row forwards. The locks are also referred to as the second-row forwards, and they help lock the scrum together. The number 8 and the two flankers form the back-row forwards.

Back positions consist of the following: (1) Scrumhalf (number 9), (2) Flyhalf (number 10), (3) Wings (number 11 and 14), (4) Inside and outside centres (number 12 and 13), (5) Fullback (number 15).

Players in scrumhalf and flyhalf positions are also known as half-backs. Players in the inside and outside centres and the two players on the wing are collectively referred to as three-quarter backs.

8.4.2 Rugby League (RL): Thirteen Players per Team

Forward positions consist of the following: (1) Prop (number 8), (2) Hooker (number 9), (3) Front-row forwards (number 10), (4) Second-row forwards (number 11 and 12), (5) Loose forwards (number 13).

Back positions consist of the following: (1) Fullback (number 1), (2) Right wing (number 2), (3) Right centre (number 3), (4) Left centre (number 4), (5) Left wing (number 5), (6) Stand-off (number 6), (7) Scrumhalf (number 7).

8.5 Similarities and Differences Between Rugby Union and League

RU and RL have similarities in game duration, field size, and goal posts. However, they also have distinct differences in rules and scoring methodology [5, 12–15]. Information regarding the current rules and regulations including the prescribed dimensions of the playing field, the ball, number, and position of players in the different formats of rugby can be obtained from the aforementioned official and regulatory organisations [5, 12–15]. In general, the match consists of two halves lasting 40 min each and a halftime of 10 min. The ball is advanced down the field by kicking or running with it or can be passed between teammates only by way of backward or lateral tosses. Points are scored by each team for advancing the ball over the opponent team's goal line with differential points awarded for a try, conversion, or goal based on the format of the rugby game. Each player in the team is assigned a particular number. Player positions can be broadly grouped into forwards and backs. The forward players are involved in gaining and maintain the possession of the ball during the course of the match.

As highlighted above, an RU team consists of 15 players, whereas RL has 2 less forward positions with 13 players. The two formats of rugby differ significantly in terms of what happens when players are involved in a tackle situation. When a player is tackled in RU game, the ball is recycled by a ruck or by a maul, with no limit to the number of phases whereas a tackled player in RL game stops play and the team in possession is able to recycle the ball up to six times before the ball is handed over to the opposition [5, 12–15]. Another significant difference between the two formats of rugby is observed when the ball goes out or 'into touch'. In RU game, the play is restarted with a lineout, whereas in RL game, the play is restarted with a scrum, retention, or changeover in possession [5, 12–15].

As the popularity of RU and RL has evolved, there has been an extensive list of players who have participated in both codes of rugby [16, 17]. These skilled players have been collectively referred to as 'dual-code rugby internationals' or 'code converts' by some authors and include players of both forward and back positions [16–18].

It must be noted that whilst some overlap and similarities exist between RU and RL, the differences between these two formats of rugby also play a role in the injuries sustained by the players. Hence, these factors are discussed in the subsequent sections of this chapter, and whilst we use the term 'rugby' to cover both codes, differences relevant to injuries in RU and RL are highlighted as appropriate.

8.6 Unique Mechanics and Risk Factors for Injury

A combination of gameplay manoeuvres and full contact/collision which is inherent to rugby makes the players prone to distinct pattern of injuries. Furthermore, player factors such as the age group, physique, physiology, field position, gender, and the competitive level of the game amongst others influence the characteristic contact situations and subsequent injuries unique to rugby [19–22]. In general, player movements during rugby have been described to be intermittent in nature, with periods of high-intensity activity (contact, sprinting, low- and high-speed running) and low-intensity recovery (standing, walking, and jogging) over two halves of the game [23]. Some of the distinct features and factors relevant to the aetiopathology of injuries in rugby players is discussed below.

8.6.1 Phase of Play

During a rugby game, contact situations can emerge depending on the phase of play [24, 25]. Tackle, ruck, and scrum are the major contact events in RU games and are associated with 50%, 9%, and 4% of all injuries, respectively [26]. Similarly, tackle has been attributed to be the cause of 38–77% of injuries in players during RL matches [27]. These factors are discussed below.

8.6.2 Tackle

Tackle is an important component of the competitive rugby game. However, it has been associated with injury irrespective of the level of competition [28–39]. The unique collision demands of rugby matches make strength a requisite quality for competitors to effectively tolerate the blunt force trauma that occurs in tackles and the physical stress associated with wrestling activities [40]. Tackles occur in open play, often involve relatively high-velocity impacts, and in many cases the tackler and ball carrier have limited time to prepare for the contact situation compared with other events [26, 41]. Current evidence suggests that player speed, mass, type of body contact, momentum, and energy transfer involved during contact are crucial factors associated with injury [21, 28, 42].

The most frequent activity immediately before tackling is striding, followed by sprinting [43]. In an extensive study, Quarrie et al. analysed 140,249 tackles in 434 professional RU matches [42]. They observed that injuries were most frequently the result of high or middle tackles from the front or side, but rate of injury per tackle was higher for tackles from behind than from the front or side. Furthermore, ball carriers were at highest risk from tackles to the head-neck region, whereas tacklers were most at risk when making low tackles. The impact of the tackle was the most common cause of injury, and the head was the most common site, but an important mechanism of lower limb injuries was loading with the weight of another player [42].

Roberts et al. reported that tackles resulted both in the greatest propensity for injury [2.3 (2.2–2.4) injuries/1000 events] and the greatest severity of rugby injuries [16 (15–17) weeks missed/1000 events] [41]. Furthermore, collision tackles (illegal tackles involving a shoulder charge) had a propensity for injury of 15.0 (12.4–18.3) injuries per 1000 events and severity was 92 weeks missed per 1000 events, which were both higher than any other event [41]. Injury risk was higher when being tackled compared with tackling. It has been demonstrated that most tackle injuries to the ball carrier are sustained when the tackler approaches from the ball carrier's peripheral vision [44] or from behind [42, 44].

Garraway et al. noted that 85% of tackling players who were injured were three quarters, and 52% of injuries occurred when the tackle came in behind the tackled player or within his peripheral vision [44]. Either the tackling or tackled player was sprinting or running in all these injury episodes. One third of injuries occurred in differential speed tackles that is, when one player was travelling much faster than the other at impact. The player with the lower momentum was injured in 80% of these cases [44].

In 2010, McIntosh et al. evaluated tackle characteristics in RU and described shoulder tackle as a tackle in which the tackler's shoulder is the first point of contact [28]. Recently, Tanabe et al. studied the role of shoulder position during tackle and the effect on resultant shoulder kinematics and injuries [45]. They reported that with the shoulder tackle as a reference, shoulder abduction on the side of impact was higher in both the arm and head-in-front tackles, whilst shoulder external rotation was lower in the head-in-front tackles. They concluded that kinematics in both the arm tackle and the head-in-front tackle were significantly different from that in the shoulder tackle. They were of the opinion that this may represent a distinct risk factor for shoulder dislocation.

Seminati and colleagues investigated the biomechanics of the shoulder during tackles based on whether it was the dominant or non-dominant side of the RU player [46]. They noted that the peak impact force was substantially higher in the stationary dominant (2.84 ± 0.74 kN) than in the stationary non-dominant condition (2.44 ± 0.64 kN), but lower than in the moving condition (3.40 ± 0.86 kN). Furthermore, muscle activation started on average 300 ms before impact, with higher activation for impact-side trapezius and non-impact side erector spinae and gluteus maximus muscles. They reported that players' technique for non-dominant side tackles was less compliant thereby posing a potential injury risk. Younger players (below 15 years) are engaged in more passive tackles and tend to stay on their feet more than experienced players. Consequently, this cohort players have been reported to have a significantly lower risk of tackle game injury (13%) compared with elite players (31%) [28].

8.6.2.1 Ruck and Maul

Rucks and mauls are phases of play peculiar to RU, and do not occur in RL. A ruck is the phase of play in which one or more players from each team contest the ball on the ground whereas maul is when the ball carrier is in contact with at least two other players on their feet [5]. Together ruck and maul represent intense non-running exertion during a game [47]. When a ruck or maul is executed, the action begins when the participant comes in purposeful contact with another player and ends with their detachment [5]. The number of rucks per match has increased almost fourfold since the introduction of professionalism [48, 49]. The number of mauls per match has decreased during the same period. Both of these changes are likely to be related to the introduction of the use-it-or-lose-it law in 1994. This law increased the risk of losing possession in mauls and made the option of a ruck preferable to that of a maul for the team in possession of the ball. It has been reported that rugby has changed from a maul-dominated to a ruck-dominated game in the post-professional era, as ball in playtime has increased [49]. Furthermore, McLean et al. noted in their study that rucks and mauls outnumbered scrums by 56% and lineouts by 44% [50].

A ruck involves twisting and straining of the upper body and unusual stress on the knees, especially when the ruck becomes unstable and collapses [51]. More than one third of the injuries to forwards and half of the injuries to loose forwards occur in rucks and mauls [52]. Seventy percent of these injuries result from kicking and trampling [51]. Fifty-five percent of the injuries are to the head, neck, and hands, which together constitute only 15% of the body area [51]. Ruck represents 1.6 injuries/1000 player hours and 0.5 injuries/1000 events [41]. It must be noted that rucks contributed the second largest proportion of concussions [41]. Roberts et al. observed that players involved in fulfilling a defensive role in the ruck are more susceptible to concussive impacts [41].

Spinal injuries have been associated with rucks and maul. Scher et al. outlined three different mechanisms in which spinal injuries were sustained during rucks and mauls [52]. These include: (1) forced flexion of the ball carrier's neck, (2) forced flexion of the neck of the player at the bottom of the ruck, (3) head and neck injury caused by charging into a mass of struggling players.

8.6.2.2 Scrum

During an RU game, scrum is the phase of play in which eight players from each team push against each other in a crouched position and contest the ball that is fed in by one team to re-start play [5] (Fig. 8.1). It is a relatively 'controlled' contact event and a highly dynamic activity with the risk of acute injury being moderate but the risk per event is high [53]. Approximately 40% of all rugby-related spinal cord injuries can be attributed to the scrum [53]. Scrums also take place in RL, but are non-competitive and involve minimal force, and therefore are not usually a source of significant injury in RL.

Clayton et al. reported that scrummaging places significant biomechanical demand on players with axial compression forces of approximately 1.8 kN during impact and 1.1 kN during the sustained push phase [54]. They observed that quads fatigue contributed towards increased cervical spine flexion and decreased muscle activation in the trunk during scrum. They hypothesised that a combination of cervical spine flexion, decreased trunk activation and the high axial compressive forces may pose a risk of cervical spinal injuries including disc herniation [54].

Milburn et al. studied the kinetics of scrummaging in university first-grade rugby union players using an instrumented scrum machine

Fig. 8.1 Formation of scrum during rugby match. (Reproduced from Hendricks et al. [55])

[19]. They observed that the primary role of the second row appeared to be application of forward force whereas the back-row ('number 8') forwards did not substantially contribute any additional forward force. However, the side-row contributed an additional 20–27% to the forward force, but at the expense of increased vertical forces on all front-row forwards. Significant amount of force (5761 N) was generated during sustained scrummaging. Interestingly, the sum of the individual player's maximum forward force was noted to be 17,725 N (approximately 1.75 tonnes) or in excess of three times the force recorded for the full scrum [19]. Using electromyography (EMG), Yaghoubi et al. analysed lower extremity muscle function of front-row rugby union players [56]. They noted that the professional props produced more synchronised muscle activation than amateur players. Furthermore, all players produced more synchronised muscle activation against the instrumented scrum machine compared to live scrummage.

In their study, Roberts et al. observed that only a small proportion (5%) of scrums collapse [41]. The propensity for injury during collapsed scrums was four times higher [2.9 (1.5–5.4) injuries/1000 events] and the severity was six times greater [22 (12–42) weeks missed/1000 events] than for non-collapsed scrums. On the other hand, Taylor et al. reported that 31% of scrums in competitive matches resulted in collapse [57]. However, similar to the previous study they noted that injury incidence associated with collapsed scrum-events (incidence: 8.6 injuries/1000 scrum-events) was significantly higher than those scrums that did not collapse (incidence: 4.1/1000 scrum-events).

Scrum is a relatively controllable event. Hence, investigators have suggested that further attempts should be made to reduce the frequency of scrum collapse and injuries associated with it [41, 57]. In January 2007, the IRB implemented a new law for scrum engagement aimed at minimising scrum collapses and the resultant injuries [58]. Fuller et al. attributed the significant reduction in cervical spine injures during scrummage to the positive implementation of this law which is aimed at overall player welfare [59].

8.6.2.3 Running

Running has been reported to be a common mechanism of non-contact injury in rugby [60]. Rugby forwards typically perform 10–15 short distance (10–20 m) sprints during a game, therefore, the initial acceleration over the first 10 m of a sprint may be a critical factor in their performance [61]. Thus, for rugby forwards, the ability to attain maximum speed quickly following a break from the opposition is an important performance requirement for this group.

Gabbett et al. demonstrated that greater amounts of very high-velocity running (i.e., sprinting) are associated with an increased risk of lower body soft tissue injury, whereas distances covered at low and moderate speeds offer a protective effect against soft tissue injury [62]. Sprint activities such as bouts of repeated high-intensity activities completed by players for up to and sometimes longer than 120 s in duration and which are separated by as little as 25 s recovery have a high physiological cost. Prolonged high-intensity intermittent running ability is a significant predictor for the risk of contact injury [63]. Higher physical demands placed on elite players during the first half could result in the earlier onset of physical fatigue towards the end of a match [64]. Body mass and body height of athletes influence sprint running performance [65]. Fatigue and muscle damage accumulate over an intensified competition, which is likely to contribute to reductions in high-intensity activities and work rates during competition [66]. Overall running activities account for 68–93% of hamstring injuries [67, 68].

8.6.2.4 Kicking

The place kick is an important skill in an RU game as it can contribute between 45% and 77% of the total points scored by the team through a penalty kick at goal or through converting a try [69–72]. A player who can produce a longer kick distance is able to attempt a penalty kick or try conversion from a greater fraction of the field of play and hence has a greater opportunity to score [72]. Kicking has been observed to be a common stretch-shortening cycle (SSC) activity [68, 73]. This movement has been associated with injuries notably to hamstrings amongst other structures [68]. Furthermore, hamstrings injuries resulting from kicking are regarded as severe leading to a significant time-loss (36 days) of player activity [68].

It must be noted that the energy demands of scrums/rucks/mauls and tackling are different from those of sprinting and high-intensity running. Nonetheless, they have a high physiological cost as suggested by Austin and colleagues [74]. This fact coupled with a complex interplay of the factors discussed above contributes to the unique injury patterns seen in rugby players.

8.6.3 Physiological Demands on Player

Rugby matches generally consist of two halves, each of 40 min, separated by a 5–10-min recovery period. The players are involved in low-intensity, aerobic exercise, combined with periods of intermittent, intensive anaerobic exercise [75]. Individual players have been shown to cover distances of approximately 5000–8000 m during a game and be involved in 20–40 tackles per match. Maximum oxygen uptake values of around 56 ml/kg/min have been reported for RL players, with no differences between the values of forwards and backs [76]. Forwards have higher body mass, subcutaneous fat and fat-free mass levels than backs [77, 78]. Backs have been found to be quicker than forwards and produce greater leg power output when related to fat-free mass [76].

Data suggests that RU players spend 47% of total time walking and jogging, 38% of their time standing, and 15% of their time in various forms of high-intensity activity [75, 79]. Similar findings have been noted in studies on RL players [75]. The mean distance covered by the backs (7336 m) has been shown to be greater than that covered by the forwards (6647 m) [80]. RL players cover between 5000 and 7000 m during an 80-min match [75]. Whilst the amount of time spent by individual players on low-intensity exercise exceeds the duration of high-intensity exercise, the nature of the high-intensity efforts (involving sprinting, lower and upper-body impacts, and high force generation) is such that the overall intensity of the game is greatly increased. Thus, the various physiological demands on players during rugby games and training period can impact their fatigability and susceptibility to injuries [62, 81–86].

8.6.4 Player Position

Positional roles play an important part in determining the amount of physical and game-specific skill involvement during match play [87–89].

RU forwards are involved in more rucks, mauls, lineouts, and scrums, which require greater body mass, body height, strength, and power in order to be successful [90]. In contrast, the backs' primary role in beating the opposition in open play requires a combination of speed, acceleration, and agility [90]. In their study, Sirotic et al. reported on the performance of professional RL team players based on five positional groups [22]. These groups consisted of backs ($n = 8$), forwards ($n = 8$), fullback ($n = 7$), hooker ($n = 8$), and service players ($n = 8$). They noted that the fullback players completed a significantly higher proportion of the very high-intensity running (VHIR) compared to all other positional groups ($p = 0.017$). Additionally, the VHIR ($p = 0.004$) and sprinting indices ($p < 0.002$) were also significantly greater in the second half of a match for the fullback players. The hooker spent more time jogging than the backs and forwards. The backs spent more time walking than the forwards, hooker, and service players. The forwards, hooker, and service players completed more tackles per minute during a match than the backs and fullback [22]. For forwards, acceleration may be less important, given their higher involvement in the physical contact aspects of the game. Sprinting performance over the shorter distances (10–15 m) is a crucial aspect for the forwards and back players. Hence, player position plays a role in their performance during match and their differential susceptibility to injuries [91, 92].

Several investigators have studied player characteristics in RU [61, 89, 90]. Some of the characteristics of players in certain positions during the game are described below.

8.6.4.1 Forwards

(1) Forwards are generally taller, heavier, and have higher body fat content than the backs with differences of ~5%, ~15%, and ~25%, respectively. (2) Typically, forwards have an endomorphic-mesomorphic physique compared to the backs [93]. (3) Forwards tend to have higher endomorphy and lower ectomorphy than backs, which is probably due to the strength demands placed upon them at the contact situation. (4) Forwards are generally stronger than backs in both upper and lower body due to requirements of strength in scrums and the higher frequency in which the forwards are involved in tackles and ruck situations [89]. (5) Forwards were involved in the ruck/maul/tackle category for a greater duration of time and at a higher frequency than backs [47]. (6) Forwards engaged in 33% more static exertion activities than backs [47].

8.6.4.2 Backs

(1) These players play crucial role in beating the opposition in open play and require speed, acceleration, and agility. (2) RU players in back positions need explosive leg power to be able to accelerate to create opportunities for the wings [94]. (3) Backs cover a greater distance than forwards during a game [94].

8.7 Top Five Rugby-Related Injuries

Several investigators have studied the common types of injuries sustained by rugby players [37, 39, 92, 95–99]. Williams et al. performed a meta-analysis of the published studies and reported the incidence rate from the pooled analysis for the following common types of injuries amongst rugby players [99].

8.7.1 Muscle and Tendon Injuries (40 per 1000 Player Hours, 95% CI 21–76)

This group of injuries is seen most commonly and includes:

8.7.1.1 Muscle Injuries

Injuries to the hip, groin, thigh and calf musculature associated with contusion/haematoma have been reported by several investigators [24, 27, 32, 95, 98, 100–104]. Muscular injuries are the predominant form of injury in rugby players accounting for 20–32% of the overall injury burden irrespective of amateur or professional nature of the game [27, 98, 100, 101, 105–108]. Calf muscle injuries have been reported to be the most common type of scrummaging injury [53].

Hamstring injury is a common problem faced by rugby players with reported incidence rate of 5.6 injuries/1000 player hours [68]. Whilst the majority (93%) of hamstring injuries are new [67], it is estimated that this injury is associated with a high recurrence rate of 25–34% [68, 104]. Hamstring strains are more likely to affect the biceps femoris and commonly occur at the distal myofascial junction [67, 68]. Hamstring strains most commonly occur during running and nearly 60% of recurrent injuries are reported within the first month of the index injury [68]. Furthermore, players in the backline positions who cover greater distance at speed compared to forwards have higher incidence (8.6 injuries per 1000 player hours) of these injuries. Additionally, fatigue, poor flexibility, inadequate warm-up, quadriceps to hamstring strength imbalance, and poor posture have been suggested as aetiological factors [109].

8.7.1.2 Tendon Injuries

High-loading conditions during scrums, mauls, sprinting, tackling, and landing following jumps have been attributed to partial or complete tendon ruptures during rugby [110]. Achilles tendon injuries accounted for 9% of all match injuries and 19% of all training injuries in one study [111]. Furthermore, 35% of these injuries can be recurrent thereby adding to the injury burden. It has been suggested that front-row forwards are susceptible to Achilles tendon injuries given the explosive and eccentric muscle loading patterns experienced during scrummaging [111]. Recently, Brazier et al. have proposed that there is a genetic component to these injuries with inter-individual variability of tendon properties amongst different rugby players thereby resulting in vastly different outcomes [110]. Achilles tendon ruptures are particularly severe injuries and may have a big impact on players as the mean time of return to full fitness has been reported to be 176 days [111].

In general, the knee joint is the most common site of injury in junior RL players [112]. Disruption of knee extensor mechanism has been described in the literature [95, 113]. The reported incidence is 183 injuries/1000 players amongst male professional RL players [113]. Unlike other knee injuries, in a recent review, Awwad et al. noted that majority of the extensor mechanism injuries (73%) in professional RL players occurred during training and were due to insidious causes [113]. However, the players who injured their knee extensor mechanism were the youngest and comparatively had the highest body mass index (BMI) [113].

Tendon and soft tissue injuries of the hand in the form of mallet finger and flexor digitorum profundus (FDP) rupture also referred to as jersey finger have also been described in rugby players [29, 114–116].

8.7.2 Ligament and Joint (Non-bone) Injuries (34 per 1000 Player Hours, 95% CI 18–65)

This group is the second most common type of injuries amongst rugby players and includes:

8.7.2.1 Ligament Injuries

Ankle and knee ligament injuries have been extensively reported in the literature [26, 98, 111, 113, 117, 118].

Injury to the lateral ankle ligament complex is a common injury and accounted for 11% of all match injuries [98] and 15% of all training injuries [111]. The reported incidence is 10 injuries per 1000 player hours [111]. This injury occurs following an inversion/plantar flexion mechanism [98]. Subsequently, the anterior talo-fibular and/or calcaneo-fibular ligaments undergo spectrum of injury ranging from sprain to complete tear [98]. Sankey et al. reported that majority (25%) of lateral ligament complex injuries in their cohort (male professional rugby union players) were grade I sprains whereas grade III sprains were relatively low at 2.4% [111]. In the same cohort, it was noted that ankle injuries were highest in second-row forwards and lowest in back-row forwards.

Roberts et al. reported an injury incidence of 2.4 per 1000 player match hours with the knee joint being the most common site of injury amongst community level rugby players [26]. Both professional male rugby league and rugby union players sustain similar pattern of knee injuries including time to return to play [113]. Common knee ligament injuries involve: (1) medial collateral ligament (MCL) (2) anterior cruciate ligament (ACL).

MCL injuries are well described comprising about 8% of overall injuries [106] with a reported injury incidence of 3.1 injuries per 1000 player hours [95]. Direct blow to the lateral aspect of the knee is a common contact mechanism in MCL injury [98]. The resultant MCL injury can vary in severity from a sprain to a complete tear from its femoral attachment [98]. A significant valgus stress during contact can result in both MCL and ACL injuries [98].

ACL injuries have been reported to constitute about 3% of overall injuries [98]. ACL injuries are relatively less frequent with an incidence of 50 injuries per 1000 players. Nonetheless, ACL injuries account for the longest time to return to play with a median of 236 days [113]. In their study, Dallalana et al. noted that with ACL injury in rugby union players the predominant mechanism of injury was contact based (being tackled, tackling, or general collision) in 86% of injuries [117]. In the remainder 14% of ACL injuries, non-contact mechanisms such as twisting and turning played a role [117]. Twisting of the player's body with a foot fixed in the ground is a common non-contact mechanism for this injury [98]. However, players sustaining a fall subsequent to an ACL injury can land in a valgus position and tear their MCL [98]. Using video analysis technique, Montgomery et al. [118] noted that 57% of ACL injuries occurred in a contact manner. They identified offensive running and being tackled as the two main scenarios of ACL injury with a higher risk to the ball carrier [118]. During non-contact injuries, lower knee flexion angles and heel-first ground contact in a side-stepping manoeuvre were associated with ACL injury [118].

8.7.2.2 Joint (Non-bone Injuries)

Shoulder joint injuries account for between 9% and 17% of all injuries [24, 119, 120]. The reported injury incidence rate is 13 per 1000 player hours [121]. The spectrum of shoulder injuries in rugby players includes haematomas, acromioclavicular joint (ACJ) injuries, instability-dislocation of glenohumeral joint, and rotator cuff tears amongst others [24, 122–124]. Common mechanisms of injury include (1) contact with the ground with the shoulder/arm in horizontal adduction, flexion, and internal rotation (2) impact to the lateral aspect of the shoulder with flexed elbow and the arm at the side [121]. Additionally, Crichton et al. [125] performed video analysis of elite rugby players and described 'try-scorer' (hyperflexion of the outstretched arm such as when scoring a try) and 'tackler' (extension of the abducted arm behind the player whilst tackling) mechanisms of shoulder injury. In a recent study, Montgomery et al. have described a new mechanism of injury (poach position) observed in 18% of all shoulder dislocations [126]. In this position, a player in the crouched rucking position with arm flexed more than 90° at the shoulder sustains a direct postero-inferior force from the opposing player [126].

Usman et al. evaluated shoulder injuries in elite RU matches using the RugbyMed injury database from New Zealand [121]. In addition to injury incidence rate (per 1000 hours), they estimated injury burden (incidence multiplied by severity) of different shoulder injuries and reported it as the number of days unavailable per 1000 hours of play. Injuries to the acromioclavicular joint (ACJ) were noted to have an injury incidence rate of 3.7 per 1000 hours. Dislocation of the glenohumeral joint was relatively less frequent (injury incidence rate of 1.8 per 1000 hours). However, the impact on the players from this injury appeared to be more (373 days unavailable per 1000 hours of play) [121].

Dislocation of interphalangeal joints in the hand amongst rugby players has been described by several investigators [115, 127, 128]. These injuries occur during contact situations like tackle, ruck, maul, and direct impact from the ball [114, 115]. It has been suggested that the vast majority of these are closed injuries treated on the field with a small proportion being open dislocations that seek medical attention [114]. Knee joint chondral and meniscal injuries have also been described in the literature [113].

8.7.3 Injuries of Central and Peripheral Nervous System (Eight per 1000 Player Hours, 95% CI 4–15)

Injuries to the central and peripheral nervous system are relatively less common. However, they may be associated with significant morbidity to the players. The common types of injuries in this group include:

8.7.3.1 Central Nervous System

Concussion is a common injury experienced by rugby players. The rugby tackle has been reported as the most common cause for concussion [21, 107, 108, 129–131], with the tackled player relatively more at risk of injury than the player making the tackle [132]. Using wireless head impact sensor, King et al. reported linear acceleration range of 10–123 g with a rotational acceleration range of 89–22,928 rad/s^2 during head impacts amongst junior rugby league players under 11 years of age [133]. They reported an average of 13 impacts per player per match with the aforementioned forces at play during such impacts.

Following a systematic review of the topic, Gardner et al. have reported incidence rates of concussion in both RU [134] and RL [135]. In men's rugby-15 s they reported an incidence rate of 4.73 and 0.07 per 1000 hours for match play and training, respectively. However, the incidence rate was relatively lower at 3.01 and 0.55 for the sevens and the women's 15 version of the game, respectively. Furthermore, they observed that the incidence of concussion varied considerably between levels of play with the sub-elite level having highest incidence of injury (2.08 per 1000 player match hours). They noted a similar rate of concussion between forwards and backs in men's rugby-15 s at 4.02 and 4.85 concussions per 1000 player match hours, respectively. During RL matches, the incidence rate for concussion has been reported to vary widely from 0 to 40 per 1000 playing hours.

The incidence rate for concussion in children and adolescent players in RU games is 0.2–6.9 per 1000 hours whereas in RL games it varies between 4.6 and 14.7 per 1000 hours [136]. Similarly, the probability of a player sustaining a concussion in the same cohort over a season is 0.3–11.4% and 7.7–22.7% for RU and RL, respectively [136]. Semi-professional RL players have concussion injury risk which is threefold and 600-fold greater compared to their amateur and professional counterparts, respectively [137].

The wide variation in the reported incidence rate of concussion is due to a combination of factors including the inconsistencies in definition of injury (time-loss vs. no time-loss), sampling and methodology of the included studies [135]. Hence, following the IRB pilot study [138], World Rugby has subsequently introduced a guideline with three-stage diagnostic process and assessment criteria to identify or rule out concussion within 48 hours of injury [139].

8.7.3.2 Peripheral Nervous System

Common peripheral spine injuries include facet fractures, disc injuries, and nerve root compressions. Additionally, acute spinal cord injuries (ASCI) have been reported by several investigators [140–143].

In general, injuries to the cervical spine are rare [59, 144] compared to other injuries. Carmody et al. reported average annual incidence of ASCI at 3.2 and 1.5 per 100,000 players for RU and RL, respectively [140]. However, they are among the most serious form of injuries noted in rugby players and are associated with poor outcomes [144]. The most common mechanism of injury is hyperflexion of the cervical spine with subsequent fracture dislocation of C4–C5 or C5–C6 [144]. Investigators have reviewed several external (phase of play, time of season, coach input, referee control of game, pitch, and environmental conditions) and player-related (age, gender, ethnicity, position, skill, anthropometric parameters, visual acuity, physiological and psychological characteristics) risk factors to evaluate spinal injuries [59, 143–146]. The majority of these injuries have been noted to occur early in the season which is due to a combination of the grounds being harder and the players lacking adequate conditioning for physical contact phases of the game [144]. Earlier studies suggested that these injuries were sustained by the forward players (predominantly the hooker) during scrum [19, 143–145]. Additionally, the scrum has been the cause of other spinal injuries (56% of thoracic, 71% of lumbar) in rugby players [53]. However, following the rule changes to 'de-power' the scrum by controlling the engagement, the risk of cervical spine injury is now relatively higher during tackle than scrum [147, 148].

8.7.4 Injuries from Bone Stresses and Fractures (Four per 1000 Player Hours, 95% CI 2–8)

Whilst relatively less frequent, this group constitutes more severe degrees of injury in terms of time-loss (42 days, 95% CI 32–51) [99]. This group of injuries includes fractures involving: (1) Axial spine, (2) upper limb, and (3) lower limb.

Cervical spine fractures associated with spinal cord injury in rugby have significant morbidity [59, 144, 148–155] and in rare cases mortality [156] associated with them. Using a porcine biomechanical model, Holsgrove and colleagues demonstrated that lordosis of the cervical spine was a key factor for anterior fractures of the vertebral body and bilateral dislocation with facet fractures [157]. They observed that the anterior fractures resulted from tension in the cervical vertebral bodies following the buckling of the cervical spine in extension [157]. Furthermore, they suggested that a large axial load transfer from the head to torso with severe movement constraints as noticed during improper engagement during scrummaging could potential cause these injuries [157]. Thoracic and lumbosacral spine injuries including stress fractures in rugby players have been described by several investigators [158–163].

Fractures involving the forearm, wrist and hand/finger account for 90% of all upper limb fractures [128]. Similar findings have been reported in a prospective cohort study of rugby players across various competition levels as part of the Rugby Union Injury Surveillance Study (RUISS) [164]. Furthermore, fractures were one of the most common form of injury to the upper limb, occurring in 17% of the cases [164]. Sixty percent of upper limb fractures occur during tackle and are seen in wing- or prop-forward positions [128]. However, the prognosis with these injuries is relatively better, with Robertson et al. reporting that 94% of players with upper limb fractures were able to return to sport by 6 months following the index injury [165].

Lower limb fractures are relatively less common, accounting for 0.8–1.8% [111, 166] of overall injuries. Nonetheless, they are associated with significant morbidity and time-loss ranging between 118 and 471 days [111, 167].

8.7.5 Laceration and Skin Injuries (One per 1000 Player Hours, 95% CI 1–3)

Lacerations to the head and face are common injuries amongst rugby players [98]. Several factors including studs have been suggested in the aetiology of skin lacerations [168]. Following their meta-analysis, Oudshoorn et al. defined the mean skin injury prevalence of 2.4 and 0.06 injuries per 1000 exposure hours during match and training sessions, respectively [169]. They noted that amateur players were more likely to sustain skin injuries during training sessions compared to professional rugby union players.

8.8 Epidemiology (Prevalence and Incidence of Injury)

Amongst the various team sports, rugby has been reported to register one of the higher overall rates of injury (69 per 1000 playing hours) compared to soccer (28 per 1000 playing hours) and ice hockey (53 per 1000 playing hours) [170]. This has been attributed to greater player size, speed with multidirectional nature of play, increased competitiveness, more aggression, and also foul play [171]. Additionally, rugby as a sport has well-established injury surveillance systems enabling documentation of training and match-related injuries to players on a regular basis [110, 120, 166, 172–179].

One of the limitations in the available literature is that the definition of injury used by different investigators has varied considerably [31, 51, 95, 101, 180–183]. Definitions ranging from the need for on-field assessment and/or treatment, to attendance at medical stations after the game, to missed games and/or training sessions have been highlighted by authors who have reviewed the epidemiological data on rugby injuries [39, 95, 184]. It has been suggested that each definition changes the 'injury' characteristics. Hence, inclusion of match injuries will increase the rate and include more minor soft tissue injuries and concussion. Contrarily, exclusion of match injuries and focusing on injuries resulting in loss of playing or training time will bias the injury patterns towards the more serious spectrum of musculoskeletal and neurological injuries [39, 135].

The International Rugby Board (IRB) established a Rugby Injury Consensus Group (RICG) to reach an agreement on the appropriate definitions and methodologies to standardise the recording of injuries and reporting of studies in RU. Subsequently, a consensus statement was published by Fuller and colleagues [185]. This document provides definition of injury, recurrent injury, and non-fatal catastrophic injury together with criteria for classifying injuries in terms of severity, location, type, diagnosis, and causation [185]. The following definition of injury was accepted:

> Any physical complaint, which was caused by a transfer of energy that exceeded the body's ability to maintain its structural and/or functional integrity, that was sustained by a player during a rugby match or rugby training, irrespective of the need for medical attention or time-loss from rugby activities. An injury that results in a player receiving medical attention is referred to as a 'medical-attention' injury and an injury that results in a player being unable to take a full part in future rugby training or match play as a 'time-loss' injury.

Irrespective of the methodological variabilities in the epidemiological studies of rugby injuries, certain general points can be inferred as below:

1. Overall there is a higher incidence of injuries reported in RU and RL compared to other team sports [30, 166, 176, 186–190].
2. Amateur players and professional players have been reported to have different injury risk (Tables 8.2, 8.3, 8.5, and 8.6) [191, 192].
3. The incidence of RL injuries typically increases as the playing level is increased [192]. In a recent meta-analysis examining RU, Yeomans et al. reported match injury incidence rate of 46.8 and 81 per 1000 player hours for amateur and professional cohorts, respectively [29].
4. The sevens version of the game has higher incidence of injuries compared to 15 member team format in women [189, 193] and men [34, 194, 195].
5. Injuries are most commonly sustained during tackles [28, 189, 196].
6. Player fatigue and overexertion are amongst the most common cause of injuries sustained during training [192, 197, 198].

It is apparent from the above that the dynamics of the game and consequently the risk of injury to players in the two popular formats of rugby such as RU and RL is considerably different. Therefore, a brief summary of studies describing the injury incidence in RU (Tables 8.1, 8.2, and 8.3) and RL (Tables 8.4, 8.5, and 8.6) players at different competitive levels has been presented separately below.

Table 8.1 Rugby union—summary of injury incidence amongst school/junior players (<19 years)

Author	Location	Study period	Age group/game type	Injury incidence (rate of injury per 1000 playing hours)
Sparks [199]	England	1950–1979	13–18	19.8
Davidson [200]	Australia	1969–1986	11–19	17.6
Nathan et al. [201]	South Africa	1982	10–19	8.2
Garraway [186]	Scotland	1993–1994	Under 16 18–19	3.4 8.67
Garraway et al. [202]	Scotland	1993–1994 1997–1998	Under 16 16–19 Under 16 16–19	4.6 10.4 10.8 16.8
McManus et al. [203]	Australia	1997	Under 16	13.26
Durie [204]	New Zealand	1998	Under 19	27.5
McIntosh et al. [39]	Australia	2002	Under 15 Under 18	40.4 52.6
Palmer-Green et al. [205]	England	2006–2008	16–18 (academy) 16–18 (school)	47 35
Nicol et al. [206]	Scotland	2008–2009	11–18	10.8
Leung et al. [207]	Australia	2016	Overall 17–18 14–16 10–13	23.7 14.8 34.9–49.2 9.1–15.5
Pringle [208]	New Zealand	NR	6–15	15.5

NR not reported

Table 8.2 Rugby union—summary of injury incidence amongst amateur/semi-professional players

Author	Location	Study period	Age group/game type	Injury incidence (rate of injury per 1000 playing hours)
Bird et al. [166]	New Zealand	1993	Amateur	
			Senior A	14
			Senior B	10.7
Garraway et al. [186]	Scotland	1993–1994	Amateur	13.95
Schneiders et al. [119]	New Zealand	2002	Amateur	52
Chalmers et al. [209]	New Zealand	2004	Amateur	
			Senior A	15.4
			Senior B	10.5
Roberts et al. [210]	England	2009–2012	Amateur	16.6
Lopez et al. [211]	USA	2010	Sevens	55.4
Swain et al. [212]	Australia	2012	Amateur	52.3
Falkenmire et al. [213]	Australia	2016	Amateur	164

Table 8.3 Rugby union—summary of injury incidence amongst professional players

Author	Location	Study period	Age group/game type	Injury incidence (rate of injury per 1000 playing hours)
Bathgate et al. [181]	Australia	1994–2000	Elite–15	74
Jakoet et al. [214]	International	1995	Elite–15	32
Targett et al. [215]	New Zealand	1997	Elite–15	120
Garraway et al. [202]	Scotland	1997–1998	Elite–15	68
Holtzhausen et al. [216]	South Africa	1999	Elite–15	
			Match	55.4
			Training	4.3
Best et al. [120]	International	2003	Elite–15	97.9
Brooks et al. [217]	England	2003	Elite–15	
			Match	218
			Training	6
Brooks et al. [24]	England	2003	Elite–15	
			Match	91
Brooks et al. [122]	England	2003	Elite–15	
			Training	2
Fuller et al. [173]	International	2007	Elite–15	
			Match	84
			Training	4
Cruz-Ferreira et al. [34]	International	2010–2013	Sevens	101.5–119.8
Fuller et al. [174]	International	2011	Elite–15	
			Match	89
			Training	2
Fuller et al. [175]	International	2015	Elite–15	
			Match	90.1
			Training	1

Table 8.4 Rugby league—summary of injury incidence amongst school/junior players (<19 years)

Author	Location	Study period	Age group/game type	Injury incidence (rate of injury per 1000 playing hours)
Gabbett et al. [218]	Australia	NR	17–19	56.8
King et al. [219]	New Zealand	2005	Under 16	217.9
King et al. [219]	New Zealand	2005	Under 18	216
Estell et al. [183]	Australia	NR	Under 19	405.6
Estell et al. [183]	Australia	NR	Under 17	343.2
Estell et al. [183]	Australia	NR	Under 15	197.8

Table 8.5 Rugby league—summary of injury incidence amongst amateur/semi-professional players

Author	Location	Study period	Age group/game type	Injury incidence (rate of injury per 1000 playing hours)
Gabbett et al. [105]	Australia	2000–2001	Semi-professional	
			Training	105.9
			Match	917.3
Gabbett et al. [100]	Australia	1995–1997	Amateur	160.6
Gabbett et al. [101]	Australia	2000–2001	Semi-professional	824.7
King et al. [220]	New Zealand	NR	Amateur Training	22.4
Gabbett et al. [108]	Australia	2000–2003	Semi-professional	55.4
Babic et al. [180]	Croatia	NR	Amateur	18.22

Table 8.6 Rugby league—summary of injury incidence amongst professional players

Author	Location	Study period	Age group/game type	Injury incidence (rate of injury per 1000 playing hours)
Gibbs et al. [106]	Australia	1989–1991	Professional	44.9
Stephenson et al. [221]	England	1990–1994	Professional	114.3
Seward et al. [104]	Australia	1992	Professional	139
Hodgson Phillips et al. [222]	UK	1993–1996	Professional	462.7
Gissane et al. [223]	Europe	1996	Professional	50.3
Gissane et al. [224]	International	1990–2000	Professional	
			First	40.8
			Reserve	38.9
			Overall	40.3
Estell et al. [183]	Australia	NR	Professional	210.7

8.9 Anatomic Locations of Common Rugby Injuries

The majority of the injuries (30–55% of injuries) in rugby players affect the lower limb [24, 34, 98, 167, 176, 181, 182, 210]. Head and spine (14–30%), upper limbs (15–20%), and trunk (10–14%) are the other common location of injuries [225] (Fig. 8.2).

Amongst professional players, the head (including concussions) is the most affected part of the body (25%), followed by the knee (14–20%), thighs (13–19%), and ankle (11%) [24, 111, 117, 225]. Roberts et al. noted that in professional rugby league players, the lower limb was the most common injury site for rucks, mauls, lineouts, scrums, and tackles [41]. However, the upper limb was the most common site for tackling injuries. Furthermore, they reported a higher incidence of upper limb injuries to the tackler compared with the tackled player ($p < 0.001$) and a higher incidence of lower limb and trunk injuries to the tackled player (both $p < 0.001$).

Lower limb injuries represent a high proportion of injuries because the fundamental elements of the game involve running and lower limb tasks such as acceleration, deceleration, tackling, and impact [109].

8.10 Unique Prevention Plans to Avoid the Most Common Injuries

Injury prevention in the different formats of rugby has been the focus of authorities and investigators alike [63, 209, 226–231]. Some of the salient aspects of injury prevention in rugby are presented below:

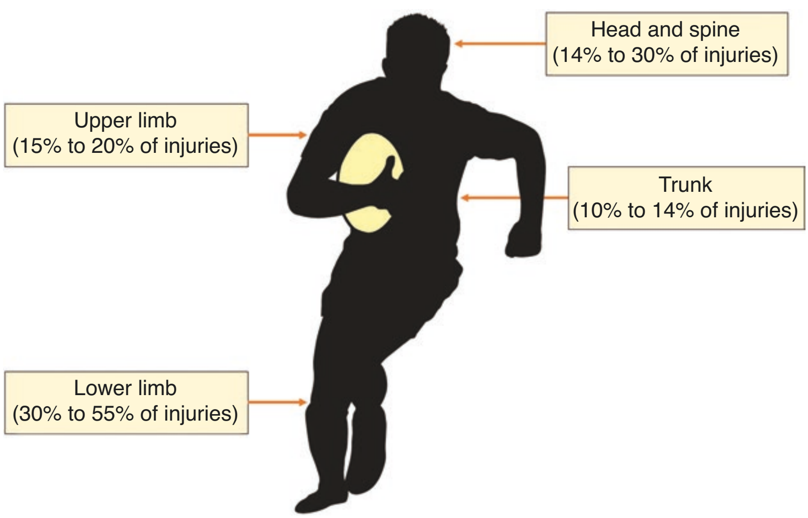
8.10.1 Contribution of International/National Governing Bodies

Injury prevention in rugby has been recognised as priority area for research and the major governing bodies including World Rugby and Rugby League International Federation have reviewed the regulations of the game and promoted systematic research in this area [173, 175, 230, 232, 233].

At a national level, the following initiatives have been undertaken with systematic research into prevention of catastrophic injuries and subsequent programmes:

1. South Africa—BokSmart was launched in 2009 [226, 234]. This programme consists of mandatory biennial courses aimed at coaches

Fig. 8.2 Anatomic locations of rugby injuries



and referees [235]. It has been associated with injury prevention behaviour amongst players and an overall decrease in catastrophic injuries in junior rugby players in South Africa [236, 237].

2. New Zealand—RugbySmart was launched in 2001 as a joint project between New Zealand Rugby Union and Accident Compensation Corporation [238]. Annual completion of the RugbySmart requirements was compulsory for all coaches and referees in order to continue with the job. This programme has been associated with reduction in number of spinal injuries [239] and injuries to the neck and back following safe scrum engagement that was implemented as part of it [58].
3. England—FMC:RUGBY project, a collaboration between Rugby Football Union and University of Bath is involved in the development of warm-up and training programmes to minimise injury risk [184].
4. Australia—SmartRugby is an occupational health and safety programme operated by Australian Rugby Union [184].

Van Mechelen and colleagues proposed a sports injury prevention model in 1992 [240]. It consists of four steps: establish the extent of the problem, establish the aetiology, and mechanism of the sports injury, introduce preventive measures, and evaluate the effectiveness of prevention strategies by repeating step one [240]. It must be noted that amongst the aforementioned programmes, only BokSmart and RugbySmart have completed all the four steps [184]. Hence, this will continue to be an area for further research and development.

8.10.2 Protective Equipment-Based Studies and Their Evidence

Several investigators have evaluated the feasibility and effectiveness of protective equipment as part of injury prevention strategy in rugby [241–247]. However, current evidence suggests that protective equipment (headgear) does not significantly reduce the risk of injuries including concussion [134, 245, 246] or spinal injuries [59]. Some studies have found mouthguards to be beneficial [248–250] whereas others [245] have found no significant difference in the reduction of orofacial injuries. Given the variability in literature, some authors have suggested that this equipment may play a 'protective' role and not necessarily a 'preventive' role in injuries and that their use needs to be encouraged [250–252]. As a part of player welfare, WR have enlisted performance specifications and general requirements for body padding, headgear, and goggles amongst other equipment [253].

8.10.3 Injury-Specific Programmes and Plans

8.10.3.1 Concussion

Over the course of a season, the probability of concussion to a child or adolescent rugby player is between 0.3% and 11.4% in rugby union and 7.7% or 22.7% in rugby league [136]. There is evidence to support educational programmes of coaches and referees to prevent concussion on rugby union [254]. It must be noted that the majority of the current evidence has been generated from just four rugby playing countries (Australia, New Zealand, South Africa, and UK) [136]. Furthermore, given the differential popularity between the two rugby codes, most of the current literature is based on rugby union [136]. Nonetheless, the scope for targeted injury prevention programmes and the beneficial impact of such programmes in limiting concussion injury is obvious [255].

Following the IRB pilot study [138], World Rugby has subsequently introduced a guideline with three-stage diagnostic process and assessment criteria to identify or rule out concussion within 48 hours of injury [139]. Currently, the fifth edition of the Sport Concussion Assessment Tool (SCAT5) is recommended for players who are 13 years of age or older whereas the child SCAT5 is intended for use in players aged 5–12 years [255]. In a recent systematic review, Patricios et al. reported that the overall strength of evidence examining sideline screening tools was very low [256]. Hence, given the lack of definitive evidence confirming the diagnostic accuracy of sideline screening tests, the authors recommended the use of consensus-derived multimodal assessment tools such as SCAT [256].

8.10.3.2 Cervical Spine Injury

Scrum has been phase of play associated with cervical spine injuries during RU games in the past [257]. In January 2007, the IRB implemented a new law for scrum engagement aimed at minimising scrum collapses and the resultant injuries [58]. Fuller et al. attributed the significant reduction in cervical spine injures during scrummage to the positive implementation of this law which is aimed at overall player welfare [59]. It has since been included in the mandatory RugbySmart programme for coaches and referees in New Zealand [58, 238]. Isometric neck strengthening programmes have been demonstrated to improve neck strength in RU players with a potential to minimise the risk of cervical spine injuries [258–260].

In comparison to RU, majority of the cervical injuries in RL players are a result of being tackled during a game [192, 261]. Following rule changes, scrum has been 'de-powered' in RL games. This has been attributed to have significantly reduced the incidence of severe cervical spine injuries in RL [261].

8.10.3.3 Shoulder Injury

Shoulder muscle strengthening has been a focus area given the predisposition of rugby players to shoulder injuries [51]. The use of shoulder pads has been explored by investigators [262, 263]. Currently, there is no demonstrable evidence to support their use to minimise injury risk [264].

8.10.3.4 Ankle Injury

Sankey et al. noted that 35% of ankle injuries were sustained during non-contact activities. There is ample evidence to suggest that proprioception-based training regimen is beneficial in reducing risk of ankle injuries [265–267]. Hence, incorporation of such regimen may play a role in minimising ankle injury risk in rugby players.

8.10.4 Training-Based Programmes

Given that tackle or being tackled is the predominant reason for injury during matches, investigators have focussed on the identification of tackler characteristics associated with positive tackle outcome thereby minimising injury risk. Hendricks et al. demonstrated that appropriate tackle training of players was associated with behaviours that reduced risk of serious injuries during matches [268]. Head positioned up, forward and facing the ball carrier, counter-acting the ball carrier, shoulder tackles targeted at the mid-torso of the ball carrier, using the arms to wrap or pull the ball carrier and leg driving after contact have been associated with positive tackle outcomes [269].

Training frequencies of two to four resistance training sessions per muscle group/week has been recommended to develop upper and lower body strength and power [270]. Tackle-related mechanisms have been reported to be amongst the leading causes of injury in rugby players with both formats of the game [39, 42, 97, 98, 112]. Hence, this has been an area of continued research and training programmes have been developed to teach players the safe tackle methods to minimise risk of injury [51, 271].

Simulation model-based studies have been performed by some investigators to enhance our understanding of the complex injury patterns in rugby and help develop better injury prevention strategies and training programmes [271–273].

8.10.5 Other Injury Prevention Strategies

It has been reported that normal variations in development observed in children of the same age can result in significant differences in physical characteristics leading to a mismatch in size [274]. Some authors have suggested that grouping of child rugby players merely on chronological age may pose a potential injury risk due to the disparity in physical size [39, 184, 275]. Hence, in New Zealand child rugby players are matched by size rather than chronological age and skills has been adopted as strategy to help reduce the frequency of severe injuries [276]. Other mechanisms such as 'weigh down' rule have been proposed and used in some competitions wherein players of higher chronological age are permitted to participate in a younger age group category if their weight is below the competition agreed threshold for a particular age group [39].

8.11 Paralympic Rugby Athletes

Wheelchair rugby as a team sport for paralympic athletes has grown in popularity since its origin back in 1976 and was included as a medal event in the 2000 Summer Paralympics in Sydney, Australia. Athletes competing in wheelchair rugby can have loss of function in the limbs or impairment from spinal cord injuries at the level of cervical vertebrae, multiple amputations, neurological conditions such as cerebral palsy, muscular dystrophy, and polio amongst other conditions. A detailed description of the laws of this sport are beyond the scope of this chapter and can be found at the International Wheelchair Rugby Federation website (www.iwrf.com) [277]. However, it is useful to note that it is a competitive team sport performed by male and female athletes with some of the aforementioned conditions [277–279]. Some of the unique features of this sport include (1) team consists of 12 players of which only 4 may be on the court at any time (2) physical contact between wheelchairs is permitted but direct physical contact between players is not permitted (3) wheelchair rugby is played indoors on a hardwood court similar in dimensions to a basketball court.

The paralympic athletes participating in wheelchair rugby are classified based on impairments that cause activity limitations in the sport into one of seven groups (from 0.5 to 3.5 points) [277]. To ensure fairness and team balance of functional levels, the total value of all players on each team cannot exceed 8 points [277]. This is vital as it has been noted that different propulsion approaches [280], asymmetries [281], speed and activity [278] exist across classification groups with arm impairment in players having a greater impact on performance compared to trunk impairment [282]. Furthermore, these players are under greater thermal strain due to their reduced heat loss capacity [279]. However, wheelchair rugby training enables players to improve cardiorespiratory function [283, 284].

The type of wheelchair prescription including design features such as seat depth, seat angle, wheel camber angle, and wheel diameter amongst others can contribute significantly to performance in these players [285–287].

Sports injuries in paralympic athletes participating in wheelchair rugby may have serious consequences and impact their ability to function independently for daily activities [288]. Nonetheless, information available in the current literature regarding injury patterns in paralympic athletes playing wheelchair rugby is limited. In a pilot study, Bauerfeind et al. [289] studied 14 male players from national team over a 9-month period and reported incidence rate 0.3 per athlete per training day. However, majority of the injuries were minor in nature and did not require medical intervention. Furthermore, injuries occurred more frequently in offensive players than in defensive players [289].

8.12 Summary

Rugby is a contact sport and the two popular formats of rugby union and rugby league are associated with a relatively high risk of injury to players compared to other team sports. The unique mechanics of this sport involve different contact situations and player characteristics, which can result in distinct pattern of injuries. The majority of these injuries are soft tissue injuries of the lower limb, including muscle, tendon, or ligament injuries, whilst head and neck injuries are less common, but frequent enough to be a significant ongoing concern. Epidemiological data and injury-related research have helped develop and implement laws and programmes aimed at minimising injury risk to players, with evidence that effective training programmes and law changes can reduce the risk of injury. In the future, incorporation of evolving technologies has the potential to enhance our understanding of the complex injury patterns, and enable development of more robust injury prevention strategies and programmes.

References

1. Nicholas CW. Anthropometric and physiological characteristics of rugby union football players. Sports Med. 1997;23(6):375–96.
2. Higham DG, et al. Physiological, anthropometric, and performance characteristics of rugby sevens players. Int J Sports Physiol Perform. 2013;8(1):19–27.
3. MacQueen AE, Dexter WW. Injury trends and prevention in rugby union football. Curr Sports Med Rep. 2010;9(3):139–43. https://doi.org/10.1249/JSR.0b013e3181df124c.
4. Baker WJ. William Webb Ellis and the origins of Rugby Football: the life and death of a Victorian myth. Albion. 1981;13(2):117–30.
5. World Rugby: Laws of the game - Rugby Union. 2020. https://laws.worldrugby.org/.
6. Collins T. Rugby's great split: class, culture and the origins of rugby league football. Abingdon: Routledge; 2012.
7. Collins T. The oval world: a global history of rugby. London: Bloomsbury Publishing; 2015.
8. WorldRugby. World Rugby Year in review. 2018. http://publications.worldrugby.org/yearinreview2018/en/56-1. Accessed 20 Dec 2019.
9. League IR. History at a glance. International Rugby League. 2019. http://www.rlif.com/the_game/history_of_the_game. Accessed 2020.
10. Ross A. Match analysis and player characteristics in rugby sevens. Sports Med. 2014;44(3):357–67.
11. Fuller CW, Taylor A, Raftery M. 2016 Rio Olympics: an epidemiological study of the men's and women's Rugby-7s tournaments. Br J Sports Med. 2017;51(17):1272–8.
12. The Rugby Football League: Laws of the game. 2020. https://www.rugby-league.com/the_rfl/rules/laws_of_the_game.
13. Rugby AU: Laws of Rugby. 2020. https://australia.rugby/participate/referee/laws.
14. World Rugby: regulations relating to the game. 2020. https://www.world.rugby/handbook.
15. World Rugby hanbook. 2020. https://www.world.rugby/handbook.
16. Howitt B, Deaker J. The converts: changing codes. New York: HarperCollins; 2011.
17. Wikipedia. List of dual-code rugby internationals. 2020. https://en.wikipedia.org/wiki/List_of_dual-code_rugby_internationals.
18. Thomas D. The rugby revolution: new horizons or false dawn? Econ Aff. 1997;17(3):19–24.
19. Milburn PD. The kinetics of rugby union scrummaging. J Sports Sci. 1990;8(1):47–60. https://doi.org/10.1080/02640419008732130.
20. Patricios JS. Rugby contact and collisions—clinical challenges of a global game. Curr Sports Med Rep. 2014;13(5):326–33.
21. Fuller CW, Ashton T, Brooks JH, Cancea RJ, Hall J, Kemp SP. Injury risks associated with tackling in rugby union. Br J Sports Med. 2010;44(3):159–67.
22. Sirotic AC. Positional match demands of professional rugby league competition. J Strength Cond Res. 2011;25(11):3076–87.
23. Waldron M. Movement and physiological match demands of elite rugby league using portable global positioning systems. J Sports Sci. 2011;29(11):1223–30.
24. Brooks JHM, Fuller CW, Kemp SPT, Reddin DB. Epidemiology of injuries in English professional rugby union: part 1 match injuries. Br J Sports Med. 2005;39(10):757–66. https://doi.org/10.1136/bjsm.2005.018135.
25. Burger N, Lambert MI, Viljoen W, Brown JC, Readhead C, Hendricks S. Tackle-related injury rates and nature of injuries in South African Youth Week tournament rugby union players (under-13 to under-18): an observational cohort study. BMJ Open. 2014;4(8):e005556. https://doi.org/10.1136/bmjopen-2014-005556.
26. Roberts SP, Trewartha G, England M, Shaddick G, Stokes KA. Epidemiology of time-loss injuries in English community-level rugby union. BMJ Open. 2013;3(11):e003998.
27. Hoskins W, Pollard H, Hough K, Tully C. Injury in rugby league. J Sci Med Sport. 2006;9(1–2):46–56. https://doi.org/10.1016/j.jsams.2006.03.013.
28. McIntosh AS, Savage TN, McCrory P, Frechede BO, Wolfe R. Tackle characteristics and injury in a cross section of rugby union football. Med Sci Sports Exerc. 2010;42(5):977–84. https://doi.org/10.1249/MSS.0b013e3181c07b5b.
29. Yeomans C, Kenny IC, Cahalan R, Warrington GD, Harrison AJ, Hayes K, et al. The incidence of injury in amateur male rugby union: a systematic review and meta-analysis. Sports medicine (Auckland, NZ). 2018;48(4):837–48.
30. Viviers PL, Viljoen JT, Derman W. A review of a decade of rugby union injury epidemiology: 2007–2017. Sports Health. 2018;10(3):223–7.
31. Ball S, Halaki M, Sharp T, Orr R. Injury patterns, physiological profile, and performance in university rugby union. Int J Sports Physiol Perform. 2018;13(1):69–74.
32. Leung FT, Franettovich Smith MM, Brown M, Rahmann A, Mendis MD, Hides JA. Epidemiology of injuries in Australian school level rugby union. J Sci Med Sport. 2017;20(8):740–4.
33. Pollock AM, White AJ, Kirkwood G. Evidence in support of the call to ban the tackle and harmful contact in school rugby: a response to World Rugby. Br J Sports Med. 2017;51(15):1113–7.

34. Cruz-Ferreira A, Cruz-Ferreira E, Santiago L, Taborda Barata L. Epidemiology of injuries in senior male rugby union sevens: a systematic review. Phys Sportsmed. 2017;45(1):41–8.
35. Burger N, Lambert MI, Viljoen W, Brown JC, Readhead C, den Hollander S, et al. Mechanisms and factors associated with tackle-related injuries in South African youth rugby union players. Am J Sports Med. 2017;45(2):278–85.
36. Booth M, Orr R. Time-loss injuries in sub-elite and emerging rugby league players. J Sports Sci Med. 2017;16(2):295–301.
37. Sabesan V, Steffes Z, Lombardo DJ, Petersen-Fitts GR, Jildeh TR. Epidemiology and location of rugby injuries treated in US emergency departments from 2004 to 2013. Open Access J Sports Med. 2016;7:135–42.
38. Pfirrmann D, Herbst M, Ingelfinger P, Simon P, Tug S. Analysis of injury incidences in male professional adult and elite youth soccer players: a systematic review. J Athl Train. 2016;51(5):410–24. https://doi.org/10.4085/1062-6050-51.6.03.
39. McIntosh AS. Rugby injuries. Med Sport Sci. 2005;49:120–39. https://doi.org/10.1159/000085394.
40. Gabbett TJ. Relationship between tests of physical qualities, team selection, and physical match performance in semiprofessional rugby league players. J Strength Cond Res. 2013;27(12):3259–65.
41. Roberts SP, Trewartha G, England M, Stokes KA. Collapsed scrums and collision tackles: what is the injury risk? Br J Sports Med. 2015;49(8):536–40.
42. Quarrie KL, Hopkins WG. Tackle injuries in professional Rugby Union. Am J Sports Med. 2008;36(9):1705–16.
43. Austin D, Gabbett T, Jenkins D. Tackling in a professional rugby league. J Strength Cond Res. 2011;25(6):1659–63. https://doi.org/10.1519/JSC.0b013e3181da781c.
44. Garraway WM, Lee AJ, Macleod DA, Telfer JW, Deary IJ, Murray GD. Factors influencing tackle injuries in rugby union football. Br J Sports Med. 1999;33(1):37–41.
45. Tanabe Y, Kawasaki T, Tanaka H, Murakami K, Nobuhara K, Okuwaki T, et al. The kinematics of 1-on-1 rugby tackling: a study using 3-dimensional motion analysis. J Shoulder Elb Surg. 2019;28(1):149–57.
46. Seminati E, Cazzola D, Preatoni E, Trewartha G. Specific tackling situations affect the biomechanical demands experienced by rugby union players. Sports Biomech. 2017;16(1):58–75.
47. Virr JL. Physiological demands of women's rugby union: time-motion analysis and heart rate response. J Sports Sci. 2014;32(3):239–47.
48. Quarrie KL. Changes in player characteristics and match activities in Bledisloe Cup rugby union from 1972 to 2004. J Sports Sci. 2007;25(8):895–903.
49. Eaves S, Hughes M. Patterns of play of international rugby union teams before and after the introduction of professional status. Int J Perform Anal Sport. 2003;3(2):103–11.
50. McLean DA, et al. Analysis of the physical demands of international rugby union. J Sports Sci. 1992;10(3):285–96.
51. Tomasin JD, Martin DF, Curl WW. Recognition and prevention of rugby injuries. Phys Sportsmed. 1989;17(6):114–26. https://doi.org/10.1080/00913847.1989.11709809.
52. Scher A. Rugby injuries to the cervical spinal cord sustained during rucks and mauls. Case reports. S Afr Med J. 1983;64(15):592–4.
53. Trewartha G, Preatoni E, England ME, Stokes KA. Injury and biomechanical perspectives on the rugby scrum: a review of the literature. Br J Sports Med. 2015;49(7):425–33.
54. Clayton JD, Kirkwood RN, Gregory DE. The influence of hip mobility and quadriceps fatigue on sagittal spinal posture and muscle activation in rugby scrum performance. Eur J Sport Sci. 2019;19(5):603–11. https://doi.org/10.1080/17461391.2018.1537379.
55. Hendricks S, Lambert MI, Brown JC, Readhead C, Viljoen W. An evidence-driven approach to scrum law modifications in amateur rugby played in South Africa. Br J Sports Med. 2014;48(14):1115–9. https://doi.org/10.1136/bjsports-2013-092877.
56. Yaghoubi M, Lark SD, Page WH, Fink PW, Shultz SP. Lower extremity muscle function of front row rugby union scrummaging. Sports Biomech. 2019;18(6):636–48. https://doi.org/10.1080/14763141.2018.1452972.
57. Taylor AE, Kemp S, Trewartha G, Stokes KA. Scrum injury risk in English professional rugby union. Br J Sports Med. 2014;48(13):1066–8.
58. Gianotti S, Hume PA, Hopkins WG, Harawira J, Truman R. Interim evaluation of the effect of a new scrum law on neck and back injuries in rugby union. Br J Sports Med. 2008;42(6):427–30.
59. Fuller CW, Brooks JH, Kemp SP. Spinal injuries in professional rugby union: a prospective cohort study. Clin J Sport Med. 2007;17(1):10–6. https://doi.org/10.1097/JSM.0b013e31802e9c28.
60. Mirsafaei Rizi R, Yeung SS, Stewart NJ, Yeung EW. Risk factors that predict severe injuries in university rugby sevens players. J Sci Med Sport. 2017;20(7):648–52.
61. Duthie GM, Pyne DB, Marsh DJ, Hooper SL. Sprint patterns in rugby union players during competition. J Strength Cond Res. 2006;20(1):208–14. https://doi.org/10.1519/R-16784.1.
62. Gabbett TJ. Relationship between running loads and soft-tissue injury in elite team sport athletes. J Strength Cond Res. 2012;26(4):953–60.
63. Gabbett TJ, et al. Identifying risk factors for contact injury in professional rugby league players—appli-

cation of a frailty model for recurrent injury. J Sci Med Sport. 2012;15(6):496–504.
64. Sirotic AC, et al. A comparison of match demands between elite and semi-elite rugby league competition. J Sports Sci. 2009;27:203–11.
65. Gabbett TJ. Sprinting patterns of National Rugby League competition. J Strength Cond Res. 2012;26(1):121–30. https://doi.org/10.1519/JSC.0b013e31821e4c60.
66. Johnston RD, et al. Influence of an intensified competition on fatigue and match performance in junior rugby league players. J Sci Med Sport. 2013;16(5):460–5.
67. Kenneally-Dabrowski C, Serpell BG, Spratford W, Lai AKM, Field B, Brown NAT, et al. A retrospective analysis of hamstring injuries in elite rugby athletes: more severe injuries are likely to occur at the distal myofascial junction. Phys Therapy Sport. 2019;38:192–8.
68. Brooks JH, Fuller CW, Kemp SP, Reddin DB. Incidence, risk, and prevention of hamstring muscle injuries in professional rugby union. Am J Sports Med. 2006;34(8):1297–306. https://doi.org/10.1177/0363546505286022.
69. Linthorne NP, Stokes TG. Optimum projection angle for attaining maximum distance in a rugby place kick. J Sports Sci Med. 2014;13(1):211–6.
70. Bezodis NE, Atack A, Willmott AP, Callard JEB, Trewartha G. Kicking foot swing planes and support leg kinematics in rugby place kicking: differences between accurate and inaccurate kickers. Eur J Sport Sci. 2019;19(4):451–60.
71. Atack A, Trewartha G, Bezodis NE. Assessing rugby place kick performance from initial ball flight kinematics: development, validation and application of a new measure. Sports Biomech. 2019;18(5):457–69. https://doi.org/10.1080/14763141.2018.1433714.
72. Quarrie KL, Hopkins WG. Evaluation of goal kicking performance in international rugby union matches. J Sci Med Sport. 2015;18(2):195–8. https://doi.org/10.1016/j.jsams.2014.01.006.
73. Werstein KM, Lund RJ. The effects of two stretching protocols on the reactive strength index in female soccer and rugby players. J Strength Cond Res. 2012;26(6):1564–7.
74. Austin D, Gabbett T, Jenkins D. Repeated high-intensity exercise in professional rugby union. J Sports Sci. 2011;29(10):1105–12. https://doi.org/10.1080/02640414.2011.582508.
75. Brewer J, Davis J. Applied physiology of rugby league. Sports Med. 1995;20(3):129–35. https://doi.org/10.2165/00007256-199520030-00001.
76. Gabbett T, King T, Jenkins D. Applied physiology of rugby league. Sports Med (Auckland, NZ). 2008;38(2):119–38.
77. Gabbett TJ. A comparison of physiological and anthropometric characteristics among playing positions in sub-elite rugby league players. J Sports Sci. 2006;24(12):1273–80.
78. Gabbett TJ. Science of rugby league football: a review. J Sports Sci. 2005;23(9):961–76. https://doi.org/10.1080/02640410400023381.
79. Docherty D, Wenger HA, Neary P. Time-motion analysis related to the physiological demands of rugby. J Hum Mov Stud. 1988;14:269–77.
80. Meir R, Arthur D, Forrest M. Time and motion analysis of professional rugby league: a case study. Strength Cond Coach. 1993;1:24–9.
81. Cunniffe B, Hore AJ, Whitcombe DM, Jones KP, Baker JS, Davies B. Time course of changes in immuneoendocrine markers following an international rugby game. Eur J Appl Physiol. 2010;108(1):113–22. https://doi.org/10.1007/s00421-009-1200-9.
82. Gabbett T. Relationship between physical fitness and playing ability in rugby league players. J Strength Cond Res. 2007;21(4):1126–33.
83. Gabbett TJ. Influence of fatigue on tackling technique in rugby league players. J Strength Cond Res. 2008;22(2):625–32. https://doi.org/10.1519/JSC.0b013e3181635a6a.
84. Granatelli G, et al. Match analysis and temporal patterns of fatigue in rugby sevens. J Strength Cond Res. 2014;28:728–34.
85. Lee AJ, et al. Influence of players' physique on rugby football injuries. Br J Sports Med. 1997;31:135–8.
86. Fuller CW. Should player fatigue be the focus of injury prevention strategies for international rugby sevens tournaments? Br J Sports Med. 2016;50(11):682–7.
87. Gabbett TJ, Polley C, Dwyer DB, Kearney S, Corvo A. Influence of field position and phase of play on the physical demands of match-play in professional rugby league forwards. J Sci Med Sport. 2014;17(5):556–61. https://doi.org/10.1016/j.jsams.2013.08.002.
88. McLellan CP, et al. Performance analysis of elite Rugby League match play using global positioning systems. J Strength Cond Res. 2011;25(6):1703–10.
89. Quarrie KL. Positional demands of international rugby union: evaluation of player actions and movements. J Sci Med Sport. 2013;16(4):353–9.
90. Vaz L, et al. Fitness profiles of elite portuguese rugby union players. J Hum Kinet. 2014;41:235–44.
91. Gabbett T, et al. A comparison of fitness and skill among playing positions in sub-elite rugby league players. J Sci Med Sport. 2008;11(6):585–92.
92. Gabbett TJ. Influence of playing position on the site, nature, and cause of rugby league injuries. J Strength Cond Res. 2005;19(4):749–55. https://doi.org/10.1519/R-16504.1.
93. Olds T. The evolution of physique in male rugby union players in the twentieth century. J Sports Sci. 2001;19:253–62. https://doi.org/10.1080/026404101750158312.
94. Cunningham DJ, Shearer DA, Drawer S, Pollard B, Eager R, Taylor N, et al. Movement demands of elite under-20s and senior international rugby union play-

ers. PLoS One. 2016;11(11):e0164990. https://doi.org/10.1371/journal.pone.0164990.
95. Brooks JH, Kemp SP. Recent trends in rugby union injuries. Clin Sports Med. 2008;27(1):51–73, vii–viii. https://doi.org/10.1016/j.csm.2007.09.001.
96. Kauffman T. Rugby injuries sustained during tournament play. J Orthop Sports Phys Ther. 1985;7(1):16–9. https://doi.org/10.2519/jospt.1985.7.1.16.
97. Kaplan KM, Goodwillie A, Strauss EJ, Rosen JE. Rugby injuries: a review of concepts and current literature. Bull NYU Hosp Jt Dis. 2008;66(2):86–93.
98. Gibbs N. Common rugby league injuries. Recommendations for treatment and preventative measures. Sports Med (Auckland, NZ). 1994;18(6):438–50.
99. Williams S, Trewartha G, Kemp S, Stokes K. A meta-analysis of injuries in senior men's professional Rugby Union. Sports Med. 2013;43(10):1043–55. https://doi.org/10.1007/s40279-013-0078-1.
100. Gabbett TJ. Incidence, site, and nature of injuries in amateur rugby league over three consecutive seasons. Br J Sports Med. 2000;34(2):98–103.
101. Gabbett TJ. Incidence of injury in semi-professional rugby league players. Br J Sports Med. 2003;37(1):36.
102. Gissane C, Jennings DC, Cumine AJ, Stephenson SE, White JA. Differences in the incidence of injury between rugby league forwards and backs. Aust J Sci Med Sport. 1997;29(4):91–4.
103. Wilson BD, Quarrie KL, Milburn PD, Chalmers DJ. The nature and circumstances of tackle injuries in rugby union. J Sci Med Sport. 1999;2(2):153–62.
104. Seward H, Orchard J, Hazard H, Collinson D. Football injuries in Australia at the elite level. Med J Aust. 1993;159(5):298–301.
105. Gabbett TJ. Influence of training and match intensity on injuries in rugby league. J Sports Sci. 2004;22(5):409–17.
106. Gibbs N. Injuries in professional rugby league. A three-year prospective study of the South Sydney Professional Rugby League Football Club. Am J Sports Med. 1993;21(5):696–700.
107. Gissane C, Jennings D, Kerr K, White J. Injury rates in rugby league football: impact of change in playing season. Am J Sports Med. 2003;31(6):954–8.
108. Gabbett TJ, Domrow N. Risk factors for injury in subelite rugby league players. Am J Sports Med. 2005;33(3):428–34.
109. Devlin L. Recurrent posterior thigh symptoms detrimental to performance in rugby union: predisposing factors. Sports Med (Auckland, NZ). 2000;29(4):273–87.
110. Brazier J, Antrobus M, Stebbings GK, Day SH, Heffernan SM, Cross MJ, et al. Tendon and ligament injuries in elite rugby: the potential genetic influence. Sports (Basel, Switzerland). 2019;7(6):138.
111. Sankey RA, Brooks JH, Kemp SP, Haddad FS. The epidemiology of ankle injuries in professional rugby union players. Am J Sports Med. 2008;36(12):2415–24.
112. Gabbett TJ. Incidence of injury in junior and senior rugby league players. Sports Med. 2004;34(12):849–59. https://doi.org/10.2165/00007256-200434120-00004.
113. Awwad GEH, Coleman JH, Dunkley CJ, Dewar DC. An analysis of knee injuries in rugby league: the experience at the Newcastle Knights Professional Rugby League Team. Sports Med Open. 2019;5(1):33. https://doi.org/10.1186/s40798-019-0206-z.
114. Shewring D, Matthewson M. Injuries to the hand in rugby union football. J Hand Surg Br. 1993;18(1):122–4.
115. Elzinga KE, Chung KC. Finger injuries in football and rugby. Hand Clin. 2017;33(1):149–60.
116. Bachoura A, Ferikes AJ, Lubahn JD. A review of mallet finger and jersey finger injuries in the athlete. Curr Rev Musculoskelet Med. 2017;10(1):1–9.
117. Dallalana RJ, Brooks JH, Kemp SP, Williams AM. The epidemiology of knee injuries in English professional rugby union. Am J Sports Med. 2007;35(5):818–30.
118. Montgomery C, Blackburn J, Withers D, Tierney G, Moran C, Simms C. Mechanisms of ACL injury in professional rugby union: a systematic video analysis of 36 cases. Br J Sports Med. 2018;52(15):994–1001. https://doi.org/10.1136/bjsports-2016-096425.
119. Schneiders AG, Takemura M, Wassinger CA. A prospective epidemiological study of injuries to New Zealand premier club rugby union players. Phys Therapy Sport. 2009;10(3):85–90.
120. Best JP, McIntosh AS, Savage TN. Rugby World Cup 2003 injury surveillance project. Br J Sports Med. 2005;39(11):812–7. https://doi.org/10.1136/bjsm.2004.016402.
121. Usman J, McIntosh AS, Quarrie K, Targett S. Shoulder injuries in elite rugby union football matches: epidemiology and mechanisms. J Sci Med Sport. 2015;18(5):529–33.
122. Brooks JHM, Fuller CW, Kemp SPT, Reddin DB. Epidemiology of injuries in English professional rugby union: part 2 training Injuries. Br J Sports Med. 2005;39(10):767–75. https://doi.org/10.1136/bjsm.2005.018408.
123. Headey J, Brooks JH, Kemp SP. The epidemiology of shoulder injuries in English professional rugby union. Am J Sports Med. 2007;35(9):1537–43.
124. Horsley IG, Fowler EM, Rolf CG. Shoulder injuries in professional rugby: a retrospective analysis. J Orthop Surg Res. 2013;8(1):9.
125. Crichton J, Jones DR, Funk L. Mechanisms of traumatic shoulder injury in elite rugby players. Br J Sports Med. 2012;46(7):538–42. https://doi.org/10.1136/bjsports-2011-090688.
126. Montgomery C, O'Briain DE, Hurley ET, Pauzenberger L, Mullett H, Moran CJ. Video analysis of shoulder dislocations in rugby: insights into the dislocating mechanisms. Am J Sports Med. 2019;2019:363546519882412.

127. Barton N. Sports injuries of the hand and wrist. Br J Sports Med. 1997;31(3):191–6.
128. Eyres KS, Abdel-Salam A, Cleary J. Upper limb fractures in rugby in Huddersfield 1986–1990. Br J Sports Med. 1991;25(3):139–41. https://doi.org/10.1136/bjsm.25.3.139.
129. McFie S, Brown J, Hendricks S, Posthumus M, Readhead C, Lambert M, et al. Incidence and factors associated with concussion injuries at the 2011 to 2014 South African Rugby Union Youth Week Tournaments. Clin J Sport Med. 2016;26(5):398–404.
130. Cusimano M, Cho N, Amin K, Shirazi M, McFaull S, Do M, et al. Mechanisms of team-sport-related brain injuries in children 5 to 19 years old: opportunities for prevention. PLoS One. 2013;8:e58868. https://doi.org/10.1371/journal.pone.0058868.
131. Kemp SP, Hudson Z, Brooks JH, Fuller CW. The epidemiology of head injuries in English professional rugby union. Clin J Sport Med. 2008;18(3):227–34.
132. King D, Hume P, Clark T. The effect of player positional groups on the nature of tackles that result in tackle-related injuries in professional rugby league matches. J Sports Med Phys Fitness. 2011;51(3):435–43.
133. King D, et al. Head impacts in a junior rugby league team measured with a wireless head impact sensor: an exploratory analysis. J Neurosurg Pediatr. 2017;19:13–23.
134. Gardner AJ, Iverson GL, Williams WH, Baker S, Stanwell P. A systematic review and meta-analysis of concussion in rugby union. Sports Med (Auckland, NZ). 2014;44(12):1717–31.
135. Gardner A, Iverson GL, Levi CR, Schofield PW, Kay-Lambkin F, Kohler RM, et al. A systematic review of concussion in rugby league. Br J Sports Med. 2015;49(8):495–8.
136. Kirkwood G, Parekh N, Ofori-Asenso R, Pollock AM. Concussion in youth rugby union and rugby league: a systematic review. Br J Sports Med. 2015;49(8):506–10.
137. King D, Hume P, Gissane C, Clark T. Semi-professional rugby league players have higher concussion risk than professional or amateur participants: a pooled analysis. Sports Med (Auckland, NZ). 2017;47(2):197–205.
138. Fuller GW, Kemp SP, Decq P. The International Rugby Board (IRB) Pitch Side Concussion Assessment trial: a pilot test accuracy study. Br J Sports Med. 2015;49(8):529–35. https://doi.org/10.1136/bjsports-2014-093498.
139. Raftery M, et al. It is time to give concussion an operational definition: a 3-step process to diagnose (or rule out) concussion within 48 h of injury: World Rugby guideline. Br J Sports Med. 2016;50(11):642–3.
140. Carmody DJ, Taylor TKF, Parker DA, Coolican MRJ, Cumming RG. Spinal cord injuries in Australian footballers 1997–2002. Med J Aust. 2005;182(11):561–4. https://doi.org/10.5694/j.1326-5377.2005.tb06814.x.
141. Armour KS, Clatworthy BJ, Bean AR, Wells JE, Clarke AM. Spinal injuries in New Zealand rugby and rugby league—a twenty year survey. N Z Med J. 1997;110(1057):462–5.
142. Noakes T, Jakoet I. Spinal cord injuries in rugby union players. BMJ. 1995;310(6991):1345–6.
143. Hermanus FJ, Draper CE, Noakes TD. Spinal cord injuries in South African Rugby Union (1980–2007). S Afr Med J. 2010;100(4):230–4. https://doi.org/10.7196/samj.4061.
144. Quarrie KL, Cantu RC, Chalmers DJ. Rugby union injuries to the cervical spine and spinal cord. Sports Med. 2002;32(10):633–53. https://doi.org/10.2165/00007256-200232100-00003.
145. Hoskins TW. Prevention of neck injuries playing rugby. Public Health. 1987;101(5):351–6. https://doi.org/10.1016/s0033-3506(87)80094-x.
146. Silver JR. Injuries of the spine sustained during rugby. Br J Sports Med. 1992;26(4):253–8. https://doi.org/10.1136/bjsm.26.4.253.
147. Madden C, Putukian M, McCarty E, Young C. Netter's sports medicine E-book. Amsterdam: Elsevier Health Sciences; 2017.
148. Shelly MJ, Butler JS, Timlin M, Walsh MG, Poynton AR, O'Byrne JM. Spinal injuries in Irish rugby: a ten-year review. J Bone Joint Surg Br. 2006;88(6):771–5. https://doi.org/10.1302/0301-620X.88B6.17388.
149. Milburn PD. Biomechanics of rugby union scrummaging. Technical and safety issues. Sports Med (Auckland, NZ). 1993;16(3):168–79.
150. Newton D, England M, Doll H, Gardner BP. The case for early treatment of dislocations of the cervical spine with cord involvement sustained playing rugby. J Bone Joint Surg Br. 2011;93(12):1646–52. https://doi.org/10.1302/0301-620X.93B12.27048.
151. Schroeder GD, Vaccaro AR. Cervical spine injuries in the athlete. Instr Course Lect. 2017;66:391–402.
152. Secin FP, Poggi EJ, Luzuriaga F, Laffaye HA. Disabling injuries of the cervical spine in Argentine rugby over the last 20 years. Br J Sports Med. 1999;33(1):33–6.
153. Silver JR. The impact of the 21st century on rugby injuries. Spinal Cord. 2002;40(11):552–9. https://doi.org/10.1038/sj.sc.3101349.
154. Bohu Y, Julia M, Bagate C, Peyrin JC, Colonna JP, Thoreux P, et al. Declining incidence of catastrophic cervical spine injuries in French rugby: 1996–2006. Am J Sports Med. 2009;37(2):319–23.
155. Scher AT. Catastrophic rugby injuries of the spinal cord: changing patterns of injury. Br J Sports Med. 1991;25(1):57–60. https://doi.org/10.1136/bjsm.25.1.57.
156. Brown JC, Lambert MI, Verhagen E, Readhead C, van Mechelen W, Viljoen W. The incidence of rugby-related catastrophic injuries (including cardiac events) in South Africa from 2008 to 2011: a cohort study. BMJ Open. 2013;3(2):e002475. https://doi.org/10.1136/bmjopen-2012-002475.
157. Holsgrove TP, Cazzola D, Preatoni E, Trewartha G, Miles AW, Gill HS, et al. An investigation into axial

impacts of the cervical spine using digital image correlation. Spine J. 2015;15(8):1856–63.
158. Hind K, Birrell F, Beck B. Prevalent morphometric vertebral fractures in professional male rugby players. PLoS One. 2014;9(5):e97427. https://doi.org/10.1371/journal.pone.0097427.
159. Castinel BH, Adam P, Prat C. A stress fracture of the lumbar spine in a professional rugby player. Br J Sports Med. 2007;41(5):337–8. https://doi.org/10.1136/bjsm.2006.032789.
160. Walsh AJ, Shine S, McManus F. Paraplegia secondary to fracture-subluxation of the thoracic spine sustained playing rugby union football. Br J Sports Med. 2004;38(6):e32. https://doi.org/10.1136/bjsm.2003.009837.
161. Shafik A, Schwabe K, de Villiers R, Viljoen J, Derman W. Stress fracture of the thoracic spine in a male rugby player: a case report. S Afr J Sports Med. 2018;30(1):1–2.
162. Geffen S, Gibbs N, Geffen L. Thoracic spinal fracture in a rugby league footballer. Clin J Sport Med. 1997;7(2):144–6.
163. Takahashi Y, Kobayashi T, Miyakoshi N, Abe E, Abe T, Kikuchi K, et al. Sacral stress fracture in an amateur rugby player: a case report. J Med Case Rep. 2016;10(1):327.
164. Usman J, McIntosh AS. Upper limb injury in rugby union football: results of a cohort study. Br J Sports Med. 2013;47(6):374–9.
165. Robertson G, Wood AM, Heil K, Aitken SA, Court-Brown CM. The epidemiology, morbidity and outcome of fractures in rugby union from a standard population. Orthop Proc. 2013;95-B(Suppl_26):21. https://doi.org/10.1302/1358-992x.95bsupp_26.csos2013-021.
166. Bird YN, Waller AE, Marshall SW, Alsop JC, Chalmers DJ, Gerrard DF. The New Zealand Rugby Injury and Performance Project: V. Epidemiology of a season of rugby injury. Br J Sports Med. 1998;32(4):319–25.
167. Whitehouse T, Orr R, Fitzgerald E, Harries S, McLellan CP. The epidemiology of injuries in Australian professional rugby union 2014 super rugby competition. Orthop J Sports Med. 2016;4(3):2325967116634075.
168. Oudshoorn BY, Driscoll HF, Dunn M, James D. Causation events of stud laceration injuries in rugby union. Proc Eng. 2016;147:496–500.
169. Oudshoorn BY, Driscoll H, Kilner K, Dunn M, James D. Prevalence of laceration injuries in professional and amateur rugby union: a systematic review and meta-analysis. BMJ Open Sport Exerc Med. 2017;3(1):e000239. https://doi.org/10.1136/bmjsem-2017-000239.
170. Duthie G, Pyne D, Hooper S. Applied physiology and game analysis of rugby union. Sports Med. 2003;33(13):973–91.
171. Brown JC, Verhagen E, Viljoen W, Readhead C, Van Mechelen W, Hendricks S, et al. The incidence and severity of injuries at the 2011 South African Rugby Union (SARU) Youth Week tournaments. S Afr J Sports Med. 2012;24(2):49–54.
172. Archbold HA, Rankin AT, Webb M, Nicholas R, Eames NW, Wilson RK, et al. RISUS study: rugby injury surveillance in Ulster schools. Br J Sports Med. 2017;51(7):600–6.
173. Fuller CW, Laborde F, Leather RJ, Molloy MG. International Rugby Board Rugby World Cup 2007 injury surveillance study. Br J Sports Med. 2008;42(6):452–9.
174. Fuller CW, Sheerin K, Targett S. Rugby World Cup 2011: International Rugby Board injury surveillance study. Br J Sports Med. 2013;47(18):1184–91.
175. Fuller CW, Taylor A, Kemp SP, Raftery M. Rugby World Cup 2015: World Rugby injury surveillance study. Br J Sports Med. 2017;51(1):51–7.
176. Leahy TM, Kenny IC, Campbell MJ, Warrington GD, Cahalan R, Harrison AJ, et al. Injury surveillance in school rugby: a systematic review of injury epidemiology & surveillance practices. Phys Therapy Sport. 2019;38:170–8.
177. Moore IS, Ranson C, Mathema P. Injury risk in international rugby union: three-year injury surveillance of the Welsh National Team. Orthop J Sports Med. 2015;3(7):2325967115596194.
178. Taylor AE, Fuller CW, Molloy MG. Injury surveillance during the 2010 IRB Women's Rugby World Cup. Br J Sports Med. 2011;45(15):1243–5.
179. Wekesa M, Asembo JM, Njororai WW. Injury surveillance in a rugby tournament. Br J Sports Med. 1996;30(1):61–3.
180. Babić Z, Misigoj-Duraković M, Matasić H, Jancić J. Croatian rugby project. Part II: Injuries. J Sports Med Phys Fitness. 2001;41(3):392–8.
181. Bathgate A, Best JP, Craig G, Jamieson M. A prospective study of injuries to elite Australian rugby union players. Br J Sports Med. 2002;36(4):265–9; discussion 9.
182. Carson JD, Roberts MA, White AL. The epidemiology of women's rugby injuries. Clin J Sport Med. 1999;9(2):75–8.
183. Estell J, Shenstone B, Barnsley L. Frequency of injuries in different age-groups in an elite rugby league club. Aust J Sci Med Sport. 1995;27(4):95–7.
184. Freitag A. Rugby injury surveillance and prevention programmes: are they effective? BMJ. 2015;350:h1587.
185. Fuller CW, Molloy MG, Bagate C, Bahr R, Brooks JHM, Donson H, et al. Consensus statement on injury definitions and data collection procedures for studies of injuries in rugby union. Br J Sports Med. 2007;41(5):328–31. https://doi.org/10.1136/bjsm.2006.033282.
186. Garraway M, Macleod D. Epidemiology of rugby football injuries. Lancet (London, England). 1995;345(8963):1485–7.
187. Barden C, Stokes K. Epidemiology of injury in elite English schoolboy rugby union: a 3-year study comparing different competitions. J Athl Train. 2018;53(5):514–20.

188. Bleakley C, Tully M, O'Connor S. Epidemiology of adolescent rugby injuries: a systematic review. J Athl Train. 2011;46(5):555–65.
189. King D, Hume P, Cummins C, Pearce A, Clark T, Foskett A, et al. Match and training injuries in women's rugby union: a systematic review of published studies. Sports Med (Auckland, NZ). 2019;49(10):1559–74.
190. King DA, Hume PA, Milburn PD, Guttenbeil D. Match and training injuries in rugby league: a review of published studies. Sports Med (Auckland, NZ). 2010;40(2):163–78.
191. Gabbett TJ. Severity and cost of injuries in amateur rugby league: a case study. J Sports Sci. 2001;19(5):341–7.
192. Gabbett TJ. Incidence of injury in junior and senior rugby league players. Sports Med (Auckland, NZ). 2004;34(12):849–59.
193. Ma R, Lopez V, Weinstein MG, Chen JL, Black CM, Gupta AT, et al. Injury profile of American Women's Rugby-7s. Med Sci Sports Exerc. 2016;48(10):1957–66.
194. Fuller CW, Taylor A, Raftery M. Epidemiology of concussion in men's elite Rugby-7s (Sevens World Series) and Rugby-15s (Rugby World Cup, Junior World Championship and Rugby Trophy, Pacific Nations Cup and English Premiership). Br J Sports Med. 2015;49(7):478–83.
195. Gabbett TJ. Incidence of injury in amateur rugby league sevens. Br J Sports Med. 2002;36(1):23–6.
196. King D, Hume PA, Clark T. Nature of tackles that result in injury in professional rugby league. Res Sports Med. 2012;20(2):86–104.
197. Gabbett TJ. Training injuries in rugby league: an evaluation of skill-based conditioning games. J Strength Cond Res. 2002;16(2):236–41.
198. Gabbett TJ, Godbolt RJ. Training injuries in professional rugby league. J Strength Cond Res. 2010;24(7):1948–53.
199. Sparks JP. Half a million hours of rugby football. The injuries. Br J Sports Med. 1981;15(1):30–2.
200. Davidson R. Schoolboy Rugby injuries, 1969–1986. Med J Aust. 1987;147:119–20. https://doi.org/10.5694/j.1326-5377.1987.tb133299.x.
201. Nathan T. The incidence and nature of rugby injuries experienced at one school during the 1982 rugby season. S Afr Med J. 1983;64(4):132–7.
202. Garraway WM, Lee AJ, Hutton SJ, Russell EB, Macleod DA. Impact of professionalism on injuries in rugby union. Br J Sports Med. 2000;34(5):348–51.
203. McManus A, Cross DS. Incidence of injury in elite junior Rugby Union: a prospective descriptive study. J Sci Med Sport. 2004;7(4):438–45. https://doi.org/10.1016/s1440-2440(04)80261-5.
204. Durie R. A prospective survey of injuries in a New Zealand schoolboy rugby population. N Z J Sports Med. 2000;28:84–90.
205. Palmer-Green DS, Stokes KA, Fuller CW, England M, Kemp SP, Trewartha G. Match injuries in English youth academy and schools rugby union: an epidemiological study. Am J Sports Med. 2013;41(4):749–55. https://doi.org/10.1177/0363546512473818.
206. Nicol A, Pollock A, Kirkwood G, Parekh N, Robson J. Rugby union injuries in Scottish schools. J Public Health (Oxf). 2011;33(2):256–61.
207. Leung FT, Franettovich Smith MM, Hides JA. Injuries in Australian school-level rugby union. J Sports Sci. 2017;35(21):2088–92.
208. Pringle RG, McNair P, Stanley S. Incidence of sporting injury in New Zealand youths aged 6–15 years. Br J Sports Med. 1998;32(1):49–52.
209. Chalmers DJ, Samaranayaka A, Gulliver P, McNoe B. Risk factors for injury in rugby union football in New Zealand: a cohort study. Br J Sports Med. 2012;46(2):95–102.
210. Roberts SP. Epidemiology of time-loss injuries in English community-level rugby union. BMJ Open. 2013;3(11):e003998.
211. Lopez V, Galano GJ, Black CM, Gupta AT, James DE, Kelleher KM, et al. Profile of an American amateur rugby union sevens series. Am J Sports Med. 2012;40(1):179–84.
212. Swain MS, Lystad RP, Henschke N, Maher CG, Kamper SJ. Match injuries in amateur Rugby Union: a prospective cohort study - FICS Biennial Symposium Second Prize Research Award. Chiropractic Manual Therap. 2016;24:17.
213. Falkenmire A, Manvell J, Callister R, Snodgrass S. Injury incidence, characteristics and timing in amateur male rugby union: a prospective cohort study. J Hum Sport Exerc. 2019;15:1. https://doi.org/10.14198/jhse.2020.153.08.
214. Jakoet I, Noakes TD. A high rate of injury during the 1995 Rugby World Cup. S Afr Med J. 1998;88(1):45–7.
215. Targett SG. Injuries in professional Rugby Union. Clin J Sport Med. 1998;8(4):280–5.
216. Holtzhausen LJ, Schwellnus MP, Jakoet I, Pretorius AL. The incidence and nature of injuries in South African rugby players in the rugby Super 12 competition. S Afr Med J. 2006;96(12):1260–5.
217. Brooks JHM, Fuller CW, Kemp SPT, Reddin DB. A prospective study of injuries and training amongst the England 2003 Rugby World Cup squad. Br J Sports Med. 2005;39(5):288–93. https://doi.org/10.1136/bjsm.2004.013391.
218. Gabbett TJ. Incidence of injury in junior rugby league players over four competitive seasons. J Sci Med Sport. 2008;11(3):323–8.
219. King D. Incidence of injuries in the 2005 New Zealand national junior rugby league competition. N Z J Sports Med. 2006;34:21–7.
220. King DA, Gabbett TJ. Training injuries in New Zealand amateur rugby league players. J Sci Med Sport. 2008;11(6):562–5. https://doi.org/10.1016/j.jsams.2007.04.011.
221. Stephenson S, Gissane C, Jennings D. Injury in rugby league: a four year prospective survey. Br J Sports Med. 1997;30:331–4. https://doi.org/10.1136/bjsm.30.4.331.

222. Phillips LH, Standen P, Batt M. Effects of seasonal change in rugby league on the incidence of injury. Br J Sports Med. 1998;32(2):144–8.
223. Gissane C, Jennings D, White J, Cumine A. Injury in summer rugby league football: the experiences of one club. Br J Sports Med. 1998;32(2):149–52.
224. Gissane C, Jennings D, Kerr K, White JA. A pooled data analysis of injury incidence in rugby league football. Sports Med (Auckland, NZ). 2002;32(3):211–6.
225. Kaux J-F, Julia M, Delvaux F, Croisier J-L, Forthomme B, Monnot D, et al. Epidemiological review of injuries in rugby union. Sports. 2015;3(1):21–9.
226. Patricios J. BokSmart - South African rugby's national rugby safety and injury prevention program. Curr Sports Med Rep. 2014;13(3):142–4. https://doi.org/10.1249/JSR.0000000000000049.
227. Chalmers DJ, Simpson JC, Depree R. Tackling Rugby injury: lessons learned from the implementation of a five-year sports injury prevention program. J Sci Med Sport. 2004;7(1):74–84. https://doi.org/10.1016/s1440-2440(04)80046-x.
228. Brown JC, Cross M, England M, Finch CF, Fuller GW, Kemp SPT, et al. Guidelines for community-based injury surveillance in rugby union. J Sci Med Sport. 2019;22(12):1314–8.
229. Brown J, Viljoen W, Readhead C, Baerecke G, Lambert M, Finch CF. 'VisionZero': is it achievable for rugby-related catastrophic injuries in South Africa? Br J Sports Med. 2017;51(15):1106–7.
230. Rugby W. Player Welfare. World Rugby. 2020. https://playerwelfare.worldrugby.org/.
231. Hislop MD, Stokes KA, Williams S, McKay CD, England ME, Kemp SPT, et al. Reducing musculoskeletal injury and concussion risk in schoolboy rugby players with a pre-activity movement control exercise programme: a cluster randomised controlled trial. Br J Sports Med. 2017;51(15):1140–6.
232. Fuller CW, Jones R, Fuller AD. Defining a safe player run-off zone around rugby union playing areas. Inj Prev. 2015;21(5):309–13. https://doi.org/10.1136/injuryprev-2015-041587.
233. Federation RLI. The Official Laws of the Game. http://www.rlif.com/about/regulations_and_laws.
234. Brown JC, et al. 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.
235. Viljoen W, Patricios J. Boksmart–implementing a national rugby safety Programme. London: BMJ Publishing Group Ltd and British Association of Sport and Exercise Medicine; 2012.
236. Brown JC, Gardner-Lubbe S, Lambert MI, Van Mechelen W, Verhagen E. Coach-directed education is associated with injury-prevention behaviour in players: an ecological cross-sectional study. Br J Sports Med. 2018;52(15):989–93.
237. Brown JC, Verhagen E, Knol D, Van Mechelen W, Lambert MI. The effectiveness of the nationwide BokSmart rugby injury prevention program on catastrophic injury rates. Scand J Med Sci Sports. 2016;26(2):221–5. https://doi.org/10.1111/sms.12414.
238. Gianotti SM, Quarrie KL, Hume PA. Evaluation of RugbySmart: a rugby union community injury prevention programme. J Sci Med Sport. 2009;12(3):371–5. https://doi.org/10.1016/j.jsams.2008.01.002.
239. Quarrie KL, Gianotti SM, Hopkins WG, Hume PA. Effect of nationwide injury prevention programme on serious spinal injuries in New Zealand rugby union: ecological study. BMJ. 2007;334(7604):1150. https://doi.org/10.1136/bmj.39185.605914.AE.
240. Van Mechelen W, Hlobil H, Kemper HC. Incidence, severity, aetiology and prevention of sports injuries. Sports Med. 1992;14(2):82–99.
241. Comstock RD. Protective equipment use among female rugby players. Clin J Sports Med. 2005;15(4):241–5.
242. Finch CF, McIntosh AS, McCrory P, Zazryn T. A pilot study of the attitudes of Australian Rules footballers towards protective headgear. J Sci Med Sport. 2003;6(4):505–11. https://doi.org/10.1016/s1440-2440(03)80275-x.
243. Jones SJ, Lyons RA, Evans R, Newcombe RG, Nash P, McCabe M, et al. Effectiveness of rugby headgear in preventing soft tissue injuries to the head: a case-control and video cohort study. Br J Sports Med. 2004;38(2):159–62. https://doi.org/10.1136/bjsm.2002.002584.
244. Marshall SW. Use of protective equipment in a cohort of rugby players. Med Sci Sports Exerc. 2001;33(12):2131–8.
245. Marshall SW, Loomis DP, Waller AE, Chalmers DJ, Bird YN, Quarrie KL, et al. Evaluation of protective equipment for prevention of injuries in rugby union. Int J Epidemiol. 2005;34(1):113–8. https://doi.org/10.1093/ije/dyh346.
246. McIntosh AS, McCrory P. Effectiveness of headgear in a pilot study of under 15 rugby union football. Br J Sports Med. 2001;35(3):167–9.
247. Pettersen JA. Does rugby headgear prevent concussion? Attitudes of Canadian players and coaches. Br J Sports Med. 2002;36(1):19–22.
248. Jennings DC. Injuries sustained by users and non-users of gum shields in local rugby union. Br J Sports Med. 1990;24(3):159–65. https://doi.org/10.1136/bjsm.24.3.159.
249. Quarrie KL, Gianotti SM, Chalmers DJ, Hopkins WG. An evaluation of mouthguard requirements and dental injuries in New Zealand rugby union. Br J Sports Med. 2005;39(9):650–1. https://doi.org/10.1136/bjsm.2004.016022.
250. Schildknecht S, Krastl G, Kuhl S, Filippi A. Dental injury and its prevention in Swiss rugby. Dent

Traumatol. 2012;28(6):465–9. https://doi.org/10.1111/j.1600-9657.2012.01115.x.
251. Navarro RR. Protective equipment and the prevention of concussion - what is the evidence? Curr Sports Med Rep. 2011;10(1):27–31.
252. Pless B. Commentary: Mad New Zealanders, tape, and grease: assessing protective equipment for rugby union players. Int J Epidemiol. 2005;34(1):119–20. https://doi.org/10.1093/ije/dyh379.
253. World Rugby: Players' equipment. 2020. https://playerwelfare.worldrugby.org/players-equipment. Accessed 2 Feb 2020.
254. Fraas MR, Burchiel J. A systematic review of education programmes to prevent concussion in rugby union. Eur J Sport Sci. 2016;16(8):1212–8. https://doi.org/10.1080/17461391.2016.1170207.
255. 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. https://doi.org/10.1136/bjsports-2017-097506.
256. Patricios J, Fuller GW, Ellenbogen R, Herring S, Kutcher JS, Loosemore M, 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.
257. Scher AT. Rugby injuries of the spine and spinal cord. Clin Sports Med. 1987;6(1):87–99.
258. Geary K, Green BS, Delahunt E. Effects of neck strength training on isometric neck strength in rugby union players. Clin J Sport Med. 2014;24(6):502–8. https://doi.org/10.1097/JSM.0000000000000071.
259. Naish R, et al. Can a specific neck strengthening program decrease cervical spine injuries in a men's professional rugby union team? A retrospective analysis. J Sports Sci Med. 2013;12(3):542–50.
260. Hrysomallis C. Neck muscular strength, training, performance and sport injury risk: a review. Sports Med. 2016;46(8):1111–24. https://doi.org/10.1007/s40279-016-0490-4.
261. Berry JG, Harrison JE, Yeo JD, Cripps RA, Stephenson SCR. Cervical spinal cord injury in rugby union and rugby league: are incidence rates declining in NSW? Aust N Z J Public Health. 2006;30(3):268–74. https://doi.org/10.1111/j.1467-842X.2006.tb00869.x.
262. Harris DA, Spears IR. The effect of rugby shoulder padding on peak impact force attenuation. Br J Sports Med. 2010;44(3):200–3. https://doi.org/10.1136/bjsm.2008.047449.
263. Pain MT, Tsui F, Cove S. In vivo determination of the effect of shoulder pads on tackling forces in rugby. J Sports Sci. 2008;26(8):855–62. https://doi.org/10.1080/02640410801910319.
264. Gerrard DF. The use of padding in rugby union. An overview. Sports Med (Auckland, NZ). 1998;25(5):329–32.
265. Schiftan GS, Ross LA, Hahne AJ. The effectiveness of proprioceptive training in preventing ankle sprains in sporting populations: a systematic review and meta-analysis. J Sci Med Sport. 2015;18(3):238–44.
266. 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(6):1385–93.
267. Eils E. The role of proprioception in the primary prevention of ankle sprains in athletes. Int SportMed J. 2003;4(5):1–9.
268. Hendricks S. The relationships between rugby players' tackle training attitudes and behaviour and their match tackle attitudes and behaviour. Saf Sci. 2015;50:266–84.
269. Hendricks S. Tackler characteristics associated with tackle performance in rugby union. Eur J Sport Sci. 2014;14(8):753–62.
270. McMaster DT. The development, retention and decay rates of strength and power in elite rugby union, rugby league and American football: a systematic review. Sports Med. 2013;43(5):367–84.
271. Tierney GJ, Simms CK. The effects of tackle height on inertial loading of the head and neck in Rugby Union: a multibody model analysis. Brain Inj. 2017;31(13–14):1925–31.
272. Cazzola D, Holsgrove TP, Preatoni E, Gill HS, Trewartha G. Cervical spine injuries: a whole-body musculoskeletal model for the analysis of spinal loading. PLoS One. 2017;12(1):e0169329.
273. Silvestros P, Preatoni E, Gill HS, Gheduzzi S, Hernandez BA, Holsgrove TP, et al. Musculoskeletal modelling of the human cervical spine for the investigation of injury mechanisms during axial impacts. PLoS One. 2019;14(5):e0216663. https://doi.org/10.1371/journal.pone.0216663.
274. Beunen G, Malina RM. Growth and biologic maturation: relevance to athletic performance. The young athlete. Malden, MA: Blackwell Publishing; 2008. p. 3–17.
275. Krause LM, Naughton GA, Denny G, Patton D, Hartwig T, Gabbett TJ. Understanding mismatches in body size, speed and power among adolescent rugby union players. J Sci Med Sport. 2015;18(3):358–63.
276. Quarrie K, Gianotti S, Murphy I. Injury risk in New Zealand Rugby Union: a nationwide study of injury insurance claims from 2005 to 2017. Sports Med (Auckland, NZ). 2019;50(2):415–28.
277. International Wheelchair Rugby Federation: Rules and documents. 2020. https://www.iwrf.com/?page=rules_and_documents.
278. Rhodes JM, Mason BS, Malone LA, Goosey-Tolfrey VL. Effect of team rank and player classification on activity profiles of elite wheelchair rugby players. J Sports Sci. 2015;33(19):2070–8. https://doi.org/10.1080/02640414.2015.1028087.
279. Griggs KE, et al. Thermoregulatory responses during competitive wheelchair rugby match play. Int J Sports Med. 2017;38:177–83.

280. Haydon DS, Pinder RA, Grimshaw PN, Robertson WSP. Overground-propulsion kinematics and acceleration in elite wheelchair rugby. Int J Sports Physiol Perform. 2018;13(2):156–62. https://doi.org/10.1123/ijspp.2016-0802.
281. Goosey-Tolfrey VL, Vegter RJK, Mason BS, Paulson TAW, Lenton JP, van der Scheer JW, et al. Sprint performance and propulsion asymmetries on an ergometer in trained high- and low-point wheelchair rugby players. Scand J Med Sci Sports. 2018;28(5):1586–93. https://doi.org/10.1111/sms.13056.
282. Mason BS. Understanding the impact of trunk and arm impairments on wheelchair rugby performance during competition. Int J Sports Physiol Perform. 2019;14(5):612–9.
283. Moreno MA, et al. Wheelchair rugby improves pulmonary function in people with tetraplegia after 1 year of training. J Strength Cond Res. 2013;27(1):50–6.
284. Barfield JP, Malone LA, Arbo C, Jung AP. Exercise intensity during wheelchair rugby training. J Sports Sci. 2010;28(4):389–98. https://doi.org/10.1080/02640410903508839.
285. Haydon DS, et al. Wheelchair Rugby chair configurations: an individual, Robust design approach. Sports Biomech. 2019; https://doi.org/10.1080/14763141.2019.1649451.
286. Vanlandewijck Y, Theisen D, Daly D. Wheelchair propulsion biomechanics: implications for wheelchair sports. Sports Med (Auckland, NZ). 2001;31(5):339–67.
287. Goosey-Tolfrey VL, Leicht CA. Field-based physiological testing of wheelchair athletes. Sports Med. 2013;43(2):77–91. https://doi.org/10.1007/s40279-012-0009-6.
288. Vanlandewijck YC, Thompson WR. Handbook of sports medicine and science: the paralympic athlete. Hoboken, NJ: Wiley; 2011.
289. Bauerfeind J, Koper M, Wieczorek J, Urbański P, Tasiemski T. Sports injuries in wheelchair rugby - a pilot study. J Hum Kinet. 2015;48:123–32.

9 Soccer/Football

Sport-Specific Injuries and Unique Mechanisms in Soccer/Football

Marshall Haden, Leonard Onsen, Joshua Lam, Melody Hrubes, and Mark R. Hutchinson

9.1 Introduction

The sport known as soccer in the United States and Canada, and football in the remainder of the world, is the most popular team sport globally, with over 265 million regular players [1]. The first organized rules were developed in London in 1863 and have evolved over time into the game played today. The sport's governing body, Fédération Nationale de Football Association (FIFA), was formed in 1904 and has since grown to become one of the largest international organizations in the world, with 211 member nations. While already massively popular, participation continues to increase, particularly among women. In just 6 years (2000–2006), the number of women players increased 54% to 4.1 million players [1].

The game itself consists of two teams of 11 players. Ten outfield players per teamwork together to gain and maintain possession of the ball, with the object of shooting the ball into the goal on the opposing team's side of the field. These players can only use their feet to pass and dribble the ball, as the use of hands constitutes a foul. The lone goalkeeper for each team is able to use his hands to block or deflect the ball from going into his/her team's goal. Multiple variations of the standard game rules exist. These include smaller fields, goals, and/or team sizes to better accommodate younger players or players with disabilities. Another variation is indoor soccer, which can be played regardless of weather conditions.

Due to both the total volume of soccer players around the world, as well as the specific overuse and traumatic injury patterns and mechanisms related to play, soccer/football accounts for a significant number of injuries that either reduces the athlete's ability to perform or must be addressed by health care providers. Players are involved in frequent, rapid changes in speed or direction as well as jumping and diving activities, which place them at risk for muscle strains and ligamentous sprains. While many cases of direct contact between opposing players constitutes a foul, collisions between players occur frequently during gameplay, leading to traumatic injuries such as concussions, contusions, and/or fractures.

9.2 Unique Mechanics and Injury Risks in Soccer/Football

Soccer is a very active sport that offers unique mechanics and injury risks for athletes. Frequent actions of players include sprinting, jumping,

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kicking, cutting movements, and slide tackles. Additional actions used by goalies include diving, reaching, and throwing. Many of these movements are performed with the added difficulty of handling a ball or attempting to gain possession of the ball from an opponent. Muscles and joints of the lower extremity play a greater role in most soccer movements as compared to those of the upper extremity, which explains the greater incidence of lower extremity injuries [2]. Be that as it may, the sport does offer unique risks to the upper extremities and head/neck as well, particularly during throw-ins and contesting a headed ball. The most common soccer injuries include ligament sprains, muscle strains, contusions, and concussion. These can be related to chronic overuse or acute sport-related trauma.

Kicking is an essential soccer movement that is done for a variety of different goals such as passing, shooting, clearing the defensive zone, and dribbling. Phases of the kick include: (1) approach, (2) back swing, (3) leg cocking, (4) leg acceleration, and (5) ball impact with contralateral leg planting [3]. Of these phases, particular injury risks come with leg cocking, acceleration, and ball impact. During leg cocking, the quadriceps and hip flexor muscle groups are stretched to extreme amounts placing them at increased risk for strains. Following cocking, the kicking leg then undergoes rapid acceleration via concentric contractions of these same muscle groups to forcefully swing the foot towards the ball. This rapid contraction is an additional risk factor of injuries such as strains or tears. Finally, in ball impact the foot directly strikes the ball. This introduces direct trauma to a participant's foot in a repetitive manner given the frequency of this movement. The ankle is also at risk during this phase depending on the location where the foot strikes the ball or in the event an athlete comes up short and strikes the ground prior hitting the ball. If the ball is struck with a more distal aspect of the foot, an external rotation force is applied to the ankle which can place both medial deltoid and lateral ligament complexes of the ankle at risk for injury. If an athlete comes up short striking the ground with the midfoot before the ball, a primarily eversion force is applied to the ankle providing a risk for deltoid ligament injury.

Another soccer-specific movement is the slide tackle. This is a defensive move where a player attempts to take the ball from an opponent while sliding to the ground. It is often used as a last effort in breakaway situations or when a player is dribbling carelessly. In this movement, a player tucks the leg at the knee of the side they will land on during the slide. As they begin to drop into the slide, the other leg is directed towards the ball in order to knock it away from the opposing player. Injury risks with this movement include direct trauma to the tucked knee and hip that strikes the ground during the slide. Improper form or landing can result in abnormal rotational force at the ankle and knee that can cause ligamentous injuries. Also, the lead leg attempting to strike the ball is at risk for shin-to-shin contact of the leg of an opposing player which can result in contusions or, in extreme cases, a fracture.

A header is a potentially high-risk movement unique to soccer. In this movement, players jump to strike an airborne ball with their head, thereby redirecting it in an intended direction within the field of play. This can be done for both offensive and defensive purposes. Risks for this movement can be divided into those related to the act of jumping and to those related to the attempt of striking the ball with the head. Jumping is an explosive movement that involves both eccentric and concentric contractions largely of the lower extremities. Particular risk comes with landing where incorrect footing or landing on another player's foot can occur. The competitive contact that occurs during a contested header often throws the athlete off-balance, increasing the risk of an awkward landing. These incidents commonly cause ankle inversion and injure the lateral ankle ligament complex. The second mechanism of injury occurs in the actual heading portion of the skill. These events occur regularly in competition and is a skill that is repeated at practices. This exposes players to repetitive head trauma of various degrees. In severe cases when the head is impacted by a high-speed ball, or more commonly when the header is contested with an opponent leading to head-to-head contact, concussions can occur. There is some evidence that frequent heading leads to decreased baseline neurocognitive function [4], as well as anecdotal reports of chronic traumatic encephalopathy in soccer players [5]. However, the overall clinical significance of repetitive heading exposure in regard to its long-term impact on cognitive function and the brain is unknown.

Finally, it is important to recognize the unique demand and potential risks of the goalie compared to field players. Since goalies are allowed to use their hands, they have increased risks of upper extremity injuries compared to other soccer players [6]. A goalie's diving save is a soccer movement that provides greater risk to the upper extremity compared to most soccer movements (Fig. 9.1). To prevent a goal, goalies will at times need to dive with arms fully extended to block away or catch a shot attempt. Direct trauma to the digits from the ball can result in sprains, dislocations, or fractures of the fingers. Landing on an outstretched shoulder in the overhead position has the potential to cause subluxations, dislocations, or labral injuries. Landing directly on the lateral border of the acromion increases the risk of a shoulder pointer, acromio-clavicular injury, clavicle fracture, or sterno-clavicular injury. The goal posts can also be a structural hazard for the goalie. When tracking the ball in competition a goalie can inadvertently run or jump into a post which can cause injuries to the head or extremities. Catastrophic injuries in children have been reported when the goal was inadequately anchored to the ground and the wind caused the goal to tip over crushing the young athlete [7].

Overall, soccer is a safe sport. However, due to unique sports-specific movements and demands, certain injury patterns are seen, mainly involving the lower extremities. Though less prevalent, injuries to the head and upper extremities do exist and have unique risk factors in soccer. Targeting the most common injuries or most severe injuries by understanding their specific mechanism and injury patterns has the best chance to reduce the overall injury-related morbidity in this athletic population.

Fig. 9.1 Goalkeeper dive. (Source: Pexels on Pixabay)

9.3 Mechanism of Most Common Soccer/Football Injuries

9.3.1 Thigh Injuries

Injuries to the thigh (femur/quadriceps/hamstrings/adductors) account for some of the most frequent complaints and sites of injuries in competitive soccer players. Indeed, they account for as many as 31% of all injuries [8]. The majority of these injuries are muscle strains, with the hamstring muscles being the most often injured. In addition to muscle strains, thigh muscle contusions frequently occur during play.

When broken down by specific injury, hamstring strains are the single most common injury in professional soccer players [8]. The vast majority of these are noncontact injuries and occur during high-speed running or sprinting. A lesser portion of strains occur during other activities with forceful hamstring contraction such as jumping, twisting, shooting, stretching, or sliding [9, 10]. The hamstrings are active throughout the entire gait cycle, with the maximum contraction strength occurring in terminal swing phase. At this point, the hamstrings are eccentrically contracting to decelerate the knee extension and hip flexion which occur during swing phase. Terminal swing phase is also when the hamstrings are at their greatest length. When the foot contacts the ground the hamstrings transition from an eccentric to a forceful concentric contraction to extend the hip, flex the knee, and propel the body forward [11]. This portion of the gait cycle is when the hamstrings are most vulnerable to injury, particularly when running at high speeds such as frequently occurs in soccer. There is also evidence that injuries occur more frequently at the end of either half in gameplay, suggesting fatigue plays an important role in hamstring injury. Multiple variables have been evaluated as possible risk factors predisposing to hamstring strains. Increased age and prior hamstring injury are key risk factors with the most supporting evidence. Other variables which may predispose to injury are muscle imbalance, hamstring inflexibility, and functional limb length discrepancy [12–14].

Quadriceps strain is another commonly encountered soccer injury due to the frequent sprinting, change in direction, and kicking during gameplay. When compared to other sports, soccer players are the most likely to sustain a quadriceps strain [15]. Similar to the hamstrings, injury often occurs during sprinting. However, the frequent kicking demand during gameplay also plays a role in quadriceps injury, as evidenced by an increased risk of quadriceps strain in the dominant kicking leg [14]. During sprinting, the maximum length of the rectus femoris occurs during early swing phase, at which point the hip is in maximum extension with the knee flexed. Eccentric contraction of the quadriceps during early swing phase make it vulnerable to injury. During deceleration, the body must rapidly absorb significant kinetic energy to quickly stop or change direction. This applies substantial eccentric forces to the quadriceps musculature, potentially inducing injury. Finally, during the windup phase of kicking the quadriceps are firing eccentrically as the hip is brought into maximum extension and the knee is flexed. This places strain across the quadriceps, particularly the rectus femoris, and predisposes to muscle injury. Similar to hamstring strains, risk factors for quadriceps strain include prior quadriceps strain, increased age, or poor flexibility.

Adductor strains, or groin strains, can involve any of the adductor muscles of the medial thigh. Unlike the quadriceps or hamstrings, the adductors are more commonly injured with quick lateral movements or change in direction. They undergo eccentric contraction when the hip abducts while the body shifts momentum from side to side. While injuries can occur acutely due to forceful contraction, adductor strains are more likely to be the result of chronic overuse when compared to other thigh strains [14, 16]. Frequent changes in direction and side-to-side motion place repetitive stress on the adductor musculature, eventually leading to pain and dysfunction. Like with other strains, prior injury and increased age are known key risk factors. In addition, decreased adductor:abductor strength ratio as well as decreased hip abduction range of motion have been shown to predispose to injury [16–18].

Contusions constitute a sizable portion of soccer injuries. These can occur in essentially any body part, but most commonly the thigh musculature is at risk. While thigh strains are predominantly noncontact in nature, thigh contusions by definition occur due to a contact mechanism. This is almost always in the form of direct contact with another player; however, in rare cases it can be secondary to contact with another object such as a ball, goalpost, or off-field equipment. Most contusions occur due to collisions with an opposing player while competing for the ball, and up to 42% are directly related to foul play. Overall, contusions account for roughly 12% of thigh injuries. Compared to muscle strains, contusions generally require less time for recovery and are substantially less likely to be recurrent [19]. For severe quadriceps contusions, the clinician should be alert for the potential complication of myositis ossificans. Heat and ultrasound should not be applied to an acute quadriceps contusion as these modalities may increase the risk of developing myositis.

9.3.2 Groin Pain: Groin Strains, Athletic Pubalgia, Sports Hernia, Osteitis Pubis, Femoroacetabular Impingement

Compared to most sports, soccer/football players have a much higher incidence of groin injuries/pain. Indeed, while a certain percentage of groin pain in soccer athletes can be related to adductor strains as outlined above in Sect. 9.3.1; groin pain as a symptom accounts for a complex myriad of diagnoses which must be considered and ruled out to optimally treat the athlete. These diagnoses include stress fractures of the pubic bone, osteitis pubis or degeneration of the symphysis pubis, adductor muscle strains, obturator nerve entrapment, insufficiency of the internal rectus fascia (sports hernia), inguinal hernia, and femoral acetabular impingement. Because of this complexity, the specific diagnosis followed by diagnosis-targeted treatment plan is often delayed. Careful physical examination with a targeted imaging work-up by a qualified, knowledgeable clinician is essential. Also critical to successful treatment in soccer players is a foundational understanding of the injury mechanism that likely led to the pathology. To be successful soccer players depends on being agile with excellent flexibility and mobility in their core musculature. They often support most of their weight on one lower limb while abducting, rotating, controlling their torso while reaching or kicking a ball. This demand plays high repetitive loads on the bones and ligaments that serve as stabilizing anchors to core stability as well as repetitive loads both in contraction and stretching of the muscle-tendon units. Indeed, with any weakness or dysfunction in the kinetic chain each link may see increased loads and risk failure. Optimal treatment and the best opportunity to return to play is based on making an accurate diagnosis and then targeting the treatment appropriately including a sports-specific return to play plan.

9.3.3 Ankle Sprains

An ankle sprain occurs when one or more of the three ligamentous complexes around the ankle is partially or completely torn, these being the lateral ligament complex, deltoid ligament, and syndesmosis. The vast majority of ankle sprains involve the anterior talofibular (ATFL) and/or calcaneofibular (CFL) ligaments of the lateral ligament complex. These occur when the ankle is subject to an inversion and/or internal rotation stress. The remainder of ankle sprains involve the deltoid ligament or syndesmosis, which occur secondary to eversion and external rotation stresses, respectively.

Like in many other sports, soccer players often sustain injuries during cutting and jumping activities. These injuries occur due to rotational stress applied to the ankle while loading or unloading the ankle joint [20]. In many sports, this is predominantly following an incidental misstep or landing on another player. While such jumping and cutting injuries still occur in soccer, it is also worth noting that players are also subject to more direct contact injuries due to the fact that soccer is played with the feet. Multiple studies have shown that the majority of ankle sprains in soccer are due to contact with another player. The most common mechanism is during a tackle, and it has been reported that anywhere from 40% to 60% of these are due to foul play [21, 22]. For these contact injuries, the injured player often has a planted foot which is then struck or stepped on by an opponent, leading to a rotational stress [23]. Injury is more likely to occur with a tackle coming from the side, as opposed to directly in front or behind [21]. Another injury mechanism unique to soccer occurs during kicking. When striking the ball, the leg and foot are moving at high velocity to impart force to the ball. If the foot is hit by another player either before or after striking the ball, this can impart a rapid rotational injury to the ankle, resulting in ligamentous sprain [23]. Of note, ankle injuries secondary to direct player contact are primarily seen in outfield players. Goalkeepers, on the other hand, have a lower overall ankle sprain incidence, and these occur predominantly due to noncontact mechanisms such as cutting, landing, and diving [24, 25].

9.3.4 Knee Injuries/ACLs/Ligament Sprains/Meniscus

Knee sprains are characterized by partial or complete tears to one of the soft tissue structures about the knee. Injury to the medial or lateral collateral ligaments (MCL, LCL), anterior or posterior cruciate ligaments (ACL, PCL), and the medial or lateral meniscus have all been described in the literature. Sprain can involve injury to any one of these in isolation or as a combination of multiple structures. The MCL is the most commonly injured ligament [8]. However, significantly more attention has been given to ACL injuries in the literature. This is likely due to the overall higher burden that ACL injuries place on athletes secondary to an increased need for surgery and greater time off of sport compared to MCL injuries, which often heal without surgical intervention.

The ACL is the primary restraint against anterior translation of the tibia in respect to the femur. Due to the poor healing capacity of the ACL, rupture often leads to knee instability requiring surgical reconstruction of the ligament. Any rotational or translational moment which applies an anterior force on the tibia relative to the femur could potentially damage the ACL. Extensive study has been completed to examine which particular movements are the highest risk. For most ACL injuries, the incident is noncontact in nature, and occurs with all or most of the body weight on the injured leg. The foot is planted, knee in extension and valgus, and the tibia externally rotated [26]. For soccer in particular, reported mechanisms include competing for possession of the ball, regaining balance after a kick, landing from a header, or direct contact from a tackle [27, 28]. When competing for the ball, the most common movement was a rapid side step when going after the ball or making a tackle. This places the hip in abduction and the knee in valgus, predisposing to an ACL injury. When kicking the ball from a position of poor balance, a player may land on an extended leg with valgus stress and be subject to ACL injury. The most common injury following a jump occurred when landing after a header. These primarily occur when landing on one leg rather than two, and may be exacerbated by player-to-player contact which forced the awkward landing. Finally, the most common direct contact ACL injury occurred when being tackled from behind, leading to valgus collapse of the knee [27].

MCL injuries are the most frequently encountered ligamentous injuries to the knee. Fortunately as noted, these often resolve without surgical intervention, meaning return to play is generally quicker than for ACL injuries. The MCL is at highest risk of injury when the knee is subjected to a valgus stress. In soccer, this is due to a direct contact injury with an opposing player in the majority of cases. The primary mechanism is being tackled from the side, causing valgus stress [29].

LCL injuries occur significantly less frequently when compared to MCL injuries, accounting for roughly 0.7% of soccer injuries. These occur due to varus stress to the knee. The most common mechanism is contact injury in which the leg is struck from the medial side, leading to varus stress. LCL sprain can also occur in a noncontact fashion while cutting or twisting in a manner that causes varus stress [30]. Mild injuries or partial strains usually do well and heal like MCL injuries; however, more severe injuries may lead to chronic posterolateral rotatory insufficiency (PLRI). When complete injuries to the lateral collateral ligament and posterior lateral corner occur, the clinician should carefully document function of the peroneal nerve and assess for the possibility of associated ligament injuries of either the ACL or PCL. Complete injuries of the lateral collateral ligament and posterolateral corner will likely require surgery if the athlete wishes to attempt to return to play.

PCL sprains are the least common, or perhaps least recognized, ligamentous knee injuries in soccer players, accounting for only 0.2% of reported injuries. Full thickness PCL ruptures are generally only seen in multi-ligamentous injuries. The PCL is subject to injury with any posterior translation of the tibia in relation to the femur. This is more likely to be due to a contact injury such as being kicked by another player or shin-to-shin contact during a slide tackle. As far as noncontact injuries, hyperextension of the knee is the primary mechanism [30].

Injuries to the meniscus most commonly occur with twisting and cutting, hyperflexion, or as an associated injury with a major ligament injury such as the ACL or MCL. Partial injuries or peripheral attachment sprains can occur in soccer players and may be treated with careful observation and gradual return to play. Complete and displaced tears should be treated surgically making every effort to save the meniscus if possible [31]. The success of meniscus repair is, in general, better when the repair is performed concurrently with an ACL reconstruction.

9.3.5 Concussion/Mild Traumatic Brain Injury (MTBI)

Interest in the impact and importance of concussion and MTBI within sport has advanced significantly in recent years secondary to the increased awareness and developing understanding regarding the long-term sequelae of repetitive head trauma. While soccer was initially thought to be at a lower risk of head injury when compared to collision sports like football or hockey, epidemiological studies have demonstrated the incidence of concussion in soccer is still quite high, accounting for anywhere from 2% to 24% of all injuries [2, 8]. The wide variability in incidence could be explained by a number of factors including: failure to report in all studies, decreased incidence with increased age, and overall differences in reporting methods. A variety of studies have demonstrated that the diagnosis of concussions is likely underreported. Indeed, there is evidence that up to 80% of athletes who had experienced concussion symptoms did not report the concussion diagnosis [32]. While historical concern for head injury pointed at the mechanism of repeatedly heading the ball, studies have shown the most common mechanism of injury is contact with another player, which accounts for anywhere from 38% to 85% of concussions in soccer [32]. The primary mechanism is head-to-head contact when competing for a header, but other player contact injuries include contact with an arm/elbow while heading, or contact with an elbow or knee during general gameplay [33]. Concussion can also occur in the absence of direct player contact. Over 95% of those that did not involve contact with another player are related to contact with the ball [34]. Head injuries from hitting the ground or goalpost have also been reported. Regarding the ball-to-head mechanism, concussion almost never occurs from intentionally heading the ball. Instead, concussion occurs when being incidentally hit in the head by a ball at close range [32]. Other factors play a role regarding both the incidence and severity of concussion in soccer/football players. It has been shown that females sustain concussion at higher rates than males and their symptoms may linger. Goalkeepers and defenders appear to be at greater risk as they are more likely to find themselves in the line of fire of a shot on goal [32, 33].

9.4 Epidemiology of Soccer/ Football Injuries

9.4.1 High School Soccer

Soccer is in the top five most popular high school sports, with over 850,000 annual participants [35]. Reported injury rates range from 1.83 to 2.39 injuries per 1000 athletic events, giving an estimated 375,000–422,000 injuries annually [2, 36–38]. The majority of all injuries are to the lower extremity or the head, with the most commonly injured sites being the ankle (17.3–24.7%), knee (13.8–21.8%), hip/thigh (13.1–25.6%), or head/face (13.7–27.7%). The most common injury types are ligament sprain (22.5–34.5%), muscle strain (16.1–31.9%), concussion (10.8–24.5%), and contusion (10.1–18.7%) (Fig. 9.2). Roughly 37.7–57.9% of all injuries required less than 1 week of missed time. However, up to 8.7% were season ending, and 5.6–6.1% required surgical intervention. The most common injuries which were season ending and/or required surgery were ACL tears and fractures. The injury rate has repeatedly been shown to be higher in competition than in practice. During practice, there are higher rates of muscle strains and non-contact injuries. During competition, there are higher rates of concussion, fracture, and injuries caused by contact with another player. Girls have been found to have higher rates of overall injury as well as knee sprains (especially ACL injuries) when compared to boys (Fig. 9.3). Boys, however, are more likely to have fractures or injuries to the hip/thigh region than girls.

Fig. 9.2 Percentage of total injuries by location in male high school and collegiate soccer [36–38]. (Source: Pexels on Pixabay)

9.4.2 Collegiate Soccer

Among National Collegiate Athletic Association (NCAA) sports, soccer has the second most female participants and fifth most male participants, with a total of over 53,000 players in the 2018–2019 season [39]. Reported overall injury rates range from 6.6 to 6.94 injuries per 1000 athletic exposures [2, 38]. 57.6–62.4% of injuries required less than 1 week of missed participation. Similar to high school athletes, the majority of injuries were in the lower extremity. The most commonly injured sites were the hip/thigh (15.1–31.3%), ankle (16.6–21.2%), knee (12–18%), and head/face (7.9–19.2%). Injury risk is up to four times higher in competition than in practice [40]. During games, players are up to 4 times as likely to sustain an ankle sprain, 6 times as likely to sustain a knee injury, and 13 times as likely to sustain a concussion when compared to practice. This is likely secondary to higher intensity of play and higher degree of player contact, as evidenced by a significantly higher proportion of injuries being directly related to contact with another player in games (53.6–61%) compared to practice (19–28.2%). In practice, players are more likely to suffer noncontact injuries, with muscle strains being far more common.

Fig. 9.3 Percentage of total injuries by location in female high school and collegiate soccer [2, 36, 37]. (Source: Unsplash)

9.4.3 Professional Soccer

Multiple studies have assessed the incidence and impact of injuries on professional soccer players. A recent review estimated that the overall injury incidence was 8.1 injuries/1000 h of exposure. The competition injury rate (36 per 1000 h) was nearly ten times the practice rate (3.7 per 1000 h) [41]. Given the length of professional league seasons, this equates to roughly two injuries per player, per year [8]. Similar to other competition levels, the majority of injuries were to the lower extremity, with the five most common sites being thigh, knee, ankle, hip, and lower leg/Achilles. Interestingly, the incidence of head/face injuries (e.g., concussion) was the lowest rate, behind torso and upper extremity injuries. As far as nature of injury, muscle/tendon strains are by far the most common, followed by contusion, joint/ligament injury, and fracture [41]. The most common overall injury is hamstring strain, accounting for up to 12% of all injuries [8] Fortunately, roughly half of all injuries are fairly minor, requiring less than 1 week away from sport. However, 15% of injuries are severe, meaning a month or more of time lost. Roughly 12% of injuries were re-injuries, with these requiring more time off than new injuries.

9.4.4 Children

There are few studies evaluating the epidemiology of injuries in pre-adolescent children. In general, these studies have similar findings as those in adolescents, such as predominantly lower extremity injuries and higher injury rates in games. However, there are some notable differences. Overall, the injury rate for children is lower than that for older players, ranging from

Fig. 9.4 Percentage of total injuries by location in pediatric (age 7–12) players [43]. (Source: Pixabay)

0.1 to 1.6 injuries per 1000 h [42]. Injury incidence increases with age in children 7–12 [43]. Children are also more likely to sustain upper extremity injuries (15–29%) and fractures (15%) than older players [42, 43] (Fig. 9.4). This is mostly attributed to increased rate of forearm, wrist, and hand fractures sustained from falls while playing. Possibly related to the increased fracture rate, the rate of severe injuries which require greater than 28 days off sport are relatively high in children (23%). Naturally, with all skeletally immature athletes, careful attention should be focused on the growth plates as a potential site of injury.

9.5 Prevention Plans to Reduce Soccer/Football Injuries

A number of different injury prevention programs exist for soccer players. In accordance with the epidemiology of soccer injuries, many of these programs focus on lower extremity injury prevention. The highest ruling body of international soccer, FIFA, has taken great interest in injury prevention by creating programs and implementing a great deal of resources into these programs. This is best demonstrated through the "FIFA 11+" Program [44]. The program involves three stages made up of 15 exercises that are completed over 20 min. It begins with 8 min of running exercises that emphasize change of direction, acceleration/deceleration, and proper form. The next stage involves various strength, balance, and plyometric exercises. These are done with the goal of improving core strength, proprioception, and neuromuscular control. Finally, the program ends with high intensity running exercises to focus on high-speed running and change of direction at this speed. There are three levels to the program increasing in difficulty based on the level of athlete.

This is a highly effective program that has been proven in multiple studies. It has been shown to be best effective when performed at least twice a week and injury risk lowers as program adherence increases [45] The program has been shown to reduce injuries overall and more specifically noncontact injuries such as ACL tears [46]. Other common injuries found to be reduced by FIFA 11 include ankle sprains and hamstring injuries [47]. Efficacy was first proven in the female population and was demonstrated in male athletes shortly thereafter [44]. It has also been shown to have wide applications with its efficacy proven in athletes of various ages from youth to adults. Furthermore, it has shown reduced injury rates in athletes of various levels of competition from youth, collegiate, and semi-professional levels [48–50].

In addition to injury reduction, the FIFA 11+ program has been shown to increase athlete performance. One study found the program increased sprint speed and vertical jump in amateur male soccer players [51]. Studies have demonstrated the program improving athlete body control and proprioception [45, 52]. These neuromuscular improvements likely help explain the injury reduction as well. Overall, the FIFA 11+ is an incredible program with wide applications that help improve athlete safety across the sport of soccer as a whole.

Another injury prevent program created by FIFA is the "FIFA 11+S" program which focuses on reducing shoulder injuries [6]. As discussed, earlier goalies are at particular risk for shoulder injuries given the different requirements of the position such as diving to prevent goals and throwing the ball to teammates. Though more recent in its creation, the program has similar organization and aims to the lower extremity-focused program. It is organized into three parts: generalized warming up, upper extremity strengthening, and core stability. Goals are to improve rotator cuff strength, scapular stabilization, and neuromuscular control for falling. Again, the total time for program completion is around 20 min with the goal of completion three times per week. Research is still lacking on the effectiveness of this program with reducing injuries. However, it is exciting opportunity to further help prevent injuries for soccer players of all positions.

Screening is another form of injury prevention in soccer. Functional movement screening (FMS) is the most commonly used in this population. This system consists of seven tests that focus on balance, mobility, stability, and neuromuscular control [53]. Example of tests include deep squat, hurdle step, and inline lunges. The tests expose weaknesses and deficiencies in athletes that can predispose them to future injury. Once identified programs can then be created to address deficiencies with the ultimate goal of reducing injury risk. Evidence is conflicting on the ability of FMS to predict injury risk [54, 55]. Despite this, the tool still holds utility in identifying deficits and optimizing neuromuscular control.

Tracking athletes' overall sports-related activity and usage offers an exciting possibility for injury prevention. Ehrmann et al. used GPS to measure high intensity distance and total distance ran by professional soccer players in training as well as game play [56]. They took averages for 1 and 4 week blocks as well as the season overall. Ultimately, they found increased distance and intensity in the weeks leading up to noncontact injuries in this population. GPS tracking could provide a great future tool in monitoring athlete usage. With this information training regimens could be further optimized to reduce injury risk and possibly improve performance.

Protective equipment offers another area of injury improvement in soccer. Shin guards are standard protective equipment required in soccer. These pads protect the lower from injuries that can occur from direct contact between the lower legs of athletes when competing. One option is ankle protection through braces or taping. These aim to provide rigid support to the ankle to protect against excessive inversion or eversion. Both methods have been shown to reduce ankle sprains. Brace options are also available for the knee which can be used to help prevent and aid multiple pathologies. Patella stabilization braces provide medial forces to help with patellar stabilization and improve patellar tracking. Other knee braces can provide rigid support to resist varus and valgus forces to the knee. Hand and finger injuries are a particular risk in goalies. Gloves can be used in this population to help avoid injuries. Gloves provide padding and rigid support to protect goalie's hands from the impact of the ball. They also provide additional grip to aid in catching.

Finally, rule changes play an important role in injury prevention. As high-risk plays are better elucidated, the governing bodies of various competitive levels have the opportunity to regulate gameplay to reduce injuries. This can be in the form of changing the enforcement of a rule, or by changing the rules themselves. One example of this occurred in 2006, when FIFA enacted a change regarding elbow to head contact. Any player who intentionally struck an opponent with their arm/elbow during gameplay would be disqualified for the remainder of the game. This led to a subsequent decrease in head injuries and concussions [57, 58] (Fig. 9.5).

9.6 Paralympic Soccer

Paralympic soccer is played in a 5-a-side format for athletes with visual impairments and in a 7-a-side format for athletes with physical impairments typically associated with neurologic disorders such as cerebral palsy, stroke, or traumatic brain injury. Both formats utilize a reduced

Fig. 9.5 FIFA 11+ Prevention Program. (Source: Bizzini M, Jiunge A, Dvorak J, Federation Internationale de Football Association (FIFA). The 11+ manual: a complete warm-up program to prevent injuries. Federation Internationale de Football Association (FIFA), FIFA-Medical Assessment and Research Centre (F-MARC))

field size and game time in comparison to standard able-bodied play [59, 60].

Injuries within the context of paralympic soccer have not been extensively studied and data concerning types of injuries typically seen in paralympic soccer players is rather limited. Included in this limited research is an investigation of the epidemiology of injuries that occurred at the London 2012 Paralympic Games for both 5-a-side and 7-a-side formats. Notably, researchers determined an injury incidence rate of 22.4 per 1000 athlete days in 5-a-side soccer, which was the highest injury rate among all paralympic sports [61]. Injury incidence rate for 7-a-side soccer was 10.4 per 1000 athlete days (WEBBORN). It was also noted that acute traumatic injuries were far more prevalent than chronic overuse injuries in players participating in the 2012 Paralympic Games. For 5-a-side soccer, the lower extremity was involved in a majority of all injuries with the knee being the most common site of injury. 7-a-side soccer injuries similarly involved mostly the lower extremity with the knee and ankle being the most commonly injured regions of the body [62].

It is perhaps most important to consider the fact that the nature in which paralympic soccer is played is different than that seen in traditional soccer, and thus produces a different pattern of injury. This discrepancy is particularly prevalent in 5-a-side soccer where the players are visually impaired and play in a more upright posture in comparison to non-visually impaired soccer athletes, resulting in a greater exposure to head collisions. In addition, visual impairment leads to athletes' reduced ability to protect themselves in anticipation of oncoming blows. Within the context of the 2012 Paralympic Games, 60% of competition-related injuries in 5-a-side soccer were associated with foul play, often associated with a player's failure to properly communicate their intent to move towards the ball as required by the rules of the game [62, 63]. For 7-a-side soccer, a majority of para-athletes have central neurologic injury such as cerebral palsy and as a result are at greater risk of muscle strain or injury secondary to spasticity and weakness [64]. Particularly in athletes with cerebral palsy, there is a greater risk for soft tissue injuries and lacerations in comparison to athletes with other types of physical impairments [65].

In order to address the relative lack of literature on paralympic athletes, a study protocol to allow for prospective longitudinal studies was adapted and published in 2016 [66]. The study in question, called the Sports-Related Injuries and Illnesses in Paralympic Sport Study (SRIIPSS), was the first of its kind and was intended to support the development of evidence-based preventive measures specifically tailored to paralympic athletes to encourage safe and healthy sport participation.

9.7 Summary

Soccer/Football is the most popular sport in the world, and the number of players continues to grow. It can easily be adapted, making it inclusive for players of multiple ages, skill levels, and levels of physical function. Gameplay is dynamic, with multiple sport-specific activities that place players at risk of injury. The majority of these injuries are sprains and strains to the lower extremities, but other severe injuries such as concussion, contusion, and fracture often occur as well. Despite its storied history, the game is still evolving as we continue to develop new ways to manage, rehabilitate, and ultimately prevent injuries.

References

1. Kunz M. Big count: 265 million playing football. FIFA Mag. 2007:10–5.
2. DiStefano LJ, Dann CL, Chang CJ, Putukian M, Pierpoint LA, Currie DW, et al. The first decade of web-based sports injury surveillance: descriptive epidemiology of injuries in US high school girls' soccer (2005–2006 through 2013–2014) and National Collegiate Athletic Association women's soccer (2004–2005 through 2013–2014). J Athl Train. 2018;53(9):880–92. https://doi.org/10.4085/1062-6050-156-17.
3. Kapidžić A, Huremović T, Biberovic A. Kinematic analysis of the instep kick in youth soccer players. J Hum Kinet. 2014;42:81–90.

4. Stewart WF, Kim N, Ifrah C, Sliwinski M, Zimmerman ME, Kim M, et al. Heading frequency is more strongly related to cognitive performance than unintentional head impacts in amateur soccer players. Front Neurol. 2018;9:240.
5. Nitrini R. Soccer (Football Association) and chronic traumatic encephalopathy: a short review and recommendation. Dement Neuropsychol. 2017;11(3):218–20. https://doi.org/10.1590/1980-57642016dn11-030002.
6. Ejnisman B, Barbosa G, Andreoli CV, de Castro Pochini A, Lobo T, Zogaib R, et al. Shoulder injuries in soccer goalkeepers: review and development of a FIFA 11+ shoulder injury prevention program. Open Access J Sports Med. 2016;7:75–80.
7. From the Centers for Disease Control and Prevention. Injuries associated with soccer goalposts—United States, 1979–1993. JAMA. 1994;271(16):1233–4.
8. Ekstrand J, Hagglund M, Walden M. Injury incidence and injury patterns in professional football: the UEFA injury study. Br J Sports Med. 2011;45:553–8.
9. Ekstrand J, Healy JC, Waldén M, Lee JC, English B, Hägglund M. Hamstring muscle injuries in professional football: the correlation of MRI findings with return to play. Br J Sports Med. 2012;46(2):112–7. https://doi.org/10.1136/bjsports-2011-090155. Epub 2011 Dec 5.
10. Woods C, Hawkins RD, Maltby S, Hulse M, Thomas A, Hodson A, Football Association Medical Research Programme. The Football Association Medical Research Programme: an audit of injuries in professional football—analysis of hamstring injuries. Br J Sports Med. 2004;38(1):36–41. https://doi.org/10.1136/bjsm.2002.002352.
11. Opar DA, Williams MD, Shield AJ. Hamstring strain injuries: factors that lead to injury and re-injury. Sports Med. 2012;42(3):209–26. https://doi.org/10.2165/11594800-000000000-00000.
12. Arnason A, Sigurdsson SB, Gudmundsson A, Holme I, Engebretsen L, Bahr R. Risk factors for injuries in football. Am J Sports Med. 2004;32(1 Suppl):5S–16S. https://doi.org/10.1177/0363546503258912.
13. van Beijsterveldt AM, van de Port IG, Vereijken AJ, Backx FJ. Risk factors for hamstring injuries in male soccer players: a systematic review of prospective studies. Scand J Med Sci Sports. 2013;23(3):253–62. https://doi.org/10.1111/j.1600-0838.2012.01487.x. Epub 2012 Jun 21.
14. Ekstrand J, Hägglund M, Waldén M. Epidemiology of muscle injuries in professional football (soccer). Am J Sports Med. 2011;39(6):1226–32. https://doi.org/10.1177/0363546510395879. Epub 2011 Feb 18.
15. Eckard TG, Kerr ZY, Padua DA, Djoko A, Dompier TP. Epidemiology of quadriceps strains in National Collegiate Athletic Association athletes, 2009–2010 through 2014–2015. J Athl Train. 2017;52(5):474–81. https://doi.org/10.4085/1062-6050-52.2.17. Epub 2017 Apr 6.
16. Hölmich P, Thorborg K, Dehlendorff C, Krogsgaard K, Gluud C. Incidence and clinical presentation of groin injuries in sub-elite male soccer. Br J Sports Med. 2014;48(16):1245–50. https://doi.org/10.1136/bjsports-2013-092627. Epub 2013 Aug 16.
17. Engebretsen AH, Myklebust G, Holme I, Engebretsen L, Bahr R. Intrinsic risk factors for groin injuries among male soccer players: a prospective cohort study. Am J Sports Med. 2010;38(10):2051–7. https://doi.org/10.1177/0363546510375544. Epub 2010 Aug 10.
18. Ibrahim A, Murrell GA, Knapman P. Adductor strain and hip range of movement in male professional soccer players. J Orthop Surg (Hong Kong). 2007;15(1):46–9. https://doi.org/10.1177/230949900701500111.
19. Ueblacker P, Müller-Wohlfahrt HW, Ekstrand J. Epidemiological and clinical outcome comparison of indirect ('strain') versus direct ('contusion') anterior and posterior thigh muscle injuries in male elite football players: UEFA Elite League study of 2287 thigh injuries (2001–2013). Br J Sports Med. 2015;49(22):1461–5. https://doi.org/10.1136/bjsports-2014-094285. Epub 2015 Mar 9.
20. Watanabe K, Kitaoka HB, Berglund LJ, Zhao KD, Kaufman KR, An KN. The role of ankle ligaments and articular geometry in stabilizing the ankle. Clin Biomech (Bristol, Avon). 2012;27(2):189–95. https://doi.org/10.1016/j.clinbiomech.2011.08.015. Epub 2011 Oct 13.
21. Giza E, Fuller C, Junge A, Dvorak J. Mechanisms of foot and ankle injuries in soccer. Am J Sports Med. 2003;31(4):550–4. https://doi.org/10.1177/03635465030310041201.
22. Waldén M, Hägglund M, Ekstrand J. Time-trends and circumstances surrounding ankle injuries in men's professional football: an 11-year follow-up of the UEFA Champions League injury study. Br J Sports Med. 2013;47(12):748–53. https://doi.org/10.1136/bjsports-2013-092223. Epub 2013 Jun 27.
23. Andersen TE, Floerenes TW, Arnason A, Bahr R. Video analysis of the mechanisms for ankle injuries in football. Am J Sports Med. 2004;32(1 Suppl):69S–79S. https://doi.org/10.1177/0363546503262023.
24. Woods C, Hawkins R, Hulse M, Hodson A. The Football Association Medical Research Programme: an audit of injuries in professional football: an analysis of ankle sprains. Br J Sports Med. 2003;37(3):233–8. https://doi.org/10.1136/bjsm.37.3.233.
25. Kofotolis ND, Kellis E, Vlachopoulos SP. Ankle sprain injuries and risk factors in amateur soccer players during a 2-year period. Am J Sports Med. 2007;35(3):458–66. https://doi.org/10.1177/0363546506294857. Epub 2007 Jan 11.
26. Alentorn-Geli E, Myer GD, Silvers HJ, Samitier G, Romero D, Lázaro-Haro C, et al. Prevention of non-contact anterior cruciate ligament injuries in soccer players. Part 1: Mechanisms of injury and underlying risk factors. Knee Surg Sports Traumatol Arthrosc. 2009;17(7):705–29. https://doi.org/10.1007/s00167-009-0813-1. Epub 2009 May 19.
27. 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. https://doi.org/10.1136/bjsports-2014-094573. Epub 2015 Apr 23.
28. Grassi A, Smiley SP, Roberti di Sarsina T, Signorelli C, Marcheggiani Muccioli GM, Bondi A, 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. https://doi.org/10.1007/s00590-017-1905-0. Epub 2017 Jan 25.
29. Lundblad M, Hägglund M, Thomeé C, Hamrin Senorski E, Ekstrand J, Karlsson J, et al. 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;27(11):3692–8. https://doi.org/10.1007/s00167-019-05491-6. Epub 2019 Apr 4.
30. Lundblad M, Hägglund M, Thomeé C, Hamrin Senorski E, Ekstrand J, Karlsson J, et al. Epidemiological data on LCL and PCL injuries over 17 seasons in men's professional soccer: The UEFA Elite Club Injury Study. Open Access J Sports Med. 2020;11:105–12. https://doi.org/10.2147/OAJSM.S237997.
31. Mitchell J, Graham W, Best TM, Collins C, Currie DW, Comstock RD, et al. Epidemiology of meniscal injuries in US high school athletes between 2007 and 2013. Knee Surg Sports Traumatol Arthrosc. 2016;24(3):715–22. https://doi.org/10.1007/s00167-015-3814-2. Epub 2015 Oct 27.
32. Maher ME, Hutchison M, Cusimano M, Comper P, Schweizer TA. Concussions and heading in soccer: a review of the evidence of incidence, mechanisms, biomarkers and neurocognitive outcomes. Brain Inj. 2014;28(3):271–85. https://doi.org/10.3109/02699052.2013.865269. Epub 2014 Jan 29.
33. Levy ML, Kasasbeh AS, Baird LC, Amene C, Skeen J, Marshall L. Concussions in soccer: a current understanding. World Neurosurg. 2012;78(5):535–44. https://doi.org/10.1016/j.wneu.2011.10.032. Epub 2011 Nov 1.
34. Comstock RD, Currie DW, Pierpoint LA, Grubenhoff JA, Fields SK. An evidence-based discussion of heading the ball and concussions in high school soccer. JAMA Pediatr. 2015;169(9):830–7. https://doi.org/10.1001/jamapediatrics.2015.1062.
35. National Federation of State High School Associations. 2018–19 High school athletics participation survey. Indianapolis, IN: National Federation of State High School Associations. https://www.nfhs.org/media/1020406/2018-19-participation-survey.pdf. 23p.
36. Yard EE, Schroeder MJ, Fields SK, Collins CL, Comstock RD. The epidemiology of United States high school soccer injuries, 2005–2007. Am J Sports Med. 2008;36(10):1930–7. https://doi.org/10.1177/0363546508318047. Epub 2008 Jul 15.
37. Khodaee M, Currie DW, Asif IM, Comstock RD. Nine-year study of US high school soccer injuries: data from a national sports injury surveillance programme. Br J Sports Med. 2017;51(3):185–93. https://doi.org/10.1136/bjsports-2015-095946. Epub 2016 Dec 28.
38. Kerr ZY, Putukian M, Chang CJ, DiStefano LJ, Currie DW, Pierpoint LA, et al. The first decade of web-based sports injury surveillance: descriptive epidemiology of injuries in US high school boys' soccer (2005–2006 through 2013–2014) and National Collegiate Athletic Association men's soccer (2004–2005 through 2013–2014). J Athl Train. 2018;53(9):893–905. https://doi.org/10.4085/1062-6050-166-17.
39. Irick E. Student-athlete participation 1981/2–2015/16: NCAA sports sponsorship and participation rates report. Indianapolis, IN: National Collegiate Athletic Association; 2016. https://www.ncaapublications.com/p-4445-2015-16-ncaa-sports-sponsorship-and-participation-rates-report.aspx. 301p.
40. Agel J, Evans TA, Dick R, Putukian M, Marshall SW. Descriptive epidemiology of collegiate men's soccer injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2002–2003. J Athl Train. 2007;42(2):270–7.
41. López-Valenciano A, Ruiz-Pérez I, Garcia-Gómez A, Vera-Garcia FJ, De Ste Croix M, Myer GD, et al. Epidemiology of injuries in professional football: a systematic review and meta-analysis. Br J Sports Med. 2020;54(12):711–8. https://doi.org/10.1136/bjsports-2018-099577. Epub 2019 Jun 6.
42. Faude O, Rößler R, Junge A. Football injuries in children and adolescent players: are there clues for prevention? Sports Med. 2013;43(9):819–37. https://doi.org/10.1007/s40279-013-0061-x.
43. Rössler R, Junge A, Chomiak J, Dvorak J, Faude O. Soccer injuries in players aged 7 to 12 years: a descriptive epidemiological study over 2 seasons. Am J Sports Med. 2016;44(2):309–17. https://doi.org/10.1177/0363546515614816. Epub 2015 Dec 8.
44. Bizzini M, Dvorak J. FIFA 11+: an effective programme to prevent football injuries in various player groups worldwide-a narrative review. Br J Sports Med. 2015;49(9):577–9.
45. Steffen K, Emery CA, Romiti M, Kang J, Bizzini M, Dvorak J, et al. High adherence to a neuromuscular injury prevention programme (FIFA 11+) improves functional balance and reduces injury risk in Canadian youth female football players: a cluster randomised trial. Br J Sports Med. 2013;47(12):794–802. https://doi.org/10.1136/bjsports-2012-091886. Epub 2013 Apr 4.
46. 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.
47. Nouni-Garcia R, Carratala-Munuera C, Orozco-Beltran D, Lopez-Pineda A, Asensio-Garcia MR, Gil-Guillen VF. Clinical benefit of the FIFA 11 pro-

gramme for the prevention of hamstring and lateral ankle ligament injuries among amateur soccer players. Inj Prev. 2018;24(2):149–54.
48. Silvers-Granelli H, Mandelbaum B, Adeniji O, Insler S, Bizzini M, Pohlig R, 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.
49. Whalan M, Lovell R, Steele JR, Sampson JA. Rescheduling Part 2 of the 11+ reduces injury burden and increases compliance in semi-professional football. Scand J Med Sci Sports. 2019;29(12):1941–51.
50. Rössler R, Donath L, Bizzini M, Faude O. A new injury prevention programme for children's football—FIFA 11+ Kids—can improve motor performance: a cluster-randomised controlled trial. J Sports Sci. 2016;34(6):549–56.
51. Nawed A, Khan IA, Jalwan J, Nuhmani S, Muaidi QI. Efficacy of FIFA 11+ training program on functional performance in amateur male soccer players. J Back Musculoskelet Rehabil. 2018;31(5):867–70.
52. Ayala F, Pomares-Noguera C, Robles-Palazón FJ, Del Pilar García-Vaquero M, Ruiz-Pérez I, Hernández-Sánchez S, et al. Training effects of the FIFA 11+ and Harmoknee on several neuromuscular parameters of physical performance measures. Int J Sports Med. 2017;38(4):278–89.
53. Cook G, Burton L, Hoogenboom BJ, Voight M. Functional movement screening: the use of fundamental movements as an assessment of function - part 1. Int J Sports Phys Ther. 2014;9(3):396–409.
54. Smith PD, Hanlon MP. Assessing the effectiveness of the functional movement screen in predicting non-contact injury rates in soccer players. J Strength Cond Res. 2017;31(12):3327–32.
55. 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(23):1661–9.
56. Ehrmann FE, Duncan CS, Sindhusake D, Franzsen WN, Greene DA. GPS and injury prevention in professional soccer. J Strength Cond Res. 2016;30(2):360–7.
57. Beaudouin F, Aus der Fünten K, Tröß T, Reinsberger C, Meyer T. Head injuries in professional male football (soccer) over 13 years: 29% lower incidence rates after a rule change (red card). Br J Sports Med. 2019;53(15):948–52. https://doi.org/10.1136/bjsports-2016-097217. Epub 2017 Jun 23.
58. Bjrneboe J, Bahr R, Dvorak J, Andersen TE. Lower incidence of arm-to-head contact incidents with stricter interpretation of the Laws of the Game in Norwegian male professional football. Br J Sports Med. 2013;47:508–14.
59. International Paralympic Committee. Football 5-a-side. https://www.paralympic.org/football-5-a-side/about. Accessed 22 Sept 2020.
60. International Paralympic Committee. Football 7-a-side at the Parapan American Games. https://www.paralympic.org/lima-2019/football-7. Accessed 22 Sept 2020.
61. Willick SE, Webborn N, Emery C, Blauwet CA, Pit-Grosheide P, Stomphorst J, et al. The epidemiology of injuries at the London 2012 Paralympic Games. Br J Sports Med. 2013;47(7):426–32. https://doi.org/10.1136/bjsports-2013-092374. Epub 2013 Mar 20.
62. Webborn N, Cushman D, Blauwet CA, Emery C, Derman W, Schwellnus M, et al. The epidemiology of injuries in football at the London 2012 Paralympic Games. PM R. 2016;8(6):545–52. https://doi.org/10.1016/j.pmrj.2015.09.025. Epub 2015 Oct 30. Erratum in: PM R. 2017;9(4):e1.
63. Mayr de Oliveira Silva G. Football for the blind: Aplikovany'ch Pohybovy'ch Aktivit. Olomouc, Czech Republic: Palacky University; 2008.
64. Carroll KL, Leiser J, Paisley TS. Cerebral palsy: physical activity and sport. Curr Sports Med Rep. 2006;5(6):319–22. https://doi.org/10.1007/s11932-006-0060-x.
65. Patatoukas D, Farmakides A, Aggeli V, Fotaki S, Tsibidakis H, Mavrogenis AF, et al. Disability-related injuries in athletes with disabilities. Folia Med (Plovdiv). 2011;53(1):40–6. https://doi.org/10.2478/v10153-010-0026-x.
66. Fagher K, Jacobsson J, Timpka T, Dahlström Ö, Lexell J. The Sports-Related Injuries and Illnesses in Paralympic Sport Study (SRIIPSS): a study protocol for a prospective longitudinal study. BMC Sports Sci Med Rehabil. 2016;8(1):28. https://doi.org/10.1186/s13102-016-0053-x. Erratum in: BMC Sports Sci Med Rehabil. 2016;8:30.

Volleyball (Indoor, Beach)

10

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10.1 History of Volleyball

Volleyball is a common sport, which is played by approximately 200 million players worldwide [1]. It was firstly introduced in Holyoke, Massachusetts in 1895 by William G. Morgan, physical director of the Young Men's Christian Association (YMCA) (Fig. 10.1). It was initially played and an indoor sport and especially attracted those players who found of basketball too hard to play and traumatic. Morgan himself proposed the first rules which were printed in the first edition of the Official Handbook of the Athletic League of the Young Men's Christian Associations of North America (1897). The game soon became really attractive for players of both sexes in schools, playgrounds, the armed forces, and other organizations in the United States, and it was subsequently introduced to other countries. The first official ball used in volleyball is disputed; some sources say Spalding created the first official ball in 1896, while others claim it was created in 1900 [2]. In 1916, the original rules were revised by the YMCA in conjunction with the National Collegiate Athletic Association (NCAA). In 1920, "three hits" rule and a rule against hitting from the back row were established. The first nationwide tournament in the

Fig. 10.1 William G. Morgan invented Volleyball in 1895

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United States took place in New York City in 1922. Volleyball was then introduced into Europe by American troops during World War I. In that period first national organizations were formed all around the world. The Fédération Internationale de Volleyball (FIVB) was founded in Paris in 1947 and moved to Lausanne, Switzerland, in 1984. International volleyball competitions began in 1913 with the first Far East Games, in Manila. However, in Asia during the early 1900s and until after World War II, volleyball was played on a larger court, with a lower net, and nine players on a team. The FIVB sponsored world volleyball championships for men in 1949 and for both men and women in 1952. Volleyball became an Olympic sport for both men and women at the 1964 Olympic Games in Tokyo.

10.2 The Game

The game is played on 9 m (30 ft) wide by 18 m (60 ft) long court. A center line divides it into two equal playing areas for the two teams. Over this central line, a 2.43 m high net is located for men's competitions, while it is 2.24 m high for women's competition [3]. A vertical tape marker is attached to the net directly above each side boundary line of the court, and, to help game officials judge whether served or volleyed balls are in or out of bounds, a flexible antenna extends 1 m (3 ft) above the net along the outer edge of each vertical tape marker. The ball used is around 260–280 g (9–10 ounces) and is inflated to about 65 cm (25.6 in.) in circumference. A ball must pass over the net entirely between the antennae.

Players may not step completely beyond the center line while the ball is in play. A line is located 3 m (10 ft) away and parallel to the center line on each half of the court, and it divides the players playing in the front row and those playing in the back row. It indicates the point in front of which a backcourt player may not drive the ball over the net from a position above the top of the net. This offensive action is called a spike and is usually performed most effectively and with greatest power near the net by the forward line of players. On the other hand, players in the back row may spike only jumping behind this line. A service area, 9 m (30 ft) long, is outside of each court end line. The service must be made from within or behind this area. A space at least 2 m (6 ft) wide around the entire court is needed to permit freedom of action, eliminate hazards from obstructions, and allow space for net support posts and the officials' stands. A clear area above the court at least 8 m (26 ft) high is required to permit the ball to be served or received and played without interference. In competition, each team consists of six players, three of whom take the forward positions in a row close to and facing the net, the other three playing the backcourt.

Figure 10.2 shows the dynamic of this sports—a moment in the volleyball match where a player is attacking and the opponents are positioned to block.

The 2000 Olympics introduced significant rule changes to international competition. A new player: the libero was introduced. He serves as a defensive specialist and may switch with all players in the back row the libero wears a different color from the rest of the team and is not allowed to serve or rotate to the front line. Another important rule change allowed the defensive side to score, whereas formerly only the serving team was awarded points.

10.3 Biomechanics of Volleyball

Volleyball is a high demanding sport with two major injury scenarios being possible: sprains and overuse syndromes. Ankles, knees, and fingers are mostly affected by traumatic events, while shoulders and knees may undergo overuse syndromes. The precise comprehension of the biomechanics of the specific skills is crucial since it allows better sport performance reducing the risk of injury.

Jumping is the most important action of volleyball since it affects serving, spiking, and blocking and is closely related to landing. The ability of jumping higher leads to increased performances [4]. Individual muscle properties, jumping technique, and playing surface all

Fig. 10.2 Volleyball dynamics of attacking and blocking

influence the height of a jump and must be therefore analyzed when trying to prevent injuries.

The first aspect to take into account is muscle function; the force generated by the muscle is transferred through the tendons to the bones and finally to the ground. These steps are influenced by muscle properties, composition of the tendons, jumping technique, and the playing surface (including both shoes and floor type). Intrinsic muscle properties such as the neural activation capacity, the force–velocity relationship, and the force–length relationship can be altered (within individual limits) by training. However, training should be individualized to address and improve any specific neuromuscular system deficiencies: while some athletes might have to increase their maximal force development, others might have to improve upon a deficit in maximum muscle contraction velocity or maximum power capacity. Repetitive jump training is important to improve performance, and it showed to have no negative effects in terms of jump's gesture or intensity among the different game sets [5]. However, this stable performance along with hard training is in opposition with the results by Wnorowski et al. who demonstrated a deterioration of jumping performance during game sets in elite male Polish players [6]. Historic studies have confirmed that explosive plyometric training increases jump height in volleyball players [7]. More recently, Krističević et al. found that completion of a 5-week plyometric training program improved selected vertical jump tests in young female volleyball players [8]. However, they did not find significant changes in spike and block jumps following the plyometric training program.

The second aspect is the jumping technique; several aspects such as a countermovement or the arm swing may have substantial effects on the spike jump (SPJ) height performance. A countermovement, i.e., a lowering of the center of mass just before the beginning of the push-off phase, increases jump height [9].

Spike jump (SPJ) height is generally greater than the height reached during a squat jump (SJ) or a countermovement jump (CMJ) from a standing position. While SPJ are reported to be approximately 25% higher than CMJ, CMJ are about 7% higher than SJ [10].

The reasons for this higher performance are increased myoelectrical activity in the stretch-shortening cycle (SSC), the storage and recoil of elastic energy, and a higher active state, i.e., increased motoneuron activity before the start of the muscle shortening [10]. Researchers have also reported a 19–23% increase in jumping height due to the use of an arm swing. The reasons for this improvement are an elevated center of mass (due to arm elevation at take-off) and a decrease of contraction velocity of the leg muscle, which leads to an increase in muscle force generation via the force–velocity relationship. Training is therefore crucial to maximize jump height the SPJ since it is a complex and unique sport gesture. In fact, although it is a two-legged jump, only the range of motion (ROM) of the right knee (flexion–extension) and the maximal angular velocity of the left (non-dominant) shoulder hyperextension were significantly related to jumping height. The reason for this is probably that the SPJ is rather asymmetrical.

Sheppard et al. showed that assisted jumping may promote the leg extensor musculature to undergo a more rapid rate of shortening, and chronic exposure appears to improve jumping ability [11].

The third aspect to take into account is the playing surface. It has been shown experimentally that jumps on sand are on average 14% lower than jumps from a rigid surface. The reason for this decrease is the energy absorbed by the sand. In addition, the instability of the sand reduces peak power output due to the differences of body configuration at the lowest body position and lower limb joints' range of motion [12]. Lesiniski et al. found no statistically significant interactions of fatigue by surface condition. They concluded that fatigue impairs neuromuscular performance during rope jumps (DJs) and countermovement jumps (CMJs) in elite volleyball players, whereas surface instability affects neuromuscular DJ performance only. Fatigue-induced changes in jump performance are similar on stable and unstable surfaces in jump-trained athletes [13].

Similar but less substantial effects can be expected from different shoe sole or indoor surface materials. Although stiff materials have advantages during the take-off, they will also absorb less energy during the landing phase, which might lead to higher stress in the athlete's lower limb joints. Hosseininezhad et al. investigated the effects of polyvinyl chloride outsole (PVC shoe) and styrene-butadiene sole (TPE shoe) on countermovement jump (CMJ) and squat jump (SJ) [14]. They showed that the height of vertical jump in CMJ was higher than SJ regardless the type of sole. The use of PVC shoe with lower hardness resulted in more tensile energy storage and energy return than TPE shoe. Therefore, shoe outsole characteristics such as material and hardness can change the height of vertical jump through affecting on storage and return of energy.

10.4 Epidemiology of Volleyball Injuries

The epidemiology of volleyball injuries is variable according to the level of athletes analyzed. In 2015, the FIVB Injury Surveillance System performed a survey on 2710 reports [15]. In total, 440 injuries were reported, 275 during match play (62.5%), and 165 during training (37.5%). The incidence of match injuries was 10.7/1000 player hours; this was greater for senior players than junior players (RR: 1.32, 95% CI 1.03–1.69), while there was no difference between males and females (RR: 1.09, 95% CI 0.86–1.38). The incidence of injuries during match play was greater for center players than for other player functions. The majority of injuries were minimal to mild, while severe injuries were rare. Ten out 440 injuries led to an absence from training of more than 4 weeks. Of these, 8 occurred during match play, corresponding to an incidence of 0.3 severe injuries per 1000 player hours (95% CI 0.1–0.5). The most common injury type was joint sprain (32.5%, *n* = 143), followed by muscle strains (14.1%, *n* = 62) and contusions (12.7%, *n* = 56).

Overall the ankle was the most commonly injured body part (25.9%), followed by the knee (15.2%), finger/thumb (10.7%), and lumbar/lower back (8.9%). This distribution was almost similar between match play (ankle: 31.3%, knee: 15.6%, fingers/thumb: 10.2%) and training (ankle: 17.0%, knee: 13.2%, lower back: 11.9%). Competition rates did not differ from practice rates among all injuries (7.48 vs. 6.91 per 1000 athlete exposures (AEs)) [16]; in addition, the injury rates in men and women were 4.69 and 7.07 per 1000 AEs, respectively. The injury rate was greater in women than men (IRR, 1.51; 95% CI, 1.19–1.90). Moreover time-loss (TL) injuries (resulting in participation restriction for at least 24 h), rates were 1.75 and 2.62 per 1000 AEs for men and women, respectively [16].

When analyzing sprains, the ankle was the most commonly affected (*n* = 87) (19.8%), followed by finger/thumb (*n* = 26) and knee (*n* = 17). When considering muscle strains, they were mostly located in the lower back (*n* = 19) and thigh (*n* = 10). In total, 23.0% of all injuries (*n* = 101) were the result of traumatic contact between players, while 20.7% (*n* = 91) were overuse injuries, and 17.3% (*n* = 76) were reported as noncontact trauma.

A Survey of Injuries Among Male Players of the Chinese Taipei National Volleyball Team showed that the incidence of injuries increased with the training sessions [17]. The percentage of injuries in the second session was greater than that in the first session when double sessions were performed daily. In the first session, 24%, 16%, 16%, and 16% of all injuries occurred in the knees, waists, fingers, and ankles, respectively. In the second session, knees and waists were the most common injury locations, which accounted for 33.3% and 23.8% of all injuries, respectively. This is the consequence of fatigue, reduced muscle performance and reduced proprioception. Based on the statistics, knee injuries were the most severe and frequent injuries of the lower extremities, which accounted for 33.3% of all injuries, followed by waists (23.8%), ankles (16%), fingers (16%), and shoulders (12%).

A difference has been also recorded according to the different playing phase [18]. The authors reported a total of 178 injuries in 121 out of 144 volleyball players. Most common location was the ankle (23.03%) followed by knee (21.91%), shoulder (11.79%), back (10.67%) hamstring (9.55%), groin (6.74%), finger (6.17%), hand (3.93%), and other (5.61%).

Most common cause for injury was spiking (33.70%), blocking (24.15%), diving (17.41%), setting (11.23%), and others (14.04%). Maximum incidence of injuries affected muscles (32.40%), ligaments (24.71%), tendon (9.55%), bones (factures) (2.80%), braises (6.17%), and other (7.40%).

Among knee injuries, patellar tendinopathy, also known as "jumper's knee," is the most common situation having been reported in about 50% of male indoor volleyball players [19, 20]. The rate is slightly inferior in elite players (around 40%) [21] and more frequent in males than females. It is more common in volleyball players who train on hard surfaces [21] and is therefore less common in beach volleyball players [22]. Middle blockers tend to suffer from jumper's knee more than do players at other positions.

On the contrary, acute trauma leading to anterior cruciate ligament (ACL) trauma is uncommon [23]. The reported incidence is about 0.1 ACL injuries/1000 athlete exposures among female collegiate volleyball athletes in the United States (compared with a rate of 0.4/1000 athlete exposures in soccer and 0.27/1000 athlete exposures in basketball) [24]. These data have been confirmed by retrospective cohort studies from Norway and the Netherlands [25, 26].

10.5 Top Five Sports-Related Injuries

Women generally have a higher rate of overuse injuries while men have a higher rate of ball contact-related injuries [16].

10.5.1 Ankle Sprains

Ankle sprains are the most common acute injury in volleyball, with a reported incidence of up to 41% of all volleyball-related injuries [26]. They usually occur when landing onto another player's foot, often a player from the opposing team, so that they are more common in players of the front row. They often result from a supination trauma with the evidence of injury to the lateral compartment ligaments with swelling and tenderness (Fig. 10.3).

Recurrent sprains are common, with one study showing a 42% risk of resprain in volleyball players within 6 months of the initial sprain [25].

10.5.2 Knee Sprains

The knee is the second most commonly injured body part among NTL injuries (men, 25.5%; women, 16.3%) [16]. However, is the majority of the published they show a considerably higher rate in women than men in landing and cutting sports [27]. In addition to female gender and prior ACL tear, a number of risk factors have been proposed including intercondylar notch width, generalized ligamentous laxity, and increased body mass index [28]. In volleyball, the mechanism for ACL tear usually is coming down awkwardly from a jump or a cutting maneuver.

Fig. 10.3 Lateral compartment swelling and tenderness are extremely common after inversion ankle sprains

10.5.3 Throwing Shoulder and Suprascapular Neuropathy

The exact rate of shoulder injuries is hard to assess. Traumatic events are uncommon, while overuse syndromes are more frequent with a reported incidence of around 12% [17, 18]. The phases of the overhead spike and serve predispose to excessive external rotation, glenohumeral internal rotational deficit (GIRD), internal impingement, labral tears, rotator cuff tears, and neurovascular structures entrapment which may lead to painful syndromes and dysfunction [29–31].

Increased external rotation and GIRD are the initial event [32]. Repetitive abduction and external rotation movement is common in hitters and servers; it increases the laxity of the anterior capsule and at the same time causes retraction of the posterior capsule. There is therefore a static translation of the humeral head into a more anterior and superior position. This trend may aggravate.

Another pathologic condition which is aggravated by this anterior subluxation is the internal impingement. It is caused by the impingement of the deep surface of the supraspinatus tendon rotator with the posterior labrum during the throwing movement [33, 34].

Suprascapular neuropathy occurs from traction and/or compression of the nerve during the extreme motions of the arm during the cocking or follow-through phases of the arm swing while serving and hitting [35, 36]. The nerve can become entrapped at different locations, but it is more common at the spinoglenoid notch. Nerve entrapment causes infraspinatus muscle weakness and atrophy which is reported in 12–30% of top-level volleyball players [37]. Biceps traction leading to a SLAP lesion and pulley pathology are often related to the deceleration/follow-through phase [38].

10.5.4 Low Back Pain

Low back pain is the fourth most common injury seen in volleyball players [18]. One study reported that low back pain is experienced in 63% of volleyball players [39]. More than half of volleyball players experience low back pain during their sports career, but less than 20% of players met with a physical therapist to receive the care they need [18].

Moreover 47.4% of volleyball players with low back pain continued to have low back pain for the rest of their sports career [40]. This constant pain can affect muscle performance, preventing athletes from reaching their maximal potential.

One common reason for low back pain in volleyball players is asymmetries in endurance for muscles that stabilize the low back [41]. Core muscles provide stability to the low back and spine with all movements. If imbalances are present in the core muscles, then players may spike or serve the ball with increased turning and bending in their spine. These extra movements caused by reduced stability in the spine cause increased pressure in the joints of the lower spine. This repetitive pressure over time can lead to low back pain.

Other muscles influence the stability of the spine [42]. The gluteal muscles prevent the trunk and hips from bending too far forward during landing. If gluteal muscles do not have the endurance to perform this motion, then your upper body will bend further forward as you land. This poor landing posture results in decreased stability of the spine and increases the risk of low back pain in volleyball players. Studies have shown that at rest, volleyball players with low back pain stand with an anterior pelvic tilt [43, 44]. It has been shown that there is a correlation between landing with an anterior pelvic tilt and low back pain in volleyball players.

10.5.5 Patellar Tendinopathy

Patellar tendinopathy is an overuse injury; its prevalence has been reported to be 11% in Swedish elite junior volleyball players [45] and 36% in male senior professional volleyball players [46]. The symptoms onset usually occurs gradually after a threshold of cumulative tissue injury has been exceeded. Histological examination on tendon samples reveals degeneration and fibrotic scarring of the tendon, particularly at the bone-tendon junction. The normally parallel collagen bundles are disorganized, and the observed tenocytes display altered morphology [47]. It has been hypothesized that excessive tendon loading induces tenocyte apoptosis (programmed cell death) [48]. An increased incidence of jumper's knee has been reported in athletes who jump highest and in those who develop the deepest knee flexion angle during landing from a spike jump [49]. Another study suggested that valgus knee strain during the eccentric loading phase of the spike jump take-off may contribute to the observed asymmetric onset of patellar tendinopathy [50].

10.5.6 Final Consideration: Concussions

They account for 19.4% and 14.8% of men's and women's volleyball TL injuries, respectively. Because volleyball includes rules to limit the amount of contact, such a finding may be unexpected [51]. However, the most common mechanism of concussion was not due to player contact [52], but rather ball contact, particularly during blocking and digging.

10.6 Prevention of Injuries

10.6.1 Ankle Sprains

Ankle sprains are the most common traumatic event in volleyball. A non-negligible number of players suffer a recurrence within 6 months from the initial trauma. Therefore, it is particularly important to complete a supervised rehabilitation program before returning to play. Balance board training to regain proprioception has shown to be effective in preventing recurrences of ankle sprains in volleyball players as well to prevent initial trauma [53]. Wearing a lace-up ankle brace or taping the ankle for the remainder of the season also may help reduce the incidence of recurrent sprain.

10.6.2 Knee Sprains

One study showed that less than 50% of athletes returned to playing sports at their preinjury level or returned to participation in competitive sport when surveyed 2–7 years after ACL reconstruction [54]. Since several athletes are not able to return to high-level sports after ACL reconstruction, and the risk of osteoarthritis is increased after an ACL tear, there has been considerable interest on prevention programs. There have been numerous studies looking at proprioceptive and plyometric training programs to decrease ACL injury risks in athletes engaging in sports with cutting, jumping, and sprinting, with many showing encouraging results. [27, 55]. One study included female volleyball players and showed that a neuromuscular training program significantly reduced ACL injury [56].

10.6.3 Throwing Shoulder and Suprascapular Neuropathy

Attention to throwing mechanics and appropriate stretching, strength, and conditioning programs may reduce the risk of injury in this highly demanding activity. Early discovery of symptoms followed by conservative management with rest and rehabilitation with special attention to retraining mechanics may mitigate the need for surgical intervention. Prevention of injury is always more beneficial to the long-term health of the thrower than is surgical repair.

It usually consists of selective stretching of the posterior capsule using “sleeper stretches” and reinforcement of the anterior “wall” (subscapularis, latissimus dorsi, and teres major) [57]. In addition, since malpositioning of the scapula can contribute to impingement and weak periscapular muscles can alter shoulder kinematics leading to shoulder pain: therapy of shoulder problems should always include scapular stabilizing exercises.

10.6.4 Low Back Pain

In order to prevent and/or treat low back pain the core muscles should be strengthened. This helps stabilizing the lower spine is stable. In addition, performing an abdominal contraction before any overhead motion (spiking, serving, setting) or landing may be beneficial. During an abdominal contraction, the core muscles rotate the pelvis posteriorly reduces compression in the joints of the lower back. This also prevents players from landing in an anterior pelvic tilt. Strengthening other lower body muscles that attach to the pelvis and provide stability to the spine such gluteal muscles is indicated.

10.6.5 Patellar Tendinopathy

Potential prevention strategies include changes in the jumping technique, training, and rehabilitation.

Changes in the jumping technique in order to prevent valgus strain on the lead knee and to keep knee flexion to a minimum on landing may be advantageous in reducing the rate and severity of patellar tendinopathy. It must be highlighted the importance of the landing phase of the jump since it is the most common reason for knee injuries in volleyball players. In any case, no clear scientific data have been published to support this fact.

Training volume on hard playing surfaces should not exceed the capacity of the patellar tendon to regenerate. However, it is still debated how often and by what percentage the volume of jump training can be safely increased over a given time period. It has been shown that a critical step occurs when young players are promoted from the junior to the senior level. During this passage, players are abruptly moved from a relatively safe training environment to an elite club or sports school that practices daily and has a structured program of weight training. Therefore, great care should be taken to muscle stretching and rest to strengthening. In addition, lack of strength and flexibility in the core muscles [58], and lack of balance or control of the body when jumping and landing has been associated with poor form in the athlete's jumps resulting in injury and reduced performance.

Eccentric training protocols (particularly those using decline squats) have shown to be effective in treating patellar tendinopathy [59, 60]. However, other studies have reported that eccentric training of the quadriceps was ineffective in treating symptomatic jumper's knee in volleyball players during the competition season [61]. There is preliminary evidence that such knee extensor eccentric training protocols, used prophylactically, can effectively prevent sports-related anterior knee pain from patellar tendinopathy [62]. Specific rehabilitation and strengthening of the core muscles may prevent functional imbalances of the lower limbs' weakness contributing to the treatment of anterior knee pain. In addition, when treating jumper's knee, it is important to rehabilitate beyond the absence of symptoms and to avoid return to play before the athlete is adequately rehabilitated, in order to maximize secondary prevention of recurrent injury and thus minimize the risk of chronicity.

Finally, although reports of benefit of external orthoses abound, there is no evidence to support the routinely use of patellar straps (ostensibly designed to redistribute the forces acting on the patellar tendon) in the treatment or prevention of jumper's knee.

Conclusions

Volleyball is one of the most common sports. Although it is a noncontact sport, it is physically demanding. Players may suffer two types of injuries: sprains and overuse syndromes. Among sprains, ankle and knee are usually affected; among overuse syndromes, patellar and Achilles tendinopathy are common. Ankle and knee sprains usually follow landing from a jump. Volleyball is not a contact sport, but players are usually in the air and while landing they can come in contact with teammates or opponents resulting in traumatic events. Overuse syndrome have a gradual onset. They are caused by extrinsic (hard surfaces, shoes, training methods) or intrinsic (muscle performance, jumping technique, core muscle weakness) factors. Prevention is essential to reduce the severity of these injuries; however, when they occur, specific rehabilitation strategies and modifications of training programs are mandatory to achieve good results and comeback to performance.

References

1. Fédération Internationale de Volleyball (FIVB). 1994. Lausanne, Switzerland: X-Press. 47:1.
2. History of volleyball. Volleyball World Wide. Archived from the original on 13 May 2011. Retrieved 21 Sept 2007.
3. volleyball | Definition, Rules, Positions, & Facts. Encyclopedia Britannica. Retrieved 2 Aug 2018.
4. Sattler T, Hadžić V, Dervišević E, Markovic G. Vertical jump performance of professional male and female volleyball players: effects of playing position and competition level. J Strength Cond Res. 2015;29(6):1486–93.
5. Lima RF, Palao JM, Clemente FM. Jump performance during official matches in elite volleyball players: a pilot study. J Hum Kinet. 2019;67:259–69. https://doi.org/10.2478/hukin-2018-0080.
6. Wnorowski K, Aschenbrenner P, Skrobecki J, Stech M. An assessment of a volleyball player's loads in a match on the basis of the number and height of jumps measured in real-time conditions. Balt J Heal Phys Activity. 2013;5(3):199–206.

7. Ziv G, Lidor R. Vertical jump in female and male volleyball players: a review of observational and experimental studies. Scand J Med Sci Sports. 2010;20(4):556–67.
8. Krističević T, Krakan I, Baić M. Effects of plyometric training on sport-specific tests in female volleyball players. Acta Kinesiol. 2016;10(Suppl 1):25–9.
9. Wagner H, Tilp M, von Duvillard SP, Mueller E. Kinematic analysis of volleyball spike jump. Int J Sports Med. 2009;30(10):760–5.
10. 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.
11. Sheppard JM, Dingley AA, Janssen I, Spratford W, Chapman DW, Newton RU. The effect of assisted jumping on vertical jump height in high-performance volleyball players. J Sci Med Sport. 2011;14(1):85–9. https://doi.org/10.1016/j.jsams.2010.07.006.
12. Giatsis G, Panoutsakopoulos V, Kollias IA. Biomechanical differences of arm swing countermovement jumps on sand and rigid surface performed by elite beach volleyball players. J Sports Sci. 2018;36(9):997–1008. https://doi.org/10.1080/02640414.2017.1348614.
13. Lesinski M, Prieske O, Demps M, Granacher U. Effects of fatigue and surface instability on neuromuscular performance during jumping. Scand J Med Sci Sports. 2016;26(10):1140–50. https://doi.org/10.1111/sms.12548. Epub 2015 Sep 15.
14. Hosseininezhad M, Eslami M, Bassiri Z, Rabiei M, Hoseninejad SE. The effect of shoe outsole hardness on vertical jump height of female handball athletes. The 3rd Iranian International Conference of Women's Health, Shiraz, Iran; 2014.
15. Bere T, Kruczynski J, Veintimilla N, Hamu Y, Bahr R. Injury risk is low among world-class volleyball players: 4-year data from the FIVB Injury Surveillance System. Br J Sports Med. 2015;49(17):1132–7. https://doi.org/10.1136/bjsports-2015-094959.
16. Baugh CM, Weintraub GS, Gregory AJ, Djoko A, Dompier TP, Kerr ZY. Descriptive epidemiology of injuries sustained in National Collegiate Athletic Association Men's and Women's Volleyball, 2013–2014 to 2014–2015. Sports Health. 2018;10(1):60–9. https://doi.org/10.1177/1941738117733685.
17. Huang H-Y, Teng T-L, Liang C-C. Volleyball injuries: a survey of injuries among male players of the Chinese Taipei National Volleyball Team. Am J Sports Sci. 2015;3(6):109–14. https://doi.org/10.11648/j.ajss.20150306.12.
18. Jadhav KG, Deshmukh PN, Tuppekar RP, Sinku SK. A survey of injuries prevalence in varsity volleyball players. J Exerc Sci Physiotherapy. 2010;6(2):102–5.
19. Reeser JC, Agel J, Dick R, Bahr R. The effect of changing the centerline rule on the incidence of ankle injuries in women's collegiate volleyball. Int J Volleyball Res. 2001;4:12–6.
20. 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. 2005;33:561–7.
21. Ferretti A. Epidemiology of jumper's knee. Sports Med. 1986;3:289–95.
22. Bahr R, Reeser JC. Injuries among world-class professional beach volleyball players. The Federation Internationale de Volleyball beach volleyball injury study. Am J Sports Med. 2003;31:119–25.
23. Ferretti A, Papandrea P, Conteduca F, Mariani PP. Knee ligament injuries in volleyball players. Am J Sports Med. 1992;20:203–7.
24. NCAA Injury Surveillance System. http://www1.ncaa.org/membership/ed_outreach/health-safety/iss/index.html. Accessed 19 Sept 2005.
25. Bahr R, Bahr IA. Incidence of acute volleyball injuries: a prospective cohort study of injury mechanisms and risk factors. Scand J Med Sci Sports. 1997;7:166–71.
26. Verhagen EA, Van der Beek AJ, Bouter LM, Bahr RM, Van Mechelen W. A one season prospective cohort study of volleyball injuries. Br J Sports Med. 2004a;38:477–81.
27. 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. 2006a;34:490–8.
28. Griffin LY, Albohm MJ, Arendt EA, Bahr R, Beynnon BD, Demaio M, Dick RW, Engebretsen L, Garrett WE Jr, Hannafin JA, Hewett TE, Huston LJ, Ireland ML, Johnson RJ, Lephart S, Mandelbaum BR, Mann BJ, Marks PH, Marshall SW, Myklebust G, Noyes FR, Powers C, Shields C Jr, Shultz SJ, Silvers H, Slauterbeck J, Taylor DC, Teitz CC, Wojtys EM, Yu B. Understanding and preventing noncontact anterior cruciate ligament injuries. A review of the Hunt Valley II meeting. Am J Sports Med. 2006;34:1512–32.
29. Seminati E, Minetti AE. Overuse in volleyball training/practice: a review on shoulder and spine-related injuries. Eur J Sport Sci. 2013;13(6):732–43.
30. Challoumas D, Stavrou A, Dimitrakakis G. The volleyball athlete's shoulder: biomechanical adaptations and injury associations. Sports Biomech. 2017;16(2):220–37.
31. Eerkes K. Volleyball injuries. Curr Sports Med Rep. 2012;11(5):251–6.
32. Lintner D. Glenohumeral internal rotation deficits in professional pitchers enrolled in an internal rotation stretching program. Am J Sports Med. 2007;35:617–21.
33. Drakos MC, Jonas RR, Allen A, Potter HG, Altchek DW. Internal impingement of the shoulder in the overhead athlete. J Bone Joint Surg Am. 2009;91:2719–28.
34. Heyworth BE, Williams RJ. Internal impingement of the shoulder. Am J Sports Med. 2009;37:1024–37.
35. Ferretti A, De Carli A, Fontana M. Injury of the suprascapular nerve at the spinoglenoid notch. The natural history of infraspinatus atrophy in volleyball players. Am J Sports Med. 1998;26:759–63.

36. Sandow MJ, Ilic J. Suprascapular nerve rotator cuff compression syndrome in volleyball players. J Shoulder Elb Surg. 1998;7:516–21.
37. Lajtai G, Pfirrmann C, Aitzetmuller Pirkl C, Gerber C, Jost B. The shoulders of professional beach volleyball players. High prevalence of infraspinatus muscle atrophy. Am J Sports Med. 2009;37:1375–83.
38. Ferretti A, DeCarli A. Shoulder injuries in volleyball. In: Reeser J, Bahr R, editors. Volleyball: Olympic handbook of sports medicine. Chichester: Wiley; 2008. p. 118–29.
39. Trompeter K, Fett D, Platen P. Prevalence of back pain in sports: a systematic review of the literature. Sports Med. 2017;47(6):1183–207. https://doi.org/10.1007/s40279-016-0645-3.
40. Noormohammadpour Rostami M, Mansournia MA, Farahbakhsh F, Pourgharib Shahi MH, Kordi R. Low back pain status of female university students in relation to different sport activities. Eur Spine J. 2016;25(4):1196–203.
41. Coreia JP, Oliveira R, Vaz JR, Silva L, Pezarat-Correia P. Trunk muscle activation, fatigue and low back pain in tennis players. J Sci Med Sport. 2016;19:311–6. https://doi.org/10.1016/j.jsams.2015.04.002.
42. Haddas R, Sawyer SF, Sizer PS, Brooks T, Chyu MC, James CR. Effects of volitional spine stabilization and lower-extremity fatigue on the knee and ankle during landing performance in a population with recurrent low back pain. J Sport Rehabil. 2017;26(5):329–38. https://doi.org/10.1123/jsr.2015-0171.
43. Movahed M, Salavati M, Sheikhhoseini R, Arab AM, O'Sullivan K. Single leg landing kinematics in volleyball athletes: a comparison between athletes with and without active extension low back pain. J Bodyw Mov Ther. 2019;23(4):924–9. https://doi.org/10.1016/j.jbmt.2019.01.012.
44. Sheikhhoseini R, Alizadeh MH, Salavati M, O'Sullivan K, Shirza E, Movahed M. Altered lower limb kinematics during jumping among athletes with persistent low Back pain. Ann Appl Sport Sci. 2018;6(2):23–30.
45. Gisslèn K, Gyulai C, Söderman K, Alfredson H. High prevalence of jumper's knee and sonographic changes in Swedish elite junior volleyball players compared to matched controls. Br J Sports Med. 2005;39(5):298–301.
46. Lian Ø, Holen KJ, Engebretsen L, Bahar R. Relationship between symptoms of jumper' knee and the ultrasound characteristics of the patellar tendon among high level male volleyball players. Scand J Med Sci Sports. 1996;6:291–6.
47. Cook JL, Feller JA, Bonar SF, Khan KM. Abnormal tenocyte morphology is more prevalent than collagen disruption in asymptomatic athletes' patellar tendons. J Orthop Res. 2004;22:334–8.
48. Scott A, Khan KM, Heer J, Cook JL, Lian O, Duronio V. High strain mechanical loading rapidly induces tendon apoptosis: an ex vivo rat tibialis anterior model. Br J Sports Med. 2005;39:e25.
49. Richards DP, Ajemian SV, Wiley JP, Zernicke RF. Knee joint dynamics predict patellar tendinitis in elite volleyball players. Am J Sports Med. 1996;24:676–83.
50. Lian Ø, Dahl J, Ackermann PW, Frihagen F, Engebretsen L, Bahr R. Pronociceptive and antinociceptive neuromediators in patellar tendinopathy. Am J Sports Med. 2006;34(11):1801–8.
51. Marar M, McIlvain NM, Fields SK, Comstock RD. Epidemiology of concussions among United States high school athletes in 20 sports. Am J Sports Med. 2012;40:747–55.
52. Reeser JC, Gregory A, Berg RL, Comstock RD. A comparison of women's collegiate and girls' high school volleyball injury data collected prospectively over a 4-year period. Sports Health. 2015;7:504–10.
53. 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. 2004b;32:1385–93.
54. Ardern CL, Taylor NF, Feller JA, Webster KE. Return-to-sports outcomes at 2 to 7 years after anterior cruciate ligament reconstruction surgery. Am J Sports Med. 2012;40:41–8.
55. Kelly AKW. Anterior cruciate ligament injury prevention. Curr Sports Med Rep. 2008a;7:255–62. https://doi.org/10.1249/JSR.0b013e318186c3f5.
56. Hewett TE, Lindenfeld TN, Riccobene JV, Noyes FR. The effect of neuromuscular training on the incidence of knee injury in female athletes. A prospective study. Am J Sports Med. 1999;27:699–706.
57. Bach HG, Goldberg BA. Posterior capsular contracture of the shoulder. J Am Acad Orthop Surg. 2006;14:265–77.
58. Sommer HM. Patellar chondropathy and apicitis, and muscle imbalances of the lower extremities in competitive sports. Sports Med. 1988;5:386–94.
59. 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;39:847–50.
60. 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.
61. Visnes H, Hoksrud A, Cook J, Bahr R. No effect of eccentric training on jumper's knee in volleyball players during the competitive season: a randomized clinical trial. Clin J Sport Med. 2005;15:227–34.
62. Fredberg U, Bolvig L. Prophylactic training reduces the frequency of jumper's knee but not Achilles tendinopathy. Br J Sports Med. 2005;39:384.

11 Water Polo

Daniel Slullitel, Mateo Escalante, and Franco Della Vedova

11.1 Introduction

Water polo is a mixed sport that contains swimming, full contact, and overhead throwing at the same time. It has bursts of very high-intensity demands (15 s) followed by low-intensity intervals that make it very physiologically demanding. Playing area is 30 m × 20 m length, to 10–20 m width, depending on the level of competition, in a 2 m minimum pool depth. Teams have six field players and a goalkeeper. The ball diameter is 68–71 cm for men, and it weighs from 400 to 450 g, making it difficult to handle and, of course, hard to be hit by. The object of the game is to score goals by putting it on the opponent's net. You can attack any player holding the ball (that cannot be immersed underwater), making it a tough collision sport. To moderate contact if a player is attacked without the ball, the offensive player is excluded for 20 s. Having three consecutive exclusions, means leaving permanently. Game is separated in four quarters of 8 min at elite level, with a 2 min interval between them [1]. Time possession by each team is 30 s, and if no shot is taken, a free throw is awarded to the opposite team, making this game very quick and demanding.

It was originated first described in 1860 as an aquatic equivalent of rugby and is the oldest team sport included in the Olympic Games. It is played since 1900 in its male version but started its woman competition lately at the Sydney Olympic Games in 2000 [2].

Swimming in water polo is different because players either carry the ball or "see" the field while playing (Photo 11.1) so it is always heads up, with neck extension. Body rolling is not performed neatly, also the stroke is shorter. Swimming postures involve greater shoulder abduction, higher elbow position that places a heavier load on the joint with less stroke efficiency [3].

Overhead shooting is also different, first, the ball is larger and heavier, then there is no support on the water. Anyway, the speed of the water polo shot can reach up to 70/km/h [4]. Another shoulder movement is passing; this is a low-intensity movement of the shoulder and could also be done by the non-dominant arm, that must be trained for that. All these movements could be stopped anytime by the opponent, as when the ball is held by a player, contact could be made anywhere. So, shooting or passing nearly always got to be compromised by a defender, making these movements prone to contact lesions and always shorter and quicker than regular shooting (Photo 11.2).

Another distinct feature is a rotational movement known as the "eggbeater kick" [4]. Water

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Photo 11.1 "Heads up" swimming

polo players, especially the goalkeeper, must maintain an above surface playing position, allowing to stand above water at different heights and move laterally, performing specific maneuvers for each player. No player is allowed to touch the bottom of the pool at any time. This specific drill is performed by rotating clockwise and counterclockwise in opposite fashion between both legs, creating high loads on groin adductors and hip, and especially on the medial side of the knee. Its dynamics may cause medial collateral laxity and patellofemoral pain, similar to breaststrokers [5, 6].

11.2 Epidemiology

Water polo was ranked fifth, in the proportion of injured athletes on 2016 Olympic [7] with an injury mean ranging from 9.6/12.9 per 100 players, while in the FINA world championship it was15.9 [7–9]. That makes us know that it is a tough contact sport. Injury rate has been increasing from 2008 to 2016, doubling its frequency [7] in a review of 8904 player matches, during FINA

Photo 11.2 Overhead shooting compromised by a defender

world and Olympic Games. Head injuries were first with a 25.6% of the overall count, followed by hand, finger, trunk, and shoulder. More than 57% were contact lesions between players and 13% with an object, so a total of 70% acute injuries. However, this is a very specific cohort of injuries that gives us concern about ruling on contact control, and probably fair play. However, it does not give us a general picture of lesion prevalence.

Regarding surveillance of athletes in longer periods of time, a report on a 4-year epidemiology, in sub-elite water polo players [9] showed that shoulders are the more affected (25%) followed by thoracic, lumbar area (17%) followed by hand, finger, wrists, then by hip, groin, knee, and elbow. This difference is explained by the nature of the sport that may have a typical training week, with up to five water polo-specific sessions, two to three swimming-specific sessions and three weight training sessions [3]. Playing needs high training loads that put overuse lesions always near.

11.3 Sports-Specific Illness

Water polo is prone to the same illnesses of any aquatic sports such as infectious respiratory or gastrointestinal tract, but usually, otitis and allergy/skin issues are the most common. Eye irritation is also very common because the use of googles is not allowed [6].

11.4 Head Injuries

Water polo is a physical sport with a great amount of contact between players and minimal protective equipment. According to the Federation International of Natation (FINA), only a swimming cap with malleable ear protectors may be worn [1].

The head and face are the most commonly injured body parts, usually as a result of physical contact [10, 11]. Contusions and lacerations are common, as well as eye injuries [12]. There also is a risk of fracture to facial or orbital bones.

There are three main categories of eye injuries seen in water polo: corneal abrasions, hyphema, and blow out fractures [13, 14].

Traumatic eardrum perforation is also common despite the use of ear protectors [15]. This typically occurs when a cupped palm slaps the side of the head, causing a sudden rise in pressure in the ear canal [10]. The use of molded earplugs, and swim caps can help these players continue to train and play while minimizing water exposure and risk of infection [10, 15].

According to the International Dental Federation (FDI) water polo is classified as a medium-risk sport for dental injury; however, according to one survey, only 7.7% of athletes wear mouth guards [16]. Nearly 50% of those players reported having witnessed a water polo-related dental injury and 21% reported that they had suffered a dental injury from the sport, most commonly a tooth fracture. In another study, a survey of 415 Swiss water polo players reported that 21% of all respondents had suffered a dental injury during play over their career [17].

11.5 Concussion in Water Polo

Water polo is a highly physical contact sport. The incidence and prevalence of concussion in the sport are not well known.

Concussions are of particular concern in water polo, with up to 36% of players reporting a history of prior concussion on a recent survey of USA Water Polo members and an average of more than two concussions per person. Goalkeepers are again at particularly high risk, with 47% reporting at least one prior concussion. Patterns of head impacts that goalies suffer are quite different than those reported by other positions, as most head impacts come from the ball rather than from player contact. In contrast, other position players report that head impacts result from a combination of the ball and hits by other players [18].

The average number of reported concussions increase with higher competition level and the number of years played [18]. An investigation of collegiate varsity men's water polo found that a single team sustained an average 18.4 head impacts per game [19].

Cecchi et al. [20] in a collegiate club water polo team were monitored during one season of intercollegiate competition. Smart Impact Monitor (SIM-G) sensors (Triax Technologies; Norwalk, CT) found that men were impacted more frequently at the back of the head, relative to the front, right, or top and by the opposing player's limb or torso, relative to their head or the ball. Women were impacted more frequently at the back of the head.

A growing body of evidence suggests that chronic, sport-related head impact exposure can impair brain functional integration and brain structure and function. In a study using cap-worn inertial sensors to measure the frequency and magnitude of head impacts sustained by 18 intercollegiate water polo athletes monitored over a single season of play, report that the frequency and magnitude of head impacts sustained during a season of water polo competition were strongly associated with changes in whole-brain functional connectivity, particularly a pattern of slow-wave synchrony associated with a loss of inhibitory control [21].

11.6 Shoulder Injuries

Water polo is an extremely demanding sport that involves several conditions that could lead to a shoulder injury. Moreover, the strokes are different from the ones of a freestyle swimmer. The statistics on shoulder damage are challenging due to its variability.

Regarding shoulder injuries, complete rolling of the body is not possible because players have to carry the ball in front or see the field; therefore, they need to put their head out of water frequently, doing abduction, high elbow position usually in a short explosive burst of speed, differing from regular swimmers' kinematics.

Overhead throwing also has different kinematics. The ball is bigger and heavier. Speed is generated with a kinematic chain suspended in an eggbeater boost kick which is no match to the counterreaction of a baseball pitch (Photo 11.3). Trunk moves from hyperextension to 20° of flexion and lateralization, with shoulder behind the body, in maximal external rotation allowing releasing the ball while giving height [22]. The speed at release shows WP with lower speed than baseball (16.5 ms^{-1} vs. 33 ms^{-1}) [23, 24], but still, reach the speed of 70 km/h [6]. Any shooting could be blocked, creating additional challenges. For shooters, contact is legal in any moment of the throw, and may cause injuries [10] such as SLAPS or instability. For defenders, due to the blocking position, the shoulder that is in a fully extended forward position is usually moved backwards [6] and may result also in these injuries (Photo 11.4).

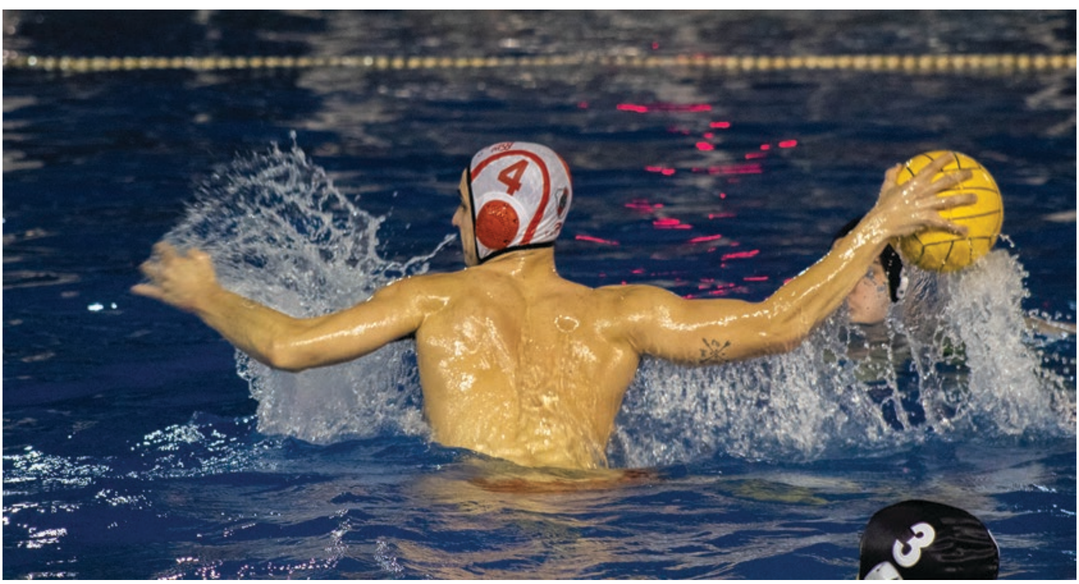

Photo 11.3 Player suspended in an eggbeater boost kick while preparing to shoot

Photo 11.4 Defender in a blocking position

Throwing could be a 7-m fast goal shoot or a 20-m precision passing throw [24].

Players have an increased bilateral external rotation usually find in swimmers, and a unilateral decreased internal rotation deficit found in throwers [3, 25]. Internal rotation vs. external rotation ratio strength shows the usual throwers imbalance [26, 27].

This explains abnormalities found in ultrasonography [28] and MRI in virtually any players although only 29% symptomatic [29]. A study performed by Galluccio et al. in 2017 [28] analyzed by ultrasound shoulder injuries in 42 players. This study found only 4 players with no shoulder modifications. When both shoulders were analyzed, the most common bilateral findings were tendinopathy of the supraspinatus (38.10%) and in second place subscapularis tendinopathy (30.95%). Isolated analysis of the throwing shoulder supraspinatus tear (21.43%) and supraspinatus impingement (21.43%) are the most frequent findings followed by supraspinatus tendinopathy (19.05%).

In MRI, reported alterations are posterior superior labrum, subscapularis tearing, and tendinopathy also in infraspinatus tendons.

Statistics about shoulder injuries are somewhat confusing if we refer to injury reported water polo lesions during FINA WORLD Championships and Olympic Games [7] shoulder injuries were in fourth place (11%). However, this is in a contact acute setting (70%). But if we take injuries as part of a more prolonged time, and not only during competitions, shoulder injuries count up to 51% of total ones in a year period [26] or 24% in a 13-year period, reaching up to 80% in older reports [30].

Wheeler et al. report shoulder "soreness" that is found in all players with a media of 2.9 in a 10-points scale, (explained by the total volume of shooting or less resting during squad selection and team game-based training camps) [31], this not to be counted as an injury report, but is an alarm to be taken to keep players playing.

Prevention should be paramount. However, it is not well defined in what is to be done.

In season shoulder risk is well defined in baseball with less IR and less ER strength in the off season, as a predictor in on season injuries [32, 33] and is one of the key points to see in water polo, but no so well defined [24]. As stated, the volume of shooting and rest intervals could be other. Besides that, overall training efforts concerning swimming, eggbeater kicks [34], core, scapular balance are also areas of interest [22, 24].

11.7 Elbow Injuries

The elbow is a complex joint composed of three bones, the humeroulnar, humeroradial, and proximal radioulnar joint. First two are in charge of flexion and extension of the elbow, and the radioulnar joint is responsible for pronosupination. Joint stability is given by huge bony congruence and its ligament complex. Stabilizers divide into *statics* (articular surfaces, joint capsule, and ligaments) and *dynamics* (biceps muscle, triceps, anterior brachial, brachioradialis, epicondyles, and epitrochlear muscles) [35].

Elbow injuries are a diagnostic and therapeutic challenge [36]. Its frequency [7] is about 6.6% of total water polo injuries. Most common are overuse injuries, ulnar collateral ligament tear (valgus instability), and osteochondritis dissecans (OCD) [36].

Ulnar collateral ligament lesions can be produced by an acute trauma in goalkeepers, or in blocking ball attempts, or as a result of repetitive microtrauma.

In water polo, the player who is throwing the ball is not standing on a fixed support or firm land; therefore, his upper limb is responsible for generating a big part of the necessary forces to execute the throwing. Moreover, the size of the ball is bigger and heavier than other sports, so this increases elbow stress [30]. The throwing of the ball involves four phases: preparation, cocking, acceleration and follow through. During the cocking and acceleration phases, forces generated in valgus exceed the intrinsic tensile stress of ulnar ligament and may cause repetitive microtears (Photo 11.5). It could result in an unstable medial ligament if these repetitive microtears were not treated adequately. Pain is located on the medial side and can be exacerbated by applying valgus with the elbow in 30° of flexion and the forearm pronated [30].

Photo 11.5 Valgus requirements of the elbow

Dominant extremity repetitive valgus stress might be the main cause to develop an osteochondritis dissecans (OCD). It manifests clinically as pain and edema and mechanic block of the joint [36]. Goalkeepers are likely to suffer OCD after repetitive hyperextension and valgus trauma. An X-ray could show fragmentation with demarcating sclerosis and formation of loose bodies. MRI can detect in more early stages the presence of OCD so it should be part of early screening [37].

Throwing technique is an essential part of the handle and prevention of these injuries as well as the training loads. Flexors and pronator muscles strength should be enhanced and the eccentric action of the biceps must also be improved. Improving the eggbeater efficiency and the timing of the shoot to get a more elevated position of the elbow with respect to the body (90–110° of abduction) are essential components in the management of this lesion prevention [3].

11.8 Hand and Wrist

Hand and wrist injuries in water polo player have a frequency of 18% [7]. The wrist has a function to put the hand in space to grab the ball.

Most common wrist injury is the acute injury of the triangular fibrocartilage complex of the carpal ligaments that occur when tackling other players or blocking a shot [3]. Clinical presentation is a pain in the ulnar compartment of the carpal bones, audible click in the same compartment, the player can manifest instability when making pronosupination or when distal ulnar epiphysis is prominent. The necessity of making X-ray to dismiss bone injury an MRI or ArthroMRI will define severity and the therapeutic behavior [38].

Repetitive gliding of tendons of the first dorsal compartment (abductor pollicis longus and extensor pollicis brevis) beneath the sheath and over the radial styloid generates de Quervain tenosynovitis [36], as well as the injury of the extensor carpal ulnaris with a similar mechanism. The initial treatment consists of splinting and rest, infiltrations and, if no progress is seen, surgical liberation of the compartment might be indicated (not so frequent in young athletes).

During blocking, catching the ball, or when fingers get caught up in other players swimsuit finger injuries ocurr. Frequently metacarpophalangeal joint luxation as well as interfalangic dislocation happens in this situation [12]. They must not only be reduced in place but also evaluated with X-ray to dismiss fracture. They can be splinted by themselves or to the next finger.

In the thumb, the most common lesion is the gamekeeper's thumb; it consists of the ulnar collateral ligament of the metacarpophalangeal joint sprain. It occurs as a cause of the large diameter of the ball and the players attempt to grab it with the hand, opening the fingers as much as they can, so the thumb lies in maximum adduction and is liable to suffer a sprain [30] (Photo 11.6).

Photo 11.6 Grabbing a big ball with thumb in max abduction and fingers fully extended

11.9 Lower Extremity Injuries

Lower extremity injuries can be traumatic injuries or injuries related to the eggbeater kick.

The lower extremity trauma can be produced when tackling other player or trying to recover the position of the ball or while swimming to get the position.

The eggbeater kick is used by water polo players to support their body in an elevated position for extended periods and then raise the body out of the water in an explosive action for defending, tackling, passing, or shooting [3].

It is an extremely complex movement and involves the coordination of both inferior limbs. It combines hip flexion, abduction, and internal rotation plus repetitive hip motion. When it is time to shoot, block or tackle, the player makes a boost action to elevate the trunk and get in position to make the action.

In the knee during the eggbeater, the repeated valgus stress and the loads (during training and matches) can produce medial knee pain. It can generate degenerative changes in the medial side of the knee and pain along or over the origin of insertion of the medial collateral ligament (overuse syndrome) [36].

One of the causes of valgus stress is the reduced range of motion in the abduction and internal rotation of the hip [3]. Therefore, the therapeutic plan not only has to include the knee pain and its study but also a complete reeducation of the eggbeater and full range of motion in the hip.

The hip and groin pain are also a common overuse pain in water polo players. The eggbeater can cause chronic adductor-related groin pain and acute adductor muscle strain. To prevent these injuries, the medical team, as well as the trainers, have to maintain range of motion in the hip and workout the power of the abductor, core stability, and internal and external rotators of the hip.

The optimization of this movement is a vital aspect of the injury prevention not only in the lower extremities (knee and hip) but also in the elbow and the shoulder because of the position of the body over the water at the time of making sports-specific movements.

Take-Home Messages

- Water polo is a rough collision sport.
- A concussion is a major concern. Its surveillance is of paramount importance.
- Overuse shoulder injuries are common. Internal rotation deficits and IR/ER strength balance are critical to avoid them. Goaling volume needs supervision.
- Eggbeater kick may predispose to the groin and hip pain, also to medial knee pain. Training is essential.
- Medial elbow injuries are common. Elbow mechanics while throwing are key to avoid them.

References

1. Federation Internationale De Natation (FINA). Water polo rules; 2013.
2. FINA. FINA and aquatics: a brief history. http://www.fina.org/content/fina-aquaticsbit-history. Accessed 7 Jan 2018.
3. Mosler A, Whiteley R. Keeping the water polo player out of the clinic and in the water. Aspetar. 2015;4:434. https://www.aspetar.com/journal/viewarticle.aspx?id=258#.Xxqxb1VKjIU.
4. Spittler J, Keeling J. Water polo injuries and training methods. Curr Sports Med Rep. 2016;15(6):410–6.
5. Nichols AW. Medical care of the aquatics athlete. Curr Sports Med Rep. 2015;14:389–96.
6. Stromberg JD. Care of water polo players. Curr Sports Med Rep. 2017;16(5):363–9.
7. Mountjoy M, Miller J, Junge A. Analysis of water polo injuries during 8904 player matches at FINA World Championships and Olympic games to make the sport safer. Br J Sports Med. 2018;2018:099349.
8. Lozovina V, Pavièiæ L. Anthropometric changes in elite male water polo players: survey in 1980 and 1995. Croat Med J. 2004;45:202–5.
9. Hams A, Evans K, Adams R, Waddington G, Witchalls J. Epidemiology of shoulder injury in sub-elite level water polo players. Phys Ther Sport. 2019;35:127–32.
10. Franic M, Ivkovic A, Rudic R. Injuries in water polo. Croat Med J. 2007;48:281–8.
11. Mountjoy M, Junge A, Alonso JM, et al. Sports injuries and illnesses in the 2009 FINA World Championships (Aquatics). Br J Sports Med. 2010;44:522–7.
12. Brooks JM. Injuries in water polo. Clin Sports Med. 1999;18:313–9.
13. Barr A, Baines PS, Desai P, MacEwen CJ. Ocular sports injuries: the current picture. Br J Sports Med. 2000;34:456–8.
14. Rodriguez JO, Lavina AM, Agarwal A. Prevention and treatment of common eye injuries in sports. Am Fam Physician. 2003;67:1481–8.
15. Dominguez RH. Water polo injuries. Clin Sports Med. 1986;5:169–83.
16. Hersberger S, Krastl G, Kuhl S, Filippi A. Dental injuries in water polo, a survey of players in Switzerland. Dent Traumatol. 2012;28:287–90.
17. Hersberger S, Krastl G, Kühl S, Fillipi A. Dental injuries in water polo, a survey of players in Switzerland. Dent Traumatol. 2012;28:287–90.
18. Blumenfeld RS, Winsell JC, Hicks JW, Small SL. The epidemiology of sports-related head injury and concussion in water polo. Front Neurol. 2016;24(7):98.
19. Cecchi NJ, Monroe DC, Metal FG. Head impacts sustained by male collegiate water polo athletes. PLoS One. 2019;14(5):e0216369.
20. Cecchi NJ, Monroe DC, Phreaner JJ, Small SL, Hicks JW. Patterns of head impact exposure in men's and women's collegiate club water polo. J Sci Med Sport. 2020;23:927. https://doi.org/10.1016/j.jsams.2020.03.008.
21. Monroe DC, Cecchi NJ, Gerges P, Phreaner J, Hicks JW, Small SL. A dose relationship between brain functional connectivity and cumulative head impact exposure in collegiate water polo players. Front Neurol. 2020;11:838–13.
22. Miller AH, Evans K, Adams R, Waddington G, Witchalls J. Shoulder injury in water polo: a systematic review of incidence and intrinsic risk factors. J Sci Med Sport. 2018;21(4):368–77.
23. Feltner ME, Taylor G. Three-dimensional kinetics of the shoulder, elbow, and wrist during a penalty throw in water polo. J Appl Biomech. 1997;13:347–72.
24. Webster MJ, Morris ME, Galna B. Shoulder pain in water polo: a systematic review of the literature. J Sci Med Sport. 2009;12(1):3–11.
25. Elliott J. Shoulder pain and flexibility in elite water polo players. Physiotherapy. 1993;79(10):693–7.
26. Hams AH, Evans K, Adams R, Waddington G, Witchalls J. Shoulder internal and external rotation strength and prediction of subsequent injury in water polo players. Scand J Med Sci Sports. 2019;29:1414–20.
27. McMaster WC, Long SC, Caiozzo VJ. Isokinetic torque imbalances in the rotator cuff of the elite water polo player. Am J Sports Med. 1991;19(1):72–5.

28. Galluccio F, Bellucci E, Porta F, et al. The water-polo shoulder paradigm: results of ultrasound surveillance at poolside. BMJ Open Sport Exerc Med. 2017;3:e000211.
29. Klein M, Tarantino I, Warschkow R, et al. Specific shoulder pathoanatomy in semiprofessional water polo players: a magnetic resonance imaging study. Orthop J Sports Med. 2014;2(5):232596711453121.
30. Colville JM, Markman BS. Competitive water polo. Clin Sports Med. 1999;18(2):305–12.
31. Wheeler K, Kefford T, Mosler A, Lebedew A, Lyons K. The volume of goal shooting during training can predict shoulder soreness in elite female water polo players. J Sci Med Sport. 2013;16(3):255–8.
32. Byram IR, Bushnell BD, Dugger K, Charron K, Harrell FE, Noonan TJ. Preseason shoulder strength measurements in professional baseball pitchers. Am J Sports Med. 2010;38(7):1375–82.
33. Wilk KE, Andrews JR, Arrigo CA, Keirns MA, Erber DJ. The strength characteristics of internal and external rotator muscles in professional baseball pitchers. Am J Sports Med. 1993;21(1):61–6.
34. Oliveira N, Saunders DH, Sanders RH. The effect of fatigue-induced changes in effbeater-kick kinematics on performance and risk of injury. Int J Sports Physiol Perform. 2016;11:141–5.
35. Varona Jose M, Simone Juan P. Inestabilidad de codo. Rev Asoc Argent Ortop Traumatol. 2006;71:353–61.
36. Franić M, Ivković A, Rudić R. Injuries in waterpolo. Croat Med J. 2007;48:281–8.
37. Rod E, Ivkovic A, Boric I, Jankovic S, Radic A, Hudetz D. Acute hyperextension/valgus trauma to the elbow in top-level adult male water polo goalkeepers: a cause of osteochondritis disecans of the capitellum? Injury. 2013;44:S46–8.
38. Esplugas M, Aixalà Llovet V. Lesiones del complejo del fibrocartílago triangular. Tipos de reparación. Revista Española de Artroscopia y Cirugía Articular. 2014;21(1):14–27.

Part II

Individual Sports

12 Athletics, Sprints, Hurdles, High Jump, Long Jump, Triple Jump, Distance Running

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12.1 Introduction

Athletics, also known as track and field, is a group of sport events including running, jumping, and throwing. Track and field athletics was included in the first modern Olympic Games in 1896, and currently can be classified into four main categories [1]:

- Track events, including sprints (100, 200, and 400 m), middle-distance running (800 and 1500 m), long-distance running (5000 and 10,000 m), hurdling (100 m for women, 110 m for men and 400 m for both), relays (4 × 100 and 4 × 400 m), and 3000 m steeplechase.
- Field events, including long jump, triple jump, high jump, pole vault, shot put and discus, javelin and hammer throw.
- Road events, including marathon, 20-km race walk for women, 20- and 50-km race walks for men.
- Combined events, including heptathlon for women and decathlon for men.

Disciplines like sprint, hurdles, and jumps have a high incidence of acute, traumatic events (e.g., hamstring or calf injuries in the 100 m), while sports like middle- or long-distance running typically predispose to overuse injuries such as bone stress fractures and tendinopathy [2–5]. The risk of injury depends on different biomechanics and technical movements, as well as the implements used, the training workload and the duration of practice [6].

Although each discipline is characterized by different physical, mechanical, technical, and psychological demands, in fact, the practice of track and field carries the risk of injury during both competing and training [5, 7–15].

Despite differences, in fact, all track and field disciplines highly involve the musculoskeletal system, composed by muscle, tendon, bone, cartilage, ligament, and soft tissue. When repeated mechanical loading exceeds the remodeling capability of the structures under stress, together with insufficient recovery and undertrained conditions, an overuse injury may develop [16].

Running disciplines require long periods of repetitive stress on musculoskeletal system, resulting in a high rate of overuse injuries. The field events are characterized by the generation of maximum force in a short time lapse instead,

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with maximal muscle contractions causing high stress [2].

Factors such as athlete's growth and maturation process, anatomical characteristics, muscle-tendon imbalance, history of prior injury, menstrual dysfunction, and psychological aspects should also be taken into account as intrinsic factors. Training workload, competition schedule, resting time, sports-specific characteristics, training environment, and equipment influence as extrinsic factors the development of such injuries [17]. In pole vault, there is also a relative risk of serious head and cervical injuries [18].

12.2 Injury Epidemiology

During elite Athletic Championships, the overall incidence is nearly 130 injuries per 1000 registered athletes [12, 19–21].

Patellar tendinopathy, Achilles tendinopathy, the medial tibial stress syndrome (MTSS), hamstring strains, plantar fasciitis, stress fractures of metatarsal bones, and the tibia and ankle sprains have been reported as the most frequently diagnosed injuries during track and field events [7, 15, 22–27].

It is widely assumed that overuse injuries represent the majority of all injuries in track and field, and they usually affect the lower extremities with rates from 60% to 100% [2, 5, 9, 14, 15, 28, 29].

Musculo-tendinous injuries are usually correlated with explosive events such as sprints and jumps, mostly due to indirect forces on the muscle-tendon junction, which is well-known as a weak point [6].

Thigh strain is the most frequent diagnosis, especially hamstring strain [12, 22]. Several factors play a role in occurrence of such injuries, like hamstring mechanics during the sprint phase [30], strength imbalances [31], flexibility, fatigue, age, ethnicity, and the severity of previous injuries [13, 32].

Other main injuries during such events are lower leg strains, ankle sprains, and trunk muscle cramps, most frequently caused by overuse [8, 11, 12, 19, 22]. Lower back injuries are common as well, especially among sprinters, jumpers, and throwers [6, 10]. This can be explained considering that all track and field disciplines require a good back and abdomen sheathing to effectively transmit the force to the lower extremities [6].

Young athletes have been reported to be at higher injury risks than adults [33], especially in middle- and long-distance running [9]. This particularly concerns the adolescent athletes. The musculoskeletal system of a growing athlete, in fact, is more vulnerable to specific injuries, such as epiphyseal traumatic injuries or apophysis overuse injuries [34–36]. Nevertheless, still very little is known about sports injury and complaint profiles of adolescent elite athletes [37].

During an athletic season, only a small percentage of injuries can be classified as traumatic [5, 9]. Considering the overall incidence, the relationship between rate and training loads seems to be directly proportional [9, 15, 38].

Furthermore, it has been reported that a higher rate of injuries occurs during training (60–91%) than during competition (9–30%) [2, 4, 10, 39]. Most of the season time, in fact, is spent in training rather than competition [10, 39].

During athletic championships, athletes competing in disciplines like combined events, steeplechase, and middle- and long-distance running are at higher risk of developing overuse injuries above all [11, 12, 19, 22]. The rate of in-competition injuries has been reported as up to three times higher than the rate of training disorders [22]. During championships, in fact, athletes spend less time in training than during season [28]. Furthermore, during competition the maximum effort required can facilitate the development of injuries, reducing the awareness of a potential disorder at an early stage as well [22].

12.3 Track Disciplines

12.3.1 Sprint

Sprinting requires a huge muscle mass for an explosive lower extremity activity (Fig. 12.1). As a result, the most frequently reported injuries involve the lower limb, in particular hamstring

Fig. 12.1 Sprint finish line

and rectus femoris strains [5, 8–10, 15, 18], as well as Achilles tendon ruptures [9] and/or back injuries [10].

Hamstring injuries are very common among sprinters, especially involving the biceps femoris [40] and are more frequent in males than female athletes [12].

It has been suggested that during both the late swing phase [41] and the early phase of sprinting [42] hamstrings are more prone to develop injury.

Referring to indirect muscle injuries, the eccentric contraction plays a key role in the pathogenesis of muscle strains. Mechanical factors based on overstretching can lead to sarcomeres fibers disruption, vessels lesion, cytoskeletal proteins, and sarcoplasmic reticulum damages [43, 44]. Fast contraction muscle fibers, as well as bi-poliarticular muscles and musculotendinous junctions can be considered as the most susceptible to injuries structures [45].

Achilles tendinopathy is usually correlated with explosive events such as sprints, hurdles, and jumps, but it may develop among middle- and long-distance runners too [2, 28]. It can be considered as the most common overuse injury occurring among sprinters and hurdlers [6]. It has been highlighted how an appropriate loading and adaptation may increase cross-sectional are and tensile strength of the tendons. Conversely, immobilization and inappropriate adaptation may cause tendon degeneration and the development of tendinopathy. Tissue adaptive changes, either physiological or pathological, are in fact influenced by the response of the peripheral nervous system and its messengers to external forces such as mechanical loading to the tendon [46]. Biomechanical causes of the development of Achilles tendinopathy may be intrinsic (such as avascularity, malalignment, hyperpronation, imbalance of the agonist–antagonist muscle action, poor running style, insufficient warm-up and stretching before sports, age and eccentric loading of the Achilles tendon with a dorsiflexed foot) or extrinsic (ground surfaces, excessive increase in sports activity, repetitive mechanical loadings, or old footgear by changing alignment) [47].

When the inflammation involves the peritendinous sheaths without any pathological changes in the tendon itself, the term *Achilles peritendinopathy* is more appropriate [48, 49].

12.3.2 Hurdles

Hurdlers and steeplechasers are frequently injured by the obstacles they must jump over [18]. Steeplechasers, as well as middle- and long-distance runners, show higher risk of injury compared to other disciplines. Despite these, events require lower intensity of exercise, the time spent in training and/or in competition is longer, causing a higher risk of developing overuse injuries [12].

Among both male and female hurdlers, most injuries are located in the thigh (especially as muscle strains), while hip and groin disorders especially affect males, and knee injuries mostly concern females [7].

Focusing on groin pain, it usually develops in sports requiring a combination of sudden and sharp movements involving the hip adductor and the abdominal musculature [50–52]. The main risk factors include adductor muscle weakness, greater hip adductor to abductor strength ratio, sport specificity of training, and the amount of pre-season sport-specific training [53]. Torsion and traction at the insertion of the involved muscle groups can cause functional overuse and repeated microtraumas [54]. The insertional tendinopathy of the adductors and rectus abdominis are the most frequent causes of groin [50].

As mentioned above, Achilles tendinopathy is a common injury above runners, including hurdlers [6]. Risk factors may be intrinsic (such as forefoot or varus deformity, *pes cavus*, leg length discrepancy, limited mobility of the subtalar joint) and extrinsic (excessive overload training, excessive eccentric loading, hard surfaces, poor shock absorption) [55–57].

Activities requiring explosive acceleration, sudden changes in direction, jumping and sprinting predispose to such an injury [58–60].

Considering the insertional Achilles tendinopathy, in particular, muscle-tendon stiffness fatigue-induced or contracture/imbalance of gastrocnemius, soleus, and anterior tibialis muscles increase the stress on the insertional zone of the tendon [58].

12.3.3 Distance Running

Middle- and long-distance runners are usually leaner, have greater endurance requirements, and are more likely to suffer from chronic injuries then athletes participating in other disciplines [3] (Fig. 12.2).

The most frequently diagnosed disorders during long-distance events are in fact patellofemoral syndrome (PFS), medial tibial stress syndrome (MTSS), Achilles tendinopathy, iliotibial band friction syndrome, plantar fasciitis and stress fractures of the metatarsal bones, the sesamoid bone, and the tibia [6, 24, 29, 61, 62] (Figs. 12.3 and 12.4). Less common disorders are ankle sprains, hamstring muscle injury and tendinopathy, gastrocnemius muscle injury, trochanteric bursitis, low back pain, tibial posterior and hip

Fig. 12.2 Three thousand meter steeplechase

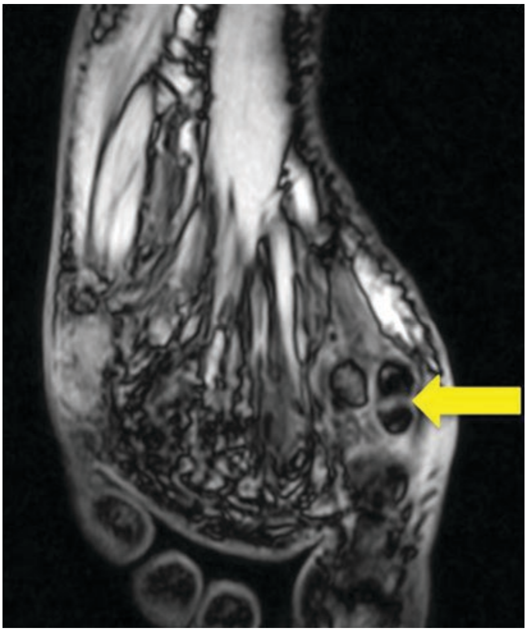

Fig. 12.3 MRI showing a sesamoid stress fracture (yellow arrow)

Fig. 12.4 MRI showing a tibial stress fracture (yellow arrow)

adductor tendinopathy, infrapatellar bursitis, and knee sprain [24].

The overall incidence of injuries may vary from 6.8 to 59 injuries per 1000 h of exposure to running [24]. This large variation is due to differences in the definition of injury, as well as study populations, type of run, and follow-up periods [63].

Acute injuries are rare among these disciplines, mainly consisting of muscle injuries, sprains, or skin lesions [64]. Eighty percent of running disorders are overuse injuries instead, mostly involving the lower extremity and the knee in particular [29].

The MTSS has been reported as the most frequently diagnosed musculoskeletal injury among middle- and long-distance runners [24]. The main pathomechanical process is related to repetitive contraction of the posterior tibial, soleus, and/or flexor digitorum longus muscles during both the landing and the propulsion phases of running. This repetitive process can generate excessive stress on the tibia, resulting in the development of inflammation at the insertion of the periosteal [65–68]. Another cause of MTSS is an inadequate capacity of bone remodeling, due to the repetitive and constant stress on the tibia after both muscle contraction and vertical ground reaction during the landing phase [66, 69]. Furthermore, several risk factors for the development of MTSS have been recently proposed: female sex, high weight, high navicular drop, previous running injuries, and great hip external rotation with the flexed hip are some examples [70].

12.4 Field Disciplines

12.4.1 Jumps

Jumping events require a running approach, thus the most frequent jumping-related injuries are usually seen among runners too [5].

Among the field events in athletics, horizontal jumps (long and triple jump), and vertical jumps (high jump and pole vault) involve the production of maximum force in a short period of time (Fig. 12.5).

The resulting maximal muscle contractions cause high stress on several body districts. Disciplines involving plyometrics, such as jumping and landing, are in fact frequently associated with musculoskeletal injuries of different locations and types [7, 71].

Most of the disorders affect the lower extremities, in particular the thigh region [5, 8–10]. The knee, the ankle and the hip joints are frequently involved too [7].

Patellar tendinopathy, also called the jumper's knee, typically affects athletes involved in repetitive jumps and with a high explosive strength required [72] (Fig. 12.6). The ground reaction force in a long jump take-off, in fact, can correspond up to ten times the body weight [73], and the resulting forces through the extensor tendons are proportional to the ground reaction force. Therefore, a correlation between the loading pattern of the knee extensors and the prevalence of jumper's knee may be considered [74].

The main risk factors for the development of patello-femoral pain (PFP) may be classified as anatomical (such as enhanced femoral anteversion, trochlear dysplasia, patella alta and baja, excessive foot pronation) and biomechanical (muscle tightness or weakness, generalized joint laxity, and gait abnormalities) [72, 75].

Pole vault can be considered as the highest risk jump event [13]. It also represents the track and field discipline with the highest risk of mortality, mainly due to the landing phase directly onto the head or neck [18]. Head injuries, spinal fractures, brain stem injuries, and pneumothorax are some examples of possible traumatic events experienced by pole vaulters [76]. Because of such potential injuries, it is imperative for the medical personnel stationed by the vault field to be highly versed in acute traumatic head and neck injury management [3].

Fig. 12.5 High jump

12.5 Prevention

Several approaches do exist to effectively prevent track and field injuries [6].

One strategy focuses on the specific characteristics and risk factors of the most common injuries: the better you know the problem, the better you can manage it.

Considering hamstring strain as one of the most frequent injuries among track and field athletes, for example, factors such as hamstring mechanics during sprinting, strength imbalances, athlete's flexibility, effort, age or ethnicity, and severity of previous disorders play an important role in developing such an injury [30–32, 77].

Ankle sprains development and gravity are influenced by the severity of previous injuries, ankle proprioceptive deficits, altered neuromuscular control, postural instability, and strength deficiency as well [78–80].

Fig. 12.6 MRI showing patellar insertional tendinopathy (yellow arrow)

Another prevention strategy focuses on the track and field disciplines with higher injury risks, such as combined events, middle- and long-distance running, pole vault, and hurdles [4, 11, 12, 14, 19, 81, 82]. Thoroughly knowing the biomechanics and metabolic needs of these disciplines may help preventing the related most common injuries [6, 12].

Above all the existing strategies, promptly treating acute injuries, reducing overtraining risks, improving strengthening and recovery schemes, and managing the first episode of injury with the appropriate treatment and rehabilitation can be considered as the milestones for an effective prevention [5, 6, 12].

Also the technical knowledge of the discipline an athlete practices is paramount for preventing injuries. This can be explained when considering the higher incidence of injuries during combined events. Besides the more intensive training load required to master several different disciplines, it should be considered that the athlete might not be as experienced and prepared in each sport as athletes competing in a single one [22].

Conclusions

Athletics has a great historical background and is one of the most fascinating individual sports coupling competition with a constant research of self-improvement. Top-level activity carries a significant risk of acute traumatic lesions or overuse injuries. The relationship between rate of injuries and training loads seems to be directly proportional.

Young athletes are at higher injury risks than adults, especially in middle- and long-distance running. The musculoskeletal system of a growing athlete is more vulnerable to specific injuries, and careful training and well-planned competition programs are mandatory.

Sprint and hurdles show a higher risk of hamstring and rectus femoris strains, as well as Achilles tendon ruptures. Long-distance events are more prone to anterior knee pain, medial tibial stress syndrome, Achilles tendinopathy, iliotibial band friction syndrome, plantar fasciitis, and stress fractures. Anterior knee pain and patellar tendinopathy affect more frequently jumpers. Pole vault can be considered as the highest risk jump event being the track and field discipline with the highest risk of mortality, mainly due to the landing phase directly onto the head or neck. Careful selection of the athlete and training in an appropriate environment are the best prevention strategies, as well as a prompt and accurate management of acute or overuse injuries is the best way to safely resume competition at the same or hopefully even higher level.

References

1. Rauh MJ, Macera C. Athletics. In: Epidemiology of injury in Olympic sports. Hoboken, NJ: Wiley; 2009. p. 26–48.
2. Zemper ED. Track and field injuries. Med Sport Sci. 2005;48:138–51.
3. Thing J, Scheer V. Track and field. In: Sports-related fractures, dislocations and trauma. Advanced on- and off-field management. Cham: Springer Nature; 2020. p. 955–8.
4. Edouard P, Morel N. Suivi prospectif des blessures en athlétisme. Étude pilote sur deux clubs durant une saison. Sci Sports. 2010;25:272–6.
5. Bennell KL, Crossley K. Musculoskeletal injuries in track and field: incidence, distribution and risk factors. Aust J Sci Med Sport. 1996;28:69–75.
6. Edouard P, Alonso J-M. Epidemiology of track and field injuries. New Stud Athletics. 2013;28:85–92.
7. Edouard P, Navarro L, Branco P, Gremeaux V, Timpka T, Junge A. Injury frequency and characteristics (location, type, cause and severity) differed significantly among athletics ('track and field') disciplines during 14 international championships (2007–2018): implications for medical service planning. Br J Sports Med. 2020;54:159–67.
8. Jacobsson J, Timpka T, Kowalski J, Nilsson S, Ekberg J, Renström P. Prevalence of musculoskeletal injuries in Swedish elite track and field athletes. Am J Sports Med. 2012;40:163–9.
9. Jacobsson J, Timpka T, Kowalski J, Nilsson S, Ekberg J, Dahlström Ö, et al. Injury patterns in Swedish elite athletics: annual incidence, injury types and risk factors. Br J Sports Med. 2013;47:941–52.
10. D'Souza D. Track and field athletics injuries—a one-year survey. Br J Sports Med. 1994;28:197–202.
11. Alonso JM, Junge A, Renström P, Engebretsen L, Mountjoy M, Dvorak J. Sports injuries surveillance during the 2007 IAAF World Athletics Championships. Clin J Sport Med. 2009;19:26–32.
12. Alonso J-M, Edouard P, Fischetto G, Adams B, Depiesse F, Mountjoy M. Determination of future prevention strategies in elite track and field: analysis of Daegu 2011 IAAF Championships injuries and illnesses surveillance. Br J Sports Med. 2012;46:505–14.
13. Opar D, Drezner J, Shield A, Williams M, Webner D, Sennett B, et al. Acute injuries in track and field athletes: a 3-year observational study at the Penn Relays Carnival with epidemiology and medical coverage implications. Am J Sports Med. 2015;43:816–22.
14. Watson MD, DiMartino PP. Incidence of injuries in high school track and field athletes and its relation to performance ability. Am J Sports Med. 1987;15:251–4.
15. Lysholm J, Wiklander J. Injuries in runners. Am J Sports Med. 1987;15:168–71.
16. Aicale R, Tarantino D, Maffulli N. Overuse injuries in sport: a comprehensive overview. J Orthop Surg. 2018;13:309.
17. Farpour-Lambert N. Adolescent athletes. In: Injury and health risk management in sports. A guide to decision making. Cham: Springer Nature; 2020. p. 7–16.
18. Pendergraph B, Ko B, Zamora J, Bass E. Medical coverage for track and field events. Curr Sports Med Rep. 2005;4:150–3.
19. Alonso J-M, Tscholl PM, Engebretsen L, Mountjoy M, Dvorak J, Junge A. Occurrence of injuries and illnesses during the 2009 IAAF World Athletics Championships. Br J Sports Med. 2010;44:1100–5.
20. Soligard T, Steffen K, Palmer D, Alonso JM, Bahr R, Lopes AD, et al. Sports injury and illness incidence in the Rio de Janeiro 2016 Olympic Summer Games: a prospective study of 11274 athletes from 207 countries. Br J Sports Med. 2017;51:1265–71.
21. Soligard T, Palmer D, Steffen K, Lopes AD, Grant M-E, Kim D, et al. Sports injury and illness incidence in the PyeongChang 2018 Olympic Winter Games: a prospective study of 2914 athletes from 92 countries. Br J Sports Med. 2019;53:1085–92.
22. Feddermann-Demont N, Junge A, Edouard P, Branco P, Alonso J-M. Injuries in 13 international Athletics championships between 2007–2012. Br J Sports Med. 2014;48:513–22.
23. Edouard P, Branco P, Alonso J-M. Muscle injury is the principal injury type and hamstring muscle injury is

the first injury diagnosis during top-level international athletics championships between 2007 and 2015. Br J Sports Med. 2016;50:619–30.
24. Lopes AD, Hespanhol Júnior LC, Yeung SS, Costa LOP. What are the main running-related musculoskeletal injuries? A systematic review. Sports Med Auckl NZ. 2012;42:891–905.
25. Pollock N, Dijkstra P, Calder J, Chakraverty R. Plantaris injuries in elite UK track and field athletes over a 4-year period: a retrospective cohort study. Knee Surg Sports Traumatol Arthrosc. 2016;24:2287–92.
26. Figueroa D, Figueroa F, Calvo R. Patellar tendinopathy: diagnosis and treatment. J Am Acad Orthop Surg. 2016;24:e184–92.
27. 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.
28. Ahuja A, Ghosh AK. Pre-Asiad '82 injuries in elite Indian athletes. Br J Sports Med. 1985;19:24–6.
29. van Gent RN, Siem D, van Middelkoop M, van Os AG, Bierma-Zeinstra SMA, Koes BW. Incidence and determinants of lower extremity running injuries in long distance runners: a systematic review. Br J Sports Med. 2007;41:469–80; discussion 480.
30. Schache AG, Dorn TW, Blanch PD, Brown NAT, Pandy MG. Mechanics of the human hamstring muscles during sprinting. Med Sci Sports Exerc. 2012;44:647–58.
31. Yeung SS, Suen AMY, Yeung EW. A prospective cohort study of hamstring injuries in competitive sprinters: preseason muscle imbalance as a possible risk factor. Br J Sports Med. 2009;43:589–94.
32. Malliaropoulos N, Isinkaye T, Tsitas K, Maffulli N. Reinjury after acute posterior thigh muscle injuries in elite track and field athletes. Am J Sports Med. 2011;39:304–10.
33. Carragher P, Rankin A, Edouard P. A one-season prospective study of illnesses acute and overuse injuries in elite youth and junior track and field athletes. Hurdling Chall 2019 IAAF World Championships. Front Media SA. 2020;86–97.
34. Caine D, DiFiori J, Maffulli N. Physeal injuries in children's and youth sports: reasons for concern? Br J Sports Med. 2006;40:749–60.
35. Engebretsen L, Steffen K, Bahr R, Broderick C, Dvorak J, Janarv P-M, et al. The International Olympic Committee Consensus statement on age determination in high-level young athletes. Br J Sports Med. 2010;44:476–84.
36. Launay F. Sports-related overuse injuries in children. Orthop Traumatol Surg Res. 2015;101:S139–47.
37. Steffen K, Engebretsen L. More data needed on injury risk among young elite athletes. Br J Sports Med. 2010;44:485–9.
38. Lundberg Zachrisson A, Ivarsson A, Desai P, Karlsson J, Grau S. Athlete availability and incidence of overuse injuries over an athletics season in a cohort of elite Swedish athletics athletes - a prospective study. Inj Epidemiol. 2020;7:16.
39. Edouard P, Morel N, Serra JM, Pruvost J, Oullion R, Depiesse F. Prevention of musculoskeletal injuries in track and field. Review of epidemiological data. Sci Sports. 2011;26:307–15.
40. Malliaropoulos N, Papacostas E, Kiritsi O, Papalada A, Gougoulias N, Maffulli N. Posterior thigh muscle injuries in elite track and field athletes. Am J Sports Med. 2010;38:1813–9.
41. Chumanov ES, Schache AG, Heiderscheit BC, Thelen DG. Hamstrings are most susceptible to injury during the late swing phase of sprinting. Br J Sports Med. 2012;46:90.
42. Orchard JW. Hamstrings are most susceptible to injury during the early stance phase of sprinting. Br J Sports Med. 2012;46:88–9.
43. Morgan DL, Allen DG. Early events in stretch-induced muscle damage. J Appl Physiol. 1999;87:2007–15.
44. Proske U, Morgan DL. Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. J Physiol. 2001;537:333–45.
45. Potvin JR. Effects of muscle kinematics on surface EMG amplitude and frequency during fatiguing dynamic contractions. J Appl Physiol. 1997;82:144–51.
46. Ackermann PW, Longo UG, Maffulli N. Aetiology of tendinopathy of the Achilles tendon: mechano-neuro-biological interactions. In: Achilles tendinopathy: current concepts. London: DJO Publications; 2010. p. 15–23.
47. Rosso C, Valderrabano J. Biomechanics of the Achilles tendon. In: Achilles tendinopathy: current concepts. London: DJO Publications; 2010. p. 25–34.
48. Canata GL, Casale V. Endoscopic Achilles tenolysis. In: EFOST surgical techniques in sports medicine foot and ankle surgery. London: JP Medical Limited; 2015. p. 95–100.
49. van Dijk CN, van Sterkenburg MN, Wiegerinck JI, Karlsson J, Maffulli N. Terminology for Achilles tendon related disorders. Knee Surg Sports Traumatol Arthrosc. 2011;19:835–41.
50. Valent A, Frizziero A, Bressan S, Zanella E, Giannotti E, Masiero S. Insertional tendinopathy of the adductors and rectus abdominis in athletes: a review. Muscles Ligaments Tendons J. 2012;2:142–8.
51. Ellsworth AA, Zoland MP, Tyler TF. Athletic pubalgia and associated rehabilitation. Int J Sports Phys Ther. 2014;9:774–84.
52. Paajanen H, Ristolainen L, Turunen H, Kujala UM. Prevalence and etiological factors of sport-related groin injuries in top-level soccer compared to non-contact sports. Arch Orthop Trauma Surg. 2011;131:261–6.
53. Maffey L, Emery C. What are the risk factors for groin strain injury in sport? A systematic review of the literature. Sports Med Auckl NZ. 2007;37:881–94.
54. Estwanik JJ, Sloane B, Rosenberg MA. Groin strain and other possible causes of groin pain. Phys Sportsmed. 1990;18:54–65.

55. Chimenti RL, Cychosz CC, Hall MM, Phisitkul P. Current concepts review update: insertional Achilles tendinopathy. Foot Ankle Int. 2017;38:1160–9.
56. Tuite DJ, Renström PA, O'Brien M. The aging tendon. Scand J Med Sci Sports. 1997;7:72–7.
57. Li H-Y, Hua Y-H. Achilles tendinopathy: current concepts about the basic science and clinical treatments. Biomed Res Int. 2016;2016:6492597.
58. Jacxsens M, Weisskopf L, Valderrabano V, Rosso C. Achilles tendon. In: Foot and ankle sports orthopaedics. New York: Springer; 2017. p. 187–99.
59. Järvinen TAH, Kannus P, Maffulli N, Khan KM. Achilles tendon disorders: etiology and epidemiology. Foot Ankle Clin. 2005;10:255–66.
60. Möller A, Astron M, Westlin N. Increasing incidence of Achilles tendon rupture. Acta Orthop Scand. 1996;67:479–81.
61. Wen DY. Risk factors for overuse injuries in runners. Curr Sports Med Rep. 2007;6:307–13.
62. Taunton JE, Ryan MB, Clement DB, McKenzie DC, Lloyd-Smith DR, Zumbo BD. A retrospective case-control analysis of 2002 running injuries. Br J Sports Med. 2002;36:95–101.
63. Hoeberigs JH. Factors related to the incidence of running injuries. A review. Sports Med Auckl NZ. 1992;13:408–22.
64. van der Worp MP, ten Haaf DSM, van Cingel R, de Wijer A, Nijhuis-van der Sanden MWG, Staal JB. Injuries in runners; a systematic review on risk factors and sex differences. PLoS One. 2015;10:e0114937.
65. Beck BR, Osternig LR. Medial tibial stress syndrome. The location of muscles in the leg in relation to symptoms. J Bone Joint Surg Am. 1994;76:1057–61.
66. Craig DI. Medial tibial stress syndrome: evidence-based prevention. J Athl Train. 2008;43:316–8.
67. Michael RH, Holder LE. The soleus syndrome. A cause of medial tibial stress (shin splints). Am J Sports Med. 1985;13:87–94.
68. Moen MH, Tol JL, Weir A, Steunebrink M, De Winter TC. Medial tibial stress syndrome: a critical review. Sports Med Auckl NZ. 2009;39:523–46.
69. Mubarak SJ, Gould RN, Lee YF, Schmidt DA, Hargens AR. The medial tibial stress syndrome. A cause of shin splints. Am J Sports Med. 1982;10:201–5.
70. Reinking MF, Austin TM, Richter RR, Krieger MM. Medial tibial stress syndrome in active individuals: a systematic review and meta-analysis of risk factors. Sports Health. 2017;9:252–61.
71. Canata GL, Pulici L, Pavei G, Casale V. Jumping sports. In: Injury and health risk management in sports. A guide to decision making. Cham: Springer Nature; 2020. p. 503–8.
72. Visnes H, Aandahl HÅ, Bahr R. Jumper's knee paradox—jumping ability is a risk factor for developing jumper's knee: a 5-year prospective study. Br J Sports Med. 2013;47:503–7.
73. McNitt-Gray JL. Musculoskeletal loading during landing. In: The encyclopedia of sports medicine. Oxford: Blackwell Scientific; 2000. p. 523–49.
74. 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. 2005;33:561–7.
75. Davis IS, Powers CM. Patellofemoral pain syndrome: proximal, distal, and local factors, an international retreat, April 30–May 2, 2009, Fells Point, Baltimore, MD. J Orthop Sports Phys Ther. 2010;40:A1–16.
76. Boden BP, Boden MG, Peter RG, Mueller FO, Johnson JE. Catastrophic injuries in pole vaulters: a prospective 9-year follow-up study. Am J Sports Med. 2012;40:1488–94.
77. Opar DA, Williams MD, Shield AJ. Hamstring strain injuries: factors that lead to injury and re-injury. Sports Med Auckl NZ. 2012;42:209–26.
78. Malliaropoulos N, Ntessalen M, Papacostas E, Longo UG, Maffulli N. Reinjury after acute lateral ankle sprains in elite track and field athletes. Am J Sports Med. 2009;37:1755–61.
79. Holmes A, Delahunt E. Treatment of common deficits associated with chronic ankle instability. Sports Med Auckl NZ. 2009;39:207–24.
80. Edouard P, Chatard J-C, Fourchet F, Collado H, Degache F, Leclair A, et al. Invertor and evertor strength in track and field athletes with functional ankle instability. Isokinet Exerc Sci. 2011;19:91–6.
81. Edouard P, Samozino P, Escudier G, Baldini A, Morin J-B. Injuries in youth and National Combined Events Championships. Int J Sports Med. 2012;33:824–8.
82. Rebella GS, Edwards JO, Greene JJ, Husen MT, Brousseau DC. A prospective study of injury patterns in high school pole vaulters. Am J Sports Med. 2008;36:913–20.

Boxing

13

George Velasco, Nitin K. Sethi, John Neidecker, Joseph Estwanik, and Donald A. Muzzi

13.1 Introduction

Boxing is a popular sport and enjoys a large fan base across the world (Fig. 13.1). Combat sports medicine is a nascent but rapidly evolving field of medicine dedicated to the care of the combat sports athlete. While this chapter will discuss principles applicable to all combat sports, the chapter will focus on boxing injuries exclusively. Ringside physicians come from all disciplines of medicine and bring their unique skill set and knowledge to the ringside setting. A good ringside physician should be knowledgeable of all the possible injuries that can be encountered during the course and in the immediate aftermath of a bout and skilled in managing these injuries. Figure 13.2a, b shows the dynamic of boxing combat. This chapter shall give a broad overview of combat sports medicine as it relates to boxing and cover commonly encountered boxing injuries, their timely recognition, and management.

Since there are limited evidence-based medicine studies in combat sports medicine, many of the recommendations in this chapter are based on the collective anecdotal experience of seasoned ringside physicians. The Association of Ringside Physicians (ARP) (https://ringsidearp.org/) is an international non-profit organization dedicated to the medical care and safety of combat sports athletes and is a continual source of expert opinion and evidence-based guidelines on topics unique to combat sports medicine. Certification as an ARP ringside physician is available through an examination jointly administered by the American College of Sports Medicine and the Association of Ringside Physicians.

This chapter addresses boxing injuries that apply to both professional and amateur boxing. Due to differences between the two levels in terms of regulations and the format of competition, differences in the types and prevalence of injuries and risk factors exist between the two. Amateur boxing matches are shorter in time duration (2 minute rounds versus 3 minute rounds) and number of rounds. Headgear is typically worn in

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Fig. 13.1 (**a**) Overhead view and (**b**) Ringside view of the ring during boxing combat. (Photo credit George Velasco MD)

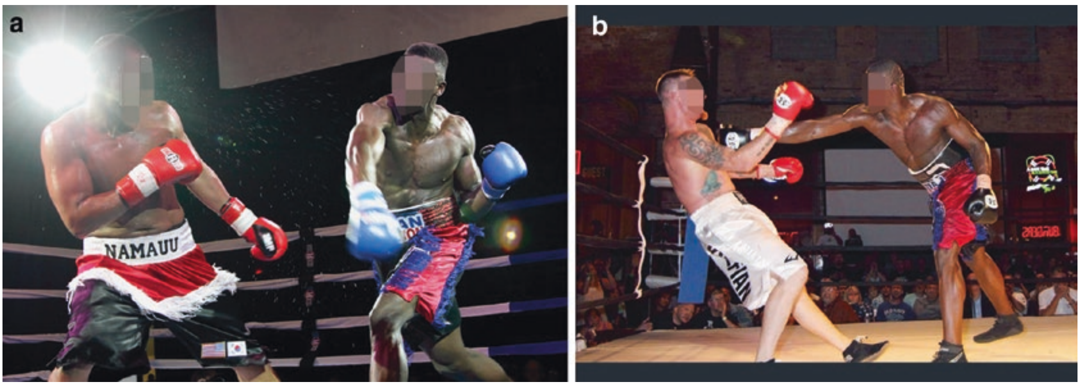

Fig. 13.2 Dynamics of boxing combat. (Photo credit Donald A. Muzzi DMD, MD)

amateur competition, whereas it is not in professional competition. While risks associated with injury exist at both levels of boxing competition, a systematic review of observational studies completed in 2007 by Loosemore et al. concluded that there is no strong evidence to associate chronic traumatic brain injury with amateur boxing [1].

The pre-participation medical testing requirements in amateur boxing vary as compared to those required by commissions regulating professional boxing bouts. Most amateur competitions around the world require only an annual physical exam for participation.

13.2 Medical Clearance of the Professional Boxer Prior to Competition

Evaluation of the professional boxer begins long before he/she actually steps into the ring. Most commissions in the United States and across the world require mandatory tests at the time of initial licensure to fight. Medical requirements for licensure vary from state commission to commission but in general include blood tests, an ophthalmological evaluation, a neuroimaging test, and an electrocardiogram (EKG). This may not be true in some countries, and there may be variations in the specific requirements between jurisdictions [2].

13.2.1 Blood Tests

Most commissions in the United States require tests for hepatitis B, hepatitis C, and HIV both at the time of initial licensure and at varying frequency thereafter. Many commissions also require female boxers to undergo a pregnancy test prior to competition. Some commissions require a complete blood count (CBC) including platelet count at the time of the initial licensure. Additionally, some commissions also require a prothrombin time and international normalized ratio (PT/INR). The goal is to detect any coagulopathy which may make that boxer more susceptible to bleeding. Given the current circumstances commissions may start to require pre-bout testing for the COVID-19 virus. This could also involve other testing for pathogens in the event of any pandemic [2–4].

13.2.2 Ophthalmological Evaluation

Nearly all commissions in the United States require an ophthalmological evaluation by a certified ophthalmologist at the time of the initial licensure and periodically thereafter [2, 5]. The goal is to ensure ophthalmological fitness to fight. Ophthalmological fitness criteria to fight vary from commission to commission but generally include the following [5]:

1. Boxers who have had surgeries which alter the structural integrity of the globe are contraindicated for participation in combat sports. Such surgeries include, but are not limited to: cataract surgery and implantation of intraocular artificial lenses.
2. Boxers who have had radial keratotomy are not permitted to compete.
3. Boxers must not present with "Major Ocular Pathologies" such as: anterior chamber angle abnormalities, glaucoma, lens abnormalities, peripheral retinal abnormalities, macular abnormalities, diplopia or extraocular muscle palsy, or active inflammation.
4. Boxers must have uncorrected visual acuity of 20/200 or better in each eye.
5. Boxers must have corrected visual acuity of 20/40 or better in each eye.

13.2.3 Special Considerations: LASIK

LASIK surgery is associated with a recognized increased risk of corneal injury (flap dislocation) after eye trauma. Due to the recognized increase risk of corneal injury in boxing, boxers are discouraged from undergoing elective LASIK surgery and should be made aware of the potential complications if they decide to participate in the sport. Some commissions do not allow boxers who have undergone LASIK procedure to participate in competition.

13.2.4 Special Considerations: Intraocular Surgery (E.g., Cataract and Retinal Detachment)

In the event of previous eye surgery, these cases should be dealt with an individual case-by-case basis. Medical clearance from an ophthalmologist to fight is required.

13.2.5 Utility of Neuroimaging in Boxing

Neuroimaging serves three distinct roles in the individualized care of the boxer.

1. Neuroimaging prior to licensure helps to identify and/or exclude coincidental or clinically suspected brain lesions which may pose a risk for rupture, bleeding, or other catastrophic brain injury during the course of the bout, representing a step towards personalized medicine and individual risk stratification of a boxer [6, 7].
2. Neuroimaging in the immediate aftermath of a bout primarily serves to rule out acute life-threating traumatic brain injury.
3. Neuroimaging may also be carried out to assess for evidence of structural brain injury/changes which may make a boxer more likely to express late-life neuropsychologic sequelae of brain injury, such as chronic traumatic encephalopathy (CTE), or dementia pugilistica/punch drunk syndrome. Serial neuroimaging could possibly help identify these at-risk athletes if progressive structural and functional changes are present over time. In these athletes, structural and functional neuro-

imaging plays a prognostic role and aids in determining whether the combatant should be allowed to continue to participate in future bouts [6, 7].

The risks for both acute and chronic traumatic brain injury are high in combat sports such as professional boxing, kickboxing, and MMA. Chronic traumatic brain injury has not been substantiated after amateur boxing careers, and there is a distinct difference in risk factors between amateur boxing and professional boxing. With that being said, all participants should be aware of the risks involved and be informed that it cannot be claimed that any amount of boxing is good for the brain [1].

Currently, there are no consensus neuroimaging guidelines for combat sports. Standardizing neuroimaging guidelines for licensure, with the goal of screening for both acute and chronic traumatic brain injury, could assist in protecting the boxer's health and safety, both in the ring, and after their professional careers have ended [6, 7].

13.2.6 Neuroimaging Prior to Licensure

Neuroimaging prior to licensure aids the clinical judgment of supervisory personnel (ringside physicians and commission officials) regarding whether the boxer should be allowed to participate in a future bout. It also helps identify and/or exclude coincidental or clinically suspected brain lesions that may pose a risk for rupture, bleeding, or other catastrophic injuries to the brain should a boxer participate in future bouts. These structural lesions include but are not limited to cerebral aneurysms, arteriovenous malformations, cavernous angiomas, mixed malformations, vein of Galen malformations, large venous malformations, large arachnoid cysts, posterior fossa arachnoid cysts, pituitary macroadenomas, and other space-occupying lesions or tumors. It is important to emphasize that the above represent a heterogeneous group of cerebral and cerebrovascular lesions, which have different natural histories and propensities to bleed. A less urgent but equally important role of neuroimaging prior to licensure is to identify evidence of prior structural injury associated with brain trauma, which may make a boxer more likely to express late-life neuropsychiatric sequelae of brain injury such as chronic traumatic encephalopathy (CTE), dementia pugilistica, chronic post-concussion syndrome, posttraumatic dementia, posttraumatic cognitive impairment, posttraumatic parkinsonism, and chronic posttraumatic headache. This information allows the physician and boxer to make decisions about the boxer's future brain health, implication for, and risk of future neurologic sequelae particularly when the athlete's clinical history is ambiguous [6, 7].

Either a computed tomography (CT) or magnetic resonance imaging (MRI) of the brain is currently included in the process of registering for a license to compete in combat sports in some jurisdictions in the United States and around the world. The imaging specifics and frequency vary widely, with some commissions requiring an MRI brain scan every 1–5 years and others only once, i.e., at the time of licensure. Some commissions do not require any imaging prior to licensure. Other commissions only require imaging if a combatant is of a certain age or deemed "high-risk" [8]. Classification of what makes a combatant "high-risk" also varies considerably from commission to commission, but usually is related to age (usually >40 years), period of inactivity (usually >1 year), and/or recent loss/multiple losses (usually >10). Some commissions detail the required MRI imaging sequences and specifically request sequences such as susceptibility weighted imaging (SWI) and gradient echo imaging (GEI) which have high sensitivity for prior traumatic brain injury (TBI). Some commissions require that all combat sports athletes undergo a magnetic resonance angiogram (MRA) of the brain in addition to an MRI of the brain at the start of their professional career (time of licensure) to primarily exclude any incidental vascular malformations of the brain [6, 7].

13.2.7 Electrocardiogram

Some commissions require an electrocardiogram at the time of the initial licensure to fight. Boxers are usually young healthy individuals with low risk of pre-existing cardiovascular pathology. The goal of the electrocardiogram is to detect that rare combat sports athlete who may harbor a tendency to a malignant cardiac arrhythmia and risk of sudden cardiac death in the ring. In amateur boxing, electrocardiograms are not routinely required. However, if the amateur boxer's history or physical exam suggest the possibility of a disqualifying condition or problem, further testing such as an electrocardiogram will be required [9, 10].

13.3 Evaluation of the Boxer Before, During, and After Competition

13.3.1 Pre-bout Evaluation of the Boxer

Medical evaluation of the boxer can take place at the time of the weigh-in which is usually a day before the fight (referred to as the weigh-in physical) or on the day of the fight itself (referred to as the pre-fight physical). At the time of the weigh-in physical, the ringside physician conducts a complete physical examination, neurological examination, and auscultation of the heart and the lungs. The medical history is reviewed and so are the results of the imaging, electrocardiogram, ophthalmological evaluation, and blood tests. Particular attention is given to the boxer's hydration and cardiovascular status. Boxers sometimes cut weight to fight at the predetermined weight. This weight-cut can be drastic, and the boxer could present with clinical signs of dehydration. Permitting such a boxer to enter the ring can threaten his/her health and safety. During the physical exam, the ringside physician should also look for any unhealed lacerations, active skin infections, and orthopedic injuries [2, 11, 12] (Fig. 13.3a).

13.3.2 Evaluation of a Boxer During a Bout

Close medical supervision of a boxer is advised during a fight (Fig. 13.3b). Boxers have died in the ring or in the immediate aftermath of a bout. The cause of death in these cases is usually neurological and acute subdural hematoma (SDH) is the most common cause of neurological morbidity and mortality. During a bout, the ringside physician should pay close attention and keep a mental tally of the number of head shots which a combatant is taking and the number of knockdowns. If the boxer is noted to exhibit signs of gross motor instability (gait is ataxic, broad based), incoordination, confusion, or disorientation, the fight should be stopped on medical grounds. In some commissions, only a referee can stop a fight, in others the referee or the ringside physician can stop a fight. The ringside physician should consider stopping a fight, if he/she cannot guarantee the health and safety of the boxer. A good medical stoppage is one which is done at the right time (neither too early, certainly

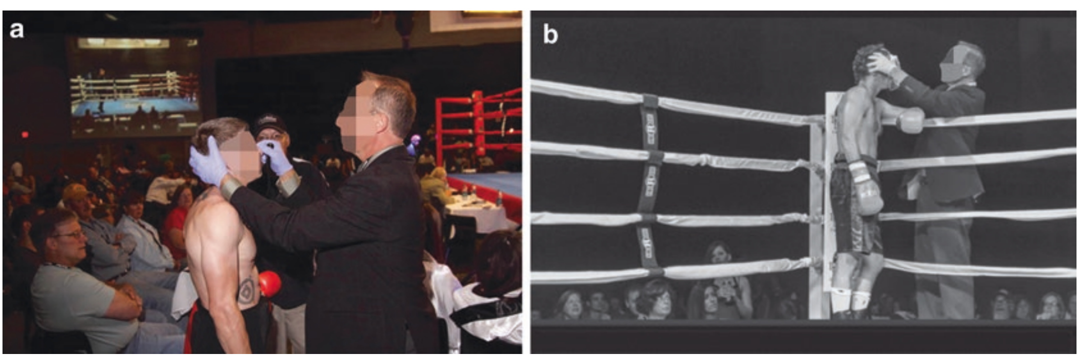

Fig. 13.3 Ringside physician assessing the boxer (**a**) after and (**b**) during a bout. (Photo credit Donald A. Muzzi DMD, MD)

never too late) and for the right indication. A bad medical stoppage is one done at the wrong time (either too early or too late) and for the wrong indication (such as a non-life threatening, non-vision impairing laceration) [13, 14].

13.3.3 Evaluation of a Boxer After a Bout

All boxers should have a post-fight medical evaluation. In the post-fight medical evaluation, the boxer is assessed for any injury he/she may have sustained during the course of the bout. At the time of the post-fight physical, the boxer should also be assessed for any facial fractures, lacerations, orthopedic injuries, or ophthalmic injuries. The neurological evaluation is the most critical aspect of the post-fight evaluation. The prime need for neuroimaging after a bout is to ascertain acute TBI. A CT scan of the head without contrast is the recommended imaging modality due to its relatively high sensitivity and specificity for identifying the presence of blood and bone fracture in the acute setting, its widespread availability, and short acquisition time. Based on the assessment by the ringside physician(s) after the post-fight physical examination, a boxer who is suspected of having sustained a TBI should be transported immediately by ambulance to the designated trauma center for an emergency assessment of possible TBI, an urgent CT scan of the head, and further care as deemed necessary by the trauma center physicians. Trauma center designation is a process outlined and developed at a state or local level, but the recommendation is that boxers in whom acute TBI is suspected be transported to a trauma center with access to specialist neurology and neurosurgery care [12].

13.3.4 Recommended Good Practice Guidelines Regarding Neuroimaging Requirements After a Bout

1. Based on the assessment by the ringside physician(s) after the post-fight physical examination, a boxer who is suspected of having sustained a TBI should be transported immediately by ambulance to the designated Level I trauma center for an emergency assessment of possible TBI/concussion, an urgent CT scan of the head, and further care as deemed necessary by the trauma center physicians. Concern for TBI is raised if the combatant manifests or reports symptoms of headache, blurred vision, double vision, nausea, vomiting, balance, or gait issues after a bout.
2. Any boxer with a Glasgow Coma Scale/Score (GCS) of less than 13 on initial assessment, posttraumatic seizure, focal neurological deficit, and/or greater than one episode of vomiting since the suspected head injury should be urgently transported to the designated Level I trauma center via an on-site ambulance for a CT scan of the head (as per National Institutes of Health and Care Excellence (NICE) guidelines for determining the need for an acute CT scan of the head in adults following a traumatic head injury) [15].

13.4 Injuries in Boxing

13.4.1 Traumatic Brain Injury

Neurological injuries associated with boxing may either be acute or chronic.

Concussion is the most common acute traumatic brain injury (TBI) encountered in boxing. Milder grades of concussions are not associated with loss of consciousness. The recovery is usually rapid with the boxer returning to full consciousness usually in the ring itself. Detecting subtle concussions (sub-concussive injuries) is difficult in a sport where every punch thrown at the head is thrown with the intention of winning by causing a concussion (KO). Concussion or mild TBI may present with a wide spectrum of signs and symptoms. Cognitive functions may be affected and can include disorientation, memory problems, impaired concentration, or loss of consciousness. Behavioral changes include sleep problems, irritability, emotional lability, anxiety, psychomotor retardation, apathy, fatigue, and

Table 13.1 "Red flag" signs and symptoms of serious brain injury [18]

Glasgow Coma Scale <15
Suspected open, depressed or basal skull fracture
Cerebrospinal fluid coming out of nose or ears
Posttraumatic seizure
Focal neurological deficit on post-bout examination
>1 Episode of vomiting since the head injury
Pupillary abnormality
Progressive increase of concussion symptoms
Deterioration of mental status/overall condition

distractibility. Physical manifestations of acute traumatic brain injury include headache, dizziness, vertigo, nausea, vacant stare, impaired skills at their sport, gait unsteadiness, impaired coordination, diplopia, photophobia, hyperacusis, and concussive convulsion/impact seizure. The Glasgow Coma Score (GCS) is insensitive to assessing for milder grades of concussion and may be 15 in a concussed boxer. Any boxer with a GCS score scale of less than 13 should be transferred to the nearest Level I trauma center for evaluation and imaging (usually a CT scan of the head) [16, 17] (Table 13.1).

The possible long-term neurologic result of multiple concussions and sub-concussive injuries includes chronic posttraumatic headache, chronic posttraumatic dizziness, posttraumatic memory impairment, posttraumatic Parkinsonism, and chronic traumatic encephalopathy (CTE). In the boxing medical literature, CTE has also been referred to as dementia pugilistica or *"punch drunk"* syndrome. It is characterized by a constellation of cerebellar and extrapyramidal signs. Chronic TBI may not appear until many years after a professional boxer retires from competitive boxing. It may also occur in an active boxer who has had a very long career with many fights [16, 17, 19].

Head trauma may also result in endocrine dysfunction. Some traumatic brain injuries sustained during boxing may cause hypopituitarism, and it is believed that the known prevalence of TBI-induced hypopituitarism is underestimated. The physician should be suspicious of any neuroendocrine abnormalities in the aftermath of a TBI. Central diabetes insipidus is a complication of traumatic brain injury and could be induced by a minor concussion with only headache and polyuria as the symptoms [20–23].

13.4.2 Further Discussion on Concussion and Return to Sport

The Association of Ringside Physicians has released a consensus statement on concussion management in combat sports which will be discussed here, as it is the most current and evidenced-based consensus on concussion management pertaining to boxing. If a boxer sustains a TKO secondary to blows to the head, it is recommended that he or she be suspended from competition for a minimum of 30 days. It is also recommended that the fighter refrain from sparring for 30 days as well. If a fighter sustains a KO without LOC secondary to blows to the head, it is recommended that he or she be suspended for a minimum of 60 days. It is also recommended that the fighter refrain from sparring for 60 days as well. If a boxer sustains a KO with LOC secondary to blows to the head, it is recommended that he or she be suspended from competition for a minimum of 90 days. It is also recommended that the fighter refrain from sparring for 90 days as well [18].

Boxers may participate in non-contact training and conditioning 1 week after sustaining a concussion or loss via TKO/KO secondary to head strikes provided his or her symptoms are improving and do not increase in severity with activity. A gradual activity progression of increased intensity is recommended, starting with light aerobic activity progressing to a more rigorous/combat sports-specific activity and finally sparring when symptoms have completely resolved. Under no circumstances should a combat sports athlete compete or engage in sparring activity or competition if he or she is experiencing signs and symptoms of concussion. A boxer's suspension should continue until a specialist physician trained in concussion management clears the fighter. Specialist physicians trained in concussion management include neurologists, neurosurgeons, and primary care sports medicine physicians [18].

13.4.3 Lacerations

Facial lacerations occur when the skin is compressed and dragged against the bony surfaces of the skull. Areas most prone to lacerations are the eyebrow area and cheekbone area because of the underlying bony structures (Fig. 13.4). Since the face and the scalp have a high degree of vascularization, lacerations tend to bleed profusely. Lacerations involving the nasolacrimal duct on the medial side of the orbit and the tarsal plates need close attention. Damage to the nasolacrimal duct can have long-term effects if the tear mechanism becomes damaged. Damage to the tarsal plates can permanently affect blinking (Fig. 13.4a, b). Simple lacerations can be sutured at the venue itself. Complex lacerations should be sutured in the hospital by a qualified physician. The boxer who has suffered a laceration is administered a medical suspension to allow time for the laceration to heal [2, 24].

13.4.4 Orthopedic Injuries

Hand injuries are common in combat sports. A common hand injury is the disruption of the metacarpophalangeal joints in the fingers. This is sometimes colloquially called "boxer's knuckle." The most common and most severe case of this is extensor hood disruption. When this occurs, immediate surgery is needed to fix the extensor unit, preventing permanent damage and restoring function. Recurrent, untreated cases can result in disabling traumatic arthritis. Another common hand injury in boxers is disruption and destabilization of the carpometacarpal joints in the fingers. This is also sometimes called "carpal boss." Severe cases require selective carpometacarpal joint arthrodesis. Injuries involving the upper limbs such as shoulder dislocation, rotator cuff tear, metacarpal fractures, and distal biceps tendon rupture are also encountered. The lower limb injuries that are more frequently reported include tears of the ligaments and menisci of the knee, and ankle sprains [17, 25–27].

13.4.5 Ophthalmological Injuries

Boxers may suffer corneal abrasions and retinal detachment both of which need evaluation and management by an ophthalmologist. If an orbital fracture is suspected, the ringside physician should rule out entrapment of the extraocular muscles. Such boxers should be referred to the emergency department for CT scan of the head, face, and orbits, and for management [28, 29].

The American Academy of Ophthalmology has issued recommendations on the prevention of eye injuries in boxing (see Table 13.2).

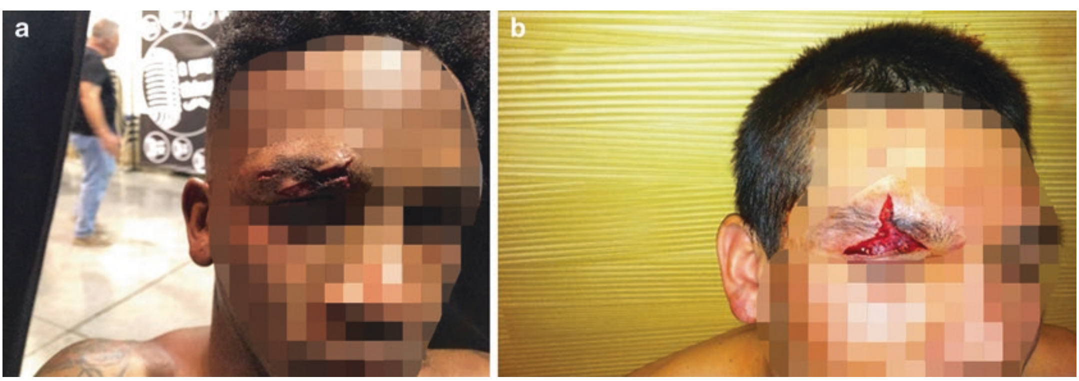

Fig. 13.4 (**a**, **b**) Cuts on the eyelid may damage the tarsal plate and cause permanent damage that may affect blinking. (Photo credit for Donald A. Muzzi DMD, MD)

Table 13.2 Recommendations from the American Academy of Opthalmology

Recommendations from the American Academy of Opthalmology's 1990 policy statement "reforms for the prevention of eye injuries in boxing" [30]
Examination of boxers before licensure and then after 1 year, six bouts, or two losses, or at the stopping of a fight because of an eye injury, or at the discretion of the ringside physician
Mandatory, temporary suspension from sparring or boxing for specific ocular pathology—30 days for a retinal tear and 60 days for a treated retinal detachment, or individualized after consultation with the athletic commission medical advisory board
Minimal visual requirements of 20/40 or better in each eye and a full central field of not less than 30° on each eye
An ophthalmologist required on each state medical boxing advisory board
Thumbless boxing gloves to minimize ocular injuries
A National Registry of Boxers for all amateur and professional boxers in the United States that records bouts, knockouts, and significant ocular injuries
A program for training and recertifying ringside physicians
A uniform safety code

13.4.6 Urological Injuries

Hematuria could result from different causes. Direct trauma to the kidneys or bladder may be responsible for hematuria associated with contact sports such as boxing. Non-traumatic causes such as Nutcracker syndrome must also be considered whenever one sees hematuria. Exercise-induced hematuria is a benign diagnosis of exclusion and the symptoms should disappear within a week. In instances where the boxer's urine is red or brown, as opposed to gross hematuria, one must rule out myoglobinuria due to rhabdomyolysis. A boxer with gross blood in the urine after a fight should be closely evaluated and if needed referred to the trauma center for further management [31].

13.4.7 Oral and Maxillofacial Injuries

Any time there are fractures of the mandible present, there should be concern about potential airway obstruction. Malocclusion or failure of the mouth guard to fit properly may be signs of a mandibular fracture. Edema, bleeding, and muscle movement may all cause upper airway obstruction. Injuries to that area also raise the possibility of a free-floating maxilla bone causing obstruction, dental evulsions, and possible aspiration of dental units or uncontrolled oral-pharyngeal bleeding that may partially obstruct the airway. Such complications underscore why a good mouthpiece is vital to the boxer's health and safety. Urgent situations in facial injuries include open fracture or cerebrospinal fluid rhinorrhea [32, 33].

13.4.8 Head and Neck Injuries

A fractured larynx will manifest with subcutaneous emphysema, hoarseness, a palpable fracture in the midline of the neck, and respiratory distress. Ventilation with the bag mask technique is always the first choice to ventilate a fighter in acute respiratory distress. Intubation of a fighter should be considered only if there is no other option. If intubation is unsuccessful an emergency cricothyrotomy can be considered. Cricothyrotomy should only be performed in an emergency when mask ventilation, bag ventilation, and intubation fail, and mental status and respiratory status continue to deteriorate. In all cases of airway compromise, an athlete should be transported to the hospital without delay. Early airway intervention could stave off the problems that would arise from delayed laryngeal edema or laryngeal hematomas. One must always try to foresee possibilities that happen with the passage of time if there is prolongation of the transport to the hospital. Current non-urgent factors could transition to a true respiratory emergency. The most dangerous orthopedic injury would be a cervical spine fracture from a direct blow to the head or neck. An airway needs to be established in the unconscious apneic boxer. Once an airway is created, ventilate, and oxygenate [32, 33].

Rupture of the tympanic membrane is also possible during sparring or a bout. The boxer may notice sudden unilateral hearing loss in the ear and complain of dizziness. If the rupture is small, it may not be seen on regular otoscopic exam. Immediate evaluation should look for any ear or nasal drainage of CSF. Further evaluation should include hearing tests at baseline as well as during follow-up to assess recovery [34].

13.4.9 Thoracic Injuries

It is important to be vigilant for rib fractures. A rib fracture is not emergent but could interfere with respiration and lead to atelectasis or pneumonia. However, rib fractures that cause splenic rupture or tension pneumothorax create an emergent situation. In the case of a tension pneumothorax, do not wait for transport. Insert a 12 or 14 gauge angiocath needle into the second intercostal space in the midclavicular line on the side of the pneumothorax. For this reason, the physician should have a 12- or 14-gauge needle readily available during the bout. After needle decompression, the fighter should be immediately taken to a trauma center for a chest tube. Commotio cordis can happen from blunt trauma to the chest causing cardiac arrest from arrhythmia. Because of that, there should be emergency medical personnel and automatic external defibrillators present at all boxing events [9, 32].

13.4.10 Abdominal Injuries

As far as abdominal injuries, possibilities include hepatic contusion or laceration, and splenic rupture. During the pre-bout evaluation the physician should have looked for history of coagulopathy or infectious mononucleosis. Splenic injury is an emergency that requires a “swoop and scoop” approach for immediate transfer to the hospital. Renal contusion may occur with or without hematuria. Pregnant women cannot box due to potential trauma to gravid uterus, and so a pregnancy test is part of any complete pre-bout evaluation for female athletes. When there is possible blunt scrotal trauma, consider hematocele or testicular torsion. Testicular torsion is an emergency and the boxer needs to go to the hospital to get a duplex Doppler ultrasound [32].

13.5 Prevention of Boxing Injuries

According to a study done by Potter et al. examining boxing injuries resulting in a visit to a US hospital emergency department from 1990 through 2008, the rate of injury was 12.7 per 1000 participants. Those injured boxers were mostly male (90.9%). The most common diagnosis in the study was fracture (27.5%), and the most common body regions injured were the hand (33.0%) and head and neck (22.5%). In their study, punching bag-related injuries accounted for 36.8% of boxing injuries [35].

Sparring should take place with the use of oversized gloves. Boxers who have not fought for over 12 months should not fight more than ten rounds. The question of inactivity raises concerns about the likelihood of increased risks of injuries based on inactivity and conditioning. A minimum of two ringside physicians should attend every boxing match [36].

The Association of Ringside Physicians supports and recommends the use of custom-fit mouthpieces to aid in the prevention of orofacial trauma. In addition, the Association of Ringside Physicians has made a consensus statement on the utility of headgear during amateur boxing competition: There are fewer head and facial injuries when using headgear in amateur boxing and eliminating headgear will make amateur boxing less safe. Headgear protects boxers against lacerations during competition and sparring [37, 38].

Conclusion
Boxing is a combat sport with a high risk of injuries to the head and extremities. Close medical supervision as outlined above helps to protect the health and safety of the boxer.

References

1. Loosemore M, Knowles CH, Whyte GP. Amateur boxing and risk of chronic traumatic brain injury: systematic review of observational studies. BMJ. 2007;335(7624):809.

2. Kelly M. Role of the ringside physician and medical preparticipation evaluation of boxers. Clin Sports Med. 2009;28:515–9.
3. King O. Infectious disease and boxing. Clin Sports Med. 2009;28:545–60.
4. Association of Ringside Physicians. Consensus statement on blood-borne infectious disease screening in combat sports. https://ringsidearp.org/wp-content/uploads/2020/01/Blood-Bourne-Infectious-Disease-Screening-in-Combat-Sports.pdf.
5. Association of Ringside Physicians. Ocular practice guidelines for participation in combative sports. https://ringsidearp.org/wp-content/uploads/2020/01/eye-position-paper.pdf.
6. Moseley G, Moseley D. Cerebral cavernoma in an aspiring boxer: 'the dream stealer'. J Combat Sports Med. 2019;1(1):1–4.
7. Sethi N, Neidecker J, Muzzi D, Horton M, Reyes P, Cantu R, deWeber K, Durkin L, Lee B, Rizzo N, Lovelace L, Golden P, Gelber J, Varlotta G, Weinstein R. Neuroimaging in combat sports: consensus statement from the Association of Ringside Physicians. BMJ. 2020;
8. Association of Ringside Physicians. Consensus statement on medical clearance of the older fighter in professional combat sports. https://ringsidearp.org/wp-content/uploads/2020/01/Medical-Clearance-of-the-Older-Fighter.pdf.
9. Siegel S. Cardiovascular issues in boxing and contact sports. Clin Sports Med. 2009;28:521–32.
10. Goodfellow R. USA boxing medical rules and medical handbook of AIBA open boxing. 2013. https://www.teamusa.org/usa-boxing/rulebook/medical-handbook.
11. Association of Ringside Physicians. Consensus statement on weight management in professional combat sports. https://ringsidearp.org/wp-content/uploads/2020/01/Weight-Management-in-Professional-Combat-Sports.pdf.
12. Schwartz M. Medical safety in boxing: administrative, ethical, legislative, and legal considerations. Clin Sports Med. 2009;28:505–14.
13. Sethi N. Boxer safety: reducing system errors in the ring. Curr Sports Med Rep. 2016;15(2):74–5.
14. Sethi N. The conflict between combat sports and ethical medicine: can the two co-exist? J Combat Sports Med. 2020;2(1):8–9.
15. Davis T, Ings A. Head injury: triage, assessment, investigation and early management of head injury in children, young people and adults (NICE guideline CG 176). Arch Dis Childhood Educ Practice Ed. 2015;100:97–100.
16. Jordan B. Brain injury in boxing. Clin Sports Med. 2009;28:561–78.
17. Lefkowitz T, Flanagan S, Varlotta G. Rehabilitation of orthopaedic and neurologic injuries. Clin Sports Med. 2009;28:623–39.
18. Neidecker J, Sethi NK, Taylor R, et al. Concussion management in combat sports: consensus statement from the Association of Ringside Physicians. Br J Sports Med. 2019;15:328–33.
19. Seifert T, Bernick C, Jordan B, Alessi A, Davidson J, Cantu R, Giza C, Goodman M, Benjamin J. Determining brain fitness to fight: has the time come? Phys Sportsmed. 2015;43(4):395–402.
20. Nieman LK. Causes of secondary and tertiary adrenal insufficiency in adults. In: Lacroix A, editor. . Waltham, MA: UpToDate; 2020.
21. Khan K, Saeed S, Ramcharan A, Gray S. A case series of closed head trauma with pituitary stalk disruption resulting in hypopituitarism. Int J Surg Case Rep. 2018;43:69–71.
22. Tanriverdi F, Kelestimur F. Neuroendocrine disturbances after brain damage: an important and often undiagnosed disorder. J Clin Med. 2015;4(5):847–57.
23. Shahid MH, Verma A, Youngblood L. From mechanical to chemical: a case of diabetes insipidus induced by concussive brain injury. Am J Med. 2018;131(7):e293–4.
24. Muzzi D. Ringside medicine for athletic commissions. Lecture presented at 2017; Minnesota, USA.
25. Melone C, Polatsch D, Beldner S. Disabling hand injuries in boxing: boxer's knuckle and traumatic carpal boss. Clin Sports Med. 2009;28:609–21.
26. Loosemore M, Lightfoot J, Palmer-Green D, Gatt I, Bilzon J, Beardsley C. Boxing injury epidemiology in the Great Britain team: a 5-year surveillance study of medically diagnosed injury incidence and outcome. Br J Sports Med. 2015;49(17):1100–7.
27. Sethi N, Wright K. Acute patellar dislocation in a boxer during a bout. J Combat Sports Med. 2020;2(1):5–7.
28. Corrales G, Curreri A. Eye trauma in boxing. Clin Sports Med. 2009;28:591–607.
29. Toldi JP, Thomas JL. Evaluation and management of sports-related eye injuries. Curr Sports Med Rep. 2020;19(1):29–34.
30. American Academy of Ophthalmology. Policy Statement: Reforms for the prevention of eye injuries in boxing. In: San Francisco: Eye Safety and Sports Ophthalmology Committee, 23 Jun 1990.
31. Mercieri A. Execise-induced hematuria. In: Glassock R, editor. . Waltham, MA: UpToDate; 2020.
32. Coletta D. Nonneurologic emergencies in boxing. Clin Sports Med. 2009;28:579–90.
33. Coletta D, Muzzi D. Management of ring/cage emergencies. Unpublished manuscript.
34. Evans AK, Handler SD. Evaluation and management of middle ear trauma. In: Bachur RG, Moreira ME, editors. . Waltham, MA: UpToDate; 2020.
35. Potter M, Snyder A, Smith G. Boxing injuries presenting to U.S. emergency departments, 1990–2008. Am J Prev Med. 2011;40(4):462–7.
36. Association of Ringside Physicians. Medical recommendations to improve boxing safety. https://ringsidearp.org/wp-content/uploads/2020/01/medrec.pdf.
37. Association of Ringside Physicians. Consensus statement on mouth guards for combat sports athletes. https://ringsidearp.org/wp-content/uploads/2020/01/Mouth-Guards.pdf.
38. Association of Ringside Physicians. Consensus statement on thxe use of headgear in amateur boxing. https://ringsidearp.org/wp-content/uploads/2020/01/Headgear.pdf.

14 Climbing

Joseph D. Cooper and Thomas Hackett

14.1 Introduction

Climbing is one of the fastest growing sports world-wide with up to 44.5 million climbers according to the International Federation of Climbing in their 2019 annual report [1]. With the recent addition of climbing to the 2020 Olympic Games, the popularity of the sport is only continuing to grow. Climbing is a sport that may be practiced across generations. Many climbers start out in childhood and may continue the sport well into their 70s or more. Genders are equally represented throughout the different disciplines and the scope of difficulty of any particular discipline can be extremely broad. Climbers as a group are highly detail oriented and deeply in-tune with their bodies similar to ballet dancers or violin players, and are focused on maximizing their body's potential. The sport often requires extreme and technical body positions which can push the body's limits.

Unlike the fields of play in many sports which are relatively controlled regarding environmental conditions, climbers may face an incredible array of climate and environmental hazards. For example, most climbing competitions are held indoors or on protected, specially constructed man-made walls with artificial holds where environmental conditions are well controlled and rarely play a role in the execution of the sport. On the other hand, Alpine mountaineering, ice climbing, and traditional climbing may take place in austere settings where extremes of temperature, altitude, and remoteness may significantly increase objective danger (Fig. 14.1). Additionally, this sport is often practiced in extremely remote international locations where first responders and rescue may be limited or non-existent. Therefore, climbing, as opposed to other more traditional sports, requires the participant to be their own first responder in the event of an injury and indeed, many climbers are highly trained as first responders.

Climbing encompasses several different disciplines all which have their own unique spectrum of injury. These different types of climbing may be broadly grouped into the following categories: Sport climbing and competition, traditional (trad) climbing, ice climbing, Alpine mountaineering, and bouldering. The various types carry their own risk categories. For instance, sport climbing and indoor climbing is relatively controlled with attachments to secure equipment and controlled surfaces, while traditional alpine climbing, or ice climbing, for example, has more risk, and more injuries due to the unpredictability of the terrain and the environments involved [1, 2]. Bouldering, while performed low to the ground, does not utilize

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Fig. 14.1 Ice climbing a frozen Niagara Falls

ropes and can consequently have the highest risk of falls and traumatic lower extremity injuries. Additionally, bouldering involves more explosive positions and higher loads and can therefore lead to more tendinopathies and overuse injuries as well. Climbing is generally performed with a belayed rope attached to another person, or to equipment that acts as a safeguard in the event of a fall. The majority of falls in climbing are wall collisions rather than ground falls, which is contrary to public perception [3]. A wall collision is when the climber falls a short distance, is caught by their safety belay rope, then swings into the wall, colliding side on with the wall. A ground fall is, as it sounds, a fall from height on to the ground.

The tendency to view climbing as a reckless or overly dangerous sport is a common misconception. There are a reported 4.2 injuries per 1000 h of climbing, compared to 15.7 and 9.8 injuries per 1000 h in football and basketball, respectively [4–6]. Different climbing environments effect this rate; indoor climbing, for instance, only has a reported injury rate of 0.2 injuries per 1000 h [7].

Injuries in rock climbing can be divided into acute and chronic. While some acute injuries do result from falls, others are due to the unique stresses and positions of climbing and unrelated to falls all together (Fig. 14.2). Chronic or overuse injuries are exceedingly common in climbing with over 2/3 of participants reporting an overuse injury requiring treatment during their career [8]. Up to 75% of all climbing injuries, acute and chronic, involve the upper extremity, with 60% of those involving the hand or fingers [9]. In the following chapter, we will review the common acute and chronic injuries specific to climbing as well as address treatment tips for caring for the climbing athlete.

Fig. 14.2 The climbers left shoulder demonstrating the gaston position. This is a common shoulder position in climbers requiring extreme internal rotation while abducting and extending. It can be a source of anterior shoulder tendonitis as well as irritation of labral injuries

14.2 Acute Injuries

14.2.1 Hand

Tendon injuries account for 52% of all hand/finger injuries in climbers [10]. Lacerations are also common at 15% of reported injuries. Fractures can occur during falls with 12% involving the wrist and 7% involving the fingers [11].

While it is easy to understand how fractures may be associated with falls, acute injuries to the tendons require an additional understanding of climbing. Climbers utilize multiple specialized grips to maintain contact with the climbing surface and facilitate movement. The type of grip a climber uses depends on the size and shape of the available hold and the climbers body positions. See Fig. 14.3 for images and descriptions of some of the common grip positions.

Pulley ruptures are common in climbers affecting approximately 25% of elite climbers [10, 11]. This is understandable when considering studies demonstrating that crimp and hanging grip positions can lead to forces in excess of 450 N on the A2 pulley and 269 N on the A4 pulley [12, 13], with reported forces required to rupture the pulley of only 400 N and 137 N, respectively, for the A2 and A4 pulley [14–17]. A pulley rupture can often be felt as a pop with pain at the time of injury. The climber will present with pain and swelling over the volar surface of the finger and point tenderness at the site of injury. If the A2 or A4 pulley is completely ruptured, bowstringing of the tendon may be seen with resisted flexion.

MRI is the most common imaging modality for diagnosing pulley injuries although ultrasound also has value in providing a dynamic exam. On MRI, a ruptured pulley can result in volar displacement of the tendon from the underlying phalanges by greater than 2 mm [11].

Conservative treatment is often adequate for grade I–III injuries with an initial immobilization period of 10–14 days in a volar splint followed by functional therapy with putty or compression balls utilizing pulley protection with tape or thermoplastic splints. Sporting activities can typically resume in 6–8 weeks after a grade III injury. Surgical reconstruction is only indicated for grade IV injuries with multiple described techniques in the literature [18, 19] (Table 14.1).

14.2.2 Shoulder

Shoulder dislocations with bankart lesions, SLAP tears, or posterior labral tears can occur in rock climbing. While many of these lesions can be

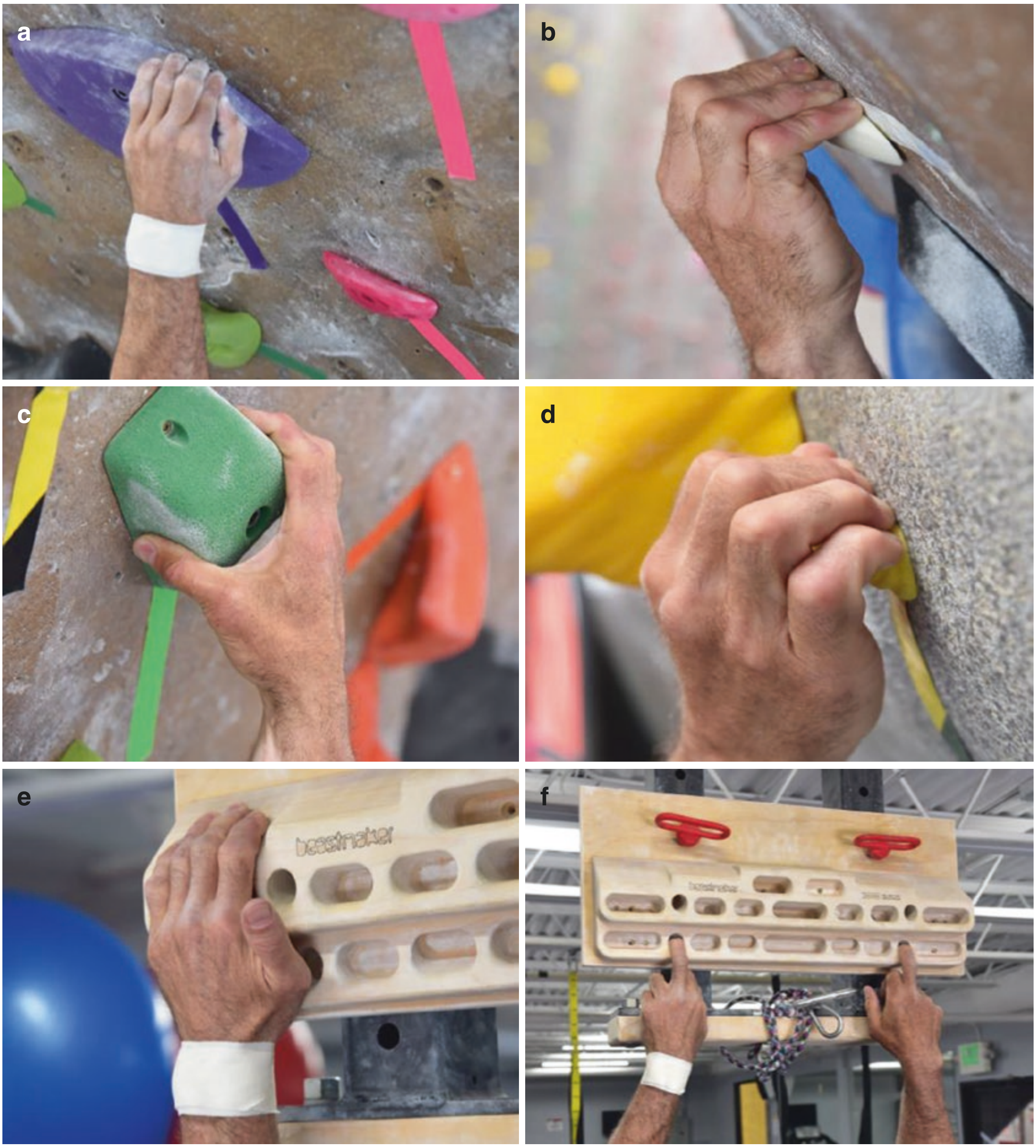

Fig. 14.3 Demonstration of various climbing grip positions. (**a**) Closed crimp/sloper, (**b**) half crimp, (**c**) pinch, (**d**) full crimp on small hold, (**e**) open crimp/sloper, (**f**) single finger/mono hang

treated conservatively, it is important to acknowledge the extreme risk a potential subsequent dislocation or instability event may have in rock climbers, possibly causing them to fall and suffer an injury or be unable to regain hold on the wall due to their dislocated shoulder. We characterize these athletes as consequence athletes, meaning that the consequence of the dislocations may be worse than the dislocation itself.

Posterior labral tears associated with shoulder pain that limit activity are treated with a short course of physical therapy, then surgery if not resolved in 4–6 weeks. Bankart lesions in first time dislocators without bone loss are

Table 14.1 Pulley injuries in climbers

Grade	Injury	Treatment
Grade I	Pulley strain	2–4 weeks of protection with tape, return to sport in 4–6 weeks
Grade II	Complete rupture of A4 or partial rupture A2 or A3	10 days of immobilization followed by 2–4 weeks of functional therapy and protection with tape, return to sport at 4–6 weeks with protection
Grade III	Complete rupture of A2 or A3	10–14 days of immobilization with 4 weeks of therapy with thermoplastic splint protection, return to sport at 2–3 months
Grade IV	Multiple ruptures as A2/A3, A2/A3/A4, or single rupture (A2 or A3) combined with lumbrical muscles or collateral ligament trauma	Surgical repair followed by 6 weeks of protection in splint and return to sport at 4–6 months

often treated more aggressively with operative labral repair in order to prevent future dislocations and potential bone loss in this population which places high demands on their upper extremities. However, if there is any concern for bone loss, we are aggressive at recommending a Latarjet procedure for these injuries. Arthroscopic bankarts have an average failure rate ranging from 10% to 20% [20] in the literature, while the Latarjet is consistently reported at 2–5% [20]. In the climbing population with the severe risks associated with subsequent dislocation as mentioned above, the reduced failure rate of the Latarjet is especially appealing. In our practice, there have been no limitations to the functional abilities of even the most elite climbers, and the procedure has consistently low failure rates.

Many asymptomatic climbers may have SLAP tears on MRI and treating physicians should be careful attributing pain to this finding. However, true symptomatic SLAP tears in climbers can occur. While some may occur acutely, the majority of these are due to repetitive microtrauma similar to other competitive overhead athletes [21, 22]. Operative treatment of SLAP tears in overhead athletes have inconsistent results with return to sport rates ranging from 22% to 75% [23]. Rather than a repair, climbers with symptomatic SLAP tears are at times better treated with a tenodesis of the long head of the biceps tendon. In our practice, we prefer a robust construct for this population. We perform a mini-open subpectotal tenodesis with a biceps button, backed up with a tenodesis screw.

Other common shoulder pathology in climbers are AC joint sprains, muscle strains of the latissimus dorsi, rhomboid, or trapezius [24]. These can generally be treated with functional therapy. Ruptures of the long head of the biceps have been reported, and these are often best addressed with an open tenodesis though some patients may do well with non-surgical management.

14.2.3 Elbow

Elbow pain is the third most common complaint of elite rock climbers [25]. Acute “climber’s elbow” is defined as tears of the brachialis muscle at the muscle-tendon junction [26]. These tears present as pain in the cubital fossa. They can be distinguished from ruptures of the distal biceps by resisted elbow motion. Pain on resisted flexion with forearm in supination is due to biceps injuries, while pain with resisted elbow flexion with the am in pronation is due to a brachialis injury [25]. Climbers should focus on pull-ups with the forearm pronated during training to help strengthen the brachialis and prevent these injuries. Acute, complete tears of the brachialis can be diagnosed with MRI and should be treated surgically. Repair is ideally performed within 1–2 weeks, patients are then placed in a splint at 90° for 1 week, and progressive extension is accomplished with an elbow brace over the next 8 weeks.

14.3 Lower Extremity

While upper extremity injuries are more common in rock climbing overall, the lower extremities are more likely to suffer fractures due to ground or wall falls [27, 28]. Falls from as little as 8 ft can lead to fracture of the calcaneus, talus, or ankle joint [27]. Ligamentous injuries such as ankle sprains are also common [29, 30]. These injuries are particularly common in bouldering, but can also occur in indoor or sport climbing if the athlete falls before reaching the first security attachment for their rope, or are dropped by their belayer [27].

14.3.1 Knee

Knee injuries are less common in climbing [10, 31]. While ligament tears and fractures can occur with fall, the majority of knee-related injuries are due to the extreme positions required in climbing. The high step position, the drop knee position, and the heel hook have been identified as especially hazardous for the climber's knee [32]. In the high step position, the athlete's weight is mostly on one leg which is position in maximum knee flexion with a flexed, externally rotated and adducted hip. The drop knee is a similar knee position with an internally rotated hip. The heel hook position uses the heel to apply pressure on a hold while pulling on the foot by flexing the knee via strong hamstring contraction [32] (see Fig. 14.4).

Medial meniscus tears are the most common knee injury with a reported incidence of 28% [32]. These occur predominately in the high step and drop knee positions. IT band sprains represent 19.5% of knee injuries and occur exclusively in the heel hook position [32].

The heel hook position is a cause of posterior cruciate ligament (PCL) injuries in climbers [33]. While PCL injuries generally occur with a posterior directed force on a flexed knee [34], the heel

Fig. 14.4 Various common knee positions in climbers. (**a**) The high step position, (**b**) the drop knee position, (**c**) the heel hook

hook position places extreme load in an anterior to posterior direction on a slightly flexed or even hyper extended knee and can overload the PCL.

In addition to PCL injuries, proximal tibial-fibular joint (PTFJ) injuries have been associated with the heel hook position. PTFJ injuries have a wide array of presentations from frank dislocations, to discomfort during activity, or symptoms related to irritation of the common peroneal nerve [35–37]. The common mechanism for injuries to the PTFJ are falling onto a flexed knee or excessive rotation of the knee with the foot planted on the ground. However, the heel hook position leads to extreme loading of the lateral knee with forced external rotation of the leg and also places the PTFJ in its most susceptible position [37, 38]. Physical exam can diagnose PTFJ instability. With the knee flexed to 90° to relax the LCL and biceps femoris, the fibular head is held between the thumb and index finger and translated anterolaterally [39]. Instability is usually encountered in the anterior direction as the posterior ligamentous complex is structurally weaker and more susceptible to injury [35, 39, 40]. Lateral and AP radiographs can help diagnose a frank PTFJ dislocation; however, MRI can be useful to confirm the diagnosis in the setting of more subtle instability. The MRI may show joint effusions of the PTFJ, bone edema at the proximal tibia, or visible ligamentous disruption of the posterior tibiofibular ligament [40]. PTFJ instability can be treated conservatively with protected weight bearing and bracing; however conflicting results have been reported with up to 23% of patients failing non-operative treatment [37]. Operative treatment is indicated when conservative measures have failed or in cases of frank and irreducible dislocations. Our preferred reconstruction technique is with bone tunnels in the fibular head and proximal tibia using a hamstring autograft or allograft fixed with tenodesis screws to reconstruct the posterior tibiofibular ligament.

14.3.2 Ankle/Foot

Fractures of the ankle, heel, and foot are common in climbers as mentioned above. These injuries should undergo standard orthopedic treatment guidelines for fractures. Many of them will require surgery considering climbers are mostly young, highly active patients with high functional demands [27].

Peroneal tendon dislocations are rare injuries, but can be seen in rock climbers [10, 27]. Patients present with pain at the lateral ankle and often report an audible pop with the foot in plantar flexion and maximum inversion, while placing extreme forces on the big toe while climbing—the pop is from a traumatic rupture of the peroneal retinaculum. Climbers generally do not tolerate conservative treatment of these injuries and operative repair is indicated [41, 42]. In acute cases, direct repair of the retinaculum is often possible, and a periosteal flap can be utilized to bolster the repair if required [27]. Patients are immobilized for 6 weeks postoperatively and can begin climbing activities again at 8 weeks.

14.4 Chronic Injuries

The majority of injuries in climbing are chronic overuse injuries [8, 43]. Much of this is related to excessive climbing specific training without adequate rest and recovery between training sessions [44]. Older climbers may be more susceptible to these injuries [45]; however, as climbing becomes more popular and younger climbers begin to specialize at an earlier age, additional chronic injuries are emerging in the younger population from early specialization [46].

14.4.1 Hand/Wrist

Tendonitis of the hand and wrist is the most common injury reported in climbers [10, 45]. Pain at the wrist or forearm can be diagnosed on exam based on knowledge of tendon insertion sites. The most common tendons involved are the FDP and FDS although FCU tendonitis is also common [25]. Chronic tendonitis can lead to hypertrophic tendons, and commonly results in carpal tunnel syndrome, reported in up to 25% of elite climbers [47].

Digital collateral ligament strains are common in high frequency climbers due to excessive torsion in certain grip positions. Once strained, the collateral ligaments rarely return to their same level of tension and stability [25]. Buddy taping to adjacent digits can help provide support to the injured collaterals as well as prevent the offending position. Chronic collateral ligament injury and subsequent IP joint instability leads to early IP joint OA seen frequently in climbers. Climbers will frequently present with IP joint effusion and early spurring and other signs of OA on imaging [47, 48]. While OA of the digits is seen commonly at the DIP joint in the general population, climbers may develop this characteristic swelling and fixed flexion at the PIP joint and at an early age.

Extensor hood syndrome is a constellation of arthritic changes affecting the tendon sheaths of the fingers [3]. Extensive climbing can predispose to OA of the digits, leading to bone spurs of the IP joints irritating the tendons and further exacerbating the tendonitis [49]. This generally presents with tenderness to palpation dorsally at the extensor hoods and is commonly accompanied by joint swelling, as well as 3–5° of extensor lag [50]. It is treated conservatively with rest, NSAIDs, and occasionally injections.

Young climbers, especially those doing excessive climbing or finger training for climbing, are susceptible to epiphyseal fractures of the phalanges [10]. They occur most frequently at the PIP joints, and the patient may present with pain or tenderness at the dorsal aspect of the joint [51]. Young climbers rarely report an acute traumatic event when diagnosed with these injuries, leading to the theory that the fractures are stress related and possibly the result of repetitive microtrauma and overloading the developing joint [46]. Injuries identified less than 4 weeks old are treated with 3 weeks of splinting followed by functional therapy. When identified over 4 weeks, malunions or fibrous unions can lead to complete separation of the epiphysis from its attachments and chronic deformity [46]. When identified late, there is no indication for splinting, and these epiphyseal fractures can be treated with rest and functional rehab only.

14.4.2 Elbow

Medial and lateral epicondylitis are both common in climbers and reported as a frequent cause of discomfort, especially in older climbers [45]. Similar to the general population, conservative treatment is typically successful with rest, bracing, and therapy. Chronic epicondylitis can lead to hypertrophy and occasionally nerve entrapment syndromes at the elbow [3].

Acute "climber's elbow" was described in the previous section as an acute rupture of the brachialis muscle. Chronic "climber's elbow" is also prevalent and is a result of a constellation of tendinopathies. Strenuous traversing climbs or any route requiring strong and prolonged flexion and pronation of the elbow with insufficient biceps brachii activation leads to compensatory firing of the brachialis and can lead to tendonitis [52]. Pain is again at the anterior elbow with flexion in a pronated forearm position. Treatment is conservative with rest for 2–4 weeks with resumption of low intensity climbing. When symptoms resolve and a gradual increase in intensity as tolerated [3, 25, 52].

Elbow osteoarthritis is common and can present at a young age in active climbers. The mechanism is likely similar to that seen in finger joints in climbers with repetitive microtrauma leading to progressive cartilage damage and destruction of the articular surface. In this active population, elbow arthritis can lead to limitations in motions with flexion contractures, and pain with terminal extension. As in the general population, treatment is conservative focusing on rest, anti-inflammatory medications, and therapy to strengthen the surrounding supporting musculature. In more advanced cases, or prior to large climbing trips, steroid injections can help with pain control. If conservative treatment fails or the loss of motion becomes too debilitating, the bony overgrowth can be treated with arthroscopic osteochondroplasty.

14.4.3 Shoulder

As with the elbow, tendonitis is common in the shoulder. Rotator cuff tendonitis, internal impingement, subacromial impingement, and biceps tendonitis are excessively common in climbers [3, 10, 26, 52, 53]. In our experience, internal impingement with contact of the underside of the rotator cuff with the glenoid rim in repetitive abducted external rotation is particularly prevalent in this population [54]. These can almost always be treated conservatively with therapy alone although targeted steroid injections can also be helpful to break the cycle of inflammation and allow pain-free therapy. Therapy is focused on scapular stabilization and proprioception. Upper trapezial dominance is particularly common and can be reversed with appropriate strengthening measures. Less common pathologies such as nerve entrapment of the supra scapular nerve as well as thoracic outlet syndromes have also been identified in climbers [55].

Subcoracoid impingement can be seen in the general population; however, it commonly has a different pathology in climbers. In the general population, subcoracoid impingement is often due to an abnormally short or excessively curved coracoid leading to abnormalities in the space between the acromial arch and the rotator cuff, leading to irritation and pain [56, 57]. In climbers, the space limitation is due to hypertrophy of the subscapularis muscle and tendon due to a combination of strength from frequent use as well as hypertrophy from chronic inflammation [55]. Additionally, asymptomatic shoulder anterior hyperlaxity common in climbers can further decrease the subacromial interval and lead to compression [55]. Coracoid impingement can be diagnosed on physical exam with pain in the cross arm adduction, forward elevation, and internal rotation position. Treatment is again conservative, with therapy and injections. An arthroscopic subcoracoid decompression can be performed if conservative measures fail.

14.4.4 Ankle

While tendonitis of the ankle is common in climbers, an injury pattern more unique to them are OCD lesions. Frequent, minor falls onto the ankle and ankle sprains predispose climbers to posttraumatic OCD [3, 27]. These injuries, which are potential complications of sprains or fractures, can be easily missed on radiographs alone and the treating provider should have a high index of suspicion, ordering an MRI when suspected [27]. The talus is a common site for climber OCD lesions. These often require operative treatment with cartilage restorative procedure such as an autologous chondrocyte transplant [58], osteochondral allograft [59], or matrix-associated autologous chondrocyte transplantation [60].

14.4.5 Foot

Chronic foot conditions in climbing are generally the result of climbing shoes (see Fig. 14.5). Climbing shoes are specifically designed and engineered to give climbers maximal performance advantages in grip and stability while standing on small edges with precision. They are generally worn two sizes smaller than casual shoes; the shoes are designed with a front point and often a semi-rigid flex similar to a ballet shoe. This causes compression of the first and fifth metatarsal joints, as well as tightening of the plantar fascia [61, 62]. Climbers are willing to suffer for advantages in performance. Between 80% and 90% of climbers reported pain while wearing their climbing shoes [27, 62]. Frequent consequences include calluses, nail bed infection, subungual hematomas, and nerve compression syndromes of the foot [27, 61–63]. Hallux valgus deformity is common in athletes who have climbed for greater than 5 years [3, 27, 61].

14.5 Pearls for Treating Climbers

Injury prevention and continued therapy in climbers is critical. Excessive climbing without additional training can lead to muscle imbalance; strengthening of opposing muscle groups, maintaining flexibility, and low weight, high repetition exercises are critical. It is important to emphasize the necessity of recovery. Rest between rigorous climbing sessions can help prevent overuse injuries. As climbers age, they must be aware that longer rest periods may be required to avoid over strain. An important aspect of treating climbers is an awareness of the medical hazards of participating in a sport in such potentially extreme envi-

Fig. 14.5 Variations of the climbing shoe. (**a**) Shows a moderate arch exaggeration with a prominent hooked distal toe. (**b**, **c**) Show various angles of a high arch variation with a slightly flatter toe. The advantage of these shoe positions is shown in (**d**) with demonstration of using the tip of the toe to support much of the climber's body weight on a hold

ronments. Frost bite, cerebral edema, pulmonary edema, altitude sickness, snow blindness, heat exhaustion, dehydration—an awareness of these medical issues must be a part of the medical knowledge of the physician hoping to take care of the climbing community.

References

1. Schöffl V, Morrison A, Schöffl I, Küpper T. The epidemiology of injury in mountaineering, rock and ice climbing. Med Sport Sci. 2012;58:17–43.
2. Schöffl V, Morrison A, Schwarz U, Schöffl I, Küpper T. Evaluation of injury and fatality risk in rock and ice climbing. Sports Med. 2010;40(8):657–79.
3. Peterson C, Ceraulo A. Caring for climbers. Curr Sports Med Rep. 2015;14(5):397–403.
4. Schöffl V, Küpper T, Hartmann J, Schöffl I. Surgical repair of multiple pulley injuries—evaluation of a new combined pulley repair. J Hand Surg Am. 2012;37(2):224–30.
5. Folkl AK. Characterizing the consequences of chronic climbing-related injury in sport climbers and boulderers. Wilderness Environ Med. 2013;24(2):153–8.
6. Schweizer A. Sport climbing from a medical point of view. Swiss Med Wkly. 2012;142:w13688.
7. Schöffl VR, Hoffmann G, Küpper T. Acute injury risk and severity in indoor climbing-a prospective analysis of 515,337 indoor climbing wall visits in 5 years. Wilderness Environ Med. 2013;24(3):187–94.
8. Grønhaug G. Self-reported chronic injuries in climbing: who gets injured when? BMJ Open Sport Exerc Med. 2018;4(1):e000406.
9. Rooks MD. Rock climbing injuries. Sports Med. 1997;23(4):261–70.
10. Schöffl V, Popp D, Küpper T, Schöffl I. Injury trends in rock climbers: evaluation of a case series of 911 injuries between 2009 and 2012. Wilderness Environ Med. 2015;26(1):62–7.
11. Chang CY, Torriani M, Huang AJ. Rock climbing injuries: acute and chronic repetitive trauma. Curr Probl Diagn Radiol. 2016;45(3):205–14.
12. Bollen SR. Injury to the A2 pulley in rock climbers. J Hand Surg Br. 1990;15(2):268–70.
13. Roloff I, Schöffl VR, Vigouroux L, Quaine F. Biomechanical model for the determination of the

forces acting on the finger pulley system. J Biomech. 2006;39(5):915–23.
14. Lin GT, Amadio PC, An KN, Cooney WP, Chao EY. Biomechanical analysis of finger flexor pulley reconstruction. J Hand Surg Br. 1989;14(3):278–82.
15. Lin GT, Cooney WP, Amadio PC, An KN. Mechanical properties of human pulleys. J Hand Surg Br. 1990;15(4):429–34.
16. Widstrom CJ, Doyle JR, Johnson G, Manske PR, McGee R. A mechanical study of six digital pulley reconstruction techniques: Part II. Strength of individual reconstructions. J Hand Surg Am. 1989;14(5):826–9.
17. Widstrom CJ, Johnson G, Doyle JR, Manske PR, Inhofe P. A mechanical study of six digital pulley reconstruction techniques: Part I. Mechanical effectiveness. J Hand Surg Am. 1989;14(5):821–5.
18. Odobescu A, Radu A, Brutus J-P, Gilardino MS. Modified flexor digitorum superficialis slip technique for A4 pulley reconstruction. J Hand Surg Eur Vol. 2010;35(6):464–8.
19. Clark TA, Skeete K, Amadio PC. Flexor tendon pulley reconstruction. J Hand Surg Am. 2010;35(10):1685–9.
20. Bliven KCH, Parr GP. Outcomes of the Latarjet procedure compared with Bankart repair for recurrent traumatic anterior shoulder instability. J Athl Train. 2018;53(2):181–3.
21. Lichtenberg S, Magosch P, Habermeyer P. [Superior labrum-biceps anchor complex]. Orthopade. 2003;32(7):616–26.
22. Schöffl V, Popp D, Dickschass J, Küpper T. Superior labral anterior-posterior lesions in rock climbers—primary double tenodesis? Clin J Sport Med. 2011;21(3):261–3.
23. Boileau P, Parratte S, Chuinard C, Roussanne Y, Shia D, Bicknell R. Arthroscopic treatment of isolated type II SLAP lesions: biceps tenodesis as an alternative to reinsertion. Am J Sports Med. 2009;37(5):929–36.
24. Shea KG, Shea OF, Meals RA. Manual demands and consequences of rock climbing. J Hand Surg Am. 1992;17(2):200–5.
25. Holtzhausen LM, Noakes TD. Elbow, forearm, wrist, and hand injuries among sport rock climbers. Clin J Sport Med. 1996;6(3):196–203.
26. Bollen SR. Soft tissue injury in extreme rock climbers. Br J Sports Med. 1988;22(4):145–7.
27. Schöffl V, Küpper T. Feet injuries in rock climbers. World J Orthop. 2013;4(4):218–28.
28. Buzzacott P, Schöffl I, Chimiak J, Schöffl V. Rock climbing injuries treated in US emergency departments, 2008–2016. Wilderness Environ Med. 2019;30(2):121–8.
29. Backe S, Ericson L, Janson S, Timpka T. Rock climbing injury rates and associated risk factors in a general climbing population. Scand J Med Sci Sports. 2009;19(6):850–6.
30. Nelson NG, McKenzie LB. Rock climbing injuries treated in emergency departments in the U.S., 1990–2007. Am J Prev Med. 2009;37(3):195–200.
31. Lutter C, El-Sheikh Y, Schöffl I, Schöffl V. Sport climbing: medical considerations for this new Olympic discipline. Br J Sports Med. 2017;51(1):2–3.
32. Lutter C, Tischer T, Cooper C, Frank L, Hotfiel T, Lenz R, Schöffl V. Mechanisms of acute knee injuries in bouldering and rock climbing athletes. Am J Sports Med. 2020;48(3):730–8.
33. Thompson RN, Hanratty B, Corry IS. ‘Heel hook’ rock-climbing maneuver: a specific pattern of knee injury. Clin J Sport Med. 2011;21(4):365–8.
34. Krämer K-L, Jani L, Grifka J, Scharf H-P, Schleberger R, Zacher J, Kommission Leitlinien der Allianz Deutscher Orthopäden. Guidelines in orthopaedic surgery. A first step. Orthopade. 1999;28(3):236–42.
35. Ogden JA. The anatomy and function of the proximal tibiofibular joint. Clin Orthop Relat Res. 1974;101:186–91.
36. Dennis JB, Rutledge BA. Bilateral recurrent dislocations of the superior tibiofibular joint with peroneal-nerve palsy. J Bone Joint Surg. 1958;40(5):1146–8.
37. Kruckeberg BM, Cinque ME, Moatshe G, Marchetti D, DePhillipo NN, Chahla J, LaPrade RF. Proximal tibiofibular joint instability and treatment approaches: a systematic review of the literature. Arthroscopy. 2017;33(9):1743–51.
38. Scott J, Lee H, Barsoum W, van den Bogert AJ. The effect of tibiofemoral loading on proximal tibiofibular joint motion. J Anat. 2007;211(5):647–53.
39. Curatolo CM, Bach G, Mutty CE, Marzo JM. Review of common clinical conditions of the proximal tibiofibular joint. Am J Orthop. 2018;47(12) https://doi.org/10.12788/ajo.2018.0105.
40. Burke CJ, Grimm LJ, Boyle MJ, Moorman CT, Hash TW. Imaging of proximal tibiofibular joint instability: a 10 year retrospective case series. Clin Imaging. 2016;40(3):470–6.
41. Brage ME, Hansen ST. Traumatic subluxation/dislocation of the peroneal tendons. Foot Ankle. 1992;13(7):423–31.
42. Arrowsmith SR, Fleming LL, Allman FL. Traumatic dislocations of the peroneal tendons. Am J Sports Med. 1983;11(3):142–6.
43. Forrester JD, Tran K, Tennakoon L, Staudenmayer K. Climbing-related injury among adults in the United States: 5-year analysis of the National Emergency Department Sample. Wilderness Environ Med. 2018;29(4):425–30.
44. Lum ZC, Park L. Rock climbing injuries and time to return to sport in the recreational climber. J Orthop. 2019;16(4):361–3.
45. Lutter C, Hotfiel T, Tischer T, Lenz R, Schöffl V. Evaluation of rock climbing related injuries in older athletes. Wilderness Environ Med. 2019;30(4):362–8.

46. Hochholzer T, Schöffl VR. Epiphyseal fractures of the finger middle joints in young sport climbers. Wilderness Environ Med. 2005;16(3):139–42.
47. Heuck A, Hochholzer T, Keinath C. [MRT of the hand and wrist of sport climbers. Imaging of injuries and consequences of stress overload]. Radiologe. 1992;32(5):248–54.
48. Rohrbough JT, Mudge MK, Schilling RC. Overuse injuries in the elite rock climber. Med Sci Sports Exerc. 2000;32(8):1369–72.
49. Allenspach P, Saupe N, Rufibach K, Schweizer A. Radiological changes and signs of osteoarthritis in the fingers of male performance sport climbers. J Sports Med Phys Fitness. 2011;51(3):497–505.
50. Schöffl V, Hochholzer T, Schöffl I. Extensor hood syndrome—osteophytic irritation of digital extensor tendons in rock climbers. Wilderness Environ Med. 2010;21(3):253–6.
51. Jones G, Johnson MI. A critical review of the incidence and risk factors for finger injuries in rock climbing. Curr Sports Med Rep. 2016;15(6):400–9.
52. Peters P. Orthopedic problems in sport climbing. Wilderness Environ Med. 2001;12(2):100–10.
53. Bollen SR. Upper limb injuries in elite rock climbers. J R Coll Surg Edinb. 1990;35(6):S18–20.
54. Drakos MC, Rudzki JR, Allen AA, Potter HG, Altchek DW. Internal impingement of the shoulder in the overhead athlete. J Bone Joint Surg Am. 2009;91(11):2719–28.
55. Schöffl V, Schneider H, Küpper T. Coracoid impingement syndrome due to intensive rock climbing training. Wilderness Environ Med. 2011;22(2):126–9.
56. Roche SJ, Kennedy MT, Butt AJ, Kaar K. Coracoid impingement syndrome: a treatable cause of anterior shoulder pain. Ir J Med Sci. 2006;175(3):57–61.
57. Radas CB, Pieper H-G. The coracoid impingement of the subscapularis tendon: a cadaver study. J Shoulder Elb Surg. 2004;13(2):154–9.
58. Koulalis D, Schultz W, Heyden M. Autologous chondrocyte transplantation for osteochondritis dissecans of the talus. Clin Orthop Relat Res. 2002;395(395):186–92.
59. Lin JS, Andersen LB, Juliano PJ. Effectiveness of composite bone graft substitute plugs in the treatment of chondral and osteochondral lesions of the talus. J Foot Ankle Surg. 2010;49(3):224–31.
60. Dickschas J, Welsch G, Strecker W, Schöffl V. Matrix-associated autologous chondrocyte transplantation combined with iliac crest bone graft for reconstruction of talus necrosis due to villonodular synovitis. J Foot Ankle Surg. 2012;51(1):87–90.
61. van der Putten EP, Snijder CJ. Shoe design for prevention of injuries in sport climbing. Appl Ergon. 2001;32(4):379–87.
62. Killian RB, Nishimoto GS, Page JC. Foot and ankle injuries related to rock climbing. The role of footwear. J Am Podiatr Med Assoc. 1998;88(8):365–74.
63. Peters P. Nerve compression syndromes in sport climbers. Int J Sports Med. 2001;22(8):611–7.

CrossFit, Spinning, Aerobics

15

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15.1 Introduction

Sports practice and regular physical activities are stimulating actions as they promote rewarding physical and mental well-being. Nowadays, more and more people are adopting the practice of physical activities in their daily lives, showing that they are becoming more aware of the benefits of healthy habits to ensure longevity with quality of life [1].

In this context, fitness centers have become an attractive and revitalizing place to workout muscles, share training programs and experiences, and, of course, lose weight and become fit. These centers have invested in their structure, design, development of new materials for sports practice, new modalities of aerobics and anaerobic exercises, and combined exercises offering different options to sport's practitioners. Consequently, the number of practitioners has grown exponentially worldwide [2, 3].

Fitness exercises consist of aerobic assignments, strengthening, stretching, endurance, and balancing exercises. Regarding the aerobic exercises, running remains the main physical activity used to prepare and improve the cardiovascular,

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pulmonary, and musculoskeletal condition in athletes and sports practitioners [4]. On a regular basis, the commonly used training programs involve weightlifting, spinning, running, jumping, and also mixing all of them, such as in CrossFit® [5].

Spinning or indoor cycling is also a popular aerobic exercise where the athletes perform movements of standing, jumping, climbing, and sprinting on a weighted flywheel stationary bike (Fig. 15.1) [6]. The spinning coach manages the physical exercises using music tracks to motivate participants to increase and decrease the exercise intensity. Each session lasts from 45 min to an hour, focusing on high-intensity exercises of endurance, speed, strength, intervals, and recovery.

Fig. 15.1 Athlete in the weighted flywheel stationary bike. (With permissions from Marcia Camargo)

On the other hand, in an anaerobic exercise, such as weightlifting, the athlete performs a brief burst of vigorous movements [7] (Fig. 15.2).

CrossFit is a physical and metabolic conditioning program, based on constantly varied functional and high-intensity movements. The name of this training program is related to Greg Glassman who created CrossFit Inc. [8]. It involves a mix of exercises such as spinning, running, squatting, rowing, weightlifting, exercises using ropes, box, bar, balls, elastic bands, and tires, and also exercises performed with practitioners' own body weight (Fig. 15.3). CrossFit® is recommended to people that aim to improve their physical and cardiorespiratory endurance, balance, speed, and muscle power. In addition, it contributes to weight loss and muscular growth when it is more intense. It has been developed to be a non-monotonous physical exercise routine; however, due to its intensity, practitioners must be aware of their own body limits, respecting them and managing their exercise program accordingly [9].

Although the athletes and regular sports practitioners train sports movements exhaustively, anatomical and functional issues could play a role on their biomechanical effectiveness. Moreover, athletes are prone to injuries when attemping to enhance their performance in order

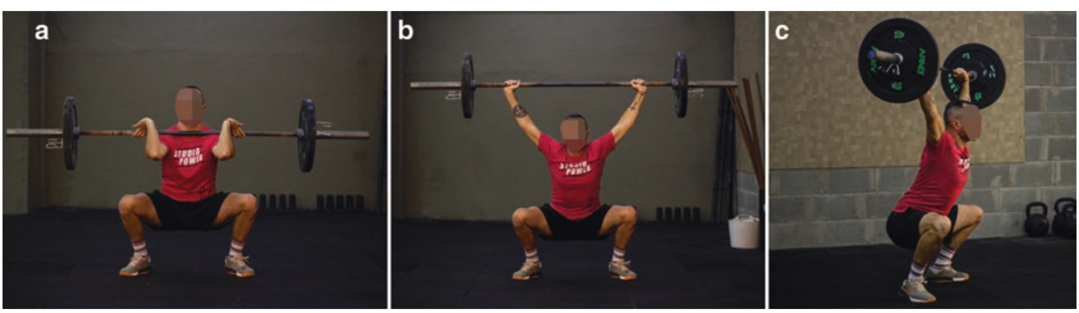

Fig. 15.2 Weightlifting: (**a**) front squat, front squat overhead: (**b**) anterior and (**c**) lateral views. (With permissions from Rodrigo Lima)

Fig. 15.3 CrossFit exercises (**a**) jumping up on a box (**b**) going up a rope, (**c**) sit-ups, (**d**) ring dip (**e**) kipping chest-to-bar pullup (**f**) push-ups. (With permissions from Rodrigo Lima)

to win competition or break their personal records [10]. Retrospective studies on CrossFit® have shown that the prevalence of musculoskeletal injuries can vary from 19% to 73%, according to period of training time, with an estimate incidence ranging from 2.1 to 3.1 injuries per 1000 h of training [11].

This chapter approaches the most common injuries in fitness exercises, focusing on shoulder, hip, and knee injuries, shin pain, and Achilles tendinopathy, discussing the importance of identifying these factors, taking preventive measures against injuries, and also, making decisions regarding the therapeutic approaches.

15.2 Shoulder

15.2.1 Introduction

The shoulder is the most mobile joint of the body. This characteristic is due to its glenoid shape that has less contact area with the humeral head when compared to the other joints. On the other hand, to get enough stability, the shoulder has dynamic forces like the rotator cuff, the scapula movement, and static forces like the labrum, glenohumeral ligaments, and lower intra-articular pressure [12].

Aerobic exercises and others whose focus are on the lower limb can lead to shoulder injuries only if acute trauma occurs. The most prevalent is the acromion-clavicular dislocation, shoulder dislocation, fractures (proximal humeral, clavicle) [13].

Athletes or regular gym practitioners can experience some shoulder lesion without acute trauma, mainly when they perform repetition movements in the extreme of the motion range. Furthermore, most of them have an unbalanced muscle group (as an example, internal rotators with more power than external rotators) [14].

15.2.2 Shoulder Injuries

The spectrum of the injuries is the inflammatory ones, rotator cuff and labral lesions and scapula dyskinesia.

15.2.2.1 Inflammatory Injuries

Inflammatory injuries take place on several anatomical sites: long head of the biceps, rotator cuff bursa or tendon or muscles around the shoulder.

In general, athletes feel pain, and it can lead to a decrease in performance or a period of training withdrawal.

A careful physical examination allows to achieve an accurate diagnosis. Muscle palpation is also useful to exclude impingement or rotator cuff diseases. However, imaging exams like ultrasonography, MRI may confirm the anatomical site of the injury [15] (Fig. 15.4).

The treatment aims to relieve pain, and mainly finds out the movement that is leading the lesion and correct it. The abusive use of anti-inflammatory drugs is expected when athletes do not change how they perform the movement. Surgical treatment is recommended when there is an anatomical lesion of the biceps long head or in case of its subluxation [16].

Fig. 15.4 MRI of subacromial impingement: supraspinatus bursa edema. (With permissions from Guilherme Grisi Mouraria)

15.2.2.2 Shoulder Impingement

Shoulder impingement can be classified into two types: external (subacromial) or internal.

The external impingement is the most prevalent when the rotator cuff tendon hits the subacromial arc. In fitness training, exercises performed with the shoulder at 90° of flexion, such as in weightlifting, may be more likely to result in shoulder impingement [17], although they rarely progress to cuff rupture. A CrossFit® epidemiological study has shown that the shoulder was the most affected joint in this training program [18], despite the need of surgery in only a few cases.

Internal impingement occurs when the posterosuperior aspect of the supraspinatus tendon hits de superior aspect of the glenoid. Typically, it happens in abduction and external rotation. In clinical practice, shoulder impingement can be assessed by two clinical tests: Neer test (Fig. 15.5a) and Hawkins test (Fig. 15.5b). Both tests are performed passively by the examiner. In the Neer test, the examiner performs an internal rotation of the patient's arm and moves it in forwarding flexion or until the patient complains of pain, while in the Hawkins test, with the arm at 90° flexed forward and the elbow at 90°, the physician performs an internal rotation of the arm to

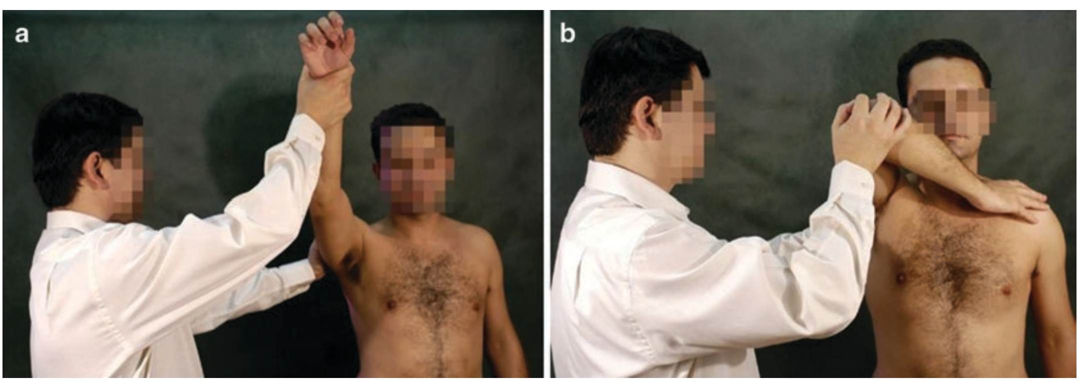

Fig. 15.5 Shoulder impingement tests: Neer (**a**) and Hawkins (**b**) tests. (Courtesy: Sergio Rocha Piedade)

Fig. 15.6 Glenohumeral internal rotation deficit on the right shoulder. (With permissions from Guilherme Grisi Mouraria)

Fig. 15.7 MRI of Partial tear of the supraspinatus tendon. (With permissions from Guilherme Grisi Mouraria)

the end of the motion range or until reports of pain.

As throwing exercises could lead to this lesion, athletes need to stretch the posterior joint capsule, strengthen internal rotators to avoid glenohumeral internal rotation deficit, and prevent and treat internal impingement [19] (Fig. 15.6).

The failure of conservative treatment is recognized when proper stretching is not achieved and there is a persistent pain associated with a structural rupture of the supraspinatus tendon. Surgical treatment is recommended when the tendon rupture reaches 50% of its transversal area, and the conservative treatment has failed (Fig. 15.7).

15.2.2.3 Labral Lesions

Labral lesions occur in contact sports leading to shoulder dislocation and labral avulsion (Bankart lesion), uncommon in Gym practitioners. However, like internal impingement, extreme motion range with the abduction and external rotation cause a traction of the insertion of the biceps long head called pell back movement. In this case, the posteroanterior and superior aspect of the labrum is unattached (SLAP lesion). Throwing exercises could also lead to this type of lesion [20] (Fig. 15.8).

Fig. 15.8 MRI of SLAP Lesion. (With permissions from Daniel Miranda Ferreira)

Nowadays, the treatment of SLAP lesion tends to be conservative. Patients with failed treatment could better be approached surgically. Likewise, when a SLAP lesion is associated with a paralabral cist that compresses the suprascapular nerve, a surgery may be required. Biceps tenodesis seems to have better clinical results and return to the sport than labral repair [21].

15.2.2.4 Scapular Dyskinesia

Scapular dyskinesia is defined by abnormal movements or position of the scapula. Its abnormal pattern can predispose to shoulder injuries. Several factors can lead to scapular dyskinesia.

Regarding gym athletes, changes in the relationship of the strength and tension of the muscles are the main cause.

Gym practitioners, like weightlifters, can present similar lesions to swimmers. For example, swimmers, especially in the propulsion phase, need to use the maximum power of the internal rotators. At this point, anterior serratus and trapezius have to act to avoid the scapula protraction. However, these protraction muscles can fatigue, causing a winning scapula [22]. Consequently, the physiopathology of the scapular dyskinesia is a result of an unbalance between the strength and stretching of the protractor and retractor muscles [23]. However, the diagnosis of scapular dyskinesia is not easily established in clinical practice.

Clinically, gym practitioners complain about pain and decrease of performance. A physical assessment performed from the posterior view of the patient's shoulder can help identify the abnormal position of the scapula, but palpation plays an essential role in the diagnosis. Positive trigger points may reveal muscle spasm. On the other hand, palpation of a weak muscle could indicate fatigue. Both show an imbalance of agonist or antagonist muscles [24].

The treatment of scapular dyskinesia is primarily conservative, and the key is to know the movement performed by the athletes very well. Afterwards, an adequate clinical examination may provide relevant information about spasm and fatigated muscles [24].

Finally, with this information, it is possible to correct the imbalance between the muscles, treat the pain, and increase the performance.

15.3 Hip

The anatomy and biomechanics of the pelvis and hip play an essential role to overcome the physical demands of sports [25]. The hip joint has a lower range of motion compared to other joints of the human body. The hip is a spheroidal diarthrosis adapted to absorb and transfer forces such as the ground reaction and the transmission of the kinetic energy generated in the axial skeleton to the lower and upper limbs [26, 27].

In sports, the loads applied to the hip may be up to eight times greater compared to the orthostatic position [28], resulted from its intrinsic joint stability and the influence of a powerful muscle surrounding this joint. Moreover, athletes are submitted to repeated high-intensity exercises with little or no recuperation time, predisposing them to injury.

In fitness exercising, physical training involves excessive hip lateral rotation associated with axial loads, hyperabduction and hyperflexion performed continually, such as kicking, changing of direction. Vigorous flexion and extension of the hip joint have been associated to a painful hip in athletes [26, 29].

In clinical practice, the sports physician must be aware that the origin of pelvic injury may be intra-articular and extra-articular. Usually, the extra-articular injuries result from repetitive overloading that causes inflammation, muscle injuries, tendinopathies, and bursitis. In contrast, intra-articular ones are related to the femoroacetabular impingement, acetabular rim injury (labrum), chondral injuries, synovitis and injuries of the round ligament. In some cases, these lesions can coexist since extra-articular disorders may appear as a result of intra-articular disorders [4, 5, 9]. However, the diagnosis may be difficult, most of the times, due to the anatomical and biomechanical particularities of the hip joint [30].

15.3.1 Epidemiology

The literature estimates that about 5–9% of athletes' injuries are located in the hip region, resulting from sports that require repetitive hip flexion, such as martial arts, CrossFit, weightlifting, and football [28] The advances of diagnostic imaging have shown that labral lesions of the hip are 73% related to femoroacetabular impingement, and this lesion is, in many cases, a prodrome of more severe injuries [31].

The increase in age, level of intensity, and competitiveness in sport are directly proportional to the rise of injuries and their complexity [32, 33].

15.3.2 Mechanism of Injury

In fitness modalities such as CrossFit, weightlifting, powerlifting, spinning, fighting, aerobic exercises, and running, repeatedly performed at high intensity, submit the hip joint and surrounding muscles to axial, translational or/and torsional loads [27, 34]. This biomechanical condition associated with muscle fatigue or inadequate sports movement may result in muscle injury, one of the most common events in these activities.

Athlete's hip pain or injury can also result from poor weight lifting technique, or supraphysiological hip movements, such as a movement of hip hyperabduction [34]. Along time, this overloading will result in intra-articular injuries and change the acetabular and femoral head morphology, causing a femoroacetabular impingement (FAI) [35], a common complaint of hip pain in CrossFitters, powerlifters, and weightlifters.

FAI is defined by bone overgrowth that is classified according to its location as Pincer (around the femoral head), CAM (along the acetabulum), and combined (around both) (Fig. 15.9).

15.3.3 Hip Muscle Injuries

Hip muscle injuries can have different degrees according to the speed and level of tension applied to the musculotendinous unit, and hamstring muscles are the most affected, especially in their proximal portion. This occurs due to the constant muscular demand during exercises of running and jumping, direction changes, acceleration, and deceleration movements performed at high speeds, and vigorous muscle force contraction. Moreover, hip flexor and adductor muscle injuries can also occur, mainly with starting movements and sudden changes of direction [27].

Pubalgia is another clinical condition that causes pain to the pelvis region. It is related to overtraining condition and adductor and abdominal rectus muscle imbalance, prevalent in male, footballers, and runners.

A differential diagnosis that should be investigated in case of hip and pelvic pain is the stress fracture pathology. In this condition, a possible target population is the long-distance runner, especially women. Again, there is an overtraining con-

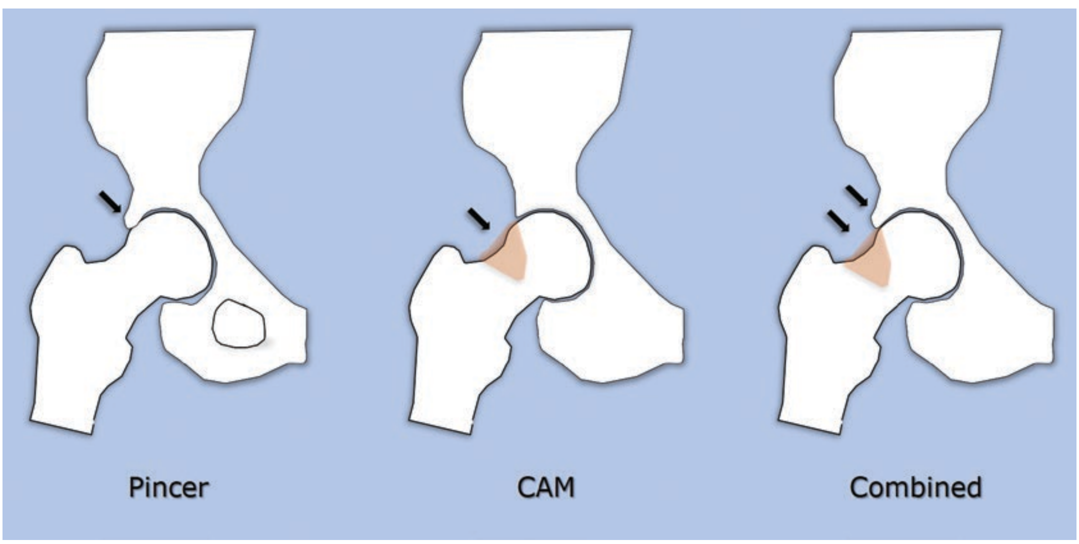

Fig. 15.9 Types of Femoroacetabular impingement (FAI), and the arrows showing the location of bone overgrowth. (Courtesy: Sergio Rocha Piedade)

dition that exceeds the bone ability to overcome the mechanical stress. In 8% of the cases, the fractures occurs in the proximal femur [34, 36].

15.3.3.1 Prevention

Early osteoarthritis of the hip joint is directly related to femoroacetabular impingement. The paramount prevention consists of avoiding great amplitudes of movements (hyperabduction and hyperflexion), in the different axes, which should not be painful but limited to the individual characteristics of each athlete. Moreover, prevention involves performing prior preparation for physical activities (global strengthening) and decreasing the intensity of physical activities, especially in corridors and jumping movements, which can overload static and dynamic stabilizers [34]. Therefore, to succeed the mechanical demands imposed by sports activity, the athlete must be aware of hip muscle strengthening and stretching, intensity and frequency of appropriately monitored training to avoid a scenario to injury.

15.3.4 Treatment of FAI

Clinical treatment is considered in patients that are in constant pain, and it involves guidelines regarding physical activities, posture, analgesia, muscle strengthening, anti-inflammatory and chondroprotective agents, to avoid clinical/radiological worsening.

Surgical treatment can be performed by open or arthroscopic means. In the open procedure, a controlled hip dislocation is performed to assess the labral repairs, free body removal, osteoplasty of the acetabulum and proximal femur. In the arthroscopic procedure, a videoarthroscopy is used for the labral repair (debridement, suture, and reconstruction), osteoplasty of the proximal femur and regularization of the acetabulum edge, a less invasive technique that optimizes the patient's recovery.

15.4 Knee

Knee pain is a common complaint in CrossFitters and indoor cyclers (spinning) and biomechanical conditions such as muscle imbalance, poorly adapted bodybuilding, overtraining, inadequate stretching are the leading causes [37, 38]. In clinical practice, the main diagnoses in this population are patellofemoral dysfunction, patellar tendonitis, and iliotibial band syndrome.

15.4.1 Patellofemoral Dysfunction

Differently from knee ligament injuries, the patellofemoral dysfunction presents a biomechanical imbalance with no history of trauma. Clinically, at the initial stages; the athletes may complain of anterior knee pain and discomfort during or after training [39]. However, it could start by crepitus during the patella tracking and no pain. Along time, they report discomfort on the anterior aspect of the knee and, progressively, the knee pain increases while physical performance decreases.

In the physical examination, the physician should be aware of the edema or effusion of knee, status of patella tracking, knee extension deficit, knee pain localization, muscle imbalance such as muscle fasciculation during knee flexion-extension movements, weak hamstring stretching, and decreased range of motion of internal hip rotators [40, 41].

Prevention and treatment focus on the quadriceps muscle balance, a critical point to achieve a well-functioning and efficient patella tracking. The intensity of training, frequency, duration, as well as the speed of training program development, should be investigated and reviewed if and when necessary. Moreover, pain relief, physiotherapy, adapting the training program, and reinforcing the importance of stretching to the athlete are the main therapeutic strategies.

15.4.2 Iliotibial Band Syndrome

Overuse tendonitis may affect mainly cyclers due to the repetitive stress of iliotibial band to the lateral femoral condyle during cycling exercises as well as long-distance runners. Most of the times, the diagnosis is based on the athlete's history, complaints, and physical assessment [42].

In the initial stages, the pain is commonly located on the lateral side of the knee and triggered by physical exercises. In the physical examination, the pain may be elicited moving the knee in flexion and extension and palpating and compressing over the lateral epicondyle at 30° of knee flexion and internally rotated. Moreover, in chronic cases, complaints of lateral knee pain and discomfort may be reported, even when descending or ascending stairs [43].

The strategy of treatment involves rest, physiotherapy focusing on stretching and strengthening exercises, pain relief, and massage. The use of anti-inflammatory medication and analgesics must be carefully managed for a brief period, 4–7 days maximum, because the level of pain is a criterion to review and define the training plan. Surgical treatment may be considered just in cases of failure of conservative treatment. It consists of partial resection of the iliotibial band in its posterior edge and bursa.

15.4.3 Patellar Tendinosis

Tendons are highly specialized structures to control joint movement and load absorption. However, in repetitive activities of running, jumping, squatting, and cycling, their physiological threshold may be exceeded, stressing their structure, modifying the extracellular matrix, and causing patellar tendinosis due to mechanical overloading [44].

Like any overuse injury, in patellar tendonitis, the tendon microtears do not heal properly because of an inadequate tissue biological response that may be followed by a progressive degeneration process of the tendon [45].

The etiology of patellar tendinosis may involve intrinsic issues such as age, sex, overweight, muscle imbalance, poor stretching, anatomical factors, and extrinsic factors intensity of training, time of practicing, equipment, inappropriate sporting gesture. Moreover, the athlete must be aware that exercises of eccentric muscle contraction, such as squatting, submit the tendons to higher tensions and, therefore, these exercises should be executed properly to avoid injuries [46].

The pain may be reported in the patellar tendon body and its bone insertions, proximally to the inferior pole of the patella, and distally to the tibial tubercle.

Ultrasonography and MRI (Fig. 15.10) are useful to investigate the extension of tendon injury and degeneration, especially in chronic cases [47].

Most patients will respond to conservative measures of physiotherapy, stretching and strengthening exercises, massage, and anti-inflammatory medication and analgesics, and changes in the training plan. The surgical treatment should be indicated in cases of motived patients that have a failed conservative treatment and may include tenotomy, resection of degenerate tissue, resection of the inferior pole of the patella [48].

15.5 Shin Pain

It is an overuse injury associated with high-intense physical activities and endurance sports. The athlete complains of pain and lower leg discomfort along with the inner board of the tibial diaphysis, front outside or medial side of the lower leg. A history of leg pain that was triggered after an intensive period of training and competitions is often reported and is characterized by an overtraining cycle. The differential diagnosis may include tendonitis, stress fracture, chronic exertional compartment syndrome, and should be investigated [49].

Once the diagnosis of shin pain has been established, the physician must guide the athlete about the importance of reducing or even stop training while the symptoms remain and impact their level of training negatively. The treatment is focused on rest, physiotherapy, stretching exercises. Moreover, it aims to prevent the injury to progress to major ones such as stress fracture. Therefore, the physician should be aware of the intensity and frequency of patient's complains. Pain is the clinical sign that will guide the treatment. Pain reduction is a good sign of treatment evolution and also a useful parameter to monitor it.

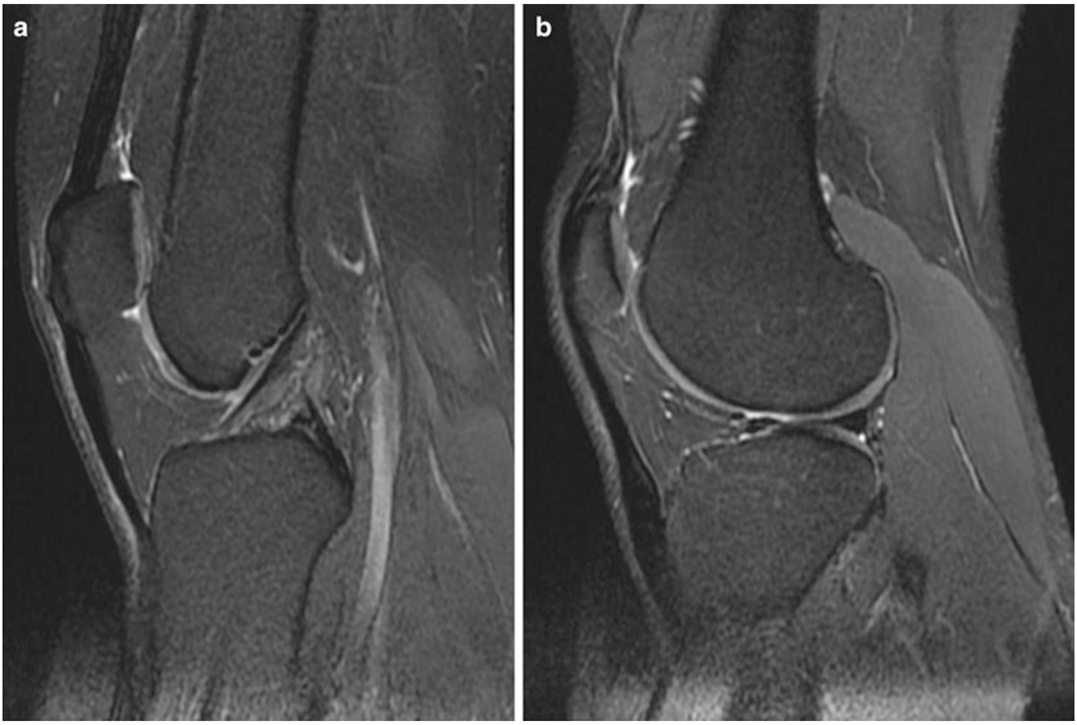

Fig. 15.10 MRI of the knee—(**a**) Sagittal DP Fat Sat. View showing fusiform thickening of the proximal segment of the patellar tendon (Patellar tendinosis), and (**b**) thickening of the distal segment of the patellar tendon at its insertion in the anterior tibial tuberosity (patellar tendinosis). (With permissions from Daniel Miranda Ferreira)

15.6 Achilles Tendinopathy

Achilles tendon is the strongest one in the body and, like any other tendon, it has a biomechanical behavior. Due to a different individual threshold, even during adequate training, the physical strain might have a positive effect, but also a negative deleterious effect on the tendon, leading to pathologic degenerative changes [50].

It is particularly relevant in high-level sports, individual or team sports due to the frequency of loads and eccentric work. The injuries can involve the tendon body, paratenon or surrounding bursas, and are divided into insertional and non-insertional ones [51].

15.6.1 Mid-Portion Achilles Tendinopathy

This clinical syndrome is characterized by localized or diffuse pain and swelling, between 2 and 7 cm from the calcaneal insertion, causing impaired performance [51]. The careful and systematic clinical evaluation of Achilles pathology is mandatory (Fig. 15.11). Histopathological diagnosis is not limited to tendinosis, and it should be noted that the fundamental lesion of tendinopathy is failed healing response [51].

15.6.1.1 Treatment for Non-insertional Achilles Tendinopathy

Treatment for non-insertional Achilles tendinopathy is largely conservative, starting with rest, modification of training regimes, specific exercises, and correction of underlying lower limb alignment issues with orthotics are some of the treatment modalities. Eccentric strength training is commonly regarded as a key element in the treatment of this disorder. The use of customized foot orthotics has also been disputed, as these seem to be less effective than sham foot orthotics in patients undergoing an eccentric exercise program [52].

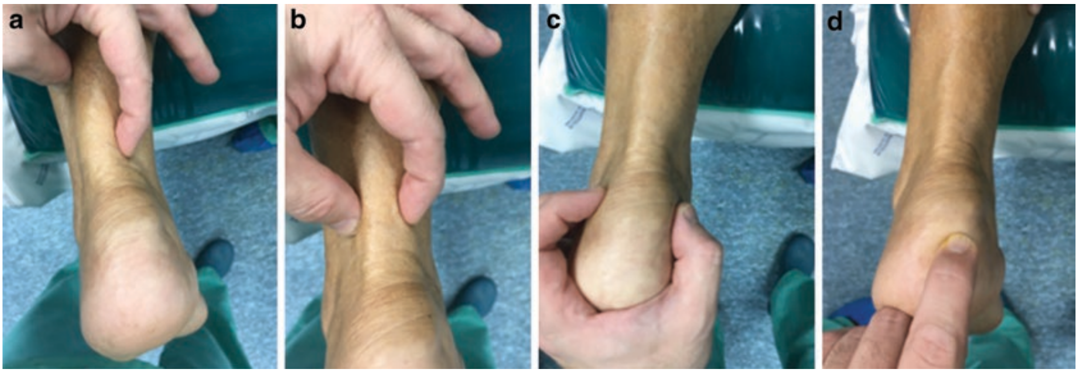

Fig. 15.11 Systematic palpation of different zones during Achilles clinical evaluation. (With permissions from Helder Pereira)

Injection therapies can also be effective treatments, and platelet-rich plasma (PRP) injections also produce good overall results, with a stable outcome at medium- and long-term follow-up [53–55]. The stromal vascular fraction extracted from adipose tissue can also be safe and effective in the treatment of recalcitrant tendinopathy. Paratendinous injections of autologous blood showed no benefits when administered in combination with an eccentric training program [56].

15.6.1.2 Conventional Surgical Treatments

Conventional surgical treatments entail the release of adhesions, which may be combined with resection of the paratenon. If using an open approach, a longitudinal tenotomy can be performed and macroscopic areas of tendinopathic areas tissue removed. Additional multiple tenotomies can be performed to initiate vascular ingrowth and a healing process. Stripping of the paratenon is thought to be effective by removing neovascularization and denervating the diseased area of the tendon [57].

15.6.2 Achilles Paratendinopathy

Paratendinopathy is defined as inflammation, either acute or chronic, with or without degeneration, of the thin membrane surrounding the Achilles tendon [51]. Most important clinical features are pain induced by exercise and swelling around the mid-portion of the tendon. Acute cases will have swelling and crepitation, with the latter not being present in mid-portion Achilles tendinopathy. Swelling and crepitations will be less pronounced in chronic cases. Typically, in patients who have acute symptoms, the area of swelling and tenderness does not move when the ankle joint is dorsiflexed [58]. It should be noted that Achilles paratendinopathy frequently coexists with mid-portion tendinopathy.

15.6.2.1 Treatment

Initial nonoperative management aims to identify and correct predisposing factors, like the ones in tendinopathy cases. Brisement has proven to be helpful in the treatment of paratendinopathy [59]. This procedure consists of taking a dilute anesthetic and injecting it into the tendon sheath to break up adhesions. *If surgical treatment is warranted*, an endoscopic approach can be used to attain release of adhesions and resection of the paratenon, similarly to what was described above for mid-portion Achilles tendinopathy [60].

15.6.3 Insertional Achilles Tendinopathy

This disorder tends to affect more active people [61]. Clinically, there is pain on palpation at the tendon insertion, swelling may be present, and a bony spur may be palpable. There are intrinsic risk factors such as structural or biomechanical foot, ankle, and lower extremity conditions. While the extrinsic ones, although anecdotal and lacking scientific evidence, are overtraining, improper stretching/preparation, shoe gear, obesity, age, and mechanical overload [62]. Several studies have suggested that the calcification seen in these individuals is related to stress shielding forces [63–65]. This adaptation increases the surface area at the bone-tendon junction, thereby protecting this area from increased mechanical loads.

15.6.3.1 Treatment

Nonoperative treatment includes eccentric stretching and strength training and heel lifts. Platelet-rich plasma and high molecular weight hyaluronic acid injections have also been shown to be beneficial in insertional Achilles tendinopathy [66, 67]. *When conservative measures fail*, surgical treatment is indicated. This can be achieved through a central incision with complete detachment of the Achilles tendon, debridement of diseased tissue, removal of spurs, and reattachment with the suture bridge technique. Achilles insertional tendinopathy surgery results in few complications with good functional results if the surgical technique is adapted to the type of tendon injury [68].

15.6.4 Retrocalcaneal Bursitis

It is a visible and painful soft tissue swelling, on the medial and lateral sides of Achilles tendon at the same level of the posterosuperior calcaneus. Patients with prominent posterior superior calcaneal tuberosities can stay asymptomatic all life, and patients with a normal calcaneus can have recalcitrant symptoms. Patients with hindfoot varus and cavus foot have a higher predisposition for this disorder [69, 70]. Repeated dorsiflexion of the ankle pushes the Achilles tendon anteriorly, compressing the bursa against the calcaneus. Some cases can happen in conjunction with insertional Achilles tendinopathy [71]. This condition is often of idiopathic origin but can also be due to inflammatory arthropathy or infection.

15.6.4.1 Treatment

Conservative treatment includes multiple physical therapy protocols. Shoe changes or modifications, especially in athletes during the competitive season. *Corticosteroid injections should be avoided* in retrocalcaneal bursitis because of a connection between the retrocalcaneal bursa and the anterior fibers of the Achilles tendon, which puts the tendon at risk of rupture [72].

Several surgical treatments have been proposed with similar success rates [73]: endoscopic calcaneoplasty and dorsal wedge or Zadek osteotomy, and the minimally invasive percutaneous approach as described by Mariano de Prado [74]. When retrocalcaneal bursitis and insertional Achilles tendinopathy coexist, the use of the central tendon-splitting approach appears to be safe and satisfactory [71].

15.6.5 Superficial Calcaneal Bursitis

It is an inflammation of an adventitious bursa between the posterior aspect of the calcaneus or Achilles tendon and surrounding skin [51], usually without involvement of the Achilles tendon. This bursa is acquired after birth as a response to friction, being frequently associated with a rigid shoe counter or poorly fitting shoes [75]. The athletes must be particularly careful with the choice of adequate shoes and equipment. Most patients with microtraumatic superficial bursitis respond to conservative management, which includes ice, elevation, activity modification, appropriate padding, compression wraps, and over-the-counter analgesics [76].

15.6.6 Achilles Rupture

The rupture of the Achilles tendon, particularly in a high-level athlete, is a serious condition, and

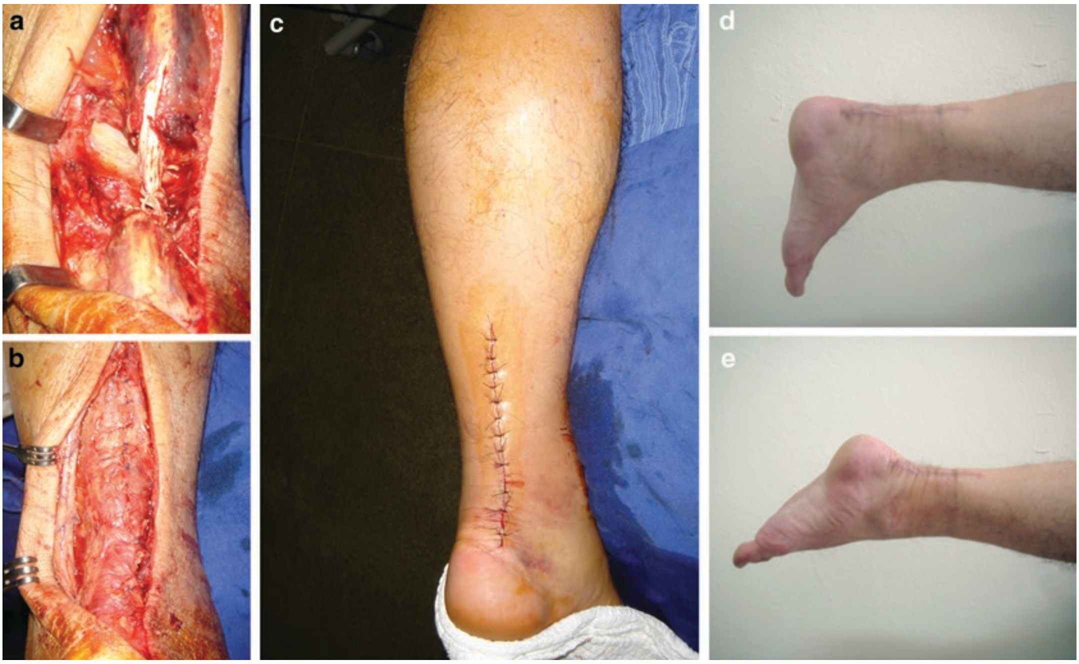

Fig. 15.12 An open approach to Achilles tendon injury (**a**) tendon injury and (**b**) repaired tendon, (**c**) surgical scar, and (**d**) and (**e**) full-recovery of Achilles tendon function at 3 months postoperative in 35-year-old male CrossFitter. (Courtesy: Sergio Rocha Piedade)

despite the positive outcome reported in all forms of treatment, from conservative, open (Fig. 15.12) minimally invasive (Fig. 15.13), it can lead to accelerating the end of competitive career [77]. Age and athletes, expectations do play a role in this setting [77].

Achilles tendon ruptures are usually severe injuries in athletes, with return-to-sports rates around 70% and the risk for lowering performance post-injury must be considered. In athletes, there is a global trend in favor of surgical treatment, once conservative treatment has been linked to some loss of function and force [77].

More recently, we have seen several advances in this field, including minimally invasive approaches such as the endoscopic transfer of *Flexor Hallucis Longus* (Fig. 15.13d) promising to enhance the biology repair with minimal comorbidities and faster return to activity [78].

15.6.6.1 Principles of Prevention and Rehabilitation

Despite some external factor related to the shoe wear, training conditions, specificities of individual sports disciplines, understanding in each moment the global fatigue level of the athlete and his recovery capacity from the implemented training program is critical to lower the injury risk [77, 79].

However, we must assume that, at the moment, there is no effective way to predict which athlete presents a higher risk for Achilles pathology, besides those linked with intrinsic anatomic factors such as poor gastrocnemius-soleus flexibility, overpronation, overweight, inflammatory diseases, or extrinsic factors like a sudden increase in training intensity, change in running/training surface, worn out or inappropriate footwear or medications (quinolones, statines) [79].

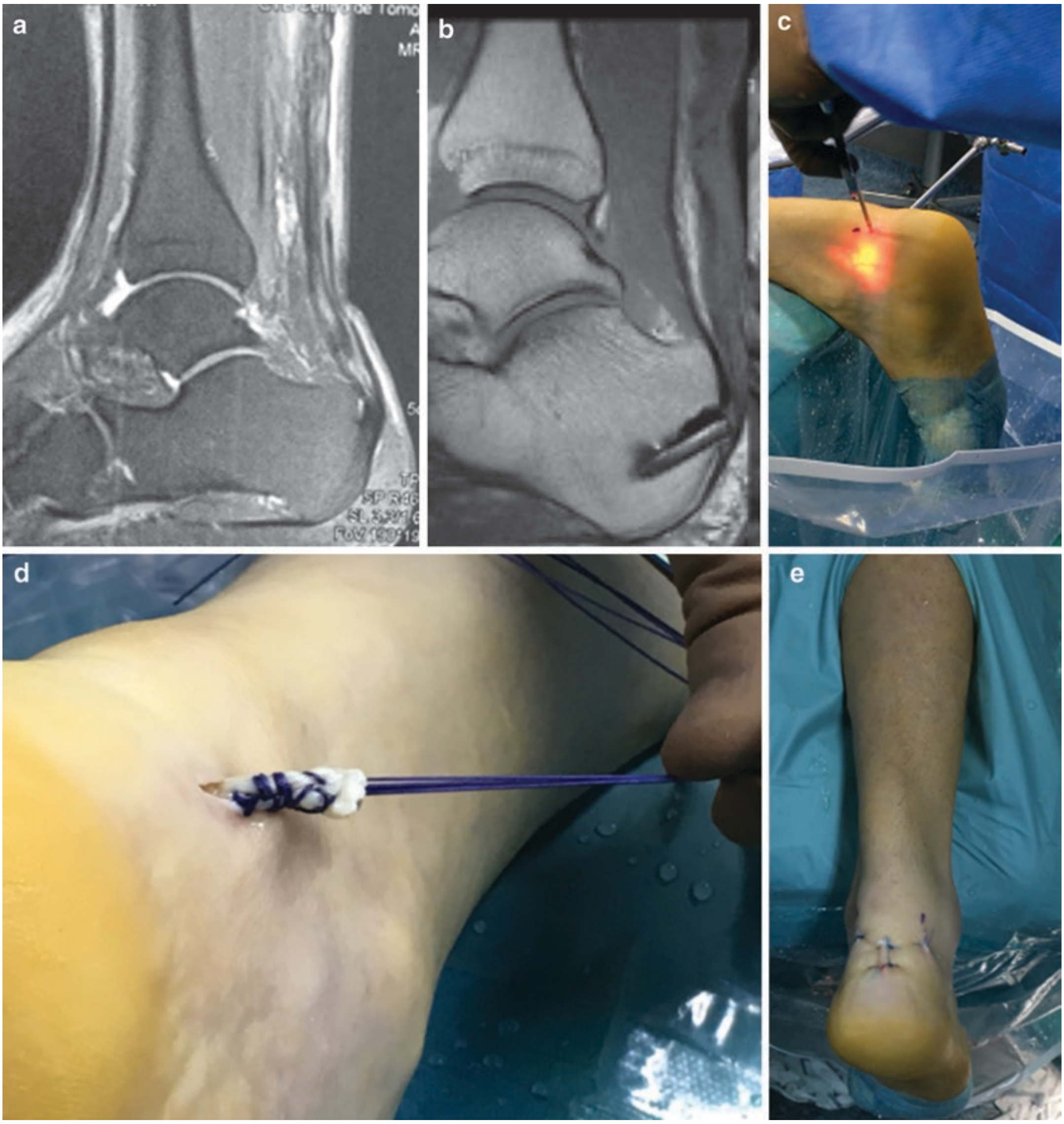

Fig. 15.13 (**a**) MRI showing complex Achilles rupture; (**b**) MRI after Flexor Hallucis Longus Transfer (FHL); (**c**) Endoscopic approach; (**d**) the harvested FHL retrieved on the endoscopic portal; (**e**) Final external look of the three portal technique for FHL endoscopic transfer. (With permissions from Helder Pereira)

Concerning the conservative treatment of several pathologies, shock wave therapy, percutaneous intra-tissue electrolysis, and biologics have been increasing their relevance [79].

Elongation of posterior chains to increase gastrocnemius-soleus flexibility, neuromuscular training to improve proprioception and strengthening exercises, adequate to the involved discipline are of paramount relevance in both prevention and rehabilitation programs [77, 79, 80].

Treatment of these disorders is largely conservative. If surgical treatment is indicated, many technical options are described in the literature.

Take Home Messages

- Although CrossFit, spinning, aerobic exercises promote rewarding physical and mental well-being, they are not free from injuries due to the high-intense functional training performed in these activities.
- The underlying conditions, such as muscle imbalance, poorly adapted bodybuilding, overtraining, inadequate stretching are the leading causes of joint and muscle pain and discomfort, and injuries.
- Athletes must be aware of symptoms of pain and discomfort that have started after a fast progression in the intensity and frequency of training.
- In the hip, the origin of extra-articular injuries is commonly related to repetitive overloading, while intra-articular ones are related to FAI, acetabular injury, chondral lesions, synovitis, and so on.
- Even during adequate training, the physical strain may cause negative deleterious effect on the tendon, such as the Achilles tendon, due to a different individual threshold.
- The history of the athlete's complaint, physical training, its intensity and frequency and speed of changes in the program helps to define the therapeutic approach.
- The program of sports training should be developed regarding the athlete's physical and mental threshold.

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References

1. Warburton DER, Nicol CW, Bredin SSD. Health benefits of physical activity: the evidence. CMAJ. 2006;174:801–9. https://doi.org/10.1503/cmaj.051351.
2. Whiteman-Sandland J, Hawkins J, Clayton D. The role of social capital and community belongingness for exercise adherence: an exploratory study of the CrossFit gym model. J Health Psychol. 2018;23:1545–56. https://doi.org/10.1177/1359105316664132.
3. Ulseth AB. Social integration in modern sport: commercial fitness centres and voluntary sports clubs. Eur Sport Manag Q. 2004;4:95–115. https://doi.org/10.1080/16184740408737471.
4. Fisher J, Sales A, Carlson L, Steele J. A comparison of the motivational factors between CrossFit participants and other resistance exercise modalities: a pilot study. J Sports Med Phys Fitness. 2017;57:1227–34. https://doi.org/10.23736/S0022-4707.16.06434-3.
5. Milanović Z, Sporiš G, Weston M. Effectiveness of high-intensity interval training (HIT) and continuous endurance training for VO2max improvements: a systematic review and meta-analysis of controlled trials. Sport Med. 2015;45:1469–81. https://doi.org/10.1007/s40279-015-0365-0.
6. Chavarrias M, Carlos-Vivas J, Collado-Mateo D, Pérez-Gómez J. Health benefits of indoor cycling: a systematic review. Medicina. 2019;55:452. https://doi.org/10.3390/medicina55080452.
7. Soriano MA, Suchomel TJ, Comfort P. Weightlifting overhead pressing derivatives: a review of the literature. Sport Med. 2019;49:867–85. https://doi.org/10.1007/s40279-019-01096-8.
8. Glassman G. Understanding CrossFit. CrossFit J. 2007;56:1–2.
9. Eather N, Morgan PJ, Lubans DR. Improving health-related fitness in adolescents: the CrossFit Teens™ randomized controlled trial. J Sports Sci. 2016;34:209–23. https://doi.org/10.1080/02640414.2015.1045925.
10. Moran S, Booker H, Staines J, Williams S. Rates and risk factors of injury in CrossFitTM: a prospective cohort study. J Sports Med Phys Fitness. 2017;57:1147–53. https://doi.org/10.23736/S0022-4707.16.06827-4.
11. de Queiroz Szeles PR, da Costa TS, da Cunha RA, Hespanhol L, de Castro Pochini A, Ramos LA, et al. CrossFit and the epidemiology of musculoskeletal injuries: a prospective 12-week cohort study. Orthop J Sport Med. 2020;8:232596712090888. https://doi.org/10.1177/2325967120908884.

12. Halder AM, Itoi E, An KN. Anatomy and biomechanics of the shoulder. Orthop Clin North Am. 2000;31:159–76. https://doi.org/10.1016/S0030-5898(05)70138-3.
13. Lemos MJ. The evaluation and treatment of the injured acromioclavicular joint in athletes. Am J Sports Med. 1998;26:137–44. https://doi.org/10.1177/03635465980260010801.
14. Haupt HA. Upper extremity injuries associated with strength training. Clin Sports Med. 2001;20:481–90. https://doi.org/10.1016/S0278-5919(05)70264-7.
15. Dinnes J, Loveman E, McIntyre L, Waugh N. The effectiveness of diagnostic tests for the assessment of shoulder pain due to soft tissue disorders: a systematic review. Health Technol Assess. 2003; https://doi.org/10.3310/hta7290.
16. Chen RE, Voloshin I. Long head of biceps injury: treatment options and decision making. Sports Med Arthrosc. 2018;26:139–44. https://doi.org/10.1097/JSA.0000000000000206.
17. Keogh JWL, Winwood PW. The epidemiology of injuries across the weight-training sports. Sport Med. 2017;47:479–501. https://doi.org/10.1007/s40279-016-0575-0.
18. Summitt RJ, Cotton RA, Kays AC, Slaven EJ. Shoulder injuries in individuals who participate in CrossFit training. Sports Health. 2016;8:541–6. https://doi.org/10.1177/1941738116666073.
19. Keller RA, De Giacomo AF, Neumann JA, Limpisvasti O, Tibone JE. Glenohumeral internal rotation deficit and risk of upper extremity injury in overhead athletes: a meta-analysis and systematic review. Sports Health. 2018;10:125–32. https://doi.org/10.1177/1941738118756577.
20. Morse KW, Eno JJ, Altchek DW, Dines JS. Injuries of the biceps and superior labral complex in overhead athletes. Curr Rev Musculoskelet Med. 2019;12:72. https://doi.org/10.1007/s12178-019-09539-5.
21. Hurley ET, Fat DL, Duigenan CM, Miller JC, Mullett H, Moran CJ. Biceps tenodesis versus labral repair for superior labrum anterior-to-posterior tears: a systematic review and meta-analysis. J Shoulder Elb Surg. 2018;27:1913–9. https://doi.org/10.1016/j.jse.2018.04.011.
22. Heinlein SA, Cosgarea AJ. Biomechanical considerations in the competitive swimmer's shoulder. Sports Health. 2010;2:519–25. https://doi.org/10.1177/1941738110377611.
23. Tooth C, Schwartz C, Croisier J-L, Stephen B, Brüls O, Denoël V, et al. Activation profile of scapular stabilizing muscles in asymptomatic people. Does scapular dyskinesis have an impact on its? Am J Phys Med Rehabil. 2020; https://doi.org/10.1097/phm.0000000000001446.
24. Panagiotopoulos AC, Crowther IM. Scapular dyskinesia, the forgotten culprit of shoulder pain and how to rehabilitate. SICOT-J. 2019;5:29. https://doi.org/10.1051/sicotj/2019029.
25. Braly BA, Beall DP, Martin HD. Clinical examination of the athletic hip. Clin Sports Med. 2006;25:199–210. https://doi.org/10.1016/j.csm.2005.12.001.
26. Murphy DF, Connolly DAJ, Beynnon BD. Risk factors for lower extremity injury: a review of the literature. Br J Sports Med. 2003;37:13–29. https://doi.org/10.1136/bjsm.37.1.13.
27. Hebert S, de Barros Filho TEP, Xavier R, Pardini AG Jr. Ortopedia e traumatologia: princípios e prática. 3rd ed. Artmed Editora: Porto Alegre; 2017.
28. Anderson K, Strickland SM, Warren R. Hip and groin injuries in athletes. Am J Sports Med. 2001;29:521–33. https://doi.org/10.1177/03635465010290042501.
29. Lynch SA, Renström PAFH. Groin injuries in sport. Treatment strategies. Sport Med. 1999;28:137–44. https://doi.org/10.2165/00007256-199928020-00006.
30. Renström P, Peterson L. Groin injuries in athletes. Br J Sports Med. 1980;14:30–6. https://doi.org/10.1136/bjsm.14.1.30.
31. Kang C, Hwang DS, Cha SM. Acetabular labral tears in patients with sports injury. Clin Orthop Surg. 2009;1:230–5. https://doi.org/10.4055/cios.2009.1.4.230.
32. Matsudo SM, Matsudo VKR, Barros Neto TL. Atividade física e envelhecimento: aspectos epidemiológicos. Rev Bras Med Do Esporte. 2001;7:2–13. https://doi.org/10.1590/s1517-86922001000100002.
33. da Costa TS, Louzada CTN, Miyashita GK, da Silva PHJ, Sungaila HYF, Lara PHS, et al. CrossFit®: injury prevalence and main risk factors. Clinics. 2019;74:e1402. https://doi.org/10.6061/clinics/2019/e1402.
34. Motta Filho GR, de Barros Filho TEP. Ortopedia e Traumatologia. 1st ed. Rio de Janeiro: Elsevier; 2017.
35. Zadpoor AA. Etiology of femoroacetabular impingement in athletes: a review of recent findings. Sports Med. 2015;45:1097. https://doi.org/10.1007/S40279-015-0339-2.
36. De Queiroz RD, De Carvalho RT, Szeles PRDQ, Janovsky C, Cohen M. Retorno ao esporte após tratamento cirúrgico de pubeíte em jogadores de futebol profissional. Rev Bras Ortop. 2014;49:233–9. https://doi.org/10.1016/j.rbo.2013.10.001.
37. Gean RP, Martin RD, Cassat M, Mears SC. Systematic review and meta-analysis of injury in CrossFit. J Surg Orthop Adv. 2020;29:26. https://doi.org/10.3113/jsoa.2020.0026.
38. Montalvo AM, Shaefer H, Rodriguez B, Li T, Epnere K, Myer GD. Retrospective injury epidemiology and risk factors for injury in CrossFit. J Sport Sci Med. 2017;16:53–9.
39. Giordano BD, Weisenthal BM. Common injuries and conditions in CrossFit participation. In: Miller TL, editor. Endurance. Sports medicine. A clinical guide. Cham: Springer International Publishing; 2016. p. 147–58. https://doi.org/10.1007/978-3-319-32982-6_12.
40. Manske RC, Davies GJ. Examination of the patellofemoral joint. Int J Sports Phys Ther. 2016;11:831–53.
41. Bronstein RD, Schaffer JC. Physical examination of the knee: meniscus, cartilage, and patellofemoral

conditions. J Am Acad Orthop Surg. 2017;25:365–74. https://doi.org/10.5435/JAAOS-D-15-00464.
42. Strauss EJ, Kim S, Calcei JG, Park D. Lliotibial band syndrome: evaluation and management. J Am Acad Orthop Surg. 2011;19:728–36. https://doi.org/10.5435/00124635-201112000-00003.
43. Khaund R, Flynn SH. Iliotibial band syndrome: a common source of knee pain. Am Fam Physician. 2005;71:1545–50.
44. Hamilton B, Purdam C. Patellar tendinosis as an adaptive process: a new hypothesis. Br J Sports Med. 2004;38:758–61. https://doi.org/10.1136/bjsm.2003.005157.
45. Pecina M, Bojanic I, Ivkovic A, Brcic L, Smoljanovic T, Seiwerth S. Patellar tendinopathy: histopathological examination and follow-up of surgical treatment. Acta Chir Orthop Traumatol Cechoslov. 2010;77:277–83.
46. Lim HY, Wong SH. Effects of isometric, eccentric, or heavy slow resistance exercises on pain and function in individuals with patellar tendinopathy: a systematic review. Physiother Res Int. 2018;23:e1721. https://doi.org/10.1002/pri.1721.
47. Khan KM, Bonar F, Desmond PM, Cook JL, Young DA, Visentini PJ, et al. Patellar tendinosis (jumper's knee): findings at histopathologic examination, US, and MR imaging. Radiology. 1996;200:821–7. https://doi.org/10.1148/radiology.200.3.8756939.
48. Figueroa D, Figueroa F, Calvo R. Patellar tendinopathy: diagnosis and treatment. J Am Acad Orthop Surg. 2016;24:e184–92. https://doi.org/10.5435/JAAOS-D-15-00703.
49. Piedade SR, Mendes LA, Carvalho LM, dos RMF S, Marques LC, Ferreira DM. Shin pain. In: Rocha Piedade S, Imhoff AB, Clatworthy M, Cohen M, Espregueira-Mendes J, editors. The sports medicine physician. Cham: Springer International Publishing; 2019. p. 211–21. https://doi.org/10.1007/978-3-030-10433-7_16.
50. Louis-Ugbo J, Leeson B, Hutton WC. Tensile properties of fresh human calcaneal (Achilles) tendons. Clin Anat. 2004;17:30–5. https://doi.org/10.1002/ca.10126.
51. van Dijk CN, van Sterkenburg MN, Wiegerinck JI, Karlsson J, Maffulli N. Terminology for Achilles tendon related disorders. Knee Surg Sport Traumatol Arthrosc. 2011;19:835–41. https://doi.org/10.1007/s00167-010-1374-z.
52. Paavola M, Kannus P, Paakkala T, Pasanen M, Järvinen M. Long-term prognosis of patients with achilles tendinopathy: an observational 8-year follow-up study. Am J Sports Med. 2000;28:634–42. https://doi.org/10.1177/03635465000280050301.
53. Munteanu SE, Scott LA, Bonanno DR, Landorf KB, Pizzari T, Cook JL, et al. Effectiveness of customized foot orthoses for Achilles tendinopathy: a randomized controlled trial. Br J Sports Med. 2015;49:989–94. https://doi.org/10.1136/bjsports-2014-093845.
54. Chaudhry FA. Effectiveness of dry needling and high-volume image-guided injection in the management of chronic mid-portion Achilles tendinopathy in adult population: a literature review. Eur J Orthop Surg Traumatol. 2017;27:441–8. https://doi.org/10.1007/s00590-017-1957-1.
55. Guelfi M, Pantalone A, Vanni D, Abate M, Guelfi MGB, Salini V. Long-term beneficial effects of platelet-rich plasma for non-insertional Achilles tendinopathy. Foot Ankle Surg. 2015;21:178–81. https://doi.org/10.1016/j.fas.2014.11.005.
56. Salini V, Vanni D, Pantalone A, Abate M. Platelet rich plasma therapy in non-insertional Achilles tendinopathy: the efficacy is reduced in 60-years old people compared to young and middle-age individuals. Front Aging Neurosci. 2015;7:228. https://doi.org/10.3389/fnagi.2015.00228.
57. Sobhani S, Dekker R, Postema K, Dijkstra PU. Epidemiology of ankle and foot overuse injuries in sports: a systematic review. Scand J Med Sci Sport. 2013;23:669–86. https://doi.org/10.1111/j.1600-0838.2012.01509.x.
58. Steenstra F, van Dijk CN. Achilles tendoscopy. Foot Ankle Clin. 2006;11:429–38. https://doi.org/10.1016/j.fcl.2006.02.001.
59. Paavola M, Järvinen TAH. Paratendinopathy. Foot Ankle Clin. 2005;10:279–92. https://doi.org/10.1016/j.fcl.2005.01.008.
60. Roche AJ, Calder JDF. Achilles tendinopathy: a review of the current concepts of treatment. Bone Joint J. 2013;95B:1299–307. https://doi.org/10.1302/0301-620X.95B10.31881.
61. Uquillas CA, Guss MS, Ryan DJ, Jazrawi LM, Strauss EJ. Everything Achilles: knowledge update and current concepts in management: AAOS exhibit selection. J Bone Joint Surg Am. 2015;97:1187–95. https://doi.org/10.2106/JBJS.O.00002.
62. Irwin TA. Current concepts review: insertional Achilles tendinopathy. Foot Ankle Int. 2010;31:933–9. https://doi.org/10.3113/FAI.2010.0933.
63. Waldecker U, Hofmann G, Drewitz S. Epidemiologic investigation of 1394 feet: coincidence of hindfoot malalignment and Achilles tendon disorders. Foot Ankle Surg. 2012;18:119–23. https://doi.org/10.1016/j.fas.2011.04.007.
64. Caudell GM. Insertional Achilles tendinopathy. Clin Podiatr Med Surg. 2017;34:195–205. https://doi.org/10.1016/j.cpm.2016.10.007.
65. Lyman J, Weinhold PS, Almekinders LC. Strain behavior of the distal Achilles tendon: implications for insertional Achilles tendinopathy. Am J Sports Med. 2004;32:457–61. https://doi.org/10.1177/0095399703258621.
66. Al-Abbad H, Simon JV. The effectiveness of extracorporeal shock wave therapy on chronic Achilles tendinopathy: a systematic review. Foot Ankle Int. 2013;34:33–41. https://doi.org/10.1177/1071100712464354.
67. Kumai T, Muneta T, Tsuchiya A, Shiraishi M, Ishizaki Y, Sugimoto K, et al. The short-term effect after a single injection of high-molecular-weight hyaluronic acid in patients with enthesopathies (lateral epicondy-

litis, patellar tendinopathy, insertional Achilles tendinopathy, and plantar fasciitis): a preliminary study. J Orthop Sci. 2014;19:603–11. https://doi.org/10.1007/s00776-014-0579-2.
68. Beitzel K, Mazzocca AD, Obopilwe E, Boyle JW, McWilliam J, Rincon L, et al. Biomechanical properties of double- and single-row suture anchor repair for surgical treatment of insertional Achilles tendinopathy. Am J Sports Med. 2013;41:1642–8. https://doi.org/10.1177/0363546513487061.
69. Gurdezi S, Kohls-Gatzoulis J, Solan MC. Results of proximal medial gastrocnemius release for Achilles tendinopathy. Foot Ankle Int. 2013;34:1364–9. https://doi.org/10.1177/1071100713488763.
70. Tallerico VK, Greenhagen RM, Lowery C. Isolated gastrocnemius recession for treatment of insertional Achilles tendinopathy: a pilot study. Foot Ankle Spec. 2015;8:260–5. https://doi.org/10.1177/1938640014557077.
71. van Dijk CN. Ankle arthroscopy: techniques developed by the Amsterdam Foot and Ankle School. Berlin: Springer; 2014. https://doi.org/10.1007/978-3-642-35989-7.
72. Van Sterkenburg MN, Muller B, Maas M, Sierevelt IN, Van Dijk CN. Appearance of the weight-bearing lateral radiograph in retrocalcaneal bursitis. Acta Orthop. 2010;81:387–90. https://doi.org/10.3109/17453674.2010.487245.
73. Wiegerinck JI, Zwiers R, van Sterkenburg MN, Maas MM, van Dijk CN. The appearance of the pre-Achilles fat pad after endoscopic calcaneoplasty. Knee Surg Sport Traumatol Arthrosc. 2015;23:2400–5. https://doi.org/10.1007/s00167-014-2908-6.
74. de Prado M, Golanó P, Ripoll PL. Minimally invasive foot surgery: surgical techniques, indications, anatomical basis. Barcelona: About Your Health; 2009.
75. Pavlov H, Heneghan MA, Hersh A, Goldman AB, Vigorita V. The Haglund syndrome: initial and differential diagnosis. Radiology. 1982;144:83–8. https://doi.org/10.1148/radiology.144.1.7089270.
76. Khodaee M. Common superficial bursitis. Am Fam Physician. 2017;95:224–31.
77. Caldwell J-ME, Vosseller JT. Maximizing return to sports after Achilles tendon rupture in athletes. Foot Ankle Clin. 2019;24:439–45. https://doi.org/10.1016/j.fcl.2019.04.003.
78. Batista JP, Del Vecchio JJ, Van Dijk N, Pereira H. Endoscopic FHL transfer to augment Achilles disorders. J ISAKOS. 2020;5:109–14. https://doi.org/10.1136/jisakos-2019-000395.
79. Abat F, Alfredson H, Cucchiarini 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;4:18. https://doi.org/10.1186/s40634-017-0092-6.
80. Abat F, Alfredson H, Cucchiarini M, Madry H, Marmotti A, Mouton C, et al. Current trends in tendinopathy: consensus of the ESSKA basic science committee. Part II: Treatment options. J Exp Orthop. 2018;5:38. https://doi.org/10.1186/s40634-018-0145-5.

16 Cycling (BMX, Mountain, Road, Track)

George A. Komnos and Jacques Menetrey

16.1 Sport Characteristics

Cycling is one of the most popular physical activities worldwide and can be either used for transport or performed as a leisure activity or sport. Cycling involves several different types. The most popular types include road cycling, track cycling, mountain biking (MTB), and BMX racing. All these four types are part of the Olympic Program. Road cycling (Fig. 16.1) is usually done in pavement roads, while track cycling in outdoor or indoor velodromes, MTB in off-road terrains and BMX in off-road tracks. Competitions include road races and time trials in road cycling, sprint, endurance, or combination in track cycling, cross-country, or downhill in MTB and sprint races on specially built off-road racetracks in BMX. With the continuous increase in cycling's popularity, cycling-related injuries are becoming more and more common.

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16.2 Epidemiology

Participation in sports activities offers many health benefits, but also involves the risk of injury. Epidemiology of bicycle-related injuries is not well documented yet. The main reason is the lack of registries to record these injuries. Most available data are from hospitals and emergency departments.

Between 2011 and 2014, 5.6 million sports injuries affected children and young adults annually [1]. In the USA, during 2010–2016, the estimated annual average of visits to the emergency department (ED) for sports injuries by patients aged 5–24 years was 2.7 million [2]. Among them, pedal cycling (9.9%) was ranked as the third cause for admission in ED, after football and basketball. Furthermore, cycling has been revealed to be the most common sport-related activity associated with injuries in children aged 5–14 years in the USA at the end of the last century [3]. Mountain biking is among the sports leading to high overall injury rates in Olympic sports [4].

Sports-related concussion accounts for 1.3–9.1% of all injuries reported during cycling events [5, 6]. Rivara et al. [7] evaluated the prevalence of head injury among 3854 injured cyclists in Seattle in 2015. They found that 35% were diagnosed with facial injuries, 22.3% with scalp, skull, forehead, or mild brain injuries, and 6% with more severe brain injuries. Moreover,

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Fig. 16.1 Cyclists during a road cycling competition

according to the National Electronic Injury Surveillance System All Injury Program database from 2001 to 2012, a sport-related traumatic brain injury from cycling was the most common cause in females and the second most common cause in males presenting to an emergency department [8].

Injuries can be classified as traumatic or overuse according to their mechanism. Overuse injuries affect mainly professional or high-level athletes because of high-intense practice. Injury rates vary by sex and age. In the first well-documented study about the incidence of injuries among professional cyclists, Barrios et al. [9] found that the commonest lesions were due to overuse mechanisms (62%) instead of trauma. Patellar and Achilles tendinopathies, and anterior knee pain were the predominant causes of pain according to their study. In another study, Clarsen et al. [10] evaluated overuse lesions in 109 professional cyclists and reported lower back pain (58%) and anterior knee pain (36%) as the most prevalent problems. De Bernardo et al. [11] revealed the iliotibial band friction syndrome as the commonest overuse injury, with the majority of overuse injuries to affect the lower limb and especially the knee. Moreover, most overuse injuries were contractures and chronic muscle shortening. Regarding traumatic injury, De Bernardo et al. [11] reported that 56% of the cases corresponded to fractures, with most of them located at the clavicle (22%), and the rest mainly at the upper limb and ribs.

In an epidemiologic study about injuries during one of the biggest and most famous cycling race, the Tour de France, showed that 49% of the withdrawals were due to fractures, with almost half of them (43%) eventually requiring a surgical treatment for the sustained fracture [12]. Clavicle was also in this study the most frequently fractured bone.

One recent study in Norway with 300 patients treated after cycling injuries [13], reported that most of the injuries were light or moderate. Nevertheless, fractures and minor head injuries dominated, and 45% of patients needed surgery.

In terms of mortality-related cycling, bicycle accidents requiring hospitalization have a high mortality rate, up to 5.7% according to Dutch epidemiological data [14]. 83.3% of the accidents occurred in regular cyclists, 9.8% in race riders, 3.9% in off-road bikers and 2.9% in e-bikes, with the majority of injured patients (92.5%) reported not to wear a helmet. Older patients, multi-trauma and cerebral hemorrhages were identified as risk factors for mortality.

16.3 Causes of Injuries

The main causes of injury during cycling can be summarized under these:

1. Speed: During the races, the athletes develop usually high speed, which can lead to severe injuries if an accident, occurs.
2. Road conditions: Sharp twists or poor road surfaces can lead to accidents.
3. Exhaustion: After intense and/or long races the athletes develop fatigue that can result in exhaustion and reduction of reflexes.

16.4 Types of Injuries

Cycling injuries can practically include all known orthopedic lesions, and a wide variety of injuries, from insignificant ones, till fatal. Some common types of injuries include skin lacerations, injury to bony prominences, friction burns, saddle sores, knee pain, bone and even skull fractures. Additionally, other conditions that can occur are sprains and foreign bodies in the eyes and sometimes exhaustion and collapse of the athletes.

As aforementioned, injuries can be divided into two categories: Traumatic injuries and overuse injuries. The first ones occur most times as a result of a fall, crash, or unexpected motion. On the other hand, overuse injury is mainly caused by repetitive micro-trauma or loading of bone, joint, and soft tissue with inadequate recovery time.

Musculoskeletal injuries include strains, fractures, and dislocations and occur at the upper and lower extremities. Clavicle, wrist, and elbow are mainly affected in upper extremity, pelvis, hip, and femur in lower extremities and ribs in the trunk [15, 16]. Head, neck, and pelvis trauma are less common but can be fatal. It has been reported that 50–73% of traumatic cycling injuries ultimately result in at least one fracture [11, 14]. Superficial soft tissue injuries, such as skin abrasion/laceration are also very frequent, even though they are usually underestimated because they don't always require medical evaluation or care. Skin lesions can be characterized as contusions, lacerations, or abrasions ("road rash") (Fig. 16.2). Even when it seems that the injury involves only the superficial soft tissue, the wound can be deep. One serious, and very difficult to manage lesion, is the Morel-Lavallée lesion, which develops in the thigh, due to shearing of superficial subcutaneous tissues away from underlying fascial layers.

Several traumatic conditions can arise in the lower limb in particular. Knee joint is the most common site of overuse injury in the cyclist. It is reported that 40–60% of riders experience knee pain [17, 18]. The patellofemoral compartment is mainly affected, with persistent anterior knee pain (cyclist's knee). The most important causes are excessive pressure to the patellofemoral joint due to hill climbing or slow pace resulting in cartilage compression and damage, and poor tracking of the patella with malalignment [19, 20]. Other not rare conditions that can appear with pain around the knee are quadriceps tendinitis, and pes anserine bursitis. Quadriceps tendinitis is

Fig. 16.2 Cyclist with skin lacerations all over his trunk and limbs after a race

diagnosed as lateral or medial knee pain, and tenderness over the quadriceps tendon. Pes anserine bursitis results from repeated friction and inflammation of the hamstring insertion in the tibia, with tenderness to palpation over the pes anserine bursa, 2–3 cm below the joint line. Iliotibial band (ITB) syndrome can also cause pain at the lateral side of the knee, usually over the femoral condyle. Hip pain in the cyclist is not as common as knee pain but can exist. Typically, this is due to trochanteric bursitis or iliopsoas tendinitis. Less common injuries of the lower leg include exertional compartment syndrome, medial tibial stress syndrome, and acute bone fractures (Fig. 16.3) or stress fractures [17]. Furthermore, Achilles tendinitis can appear, especially when an improperly fitted bicycle is used.

Regarding upper extremity, the most common injury is clavicle fracture. In general, clavicle fractures are caused mostly by sports activities (45.3%) and most commonly result from bicycling (16%) [21, 22]. These fractures are managed operatively in most cases in athletes, in an attempt for a quicker recovery and earlier return to sports [23, 24] (Fig. 16.4).

Spinal injuries are most common in mountain biking. Dodwell et al. reported that mountain biking-related spine injuries consisted of almost

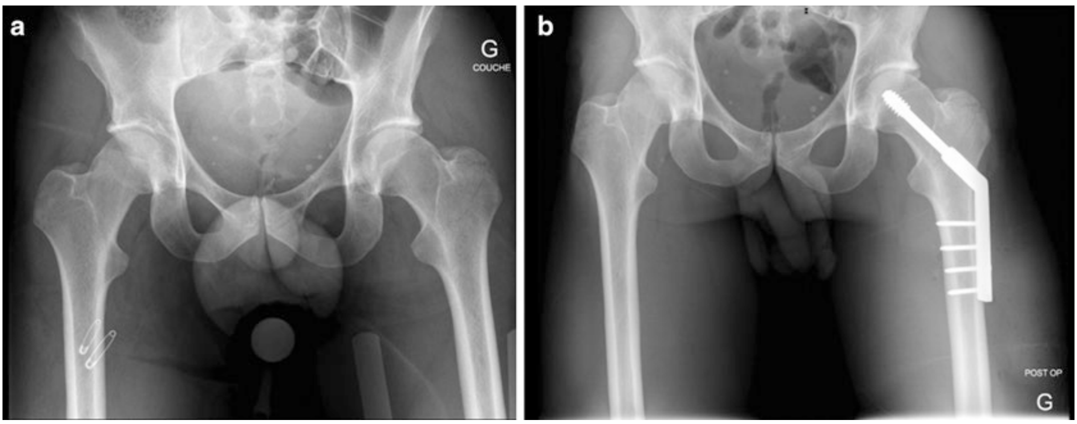

Fig. 16.3 Hip intertrochanteric fracture sustained by a cyclist during the race (**a**). Treated with DHS fixation (**b**) and returned to high-level cycling 3 months later

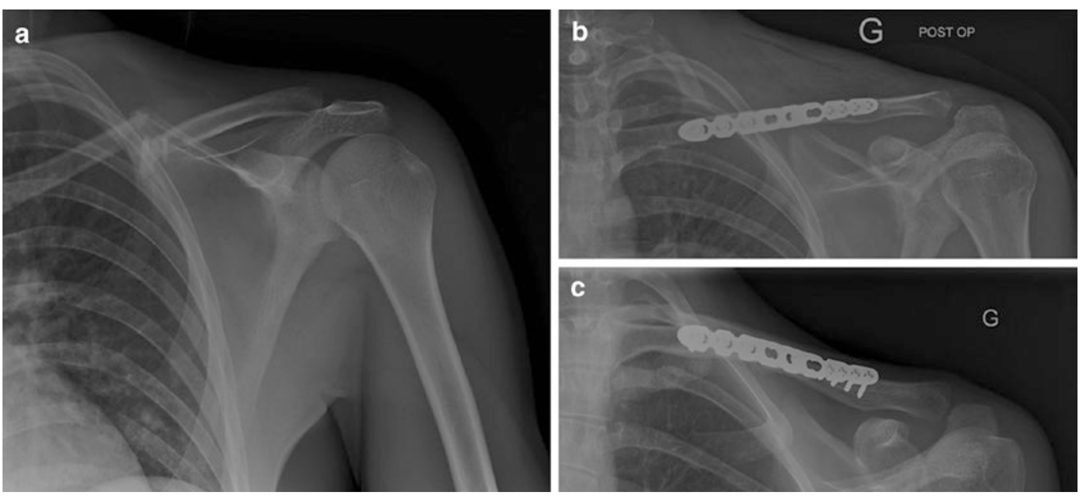

Fig. 16.4 Clavicle fracture sustained during a cycle race (**a**), treated with a plate fixation (**b**, **c**)

4% of all spinal injuries [25], with cervical spine injuries accounting for 74% of them.

Concussions are of huge importance during cycling races. However, not scarce, concussions are underestimated. Cyclists disregard head injuries and prefer to return on their bike feeling willing and able to ride. Nevertheless, they can suffer from impaired performance and reaction time, placing them at risk for further, severe injuries [26]. In professional cycling, helmets are mandatory since 2003, following the death by brain injury of a professional road cyclist in the Paris-Nice race. Their main characteristic is to prevent skull fractures and intracranial hemorrhage (Fig. 16.5).

Heat-related injuries, such as cramping, dehydration, and heat exhaustion can occur during races in extremely hot environmental conditions. According to a survey among elite cyclists who competed in the heat at the 2016 UCI Road World Cycling Championships in Qatar, 22% of respondents reported illness symptoms in the 10 days preceding the Championships, 57% of respondents had previously experienced heat-related symptoms while 17% had previously been diagnosed with exertional heat illness [27].

In terms of mountain biking, the patterns of injuries are similar. However, mountain biking appears to be a high-risk sport for severe spine injuries [16]. The most common mechanism of acute severe injury is by falling forward, mainly during downhill riding [28]. Besides spinal injuries, blunt abdominal and chest trauma are also more common among bikers of this style, caused mostly by bicycle handlebars and bar ends [29, 30].

Same injuries pattern applies to BMX riders, with abrasions and contusions to be the most predominant causes of injury [31]. However, as BMX bicycles are quite popular among young athletes and children, spinal injuries can be devastating and even life threatening in this population, especially after being involved in road traffic accidents [32].

Fig. 16.5 Helmets are the most important equipment for prevention of head injuries

16.5 Rehabilitation and Return to Sport

The protocol of rehabilitation and return to sport for traumatic or overuse injuries depends upon the nature of the lesion (Table 16.1). Time to return to competition varied widely, 7–316 days, among cyclists who suffered an injury during

Table 16.1 Return to sport correlated to the sustained injury

Lesion	Return to sport
Clavicle fractures	Stationary cycles within 1 week, outdoor cycles after 2–3 weeks, and race from 4 to 6 weeks after the trauma. In case of operative treatment, return to training and competition may be sooner
Acromioclavicular dislocations	Stationary workout after 1–2 weeks
Radial head fractures	Return to riding after 1–2 weeks (if non-displaced)
Pneumothorax and ribs fractures	Return to racing may take 4–6 weeks
Patellar tendinopathy	Home exercises for at least 12 weeks before starting to ride, or sooner if other medical therapies are also applied
Overuse injuries	Return to racing depends on the symptoms after and the specific treatments according to the symptoms
Concussion	Physical and cognitive rest until acute symptoms resolve and frequent assessments with a stepwise return to play, initially with stationary bike. Clinical evaluation is essential for a safe return to cycling

Tour de France [12]. Athletes who withdrew from the race because of an injury returned to competition significantly later (52 days on average) in comparison to those who withdrew for non-traumatic reasons. Overall, athletes who had to undergo operative treatment of their injury experienced a longer time to return to competition (77 days) compared to those treated non-operatively (44 days).

As aforementioned, clavicle fractures are of the most frequent and significant injuries that can occur in cyclists. Among cyclists from Tour de France who sustained such a fracture, the time to return to competition managed operatively, was significantly shorter than those whose the same injury was treated non-operatively [12]. Moreover, in another study it is demonstrated that plate fixation accompanied by an early post-operative rehabilitation protocol gives professional cyclists the chance to resume athletic activity 2 weeks postoperatively and return to competition 3 weeks later [21].

Concussion can be a life-threatening trauma. Subsequently, the proper evaluation and decision to return to sport after sustaining a concussion are of huge importance. Evaluation of neurological status must always be performed. Cyclists who have a history of prior concussion are at an increased risk and should undergo cognitive and motor control assessment prior to any race.

One of the most challenging situations is the assessment of the cyclist after an injury during the race. It is not a rare phenomenon that the athlete wants to resume his activity and therefore has a tendency to underestimate any early symptoms. In these cases, on-field SCAT-5 assessment and Maddock's questions can be used. However, Maddock's questions have been validated in a team sport setting, such as football, ice-hockey, and rugby and therefore cannot thoroughly apply to cycling. A recent systematic review [33] suggested the following questions as road cycling specific Maddock's questions:

1. What is the name of this race?
2. How many kilometers are there still to go in today's stage?
3. Who is the road captain today for the race?
4. What was your last race?
5. What is your coach's name?

Table 16.2 RIDE Protocol for head injury (concussion) in cyclists [6]

RIDE 1	Road-side assessment	Symptoms checklist Medical evaluation Cognitive tests
RIDE 2	Immediately after race	SCAT 5 tool Digit Symbol Substitution Testing Full neurological examination
RIDE 3	After one night rest	SCAT 5 tool Digit Symbol Substitution Testing Full neurological examination

In a very recent study, a cycling-specific RoadsIde heaD injury assEssment (RIDE) protocol has been proposed [6]. According to that, there is a three-stage diagnostic process, which consists of three phases (Table 16.2):

1. Initial road-side assessment immediately after the head impact event (RIDE 1).
2. Reassessment immediately following completion of the stage on the same day of the injury (RIDE 2).
3. Reassessment the day following the initial injury (RIDE 3).

However, in the setting of more severe situations, evaluation can be made more than three times as described above.

16.6 Prevention Strategies

In general, self-taken measures include the use of protective gear, especially helmet, the placement of correct position on the bike and adjustment of it to individual morphology. Mountain riders should not overestimate their ability, use well-fitted bikes, avoiding handlebars ends and wear the appropriate equipment, including helmets with facial protection, padded gloves and shorts, and shin pads [34]. Another essential issue is that athletes should also try to maintain a high level of fitness, which makes them less vulnerable to the aforementioned injuries.

Extremely important are the educational programs that teach youth population to be safe drivers and cyclers, especially encouraging them to wear helmets. Refusal to wear a bicycle helmet has been proven to be the biggest predisposing factor for bicycle head injury (Fig. 16.5) [33, 34]. Specifically built bicycle roads and bicycle-friendly riding environments could lower the risk of traumatic injuries by separating bicycles from motor vehicles.

In professional racing, basic rules of prevention consist of a precise preparation of the race route and a highly experienced security team. Regarding compression and overuse injuries, bike adjustments are the most essential measure. Adjustment of the saddle (posture height) can address problems related to the lower extremity, adjustment of the handlebars (posture length) can address those related to the upper part of the trunk (extremities, neck and spine) [35].

To prevent adverse events resulting from weather conditions, the Union Cycliste Internationale (UCI), cycling's governing body, has established an *Extreme Weather Protocol* that incorporates a discussion among all members involved in a cycling race to discuss and propose modifications to the race route, depending on extreme weather conditions, such as extremely hot conditions [27]. In races that take place under high temperatures, cyclists should use a pre-cooling strategy before competing in the heat. In addition, team doctors should be aware of the four "golden rules" of heat stroke management, which consist of early recognition, early diagnosis, rapid cooling, and on-site cooling [36].

16.7 Equipment and Protection Considerations

In terms of protective equipment, the most efficient one for cyclists is the helmet. Among others, protective equipment can include:

- Eye protection against weather, foreign bodies, and ultraviolet lights.
- Gloves.
- Bicycle shorts with padding integrated.
- Cycling shoes with toe clips or sole cleats that attach the foot to the pedals.
- Bright color clothes and reflective vests to improve cyclists' visibility on the road, in particular when poor weather or in the evenings.

Regarding pedals, the ideal position of the foot on the pedal is the anterior foot position. It decreases the stress across the knee ligaments and gives a mechanical advantage to gastrocnemius and soleus muscles to turn the crank.

Besides the personal prevention equipment, medical cover during races is of high importance. Well-trained and equipped medical personnel should be present during cycling events, with the accompaniment of at least one ambulance.

References

1. Sheu Y, Chen LH, Hedegaard H. Sports- and recreation-related injury episodes in the United States, 2011–2014. Natl Health Stat Report. 2016;(99):1–12.
2. Rui P, Ashman JJ, Akinseye A. Emergency department visits for injuries sustained during sports and recreational activities by patients aged 5–24 years, 2010–2016. Natl Health Stat Report. 2019;(133):1–15.
3. Conn JM, Annest JL, Gilchrist J. Sports and recreation related injury episodes in the US population, 1997–99. Inj Prev. 2003;9:117.
4. Engebretsen L, Soligard T, Steffen K, Alonso JM, Aubry M, Budgett R, et al. Sports injuries and illnesses during the London Summer Olympic Games 2012. Br J Sports Med. 2013;47:407.
5. Roi GS, Tinti R. Requests for medical assistance during an amateur road cycling race. Accid Anal Prev. 2014;73:170.
6. Heron N, Elliott J, Jones N, Loosemore M, Kemp S. Sports-related concussion (SRC) in road cycling: the RoadsIde heaD Injury assEssment (RIDE) for elite road cycling. Br J Sports Med. 2020;54:127.
7. Rivara FP, Thompson DC, Thompson RS. Epidemiology of bicycle injuries and risk factors for serious injury. Inj Prev. 2015;21:47.
8. Coronado VG, Haileyesus T, Cheng TA, Bell JM, Haarbauer-Krupa J, Lionbarger MR, et al. Trends in sports- and recreation-related traumatic brain injuries treated in US emergency departments: the National Electronic Injury Surveillance System-All Injury Program (NEISS-AIP) 2001–2012. J Head Trauma Rehabil. 2015;30:185.

9. Barrios C, Sala D, Terrados N, Valenti JR. Traumatic and overuse injuries in elite professional cyclists. Sport Exerc Inj. 1997;3(4):176–9.
10. Clarsen B, Krosshaug T, Bahr R. Overuse injuries in professional road cyclists. Am J Sports Med. 2010;38:2494.
11. de Bernardo N, Barrios C, Vera P, Laíz C, Hadala M. Incidence and risk for traumatic and overuse injuries in top-level road cyclists. J Sports Sci. 2012;30(10):1047–53.
12. Haeberle HS, Navarro SM, Power EJ, Schickendantz MS, Farrow LD, Ramkumar PN. Prevalence and epidemiology of injuries among elite cyclists in the Tour de France. Orthop J Sport Med. 2018;6(9):2325967118793392.
13. Mjåland O, Nygaard A, Storm-Larsen C, Brommeland T. Sykkelrelaterte skader på Sørlandet sykehus Kristiansand. Tidsskr den Nor Laegeforening. 2019.
14. de Guerre LEVM, Sadiqi S, Leenen LPH, Oner CF, van Gaalen SM. Injuries related to bicycle accidents: an epidemiological study in The Netherlands. Eur J Trauma Emerg Surg. 2020;46(2):413–8.
15. Sillén K, Wallenius V. Rates and types of injuries during the three consecutive years 2016 to 2018 of the Vätternrundan—one of the world's largest and longest bicycle races. Traffic Inj Prev. 2019;20:749.
16. Ansari M, Nourian R, Khodaee M. Mountain biking injuries. Curr Sports Med Rep. 2017;16:404.
17. Wanich T, Hodgkins C, Columbier JA, Muraski E, Kennedy JG. Cycling injuries of the lower extremity. J Am Acad Orthop Surg. 2007;15:748.
18. Wilber CA, Holland GJ, Madison RE, Loy SF. An epidemiological analysis of overuse injuries among recreational cyclists. Int J Sports Med. 1995;16(3):201–6.
19. Dettori NJ, Norvell DC. Non-traumatic bicycle injuries: a review of the literature. Sports Med. 2006;36:7.
20. Ruby P, Hull ML, Kirby KA, Jenkins DW. The effect of lower-limb anatomy on knee loads during seated cycling. J Biomech. 1992;25(10):1195–207.
21. van der Ven DJC, Timmers TK, Broeders IAMJ, van Olden GDJ. Displaced clavicle fractures in cyclists. Clin J Sport Med. 2017;29(6):465–9.
22. Postacchini F, Gumina S, De Santis P, Albo F. Epidemiology of clavicle fractures. J Shoulder Elb Surg. 2002;11:452.
23. Ranalletta M, Rossi LA, Piuzzi NS, Bertona A, Bongiovanni SL, Maignon G. Return to sports after plate fixation of displaced midshaft clavicular fractures in athletes. Am J Sports Med. 2015;43:565.
24. Meisterling SW, Cain EL, Fleisig GS, Hartzell JL, Dugas JR. Return to athletic activity after plate fixation of displaced midshaft clavicle fractures. Am J Sports Med. 2013;41:2632.
25. Dodwell ER, Kwon BK, Hughes B, Koo D, Townson A, Aludino A, et al. Spinal column and spinal cord injuries in mountain bikers: a 13-year review. Am J Sports Med. 2010;38:1647.
26. Silberman MR. Bicycling injuries. Curr Sports Med Rep. 2013;12:337.
27. Racinais S, Nichols D, Travers G, Moussay S, Belfekih T, Farooq A, et al. Health status, heat preparation strategies and medical events among elite cyclists who competed in the heat at the 2016 UCI Road World Cycling Championships in Qatar. Br J Sports Med. 2020;54:1003.
28. Chow TK, Kronisch RL. Mechanisms of injury in competitive off-road bicycling. Wilderness Environ Med. 2002;13:27.
29. Kim PTW, Jangra D, Ritchie AH, Lower ME, Kasic S, Brown DR, et al. Mountain billing injuries requiring trauma center admission: a 10-year regional trauma system experience. J Trauma. 2006;60(2):312–8.
30. Kotlyar S. Cycling injuries in Southwest Colorado: a comparison of road vs trail riding injury patterns. Wilderness Environ Med. 2016;27:316.
31. Brøgger-Jensen T, Hvass I, Bugge S. Injuries at the BMX cycling European Championship, 1989. Br J Sports Med. 1990;24:269.
32. Piggot J, Graham HK, Henderson SA. Death and serious injury in child motorcyclists. Br Med J. 1987;294(6582):1259.
33. Elliott J, Anderson R, Collins S, Heron N. Sports-related concussion (SRC) assessment in road cycling: a systematic review and call to action. BMJ Open Sport Exerc Med. 2019;5:e000525.
34. Carmont MR. Mountain biking injuries: a review. Br Med Bull. 2008;85:101.
35. Nehoda H, Hochleitner BW, Hourmont K, Weiß H, Lanthaler M, Tschmelitsch J. Central liver hematomas caused by mountain-bike crashes. Injury. 2001;32(4):285–7.
36. Racinais S, Casa D, Brocherie F, Ihsan M. Translating science into practice: the perspective of the Doha 2019 IAAF world championships in the heat. Front Sport Act Living. 2019; https://doi.org/10.3389/fspor.2019.00039.

17 Dancing

Victoria B. Duthon and George A. Komnos

17.1 Dance Modality

Dance is a mix of art and sport, and dancers can be considered both artists and athletes. Their sports performance has to look attractive and at the same time, easy to the audience. The dance movements place specific demands on the body in terms of endurance and aerobic capacity, muscle strength, overall flexibility, joint stability, somatosensory integration, and neuromuscular coordination [1]. Dancers possess unique physical and anatomical qualities because they have to be strong, powerful, and flexible at the same time. And interestingly, this applies to every part of their body, from head to toes. Professional dancers usually begin dancing during childhood, and the intensive training and stretching from the youngest age leads to a flexible spine, both in extension and in flexion, but also to flexible muscle units, such as hamstrings. Combining these criteria is unique to dance although artistic gymnastics has similarities. Dance requires great flexibility of all joints, and it is important to perform the classical ballet technique. Nevertheless, dancers are not necessarily hypermobile [2]. Briggs et al. [3] reported the prevalence of joint hypermobility as assessed by skin hyperextensibility and joint dislocation as 33% of females and 32% in males. Those who are hypermobile, are at a higher risk of injury, because the extreme dance movements combined with ligamentous laxity place increased demand on the stabilizing and supporting structures of the joints [4].

Knowledge of the basic dance positions and techniques from the traditional ballet repertoire contributes to understanding dance injury mechanisms. Figure 17.1 shows the basic positions of ballet dancing.

Ballet dance technique is characterized by the use of extreme positions, and the permanent "turnout" position (lower limb external rotation from hips to ankles) that is maintained in every position and dance movement. Another ballet specificity is the "pointe work" (maximal ankle and foot plantarflexion). These positions, described below, can potentially place undue stress on muscles, joints, and tendons. Ballet dancers who cannot reach these specific esthetic standards may fail to execute the proper technique with an increased potential for injury [1].

- *Dancing on pointe* (on the toes) is a technique that separates classical ballet from all other dance forms, both technically and medically. Female dancers require 90° of ankle plantarflexion to do pointe work: full equinus of the foot and full ankle plantarflexion attained

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Fig. 17.1 Basic ballet positions: first position, second position, third position, fourth position, fifth position

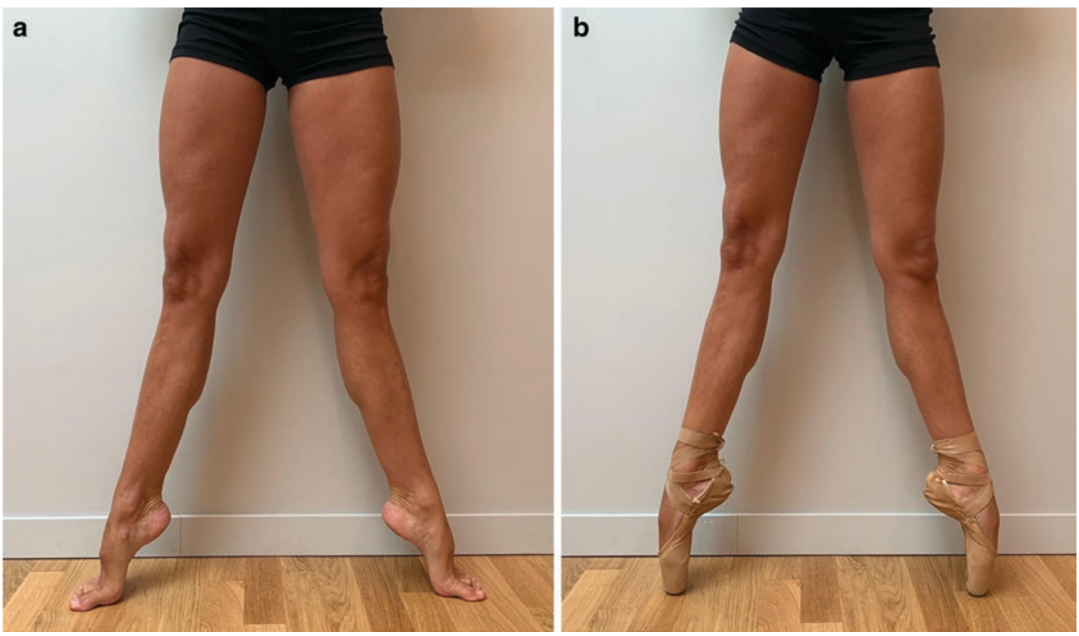

Fig. 17.2 (**a**) Demi-pointe, (**b**) pointe

from the combined motion of the tibiotalar, subtalar, and midtarsal joints [5] (Fig. 17.2b). Pointe work is performed while wearing pointe shoes, which employ structural reinforcing to distribute the dancer's weight load throughout the foot. It reduces the load on the toes enough to enable the dancer to support all body weight on fully vertical feet. Extensive training and practice are required to develop the strength and technique needed for pointe work. Dance teachers take into consideration factors, such as age, experience, strength, and alignment before deciding whether to allow a dancer to begin pointe work or not. The demi-pointe position (Fig. 17.2a) is similar to pointe, except for 90° hyperextension of the metatarsophalangeal (MTP) joints transferring weight-bearing to the metatarsal heads. When dancers lack sufficient ankle and/or foot plantarflexion for pointe or demi-pointe positions, they may attempt to force plantarflexion, placing greater stress on the posterior ankle structures. Forcing plantarflexion has also been associated with "sickling," a malalignment at the foot-ankle complex in which the dancer fails to balance correctly on pointe or demi-pointe positions. "Sickling in" refers to varus alignment of the foot and increases stress of lateral structures of the ankle [6].

- *Turnout* is a term that refers to the maximal external rotation achieved at the lower extremity, which is of major significance for ballet esthetics, and dancers have to maintain maximal turnout in all ballet positions (Fig. 17.1). Turnout is the combination of external rotation from all lower limb joints, with the hip providing the greatest contribution (60%) and the rest obtained through the knee, ankle, and foot [7].

  Hip turnout provides a functional advantage for a dance performance; greater external rotation at the hip correlates with greater abduction of the leg because the greater trochanter is cleared away from a position of impingement [8]. The anatomic basis for dancers' ample turnout has been attributed to various factors, including soft tissues as well as osseous architecture. Soft tissue adaptations (ligamentous and hip capsule stretching) may be gained through dance training. Muscle strength has a critical role in a dancer's ability for hip turnout. Specificity of muscle training is suggested by the preferential strengthening of hip external rotators, abductors, and adductors as recorded in young (8–11-year old) female dancers [1]. Dancers who do not have an ideal turnout of the lower extremity cheat by using compensatory strategies along the kinematic chain. In the weight-bearing position, dancers may compensate by augmenting the anterior pelvic tilt or lumbar lordosis, the external rotation of the knee ("screwing the knee"), or the valgus heel with forefoot pronation ("rolling in") [9]. As for the other mentioned strategies, the augmented torsional forces on these lower extremity joints increase the risk of an overuse injury, particularly at the medial aspect of the knee, ankle, and foot [1]. Carter et al. [10] used 3D kinematic analyses to investigate the lower leg and foot compensations to turnout and found that dancers were more likely to pronate about the foot/ankle complex than externally rotate the knee to achieve greater turnout. However, dancers with limited capacity to pronate in turnout may force additional rotation via the knee.

Knowledge about the technical requisites of dance is an important consideration in the care of the dancer. Understanding the biomechanics of dance is essential for dance medicine practitioners to identify the specific anatomic demands placed on body structures and to uncover pathomechanics leading to injury [1].

Fact Box

Ballet dancers are highly trained athletes and artists who are at significant risk of musculoskeletal injury because of extreme positions, always maintaining the "turnout." The "pointe" work is characteristic of ballet and requires 90° of ankle plantarflexion with full equinus of the foot.

17.2 Top Five Dance-Related Injuries

Professional ballet dance is a highly demanding performance art. Given the high physical and psychological stress, injuries are common in ballet. Ballet dancers exploit their locomotor system, which leads to breaking the limits of the adaptive mechanisms and results in motor system dysfunctions and injuries [11]. Most musculoskeletal injuries are soft tissue injuries, such as sprains, strains, and tendinopathies although stress fractures have also been reported in the literature. More common injuries occur as a result of systematic overload (i.e., overuse injuries) and involve injuries of the lower extremities and lower back. Overuse and chronic injuries are commonly reported because professional ballet dancers train up to 40 h per week, in addition to performances. The dancer's body may be exposed to a highly physical demanding activity for greater than 6 h per day. Many dancers may also have only 1 day of rest per week. Most injuries affect the lower extremities and back. Traumatic injuries (e.g., injuries that occur as a result of acute stress) are less common and are mostly associated with loss of balance during the practice or performance (e.g., ankle strain, hamstring strain, patella dislocation) [12]. Most dancers' injuries are mild or minor and require minimal time off.

Here are the top five dance-related injuries that dance medical team should be aware of:

(a) FOOT AND ANKLE:

Ankle sprain: Ankle sprain is the most common traumatic injury in dance, including classical ballet and theatrical dance [13]. The mechanism for inversion ankle sprains involves foot plantarflexion and inversion, such as when the dancer performs in demi-pointe or while landing from a jump. It is the same mechanism that leads to the "dancer's fracture" which is an acute spiral fracture on the fifth metatarsal neck that is associated with twisting and inversion of the foot on demi-pointe. O'Malley and colleagues [14] reviewed the outcomes of this fracture in dancers and described that this fracture has a high rate of union and can be treated conservatively with positive outcomes. The dancer is less susceptible to inversion ankle sprains in the full plantarflexed position (i.e., full pointe in ballet), however, because the ankle is stabilized by the posterior lip of the tibia resting over the talus and the subtalar joint is locked [15]. From this position, the dancer is more likely to sustain a midfoot sprain, especially at the capsules of the base of the fourth and fifth metatarsals. Ankle sprains can be classified into three grades. Grade I sprain involves microscopic partial tearing of the anterior-talo-fibular ligament (ATFL) fibers. Grade II sprain includes a positive anterior drawer sign corresponding to tearing of the ATFL. Grade III sprains involve a complete tear of the lateral ligament complex, such that the anterior drawer sign and the result of the talar tilt test are positive. X-rays are mandatory to rule out a fracture or a widening of the mortise which would traduce a syndesmosis sprain; MRI may be useful to confirm the exact lesions and their severity and to plan more precisely the time off and the delay until the return to dance. Grade I and II sprains can be managed with early mobilization. Nevertheless, external support measures can be helpful in the acute phase and may include taping, elastic wrapping, and orthoses. Grade I and II sprains generally have a favorable prognosis after conservative management that includes rehabilitation. Grade III sprains may require immobilization of the leg, with a short leg cast or an air boot for a short period until early mobilization and return to function. The rehabilitation phase of grade I through III sprains may include therapeutic exercises and dancing activities within a pool, which allow the dancer to maintain a conditioning program with the advantage of reduced weight-bearing conditions. Exercises in plantarflexion position help to progress strengthening rapidly, with the emphasis being on improving gastrocnemius, soleus and peroneal muscles strength. A functional goal of rehabilitation for the dancer may include the ability to maintain a stable pointe position against varus and/or valgus testing forces. Continuing in a program for proprioception and coordination exercises may be beneficial. Hamilton [15] described that other complications after ankle sprains possibly associated with chronicity of the condition, include (1) varus instability attributable to combined ligamentous laxity and peroneal weakness and (2) rotatory instability with subluxation of the anterior talar dome when the dancer turns on the affected foot in demi-pointe position. According to his study, peroneal strengthening to stabilize the ankle in plantarflexion positions is recommended [15].

Os trigonum impingement syndrome refers to the impingement of the accessory bone called the os trigonum, which can become entrapped between the tibia and calcaneus during plantarflexion. This accessory bone can be found in 3–13% of the general population, and unilateral incidence is more common than bilateral incidence [16]. In an MRI series review, up to 30% of ballet dancers had an os trigonum [17]. When symptomatic, the clinical presentation consists of posterior ankle pain exacerbated by plantarflexion (may restrict performing on pointe) and a positive plantarflexion test. Lateral plain radiographs demonstrate the presence of the os trigonum (Fig. 17.3) and in plantarflexion (e.g., standing in demi-pointe) may demonstrate impingement of the os trigonum. Initial treatment is conservative, as previously described for posterior ankle impingement syndrome. Surgical excision of the os trigonum has been successful for treating dancers [18]. Nowadays, it can be removed by both arthroscopic and posterior endoscopic excisions (Fig. 17.3): both techniques are safe and effective in treating. The arthroscopic procedure is more demanding, especially in cases of a large os trigonum. The posterior endoscopic approach has the advantage of addressing problems in the posterior ankle joint and allows a more extensive release of the flexor hallucis longus [19].

FHL tendinitis, also known as "dancer's tendinitis," may occur by itself or secondary to impingement by an os trigonum. The FHL tendon may become inflamed along its course while traveling medial to the os trigonum, beneath the sustentaculum tali, or as it crosses the sesamoids to the first interphalangeal joint of the big toe. Typically, areas of tendon degeneration occur at avascular zones: where the FHL tendon courses near to bony prominences (behind the talus and at the metatarsal head between the sesamoids). Physical examination may reveal tenderness along the course of the tendon, particularly at the posteromedial ankle (often being misdiagnosed as posterior tibial tendinitis) and passive extension of the great toe may be painful. In the setting of FHL tenosynovitis and formation of nodules, other associated findings may include crepitus, trigger hallux, and functional hallux rigidus (reduced dorsiflexion at first MTP joint when the knee is extended and the ankle is in maximal dorsiflexion) [20]. Treatment is initiated with conservative management, with specific FHL stretching exercises that optimize FHL excursion by simultaneous dorsiflexion of the ankle and first MTP joint: it improves symptoms associated with FHL tendinitis and functional hallux rigidus [20]. The training regimen of dancers should be modified to limit pointe work. Malalignments related to forced turnout and rolling in of the foot should be corrected. For dancers returning to dance activity, proper jump techniques must be emphasized because the FHL is involved in takeoff and landing. If conservative treatment fails, a brief trial with immobilization (3–4 weeks) can be considered. For refractory cases, surgery may be indicated for the release of the FHL tendon sheath and debridement of the tendon. Operative release of the FHL has been effective in treating isolated stenosing tenosynovitis in ballet dancers [21].

(b) *SPINE: Low back pain and low back injury* have been identified as a common and often severe cause of time loss injury in both preprofessional and professional dancers. This problem has been attributed to the unique and highly physical movement demands of dance because positions with hyperlordosis (for example, arabesque, Fig. 17.4) generate undue stress on the posterior elements of the spine. Approximately 73% of dancers will experience at least one episode of LBP each

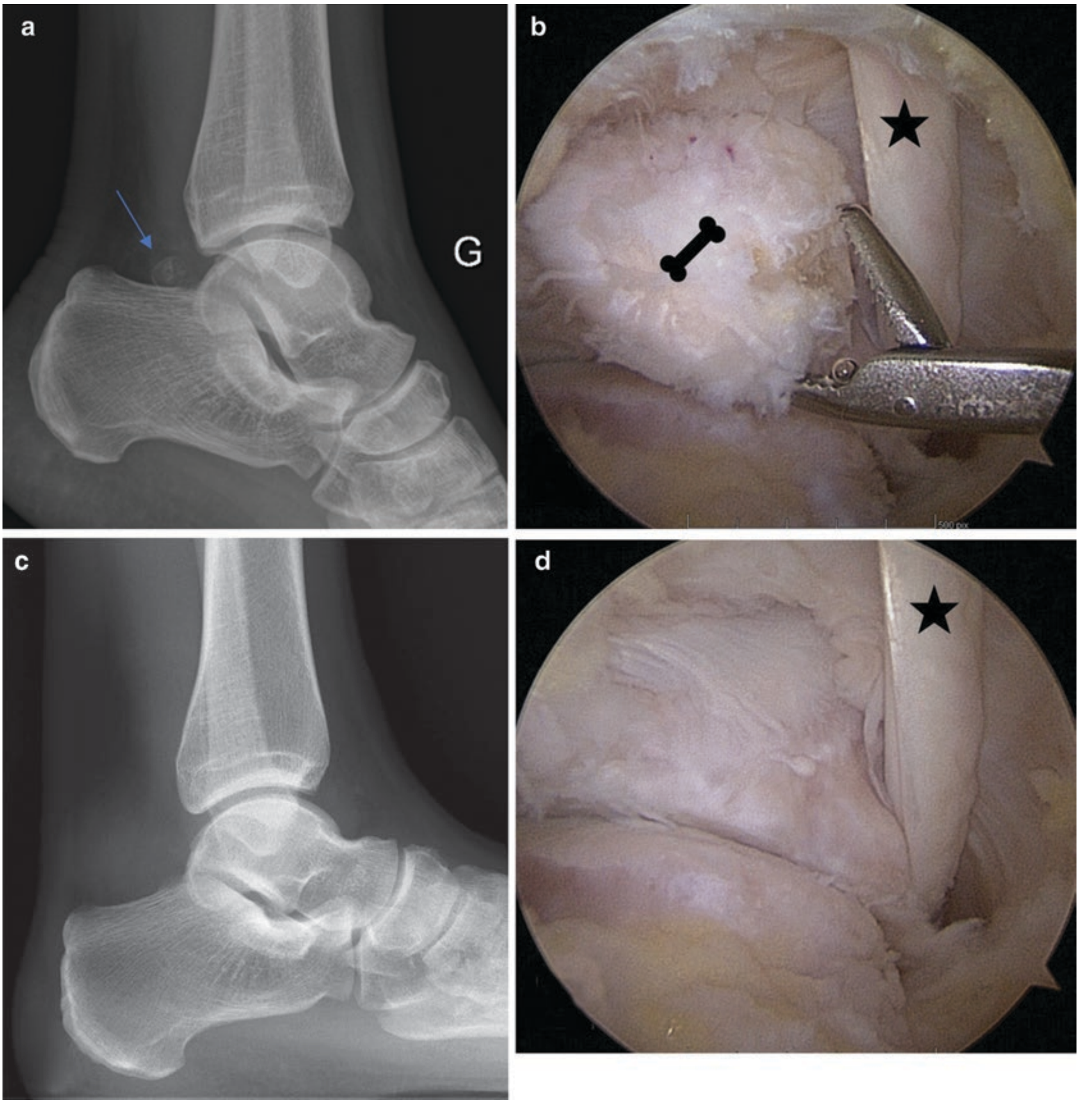

Fig. 17.3 Os trigonum: (**a**) lateral ankle X-ray with os trigonum (blue arrow); (**b**) posterior ankle arthroscopic view of os trigonum (bone) and flexor hallucis longus (star); (**c**) lateral ankle X-ray after os trigonum removal; (**d**): posterior ankle arthroscopic view after os trigonum removal

year; however, the lower back will only be identified as the cause of time loss or medical attention for 11% of cases [22]. Indeed, spinal pathologies such as spondylolysis, a defect caused by alternating full flexion and extension movements, are more common in ballet dancers than the general population. Further, the incidence of spine stress fractures in professional ballet dancers appears to increase with dance hours completed. Male dancers experience a greater percentage of injuries to the low back than female dancers [22]. In pre-professional dancers, adolescent idiopathic scoliosis (AIS) should be examined because several studies report a higher prevalence of adolescent idiopathic scoliosis (AIS) in adult classical ballet dancers who are women than the general population, finding scoliosis in 24–50% of adult participants [23]. Similar findings have been

Fig. 17.4 Arabesque on pointe

found in sports sharing similar characteristics to dance, including rhythmic gymnastics, where the incidence of scoliosis was reported to be ten times that of a nongymnast group. Longworth et al. [24] found 30% of dancers tested positive for scoliosis compared with 3% of nondancers. Odds ratio calculations suggest that dancers were 12.4 times more likely to have scoliosis than nondancers of the same age. There was a higher rate of hypermobility in the dancer group (70%) compared with the nondancers (3%); however, there were no statistically significant correlations between scoliosis and hypermobility, age of menarche, BMI, or hours of dance per week. This study has found a high prevalence of AIS in adolescent ballet dancers similar to the prevalence of scoliosis reported in adult ballet dancers. Another source of pain is the sacroiliac joint which can cause local and referred pain. The sacroiliac joint is intimately related to the biomechanics of the pelvis and lumbar spine, and for dancers who move throughout the ROM of these joints, sacroiliac dysfunction may contribute to pain interfering with dance activity [25]. Dancers may experience pain on jumps and limitation to achieve leg extension (arabesque) on the affected side. Diagnosis is set by performing stress maneuvers, such as the Gillet test and Gaenslen's tests. Treatment with physical therapy should include modalities strengthening weak muscles through pelvic stabilization exercises and mobilization of the joint.

(c) *HIP: The "snapping hip"* refers to a click with a snapping sensation that occurs during movement of the thigh. Lateral (external) snapping on the hip corresponds to motion of the iliotibial band (ITB) over the greater trochanter: it more commonly affects the supporting leg while attempting rotation movements and when landing from jumps, as the hip extends from a flexed position. Medial (internal) snapping that occurs medial or anterior to the hip is caused by the iliopsoas tendon moving across the femoral head or iliopectineal eminence: it affects the gesturing leg (non-weight-bearing limb), causing a painful arc when performing semicircular motion around the torso to bring the hip into extension from a flexed, adducted, and externally rotated position (ronde de jambe) [1]. The snapping sensation is usually audible and palpable and is usually painless, but it can become painful and limit dance activities, if chronic. Treatment is usually conservative with physiotherapy to correct the posture, to strengthen the hip musculature, to stretch the iliopsoas and the iliotibial band; the use of anti-inflammatory drugs, or even crutches to unload the joint are also recommended occasionally. For refractory cases of medial snapping, an MRI may rule out any intra-articular pathology and surgical release or lengthening may be an option. An MRI may show an underlying labral tear which also could cause hip pain and mechanical instability because of a catching and "giving away" sensation at the anterior hip or groin [26]. It was shown using 3D motion capture that some dancers have a *dynamic femoro-acetabural impingement* caused by repetitive extreme movements which can cause femoral head subluxations and femoroacetabular abutments even with normal hip morphologic features (normal α neck angle, acetabular depth, acetabular version, and femoral neck anteversion) which could result in early hip osteoarthritis [27, 28].

(d) *KNEE: Patellofemoral syndrome:* An esthetic feature of classical ballet is knee full extension, or even knee recurvatum. This position may be associated with posterior capsular strain and pain. Grand-plié involves deep flexion of the knees (Fig. 17.5). The required degree of knee flexion increases the patellofemoral joint reaction force, and repetitive movement into these positions may contribute to extensor mechanism overuse syndrome as the patellofemoral syndrome. An increased Q angle is associated with further patellar dysfunction because lateral patellar tracking is accentuated by the turnout technique. Steinberg et al. [29] found that 23.6% of dancers experience patella-femoral syndrome, and that prevalence of the syndrome increase with dancer's age ($p < 0.001$). Dancers with hypo range of motion in hip external rotation, ankle plantarflexion, ankle/foot pointe, hip abduction, hip extension, and limited hamstrings and lumbar spine are significantly less prone to developing patellofemoral syndrome compared to dancers with

Fig. 17.5 (**a**) Demi-plié, (**b**) grand-plié

average range of motion. Unilateral and bilateral patellofemoral syndrome is common among young dancers. Body morphology, reduced ankle proprioception ability, dynamic postural balance asymmetry, and increased hour/day of practice are associated with patellofemoral pain.

(e) *Stress fractures:* Stress fractures are the partial or complete fractures as a result of submaximal loading. This injury is often compared to fatigue fractures that occur in engineering materials, such as bridges and buildings, although some would argue that the mechanisms are different. Normally, submaximal forces do not result in the fracture; however, with repetitive loading and inadequate time for healing and recovery, stress fractures can potentially occur. The debate continues whether the cause is contractile muscle forces acting on a bone or increased fatigue of supporting structures; it is most likely that both contribute [30]. Stress fractures have been reported as a common overuse injury within certain athletic populations: military personnel, runners, and ballet dancers. A self-report survey of female ballet dancers reported an overall incidence of stress fractures in 45% of the respondents. Dancers have the greatest incidence of stress fractures at the metatarsals, particularly the second metatarsal. Other common sites for stress fractures in dancers are the distal third of the fibula, sesamoid, and pars interarticularis [31]. The earliest symptom of stress fractures is pain after physical activity, but as the lesion progresses, pain may eventually start interfering with physical activity. Local tenderness may be present at physical examination and is also reproduced by vibration stimuli. MRI is the most sensitive imaging modality (approximately 88%) and is replacing bone scintigraphy as standard practice for workup of suspected stress injuries [32]. The fracture line usually extends through the cortex into the medullary canal with surrounding bone edema. MRI can also be used to evaluate other possible soft tissue injuries including muscle, ligament, and cartilage injuries. Other advantages of MRI include the fact that it can depict the anatomic details of the stress fracture (e.g., extent, angulation, displacements of the fracture) and differentiate a stress response from a stress fracture. Particularly when evaluating hip fractures, MRI has become the study of choice when radiographs are equivocal [33]. Although most stress fractures heal within 6–8 weeks, some critical skeletal sites generally have a protracted course and higher risk of completion of fracture. Some high-risk sites are the great toe sesamoids, navicular, base of the second metatarsal, fifth metatarsal, anterior tibial cortex, and femoral neck [34]. Initial treatment for stress fractures should allow for a 6- to 8-week rest period, including protected weight-bearing. Eventually, low-impact training (e.g., dance exercise in a pool) is initiated and progressed slowly to regular physical activities. Special consideration must be given to high-risk cases, because surgical intervention (e.g., fixation, bone grafting) may be required when conservative treatment does not yield the desired outcomes. Stress fractures in dancers, especially in females, have a multifactorial etiology: intensity of physical activity, and dietary restrictions lead to metabolic and hormonal imbalances, well known as the “female athlete triad” (disordered eating, amenorrhea, and osteoporosis), frequent in sports activities that place a high emphasis on esthetics [35].

Fact Box

Ankle sprain is the most common traumatic injury in dance and its incidence decreases with years of experience. Spine strain is the second most common diagnosis.

17.3 Epidemiology

Medical literature profiling the incidence of injury within modern, theatrical, and classical ballet companies reports that the incidence of injuries ranges from 17% to 95% [36]. There is a high prevalence and incidence of lower extremity and back injuries, with soft tissue and overuse injuries predominating. For example, in a study, lifetime prevalence estimated for injury in professional ballet dancers ranged between 40% and 84%, while the point prevalence of minor injury in a diverse group of university and professional ballet and modern dancers was 74% [37]. The patterns of injury seem to be fairly consistent, particularly in ballet, and the incidence of injuries in a variety of studies has been found to be greatest for the lower extremity (57–75%), followed by the ankle and/or foot (34–54%), and, less frequently, the lower back and/or pelvis (12–23%) [1].

Age and gender differences in patterns of injury within a classical ballet company were described by Nilsson and coworkers [13]. In their study, younger dancers incurred traumatic injuries more often. They reported that acute knee injuries occurred most frequently in male dancers, particularly traumatic knee injuries in soloist men, possibly because of the demands of their dance roles (e.g., performing high jumps). Acute or recent injuries are more likely to occur during rehearsal and performances rather than in class. Conversely, they found that overuse injuries, particularly of the foot and ankle, were most common in ballerinas.

Trentacosta et al. [38] did a systematic review on hip and groin injuries in dancers and found an overall rate of injury of 17.2%, with an incidence rate of 0.09 hip and groin injuries per 1000 dance hours. Eighty-five percent of hip injuries were overuse in nature, with the majority of diagnoses being tendinitis. The injury rate in professional dancers was 27.7%, and the injury rate in student dancers, was 14.1% ($p < 0.01$). Professional dancers were more prone to hip/groin injuries than their student counterparts, the etiology is unclear: it may be secondary to higher levels of training or skill, increased exposure time, or older age.

The prevalence of low back injury (LBI) in professional contemporary is 23%, in ballet dancers 32% and history of major LBI (causing more than 1 month away from dance) was reported to be 20%. Incidence of low back injury of 0.78 per 1000 dance exposures and 0.53 per 1000 dance hours were observed in ballet students. Reported incidence in professional ballet dancers was 0.63 and 0.55 per 1000 dance hours in females and males, respectively [22].

17.4 Percentage of Sports-Related Injury and Their Anatomic Locations

Ramkumar et al. [39] collected and published data regarding dancers from a single company with the dancers' age, gender, location of injury, and diagnosis. The study encompassed a 10-year period from January 2000 to December 2010. Over the 10-year span in the dance company, 574 injuries occurred. Given that the average number of dancers, including dancer turnover, was 52 per year for 10 years (520 dancer-years), the injury incidence per dancer per annum was calculated at 1.10 (574 injuries/520 dancer-years). That is to say that every year a dancer can be expected to sustain at least one new injury. The dancers' rate of injury per 1000 h was calculated to be 0.91. There were 220 foot and ankle (38%), 117 lumbar (20%), and 55 cervical (10%) injuries (Fig. 17.6).

Nilsson et al. [13] performed a combined retro- and prospective study of injuries in a Swedish professional, classical ballet company during five consecutive years. There were 390 injuries incurred by 98 dancers over a 5-year period, i.e., 0.6 injuries/1000 dance hours. Most injuries were considered to be due to overuse. The median sick leave was 2.3 weeks per injury. The foot and ankle region is vulnerable in classical ballet dancers, and overuse injuries can result in long periods of sick leave. They found considerable differences in the injury profile between male and female and between younger and older dancers. Male dancers suffered more frequently from acute injuries to the knee joint: jumper's knee was the most common diagnosis; the traumatic knee injuries included cases of distorsions and

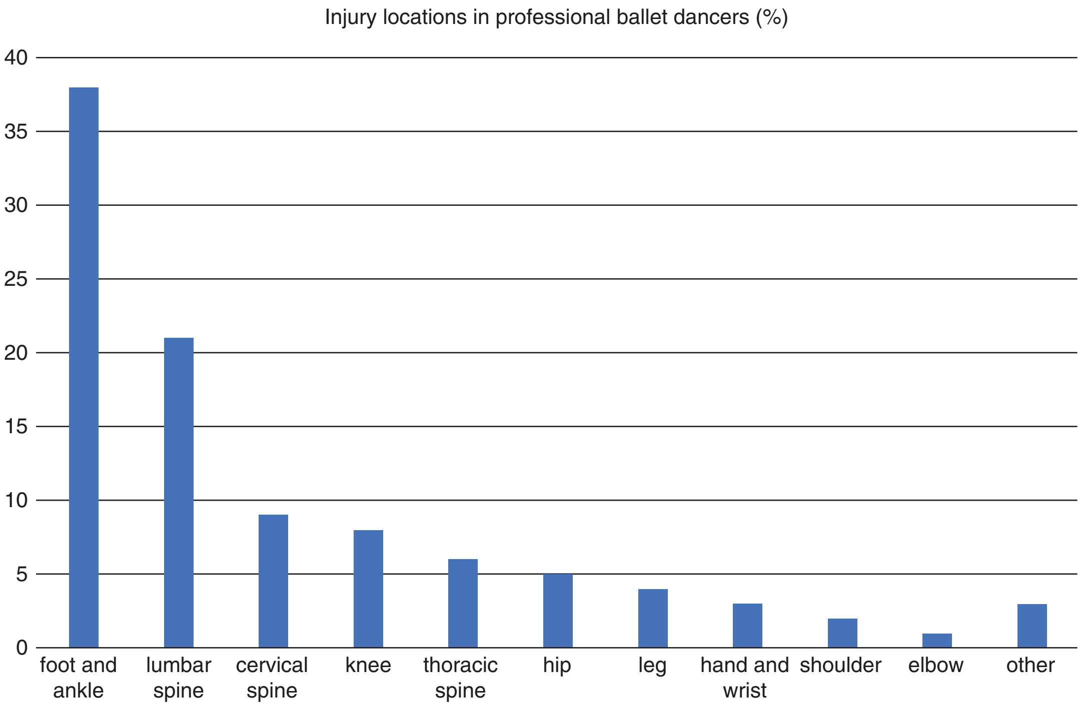

Fig. 17.6 Frequency of injury locations in professional ballet dancers. (Adaptation from Markumar et al. [39])

ruptures of the medial or lateral menisci. Traumatic injuries were seen most frequently in male soloists. Female dancers more often suffered overuse injuries, especially to the foot and ankle region. The younger dancers more often suffered traumatic injuries, for example, ankle sprain, and also stress fractures.

17.5 Prevention

The health problems of dancers are noteworthy for several reasons. First, since most dancers begin training at a young age, there is a potential for a great impact on their future health. Second, the interplay of physical and esthetic demands in dance may lead to various health issues especially relevant to dancers [37]. Injury prevention has focused on educating the performer, teachers, and staff to modify activity levels to allow for adequate rest and recovery time for the dancer's body. However, many ballet schools and touring ballet companies do not have optimal conditions that would help to prevent injuries because of lack of financial support and sometimes lack of knowledge of prevention plans. For instance, they may not have correct floor surfaces or large and ventilated training rooms. Additionally, many have inadequate warm-up spaces and no access to ice or cold packs.

Injury prevention strategies, working towards optimizing proprioceptive and core-stability capabilities, can be important in reducing the risks of injury [37]. In a review paper, Miller et al. [5] propose the following tips:

- Proper warm-up/cool-down before dancing. Dancers can use heat to warm up muscles/tendons and gently stretch calves, hamstrings, quadriceps, hips, and low back. Do for 5–10 min. Prolonged stretches greater than 30 s after class for all muscles. Ice sore areas for 20 min or 5 min of ice massage.
- Muscle soreness that goes away after 5–10 min is okay. Pain lasting longer may lead to injury. Sharp pain or persistent pain may indicate

possible injury requiring rest and medical attention.

- Avoid hard dance surfaces or obtain proper sprung flooring.
- Proper fitting footwear and possible inserts/shoe modifications.
- Proper nutrition (includes a minimum of 1200 mg calcium with 800 U vitamin D for females) and nutritionist follow-up to avoid the female athlete triad.
- Proper ballet technique. Avoid rolling in off feet; do not force turnout.
- Keep heels on the floor in plie; do not grip floor with toes. Do not hyperextend the back (sway back); do not tuck under pelvis. Keep the knee over the second toe.
- Avoid recreational activities that may add stress to the body.
- Counseling for stress management and eating disorders.

Injuries can be reduced (and medical insurance premiums) for a professional ballet company with a self-insured and company-based medical clinic on-site because dancers are used to coping with pain, muscle soreness, and minor injuries. But if left untreated, the injuries may become chronic and even more difficult to treat. Therefore, having an on-site physiotherapist and a weekly medical on-site consultation, which allows the dancers an easy access to healthcare, is mandatory for professional dancers.

Regarding adolescent idiopathic scoliosis (AIS), given that treatment outcomes improve with earlier detection, benefit would be gained from implementing formal screening in dance schools and improving the education of dance teachers and parents of dance students regarding the high rate of scoliosis in dancers. Earlier detection may allow to begin treatment sooner, reducing the likelihood of surgery and complications such as pain, reduced range of motion, pulmonary compromise, and maximizing outcomes for the dancer in terms of health, function, and career [22].

Another concern is the transition to full-time training or professional level dance. Fuller et al. [40] did a systematic review on that topic. Pre-professional dance students spend most of their day in dance class, with only 1.4% of their time spent performing. Contrastingly, professional dancers may perform seven times per week and up to 145 performances per year across 15 different programs. Adolescent dancers may increase their training to 20–30 h per week when beginning pre-professional full-time training. The transition to professional dance generally involves an increase in performance demands. Professional dancers have better aerobic fitness compared to pre-professional dancers, while lesser aerobic fitness levels have been associated with the number of injuries sustained in ballet students [41]. Therefore, dancers may benefit from undertaking supplemental cardiovascular fitness and strength training as they begin full-time pre-professional training. Vera et al. [42] evaluated the benefit of an injury prevention program consisting of a 30-min exercise program three times per week over the 52-week study period. Injuries were recorded and they found the injury rate was 82% less after adjustment for confounding variables, and time between injuries was 45% longer than for controls.

Diet and eating habits should always be evaluated because energy deficiency through diet (i.e., low caloric intake based on the amount of exercise performed) and hypovitaminosis D are among the risk factors for stress injuries. The female athlete triad consisting of disordered eating, amenorrhea or oligomenorrhea, and decreased bone mineral density also increase the risk for stress injuries. Rapid changes in training programs including increased distance, pace, volume, or cross training without adequate time for adaptation can contribute. Failure to follow intense training days with easy ones for recovery can also contribute to injury [30].

Finally, besides the physical problems, special attention to psychological disorders is mandatory, because dancers perform within a culture of excellence. Ballet dancers are committed to achieve high performing standards but regularly must reach societal standards of esthetics (i.e., lean and elegant body figure), which results in high physical and emotional stress. This promotes behavior that ignores pain and injuries.

Dancers have cumulative injuries to multiple regions of their bodies, and compared to athletes, dancers experience more anxiety and emotional difficulties. Dancers who underwent orthopedic surgery also had more cumulative trauma and injuries as well as more difficulty with emotional regulation. Increased exposure to childhood and adult traumatic events were significant predictive factors associated with an injury. It is recommended that coaches, educators, and healthcare workers understand the influence of cumulative trauma on risk for orthopedic injury and incorporate trauma-informed care [43].

Fact Box

Prevention of dance injuries is crucial and goes through educating the performer, teachers, and staff to allow for adequate rest and recovery time for the dancer's body, offering them ideal dance conditions (floor, footwear, ventilation), learning them proper ballet technique and counseling them for stress management and eating disorders.

Take-Home Message

Dance medicine is a very specific branch of sports medicine because dance is both a sport and an art. Dancers are as strong as flexible, use extreme joint range of motion, need to have a perfect balance and proprioception, have to be muscled without too much muscular volume, and with the less body fat as possible for esthetic reasons. They have to perform fluently and synchronized to music, in order to create emotions, without ever showing the difficulty of their sport and the pain they may feel due to injuries. A dancer experiences at least one new injury every year. The most frequent anatomical regions injured by ballet dancers are ankle and foot injuries accounting for 40% of all injuries, followed by lumbar and cervical spine. Dance medicine is very developed and specific journals and websites offer evidence-based knowledge and updates (www.artsmed.org., Journal of Dance Medicine and Science).

References

1. Motta-Valencia K. Dance-related injury. Phys Med Rehabil Clin N Am. 2006;17(3):697–723.
2. Garrick JG, Requa RK. Ballet injuries. An analysis of epidemiology and financial outcome. Am J Sports Med. 1993;21(4):586–90.
3. Briggs J, McCormack M, Hakim AJ, Grahame R. Injury and joint hypermobility syndrome in ballet dancers—a 5-year follow-up. Rheumatology (Oxford). 2009;48(12):1613–4.
4. Grahame R, Jenkins JM. Joint hypermobility—asset or liability? A study of joint mobility in ballet dancers. Ann Rheum Dis. 1972;31(2):109–11.
5. Miller C. Dance medicine: current concepts. Phys Med Rehabil Clin N Am. 2006;17(4):803–11, vii.
6. Brown TD, Micheli LJ. Foot and ankle injuries in dance. Am J Orthop (Belle Mead NJ). 2004;33(6):303–9.
7. Khan K, Roberts P, Nattrass C, Bennell K, Mayes S, Way S, et al. Hip and ankle range of motion in elite classical ballet dancers and controls. Clin J Sport Med. 1997;7(3):174–9.
8. Reid DC. Prevention of hip and knee injuries in ballet dancers. Sports Med. 1988;6(5):295–307.
9. Negus V, Hopper D, Briffa NK. Associations between turnout and lower extremity injuries in classical ballet dancers. J Orthop Sports Phys Ther. 2005;35(5):307–18.
10. Carter SL, Duncan R, Weidemann AL, Hopper LS. Lower leg and foot contributions to turnout in female pre-professional dancers: a 3D kinematic analysis. J Sports Sci. 2018;36(19):2217–25.
11. Novosel B, Sekulic D, Peric M, Kondric M, Zaletel P. Injury occurrence and return to dance in professional ballet: prospective analysis of specific correlates. Int J Environ Res Public Health. 2019;16(5):765.
12. Arendt YD, Kerschbaumer F. [Injury and overuse pattern in professional ballet dancers]. Z Orthop Ihre Grenzgeb. 2003;141(3):349–356.
13. Nilsson C, Leanderson J, Wykman A, Strender LE. The injury panorama in a Swedish professional ballet company. Knee Surg Sports Traumatol Arthrosc. 2001;9(4):242–6.
14. O'Malley MJ, Hamilton WG, Munyak J. Fractures of the distal shaft of the fifth metatarsal. "Dancer's fracture". Am J Sports Med. 1996;24(2):240–3.

15. Hamilton WG. Sprained ankles in ballet dancers. Foot Ankle. 1982;3(2):99–102.
16. Vosseller JT, Dennis ER, Bronner S. Ankle injuries in dancers. J Am Acad Orthop Surg. 2019;27(16):582–9.
17. Peace KA, Hillier JC, Hulme A, Healy JC. MRI features of posterior ankle impingement syndrome in ballet dancers: a review of 25 cases. Clin Radiol. 2004;59(11):1025–33.
18. Marotta JJ, Micheli LJ. Os trigonum impingement in dancers. Am J Sports Med. 1992;20(5):533–6.
19. Ahn JH, Kim YC, Kim HY. Arthroscopic versus posterior endoscopic excision of a symptomatic os trigonum: a retrospective cohort study. Am J Sports Med. 2013;41(5):1082–9.
20. Michelson J, Dunn L. Tenosynovitis of the flexor hallucis longus: a clinical study of the spectrum of presentation and treatment. Foot Ankle Int. 2005;26(4):291–303.
21. Rietveld A, Hagemans FMT. Operative treatment of posterior ankle impingement syndrome and flexor hallucis longus tendinopathy in dancers open versus endoscopic approach. J Dance Med Sci. 2018;22(1):11–8.
22. Swain CTV, Bradshaw EJ, Ekegren CL, Whyte DG. The epidemiology of low back pain and injury in dance: a systematic review. J Orthop Sports Phys Ther. 2019;49(4):239–52.
23. Burwell RG, Dangerfield PH. The NOTOM hypothesis for idiopathic scoliosis: is it nullified by the delayed puberty of female rhythmic gymnasts and ballet dancers with scoliosis? Stud Health Technol Inform. 2002;91:12–4.
24. Longworth B, Fary R, Hopper D. Prevalence and predictors of adolescent idiopathic scoliosis in adolescent ballet dancers. Arch Phys Med Rehabil. 2014;95(9):1725–30.
25. DeMann LE Jr. Sacroiliac dysfunction in dancers with low back pain. Man Ther. 1997;2(1):2–10.
26. Merckaert S, Zambelli PY. Acetabular labral tear secondary to repeated lateral “grand ecart” split exercises in an adolescent ballet dancer: case report and review of the literature. J Dance Med Sci. 2019;23(3):126–32.
27. Duthon VB, Charbonnier C, Kolo FC, Magnenat-Thalmann N, Becker CD, Bouvet C, et al. Correlation of clinical and magnetic resonance imaging findings in hips of elite female ballet dancers. Arthroscopy. 2013;29(3):411–9.
28. Charbonnier C, Kolo FC, Duthon VB, Magnenat-Thalmann N, Becker CD, Hoffmeyer P, et al. Assessment of congruence and impingement of the hip joint in professional ballet dancers: a motion capture study. Am J Sports Med. 2011;39(3):557–66.
29. Steinberg N, Siev-Ner I, Peleg S, Dar G, Masharawi Y, Zeev A, et al. Joint range of motion and patellofemoral pain in dancers. Int J Sports Med. 2012;33(7):561–6.
30. Kiel J, Kaiser K. Stress reaction and fractures. Treasure Island, FL: StatPearls; 2020.
31. Hardaker WT Jr. Foot and ankle injuries in classical ballet dancers. Orthop Clin North Am. 1989;20(4):621–7.
32. Fredericson M, Jennings F, Beaulieu C, Matheson GO. Stress fractures in athletes. Top Magn Reson Imaging. 2006;17(5):309–25.
33. 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.
34. Boden BP, Osbahr DC, Jimenez C. Low-risk stress fractures. Am J Sports Med. 2001;29(1):100–11.
35. Hincapie CA, Cassidy JD. Disordered eating, menstrual disturbances, and low bone mineral density in dancers: a systematic review. Arch Phys Med Rehabil. 2010;91(11):1777–89. e1
36. Bronner S, Ojofeitimi S, Rose D. Injuries in a modern dance company: effect of comprehensive management on injury incidence and time loss. Am J Sports Med. 2003;31(3):365–73.
37. Hincapie CA, Morton EJ, Cassidy JD. Musculoskeletal injuries and pain in dancers: a systematic review. Arch Phys Med Rehabil. 2008;89(9):1819–29.
38. Trentacosta N, Sugimoto D, Micheli LJ. Hip and groin injuries in dancers: a systematic review. Sports Health. 2017;9(5):422–7.
39. Ramkumar PN, Farber J, Arnouk J, Varner KE, McCulloch PC. Injuries in a professional ballet dance company: a 10-year retrospective study. J Dance Med Sci. 2016;20(1):30–7.
40. Fuller M, Moyle GM, Hunt AP, Minett GM. Ballet and contemporary dance injuries when transitioning to full-time training or professional level dance: a systematic review. J Dance Med Sci. 2019;23(3):112–25.
41. Twitchett E, Brodrick A, Nevill AM, Koutedakis Y, Angioi M, Wyon M. Does physical fitness affect injury occurrence and time loss due to injury in elite vocational ballet students? J Dance Med Sci. 2010;14(1):26–31.
42. Vera AM, Barrera BD, Peterson LE, Yetter TR, Dong D, Delgado DA, et al. An injury prevention program for professional ballet: a randomized controlled investigation. Orthop J Sports Med. 2020;8(7):2325967120937643.
43. Thomson P, Jaque SV. Cumulative psychological trauma, emotional regulation, and orthopedic injury in a sample of pre-professional and professional dancers and college athletes. Med Probl Perform Art. 2020;35(2):89–95.

18

Equestrian (Dressage, Eventing, Jumping)

Sports-Specific Injuries and Unique Mechanisms in Equestrian Athletes

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18.1 Introduction

Equestrian sports represent a uniquely popular group of sporting event with a wide variety of participants and injury patterns. The inter-relationship between the horse and rider is unusual in sports in that the athlete's safety depends on the performance of the horse and maintaining contact with the horse. The rider is, by definition, in at elevated position on the horse which places the athlete at an increased potential energy and elevated risk of higher energy injury due to falls from heights (rider's head is approximately 3 m above the ground), falls moving at high speeds (40–50 mph), or falls related to being entangled beneath a 1000-lb (500 kg) animal [1]. According to the United States' Center for Disease Control and Prevention (CDC), approximately 30 million people per year participate in horse riding in the United States alone, and account for nearly 50,000 yearly emergency department visits [2]. Indeed, equestrian sports have been classified as more dangerous than car racing, motorcycle riding, football, rugby, and skiing [1, 3]. The most common injured equestrian is a young amateur (20s–30s) female rider, but given its popularity as a recreational activity, people of all ages and genders are at risk for injury [4, 5]. In fact, it is one of the few gender-neutral sports and Olympic level athletes are often competitive into their 50s. While the sport's popularity continues to rise, literature on equestrian sport injuries remains sparse. Indeed, if you perform an online search of equestrian injuries, there is a much larger body of research regarding injuries to the horses than the riders. Due to the expense of owning and maintaining competitive horses, this veterinary focus is not surprising. Nonetheless, we can't lose focus on the critical importance of athlete wellness and injury prevention in equestrian athletes. At the elite and competitive level, riders compete in a variety of events or categories of equestrian activities each with their own unique injury patterns. The most commonly sustained orthopedic injuries during equestrian activities are extremity fractures, joint dislocations, spine and head injuries, as well as chest and torso injuries [6]. The purpose of this chapter is to delineate the unique mechanics and injury risk factors associated with equestrian sports, discuss the most commonly sustained orthopedic injury patterns within the sport including their epidemiology, and offer injury-specific prevention strategies.

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18.1.1 Mechanics and Injury Risk Factors

The injury mechanisms found within equestrian sports differ greatly from other sports due the high speeds, athlete elevation, and involvement of large animals; indeed, injury mechanisms vary within different categories of equestrian sport based on the unique demands and requirements. For example, dressage has less falls but an increased number of low back pain complaints due high core demands compared to rodeo which would have more traumatic injuries due to the elevated risks of falls and direct collision/contact with the horse. In horse racing, the jockeys are traveling at high rates of speeds with numerous other animals in close proximity which increases the potential energy of injury and risk of being trampled. As discussed earlier, all equestrian sports require direct interaction between athletes and an extremely powerful and sometimes unpredictable animal. While mounted on horses, equestrian athletes are at elevated heights and can travel at speeds that greatly exceed the top speeds achieved in other sports. Therefore, collisions or falls at these speeds exert a far greater energy to the participant leading to potentially more severe injuries. Falls represent the most common mechanism of significant injury across all equestrian sports. Falls can commonly result in orthopedic injuries of the upper extremity and spine, as well as a variety of head injuries (Fig. 18.1). The mechanics of riding or jumping a horse also pose unique injury risks for athletes. With each stride of the horse the rider must stabilize himself to withstand the impact using muscles throughout all extremities and the core. The repetitive wear and tear can place athletes at risk for a variety of different muscle imbalances, muscle sprains, and tendinopathies. Furthermore, while the athlete is stabilizing his or herself on the horse during competition or training, the athlete is continually and repetitively loading joints throughout the axial and appendicular skeleton. This loading through many years results in increased risk of developing arthritis in any of these joints in an accelerated manner particularly in the lumbar spine.

In addition, to the athletes riding the horses, we cannot overlook the potential risk to handlers who work in close proximity with the animals. They are integral to the success of the equestrian athlete but place themselves in harm's way when the horse becomes agitated or out of control. Kicking and rearing is a potentially dangerous horse behavior that can deliver up to 1000 N of force [7]. Overall, equestrian sports offer a wide variety of injury risk factors particularly to the musculoskeletal system due to the powerful and sometimes unpredictable nature of horses, which can place athletes and

Fig. 18.1 Fall from horses are high-energy injuries, are the most common cause of injury in equestrian athletes, and account for some of the most severe and catastrophic injuries. (© Mark R. Hutchinson, 2021. All Rights Reserved)

those that work around horses at increased risk for high-energy injuries found less commonly in other sports.

Beyond musculoskeletal injuries and head injuries, equestrian athletes, but particularly horse racing jockeys, may be at increased risk of problems related to relative energy deficits, poor nutrition, and complications related to relative energy deficit. Athletes may try to lose weight to reduce the load on the horse or as is the case in horse racing to meet a specific riding weight [8]. While weight loss is commonly pursued in a healthy controlled manner, decreased caloric intake, purging behaviors, and the use of sweat boxes, are not uncommon in horse racing jockeys which places them at increased risk of illness, overuse injury, and catastrophic injuries [9, 10]. In the extreme scenarios, athletes can drop below a healthy weight and experience symptoms of malnourishment with possible organ or endocrine dysfunction.

18.2 Categories of Equestrian Athletes/Competition

The horse is a central part of equestrian sports and the historical range of competitions is broad and extensive. There are 22 types or variations of commonly referenced equestrian sports underneath the general equestrian sports classification, which include three categories of Olympic or Paralympic disciplines (dressage, jumping, eventing). Additionally, 11 subcategories of racing further subdivided into two broad variations of English and Western riding. Rodeo includes nine events in the American style. Finally, equestrian includes the defined team sports of polo, equestrian drill team, jousting, or mounted archery. For the purposes of this chapter, we will include more in-depth discussion regarding the most popular of these sports including the Olympic sports of dressage, jumping, and eventing; horse racing with special focus on thoroughbred jockeys; common rodeo-related injuries, and finally polo as a representative of team equestrian sport.

Dressage has been described as the ballet of the equine events which requires horse and rider be judged by performing a prescribed series of specific moves and gait changes without voice commands using the reins and rider postural cues. It demands strong core control and balance for the rider and a close postural interaction with the horse. While falls may occur, the most common medical condition faced by dressage riders are chronic overuse injuries of the core muscles including low back pain. Dressage is the only equestrian event competed at the Paralympic Games. Show jumping occurs in a flat arena with the horse and rider jumping over a course of obstacles (usually colored rails) in the fastest time knocking down the fewest rails. Cross-country is the “triathalon” of the equestrian sports consisting of the composite results from three phases competed by the same horse/rider combination including dressage, showjumping, and the unique phase of cross-country. The cross-country course at the upper competitive levels is approximately four miles (6 km) long comprising approximately 34 jumping efforts over fixed and solid obstacles that mimic those found riding across the countryside including water, trees, logs, ditches, and banks at speeds averaging 550 m/min. Falls during jumps account for the most significant injuries during eventing, jumping, and cross-country competitions (see Fig. 18.1).

Horse racing comprises many disciplines with the most popular being thoroughbred track racing and steeplechase which consists of track racing with the addition of multiple fixed and solid obstacles jumping at full speed. The ultimate goal of these events is to complete the course as fast as possible. Thoroughbred refers to a group of horses whose bloodline can be traced back to one of the three original male thoroughbred horses of England. Track racing occurs over standard distances most commonly ranging from a mile to a mile and a quarter and on various flat surfaces that include turf, dirt, and artificial. Steeplechase races occur over longer distances up to four miles with fences up to 52 in. high or natural obstacles such as hedges interspersed. These events occur at high speeds up to 40–50 mph at times.

Rodeo equestrian sports include the classic "American" rodeo, bull-riding, barrel racing, reining, and roping. American rodeo includes bronc riding with or without a saddle where the athlete attempts to ride a bucking horse for 8 s. During this time, the rider uses one hand on a single rein to help stabilize himself while the other hand is free and not allowed to touch the horse. After a complete ride, both the athlete and horse are scored and ultimately combined for the total score. These general rules hold true for bull-riding as well. In these events, significant core strength is required to help stabilize an athlete while mounted and falls frequently happen. Barrel racing requires horse and rider to maneuver a predetermined pattern around three barrels for time. Reining is a form of dressage where a rider must direct a horse through a variety of movements that imitate those that could be required when herding cattle. Such movements include circles, rundown, sliding stop, backup, spins, and pauses. Scoring takes into account difficulty and precision of each movement along with the horse's cooperation. Finally, roping requires horse and rider pursue a calf with the ultimate goal of the rider capturing the calf with a lasso and restraining it. Overall rodeo comprises a group of high-risk sports that involve athletes competing both with and against powerful animals.

Polo is a popular international equestrian team sport that involves multiple horses traveling in close proximity of each other at high speeds with riders wielding mallots to score goals by striking a ball through a stationary goal. The modern format of the game can be traced back to India; however, other variations date back to a far earlier time. Teams of four mounted riders compete in field polo whereas in the arena setting there are teams of three due to the smaller playing surface. General gameplay rules center around the "line of the ball," which refers to the trajectory of the ball while in play after it is struck. The player who struck the ball has the right of way and sets the line of the ball. Opposing players cannot cross the line of the ball in front of the player with right of way unless there is adequate spacing present so as to comfortably avoid any collision. Instead one must approach parallel to the line of the ball on either side to play defense via stealing the ball, riding the opposing player off of the line, or hooking an opponent's mallet to prevent them from striking the ball. Penalties or fouls are related to improper crossing of the line of the ball which results in potentially dangerous contact between competitors. Fouls result in a stoppage of play and a penalty shot may be awarded based on the location and severity of the foul. Rules are enforced by umpires who are also horseback on the field of play. A game is divided into periods known as chukkas each lasting 7 min. Games range from 4 to 8 chukkas with the winner determined as the team with the most goals at the completion of gameplay. Polo presents unique injury risks compared to other equestrian sports given multiple mounted riders competing at a given time and the possibilities of increased contact due to the style of gameplay.

18.3 Epidemiology and Injury Patterns

Due to the rider's height from the ground, the speed of movement, and the relationship with a large animal, injuries in equestrian sports tend to be higher energy and more severe (severity score 20 and mortality rate of 7%) when compared to other high-risk sports such as football, rugby, and skiing [1]. Indeed, the distribution of injuries by anatomic body part tends to demonstrate increased percentage injuries in the more catastrophic areas of the body including head, neck, chest, and abdomen when compared to other sports (Fig. 18.2). Young et al. reviewed 27 studies relating to injuries in equestrian sports and noted a preponderance of injuries in female athletes (64.5%), the most common mechanism being falls, the most common injury type being fractures, and the most common anatomic site being the upper extremity [6]. When totaling injury totals from ten studies; 50.7 of the injuries involved the upper extremity, 22.9% involved the lower extremity, 9.4% involved the spine, 11.6% involved the chest/torso, and 4.7% involved the pelvis. McCrory et al. charted the rate of eques-

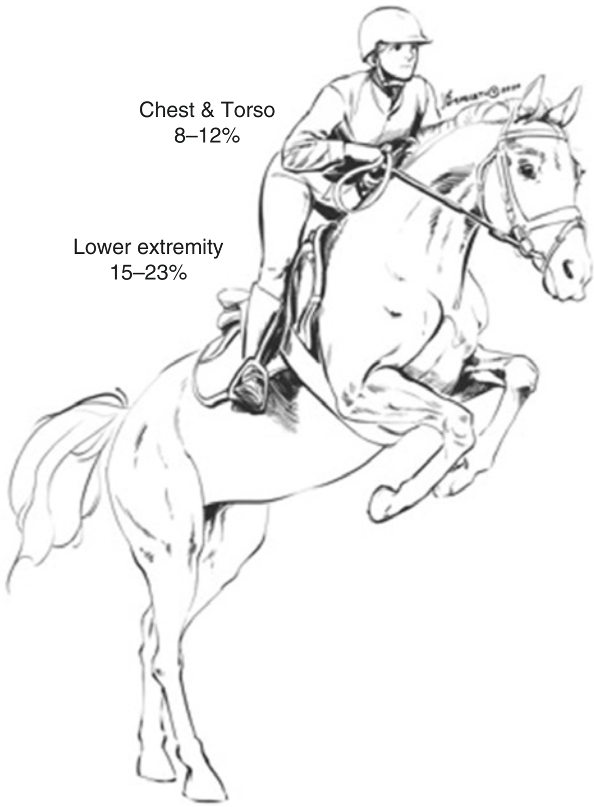

Fig. 18.2 Injuries in equestrian sports by anatomic site. (© Mark R. Hutchinson, 2021. All Rights Reserved)

trian injuries per 100,000 population versus rider's age and noted an alarming peak of intracranial injuries, forearm fractures, and ankle fractures between the ages of 10–14, particularly in females [11]. This suggests that young athletes are at elevated risk and adults need to assure these children are trained and protected to avoid and reduce the risk of these injuries when participating in equestrian activities and interacting with these large animals.

In thoroughbred and quarter horse racing, falls occur 1.99 times per 1000 rides and 3.14 per 1000 rides, respectively [12]. Thirty-nine percent of jockeys reported only one fall and 61% reported multiple falls. Indeed, there is a 51% and 59% chance respectively that a fall will cause a jockey injury. Fractures represent the most common type of injury. Catastrophic injury and death occurred in 441 per 100 million rides and 0.20% of all falls. Finally, while falls and catastrophic injuries are certainly a primary concern with these athletes, relative energy deficits and eating disorders are much more common in the jockey population [8]. Weight requirements and routine weigh-ins place significant pressures on jockeys to maintain or lose weight which can lead to energy restriction behaviors such as excessive exercise, poor energy intake, the use of sweat suits and seat boxes, laxative use, restricted hydration, and forced emesis [9, 10]. Ultimately, these behaviors can become hazardous to one's health and may increase the risk of falls during competition.

In horse racing, injury events occur 606 times per 1000 jockey years with 19% being related to the head and neck, 16% leg, 11% foot and ankle, 11% back, 11% arm/hand, and 10% shoulder [13]. Thirty-five percent of the time, the accident occurs at the starting gate which accounts for 30% of the head injuries, 40% of the arm and hand injuries, and 52% of the leg and foot injuries. Most head injuries (42%) were related to being thrown from

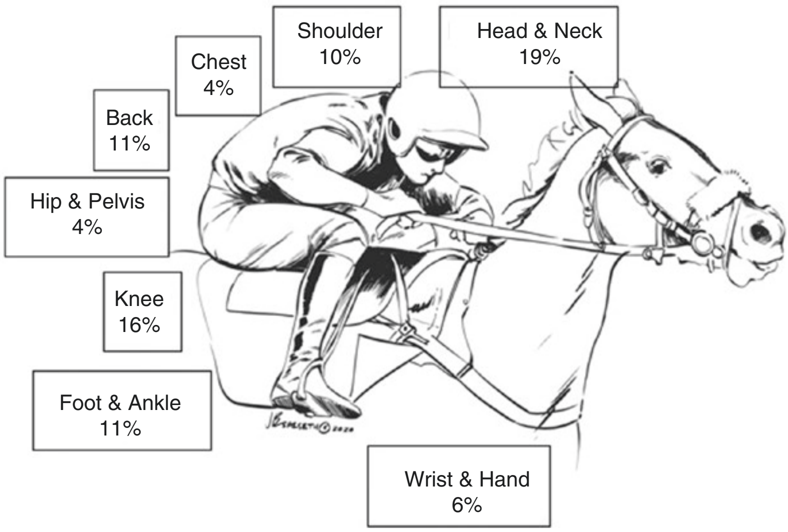

Fig. 18.3 Injuries in horse racing by anatomic site. (© Mark R. Hutchinson, 2021. All Rights Reserved)

the horse or being struck by the horse's head (23%). Being thrown from the horse accounted for 55% of the back injuries and 50% of the chest injuries in horse racing jockeys (Fig. 18.3).

Polo is an equestrian sport, a team sport, a mallet sport, and a ball sport; each facet of which introduces the potential of a unique pattern of injury risk. The risk of injury in Argentine polo players was 7.8/1000 playing hours which was less than soccer (17–29/1000 h) and rugby (53.5/1000 h) [14]. Fifty-eight percent of players reported a fall in the prior season of play with 2.5% reporting over ten falls with a slight predilection of females over males. Upper extremity injuries accounted for a majority of injuries with 39% involving the upper extremity, 31% involving the lower extremity, 25% involving the head and face, and 5% involving the spine [15].

Rodeo is a high-velocity, high-energy, high-impact sport in which the athlete competes against the clock with a usually uncooperative animal making the sport exciting but also dangerous at high risk of severe or catastrophic injury. The injury risk varies by the specific event with bull-riding carrying the greatest risk which had a relative risk ratio of 1.32 when compared to bareback riding which held a relative risk ratio of 1.39 compared to saddled bronco riding [16]. The incidence of injury in rodeo is reported to be 32.2 per 1000 competitions with most injuries occurring during the dismount [17, 18]. Furthermore, animal human contact accounts for 80% of reported rodeo injuries and again are usually associated with falls [19] Injury patterns have been found to be consistent throughout various levels of competition which includes the high school, collegiate, and professional levels [20]. Head and neck injuries account for 10–29% of injuries with concussion rates of 3.4/1000 athletic exposures. Upper body injuries are most common accounting for 63% of all injuries with thoracic, back and abdominal injuries accounting for between 11% and 84% of all traumatic injuries. Shoulder injuries accounted for 8–15% of all upper extremity cases. Lower extremity trauma accounted for between 26 and 34% of cases with a preponderance of knee injuries [19].

Key concerns and injuries by equestrian sport	
Equestrian sport	Key concerns and injuries
Dressage	Core conditioning, overuse injuries to core muscles, low back pain
Show jumping	Falls, extremity fractures, concussions, cervical spine injury
Horse racing	Falls, trampling, struck by horse, extremity fractures, head injuries, relative energy deficit, unhealthy weight loss strategies
Rodeo	Falls, trampling, concussions, fractures, cervical spine injuries
Polo	Falls, collisions, fractures, Mallot and Ball injuries

18.4 Most Common Injuries by Anatomic Location

18.4.1 Upper Extremity

Broadly, there has been some disparity regarding the most and least common injuries sustained during equestrian activities, as many factors contribute to injury pattern or prevention—such as specific disciplines, rider and horse experience level, age, and gender [3, 6, 7]. The differences may also be explained by changing injury patterns over time. Many decades ago, there was little use of protective equipment during horseback riding, and concussions and head injuries dominated injury lists. In recent years, many studies have emphasized upper extremity fractures, joint dislocations, and sprains as the most commonly sustained equestrian orthopedic injuries [6, 7]. This could partly be explained by more frequent use of protective equipment such as helmets (which are more commonly used than upper extremity protective equipment), even amongst recreational riders. One of the only comprehensive review studies on orthopedic injuries in equestrian sports by Young et al. found that fractures accounted for 33.6% of injuries in the United States, and over 50% of fractures occurred in the upper extremities [6]. Young et al. reviewed 27 studies in their evaluation of risk factors and orthopedic injury patterns in equestrian sports, and in discussing the upper extremity, they and other authors comment on the common mechanism of injury involving impact with an outstretched arm after either being thrown from a saddle or falling. Upper extremity injuries are common in pediatric patients as well. Ghosh et al. reviewed 315 patients from the National Pediatric Trauma Registry aged 19 or younger who sustained horse-related trauma. The authors discussed that in pediatric patients, injuries to the head, neck, and face were most common—but injuries to the upper extremity were the next most frequent. These injuries involved humeral fractures, fractures of the radius-ulna, clavicular fractures, shoulder dislocations, and even peripheral nerve injuries involving the brachial plexus, median, radial, and ulnar nerves [21]. Sandiford et al. further highlight upper limb injuries as most common, stating the majority of these were sustained after falls [22]. They also comment, as previous studies have, on the scarcity of protection that riders wear over their upper extremities in comparison to other protective equipment. Accordingly, when discussing prevention, there must be consideration of additional protective equipment involving the upper extremities—this could include vest protection or protective upper extremity braces but must balance the need for the rider to have flexibility and full range of motion to reduce falls [7]. Presently, no clear guidelines exist on extremity protective equipment—with respect to both recreational and professional riders.

18.4.2 Spine Injuries

Spinous injuries make up the majority of equestrian-related injuries to the torso. More specifically, a study by Gates et al. found that spinous injuries account for nearly half of all torso injuries in equestrian sports [23]. Amongst all equestrian injuries, it falls behind just upper extremity and head injuries in terms of prevalence. Within acute spine injuries, fractures are the most common pathology. Various subtypes of fractures have been reported in these cases including burst, wedge, distraction, and facet fracture dislocations. The most common mechanism for these types of injuries is falls, which can result in hyperflexion, axial loading, rotation, and shearing forces to the spine [24]. Lumbar spine has been shown to have the greatest percentage of injuries followed by thoracic then cervical spine. These injuries have been found to occur at similar rates across all age ranges [25]. Little has been studied on the long-term effects of these injuries, but Siebenga et al., found that 22% (6/36 patients) of their population had permanent occupational disability following a spine fracture [26].

Though far more rare, spinal cord injuries (SCI) do occur in equestrian sports especially in certain disciplines often related to rotational falls and can have far more severe consequences. In a review of the National SCI Statistical Center database, 121 equestrian-related injuries were found from 1973 to 2008 [24]. In comparison, a similar number of injuries were found in snow skiing and American football. These injuries most commonly resulted in incomplete tetraplegia (partial or complete loss of all limbs with retention of some sensation below the level of injury), which coincides with the general trend amongst all spinal cord injuries. Within this group C5 level was the most frequent level of preserved function. Women have been found to be more likely to sustain a spinal injury, which is likely related to a significantly higher number of participants being female. One effort to reduce spine injuries has been through the use of protective safety vests and inflatable vests. Use of this equipment is becoming increasingly required amongst certain equestrian sport governing bodies where falls are more prevalent, yet past investigations have found low use overall. Studies have found 3.6% to 14% of riders wearing a vest at the time of injury and one report found only 7% of all riders wear a vest [27–29]. However, to date no studies have shown any form of protective equipment to specifically reduce spine injuries in this population [27, 30]. This group of injuries includes a wide variety of pathologies that can have a significant long-term impact on athletes and is an area where protection efforts must continually be improved.

18.4.3 Head Injuries (Concussion, Traumatic Brain Injury)

Head injuries are frequently reported in equestrian trauma literature. In fact, many studies point to head injuries as the most commonly sustained, especially amongst pediatric patients and can occur both while mounted on the horse and while handling the horse on the ground [31–34]. Concussions are alarmingly common in rodeo athletes with a rate of 3.4 per 1000 competitive exposures as previously documented. Havlik et al. discuss that while mounted, a rider's head sits nearly 9 ft off of the ground completely unrestrained. Additionally, the rider may be traveling at speeds of up to 50 mph, placing him or her at great risk for falls and unintentional dismounts with dangerously high forces of impact [7]. Winkler et al. studied nearly 5000 cases of adult sports-related traumatic brain injuries from the National Trauma Data Bank from 2003 to 2012, and the authors found that equestrian sports were connected to the majority of sports-related TBI at approximately 45% [35]. Although the mortality rate for those who sustained equestrian-related TBI was lower than for other sports (specifically roller sports and aquatic sports), the importance of helmet use in equestrian sports cannot be overstated, as even mild TBI may carry significant long-term health risks.

While it is clear that those who participate in equestrian sports are accepting greater risk of head injuries including TBI and concussion, the focus on head injury prevention is somewhat lacking. Helmets remain the gold standard of protection in equestrian sports, reducing the risk of head injuries in both recreational and professional participants [6, 7, 35, 36]. Equestrian helmets are constructed a bit differently from helmets in other sports given the risk of both skull fractures and concussive injuries. Equestrian helmets have a rigid outer shell, and a two-layer inside made of an inner liner which absorbs energy and surrounding padding which makes direct contact with the rider's head. The energy absorbing inner liner is made from expanded polystyrene or expanded polypropylene, each of which assists with energy absorption on impact [36]. Connor et al. recently studied head injuries in equestrian athletes in the United Kingdom and United States via damaged helmet return systems. They collected over 200 damaged helmets between 2015 and 2018 in addition to accident reports from the various incidents. Associated injuries included TBI, concussions, skull fractures, and facial/soft tissue injuries. The authors performed an in-depth analysis of helmet damage patterns and resulting head trauma in athletes, and they comment that equestrian head injuries occur mainly from rotational acceleration versus linear or translational acceleration. The brain is at greater risk of injury during rotational acceleration, including more serious injuries such as cerebral edema, subdural hematomas, or diffuse axonal injuries [36]. Accordingly, helmet research and certification standards and helmet use in general must improve. Zuckerman et al. comment that rates of helmet use are at a mere 25% across equestrian sports, even given the above-mentioned benefits that helmets may provide [37]. In order to make continued efforts to decrease head injuries in equestrian trauma, the focus must remain on riders wearing protective headgear at all times while mounted. Indeed, the equestrian international governing body (FEI) recently passed rules which will become effective in 2021 that all riders are required to wear approved headgear anytime while on a horse at all sanctioned competitions. These expanding guidelines need to include not only professional riders but also recreational riders where a majority of equestrian head injuries occur.

18.4.4 Lower Extremity Injuries

Lower extremity injuries including the bony pelvis represent another major group of equestrian-related injuries. They fall behind upper extremity, head, and spine injuries in terms of overall prevalence with most studies reporting the group accounting for 15–20% of all injuries. Similar to the upper extremity, fractures are the most common pathology for this group, followed by contusions/abrasions in similar prevalence, sprains/strains less so, and finally dislocations being the least common of the group [38]. These injuries are most likely to occur from a fall while mounted. However, injuries to this region can also occur during general handling of the horse by being stepped on or struck by the horse. Loder et al. found the prevalence of various lower extremity fractures amongst all equestrian-related fractures as follows: pelvis (6.6%), ankle (4.1%), foot (2.1%), femur (1.8%), toes (0.8%), and knee (0.6%). More specifically, pelvic fractures and pelvic core muscle injuries have been found to be more associated with being thrown from or crushed by the horse in a fall [39]. Risk factors for this group are similar to those for all equestrian injuries such as less experienced younger riders and younger more unpredictable horses.

Proper riding attire remains the standard protective equipment for lower extremity injuries [7]. This includes long pants with boots. More specifically these boots commonly include a hard toe to protect the area if stepped on during general horse handling. A heel is also commonly found with these boots to help prevent the foot getting caught in stirrups during a fall, which could lead to the rider being dragged. Boots much like helmets are much more commonly worn by riders compared to protective vests. The specific impact of boots has not been studied, as it is an accepted standard with nearly all athletes wearing them. Safety stirrups have also been designed to better facilitate foot release during a fall but their effectiveness in injury reduction has not yet been studied. No other safety measures or products have been documented for injuries involving the pelvis or thigh. As one can see this group follows similar patterns to the upper extremity group in terms of pathology, risk factors, and prevention but is less common overall.

18.4.5 Chest and Torso Injuries

Chest and torso injuries represent a smaller fraction of orthopedic injuries sustained by equestrian athletes, but deserve mention nonetheless. There has been large variation in the reported incidence of chest and torso injuries compared to other orthopedic equestrian injuries, with some studies reporting a 2% incidence and the mean incidence hovering between 8% and 11% [6, 31]. Chest and torso injuries mainly occur with falls off of the horse—especially when jumping—but may also occur when the participant is interacting with the horse from the ground [7]. These injuries can be especially dangerous with respect to non-orthopedic trauma including intrathoracic or intra-abdominal hemorrhage, namely when riders fall off of the horse and are either kicked or trampled by the animal or with rotational falls [21].

While body protection is now mandatory while competing by the United States Equestrian Federation and United States Eventing Association in the discipline of eventing, literature regarding its preventative role remains sparse. In a study comparing over 600 incidents of riders wearing vests versus riders without body protection, with respect to the chest and torso specifically, the authors found that wearing body protective vests can help to prevent injuries. This effect was especially pronounced during cross-country riding, suggesting that the risk of chest and torso injuries may be given the nature of jumping solid obstacles at high speeds [40]. Further efforts need to be made to implement more widespread body protection and follow evidence based after more extensive research as well as safer jump design to reduce horse and rider falls. Andres et al. discuss experience level and cost as the main reasons for lack of body protection in equestrian athletes. While many professional organizations mandate helmet and body protection, recreational riders are not abiding by these same rules. Furthermore, equestrian vests can reach costs of $1000 or greater, partially explaining the hesitancy of sporadic participants to purchase them. Education research on injury prevention is critical and the equestrian organizations need to lead this charge to protect both the riders and horses.

Conclusion

Equestrian refers to a variety of sports that involve horseback riding. Some of the competitive sports included in this group are dressage, show jumping, eventing, horse racing, rodeo, and polo. The direct interaction with horses place equestrian athletes at unique risks not found in other sports. These risks include competing at heights and speeds that far exceed those found in more traditional sports. General interaction between athlete and horse comes with additional risks due to the powerful and unpredictable nature of the animal. A fall from the horse has been consistently found to be the most common mechanism of injury in this population. Injuries throughout the body have been reported with fractures to the upper extremity being most prevalent followed by head and spine injuries. Helmets and vests are the most common forms of protective equipment used though their use remains low amongst participants overall. Vests have been found to have variable effectiveness in injury reduction whereas helmets have been shown to have a significant impact on reducing overall severity of head injuries although the risk of concussions and traumatic brain injury remain present. Overall, equestrian sports place athletes at significant risk and further work is needed to help reduce these risks whether it be through protective equipment or regulations.

References

1. Ball CG, Ball JE, Kirkpatrick AW, Mulloy RH. Equestrian injuries: incidence, injury patterns, and risk factors for 10 years of major traumatic injuries. Am J Surg. 2007;193:636–40.
2. Carrillo EH, Varnagy D, Bragg SM, Levy J, Riordan K. Traumatic injuries associated with horseback riding. Scand J Surg. 2007;96:79–82.
3. Hasler RM, Gyssler L, Benneker L, et al. Protective and risk factors in amateur equestrians and description of injury patterns: a retrospective data analysis and a case-control survey. J Trauma Manag Outcomes. 2011;5:4.
4. Loder RT. The demographics of equestrian-related injuries in the United States: injury patterns, orthopedic specific injuries, and avenues for injury prevention. J Trauma. 2008;65(2):447–60.
5. Moss PS, Wan A, Whitlock MR. A changing pattern of injuries to horse riders. Emerg Med J. 2002;19(5):412–4.
6. Young JD, Gelbs JC, Zhu DS, Gallacher SE, Sutton KM, Blaine TJ. Orthopaedic injuries in equestrian sports: a current concepts review. Orthop J Sports Med. 2015;3:2325967115603924.
7. Havlik HS. Equestrian sport-related injuries: a review of current literature. Curr Sports Med Rep. 2010;9:299–302.
8. Hutchinson M, Benardot D, Thompson WR, Roman SB, Reynaud C, Hedrick T. Intake of energy and energy substrates in male and female professional racehorse jockeys: 571. May 28 2:45 PM–3:00 PM.
9. Benardot D, Thompson WR, Hutchinson M, Roman SB, Hedrick T, Reynaud C. Urine specific gravity is unrelated to BIA or skinfold-derived body fat percent,

but is related to weight in professional racehorse jockeys. 2251Board #31 May 30 3:30 PM–5:00 PM.
10. Benardot D, Thompson WR, Hutchinson M, Doizer LM, Speake NK. Weight loss practices of professional racehorse jockeys. 2903Board #207 June 1 8:00 AM–9:30 AM.
11. McCrory P, Turner M. Equestrian injuries. In: Caine DJ, Maffulli N, editors. Epidemiology of pediatric sports injuries. Individual sports, Med Sports Science, vol. 48. Basel: Karger; 2005. p. 8–17.
12. Hitchens PL, Hill AE, Stover SM. Jockey falls, injuries, and fatalities associated with thorougbred and quarter horse racing in California, 2007–2011. Orthop J Sports Med. 2013;1:232596711349262. https://doi.org/10.1177/2325967113492625.
13. Waller AE, Daniels JL, Weaver NL, Robinson P. Jockey injuries in the United States. J Am Med Assoc. 2000;283(10):1326–8.
14. Inness CM, Morgan KL. Falls and injuries to polo players: risk perception, mitigation, and risk factors. Sports Med Open. 2015;1:2. https://doi.org/10.1186/s40798-014-0002-8.
15. Costa-Paz M, Aponte-Tinao L, Musculo DL. Injuries to polo riders: a prospective evaluation. Br J Sports Med. 1999;33(5):329–32.
16. Butterwick DJ, Hagel B, Nelson DS, LeFave MR, Meeuwisse WH. Epidemiologic analysis of injury in five years of Canadian professional rodeo. Am J Sports Med. 2002;30(2):193–8.
17. Downey DJ. Rodeo injuries and prevention. Curr Sports Med Rep. 2007;6:328. https://doi.org/10.1007/s11932-007-0071-2.
18. Sinclair Elder AJ, Nilson CJ, Elder CL. Analysis of 4 years of injury in professional rodeo. Clin J Sports Med. 2018; https://doi.org/10.1097/JSM.0000000000000654.
19. Meyers MC, Laurent CM. The rodeo athlete: injuries. Sports Med. 2010;40:817–39.
20. Sinclair AJ, Smidt C. Analysis of 10 years of injury in high school rodeo. Clin J Sports Med. 2009;19(5):393–87.
21. Ghosh A, Di Scala C, Drew C, Lessin M, Feins N. Horse-related injuries in pediatric patients. J Pediatr Surg. 2000;35:1766–70.
22. Sandiford N, Buckle C, Alao U, Davidson J, Ritchie J. Injuries associated with recreational horse riding and changes over the last 20 years: a review. JRSM Short Rep. 2013;4:2042533313476688.
23. Gates JK, Lin CY. Head and spinal injuries in equestrian sports. Curr Sports Med Rep. 2020;19(1):17–23. https://doi.org/10.1249/jsr.0000000000000674.
24. Lin CY, Wright J, Bushnik T, Shem K. Traumatic spinal cord injuries in horse-back riding: a 35-year review. Am J Sports Med. 2011;39:2441–6.
25. Liaw F. Injury patterns of equine-related trauma. Open Orthop J. 2019;13(1):130–5. https://doi.org/10.2174/1874325001913010130.
26. Siebenga J, Segers MJM, Elzinga MJ, Bakker FC, Haarman HJTM, Patka P. Spine fractures caused by horse riding. Eur Spine J. 2006;15(4):465–71.
27. Puschel V, Michaelsen U, Giensch M, et al. The question of safety in horse-riding sports. Sportverletz Sportschaden. 2012;26:159–63.
28. Mayberry JC, Pearson TE, Wiger KJ, Diggs BS, Mullins RJ. Equestrian injury prevention efforts need more attention to novice riders. J Trauma. 2007;62(3):735–9.
29. Kiss K, Swatek P, Lénárt I, Mayr J, Schmidt B, et al. Analysis of horse-related injuries in children. Pediatr Surg Int. 2008;24(10):1165–9.
30. Kate CA, de Kooter TA, Kramer W. Prevention of injuries associated with horseback riding. Ned Tijdschr Geneeskd. 2015;159:A8624.
31. Barone GW, Rodgers BM. Pediatric equestrian injuries: a 14-year review. J Trauma. 1989;29:245–7.
32. Clarke CN, Tsuei BJ, Butler KL. Equine-related injury: a retrospective analysis of outcomes over a 10-year period. Am J Surg. 2008;195:702–4.
33. Grossman JA, Kulund DN, Miller CW, Winn HR, Hodge RH Jr. Equestrian injuries. Results of a prospective study. JAMA. 1978;240:1881–2.
34. Hobbs GD, Yealy DM, Rivas J. Equestrian injuries: a five-year review. J Emerg Med. 1994;12:143–5.
35. Winkler EA, et al. Adult sports-related traumatic brain injury in United States trauma centers. Neurosurg Focus. 2016;40(4):E4.
36. Connor TA, et al. Do equestrian helmets prevent concussion? A retrospective analysis of head injuries and helmet damage from real-world equestrian accidents. Sports Med Open Access. 2019;5:19.
37. Zuckerman SL, Morgan CD, Burks S, Forbes JA, Chambless LB, Solomon GS, et al. Functional and structural traumatic brain injury in equestrian sports: a review of the literature. World Neurosurg. 2015;83:1098–113.
38. Loder RT. The demographics of equestrian-related injuries in the United States: injury patterns, orthopedic specific injuries and avenues for injury prevention. Trauma. 2008;65:447–60.
39. Meredith L, Ekman R, Brolin K. Epidemiology of equestrian accidents: a literature review. Internet J Allied Heal Sci Pract. 2019;17:1–15.
40. Andres SA, et al. Effects of body protection vests and experience levels in prevention of equestrian injuries. BMJ Open Sport Exerc Med. 2018;4(1):e000426.

Fencing

Epidemiology and Organizational Prophylaxis

19

Peter A. Harmer

19.1 Introduction

Although an exact date for the "beginning" of modern competitive fencing is the subject of considerable debate by fencing historians, two distinct international competitions, the Olympic Games (OG) and the World Championships (WC), are readily verifiable as foundational episodes in its history, and both can be used to track its evolution and growth from an exemplar of European aristocratic privilege to international egalitarian contests. For example, fencing was included in the first OG of the modern era in 1896, but only involved athletes from Europe, with an event for professionals (an exception for participation in the OG, also allowed for fencing in 1900, which would not occur again for any sport for more than 90 years)—and no events for women. It was not until the Games of the fifth Olympiad in 1912 that the fencing program was standardized to the three disciplines currently contested: foil, epee, and saber [1]. In contrast, the Fédération Internationale d'Escrime (FIE), the international governing body for modern competitive fencing responsible for organizing the World Championships, was not founded until 1913 and did not organize its first European Championships until 1921. These competitions between 1921 and 1936 were retroactively titled World Championships [2].

Each organization has pioneered different aspects of the development of the sport—the OG had the first international competitions for men's foil (individual, 1896; team, 1904), men's epee (individual, 1900; team, 1908), men's saber (individual, 1896; team, 1908), and women's foil (individual, 1924). However, the FIE WC was first to include a women's foil team event (1932; 38 years before the first such event in the OG), women's epee (both individual and team, 1988 vs. 1996 for OG), and women's saber (individual and team, 1999 vs. 2004 and 2008 for OG, respectively). While the time lag from the first women's epee and saber WC to inclusion in the OG program was less than a decade, neither organization was particularly proactive across the twentieth century in terms of gender equality with gaps of 70–100 years from the introduction of these events for men in OG and WC until they were made available for women. However, the FIE WC have had full gender parity since 1999. In addition to expanding opportunities for women in international fencing, the FIE has continued to nurture the sport around the world. As a

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result of its diversity and expansion plan beyond its traditional European base, in the past 20 years it has increased the number of member nations by approximately 42%, to the current total of 157, primarily from Africa and Asia [3].

The success of the FIE's diversity and growth plan can also be gaged by examining the number of nations qualifying to compete in the OG. Using the OG boycotts of 1980 and 1984 as a starting point, the Games of the eight Olympiads prior (1948–1976) had an average of 33 nations represented (range 23–42). However, the eight OG since (1988–2016) have had an average of 44 nations included (range 40–48), even with more stringent controls on the total numbers of athletes permitted [1]. In 2017, the International Olympic Committee announced the OG for Tokyo 2020 would be the first for fencing to have full parity for events and gender (all three disciplines, individual and team, for both men and women).

19.2 The Sport

Fencing consists of three disciplines, foil, epee and saber, each with distinct performance requirements and potential injury risk. Both foil and epee are "point" weapons, that is, a thrusting motion is used to push the tip of the blade onto an approved target area to register a valid touch. The saber is considered primarily a "cutting" weapon, that is, although the point may be used to score, touches in saber are typically scored with the edge of the blade by a cutting action of the arm and wrist. The target area for foil is the torso, front and back, but not the head. For epee, the entire body is valid, and for saber, the torso above the waist, including the head, is valid. A point is scored when the referee determines that a fencer has hit the opponent's valid target area in accordance with the rules. Hits are registered electronically and instantaneously activate a light on a scoring machine to assist referees in their assessments.

Competitions are generally conducted in two phases: (a) a preliminary round of pools of 6–7 fencers competing in a round robin format of bouts to a maximum of 5 points or 3 min, whichever comes first. If time expires before one of the fencers reaches 5 points, the competitor with the lead is declared the winner. (b) the top 70–80% of competitors in the preliminary round will be promoted to a direct elimination format consisting of three, 3-min periods, with a 1-min rest between periods, to a maximum of 15 points. Because of the rapidity of saber fencing in general, there is an exception in the regulations to allow the break when one fencer reaches eight points. Team events entail three athletes per team in a relay-type format, that is, each fencer on a team will fence against each fencer on the opposing team for a total nine individual bouts. Each individual bout is a maximum of 3 min; the score is a running tally—the first team to reach 45 is the winner.

The field of play for fencing (the piste) is 1.5 m wide × 14 m long and constructed of a conductive material so that if a point hits the floor during a bout, nothing will register on the scoring machine. To minimize the risk of fencing-specific injury (i.e., blade-related laceration or punc-

Fig. 19.1 Minimizing the risk of fencing-specific injury involves extensive and mandatory personal protection. Photo credit: Serge Timacheff. Used with permission

ture), all areas of the body directly accessible to an opponent's point (i.e., anterior and lateral surfaces), except for the non-fencing hand, must be completely covered by approved clothing (mask, jacket, pants, socks, shoes, and glove) (Fig. 19.1).

19.3 Biomechanics

Fencing can be classified as a combat sport and a dual sport, but it has unique characteristics within each category. As a combat sport it involves only one arm, and as a dual sport it involves direct contact with the opponent. Moreover, it is different from other sports in each category in that its field of play is narrow and linear. All other combat and dual sports have greater capacity for lateral activity. Additionally, to minimize the available target area presented to an opponent, the standard fencing position is, in effect, a stance in profile which is thought to place the back leg, especially the ankle and knee, in vulnerable positions. These characteristics emphasize the typical asymmetrical biomechanical demands of the sport which are thought to influence injury risk profiles (Fig. 19.2). For example, the ankle of the back leg is usually slightly plantar-flexed and set perpendicular to the line of movement on the piste to facilitate rapid attacks, especially in lunging, but this position also potentially increases the risk of inversion sprain from foot misplacement during retreats from an opponent's attack. However, recent research indicates that 68% of time-loss ankle sprains occur in the front foot, primarily from slipping or contact with the opponent in an attack [4]. By contrast, the prevalence of strains in the front thigh (91%) [4] fit with the understanding of high eccentric loads in the quadriceps and hamstrings during a lunge.

The ballistic nature of lunging also involves high loading forces in the front leg on landing which may increase the risk of impact-related injury (Fig. 19.3). For example, Hayashi et al. [5] found fencers accounted for 2 of 25 cases of stress reaction in the leg diagnosed by magnetic resonance imaging across 11,274 athletes at the 2016 Olympic Games. While the significance of this finding is unclear

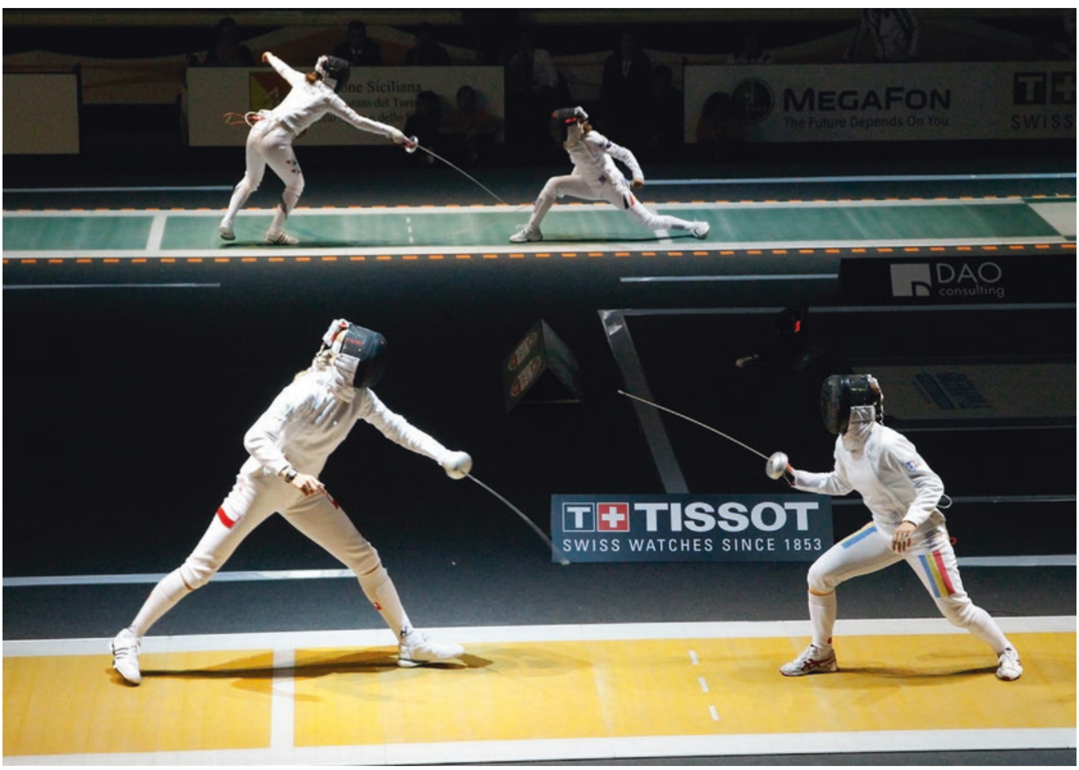

Fig. 19.2 The narrow field of play and asymmetrical nature of fencing impose unique biomechanical demands on participants. Photo credit: Serge Timacheff. Used with permission

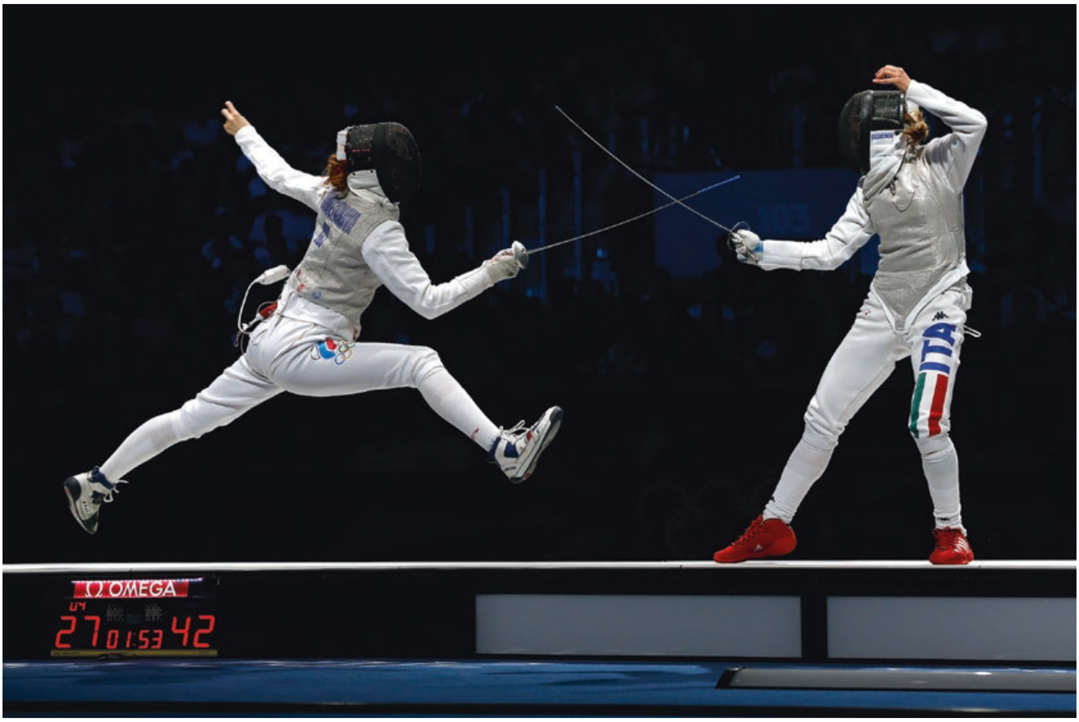

Fig. 19.3 Aggressive attacking may induce acute or overuse injuries through high impact forces in the lead leg. Photo credit: Serge Timacheff. Used with permission

because of the low numbers involved, it has been demonstrated that this theoretical risk can be attenuated by using shoes and/or pistes with greater cushioning [6, 7]. However, these solutions are problematic in that better cushioned shoes impede performance and universal piste cushioning has significant technical and financial barriers.

Fact Box

1. The asymmetrical biomechanical forces of fencing and its narrow field of play have variable influence on the risk and nature of injuries.
2. Options for reducing injury risk identified by biomechanical analysis may be limited by higher priorities including enhancing performance and limiting costs.

19.4 Epidemiology

The general conventions of fencing technique have not changed significantly over the past 125 years. However, the technical developments in equipment (especially the move to electric scoring: epee 1936, foil 1956, saber 1988) and changes in competition format over the past 30 years make it very problematic to use data prior to 1990 as a basis for estimating injury risk of fencing. Additionally, methodological limitations, including small sample sizes, short time lines, indistinct criteria for reportable cases and inconsistent metrics, in the majority of the few studies in the literature with exposure data have produced questionable incidence estimates [4, 8–10]. Nonetheless, older studies can augment recent research for descriptive epidemiological parameters (specifically injury type and location) to highlight both rare and robust phenomenon associated with injury connected to the fundamentals of fencing movements.

As the most basic research design in descriptive epidemiology, case reports are especially useful for identifying atypical findings that may be completely unique or may be the foundation for a case-series analysis that uncovers previously unknown injury relationships. For example, case reports in fencing contain "one-off" situations such as osteochrondritis dessicans of the patella [11], Masson's tumor in fencing hand [12], tibialis anterior tendon rupture [13], hamstring avulsion [14], and isolated rupture of radial collateral ligament of the metacarpophalangeal joint of the index finger [15]. However, they have also captured significant puncture and laceration events, the pre-eminent sport-specific injury concern in fencing, such as a distant entry pneumothorax [16], translaryngeal puncture [17], and chest and groin lacerations [18] that are important for developing insights for prevention interventions. For example, although there have been only 11 reported fatalities in fencing worldwide [19] since the first in an official international competition in 1937 [2], case report details indicating that all involved head, neck, or chest penetration (9 by a broken blade) and that the three most recent cases (2004–2009) had no plastron (a mandatory piece of clothing to provide additional protection for the neurovascular bundle in the axilla of the fencing arm, the lateral chest wall on the same side and the neck) [19] have lead the FIE to focus on equipment standards as the most effective approach to mitigating this primary risk.

19.4.1 Incidence

Eleven studies with exposure data have been published since 1990 [4, 10, 20–28], but two [27, 28] have significant methodological concerns that negate their utility [4]. Of the remaining nine, although three [21–23] employed a case definition of any injury for which medical attention was sought, two [21, 23] also provided data from which the more meaningful standard of "time-loss injury" [26] could be calculated (as was the criterion in the other six studies). Only 4 of the 11 studies involved more than 200 participants and/or more than 1 year of data collection [4, 24–26].

Overall, the available literature indicates that the risk of sustaining any time-loss injury in fencing is very low, with estimates in the range of 0.0–0.25/100 participants [21, 23] to 1.2–2.4/1000 h participation [10, 20] for the smaller studies and 0.2–0.3/1000 athlete exposures [AE] for the four largest (which reported across a wide array of competition settings and participants, and also had values of 0.1–0.2/100 participants) [4, 24–26]. Concomitantly, only two of these were large enough to calculate the incidence of fencing-specific injuries, which averaged 0.009/1000AE (0.008/1000AE [4] and 0.01/1000AE [26] from 11 incidents (9 penetrations (7 involving the hand); 2 lacerations) across 163,909 participants over 10 years of surveillance.

19.4.2 Sex and Age

The impact of sex and age as risk factors has varied as competitive opportunities and performance standards have increased for women and younger athletes over the past 30 years. For example, in a small study of collegiate fencers, Lanese et al. [20] found women had an 80% greater risk of sustaining a time-loss injury than men (0.18 vs. 0.10/100 person hours of participation) but that men had more than double the time loss (0.43 vs. 0.21 disability days/100 person hours), indicting men sustained more severe injuries overall. The complexity of the relationship between sex and injury is highlighted by the findings from the two largest studies ever conducted on fencing injuries. A 5-year study of national competitions in the USA from 2001 to 2006 [26] reported women at 33% greater risk than men (0.36 vs. 0.27/1000AE), whereas a comparable 5-year study of FIE international competitions from 2010 to 2014 [4] found men at a 45% greater risk (0.32 vs. 0.22). A potential explanation may be related to the influence of women's saber results. The first World Championship for the event was not until 1999, so few of the women participating in the USA study had an extensive background in the weapon, which may account for this group having a statistically significant higher incidence of time-loss injury than any other sex/weapon combination (e.g., 0.51 vs. 0.37 for men's sabee, or 0.33 for women's foil). However, by the time of the FIE study a generation of women had grown up specializing in this event, and the incidence at this higher level of competition was 0.22/1000AE.

Similarly, age as a risk factor is uncertain, and the available incidence data do not support the suppositions that younger athletes are more at risk because of inadequate physical development or that older athletes may be because of declining physical capacity. Additionally, the influence of age is often confounded by skill and experience, with elite adults being stronger and faster than adolescent and youth participants. For example, although no significant differences were found in the incidence of time-loss injuries across three age grouping in a large national sample (youth: 0.27/1000AE; adult: 0.20–0.35; adults 50+: 0.21) [24] or national and international championships for adults 50+ (0.23) [25], results from the FIE international study indicated fencers in senior open events were at 74.3% greater risk than those in junior competitions for athletes aged 13–20, although the incidence in the adult group was still low (0.33 vs. 0.19/1000AE) [4].

19.4.3 Injury Type

Although understanding the frequency of different types of injury is important for medical coverage plans for competitions, identifying those most associated with time loss is key to developing potential substantive prevention protocols. In fencing, minor insults to the integumentary system (blisters, abrasions, contusions) are the most common injuries, accounting for ~48–65% of treatments at competitions, but they rarely require any other than minimal treatment or result in time loss [21, 29].

In contrast, the research has consistently shown acute musculoskeletal injuries as the primary causes of time-loss injuries, with prevalence ranging from ~20% to 44% for first and second degree strains and ~25.5–45% for sprains [4, 10, 26, 30]. Third degree strains and sprains account for an additional 4–10% of all time-loss injuries [4, 26].

Overuse injuries, especially tendinitis of the lateral elbow and patellar tendon, and plantar fasciitis, are well known in fencing [31, 32] but not well documented as causes of time loss from participation.

19.4.4 Injury Location

In studies that use the "any request for medical assistance" criterion, the wrist/hand accounts for up to 60% of injuries [21]. However, these are usually minor contusions and abrasions that do not affect participation (only ~5% of wrist/hand injuries are time loss) [4, 26]. Conversely, the research is consistent in identifying the lower extremities as the primary location for time-loss injuries, with prevalence ranging from 40 to 72% [4, 10, 26, 30, 31]. The most important specific regions within the lower extremities are the knee (20–42% of all time-loss injuries) and the ankle (16–26%) [4, 10, 22, 26, 30].

19.4.5 Training vs. Competition

Few fencing studies have documented the risk of injury in training and competition, but the available data are congruent with findings from other sports that training accounts for the most injury events (~75%) [10, 22], but competition has the greatest risk (for example, RR from 2.55 [10] to 3.25 [33].

19.4.6 Mechanisms

To date, only one study has fully documented the mechanisms of time-loss injury in fencing competition. In the study of international events, Harmer [4] found 47.1% of cases were non-contact (which is congruent to the 39% reported by Engebretsen et al. [32] from the fencing competition at the 2012 Olympic Games), 28.2% due to the athlete slipping/falling, 19.5% resulting from contact with the opponent (Fig. 19.4), and 5.2% from the opponent's blade.

19.4.7 Prevention

The unique "profile" positioning of fencers and the preponderance of acute musculoskeletal injuries as primarily responsible for time loss from training and competition has led to standard recommendations for injury prevention programs to focus on whole-body strength and conditioning to address the perceived risks associated with muscle imbalances [30, 34]. While such advice has common-sense appeal, there have been no studies to demonstrate the veracity of this posi-

Fig. 19.4 Contact with the opponent in close quarter actions is a leading mechanism for injuries. Photo credit: Author

tion [9, 35]. Overall, given the low incidence and stochastic nature of time-loss injuries in fencing, it is unlikely that experimental evidence will identify interventions that can substantially decrease the risk of these injuries.

Fact Box

1. Fencing-specific injuries are very uncommon and rarely result in significant consequences.
2. The most common injuries in fencing are minor skin trauma (abrasions, blisters, contusion) that require minimal treatment.
3. Acute musculoskeletal trauma (sprains, strains), typical of dynamic change-of-direction sports, account for the majority of time-loss injuries in fencing.

19.5 Safety

Because of the continuing misperception in the general public of the risks associated with fencing based on its historical origins as a means of lethal combat, the FIE is acutely aware of the need to ensure that fencing-specific injuries (i.e., lacerations and penetrations) are minimized/eliminated [2]. Incidents involving significant injuries of these types often receive wide media coverage (e.g., [36]) that overshadows the reality of how safe fencing is as a sport and potentially dissuades participation, especially in terms of parents concerned about their children's safety. Therefore, the FIE has utilized three main strategies to achieve its primary goal: (a) regulations related to the manufacturing and physical characteristics of fencing equipment (blades, masks, clothing, gloves, and piste) and the administration of competitions, (b) rules regulating athlete behavior in competition, and (c) specifications for medical coverage at competitions.

Although recent data on the incidence of fencing-specific injuries [4] indicate these measures appear to have been successful, changes have generally been in response to a significant failure in the existing equipment or rules rather than as a result of a proactive agenda based on research. For example, the plastron was introduced after a fatal injury at the 1951 World Championships [37, 38], but a standard for integrity of 800 N was not introduced until 1987. Similarly, it took the death of World and Olympic Champion Vladimir Smirnov at the 1982 World Championships due to the penetration of a broken blade through his mask [37, 39] for significant structural changes to masks to be initiated and standardized testing for mask integrity to be instituted, initially with a 7 kg punch test in 1984 and subsequently to 12 kg in 1996. Concomitantly, a move to nickel maraging steel, which has a significantly lower fracture rate than the traditional carbon steel used for blades, was not integrated into the rules for foil and epee until 1993. More recently, evidence to extend the requirement for maraging steel blades for saber was presented to the FIE in 2011 [40], but this change was not implemented until the 2020–2021 season [41]. Delays in the implementation of safety changes are often the result of conflicting priorities for the federation, such as financial and political considerations [2, 42].

19.5.1 Equipment Regulations

The physical characteristics of the weapons, including length, weight, cross-sectional area, flexibility, size, and shape of the guard and the tip (as well as spring force of the tip for foil and epee), and the composition of the steel used are precisely detailed in the Materials Rules [41], and all blades used in official competitions must be certified as conforming to the regulations. Of particular interest is the bending test to gage resistance to fatigue (breaking), with the standard for foil being 18,000 cycles and 7000 cycles for epee. Similarly, the chemical composition and gage of the wire used for masks, as well as the shape of the mask and mesh size, are specified within very narrow parameters to minimize the possibility of a blade penetration. The bib (the extension of the mask covering the neck) must be 10–12 cm in length inferiorly and be resistant to penetration of a force of at least 1600 N. As a result of several episodes of masks falling off during rapid change-of-direction actions during bouts in recent years (Fig. 19.5), each mask must have two different safety systems at the back to keep it securely in place.

To complement the strict standards designed to minimize the potential for broken blade injuries, the current regulations dictate that all clothing (jackets, plastron, trousers) must be resistant to 800 N, which means vulnerable areas covered by the plastron are effectively protected to 1600 N. Furthermore, a rigid chest protector, commonly constructed of thermoplastic and worn under the jacket, is obligatory for all female fencers (optional for male athletes). Additional safety specifications for clothing are event-specific. For example, there are no integrity requirements for gloves for foil (the hand is not a target) or epee (the guard is uniquely designed to protect the hand) but following a case-series analysis of unusual non-broken blade hand penetrations in 2011 [42], an 800 N standard was instituted for saber gloves in September 2013. Similarly, there are no standards for socks (although the leg is susceptible to blade injuries as a target in epee; Fig. 19.6) or shoes.

In addition to the dimensional standards noted previously, pistes must conform to requirements for thickness, traction, and tensile strength to reduce the risk of athletes slipping or tripping in the dynamic back-and-forth actions in a bout. However, they must also meet a standard for conductivity, which requires they be made of metallic mesh, metal plates, or conductive fabric. Initially woven copper mesh was used, but these pistes were expensive, heavy, and susceptible to tearing, which could result in significant musculoskeletal injury. The development of aluminum plates solved several of these problems but increased the risk of ankle and knee sprains when fencers mis-stepped on the edge of the plates [22]. The evolution of the current genera-

Fig. 19.5 As fencing competition becomes more dynamic, equipment upgrades for safety, such as additional security constraints for masks, are required. Photo credit: Serge Timacheff. Used with permission

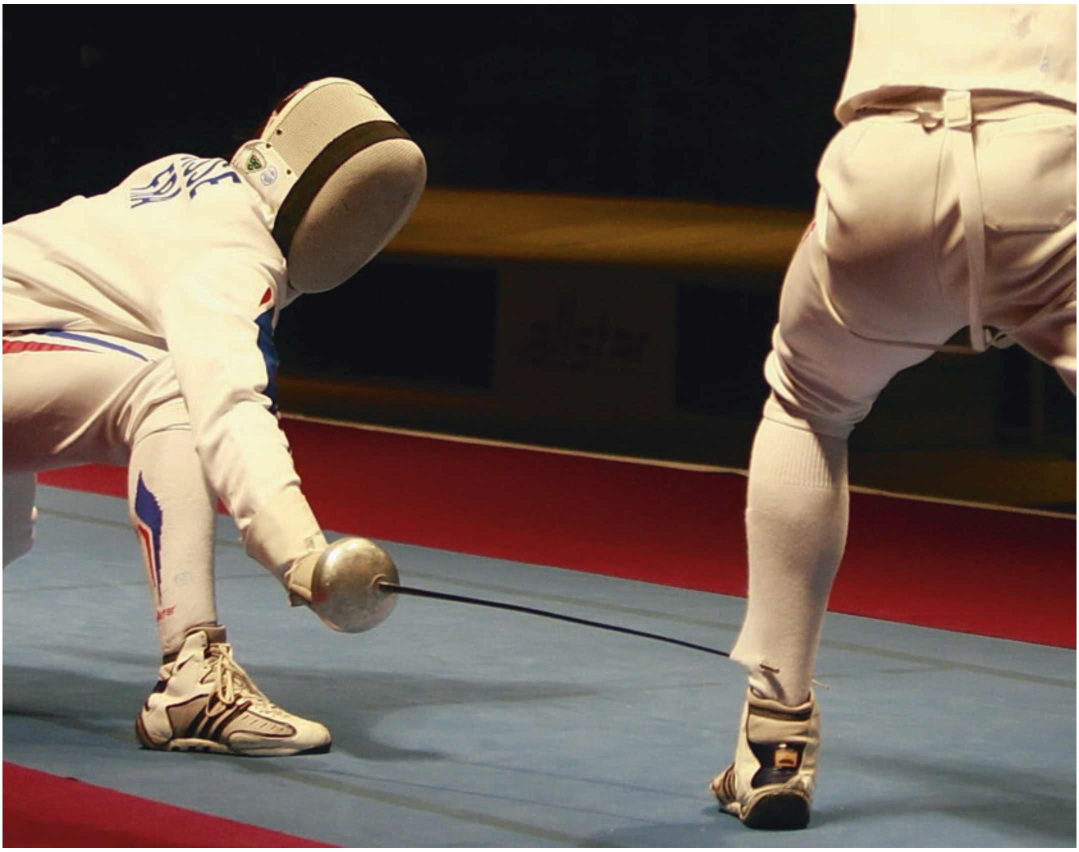

Fig. 19.6 Although event-specific vulnerabilities exist, current epidemiological evidence does not support changes in protective equipment rules to address them. Photo credit: Serge Timacheff. Used with permission

tion of conductive fabric for pistes, prompted by cost and ease-of-use considerations, seems to have alleviated the injury risk potential associated with the physical characteristics of previous piste construction.

19.5.2 Rules Regulating Athlete Behavior in Competition

Rules regulating athlete behavior can be divided into two general categories: (a) those designed to ensure adherence to equipment regulations, and (b) those designed to limit injury as a result of mal-intent [43]. The logic of the rules in the first category is readily apparent—the value of protective equipment is nil if it is not used, or not used appropriately. Therefore, there is a variable scale of penalties if athletes use non-conforming clothing or equipment or warm-up/train without protective clothing. Similarly, turning the back to an opponent during a bout or removing the mask without permission are prohibited as these actions remove protection from the head and/or back of the neck. The second category regulations arise from the dynamic physical and intense psychological nature of fencing and the fact that although the blades are very safe to use as intended they are still made of steel and could cause serious injury if misused. Thus, there are rules against body contact with an opponent during a bout (even if inadvertent) and significant penalties, including immediate disqualification and variable suspension from future competitions, for dangerous, violent, or vindictive actions or deliberate brutality.

19.5.3 Specifications for Medical Coverage at Fencing Competitions

Recognizing that serious injury can occur in fencing competitions, even with the use of extensive protective equipment, the FIE established a Medical Handbook of Specifications [44] to ensure appropriate medical care at its major competitions (World Championships, Zonal Championships, Grand Prix/World Cups) and to provide a model for other fencing organizations (e.g., national federations, regional clubs). The requirements are presented in a matrix, cross-referencing six levels of medical service (medical emergencies, sports trauma, general medical care, additional medical services, doping control, and medical supervisors) with the three competitions covered. Each cell is designated as essential, desirable, or not required. Reflecting the primary focus on preventing significant/potentially fatal fencing-specific injuries, provision of a locally licensed specialist health-care provider trained in advanced life support with requisite resuscitation equipment for cardiac and respiratory arrest is essential for all competitions, as is an on-site fully equipped ambulance (this requirement is waived in instances of verifiable response times of less than 10 min). Similarly, the presence of an appropriately equipped sports medicine professional to care for the most common competition-related injuries (sprains, strains) is also designated essential for all three competitions. By contrast, addressing general medical care is rated essential for World Championships, desirable for Zonal Championships and not needed for Grand Pix/ World Cups. The variations in requirements across this matrix reflect considerations of the available epidemiological evidence, the public relations implications, and the financial impact. The relative importance of each of these factors differs depending on the characteristics of the participants (children, beginners, veterans, elite athletes) and events (local, regional, national) with the result that there are considerable disparities in the level of medical care available across the spectrum of sanctioned competitions.

Despite these differences, the rule governing medical intervention during a bout (i.e., immediate acute care) is universal. Item t.45 of the Technical Rules [43] indicates that a fencer must be evaluated on the piste by the medical professional on duty at a competition to verify an injury, cramp, or acute medical incident before any treatment can be undertaken. Subsequent treatment on the piste for the same injury during the same day is not permitted. While these requirements are designed to balance the well-being of competitors with the smooth running of competitions, the decision to reduce the maximum on-piste treatment time by 50% (from 10 min to 5 min), effective from January 2018, indicates a shift in the priorities of the FIE. To date, no data on the impact of this change on the incidence of time-loss injuries or other metrics of athlete health have been reported.

19.6 Wheelchair Fencing

Although wheelchair fencing is governed by the International Wheelchair and Amputee Sports Federation (IWAS) rather than the FIE, it shares several parallels with able-bodied fencing: the same three disciplines are contested as both individual and team events, the same general rules are utilized, it has been included in every Paralympic Games since they started in 1960, and Tokyo 2020 will be the first time there will be full gender parity [45]. However, there are important differences, particularly the facts that athletes compete while being seated and in classes based on the level of functional ability. Athletes are eligible to be classified to compete if they have any of the following: leg length differences, limb deficiency, ataxia, hypertonia, athetosis, impaired muscle power, or impaired passive range of motion. IWAS has three classifications for wheelchair fencing: A, B, & C (although only A & B classes are included in the Paralympic program) [46]. In general, category A athletes have good arm and trunk control (e.g., a limb deficiency or spinal cord injury T10-L2), while category B fencers have a qualifying functional impairment of the fencing arm or trunk control (e.g., spinal cord injury T1–T9; cerebral palsy). Category C athletes have minimal arm control and no sitting balance (e.g., spinal cord injury C5–C8) [46]. Wheelchair fencing is unique among dual wheelchair sports (e.g., tennis, badminton, table tennis) in that the athletes cannot move their chairs, which are locked into a frame to maintain a fixed distance between the competitors (Fig. 19.7).

Despite a 60-year history of expanding opportunity and growing participation in wheelchair fencing, epidemiological research of the sport is even more sparse than for able-bodied fencing. This may be partly due to the still relatively small number of wheelchair fencers overall, lack of urgency because of anecdotal evidence of low injury risk, and/or the priorities of disability sport researchers to build an understanding of disability sports in general or to focus on those with greater participation or risk profiles [47]. For example, Nyland and colleagues [48] analyzed soft tissue injuries in the USA team at the 1996 Paralympic Games and listed study participants (i.e., team members) by sport. However, the researchers provided no breakout data for any specific sport but rather compared results across the seven national disability sports organizations responsible for different types of athletes (e.g., wheelchair, visual impairment, deaf, dwarf, cerebral palsy, and intellectual disability). Similarly, Gawrónski et al. [49] examined injuries and illness in the Polish team across the Paralympic Games in Beijing and London and noted that fencers comprised 10% of the athletes from the 12 sports represented in the two teams but provided no breakout data. In this instance, the researchers reported data by disability category (i.e., amputee, spinal cord injury, les autres, cerebral palsy, visual impairment, and intellectual disability).

In a report on medical issues with the British team at the 1992 Paralympic Games in Barcelona, Reynolds et al. [50] noted that 71% (5/7) of the team's wheelchair fencers sought medical care during training and competition, but no details on type, location, or severity of their injuries were provided. An estimation of the "relative dangerousness" of wheelchair fencing may be gleaned from the fact that this percentage was similar to that for table tennis and swimming (69% each; 9/13 and 30/43, respectively). However, more recent data seem to challenge that perspective.

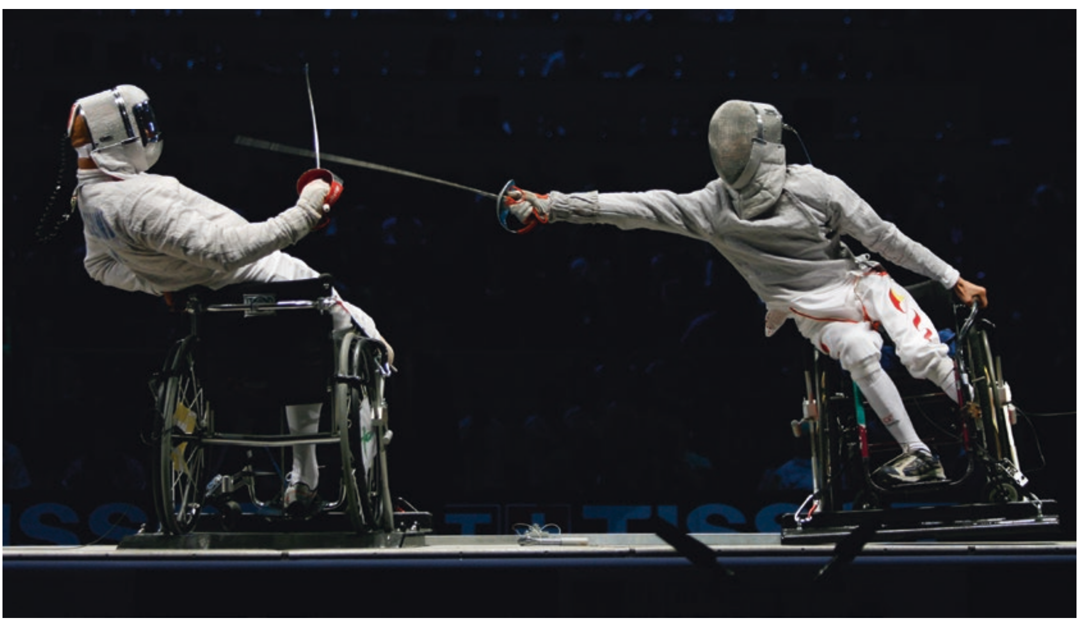

Fig. 19.7 Wheelchairs for fencing are fixed in place requiring vigorous arm and trunk action to change distance in attack and defense. Photo credit: Serge Timacheff. Used with permission

The first study to include exposure data for all participants at a Paralympic Games (London 2012) reported that wheelchair fencing had an injury incidence rate of 18/1000 athlete days [95% CI 11.6–26.7], the fourth highest among the 21 sports covered [51]. However, the criterion for a reportable injury was any musculoskeletal or neurological problem for which medical care was sought, without attention to the severity of the complaint. As with injury research with able-bodied fencers, this standard tends to overestimate risk in the meaningful sense, that is, severe enough to interfere with training or competing as opposed to minor physical injury, such as contusions, abrasions, or non-debilitating sprains and strains, which are a common consequence of intense physical activity. Interestingly, wheelchair fencing was also found to have the second highest proportion of chronic injuries (58%) in the sample, which could account for the high injury incidence rate. Daily treatment for ongoing problems, such as tendinitis or impingement syndrome, which do not necessarily prevent athletes from training or competing would inflate the injury risk data. By contrast, fencing had the fourth lowest percentage of acute injuries (42%), exceeding only powerlifting (14%), wheelchair archery (33%), and wheelchair tennis (37%). Additional research using the same definition of a reportable injury was conducted at the 2016 Paralympic Games in Rio de Janeiro [52] with wheelchair fencing accounting for the second highest injury rate (15.9/1000 athlete days; 95%CI 9.7–25.9) of the 22 sports contested, but it was not in top group for time-loss injuries, indicating that while fencers may have to frequently seek medical care it is not generally for significant problems. Unfortunately, no additional breakout information by sport that might be used to develop effective prevention strategies is provided.

To date, the most complete research on injury risk in wheelchair fencing is the pilot study by Chung and colleagues [10] who tracked 14 A & B class wheelchair foil fencers on the Hong Kong national team over 3 years. Although the study has some limitations, specifically the small sample size and the potential of recall bias, these are well balanced by the incorporation of a strong exposure metric (injuries per 1000 h of exposure), data from training and competition, a reportable injury criterion that incorporates time loss and, consequently, the ability to categorize injuries as minor (>1 day ≤7 days away from training), moderate (8–21 days) or major (≥22 days), and comparisons across classification categories. Unsurprisingly, all of the reported injuries were above the waist (74% in the upper extremities; 26% involving the trunk and head); strains (59%) and sprains (28%) were the most prevalent injuries types, with strains of the elbow/forearm the most common specific problem, accounting for approximately 33% (31/95) of all of their reported injuries. Consistent with the general sports epidemiology literature, training accounted for more injuries than competition (83 vs. 12), but there was a statistically significant 35% greater risk of being injured in competition. Similarly, Category B fencers had a 64% greater risk of sustaining a reportable injury than their Category A teammates, although Category A athletes had almost a five times greater risk of sustaining a shoulder injury (RR = 4.97; 95%CI 1.8–16.9). Finally, in contrast to the findings from the 2016 Paralympic Games [51], only 15% of the injuries were classified as chronic, possibly due to the ongoing care provided by the clinicians working with the national team.

It is clear that the current knowledge base of the epidemiology of wheelchair fencing is inadequate. There is broad consensus among researchers that, as with epidemiological research on able-bodied fencers, future projects must move beyond single event/short-term studies to large-scale longitudinal work with consistent methodological parameters to allow meaningful comparisons across projects and, ultimately, develop effective injury prevention and performance enhancement strategies [10, 47, 51, 53, 54]. Within this overarching framework, which may be coordinated by a global entity such as the International Paralympic Committee, it is important that a sport-specific focus is emphasized, and parallel work by individual international sports federations is undertaken as injury risk is sport-specific [51, 53]. Additionally, classification systems and modifiable and non-modifiable risk factors vary across sports, and the risk profiles of these differences can only be explored within the context of each specific sport [47]. Finally, Sobiecka et al. [54] argue that medical care for all athletes with disabilities, not just those in elite competition, needs to extend beyond their involvement with sport. As their functional limitations are part of their everyday life, they need regular medical screening and care by sports medicine professionals to minimize the potential for injury.

Conclusion
Many people not familiar with fencing would be surprised to know that at the 2008 Olympic Games, only canoe/kayak, rowing, sailing, diving, and synchronized swimming had a lower prevalence of time-loss injuries than that of the 0.8% of fencers [55] or that soccer and basketball players had 20–50 greater risk of a time-loss injury than fencers [26]. The low risk, especially of fencing-specific injuries, is primarily due to the imposition of regulations dictating equipment standards by the international governing body. The singular nature of the sport is such that these changes could only be implemented at this organizational level. The traditional measures for decreasing injury risk through individual athlete action, such as strength and conditioning programs and nutritional protocols, are only tangentially germane. Although it is prudent for fencers to continue to utilize these approaches to enhance performance, their relevance to injury prevention is empirically unresolved.

Take-Home Message
Despite appearances, modern competitive fencing is very safe. Fencing-specific injuries are rare, and the characteristics of typical time-loss injuries (type, location) are similar to those in other dual sports involving ballistic action and a high degree of agility.

References

1. Wikipedia: https://en.wikipedia.org/wiki/Fencing_at_the_Summer_Olympics
2. Ottogalli C, Six G, Terret T. The history of fencing. 1st ed. Biarritz, FRA: Atlantica; 2013.
3. FIE: https://fie.org/fie/structure/federations-map
4. Harmer P. Epidemiology of time-loss injuries in international fencing: a prospective, 5-year analysis of Fédération Internationale d'Escrime competitions. Br J Sports Med. 2019;53:442–8. https://doi.org/10.1136/bjsports-2018-100002.
5. Hayashi D, Jarraya M, Engebretsen, Crema M, Roemer F, Skaf A, Guermazi A. Epidemiology of imaging-detected bone stress injuries in athletes participating in the Rio de Janeiro 2016 summer Olympics. Br J Sports Med. 2018;52:470–4. https://doi.org/10.1136/bjsports-2-17-098189.
6. Sinclair J, Bottoms L, Taylor K, Greenhalgh A. Tibial shock during the fencing lunge: the influence of footwear. Sports Biomech. 2010;9(2):65–71. https://doi.org/10.1080/14763141.2010.491161.
7. Greenhalgh A, Bottoms L, Sinclair J. Influence of surface on impact shock experienced during a fencing lunge. J Appl Biomech. 2013;29:463–7. https://doi.org/10.1123/jab.29.4.463.
8. Zemper E, Harmer P. Fencing. In: Caine D, Caine C, Lindner K, editors. Epidemiology of sports injuries. Champaign, IL: Human Kinetics; 1996. p. 186–95.
9. Harmer P. Fencing. In: Caine D, Harmer P, Schiff M, editors. Epidemiology of injury in Olympic sports. West Sussex, UK: IOC Medical Commission; 2009. p. 124–32.
10. Chung WM, Yeung S, Wong AUL, Lam IF, Tse PTF, Dasawni D, Lee R. Musculoskeletal injuries in able-bodied and wheelchair fencers – a pilot study. Clin J Sports Med. 2012;23(3):278–80. https://doi.org/10.1097/jsm.0b013e31824a577e.
11. Gray W, Bassett F. Osteochondritis dissecans of the patella in a competitive fencer. A case report. Orthop Rev. 1990;19(1):96–8.
12. Borelli L, Ciniglio M, Maffulli N, Del Torto M. Intravascular papillary endothelial hyperplasia in the hand of a fencer. Pathologica. 1992;84:551–6.
13. Kelm J, Anagnostakos K, Deubel G, Schließing P, Schmitt E. The rupture of the tibial anterior tendon in a world class veteran fencer [Ruptur der sehne des musculus tibialis anterior bei einem seniorenfechter der weltklasse]. Sportverletzung-Sportschaden. 2004;18(3):148–52.
14. Lefevre N, Naoury J-F, Vincent D, Ribette P, Herman S. Rupture of the ischial origin of the hamstring muscles among athletes: a report of 9 cases of surgical repair [rupture haute des tendons ischiojambiers chez le sportif. À propos d'une série de neuf cas opérés]. Journal de Traumatologie du Sport. 2007;24(3):143–7.
15. Stepień K, Śmigielski R. 28-year-old woman training fencing with isolated rupture of radial collateral liga-

ment of the metacarpophalangeal joint of the index finger. Sports Ortho Traumatol. 2015;31(3):228–32. https://doi.org/10.1016/j.orthtr.2015.06.009.
16. Harmer P, Moriarty J, Walsh M, Bean M, Cramer J. Distant entry pneumothorax in a competitive fencer. Br J Sports Med. 1996;30:265–6. https://doi.org/10.1136/bjsm.30.3.265.
17. Campbell JP, Brownlee RE, Weissler MC. Translaryngeal puncture in a collegiate fencer. Otolaryngol Head Neck Surg. 1997;116:120–2. https://doi.org/10.1016/S0194-5998(97)70363-2.
18. Wild A, Jaeger M, Poehl C, Werner A, Raab P, Krauspe R. Morbidity profile of high-performance fencers. Sportverl Sportschad. 2001;15(3):59–61. https://doi.org/10.1055/s-2001-17277.
19. Harmer P Fencing fatalities. Unpublished data 2020
20. Lanese RR, Strauss RH, Leizman DJ, Rotondi A. Injury and disability in matched men's and women's intercollegiate sports. Am J Pub Health. 1990;80(12):1459–62. https://doi.org/10.2105/AJPH.80.12.1459.
21. Majorano M, Cesario S. Lesioni traumatiche acute nella pratica sportive agnostica della scherma [in Italian]. Med Del Sport. 1991;44(3):281–9.
22. Gambaretti R, Berlusconi M, Lanzani A. La traumatologia da scherma: Studio della patologia tipica della disciplina in rapporto all'evoluzione del gesto atletico [in Italian]. J Sports Traumatol Rel Research. 1992;14(2):139–41.
23. Naghavi SH. A study of traumatic events in junior fencing competitions. Escrime. 2000;34(12):12–3.
24. Harmer P. Rate and characteristics of time-loss injuries do not differ across age groups in competitive fencing. Med Sci Sports Exercise. 2008;39(5):S234. (abstract)
25. Harmer P. Minimal risk of time-loss injury in Masters fencing: USA National and World Championship data 2002–2006. Med Sci Sports Exercise. 2007;9(5):S412. (abstract)
26. Harmer P. Incidence and characteristics of time-loss injuries in competitive fencing: a prospective, 5-year study of national competitions. Clin J Sports Med. 2008;18(2):137–42. https://doi.org/10.1097/JSM.0b013e318161548d.
27. Park KJ, Byung SB. Injuries in elite Korean fencers: an epidemiological study. Br J Sports Med. 2017;51:220–5. https://doi.org/10.1136/bjsports-2016-096754.
28. Walrod B, Turner W, Hartz C. A prospective cohort of collegiate fencing injuries. Curr Sports Med Rep. 2019;18(10):361–6. https://doi.org/10.1249/JSR.0000000000000637.
29. Roi GS, Fasci A. Indagine sulle richieste di intervento del medico durante le gaare di scherma [survey of requests for medical assistance during fencing matches]. Ital J Sports Trauma. 1988;10(1):55–62.
30. Moyer JA, Jaffe R, Adrian M. Fencing. In: Fu FH, Stone DA, editors. Sports injuries – mechanisms, prevention, treatment. Baltimore: Williams & Wilkins; 1994. p. 333–48.
31. Müller-Strum AE, Fechtsportunfälle BK. Epidemiologie und prävention [in German]. Deutsche Zeitschrift fur Sportmedizin. 1991;42(2):48–52.
32. Engebretsen L, Soligard T, Steffen K, Alonsso JM, Aubry M, Budgett R, Dvorak J, Jegathesan M, Meeuwisse W, Mountjoy M, Palmer-Green D, Vanhagen I, Renström P. Sports injuries and illnesses during the London summer Olympic games 2012. Br J Sports Med. 2013;47:407–14. https://doi.org/10.1136/bjsports-2013-092380.
33. Soligard T, Steffen K, Palmer D, Alonso JM, Bahr R, Dias Lopes A, Dvorak J, Grant M-E, Meeuwisse W, Mountjoy M, Pena Costa LO, Salmina N, Budgett R, Engebretsen L. Sports injury and illness incidence in the Rio de Janeiro 2016 Olympic summer games: a prospective study of 11274 athletes from 207 countries. Br J Sports Med. 2017;51:1265–71. https://doi.org/10.1136/bjsports-2017-097956.
34. Turner A, James N, Dimitriou L, Greenhalgh A, Moody J, Fulcher D, Mias E, Kilduff L. Determinants of Olympic fencing performance and implications for strength and conditioning training. J Strength Cond Res. 2014;28(10):3001–10. https://doi.org/10.1519/jsc.0000000000000478.
35. Harmer P. Getting to the point: injury patterns and medical care in competitive fencing. Curr Sports Med Reports. 2008;7(5):303–7. https://doi.org/10.1249/jsr.0b013e318187083b.
36. Brazilian fencer stabbed by brother in training for Pan Ams. 2007 Associated press, July 10.
37. Parfitt R. The fencer at risk. In: Armstrong J, Tucker W, editors. Injuries in sports. London: Staples; 1964. p. 173–90.
38. Terret T, Ottogalli-Mazzacavallo C. L'effet Smirnov en question? Perception et impact des accidents fatals en escrime [in French]. Int Rev Sport Violence. 2011;4:1–10.
39. Safra J-M. La securite en question aprés l'accident de Vladimir Smirnov [in French]. Escrime. 1982;42:26–8.
40. Harmer P. Special report on sabre injuries - FIE executive committee meeting. Feb. 2011;26
41. FIE: https://fie.org/fie/documents/rules (Material Rules – Book 3, Dec. 2019).
42. Harmer P. Preventing penetrating hand injuries in sabre fencing: an application and critique of the van Mechelen model by the Fédération Internationale d'Escrime. Br J Sports Med. 2015;49:1138–43. https://doi.org/10.1136/bjsports-2015-094823.
43. FIE: https://fie.org/fie/documents/rules (Technical Rules – Book 1, Dec. 2019).
44. FIE: https://fie.org/fie/documents/handbooks (Medical Handbook of Specifications, September 2014).
45. IWASF https://wheelchairfencing.iwasf.com/about/history/ 2020.
46. IWASF https://wheelchairfencing.iwasf.com/about/classification/ 2020.
47. Fairbairn JR, Huxel Bliven KC. Incidence of shoulder injury in elite wheelchair athletes differ between sports: a critically appraised topic. J Sports

Rehabil. 2019;28:294–8. https://doi.org/10.1123/jssr.2017-0360.
48. Nyland J, Snouse S, Anderson M, Kelly T, Sterling J. Soft tissue injuries to USA Paralympians at the 1996 summer games. Arch Phys Med Rehabil. 2000;81:368–73. https://doi.org/10.1016/S0003-9993(00)90086-8.
49. Gawrónski W, Sobiecka J, Malesza J. Fit and healthy Paralympians – medical care guidelines for disabled athletes: a study of the illnesses and injuries incurred by the polish Paralympic team in Beijing 2008 and London 2012. Br J Sports Med. 2013;47:844–9. https://doi.org/10.1136/bjsports-2013-092298.
50. Reynolds J, Stirk A, Thomas A, Geary F. Paralympics – Barcelona 1992. Br J Sports Med. 1994;28(1):14–7. https://doi.org/10.1136/bjsm.28.1.14.
51. Willick S, Webborn N, Emery C, Blauwet C, Pit-Grosheide P, Stomphorst J, Van de Vliet P, Patino Marques NA, Martinez-Ferrer JO, Jordaan E, Derman W, Schwellnus M. The epidemiology of injuries at the London 2012 Paralympic games. Br J Sports Med. 2013;47:426–32. https://doi.org/10.1136/bjsports-2013-092374.
52. Derman W, Runciman P, Schwellnus M, Jordaan E, Blauwet C, Webborn N, Lexell J, Van de Vliet P, Tuakli-Wosornu Y, Kissick J, Stomphorst J. High precomeptition injury rate dominates the injury profile at the Rio 2016 summer Paralympic games: a prospective cohort study of 51,198 athlete days. Br J Sports Med. 2018;52:24–31. https://doi.org/10.1136/bjsports-2017-098039.
53. Webborn N, Emery C. Descriptive epidemiology of Paralympic sports injuries. PM&R. 2014;6:S18–22. https://doi.org/10.1016/j.pmrj.2014.06.003.
54. Sobiecka J, Plinta R, Kadziolka M, Gawrónski W, Kruszelnicki P, Zwierzchowska A. Polish Paralympic sports in the opinion of athletes and coaches in retrospective studies. Int J Environ Res Public Health. 2019;16:4927. https://doi.org/10.3390/ijerph16244927.
55. Junge A, Enegbretsen L, Mountjoy M, Alonso JM, Renström P, Aubry M, Dvorak J. Sports injuries during the summer Olympic games 2008. Am J Sports Med. 2009;37(11):2165–71. https://doi.org/10.1177/0363546509339357.

20 Field Sports Throwing Injuries

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20.1 Introduction

Throwing events in track and field are some of the earliest recorded sporting events in history. Discus and javelin were included in the Ancient Olympic Games dating back to ~708 B.C [1]. Fast forwarding to modern times, the first Olympic Games in 1896 featured shot put and discus. Hammer throw was introduced shortly afterward in 1900, followed by javelin in 1908.

In today's high school climate, track and field comprises the most athletic participants for girls' sports and the second most in boys' sports behind football [2]. Despite its popularity, the literature on injuries in throwing events is relatively sparse. Most of the literature is specific to competitive events, which occur less frequently than in most other popular sports, such as basketball, baseball, or football. However, track and field injuries happen more often in practice than in competition [3]. This chapter attempts to synthesize the body of knowledge related to common injuries sustained by field throwing athletes.

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20.1.1 Throwing Events: The Basics

When attempting to understand the injuries common to the different field throwing events, it is first important to understand the basics of each of the throwing event.

Shot Put: The shot put involves throwing a metal ball (7.26 kg for men and 4 kg for women) while staying within a throwing circle spanning 2.135 m. The goal is to bring the throwing arm and shot put from a stand-still to maximum velocity at release, with an optimal release angle of 31–26° [4, 5] Fig. 20.1.

Javelin: The javelin throw occurs on a runway measuring 4 feet wide and 30+ feet long, which ends in a curved arc from where the throw is measured. The javelin itself measures 2.6–2.7 m for men with a mass of 800 g, versus 2.2–2.3 m for women with a mass of 600g [6] Fig. 20.2.

Discus: The discus measures 22.0 cm for men and 18.1 cm for women, with masses of 2 and 1 kg, respectively. The throwing circle is 2.5 m, and the optimal release angle is 35–40° [7] Fig. 20.3.

Hammer: Lastly, the hammer throw is unique within the throwing sports in that it involves a centrifugal force from a wire rather than from the thrower's hand. For men, a 7.3 kg metal ball is attached to a handle by a 4-foot steel wire. For women, a 4 kg ball is attached to a wire measuring 3 feet, 11 inches. Farther hammer throwing

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Fig. 20.1 Shot Put (female): Photos courtesy of Linh Vuong

Fig. 20.2 Javelin: Photo courtesy of Linh Vuong

distances are associated with a higher center of gravity and height of the hammer at release [8] Fig. 20.4.

20.2 Unique Mechanics and Injury Risks

Proficiency in all of the throwing events involves a unique combination of a large number of repetitions and the generation of maximum force over a short period. As a result, injuries stemming from throwing include not only repetitive stress injuries but also more explosive acute injuries. As with most throwing sports, the common theme of these sports is generation of power from the lower extremities and subsequent transfer of energy to the upper extremity for release. The throwing motion involves activation and coordination of multiple muscle groups in a complex sequence to efficiently transfer energy and achieve maximum power and accuracy.

Fig. 20.3 Discus: Photo courtesy of Linh Vuong

Fact Box 1 Throwing Mechanics Involve a Complex Sequence of Power Generation from the Lower Extremities, Followed by Transfer of Energy to the Upper Extremity for High-Velocity Release

Shot put: There are two primary shot put techniques: the glide technique and the rotational technique [7]. The glide technique involves driving with one leg to "glide" from the back to the front of the throwing circle. During this process, the thrower is briefly airborne as the lower body is generating force and the upper body is generating torque. The front leg then collides with the toe board at the front of the throwing circle, initiating the throwing motion. During the throw, both legs extend and lift off the ground, while the elbow extends with shoulder remaining adducted to release the shot. This is in contrast to the rotational technique, which generates higher rotational energy but requires more coordinated footwork. One leg is planted while the other is airborne and engaging in a long sweeping rotational movement. This technique stretches the core muscles and creates potential energy, which is subsequently converted to kinetic energy with the release of the shot put [5, 9]. According to one EMG study, there was a positive correlation between peak performance and the activity of vastus lateralis and pectoralis major during delivery, as well as decreased time to maximal triceps activation during the throw itself [10].

Javelin: Javelin technique begins with a run-up that generates momentum. Toward the end of the run-up, the athlete completes a series of sideways crossover steps with the throwing arm above shoulder height. Before the throw, the dominant leg lands more softly in a bent position to allow the thrower's body weight and momentum from the run to continue without significant slowing. The throwing arm remains behind and straightened while the nondominant arm and leg extend toward the target for balance. The nondominant side of the body acts as a fulcrum, which anchors and allows acceleration of the dominant side into the release of the javelin. The energy travels from the lower to the upper extremities, starting with the hips rotating forward, followed by the chest, and finally followed by the throwing arm, which completes the throwing motion much like a baseball pitcher or tennis serve. There is arm abduction and external rotation in late cocking phase, which is associated with anterior stress in the glenohumeral joint [11]. The anterior stress on the shoulder seen in maximal external rotation is resisted by static and dynamic stabilizers including the labrum, glenohumeral ligaments, anterior joint capsule, rotator cuff muscles, and tendon of the long head of the biceps. This is followed by acceleration and follow-through with elbow extension and shoulder internal rotation. This last stage of throwing generates the largest joint forces due to eccentric contraction required to decelerate the arm [12].

Discus: The athlete grips the discus with their palm and fingers, throwing it after rotating several times while traversing the throwing circle spanning 2.5 m. For the majority of the motion the arms are extended and the trunk is rotated to maximize potential energy, which supplements the body's rotational energy. Both legs support the body in the beginning of the motion, which starts with a backswing to extend the arms and engage the core. Each leg then supports the body during the rotational portion of the motion, while the arms remain extended and trunk rotated. Immediately prior to the release, the nondominant foot plants while the body is perpendicular to the throwing direction. On the release, energy is transferred cranially, from the pelvis, to the trunk, to the chest, and finally through the arm.

Hammer Throw: Hammer throw technique involves preliminary arm swings to start the momentum of the hammer, followed by a series of body rotations, and finally the release. During rotations, the wire is in full extension to maximize angular acceleration, which creates the energy necessary to maximize distance of the throw. During the rotations, the nondominant leg is planted and bears most of the body weight, while the dominant leg swings and lands every 360°. Coordinating the legs during these rotations aids in balance as well as acceleration. Ultimately, the final turn produces the most momentum going into the release [13].

Fig. 20.4 Hammer Throw: Photo courtesy of Alisha Lovrich

20.3 Common Injuries in Track and Field Throwing Athletes

The unique biomechanics of these track and field throwing events render athletes susceptible to certain specific injuries detailed below.

20.3.1 Shoulder

Most injuries experienced by track and field throwers involve the upper extremity, and the majority of these are in the shoulder [14]. Overhead and throwing athletes subject their shoulders to a unique set of repetitive forces. This results in adaptive changes around the glenohumeral joint, which may become pathologic over time related to overuse and faulty mechanics. The result in many cases is an overhead athlete with shoulder pain.

To create velocity whether throwing a javelin, shot put, or baseball, the throwing athlete harnesses ground reactive forces which are then channeled through the lower body and core and finally into the arm. Efficiency of energy transfer can result in significant velocity of the thrown object. However, there is also significant repetitive stress imparted to the shoulder and elbow. Angular velocity of shoulder internal rotation can be as high as 6000°/s during acceleration [15]. These high forces and torques place considerable stress on the glenohumeral joint, rotator cuff, labrum, and muscles of the shoulder girdle. The successful overhead athlete strikes a balance between the increased external rotation and relative anterior shoulder laxity needed to produce high torques and the action of the rotator cuff and labrum necessary to produce stability and glenohumeral centration.

The shoulder structures adapt to repetitive overhead throwing. For example, many throwing athletes have a notable increase in retroversion of the humerus and may show glenoid retroversion as well [16]. Overhead athletes also generally will have increased external rotation in an abducted position [16]. In asymptomatic throwers, partial-thickness rotator cuff tears and labral tears are also commonly seen but are not necessarily well correlated with decrements in performance [17].

The internal impingement seen in throwing athletes occurs when the shoulder becomes unbalanced [18]. This allows the humeral head to ride posterior and superior and impinge upon the superior labrum and rotator cuff. This may result in damage to these structures as well as thickening and tightness of the posterior capsule [19]. Clinically, this commonly presents as shoulder pain during the late cocking phase of throwing at maximum abduction-external rotation. Pain is most commonly posterior or posterolateral but may be anterior. Overhead athletes may also complain of performance decline or a loss of velocity or power. Athletes should be asked about their volume of throwing and any changes in mechanics or lower body conditioning.

On physical exam, it may be difficult to determine normal physiologic changes in the overhead athlete from pathologic changes. In particular, an increase in external rotation should be expected, but ideally, the total arc of motion should be maintained [18]. Evaluation of rotator cuff strength is also important as this may decline due to injury. Finally, an examination of the scapula is critical. The scapula is a necessary stable platform from which the shoulder functions. When there is scapular dyskinesia or weakness, the shoulder is often affected.

Radiographs are commonly obtained as first-line imaging and are generally normal. A Bennett lesion, or calcification of the posterior capsule, may be seen on axillary view [18]. Notching or cystic change in the posterior greater tuberosity may be noted in long-term overhead athletes. MRI or MRI arthrogram is the most common modality employed when evaluating the intra-articular structures of the shoulder. This allows a thorough examination of the rotator cuff and labrum, where pathology is most commonly seen. Comparison with previous imaging if available is generally helpful given a high prevalence of pathology in asymptomatic throwers. History and physical exam more often guide treatment than imaging in internal impingement.

The first line of treatment for internal impingement is rest from overhead activities and rehabilitation. Rehabilitation focuses on stretching of the posterior capsule, for example, with sleeper stretches. Therapy should also focus on scapular stabilization and rotator cuff strengthening. Rest from throwing and focus on strength and stabilization are often successful in returning overhead athletes to sport. While surgical care for internal impingement was previously common, the results were highly variable and in most cases equivalent to non-operative care [20, 21]. Therefore, currently, non-operative care is the mainstay of treatment. In severe cases that have failed significant conservative therapy, shoulder arthroscopy may be considered for labral treatment and rotator cuff debridement.

20.3.2 Elbow

Ulnar collateral ligament (UCL) injuries of the elbow are well recognized as one of the most significant injuries in throwing sports. These can occur in javelin throwers, baseball pitchers, and shot putters but are rare in the hammer and discus where a different rotational throwing action is employed. In 1974, Frank Jobe performed the first UCL reconstruction, and surgical treatment of UCL-related instability has continued to evolve since that time [22].

The UCL is the primary restraint to valgus stress in the elbow. It is made up of the anterior, posterior, and oblique band. The anterior band, and in particularly the anterior bundle of the anterior band, plays the most significant role in resisting valgus stress [23]. The ulnar collateral ligament originates on the medial epicondyle of the humerus and terminates at the sublime tubercle of the ulna.

A careful history is critical to proper diagnosis of an UCL injury. Athletes may have an acute injury where they feel or hear a pop. This is generally followed by medial elbow pain, loss of motion, and swelling. Athletes may also have a more chronic course where they slowly have increasing pain in the medial elbow associated with throwing and decreased performance. Pain is generally present in late cocking and acceleration.

Both shoulders and elbows should be examined when UCL injury is suspected. Faulty mechanics in other areas such as the lower body and hips may lead to increased stress at the elbow. Many healthy throwers also have a lack of full elbow extension, but this is generally more striking after injury. Patients may be tender at the medial epicondyle, distally along the UCL or at the sublime tubercle. Pain with application of valgus stress or a milking maneuver indicates injury to the UCL. Flexor-pronator injuries are also common both independently or concurrently in this population and may be diagnosed with resisted strength testing. Finally, posterior pain related to olecranon spurs and radiating pain due to ulnar neuritis are common.

X-rays and MRI may be indicated for patients with an acute injury or worsening chronic pain. X-rays may demonstrate an ossicle within the UCL, which may denote prior injury or an avulsion in acute injuries in younger throwers. X-rays may also show an olecranon spur (a sign of valgus extension overload) or in some long-term throwers, frank osteoarthritis [24]. MRI or MRI arthrogram may be obtained to further delineate damage to the ligament. Many throwers do not have a normal ligament at baseline due to years of increased stress in this area, making prior imaging very useful. Peri-ligamentous edema, frank ligament injury, or injury to the flexor-pronator may be seen. Injuries are often graded as mild (grade 1), moderate (grade 2), or severe/full-thickness tears (grade 3) [25]. Proximal injuries have a more likely chance of healing than distal tears, which have a high rate of failure with nonsurgical care [26].

Once the diagnosis of an UCL injury is made, treatment is generally dependent on degree of symptoms and degree of injury on MRI. In mild or grade 1 strains, the majority of patients will improve without operative treatment. Most athletes are placed on 4–6 weeks of rest and then start a gradual throwing program. During this time, a biomechanical evaluation can also be undertaken. In some low-grade sprains, platelet-rich plasma (PRP) may be considered. Although commonly used, the evidence for use of PRP in low-grade UCL sprains is variable with some publications indicating a high rate of success and others indicating no benefit [27–30]. As previously noted, high-grade injuries and distal injuries have a high rate of failure with nonsurgical care. In individuals with full-thickness tears or those who have failed nonsurgical care, surgical reconstruction is generally indicated. There are multiple techniques, but the most common are the figure-of-eight technique popularized by Jobe and the docking technique popularized by Altchek [30]. In most cases, return to play takes approximately 1 year after surgical reconstruction. Rates of return to play after reconstruction are best studied in the baseball pitcher population and are quite high, from 70 to 90% [31–33].

20.3.3 Wrist and Fingers

Javelin, discus, and shot put involve extension of wrist and long fingers during release. This risks hyperextension injury of long finger, or full or partial volar plate (VP) rupture at the proximal interphalangeal joint (PIP) with or without avulsion fracture.

The VP is a small fibrocartilaginous structure that forms the floor of the PIP, separating the joint space from the flexor tendon sheath. It has a ligamentous origin on the proximal phalanx and a capsular insertion onto the middle phalanx. Williams et al. summarized the four main functions of the volar plate: (1) provides crucial stability against hyperextension; (2) acts as a meniscus in the PIP joint; (3) forms part of the intracavity lining of the PIP joint; and (4) provides a gliding surface for the flexor tendon [34].

A thorough history should be obtained with focus on the mechanism of injury and an understanding of the requirements of the PIP joint during the specific sport of participation. Most PIP joint injuries are caused by hyperextension and axial loading resulting in impaction of the volar articular surface of the middle phalanx against the condyles of the proximal phalanx [35]. On examination, one should note the location of swelling, tenderness, and deformity. Stability of the joint is first assessed by having the patient actively flex and extend the finger, with full painless motion indicating functional stability. Care should be given in the assessment of the last 20° of extension where instability is most likely to occur [36]. Passive stability is assessed by applying stress to each collateral ligament in both extension and 30° of flexion [37]. Adjacent and contralateral fingers may be used to provide comparison.

Radiographs should always be obtained in the assessment of PIP joint injuries, and particular focus should be placed on the true lateral radiograph of the finger to assess for subluxation and the common volar lip fracture. Dynamic fluoroscopy may be considered in complex fractures to better characterize the fracture and intra-articular stability.

Treatment is guided by the (i) Eaton's and (ii) the Keifhaber-Stern classifications [38]. Conservative management remains the treatment of choice and is most appropriate for injuries that involve less than 30–40% PIPJ surface and are reducible in less than 30° flexion. In these instances, splinting with extension-blocking immobilization (PIPJ in 20° to 30° flexion) is advised and, as swelling subsides, mobilization with protective splinting may be initiated [39]. Splinting is not generally needed for more than 4 weeks, and early motion is critical to avoiding stiffness. Even in mild or moderate injuries, the joint may remain swollen for several months, which can impair athletic activity. Open injuries, severe fractures which involve more than 40–50% PIPJ surface, irreducible dislocations, or those which require more than 30° flexion to reduce the fragment require surgical treatment. Techniques include volar plate arthroplasty, screw fixation of the volar fragment, and/or k-wire to temporarily stabilize the PIP [39].

The wrist remains at risk for these athletes as well. For example, during shot put the wrist is forcefully extended as it prepares to project the weight of a heavy spherical ball. During release the wrist is then required to push the ball, requiring significant forces to be transmitted through and generated at the wrist. As a result, the wrist is at risk of both acute and chronic injuries, including flexor and extensor tendinitis, intersection syndrome, and TFCC injury.

20.3.4 Lumbar Spine

Track and field throwing athletes are susceptible to lumbar spine pathologies because of repetitive hyperextension and rotation as well as heavy loading. These pathologies include spondylolysis, spondylolisthesis, and herniated disks. Moreover, throwers are often larger and heavier relative to other track and field athletes, increasing susceptibility. Previous studies have validated throwing sports as having the highest prevalence of lumbar spine pathology. Javelin was associated with the most radiographic changes in the lumbar spine, while shot putters and discus throwers were found to have significantly more lumbar osteophytes [40, 41].

Lumbar spine injuries in the throwing in the track and field athlete may be either acute or chronic. Acute lumbar disc herniations may occur when a sudden increased force ruptures the annulus fibrosis of the intervertebral disc, such as during a throw or heavy weightlifting [42]. Athletes generally will experience significant low back pain, which can radiate down one or both legs. In severe cases, there may be a loss of strength or sensation or even a loss of bowel or bladder control, indicative of possible cauda equina syndrome, an orthopedic emergency. More commonly, there is sudden back pain, loss of motion, and a positive straight leg raise on exam. Other examination findings include a subtle loss of sensation or strength, or a change in deep tendon reflexes compared to the contralateral side. Imaging for acute back pain generally begins with X-rays. As noted above, athletes in the track and field throwing sports may have osteophytes or narrowed joint spaces in the lumbar spine indicative of chronic degenerative disease [40]. MRI is generally obtained when there is high suspicion of a disc herniation or other spinal pathology. While lumbar disc herniations can be seen on MRI, some athletes may have baseline pathology and acuity may be difficult to ascertain. In most cases, lumbar disc herniations can be treated conservatively with rest and physical therapy. Use of oral anti-inflammatories or epidural corticosteroid injection may also be considered [42]. In athletes with larger disc herniations, neurologic findings, or failure to improve with conservative care, surgical microdiscectomy may be considered [43].

Also common in the younger track and field throwing athlete is spondylolysis, a stress injury of the vertebral pars segment, or spondylolisthesis. Athletes generally present in subacute fashion with low back pain that may be midline, bilateral, or unilateral. Pain may radiate into the buttock but generally does not radiate into the legs. Spondylolysis is most commonly seen in the teenage years [44]. On examination, athletes will often have pain with lumbar extension. They may or may not be tender over the lumbar spine. Straight leg raise is less commonly positive, and similarly, neurologic signs are not frequent. Spondylolysis may be seen on oblique radiographs of the spine. It most commonly occurs at L5 and maybe unilateral or bilateral [45, 46]. If X-rays are negative and there is high suspicion, MRI or SPECT scan will often identify a subtle pars fracture or stress reaction [44]. Bilateral pars fractures can result in spondylolisthesis or slippage over time. This places the athlete at higher risk for non-healing and continued symptoms. When a unilateral partial or complete pars fracture is noted, most athletes do well with conservative care [47]. This involves rest from weightlifting or throwing. Bracing is commonly used, but evidence for bracing is slight [48]. Rehabilitation to strengthen the core and back musculature is often helpful. Athletes may return to play when they are asymptomatic, but return to sports-related activities should be gradual.

20.3.5 Lower Extremity

Lower extremity injuries are less common than upper extremity for throwing athletes. However, these injuries do occur and must be well understood by the treating providers. The most common lower extremity injuries involve the knee and ankle.

Knee injuries often involve improper mechanics, and thus proper coaching and technique is vital to prevent these injuries. Acute ACL, MCL, and meniscus tears can occur when the athlete lands with improper foot position, due to rotational forces and valgus stress.

Ankle injuries can also occur in throwing athletes. In fact, ankle injuries were the most common injury in a study involving British shot putters [49]. The toe board in front acts as a brake for the leg while the athlete is traveling at a high velocity. This can lead to rotational stresses on the ankle. Multiple repetitions of the shot putting motion lead to repetitive contact with the toe board, causing microtrauma and osteoarthritis in the ankle and midfoot [7]. Because of the chronicity of this issue, the treatment for this injury is often conservative and may occur years after the thrower's career has ended.

20.4 Epidemiology

Overall, injuries in track and field throwing athletes are not very common. They occur in 47 per 1000 males and 32 per 1000 females, and have a 1-year prevalence of 35%, which is lowest of all track and field events [50, 51]. Throwers are injured roughly five times less commonly than sprinters who run the 110-meter hurdles and nine times less than athletes participating in the steeplechase [52]. Retrospective studies have concluded that 62% of throwers have had some form of injury in the last year, 75% had an injury at some point during their career [14.52]. Roughly half of injuries result in no time away from the sport [50] Table 20.1.

Throwers are susceptible to injuries throughout the entire body, given the complex whole body involvement associated with the throwing techniques. In male throwers, the distribution of injuries includes 20% upper extremity, 20% trunk, and 16.4% knee injuries. Almost half (47.3%) of injuries involve muscles, while 21.8% involve ligaments. The most frequent mechanism is overuse (49.1%). Most injuries result in no time loss (49.1%), while a smaller subset (27.3%) result in 1–4 weeks away from sport. In female throwers, injury distribution involves the trunk most commonly (28.1%), followed by upper extremity (18.8%), and lower leg (15.6%). Again, muscles are most commonly involved (31.3%), followed by tendons and ligaments (18.8% each). For females, overuse (53.1%) and trauma (40.6%) are the most common mechanisms of injury, and over half (53.1%) of athletes experience no time loss. The vast majority of upper extremity injuries involve the shoulder (70%), followed by elbow (15%), wrist, and hand (7% each) [14, 50].

Table 20.1 Injuries in throwing sports as a percentage of all track and field injuries [3]

	Male	Female
All	5.9%	6.7%
Shot put	2.9%	3.6%
Discus	1.6%	1.6%
Javelin	1.3%	1.5%

Fact Box 2 Throwing Injuries Involve the Entire Body and Are Often Overuse Injuries Involving Ligaments or Tendons. Shoulder Injuries Are the most Common Upper Extremity Injury

Prevention

Prevention for throwing injuries emphasizes striking the delicate balance between limiting the number of repetitions to prevent overuse injuries while having enough repetitions to perfect the unique and demanding throwing techniques. Prior studies have shown that having a coach readily available during practice and competition is protective against injury, likely due to the ability to provide constant feedback on technique [49]. Admittedly, this can be challenging for high school athletes who may not have access to high-level technical instruction.

Another important aspect of injury prevention is proper surface maintenance. Ensuring optimal conditions and equipment is essential for minimizing preventable injuries. This includes keeping throwing surfaces flat and dry, as well as encouraging proper footwear.

A diverse, full body training regimen is also key to preventing injury. Although upper body injuries are most common, the lower body drives energy production in throwers and cannot be ignored. Prior studies have recommended programs that include appropriate strength and plyometric training via periodization and structure to achieve optimal performance with few injuries [53–55]. Training programs are often aimed at achieving specific goals at certain times throughout the competitive season. For instance, an in-season program involves a focus on general strength endurance, a competition phase to emphasize speed and power, as well as a brief taper before the most important competitive events [54]. Year-round regimens include a period of rest and rehabilitation in the offseason, with injury screening included [55]. In addition to strength and endurance, flexibility is a key component of a thrower's training program, due to the unusual positions involved in the throwing technique. Flexibility exercises include core rotation, hip flexors, hamstrings, and internal rotation of the shoulder, as well as low back stretching and strengthening to mitigate chronic low back pain.

Conclusion

Track and field throwing athletes are a relatively small population of athletes with unique mechanics, training requirements, and injury patterns. Significant strength and mass are needed to propel items as diverse as a hammer and javelin. Throwing in track and field shares some qualities with traditional throwers such as baseball pitchers and also may add a rotational element. Injuries in these athletes occur throughout the body, reflecting the importance of all body zones in generating and delivering energy to the arm and projectile. Upper extremity injuries related to soft tissue and overuse are most common. Appropriate technique, conditioning, and avoidance of overuse are key to keeping these unique athletes healthy and on the field.

References

1. Miller SG. Ancient greek athletics: Yale University Press; 2004.
2. The National Federation of State High School Associations. 2018–19 High School Athletics Participation Survey. 2019; https://www.nfhs.org/media/1020412/2018-19_participation_survey.pdf.
3. Pierpoint L, Williams C, Fields S, Comstock R. Epidemiology of injuries in United States high school track and field: 2008-2009 through 2013-2014. Am J Sports Med. 2016;44(6):1463–8.
4. Zatsiorsky VM, Lanka GE, Shalmanov AA. Biomechanical analysis of shot putting technique. Exerc Sport Sci Rev. 1981;9:353–89.
5. Young M, Li L. Determination of critical parameters among elite female shot putters. Sports Biomech. 2005;4(2):131–48.
6. World Athletics. Javelin Throw. 2020; https://www.worldathletics.org/disciplines/throws/javelin-throw. Accessed 20 May 2020, 2020.
7. Meron A, Saint-Phard D. Track and field throwing sports: injuries and prevention. Curr Sports Med Rep. 2017;16(6):391–6.
8. World Athletics. Hammer Throw. 2020; https://www.worldathletics.org/disciplines/throws/hammer-throw. Accessed 20 May 2020, 2020.
9. Kyriazis T, Terzis G, Boudolos K, Georgiadis G. Muscular power, neuromuscular activation, and performance in shot put athletes at preseason and at competition period. J Strength Cond Res. 2009;23(6):1773–9.
10. Terzis G, Karampatsos G, Georgiadis G. Neuromuscular control and performance in shot-put athletes. J Sports Med Phys Fitness. 2007;47(3):284–90.
11. Meister K. Injuries to the shoulder in the throwing athlete: part one: biomechanics/pathophysiology/classification of injury. Am J Sports Med. 2000;28(2):265–75.
12. Wolf BR, Wolstenholme K. Throwing injuries: biomechanics, injury mechanisms and rehabilitation. Current Orthopaedic Practice. 2010;21(5):467–71.
13. Gutierrez M, Soto VM, Rojas FJ. A biomechanical analysis of the individual techniques of the hammer throw finalists in the Seville athletics world championship, 1999. New studies in Athletics. 2002;17(2):15–28.
14. Edouard P, Depiesse F, Serra J-M. Throwing arm injuries in high-level athletics throwers. Science & Sports. 2010;25(6):318–22.
15. Scarborough DM, Bassett AJ, Mayer LW, Berkson EM. Kinematic sequence patterns in the overhead baseball pitch. Sports Biomech. 2018:1–18.
16. Tokish JM, Curtin MS, Kim YK, Hawkins RJ, Torry MR. Glenohumeral internal rotation deficit in the asymptomatic professional pitcher and its relationship to humeral retroversion. Journal of Sports Science & Medicine. 2008;7(1):78.

17. Del Grande F, Aro M, Farahani SJ, Cosgarea A, Wilckens J, Carrino JA. High-resolution 3-T magnetic resonance imaging of the shoulder in nonsymptomatic professional baseball pitcher draft picks. J Comput Assist Tomogr. 2016;40(1):118–25.
18. Corpus KT, Camp CL, Dines DM, Altchek DW, Dines JS. Evaluation and treatment of internal impingement of the shoulder in overhead athletes. World J Orthop. 2016;7(12):776.
19. Burkhart S, Morgan C, Kibler W. The disabled throwing shoulder: spectrum of pathology part I: pathoanatomy and biomechanics. Arthroscopy. 2003; 19(4):404–20.
20. Jobe C. Superior glenoid impingement. Orthop Clin North Am. 1997;28(2):137–43.
21. Andrews J, Dugas J. Diagnosis and treatment of shoulder injuries in the throwing athlete: the role of thermal-assisted capsular shrinkage. Instr Course Lect. 2001;50:17–21.
22. Jobe FW, Stark H, Lombardo SJ. Reconstruction of the ulnar collateral ligament in athletes. J Bone Joint Surg. 1986;68(8):1158–63.
23. Schwab G, Bennett J, Woods G, Tullos H. Biomechanics of elbow instability: the role of the medial collateral ligament. Clin Orthop Relat Res. 1980;146:42–52.
24. Matsuura T, Iwame T, Suzue N, et al. Clinical outcome of arthroscopic treatment for posteromedial elbow impingement in adolescent baseball players. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2018;34(1):105–10.
25. Kheterpal AB, Bredella MA. Overuse injuries of the elbow. Radiologic Clinics. 2019;57(5):931–42.
26. Ramkumar PN, Haeberle HS, Navarro SM, Frangiamore SJ, Farrow LD, Schickendantz MS. Clinical utility of an MRI-based classification system for operative versus nonoperative Management of Ulnar Collateral Ligament Tears: a 2-year follow-up study. Orthop J Sports Med. 2019;7(4):2325967119839785.
27. Chauhan A, McQueen P, Chalmers PN, et al. Nonoperative treatment of elbow ulnar collateral ligament injuries with and without platelet-rich plasma in professional baseball players: a comparative and matched cohort analysis. Am J Sports Med. 2019;47(13):3107–19.
28. McCrum CL, Costello J, Onishi K, Stewart C, Vyas D. Return to play after PRP and rehabilitation of 3 elite ice hockey players with ulnar collateral ligament injuries of the elbow. Orthop J Sports Med. 2018;6(8):2325967118790760.
29. Kato Y, Yamada S, Chavez J. Can platelet-rich plasma therapy save patients with ulnar collateral ligament tears from surgery? Regenerative therapy. 2019;10:123–6.
30. Hurwit DJ, Garcia GH, Liu J, Altchek DW, Romeo A, Dines J. Management of ulnar collateral ligament injury in throwing athletes: a survey of the American shoulder and elbow surgeons. J Shoulder Elb Surg. 2017;26(11):2023–8.
31. Cain EL, McGonigle O. Return to play following ulnar collateral ligament reconstruction. Clin Sports Med. 2016;35(4):577–95.
32. Marshall NE, Jildeh TR, Okoroha KR, et al. Performance, return to play, and career longevity after ulnar collateral ligament reconstruction in professional catchers. Arthroscopy: The Journal of Arthroscopic & Related Surgery. 2018;34(6):1809–15.
33. Saper M, Shung J, Pearce S, Bompadre V, Andrews JR. Outcomes and return to sport after ulnar collateral ligament reconstruction in adolescent baseball players. Orthop J Sports Med. 2018;6(4):2325967118769328.
34. Palmer R. Joint injuries of the hand in athletes. Clin Sports Med. 1998;17(3):513–31.
35. Williams EH, McCarthy E, Bickel KD. The histologic anatomy of the volar plate. J Hand Surg Am. 1998;23(5):805–10.
36. Eaton RG. Joint injuries of the hand. Clin Orthop Relat Res. 1972;84:280.
37. Minamikawa Y, Horii E, Amadio PC, Cooney WP, Linscheid RL, An KN. Stability and constraint of the proximal interphalangeal joint. J Hand Surg Am. 1993;18(2):198–204.
38. Kiefhaber TR, Stern PJ. Fracture dislocations of the proximal interphalangeal joint. J Hand Surg Am. 1998;23(3):368–80.
39. Pattni A, Jones M, Gujral S. Volar plate avulsion injury. Eplasty. 2016;16
40. Schmitt H, Dubljanin E, Schneider S, Schiltenwolf M. Radiographic changes in the lumbar spine in former elite athletes. Spine (Phila Pa 1976). 2004;29(22):2554–9.
41. Schmitt H, Brocai D, Carstens C. Long-term review of the lumbar spine in javelin throwers. J Bone Joint Surg Br. 2001;83(3):324–7.
42. Huang P, Anissipour A, McGee W, Lemak L. Return-to-play recommendations after cervical, thoracic, and lumbar spine injuries: a comprehensive review. Sports health. 2016;8(1):19–25.
43. Weinstein JN, Tosteson TD, Lurie JD, et al. Surgical vs nonoperative treatment for lumbar disk herniation: the spine patient outcomes research trial (SPORT): a randomized trial. JAMA. 2006;296(20): 2441–50.
44. Eddy D, Congeni J, Loud K. A review of spine injuries and return to play. Clin J Sport Med. 2005;15(6): 453–8.
45. Fredrickson B, Baker D, McHolick W, Yuan H, Lubicky J. The natural history of spondylolysis and spondylolisthesis. J Bone Joint Surg Am. 1984;66(5):699–707.
46. Ciullo J, Jackson D. Pars interarticularis stress reaction, spondylolysis, and spondylolisthesis in gymnasts. Clin Sports Med. 1985;4(1):95–110.
47. d'Hemecourt P, Zurakowski D, Kriemler S, Micheli L. Spondylolysis: returning the athlete to sports participation with brace treatment. Orthopedics. 2002;25(6):653–7.

48. Eck JC, Riley LH 3rd. Return to play after lumbar spine conditions and surgeries. Clin Sports Med. 2004;23(3):367–79.
49. D'Souza D. Track and field athletics injuries--a one-year survey. Br J Sports Med. 1994;28(3):197–202.
50. Edouard P, Navarro L, Branco P, Gremeaux V, Timpka T, Junge A. Injury frequency and characteristics (location, type, cause and severity) differed significantly among athletics ('track and field') disciplines during 14 international championships (2007-2018): implications for medical service planning. Br J Sports Med. 2020;54(3):159–67.
51. Jacobsson J, Timpka T, Kowalski J, Nilsson S, Ekberg J, Renstrom P. Prevalence of musculoskeletal injuries in Swedish elite track and field athletes. Am J Sports Med. 2012;40(1):163–9.
52. Opar D, Drezner J, Shield A, et al. Acute injuries in track and field athletes: a 3-year observational study at the Penn relays carnival with epidemiology and medical coverage implications. Am J Sports Med. 2015;43(4):816–22.
53. Turner A. The science and practice of periodization: a brief review. Strength and Conditioning Journal. 2011;33(1):34–46.
54. Judge L. Developing speed strength: in-season training program for the collegiate thrower. Strength and Conditioning Journal. 2007;29(5):42.
55. Zaremski JL, Zeppieri G Jr, Tripp BL. Sport specialization and overuse injuries in adolescent throwing athletes: a narrative review. J Athl Train. 2019;54(10):1030–9.

21 Golf: Injuries and Treatment

R. Zackary Unger, Zachary T. Skipper, and Mary Lloyd Ireland

21.1 Introduction

Golf is a sport that can be played by athletes of all ages, both males and females, and by various income earners. It continues to be one of the most popular sports worldwide, with Golf Digest reporting 25 million in the United States alone in 2015 and around 55 million worldwide. There have been various articles and book chapters looking at injuries in general and specific to golf. Most golf injuries are related to overuse syndromes, but can result from poor swing mechanics, poor core stability and strength, improper warm-up, and striking foreign objects. Injuries can be acute or chronic. When caring for golfers, it is important to understand the golf swing, swing biomechanics, and injuries, both general and specific to golfers.

21.2 The Golf Swing

When caring for golfers, it is important to consider the biomechanics of the golf swing just as in any sports injury. Several studies have assessed muscle activity in the forearm, shoulder, scapula, and trunk [1–7]. We will break down the swing into phases including the backswing, downswing, impact, and follow through as well as provide an overview of the muscles assisting with each phase.

21.2.1 Backswing

The backswing begins with the setup and continues throughout the takeaway to the top of the swing arc. With the shoulder turn during takeaway, rotational motion of the thoracic spine occurs in relation to a relatively stable lumbar spine and relatively stationary head position. Classic swings shift weight to the back foot (right for right-handed golfers); more modern swings are often more centered with weight and even some (i.e., Stack-and-Tilt) keep most of the weight on the front foot (left foot for right-handed golfers). As the athlete rotates the hips and torso for the backswing, the right elbow bends remaining tucked while the left elbow remains extended to the top of the backswing. As the golfer continues the backswing toward the top, they achieve a cocking position which results in radial deviation of the left wrist with the left thumb in hyperabduction and the right wrist is in an extended position. Skilled golfers store power during the backswing with a large shoulder turn while maintaining a more stable lower body position creating a coiling effect which can be released during the downswing generating power and increased distance (Figs. 21.1 and 21.2). Injuries can occur

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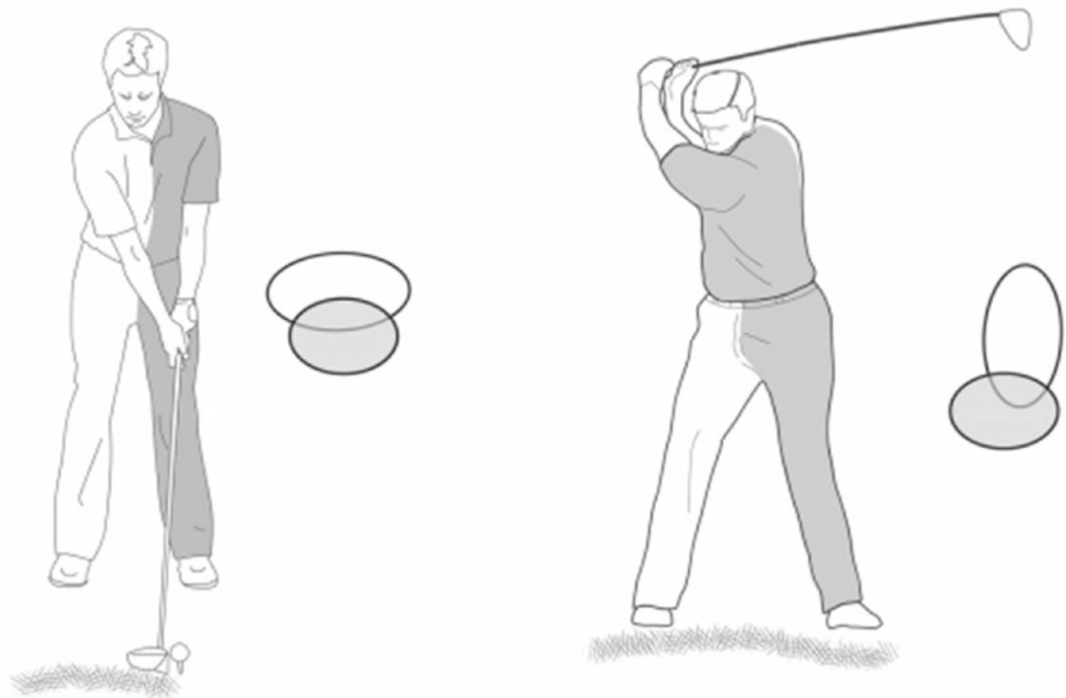

Fig. 21.1 The modern swing with large shoulder turn while rotating around a stable axis and lower body generating stored energy to be released when uncoiled during the downswing. (Courtesy of Thomas Dolan and Matt Hazzard)

in the backswing, but they are much less frequent than other phases of the swing and reportedly make of less than 25% of injuries [8].

Downswing:

Classically, the downswing begins from the ground up in a coordinated effort to release the stored energy created by the backswing. As weight is transferred toward the target, the hips and torso uncoil, and the shoulders rotate while the arms lower the club toward the ball. The elbows extend and the forearms rotate to square the clubface as it approaches the ball for impact.

21.2.2 Impact

Approaching impact trunk muscle activity remains very high. Weight is transferring to the front foot as the torso continues to rotate toward the target with ideally a clubface square to the target. The left (front) knee is in slight varus bracing for impact while the right (back) knee is in significant valgus as one pushes off the back foot. The left (front) wrist is now in a neutral position while the right wrist is near fully extended with the hands slightly ahead of the clubhead helping to compress the golf ball (Fig. 21.3).

21.2.3 Follow-through

After impact, the torso, shoulders, and forearms continue to rotate as continuation of energy released at impact. The left forearm supinates while the left elbow flexes; the right forearm pronates with the elbow remaining extended until folding in late follow-through. Hip rotation is complete, and the golfer moves almost full weight onto the left side. The knees rotate with the left knee achieving near full extension, while the right knee is flexed and pointing toward the target while coming up onto the right toe. Additionally, as the club decelerates some swing types result in a “reverse-C” position of the spine resulting in the majority of back injuries in the golf swing (Fig. 21.4) [8].

Fig. 21.2 Skilled modern golfer demonstrating large shoulder turn and relatively stable center of rotation and lower body. (Courtesy of Jeffrey Pierce)

21.2.4 Breakdown of Muscle Involvement in the Swing

21.2.4.1 Shoulders

During the backswing a coordinated effort of the rotator cuff and periscapular muscles is required. Studies have shown that during the backswing in a right-handed golfer, the right infraspinatus and supraspinatus fire most forcibly to externally rotate and abduct the right shoulder, while the left subscapularis fires to internally rotate the left shoulder [5]. Additionally, the trapezius acts to help retract the scapula of the right shoulder while all scapular muscles assist in a coordinated effort to help rotate around the trunk to maximize the swing arc adding the power and distance created by the swing [6].

21.2.4.2 Forearms

Forearm musculature firing was examined using EMG by Farber et al. [2] Extensor carpi radialis

Fig. 21.3 Skilled golfer approaching impact with hands slightly ahead of the squaring clubface. (Courtesy of Jeffrey Pierce)

brevis muscle activity peaks during the downswing through early follow-through in the front extremity (left for right-handed golfer). It peaked earlier in amateurs than professionals as evidence of premature uncocking of the wrists prior to impact in amateurs. Pronator teres activity was relatively low in the lead forearm, while its activity peaked in the trailing forearm during the downswing through follow-through. Professional golfers were shown to have increased pronator teres activity in their lead forearm and decreased activity in their trail arm compared with the amateurs as demonstrating pulling through impact with the lead arm versus pushing through with the trail arm seen in amateurs. Flexor carpi radialis muscle activity was higher in the lead forearm closer to impact in professionals, while activity in the trailing forearm peaked during the early downswing in both groups. Flexor carpi ulnaris muscle activity peaks in both forearms during the early downswing.

21.2.4.3 Trunk

Trunk muscle activity is relatively low on electromyography in professional golfers during the backswing [3]. However, during the downswing and acceleration all trunk muscles are active helping generate power for impact. The left gluteus muscles show high activity helping to stabilize the core and left hip as the right lower extremity pushes off. Early in the follow-through, most trunk muscle activity decreases, but the abdominal oblique muscles remain relatively active with the left remaining active through most of the forward swing and follow-through.

21.2.4.4 Hip and Knee

Bechler et al. looked at EMG activity in the hip and knee musculature [9]. Pelvic rotation is initiated during the downswing with left adductor magnus in coordination with the right hip extensors and abductors. The lead hamstrings help keep the left knee stable during pelvic rotation in the early downswing. Peak EMG activity occurs earlier than the trunk and shoulder muscles in more competitive golfers. Another study looking at hip rotational velocities has demonstrated that the lead hip experiences substantially higher internal rotation velocity which can result in injury [10].

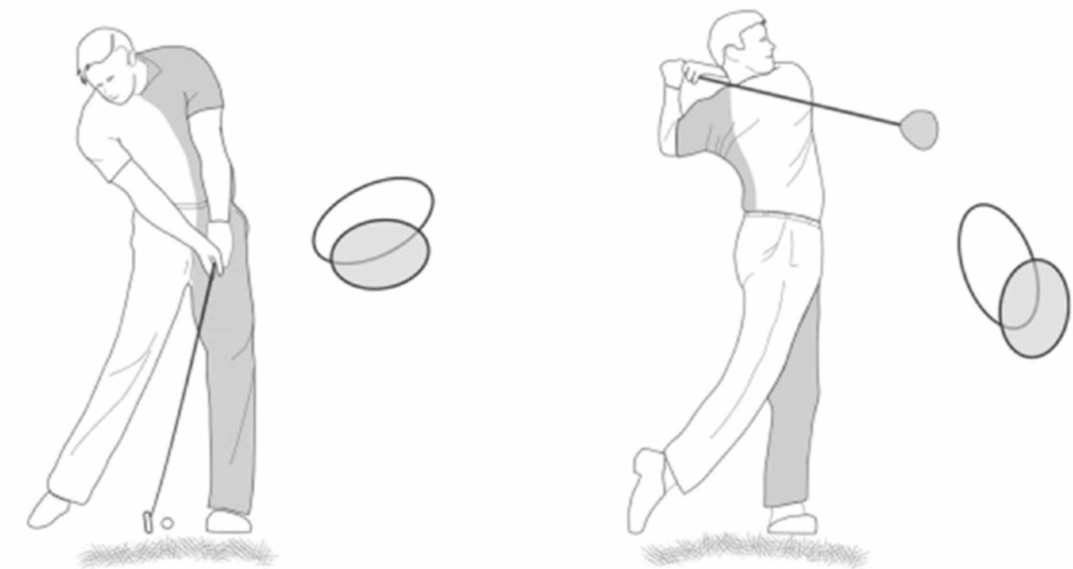

Fig. 21.4 The "reverse-C" created during the follow-through that can result in spine injury. (Courtesy of Thomas Dolan and Matt Hazzard)

21.3 Epidemiology of Injuries

There is a small volume of epidemiologic literature on golf injuries currently. In 2018, Zouzias et al. [1] published a literature review that compiled the most recent studies on golf-related injuries. Results from a 1-year prospective study performed by McHardy et al. [11] on 588 golfers show an overall incidence rate of 15.8 injuries per 100 golfers. They showed that 42.6% of injuries were sustained during the golf swing, and injury was most likely to occur at ball contact during the swing with a rate of 23.7%. Previous studies have shown that the most common sites for injuries include the low back, elbow, forearm, shoulder, and foot & ankle.

McCarroll [12] surveyed both professional and amateur golfers and out of those that responded, too much play or practice was the most common reported mechanism. In a different survey study performed by Batt [13], they found that of the 193 amateur golfers that responded stated that poor swing mechanics and overuse were the two most common causes of injury. Gosheger et al. [14] showed in a retrospective study of 703 golfers (643 amateurs and 60 professionals) that 82.6% of reported injuries were due to overuse and 17.4% were due to single trauma events. Professional golfers were injured more often overall and amateur golfers sustained more elbow injuries. It was also shown that golfers who carried their own golf bag had a higher risk of low back, shoulder, or ankle injury. Warming up for greater than 10 minutes had a positive effect for reducing injury rates. They also found that there were differences in swing biomechanics comparing amateur and professional golfers that affect the relative risk of injury. Amateur golfers attempted to generate more power and speed using their upper extremities which results in greater spinal torque and lateral bending forces on the lumbar spine. On the other hand, professional golfers' swings allow for greater trunk rotation and swing velocities consistently [1].

21.4 Low Back Injuries

The back is one of the most common injury locations for golfers. This is due to high compression, shear, lateral bending, and rotational loads that are created in the lumbar spine during the normal golf swing. It has been shown that compression loads up to approximately eight times the force equivalent of an athlete's body weight can be generated during the golf swing. Low back pain is occasionally due to a traumatic event, but it is usually a slow process that is due to a repetitive process referred to as cumulative load theory. The stress distribution to the lower back is asymmetric due to the difference in trunk velocity during the backswing compared to the forward swing and follow-through. Gluck et al. [15] found that lower back pain is more common on the trail side. Due to the strain from the golf swing on the lumbar spine, golfers have been found to be predisposed to muscle strains, herniated discs, stress fractures of the vertebral body and pars interarticularis, spondylolisthesis, and facet arthropathy [1].

When providing care for golfers with back pain, it is helpful to understand their swing style. It has been shown that the modern swing increases the torsional loads in the spine caused by focusing on a large shoulder turn with restricted hip turn by keeping the lead foot flat on the ground throughout the swing. This creates a large hip-shoulder separation angle increasing the load of the spine. In contrary, the classic golf swing focuses on reducing the hip-shoulder separation angle by raising the lead heel during the takeaway to increase hip turn which reduces the torque on the spine. These differences cause the modern swing golfer to experience increased lateral bending and exaggerated hyperextension of the back on follow-through, whereas in the classic swing finishes with the shoulder parallel to the ground, and this more erect posture is believed to prevent low back pain (Fig. 21.5) [1].

For the treatment of low back pain in golfers after the usual rest, ice, and NSAIDs the key is preventive exercises. After a golfer presents with their first episode of low back pain, initiation of stabilization exercises has been shown to reduce recurrence. It is

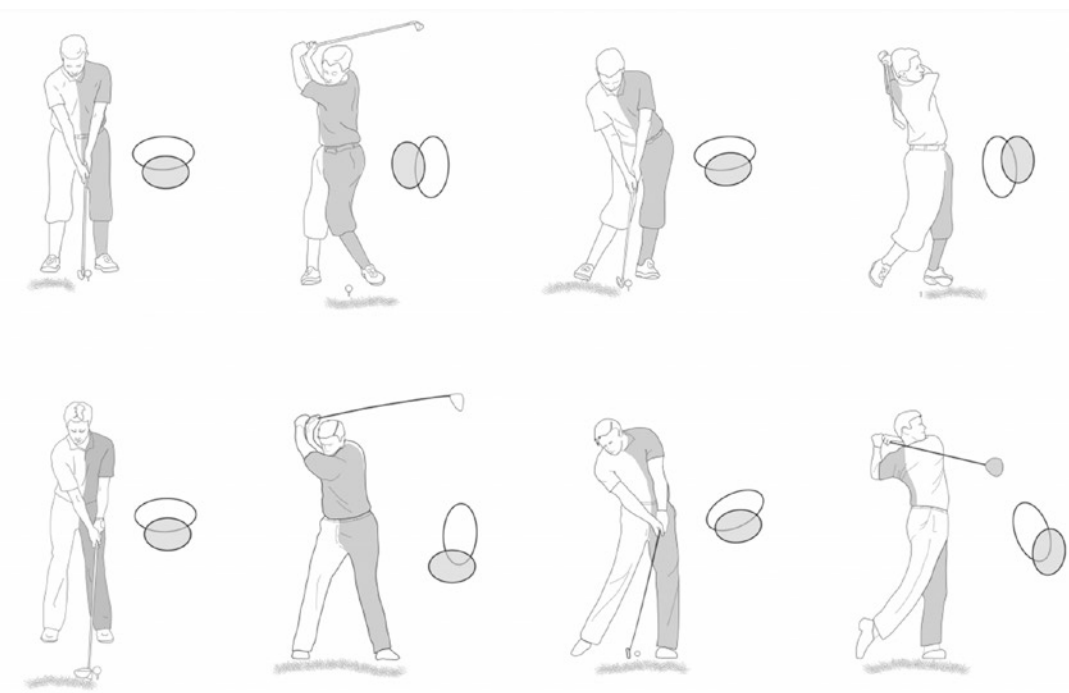

Fig. 21.5 The classic (top) and modern (bottom) golf swings compared. (Courtesy of Thomas Dolan and Matt Hazzard)

important to identify strength and mobility deficits elsewhere in the kinetic chain, which after therapy can also help improve low back pain. It is common for professional golfers to perform pre-competition exercise routines that emphasize activation and mobility of the scapular, trunk, and hip musculature. This is followed by post play exercises focused on improving flexibility in the large muscles of the low back and hips. It is also important to treat muscular asymmetries created from repetitive swinging, which if left untreated can possibly increase the risk of injury [1]. Modern training regimens use a significant amount of medicine ball core strengthening as well as yoga and foam roller stretching.

21.5 Shoulder Injuries

The shoulder is one of the more common anatomical locations for golfers to experience pain. Most of the time pain in the shoulder is due to rotator cuff pathology or subacromial impingement of the front shoulder. Golfers with shoulder injuries often experience pain in the extremes of motion for the shoulder at the top of the backswing and at the end of the follow-through of the swing [1].

The golfer will experience pain in the shoulder from subacromial impingement whenever the rotator cuff becomes impinged between the greater tuberosity and the acromion which can eventually result in rotator cuff tendinitis and partial rotator cuff tears. Initially, nonoperative treatment modalities are used including physical therapy, NSAIDs, and corticosteroid injections. After all nonoperative treatments have failed, then surgical options can be discussed with the patient.

A study by Vives et al. [16] looked at 29 amateur golfers with an average age of 60 who underwent surgical intervention for impingement and rotator cuff tears. At a mean follow-up of 3 years, only three of the patients had not returned to their previous level of play. This shows that surgical rotator cuff repair could result in return to golf without pain. Internal and external shoulder impingement can also occur during the golf swing. During the backswing, the front shoulder proceeds to significant adduction and internal rotation which leads to the humeral head and rotator cuff to impinge on the glenoid and anterior labrum. Also, at the end of the follow-through, the front shoulder moves in an abduction and external rotation position causing rotator cuff impingement on the posterior labrum and glenoid. Both external and internal impingement can cause articular-sided cuff tears, labral tears, and cartilage damage to the humeral head [1].

Another common source of pain in the shoulder for golfers is the acromioclavicular (AC) joint. A study by Mallon and Colosimo [17] showed that repetitive adduction of the front shoulder at the top of the backswing caused an increase in load across the AC joint. In the study, out of the 53 professional golfers, 53% had AC joint disease causing their shoulder pain. Only one golfer did not return to competition post-treatment. Treatment options include physical therapy, swing modification, steroid injection, and distal clavicle excision if all other conservative treatments failed [1].

Glenohumeral instability has also been reported in golfers. It is believed that golfers try to maximize their shoulder turn in relation to their hip turn in order to create more power during the swing, but this can also cause microtrauma to the capsule and labral tissues. This repetitive overuse which is exaggerated in those that have baseline hyperlaxity can result in symptomatic instability. Treatment begins with physical therapy focused on rotator cuff and periscapular stabilization and strengthening exercises. If the patient fails to improve with conservative management, then surgical options can be discussed. Surgical options include arthroscopic capsular plication, labral repair, debridement of rotator cuff, and subacromial decompression [1].

Superior labral pathology and the biceps can also be the source of pain in the golfer's shoulder. Nonoperative treatment options include physical therapy, NSAIDs, and corticosteroid injections to the glenohumeral joint and/or the biceps tendon sheath. If nonoperative management fails to relieve the symptoms, surgical treatment options include arthroscopic debridement, repair, biceps tenotomy, or tenodesis. For golfers with superior labral anterior to posterior (SLAP) tears, pain is often in the front shoulder at the end of the backswing or beginning of the forward swing when the arm is in cross-body adduction. In isolated biceps tendinitis, pain is most often experienced during late follow-through when the front shoulder is in maximum abduction, external rotation, and extension [1].

Golf is a unique sport in that it can be played by individuals of all ages. Given that many golfers are older in age, glenohumeral osteoarthritis is also a concern. This can be managed conservatively with NSAIDs and/or injections, but often requires shoulder arthroplasty when end-stage arthritis fails conservative measures. Arthroplasty has been shown to be successful in treating this patient population with one retrospective study showing 23 of 24 patients returning to golf after total shoulder arthroplasty [1, 18].

21.6 Elbow Injuries

Professional and amateur golfers both often suffer from elbow injuries most commonly from overuse. A common theme among some golfers is to grip the club too tightly which can cause increased strain on the elbow. Occasionally, trauma to the elbow can occur due to striking the clubhead against the ground prior to hitting the ball which causes sudden deceleration and places strain on the forearm flexors [1].

One of the most common elbow problems that golfers encounter is lateral epicondylitis which normally affects the lead arm. As noted above, common causes of lateral epicondylitis other than overuse is striking the ground before the ball or a grip that is too tight which causes stress at the elbow. The signs and symptoms of lateral epicondylitis are tenderness at the lateral elbow over the Extensor Carpi Radialis Brevis (ECRB) origin and pain with resisted wrist and finger extension. Conservative management includes rest and NSAIDs initially followed by stretching of the forearm extensors, strengthening, ice, ultrasound, deep tendon friction, steroid injection, platelet-rich plasma injection, acupuncture, and electrotherapy. Occasionally, lateral epicondylitis from gripping the club too tight is due to proximal weakness. In this case physical therapy sessions should include not only elbow treatment but also focus on scapular stability and rotator cuff strengthening. Others have had success with forearm and wrist braces, changing their grip on the club, or changing golf clubs from heavier steel shafts to lighter graphite shafts. After all conservative modalities have failed, both open and arthroscopic surgical intervention have had successful outcomes. Surgical intervention consists of surgical debridement of the lateral epicondyle with removal of pathologic tissue and then reattachment of tissue back to the epicondyle [1].

Medial epicondylitis or golfer's elbow normally involves the trailing arm in golfers. Once again, striking the ground or repetitive hitting off of artificial mats can cause medial epicondylitis. Most players who develop medial epicondylitis make the mistake of pushing the club through the swing with the back arm during the forward swing and acceleration, while players with proper mechanics will use the lead arm to pull the club through the swing. Conservative management like lateral epicondylitis consists of rest, NSAIDs, physical therapy, bracing, and injections. When nonsurgical management fails, open debridement of pathologic tissue of the common flexor origin and repair of the defect to the medial epicondyle is recommended [1].

21.7 Wrist Injuries

As we move down the upper extremity, wrist injuries are also common in both amateurs and professionals. The lead wrist is more often injured than the trailing wrist. Like the elbow, amateurs are more likely to injure their wrist from striking the ground with the club especially in those who have premature release of the clubhead resulting in lead wrist extension at impact and early follow-through (Fig. 21.6); however, professional golfers can also injure their wrist in this manner or swinging through thick rough. The wrist is also susceptible to overuse injuries leading to tendinitis more often in the front hand. There are certain positions during the swing that can cause pain from tendinitis. Specifically, tendinitis can occur in the extensor carpi ulnaris (ECU) and flexor carpi ulnaris (FCU) during the top of the backswing when the lead wrist moves into exces-

Fig. 21.6 Amateur golfer demonstrating excessive radial deviation and extension of the lead wrist during the backswing (left) followed by compensation with early release of the clubhead at impact and early follow-through (right) putting her at risk of wrist injury. (Courtesy of Jeffrey Pierce)

sive radial deviation (Fig. 21.6). Also, pain can occur immediately after ball impact and early follow-through when the lead wrist goes into ulnar deviation and supination from ECU instability and tears of the triangular fibrocartilage complex (TFCC) [1].

Disruption of the ECU tendon sheath can occur, if the ground is struck by the club with enough force, which can result in painful snapping sensation when impact causes supination, ulnar deviation, and flexion at the wrist. Initial treatment of ECU tendon sheath injury consists of rest and splinting in extension, radial deviation, and supination. Repair of the tendon sheath may be required if conservative management fails [1].

The TFCC is at risk for tears due to the repetitive rotation of the wrist throughout the normal golf swing. The normal presentation for TFCC injuries is ulnar-sided wrist pain with possible palpable click with forearm rotation. Treatment for TFCC injuries is rest, immobilization, NSAIDs, and steroid injections. Most of these injuries do well with therapy and rest alone. For returning to play, supportive taping and bracing can be used for professional golfers to complete tournaments, etc. If nonsurgical management fails, operative intervention involving debridement or repair can be recommended depending on the size and location of the TFCC tear [1].

Another possible injury due to striking the ground with the club includes hook of hamate fractures. Golfers will present with tenderness to palpation directly over the hook of the hamate on the volar aspect of the wrist. Carpal tunnel radiograph views can be obtained as well as a CT scan to confirm the diagnosis. Initial treatment consists of immobilization and rest. If this fail to relieve the symptoms, then surgery can be discussed to excise the hook of the hamate [1].

Two other wrist issues that can present in golfers include intersection syndrome and DeQuervain's tenosynovitis. Intersection syndrome is an overuse injury caused by friction at the intersection of the first extensor compartment containing the abductor pollicis longus and extensor pollicis brevis and the second compartment containing the ECRB and ECRL. This presents with pain proximal to the radial styloid, swelling about the wrist and forearm, and occasional redness [19]. DeQuervain's presents with pain in a similar region of the radial wrist and is caused by stenosis of the first extensor compartment. Treatment for both includes rest, NSAIDs, splinting, therapy, and steroid injection. If all nonsurgical treatment options fail, surgical intervention has been successful for both pathologies [20].

21.8 Hip Injuries

During the course of a golf swing, golfers experience rotational velocities at the hip that can result in increased joint stress. Despite the stress placed on the hip joint throughout the swing, the incidence of hip injuries in golf is just under 3%. Overuse is the main cause for hip injuries in golf which attributes for up to 78% of hip injuries. A study in 2004 found a significant correlation between limited lead hip internal rotation range of motion and the presence of low back pain [21]. Now improving hip range of motion is included in the pre- and post-round exercise routine for many professional golfers [1].

The most common hip pathology for golfers includes femoroacetabular impingement, labral tears, chondral defects, loose bodies, piriformis syndrome, and arthritis. In the past, total hip arthroplasty was the only option for significant hip pain that had failed conservative treatment. However, with the advances in hip arthroscopy techniques, it has now become an option for more golfers of all ages with positive results for almost all of the above listed pathologies with the exception of arthritis for which total hip arthroplasty is still the surgery of choice [1].

21.9 Knee Injuries

Knee injuries in golfers are relatively uncommon and only comprise approximately 4–9% of golf injuries. Like the other joint injuries in golf, most knee injuries are due to overuse or improper swing mechanics (Fig. 21.7). During the process of the golf swing the force about the knee generates internal and external rotation of the tibia on the femur which is resisted by the ligaments and menisci of the knee. Most pathology of the knee, except for a case report documenting acute bucket handle tear of the meniscus, is managed nonsurgical with rest, NSAIDs, physical therapy, and possibly corticosteroid injections. If the pain is believed to be due to early osteoarthritis, it is reasonable

Fig. 21.7 Amateur golfer with improper lower body stability that can lead to hip and knee injuries. (Courtesy of Jeffrey Pierce)

to discuss strengthening and stretching program, intraarticular injections, knee braces unloading the involved compartment, knee sleeves, shoe modifications, and orthotics although evidence in the literature is lacking and controversial. For patients with meniscal pathology that fails to improve after all conservative treatment measures, knee arthroscopy with meniscal repair vs partial meniscectomy is an option. For those with significant pain and advanced arthritis that has failed conservative treatment, total knee arthroplasty can be a good option with most people able to return to golf with minimal modifications [1].

21.10 Foot and Ankle Injuries

There is a general lack of literature describing golf-related injuries to the foot and ankle; however, there is a 1-year prospective study including 588 golfers that found the foot and ankle to be the third most injured site [11]. The majority of these injuries occurred mostly by accidents on the golf course including slipping or tripping over an object while walking.

The stability of the ankle is provided by both static and dynamic restraints. The static restraints on the lateral aspect of the ankle include the anterior and posterior talofibular ligaments and the calcaneofibular ligament followed by the deltoid ligament on the medial side of the ankle. The peroneal tendons on the lateral aspect of the ankle provide the primary dynamic restraint. There is not significant literature documenting the injuries, but golfers can sustain ankle sprains and experience tendinopathies about the ankle.

Plantar fasciitis is a common foot pathology that typically presents with pain and stiffness in the medial calcaneal tuberosity and plantar foot [22]. It can be caused by chronic overuse in golfers that walk the course and commonly described as pain when first getting out of bed or at the end of the day after prolonged standing.

Most foot and ankle injuries can be managed with conservative measures such as rest, ice, NSAIDs, proprioceptive training, stretching, braces or orthotics. If these measures fail, advanced imaging is ordered and surgical intervention can be considered.

21.11 Golf Cart Accidents

Golf cart accidents represent another main non-swing-related mechanism of injury, although not always on the golf course. Watson et al. [23] reviewed all golf cart-related injuries recorded in the USA from 1990 to 2006. They collected data on 147,696 injuries over that time from people of all ages that were treated in the emergency department for golf cart-related injuries. Children less than 16-years-old made up 31.2% of all cases. Soft tissue injuries were the most common type comprising 47.7% of all injuries. Patients required hospital admission in 7.8% of cases. When reviewing the cases that had a reported location for the accident, 70.3% occurred at sport facilities, 15.2% occurred on streets or public property, and 14.5% occurred around a home or farm. Over the 17-year course of the study, golf cart-related injuries in the USA showed an increase of 132.3%. However, this study looked at overall golf cart injuries and not specifically at golfers riding on a course.

21.12 Skin Conditions

One of the most common golf injuries not related to the golf swing or mechanics is related to sun exposure. According to a 2016 article by Dr. Mindy Clark [24], it is estimated that professional golfers received 217 times the amount of ultraviolet (UV) radiation needed to cause sunburn over the course of the year. The study also reported that for the amateur golfer for every hour on the golf course or driving range received 3.5 to 5.4 times the amount of UV radiation required to develop a sunburn. On the course water hazards and sand traps reflect the UV radiation back at the golfer giving them a second dose of radiation. Also, on cloudy days 80% of UV radiation still makes its way through the clouds to the ground. It is commonly recommended that while playing golf, one should wear the proper attire and apply sunscreen for UV protection. Skin cancers can occur as a result of repeated damage to the skin by the sun from sunburn. Skin cancer types include basal cell carcinoma, squamous cell carcinoma, and melanoma. Also notable is the precancerous growth, actinic keratosis that can develop into squamous cell carcinoma. Early identification and treatment are key to increase the likelihood of good outcomes, but prevention with appropriate clothing, hats, and sunscreen is important.

21.13 Warm-Up and Injury Prevention

The golf swing is a highly coordinated, multisegmental, rotational, closed chain activity, requiring strength, explosive power, flexibility, and balance. There is a large volume of literature that discusses the importance of appropriate range of movement and flexibility at specific joints and soft tissues in order to perform an optimal golf swing. It has been shown that static stretching during warm-up can lead to decreased performance compared to active, dynamic stretches during pre-competition warm-up. A study by Tillery & Macfarlane [25], compared three different warm-up routines in 15 elite golfers each performing all three warm-ups at different time points. The three routines consisted of active dynamic stretching, active dynamic stretching plus functional resistance bands, and active dynamic stretching with weights. They found that the active dynamic stretching plus functional resistance bands led to significant increases in consistent ball strike, maximal driving distance and smash factor with improvement but not a significant difference in maximum clubhead speed and driving accuracy. They concluded that it is beneficial to use a warm-up program that includes activation of key rotational and stability muscle groups and motor patterns in addition to dynamic flexibility in order to prepare the upper body, trunk, and legs for power production [25]. However, this study was just evaluating immediate pre-competition routine and driving distance. Another study by Doan et al. [26] developed a longitudinal training program for a group of intercollegiate golfers. Sixteen collegiate golfers (10 males and 6 females) participated in a supervised strength, power, and flexibility training for 11 weeks performed three times a week. The program was broken down into full body flexibility, strength, and core strength. Performance tests were conducted pre- and post-program and showed significant increases in overall strength, power, and flexibility testing with improvements between 7.3% and 19.9%. Clubhead speed increased significantly by 1.6% equating to approximately a 4.9 m increase in driving distance [26]. The American Academy of Orthopaedic Surgeons recommends to always warm up before a round of golf, which should consist of increasing blood flow to all muscles, raising muscle temperature, and stretches focusing on shoulder, back, and legs followed by hitting a few balls on the driving range [27].

21.14 Summary

Although one does not usually think of golf as a sport plagued with injuries, chronic impactful musculoskeletal injuries can occur. The majority of golf-related injuries are secondary to general overuse. The most common sites of injury are the back and upper extremity. Proper warm-up and conditioning can help the golfer's body tolerate the repetitive forces one experiences during the golf swing. The best golf-specific exercise program should concentrate on a combination of strength and stability in the core and lumbar spine, periscapular musculature, and hips. One should also focus on mobility in the hip joints and the thoracic spine. From a medical standpoint, it is important for treating physicians to be familiar with the phases of the golf swing, and have a general understanding of the forces applied to the various joints and body parts throughout the swing in order to identify the right diagnosis and treatment for golfers at all levels of play.

References

1. Zouzias IC, et al. Golf injuries. J Am Acad Orthop Surg. 2018;26(4):116–23. https://doi.org/10.5435/jaaos-d-15-00433.
2. Farber AJ, Smith JS, Kvitne RS, Mohr KJ, Shin SS. Electromyographic analysis of forearm muscles in professional and amateur golfers. Am J Sports Med. 2009;37(2):396–401.19022991.

3. Watkins RG, Uppal GS, Perry J, Pink M, Dinsay JM. Dynamic electromyographic analysis of trunk musculature in professional golfers. Am J Sports Med. 1996;24(4):535–538.8827315.
4. McHardy A, Pollard H. Muscle activity during the golf swing. Br J Sports Med. 2005;39(11):799–804.16244187.
5. Pink M, Jobe FW, Perry J. Electromyographic analysis of the shoulder during the golf swing. Am J Sports Med. 1990;18(2):137–140.2343980.
6. Kao JT, Pink M, Jobe FW, Perry J. Electromyographic analysis of the scapular muscles during a golf swing. Am J Sports Med. 1995;23(1):19–23.7726345.
7. Jobe FW, Moynes DR, Antonelli DJ. Rotator cuff function during a golf swing. Am J Sports Med. 1986;14(5):388–392.3777315.
8. McCarroll JR. "Golf." The Female Athlete, by Mary Lloyd. W.B. Saunders: Ireland and Aurelia Nattiv; 2002. p. 661–7.
9. Bechler JR, Jobe FW, Pink M, Perry J, Ruwe PA. Electromyographic analysis of the hip and knee during the golf swing. Clin J Sport Med. 1995;5(3):162–166.7670971.
10. Gulgin H, Armstrong C, Gribble P. Hip rotational velocities during the full golf swing. J Sports Sci Med. 2009;8(2):296–299.24149541.
11. McHardy A, Pollard H, Luo K. One-year follow-up study on golf injuries in Australian amateur golfers. Am J Sports Med. 2007;35(8):1354–60.
12. McCarroll JR. The frequency of golf injuries. Clin Sports Med. 1996;15(1):1–7.
13. Batt ME. A survey of golf injuries in amateur golfers. Br J Sports Med. 1992;26(1):63–65.1600459.
14. Gosheger G, Liem D, Ludwig K, Greshake O, Winkelmann W. Injuries and overuse syndromes in golf. Am J Sports Med. 2003;31(3):438–43.
15. Gluck GS, Bendo JA, Spivak JM. The lumbar spine and low back pain in golf: a literature review of swing biomechanics and injury prevention. Spine J. 2008;8(5):778–788.17938007.
16. Vives MJ, Miller LS, Rubenstein DL, Taliwal RV, Becker CE. Repair of rotator cuff tears in golfers. Arthroscopy. 2001;17(2):165–72.
17. Mallon WJ, Colosimo AJ. Acromioclavicular joint injury in competitive golfers. J South Orthop Assoc. 1995;4(4):277–82.
18. Jensen KL, Rockwood CA Jr. Shoulder arthroplasty in recreational golfers. J Shoulder Elb Surg. 1998;7(4):362–7.
19. Balakatounis K, Angoules AG, Angoules NA, Panagiotopoulou K. Synthesis of evidence for the treatment of intersection syndrome. World J Orthop. 2017;8(8):619–23.
20. Oh JK, Messing S, Hyrien O, Hammert WC. Effectiveness of corticosteroid injections for treatment of de Quervain's tenosynovitis. Hand (N Y). 2017;12(4):357–61.
21. Vad VB, Bhat AL, Basrai D, Gebeh A, Aspergren DD, Andrews JR. Low back pain in professional golfers: the role of associated hip and low back range-of-motion deficits. Am J Sports Med. 2004;32(2):494–497.14977679.
22. HSS PGA Portal: Plantar Fasciitis in the Golfer. (n.d.). Retrieved from https://www.hss.edu/golfportal/plantar-fasciitis-in-the-golfer.htm.
23. Watson DS, et al. Golf cart–related injuries in the U.S. Am J Prev Med. 2008;35(1):55–9.
24. Clark, M. (2016, May 29). Skin In The Game: The Overlooked Risk Of Golf. Retrieved February 9, 2020, from http://www.mytpi.com/articles/health/skin_in_the_game_the_overlooked_risk_of_golf
25. Tillery NR, Macfarlane A. Effects of different warm-up programs on golf performance in elite male golfers. International Journal of Sports Physical Therapy. 2012;7:388–95. 4th
26. Doan BK, et al. Effects of physical conditioning on intercollegiate golfer performance. J Strength Cond Res. 2006;20(1):62–72. https://doi.org/10.1519/00124278-200602000-00011.
27. "Golf Injury Prevention - OrthoInfo - AAOS." *OrthoInfo*, 2016., orthoinfo.aaos.org/en/staying-healthy/golf-injury-prevention/.

Suggested Readings

Ireland ML, Nattiv A, editors. The female athlete, chapter 56: golf. Philadelphia, PA: Saunders; 2002.

Stover CN, McCarroll JR. Mallon W (eds): feeling up to par: medicine from tee to green. Philadelphia: PA., FA Davis; 1994.

Suggested Videos

Medicine Ball Golf Workout: https://youtu.be/NqtjGmP24F8.

6 Simple Exercises for Golfers: https://youtu.be/RK8ld0M_n_4.

22 Judo, Karate, and Taekwondo

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and Carlos Henrique Fernandes

22.1 Introduction

Each martial art has a DNA that reflects how their methods of combat are organized and principles of physical, mental, and spiritual balance are guided. Martial arts are elaborated systems, schools of combat developed to achieve specific purposes as individual self-defense and military targets. Most of them have become popular worldwide, and three of them have become an Olympic Sport. This chapter approaches these three Olympic martial arts, their dynamics of combat, physical demands, and common injuries, mostly because of their dynamics: **Judo** with gripping, throwing, and being thrown, **Karate** with punching, kicking, and jumping, and **Taekwondo** with intense full-contact sparring and higher incidence of injuries during competition.

22.1.1 Judo

Created by Jigoro Kano, Judo has the philosophy of "the gentle way." This martial art was adapted from Ju-Jutsu, in 1882, as a physical, mental, and moral pedagogy method. Judo was the first martial art that has become an Olympic Sport, in Tokyo, Japan, in 1964 [1].

The techniques of Judo apply the mechanics of dynamic and static forces. The fighter learns that the movement causes imbalance, and also allows the judoka, who has his forces balanced in the static position, to perform a throwing technique more efficiently. Therefore, the judoka trains to overcome the attack of the opponent fighter using different lever mechanisms [1–3].

Judo has amazing throwing techniques that have helped to popularize it worldwide. However, Judo also has ground techniques utilizing specific pins, control holds, arm locks, and choking techniques (Fig. 22.1a and b). A particular dynamic of this fight is the dispute of grasping the opponent's kimono in an advantageous position to attack using throwing techniques as

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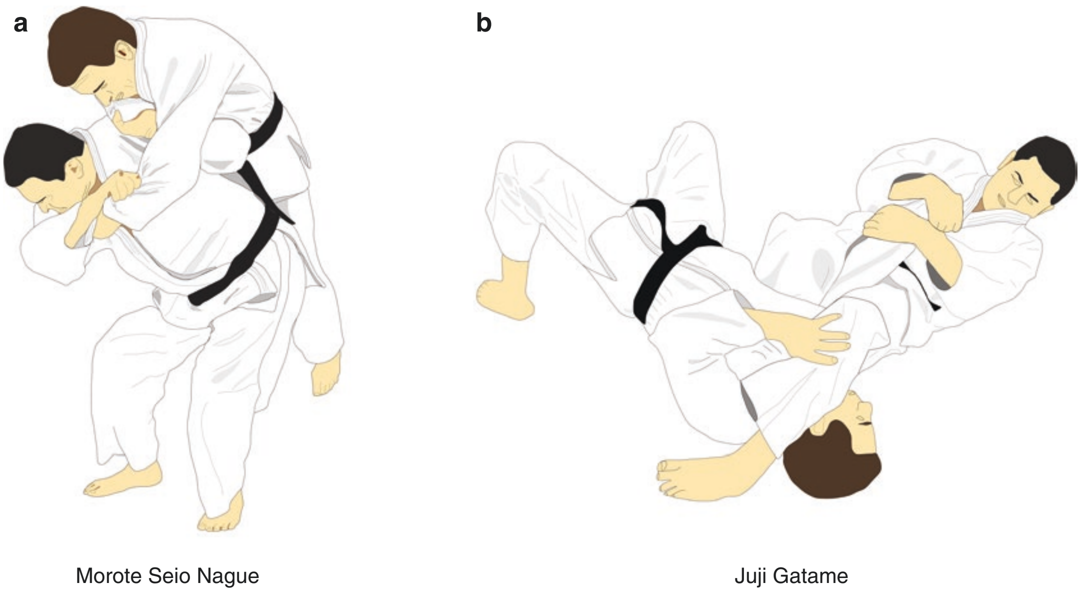

Fig. 22.1 Showing a throwing technique (**a**) and an arm lock (**b**). With permission from Ana Karina Piedade

well as ground ones, and also to block the opponent's attack. Due to the dynamics of Judo, the upper limbs have been described as usual sites of injury. However, spine and knee injuries can also take place, especially in cases of a fall over the head, an opponent's fall on the fighter's knee, or even due to an inaccurate execution of a throwing technique [3–5]. Because of the characteristics of judo combat, no specific system of protection is used regularly for training or competition.

22.1.2 Karate

Karate is a Japanese martial art, self-defense without the use of weapons ("empty hand") that perform striking sensitive areas of opponent's body using the hands, feet, elbows, and knees to overcome or disable the opponent. Karate is a martial art that has many different styles; the most popular is the Shotokan karate, also known as sports Karate, ruled by the World Karate Federation (WKF) in more than 190 countries. It will be included in the program of the Olympic games in Tokyo 2020 [6].

Karate has two competition modalities: Kata (a choreographic presentation with a sequence of techniques), and Kumite or Combat. Usually, the WKF reviews and establishes new rules to improve the athletes' safety, controlling and avoiding the use of unproportional force that can cause disqualification or even a knockout [6].

Usually, the injuries occur in a defensive position to an offensive attack produced by punches and kick traumas [3, 7] (Fig. 22.2). In Kata and Kumite, the most common anatomical sites of injuries are the face, head, neck, hand and wrist, and knee.

22.1.3 Taekwondo

Taekwondo is a Korean martial art that is practiced in more than 200 countries worldwide. In the Sidney Olympics in 2000, Taekwondo stars in the Olympics program, confirming the status of a global sport. This efficiency of Taekwondo is based on the fighter's skill to execute and coordinate the physical actions that comprise punches, jumps, and block against the opponent's attack by hands and feet. Moreover, taekwondo techniques demand balancing the speed, flexibility, agility, and endurance to overcome combat. Hand, knee, and head injuries are regularly seen in taekwondo combat or training [7, 8].

Fig. 22.2 Karate kick hitting the opponent's head. With permission from Ana Karina Piedade

Judo, Karate, and Taekwondo are sports activity where some specific injuries are more prevalent due to their dynamics such as kicking, punching, throwing techniques, falling, and so on. This chapter explores common injuries to the face, cervical spine, elbow, hand and wrist, and knee reported in Judo, Karate, and Taekwondo competitions and trainings.

22.2 Common Injuries in Judo, Karate, and Taekwondo

22.2.1 Face Injuries

In Karate and Taekwondo, the rules favor punches, kicks, and spinning kicks that hit the opponent's head and face with higher scores. This condition encourages fighters to train more intensively to improve the technique to hit this anatomical region more precisely and efficiently to win the combat faster. Thus, in Karate and Taekwondo, face trauma is a frequent event due to its dynamics, while in judo it more often results from an involuntary shock or a fall. Although minor face traumas are more commonly seen in martial arts, presenting no problem to the athlete to return and continue fighting, violent face traumas are part of the game.

In martial arts, a violent trauma is caused by a combination of factors such as the athletes' position and movement at the time of the blow, leg or hand technique employed, and blow speed. Moreover, the physician must be aware that the mechanisms of face trauma can also promote injuries to the cervical spine, and head, causing associated injuries.

22.2.1.1 Facial Trauma Assessment

Regarding minor face trauma, the assessment begins with delicate palpation, performed with both hands, to verify bone stability, depression, and mobility (crackling). It allows identifying areas of sensibility and ruling out fractures. On the other hand, the physical assessment of facial trauma that has caused fighter's loss of consciousness or knockdown must focus the attention on fighter's airways, breathing, disability, and need of prior cervical stabilization [9–11].

Moreover, it is essential to state that facial traumas can also be associated with soft tissue bleeding and be well-controlled by direct pressure and ice directly applied to the region to minimize the soft tissue edema and swelling.

22.2.1.2 Radiological Evaluation

Due to the complex anatomy of the face, computed tomography is the exam of choice for this purpose [12] as it allows a multiplanar evaluation without overlapping the structures (Fig. 22.3).

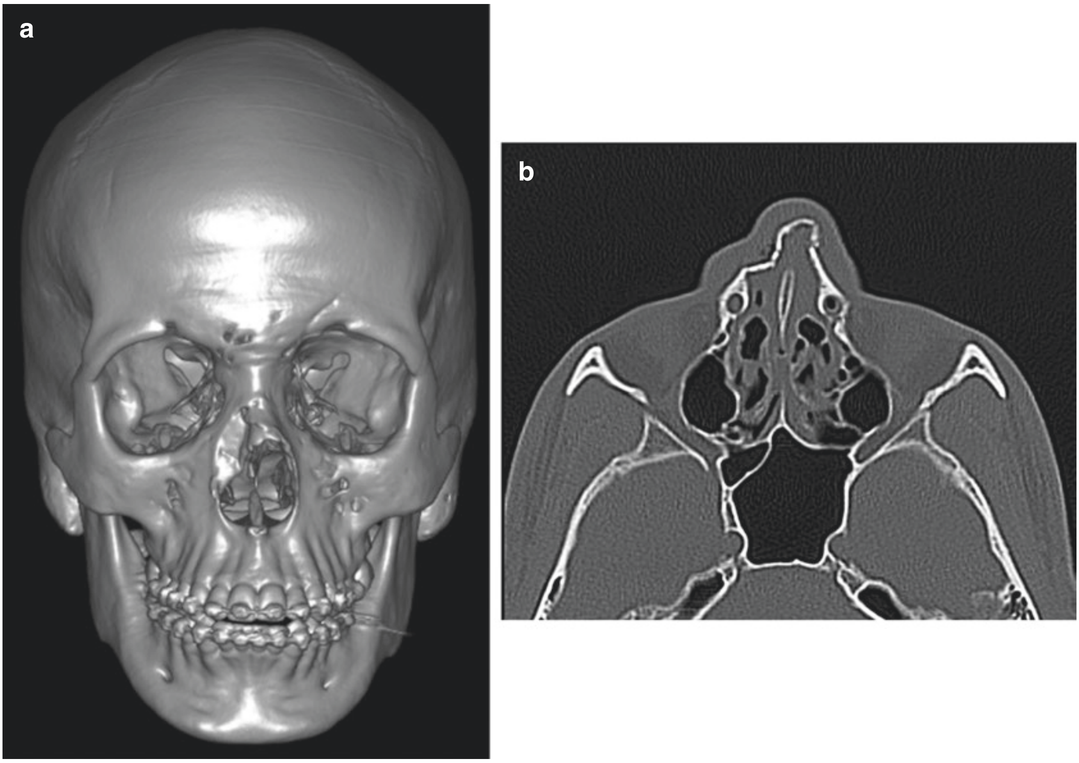

Fig. 22.3 CT images showing a comminuted nasal fracture in a karate fighter in (**a**) 3D reconstruction and (**b**) axial views. With permission from Daniel Miranda Ferreira

22.2.1.3 Nasal Fracture

The face is a well-vascularized region and, therefore, bleeding is often present, particularly in cases of nasal trauma or fracture. The initial management of nasal trauma is controlling the hemorrhage and septal hematoma drainage to avoid obstruction or severe pain. Nasal fractures can be addressed with closed reduction or an open approach [10, 12] (Fig. 22.3).

22.2.1.4 Orbital Fracture and Eye Injuries

Orbital fractures result from direct traumas to the periorbital regions. The floor and the medial wall of the orbit are less thick and resistant and, therefore, more susceptible to fracture. Herniation of the orbit contents through the fracture can cause functional deficit resulting in diplopia and loss of ocular motricity due to the entrapment of periocular muscles. In these cases, the athlete should be referred to an ophthalmologist [13].

Regarding the assessment of eye injuries, the sports medicine physician should test the visual acuity using a chart or written texts, and examine the visual field, pupil symmetry and reflex, conjunctiva, cornea, and eyelids. In case of tearing and ocular fluid drainage, photophobia, blurred vision, diplopia, pain, nausea, scotomas, and flashes of light, an ophthalmological assessment should be carried out.

22.2.1.5 Mandible Fracture

Condyles and mandibular angle are the most common sites of mandible fractures. The nondisplaced fractures can be managed conservatively, while the displaced ones need closed reduction and fixation.

22.2.1.6 Ear Injury

Injuries in the ear could result in functional hearing and balance impairment. In case of repeated traumas to the external ear, the connections between the perichondrium and the cartilage can be disrupted, causing blood accumulation in the subperichondral space. If the blood is not drained correctly, it will cause an irreparable deformity of the external portion of the ear named "cauliflower ear," commonly seen in judo fighters. Therefore, the best approach is early drainage done within 1 or 2 days [14].

22.2.1.7 Concussion

Concussion is defined as a transient neurometabolic crisis in the brain trigger by direct or indirect trauma to the head, causing violent movement inside the skull. Epidural hematoma, subdural hematoma, subarachnoid hematoma, and intracerebral hematoma are types of blood collections inside the skull, often associated with intracranial injuries [15, 16].

However, the diagnosis of concussion may be unclear as not every sports-related concussion presents clinical signs of this injury. Moreover, the physician must be aware that loss of consciousness cannot be considered to predict the severity and recovery from it. In case of a suspicious sports-related concussion, the athlete should be removed from the fight, and carefully assessed focusing on the neurological and mental status and symptoms.

Tooth Injuries

Dental injuries may be presented as dentoalveolar, tooth fracture (Fig. 22.4), tooth displacement, or even tooth avulsion and fracture of the alveolar process. Tooth avulsion is a critical dental injury where the prognosis is strictly related to adequate first aid. In these cases, the physician should deal with these following rules: avoid touching the root of the teeth, clean the teeth with water, make reposition of the teeth in the alveolar process, if it is not possible, store it in a recipient with milk or patient's saliva [17].

Fig. 22.4 Tooth fracture in a judo fighter (Courtesy: Sergio Rocha Piedade)

Prevention Measures

In Karate and Taekwondo, the use of protective equipment such as mouthguards, helmets, and headgear are used in competition and training, while in judo there is no additional equipment. Moreover, referees and athletes' education and constant development and improvements in martial arts rules also play an essential role in prevention.

22.2.2 Cervical Spine Injuries

Although cervical injuries are uncommon in martial arts, some of their mechanisms and severity are related to a fall over the head, and with specific offensive maneuvers [3, 18, 19] and, therefore, cervical spine fractures and dislocations must be a concern for a physician that works with any contact sport.

22.2.2.1 Applied Anatomy and Biomechanics of the Cervical Spine

The cervical spine is divided into two segments: upper cervical that includes craniocervical junction occiput condyle joint and C1-C2 joint, and lower cervical spine from C3 to C7. Cervical stability depends on bones, intervertebral discs, joints, and ligaments.

Injuries can occur in flexion-extension, rotation, compression, or combined mechanisms. Upper cervical subluxation and SCIWORA (Spinal Cord Injury Without Radiologic Abnormalities) are more often in the pediatric population due to the laxity of ligaments [18], while in elderly odontoid fractures and compression fractures are more often because of the bone quality and ligament stiffness [19, 20].

22.2.2.2 Epidemiology of Cervical Spine Injuries

In karate, most severe neck injuries are associated with vessel or airway trauma [20–23]. In Judo, during combat, both opponents could fall over the head, especially trying to avoid falling over back or in striking movements which may evolve using the head as support to throw the opponent, especially in the throwing technique called Uchi Mata [24] (Fig. 22.5).

Moreover, in Japan, a study has shown that spinal cord injury and cervical spine injuries in Judo reached 6.8% of all spinal cord injury in the country [25]. Most injuries in Tae Kwon do occur in lower limbs head and face; the number of concussions is four times higher than the American Football [26]. And the overall injury rates are higher than any other collision sports as wrestling and American football [27]. In Taekwondo competition, although cervical spine injury with **spinal cord injury is rare**, they are the third most common injuries, with an incidence of 13.8 injuries per 1000 athletes' expositions [28]. Biomechanical studies on the effects of Taekwondo kicks display alarming injury potential, with linear accelerations that exceed the uppercut in boxing [29]. Moreover, the **spinning kick** is the cause of the most severe injuries such as concussion, cervical trauma, and even death [29] (Fig. 22.6).

22.2.3 Elbow Dislocation

Isolated elbow dislocation is the second most common dislocation in adults, with a peak incidence between the first and third decades of life [30, 31]. It is prevalent in combat sports, presenting the second most common dislocation in judo athletes only behind shoulder dislocations [32–35].

In clinical practice, the most prevalent type is a posterior dislocation of the elbow. A fall on the hand or wrist with the elbow slightly hyperextended or fully extended seems to be condition that makes the elbow more vulnerable to this injury [35, 36].

Fig. 22.5 Fighter's fall over the head to perform an Uchi Mata technique. With permission from Alexandre Fogaça Cristante

Fig. 22.6 Taekwondo spinning kick. With permission from Alexandre Fogaça Cristante

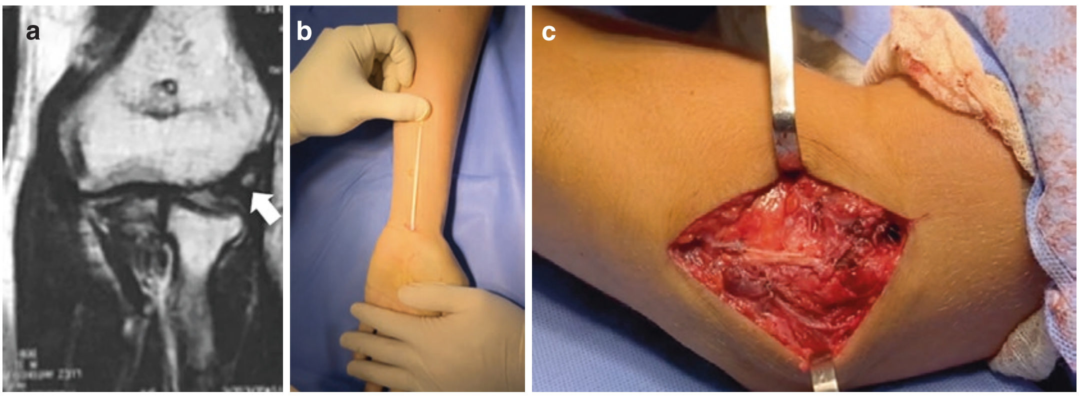

Fig. 22.7 (**a**) Preoperative MRI views of left elbow joint: bone avulsion fracture of the lateral epicondyle of humerus (white arrow); (**b** and **c**) lateral collateral ligament reconstruction of the left elbow using a graft of palmaris long in a judo fighter. With permission from Roberto Yukio Ikemoto

This mechanism results in a combination of forces that causes a sequence of soft tissue rupture that begins in the region of the lateral collateral ligament and progresses to the anterior and posterior capsule, and finally to the medial collateral ligament. Elbow dislocations can be divided into simple, when they are not associated with fractures, and complex, when associated with fractures [32].

In case of elbow dislocation, deformity is generally easily observed. The neurovascular status must be carefully assessed before and after the reduction. Although the ulnar nerve has been described as the most frequently injured, the median and radial nerve can also be damaged and must be assessed. Arterial injuries are uncommon and occur in 0.3–6% of closed elbow dislocations [35].

The anteroposterior (AP) and lateral radiographs view of the elbow as well as an oblique lateral view must be performed before and after closed reduction to rule out any associated fractures as well as to assess the quality of joint congruence. The oblique view of the elbow, Greenspan, offers a better view of the radial head, especially in cases of minimally displaced fractures of the radial head, coronoid, or capitulum. On lateral radiographs, an ulnar-humeral distance greater than or equal to 4 mm is referred to as a positive "**drop sign**" and suggests instability, which may require surgical stabilization [35, 36].

In the acute setting, MRI can offer additional information about the severity of ligament injury and avulsed bone or bone fractures (Fig. 22.7a). Regarding the vascular status, additional exams should be requested in cases of compromised limb vascularization. Additionally, in order to assess the presence of joint bone fragments and ligaments integrity, fluoroscopy can be used during or after the reduction evaluating elbow stability through its range of motion, and varus and valgus stress ligament tests [35–38].

The reduction of elbow dislocation must be performed with local intra-articular anesthesia or sedation as soon as possible to avoid the occurrence of edema and consequent suffering of the soft tissues. The reduction usually begins by correcting the medial or lateral dislocation followed by longitudinal traction and elbow flexion. After the reduction, if the elbow is stable during the entire arc of movement, it is immobilized with a simple sling for greater comfort, and then the mobility of the elbow begins [38].

If the elbow is subluxated or dislocated in extension, stability should be assessed with the forearm in pronation; if this guarantees stability, a joint orthosis can be used with blocking the extension, and the forearm in full pronation. In cases where the elbow is more stable in supination, the injury probably occurred in the medial ligament complex. The main indications for surgical treatment are when there is a restriction of 30–45° to maintain the reduction, and a positive "drop sign." In both cases, surgical repair of collateral ligaments should be considered in order to guarantee stability and early mobility. The other indication is the presence of articular bone fragments, when surgical intervention is necessary to remove the fragments [38]. The average time away from sport after an elbow dislocation is around 2 and 4 weeks when the surgical treatment is not necessary [39].

Regarding the surgical treatment in acute cases, the lateral collateral ligament is repaired by an anchor or using a tendon graft augmentation on the lateral epicondyle, using the Kocher approach, with the elbow pronated (Fig. 22.7b and c). In case of medial collateral ligament rupture, the surgeon performs a medial approach to repair the injury, using anchor or tendon graft, with the elbow in supine position. Using these surgical approaches, associated fractures can also be addressed and fixed. However, the elbow remains unstable, an external articulated fixator can be used [38, 39]. Moreover, the surgeon must remember the importance of the coronoid process for the anterior stabilization of the elbow [40–42].

22.2.4 Hands and Wrist Injuries

22.2.4.1 Phalanx Fractures

The articular fractures are the most severe that affect the fingers. Treatment decisions regarding volar base fractures of the middle phalanx depend on whether the proximal interphalangeal (PIP) joint is reduced. Fractures with less than 15–20% of articular involvement are considered stable and can be treated with immobilization as joint in flexion. When the fracture involves 30–50% of the articular surface, they are considered potentially unstable. Fractures involving greater than 50% of the articular surface are generally unstable, and the operative treatment is indicated [43] (Fig. 22.8a and b).

22.2.4.2 Metacarpal Fractures

In general, these fractures are stable due to the presence of inter-metacarpal ligaments. "Acceptable angulation" depends on the metacarpal involved with no greater than 10° tolerated in the index, and up to 30° in the small finger. Shortening of greater than 2 mm is generally not well tolerated. Mild rotation in the metacarpal can lead to significant finger overlapping when the patient makes a fist. Immobilization of isolated fractures in acceptable alignment is usually the best treatment. For unstable or rotated fractures, Kirchner wires are largely used. They are inexpensive multipurpose implants that have a relatively easy application with minimal damage to surrounding soft tissue. One main drawback of this type of fixation is related to its lack of stability, which may require a period of postoperative immobilization. In an effort to increase stability, multiple pins, thicker diameter wires, and numerous geometric configurations can be adopted. More common complications following metacarpal fractures treated with percutaneous fixation are stiffness and pin track infection. Fracture fixation with plates and screws may provide imme-

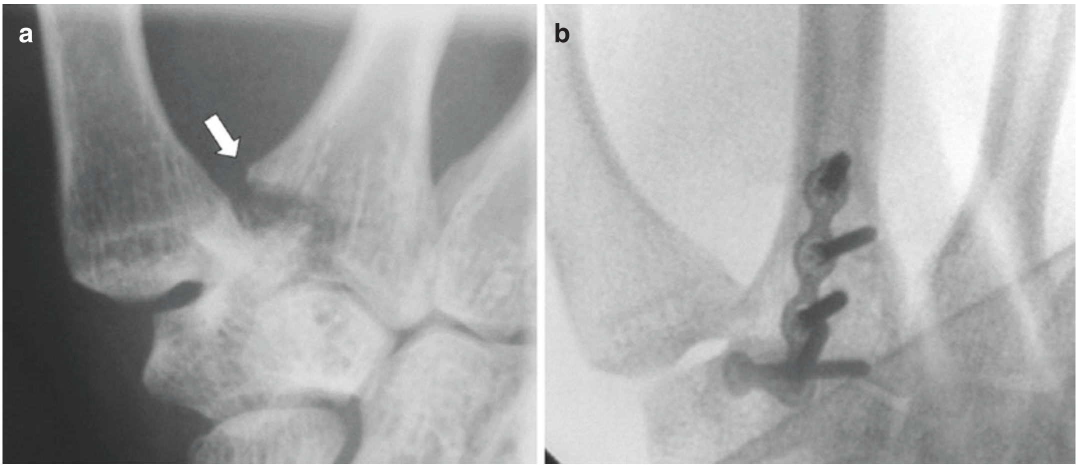

Fig. 22.8 (**a**) An AP X-ray view of fracture of base of the second metacarpal, and (**b**) postoperative X-ray showing internal fixation. With permission from Carlos Henrique Fernandes

diate stability while granting anatomic reduction. Care must be taken with plate positioning in order to avoid potential complications, such as tenosynovitis, scar formation, tendon adhesions, and tendon ruptures [44].

22.2.4.3 Sprain and Dislocation of Fingers

Finger Sprain

Finger sprain is an incomplete lesion of the capsular and ligament layers without loss of joint congruence, resulting from a direct axial impact or contact during the fight. According to the direction of the forces and the energy expended, one or more structures of the PIP joint can be injured to varying degrees, the collateral ligaments and central slip of the extensor mechanism. Sprains can cause persistent pain associated with local swelling, and there may be lateral laxity in case of complete rupture of the collateral ligament. Partial ruptures can result in voluminous joints due to capsuloligamentous scarring that may persist for several months associated with joint stiffness.

Finger Dislocation

Finger dislocation is a complete breakdown in the joint structure. Finger dislocations result in functional disability and dorsal deformity in 90% of the cases. In clinical practice, a finger dislocation can be promptly reduced after the injury. Plain anteroposterior (AP) and lateral X-rays of the finger are often sufficient. Ultrasound may detect injuries to the radial collateral ligament and volar plate. The treatment is almost always conservative. The aim is to recover digital mobility as quickly as possible. To avoid retraction of volar plate and stiffness, an immobilization of the PIP in extension is applied for a maximum of 7–10 days and may continue at night for 3 weeks. Buddy taping to the neighboring finger takes the relay and allows early mobilization. The indications of surgery are to complex or open injuries [45] (Fig. 22.9).

Fig. 22.9 A stress test in a patient with complete lesion of collateral ligament. With permission from Carlos Henrique Fernandes

22.2.4.4 Finger Osteoarthritis

A Judoka seems to be at risk of developing finger osteoarthritis due to continuous stress and injuries that can occur in hand joints along time. Soft tissue changes including Heberden nodes have been clinically observed on Judokas' fingers. The lesions are symmetrical and are not restricted to joints with tendon ruptures or fractures. Subjectively, symptoms are usually mild. The level of distal interphalangeal osteoarthritis (DIP) can be evaluated in plain anteroposterior (AP) and lateral X-rays of the finger [46].

22.2.4.5 Thumb Ulnar Collateral Ligament Injury

A forced abduction and radial deviation of the thumb can cause an injury of the ulnar collateral ligament (UCL). The UCL lesion can be associated to injury to the ulnar halves of the joint capsule, the volar plate, and the adductor aponeurosis. Athletes complain of pain, swelling at thumb MCP joint. Complete ligament injuries differ from mild sprains through a careful valgus stress test. If 35° of joint angulation was noted on valgus stress of the flexed MCP joint or if instability of the injured thumb exceeded stability of the uninjured thumb by at least 15°, we should suspect a complete ulnar collateral ligament injury. A plain X-ray can show a nondisplaced avulsion fracture. Treatment of nondisplaced avulsion fractures and partial tears of UCL entails 4 weeks of immobilization in a thumb-spica cast or splint. Unstable injuries, however, require operative intervention to repair or reconstruct the UCL. The "Stener lesion" occurs when the aponeurosis of the adductor policies is interposed between the ligament and its insertion on the proximal phalanx. The presence of the interposed tissue is therefore an indication for surgical treatment. MRI can provide the diagnosis of a Stener lesion. The surgical approach is through a lazy-S incision in a dorsal/ulnar aspect of the MCP joint. The adductor aponeurosis is taken down to allow visualization of the UCL. The UCL Mid-substance tears can be primarily repaired with 3–0 nonabsorbable suture. Distal avulsions of the base of the metacarpal can be repaired back to bone using suture anchors. After surgery, athletes involved in contact sports should be casted for 4 weeks with an additional 2 weeks of splinting [47] (Fig. 22.10).

Fig. 22.10 The abductor pollicis muscle (aponeurosis) is interposed between the ulnar collateral ligament of the thumb (white arrow). ("Stener lesion"). With permission from Daniel Miranda Ferreira

22.2.4.6 Ulnar Artery Thrombosis (Hypothenar Hammer Syndrome)

Repetitive blunt trauma or single severe trauma to the hypothenar region may lead to traumatic thrombosis of the distal ulnar artery. The onset

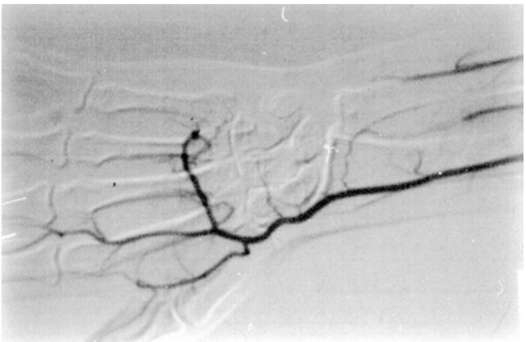

Fig. 22.11 An angiography revealing a filling defect of the distal ulnar artery (white arrow). With permission from Carlos Henrique Fernandes

of symptoms may be prolonged and misleading if the ischemic symptoms in the hand are not present. An angiography is useful to reveal filling defects of the distal ulnar artery. Treatment by just resection of the thrombosed segment or resection and replacement with an autologous vein graft resulted in complete relief of symptoms [48] (Fig. 22.11**).**

22.2.4.7 "Karate Kid" Finger

The ulnar dorsal digital nerve of the little finger overlies the prominence of the ulnar hemicondyle of the middle phalanx at the proximal interphalangeal joint level and is vulnerable to contusion injury when the hand is used to perform karate chops. Localized perineural and interfascicular fibrosis may ensue. The non-surgical treatment is avoiding trauma or using protection. If the symptoms are persistent, neurolysis is indicated [49].

22.2.5 Knee Injuries

Knee joint stability is achieved by static restrictors (ligaments) and dynamic ones (muscle actions). In a standing position, the knee helps control movements to change the force direction, supports the fighter and opponent's body weight, and performs leg and throwing techniques such as O Soto Gari, Uchi Mata, and Seoi Nage techniques.

However, in all of these conditions, the knee joint is exposed to significant loads and may become more vulnerable to injury. Usually, the mechanism of injury is valgus stress to the knee joint that can result from direct trauma to the lateral aspect knee or during the fighter's jump landing [50, 51].

According to the energy of trauma, the ligament injury varies from a sprain (different grades) to a complete ligament disruption. The medial collateral ligament (MCL) and anterior cruciate ligament (ACL) are the ligaments more commonly injured in the mechanism of valgus stress [50].

At the time of injury, the physical assessment is more accurate because there is no swelling or muscle pain contraction to affect the clinical exam. The ACL injury can be confirmed by a positive Lachman test or a positive pivot shift test, while a positive valgus stress test and tenderness on the medial side of the knee joint confirms the MCL damage. The swelling and pain tend to increase in about 6 hours after injury, interfering in the physical assessment due to pain muscle guarding. A radiological evaluation performed by X-ray and MRI (Fig. 22.12a, b and c) of the knee joint is useful to confirm the diagnosis, and to investigate associate injuries.

Regarding the MCL injuries, the conservative treatment of sprains, graded as I and II, offers satisfactory outcomes and return to sports practice. However, in some cases MCL, classified as II, associated with an ACL rupture, the surgical approach should be discussed. (Fig. 22.12d).

In case of ACL injury, there is a consensus in the literature that the ACL injury is an impairing condition to athletes' performance. Therefore, ligament reconstruction is commonly the treatment of choice. Different tendons autografts can be used in ACL reconstruction with similar results. However, the graft choice can vary according to the surgeons' options or even in case of previous knee surgery. (Fig. 22.12d).

22.3 Prevention Measures

Prevention measures focus on the constant review and renewal of rules to preserve the fighter's integrity. Moreover, investing in referees' educa-

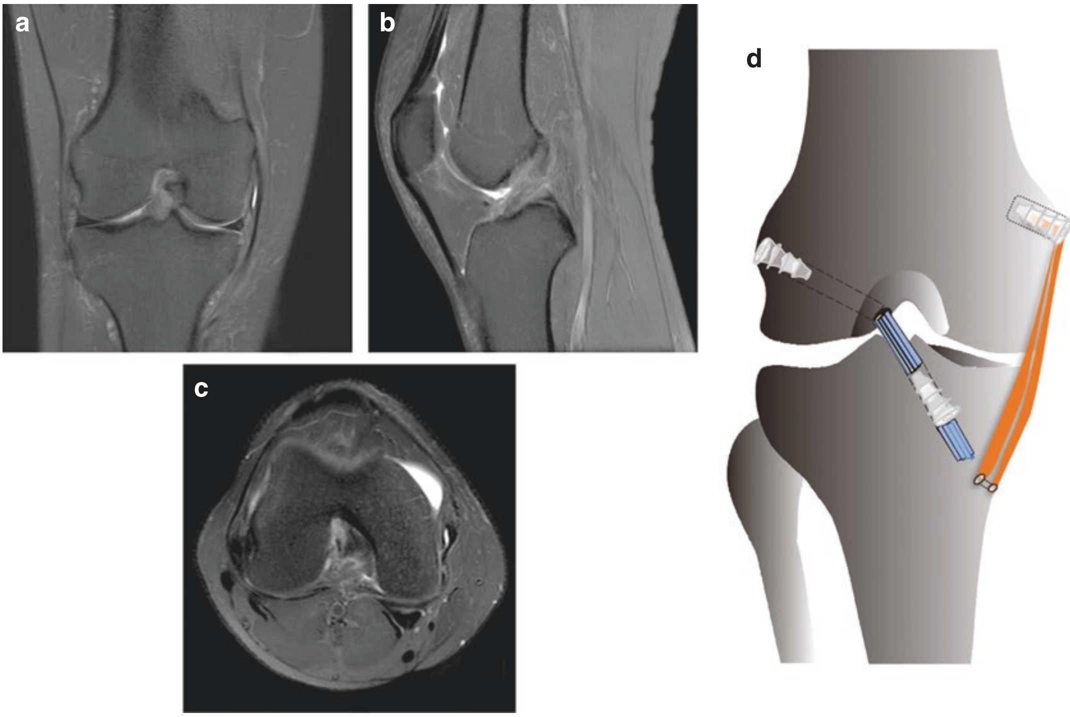

Fig. 22.12 MRI views showing MCL injury on AP view (**a**), ACL injury on the sagittal view (**b**), and axial (**c**) view and drawing of surgical reconstructions of MCL (orange) and ACL with using the tendon grafts (**d**) (Courtesy: Sergio Rocha Piedade)

tion, punishing athletes who use excessive contact and unproportional force, and athletes that perform head diving to throw or using the head to avoid falling.

Warning judges of the risks of certain injuries and their possible mechanisms during the fight.

Regular use of a system of protection (headgear, mouth guard, chest protector, hand guards, shin guard, foot guards, and so on) for training or competition, especially in Karate and Taekwondo.

Development of new equipment and materials to offer additional safety to fighters.

Well defined rules that contribute to lower risks of injuries.

Even video analysis can also contribute to the improvement of combat judgment and rules.

Take-Home Message

Judo, Karate, and Taekwondo are combat sports where some specific injuries are more prevalent due to their dynamics such as kicking, punching, throwing techniques, falling, and so on.

Knowledge of judo, Karate, and Taekwondo combat dynamics allows the physician to identify the possible mechanism of more severe injuries, and act in advance to preserve the athlete's physical integrity.

In Judo, the upper limbs are usual sites of injury, but spine and knee injuries can also take place, especially in cases of a fall over the head, an opponent's fall on the fighter's knee, or even due to an inaccurate execution of a throwing technique.

In Karate, the injuries occur in a defensive position to an offensive attack produced by punches and kick traumas.

In Taekwondo, the spinning kick is the cause of the most severe injuries such as concussion, cervical trauma, and even death.

References

1. Bergfeld JA, Gelber J, Lynch SA, Seidenberg PH, Piedade SR. In: Piedade SR, Imhoff A, Clatworthy M, Cohen M, Espregueira-Mendes J, editors. Judo, Brazilian Jiu-Jitsu, wrestling, and mixed Martial Arts. Cham: Springer; 2019.
2. Akoto R, Lambert C, Balke M, Bouillon B, Frosch KH, Hoher J. Epidemiology of injuries in judo: a cross-sectional survey of severe injuries based on time loss and reduction in sporting level. Br J Sports Med. 2018;52(17):1109–15.
3. Hammami N, Hattabi S, Salhi A, Rezgui T, Oueslati M, Bouassida A, et al. Combat sport injuries profile: a review. Epidemiology of injuries in judo: a cross-sectional survey of severe injuries based on time loss and reduction in sporting level. Science & Sports. 2018;33(2):73–9.
4. Cierna D, Stefanovsky M, Matejova L, Lystad RP. Epidemiology of competition Injuries in elite European Judo athletes: a prospective cohort study. Clin J Sport Med. 2019;29(4):336–40.
5. Ingvaldsen CA, Tonseth KA. Auricular haematoma. Tidsskr Nor Laegeforen. 2017;137(2):105–7.
6. World Karate Federation - WKF: [Internet]; 2020 [https://www.wkf.net].
7. Diesselhorst MM, Rayan GM, Pasque CB, Peyton Holder R. Survey of upper extremity injuries among martial arts participants. Hand Surg. 2013;18(2): 151–7.
8. Gesslein M, Ruther J, Bail HJ, Schuster P, Krutsch W, Wolpert AK. Injury incidence rates and profiles in elite taekwondo during competition and training. Int J Sports Med. 2020;41(1):54–8.
9. Bhamra J, Morar Y, Khan W, Deep K, Hammer A. Cervical spine immobilization in sports related injuries: review of current guidelines and a case study of an injured athlete. Open Orthop J. 2012;6:548–52.
10. Marston AP, O'Brien EK, Hamilton GS. Nasal Injuries in sports. Clin Sports Med. 2017;36(2):337–53.
11. Piedade SR, Carvalho LM, Mendes LA, Possedente M, Ferreira DM, Trauma F. In: Rocha Piedade S, Imhoff AB, Clatworthy M, Cohen M, Espregueira-Mendes J, editors. The sports medicine physician. Cham: Springer International Publishing; 2019. p. 261–73.
12. Patel Y, Goljan P, Pierce TP, Scillia A, Issa K, McInerney VK, et al. Management of Nasal Fractures in sports. Sports Med. 2017;47(10):1919–23.
13. Petrigliano FA, Williams RJ, 3rd. Orbital fractures in sport: a review. Sports Med 2003;33(4):317–322.
14. Cassaday K, Vazquez G, Wright JM. Ear problems and injuries in athletes. Curr Sports Med Rep. 2014;13(1):22–6.
15. Bailes JE, Hudson V. Classification of sport-related head Trauma: a Spectrum of mild to severe injury. J Athl Train. 2001;36(3):236–43.
16. Sherry N, Collins MW. Concussion. In: Rocha Piedade S, Imhoff AB, Clatworthy M, Cohen M, Espregueira-Mendes J, editors. The sports medicine physician. Cham: Springer International Publishing; 2019. p. 429–38.
17. Piedade SR, Ferreira DM, Filho MF, Zogiab RK, Martínez IC, Zayats V, et al. Pre-participation evaluation in sports practice. In: Rocha Piedade S, Imhoff AB, Clatworthy M, Cohen M, Espregueira-Mendes J, editors. The sports medicine physician. Cham: Springer International Publishing; 2019. p. 13–25.
18. Barros Filho TEP, Cristante AF, Marcon RM, Ono AHDA. Pediatric spinal cord Injuries surgical perspective. 1st ed. New Delhi India: LWW; 2015.
19. Zhou J, Agarwal N, Gandhoke GS, Koltz MT, Kanter AS, Okonkwo DO, et al. Long-term clinical outcomes following surgical management of cervical spine fractures in elderly patients injury incidence rates and profiles in elite taekwondo during competition and training. Interdisciplinary Neurosurgery. 2018;12(1):10–4.
20. Landim E, Cavali PTM, Neto MIR, Lima MC, Injuries S. In: Rocha Piedade S, Imhoff AB, Clatworthy M, Cohen M, Espregueira-Mendes J, editors. The sports medicine physician. Cham: Springer International Publishing; 2019. p. 241–57.
21. Thomas RE, Ornstein J. Injuries in karate: systematic review. Phys Sportsmed. 2018;46(3):279–303.
22. Blumenthal DT, Riggs JE, Ortiz O. Carotid artery occlusion following a karate punch to the neck. Mil Med. 1996;161(9):562–3.
23. Pentore R, Nichelli P. De-afferented state syndrome (locked-in syndrome) following sudden cervical

sprain trauma during a karate training session. Riv Neurol. 1991;61(2):66–70.
24. Kamitani T, Nimura Y, Nagahiro S, Miyazaki S, Tomatsu T. Catastrophic head and neck injuries in judo players in Japan from 2003 to 2010. Am J Sports Med. 2013;41(8):1915–21.
25. Katoh S, Shingu H, Ikata T, Iwatsubo E. Sports-related spinal cord injury in Japan (from the nationwide spinal cord injury registry between 1990 and 1992). Spinal Cord. 1996;34(7):416–21.
26. Pieter W, Fife GP, O'Sullivan DM. Competition injuries in taekwondo: a literature review and suggestions for prevention and surveillance. Br J Sports Med. 2012;46(7):485–91.
27. Lystad RP, Pollard H, Graham PL. Epidemiology of injuries in competition taekwondo: a meta-analysis of observational studies. J Sci Med Sport. 2009;12(6):614–21.
28. Kazemi M, Pieter W. Injuries at the Canadian National Tae Kwon do Championships: a prospective study. BMC Musculoskelet Disord. 2004;5:22.
29. Fife GP, Apos, Sullivan D, Pieter W, Kaminski TW. Effects of taekwondo kicks on head accelerations and injury potential: a pilot study. International SportMed Journal. 2013;14(2):53–66.
30. O'Driscoll SW, Morrey BF, Korinek S, An KN. Elbow subluxation and dislocation. A spectrum of instability. Clin Orthop Relat Res. 1992;(280):186–97.
31. Chang ES, Bishop ME, Dodson CC, Deluca PF, Ciccotti MG, Cohen SB, et al. Management of Elbow Dislocations in the National Football League. Orthop J Sports Med. 2018;6(2):232596711875545.
32. Frey A, Lambert C, Vesselle B, Rousseau R, Dor F, Marquet LA, et al. Epidemiology of Judo-related Injuries in 21 seasons of competitions in France: a prospective study of relevant traumatic Injuries. Orthop J Sports Med. 2019;7(5):232596711984747.
33. Morris MS, Ozer K. Elbow dislocations in contact sports. Hand Clin. 2017;33(1):63–72.
34. Skelley NW, Chamberlain A. A novel reduction technique for elbow dislocations. Orthopedics. 2015;38(1):42–4.
35. Skelley NW, McCormick JJ, Smith MV. In-game Management of Common Joint Dislocations. Sports Health. 2014;6(3):246–55.
36. McGuire DT, Bain GI. Management of dislocations of the elbow in the athlete. Sports Med Arthrosc Rev. 2014;22(3):188–93.
37. Goodman AD, Lemme N, DeFroda SF, Gil JA, Owens BD. Elbow dislocation and subluxation Injuries in the National Collegiate Athletic Association, 2009-2010 through 2013-2014. Orthop J Sports Med. 2018;6(1):232596711775010.
38. O'Driscoll SW, Jupiter JB, Cohen MS, Ring D, McKee MD. Difficult elbow fractures: pearls and pitfalls. Instr Course Lect. 2003;52:113–34.
39. Regan W, Morrey B. Fractures of the coronoid process of the ulna. J Bone Joint Surg Am. 1989;71(9):1348–54.
40. Parsons BO, Ramsey ML. Acute elbow dislocations in athletes. Clin Sports Med. 2010;29(4): 599–609.
41. Oflazoglu K, de Planque CA, Guitton TG, Rakhorst H, Chen NC. Dorsal subluxation of the proximal interphalangeal joint after Volar Base fracture of the middle phalanx. Hand (N Y). 2020:155894471989562.
42. Fernandes CH, Sabongi RG. Fracture after prior Kirschner fixation. 1 st. New York: Thieme; 2020.
43. Breeze SW, Ouellette T, Mays MM. Isolated avulsion fracture of the extensor carpi radialis brevis insertion due to a boxer's injury. Orthopedics. 2009;32(3):210.
44. Strasser P, Hauser M, Hauselmann HJ, Michel BA, Frei A, Stucki G. Traumatic finger polyarthrosis in judo athletes: a follow-up study. Z Rheumatol. 1997;56(6):342–50.
45. Halim A, Weiss AP. Return to play after hand and wrist fractures. Clin Sports Med. 2016;35(4):597–608.
46. Fernandes CH, Tinos MS, Meirelles LM. Trombose da arteria ulnar por digitacao: relato de caso. Rev Bras Ortop. 1998;33(11):911–3.
47. Chiu DT. “Karate kid” finger. Plast Reconstr Surg. 1993;91(2):362–4.
48. Pierantozzi E, Muroni R, Chiu DT. Judo high level competitions injuries "karate kid" finger. Medit J Musc Surv. 2009;7(2):26–9.
49. Green CM, Petrou MJ, Fogarty-Hover ML, Rolf CG. Injuries among judokas during competition. Scand J Med Sci Sports. 2007;17(3):205–10.
50. Koshida S, Deguchi T, Miyashita K, Iwai K, Urabe Y. The common mechanisms of anterior cruciate ligament injuries in judo: a retrospective analysis. Br J Sports Med. 2010;44(12):856–61.
51. Piedade SR, Servien E, Lavoie F. Neyret P. classification of knee laxities. The knee joint: surgical techniques and strategies. Paris: Springer Paris; 2012. p. 85–93.

23 Luge, Bobsleigh, Skeleton

Ayoosh Pareek, R. Kyle Martin, and Lars Engebretsen

23.1 Introduction

Luge, bobsleigh, and skeleton are collectively known as winter sliding sports or sledding sports. Events are performed on frozen, U-shaped tracks of ice with steep banks and twists along hills or mountainsides. There are currently 16 tracks operating throughout the world with one additional track under construction for the 2022 Winter Olympics. All three sports are performed on sleds carrying the athletes down the track, with the individual or team with the fastest time being crowned the victor.

23.1.1 Luge

Luge competitions consist of single- and double-rider formats and competitors lie supine as they travel down the track in a feet-first position (Fig. 23.1). Luge is the only sport of the three in which the athletes start the race on the sled. Special gloves with spikes are used to propel the sled forward on the ice at the beginning of the race. Once in motion, athletes recline on the sled. Steering is achieved using a combination of handles, body weight transfer, and differential leg pressure on the runners. The runners are curved pieces of fiberglass at the front of the sled and make up the main steering mechanism. Luge sleds typically do not have a braking system, and athletes must use their body position and legs to increase wind resistance and friction in order to slow down. In competition, the start point is lower down the track for women as compared with men.

23.1.2 Bobsleigh

Bobsleigh, or bobsled, athletes race down the track in a seated position inside sleds. Events traditionally consist of four-man, two-man, or

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Fig. 23.1 A luge athlete speeds feet-first down the track. Courtesy of Chris Graythen, International Olympic Committee (IOC) multimedia library, Lausanne, Switzerland, accessed March 2020

Fig. 23.2 A 4-person bobsleigh team gets a running start. Courtesy of Chris Graythen, International Olympic Committee (IOC) multimedia library, Lausanne, Switzerland, accessed March 2020

two-women competitions, while monobob, a single-rider event, has emerged in youth and para competitions. Athletes begin outside the sled and push it through a 50-m running start to gain speed before entering the bobsleigh (Fig. 23.2) [1]. The pilot uses a pulley system for steering through the track, and the brake person uses a braking system once the sled has passed the finish line. Top speeds during competition can reach up to 150 km/hr. [1]

23.1.3 Skeleton

Skeleton is a single-rider sport and the only sliding sport where the athlete races in a prone, head-first position (Fig. 23.3). As with bobsleigh, athletes get a running start and use their head, shoulders, and body weight to steer. Toe-dragging is another mechanism which is used commonly for selective steering and braking as there is no formal braking system on the sled.

Fact Box

Characteristics and comparison of sledding sports. Adapted from McCradden et al.[2]

	Luge	Bobsleigh	Skeleton
Rider position	Supine, feet-first	Seated	Prone, head-first
Events	Single-rider, double-rider	Monobob, two-person, four-person	Single-rider
Initiation	Paddle with spiked gloves	50 m running start	50 m running start
Steering mechanism	Runners, handles, body weight	Pulley system	Head and shoulder control, body weight, toe drag
Braking	None	Yes	None
Speed	Over 100 km/hr	Over 150 km/hr	Over 100 km/hr

Fig. 23.3 A skeleton athlete races head-first toward the finish line. Courtesy of Polina Golovina, International Olympic Committee (IOC) multimedia library, Lausanne, Switzerland, accessed March 2020

Winter sliding sports present unique challenges for providing medical care that a sports medicine physician must be familiar with. All three sports involve high speeds which can lead to acute, high-energy injuries and may involve multiple extremities or organs. Additionally, the outdoor nature imparts environmental concerns that may be unpredictable and rapidly changing. Weather conditions may affect the quality of the track and the nature of the injury, and can impose extrication barriers in extreme situations. The icy track is long and difficult to navigate on foot, which creates further challenges for the medical team to reach an athlete in a timely manner.

The Olympic Games and International Ski Federation (FIS) have implemented comprehensive injury surveillance systems across winter sporting events which have increased our knowledge regarding injury types, rates, and risk factors. The overriding goal of these injury registries is to use this information to help mitigate the risk of future injuries, improving athlete health and safety. This is accomplished through continuously evolving injury prevention initiatives and reassessment through ongoing surveillance.

This chapter outlines the epidemiology, etiology, and treatment of common and catastrophic injuries associated with the sliding sport injuries—luge, bobsleigh, and skeleton. Knowledge in these areas will assist the team or event physician providing care for these winter sport athletes in understanding and managing these injuries.

23.2 Mechanism

Crashes are the most common cause of injury in the sliding sports, and these may occur during training or competition runs. Crashes can range from mild to severe. Mild crashes may involve simple contact with the track edge, while more severe crashes can propel the athlete off of the sled and may result in an ejection of the athlete from the track [3]. Resulting injuries often affect exposed body parts which reside outside the confines of the sled during runs (Fig. 23.4). The extremities and head/neck regions are therefore most at risk of injury due to this mechanism; however, with ejection from the sled and/or track the entire body is at risk [2, 4–8].

It is unclear if there are sex-based differences in injury risk among sliding sport athletes. In one study, while the overall incidence of injury during the PyeongChang 2018 Winter Olympic Games did not differ significantly between female and male athletes, female athletes in luge were at a higher risk of injury compared to their male counterparts (risk ratio, RR = 5.33, 95% CI 1.61–17.71) [8]. In another study reporting on

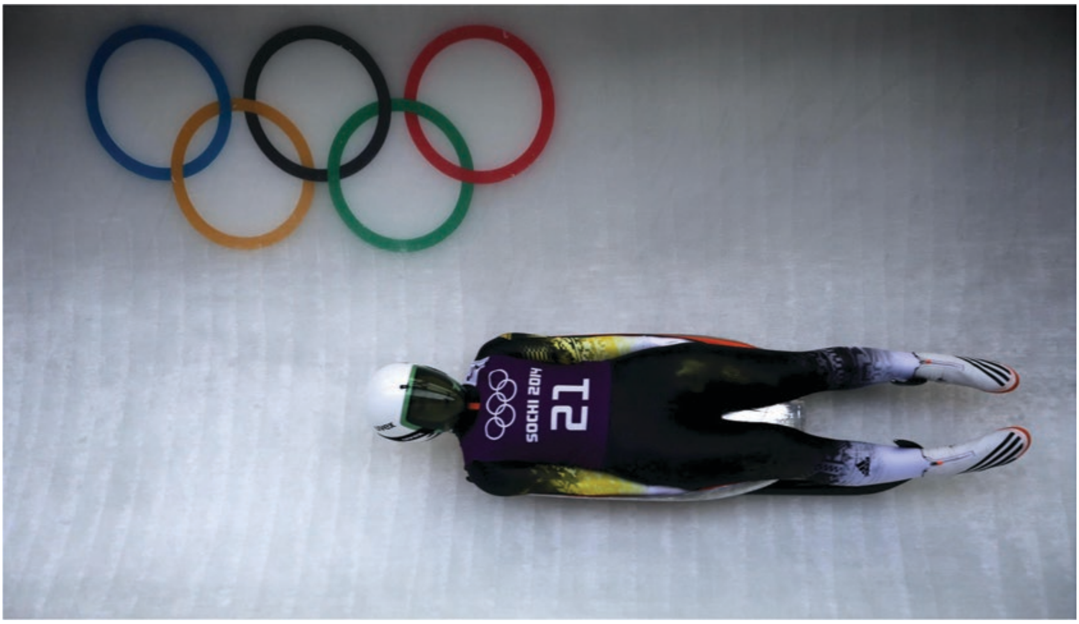

Fig. 23.4 As a luge athlete races through a curve on the track, the head and neck, along with the extremities, are exposed and vulnerable to injury in the event of a crash. Courtesy of Chris Graythen, International Olympic Committee (IOC) multimedia library, Lausanne, Switzerland, accessed March 2020

the injury rates in the 2010 Winter Olympics, females had a higher incidence of injury at 131.1 per 1000 athletes compared to male athletes at 111.8 per 1000 (RR = 1.4, 95% CI 1.1–1.8) [4]. While the prior two studies have the strength of analysis of an epidemiological cohort, they were not specific to sliding sport athletes. In addition, though these two studies did not delve into the possible mechanism behind the observed difference, other studies have noted multiple possible explanations including anatomical, neuromuscular, and hormonal differences [9]. Conversely, other studies have reported contradictory differences in the rates of injury between men and women, with some reporting that men may have a higher rate of major injuries if minor injuries are excluded [2, 4–8]. An older study found that when evaluating specifically major injuries, men were four times as likely to sustain an injury compared to women [3]. The authors suggested the higher kinetic energy was a possible reason for this difference as the men started their runs at 50% higher elevation in luge and may achieve higher speeds [3]. In addition, men are typically heavier and therefore carry higher kinetic energy and sustain higher centripetal forces in turns, possibly resulting in higher impulse loads to their bodies [10].

Injuries to the neck and spine can occur in all three sliding sports. Cummings et al. reported on luge injuries and found that non-crash-related injuries most often involved the neck and were due to “hyperextension of the athlete’s neck while driving through the curve.” [3] In bobsleigh, injuries to the neck and back may occur due to extreme centripetal forces [10]. Specifically, force is equal to a product of mass and acceleration, and by calculation, a 70 kg male sustaining a peak “g force” of 4.67 experiences a force of approximately 3200 N with greater forces for heavier riders and faster acceleration [10]. Moreover, the force is not unidirectional and often occurs with a combination of axial compression, flexion, and torsional loading to the spine [10]. These centripetal forces occur during both training and competition, and this may contribute to the relatively high proportion of head and neck injuries relative to other body regions even with helmet and other protective equipment use.

Head injuries, including concussions, are also becoming increasingly recognized in sliding sport athletes. In one study, while the rate of injury to the head was low overall (5.2% of all injuries), head injuries were disproportionately more severe as moderate or major (20%) compared to body parts like the hip (2.2%) and knee (8.8%) [3]. Across all sports, the concussion rate for the 2010 Winter Olympic Games was low at 0.8 injuries per 100 athletes, and concussions accounted for 7% of the reported injuries overall [4]. In contrast, the rate of concussion was 1.6 per 100 athletes among the sliding sports, representing 14% of the injuries sustained by sliding sport athletes overall during the 2010 Winter Olympic Games. In addition, McCradden et al. suggest that subclinical concussions, or "sled head," may be underrecognized due to the somewhat benign presentation. Athletes experiencing "sled head" typically report headaches which may last for minutes to days, beginning following sliding sport runs [2]. The proposed mechanism remains controversial with some authors citing collisions as the primary cause, while others raise the possibility that the high acceleration/deceleration forces may cause cumulative damage to the central nervous system [2, 10].

23.3 Injury Rates

23.3.1 Luge

Most of the reported injury data in the English language comes from surveillance studies of Winter Olympic Games. In the 2010 Winter Olympic Games in Vancouver, the injury rate among luge athletes was 2%. One of these injuries was a fatal track ejection during a training run [4]. The Lillehammer 2016 Winter Youth Olympic Games saw 2.9% of the luge athletes sustain an injury [7]. During the 2018 Winter Olympic Games in PyeongChang, injuries affected approximately 11% of the luge athletes with 2% missing greater than 7 days from training or competition [8].

Similar injury rates have been observed in non-Olympic events. A 1997 study examining more than 1000 luge athletes over a 7-year span recorded 407 injuries out of 57,000 track runs [3]. This corresponded to a rate of 0.39 injuries per person per year. Similarly, Stuart et al. evaluated injuries at Whistler Sliding Center from 2007 to 2011 and reported that while the risk of injury was low (4.1%) for double-rider luge, this dramatically increased to 48.6% for single-rider luge [6].

23.3.2 Bobsleigh

During the 2010 Winter Olympic Games, 20% of the bobsleigh participants sustained injuries, and the rate of injury was slightly higher among female athletes (24%) compared to the male competitors (17%). Another study followed Great Britain's Olympic Team through the 2014 Winter Olympic Games and reported an injury rate of 40% among bobsleigh athletes (4 out of 10 athletes) [5]. This rate is higher than the reported rate of 18.1% sustained by all bobsleigh athletes in the 2014 Winter Olympic Games and 14% reported for bobsleigh athletes in 2016 Youth Winter Olympic Games [7, 11]. During the 2018 Winter Olympic Games, 14% of the bobsleigh athletes sustained an injury with approximately 4% missing >1 day due to injury [8]. The injury rate of 4-person and 2-person bobsleigh at Whistler Sliding Center from 2007 to 2011 was 13.5% and 27%, respectively [6].

23.3.3 Skeleton

Few studies have reported on the rates of injury in skeleton. The largest and most recent study on these athletes was conducted during the 2018 PyeongChang Olympic Games. In PyeongChang, skeleton athletes had an injury rate of 8 per 100 athletes with approximately one-quarter of these injured athletes missing >1 day due to injury [8]. Great Britain's Olympic skeleton team reported an injury rate of 75% (3 out of 4 athletes) in the 2014 Winter Olympic Games [5]. In comparison, the injury rate among luge athletes competing at the 2016 Winter Youth Olympic Games was reported as 17.5% [7]. The authors noted that most skeleton injuries occurred during training (11 injuries) rather than competition (2 injuries). This is in contrast to the proportionally higher rate of injuries occurring in games compared with practices or training sessions observed in other sports such as ice hockey [7]. A longitudinal study over 4 years at Whistler Sliding Center reported an injury rate of approximately 6.8% in skeleton athletes.

23.4 Common Injuries

23.4.1 Luge

Cummings et al. reported that the most commonly injured body regions among USA Luge athletes were the neck (13%), hand (11%), back (9%), shoulder (7.6%), and knee (7.6%). The least common injuries involved the wrist (3%), thigh (2.2%), and face (1.7%) [3]. Contusions (51.1%) and strains/sprains (32%) were the most commonly observed injuries, but athletes also sustained more severe injuries including lacerations (3.9%), fractures (3.4%), and concussions (2.5%) [3]. The majority (89%) of the injuries were classified as "minor," resulting in no time away from sport. While 64% of the overall injuries were crash-related, 91% of the "moderate" and "major" injuries involved crashes [3]. Over a 4-year time period at the Whistler Sliding Center, concussion/head injury and abrasion/contusion were most common injuries overall, while fractures were less common among luge athletes [6]. The head injury and/or concussion rate was 12% for single-rider luge and 1.4% for double-rider luge. Similarly, the rate of contusions and/or abrasions was 18.9% for single-rider luge and 2.7% for double-rider luge. The rate of fracture was also higher in single-rider luge (8.1%), with no fractures occurring among double-rider luge athletes (Fig. 23.5) [6].

23.4.2 Bobsleigh

Similar to the other sliding sports, bobsleigh athletes commonly sustain injuries to their extremities and head/neck region. Engebretsen et al. reported that the most commonly injured body parts were the head, neck, and lower leg in

Fig. 23.5 A double-rider luge team accelerates down the track. Courtesy of Chris Graythen, International Olympic Committee (IOC) multimedia library, Lausanne, Switzerland, accessed March 2020

sliding sports, though did not specifically mention injury patterns for bobsleigh athletes [4]. Strains and contusions accounted for approximately 55% of the injuries overall, and no fractures were observed over the study period [4]. Another study reported that abrasions and contusions in 2-person and 4-person bobsleigh athletes occurred at rates of 10.8% and 2.7%, respectively [6]. The rates of concussion (2.7%) and fracture (5.4% in 2-man, 0% in 4-man) were lower than rates of strains/sprains (15.7%) and abrasions/contusions/lacerations (52%) [6].

23.4.3 Skeleton

There is a paucity of published information regarding the specific breakdown of skeleton athlete injuries by body part or injury type. Most available data suggest that athletes competing in skeleton sustain similar injuries to luge and bobsleigh athletes. In particular, contusions, sprains, fractures, and head/neck injuries including concussions occur most frequently [4–6, 8, 12]. Only one study reported on rates of injury specifically for skeleton athletes and found a 4.1% rate of abrasions/contusions, 1.4% rate of concussion/head injury, and no fractures [6].

23.5 Catastrophic Injuries

A catastrophic injury is a severe injury to the bony spine, spinal cord, or brain. The National Center for Catastrophic Sports Injury Research (NCCSIR) in the United States classifies catastrophic injuries according to fatality, permanent severe functional disability, and severe head/neck trauma with no permanent disability [13]. An epidemiological study examining the population of Ontario, Canada, reported that the greatest incidence of catastrophic injuries occurred in snowmobiling, cycling, ice hockey, and skiing, with the majority of these injuries occurring in males, and 79.2% of the injuries being preventable [14]. It is critical to be prepared to manage these injuries quickly and efficiently as these injuries can result in fatality, with a majority of them resulting in at least some form of permanent deficit in physical or mental function [15].

The thrill associated with participation in winter sports is accompanied by a risk of catastrophic injury, which may result in death [4]. In a study evaluating over 2200 patients who sustained winter sports trauma injuries, 16.7% involved head injuries, 5.5% had thoracic vertebral fractures, 5.1% had lumbar vertebral fractures, and 1.2% sustained vascular injuries [16]. One patient (0.04%) subsequently underwent an amputation and 15 patients (0.7%) died as a result of their injuries. Mortality was significantly higher in patients with a vascular injury compared to those without (7.1% vs 0.6%, $p = 0.01$) [16]. Regarding the sliding sports, Weber et al. analyzed an international trauma database and reported a higher proportion of facial and lower extremity trauma compared to snowboarding which had the highest rate of intubation and upper extremity trauma [17].

Sliding sport athletes are at risk of sustaining head, neck, and spine injuries which have the potential to be severe. Severson et al. found that sliding sport athletes can experience forces four to five times that of gravity during turns which imposes forces of greater than 3000 N. This has been associated with thoracic or lumbar spine fractures [10]. Other injuries involving the face, head, or neck are common, and are more often classified as major compared to other injury locations [3] In a study evaluating injuries in Youth Olympic games, 2 of 13 reported injuries (15%) were in the neck/cervical spine, while 3 of 13 injuries (23%) were in the lumbar spine/lower back though the overall incidence of these injures was low at 5 injuries out of 139 athletes (3.6%) (Fig. 23.6) [7].

It is critical for the sliding sport physician to be prepared for quick and effective evaluation of the injured athlete. Initial management should follow the Advanced Trauma Life Support (ATLS) and Advanced Cardiac Life Support (ACLS) algorithms and principles. After ruling out life threatening injuries, the treating physician should use the mechanism of injury, symptoms, and physical exam to guide appropriate treatment in a safe and efficient manner.

Fig. 23.6 A skeleton athlete jumps onto the sled following his running start. It is easy to visualize the strain placed on his body, particularly the head and neck, as he speeds through the race. Courtesy of Polina Golovina, International Olympic Committee (IOC) multimedia library, Lausanne, Switzerland, accessed March 2020

23.6 Revention and Treatment Principles

Protecting the health and safety of the athlete is the overriding goal of sports medicine physicians. This is accomplished through injury prevention strategies coupled with proficient recognition and treatment of illness and injury. This is especially important in sliding sport athletes who are at risk of sustaining sudden high-energy injuries. While most sliding sport injuries are localized to extremities and are minor in nature, more severe head (concussions), neck, and spine injuries do occur [2, 4–8]. In rare instances, these injuries can be fatal [4].

Protection of the athlete begins with injury prevention. Injury prevention typically proceeds through a four-step approach. The determination of injury incidence represents the first step in the sequence of injury prevention research and intervention. After establishing the extent of the injury problem including incidence and severity, the next step is to determine the common etiology and injury mechanisms. Injury prevention initiatives can then be introduced to focus on reducing common and serious injuries. The final step is to assess the effectiveness of the initiative through re-evaluation of injury incidence.

Injury prevention initiatives may focus on equipment, training, or optimization of environmental and venue factors. One study reported contusions to be the most common injury in sliding sports, often occurring in parts of the body that are exposed beyond the sled [3]. The same study proposed that more protective clothing (gloves, padded suits) in addition to wider sleds that cover more of the body could drastically reduce contusions as protective clothing is limited in sliding sport athletes due to performance concerns [3]. Due to the high number of contact-related injuries, athletes may benefit from more padded protective equipment for the head and body [4, 7, 8]. Athlete experience level has been identified as a risk factor for injury, with less experienced athletes sustaining more injuries and a higher proportion of these injuries being classified as severe [6]. Increasing exposure gradually for less experienced athletes, such as lowering the start point and sequentially moving it up as experience increases, may go a long way in limiting injuries [6]. Training of coaches and medical staff is also an important part of injury prevention, as all parties should be familiar with the emergency action plan and venue resources to limit secondary injury in the case of a catastrophic event.

Track conditions and design can be optimized to avoid injury, specifically related to the avoidance of athlete ejection from the track. One study evaluated two track ejections (including one fatal event) and noted that both ejections occurred at the same corner when the athlete contacted the ice wall. This resulted in significant vertical and rebound motion, propelling the athlete from the track [6]. In response, the track was modified and the barrier height was increased to prevent future ejections. Additionally, regulating ice conditions to keep the track in optimal condition may be critical in preventing surface condition injuries [3].

Medical providers must be familiar with the management of head and neck injuries, particularly concussions as these injuries are relatively common in luge, bobsleigh, and skeleton [2–4, 6–8]. Injury prevention strategies include improving protective equipment and decreasing the kinetic energy of the athletes during the run. Appropriate protective equipment has been found to limit both the incidence and severity of head and face trauma [18]. Hyperextension injuries to the neck and back related to high centripetal forces can be decreased if the head/neck support in the sled is increased [10]. In addition, the control of kinetic energy can be accomplished through track or rule modification. The belief is that by decreasing the gravitational forces experienced by the athletes, the incidence of headaches and subclinical concussions, in addition to neck injuries, may be reduced along with the impact force in the event of collisions or ejections [2, 3].

Fact Box

Haddon's Matrix of concussion prevention in sledding sports [2], modified from Haddon's Matrix of concussion prevention [19]

	Host	Agent	Environment
Pre-event	• Track experience. • Visualization strategies. • Strength/technique. • Concussion education.	• Improved shock absorption. • Energy absorption layer. • Retractable shield (bobsled).	• Presence of medical staff. • Compliance with track safety standards. • Ice condition standardized.
Event	• Technique. • Mouthguard use. • Helmet use. • Mental state.	• Prompt retrieval of sled.	• Maintenance of ice conditions.
Post-event	• Recognize, facilitate, and encourage reporting of concussion.	• Inspection.	• Injury documentation. • Regular performance and documentation of safety audits.

Concussion prevention initiatives can be managed using Haddon's Matrix. This considers the role of the host (athlete), agent (sled), and environment (track) in a pre-event, event, and post-event approach [2, 19]. If a concussion is suspected, early recognition and prompt removal from competition or training are the most important initial steps. At elite levels of competition, the athlete may be removed from play and assessed using tools such as the Sports Concussion Assessment Tool version 5 (SCAT5) and/or the Child SCAT5 [20]. Concussed athletes should enter a supervised gradual return-to-play protocol to minimize additional risk and prioritize the safety of the athlete upon their return.

Fact Box

Players sustaining a concussion should follow a graduated return-to-play protocol. [20] Players should be symptom-free at each stage for at least 24 hours before progressing to the next level. If symptoms reoccur, players should revert back to the previous stage until symptom-free for 24 hours. Clearance from a sports medicine professional must be obtained before progressing to Stage 5. Symptoms persisting for longer than 10–14 days in adults and >4 weeks in children warrants referral to a specialist experienced with sport-related concussions.

- Stage 1: No activity, complete rest
- Stage 2: Light aerobic activity such as walking or stationary bicycle
- Stage 3: Sports-specific training (skating)
- Stage 4: Non-contact advanced training drills and progressive resistance training
- Stage 5: Full contact training drills
- Stage 6: Return to full competition

When an injury occurs, the focus shifts quickly to management. Regardless of the specific injury circumstances, initial treatment should always follow the same basic principles. This includes prompt evaluation with initiation of the emergency action plan as indicated. Vigilance from the medical staff is vital and management should proceed as per the ATLS principles [21]. The primary and secondary surveys should be balanced with the need for extrication from the track and/or venue in a timely manner. When a cardiovascular event is suspected, the ACLS principles and algorithms should be implemented [22]. Avoidance of secondary injuries during injury assessment and management is paramount, and proper cervical spine precautions should be followed as indicated. Identification of all medical personnel with clarification of roles and responsibilities should be performed prior to the event, and, when possible, emergency scenario simulation with the team should be attempted.

Take-Home Message

Sports medicine physicians providing care for sliding sport athletes must be knowledgeable of each event including rules, regulations, sport-specific injury rates, common mechanisms, and available immediate and delayed treatment resources. In outdoor sliding sports such as luge, bobsleigh, and skeleton, environmental factors including ice and weather conditions may play a role. Therefore, medical teams must have practiced and publicized event-specific emergency action plans. Lastly, physicians should serve as leaders in the greater athletic community by encouraging proper equipment use and advocating for athlete welfare.

References

1. (IBSF) IBaSF. International Bobsleigh and Skeleton Foundation (IBSF) | Bobsleigh Info Graphics. https://www.ibsf.org/en/our-sports2020.
2. McCradden MD, Cusimano MD. Concussions in sledding Sports and the unrecognized "sled head": a systematic review. Front Neurol. 2018;9:772. https://doi.org/10.3389/fneur.2018.00772.
3. Cummings RS, Shurland AT, Prodoehl JA, Moody K, Sherk HH. Injuries in the sport of luge. Epidemiology and analysis. Am J Sports Med. 1997;25(4):508–13. https://doi.org/10.1177/036354659702500414.

4. Engebretsen L, Steffen K, Alonso JM, Aubry M, Dvorak J, Junge A, et al. Sports injuries and illnesses during the winter Olympic games 2010. Br J Sports Med. 2010;44(11):772–80. https://doi.org/10.1136/bjsm.2010.076992.
5. Palmer-Green D, Elliott N. Sports injury and illness epidemiology: Great Britain Olympic team (TeamGB) surveillance during the Sochi 2014 winter Olympic games. Br J Sports Med. 2015;49(1):25–9. https://doi.org/10.1136/bjsports-2014-094206.
6. Stuart CA, Richards D, Cripton PA. Injuries at the whistler sliding center: a 4-year retrospective study. Br J Sports Med. 2016;50(1):62–70. https://doi.org/10.1136/bjsports-2015-095006.
7. Steffen K, Moseid CH, Engebretsen L, Søberg PK, Amundsen O, Holm K, et al. Sports injuries and illnesses in the Lillehammer 2016 youth Olympic winter games. Br J Sports Med. 2017;51(1):29–35. https://doi.org/10.1136/bjsports-2016-096977.
8. Soligard T, Palmer D, Steffen K, Lopes AD, Grant M-E, Kim D, et al. Sports injury and illness incidence in the PyeongChang 2018 Olympic winter games: a prospective study of 2914 athletes from 92 countries. Br J Sports Med. 2019;53(17):1085–92. https://doi.org/10.1136/bjsports-2018-100236.
9. Ireland ML. The female ACL: why is it more prone to injury? Orthop Clin North Am. 2002;33(4):637–51. https://doi.org/10.1016/s0030-5898(02)00028-7.
10. Severson EP, Sofianos DA, Powell A, Daubs M, Patel R, Patel AA. Spinal fractures in recreational bobsledders: an unexpected mechanism of injury. Evid Based Spine Care J. 2012;3(2):43–8. https://doi.org/10.1055/s-0031-1298617.
11. Soligard T, Steffen K, Palmer-Green D, Aubry M, Grant ME, Meeuwisse W, et al. Sports injuries and illnesses in the Sochi 2014 Olympic winter games. Br J Sports Med. 2015;49(7):441–7. https://doi.org/10.1136/bjsports-2014-094538.
12. Derman W, Schwellnus MP, Jordaan E, Runciman P, Van de Vliet P, Blauwet C, et al. High incidence of injury at the Sochi 2014 winter Paralympic games: a prospective cohort study of 6564 athlete days. Br J Sports Med. 2016;50(17):1069–74. https://doi.org/10.1136/bjsports-2016-096214.
13. McKeag D, Moeller JL. American College of Sports Medicine. ACSM's primary care sports medicine. 2nd ed. Philadelphia: Wolters Kluwer Health/Lippincott Williams & Wilkins; 2007.
14. Tator CH, Edmonds VE. Catastrophic Sports and Recreational injuries in Ontario - causes and prevention. J Trauma. 1988;28(10):1511.
15. Pfeiffer RP, Mangus BC. Concepts of athletic training. 5th ed. Sudbury, Mass: Jones and Bartlett Publishers; 2008.
16. Eun JC, Bronsert M, Hansen K, Moulton SL, Jazaeri O, Nehler M, et al. Vascular injury is associated with increased mortality in winter sports trauma. Ann Vasc Surg. 2015;29(1):109–13. https://doi.org/10.1016/j.avsg.2014.10.003.
17. Weber CD, Horst K, Lefering R, Hofman M, Dienstknecht T, Pape H-C, et al. Major trauma in winter sports: an international trauma database analysis. Eur J Trauma Emerg Surg. 2016;42(6):741–7. https://doi.org/10.1007/s00068-015-0596-7.
18. Hagel B, Pless IB, Goulet C, Platt R, Robitaille Y. The effect of helmet use on injury severity and crash circumstances in skiers and snowboarders. Accid Anal Prev. 2005;37(1):103–8. https://doi.org/10.1016/j.aap.2004.04.003.
19. Haddon W Jr. Advances in the epidemiology of injuries as a basis for public policy. Public Health Rep. 1980;95(5):411–21.
20. McCrory P, Meeuwisse W, Dvorak J, Aubry M, Bailes J, Broglio S, 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. https://doi.org/10.1136/bjsports-2017-097699.
21. Galvagno SM Jr, Nahmias JT, Young DA. Advanced trauma life support((R)) update 2019: Management and Applications for Adults and Special Populations. Anesthesiol Clin. 2019;37(1):13–32. https://doi.org/10.1016/j.anclin.2018.09.009.
22. Craig-Brangan KJ, Day MP. Update: 2017/2018 AHA BLS, ACLS, and PALS guidelines. Nursing. 2019;49(2):46–9. https://doi.org/10.1097/01.NURSE.0000552705.65749.a0.

24 Marathon

Sérgio Rocha Piedade, Daniel Miranda Ferreira, Daniela Oliveira Magro, and Clea Simone S. S. Colombo

24.1 Introduction

An astonishing event of human musculoskeletal development occurs when a child becomes independent to stand up and start walking; a fantastic moment to the parents and family is to watch a child taking the first steps by themselves [1]. As time goes by, the brain maturity establishes the typical human gait pattern [2] that is followed by the innate human desire to go faster, consequently, it drives us to try the first running of our life. This physiological process strengthens the statement that running is one of the first physical activities that we learn at early ages [3].

Running is, by far, an exciting physical activity that promotes health benefits to cardiovascular, respiratory, musculoskeletal, and mental balance [4]. Most of us have already experienced how good running is. It burns calories, tones the body, sets socialization, emotional well-being, longevity. Moreover, in sports, running works as the basis for physical fitness and sports training of elite athletes as well as recreational sports practitioners.

All of this has contributed to a remarkable increase in the number of runners, making it a "big business" for the sports industry of clothing, shoes, books, energy drinks, and so on, worldwide. Consequently, new runners are born every single day, driven by the health benefits of running.

One reason why running is such an engaging, inspiring activity is the marathon race. According to a Greek legend, the concept of the marathon race was born in 490 B.C., when a messenger soldier named Pheidippides ran around 25 miles from a battlefield near Marathon to Athens to inform that the Athenians had won the battle against the Persian army. As soon as he announced the Greek victory, the exhausted soldier collapsed and died.

While a marathon is a type of endurance running race that defies athletes' body limits, it also offers them an amazing moment of joy and celebration for overcoming this challenge. All of this explains why the marathon race inspires many elite and recreational athletes [5].

This chapter explores a runners' line of thinking; The "Forrest Gump" syndrome; physiological demands in the marathon; relevant

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clinical injuries in marathon such as dehydration/hyponatremia; sudden cardiac arrest; knee pain; shin pain, muscle injury.

24.1.1 Runners' Line of Thinking

Emotions are part of human beings' character and guide most of our decision-making, mainly when we are testing and defying our body limits.

For most recreational runners, overcoming their first 5-km race works as a trigger to develop *their passion* for running. Moved by that, some runners will set their minds to finish a new challenge: a 10-km race. Due to innate human nature to face new challenges, it will be followed by a new achievement, completing a 15-km run (Fig. 24.1), and then why not a half marathon.

A half marathon race seems to be the turning point for most runners who desire to continue, exceeding their body limits and dream of running a marathon. It defines the moment in a runner's career where they must be aware of how prepared they are and their ultimate physical limit. Before facing a challenge that doubles the

Fig. 24.1 Recreational runner checking his running time during his first 15-km run

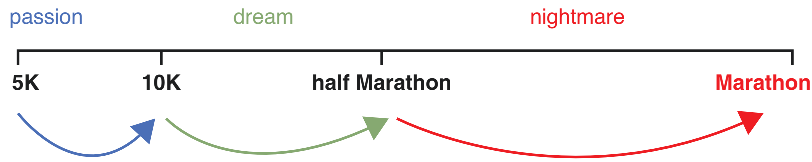

Fig. 24.2 Runners' line of thinking: passion, dream, and nightmare

physical requirement, athletes should be aware that there is a price to be paid running a marathon as it could become a "nightmare" like in the Pheidippides' story, the first Marathon runner. Figure 24.2 below shows the line of thinking of most new runners when they start facing progressive levels of running.

24.1.2 The "Forrest Gump" Runner's Behavior Syndrome—A Personal Description

In my clinical practice, I have identified a common runners' behavior that I refer to as "Forrest Gump" syndrome. In this clinical condition, after a long period of sedentarism or inactivity, a person takes up running and quickly increases the rate of training to more than four times a week as well as its intensity, adopting a non-stop running behavior, commonly triggered by the stresses of the daily working activities, need of losing weight, and so on.

Along time, without respect to the body limits, the runner becomes more vulnerable to injuries such as shin pain, knee pain, bad sleeping, insomnia, confirming that the psychological imbalance plays a vital role in this syndrome; therefore, the physician should take this into account when assessing the runner's musculoskeletal complaints.

In the elite runner's group, this scenario is not common as they have a professional entourage that guides their career and training program to overwhelm above-average of physical and mental demands.

24.1.3 Olympic Games [6]

Men's Olympic marathon debuted at the first Olympic games of modern times held in Athens, Greece, in 1896. In those Olympic games, a Greek runner, **Spyridon Louis**, was the winner and completed the first Olympic marathon in 2:58:50 h. Ninety years later, in the National Stadium of Beijing, China, the running time was reduced in almost 52 min by **Samuel Kamau Wanjiru**, a Kenyan runner, who finished this race in 2:06:32 h.

However, the first women's Olympic marathon happened in 1984, in the Memorial Coliseum Los Angeles, and the American runner **Joan Benoit Samuelson** won this race in 2:24:52 h. Twenty-eight years later, in 2012, in the London Olympic games, an Ethiopian named **Tiki Gelana** established a new women's Olympic record time: 2:23:04 h. Figure 24.3 presents the time of men's and women's Olympic marathon winners.

24.1.3.1 Amazing Moments in Olympic Marathon

Olympic marathons offer amazing and unforgettable moments to the audience such as the striking finish of **Gabrielle Andersen-Schiess**, a Swiss marathoner, in Los Angeles - 1984; and the Brazilian marathoner, **Vanderlei Cordeiro de Lima**, who was helped by a Greek spectator to get rid of a "crazy" priest who prevented him from winning the Olympic marathon of Greece in 2004.

24.1.3.2 Men's and Women's Marathon World Record

Besides the Olympics, **Eliud Kipchoge** from Kenya has two amazing men's marathon world records. In September 2018, he finished the marathon of Berlin in 2:01:39 h, and in October 2019, in Vienna, Austria, **Eliud Kipchoge** broke the two-marathon barrier, completing the running in 1:59:40 h, but it not considered an official world record. The women's marathon world record belongs to a Kenyan runner, **Brigid Kosgei** who completed this race in 2:14:04 h in Chicago, United States, in October 2019.

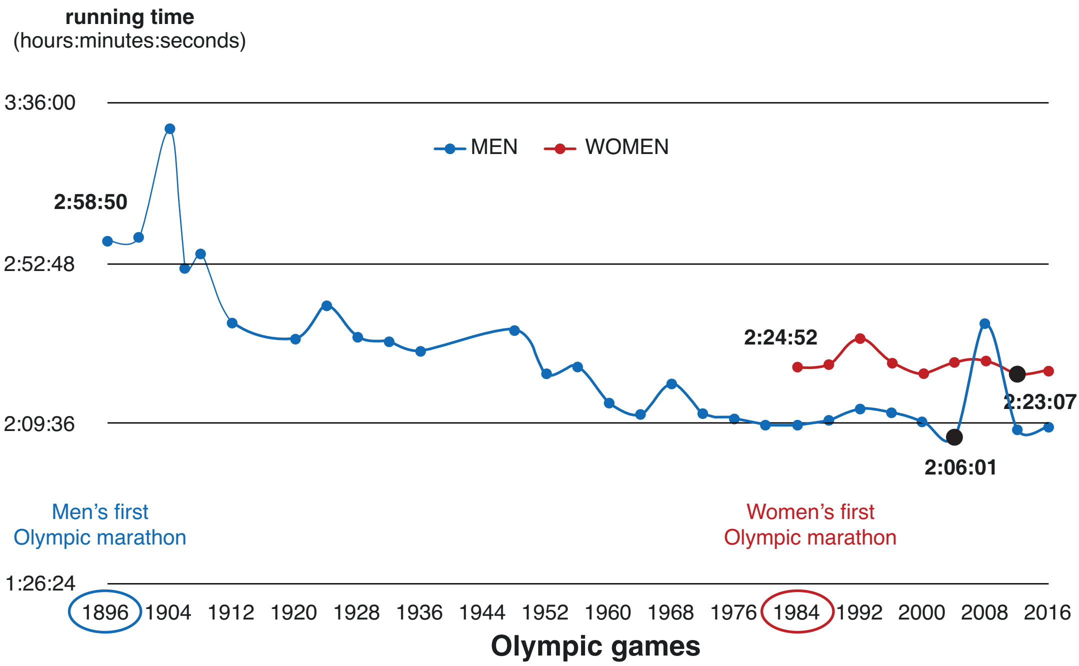

Fig. 24.3 Time of men's and women's Olympic marathon winners of modern times from 1896 to 2016. (https://www.worldathletics.org/) [6]

24.2 Physiological Demands in Marathon

Marathon is an endurance activity that drives athletes to their physical limits that require a higher level of adaptability to manage the body energy to overcome this race [7]. It is well-known that endurance training enhances muscle strength coordination, balance, and muscle blood supply that optimizes the runners' performance. Regarding the cardiopulmonary system, this training causes cardiac hypertrophy and boosts the efficiency of the cardiac pump, confirmed by reduced resting heart rate and blood pressure [8, 9]. Although endurance training offers all of these health benefits, the body response to physical overloading is not uniform for each athlete. When runners start training to overcome a marathon challenge, they could experience the effect called hitting the wall—when the oxidative system (carbohydrate) is not efficient to produce energy. Consequently, muscle fatigue takes place due to the increasing level of acid lactic in circulation, higher cost of energy, and loss of performance that will be worse if the athlete's fluid intake is not suitable because even when consuming a fluid of volume of 1 L/h, marathon runners may lose 0.3 and 1.7% of the body weight in cold and warm conditions, respectively. However, it is estimated that about 20% of endurance runners monitor their hydration status [10].

Hence, all these adverse conditions will create a "perfect" scenario to metabolic imbalance and increase the vulnerability of musculoskeletal and cardiovascular system to injuries. In the following topics, this chapter explores relevant clinical injuries in marathon such as dehydration/hyponatremia, sudden cardiac arrest, knee pain, shin pain, and foot injuries.

24.3 Relevant Clinical Injuries in Marathon

24.3.1 Dehydration

Insufficient fluid replacement during training or race events can lead to a net loss of body water and consequently induce dehydration [11]. Races in hot environments (when environmental temperature exceeds body temperature heat) and/or humid conditions increase the risk of impaired performance, heat illness, and rhabdomyolysis [12, 13]. Moreover, dehydration may diminish cognitive performance and increase perceived exertion [11].

Several factors can increase the risk of hypohydration in cold environments, such as cold-induced diuresis, impaired thirst sensation, reduced desire to drink, limited access to fluids, self-restricted fluid intake to minimize urination, sweat losses from overdressing and increased respiration with high altitude exposure. Fluid intakes and changes in body weight and hydration status should be assessed regularly when exercising in both hot and cold environments [13].

Runners should intake a fluid volume equivalent 5–10 mL/Kg body weight (~2 to 4 mL/Ib) for 2–4 h pre-race to achieve pale yellow urine color allowing enough time to empty excess fluid. Sodium consumed in pre-exercise fluids and food may help with fluid retention [13]. Fluid volumes of 0.4–0.8 L/h [7], or 150–250 mL every 20 min are recommended during racing [11]. Skeletal muscle cramps are typically caused by muscle fatigue and may be associated with hypohydration and electrolyte imbalances. Sports drinks with sodium (e.g., 300–500 mg sodium per 500 mL liquid) may help reduce the risk of cramps [12, 14].

24.3.1.1 Hyponatremia

Fluid ingestion that results in diluted plasma sodium may be indicative that runners are not meeting their sodium needs. The problems can occur when the fluid intake is rapid and excessive leading the hyponatremia (dilution of blood sodium concentration < 135 mmol/L) also known as water intoxication, although this can be exacerbated in cases where there are excessive losses of sodium in sweat and fluid replacement involving low sodium beverages. Symptoms of hyponatremia include bloating, puffiness, weight gain, nausea, vomiting, headache, confusion, delirium, seizures, respiratory distress, loss of consciousness, and possibly death if untreated [9]. In order to reduce the risk of hyponatremia during the marathon, runners should consume sodium in concentrations of 500–700 mg/L of fluid [11].

Electrolyte concentrations (particularly sodium) from commercial products may not be enough for optimal hydration, especially in hot/humid conditions, and additional sources of sodium should be considered with the aim of ingesting 500–700 mg/L (Evidence statement—category C) [11].

The incorrect hydration can lead to hyperhydration state or dehydration process. A personalized drinking plan may be adjusted to all levels of runners to avoid drinking volumes that exceed their sweat rates and could cause hyponatremia [15, 16].

24.3.1.2 Hydration

Ideally, runners must be hydrated before, during, and after exercise as it optimizes performance, prevents metabolic strain, and works on thermoregulation during the race. According to the International Society of Sports Nutrition (ISSN), for a marathon runner, hydration monitoring strategies are recommended to avoid dehydration, hyponatremia, and failure of the renal system [11]. However, insufficient hydration resulted in moderate dehydration of 3.3% and 5.3%, respectively [17].

Postexercise Fluid Intake

Usually, it is difficult for fast runners to intake a large volume of hydration during training and race events. Thermoregulatory sweat comprises water, sodium, potassium, calcium, magnesium, and chloride, so runners should replace both fluids and electrolytes as part of their recovery strategy [18]. Runners should replenish the lost fluid volume, in training, measuring pre- to postexercise body mass losses (Evidence statement—category A/B). After crucial training sessions, ingesting a fluid volume higher than that lost (150%) is necessary to restore water balance, associated at least 460 mg/L of sodium (food or a supplement) (Evidence statement—category A/B) [11, 18, 19].

In clinical practice, the urine color is well-accepted as an important and easy hydration indicator post-training. The darker color (copper brown until ruby brown) is associated with more dehydration and, therefore, requires larger quantities of liquids to recovery [11, 12].

Day-to-Day Fluid Intake

The absolute daily fluid intake (from food and drink) to attain euhydration will vary among individuals. According to Cheuvront et al., there are also daily fluctuations in total body water of ±1% of body weight (i.e., 0.6–0.9 kg in an adult of 60–90 kg) [20]. For the marathon runner, hydration monitoring strategies are recommended, by combining measures of nude body weight, thirst perception, and urine color. General day-to-day hydration, in most instances, can be obtained by the sensation of thirst (ad libitum) strategy (Evidence statement—category B/C) [11]. Recommendations are to avoid excessive dehydration (>2% body weight) as well as weight gain [15, 17]. ISSN recommends two components of hydration that are considered in the periodized nutrition program: hydration strategies to facilitate postexercise recovery and day-to-day hydration requirements that are independent of training [11].

24.3.2 Sudden Cardiac Arrest

Sudden cardiac death (SCD) is a rare event in marathon running, but it can occur unpredictably. The literature has reported a prevalence rate of 0.67 per 100,000 finishers (0.98/100,000 males and 0.41/100,000 females) [21]. Marathon race explores body limits, enhancing 10- to 15-fold in energy demands. Moreover, intrinsic and extrinsic factors may interfere in the body response to the physical stress such as the amount, intensity, and frequency of training, individual acclimatization capacity, adequate fluid and fuel replacement, and external ambient factors.

24.3.2.1 Coronary Atherosclerotic Disease (CAD)

This is the most common cause for sudden cardiac arrest in about 60% of the cases. The prevention of SCD secondary to CAD is based on the risk factor control and regular cardiovascular assessment. Keeping the blood pressure, glucose, and cholesterol levels under control, quitting smoking, and having proper training guidance are crucial to avoid the evolution of CAD and to prevent SCD. Pre-participation cardiovascular screening is the most effective preventable tool and must be encouraged before marathons. A proper cardiovascular evaluation may identify previous silent cardiac diseases, provide adequate treatment and guidance [22]. Multidisciplinary accompanying helps to minimize the occurrence of events during a marathon.

However, there are other common and unrecognized non-cardiac causes such as "**heat stroke**" and "**exercise-associated hyponatremia**" that may be easily preventable [23]. Age, running time, heat, electrolytes disturbance, and use of drugs are contributing factors to its occurrence. It is important to emphasize that the average age of marathon runners is around 45 years old, and the presence of comorbidities or the use of stimulating substances to enhance performance is frequently reported.

24.3.2.2 Exertional Heat Stroke

Heat stroke is characterized as the presence of core body temperature over 40 °C and multiple organ dysfunction, always associated with central nervous system changes.

Environmental temperature and relative humidity, heat acclimatization status, hydration status, and aerobic conditioning are determinants for body temperature and contribute for its occurrence. Heat load, low physical fitness, overweight, improper rehydration regimen, improper work/rest cycles, and sleep deprivation are predisposing factors for exertional heat stroke. The mortality rate is higher than 20% of the cases, reported as 3% of SCD in marathons [24].

Exercise practice produces heat, promotes skin vasodilation and sweating for body temperature control. During endurance exercises, especially for males with a high body surface, this mechanism may predispose to dehydration, volume depletion, and reduced muscle pump activity. Consequently, it may lead to a reduced stroke volume, cerebral hypoperfusion, and cardiovascular collapse, evolving to the loss of organ function, coma, and even death.

Clinical Manifestation

General clinical manifestations of heat stroke may comprehend fatigue, flushing, chills, dizziness, hyperventilation, impaired judgment, and collapse. All the cases have neurological manifestations ranging from unsteady gait, bizarre behavior, amnesia, and delirium to stupor, seizures, and coma.

Reduced organ perfusion leads to organ injuries such as rhabdomyolysis, hepatic necrosis, renal failure, myocardial depression, arrhythmias, and hypothalamic dysfunction, which can be fatal [25].

Management

Rectal temperature assessment is the most accurate measure of body core temperature because it is not affected by environmental or skin surface influences, such as sweating. Rapid and aggressive cooling is the key to survival. The absence of proper diagnosis and the delay in treatment have been reported in 100% of the HS fatal cases.

Immediate cooling must be done using towels or ice packs in axillae, groin, and neck, but with cold/ice water immersion is preferable [26]. It is recommended to initiate the cooling within 10 min (before transportation) and to keep it until the core body temperature is 39 °C, measuring rectal temperature every 5–10 min. Rehydration with intravenous fluids, blood glucose monitoring, and replacement are part of the treatment. The use of Diazepam may be necessary for severe cramps or seizures.

24.3.2.3 Exercise-Associated Hyponatremia (EAH)

EAH is defined when plasma or serum concentration of sodium is <135 mmol/L, symptomatic or not, up to 24 h after the end of prolonged exercise practice. In the London marathon, 12.5% of asymptomatic volunteers presented it [26]. The prevalence of EAH in marathons is around 8%, and it is reported as the more common cause of death in younger runners.

The mean cause is dilutional hyponatremia secondary to the combination of excess intake of hypotonic fluids (water) and the loss of sodium through sweat (eventually, vomiting) during endurance exercises. The extracellular fluid volume that should decrease in the normal body's response remains the same or increases and may lead to encephalopathy due to brain oedema. Eventually, the syndrome of inappropriate antidiuretic hormone secretion (SIADH) may contribute to its occurrence, but this mechanism is not completely understood. Interleukin-6 was described as an important stimulator of arginine vasopressin in marathoners, and so non-steroidal anti-inflammatory medications (NSAIDs) play a role on hyponatremia's pathogenesis and should be avoided before running [27].

Female sex, low body weight, slow running pace (> 4 h for marathons), excessive drinking (>3.5 L), NSAIDs intake, long duration of the event, and extreme heat or cold are a predisposing factor to develop EAH.

Clinical Manifestations

Symptoms are very nonspecific and may look like other conditions such as HS, hypoglycemia, and altitude sickness. Mild headache, malaise, fatigue, dizziness, tremor, increase in body weight, and vomiting may be present in the early stages. More severe degrees of hyponatremia may present confusion, agitation, cerebral and pulmonary oedema, altered behavior, seizures, coma, severe respiratory failure, and death [28].

Management

Prompt diagnosis and urgent treatment are crucial to a good outcome. Plasma sodium concentration dosage should be available at the venue, and replenishment should be initiated if it is <135 mmol/l and neurological symptoms or signs are present. It is recommended to administrate 100 mL of 3% saline solution in bolus and repeat it at 10 min intervals twice when no improvement is observed. If the patient remains symptomatic, transportation to the hospital must be done.

In conclusion, the key to sudden cardiac arrest prevention during a marathon is planning. Runners should seek for nutritional and training guidance and undergo cardiovascular preparticipation evaluation. In contrast, marathon's organizers must provide good conditions to recognition and initial treatment of the most common problems at the venues.

24.3.3 Knee Pain

Functional knee balance plays a vital role in absorbing charges, accelerating/decelerating, changing direction, and guiding the runner's performance [29]. In clinical practice, biomechanical and metabolic conditions can deteriorate the knee balance, such as overtraining, poorly adapted bodybuilding, insufficient heating and stretching, inadequate sleep, poor diet, doping with anabolic [30, 31]. All of this explains why knee pain is one of the most common complaints of long-distance runners, especially the recreational and non-elite ones [32].

The physician must be aware of that knee pain complaints are not uniform (dull, sharp, creaky, shocking) as well as the area of soreness. Usually, the leading causes of knee pain in runners are the patellofemoral pain syndrome; iliotibial band syndrome; tendinitis of patellar and quadriceps tendons (Fig. 24.4), that are commonly triggered by overtraining and muscle imbalance (poorly adapted).

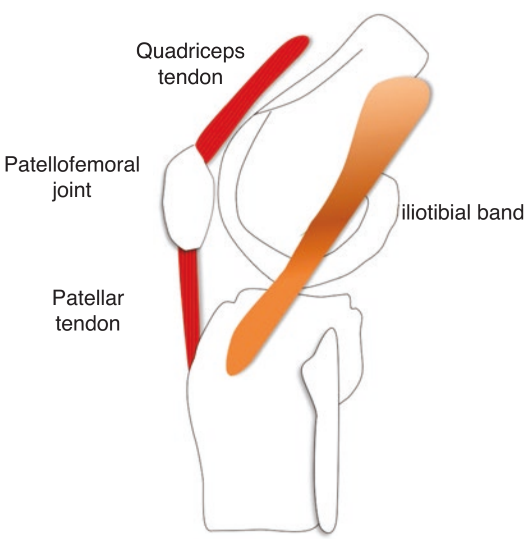

Fig. 24.4 Drawing of the sagittal view of the knee joint, and its common anatomical sites of pain in runners: patellofemoral joint; patellar and quadriceps tendons; and iliotibial band

24.3.3.1 Patellofemoral Pain Syndrome

Patellofemoral pain syndrome is the number one overuse knee injury in runners, mainly in long-distance ones. Biomechanically, the patella bone enhances the function of the knee extensor mechanism, and its ability to absorb and control the compressing and shearing forces over the patella and the femoral trochlea; however, the key point of patellofemoral pain syndrome is how well the quadriceps muscles are controlling the patella tracking [33, 34]. It reinforces the concept that muscle balance improves knee muscle power, and runner's performance. In the presence of a functional imbalance of quadriceps muscles, the femoropatellar joint is submitted to significant overloading, creating a harmful field to chondral and osteochondral injury, and with time the patellofemoral degeneration takes place [34, 35]. The severity of symptoms will increase if the athlete adopts a higher intensity of sports training (duration, intensity, frequency) and, of course, a rebel behavior—never give up and go on non-stop running like "Forrest Gump Syndrome." This behavior is commonly seen in recreational and non-elite runners.

Runners' complaints of pain and discomfort that have started insidiously, and increased progressively, lead to a loss of performance and limit their training program [36]. Crackly, dull, sharp are knee symptoms commonly reported; and sometimes it comes with swelling. Physical examination shows pain over the patella and its surroundings, and quadriceps muscles fasciculation that confirms the muscle imbalance. Although the physical examination allows for establishing the diagnosis, additional exams could request to evaluate the patellofemoral joint (X-rays, MRI) (Fig. 24.5). The treatment focuses on analgesia, physiotherapy to reestablish the knee extensor mechanism balance (stretching, strengthening), and careful reviewing and adequating the training program according to knee symptoms.

24.3.3.2 Iliotibial Band Syndrome

Iliotibial band syndrome is described as an overuse tendonitis, the leading cause of lateral knee pain in runners as well as cyclist, and military

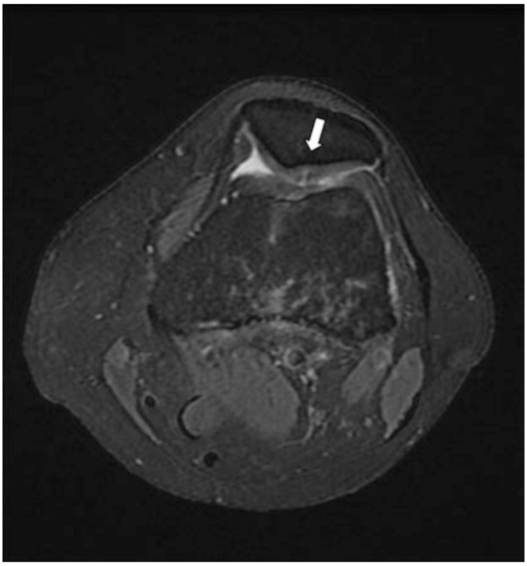

Fig. 24.5 T2 Fat Sat MRI of the knee in axial view, a defect involving more than 50% of patellar cartilage thickness, with no bone edema (grade 3 chondromalacia)

recruits. Women are more slightly affected than men. Its etiology is multifactorial, controversial, where the repetitive or undue compression of the iliotibial band to the lateral epicondyle of the femur during running seems to be one of the possible etiologies [37].

The diagnosis of iliotibial band syndrome is clinical, and most of the times, no additional exam of the image is needed to confirm the diagnosis, except to exclude knee joint injuries. Usually, the patient reports that lateral knee pain has started after a change on intensity, frequency, and duration of exercise training—moreover, the pain worse with running and ascending and descending stairs [38]. In the physical assessment, the Ober' test is a valuable tool to assess how tightness the tensor fascia lata and the iliotibial band are. Moreover, the compressive and provocative test of Noble will help to establish the diagnosis of iliotibial band syndrome ("friction"). Any other cause of knee pain should be ruled out as a differential diagnosis. However, the patient's history and physical assessment are enough to establish the diagnosis of iliotibial band syndrome in a vast majority of cases. The key to the treatment is rest and changes in physical activities, physiotherapy with a focus on stretching and strengthening exercises to achieve muscle balance, and suitable doses NSAIDs and analgesics. Surgery is an exceptional approach to this clinical problem [39].

24.3.3.3 Tendonitis of Patellar and Quadriceps Tendons

Tendonitis of patellar and quadriceps tendons is one more overuse syndrome related to long-distance knee runners. Usually, it results from repetitive mechanical stress to tendon structure, muscle imbalance, and inadequate stretching that will impact the natural ability of tendons to support and reply to running physical demands, negatively [40, 41]. Moreover, poor athlete nutrition, inadequate sleep, as well as psychological imbalance (anxiety, depression) can also be associated with this clinical syndrome and should be carefully investigated.

In the physical examination, the patient refers to pain over the tendon body that could be elicited by palpation, and when performing active knee extension against resistance. The physician must be aware of a deficient hamstring stretching, a common finding, as it plays a vital role in this pathology. Usually, the treatment focus rest by reducing the intensity and frequency of physical activity as well as avoiding conditions that cause significant overload to the tendons. Analgesics and NSAIDs must be used in suitable doses; remember pain relief is an essential parameter to assess tendon healing; physiotherapy and careful adapting of the training program are the keys to achieve the best outcomes [42, 43].

24.3.4 Shin Pain

Another common complaint reported by long-distance runners is discomfort and pain on the anterior or medial side of the lower leg, so-called as medial tibial stress syndrome (MTSS], "shin splints," or shin pain (Fig. 24.6). Shin pain has been associated with a higher level of physical demands in long-distance runners, jumpers, endurance sports, dancers, and military. This entity may affect recreational and professional

Fig. 24.6 Common anatomical area of shin discomfort and pain on the lower leg

athletes, indistinctly. Moreover, fast changes in the training program regarding the amount, type, and intensity of the physical activity, and a huge number of competitions can be the trigger to develop the symptoms of pain and discomfort on the lower leg. Novice runners are commonly affected by this type of pain as they increase their distance and intensity of running without guidance [44].

24.3.4.1 Clinical Diagnosis

In clinical practice, different diagnosis may be considered according to the runner's complaints. The shin pain increases with sports activity and relieves with rest. It is described as vague and diffuse, and comprises redness, swelling, and tenderness along the inner edge of the two-thirds of the shinbone. On the other hand, in the exertional compartment syndrome, the pain starts 10 min after running, followed by sensory and motor loss, and relief with 30 min of rest. In tendinopathies, the pain is related to the tendon structure and its movement. Finally, in the medial tibial stress, the pain is vague and diffuse, and a functional test can be used to distinguish it from a stress fracture. A patient with MTSS can hop at least ten times over the affected leg while a patient with a stress fracture cannot without severe pain [45].

Therefore, the anamnesis plays an essential role to guide the athletes' physical assessment and to answer some critical questions before establishing the diagnosis and treatment. The body mass index (BMI), gender, intensity of training, type of shoes, type of training surface, regularity, supervision from a coach, adaptation of the training program, and the athlete's ability to define the onset and timing of symptoms are essential parts of these approaches [46, 47].

24.3.4.2 Radiological Assessment

In most cases of medial tibial stress syndrome, plain radiograph (X-rays) and computed tomography (CT) may show no changes, or even, a slight cortical thickening, and a laminar periosteal reaction in the medial tibial. Bone scintigraphy is relatively sensitive and may demonstrate uptake in the affected region. MRI is the most sensitive radiological assessment, displaying from a normal exam to periosteal fluid (Fig. 24.7), and from marrow edema to a line of fracture. The tendinopathies are well evaluated by both MRI and ultrasound (US), and exertional compartment syndrome can be evaluated by MRI to analyze the structure of the muscles in the compartments. Fredericson et al., based on radiological findings such as periosteal edema, bone marrow edema, and intracortical signal abnormality, developed an MRI classification for tibial stress injuries [45].

24.3.4.3 Treatment and Prevention

The athletes should rest while pain and discomfort persist. Initially, the treatment focuses on rest and pain relief (use of analgesics and NSAIDs for a week), followed by physiotherapy to achieve pain relief, stretching exercises, muscle strengthening, and muscle balance.

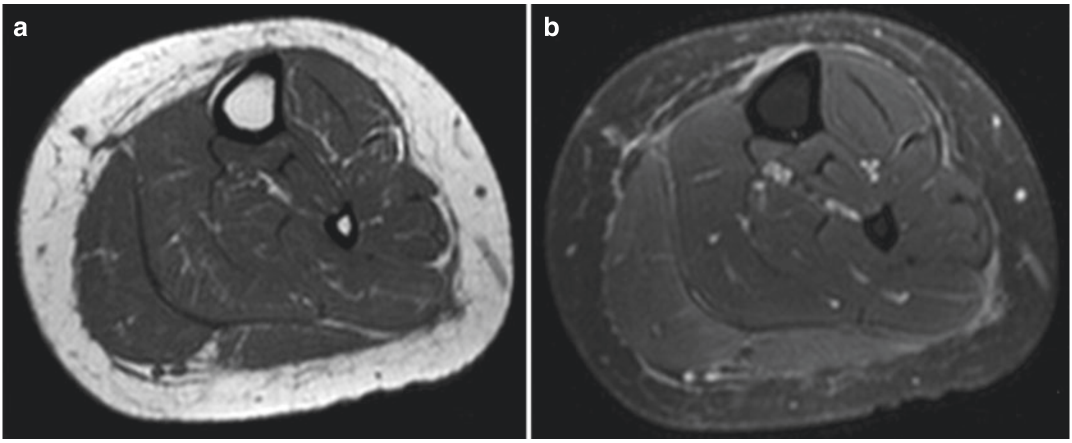

Fig. 24.7 MRI images showing periosteal edema along the anteromedial border of the tibial diaphysis in (**a**) axial T1 and (**b**) Axial T2 Fat Sat views

Prevention measures are based on reviewing the training program, progressive escalating the amount, type, and intensity of training as well as reducing the periodicity of running according to the athlete's response to the treatment.

24.3.5 Runner's Toe and Blisters

Runner's toe (subungual hematoma) and blisters are one of the more common dermatologic injuries in long-distance runners. Blisters can cause severe pain and discomfort with every step, infection, withdrawal from the competition, and have been attributed as an essential risk factor in the development of overuse injuries.

Subungual hematoma is often caused by a crush injury resulting from repetitive trauma in athletes such as runners and dancers. The nail plate rips away from the nail bed and causes the bleeding. Runners should wear running shoes that are a full size larger than their regular shoes to avoid runner's toe (Fig. 24.8).

Blood is by far the most common cause of dark nail pigmentation or color. Subungual hematomas usually do not pose diagnostic problems because acute traumas are usually evident. However, hemorrhages mimicking longitudinal streaks may also result from repeated minor traumas frequently caused by frictions with shoes and that are easily unnoticed.

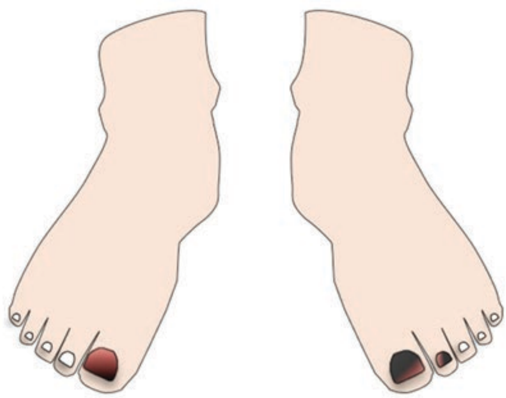

Fig. 24.8 Runner's toe (subungual hematoma)

Subungual pigmentation is a very common problem and can result from pigments derived from melanin, blood, or fungi. Subungual hematoma usually appears as a reddish to reddish-black pigment (color) depending on the age of the bleeding, and it can easily be misdiagnosed as melanoma. Subungual melanomas are the differential diagnosis that most commonly starts as a brown to black nail pigmentations such appearances may be simulated by several other conditions such as bacterial, mycotic, or blood pigments. The presence of dermoscopic features of subungual hematoma is not pathognomonic for the diagnosis of hematoma. The diagnosis of subungual hematoma should be made merely after the exclusion of the presence of criteria typical of melanocytic lesions.

Subungual hemorrhages are characterized by well-circumscribed dots or blotches with a red to red-black pigmentation. Dermoscopy has proven to be a useful, noninvasive tool in the diagnosis of pigmented lesions in the nail; however, few dermoscopic studies of subungual hemorrhages have been reported.

Blisters prevention may be done by athletic socks, emollients, and aluminum-based antiperspirants to decrease friction. Runners use cushioned insoles for cushioning and shock absorption, but not typically for blister prevention. Silicone ointment appears to have a protective effect against maceration and blisters that form as a result of extreme exposure to moisture.

Recommendations for blister treatment for hot spots and blisters less than 5 mm in size, one should (1) clean the area with hot soap and water or antibacterial scrub, (2) apply a hydrocolloid or hydrogel patch, or a small amount of petrolatum covered with either a donut pad or moleskin, (3) check the athlete's shoes for abnormal pressure points or defects in craftsmanship, and (4) re-examine the blister in 24 h for signs of infection. For larger blisters (> 5 mm), (1) clean the area with hot soap and water or antibacterial scrub, (2) at approximately 24 h drain the blister of fluid with a sterile technique and an 18-gauge needle or syringe (do not unroof the blister unless the roof is mostly torn or there are signs of infection), (3) apply a hydrocolloid or hydrogel patch or a thin layer of petrolatum followed by a sterile gauze or moleskin, and (4) re-check the blister in 24 h but do not remove the patch unless there are signs of infection; this will allow for healing of the roof and the floor of the blister, and (5) re-check the blister at 72 h. The patch or dressing can be removed, the blister cleaned, and a new bandage or moleskin can be applied. Be careful not to tear off the roof of the blister when changing the dressing. As long as there are no signs of infection, and the athlete is relatively pain-free, he or she may safely return to sporting activities [48].

Take-Home Message

- Marathon is an endurance activity that drives athletes to their physical limits.
- Dehydration deteriorates thermoregulation during the race, enhances the metabolic strain, and worsens the runners' performance and, therefore, runners must be hydrated before, during, and after exercise.
- a pale yellow urine color is well-accepted as an important and easy hydration indicator post-training.
- Sudden cardiac death (SCD) during marathons is rare, but it may occur unpredictably.
- Lower limb pain in runners is commonly triggered by overtraining, poorly adapted bodybuilding, insufficient heating and stretching, inadequate sleep, poor diet, doping with anabolic.
- Shin pain may result from different pathologies.
- Runner's toe (subungual hematoma) and blisters are common dermatologic injuries in long-distance runners that may become a risk factor in the development of overuse injuries.

References

1. Lubans DR, Morgan PJ, Cliff DP, Barnett LM, Okely AD. Fundamental movement skills in children and adolescents: review of associated health benefits. Sports Med. 2010;40(12):1019–35.
2. Van de Walle P, Meyns P, Desloovere K, De Rijck J, Kenis J, Verbecque E, et al. Age-related changes in arm motion during typical gait. Gait Posture. 2018;66:51–7.
3. AIMS: The home of world running 2020 [Available from: https://aims-worldrunning.org/aims.html.
4. van Gent RN, Siem D, van Middelkoop M, van Os AG, Bierma-Zeinstra SM, Koes BW. Incidence and determinants of lower extremity running injuries in

long distance runners: a systematic review. Br J Sports Med. 2007;41(8):469–80. discussion 80
5. Sjodin B, Svedenhag J. Applied physiology of marathon running. Sports Med. 1985;2(2):83–99.
6. World Athletics: worldathletics.org; 2020 [Available from: https://worldathletics.org/.
7. Mahler DA, Loke J. The physiology of Marathon running. Phys Sportsmed. 1985;13(1):84–97.
8. Tiller NB. Pulmonary and respiratory muscle function in response to Marathon and ultra-Marathon running: a review. Sports Med. 2019;49(7):1031–41.
9. Millet GP, Millet GY. Ultramarathon is an outstanding model for the study of adaptive responses to extreme load and stress. BMC Med. 2012;10:77.
10. O'Neal EK, Wingo JE, Richardson MT, Leeper JD, Neggers YH, Bishop PA. Half-Marathon and full-Marathon Runners' hydration practices and perceptions. J Athl Train. 2011;46(6):581–91.
11. Tiller NB, Roberts JD, Beasley L, Chapman S, Pinto JM, Smith L, et al. International Society of Sports Nutrition Position Stand: nutritional considerations for single-stage ultra-marathon training and racing. J Int Soc Sports Nutr. 2019;16(1):50.
12. Olympic World Library - Nutrition for athletes: a practical guide to eating for health and performance : based on an International Consensus Conference held at the IOC in Lausanne in June 2003 / prepared by the Nutrition Working Group of the International Olympic Committee - Detail. 2020.
13. 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(3):501–28.
14. Heaton LE, Davis JK, Rawson ES, Nuccio RP, Witard OC, Stein KW, et al. Selected in-season nutritional strategies to enhance recovery for team sport athletes: a practical overview. Sports Med. 472017:2201–18.
15. Burke LM, Jeukendrup AE, Jones AM, Mooses M. Contemporary nutrition strategies to optimize performance in distance runners and race walkers. Int J Sport Nutr Exerc Metab. 2019;29(2):117–29.
16. Belinchón-deMiguel P, Tornero-Aguilera JF, Dalamitros AA, Nikolaidis PT, Rosemann T, Knechtle B, et al. Multidisciplinary analysis of differences between finisher and non-finisher ultra-Endurance Mountain athletes. Front Physiol. 2019:10.
17. Cheuvront SN, Montain SJ, Sawka MN. Fluid replacement and performance during the marathon. Sports Med. 2007;37(4–5):353–7.
18. McCubbin AJ, Allanson BA, Caldwell Odgers JN, Cort MM, Costa RJS, Cox GR, et al. Sports dietitians Australia position statement: nutrition for exercise in hot environments. Int J Sport Nutr Exerc Metab. 2020:1–16.
19. Puchalska P, Crawford PA. Multi-dimensional roles of ketone bodies in fuel metabolism, signaling, and therapeutics. Cell Metab. 2017;25(2):262–84.
20. Cheuvront SN, Kenefick RW. Dehydration: physiology, assessment, and performance effects. Compr Physiol. 2014;4(1):257–85.
21. Dayer MJ, Green I. Mortality during marathons: a narrative review of the literature. BMJ Open Sport Exerc Med. 2019;5(1):e000555.
22. Colombo CSSS, Papadakis M, Ghorayeb N. Sudden Cardiac Arrest. In: Rocha Piedade S, Imhoff AB, Clatworthy M, Cohen M, Espregueira-Mendes J, editors. The sports medicine physician. Cham: Springer International Publishing; 2019. p. 413–28.
23. Yankelson L, Sadeh B, Gershovitz L, Werthein J, Heller K, Halpern P, et al. Life-threatening events during endurance sports: is heat stroke more prevalent than arrhythmic death? J Am Coll Cardiol. 2014;64(5):463–9.
24. Alonso JM, Dijkstra P. Alternative noncardiac medical causes of sudden death: sickle-cell trait, exertional heatstroke, exercise rhabdomyolysis, asthma and extreme environmental conditions. IOC Manual of Sports Cardiology. 2016:359–69.
25. Zeller L, Novack V, Barski L, Jotkowitz A, Almog Y. Exertional heatstroke: clinical characteristics, diagnostic and therapeutic considerations. Eur J Intern Med. 2011;22(3):296–9.
26. Kjertakov M. Exertional heat stroke in athletes. Macedonian Journal of Medical Sciences. 2013;6:473–7.
27. Kipps C, Sharma S, Tunstall Pedoe D. The incidence of exercise-associated hyponatraemia in the London marathon. Br J Sports Med. 2011;45(1):14–9.
28. Whatmough S, Mears S, Kipps C. Serum sodium changes in marathon participants who use NSAIDs. BMJ Open Sport Exerc Med. 2018;4(1):e000364.
29. Knechtle B, Chlibkova D, Papadopoulou S, Mantzorou M, Rosemann T, Nikolaidis PT. Exercise-associated hyponatremia in endurance and ultra-endurance performance-aspects of sex, race location, ambient temperature, sports discipline, and length of performance: a narrative review. Medicina (Kaunas). 2019;55(9)
30. Flandry F, Hommel G. Normal anatomy and biomechanics of the knee. Sports Med Arthrosc Rev. 2011;19(2):82–92.
31. Babel P, Bajcar EA, Smieja M, Adamczyk W, Swider K, Kicman P, et al. Pain begets pain. When marathon runners are not in pain anymore, they underestimate their memory of marathon pain--a mediation analysis. Eur J Pain. 2018;22(4):800–9.
32. Mellinger S, Neurohr GA. Evidence based treatment options for common knee injuries in runners. Ann Transl Med. 2019;7(Suppl 7):S249.

33. Fredericson M, Misra AK. Epidemiology and aetiology of marathon running injuries. Sports Med. 2007;37(4–5):437–9.
34. Dutton RA, Khadavi MJ, Fredericson M. Patellofemoral pain. Phys Med Rehabil Clin N Am. 2016;27(1):31–52.
35. Bonacci J, Hall M, Fox A, Saunders N, Shipsides T, Vicenzino B. The influence of cadence and shoes on patellofemoral joint kinetics in runners with patellofemoral pain. J Sci Med Sport. 2018;21(6):574–8.
36. Witvrouw E, Werner S, Mikkelsen C, Van Tiggelen D, Vanden Berghe L, Cerulli G. Clinical classification of patellofemoral pain syndrome: guidelines for non-operative treatment. Knee Surg Sports Traumatol Arthrosc. 2005;13(2):122–30.
37. Loudon JK, Gajewski B, Goist-Foley HL, Loudon KL. The Effectiveness of Exercise in Treating Patellofemoral-Pain Syndrome. 2004;13(4):323.
38. Baker RL, Fredericson M. Iliotibial band syndrome in runners: Biomechanical Implications and Exercise Interventions. Phys Med Rehabil Clin N Am. 2016;27(1):53–77.
39. Pegrum J, Self A, Hall N. Iliotibial band syndrome. BMJ. 2019;364:l980.
40. Beals C, Flanigan DA. Review of treatments for iliotibial band syndrome in the athletic population. J Sports Med (Hindawi Publ Corp). 2013;2013:367169.
41. Figueroa D, Figueroa F, Calvo R. Patellar tendinopathy: diagnosis and treatment. J Am Acad Orthop Surg. 2016;24(12):e184–e92.
42. Peers KH, Lysens RJ. Patellar tendinopathy in athletes: current diagnostic and therapeutic recommendations. Sports Med. 2005;35(1):71–87.
43. Maffulli N, Wong J, Almekinders LC. Types and epidemiology of tendinopathy. Clin Sports Med. 2003;22(4):675–92.
44. Piedade SR, Mendes LA, Carvalho LM. dos Santos RMF, Marques LC, Ferreira DM. Shin Pain. In: Rocha Piedade S, Imhoff AB, Clatworthy M, Cohen M, Espregueira-Mendes J, editors. The sports medicine physician. Cham: Springer International Publishing; 2019. p. 211–21.
45. Batt ME, Ugalde V, Anderson MW, Shelton DK. A prospective controlled study of diagnostic imaging for acute shin splints. Med Sci Sports Exerc. 1998;30(11)
46. Thacker SB, Gilchrist J, Stroup DF, Kimsey CD. The prevention of shin splints in sports: a systematic review of literature. Med Sci Sports Exerc. 2002;34(1):32.
47. Lieberman DE, Venkadesan M, Werbel WA, Daoud AI, D'Andrea S, Davis IS, et al. Foot strike patterns and collision forces in habitually barefoot versus shod runners. Nature. 2010;463(7280):531.
48. Brennan FH Jr. Managing blisters in competitive athletes. Curr Sports Med Rep. 2002;1(6):319–22.

25 Shooting Sports and Archery

K. C. Ravella and Mark R. Hutchinson

25.1 Introduction

Shooting sports represent unique sporting events with a wide variety of participants and injury patterns. According to the National Shooting Sports Foundation, more than 50 million people participate in shooting sports annually, an estimate that includes participation in archery competition. Furthermore, annual reports from the past two decades reveal a steady increase in shooting sport popularity among males, females, and individuals of all ages, with overall participation up nearly 30% since the start of the new millennium [1]. Literature on injuries that athletes incur during shooting sports and archery remains sparse. Nonetheless, focus on athlete wellness and injury prevention is critical, especially as shooting sport participation continues to rise. In this chapter, we will focus on three main shooting disciplines as well as archery. The three main shooting disciplines involve shooting at stationary targets with a rifle, shooting at stationary targets with a pistol, and a shotgun discipline during which the competitor hits moving targets [2, 3]. These three shooting disciplines, in addition to an archery discipline, represent the main shooting sports that were originally scheduled to be held at the 2020 Tokyo Olympics prior to its cancellation due to the COVID-19 pandemic [2–4].

The most commonly sustained orthopedic injuries during shooting sports involve the shoulder, hand, and wrist, with a minority of injuries involving the lower back and lower extremity. We will also discuss penetrating trauma and other injuries. The purpose of this chapter is to outline the unique mechanics and injury risk factors associated with shooting sports, discuss the most commonly sustained orthopedic injury patterns within the sports including their epidemiology, and offer injury-specific prevention strategies.

25.2 Mechanics and Injury Risk Factors

The injury mechanisms found within shooting sports differ significantly from other sports due to the unique nature of the sporting events. Although the disciplines are non-contact, shooting sport athletes require intense concentration and often engage in rapid-fire movements, rendering them susceptible to distinctive injury patterns. In addition, athletes are required to maintain this intense concentration in isolated positions for long periods of time. These athletic feats demand sustained isometric contraction, defined as contraction during which muscle length does not change [5]. Unremitting isometric contraction can lead to muscle spasms, soreness, and fatigue, predispos-

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ing the athletes to further injury. Furthermore, athletes typically remain on either their right or left sides during the entirety of competition. As emphasized earlier, spending lengthy periods of time in one position can predispose these athletes to painful muscle and joint problems.

Another important factor to consider is the recoil that athletes experience while firing their weapons during competition. This is especially relevant in recreational shooters, who may lack the upper body strength that would otherwise offer a protective effect against weapon recoil [6]. Recoil is a reactive backward force felt by a shooter once he or she fires a weapon. At the crux of recoil is Newton's Third Law of Motion, which states that every action necessitates an equal and opposite reaction. Several studies have examined the effects of gun recoil on both professional and recreational shooters [7–11]. In fact, other than gunshot wounds, gun recoil injuries make up the largest group of non-fatal firearm related injuries nationally at 43% [8]. These injuries include both acute injuries such as lacerations to the head, face, or eyes from gun kickback, as well as more chronic injuries such as nerve palsies or tendon problems. Pain and soft tissue injuries have also commonly been reported. Wanamaker et al. cite numerous examples of gun recoil injuries, including a case in which an athlete's right shoulder pain and weakness persisted for multiple years. EMG findings in this athlete were abnormal, and the postulated mechanism of injury was recoil causing clavicle retraction, potentially impinging on the upper trunk of the brachial plexus causing neuropathy [9].

25.3 Categories of Shooting Sports and Archery

Shooting sports have evolved dramatically since the first World Shooting Championships were held in 1897, and shooting events have been a part of nearly every Olympic Games since the first was held in 1896. For the purposes of this chapter, we will include an in-depth discussion regarding the most popular Olympic shooting sports including the rifle and pistol shooting sports involving athletes shooting at stationary targets, shotgun sport involving athletes aiming at moving targets, and archery—where athletes aim their arrows at the center of a target.

The rifle shooting sport includes 50 m rifle with three positions—athletes shoot at targets from kneeling, prone, and standing positions. In 10 m air rifle, athletes shoot at a target exactly 10 m away using air guns. The pistol shooting sport includes 10 m air pistol, where athletes use one hand to shoot at a target 10 m away. In 25 m rapid-fire pistol, athletes fire consecutive shots over periods of 8, 6, and 4 seconds—testing speed. In the 25 m pistol women's event, athletes shoot at a target 25 m away while standing. Finally, the shotgun shooting sport spans five separate trap events, where clay targets are launched into the distance from a single trap—and skeet, where clay targets are released from multiple right and left-sided traps. In archery, there are individual and team events at the Olympics. Athletes shoot arrows at a target located exactly 70 m away. The target itself is exactly 12 cm in diameter.

In addition, shooting sports have been held at every Paralympic games since 1976 and continue to grow in popularity. Paralympic shooting sport participation has grown from 39 participants from 10 countries in 1976 to 147 participants from 42 countries in 2016 [12]. There are multiple shooting Para sport events—athletes may shoot from distances of 10 m, 25 m, and 50 m, in individual men's and women's as well as mixed competition disciplines. Similar to shooting events of the Olympic Games, Para shooting sports are meant to test shooting skill and accuracy as athletes aim their shots as close to the center "bullseyes" of targets as possible at specific distances. Appropriately, there are three main categories of Paralympic shooting which allows athletes from different disability classes to fairly compete. The SH1 category encompasses Para athletes who do not necessitate the use of a shooting stand. The SH2 category encompasses Para athletes who are not able to support the weight of a firearm, and thus require shooting stands to compete. The SH3 category encompasses Para athletes with visual impairment. Broadly, Paralympic shooting competition allows male and female athletes with physical disabilities to compete against one another. With respect to sports injuries, risks in Para shooting sport athletes are closely related to the athletes' physical limitations, which may or may not involve body trunk balance, muscle strength, and upper and lower limb functionality [13].

Archery has also been a popular sport in the Paralympic games—it has been a medal sport since the first Paralympic Games were held in 1960. The rules of the archery discipline in the Paralympic Games are nearly identical to that of the Olympic Games. Para athletes aim their 72 arrows at a 122-centimeter target, from exactly 70 meters away. Similar to shooting sports, there are different Para archery competition classifications, including open, W1, and three visually impaired categories. The open classification encompasses athletes who may use a wheelchair due to lower extremity or balance impairment, or shoot standing if they wish. The W1 category encompasses athletes who require the use of a wheelchair. The V1 visually impaired category encompasses athletes who necessitate the use of blindfolds or black-out glasses during competition. The V2/V3 categories encompass athletes who use senses other than sight during competition. These athletes may or may not choose to utilize an assistant to convey information about the targets. The V2/V3 categories are the only mentioned categories not currently held at the Paralympic games [14].

25.4 Epidemiology and Injury Patterns

Literature regarding epidemiology and injury patterns sustained during recreational, training, and competition-level shooting sports is sparse. Relatedly, shooting sports offer a significantly lower risk of injury compared to other sports. Injury patterns vary significantly. Types of injuries reported during shooting sports include strains, muscle tears, tendinitis, sprains, and rarely—fractures, dislocations, and contusions [15] (Fig. 25.1). Kabak et al. performed a comprehensive analysis on 729 shooting athletes during the 2010–2011 Turkish Shooting Sports competi-

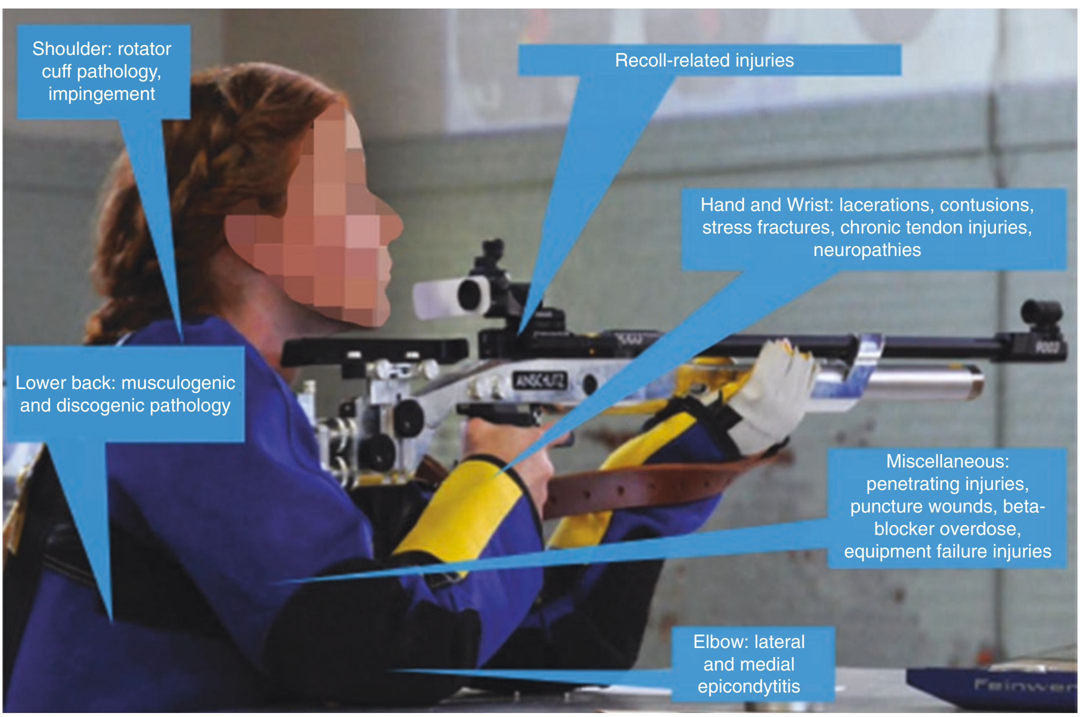

Fig. 25.1 Common sites of musculoskeletal injuries in shooting sports. (Source: Pikist)

tion period. They compared injuries that occurred during competition to injuries that occurred during training leading up to competition, and found that dislocations, strains, muscle tears, tendinitis, and impingement pathologies occurred more commonly during training. In contrast, sprains, ligament ruptures, and lower back pathology occurred more commonly during competition. The majority of injuries were observed in the shoulder region, followed by hand & wrist, foot & ankle, calf & thigh, and finally the lower back region. As mentioned earlier, shooting athletes maintain isolated positions for prolonged periods of time, subjecting their muscles and tendons to unique yet significant stresses [16–19]. Shooting requires a great deal of mental and physical fortitude, as biomechanical studies performed on shooting athletes have demonstrated that even the slightest movement deviations will affect shooting accuracy [16].

Similar to gun and rifle sports, archery confers a relatively low risk of injury in comparison to other sports. The majority of acute injuries in archers are soft tissue injuries including contusions and lacerations (Fig. 25.2). Conversely, chronic injuries have a similar distribution to those seen in gun and rifle sports, with the majority involving the shoulder, elbow, hand & wrist, and lower back. Nearly half of injuries in these athletes involve the shoulder. Wrist injuries make up 12% of archery injuries, followed by finger injuries at 6%, back injuries at 5%, and forearm injuries at 4% [20]. In the sections to follow, this chapter will examine the abovementioned injury patterns in both shooting sports and archery, based on the most commonly involved anatomic locations in each sport.

25.5 Most Common Injuries by Anatomic Location

25.5.1 Shoulder

Shoulder injuries are by far the most common type of injury seen in shooting sports, with the shoulder

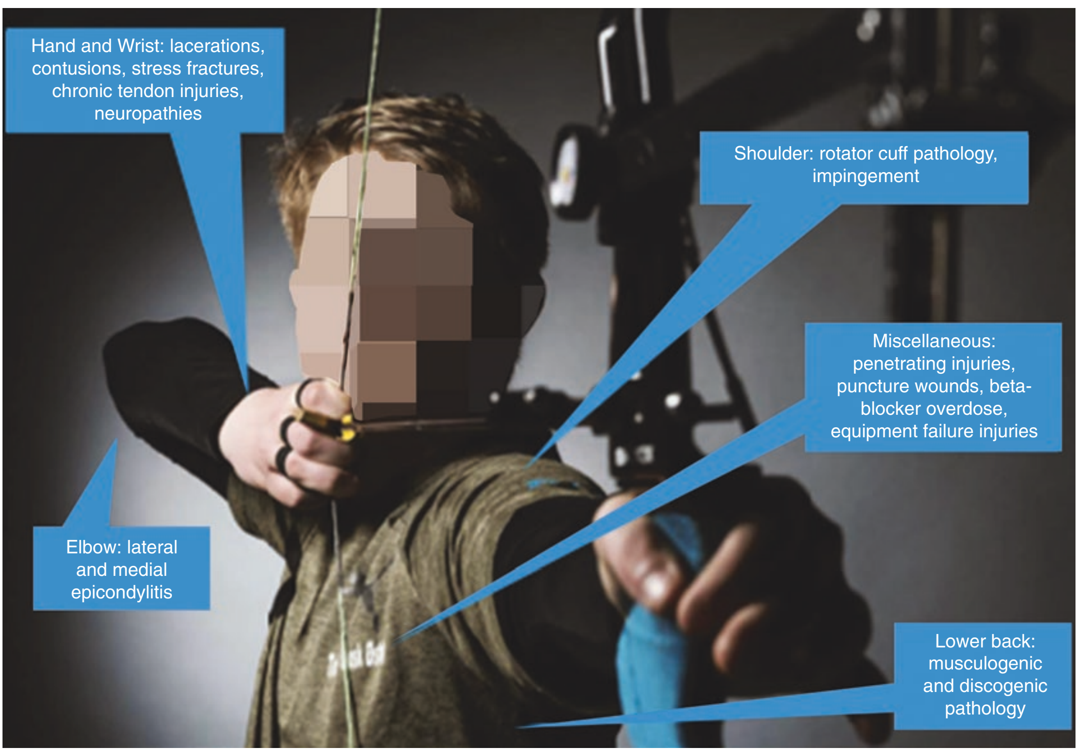

Fig. 25.2 Commons sites of musculoskeletal injuries in archery. (Source: Pixabay)

involved in nearly 50% of shooting injuries [15]. As discussed earlier, recoil plays a large role in the mechanism of shoulder injury in shooters. In 2004, the United States Army together with Walter Reed Army Medical Center in Washington, D.C., and the Army Research Laboratory in Aberdeen, MD, conducted a study on infantrymen in order to assess the injury risks associated with weapon recoil [7]. Although this study was not specifically performed on shooting athletes, many of the findings can be applied as both groups of individuals experience the effects of recoil during weapon firing. Out of the 15 volunteers in the study, all of them showed evidence of bruising over the anterior shoulder, and all but four reported pain after firing. All but one infantryman had abnormal MRI soft tissue findings in the form of muscle edema. Although the aforementioned study only documented findings up to 96 hours post-shooting, they did find that shooting and recoil had a cumulative effect on shooters' subjective reports of pain and recoil intensity. Thus, recoil can be dangerous to both the recreational, occasional shooter as well as the trained, competitive shooter in the form of acute kickback injuries and aggregate chronic stresses, respectively. Protective measures against recoil have been discussed. Heavier weapons may actually prevent larger counterforces during the act of shooting. Padding shooting vests can help to prevent anterior shoulder bruising and recoil-related lacerations. Finally, the importance of proper technique cannot be understated, especially for recreational shooters. It should be mentioned that shooting athletes are fortunate to fire their weapons on flat surfaces during competition in comparison to infantrymen and military soldiers, who are often forced to fire their weapons on uneven ground or in imbalanced positions. Both recreational and competitive shooting athletes should focus on maintaining proper posture and technique during the act of shooting in order to mitigate the risk of shoulder injury.

Relatedly, the most common injuries in archers involve rotator cuff pathology related to repetitive activity during shooting [21, 22]. In recreational athletes, injury may be related to poor shooting techniques in the form erratic shooting motion, or using equipment that is not well fitting (i.e., large or heavy bows). Shoulder kinematics during shooting may also play a large role in injury prevention. In fact, studies have shown that injured archers possess alterations in shoulder kinematics and muscle activity when compared to uninjured peers [23]. Shinohara et al. discuss that loading of the shoulder girdle can put up to 42 pounds of stress on the joint. Over time, repetitive loads of this magnitude can lead to shoulder pain and rotator cuff dysfunction. In addition, the authors found that external rotation and posterior tilt of the scapula measured lower in the group of archers who experienced impingement; moving forward, shoulder injury prevention may center on muscle strengthening exercises that focus on the rotator cuff musculature and deltoid and efforts to alter or increase the abovementioned angles. Treatment plans center on rest and NSAIDs initially. Physical therapy exercises centered on rotator cuff strengthening, and education also play a role in initial conservative treatment, in addition to subacromial corticosteroid injections. Surgery is reserved for those who fail non-operative management, and generally consists of arthroscopic or mini-open rotator cuff repair versus tendon transfer or reverse total shoulder arthroplasty for massive cuff tears.

Elbow injuries are also quite common in archers. It rivals the shoulder in terms of magnitude of repetitive stresses that the joint experiences during shooting activity [21]. Lateral and medial epicondylitis represent the most common elbow pathologies in archers, and the archers sustaining these injuries are typically treated using the standard regimen of conservative treatment at rest initially, followed by surgical management for those injuries that are refractory. In what appears to be a recurring theme involving injury patterns affecting other joints in shooting athletes and archers, knowledge and prevention of these elbow injuries in the form of strengthening of the surrounding musculature is key.

25.5.2 Hand-Wrist

Hand and wrist injuries are also commonly seen in shooting sport athletes, with reported rates of about 10% in literature [15]. Acutely, lacerations and contusions can be seen. Rarely, stress fractures have been reported [24]. Chronically, carpal tunnel syndrome and can be seen due to the repetitive nature of the sport and isolated stresses placed on the hand and wrist. Rest is important in protecting against chronic tendon injuries or neuropathies. Spending time away from shooting in between competitions or training sessions will allow the shooters to adequately recuperate and protect against overuse. Protective gear has been discussed in protecting other areas of the body during shooting, and again is seen to have a benefit for the hand and wrist. Special grips and padding can help to offset the kickback effects of gun recoil and protect against bruising or lacerations.

Similarly, due to the nature of the sport involving fine control of the bow and arrow, hand and finger injuries represent a significant proportion in archers, especially recreational athletes. The majority of these injuries are minor, such as abrasions on hand or wrist from the bowstring after the arrow is released. Injury patterns range from lacerations and contusions to chronic overuse tendon injuries. Acutely, lacerations and contusions are more common. Literature has discussed the incidence of both superficial skin lacerations as well as deeper lacerations including injuries to digital nerves and arteries. Rarely, finger metacarpal fractures have also been reported to occur to athletes Chronically, conditions such as de Quervain's tenosynovitis and compressive neuropathies involving the median nerve are widely seen, in addition to extensor tendon tenosynovitis. The suspected mechanism through which these conditions develop appears to be related to persistent stresses placed on individual tendons and soft tissue structures, which over time undergo structural changes—the affected structures may ultimately fatigue or break down, causing pain and discomfort in athletes. Exact prevalence of these injuries vary widely in the literature, but overall acute injuries tend to be more common in recreational archers, in contrast to chronic overuse injuries and syndromes being more common in competitive archers [25–27]. Acute injuries such as lacerations and contusions are typically treated conservatively in the form of either laceration repair or rest and ice for contusions. Chronic tendon injuries and neuropathies are also treated conservatively initially in the standard fashion, with either rest and immobilization or occasionally corticosteroid injections. However, operative treatment may be considered in the event that the athlete fails conservative management.

In terms of prevention for hand and wrist injuries specifically, protective gear such as wrist guards or gloves appears to offer some benefit, especially in prevention of the majority of minor abrasions discussed earlier. Lightweight bows have also been supported, with the idea that commonly utilized tendons and muscles would receive less loading during training and competition. Authors have also advocated for archery education programs to minimize the risk of injury in recreational archers. These programs could involve instruction on proper shooting form in addition to tailored strengthening programs that focus on the muscles, tendons, and joints that are most commonly put under stress during archery [27]. Proper stretching and preparation in the form of pre-participation strengthening is recommended.

25.5.3 Lower Back

Archers frequently orient their bodies in positions that exert considerable stresses on the lower back. Accordingly, acute and chronic injuries in archers can be seen in this area. These injuries range from intervertebral disk injuries to chronic degenerative pathologies of the lower back. Singh et al. explain that in a single day of international level competition, male archers will fire their 45-pound bow and arrow weapons roughly 75 times, which amounts to the male athlete pulling about 3400 pounds. The female athlete will pull about 2625 pounds using their 35-pound bows [21]. Over time, this repetitive strain can cause musculogenic pain, discogenic pain, and mechanical wear-and-tear of the lower back. Treatment of chronic low back pain in archers is largely dictated by the root cause of pain. Radiographs or advanced imaging such as MRI may be ordered to further guide treatment plans. Injury prevention in the form of education is critical, specifically regarding proper technique and posture. Improper shooting form may lead to an imbalanced distribution of weight throughout the lower back, further contributing to pain and injury. Treatment of acute pathologies such as disc herniations initially involves rest and physical therapy in addition to anti-inflammatory medications. While the majority of these patients improve with conservative management, those who do not may benefit from operative intervention in the form of discectomy.

25.5.4 Lower Extremity

Lower extremity injuries are infrequent in shooting athletes and archers but have been reported in literature [15, 28]. They fall far behind upper extremity and lower back injuries in terms of overall prevalence but should be mentioned nonetheless. Kabak et al. discuss that the majority of lower extremity injuries that occur during training involve the calf and thigh, while the majority of injuries that occur during competition involve the foot and ankle or the knee. Chronic injuries are often related to the fact that shooting athletes are often required to maintain standing positions for prolonged periods of time, placing increased stress on the lower extremity joints and tendons which are maintained in rigid, immobile positions. Furthermore, the lower extremities can fall victim to strains and sprains with improper stretching and warm-up sessions, poor shooting technique, and other modifiable factors. This is especially true of the calf and thigh area, where muscle tears and sprains were found to be the most common injury pattern. Authors have attributed this to inadequate stretching routines coupled with lengthy training and competition sessions with little rest in between [15]. Acutely, ankle sprains appear to be the most common lower extremity pathology—especially during competition when the limits of physical and mental fortitude are pushed. Interestingly, some shooting athletes are more prone to ankle sprains and lower extremity muscle strains based on their anatomy [28]. Flatfoot deformity is a common condition that predisposes shooting athletes to acute ankle sprains. Although lower extremity injuries are less common given the upper body-focused nature of these sports, preventative education can have a positive effect on the athlete as a whole. Placing an emphasis on flexibility is critical—flexible joints, muscles, and tendons help to offer a protective effect against acute injuries. Furthermore, knowledge on proper training and resting between training sessions and competitions may help to avoid chronic stresses on the body that can predispose to overuse injuries.

25.5.5 Penetrating and Other Injuries

Lastly, we will discuss penetrating and other less commonly sustained injuries during shooting sports and archery. Penetrating injuries occur rarely in competition due to athlete experience and competition safety guidelines. In recreational archers, there have been reports of puncture wounds and instances where athletes accidentally fall forward onto arrows or bows [27]. Gunshot wounds during shooting sport competition is similarly rare, but these injuries understandably need to be taken seriously. Prompt emergency department management is critical.

The use of beta-blockers in shooting sports should also be briefly discussed. Beta-blockers are drugs that block norepinephrine and epinephrine, two naturally occurring chemicals in the body that are involved in increasing heart rate and other physiological fight or flight characteristics. By blocking these chemicals, beta-blockers reduce the effects of anxiety and work to improve concentration in competitors. As mentioned earlier, shooting sports and archery require an immense amount of focus, deliberation, and both physical and mental fortitude. Beta-blocker use has been reported in Olympic shooters and archers but are prohibited by the International Shooting Sport Federation and World Archery

Federation as performance-enhancing drugs [29]. Given its illegality in competition, beta-blocker use in archers and shooting athletes is rare. However, beta-blocker toxicity has been well-studied in literature [30, 31]. Toxicity can manifest in the form of hypotension, bradycardia, and even CNS depression. In severe cases toxicity can be life threatening. Treatment focuses on promptly addressing the symptoms of beta-blocker toxicity—such as protecting the airway in obtunded patients or administering bronchodilators to treat bronchospasm [30].

Interestingly, injuries have also been reported to occur due to equipment failure [32]. Therefore, in addition to abovementioned archery education and pre-participation strengthening programs, proper maintenance of sports equipment holds great importance.

As mentioned earlier, Para shooting and archery has risen in popularity over the past few decades and remains a staple at the Paralympic Games. Similar to their Olympic sport counterparts, para shooting and para archery are among the safest Paralympic sports, and overall injury patterns are similar to those seen in both recreational and Olympic shooters and archers. Literature on injuries associated with Paralympic archery is scarce, but authors have discussed similar incidences of injuries in various anatomic locations in comparison to non-paralympic athletes, with shoulder and other upper extremity pathologies being the most common [33]. Chronic rotator cuff tendinitis and acute injuries such as bowstring-induced lacerations to the hand, wrist, and fingers are widespread. Treatment options also follow a similar framework to those outlined in non-paralympic athletes, with conservative management in the form of physical therapy and over-the-counter anti-inflammatory medications recommended initially, followed by surgical management for those who fail conservative management. Prevention is critical in Paralympic athletes as well; protective equipment and proper posturing and shooting form can significantly diminish the risk of injury.

Conclusion

Shooting sports are rising in popularity and involve participants of all ages, both recreational and competitive. Both shooting sports and archery events have been held regularly at the Olympic and Paralympic level. These sports involve intense concentration—at the competitive level especially, even the smallest of movements can affect outcome and success in dramatic fashion. Although these sports involve a significantly lower injury risk compared to contact sports such as football, basketball, soccer, and others—the unique nature of the sports predispose these athletes to distinctive injury patterns. Chronic shoulder rotator cuff pathology or impingement syndromes are by far the most common injuries seen in these athletes, with tendon and neuropathic injuries in the hand and wrist close behind. In shooting athletes, weapon recoil plays a large role in mechanism of injury and has been a focus in shooting-specific injury literature. Prevention in the form of cushioned or padded equipment has been evaluated in lessening the damaging effects of recoil [8]. Lower back and lower extremity injuries are also seen and predominantly relate to the rigid posture that athletes are forced to maintain for lengthy periods of time. Injury prevention primarily consists of pre-participation education, flexibility and stretching programs, and adequate rest between participation. Finally, pathologies such as penetrating trauma or beta-blocker toxicity are seen in shooting athletes and archery, and management of these unique injuries should be understood by physicians or athletes involved in training and competition. Overall, injury literature involving shooting sports and archery is sparse; further work is needed to help understand the preventability of these injuries and mitigate their risks.

References

1. Allen T, Olds E, Southwick R, Scuderi B, Howlett D, Caputo L. An economic force for conservation. NC: Huntersville; 2018.
2. Shooting. https://tokyo2020.org/en/sports/shooting/. Accessed 10 Jun 2020.
3. ISSF - International Shooting Sport Federation - issf--sports.org. https://www.issf-sports.org/competitions/venue/information.ashx?cshipid=2626. Accessed 10 Jun 2020.
4. Archery. https://tokyo2020.org/en/sports/archery/. Accessed 10 Jun 2020.
5. Faulkner JA. Terminology for contractions of muscles during shortening, while isometric, and during lengthening. J Appl Physiol. 2003;95:455–9.
6. Wilson-Cohen T Recreational shooting injuries with women | greater Alexandria, VA Patch https://patch.com/virginia/greateralexandria/recreational-shooting-injuries-with-women_0317de7a. Accessed 18 Jun 2020.
7. Blankenship, Evans, Allison, Murphy, Isome, Dinauer, Harper. Shoulder-fired weapons with high recoil energy: Quantifying injury and shooting performance. Tech Rep. 2004;63
8. Hootman JM, Annest JL, Mercy JA, Ryan GW, Hargarten SW. National estimates of non-fatal firearm related injuries other than gunshot wounds. Inj Prev. 2000;6:268–74.
9. Wanamaker WM. Firearm recoil palsy. Arch Neurol. 1974;31:208–9.
10. Morelli F, Neugebauer JM, Haynes CA, Fry TC, Ortega SV, Struve DJ, LaFiandra ME, Larkin GB. Shooter-system performance variability as a function of recoil dynamics. Hum Factors. 2017;59:973–85.
11. Shyu WC, Lin JC, Chang MK, Tsao WL. Compressive radial nerve palsy induced by military shooting training: clinical and electrophysiological study. J Neurol Neurosurg Psychiatry. 1993;56:890–3.
12. Shooting Para Sport - About the Sport | International Paralympic Committee. https://www.paralympic.org/shooting/about. Accessed 14 Jun 2020.
13. Paralympic Shooting | USA Shooting. https://www.usashooting.org/about/paralympicshooting. Accessed 14 Jun 2020.
14. Para Archery | World Archery. https://worldarchery.org/Para-Archery. Accessed 14 Jun 2020.
15. Kabak B, Karanfilci M, Ersöz T, Kabak M. Analysis of sports injuries related with shooting. J Sports Med Phys Fitness. 2016;56:737–43.
16. Zatsiorsky VM, Aktov AV. Biomechanics of highly precise movements: the aiming process in air rifle shooting. J Biomech. 1990;23:35–41.
17. Sattlecker G, Buchecker M, Müller E, Lindinger SJ. Postural balance and rifle stability during standing shooting on an indoor gun range without physical stress in different groups of biathletes. Int J Sport Sci Coach. 2014;9:171–83.
18. Sadowska D, Krzepota J, Klusiewicz A. Postural balance and rifle stability in a standing shooting position after specific physical effort in biathletes. J Sports Sci. 2019;37:1892–8.
19. Era P, Konttinen N, Mehto P, Saarela P, Lyytinen H. Postural stability and skilled performance-a study on top-level and naive rifle shooters. J Biomech. 1996;29:301–6.
20. Sports injury in archery. https://www.qsl.net/gi4fum/page5.html. Accessed 18 Jun 2020.
21. Singh A, Lhee S-H. Injuries in archers. Saudi J Sport Med. 2016;16:168.
22. Fukuda H, Neer CS. Archer's shoulder. Recurrent posterior subluxation and dislocation of the shoulder in two archers. Orthopedics. 1988;11:171–4.
23. Shinohara H, Urabe Y, Maeda N, Xie D, Sasadai J, Fujii E. Does shoulder impingement syndrome affect the shoulder kinematics and associated muscle activity in archers? J Sports Med Phys Fitness. 2014;54:772–9.
24. Wu J-L, Huang G-S, Wu C-C, Lee C-H, Wu S-S. Stress fracture of the ulna occurring in military recruits after rifle drill training. Mil Med. 2004;169:839–41.
25. Rayan GM. Archery-related injuries of the hand, forearm, and elbow. South Med J. 1992;85:961–4.
26. Kaymak B, Özçakar L, Ertan H, Karabulut I, Akinci A. Sonographic assessment of finger flexor tendons in Olympic archers. Turkiye Fiz Tip ve Rehabil Derg. 2012;58:85–7.
27. S.E. P (2012) Epidemiology of recreational archery injuries: implications for archery ranges and injury prevention. J Sports Med Phys Fitness 52:293–299.
28. Arnot C Postural Considerations for Rifle and Pistol Shooters. Accessed 18 Jun 2020.
29. Lally JM Bull's eye on doping the ISSF IPOD Accessed 18 Jun 2020.
30. Khalid MM, Hamilton RJ (2018) Beta-Blocker Toxicity.
31. Reith DM, Dawson AH, Whyte IM, Buckley NA, Sayer GP. Relative toxicity of beta blockers in overdose. J Toxicol - Clin Toxicol. 1996;34:273–8.
32. Niestroj CK, Schöffl V, Küpper T. Acute and overuse injuries in elite archers. J Sports Med Phys Fitness. 2018;58:1063–70.
33. Chen Y-T, Mordus D. Shooting sports (archery, air rifle, trapshooting). In: Adapt. Sport. Med; 2018. p. 313–22.

26 Skating, Speed Skating, Figure Skating

Peter Gerbino

Gliding on ice as a means of transportation has been used for millennia [1] and can be found described in ancient Nordic myths [2]. Over the centuries it has grown to become several distinctive sports and activities. Ice skating activities include recreational skating, speed skating, figure skating, and ice hockey. When ice was not available, skaters affixed wheels to their boots and created roller skates. That has evolved and there are several sports that use roller skates.

Injuries that occur on skates have to do not only with the activity of skating itself, but also with the specific needs of the particular sport. In the United States in 2018 there were 76,736 skating injuries requiring an Emergency Department visit. This number excluded skating injuries from inline skating [3]. Within each type of skating activity there are different types of ice skates or roller skates, different activities and intensity of activity, different skills, different levels of ability, different body types, and additional different risk factors. All of these factors must be considered when trying to understand, manage, and prevent injuries in these various athletes. For each sporting activity, it is necessary to understand how the sport is played, the specific mechanics of the sport, injury risks and injury patterns for that sport, prevalence and incidence of injuries in that sport, and prevention strategies that have been successful.

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26.1 Skating Sports and Activities

The most common form of skating is recreational ice skating. This is where novice to accomplished ice skaters periodically skate on rinks, ponds, or rivers for enjoyment. In terms of injuries, this group can be subdivided into inexperienced skaters, young skaters, and older skaters. Among the skating sports, speed skating is where skaters race against each other and the clock. It is divided into long track and short track skating. The athletic skills and risks of these two types of speed skating are very different from each other. The sport of figure skating is another very different type of skating. It can be divided into four separate activities. There are singles skaters, pairs skaters, dancers, and synchronized skaters. Each activity is different with different needs and injuries. Beyond competitive figure skating, there is also adult figure skating and exhibition skating. The roller skaters are either inline or quad skaters and recreational, hockey or roller derby skaters. While ice hockey is a major sport with skating, it will not be discussed in this chapter, but rather as its own separate entity with a distinct chapter.

When discussing any issue regarding skating, it is necessary to utilize the immense experience and knowledge base of the International Skating Union

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(ISU). The ISU regulates all activities of both speed skating and figure skating worldwide. The organization was founded in 1892 and has worked with figure and speed skating groups to promote, organize, and improve the sports. Through ISU Communications and Special Regulations & Rules, the organization has improved the safety of the skaters and decreased injury. As a recent example, the ISU has mandated that all participants in speed and figure skating, including coaches and volunteers, are SafeSport certified. The U.S. Center for SafeSport is non-profit United States organization whose mission is to prevent physical and psychological injury to athletes from physical, emotional, and sexual abuse. It is part of Safe Sport International. Certification involves taking and passing an online course covering preventing abuse of athletes [4].

In reviewing the literature on skating injuries, it is interesting to note that figure skating has by far the largest number of publications regarding mechanics and injuries in the sport. In fact for figure skating, there is now an entire textbook on the science of figure skating [5]. The next greatest number of publications are for short and long track speed skating. By comparison, the other types of skating have few publications in the scientific literature. PubMed, Google Scholar, and the Cochrane databases were searched for articles on skating, skating injuries, skating technique, roller skating, and rollerblading.

Fact Box
- Skating is one of the oldest sports in the world.
- Relatively few scientific studies have been published on skating injuries.
- The ISU and Safe Sport International are involved in promoting safe skating.

26.2 Recreational Ice Skating

When one thinks of skating, the high-profile skating sports of figure skating and speed skating come to mind rather than recreational skating. In terms of numbers, however, millions more participate in recreational skating than in the high-profile sports. Athar et al. have stated that 4% of the UK population participates in ice skating each year [6]. That is before considering Scandinavia, Europe, Russia, former Soviet countries, Asia, Canada, and the United States. Recreational skating includes first-time skaters, seasonal skaters, and those with access to skating rinks who enjoy it routinely year round. The skates used by recreational skaters vary widely. They can be high-quality figure or hockey skates, but more often are poor quality rental skates with poor support (Fig. 26.1). As in most sports, recreational skaters who are skilled are only injured in occasional falls and from overuse if they increase their activity level too quickly. Inexperienced skaters are typically injured from falls. These falls result in fractures, contusions, lacerations, and concussions. Barr et al. found that 80% of injuries to inexperienced skaters were fractures to the upper extremities, almost always distal radius fractures. There were occasional ankle fractures, concussions, and lacerations. They recommended that all novice skaters be required to wear wrist guards [7]. They and others have added that novice skaters should be taught how to fall safely [6, 7].

Recreational skaters over age 50 have been found to be at increased risk for severe head injury with falls [8]. Roller skating is a bit different. A descriptive epidemiologic study has reported that inline roller skating has the highest

Fig. 26.1 Typical recreational or rental skate with limited support due to excessive wear. Reproduced with permission from Derek Marsano

odds ratio in any sport of sustaining a fracture at 6.03, where most injuries are to the fingers, wrist, and forearm. Ice skating fractures had an odds ratio of 2.82, also mostly to the upper extremities [9]. Several authors have claimed that up to 80% of traumatic injuries in both experienced and inexperienced recreational skaters could be prevented by use of protective gear such as wrist guards and helmets [8, 10, 11], but whether this is true or not has not been rigorously studied.

26.3 Short Track Speed Skating

Short track speed skating is an Olympic team sport where individual skaters race each other in a pack of 4–6 skaters. The track length is 111.111 meters, and the rink is the same rink used by figure skaters and international hockey teams. The low-cut skate boots have flexible ankles and have 16–18 inches long, very sharp, fixed blades that are set at an angle to offset the extreme leaning to the left as the skaters race counterclockwise (Fig. 26.2). Skaters are skating very close together and reach speeds in excess of 50 km/hr [12]. This leads to collisions and falls which account for the majority of injuries. In 2003 Quinn et al. published a retrospective review of short track injuries. Most injuries occurred to the knee, ankle, spine, leg, and groin. They found that injury patterns were slightly different between practice and competition. In competition, the greatest number of injuries were shoulder dislocations and separations. This was followed by groin strains, concussions, and knee contusions. In practice, the greatest number of injuries were groin strains and knee contusions. Off-ice injuries (using inline roller skates) were ankle sprains, groin strains, hand and wrist fractures, and knee pain. Their group has postulated that extrinsic risk factors for injury include lack of cut-resistant suit material, poor ice quality, lack of safety equipment worn, increased training time, increased number of skaters on the ice, greater number of skaters falling in a collision, and lack of adequate padding material over the boards [12]. Prevalence and incidence values for injuries in this sport have not been published.

Fig. 26.2 Short track speed skate. Sergey Ryzhov/Shutterstock.com

Most of the scientific research in all skating sports has been centered on improving performance. Most of the hypotheses concerning injury prevention have been expert opinion based on the mechanisms of injury. For speed skating, the ISU has implemented the following regulations. All competitors must wear a cut-resistant suit, knee and shin guards, cut-resistant gloves, cut-resistant neck and ankle protection, and an approved helmet. In addition, the front and rear parts of the skate blade must be rounded off with a radius of 1cm [13]. These rules are to decrease lacerations, fractures, and knee injuries. The rules have evolved from empirical observations rather than Level I scientific studies. The boards are padded to absorb energy from falls, and many skaters also wear shatterproof eye protection.

As speed skaters get faster, they have more kinetic energy and, consequently, greater risk of injury. Attempts to recruit skaters with more fast type IIx muscle fibers [14], modify skating position to improve blood flow [15], increase unilateral squat exercises [16], improve mechanics through the skating cycle [17], change pacing behavior [18], and modify push-off angle [19] have all been used to increase speed and performance. Specially instrumented skates and devices have been invented to measure push-off forces, foot pressure and center of pressure, stroke time, knee forces, and speed [19–21]. These devices improve performance and speed leading to increased peak forces in a fall and severity of trauma.

With regard to ankle injuries, the low-cut shoe (Fig. 26.2) may predispose speed skaters to injury. Ankle strengthening exercises and balance training are the typical training and physical therapy interventions to try to minimize ankle injury. Spine and groin injury prevention has been limited to general strength training and flexibility.

26.4 Long Track Speed Skating

The sport of long track speed skating is an Olympic sport where racers skate in pairs a defined distance against the clock. The track is an oval 400 meters long. Race distances are 500, 1500, 5000, and 10,000 meters. In addition to competitive long track events, ice skating marathon and wheeled inline and quad speed skating are similar sports with similar needs and injuries. Long track speed skaters can reach speeds of 59 km/hr. [22, 23]

Skaters can use either fixed-blade or clap skates, but all long track skaters now use clap skates. Clap skates are hinged at the toe so that the heel can rise while the blade remains longer in contact with the ice for greater speed. The blades are 38–45 cm long and about 1.25 mm thick. The edges are kept extremely sharp for maximum bite. The boot is short, leather, and very stiff at the heel (Fig. 26.3). As in short track, the skaters lean dramatically to the left and so the blades are offset to adjust to this angle (Fig. 26.4). The skin suits can be highly sophisticated, comprised of rubber, plastic, Kevlar, Dyneema, and honeycomb stretch with 1.5 mm ceramic dots on the arms for minimal wind resistance [24].

Fig. 26.3 Long track clap skate. Sportpoint/Shutterstock.com

Injuries in long track speed skating are usually overuse injuries. These include low-back strain and muscle strains to the lower extremities. In addition, these athletes sustain patella and Achilles tendinitis, patellofemoral pain, and exertional compartment syndrome [25]. After skating for more than 12 years, speed skaters can develop “skater’s cramp,” a task-specific dystonia with acute, disabling cramping of the calves and feet. It is thought to be caused by specific technique factors that could be modified [26]. Long track skaters do have their share of acute trauma. Complete laceration of the patella tendon in a long track skater has been reported [27]. As in all skaters, ill-fitting boots can cause blisters, corns, and Haglund’s deformity.

Fact Box

- Speed skaters reach incredible speeds.
- The blades are long and sharp causing lacerations.
- High kinetic energy and skating position lead to injuries.

26.5 Figure Skating

United States Figure Skating, the National Governing Body for competitive figure skaters, has 203,023 members, 750 clubs, and 1000 Learn to Skate programs as of 2020 [28]. As previously mentioned, figure skating includes singles, pairs, dance, and synchronized skating. Injury patterns are different at different skill levels. Less skilled figure skaters get more acute injuries like ankle sprains, and more skilled skaters get overuse injuries like stress fractures [29]. Understanding the elements which are needed for a given performance and technical skills needed for those elements is critical to understanding how to care for

Fig. 26.4 Extreme lean in speed skating requiring blades to be angled to achieve optimum efficiency. Coolakov_com/ Shutterstock.com

these athletes [30]. Elite-level figure skating is now so sophisticated that there are different boots and blades for each event within figure skating (Fig. 26.5).

Singles skaters are males and females who skate alone on the ice performing jumps, spins, and other element skills which are judged on degree of difficulty and precision of execution and aesthetics. Their injuries tend to be overuse injuries. Pairs skaters, ice dancers, and synchronized skaters have a partner or several teammates on the ice and in questionnaire studies have been found to get more acute injuries [31–33]. The singles skaters are performing more and more complex jumps, and quadruple rotations have become the new minimum standard at the highest levels. To successfully complete a quadruple (quad) jump, the skater must jump up to 24 inches in the air and spin faster than five revolutions per second. This has been estimated to require generating 150 foot-pounds of torque and 300 pounds of force on take-off. The spinning generates 180 pounds of centrifugal force requiring that much force to hold the arms tight to the body [34]. Landing requires transmitting torque and force through the ankle, knee, hip, and back (Fig. 26.6). Peak landing forces from jumping have been measured as high as almost 10 times body weight [35]. These stresses raise concerns about possible increases in hip labral tears [36].

In pairs skating the female partner can be thrown great distances by the male. This could result in even higher peak forces on landing. In pairs, dance and synchronized skating, the close proximity of skaters leads to lacerations and contusions. In all figure skaters, the most commonly injured areas are the lower extremities and back. Kowalczyk et al. have recently published an extensive review of figure skating injuries. They found 68.9% overuse injuries and 31.1% acute injuries. In females, injuries were 29.6% foot/ankle, 19.3% knee, and 15.8% back. In males, injuries were 25.4% foot/ankle, 16.4% hip, and 14.9% knee. Their data were collected from 2003 to 2017 [37]. During that time period only males were performing quad jumps. It is interesting to speculate if the number of female hip injuries has increased since they have also started doing quad jumps. Recent anecdotal observations have noted an increase in hip labral tears and femoroacetabular impingement in both male and female singles skaters.

Foot and ankle injuries have been found to be most common among all figure skaters and include bursitis, corns, blisters, and Haglund's deformity. Tendinitis of the peroneals, tibialis posterior, Achilles, extensor tendons, and flexor halluces longus were common. Ankle sprains, plantar fasciitis, and syndesmosis sprains were the most common ligament injuries. Metatarsal

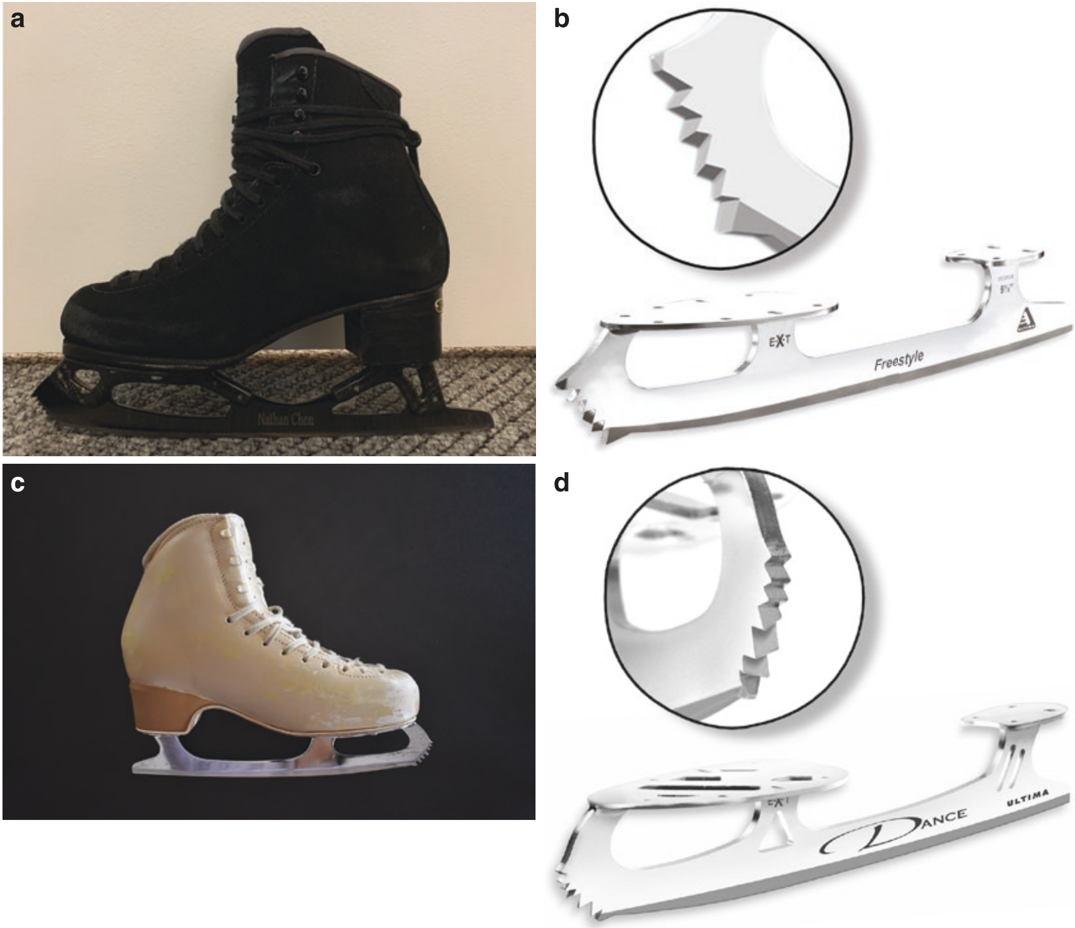

Fig. 26.5 (**a**) Skate used by World Champion men's single figure skater Nathan Chen. Reproduced with permission from Nathan Chen. (**b**) Specialized blade specifically for singles skaters. Reproduced with permission from Jackson Ultima Skates. (**c**) Skate used by World Medalist ice dancer Madison Hubbell. Reproduced with permission from Madison Hubbell. (**d**) Specialized blade specifically for ice dancers. Reproduced with permission from Jackson Ultima Skates

and navicular bone stress reactions and stress fractures were the most common bone injuries in the foot [33, 37]. Retrocalcaneal bursitis and other heel overuse injuries were found to be caused by poor boot fit and design and from excessive training [10, 38]. Lace bite is a specific irritation of the tibialis anterior and toe extensor tendons. It is caused by pressure from the tongue of the boot and treated by protecting the foot dorsum from the laces by various methods [10, 39].

The knee injuries were extensor mechanism problems 70.1% of the time. Most were patellofemoral pain, patellar tendinosis, contusion, and Osgood-Schlatter Syndrome. Hip injuries were not reported in detail. Bone stress injuries were 11.8% of all injuries and of that 42.2% were in the low back, 32.4% in the feet, and 15.7% in the tibia and fibula [33, 37]. Spondylolysis and pars stress reaction are the most common bone stress reactions in the low back, and recent evidence has shown that many athletes with these problems have low Vitamin D3 levels [37, 40]. Some increases in hip muscle strains have been reported [41], but femoroacetabular impingement is the more recent concern. It is known that figure skaters, as well as dancers and gymnasts, require extreme amounts of hip external rotation and that can lead to impingement, instability, or

Fig. 26.6 World Champion Nathan Chen performing quadruple spin. Areas of high stress and potential injury include ankles, knees, hips and back. Courtesy of Nathan Chen. Reproduced with permission from David Carmichael

both [42]. These hip problems are getting treated by hip arthroscopy, but not all are not returning to their previous performance levels [43].

Prevention of injury in figure skaters has been approached in several ways. Adequate training and nutrition, proper supervision, and avoidance of overuse are always appropriate. More specifically, checking Vitamin D level, ferritin level, and thorough physical examination on a yearly basis has helped [44]. Thorough analysis of each aspect of figure skating technique has been undertaken. This should not only improve performance but also decrease injury by minimizing high-risk actions. Such interventions include wearable jump monitors [45, 46], assessment of Y-balance [47], comparing different boot types for kinematics and shock absorption [48, 49], evaluating joint laxity [50], video analysis [51, 52], and thorough analysis of the science of figure skating jumps [34]. It is known that the shear modulus of the semimembranous muscle measured at 60 degrees of knee flexion is lower in figure skaters than controls [53]. Whether any of these metrics can be used to predict or prevent injury is as yet unstudied. Analysis of a skater's anatomy has long been used to modify biomechanics to improve performance and prevent injury [54], but, here again, no scientific analyses have been done.

Fact Box
- Singles skaters get more overuse injuries.
- Pairs, dance, and synchronized skaters get more traumatic injuries.
- Technique analysis may improve performance and decrease injuries.

26.6 Roller Skating

Roller skating includes inline skating and quad wheeled skating. Quad skates (Fig. 26.7a) are used for recreational skating and roller derby. Inline skates (Fig. 26.7b) are used for recreational skating, hockey, and racing. The major difference between roller skating and ice skating is that roller skaters are on dry, hard surfaces rather than ice. Despite the fact that ice is also very hard, it is also very slippery so that in a fall the athlete slides more, absorbing some of the impact with less friction. That is one reason that probably explains the finding of an odds ratio of 6.03 for fracture in inline skating which is three times higher than for ice skating and higher than for any other sport [9]. The difference in surface friction has been linked to types of injury. Ice skaters and roller skaters both fall forward, and both groups attempt to break their falls with their hands, but ice skaters are less successful resulting in almost five times as many head injuries as roller skaters. Roller skaters do get their hands out in the falls resulting in many more upper extremity injuries [51]. Aggressive inline skat-

Fig. 26.7 (**a**) Quad roller skates used in roller derby. Reproduced with permission from Mark Nockleby. (**b**) Inline roller skate. 3DMAVR/Shutterstock.com

ers have been found to average 1.4 injuries per skater per year [55]. The Inline Skating Resource Center has reported a New York Times survey showing that inline skaters will have 3.4 injuries per 1000 skaters. They also found that most injuries occurred outdoors on streets or paths with a surface defect [56]. In 1995 Schieber and Branche-Dorsey published the epidemiology of inline injuries. They found that in 1993 there were 12.6 million inline skaters in the USA who had 31,000 injuries requiring hospital visits. They recommended inline skaters wear wrist guards, knee pads, elbow pads, and helmets [57]. They looked again in 1995 and found 22.5 million skaters and 100,000 emergency room visits; wrist injuries being most common [58]. While inline skating may be less popular in 2020, the recommendations are still appropriate. The United States Center for Disease Control and Prevention (CDC) has studied the nature and body site distribution of inline skating injuries using Schrieber and Branche-Dorsey's data [57]. They found fractures, dislocations, sprains, strains, and avulsions constituted 63% of all serious injuries, most of which were to the wrist and forearm [59].

Quad roller skating shares all the same risk factors as inline skating [60]. A recreational quad skater has the same risk of falling on broken pavement as an inline skater. These skaters should be viewed as part of the inline population and given the same recommendations. Roller derby participants are a bit different. Women's flat track roller derby is a world-wide growing sport with over 50,000 participants in the U.S. alone [61]. The skaters wear ankle-high quad skates (Fig. 26.7a) and race around a track trying to have their "jammer" pass the other team's blockers while blocking that team's jammer. This leads to constant physical contact and falling (Fig. 26.8). While wrist guards, knee pads, elbow pads, and helmets are mandatory, many injuries still occur. Little has been published about roller derby injuries, but one early study has found that 50% of participants have had multiple injuries, occurring mostly in the lower extremity and causing 19% to give up the sport permanently [61]. Another study reported a prevalence of 53% injuries in a 12 months period in 14,000 derby skaters [62]. One study examined derby skaters' beliefs about how they got injured. Most often skaters felt that they had pushed the envelope too far in terms of skill or body awareness and consequently were injured [63].

A major difference between roller skating and ice skating is the intensity behind safety recommendations for each. In roller skating, multiple groups have given recommendations about safety. The United States Consumer Product Safety Commission has twice published safety recommendations for roller skating. They recommend taking lessons, protective padding, helmets, falling relaxed rather than stiffened, falling on flesh rather than bone, and skating on smooth surfaces and not at night [64, 65]. InlineSkates.com recommends taking lessons, avoiding rough surfaces, using helmets and padding, stretch-

Fig. 26.8 Roller derby is a collision sport with many opportunities for injury. Reproduced with permission from Mark Nockleby

ing, proper fitting boots, and avoiding crowded areas [66]. The National Safety Council, American Academy of Orthopaedic Surgeons, and American Orthopaedic Society for Sports Medicine all concur and add the need for proper etiquette when skating to avoid collisions [67, 68, 69]. The most extensive list of recommendations was published by the American Academy of Pediatrics. They listed nine items summarized as follows [70].

1. Parents need to learn risks and benefits.
2. Protective gear: helmets, wrist guards, knee pads, elbow pads.
3. Skate on streets without cars.
4. Prefer rinks to streets and avoid tricks.
5. Prohibit truck-surfing and skitching.
6. Get appropriate and well-fitted skates.
7. Take lessons and avoid road debris and defects.
8. If there are muscle or balance problems have extra protection.
9. Encourage state legislation to mandate helmet usage.

Unfortunately, this level of concern has not been seen in ice skating. While as previously mentioned, several authors have advocated for wrist guards and helmets for ice skaters, it is not a general consensus.

Fact Box

- Roller skating has the same injuries as ice skating.
- Dry, uneven skating surfaces lead to increased falls.
- Roller skating has embraced using more protective gear than has ice skating.

26.7 Skaters with Disabilities

Disabilities in skaters are intellectual, physical or both. The Special Olympics is for athletes with intellectual disabilities. The Paralympics is for athletes with physical disabilities. The Special Olympics includes three skating sports: figure skating, roller skating, and short track speed skating. In 2011, 7304 Special Olympic Athletes participated in figure skating competitions, 44,231 athletes competed in roller skating and 14,496 athletes competed in speed skating. Every year the numbers are increasing. Ice hockey is the only Paralympic sport with skating, but figure and speed skaters are trying hard to become part of the Paralympics. In 2010 Margarita Sweeney-Bird began Inclusive Skating, an organization to include skaters with all types of disabilities. In 2018 the ISU recognized Inclusive Skating and began to include their events on their calendar. Inclusive Skating holds competitions for skaters with physical disabilities and is working to have these events become part of the Paralympics [71]. US Figure Skating has published the online Adaptive Skating Manual for skating directors and instructors to teach how to coach athletes with both intellectual and physical disabilities [72]. US Figure Skating also promotes Therapeutic Skating for skaters with physical disabilities to improve posture, strength, and confidence [73].

The skaters and their advocates are working hard to get recognition. It is an uphill fight. Rinks are concerned about liability, and trained coaches are in short supply. Some feel that these skaters do not have the same allure of abled skaters. Those involved with Inclusive Skating note that these skaters experience true joy while skating and gain more from the experience than do the abled skaters [74]. It has been well known for some time that skating improves motor skills and mental and social abilities [75].

In terms of injuries, skaters with disabilities should be at risk for the same injuries other skaters get. There have been no epidemiological studies of injuries in this group, but it is unlikely that they would have different injury patterns. The skaters with balance problems would most likely sustain the same pattern of injuries as novice skaters who have more falls, concussions, and fractures. It is for that concern that these athletes require greater supervision [72]. Prevention and rehabilitation would be similar to that of all skaters.

26.8 Summary

There are many different types of skating, levels of skater skill and risk factors for injury. All skaters are at risk for falling, and most of the serious injuries occur from falls. These include concussion, wrist fractures, and ankle fractures more often than other major injuries. In speed skating lacerations are a concern. In singles figure skating hip problems are becoming more concerning. In roller skating uneven skating surfaces are a major risk factor for falls. All skaters can develop blisters and calluses from poorly fitted skates. Learning how to fall safely and use of helmets, wrist guards, elbow pads, and knee pads should decrease injuries from falls in all skaters.

References

1. Formenti F, Minetti AE. Human locomotion on ice: the evolution of ice skating energetics through history. J Exp Biol. 2007;210:1825–33.
2. Hines J. Figure Skating, A History: University of Illinois Press; 2006.
3. (2018) Sports and Recreational Injuries. In: Injury Facts. https://injuryfacts.nsc.org/home-and-community/safety-topics/sports-and-recreational-injuries/. Accessed 22 Mar 202.
4. Training Resources for NGBs. In: NGB Services. https://uscenterforsafesport.org/ngb-services/training/. Accessed 22 Mar 2020.
5. Vescovi JD, VanHeest JL. The science of figure skating. London: Routledge, Taylor & Francis Group; 2018.
6. Athar S, Mirhadi S, Ashwood N. Ice skating injuries: a review of the literature on nature of injuries and the role of preventive strategies. Annals of Trauma & Acute Care. 2017;1:1–4.
7. Barr LV, Imam S, Crawford JR, Owen PJ. Skating on thin ice: a study of the injuries sustained at a temporary ice skating rink. International Orthopaedics (SICOT). 2010;34:743–6.
8. Ostermann RC, Hofbauer M, Tiefenböck TM, Pumberger M, Tiefenböck M, Platzer P, Aldrian S. Injury severity in ice skating: an epidemiologic analysis using a standardised injury classification system. International Orthopaedics (SICOT). 2015;39:119–24.
9. Meixner C, Loder RT. The demographics of fractures and dislocations across the entire United States due to common sports and recreational activities. Sports Health. 2020;12:159–69.
10. Porter EB, Young CC, Niedfeldt MW, Gottschlich LM. Sport-specific injuries and medical problems of figure skaters. WMJ. 2007;106:330–4.
11. Porter EB (2013) Common Injuries and Medical Problems in Singles Figure Skaters: Current Sports Medicine Reports 12:318–32013.
12. Quinn A, Lun V, McCall J, Overend T. Injuries in short track speed skating <sup/>. Am J Sports Med. 2003;31:507–10.

13. (2020) Communication No. 2195. In: Mass Start and Team races at ISU Speed Skating Events -- Safety measures, promotion initiatives and organization of races. https://www.isu.org/speed-skating/rules/ssk-communications/17969-isu-communication-2195/file.
14. Hausken K. Evolutions in the physiology of skiing, skating and running in the Olympics. J Sports Med Phys Fitness. 2019;59 https://doi.org/10.23736/S0022-4707.18.08977-6.
15. Richard P, Billaut F. Combining chronic ischemic preconditioning and inspiratory muscle warm-up to enhance on-ice time-trial performance in elite speed skaters. Front Physiol. 2018;9:1036.
16. Gimenez P, Chicoine E, Amarantini D, Maso F, Tremblay J. Unilateral conditioning contractions enhance power output in elite short track speed skaters. Sports Med Int Open. 2018;02:E185–90.
17. Khuyagbaatar B, Purevsuren T, Park WM, Kim K, Kim YH. Interjoint coordination of the lower extremities in short-track speed skating. Proc Inst Mech Eng H. 2017;231:987–93.
18. Menting SGP, Konings MJ, Elferink-Gemser MT, Hettinga FJ. Pacing behavior of elite youth athletes: analyzing 1500-m short-track speed skating. Int J Sports Physiol Perform. 2019;14:222–31. https://doi.org/10.1123/ijspp.2018-0285. Epub 2019 Jan 2
19. Kim K, Kim JS, Purevsuren T, Khuyagbaatar B, Lee S, Kim YH. New method to evaluate three-dimensional push-off angle during short-track speed skating using wearable inertial measurement unit sensors. Proc Inst Mech Eng H. 2019;233:476–80.
20. Purevsuren T, Khuyagbaatar B, Kim K, Hyuk Y (2018) Investigation of Knee Joint Forces and Moments during Short-Track Speed Skating Using Wearable Motion Analysis System International J Precision Eng Manufacturing 19:1055–1060.
21. van der Kruk E, Reijne MM, de Laat B, Veeger D (H. EJ) (2019) Push-off forces in elite short-track speed skating. Sports Biomech 18:527–538.
22. Huebsch T. How much faster is speed skating than running? In: Canadian Running; 2018. https://runningmagazine.ca/the-scene/olympic-speedskating-running-comparison/. Accessed 3 Feb 2020
23. About long track speed skating. In: Faster Forward. https://www.speedskating.ca/teams/long-track/about-long-track. Accessed 3 Feb 2020.
24. Spencer D (2018) Canada's speed skating suits harbour a high-tech secret. In: The Gelobe and Mail. https://www.theglobeandmail.com/sports/olympics/canadas-speed-skating-suits-harbour-a-high-tech-secret/article38026807/. Accessed 6 Feb 2020.
25. Smith W. Speed Skating, In-Line skating, Figure Kkating. In: McKeag DB, Van Camp SP, editors. Safran MR. Manual of Sports Medicine: Lippincott-Raven Publishers; 1998. p. 597–8.
26. Nijenhuis B, Schalkwijk AHP, Hendriks S, Zutt R, Otten E, Tijssen MAJ. Skater's cramp: a possible task-specific dystonia in Dutch ice skaters. Mov Disord Clin Pract. 2019;6:559–66.
27. Tandberg AN, Grindem H, Wiig C, Figved W. Knee sliced open by skate blade: complete patellar tendon rupture in an elite long track speed skater. BMJ Case Rep. 2019;12:e228611.
28. US Figure Skating (2020) Organized Figure Skating in the United States.
29. King DL, DiCesaro SF, Getzin AR. Self-reported injuries of competitive US figure skaters. Cogent Medicine. 2017;4 https://doi.org/10.1080/2331205X.2017.1419420.
30. Lambrinakos-Raymond K, FitzGerald B, Geminiani ET (2019) Sideline Coverage of Figure Skating Events: Current Sports Medicine Reports 18: 166–171.
31. Dubravcic-Simunjak S, Kuipers H, Moran J, Simunjak B, Pecina M. Injuries in synchronized skating. Int J Sports Med. 2006;27:493–9.
32. Dubravcic-Simunjak S, Pecina M, Kuipers H, Moran J, Haspl M. The incidence of injuries in elite junior figure skaters. Am J Sports Med. 2003;31:511–7.
33. Han JS, Geminiani ET, Micheli LJ. Epidemiology of figure skating injuries: a review of the literature. Sports Health. 2018;10:532–7.
34. King D (2018) The science of figure skating: jumps. ACSM Blog.
35. Ortega DR, Rodríguez Bíes EC, de la Rosa FJ B. 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. eCollection 2010
36. AOSSM Preventing figure skating injuries. In: Stop Sports Injuries. https://www.stopsportsinjuries.org/STOP/STOP/Prevent_Injuries/Figure_Skating_Injury_Prevention.aspx. Accessed 4 Feb 2020.
37. Kowalczyk AD, Geminiani ET, Dahlberg BW, Micheli LJ, Sugimoto D. Pediatric and adolescent figure skating injuries: a 15-year retrospective review. Clin J Sport Med. 2019;1
38. Campanelli V, Piscitelli F, Verardi L, Maillard P, Sbarbati A. Lower extremity overuse conditions affecting figure skaters during daily training. Orthop J Sports Med. 2015;3:232596711559651.
39. Smith AD. The Young skater. Clin Sports Med. 2000;19:741–55.
40. Amoli MA, Sawyer JR, Tyler Ellis R, Seymour J, Spence DD, Kelly DM, Warner WC. Pediatric patients with Spondylolysis have high rates of vitamin-D deficiency. J Surg Orthop Adv. 2019;28:257–9.
41. Lipetz J, Kruse RJ. Injuries and special concerns of female figure skaters. Clin Sports Med. 2000;19: 369–80.
42. Weber AE, Bedi A, Tibor LM, Zaltz I, Larson CM. The Hyperflexible hip: managing hip pain in the dancer and gymnast. Sports Health. 2015;7: 346–58.
43. Ishøi L, Thorborg K, Kraemer O, Hölmich P. The association between specific sports activities and sport performance following hip arthroscopy for femoroacetabular impingement syndrome: a secondary analysis of a cross-sectional cohort study includ-

ing 184 athletes. Journal of Hip Preservation Surgery. 2019;6:124–33.
44. Burke J (2020) Medical evaluation of elite figure skaters. Personal Communication.
45. Bruening DA, Reynolds RE, Adair CW, Zapalo P, Ridge ST. A sport-specific wearable jump monitor for figure skating. PLoS One. 2018;13:e0206162.
46. Acuña SA, Smith DM, Robinson JM, Hawks JC, Starbuck P, King DL, Ridge ST, Charles SK. Instrumented figure skating blade for measuring on-ice skating forces. Meas Sci Technol. 2014;25:125901.
47. Slater LV, Vriner M, Schuyten K, Zapalo P, Hart JM (2018) Sex Differences in Y-Balance Performance in Elite Figure Skaters: Journal of Strength and Conditioning Research 1.
48. Spiegl O (2016). The effects of different figure skating boots on the human body during the landing impact. - a comparison between two figure skating boots, Edea and Graf.ResearchGateThesis. https://www.researchgate.net/publication/308611586_The_effects_of_different_figure_skating_boots_on_the_human_body_during the_landing_impact_A_comparison_between_two_figure_skating_boots_Edea_and_Graf.
49. Bruening DA, Richards JG. Redesigning figure skating boots to reduce impact forces: 1143 board #6. Med Sci Sports Exerc. 2005;37:S215.
50. Okamura S, Wada N, Tazawa M, Sohmiya M, Usuda S, Shirakura K, Shimizu T, Ibe Y (2014) Injuries and disorders among young ice skaters: relationship with generalized joint laxity and tightness OAJSM 191.
51. Knox CL. Video analysis of falls experienced by paediatric iceskaters and roller/inline skaters. Br J Sports Med. 2006;40:268–71.
52. Knox CL, Comstock RD, McGeehan J, Smith GA. Differences in the risk associated with head injury for pediatric ice skaters, roller skaters, and inline skaters. Pediatrics. 2006;118:549–54.
53. Avrillon S, Lacourpaille L, Hug F, Le Sant G, Frey A, Nordez A, Guilhem G. Hamstring muscle elasticity differs in specialized high-performance athletes. Scand J Med Sci Sports. 2020;30:83–91.
54. Larkin P Identifying Lower Extremity Imbalances in your Skater. http://www.houstonskatesports.com/biomechanics?sitecookie=f5542c8ef96d83675f3ca00f0c818d13. Accessed 22 Mar 2020.
55. Hilgert RE, Besch L, Behnke B, Egbers HJ (2004) Injury pattern caused by aggressive inline skating. Sportverletz Sportschaden. 18:196-203.
56. Inline Skating Safety Statistics. In: Inline Skating Resource Center. https://www.iisa.org/resources/safety.htm. Accessed 20 Mar 2020.
57. Schieber RA, Branche-Dorsey CM. In-line skating injuries: epidemiology and recommendations for prevention. Sports Med. 1995;19:427–32.
58. Schieber RA, Branche-Dorsey CM, Ryan GW, Rutherford GW, Stevens JA, O'Neil J. Risk factors for injuries from in-line skating and the effectiveness of safety gear. N Engl J Med. 1996;335:1630–5.
59. CDC (2016) In-line skating injuries: epidemiology and recommendations for prevention. In: Centers for Disease Control and Prevention. https://wonder.cdc.gov/wonder/prevguid/p0000450/p0000450.asp. Accessed 12 Mar 2020.
60. Houshian S, Andersen HM. Comparison between in-line and rollerskating injury. A prospective study. Scand J Med Sci Sports. 2000;10:47–50.
61. Markey SM, Hutchison RL (2017) Epidemiology of Women's Flat Track Roller Derby Injuries in Kansas City. Mo Med. 114:66–69. PMID: 30233105; PMCID: PMC6143569.
62. Cathorall ML, Peachey AA. Incidence and predictors of roller derby injuries among female roller derby athletes. Int J Inj Control Saf Promot. 2018;25:387–92.
63. Cathorall ML, Punches M. Descriptive study of female roller derby athletes' beliefs about risk factors for injury in roller derby. BMJ Open Sport Exerc Med. 2017;3:e000294.
64. CPSC (1980) Roeller skates pass skateboards as major cause of recreational injury. In: U.S. Consumer Product Safety Commission. https://www.cpsc.gov/content/roller-skates-pass-skateboards-as-major-cause-of-recreational-injury. Accessed 12 Mar 2020.
65. CPSC Skate safely – always Wear safety gear . In: CPSC Safety Alert https://www.cpsc.gov/s3fs-public/5014.pdf. Accessed 12 Mar 2020.
66. Preventing Inline Skating Related Injuries. In: Inlineskates. https://www.inlineskates.com/Preventing-Inline-Skating-Related-Injuries/article-4-30-2015,default,pg.html. Accessed 12 Mar 202.
67. Inline Skating Safety. In: National Safety Council Our Mission is Safety. https://www.nsc.org/NSCDocuments_Advocacy/Fact%20Sheets/Inline-Skating-Safety.pdf. Accessed 24 Feb 2020.
68. AAOS (2009) Inline skating injury prevention. In: OrthoInfo. https://orthoinfo.aaos.org/en/staying-healthy/inline-skating-injury-prevention. Accessed 12 Mar 2020.
69. AOSSM Preventing inline skating injuries. In: Stop Sports Injuries https://www.stopsportsinjuries.org/STOP/STOP/Prevent_Injuries/Inline_Skating_Injury_Prevention.aspx. Accessed 4 Feb 2020.
70. American Academy of Pediatrics Committee on Injury and Poison Prevention and Committee on Sports Medicine and Fitness. In-line skating injuries in children and adolescents. Pediatrics. 1998;101(4 Pt 1):720–1.
71. Inclusive Skating started as a Global Project and achieved World-Wide recognition in 2018. In: International. https://inclusiveskating.org/about/international. Accessed 12 Mar 2020.
72. https://usfigureskating.org/sites/default/files/media-files/Adaptive%20Skating%20Manual.pdf. Accessed 20 Mar 2020.
73. US Figure Skating Adaptive Skating. In: Skating OppCapitalize "Someword" through "Otherword"

ortunities. https://www.usfigureskating.org/skate/skating-opportunities/adaptive-skating. Accessed 20 Mar 2020.

74. Frizzell N (2015) Why disabled figure skaters are fighting for the right to fall. In: Vice. https://www.vice.com/en_us/article/9ae743/why-disabled-figure-skaters-are-fighting-for-the-right-to-fall. Accessed 20 Mar 2020.

75. Ionescu A, Badau D. Student perceptions of motor, mental and social benefits and the impact of practicing recreational figure skating. Behavioral Sciences. 2018;8:110.

27 Skiing (Alpine, Nordic, Road, Track)

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27.1 Some Key Elements in the History of Skiing

27.1.1 Skiing: A Necessity and Survival Tool

For more than 10,000 years, the word "ski" has been used in the dialects of many peoples in northern Europe and Asia (skis: suski, suks, sok, suksildae). The first rock engravings showing skis in skis date from 4000 BC and depict men wearing long planks (Fig. 27.1).

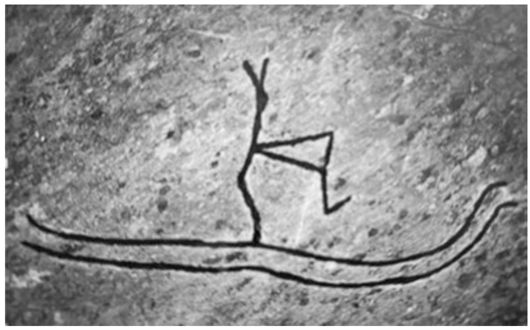

Fig. 27.1 Cave painting showing "man on ski"—4000 BC

These men who lived at altitude after the ice ages and were led in the face of significant snow conditions to develop tools to improve their mobility and their living conditions. Skiing was born in the Nordic countries, especially the peoples who lived between Lake Baikal and Mount Altai. Ski remains to date back more than 4000 years have been found in Norway and also in Scandinavian peat bogs with shorter or shorter fossil skis attesting to a different evolution of the equipment according to the regions.

From the tenth century, the use of skiing became more popular in Norway and Sweden.

27.1.2 Skiing: A Fast and Silent Means of Mobility Useful for the Military

In 1552, skiing played a decisive role in the uprising organized by Gustav Vasa for the liberation of

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Fig. 27.2 1900: army soldiers help democratize and spread the practice of skiing

Sweden (celebrated every year since 1922 by one of the most important international cross-country ski races: "La Vasaloppet").

It was at the end of the nineteenth century that the French army became interested in this means of fast and silent transport. The army of the Alps is responsible for monitoring the high valleys. The Ministry of War bases the first military skiers at the Col du Lautaret in 1900, and created in 1904 the first army ski school to train several generations of military skiers. These soldiers, often ski instructors in their spare time, will help democratize and disseminate the practice of skiing to tourists and local mountain populations (Fig. 27.2).

27.2 When Skiing Becomes Leisure and a Sport

In the nineteenth century, the Scandinavians came up with the idea to transform the practice of skiing into a sport. This shift of skiing to sports and leisure activities will be punctuated by developments initiated by men who propose technical modifications to the equipment and also changes to the practice and technical gesture.

Norwegian skis are 3-m long and attached to the feet with a simple strap (Fig. 27.3). Their instructions are rustic:

"Skiers let skis drive them where they want until the air acts as a natural brake and causes them to stop."

"When going downhill, the skier leans on his pole and closes his eyes. Then he goes straight and continues until he can no longer breathe. He then throws himself on the side in the snow and waits to catch his breath, then he launches straight on until he still loses his breath and throws himself in the snow, and so on to the bottom."

In 1850, the Norwegians mold the first arched skis: the weight of the skier is better distributed over the snow, and the "gliding" is better.

In 1868: Sondre Norheim manufactures "wasp size" skis with variable dimensions.

The skis are wider in the heel and the tip than under the foot. The turns are easier: placed on the side, the ski follows the curve under the pressure of the skier. On the binding side, it braids a

Fig. 27.3 Norwegian skis were 3 m long

wicker strap that goes behind the heel so that the ski comes back naturally under the foot.

An ultimate and brilliant finding, he developed a turn, which he called telemark: on his knees on the inside ski, the pivoting was obtained by pushing the outside foot in the direction of the curve (Fig. 27.4).

February 9 is the first national ski competition in Iverslokken.

1878: Grenoble mountaineer Henry Duhamel descends with 3 m boards on the slopes of the Croix de Chamrousse near Grenoble and succeeds in one of the first long ski descents.

1882: ash wood, which is most often used for its flexibility and lightness, is replaced by hickory (already imported from Louisiana to make golf clubs) because it allows to cut narrower, more flexible skis while remaining robust.

1889: Mathias Zdarsky invents alpine skiing (Fig. 27.5).

He understands that the skis are too long to face the steep slopes, and he reduces their size from 3 m to 1.80 m, removes the grooves from the sole, and makes a metal stirrup to lock the foot on the ski (he will deposit one hundred and four–twenty fastening patents).

Fig. 27.4 Sondre Norheim invented telemark and wasp-sized skis

Several technical schools compete: the Norwegian school, called "Telemark," that of Lilienfeld, called "Alpine technique," and that "of Christiania" (first parallel turn downstream to embark on big slopes).

1905: Zdarsky, to objectively quantify the difference between these techniques, plants stake on the slope: he has just invented the giant slalom.

1908: Arnold Lunn founded the Alpine ski club, invented timed slalom, downhill events, and created the most prestigious alpine ski competition: the Kandahar.

1928: Rudoph Lettner screws steel blades on each side of the ski and has just invented the metal edges.

This year, the ski lifts appeared in the Alps.

1933: first Rochebrune cable car in Megève.

1934: first ski lift in Davos.

Things are started, skiing becomes an essential economic activity (the white gold rush) and since then has not stopped evolving and democratizing.

Today, all over the world, skiing and snowboarding have become the most practiced winter sports.

Practitioners, whether amateur or professional, are particularly exposed to trauma in the alpine modalities, freestyle skiing and snowboarding, and also to overuse pathologies in the context of Nordic skiing.

Fig. 27.5 Mathias Zdarsky: precursor of alpine skiing

These activities have become more and more accessible, with more and more efficient equipment which allows an ever-increasing number of practitioners to go faster and faster on every larger ski area.

This evolution of practices and equipment has considerably modified the accidentology and traumatic pathology linked to the practice of skiing and snowboarding.

27.3 We Propose to Analyze in this Work

– The current traumatic pathology of a standard population based on an analysis over a period of 60 years, of the activity of a trauma and sports service located "at the foot of the slopes" at the southern hospital of Grenoble.
– The more specific traumatic pathology of high-level skiers from the monitoring of competitors in the French team.

27.4 Accidentology and Traumatic Pathologies Linked to the Practice of Snow Sports in a Standard Population

The trauma of snow sliding sports has evolved considerably over the past 50 years. Before the 1970s, the practice of skiing was relatively confidential, and only a few initiates engaged in this practice. In Alpine skiing, bimalleolar fractures were by far the most frequent fractures. Gradually, new sliding techniques appeared with cross-country skiing at the end of the 1970s, then snowboarding at the end of the 1980s and finally snowblade (short skis, less than 1 m long) at the end of the 1990s. In parallel with this evolution of practices, the equipment has considerably evolved as well as the preparation of the ski area, with an increasingly pronounced craze for this leisure activity. Therefore, the trauma has changed over time. Even if snow sports accidents are currently commonplace, they only represent 2.5 accidents per 1000 ski days which are far behind the seven accidents per 1000 days of the 1970s. Alpine skiing remains by far the most practiced with snowboarding, and we will, therefore, focus on these two disciplines which summarize the major evolution of snow sports over the past 25 years.

Given the significant frequency of ruptures of the anterior cruciate ligament (ACL), we will discuss in a specific chapter, the mechanisms of ACL rupture during alpine skiing.

27.5 Epidemiology, Evolution, Prevention of Downhill Skiing Accidents (from 1968 to 2012)

The epidemiological study is based on the analysis of the activity of the same hospital service at the Grenoble University Hospital, which has documented ski track accidents since 1968 until today. We were, thus, able to compare the evolution of trauma-based on three studies: the first carried out in the 1970s by Professor Bèzes (between 1968 and 1976); the second and the third carried out by Professor Saragaglia in the 1990s on the one hand (between 1990 and 1997), and in the 2010s on the other hand (from the 1998–1999 season to the present day).

27.6 In the Seventies (the Series of Prof H. Bèzes)

The cohort includes 5200 injured people, victims only of a ski accident (no cross-country ski accident recorded at that time), 59% men, and 41% women, average age 22 years (2–77) (Table 27.1).

Table 27.1 Evolution of snow sports accidentology in recreational practice

	1968–1976	1990–1997	1998–1999	2008–2009
Number of patients	5200	4647	731	591
Alpine skiing	5200	3570	567	425
Cross-country skiing	0	322	NC	NC
Snowboard	0	535	134	139
Nordic skiing	0	110	25	26
Artistic skiing	0	86	0	0
Snow blade	0	1	6	1
Monoski	0	19	0	0
Telemark	0	3	0	0
Skwal	0	1	0	0
Alpine Skiing (*N*)	5200	3570	567	425
Male (%)	59	53	58	55
Female (%)	41	47	42	45
Mean age (years)	22	29	30	30
Anatomical site	Injuries (percentage values)			
Body axis	9%	7%	Not included	Not included
Upper limb (%)	15	31	29	35
Scapular girdle (%)	45	37	44	51
Wrist fracture (%)	16	11.4	10	17
Hand (%)	21	29	24	16
Sprain of MCP of the thumb (%/hand)	40	67	72	46
Others (%)	60	33	28	54
Lower limb (%)	76	62	71	65
Femur fracture (%/lower limb)	3.3	8.1	4.2	4.7
Knee (%/lower limb)	18.8	60	60	66
Severe knee sprain (% knee)	10.2	36.2	35	40.5
Tibial plateau fracture (%/knee)	1.2	5	7.1	12.5
Leg fracture (%/lower limb)	57.6	19	25	16.5
Ankle (%/lower limb)	18.5	7.3	5.5	2.2

Group 1 lesions (body axis: head, face, spine, thorax, abdomen, pelvis) represented 9% of all lesions, including 33% of spine lesions and 5.5% of pelvis lesions.

Lesions of the upper limb represented 15% of all lesions including 45% of lesions of the shoulder girdle and 21% of lesions of the hand (40% of sprains of the metacarpophalangeal joint (MCP) of the thumb.

Lesions of the lower limb represented 76% of all lesions with, 57.6% of fractures of the leg, 18.8% of lesions of the knee, 18.5% of lesions of the instep, and 3.3% fractures of the femur.

27.7 In the Nineties (Prof Saragaglia Series)

The cohort included 4647 injured people (4920 injuries) including 3570 alpine skiers (3788 injuries), 535 snowboarders, and 322 cross-country skiers. Among alpine skiers, we had 53% men and 47% women whose average age was 29 (2–84) (Table 27.1).

Group 1 lesions represented 7% of all lesions, including 29% of spine fractures and 20% of pelvic fractures.

Lesions of the upper limb represented 31% of all lesions including 37% of lesions of the shoulder girdle and 29% of lesions of the hand (67% of sprains of the MCP of the thumb).

Lesions of the lower limb represented 62% of all lesions, including 60% of knee lesions, 19% of leg fractures, 8.1% of femur fractures, and 7.3% of instep.

27.8 In the 2000s (Series by Prof Saragaglia)

This series analyzes the epidemiology of alpine skiing and snowboarding accidents 10 years apart by comparing the 1998–1999 and 2008–2009 seasons. Overall, we have seen an increase in serious accidents (fractures, dislocations, severe sprains) going from 62.1% to 71.5% ($p < 0.001$), or from 26.1% of surgical treatments to 36.5% (Table 27.1).

Regarding ski trauma, we have witnessed a "sliding" of the lesions up the body with an increase in damage to the upper limb (from 29% to 35%) knowing that the lower limb remains the most affected. This "sliding" toward the top of the body corresponds to a significant increase in lesions of the wrist ($p = 0.021$), shoulder, and shoulder girdle ($p = 0.036$) (Fig. 27.6). Conversely, sprains of the metacarpophalangeal thumb have decreased significantly ($p = 0.006$). The knee remains the most affected joint, but

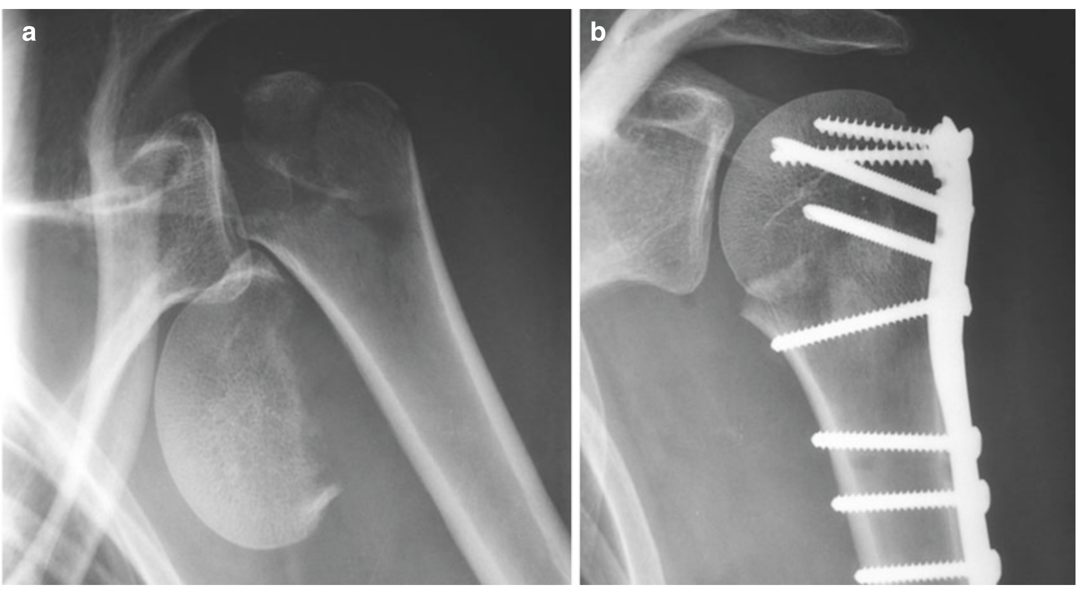

Fig. 27.6 fracture-dislocation of the humeral head (anatomical neck) (**a**) preoperative and (**b**) postoperative X-rays

its proportion remains stable (42.32% in 1998–1999 and 43.06% in 2008–2009) with a majority of ACL lesions. There is indeed a decrease in the proportion of benign sprains ($p = 0.041$) in favor of severe sprains which go from 20.9% to 26.81% of lesions of the lower limb. We will see in the analysis of risk factors that the appearance of parabolic skiing, which constitutes the main technical evolution between the two studied seasons, seems to have a responsibility in alpine skiing trauma modifications.

27.9 Currently

Our database includes 12,352 alpine skiing accidents and 3290 snowboard accidents. Over this period, in alpine skiing, the knee was most frequently injured with 3851 sprains (31.2%) including 235 avulsions of the anterior tibial spines and 496 fractures of the tibial plateau (4%) (Fig. 27.7). Regarding leg fractures, we counted 1199 fractures, or 9.7%. In snowboarding, we counted 669 fractures or epiphyseal detachment of the distal end of the radius (20.5%). These figures, over a long period, corroborate what has been demonstrated elsewhere over shorter periods and with a smaller population.

27.10 Evolution of Accidents

Body axis lesions, despite tending to bend, remain relatively stable over time. The decrease in their frequency, observed in our study, is linked to a recruitment bias since hyper specialization has led us over time to direct the management of these craniocerebral traumas and serious trauma to the spine, to other more specialized structures equipped with dedicated multidisciplinary technical platforms.

On the other hand, the isolated trauma of the limbs has worsened to represent in certain centers and according to the data in the literature up to 25% of recruitment.

Lesions of the upper limb, after a considerable increase was observed between 1976 and 1997 (from 15% to 31%), stabilized slightly above 30% (29% in 1999 and 35% in 2009). However, the distribution of lesions has changed. In fact, there has been an increase in lesions of the shoulder girdle (fractures of the clavicle, shoulder dislocations, acromioclavicular disjunctions), with a rate which

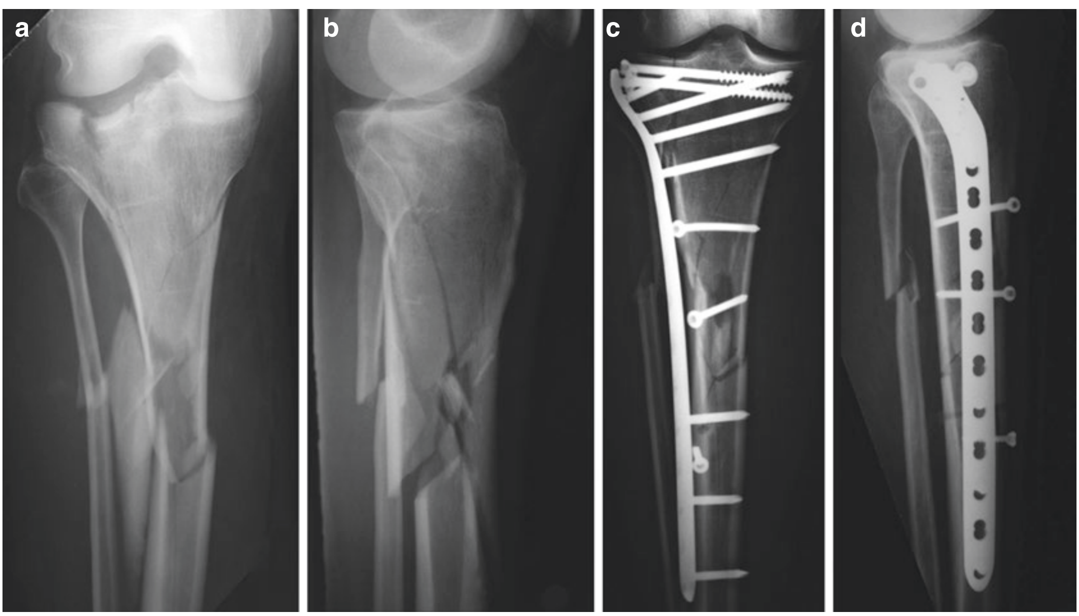

Fig. 27.7 (**a** and **b**) Preoperative AP and lateral radiograph views of complex T-shaped fracture of the tibia plateau and diaphysis. (**c** and **d**) postoperative AP and lateral radiograph views of fracture osteosynthesis

increased from 6.8% in 1976 to 13% in 1999 and 18% in 2009. There has also been an increase in fractures of the lower extremity of the radius, especially in the past 15 years because this rate increased from 2% in 1997 to 3% in 1999 and 6% in 2009. On the other hand, sprains of the MCP of the thumb, after a significant increase between 1976 and 1997 (from 1.3% to 6%), decreased with a rate which rose from 5% in 1999 to 2.5% in 2009.

Lesions of the lower limb stabilized around 65% (71% in 1999 and 65% in 2009) after a significant drop between 1976 and 1997 (from 76% to 61%). The rate of leg fractures stabilized at around 10% (15.5% in 1999 and 9.2% in 2009) after a considerable drop between 1976 and 1997 (from 44% to 11.5%). Knee injuries (mainly sprains and fractures of the tibial plateau) also stabilized around 42% (42.3% in 1999 and 43% in 2009) after a considerable increase between 1976 and 1997 (from 14.5% to 37%). Knee sprains remain the most frequent lesions with 37.2% of sprains in 1999 (including 15% of serious sprains with at least a rupture of the ACL) and 34.6% in 2009 (including 18.2% of severe sprains). The fractures of the tibial plateau were multiplied by 5 between 1976 and 2009 (from 0.2% to 5.3%), and the fractures of the femur were multiplied by 2 (from 2.5% to 5%).

27.11 The Reasons for this Development

Several factors explain this evolution of lesions:

– the evolution of the material,
– the evolution of the ski area,
– frequentation of the slopes.

27.12 The Evolution of the Material (Equipment) Has Changed the Mechanism of Injuries in Alpine Skiing

First of all, the shoe which went from the 1950s boot to the shoe with a high and rigid upper from the 1990s and 2000s (Fig. 27.8).

This development helped move the lesions up the lower limb. Thus, this type of shoe protects the ankle and the leg (reduction of fractures of the instep and the leg), but increases the stresses especially at the knee level, and to a lesser degree at the level of the femur.

Then, the bindings of the boot on the ski are triggered more quickly and in several planes of space, and not in the frontal and sagittal planes. This faster release of the bindings explains

Fig. 27.8 Evolution of ski equipment and boots: from a loose ankle to a blocked ankle

some falls forward, real dives with reception on the stump of the shoulder on groomed snow. Furthermore, improper adjustment of the fasteners contributes to increasing the incidence of ACL tears.

Finally, the ski itself developed from VR7, VR17, Strato in the seventies to parabolic ski to the end of the 1990s (Fig. 27.9).

We can consider that there were few skiers who used these skis during the 1998–1999 season whereas in 2008–2009 this type of ski is widely used since it represents almost all skis today, rentals and new purchased skis. These skis involve a new way of sliding, it is the "carving effect" which is inspired by snowboarding. The principle is based on a spatula and a widened heel while the middle of the ski is narrowed. This curvature will make it possible to decrease the radius of the turn, which is of the order of 45 m with traditional skis and from 10 to 30 m with the "carve" of parabolic skis. This finer handling in cut turns will considerably increase the skier's speed by decreasing the skid. During a "carving" turn, the skier leans as far as possible inside, giving way to a new trauma (closer to that of snowboarding) linked essentially to the stalling of support at high speed. There is, therefore, trauma by direct impact on the shoulder girdle and wrists (fractures). Although it is a more accessible ski for beginners to progress faster and reach high speeds in a short time, this increases the severity of the lesions. This type of skiing *does not spare the knee* (persistence of a majority of ACL lesions) because to drive the turn, the skier must print a movement of valgus external rotation to the knee of the lower limb downstream of the turn, stressing the central pivot. Paradoxically, despite the presence of the wrist strap, there has been a reduction in sprains of the metacarpophalangeal joint of the thumb.

The ski area has evolved considerably over the decades. Today, the ski slopes are wide,

Fig. 27.9 Evolution of bindings and skis: from a free foot and from a straight ski to a held foot and to a carved ski

groomed, often prepared with snow cannons. This leads to an increase in the speed of skiers which has almost doubled in more than 40 years. Falls are more dangerous and more serious as are collisions (between skiers or against a tree or rock). These falls at high speed on snow that looks like concrete, and the modification of practices (free ride with jump of rocky bar) largely explains the increase in serious trauma (fractures-dislocations of the shoulder, complex fractures of the plateau tibial, fractures of the pelvis, fractures of the femoral shaft or the neck of the femur) and polytrauma.

Overcrowded slopes, lack of snow and unsuitable weather conditions further increase the risk of serious accidents.

27.13 Prevention

The prevention of serious accidents involves reducing and harmonizing the speed of skiers. It is also essential to regulate the density and traffic on the ski slopes. The systematic wearing of helmets in children has helped reduce serious head injuries. Finally, the education of skiers must remain essential with the learning of a certain civic spirit and a code of good conduct on the slopes.

The prevention of serious knee sprains requires regular maintenance and adjustment of the bindings. Perhaps we should further improve the multi-directional release of the bindings or develop shoes with rear opening in the event of a backward fall? Finally, educational programs could be of significant help.

27.14 Epidemiology, Evolution, and Prevention of Snowboard Accidents (Snowboard)

Snowboarding traumas represents between 20% and 25% of snow sport accidents. It is marked by a significant upper body involvement that increases over time (from 60.5% to 81.3%—$p < 0.001$). In the foreground, the shoulder and shoulder girdle injuries increased significantly between the 1998–1999 and 2008–2009 seasons, from 14.2% to 31% ($p = 0.002$). The shoulder lesions increased from 4.5% to 13.5% ($p < 0.05$). In our series, the wrist fractures represented the second lesion location, and also increased between 1998–1999 and 2008–2009, going from 17% to 20%.

The involvement of the lower limbs is much less significant in snowboarding and decreases over time since it represented 39.5% of all lesions in 1998–1999 and 18.7% in 2008–2009. We are currently witnessing a significant reduction in knee injuries and more particularly benign sprains since the proportion of involvement of the medial collateral ligament drops from 16.4% to 1.5%. These figures do not agree with the statistics of mountain physicians who find a stability of this type of sprain at around 6.5%. ACL rupture remains rare in snowboarding since it represents only 1.5% of all lesions in our 2008–2009 series and is rather the result of high energy trauma.

In recent years, the evolution of snowboarding equipment has been more progressive, with no major revolution, but rather with a refinement of the technical characteristics of the different types of boards. These have improved by becoming more responsive and faster than before. As its technical development progressed, snowboarding was divided into three main specialties: “free-ride,” “alpine,” and “free-style.” Alpine and competitive surfboards, hard and narrow, with a rounded shape at the front, are precise and fast for surfing on edges. Their proportions have decreased over the past 15 years. This type of board is used with bindings and rigid boots like those used in skiing, which perfectly protect the foot and forefoot. The more flexible free-ride board makes it possible to exploit all the possibilities of the discipline: from off-piste in deep snow to carving on hard tracks. As for jumpers and snow-park enthusiasts, they preferably use a “free-style” board, with more flexible shoes allowing significant back flexion. This type of shoe leads to tearing lesions and fragmented fractures of the lateral tubercle of the talus which are entirely specific to snowboarding (5.6% of lesions of the lower limb in snowboarding in

1998–1999). There are also malleolar fractures (7.7% of lesions of the lower limb in snowboarding in 2008–2009), which are favored by the starting position of the surfer who descends the track in a position of pronounced rotation of the ankles relative to the axis of descent and body.

Regardless of the type of board used, the "carving" effect is used by surfers who are very leaning in turns, with the risk of direct impact on the upper limb, which explains why the upper body is most affected and that rates are still increasing.

Prevention essentially involves educating surfers because the prevention of wrist fractures by protective splints has not been proven to be effective and tend to move fractures up the upper limb.

27.15 Mechanisms and Risk Factors for ACL Tears in Alpine Skiing

Johnson summarizes the ACL rupture mechanisms in skiing in six major types.

Four are not specific to skiing: The *valgus-external rotation* (Fig. 27.10) where the skier makes an inside edge fault, which brings the involved leg outwards and causes the skier to fall forward. The medial collateral ligament of the knee (MCL) is the first affected, but it can be associated with a rupture of the ACL. Ruedl et al. (Int J Sports Med, 2011) have shown that the most frequent mechanism of injury found in ACL tears is the fall forward in Valgus-External Rotation or "forward twisting fall." It seems that it has become the most frequent lesion mechanism since the introduction of carving skis, regardless of gender; *knee hyper-extension; the varus-flexion-internal rotation* (Fig. 27.10) which is often a trauma with low kinetic energy, even in snowplow, the external edge of the downhill ski is blocked, and the body weight by tilting downstream prints in a varus movement of the knee with internal rotation of the tibia which puts tension on the ACL; *complex distortions* that cause multiligament knee injury.

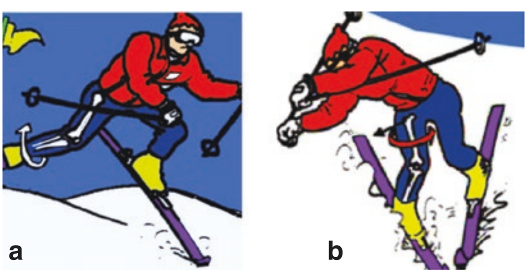

Fig. 27.10 Mechanisms and risk factors for ACL tears in alpine skiing: (**a**) valgus-external rotation and (**b**) varus-flexion-internal rotation

Two are specific to the practice of skiing: the "phantom foot" (Fig. 27.11) which corresponds to a forced internal rotation of the tibia under the femur with the knee bent more than 90° (downstream member), when the skier is unbalanced rear and only has the internal edge of its downstream ski in contact with the snow; "boot induced" (Fig. 27.11) which corresponds to a thrust of the tibia forward, when landing a jump, the tail of the ski first touches the snow while the knee is close to the extension.

Two other mechanisms have been described by *Chambat* in competitive skiers: hyperflexion combined with a strong contraction of the quadriceps at the start of a race can be at the origin of a bilateral rupture of the ACL; the "click-clack" which occurs during a stealthy episode of valgus-external rotation of the downstream knee which disengages from the upstream knee during a tight turn in competition.

The risk factors for ACL injury are gender first. Statistically significantly, epidemiological studies find more ACL injuries in women. According to the Association of Mountain Physicians, women's risk of ACL rupture is 3.5 times greater than men. Several studies show that this is not specific to skiing since, at sport and at the same level, reaching the ACL is more frequent in women (up to 4–6 times more frequent). The reasons are multifactorial: anthropometric (larger pelvis, tighter femoral arch, genu valgum), hormonal (more ACL lesions in the luteal phase, hormonal impregnation of the ACL), and neuromuscular (studies have shown differences in mechanoreceptors). Then, the lack of physical

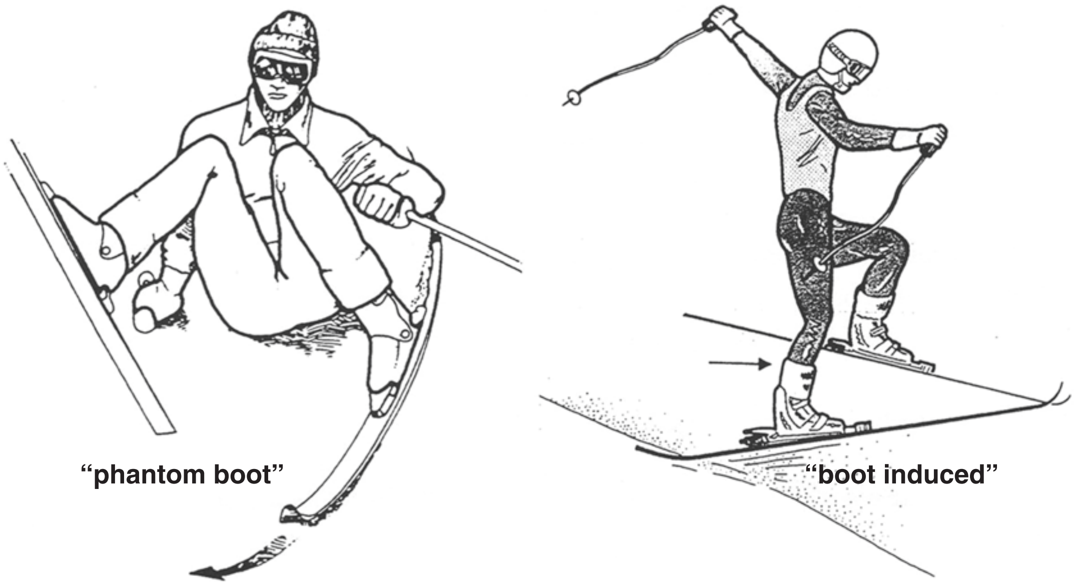

Fig. 27.11 Mechanisms of ACL injury in alpine skiing: "phantom foot" and "boot induced"

preparation: a lack of muscular preparation, especially at the quadricipital level, increases the risk of ACL lesions. Finally, unsuitable equipment: excessively long skis increase the stresses on the LCA, and improperly adjusted bindings (which do not trigger) are the cause of almost half of knee sprains. The binding adjustment criteria must consider the skier's gender, weight, height, and ski level.

27.16 Accidentology and Traumatic Pathologies Linked to the "Competitive" Practice of Snow Sports in a Group of High-Level Athletes

Since 2006, the International Ski Federation (FIS) has carried out retrospective monitoring of the trauma of professional athletes during the winter season 1–8. This work made it possible to better assess the incidence of different injuries during the season in the different disciplines.

Similarly, within the French Ski Federation (FFS), continuous prospective monitoring of ruptures of the anterior cruciate ligament (ACL) has been carried out since 1980 with French alpine ski teams. Since 2017, monitoring has expanded, on the one hand, to all disciplines (alpine skiing, freestyle skiing, snowboarding, and Nordic skiing), and on the other hand, to all traumatic and overuse pathologies.

27.17 Prospective Trauma Monitoring Within the French Ski Teams (Epitraumatic Cohort [1])

If the monitoring carried out by the FIS allows a global evaluation of the trauma of skiing, it is perfectible and incomplete since it is, on the one hand, a retrospective analysis and, on the other hand, an analysis relating to winter season only. Since 2017, the Medical Commission of the French Ski Federation has been carrying out a prospective follow-up on the trauma of all the athletes of French ski teams in all disciplines.

A first analysis was carried out between May 1, 2017, and April 30, 2018. Three hundred and twenty-four athletes who were divided into five disciplines: alpine skiing, freestyle skiing, snowboarding, jumping, and Nordic skiing were included in this study.

Two hundred and four injuries were recorded, an absolute incidence of 63 injuries per 100 athletes/season. In the literature, this incidence is much higher than those published in other studies which only took into account the winter season [2, 3]. One hundred and thirty-six athletes, or 42% of the population, presented at least one injury. One hundred and ninety injuries required a sports interruption, and 32.6% of them were considered severe, that is to say, they required a sports interruption of more than 28 days.

Traumatic injuries accounted for 77% of the cases. In alpine skiing, freestyle skiing, and snowboarding, these traumatic injuries represented most cases, while in Nordic skiing overuse injuries were the majority (Table 27.2).

Alpine skiing represented 37.7% of injuries, snowboarding 21.5%, freestyle skiing 20%, Nordic skiing 18.1%, and ski jumping 2.4%.

The analysis of the relative incidence reported on 1000 runs highlighted an over-risk of injury and severe injury (stopping>28 days) for snowboarding and freestyle skiing compared to alpine skiing (Table 27.3).

One hundred and four injuries (51%) involved the lower limb, including 43 knee injuries. The upper limb was affected in 19.6% of the cases. These findings are corroborated by the results of the literature [2–5]. The skull and cervical spine were affected in almost 20% of the cases for the freestyle (19.5%) and snowboard (22.7%) disciplines (Table 27.4). The Steenstrup [6] series also found a higher incidence of head injuries in the snowboard and freestyle disciplines compared to the alpine. It was concussions in over 80% of cases.

There was no difference in the distribution of injuries between summer and winter (46.8% vs 53.2% (ns)). On the other hand, there were significantly more traumatic injuries during the winter period (86% vs 65%) and more overuse injuries during the summer period (34.4% vs 14%) ($p = 0.001$). Almost a quarter of traumatic injuries occurred in January and February. The Pujol

Table 27.2 Number and percentage values of traumatic and overuse injuries according to the skiing sports modalities

	Alpine	Freestyle	Nordic	jumping	Snowboard	Total
Overuse (%)	21 (27.3)	2 (4.9)	19 (51.4)	0 (0)	5 (11.4)	47 (23)
Traumatic (%)	56 (72.7)	39 (95.1)	18 (48.6)	5 (100)	39 (88.6)	157 (77)
Total (%)	77 (100)	41 (100)	37 (100)	5 (100)	44 (100)	204 (100)

Table 27.3 Relative incidence of all injuries and severe injuries in every 1000 runs in each discipline: Alpine. Freestyle and Snowboard

	Runs (*n*)	All injuries (*n*)	Relative incidence	Relative risk	*p*-Value
Alpine	4642	72	15.5 [12.1–19.5]	1	Not significant
Freestyle	503	41	81.5 [58.5–110.6]	5.26 [3.58–7.71]	**<0.001**
Snowboard	616	43	69.8 [50.5–94]	4.5 [3.08–6.57]	**<0.001**
		Severe injuries (*n*)			
Alpine	4642	24	5.2 [3.3–7.7]	1	Not significant
Freestyle	503	11	21.9 [10.9–39.1]	4.23 [2.07–8.63]	**<0.001**
Snowboard	616	10	16.2 [7.8–29.9]	3.14 [1.5–6.57]	**0.002**

Table 27.4 The injury distribution according to the localization, and discipline of skiing

	Alpine	Freestyle	Nordic	Jumping	Snowboard	Total
Cranial and cervical spine (%)	3 (3.9)	9 (22)	2 (5.4)	0	11 (25)	25 (12.3)
Upper limb (%)	16 (20.8)	9 (22)	5 (13.5)	0	10 (22.7)	40 (19.6)
Trunk (%)	19 (24.7)	5 (12.2)	5 (13.5)	1 (20)	5 (11.4)	35 (17.2)
Lower limb (%)	39 (50.6)	18 (43.9)	25 (67.6)	4 (80)	18 (40.9)	104 (51)
Total (%)	77 (100)	41 (100)	37 (100)	5 (100)	44 (100)	204 (100)

study [7], with 25 years of experience, reported the same finding. Overuse injuries occurred mainly during the months of May and September.

27.18 Monitoring of ACL Ruptures Within the French Ski Teams

The development of the equipment played *a key role in the evolution* of the trauma of ski "competition." The turning point appeared in the late 1970s with the modification of shoes and the appearance of tall stems. While ankle and leg injuries have decreased significantly, knee sprains have increased. ACL ruptures now account for almost 35% of all trauma and 68% of knee trauma [2, 8].

The high incidence of ACL tears in skiers has prompted the FFS Medical Commission to set up a prospective collection of these lesions among French teams since 1980.

The results of this cohort analysis were published in 2007 and 2013, respectively [7, 9]. The study by Pujol and Chambat [7] assessed trauma in the World Cup and European Cup groups between 1980 and 2005. Three hundred and seventy-nine athletes (191 men and 188 women) were followed over this period. One hundred and five athletes presented at least one ACL rupture, i.e., 27.7% of the athletes followed. The gender distribution was homogeneous with 28.2% of the affected women and 27.2% of the men ($p = 0.21$). Women were affected significantly earlier (20 years vs 22 years ($p < 0.01$)). The prevalence of iterative ruptures was 19% and that of contralateral ruptures 30.5%. Regarding a first rupture operation, 39% of athletes were re-operated for a new ACL rupture (homo or contralateral).

The authors reported an incidence of 6.1 ACL tears per year. This figure remained broadly stable over the period analyzed, even though there was a downward trend in the female population while there was an upward trend among men. This incidence remained stable despite the evolution of ski equipment and techniques.

There were also three traumatic peaks during the season: first, in autumn for women, which corresponds to the end of the period of intense physical preparation and the resumption of skiing; second, at the start of the year, a busy period in races and a source of fatigue for athletes; and third, at the end of the season, which can be a sign of a certain relaxation and a lack of concentration.

Finally, the authors analyzed the incidence of ACL tears based on the level of the athletes. The rate of first ACL rupture was significantly higher in the group of athletes ranked in the 30 best skiers in the world ($p = 0.002$). Fifty percent of athletes ranked in the top 30 had at least one ACL rupture compared to 23% for the others. The rates of re-ruptures and contralateral ruptures were also significantly higher in the first group (38.5% vs 12.8% and 33.3% vs 11.4%). The authors noted that athletes ranked in the top 30 in the world had a longer career and that the occurrence of an ACL rupture had no influence on the length of their careers.

Similarly, Haida's study [9] analyzed the influence of the occurrence of an ACL rupture on the career of skiers in the French team. The study involved 477 skiers who joined the national alpine ski teams between 1984 and 2013. One hundred and forty-eight skiers had at least one ACL rupture during their careers. Athletes with an ACL rupture had a significantly longer career than those without an ACL rupture. According to the authors, the mental and physical capacities of very high-performance athletes as well as the optimization of the return to sport process by all medical and sports staff are the keys. In addition, all the athletes who had a rupture of the ACL continued their careers after the rupture.

Age is described in the literature as being closely correlated with performance level [10, 11]. Skiers who improved their performance were significantly younger (men: 22.2 ± 3 years; women: 18.7 ± 2.2 years) than those who did not improve (men: 25.3 ± 4.2 years; women: 22 ± 4 years). The authors emphasized that the time to return to the best level was 3.8 ± 3.1 years for men and 3.1 ± 2.5 years for women, and that the peak performance was reached on average at 25.1 years for men and 25.3 years for women.

Conclusion

The trauma of skiing and snowboarding is constantly evolving due to the modifications made to the equipment, from the boot to the ski or to the board, via the bindings.

The development of the equipment played a key role in the evolution of the trauma of "leisure" or "competition" skiing. The turning point appeared at the end of the 1970s with the modification of shoes and the appearance of tall uppers which limit trauma to the ankle and leg, but which considerably increased severe knee sprains. The evolution of the board and the arrival of carved ski, when it increased the speed and the inclination of the skier, are responsible for an increase in upper limb injuries, and the severity of the trauma that often occurs at speeds is more important.

Trauma is also influenced by external factors such as the preparation and congestion of the slopes, snow conditions, and weather conditions.

Education, good citizenship, and self-discipline should make it possible to reduce the accidentology of snow sports, which has already decreased considerably over the years since 1960.

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References

1. Guyon A. Traumatologie des athlètes élites de la fédération française de ski toutes disciplines confondues sur la saison 2017–2018: cohorte EPITRAUMA. 2018.
2. Flørenes TW, Nordsletten L, Heir S, Bahr R. Injuries among World Cup ski and snowboard athletes. Scand J Med Sci Sports. 2012;22(1):58–66. https://doi.org/10.1111/j.1600-0838.2010.01147.x.
3. FIS_ISS_report_2017–18_English.pdf. https://assets.fis-ski.com/image/upload/v1537433416/fis-prod/assets/FIS_ISS_report_2017-18_English.pdf. Accessed April 11, 2020.
4. Flørenes TW, Heir S, Nordsletten L, Bahr R. Injuries among World Cup freestyle skiers. Br J Sports Med. 2010;44(11):803–8. https://doi.org/10.1136/bjsm.2009.071159.
5. Torjussen J, Bahr R. Injuries among elite snowboarders (FIS Snowboard World Cup). Br J Sports Med. 2006;40(3):230–4. https://doi.org/10.1136/bjsm.2005.021329.
6. Steenstrup SE, Bere T, Bahr R. Head injuries among FIS World Cup alpine and freestyle skiers and snowboarders: a 7-year cohort study. Br J Sports Med. 2014;48(1):41–5. https://doi.org/10.1136/bjsports-2013-093145.
7. Pujol N, Blanchi MPR, Chambat P. The incidence of anterior cruciate ligament injuries among competitive alpine skiers: a 25-year investigation. Am J Sports Med. 2007;35(7):1070–4. https://doi.org/10.1177/0363546507301083.
8. Heijne A, Axelsson K, Werner S, Biguet G. Rehabilitation and recovery after anterior cruciate ligament reconstruction: patients' experiences. Scand J Med Sci Sports. 2008;18(3):325–35. https://doi.org/10.1111/j.1600-0838.2007.00700.x.
9. Haida A, Coulmy N, Dor F, et al. Return to sport among French alpine skiers after an anterior cruciate ligament rupture: results from 1980 to 2013. Am J Sports Med. 2016;44(2):324–30. https://doi.org/10.1177/0363546515612764.
10. Baker AB, Tang YQ. Aging performance for master's records in athletics, swimming, rowing, cycling, triathlon, and weightlifting. Exp Aging Res. 2010;36(4):453–77. https://doi.org/10.1080/0361073X.2010.507433.
11. Lafollie D. Detection of high-risk personalities in risky sports. L Encéphale. 2007;33(2):135–41. https://doi.org/10.1016/s0013-7006(07)91543-2.

Bibliographic Supplement

Bèzes H, Julliard R. Les accidents de ski: à propos d'une statistique de 5200 observations. Ann Chir. 1976;30:583–605.

Haaland B, Steenstrup SE, Bere T, Bahr R, Nordsletten L. Injury rate and injury patterns in FIS World Cup alpine skiing (2006–2015): have the new ski regulations made an impact? Br J Sports Med. 2016;50(1):32–6. https://doi.org/10.1136/bjsports-2015-095467.

Head impact velocities in FIS World Cup snowboarders and freestyle skiers: do real-life impacts exceed helmet testing standards? https://www.ncbi.nlm.nih.gov/pubmed/?term=Head+impact+velocities+in+FIS+World+Cup+snowboarders+and+freestyle+skiers%3A+Do+real-life+impacts+exceed+helmet+testing+standards. Accessed April 11, 2020.

Major DH, Steenstrup SE, Bere T, Bahr R, Nordsletten L. Injury rate and injury pattern among elite World

Cup snowboarders: a 6-year cohort study. Br J Sports Med 2014;48(1):18–22. doi:https://doi.org/10.1136/bjsports-2013-092573.
Müller E, Spörri J, Kröll J, Hörterer H. Equipment designed to reduce risk of severe traumatic injuries in alpine ski racing: constructive collaboration between the International Ski Federation, industry and science. Br J Sports Med. 2016;50(1):1–2. https://doi.org/10.1136/bjsports-2015-095689.
Ruedl G, Webhofer M, Linortner I, Schranz A, Fink C, Patterson C, Nachbauer W, Burtscher M. ACL injury mechanisms and related factors in male and female carving skiers: a retrospective study. Int J Sports Med. 2011;32(10):801–6. https://doi.org/10.1055/s--0031-1279719. Epub 2011 Jun 21
Saragaglia D, Diemer C, Huboud-Peron A, Kirchmeier M, Vouillat H. Évolution de la traumatologie du ski alpin. Comparaison de deux études réalisées à 20 ans d'intervalle à l'hôpital sud de Grenoble (1968–1976 et 1990–1997). J Traumatol Sport. 2000;17:16–28.
Shea KG, Archibald-Seiffer N, Murdock E, Grimm NL, Jacobs JC Jr, Willick S, Van Houten H. Knee injuries in downhill skiers: a 6-year survey study. Orthop J Sports Med. 2014;2(1):2325967113519741. https://doi.org/10.1177/232596711351974.
Stenseth OMR, Barli SF, Martin RK, Engebretsen L. Injuries in elite women's ski jumping: surveillance through the 2017-18 FIS World Cup season. Br J Sports Med. 2020;54(1):44–8. https://doi.org/10.1136/bjsports-2019-100799.

Theses from Grenoble-Alpes University

Arnaud Huboud-Péron: Les accidents de ski et de glisse sur neige: étude épidémiologique rétrospective (1990–1997), à propos de 4647 accidents totalisant 4920 lésions. Holded December, 1rst, 1998 (jury president: Pr D. Saragaglia).
Christelle Diemer : Évolution de la traumatologie du ski : Comparaison de deux séries de l'hôpital sud de Grenoble à 20 ans d'intervalle. Évolution du matériel et des techniques de ski. Perspectives de prévention. Holded December, 17, 1998 (jury president: Pr D. Saragaglia).
Florent Véjux et Nicolas Picard: Évolution de la traumatologie des sports d'hiver: comparaison de 2 séries à 10 ans d'intervalle à Grenoble (1998–1999 et 2008–2009). Holded, September, 10, 2012 (Jury president: Pr D. Saragaglia).

Sumo

28

Yasuaki Nakagawa, Kazufumi Minami,
and Yuji Hattori

28.1 Introduction (a Brief Presentation of Sumo Wrestling)

The origin of Sumo is transmitted the bout with "Nomi-no-Sukune" and "Taima-no-Kehaya" in front of the Emperor Suinin at 23 B.C. in Japan. The winner was "Nomi-no-Sukune." Thereafter, Sumo has long been a traditional sport in Japan. Today, Nihon Sumo Kyokai is an organization that controls professional sumo wrestling, and the International Sumo Federation controls amateur sumo wrestling. Approximately 1000 sumo wrestlers belong to Nihon Sumo Kyokai, and 84 countries belong to the International Sumo Federation.

The first World Sumo Championship was held approximately 30 years ago in 1992, when the International Sumo Federation was established. The World Sumo Championships have been held in various countries such as Japan, Germany, and Brazil. The 23th World Championship was held in Japan at October in 2019. In 1996, in addition to the Asian Sumo Championships, championships were held in Africa, America, Europe, and the Pacific. People from many regions of the world have the opportunity to experience the emotionally charged atmosphere of sumo. We believe that sumo will bring about friendship that crosses racial boundaries and contribute to the advancement of international peace. With the aim of having the sport included in the Olympics, women's sumo has also been established. The first World Women's Sumo Championships were held in Aomori, Japan, in 2001. The 14th World Women's Sumo Championship was also held in Japan in 2019. Thus, sumo is enjoyed and practiced by many people, and has recently grown to become a worldwide sport.

The Continental Championships at each of the five continents are held every year. These championships are a great pleasure for the Japan Sumo Federation that has missions of promoting and developing the amateur Sumo wrestler in Japan. One attraction of Sumo is that nursed athlete fight with sense of courtesy, respect, and fair play in

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rounds that last only a few seconds. This is highly recognized as “body, soul and skill” as an essence of Sumo. As the International Sumo Federation was authorized as the fully recognized member of IOC in 2019, we expect Sumo will be an official sport in the Olympic Games in the near future, and we believe Sumo deserves the status.

In Sumo, the playing area is called “dohyo” (ring) which is 455 cm in diameter. There are two types of dohyo, “mori-dohyo” (Fig. 28.1a) and “hira-dohyo” (Fig. 28.1b). “Mori-dohyo” involves a regular square (6.7 × 6.7 m) and its height is 30–50 cm, in contrast, the height of “hira-dohyo” is 0 cm. “Mori-dohyo” is almost

Fig. 28.1 (**a**) “Mori-dohyo” has a regular square (6.7 x 6.7 m) and its height is determined from 30 to 50 cm. (**b**) The height of “hira-dohyo” is 0 cm

always used in competitions, and "hira-dohyo" is almost always used in training.

In official uniform, all sumo wrestlers wear a "Mawashi" (loincloth belt). Women sumo wrestlers wear a "Mawashi" over a leotard (Fig. 28.2a), and foreign sumo wrestlers are permitted to wear a "Mawashi" over underpants (Fig. 28.2b). Sumo wrestlers have little in the way of protective clothing; their main garb is the "Mawashi," making them prone to microorganism exposure in the

Fig. 28.2 (**a**) Women sumo wrestlers wearing "Mawashi" over their leotards. (**b**) Foreign sumo wrestlers are permitted to wear "Mawashi" over their underpants

Fig. 28.2 (continued)

dohyo. Sixteen bacterial genera were identified using a bacterial identification system that tested dohyo soil samples during the year. The number of identified bacterial species was 32. Even when the sumo wrestler scatters salt in the dohyo before the bout, there is a measurable amount of bacterial flora in dohyo soil; the salt does not act as an antibacterial agent [1].

28.2 Introduction of Unique Mechanics and Injury Risks in Sumo Wrestling

In sumo wrestling, there is a risk of impact to the cervical spine. Sumo wrestlers make contact with their heads and upper trunks during a "Tachiai" (initial charge) (Fig. 28.3), which is the same type of contact that occurs in American football, and their cervical spines suffer from the axial load. In Japan, many sumo wrestlers start to play sumo during their junior high school years. Professional sumo wrestlers have six championships per year, and the period of one championship is 15 days. Many collegiate sumo wrestlers have about 20 competitions per year, and about 20 matches per competition. In training, they have about 50 initial charges per day.

28.3 Epidemiology Including Percentage of Sports-Related Injury and Their Anatomic Locations

A high occurrence of injuries in sumo wrestlers has been suspected to occur because sumo wrestlers of very different heights and weights (for example, tall with short, heavy with light) fight each other. We investigated the characteristics and statistics of sumo injuries in 101 collegiate and 203 high school sumo wrestlers [2]. In sumo

Fig. 28.3 Sumo wrestlers make contact with their heads and upper trunks during a "Tachiai" (initial charge), and the right wrestler has an ideal "Tachiai"

wrestling, injury associated pain is most frequently reported in the lower back, neck, knees, and shoulders, while in major class collegiate wrestling, disabilities from injuries of the knee, shoulder, neck, and ankle are most commonly reported [2]) (Fig. 28.4a). In major class high school wrestling, injury-associated pain is most frequently reported in the lower back, wrist, neck, and fingers, and disabilities from injuries of the neck, lower back, finger, and shoulder are most commonly reported (Fig. 28.4b). The risk factors for cervical spine injuries were smaller height and lighter weight. The risk factors for lumber spine injuries were taller height and heavier weight. The risk factors for knee injuries were heavier weight and larger body mass index. The lighter sumo wrestlers tended to injure their shoulders. Key points: The longer a sumo wrestler's career is the higher the risk factor for neck, wrist, finger, and shoulder injuries; however, these risks are not seen for lower back and knee injuries. Therefore, we take care in teaching the beginner sumo wrestlers, especially in terms of lower back and knee injury prevention, especially when the beginner has a heavy weight.

We examined the pain and disability of experienced postgraduate student sumo wrestlers in their daily and sporting activities [3]. The three major problem sites in sumo wrestlers were the neck, lower back, and knees; however, neck problems did not become worse during postgraduate or retirement periods. Inversely, lower back and knee joint problems may become worse because of degenerative conditions such as spondylolysis and knee ligament injuries.

In the following, we discuss the top five sports-related injuries including their mechanisms such as cervical spine injuries, Burner syndrome, lumber spondylolysis (including lumbar spine disorder), anterior cruciate ligament injury (including knee osteoarthritis), and shoulder dislocation. Concussion and prevention plans are also reported.

28.4 Cervical Spine Injury

We examined radiological changes in the cervical spines of freshman collegiate and high school sumo wrestlers and the relationship between the radiological changes and the cervical spine

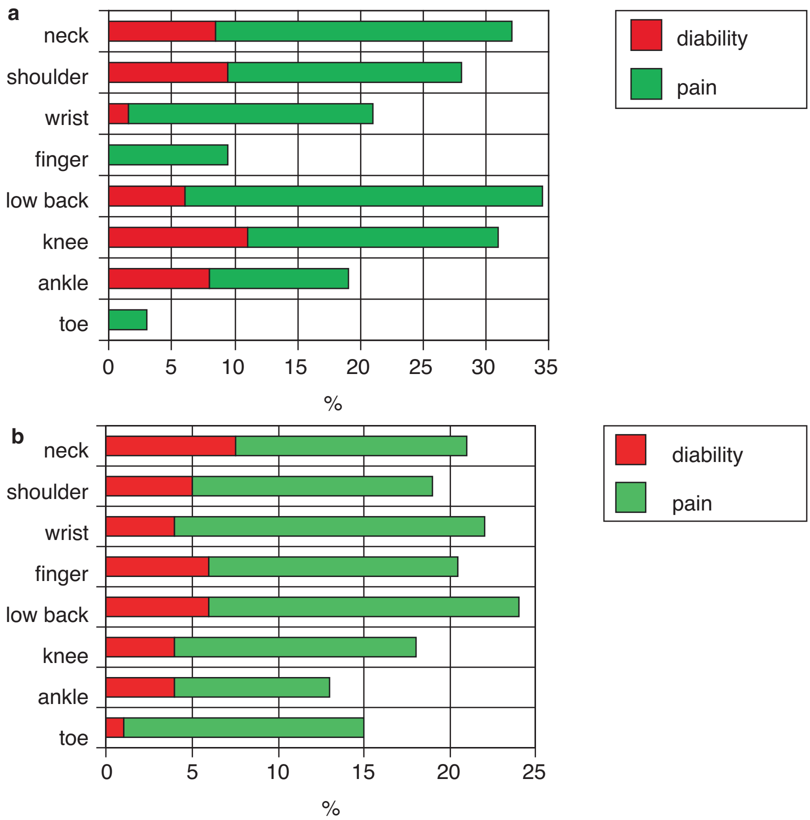

Fig. 28.4 The characteristics and statistics of sumo injuries. (**a**) Collegiate sumo wrestlers; (**b**) High school sumo wrestlers

Table 28.1 The mean physical items in high school and collegiate sumo wrestlers

	High school	Collegiate	*P* value
Height (cm)	173.3	175.1	0.1231
Weight (kg)	99.9	113.9	0.0001
BMI (kg/m^2)	33.1	37.1	0.0001
Sumo career (years)	6.4	8.5	0.0002
Training time (hours/week)	17.5	15.0	0.0028

symptoms [4]. For the medical check, 116 freshmen collegiate and 41 high school sumo wrestlers who belonged to a major class in the Japan Sumo Federation were examined using the following: cervical radiographs, a questionnaire about cervical symptoms, and their physique. We assessed their height, weight, body mass index, sumo career, and training time. The mean physical items in high school and collegiate sumo wrestlers are shown in Table 28.1. In the cervical radiographs, we assessed alignment, osteophyte formation, deformity of cervical body, disc space narrowing, and narrowing of the nerve root foramen. Fourteen high school (34%) and 46 collegiate (40%) wrestlers had some cervical symptoms. In 73%, lordosis disappeared; in 39%, osteophyte formation was observed (mainly in the third and fourth cervical spaces). In 6%, disc space narrowing was present (mainly in the fifth and sixth cervical spaces), and in 54%, cervical nerve root foramen narrowing was present (mainly in the third and fourth cervical foramen) (Fig. 28.5a). Disappearance of cervical lordosis, narrowing of nerve root foramen, and cervical body deformities occurred from an early stage. Osteophyte formation and disc space narrowing also occurred at a later stage. The correlation between cervical spine deformity and cervical symptoms was significant. Generally, sumo wrestlers make contact with their heads and upper trunks during an initial charge, the same type of contact that occurs in American football.

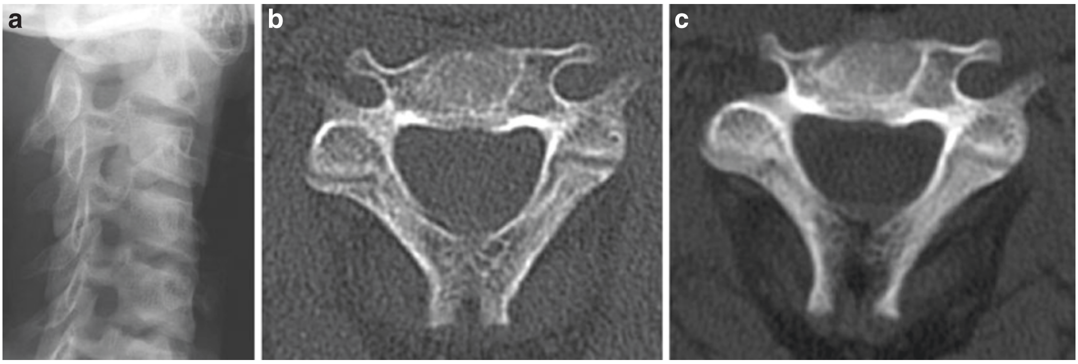

Fig. 28.5 (**a**) Narrowing in the third and fourth cervical foramen is shown. (**b**) A 12-year-old boy sumo player suffered from a C4 lamina fracture in "Tachiai" training. (**c**) His C4 fracture is showing almost union 6 months after his injury

Therefore, sumo wrestlers have severe axial load to their cervical spine in the initial charge. Compared with our previous study, we conclude here that the disappearance of lordosis and narrowing in the cervical nerve root foramen of C3/4 were due to the axial load wrestlers incurred in training during high school, and osteophyte formation and disc space narrowing of C5/6 that occurred in training during collegiate and postgraduate sumo wrestling.

The following discussion is based on seven previous reports of cervical spinal fractures or cord injuries associated with sumo wrestling [5–11]. One injury occurred in a 16-year-old recreational sumo wrestler who pushed his opponent with his head on the initial charge and suffered neck hyperextension, which led to a fracture of C2 and cervical cord injury [5]. Another case involved a C1-C2 dislocation and a cervical spinal cord injury in an 18-year-old boy with Os odontoideum who was thrown down [10]. A third case was a C4-C5 cervical disc herniation in a 25-year-old professional sumo wrestler due to repeated constant force on the neck during the initial charge [11]. Nakagawa reported another two cases. One involved a 31-year-old amateur sumo wrestler who fell on his crown with his neck held by his opponent and suffered an atlantoaxial dislocation and a central cervical spinal cord injury [6]. The other case involved a high-level collegiate sumo wrestler who suffered anterior dislocation of C7 on Th1 and a cervical spinal cord injury [7]. This wrestler was pushed down near the edge of the dohyo while the back of his head was placed on the abdomen of his opponent, resulting in hyperflexion of his cervical spine. Tateishi reported on six professional sumo wrestlers who developed a Jefferson fracture [9]. Shimizu also reported four other professional sumo wrestlers with Jefferson fractures, one with an odontoid fracture and one with a cervical spinal process fracture [8]. Almost all injuries occurred during a head crash referred to as "Tachiai." All Jefferson fractures were diagnosed using CT scans. The wrestlers did not develop degenerative changes and did not have congenital abnormalities except for one wrestler who had Os odontoideum.

Recently, we treated a 12-year-old boy sumo wrestler who suffered from a C4 lamina fracture (Fig. 28.5b). In training, he made contact the chest of a professional sumo wrestler whose weight was about 180 kg using his head and upper trunk, the move is known as a "Tachiai." Immediately after, he had severe neck and back pain, but no motor and sensory dysfunction. He subsequently wore a hard brace from his neck to his chest for about 4 months, by the end of the 4 months, there was almost union with the C4 fracture (Fig. 28.5c). These outcomes show that closer attention to training of elementary and junior high school players is needed.

28.5 Burner Syndrome

As sumo wrestlers make contact with their heads and upper trunks during an initial charge, it is assumed that many experience Burner syndrome. We examined the frequency and degree of severity of the Burner syndrome in sumo wrestlers [12]. Questionnaires were completed by Japanese collegiate (N = 101) and high school (N = 203) sumo wrestlers. We assessed aspects of their physique such as height, weight, body mass index, and age, and the frequency of Burner pain about the neck and pain radiating into the hand. Ninety-one percent of collegiate major-class sumo wrestlers and 79% of high school major-class sumo wrestlers experienced intense burning neck pain. *Key point: The risk factors for this pain were smaller height and lighter weight*. Fourteen percent of those in the collegiate major class and 3.5% of the high school major-class sumo wrestlers could not wrestle for more than 1 week because of the Burner syndrome. On examination, eight wrestlers (11%) from the former group had prolonged symptoms, such as loss of grasping power, with eight wrestlers (4%) in the high school group also being affected by prolonged symptoms. This "Burner syndrome" represents one of the most common injuries seen in sumo. Although many sumo wrestlers have only mild symptoms, a few have prolonged symptoms.

28.6 Lumber Spondylolysis

The mechanism of spondylolysis in sumo is torsion, extension, or sometimes flexion of the lumber spine [13]. Sumo wrestlers often experience hyperextension of the lumber spine when pushed down near the edge of the dohyo. Hyperextension from torsion of the lumber spine can also occur if a wrestler throws his heavy opponent down. Because "Mawashi" movements are associated with restriction of the lower lumber spine of a sumo wrestler, this can protect the region from L4 to S1. However, herniation of the upper lumber spine (from L2 to L4) can sometimes occur. It is characteristic of lumber spinal injuries in sumo wrestling (Fig. 28.6a).

We examined the radiological changes in the lumbar spines of freshman collegiate and high school sumo wrestlers, and evaluated the relationship between the radiological changes and the lumbar spine symptoms [14]. For the medical checks, 78 freshman collegiate sumo wrestlers and 16 high school sumo wrestlers who belonged to the major class of the Japan Sumo Federation were examined using the following: physical assessments, questionnaires about lumbar spine symptoms, and radiographs of the lumbar spine. We assessed parameters such as height, weight, body mass index, length of sumo career, and training time. In the lumbar radiographs, we assessed for osteophyte formation, deformity of the lumbar spine, spondylosis, and narrowing of the disc space. The weight, body mass index, and length of sumo career of the collegiate sumo wrestlers were significantly larger and longer those of the high school sumo wrestlers. The high school group that had symptomatic lumbar spine injuries consisted of 10 cases (63%), and the symptomatic collegiate group had 43 cases (55%). There were no significant differences between the groups. One high school wrestler (6%) and seven collegiate wrestlers (9%) demonstrated osteophyte formation in the lumbar bodies (mainly in the third lumbar body). Seven high school wrestlers (44%) and 25 collegiate wrestlers (32%) lumbar body deformities found mostly in the fourth and fifth lumbar bodies. Five high school sumo wrestlers (31%) and 9 collegiate sumo wrestlers (12%) had spondylolysis (Fig. 28.6b) that existed mainly in the fifth lumbar body; this consisted of mainly the pseudoarthrosis type. In the only occurrence rate of spondylolysis, there was a significant difference between high school sumo wrestlers and collegiate wrestlers. No sumo wrestlers had spondylolisthesis. None of the high school wrestlers (0%) and four collegiate wrestlers (5%) had disc space narrowing. There were 9 high school wrestlers (56%) and 32 collegiate wrestlers (41%) who had degenerative radiographic changes, such as osteophyte formation, lumbar spine deformities, spondylolysis, and the disc space narrowing. There was no relationship between degenerative radiographic changes and the physical parame-

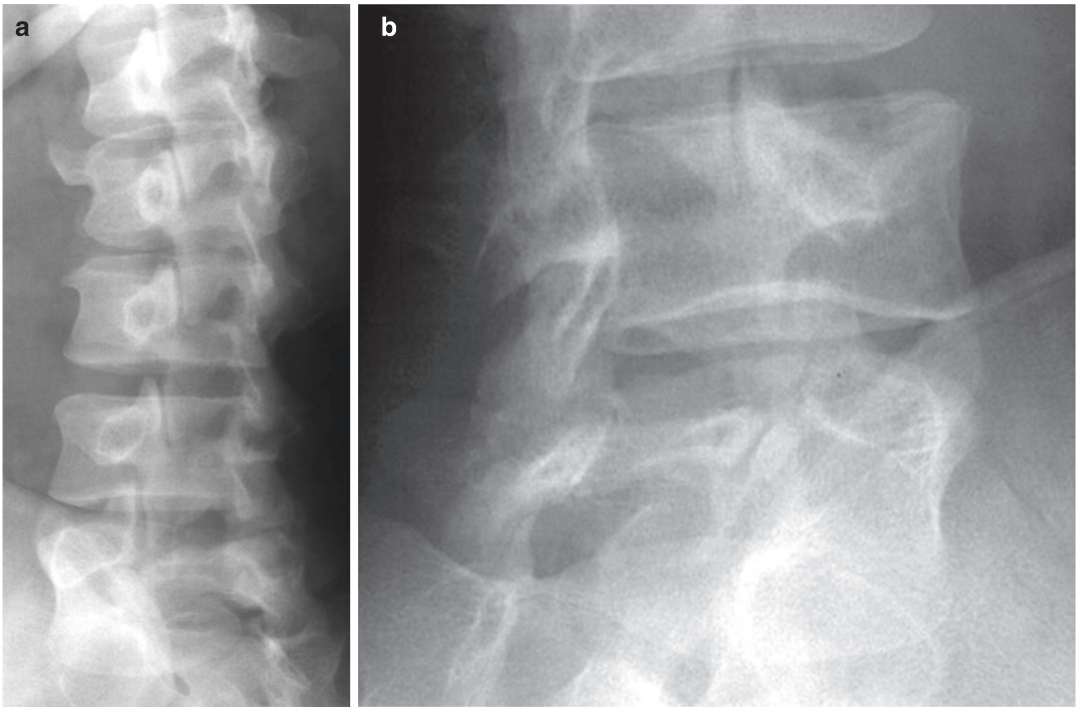

Fig. 28.6 (**a**) Because "Mawashi" restricts lower lumber spine movement of the sumo wrestler, no degenerative changes in the lower spine were observed. However, the wrestler had a large osteophyte formation in the upper body of L2. He was 28-years-old, and his sumo career was 16 years. (**b**) Spondylolysis with pseudo-arthrosis in L5

ters assessed. In assessing the relationship between lumbar symptoms and radiological changes, a correlation between spondylolysis and lumbar symptoms was the only significant correlation observed. Key point: The occurrence ratio of spondylolysis in high school sumo wrestlers was significantly larger than that for collegiate wrestlers, and the correlation between spondylolysis and lumbar symptoms was significant. Therefore, it may be recommended that sumo wrestlers discontinue this sport if symptomatic spondylolysis develops. Elementary school age sumo wrestlers have increased in Japan, and some of them had spondylolysis at approximately 10 years old. Early detection and treatment of spondylolysis are therefore needed. In the radiological changes noticed for professional sumo wrestlers who had lumbago, the occurrence ratios of spondylolysis and disc ballooning were larger than those who had not lumbago. In assessing the relationship between the physical parameters and radiological changes in professional sumo wrestlers, older age, longer sumo career, taller body height, and heavier body weight were risk factors. We also examined the radiological changes of the lumbar spine in 94 freshman sumo wrestlers who belonged to a major class of the Japan Sumo Federation. Half of the wrestlers had some abnormal findings on their lumbar radiography. Early detection and treatment of spondylolysis are therefore needed because it may decrease sports performance in sumo wrestlers.

28.7 Knee Osteoarthritis and Ligament Injury

We previously reported that even in freshman sumo wrestlers, 44% had some abnormal knee joints findings, and that these risk factors were heavy bodyweight and large body mass index [15]. The purpose of this study was to examine the radiological changes of the knee joint in high school sumo wrestlers, and to compare their radiological changes with those of freshman collegiate sumo wrestlers. Thirty-five high school sumo wrestlers and 106 freshman sumo wrestlers who belonged to the Japan Sumo Federation underwent routine radiographic examination of their knee joints and answered questionnaire on knee symptoms. In the knee radiographs, we assessed joint space narrowing, osteophyte formation, bony sclerosis, sharpening of intercondylar eminence. High school sumo wrestlers were significantly smaller than collegiate wrestlers in weight, body mass index, and sumo career; however, they had significantly longer training times than collegiate wrestlers. Seventeen high school wrestlers (48.6%) and 51 collegiate wrestlers (48.1%) had some type of knee symptom. Overall, 4 knees of 3 high school wrestlers (8.6%) and 23 knees of 15 collegiate wrestlers (14.2%) were observed to have joint space narrowing. In addition, 4 knees of 4 high school wrestlers (11.4%) and 24 knees of 19 collegiate ones (17.9%) were observed to have osteophyte formation (mainly in the medial compartment). The risk factors for these phenomena were heavy bodyweight, large body mass index, and short sumo career. One knee of one high school wrestler (2.9%) and six knees of four collegiate wrestlers (3.8%) had bony sclerosis. There were 9 knees of 7 high school wrestlers (20.0%) and 52 knees of 37 collegiate wrestlers (34.9%) that had a sharp intercondylar eminence. Eleven high school wrestlers (31.4%) and 46 collegiate wrestlers (43.4%) were observed to have some abnormal findings on their knee radiography. The correlation between the osteophyte formation, bony sclerosis, and their knee joint syndrome was significant. Only the rate of intercondylar eminence sharpening of collegiate wrestlers was significantly larger than high school wrestlers. The intercondylar eminence sharpening of their knee joints increased chronologically. However, the risk factor for joint space narrowing and osteophyte formation was short sumo career. Probably because of their heavy weight and large body mass index, several sumo wrestlers had joint space narrowing and osteophyte formation in their knee joints at an early stage such as high school years. In conclusion, one-third or two-fifths of sumo wrestlers had some abnormal findings on their knee joint radiography. Knee osteoarthritic changes of sumo wrestlers appeared as early as high school.

Anterior cruciate ligament (ACL) rupture is one of the most important injuries in sumo wrestlers. ACL rupture occurs frequently, just as in other contact sports. The mechanism of almost all ACL ruptures in sumo wrestlers resembled non-contact injury patterns as seen in handball, basketball [16] and rugby [17]. Shimizu recommended conservative or surgical treatment for ACL injuries in professional sumo wrestlers [18]. The age of wrestlers treated in the conservative (C) group was significantly older than the age of those in the reconstructive (R) group. The time it took to return to play was 2 months in C group and 7 months in R group. The time in the C group is significantly shorter than that in R group. At 2 years after their injuries, the rank in R group became comparable to that in C group, and thereafter the rank in R group is prone to outrun that in C group. In the R group, wrestlers were more likely to experience left knee injuries more so than right injuries. The left knee may need more stability than the right since sumo wrestlers maintain their balance with the left leg near the edge of dohyo when he plays the defensive side during pushing training. However, the re-tear rate of reconstructed ACLs in professional sumo wrestlers for at approximately 30 months was found to be 11%, and the rate of contralateral ACL rupture was 11%. Sumo wrestlers who had ACL reconstruction took approximately 18 months to experience complete healing of their ACL [19]. Nagase reported that the period to return to play from ACL reconstruction using bone tendon bone (BTB) was 6.6 months in players more than 100 kg [20]. Mae discussed the rates of returning to play in contact sport players who underwent anatomic rectangular-tunnel BTB ACL reconstruction [21]. Most players could resume their previous sport activities (returning to play (RTP) 8.4 months), but had a very high rate (19%; 17 months after reconstruction) of graft tearing after they had returned to play.

It appears that sumo wrestlers with ACL dysfunction may feel no dysfunction when pushing opponent. However, it is likely that sumo wrestlers with ACL dysfunction cannot protect their body when their opponent pushes them, or when they move to the side. Previously, we recommended a 6 months period of rest in sumo wrestlers after ACL reconstruction before returning to play. Following these protocols, they maintained their good condition for 1 year after their ACL reconstruction, but thereafter, the reconstructed ACL became loose probably because of their heavy weight. Therefore, today we recommend the following therapy for sumo wrestlers who experience ACL rupture: conservative groups include more than 25-year-old sumo wrestlers, or those utilizing pushing type of sumo technique, and reconstructive groups involve less than 25-year-old or lighter weight sumo wrestlers or those who do not utilize pushing type of sumo technique. If we decide to perform ACL reconstruction, we generally aim for a 1-year return to play schedule. Fortunately, with this protocol, all our sumo wrestlers who underwent ACL reconstruction using their hamstrings recovered completely and were able to return to play 1 year later with no re-tear occurrence.

However, returning to play for sumo wrestlers with a knee dislocation is difficult. Shimizu reported on two cases of multiple ligament injured knees with common peroneal nerve avulsion injuries in professional sumo wrestlers [22]. Unfortunately, two wrestlers could not return to play completely. We also experienced a 20-year-old collegiate sumo wrestler with a knee dislocation. His left knee was added in severe valgus stress, which led to left knee dislocation when he endured the opponent's pushing near the edge of dohyo (Fig. 28.7b). His anterior cruciate, posterior cruciate, and medial collateral ligament ruptures are shown in Fig. 28.7c, d. At 1 month after his injury, we reconstructed his ACL (using the ipsilateral semitendinosus and gracilis (STG)), PCL (using the contralateral STG) (Fig. 28.7e), and MCL (using the advancement technique) (Fig. 28.7f). At 5 months after his surgery, he started basic sumo training. At 17 months after his surgery, he was able to participate in sumo competitions. He had no knee pain and no feelings of instability; however, he rated his sumo performance at only 80% at 2 years after his surgery (Fig. 28.7g).

28.8 Shoulder Dislocation

Shoulder pain was fourth, and shoulder disability was the second most common problem for sumo wrestlers [23]. In this report, 23% of sumo wrestlers had anterior shoulder dislocation [23]. Sumo wrestlers generally have a 90-degree external rotation and abduction as a typical anterior shoulder dislocation position when the pushed arm is driven up to his opponent, or when his outside arm is pushed up by the externally rotated inside aspect of his opponent's arm. If the sumo wrestler is able to change his playing style as he tightens his axilla, he may avoid a typical anterior shoulder dislocation position, and thereafter, wrestle relatively safely. If he cannot play so, he must undergo the operation. We recommend the Bristow–Bankart method to sumo wrestlers who have recurrent anterior shoulder dislocation. In this technique, the wrestler can start to play 3 months after the operation, and return completely 6 months after.

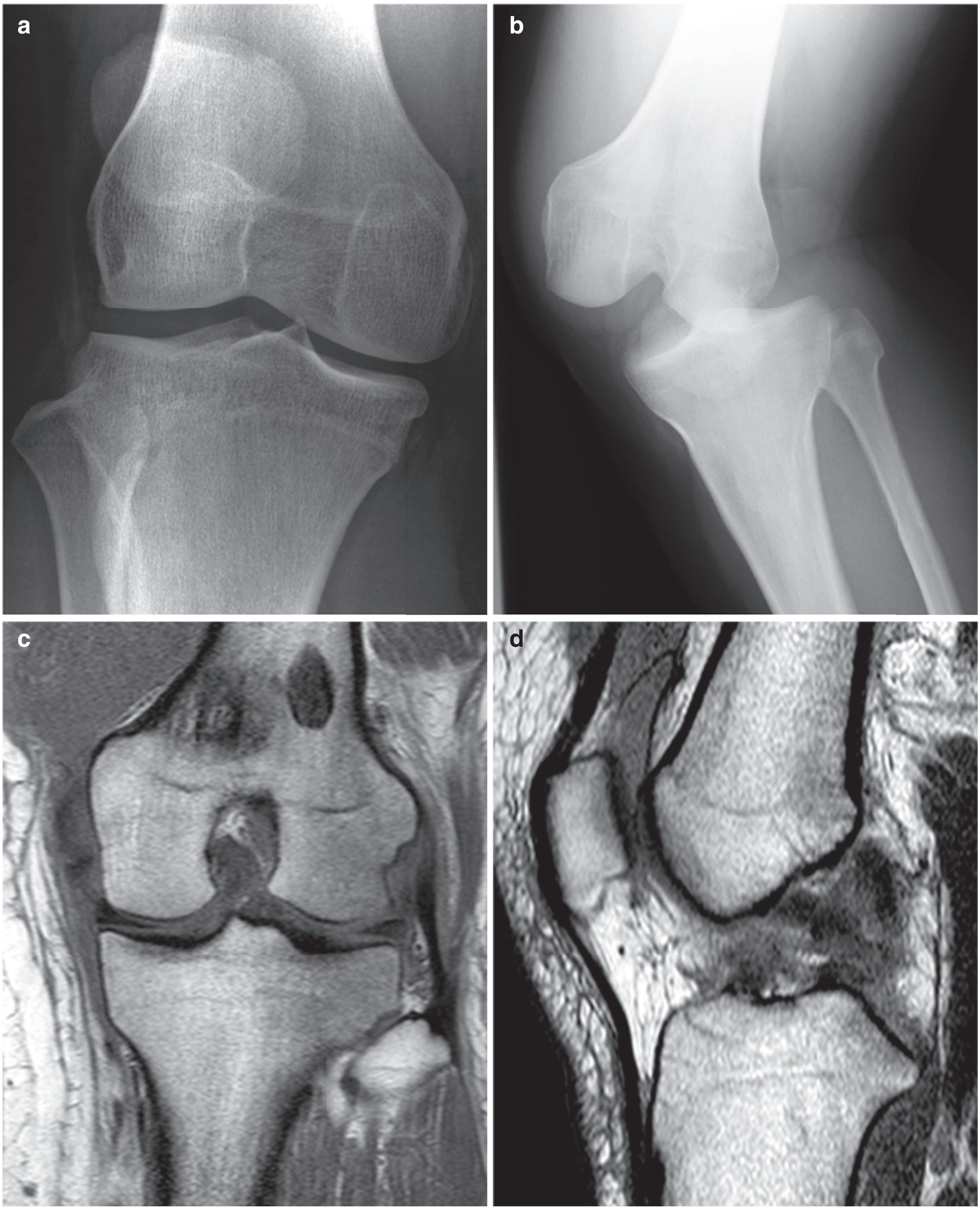

Fig. 28.7 (**a**) Osteophyte formation within the medial tibia of the right knee, (**b**) left knee dislocation indicated on radiography, (**c**) MCL rupture shown on MRI, (**d**) ACL and PCL ruptures shown on MRI, (**e**) ACL and PCL reconstruction in arthroscopy, (**f**) MCL reconstruction with the advancement technique, and (**g**) left knee radiography at 2 years after surgery

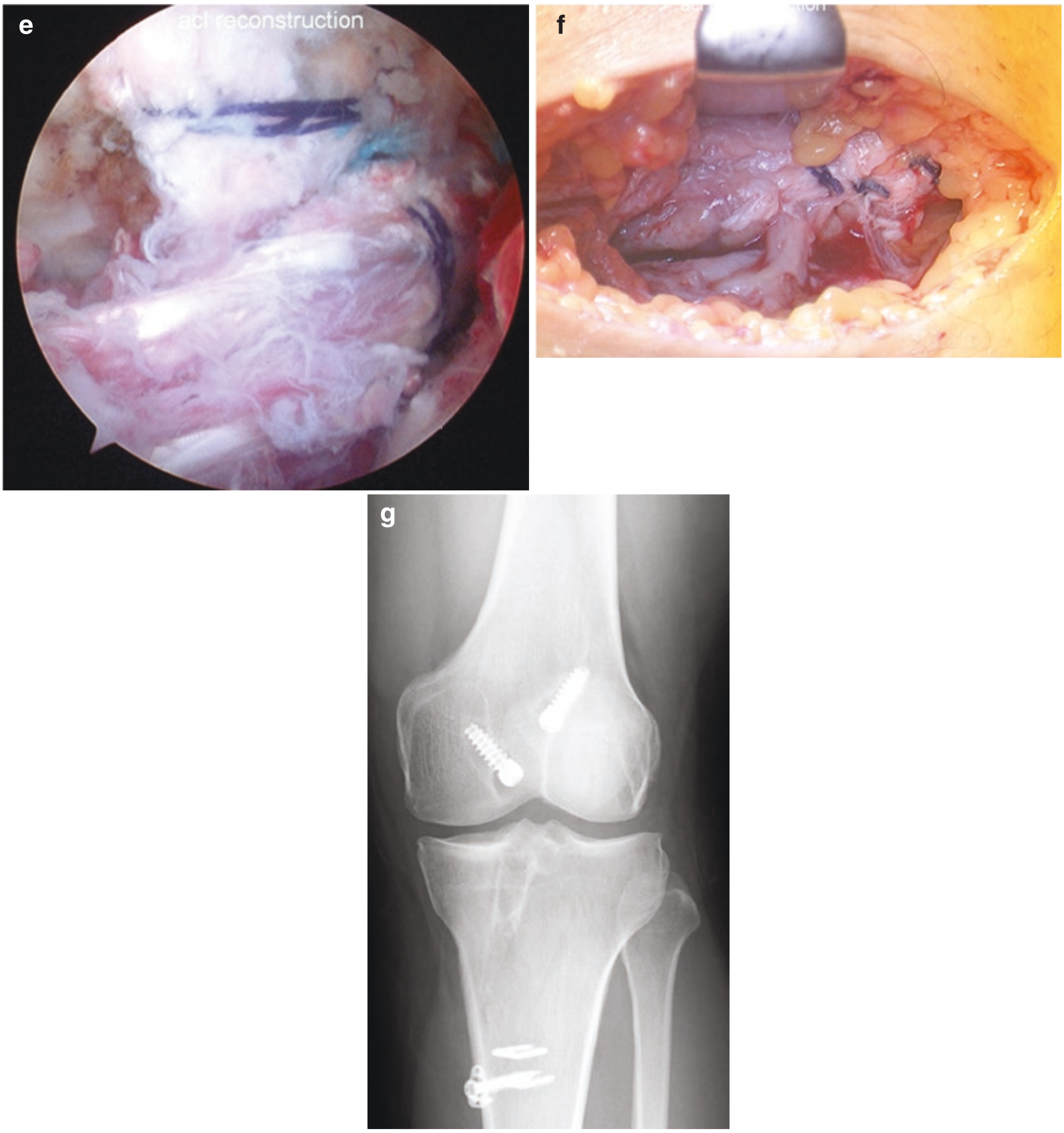

Fig. 28.7 (continued)

28.9 Concussion

Because sumo wrestlers cannot participate in position lying down, they work hard defensively when he falls down. Therefore, the occurrence rate of severe head and cervical spine injuries is small [13].

Concussions occur sometimes in sumo wrestling; therefore, the Japan Sumo Federation created concussion guidelines in 2019. We experienced the suggestive report of the occurrence rate of concussion in Japan women sumo championship in 2016. The occurrence rate of concussions is shown in Table 28.2. We interpreted this data as follows: Because the elementary school player has small power, and more than high school player has high power and the technique of her defensive position, she has no concussion when she falls down. Because junior high school players have high power, but very limited technique for maintaining a defensive position, concussions may occur when the player falls down. Therefore, it is important to teach players the correct defensive position and techniques when she falls down, especially junior high school players.

Yamashiro reported on a 14-year-old sumo wrestler who had a traumatic vertebral artery dissection due to sumo practice [24]. His early symptoms were similar to a concussion. Traumatic brain injury has been of great concern in recent years as certain contact sports have been gaining in popularity. Sumo is one of the most physically demanding sports involving frequent head-on-head impact. The risk of vertebra-basilar artery dissection during sumo wrestling should be addressed to secure the safety of young players.

Table 28.2 The occurrence rate of concussion in Japan women sumo championship in 2016

	Participate wrestlers	Concussion occurred	Occurrence rate (%)
Elementary school	86	0	0
Junior high school	60	5	8.3
More than senior high school	66	0	0

The occurrence rate of junior high school is significantly higher than another two groups

28.10 Unique Prevention Plans

Some reports have mentioned that the changes in rules have decreased cervical spine injuries in both rugby [25] and American football [26]. The refereeing regulations of the Japan Sumo Federation prohibit techniques that are considered risky with the aim of preventing injuries, and these regulations are applied at sumo championships to all wrestlers below junior high school level. Techniques thought to be risky include strong gripping of the neck of an opponent below the axilla (Fig. 28.8a), fixing the back of one's head onto the abdomen of an opponent (Fig. 28.8b), and fixing both partners' necks under both their axillae. If a junior sumo wrestler uses one of these techniques, the bout is immediately stopped and a rematch is played. If such bouts are allowed to continue, they may result in an injury similar to the above described in our patients. When a small wrestler fights a larger one, the ideal style is fixing one's forehead under the chin of the opponent (Fig. 28.8c). To decrease cervical spinal cord injuries in sumo, these refereeing regulations should be strictly obeyed, and all sumo wrestlers should be taught the above ideal style. If a sumo wrestler is placed in a risky situation as in our patient's case [7], he should be instructed to remove his head from the abdomen of his opponent. We have also previously described a case of a high-level collegiate sumo wrestler who suffered anterior dislocation of C7 on Th1 and a cervical spinal cord injury [7]. The wrestler was pushed down near the edge of the dohyo while the back of his head was placed on the abdomen of his opponent, resulting in hyperflexion of his cervical spine. We

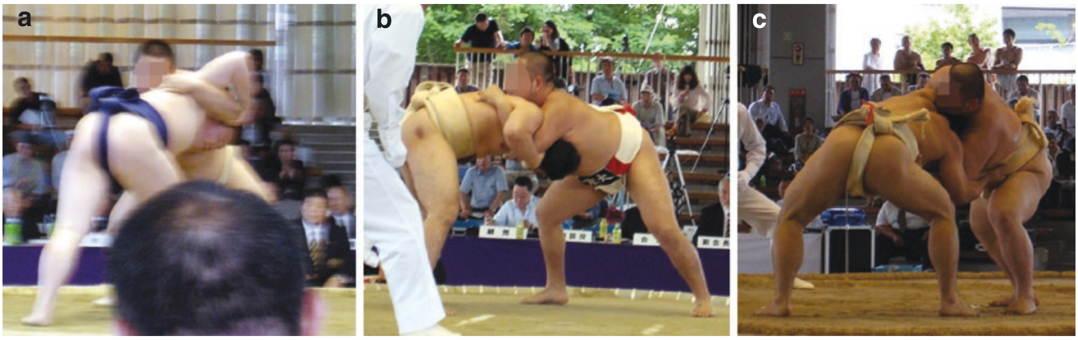

Fig. 28.8 (**a**) The neck position of left wrestler is risky, strong neck gripping of an opponent below the axilla. (**b**) The neck position of left wrestler is risky, fixing the back of one's head onto the abdomen of an opponent. (**c**) The ideal style (left wrestler) is fixing one's forehead under the chin of the opponent

suggest that measures be taken to improve the education of sumo wrestlers in preventive techniques to decrease the incidence of cervical spinal cord injuries.

Tsuchiya investigated knee muscle strength and body fat in 69 highly-ranked professional sumo wrestlers using CYBEX NORM and BIODEX, and for body fat by BODPOD [27]. Peak torque of the knee extensor averaged 461 Nm in the "Makuuchi" rank (Division I), 457 in the "Juryo" (Division II), and 457 in the "Makushita" (Division III). WBI (peak torque of knee extensor per body weight) averaged 0.88, 0.86, and 0.75, respectively. Isokinetic muscle strength decreased according to an increase in the angular velocity; however, higher ranked sumo wrestlers were stronger than the lower ranked ones at the high angular velocity. When the percentage body fat increased, the peak torque and WBI decreased statistically. The relation between WBI and body weight was significantly linear. When body weight was below 180 kg, there were many sumo wrestlers who had a high value for WBI. However, when body weight exceeded 180 kg, all sumo wrestlers except one were not able to score beyond 0.7 for WBI. Therefore, sumo wrestlers must keep their body weight under 180 kg. Considering the results after the muscle examination for 2 years, those wrestlers who had good muscle strength were promoted in their sumo rank compared to the weaker ones. Patterns in promotions in sumo ranking over a 2-year period indicated that sumo wrestlers did not suffer from any severe sumo injuries during that time. Therefore, muscle strengthening exercises are useful for preventing sumo injuries.

ACL injury occurs frequently, and is one of the most serious problems in sumo wrestling. A preventive counterplan for ACL injury is coming major theme to be considered in future.

Take-Home Messages

1. Sumo wrestlers have severe axial load to their cervical spine in the initial charge. The disappearance of lordosis and narrowing in the cervical nerve root foramen of C3/4 was due to the axial load wrestlers incurred in training during high school, and osteophyte formation and disc space narrowing of C5/6 that occurred in training during collegiate and postgraduate sumo wrestling.
2. Because "Mawashi" movements are associated with restriction of the lower lumber spine of a sumo wrestler, this can protect the region from L4 to S1.

However, herniation of the upper lumber spine (from L2 to L4) can sometimes occur. It is characteristic of lumber spinal injuries in sumo wrestling.
3. It appears that sumo wrestlers with ACL dysfunction may feel no dysfunction when pushing opponent. However, it is likely that sumo wrestlers with ACL dysfunction cannot protect their body when their opponent pushes them, or when they move to the side. We recommend the following therapy for sumo wrestlers who experience ACL rupture: conservative groups include more than 25-year-old sumo wrestlers, or those utilizing pushing type of sumo technique, and reconstructive groups involve less than 25-year-old or lighter weight sumo wrestlers or those who do not utilize pushing type of sumo technique. If we decide to perform ACL reconstruction, we generally aim for a 1-year return to play schedule.

References

1. Osafune T, Mitsuboshi M, Ito T, Aoki S, Ehara T, Hashiguchi H, Minami K. Analysis of bacterial flora in dohyo soll. Environ Health Prev Med. 2007;12:11–6.
2. Nakagawa Y, Matsusue Y, Nakamura T. An epidemiological study of injuries in student sumo wrestlers. Jpn J Orthop Sports Med. 1998;18:313–9.
3. Nakagawa Y, Hattori Y, Tanaka E, Suzuki T, Gotou T, Okamoto Y, Nakamura T. Orthopaedic study of experienced post-graduate student sumo wrestlers. Japanese J Clin Sports Med. 2005;13:56–60. (in Japanese)
4. Nakagawa Y, Mukai S, Minami K, Hattori Y, Nakamura T. Radiological changes in the cervical spine in freshman collegiate sumo wrestlers. Orthop J Sports Med. 2017 Dec 14;5(12):232596711774421.
5. Kidokoro Y, Kobayashi K, Yamagishi M. Injuries of the spine and the spinal cord in sports. Seikeigeka. 1979;30:629–37. (in Japanese)
6. Nakagawa Y, Miki T, Nakamura T. Atlantoaxial dislocation in a sumo wrestler. Clin J Sports Med. 1998;8:237–40.
7. Nakagawa Y, Minami K, Arai T, Okamura Y, Nakamura T. Cervical cord injury witnessed in the field in sumo wrestling: a case report. Am J Sports Med. 2004;32:1054–8.
8. Shimizu S, Tateishi T, Nagase T, Nakagawa T, Tsuchiya M. Study of cervical spine disorder in sumo wrestlers. Japanese J Orthop Sports Med. 2009;29:164–7. (in Japanese)
9. Tateishi T, Motosugi N, Asahina S, Nakagawa T, Tsuchiya M. Jefferson's fracture in professional sumo wrestlers: report of 6 cases. Japanese J Orthop Sports Med. 2005;25:231–5. (in Japanese)
10. Tominaga S, Date K, Katayama M. Cervical spinal and cord injuries due to sports in middle and high school-boys. Jpn J Orthop Sports Med. 1989;8:157–61. (in Japanese)
11. Yanagisawa Y, Yamauchi H, Tsuji T. A case report of cervical disc herniation caused by sumo. Jpn J Orthop Sports Med. 1991;10:73–6. (in Japanese)
12. Nakagawa Y, Matsusue Y, Nakamura T. Burner syndrome in sumo players. International meeting of sports science commemorating the 1998 winter Olympics in Nagano 1998; 237–245.
13. Minami K. Lumbago in Sumo. Clin Sports Med 2012; 29: suppl. 352–359 (in Japanese).
14. Nakagawa Y, Hattori Y, Nakamura T. Radiological changes of lumber spine in high school and collegiate sumo wrestlers. Japanese J Orthop Sports Med. 2009;29:83–7. (in Japanese)
15. Nakagawa Y, Suzuki T, Goto T, Okamoto Y, Hattori Y, Nakamura T. Radiological changes in the knee joints in freshman collegiate sumo wrestlers. Japanese J Orthop Sports Med. 2004;24:296–301. (in Japanese)
16. Goshima K, Kitaoka K, Shima Y, Tomita K. The mechanisms of anterior cruciate ligament injuries in student sumo. Japanese J Clin Sports Med. 2010;18:310–3. (in Japanese)
17. Sunagawa N, Hirose N, Kuramochi R, Takemura M, Tsuchiya A, Fukubayashi T. Analysis of indirect ACL injuries in rugby layers-classification of injury mechanisms- Japanese J. Clin Sports Med. 2015;23:427–32. (in Japanese)
18. Simizu S, Tateishi T, Nagase T, Motosugi N, Nakagawa T, Tsuchiya M. Study of anterior cruciate ligament injuries in professional sumo wrestlers. Japanese J Orthop Sports Med. 2009;30:119–23. (in Japanese)
19. Nagase T, Tateishi T, Shimizu S, Saito R, Tsuchiya M, Nakagawa T. Analysis of complications and return to match after anterior cruciate ligament reconstruction for professional sumo wrestlers. Japanese J Orthop Sports Med. 2017;37:235–8. (in Japanese)
20. Nagase T, Tateishi T, Yuki A, Nakagawa T, Tsuchiya M. Clinical outcome of anterior cruciate ligament reconstruction for super-heavy weight athlete. Japanese J Orthop Sports Med. 2015;35:181–3. (in Japanese)
21. Mae T, Shino K, Hiramatsu K, Tachibana Y, Kita K, Nakata K. Return to play after anterior cruciate ligament reconstruction in contact sport players. Japanese J Clin Sports Med. 2013;21:165–9. (in Japanese)

22. Shimizu S, Nagase T, Tateishi T, Nakagawa T, Tsuchiya M. Multiple-ligament injured knee with common peroneal nerve avulsion injury in professional sumo wrestlers: report of two cases. Japanese J Orthop Sports Med. 2017;37:158–62. (in Japanese)
23. Nakagawa Y, Matsusue Y, Nakamura T. The problems in the shoulder joints of student sumo wrestlers. Shoulder Joint. 1998;22:161–4. (in Japanese)
24. Yamashiro S, Hitoshi Y, Yoshida A, Kuratsu J. Traumatic vertebral artery dissection due to Sumo practice: a case report. Japanese J Clin Sports Med. 2015;23:261–5. (in Japanese)
25. Silver JR, Stewart D. The prevention of spinal injuries in rugby football. Paraplegia. 1994;32:442–53.
26. Torg JS, Vegso JJ, O'Neill MJ, Sennet B. The epidemiologic, pathologic, biomechanical, and cinematographic analysis of football-induced cervical spine trauma. Am J Sport Med. 1990;18:50–7.
27. Tsuchiya M, Onishi S, Otani T, Saito A, Wakano K, Kinoshita N, Miyake F, Yamazaki H, Yoshida H. Investigation of knee muscle strength and body fat in sumo wrestlers. Japanese J Clin Sports Med. 2003;11:469–76. (in Japanese)

29 Swimming, Open-Water Swimming, and Diving

Ivan Córcoles Martínez and Jaume Calmet Garcia

29.1 Epidemiology

Between 2009 and 2014, 1.54/1000 male swimmers exposed got injured (83.2% swimmers, 16.8% divers) and 1.7/1000 female swimmers (82.2% swimmers, 17.8 divers) without significant differences between men and women. [1, 2].

In Junge's study during the Olympic Games in Beijing, 73.8% of Olympians were injured during competitions and 26.2% during training. However, it was not like this in water sports, in swimming 62.5% of injuries occurred during training and in divers was 100% of injuries [3]. This data is similar to 83.2% training injuries and 16.2% competitions injuries in Kerr's study [1].

Besides of injuries, athletes may also have sports-related illnesses. For example, in the last Olympic Games in Rio de Janeiro 2016, 12% of jumpers (diving) and 12% of open-water marathon swimmers suffered respiratory problems (47%), gastrointestinal problems (21%) among other pathologies [4].

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29.2 Swimming

29.2.1 Introduction

Swimming is a very practiced sport around the world and highly recommended by many health professionals because buoyancy in water makes it a sport without impact. However, swimming requires a very high training load at some levels of competition, which can cause a very specific pathology in different joints such as shoulder, spine, knee, and hip.

There are four different styles, freestyle, backstroke, breaststroke, and butterfly, and swimmers tend to specialize in one particular stroke during competitions.

29.2.2 Shoulder

29.2.2.1 Epidemiology

Shoulder is the most often injured joint in swimmers. It represents 31–36% of swimmer injuries, and the prevalence of suffering shoulder pain is 40–90%. There is no different predisposition between men or women, between non-dominant or dominant limb, long distances or in short distances, and at any phase of stroke [5, 6].

The shoulder is the most affected joint because 90% of the swimming propulsion strength is done with the upper limbs [7].

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Table 29.1 Shoulder pain causes

Extrinsic	
	Training volume
	Technique details
	Hand paddles
Intrinsic	
	Excessive laxity/general joint hypermobility
	Scapular dyskinesis
	Rotator cuff imbalance
	Thoracic kyphosis

29.2.2.2 Etiology

Shoulder pain can be caused by different factors, some extrinsic to the shoulder (training volume, technique details, hand paddles) and other intrinsic (hyperlaxity, scapular dyskinesis, rotator cuff imbalance) Table 29.1 [8, 9].

Extrinsic Factors

- Training volume:
  - It is the main reason swimmers have shoulder pain. Many swimmers make high-volume of training, not just elite ones. They can do until 100 km per week complemented with dryland training program. It is estimated that a swimmer can make one million strokes a year with each arm [1, 4, 6, 10, 11]. The number of hours swum/week is correlated significantly with supraspinatus tendinopathy [12] .
  - The repetition of the same movements produces several changes in the glenohumeral joint at the rotator cuff, the antero-inferior joint capsule, and the thoracic scapula joint [13, 14].
- Technique details:
  - The main cause of pain is subacromial impingement that occurs during 23.2–26.5% of the stroke cycle [15].
  - Shoulder pain will depend on individual and the personal anatomical characteristics that delimit subacromial space and hyperlaxity.

Freestyle Stroke

There are three phases in the freestyle swimmer stroke:

1. Initial phase stroke:
   - The hand goes into the water forward and lateral to the head, medial to the shoulder and with all the fingers at once. If the hand enters more medial to the head increases the subacromial impingement and if the thumb enters first we are placing the shoulder in internal rotation and increases the stresses of the biceps attachment to the anterior labrum (Fig. 29.1) [16].
   - In this initial phase the shoulder gets its highest elevation angle and could originate an impingement. To avoid this, you should limit the extension and train the strength and stretching of the antagonistic muscles to elevation (latissimus dorsi, pectoralis major, teres major, biceps brachii) [15].
2. Pull phase.
   - During this phase the hand describes an S-shape that goes first from the middle line to deep side and outside. When the hand reaches the deepest area, it goes toward the surface and the midline (at chest height) to finally goes to the lateral side of the thigh with the elbow fully extended. This phase ends when the hand comes out of the water.

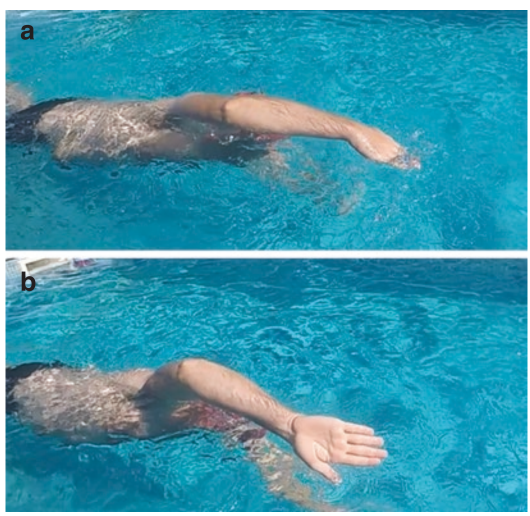

Fig. 29.1 Hand entry. (**a**) Correct. (**b**) Incorrect

– Early pull phase:
  During the first part of this phase the coaches insist on keeping the elbow in a high position and tilting the hand. This position will make the propellant muscles work well mechanically. For some studies this would be the most correct position to perform this stroke phase [6, 7].
  If we keep the elbow in a high position, we are placing the shoulder in internal rotation and causing subacromial impingement. Keeping the elbow down ("dropped elbow") the propulsion will be worse, but increases the external rotation of the shoulder and improves the pressure on the subacromial space (Fig. 29.2) [15].
– Middle pull phase:
  If the hand overtakes the body midline increases the time the shoulder remains with impingement (Fig. 29.3). In this phase the stabilizing muscles of the scapula work such as the

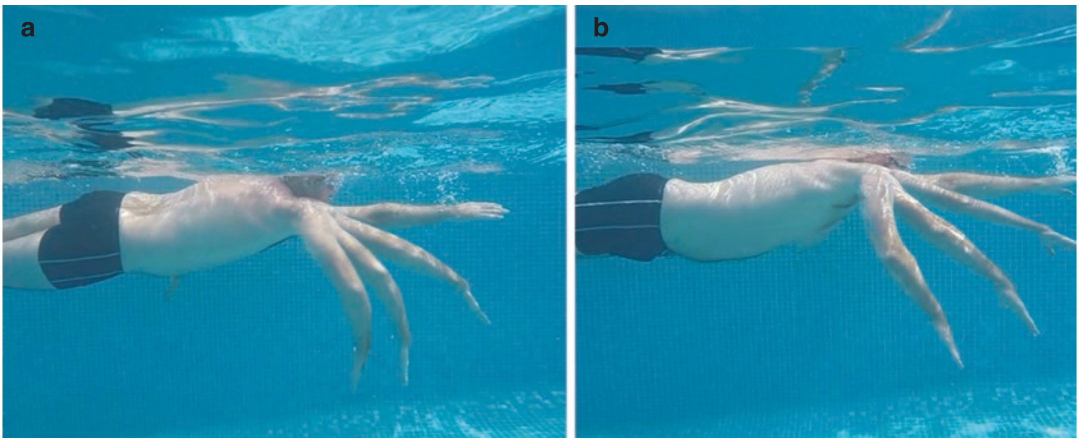

Fig. 29.2 Elbow position. (**a**) Elbow higher than wrist. (**b**) Dropped Elbow

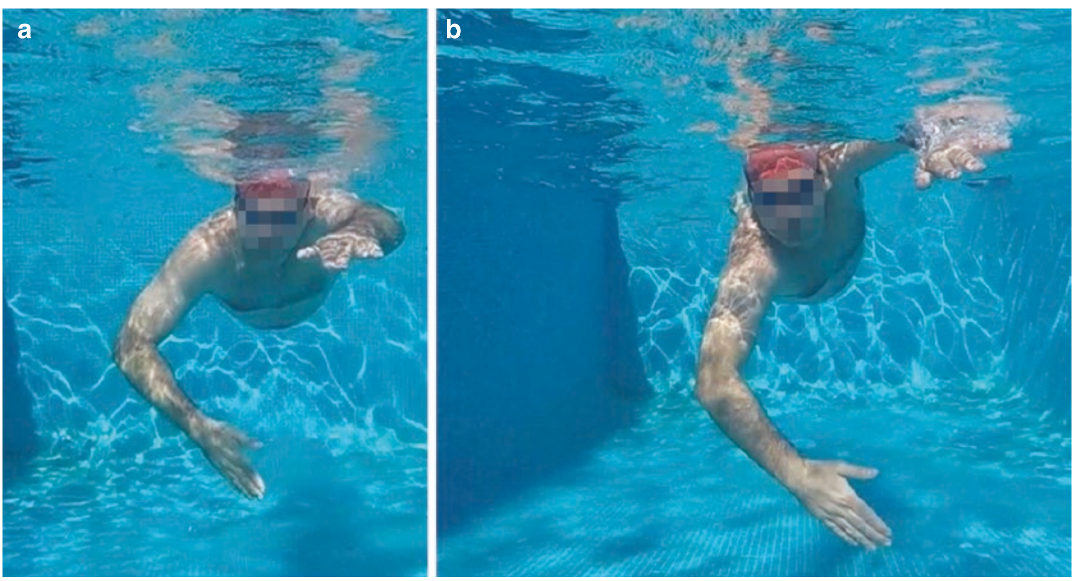

Fig. 29.3 S-shape. (**a**) Correct. (**b**) Incorrect

Serratus Anterior, Trapezium, and *Major Rhomboid*. A malfunction of these muscles can give shoulder pain as we will describe later.

– Late pull phase:

The elbow gets extended almost completely, the shoulder performs a slight internal rotation and the hand is completely extended. If the swimmer brings out the hand too early, it may be indicative of a subacromial space problem [17].

3. Recovery phase.
   - The hand comes out of the water to go back to the initial phase doing an abduction and external rotation of the shoulder. If elbow precedes the wrist for a long time, there will be more shoulder internal rotation; however, if the elbow precedes the wrist for a short time, there will be a correct external rotation [15] .

Butterfly Stroke

There are not many differences between crawl and butterfly stroke. In this style the stroke is done with the two arms at the same time, the body is moved using the hip as the axis, and the breathing will be done forward instead of sideway.

Sometimes the hands go into water laterally, this can be a sign of shoulder pain in swimmers, it is because of the increased activity in the *Posterior Deltoid* and less activity in the *Supraspinatus* and *Teres minor* [18].

In the pull phase (especially the middle pull phase), swimmers who have pain have less potency due to a weakness of the scapula stabilizers such as the *Serratus anterior* and the *Teres minor*.

Backstroke

This style is similar to crawl biomechanically, but the athlete swims on his back. In the initial phase of the stroke, the shoulder is placed at the highest extension and internal rotation to allow the hand go into the water with the little finger (Fig. 29.4). During all the recovery phase, the elbow is in extension and the shoulder in internal rotation to reach the initial phase into the right position [7].

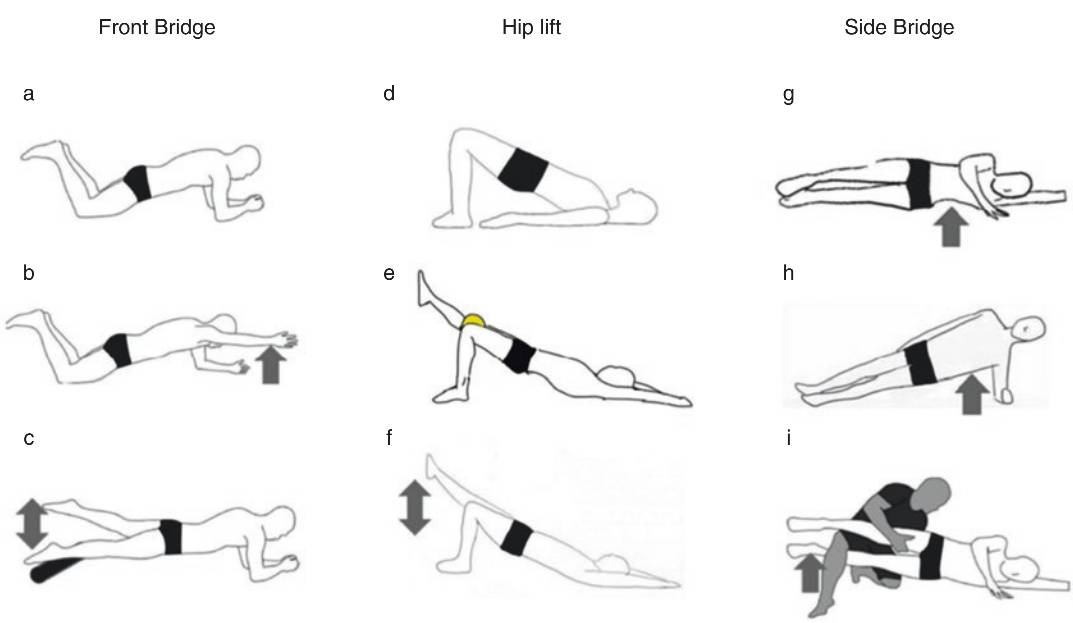

Fig. 29.4 Lumbar injury prevention exercises

Breaststroke

It is different from the rest of the styles. Most of waste of energy comes especially from the lower extremities, that is why most of the injuries will be located in the knee.

Upper extremities have a shorter pull phase than freestyle and the recovery phase is longer.

In the pull phase of swimmers with pain, we see a decrease in electromyograph activity of the *Teres Minor muscle*, *Serratus Anterior*, and an increase in the activity of *Subscapularis* and *Latissimus Dorsi*. With this balance of strength, the shoulder will have more internal rotation than in patients who have no pain.

In the recovery phase we can find a decrease in the activity of *Supraspinatus, Teres Minor, Middle Deltoid*, and *Upper Trapezius* seen in swimmers with a lower elbow position. At the same time, we can find an increase in *Infraspinatus* and *Latissimus dorsi* that produces an increase in subacromial impingement in the swimmers with pain [19].

- Hand paddles:
  - To improve stroke strength, swimmers often use hand paddles. Using this can cause increased shear forces at the joint surface, particularly in an anterior direction, which may potentiate joint subluxation [20].
  - The use of hand paddles causes, in the recovery phase, that external rotation get to delayed, increasing risk of impingement [15].
  - In recent studies, considering that the number of subjects is very low, it has been questioned that the use of hand paddles improves the speed, frequency, length, or strength of the stroke. Although the use of hand paddles increases swimming velocity and propelling efficiency, it does not increase the muscle power [21, 22].

Intrinsic Factors:

- Excessive laxity/general joint hypermobility:
  - There are significant differences of laxity between swimmers with shoulder pain and swimmers without pain. Those who have pain have higher laxity. There are also differences between non-professional athletes and professionals. Professionals have greater mobility [12, 23].
  - To reduce water resistance, improve buoyancy, and basically increase speed, swimmers try to have higher laxity/flexibility in the shoulder to increase the range of mobility and improve stroke. This search for laxity is what can lead to a capsuloligamentous complex stretch that will produce a shoulder instability. This instability can be partially compensated at the beginning by increasing the antagonistic strength until the musculature gets fatigued and pain appears. When reducing stability, the strength of rotator cuff muscles decreases [8, 24–26].
  - There is also a genetic predisposition of hyperlaxity, but this is only seen in 20% of swimmers [27].
- Scapular dyskinesis:
  - Muscle imbalance of scapula stabilizers is seen in most of the athletes with overuse pain syndromes [5]. Scapula stabilizers fatigue increased scapular dyskinesis as swim training season progressed in a study with 78 swimmers with no history of shoulder pain [28].
  - Protraction is mostly done by the Serratus Anterior muscle (innervated by the Long Thoracic Nerve) and also by the Pectoralis Minor muscle (innervated by the Medial Pectoral nerve) [29]. Retraction is mainly carried out by the most medial fibers of the Trapezium muscle (innervated by Spinal Accessory Nerve) and by the Major and Minor Rhomboid (innervated by the Dorsal Scapular Nerve) [29]. An alteration in any of these components can result in a malfunction of the scapula, as we can see in a study where painful shoulders had higher abduction, protraction, and lateral displacement than asymptomatic shoulders [30].
  - The Serratus *Anterior* muscle has a reduced electromyographic activity and increased fatigue during activity in swimmers with

shoulder pain because *Long Thoracic Nerve* is very superficial and the overuse can easily injure it [18, 29, 31].

- Rotator cuff imbalance:
  - After a period of exclusively aquatic training, there is a significant asymmetry of strength in the rotator cuff, increasing the strength of the internal rotator muscles than their antagonists [32, 33]. The weakness of the rotator cuff musculature can be compensated by the position of the scapula, but to a certain degree. After this the shoulder becomes subluxated, causes inflammation around the joint, and pain appears [9, 18].
  - Swimmers with painful shoulders have less muscle activity in more than half of the muscles involved. There are 7 of the 12 muscle groups studied in butterfly swimmers (*Anterior Deltoid, Middle Deltoid, Infraspinatus, Subscapularis, Upper Trapezius, Rhomboids*, and *the Serratus Anterior*) that have a decreased electromyographic activity [18, 31].
- Thoracic kyphosis:
  - Protraction and retraction of the scapula are strongly linked to the thorax, therefore, an alteration in its shape can worsen subacromial space. Patients with kyphosis and slouched sitting posture have higher internal scapular rotation and previous inclination and less upward scapular rotation [34].
  - Subacromial space size, in patients with hyperkyphosis, get decreased significantly as kyphosis is getting worse [35].

29.2.2.3 Clinical Evaluation

Clinical findings are often displayed as impingement signs, anteroinferior instability, dysfunction of the scapulothoracic joint, and significant muscle imbalance of the rotator cuff. We asked patients about the intensity, the time elapsed since the pain began, the location of the pain, and the moment during the stroke when the pain improves or worsens [9].

The physical examination should be complete, and it should be done in headquarters and also in supine position, looking for the intrinsic factors of shoulder pain, described previously, as well as observe the scapular movement ("snapping scapula" or "winged scapula").

We will look for signs of hyperlaxity by:

- sulcus sign (inferior displacement of the humeral head),
- drawer test (anterior/posterior displacement of the humeral head),
- apprehension test in swimmers is often found at 135° abduction, not at 90° because it is the position that the stroke starts [8].
- Beighton criteria for hyperlaxity [36].

Measures of the balance joint and force will be more objective by using goniometer, dynamometer, and laxometer, so we can evaluate mobility deficits, in particular in internal rotation [9, 12, 37–39].

29.2.2.4 Imaging

Simple radiology and ultrasound are used to rule out different pathologies such as calcifications, complete tendon tears, or partial tears.

Magnetic resonance shows how the thickness of the supraspinatus tendon correlates significantly with the time the swimmer has been training, hours trained a week, and the level the swimmer competes. In addition, in this study, all patients who have an increase of thickness had shoulder pain and supraspinatus tendinopathy [12].

Magnetic Resonance Arthrogram will be necessary when we suspect capsule-labral injuries, and the patient does not improve clinically. Many of swimmer's asymptomatic shoulders may have a redundant capsule, labral lesions, or cuff injuries [40].

Arthroscopy is a diagnostic test and a treatment for patients who were refractory to conservative treatment.

29.2.2.5 Treatment

The main treatment is based on detecting technical fails, decreasing the volume of training, and enhancing the strength and stretch of the rotator cuff, of the scapula stabilizers and core. Only half of the athletes reported that injury prevention exercises were always (29.9%) or often (23.4%) a regular part of their daily training. The prevention would help avoid this type of injury. Swimmers should not increase distances covered in training by 10% per week [28, 41].

Swimmers who are subjected to a compensatory strength training program get an improvement in the strength and joint balance of the rotator cuff. If they continue doing only aquatic exercise without strength training, in the middle of the season, strength and joint balance will get worse [33].

The swimmer's stroke will tell us which set of muscles will have to work depending on the moment of stroke or what kind of technical error is being produced. It is important that trainers and sports therapists to be correctly instructed, so they can detect as soon as possible technique alterations and can prevent the appearance of consequential pains [42].

Treatment of swimmer's shoulder pain in three phases:

Phase 1. The patient is in pain only while swimming [5]:

- Reduce training volume.
- Ice pack after training.
- Detecting technical errors.
- Find out if there is any dysfunction (subacromial, instability, scapular) and exercise or stretch weakened muscles.

Phase 2. The patient is in pain also when he is not swimming. The same as Phase 1 plus:

- Swimming rest for 2–4 weeks.
- NSAIDs +/− corticosteroid infiltration in subacromial bursa [43].

Phase 3. The patient does not improve after 3 months in Phases 1 and 2:

- Same rehabilitation and rest as in the previous phases.
- If we have imaging tests and there are injuries to repair, it is time to consider the surgery. If there are signs of instability, they will also need to be repaired.
  – Athletes involved in overhead sports (baseball, basketball, racquet sports, volleyball, weightlifting, and swimming), with instability (recurrent dislocations or recurrent subluxation) were operated by a capsular shift and 92% returned to the same sport, 71% of them did it at the same level [44].
  – The swimmers for whom conservative treatment failed and showed signs of multidirectional instability and were not able to swim, they were operated by a capsular plication. Of them 80% competed again, but only 20% did it at the same pre-injury training intensity [45].
  – Patients who were brought under debridement, partial release of the coraco-acromial ligament and bursectomy, after the failure of rehabilitation treatment during more than 6 months, 56% returned to the pre-injury level, and 44% did not compete again [46].
  – In a study where athletes were operated after the first episode of dislocation and presents anteroinferior capsulolabral lesions +/− SLAP lesions type 5, the return-to-sports rate was 83%. The return-to-sports rate did not show an association with the type of sport (overhead, contact) (Table 29.2) [47].

29.2.3 Spine

29.2.3.1 Epidemiology

Spine is the second most common area injured in swimmers (21–24%). 33.3% of butterfly swimmers and 22.2% of breaststroke swimmers had back pain. [2, 48, 49].

29.2.3.2 Etiology

- Overuse: the repeated microtraumas in long sessions of water and dryland training produce an injury of the annulus that has no time to get repair and get erodes. Torsion and rotation movements added to with bending, and exten-

Table 29.2 Muscles involved and biomechanical parameters in freestyle stroke

Phase	Muscles involved	Technique details that produce pain	Intervention
Initial phase	Ant/med deltoid Upper trapezius Rhomboids	Too high angle elevation Cross hand Thumb first	• Train the strength and stretching of the antagonistic muscles to elevation • Correct body roll • Entry the hand with no extension of the elbow • Entry the hand lateral to the head and medial to the shoulder
Pull phase arly	Pectoralis major. Teres minor	Elbow remain high	Keep the elbow down (dropped elbow)
Middle	Serratus anterior, pectoralis major, latissimus dorsi	Cross the hand to middle line	Do not cross the hand to middle line Stretch and strength of the scapula stabilizers
Late	Subscapular, latissimus dorsi, posterior and middle deltoid	Low stroke power Brings out the hand too early	Stretch and strength of the scapula stabilizers
Recovery phase	Deltoid, rhomboid, serratus anterior	Elbow go before hand	Increase external rotation, then the elbow goes before hand less time. Increase body roll (around 45°)

sion movements can cause annulus delamination [50].

- Swimming style: there are two swimming styles that perform the most flexion-extension motion: butterfly and breaststroke. These repeated microtraumas can cause stress fractures in pars interarticularis (spondylolysis) [51].
- Hip stiffness: Lack of hip mobility or contraction of the iliopsoas muscle causes an overload in the lower back.
- Training devices: kicking boards, pull-boy or fins produces overload and hyperextension of lumbar spine with an increase of compression on the facets.

29.2.3.3 Clinical Evaluation

Oswestry disability questionnaire is the best way to objectively and systematically assess lower back pain. As we did with shoulder, swimmers should be questioned about the pain intensity, and about his location and position when they are in pain. Most of the time, low back pain occurs during hyperextension or hyperflexion positions [52].

Evaluation for symptomatic neck and back pain is the same for athletes as in the general population including full assessment of range of motion and neurologic exam.

Differential diagnosis of lumbar pain in swimmers should be made with muscle and ligaments sprains, facet joint injury, herniated disc, spondylolysis, Scheuermann disease, infections, and tumors.

29.2.3.4 Imaging

The prevalence of lumbar degeneration in swimmers is not resolved yet, there are studies that do not find differences between swimmers and non-athletes and other studies that do. The prevalence of disc degeneration in asymptomatic population is 25%, and specifically in lumbar spine ranges from 35 to 54% [53, 54]. Some works show that this prevalence in swimmers can range from 48 to 68%, being this different with asymptomatic population significant for some of these works but not for the others [55, 56].

As the training volume increases in overuse athletes, also increases disc degeneration incidence, predominantly on the L5-S1 level, having no relation with lower back pain [57, 58]. It is not clear what lumbar level is the most affected; sometimes these are high levels (L1-L2) and sometimes the lower ones (L5-S1). Most of the studies use Pfirmann's graduation to classify disc degeneration [55, 56, 59].

29.2.3.5 Treatment

Prevention and a correct planning of dryland exercises are the key to avoid lower back pain. In Matsuura's study, they achieve to decrease the prevalence of lower back pain in the national team of Japanese swimmers from 23.5% (2002–2008) to 14.8% (2009–2016) by implementing deep trunk muscle exercises (transversus abdominis and lumbar multifidus) that help stabilize the lumbar spine (Fig. 29.4) [60].

A high percentage of athletes with a spondylolysis and low-grade spondylolisthesis after initial treatment with rest, application of bracing and rehabilitation manage to return to sports. After 6 months of nonoperative treatment, if the pain persists or non-union achieved at 9–12 months, a posterolateral fusion can be performed with a high success rate [61, 62].

29.2.4 Knee

29.2.4.1 Epidemiology

The knee is the third most common location of injury in swimmers. The breaststrokes swimmers are the most affected; they have a fivefold higher risk of knee pain [1, 63–65].

29.2.4.2 Etiology

The knee injuries also have a multifactorial etiology, a combination of overtraining, technical details, and intrinsic factors:

- Overuse: it is the main cause of knee pain. There is a positive correlation between knee pain and age, years of training, and level of competition. As they increase, so does the incidence of knee pain [66].
- Hip position: the incidence of knee pain has a bimodal distribution, increases if the abduction angle of the hip at kick initiation is lower than 37° and greater than 42°. If the knees remain tightly attached or separated (greater or lower hips abduction), the incidence of pain increases because it will condition the forces that will be generated in the propulsion phases of the kick [64].

Internal hip rotation is significantly lower in those breaststroke swimmers who have knee pain frequently. These present tight hamstrings, tight calf muscles, and tight hip adductors [66].

- External tibial rotation and valgus stress: to get prepare for the propulsion phase, the knee and hip flex, the tibia do an external rotation and the foot a dorsal flexion. When performing the propulsion, the hip must be extended and adducted, the knee full extended and the foot plantar flexed. This rapid movement creates a stress at the level of the medial face of the knee, in the superficial and deep fascicles of the medial collateral ligament and the medial face of the extensor mechanism of the knee (medial facet, medial retinaculum, medial capsule) [63, 67].
- Patellofemoral tracking: The repeatedly sudden contraction of quadriceps generates an increase in patellofemoral contact stresses. Patellar instability, patellar malalignment, or an imbalance in the quadricipital musculature can predispose to knee pain.

29.2.4.3 Clinical Evaluation

Knee pain usually has a medial location, and it usually occurs during kicking. Exploring the stability and position of the patella will helps us to dismiss malalignment or subluxations. Valgus stress testing can trigger pain in the medial face of the knee that will suggest medial collateral ligament injury. A mechanical pain in the lateral side may indicate an external meniscal injury or an external discoid meniscus [63, 66, 68].

With repeated flexion-extension movements, swimmers can also present injuries to the iliotibial band, so we will keep exploring the lateral side of the knee.

We should differentiate knee pain with other apophyseal injuries that can present adolescence such as Osgood-Schlatter disease and Sinding-Larsen-Johansson disease.

29.2.4.4 Imaging

In a cross-sectional case–control study, the authors found significantly more MRI abnormalities in the knee joints of asymptomatic adolescent elite swimmers than in the control group. The most common abnormalities were infrapatellar fat pad edema, bone marrow edema, edema of prefemoral fat pad, and joint effusion [69]

29.2.4.5 Treatment

Prevention keeps remaining basic in overuse injuries. Progressive training increase, correct technique, and early diagnosis of minor injuries prevent knee pains from worsening.

Once the knee pain is established, the swimmer should do relative rest (depending on the pain may decrease the intensity of training or even stop it), use ice pack and try to improve the technique with the help of trainer and physical therapist. Performing stretching exercises to increase internal hip rotation and strengthen the quadriceps muscle may be appropriate [66].

29.2.5 Other Musculoskeletal Conditions less Frequent

- Hip:
  - To perform the kick, the swimmers do a hip adduction movement repeatedly, and this increases the forces at the adductor's insertion into the breaststroke swimmers. According to Grote, breaststroke swimmers are 6.92% more likely to have groin pain than swimmers who did not compete in pure breaststroke events [70].

Preventing hip injuries through stretches and a correct technique can also prevent future knee injuries, which as we saw above is closely linked to the hip position.

- Cervical spine:
  - Repetitive head rotation movements in freestyle and flexion-extension movements in butterfly stroke and breaststroke for breathing predispose to inflammatory and degenerative cervical lesions. More moderate movements will improve cervical pain.
- Ankle:
  - The repetitive ankle dorsiflexion and plantar flexion in swimmers' kick can trigger inflammatory injuries to flexor tendons and extensors.

29.2.6 General Medical Conditions in Swimmers

- Respiratory illness: trainings in chlorinated pools predispose swimmers to suffer episodes of bronchospasm associated with exercise [71].
- Ear conditions: water accumulation in the ear canal propitiate the cultivation of microorganisms, especially *Pseudomonas aeruginosa*. Prevention involves keeping the external ear canal dry by using a hair dryer and then instill prophylactic eardrops (isopropyl alcohol + acetic acid). Eardrops alone are the most effective treatment for acute otitis externa and may contain antibiotics, antiseptics, steroids, or a combination [72].
- Dermatology illness: this is due to the chlorine effect on the skin, the exposure to bacteria, or allergic reactions [73].
  - Swimmer's xerosis or dry skin: after training and after long showers with very hot water the skin gets dry and causes itching. It can be relieved with showers with cooler water or using an oil-based protective emollient after drying.
  - Bacterial folliculitis on bikini bottom: due to the humidity of the swimsuits, the streptococcus or *Staphylococcus aureus* causes nodules under the buttock folds. They should be treated with antibiotherapy. In those swimmers who use hot tubs or whirlpool, the cause may be *Pseudomonas aeruginosa*, and it appears presented as pustules or papules that self-limit in 7–10 days.
  - Dermatitis: Swimmer's googles can produce an allergic periorbitari erythema with pruritus and occasionally vesicles.
  - Swimming pool granuloma: it is the result from exposure to *Mycobacterium marinum*. It is presented as an indurated erythematous nodule in knees, elbows, dorsal surface of the hands and feet. It should be treated with oral antibiotherapy.
  - Seabather's eruption: mostly in saltwater swimmers. Clinically an urticarial or vesiculopapular eruption appears with itching at 24 hours after bathing. It is caused by a larva trapped in the swimsuit.

The best prevention is to shower without swimsuit after bathing. To relieve symptoms, we should apply ice, antihistamines, or topical corticosteroids depending on the pain intensity.

- Thoracic outlet syndrome: swimmers refer arm pain or arm cold sensation when they do over-the-head activities. It is due to a vascular or nerve compression in the interscalene triangle that can be confirmed by electromyography of the brachial plexus or an ultrasound in case of venous axillary thrombosis. Cervical X-rays are also useful to identify cervical rib.

29.3 Open-Water Swimming

It has many similarities with pathology explained previously for swimming, especially the one related to freestyle. In this discipline, we also must consider that the distances of the competitions in the Olympic Games are 5, 10, and 25 km. There are even other greater official distances, such as 30, 32, 57, and 88 km. These long distances make it the aquatic discipline with the highest risk of injury in competition [74]. They have an increased risk of dehydration, hypo/hyperthermia, and by the environment may have injuries such as sunburn, jellyfish stings, marine envenomation, and otitis [4].

Open-water swimming is the first of the triathlon disciplines and the one with more episodes of sudden death. The etiology of this sudden death in open-water swimmers is unknown; there are several theories. One of them is immersion pulmonary edema. A higher proportion than expected of left ventricular hypertrophy has been found in triathletes that died during open-water swimming [75]. Another theory would be an Autonomic Conflict; both the autonomic sympathetic system (tachycardia), which is activated by water temperature, exercise, the stress of competition, and parasympathetic (bradycardia), which is activated by the face immersion and breath holding (diving response), would produce fatal cardiac arrhythmia [76].

29.4 Diving

29.4.1 Epidemiology

Diving is not as frequent as swimming, although it presents a great number of injuries. Current literature on diving injuries is scarce. FINA consensus authors has established a methodologically way to collect the injuries in all water sports [77].

Most of the diving injuries are similar to swimmers ones. Shoulder and lower back are the most common injured areas [1].

29.4.2 Etiology

Injuries are produced by overuse (repeated trauma) at the moment of takeoff, during flight and at entry to the water. During daily training, between 50 and 150 dives are performed. In addition, we must add the dryland training activities that represent 50% of them.

- Takeoff: in this phase approach and propulsion is done. The propulsion can be performed with one or two feet. The search for acceleration overloads the lower limbs (most common injuries in this phase), especially the extensor mechanism of the knee (patellar tendinopathy, quadriceps tendinopathy, and patellofemoral compression syndrome) and ankle (Achilles and posterior tibialis tendinopathy). Handstand dive requires isometric force and balance to support weight. Wrist dorsiflexion, elbow hyperextension, and shoulder stability increase the incidence of overuse injuries in this position.
- Flight: maneuvers are performed at this stage. The body position can determine the injury location. For example, in the pike position (stretched legs, bent waist, feet together, and pointed fingers), the lumbar area bears a higher pressure on anterior structures, and it is more exposed to injury.
- Placing your hand behind your head for twisting, with your shoulder in abduction and

external rotation, irritates the long portion of the biceps.

- Although it is not common in high-level athletes, at this stage there could be concussion against springboard or platform.
- Entry: A deceleration of 50% occurs in a fraction of a second that is why this phase is the most susceptible to suffer injuries [78].
  – To avoid head injuries and to perform a splashless entry, in this phase the hands are placed in flat-hand position (wrist dorsal flexion, radial deviation, pronation), the elbows in hyperextension, and the shoulders in abduction and internal rotation. The transmission of forces through this kinetic chain requires strength and a correct technique to avoid any injury.
  – Shoulders are the last point of kinetic chain and repeated microtraumas in abduction and internal rotation can make it unstable. To reduce the risk of instability at the moment of entry, the divers stabilize the scapular waist with a scapula elevation that places the glenoid cavity behind the head of the humerus.
  – The entrance to the water should not splash. If the position is not right, the divers usually try to correct it by performing a lumbar hyperextension causing a stress in the posterior lumbar (facet joints, interarticularis pars).
  – Most of the lesions are on the wrist because of the repetitive microtrauma, rarely from direct trauma. These can be contusions of the carpal bones, carpal instability, triangle fibrocartilage complex tears, flexor carpi ulnaris tendinitis, extender pollicis longus ruptures, microfracture of the radial styloid, and scaphoid stress fractures [79, 80].
  – Microtraumas in elbows hyperextension cause an overload in the insertion tricipital and also at the ulnar collateral ligament, which causes tricipital tendinitis and valgus instability of the elbow [81].

29.4.3 Treatment

It is important, to prevent injury, to know the correct technique so the errors can be detected early. It is also important to know the specific physical demands of each phase and to train the flexibility and the strength of the most demanding areas.

Take-Home Message
In overuse sports seen in this chapter, the main treatment is based on preventing injuries by watching over the technical details, increasing the volume of training progressively, and be aware at the initial symptoms. This symptom should be early treated by the trainer/physiotherapist, so we can prevent the injury, and the athlete does not lose the training rhythm.

References

1. Kerr ZY, Baugh CM, Hibberd EE. Epidemiology of National Collegiate Athletic Association men's and women's swimming and diving injuries from 2009/2010 to 2013/2014. Br J Sports Med. 2015;49:465–71.
2. Wolf BR, Ebinger AE, Lawler MP, Britton CL. Injury patterns in Division I collegiate swimming. Am J Sports Med. 2009;37(10):2037–42.
3. Junge A, Engebretsen L, Mountjoy ML, Alonso JM, Renström PA, Aubry MJ, et al. Sports injuries during the Summer Olympic Games 2008. Am J Sports Med. 2009;37:2165–72.
4. Soligard T, Steffen K, Palmer D. Sports injury and illness incidence in the Rio de Janeiro 2016 Olympic Summer Games: a prospective study of 11274 athletes from 207 countries. Br J Sports Med. 2017;51:1265–71.
5. Bak K. The practical management of swimmer's painful shoulder: etiology, diagnosis, and treatment. Clin J Sport Med. 2010;20:386–90.
6. Richardson AB, Jobe FW, Collins HR. The shoulder in competitive swimming. Am J Sports Med. 1980;8:159–63.
7. Counsilman J. The science of swimming. Englewwodd Cliffs: PrenticeHall; 1968.

8. Bak K, Faunø P. Clinical findings in competitive swimmers with shoulder pain. Am J Sports Med. 1997;25:254–60.
9. Rupp S, Berninger K, Hopf T. Shoulder problems in high level swimmers—impingement, anterior instability, muscular imbalance? Int J Sports Med. 1995;16:557–62.
10. Tate A, Turner GN, Knab SE. Risk factors associated with shoulder pain and disability across the lifespan of competitive swimmers. J Athl Train. 2012;47:149.
11. Walker H, Gabbe B, Wajswelner H. Shoulder pain in swimmers: a 12-month prospective cohort study of incidence and risk factors. Phys Ther Sport. 2012;13:243–9.
12. Sein ML, Walton J. Linklater. Shoulder pain in Elite swimmers: primarily due to swim-volume-induced supraspinatus tendinopathy. Br J Sports Med. 2010;44:105–13.
13. Jobe FWKRGC. Shoulder pain in the overhand or throwing athlete. The relationship of anterior instability and rotator cuff impingement. Orthop Rev. 1989;18:963–75.
14. Kvitne RSJFJC. Shoulder instability in the overhand or throwing athlete. Clin Sports Med. 1995;14:917–35.
15. Yanai T. Hay JG. Shoulder impingement in front-crawl swimming: II. analysis of stroking technique. Med Sci Sports Exerc. 2000;32(1):30–40.
16. Johnson JN, Gauvin J, Fredericson M. Swimming biomechanics and injury prevention: new stroke techniques and medical consideration. Phys Sportsmed. 2003;31:41–6.
17. Rockwood and Matsen's. The shoulder. Philadelphia PA: Rockwood; 2016.
18. Pink M, Jobe FW, Perry J. The painful shoulder during the butterfly stroke: an electromyographic and cinematographic analysis of twelve muscles. Clin Orthop Relat Res. 1993;288:60–72.
19. Ruwe PA, Pink M, Jobe FW, Perry J, Scovazzo ML. The normal and the painful shoulders during the breaststroke. Am J Sports Med. 1994;22(6):789–96.
20. McMaster WC. Shoulder injuries in competitive swimmers. Aquat Sports Inj Rehab. 1999;18:349–59.
21. Barbosa AC, Ferreira THN, Leis LV, Gourgoulis V, Barroso R. Does a 4-week training period with hand paddles affect front-crawl swimming performance? J Sports Sci. 2020;38(5):511–7.
22. Tsunokawa T, Tsuno T, Mankyu H, Takagi H, Ogita F. The effect of paddles on pressure and force generation at the hand during front crawl. Hum Mov Sci. 2018;57:409–16.
23. Zemek MJ, Magee DJ. Comparison of glenohumeral joint laxity in elite and recreational swimmers. Clin J Sport Med. 1996;6(1):40–7.
24. Wakayoshi K. Adaptations to six months of aerobic swim training. Changes in velocity, stroke rate, stroke length and blood lactate. Int J Sports Med. 1993;14(7):368–72.
25. Crowley E, Harrison AJ, Lyons M. The impact of resistance training on swimming performance: a systematic review. Sports Med. 2017;47(11):2285–307.
26. McMaster WC, Roberts A, Stoddard T. A correlation between shoulder laxity and interfering pain in competitive swimmers. Am J Sports Med. 1998;26(1):83–6.
27. Pink MM, Tibone JE. The painful shoulder in the swimming athlete. Orthop Clin North Am. 2000;31(2):247–61.
28. Madsen P, Jensen S, Bak K. Training induces scapular dyskinesis in pain-free competitive swimmers. Clin J Sport Med. 2011;21(2):109–13.
29. Martin RM, Fish DE. Scapular winging: anatomical review, diagnosis, and treatments. Curr Rev Musculoskelet Med. 2008;1(1):1–11.
30. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology part I: pathoanatomy and biomechanics. Arthroscopy. 2003;19:404–20.
31. Scovazzo ML, Browne A, Pink M, Jobe FW, Kerrigan J. The painful shoulder during freestyle swimming. An electromyographic cinematographic analysis of twelve muscles. Am J Sports Med. 1991;19(6):577–82.
32. Ramsi M, Sawanik KA, Sawanik CB, Straub SJ, Mattacola CG. Shoulder-rotator strength of high school swimmers over the course of a competitive season. J Sport Rehabil. 2004;13(1):9–18.
33. Batalha NM, Raimundo AM, Tomas-Carus P, Marques MA, Silva AJ. Does an in-season detraining period affect the shoulder rotator cuff strength and balance of young swimmers? J Strength Cond Res. 2015;28(7):2054–62.
34. EPM C. Functional anatomy of the shoulder complex. J Orthop Sports Phys Ther. 1993;18:342–50.
35. Gumina S, Di Giorgio G, Postacchini F, Postacchini R. Subacromial space in adult patients with thoracic hyperkyphosis and in healthy volunteers. Chir Organi Mov. 2008;91(2):93–6.
36. Beighton P, Solomon L, Soskolne CL. Articular mobility in an African population. Ann Rheum Dis. 1973;32(5):413–8.
37. Jansson A, Saartok T, Werner S. Evaluation of general joint laxity, shoulder laxity and mobility on competitive swimmers during growth and in normal controls. Scand J Med Sci Sports. 2005;15:169–76.
38. Beach ML, Whitney SL, Dickoff-Hoffman S. Relationship of shoulder flexibility, strength and endurance to shoulder pain in competitive swimming. J Orthop Sports Phys Ther. 1992;16:262–8.
39. Torres RR, Gomes JL. Measurement of glenohumeral internal rotation in asymptomatic tennis players and swimmers. Am J Sports Med. 1998;1:1017–23.
40. Connor PM, Banks DM, Tyson AB. Magnetic resonance imaging of the asymptomatic shoulder of overhead athletes: a 5-year follow-up study. Am J Sports Med. 2003;31:724–7.
41. Mountjoy M, Junge A, Miller J. An uneven playing field: athlete injury, illness, load, and daily training environment in the year before the FINA (Aquatics) World Championships, 2017. Clin J Sport Med. 2009; https://doi.org/10.1097/JSM.0000000000000814.

42. Bak K. Nontraumatic glenohumeral instability and coracoacromial impingement in swimmers. Scand J Med Sci Sports. 1996;6:132–44.
43. Kenal KAF, Knapp LD. Rehabilitation of injuries in competitive swimmers. Sports Med. 1996;22:337–47.
44. Bigliani LU, Kurzweil PR, Schwartzbach CC, Wolfe IN, Flatow EL. Inferior capsular shift procedure for anterior-inferior shoulder instability in athletes. Am J Sports Med. 1994;22(5):578–84.
45. Montgomery SR, Chen NC, Rodeo SA. Arthroscopic capsular plication in the treatment of shoulder pain in competitive swimmers. HSS Journal. 2010;6(2):145–9.
46. Brushøj C, Bak K, Johannsen HV, Faunø P. Swimmers' painful shoulder arthroscopic findings and return rate to sports. Scand J Med Sci Sports. 2007;17(4):373–7.
47. Terra BB, Ejnisman B, Belangero PS, Figueiredo E, De Nadai A, Ton A, et al. Arthroscopic treatment of first-time shoulder dislocations in younger athletes. Orthop J Sports Med. 2019;7(5):2325967119844352.
48. Capaci K, Ozcaldiran B, Durmaz B. Musculoskeletal pain in elite competitibe male swimmers. Pain Clin. 2002;14:229–34.
49. Drori A, Mann G, Constantini N. Low back pain in swimmers: is the prevalence increasing? In The 12th International Jerusalem Symposium on Sports Injuries; 1996; Tel Aviv. p. 75.
50. Marshall LW, McGill SM. The role of axial torque in disc herniation. Clin Biomech (Bristol, Avon). 2010;25(1):6–9.
51. Cyron BM, Spondylolysis HWC. The fatigue strength of the lumbar neural arch in spondylolysis. J Bone Joint Surg. 1978;60:234–8.
52. Hudson-Cook N, Tomes-Nicholson K. Breen A. A revised Oswestry disability questionnaire. In Back Pain: New Approaches to Rehabilitation and Education. Manchester: Manchester University Press. Roland M: Jenner JR; 1989. p. 187–204.
53. Matsumoto M, Fujimura Y, Suzuki N, Nishi Y, Nakamura M, Yabe Y, et al. MRI of cervical intervertebral discs in asymptomatic subjects. J Bone Joint Surg (Br). 1998;80(1):19–24.
54. Takatalo J, Karppinen J, Niinimäki J, Taimela S, Näyhä S, Järvelin MR, et al. Prevalence of degenerative imaging findings in lumbar magnetic resonance imaging among young adults. Spine. 2009;34(16):1716–21.
55. Kaneoka K, Shimizu K, Hangai M, Okuwaki T, Mamizuka N, Sakane M, et al. Lumbar intervertebral disk degeneration in elite competitive swimmers: a case control study. Am J Sports Med. 2007;35(8):1341–5.
56. Folkvardsen S, Magnussen E, Karppinen J, Auvinen J, Larsen RH, Wong C, et al. Does elite swimming accelerate lumbar intervertebral disc degeneration and increase low back pain? A cross-sectional comparison. Eur Spine J. 2016;25(6):2849–55.
57. Elfering A, Semmer N, Birkhofer D, Zanetti M, Hodler J, Boos N. Risk factors for lumbar disc degeneration: a 5-year prospective MRI study in asymptomatic individuals. Spine. 2002;27:125–34.
58. Powell MC, Wilson M, Szypryt P, Symonds EM, Worthington BS. Prevalence of lumbar disc degeneration observed by magnetic resonance in symptomless women. Lancet. 1986;2(8520):1366–7.
59. Pfirrmann CW, Metzdorf A, Zanetti M, Hodler J, Boos N. Magnetic resonance classification of lumbar intervertebral disc degeneration. Spine. 2001;26:1873–8.
60. Matsuura Y, Hangai M, Koizumi K, Ueno K, Hirai N, Akuzawa H, et al. Injury trend analysis in the Japan national swim team from 2002 to 2016: effect of the lumbar injury prevention project. BMJ Open Sport Exerc Med. 2019;5(1):e000615.
61. Grazina R, Andrade R, Santos FL, Marinhas J, Pereira R, Bastos R, et al. Return to play after conservative and surgical treatment in athletes with spondylolysis: a systematic review. Phys Ther Sport. 2019;(37):34–43.
62. Bouras T, Korovessis P. Management of spondylolysis and low-grade spondylolisthesis in fine athletes. A comprehensive review. Eur J Orthop Surg Traumatol. 2015;25(Suppl 1):S167–75.
63. Keskinen K, Eriksson E, Komi P. Breaststroke swimmer's knee: a biomechanical and arthroscopic study. Am J Sports Med. 1980;8(4):228–31.
64. Vizsolyi P, Taunton J, Robertson G, Filsinger L, Shannon HS, Whittingham D, et al. Breaststroker's knee. An analysis of epidemiological and biomechanical factors. Am J Sports Med. 1987;15(1):63–71.
65. Knobloch K, Yoon U, Kraemer R, Vogt PM. 200–400 m breaststroke event dominate among knee overuse injuries in elite swimming athletes. Sportverletzung-Sportschaden. 2008;22(4):213–9.
66. Rovere GD, Nichols AW. Frequency, associated factors, and treatment of breaststroker's knee in competitive swimmers. Am J Sports Med. 1985;13(2):99–104.
67. Wethelund JO, de Carvalho A. An unusual lesion in the knee of a breaststroke swimmer. Int J Sports Med. 1985;6(3):174–5.
68. Stulberg SD, Shulman K, Stuart S, Culp P. Breaststroker's knee: pathology, etiology and treatment. Am J Sports Med. 1980;8(3):164–71.
69. Soder RB, Mizerkowski MD, Petkowicz R, Baldisserotto M. MRI of the knee in asymptomatic adolescent swimmers: a controlled study. Br J Sports Med. 2012;46(4):268–72.
70. Grote K, Lincoln TL, Gamble JG. Hip adductor injury in competitive swimmers. Am J Sports Med. 2004;32(1):104–8.
71. Boulet LP, O'Byrne PM. Asthma and exercise-induced bronchoconstriction in athletes. N Engl J Med. 2015;372:641–8.
72. Rosenfeld RM, Schwartz SR, Cannon CR, Roland PS, Simon GR, Kumar KA, et al. Clinical practice guideline: acute otitis externa executive summary. Otolaryngol Head Neck Surg. 2014;150(2):161–8.
73. Freiman A, Barankin B, Elpern DJ. Sports dermatology part 2: swimming and other aquatic sports. CMAJ. 2004;171(11):1339–41.

74. Mountjoy M, Junge A, Alonso JM, Engebretsen L, Gerrard D, Kouidri M, et al. Sports injuries and illnesses in the 2009 FINA World Championships. Br J Sports Med. 2010;44(7):522–7.
75. Moon RE, Martina SD, Peacher DF, Kraus WE. Deaths in triathletes: immersion pulmonary oedema as a possible cause. BMJ Open Sport Exerc Med. 2016;2(1):e000146.
76. Shattock MJ, Tipton MJ. 'Autonomic conflict': a different way to die during cold water immersion? J Physiol. 2012;590(14):3219–30.
77. Mountjoy M, Junge A, Alonso JM. Consensus statement on the methodology of injury and illness surveillance in FINA (aquatic sports). Br J Sports Med. 2016;(50):590–6.
78. Baranto A, Hellström M, Nyman R, Lundin O, Swärd L. Back pain and degenerative abnor-malities in the spine of young elite divers: a 5-year follow up magnetic resonance imaging study. Knee Surg Sports Traumatol Arthrosc. 2006;14(9):907–14.
79. le Viet DT, Lantieri LA, Loy SM. Wrist and hand injuries in platform diving. J Hand Surg. 1993;18(5):876–80.
80. Mohamed Haflah NH, Mat Nor NF, Abdullah S, Sapuan J. Bilateral scaphoid stress fracture in a platform diver presenting with unilateral symptoms. Singapore Med J. 2014;55(10):e159–61.
81. Jones NS. Competitive diving principles and injuries. Curr Sports Med Rep. 2017;16(5):351–6.

Racquet Sports: Tennis, Badminton, Racquetball, Squash

30

Elisabeth A. Wörner and Marc R. Safran

30.1 Introduction on Racquet Sports

Racquet sports are games in which players use racquets to hit a ball or other object, major racquet sports include tennis, badminton, squash, and racquetball. For the purpose of describing the most common injuries related to racquet sports, the chapter will start with an introduction of the most commonly played sports. These racquet sports are net and wall games that are score-dependent rather than time-dependent. The sports are played individually but can also be played in doubles (two players on each team).

It is estimated that over 75 million people participate in tennis worldwide [1]. In the US alone, more than five million people play tennis at least twice a month. Tennis is a sport played on a court (hard court, clay (and har-tru), grass, or carpet), that is 78 feet long with a net 3 feet in height dividing the court in two equal parts. The court is 27 feet wide for singles, with one player on each side of the net. Doubles is played with 2 players on each side of the net with the court 36 feet wide. Racquets are made of fiberglass, graphite, and titanium. The racquets measure 27–29 inches long, though the maximum length allowed is up to 32 inches. There are no weight restrictions, though current technology has resulted in racquets weighing 7 ½ to 13 ounces. Strings are made from resilient gut or nylon. The tennis ball is hollow, composed of inflated rubber and covered with fabric. The ball may travel at a velocity of up to 150 mph in adults on the serve and 90 mph on the return.

The World Badminton Federation estimates that 220 million people play badminton worldwide [2]. Badminton is especially a very popular sport in China, Indonesia, Malaysia, and India. Badminton is a non-contact sport played on a court of 20 feet wide and 44 feet long with the court evenly divided into two, lengthwise, by a net 5 feet high at the center. Opponents are on opposite sides of the net. This sport is usually played indoors on a wooden floor. The shuttlecock is made of feathers with a fixed base. The racquet is much lighter than the tennis racquet (3–3.7 ounces). The racquets are 26 ¾ inches long and the head length cannot exceed 11 3/8 inches. The shuttlecock may travel up to 200 mph in world class adult play.

Squash is played by approximately 18 million people worldwide, and in the USA, 500,000 play at least once a year, and 150,000 play regularly. Squash is a sport played in an indoor enclosed court. The international sized court and measures 21 feet by 32 feet enclosed by a ceiling, 18 feet, that is used in play. Racquets measure 27 inches in length and weighs 7 ½ to 9 ounces. The ball is relatively soft and owing the player more time to

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retrieve the ball. The ball velocity has been measured at over 110 mph after direct contact. The players are in close proximity in singles and especially in doubles..

Racquetball was invented in 1949 by an individual, in the USA, who sought a fast-paced game that was easy to learn on a handball court. Racquetball has grown to a total of approximately seven million players a year in the United States, and eight million worldwide. More than 1.5 million of the participants are estimated to play at least twice a month. The racquetball court is enclosed with a ceiling which is used in play. The court is 40 feet in length and 20 feet in width. The height of the front wall is 20 feet, while the back wall is equal to or greater than 12 feet. The racquet cannot exceed 22 inches in length, and the racquet weighs between 6 and 8 ounces. The ball is softer than a squash ball. The ball has been shown to travel consistently at 127 mph during a highly competitive play after a direct hit.

Squash, racquetball, and badminton are relatively faster games than tennis and more taxing on the joints, with more sudden stops, starts, pivoting, and a harder swing of the racquet. In general, conditioning is vital for singles play, whereas doubles is more forgiving.

This chapter reviews orthopedic injuries in racquet sports (tennis, badminton, squash, racquetball). Since the injuries are very similar between the sports, the main focus will be on tennis injuries, but the differences will be elucidated. The incidence rate of tennis injuries varies from 0.04 to 3.00 per 1000 hours played, with the highest incidence in the lower extremity, followed by the upper extremity and the trunk [3].

30.2 Biomechanics in Racquet Sports

All of these above mentioned racquet sports require jumps, lunges, quick changes of directions, and rapid arm movements from a wide variety of body positions. Excelling in skill, stamina, speed, strength and spirit are important qualities for a player. In badminton a correlation was found between the quality of a player and its "explosive strength" and "agility." [2] The duration of an actual play during a match is not determined by any time limit. Thus, matches can last for several hours, the aerobic and anaerobic requirements of racquet sports, combined with the variety of strokes, result in a unique profile of qualities of a player. It also results in a unique profile of injuries [3]. The shoulder and elbow can be adversely affected by the repetitive trauma of chronic overuse; acute injuries tend to involve the lower extremities.

Minor injuries, such as sunburns, abrasions, and blisters, are common in all players and can be of importance in a game or practice. In all racquet sports, and especially courts with poor ventilation, there is a significant cardiovascular and thermal stress to the body. Heat-related illness such as cramps, heat syncope, exhaustion, and heat stroke may result. Although it is beyond the scope of this chapter, optimal heat regulation is important.

Understanding the kinetic chain is important for determining the sports-related injury patterns. The kinetic chain links the upper extremity through the core muscle segments and the hip to the lower extremity. In this regard, any pathologic process that disturbs the groin, hip, and abdominal musculature can affect power generation of the lower extremities to be transmitted to the upper extremities and racquet, resulting in an increased risk of injury to the shoulder and upper extremity. In particular in power shots, such as the serve, overhead smash, and groundstrokes, the player must efficiently use the kinetic chain to minimize load on the shoulder and elbow. Tennis players with more effective knee flexion-extension during the service action were associated with lower loading on the shoulder and elbow [4]. Throughout the game, players are reacting and adjusting their body position rapidly and continuously. The kinetic chain is active during every stroke [5]. An injury somewhere within the kinetic chain may result in injury to another area within the kinetic chain due to attempts to compensate for the injury while trying to maintain power.

30.3 Sports-Related Injuries

Descriptive epidemiological studies of tennis injuries have found that injuries occur most frequently in the lower extremity, followed by the upper extremity, then trunk [3, 6]. Upper extremity injuries are mostly chronic and a result from repetitive overuse. In badminton, squash and racquetball, injuries to the upper extremity are frequent due to the repetitive fast swinging of the lighter racquets with more whipping or snapping motions to attain power for the swing. In tennis, stresses increase to the upper extremity during the acceleration and follow-through stages, especially the serve and overhead smash. In all racquet sports, ankle sprains are the most common acute injury and occur most often on hard courts. Squash and racquetball participants are more likely to sustain acute injuries, as opposed to the predominance of overuse injuries with tennis and badminton. In squash and racquetball, the players are not separated by a net, are in close quarters, swing the racquet powerfully, and since balls can be played off the walls and ceiling, there is a lot of pivoting and sudden stops and starts. In top-level tennis players, female players experience more injuries than male players. Upper and lower extremity injuries occurred more in females, while lower limb injuries were more prominent in males [7].

30.3.1 Upper Extremity

30.3.1.1 Shoulder

The shoulder has the widest range of motion of any major joint in the body and does so at the expense of stability. The shoulder girdle is in that way especially prone to injury because it has to maximally accelerate and decelerate the arm while maintaining precise control over the racquet at ball strike. A careful balance between mobility and stability is necessary to maximize performance. A player performs repetitive motions that generate high-magnitude forces about the shoulder during the various strokes. Increased range of motion, particularly shoulder external rotation for serving, is beneficial to generate maximal velocity and spin (Fig. 30.1). The serve, high forehand and backhand volley strokes and overhead smash place large stresses on the shoulder and rotator cuff. Due to the repetitious nature of these forces, it is important to maintain the balance between motion and stability in the shoulder.

Overuse injuries to the shoulder are prevalent among tennis players of all skill levels and have been shown to contribute to nearly 9–17% of all tennis injuries [8]. As many as 50% of adult tennis players and 30% of junior players complained of shoulder pain at some time in their career [9]. It is usually the older players that may have symptoms such as rotator cuff impingement, rotator cuff tear, or degenerative arthritis of the glenohumeral and/or AC joints. The young player's rotator cuff symptoms are more often secondary to mild instability of the glenohumeral joint. Instability may result in labral degeneration or tears.

The primary overuse injury to the shoulder is rotator cuff inflammation. This usually occurs as a result of chronic repetitive swinging of the racquet. In tennis it is associated with shoulder abduction such as with serves, overhead smashes, high backhand volley, and the follow-through with the backhand stroke. In racquetball it occurs commonly due to lengthy ceiling rallies in this sport. While rotator cuff inflammation may be the result of overuse, or outlet impingement, it may also be seen in other situations, such as rotator cuff tears, instability or micro-instability, superior labral anterior to posterior injuries (SLAP), internal impingement, glenohumeral internal rotation deficit (GIRD), SICK scapula (see below), shoulder stiffness, or scapular dyskinesis [10].

Scapular dyskinesis has been shown to contribute to rotator cuff pathology, as the rotator cuff muscles synchronicity is disrupted by abnormal scapular range of motion. Alterations in scapular motion or position can significantly affect overall glenohumeral biomechanics, as it is critical in shoulder function [11]. The term "SICK scapula" was introduced to describe a pathological state of the scapula seen in overhead athletes which is characterized by [1] Scapular

Fig. 30.1 Serving of a professional tennis player: note the external rotation of the shoulder in late cocking phase/early acceleration phase. This increased external rotation allows for more time to accelerate the arm and thus racket, to hit the serve hard. (Picture used with permission from Marc R. Safran, MD)

malposition, [2] Inferior medial border prominence, [3] Coracoid pain and malposition, and [4] Kinesis abnormalities of the scapula [12]. This may potentiate rotator cuff symptoms due to the protracted scapula not allowing the acromion to rotate sufficiently out of the way from the greater tuberosity.

Tennis players often develop increased glenohumeral external rotation in the dominant shoulder on the expense of internal rotation. The repetitive loading in the late cocking phase of the tennis serve causes microtrauma to the anterior shoulder capsular structures and the anterior labrum, causing subtle anterior instability [10]. This instability allows the humeral head to translate anteriorly, bringing the greater tuberosity of the humerus and the rotator cuff in close proximity to the posterior glenoid. This can cause internal impingement of the undersurface of posterosuperior rotator cuff (supra- and infraspinatus) between the humeral head and the posterosuperior rim of the glenoid. Pathologic glenohumeral internal rotation deficit is associated with internal impingement [13]. This damages the rotator cuff tendons and may also lead to posterior superior labral pathology [14]. Players usually complain of pain in the posterior part of the shoulder with overhead activity. Alternatively,

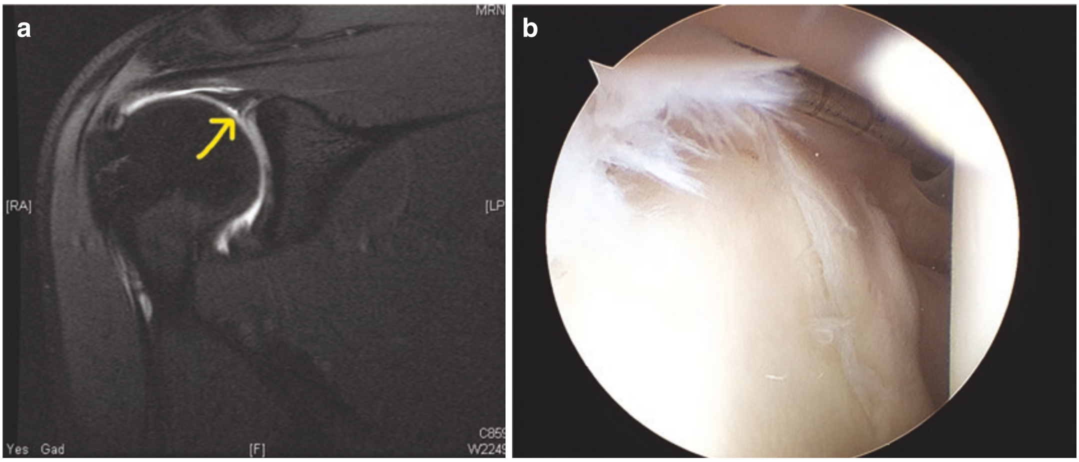

Fig. 30.2 SLAP Lesion—MRI (**a**) and arthroscopic photo (**b**) of SLAP lesion in a professional women's tennis player. (Images used with permission from Marc R. Safran, MD)

anterior instability could also cause overuse of the rotator cuff because of trying to maintain shoulder stability. It has also been shown that posterior capsular contracture results in posterior shoulder tightness and internal rotation contracture that results in altered humeral head motion. The humeral head moves in a posterior and superior direction with posterior inferior glenohumeral ligament (IGHL) contracture leading to increased posterior-superior labral wear, internal impingement, and possibly SLAP tears (Fig. 30.2). [15]

Initial treatment of these shoulder pathologies is usually nonoperative. Rehabilitation exercises should focus on the inciting factors, such as the posterior capsular tightness and any scapular dyskinesis which may be present. Posterior capsular stretching exercises include the "sleeper stretch" and "cross body stretch", while scapular dyskinesis can be addressed by several exercises which specifically target the scapular stabilizers. Restoration of rotator cuff muscle balance and proprioceptive exercises are also important parts of the rehabilitation protocol. Should nonoperative management fail to improve symptoms, then surgical management is necessary. This may include repairing the SLAP lesions, if present, and possibly cutting the posterior IGHL to gain internal rotation. Patient satisfactory results are good following arthroscopic rotator cuff repair (RCR) in both small and larger tears. Rotator cuff repair in middle-aged tennis players showed a good return to tennis rate and a high satisfaction score. Smaller rotator cuff tears have a better return, rate to sports compared to larger tears [16, 17]. Another study also found that most athletes (70.2%) were able to return to a preinjury level of play after arthroscopic RCR. While recreational sports participation (73.3%) was associated with higher return, competitive sports (61.5%) and overhead sports (38%) were associated with lower return. Exactly why all athletes do not return remains uncertain and likely multifactorial [18]. A study in professional female tennis players that underwent arthroscopic shoulder surgery showed that 88% returned to play with a mean time of 7.0 months. However, the study showed a prolonged, and often incomplete, recovery time to previous level; only 50% of the players were able to return to their pre-injury ranking after 2.5 years [19].

Biceps tendonitis is another common complaint in tennis players and may be due not only to the overhead service motion, but also the pronation/supination motions of the forearm required for forehands and backhands. Rotator cuff dysfunction, as a result of strain, tendinopathy, or tearing, may also be a result of biceps tendonitis.

The biceps may also become impinged by the humeral head and acromion or due to its proximity to the inflamed rotator cuff.

To prevent and rehabilitate the tennis player's shoulder overuse syndromes imbalances must be corrected. Specifically, it is important to:

1. improve glenohumeral internal rotation;
2. stretch the posterior capsule;
3. strengthen the posterior rotator cuff and scapular stabilizing muscles.

Acute shoulder injuries are uncommon in tennis, though shoulder dislocations and AC Joint separations may occur from falls.

30.3.1.2 Elbow

Injuries to the elbow are primarily due to overuse and often involve tendinous structures of the lateral and medial humeral epicondyle. Lateral epicondylitis (tennis elbow), medial epicondylitis (golfer's elbow), and injury to the medial epicondylar apophyseal growth plate in skeletally immature players are common injuries about the elbow seen in tennis players. Due to the rapid, repetitive arm movements required in racquet sports, medial and lateral epicondylitis has been reported in tennis, badminton, squash, and racquetball. However, the term "tennis elbow" for lateral epicondylitis is misleading since only 10% of all patients with lateral epicondylitis play tennis [20]. The pathology is caused by degeneration of the deeper fibers of the extensor carpi radialis brevis (ECRB) and is attributable to overuse of wrist extension, gripping the racquet (usually if grip size incorrect) and excessive pronation/supination. Non-optimal contributions from other segments of the kinetic chain and poor overall stroke biomechanics and whole-body fitness can contribute to medial or lateral epicondylitis.

The incidence of lateral epicondylitis in elite and recreational tennis players ranges from 37% to 57% [21]. Lateral epicondylitis involves the wrist extensors, and is thought to be the result of repetitive microtraumatic injury that results in microtears of the muscular origin. Focal degeneration and healing with vascular and fibroblastic proliferation suggests this is a degenerative process; however, it has been suggested that there may be an inflammatory component [22, 23]. Technique flaws are considered to predispose epicondylitis lateralis. It can be the cause of a faulty backhand technique with the elbow leading, excessive forearm pronation during a forehand topspin, and excessive wrist flexion during a service. Additional potential risk factors are racquet type, grip size, string tension, court surface, and weight of the ball. On the other hand, it has been shown that there was a 63% higher incidence of shoulder injury among recreational players with a tennis elbow than among players without a history of tennis elbow [24]. Medially, the medial epicondylitis is caused by overuse of the wrist flexor/pronator muscles. Medial epicondylitis occurs much less frequently than lateral epicondylitis in the recreational athlete, though it tends to occur more often than lateral epicondylitis in higher level tennis players. In tennis, it is associated with snapping the wrist during the serve or hitting a forehand with a lot of "top spin". Acute overload to the medial elbow may also result in an acute muscle strain of the flexor pronator muscle group (Fig. 30.3). Medial elbow pain is common in squash and racquetball due to the snapping mechanism of kill shots and to the lighter racquet.

Treatment of epicondylitis is primarily nonoperative, with 90% of patients responding to conservative measures [25]. These measures include rest, anti-inflammatory medications, cock-up wrist splints, physical therapy focusing on stretching and eccentric strengthening of the wrist extensor and flexor muscle groups. In lateral epicondylitis, a counterforce brace can be used when returning to activity. Prevention of lateral and medial epicondylitis consists mostly of stretching and strengthening of the wrist flexors and extensors. The muscular endurance of the wrist and forearm musculateur should be increased. Steroid injections are effective on short term, but long-term effects are worse than patients without injection. Platelet-rich plasma (PRP) has been shown to have good results although more research is needed [20, 26]. Surgery tends to be less effective in the medial aspect, whereas a lateral tennis elbow release is usually a satisfactory

Fig. 30.3 Acute Pronator Muscle Strain in professional male tennis player (pt = pronator teres). (Images used with permission from Marc R. Safran, MD)

procedure in the 10% of cases that are resistant to conservative therapy [27].

Tennis players place tremendous tensile strain on the medial elbow, particularly on the ulnar collateral ligament (UCL). These forces are highest during the late cocking and early acceleration phases of the service motion and may also be notable during the forehand groundstroke, especially when hitting the ball late [28]. The most common causes of an UCL injury in the tennis player is chronic attenuation due to repetitive performance of the service motion. Players with UCL injuries will typically complain of a loss of "pop" or "zip" on the serve, loss of power, and pain during the late cocking or early acceleration phase. Treatment consists of nonoperative therapy and sometimes followed by surgical reconstruction. Nonoperative measures include rest of overhead sport activities, anti-inflammatory medications, and physical therapy. Operative treatment consists of reconstruction of the torn ligament, usually using a free graft [29]. Return to sport (including tennis) following operative intervention has been reported to be 80–90% [30].

30.3.1.3 Hand and Wrist

Racquetball and squash require a snapping motion of the wrist as part of the normal stroke. This puts stress on the tendons and ligaments of the wrist that may result in an overuse injury. This is often manifested as tendinitis in the wrist extensor tendons either in the dorsal compartment or at the distal insertions. Tendinitis of the wrist may develop in tennis players who place a lot of spin on their shots or in novices with mechanically improper technique. Wrist extensors are more frequently involved than flexor tendons. DeQuervain's stenosing tenosynovitis is one of the most common tendon problems seen in the tennis player. This usually occurs due to shearing within the fibro-osseous sheath from repeated ulnar deviation. It can be treated by cortisone injection and physical therapy but often needs surgical release. Wrist injuries can be classified as radial-sided and ulnar-sided. A study found that radial-sided wrist injuries occurred most often in players who utilized an Eastern grip. Ulnar-sided wrist injuries were more frequently associated with Western or Semi-Western grips [31].

In tennis, wrist injury to the extensor carpi ulnaris (ECU) tendon is often involved. It may occur during forehand groundstrokes or in the non-dominant wrist of players with two-handed backhands, possibly due to the overuse during the backswing. It presents itself as ulnar-sided wrist pain; overuse and/or improper technique is often the cause of the tendonitis. The forehand stroke is the most frequently utilized groundstroke in tennis and is performed with the dominant forearm in full supination and the wrist flexed in ulnar deviation. Wrist flexion and extension are important components of ball velocity after ball-racquet impact. Dynamic repetition of this stroke depends largely on the integrity of the ECU and its ability to contribute to wrist flexion and extension. Attenuation of the ECU subsheath may be caused by a sudden volar flexion and ulnar deviation stress, such as hitting a low forehand. ECU tendonitis is frequently associated with triangular fibrocartilage complex (TFCC) tears. The TFCC is a stabilizer of the distal radioulnar joint. An MRI arthrogram is recommended to confirm the diagnosis of a TFCC tear [9, 32]. TFCC tears can be accompanied by instability of the distal radioulnar joint (DRUJ). This often needs acute surgical treatment. If DRUJ instability is absent, nonsurgical treatment is the initial management. Conservative treatment includes temporary splint immobilization of the wrist and forearm, corticosteroid joint injection, and physical therapy [33]. Surgical management includes arthroscopic techniques of repair or debridement, depending on the pathology present. In case of DRUJ instability, open surgical repair is indicated. The return-to-play prognosis is good following treatment after an average of 3.3 months [34].

Subluxation of the extensor carpi ulnaris tendon has been reported in tennis players and is associated with hypersupination and ulnar deviation, such as with a back-spin slice, low forehand, slice, or topspin service motion. A player may notice a snapping wrist; it usually requires surgical correction for return to play [32].

30.3.1.4 Central Region Injuries (Back and Trunk)

Overuse injuries of the central region are common in the racquet sports athlete. In badminton, musculoskeletal back pain has been implicated among badminton players due to overuse, overreaching, lunging for the shuttlecock, hyperextension, and bending to reach low [5]. It has been reported that 38% of professional male tennis players reported missing at least one tournament because of low back pain and up to 50% of elite junior tennis players noted a history of low back pain [9, 35]. There are a variety of sources causing low back pain in the player. High demands placed on the lower back and trunk combined with low flexibility patterns result in frequent overuse-type injuries. The tennis serve can particularly stress the spine; it has combined movements of extension, lateral flexion, and rotation that are inherent in the cocking or loading phase (Figs. 30.1 and 30.4). The repetitive motions and stresses to the lumbar spine are thought to cause spondylolysis and spondylolisthesis, in tennis and other racquet sports [36].

The three areas most often involved are as follows:

1. The posterior midline paraspinal musculature (used in the service motion, when charging the net or when dropping straight back for a volley);
2. The peripheral trunk musculature, i.e., the quadratus lumborum or oblique muscles (used during the service motion or in ground strokes);
3. The rectus abdominus (tears in this muscle may be associated with hitting overhead strokes, volleys, or serves).

Other potential causes of low back pain include intervertebral disk degeneration and herniation, facet impingement, and spondylolysis due to the repetitive hyperextension and rotation of the spine. Injuries to the abdominal muscles occur frequently during serves, particularly to the non-dominant rectus abdominus muscle and obliques (Fig. 30.5). Open stance forehand strokes are purported to be the cause of the increasing incidence of abdominal muscle injury as well [8]. Extensive strengthening of the abdominal muscles contributes to the prevention of pain and injury to the low back and abdomen.

30.3.2 Lower Extremity

Racquet sports place unique stress on the soft tissues of the lower leg. The extreme ranges of motion that the hip, knee, ankle, lower leg, and foot must go through can result in numerous injuries. In racquet sports, lower extremity injuries are about twice as frequent as upper extremity or central region overuse injuries [9]. Lower extremity injuries are common for two reasons. First, each of these sports involves repeated short bursts of activity with quick stop-start and sharp, lateral movements, and accelerations which place high demands on the lower extremities (Fig. 30.6). Second, athletes may be less flexible and weaker in specific anatomic areas, including the lower

Fig. 30.4 Back extension and rotation with tennis serve in professional woman tennis player. (Picture used with permission from Marc R. Safran, MD)

extremity, which may predispose them to overuse injuries.

30.3.2.1 Hip/Thigh

The loading and multidirectional movement patterns, abrupt stopping, starting, and twisting in racquet sports put great forces on the hip. The hip is important in the kinetic chain and subjected to extremes of motions: flexion, extension, and rotation. In 8–27% of injuries the hip/groin is involved in high-level tennis players [8]. Hip pain can be a cause of intra-articular pathology or extra-articular pathology. There are numerous other factors that can cause a painful hip, such as hip flexor strain, peri-trochanteric pain, adductor strain, and core muscle injury/athletic pubalgia ("sports hernia") (Fig. 30.7) or referred pain from the lumbar spine [37].

Most of the injuries are muscle strains. The most common areas for strains in the thigh are the adductor muscles (groin pulls) and the hamstrings (Fig. 30.8). Quadriceps and groin strains are particularly common in badminton due to the leaping and lunging required in this quick sport. Adductor muscle strains usually result from sudden changes in direction, particularly when attempting to stop lateral movement by sliding or posting the lead foot [37]. Slipping on clay courts, resulting in "the splits" (Fig. 30.6a), may also strain the adductor muscles. Hamstring tears may occur at either end of the muscle and are usually associated with explosive acceleration, for example, when sprinting or charging toward the net. Quadriceps strains, and potentially ruptures in the older athlete, are known to occur, particularly when a player slides on clay courts with the knee flexed and then the player tries to forcefully extend the knee (Figs. 30.6b and 30.9) [8]. Decreased hip range of motion is associated with an increased risk for groin injury and low back injury (Fig. 30.7) [38]. Thus, stretching, core training, hip abduction strength, and training of muscular endurance around the hip are important for prevention of hip or thigh injuries. Lunge with rotations mimics joint angles and movement patterns used during tennis ground strokes.

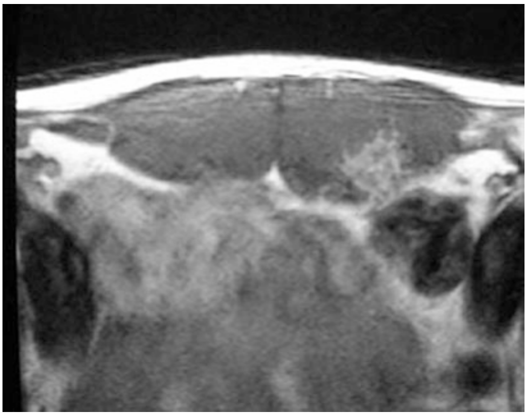

Fig. 30.5 MRI of the lower abdomen of a right-handed professional female tennis player. The left (non-dominant) rectus abdominus demonstrates hypertrophy (regularly seen in high level tennis players) and edema, consistent with a strain of the rectus abdominus. (Image used with permission from Marc R. Safran, MD)

Another cause for hip pain in the player can be femoroacetabular impingement (FAI). The typical presentation is a tennis player reporting groin pain or anterolateral hip pain. Lunging for a low ball/shuttle may be painful, and it affects other structures of the kinetic chain. The hip shows limited internal rotation (measured in 90-degree flexion) and pain in flexion. Imaging should include radiographs to evaluate the cam or pincer lesion. Further imaging should include a magnetic resonance arthrogram (MRA) to detect

Fig. 30.6 Stresses on lower extremities moving for tennis ball in male (**a**) and female (**b**) professional tennis players. The male is doing a mild "split" and the female a lunge. (Pictures used with permission from Marc R. Safran, MD)

Fig. 30.6 (continued)

intra-articular pathology, such as labral tears. Treatment of symptomatic FAI with intra-articular pathology is surgical in order to address the cause of the pathology and resolve the damage to the cartilage or labrum. Return rate to sports is usually good [39].

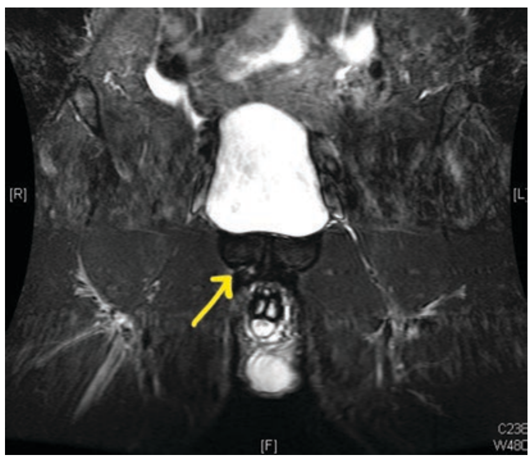

Fig. 30.7 MRI of the pelvis of a male ATP tennis player who had limited hip range of motion consistent with femoroacetabular impingement. However, he had no hip pain, and this MRI demonstrates a core muscle injury on the right (Arrow). (Image used with permission from Marc R. Safran, MD)

30.3.2.2 Knee

Nearly 20% of all injuries are knee injuries, with 70% of the injuries being traumatic and 30% overuse [9]. Due to the sudden changes in direction, repetitive stop–start activity, lunging and jumping, the knee is susceptible to overload and overuse injuries.

Knee ligament and meniscal injuries are more common in squash, badminton, and racquetball than in tennis. The frequent pivoting maneuvers in racquetball and squash, particularly on wooden floors with excellent traction, predispose players to meniscal and ligamentous injuries [5]. Medial collateral ligament injuries are the most common injuries, though ruptures of the anterior cruciate ligament have been reported. Patellofemoral pain is more common in tennis compared to the other racquet sports. Patellofemoral pain may be related to the sudden start and stops of these sports as well as lunging and jumping. The patel-

Fig. 30.8 Professional Women's Tennis player with taping of the upper thigh for groin strain. (Picture used with permission from Marc R. Safran, MD)

lofemoral joint is susceptible to overuse injuries. This may be commonly manifested as Osgood-Schlatter's syndrome (tibial tubercle apophysitis) in young racquet sports players, patellar tendinitis (jumper's knee) (Fig. 30.10) and quadriceps tendinitis in adults, and patellofemoral syndrome or chondromalacia patellae [9]. Treatment is limitation of play, anti-inflammatory medication, and physical therapy for strengthening of the muscles about the knee. It should especially focus on the quadriceps and hip external strengthening. However, as stated earlier, the whole kinetic chain is of importance in injuries. Core hip stability, proprioception, and abdominal strength are important in controlling knee movement in tennis.

30.3.2.3 Leg

Gastrocnemius muscle strains are common and occur during repeated, explosive accelerations of the leg, such as sprinting or jumping. A "Tennis leg" is described as a strain or partial tear of the gastrocnemius at its medial origin. These strains and injuries occur typically when a foot is sud-

Fig. 30.9 MRI of upper thigh of professional women's tennis player demonstrating an acute Rectus Femoris Strain. (Image used with permission from Marc R. Safran, MD)

denly forced from plantarflexion into dorsiflexion while the knee is in full extension. This motion occurs during the serve during the first step forward and during ground strokes as forward and lateral lunges are executed. Players usually feel a sudden onset of pain in the upper to middle third of the calf, and players note that it feels like they were shot or hit in the leg by the ball or a racquet. These injuries are self-limiting, treatment involves rest, ice, elevation, and physical therapy, and take about 6 weeks to recover.

Achilles tendinitis and achilles tendon ruptures are more frequently seen in racquetball and squash. Achilles tendinitis can be chronically debilitating and is often associated with recent change in the amount or intensity of play, especially to clay courts in tennis. A sudden increase in activity, including changing surfaces from hard court to clay, or the effect of long-term

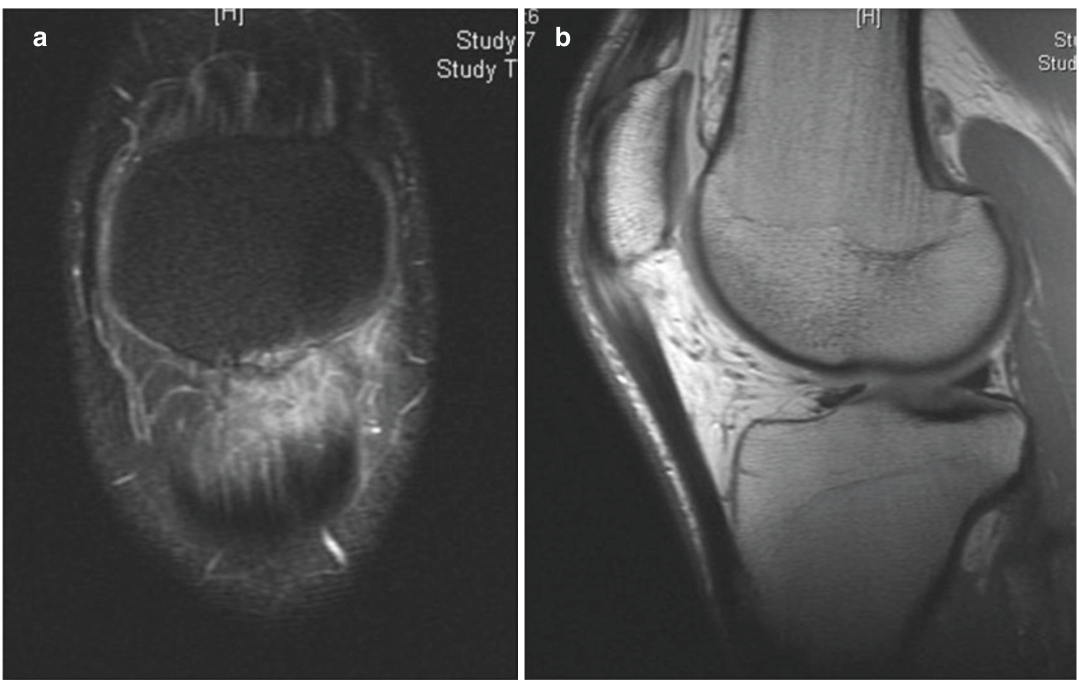

Fig. 30.10 MRI of a male professional tennis player with patellar tendinitis— (**a**) demonstrating a coronal view with T2 weighting and (**b**) showing a sagittal view with T1 weighting, showing the thickened and inhomogeneous proximal patellar tendon. (Images used with permission from Marc R. Safran, MD)

repetitive stress on the tendon may lead to the development of microtears or degeneration of the tendon. An achilles tendon rupture occurs typically during a strong dorsiflexion force that is applied to the ankle as the gastrocnemius–soleus complex simultaneously contracts to plantarflex the ankle: an eccentric contraction. Patients note a snap and inability to push off on the affected foot. Achilles tendon ruptures are common in older racquet sports participants, usually over 40 years of age, or the "weekend warriors", due to the sudden bursts of speed that are necessary in each of the sports [40]. This is a more severe injury, and return to play is prolonged after this injury, regardless if treated surgically or in a cast.

30.3.2.4 Ankle

Ankle sprains are the most common macrotrauma injury in all racquet sports. Each of these sports demands frequent running, pivoting, stopping, and starting movements as well as lunging and jumping [9]. As a result of high twisting forces, injuries to the ankle occur often [9]. Most injuries occur during twisting while the ankle is in plantarflexion, resulting in lateral ankle sprains. Initial treatment of ankle sprains includes a combination of rest, ice, elevation, compression, immobilization. Secondary treatment, usually after 2–3 weeks, is to restore ankle proprioception, strength, and flexibility [41]. Chronic, recurrent ankle sprains usually occur secondary to incomplete rehabilitation. If chronic instability persists as a result of an ankle sprain, surgical reconstruction of the ligaments might be indicated [42].

30.3.2.5 Foot

Orthopedic foot injuries in players may include stress fractures, plantar fasciitis, and hallux rigidus. Stress fractures in racquet sports athletes occur most commonly at the base of the fifth metatarsal, the second or third metatarsal diaphysis, lateral process of the talus, and, occasionally, the navicular neck (Fig. 30.11). Older players with poorly cushioned footwear with absent medial arch support may be prone to plantar fasciitis or rupture of the plantar fascia. Tennis players are particularly susceptible to this injury due to the great amount of time spent on the balls of their feet while making quick changes in different directions. Hallux rigidus, degeneration of the first metatarsal phalangeal joint with dorsal exostosis, occurs frequently in tennis players due to the excessive dorsiflexion of the first toe during play. Players have pain during push-off. Another injury that players are subject to is the "tennis toe". This is an injury to the hallux or second toe from forceful and repetitive abutment of these toes against the toe box of the shoe. This can lead to subungual hematomas, nail bed injuries, or to injury to the interphalangeal or metatarsophalangeal (MTP) joints. A right shoe size and adequate padding of the toebox are important to prevent this injury.

Fig. 30.11 MRI of the foot of a professional female tennis player demonstrating navicular bone edema of a navicular stress fracture. (Image used with permission from Marc R. Safran, MD)

30.4 Prevention of Injury

Flexibility is an important part of injury treatment and prevention. For stretching to be effective, it must be done on a regular basis and should be done when warmed up. It is important to stretch before and after activity. Stretching should not be painful. All stretches should be done in a slow static manner, with no bouncing. It is important to hold a stretch for at least 30–60 seconds and repeated several times.

Exercises of the whole kinetic chain are important in order for prevention of injuries. These exercises include squats for leg strengthening, to recruit power generation, and load absorption; trunk rotations; scapular stabilization; and shoulder and wrist co-contractions [43]. However, also strengthening and balancing tennis-specific muscle groups are important. Weak or inflexible muscles of the arm are a major contributor to epicondylitis of the elbow [25]. The exercises will not only strengthen the damaged muscle tendon unit to prevent injury but will also help improve your game. Weak and inflexible muscle in the back of the shoulder may result in rotator cuff inflammation, pain, and limited function. Weak and inflexible abdominal and back muscles may predispose to these muscles being injured. Patellofemoral pain may be diminished or prevented by maintaining flexibility of the hamstrings and strength of the quadriceps muscles, particularly the vastus medialis obliquus. Achilles tendon and gastroc-soleus injuries may be prevented by stretching before play and maintaining good cardiovascular fitness to prevent fatigue.

Management of the tennis player, junior, professional, or recreational, may require more information than the usual musculoskeletal examination of the non-tennis player with similar maladies. The evaluation of the tennis player should include information about the type of strokes used for the serve, forehand (closed versus open stance), and backhand (one- versus two-handed), how much they play per week (singles and doubles), the amount of other sports or training they do, whether or not they stretch and what areas they stretch, do they stretch before and/or after practice and/or play, court surface played upon, type of racquet (including length of time used and grip size) and string (include tension), and any recent injuries. It is important to observe the players stroke mechanics and grip size. Often players with symptoms may have had another injury along the kinetic chain, clinically apparent or not, and as result of compensating for another injury, they may develop symptoms somewhere else along the kinetic chain.

References

1. Pluim BM, Miller S, Dines D, Renström P, Windler G, Norris B, et al. Sport science and medicine in tennis. Br J Sports Med. 2007;41(11):703–4.
2. Phomsoupha M, Laffaye G. The Science of Badminton: game characteristics, anthropometry, physiology, visual fitness and biomechanics. Sport Med. 2015;45(4):473–95.
3. Pluim BM, Staal JB, Windler GE, Jayanthi N. Tennis injuries: occurrence, aetiology, and prevention. Br J Sports Med. 2006;40(5):415–23.
4. Reid M, Elliott B, Alderson J. Shoulder joint loading in the high performance flat and kick tennis serves. Br J Sports Med. 2007;41(12):884–9.
5. Marchena-Rodriguez A, Gijon-Nogueron G, Cabello-Manrique D, Ortega-Avila AB. Incidence of injuries among amateur badminton players: a cross-sectional study. Medicine (Baltimore). 2020;99(18):1–5.
6. Lynall RC, Kerr ZY, Djoko A, Pluim BM, Hainline B, Dompier TP. Epidemiology of National Collegiate Athletic Association men's and women's tennis injuries, 2009/2010–2014/2015. Br J Sports Med. 2016;50(19):1211–6.
7. Gescheit DT, Cormack SJ, Duffield R, Kovalchik S, Wood TO, Omizzolo M, et al. Injury epidemiology of tennis players at the 2011–2016 Australian Open Grand Slam. Br J Sports Med. 2017;51(17):1289–94.
8. Abrams GD, Renstrom PA, Safran MR. Epidemiology of musculoskeletal injury in the tennis player. Br J Sports Med. 2012;46(7):492–8.
9. Kibler WB, Safran MR. Tennis injuries. Med Sport Sci. 2005;48:120–37.
10. Kvitne RS, Jobe FW. The diagnosis and treatment of anterior instability in the throwing athlete. Clin Orthop Relat Res. 1993;(291):107–23.
11. Van Der Hoeven H, Kibler WB. Shoulder injuries in tennis players. Br J Sports Med. 2006;40(5):435–40.
12. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology part III: the SICK scapula, scapular dyskinesis, the kinetic chain, and rehabilitation. Arthrosc J Arthrosc Relat Surg. 2003;19:641–61.
13. Kibler WB, Kuhn JE, Wilk K, Sciascia A, Moore S, Laudner K, et al. The disabled throwing shoulder: spectrum of pathology – 10-year update. Arthrosc J Arthrosc Relat Surg. 2013;29(1):141–161.e26.
14. Kibler WB. The role of the scapula in athletic shoulder function. Am J Sports Med. 1998;26:325–37.
15. Burkhart SS, Morgan CD, Kibler WB. The disabled throwing shoulder: spectrum of pathology part I-III. Arthrosc J Arthrosc Relat Surg. 2003;19:404–20.
16. Bigiliani LU, Kimmel J, McCann PD, Wolfe I. Repair of rotator cuff tears in tennis players. Am J Sports Med. 1992;20(2):112–7.
17. Sonnery-Cottet B, Edwards TB, Noel E, Walch G. Rotator cuff tears in middle-aged tennis players: results of surgical treatment. Am J Sports Med. 2002;30:558–64.

18. Altintas B, Anderson N, Dornan GJ, Boykin RE, Logan C, Millett PJ. Return to sport after arthroscopic rotator cuff repair: is there a difference between the recreational and the competitive athlete? Am J Sports Med. 2020;48(1):252–61.
19. Young SW, Dakic J, Stroia K, Nguyen ML, Harris AHS, Safran MR. High incidence of infraspinatus muscle atrophy in elite professional female tennis players. Am J Sports Med. 2015;43(8):1989–93.
20. Keijsers R, de Vos RJ, Kuijer PPFM, van den Bekerom MPJ, van der Woude HJ, Eygendaal D. Tennis elbow. Shoulder Elb. 2019;11(5):384–92.
21. Ellenbecker TS, Pluim B, Vivier S, Sniteman C. Common injuries in tennis players: exercises to address muscular imbalances and reduce injury risk. Natl Strenth Cond Assoc. 2009;31(4):50–8.
22. Rineer CA, Ruch DS. Elbow Tendinopathy and tendon ruptures: epicondylitis, biceps and triceps ruptures. J Hand Surg Am. 2009;34(3):566–76.
23. Bazancir Z, Fırat T. A potential factor in the pathophysiology of lateral epicondylitis: the long sarcomere length of the extensor carpi radialis brevis muscle and implications for physiotherapy. Med Hypotheses. 2019;130:75–8.
24. Priest J, Braden V, Gerberich S. An analysis of players with and without pain. Phys Sport. 1980;8:81–91.
25. Keijsers R, Eygendaal D. Conservative treatment in Lateral Elbow Pain. Elb Sport. 2016:125–31.
26. Mishra AK, Skrepnik NV, Edwards SG, Jones GL, Sampson S, Vermillion DA, et al. Efficacy of platelet-rich plasma for chronic tennis elbow: a double-blind, prospective, multicenter, randomized controlled trial of 230 patients. Am J Sports Med. 2014;42(2):463–71.
27. Baker CL Jr, Baker CL III. Long-term follow-up of arthroscopic treatment of lateral epicondylitis. Am J Sports Med. 2008;36(2):254–60.
28. Elliott B, Fleisig G, Nicholls R, Escamilia R. Technique effects on upper limb loading in the tennis serve. J Sci Med Sport. 2003;6:76–87.
29. Rohrbough JT, Altchek DW, Hyman J, Williams RJ, Botts JD. Medial collateral ligament reconstruction of the elbow using the docking technique. Am J Sports Med. 2002;30(4):541–8.
30. Dines JS, ElAttrache NS, Conway JE, Smith W, Ahmad CS. Clinical outcomes of the DANE TJ technique to treat ulnar collateral ligament insufficiency of the elbow. Am J Sports Med. 2007;35:2039–44.
31. Tagliafico AS, Ameri P, Michaud J, Derchi LE, Sormani MP, Martinoli C. Wrist injuries in nonprofessional tennis players: relationships with different grips. Am J Sports Med. 2009;37(4):760–7.
32. Montalvan B, Parier J, Brasseur JL, Le Viet D, Drape JL. Extensor carpi ulnaris injuries in tennis players: A study of 28 cases. Br J Sports Med. 2006;40:424–9.
33. Henry MH. Management of acute triangular fibrocartilage complex injury of the wrist. J Am Acad Orthop Surg. 2008;16(6):320–9.
34. McAdams TR, Swan J, Yao J. Arthroscopic treatment of triangular fibrocartilage wrist injuries in the athlete. Am J Sports Med. 2009;37(2):291–7.
35. Hutchinson MR, Laprade RF, Burnett QM, Moss R, Terpstra J. Injury surveillance at the USTA boys' tennis championships: a 6-yr study. Med Sci Sports Exerc. 1995;7:826–30.
36. Alyas F, Turner M, Connell D. MRI findings in the lumbar spines of asymptomatic, adolescent, elite tennis players. Br J Sports Med. 2007;41(11):836–41.
37. Safran MR. Evaluation of the painful hip. Aspetar Target Top Sport Med Tennis. 2014;3:517–25.
38. Verrall GM, Slavotinek JP, Barnes PG, Esterman A, Oakeshott RD, Spriggins AJ. Hip joint range of motion restriction precedes athletic chronic groin injury. J Sci Med Sport. 2007;10:463–6.
39. Philippon M, Schenker M, Briggs K. Femoroacetabular impingement in 45 professional athletes: Associated pathologies and return to sport following arthroscopic decompression. Knee Surgery, Sport Traumatol Arthrosc. 2007;15:908–14.
40. Pedowitz D, Kirwan G. Achilles tendon ruptures. Curr Rev Musculoskelet Med. 2013;6(4):285–93.
41. Safran MR, Zachazewski JE, Benedetti RS, Bartolozzi AR, 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):S438–47.
42. Pijnenburg A, Bogaard K, Krips R, Marti R, Bossuyt P, van Dijk N. Operative and functional treatment of rupture of the lateral ligament of the ankle. A randomised, prospective trial. J Bone Joint Surg Br. 2003;85:525–30.
43. Dines JS, Bedi A, Williams PN, Dodson CC, Ellenbecker TS, Altchek DW, et al. Tennis injuries: Epidemiology, pathophysiology, and treatment. J Am Acad Orthop Surg. 2015;23(3):181–9.

Triathlon

31

Philip Friere Skiba and Kaleigh Suhs

31.1 Introduction

Triathlon is relatively a new sport in the pantheon of endurance exercise, with the first competitions held in the 1970s [1]. Indeed, it did not appear in the Olympics until the 2000 Sydney games. The triathlon consists of a swim, bike, and run, typically executed in that order (Fig. 31.1a–c). Races may be characterized as sprint, Olympic, half-iron, or iron distance (Table 31.1), noting that the moniker "Ironman" is a registered trademark of the World Triathlon Corporation. A triathlon may be completed with little more than a swimsuit, a bicycle, and running shoes, although rules do exist with respect to the type and construction of the cycling equipment and wetsuit used.

At first glance, the injury risks of triathlon would seem to be the sum of those of the individual sports, discussed elsewhere in the present work. However, there are particular physiological and biomechanical challenges to training in several disciplines concurrently [2]. With this in mind, careful planning is necessary to properly prepare an athlete [3]. This is problematic: according to one study, 47% of amateur triathletes lacked a precise training plan [4]. As ability varies widely due to the amateur nature of the sport and the background of the athletes, and the volume of training daunting, this can have particular consequences. With this in mind, it is reasonable to divide athlete injury between those that are the result of physiological insult and those that are the result of mechanical insult.

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31.2 Exercise Intolerance

Broadly speaking, athletes often present with an inability to execute desired workouts, or of being unable to keep up with training partners or competitors. While there can be a number of reasons for this, ranging from cardiopulmonary pathology to musculoskeletal injury, the first (and most commonly ignored) step is to document that there is in fact a bona-fide performance incompetence evident. One way to do this is through the use of the two-parameter critical power (CP) model (for review, see Jones et al. 2010 [5]). An athlete's personal best performances over different durations are graphed. For example, swim velocity over 200, 400, and 800 m, or cycling power outputs for different time points. A curve is fit to this data in a spreadsheet program, and the athlete's desired velocity compared to the curve (Fig. 31.2). If the desired performance velocity falls above the curve, the athlete is attempting an effort currently beyond their physiological ability. They may be advised to shorten the work interval to a duration the model indicates is possible for them. However, if the desired performance falls below

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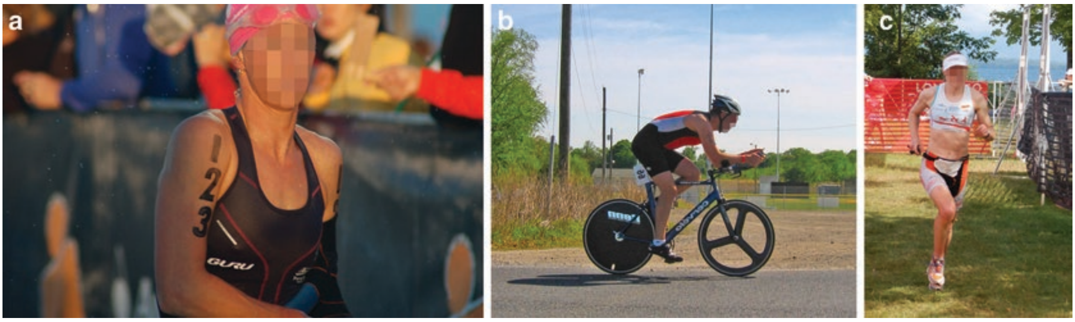

Fig. 31.1 Triathlon: swimming (**a**), cycling (**b**), and running (**c**)

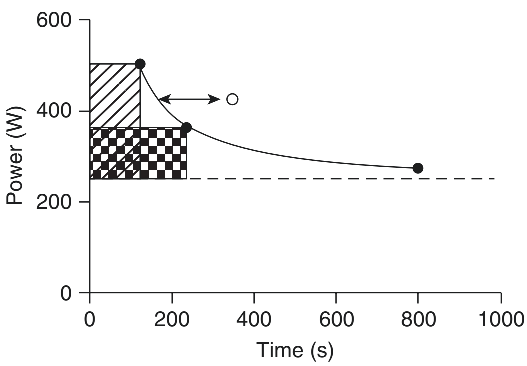

Fig. 31.2 Two-parameter critical power model. Filled circles indicate a cyclist's reported personal best power outputs for different time points. A regression is calculated (solid black line) using the equation P = (W′/t) + CP, where P is measured power output, CP is the critical power, W′ is the work capacity (in joules, hatched boxes) available above CP (dashed line), and *t* is time in seconds. Open circle indicates desired performance, which is currently impossible for the athlete. The athlete may be advised to attempt the performance at time point that would fall on the curve (arrows), and then build up the tolerable duration through interval training. Equation may be altered for running or swimming such that speed replaces power, distance in meters replaces W′, and critical speed replaces CP

Table 31.1 Sprint, Olympic, half-iron, or iron distance races

	Swim (m)	Bike (k)	Run (k)
Sprint	800	24	5
Olympic	1600	40	10
Half-iron	1900	90	21.1
Iron	3800	180	42.2

the curve, this may be indicative of a medical or physiological issue requiring investigation. A similar approach can be applied to intermittent exercise (i.e., a high intensity interval workout or race with surges and periods of recovery) using the methodology of Skiba et al. [6–8].

31.3 Overtraining

Human performance has been described mathematically as the difference between fitness and fatigue using impulse–response (IR) modeling [9, 10]. Initially, fatigue from training exceeds the fitness gained, leading to a decrement in performance. However, the effects of fatigue fade more quickly than those of fitness, eventually resulting in improved performance [9, 11–13] (Fig. 31.3). Over time, the effects of fitness and fatigue are additive. Therefore, the athlete's present performance capacity can be considered to be the residual sum of all of the positive and negative effects of all of the training they have ever done [9, 11–13]. Adaptations of these models have been used to examine the relationships between "acute" and "chronic" workloads undertaken by the athlete [14].

Overload is required to elicit new adaptation [15]. However, many IR models share a shortcoming more commonly attributed to athlete (and coach) psychology: the idea that additional training will always result in better performance [10]. This can lead to the stacking of workouts or races, without adequate time to absorb the training load imposed [16]. One particularly undesirable type of resultant exercise intolerance is the overtraining syndrome.

The overtraining syndrome was first described as "stillness," and was reported in the United

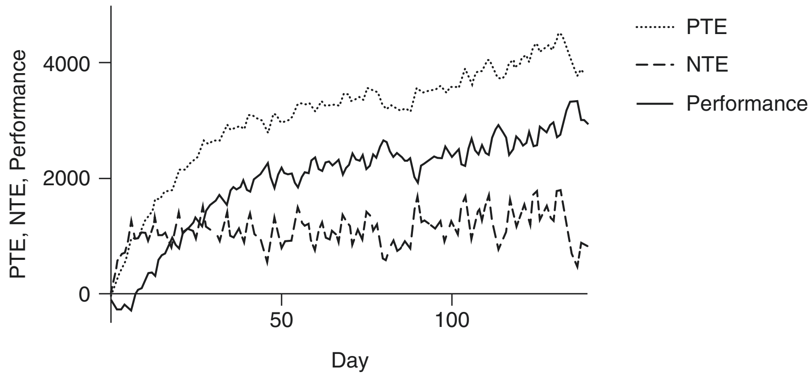

Fig. 31.3 Sample impulse–response model showing athlete development over time. Note that initially, NTE (i.e., fatigue) exceeds gains in PTE (i.e., fitness), leading to a decrement in performance. Eventually, fitness exceeds fatigue and performance begins to improve. Note the effect of tapering to race near the end of the plot: a substantial fall in NTE, relative preservation of PTE, and a peak in performance

States in the 1920s, while the term overtraining was preferred in the European literature [15]. Overtraining typically begins with "overreaching," which can occur after several days of hard training with insufficient recovery, resulting in muscle soreness and fatigue [17]. The transition to an overtrained state is somewhat gradual, and may be defined as a decrement in performance of longer than 2 weeks in the face of an increased training load [18].

Diagnosis of overtraining is complex, owing to the combination of psychological and physiological factors that are the result of training to excess. In addition to the aforementioned decline in physical performance, the signs and symptoms include insomnia, change in appetite, anxiety, and loss of motivation and/or concentration [19]. There appear to be parasympathetic or sympathetic dominated presentations. For example, signs and symptoms associated with endurance aerobic sports may include fatigue, depression, changes in resting heart rate or apathy, and may be linked to changes in parasympathetic activity [15, 20]. In contrast, symptoms associated with "anaerobic" or power sports are often attributed to sympathetic changes such as insomnia, irritability, agitation, and increased heart rate and blood pressure [15, 20].

Overtraining is almost certainly more than a functional disorder. However, perhaps in part due to the broad symptom constellation, it is difficult to pinpoint discrete pathophysiological mechanisms (for review, see Lehman et al. [20]). Overreaching has been associated with increased sex hormone binding globulin, and decreased insulin-like growth factor I and cortisol [21, 22]. Catecholamine response to submaximal exercise has been shown to be increased over baseline in overtraining [23, 24]. Somewhat more reliable markers appear to be a reduction in maximum lactate concentration and maximum heart rate during graded exercise testing [25]. As many of these tests are not typically done in the sports medical clinic setting, and indeed are not available outside of a dedicated physiology laboratory, diagnosis has remained largely clinical, and often one of exclusion. Treatment consists of long-term rest, sometimes for a year or more [26, 27]. Prevention is therefore crucial, and consists of appropriate long-term building and distribution of training, termed "periodization," discussed below.

31.4 Musculoskeletal Injury

Musculoskeletal triathlon injuries can be classified into acute injuries and overuse injuries. Upwards to 85% of all triathlon-associated injures are overuse [28, 29]. Of these injuries, most occur in the lower extremities. Running has been shown to cause the highest incidence of overuse injuries, followed by cycling and then swimming [28, 30–32].

31.5 Injury Sites and Mechanisms

The most common regions of injury are the knee, ankle/foot, lower leg, low back, and shoulder [30–33]. Among the individual triathlon segments, the most commonly reported injuries are the knee in running, low back in cycling, and shoulder in swimming [34]. The highest reported mechanisms of musculoskeletal injury include inflammatory pain (tendinitis), soft tissue strains, sprains, and fractures [31]. During the Kona Ironman in 1985, approximately 85% of the injuries reported were non-traumatic overuse injuries. Of the overuse injuries, muscle strain (with accompanying tendonitis) was the most prominent, followed by ligament sprain [28].

Lower extremity injuries have been shown to account for 36–90% of overuse injuries, whereas upper extremity injuries have accounted for 6.5–24.4% of injuries [34, 35]. In a study looking at the Hawaii Ironman in 1986, 91% of triathletes reported an overuse injury in previous year of training. Seventy-two percent of these triathletes reported multiple injuries during the competition year, many with more than one affected region [34]. If an athlete reported two injuries at a time, the most common regions were back and knee/thigh as well as back plus ankle/foot [29]. Achilles and knee injuries have been reportedly linked to running over cycling, whereas low back injuries have been attributed to cycling in Elite athletes [36].

Swimming tends to contribute to the least number of injuries with most swimming injuries localized to the shoulder [33, 34, 37]. A majority of shoulder injuries tend to be secondary to overuse [1]. The repetitive overhead movement involved in the swimming stroke predisposes the rotator cuff to impingement underneath the acromial arch, particularly in swimmers using old-style thumb-first entry on the catch, which is essentially an impingement maneuver. A subgroup analysis showed that athletes with overuse injuries to the shoulder had a higher weekly duration of swimming training [1]. They also associated the use of paddles during swimming training with shoulder injury, noting that this may lead to increased strain on rotator cuff due to increased resistance during the pull phase of the stroke.

Most acute traumatic injuries have been reported to be from cycling [28]. In a study by Egermann et al., fractures were reported by 11.9% of athletes with an incidence of 0.04 per 1000 h of exposures [38]. A majority (75.8%) of these fractures were linked to cycling, and 12.1% were associated with running. A 26-week prospective study by Anderson et al. demonstrated that of the 41 acute injuries, 60.9% were due to bike accidents [30]. Of the athletes who sustained a bike accident, one-third suffered multiple injuries. Although cycling has shown to account for only 12–29% of overuse injuries in triathletes [31, 33, 35], it may predispose triathletes to low back pain. Manninen et al. evaluated the incidence of low back pain in Japanese triathletes and found 32% of athletes reported low back pain in the previous year [39]. In these athletes with low back pain, 74% attributed it to cycling, 45% with weight training, and 43% with running. Mid-season training phase was the peak period for low back pain. They did not find any association of low back pain with cycling position, degree of trunk flexion, or use of aerobars. Athletes who experienced low back pain did tend to spend more time weekly in trunk flexor musculature training, suggesting they were performing the exercises incorrectly, or that they started to train this muscle group after developing low back pain.

In most studies, the highest number of injuries during triathlon competition and training involve running [28, 31–33, 40]. The knee and ankle have been shown to be the most common sites of overuse injuries in runners [41]. A recent 2-year prospective study showed that 66% of runners sustained at least 1 overuse injury during the trial period. The knee was most often injured at 28% followed by the foot at 21% of injuries [42].

31.6 Factors Contributing to Overuse Injury

Data about injury occurrence in relation to triathlon training habits vary. A previous study on runners showed that overuse injury was associated with higher weekly mileage and days ran per week in addition to participation in more races in the previous year [41]. Multiple triathlon-specific studies have shown no statistically significant evidence that injury is related to age, sex, training mileage, or competition level [28, 32, 33, 36, 40]. Others have also shown no relationship between stretching or pre-training warm-up and injuries [31]. A prospective study found that athletes who reported an injury in the previous year were more likely to sustain an injury during the training and competition period [32]. Other studies have also shown higher incidence of injuries in athletes who sustained previous injury, reporting that athletes who sustained a preseason injury were 2.5 times more likely to sustain an injury during the competition season [40]. Although most studies have found no training patterns identifiable to injury, one found that faster pace and use of higher gears during cycling showed a slightly higher incidence of foot and ankle injuries [28]. Although data was limited by small sample size, this study also noted that a majority of the triathletes came from a running background, therefore may have been more inexperienced in cycling and less knowledgeable about gear selection which could predispose them to injury. A study by Williams et al. found a relationship between weekly training distances in cycling and reported injuries [34]. Burns et al. reported that number of years of triathlon experience increased risk of preseason injury, although this might be secondary to summative training over multiple seasons causing overuse and deterioration of body regions [28].

31.7 Injury Incidence

Preseason injury incidence has been reported between 47% and 91% [29, 32]. Exposure to injury during training has ranged from 2.5 injuries per 1000 training hours to 5.4 injuries per 1000 hours, whereas acute competition injury rates have ranged between 1.0 injuries per 1000 hours and 17.4 per 1000 hours of competition [30, 32, 40]. An injury surveillance study of shorter triathlon races (fun, sprint, olympic) over the course of the 2006–2007 race season in Australia showed that the injury presentation rate was 20.1 per 1000 hours of competition or 2.3% of total race starts. Most injuries occurred during the run or cycle legs of the race. The injury rates across the three legs ranged from 3.24 per 1000 race starts in the swim to 10.61 per 1000 starts in the run leg [43]. There are limitations on previous data reported on injury incidence as results can be varied due to the risk of recall bias for retrospective studies, along with difficulty in self-reporting of injuries by the athletes. Large-scaled prospective studies on injury incidence will provide the community with more data on injuries.

Fact Box

- A majority of triathlon-related injuries are due to overuse.
- Running has been associated with highest injury rate.
- Lower extremity injuries are the most common.

31.8 Prevention

While it may be difficult to avoid traumatic injuries, overuse injuries and overtraining may be minimized through a logical approach to athlete development, even at high workloads [44]. The logical building and cycling of workloads over time is termed periodization. In fact, periodization has been part of the canon of sports training since ancient times. In Roman times, Galen himself discussed a logical progression from strength training, to speed training, to a combination [45]. The Greek scientist Philostratus discussed pre-Olympic preparations that were approximately 11 months in length: 10 months of general training, and 1 month of event-specific work. It is here that we see one of the earliest examples of the weekly distribution of training load: Philostratus advocated a pattern of workouts that moved from easy to difficult over a 4-day training window [45].

In modern times, periodization was first formalized by L.P. Matveyev, a Russian social scientist. However, Matveyev's work was not based upon scientific study. He simply distributed questionnaires to the Russian athletics team before the 1952 Helsinki Olympics, and published this as the basis for proper athlete preparation [45–47]. Although criticized today, his work demonstrated the now widely held belief that there should be a reciprocal relationship between the volume of training done and the intensity of that training [47, 48]. This said, Matveyev understood the process of sports training as one of pedagogy rather than physiology: in the early 1990s, he still warned against founding training theory in the "newly found" principles of biology [49]!

Modern reviews of periodization are available [45, 50–52], with step-by-step protocols available for triathlon in particular [48], and in-depth analyses for a variety of sports [53, 54]. In the general case, successful periodization protocols typically include blocks of general preparation, specific preparation, tapering, competition/peak, and a transition period between seasons [50]. Workloads and types of exercise are cycled over short, medium, and long-term time frames in an effort to deliver the athlete to competition in a particular physical condition for an event [52, 53]. Different protocols have been compared for triathlon in particular (i.e., traditional vs. reverse) [55]. One of the present authors (PS) offered a few basic guidelines, which were anecdotally successful with a large population of triathletes ranging from rank amateurs to Olympians and World Champions, and which are relatively easy to implement [48]:

1. **Training must go from general to specific:** Early or "base" training should not exclusively be *slow* training per se. Rather, the athlete should touch upon all aspects of fitness, including endurance, strength, and speed. As the season progresses, aspects most critical for success are emphasized, while those that are less so are taken down to "maintenance dose."
2. **Training must be specific to the sport(s):** Cross-training is in large part a convenient myth [56–58]. The principle of specificity dictates that the body adapts precisely to the demand imposed. Therefore, specific sport training must be maximized (i.e., actual swimming versus dryland) [58].
3. **Training must be specific to the physiological demands of the event:** Iron distance racing does not often require large variations in pace or surges in effort, whereas Olympic style racing does. Training should be developed accordingly.
4. **The training schedule must account for the positive and negative effects of all training:** Improvements in performance do not come from training, but rather from the supercompensation that comes during recovery [59]. Sport-specific planning of rest and recovery is therefore paramount. A variety of mathematical modeling techniques can help in this enterprise, and have been used to good effect by one of the present authors (PS; Fig. 31.3) [10, 14, 44, 48, 60].

31.9 Summary

Although all sporting enterprises require an element of planning, triathlon requires successful management of at least three training schedules, as well as the nutrition and recovery strategies necessary to maintain such. Above all, it is crucial to instill in the athlete a long-term vision of development, emphasizing patience and a respect for the time it takes to master so many skills. Triathlon is relatively unique in that professionals and amateurs complete the same course. It is important for most athletes to understand that they should not (and indeed, typically cannot) tolerate the type of training they may see the professionals executing. Even under the best of circumstances, injuries are common and triathletes often require a substantial amount of emotional support. This is due in part to the significant personal sacrifices required to manage a high volume, complex training schedule. Moreover, it should not be lost on the practitioner that the triathletes must register for many events a year or more in advance, at substantial cost (US$500+ entry fees are not uncommon), without hope of recouping the funds. This can create a paradoxical, dangerous motivation to hide or deny injury, which may lead to a greater chance of missed events or the premature end of a racing career.

References

1. Schorn D, Vogler T, Gosheger G, Schneider K, Klingebiel S, Rickert C, et al. Risk factors for acute injuries and overuse syndromes of the shoulder in amateur triathletes – a retrospective analysis. PLoS One. 2018;13(6):e0198168.
2. Etxebarria N, Mujika I, Pyne DB. Training and competition readiness in triathlon. Sports (Basel). 2019;7(5):101.
3. Mujika I. Olympic preparation of a world-class female triathlete. Int J Sports Physiol Perform. 2014;9(4):727–31.
4. Vleck V, Millet GP, Alves FB. The impact of triathlon training and racing on athletes' general health. Sports Med. 2014;44(12):1659–92.
5. Jones AM, Vanhatalo A, Burnley M, Morton RH, Poole DC. Critical power: implications for the determination of V O2 max and exercise tolerance. Med Sci Sports Exerc. 2010;42:1876–90.
6. Skiba PF, Chidnok W, Vanhatalo A, Jones AM. Modeling the expenditure and reconstitution of work capacity above critical power. Med Sci Sports Exerc. 2012;44(8):1526–32.
7. Skiba PF, Jackman S, Clarke DC, Vanhatalo A, Jones AM. Effect of work & recovery durations on W′ reconstitution during intermittent exercise. Med Sci Sports Exerc. 2013;46(7):1433–40.
8. Skiba PF, Clarke D, Vanhatalo A, Jones AM. Validation of a novel intermittent W' model for cycling using field data. Int J Sports Physiol Perform. 2014;9:900.
9. Morton RH, Fitz-Clarke JR, Banister EW. Modeling human performance in running. J Appl Physiol. 1990;69(3):1171–7.
10. Clarke DC, Skiba PF. Rationale and resources for teaching the mathematical modeling of athletic training and performance. Adv Physiol Educ. 2013;37(2):134–52.
11. Banister EW, Carter JB, Zarkadas PC. Training theory and taper: validation in triathlon athletes. Eur J Appl Physiol Occup Physiol. 1999;79(2):182–91.
12. Fitz-Clarke JR, Morton RH, Banister EW. Optimizing athletic performance by influence curves. J Appl Physiol. 1991;71(3):1151–8.
13. Banister EW. Modeling elite athletic performance. In: MacDougall JD, Wenger HA, Green HJ, editors. Physiological testing of the high-performance athlete. 2nd ed. Champaign, IL: Human Kinetics; 1991. p. 403–24.
14. Griffin A, Kenny IC, Comyns TM, Lyons M. The association between the acute:chronic workload ratio and injury and its application in team sports: a systematic review. Sports Med. 2020;50(3):561–80.
15. Purvis D, Gonsalves S, Deuster PA. Physiological and psychological fatigue in extreme conditions: overtraining and elite athletes. PM R. 2010;2(5):442–50.
16. Ingjer F. Maximal oxygen uptake as a predictor of performance ability in women and men elite cross-country skiers. Scand J Med Sci Sports. 1991;1(1):25–30.
17. Kuipers H. Training and overtraining: an introduction. Med Sci Sports Exerc. 1998;30(7):1137–9.
18. Xiao W, Chen P, Dong J. Effects of overtraining on skeletal muscle growth and gene expression. Int J Sports Med. 2012;33(10):846–53.
19. Meeusen RDM, Gleeson M, Rietjens G, Steinacker J, Urhausen A. Prevention, diagnosis and treatment of the overtraining syndrome: ECSS position statement 'Task Force'. Eur J Sport Sci. 2006;(6):1–14.
20. Lehmann M, Foster C, Dickhuth HH, Gastmann U. Autonomic imbalance hypothesis and overtraining syndrome. Med Sci Sports Exerc. 1998;30(7):1140–5.
21. Tanskanen MM, Kyrolainen H, Uusitalo AL, Huovinen J, Nissila J, Kinnunen H, et al. Serum sex hormone-binding globulin and cortisol concentrations are associated with overreaching during strenuous military training. Journal of strength and conditioning research / National Strength & Conditioning Association. 2011;25(3):787–97.
22. Hedelin R, Kentta G, Wiklund U, Bjerle P, Henriksson-Larsen K. Short-term overtraining: effects on performance, circulatory responses, and heart rate variability. Med Sci Sports Exerc. 2000;32(8):1480–4.
23. Lehmann M, Baumgartl P, Wiesenack C, Seidel A, Baumann H, Fischer S, et al. Training-overtraining: influence of a defined increase in training volume vs training intensity on performance, catecholamines and some metabolic parameters in experienced middle- and long-distance runners. Eur J Appl Physiol Occup Physiol. 1992;64(2):169–77.
24. Lehmann M, Gastmann U, Petersen KG, Bachl N, Seidel A, Khalaf AN, et al. Training-overtraining: performance, and hormone levels, after a defined increase in training volume versus intensity in experienced middle- and long-distance runners. Br J Sports Med. 1992;26(4):233–42.
25. Urhausen A, Gabriel HH, Weiler B, Kindermann W. Ergometric and psychological findings during overtraining: a long-term follow-up study in endurance athletes. Int J Sports Med. 1998;19(2):114–20.
26. Kuipers H, Keizer HA. Overtraining in elite athletes. Review and directions for the future. Sports Med. 1988;6(2):79–92.
27. Lehmann M, Foster C, Keul J. Overtraining in endurance athletes: a brief review. Med Sci Sports Exerc. 1993;25(7):854–62.
28. Massimino FAAM, O'Toole M, et al. Common triathlon injuries: special considerations for multisport training. Ann Sport Med. 1988;4:82–6.
29. O'Toole ML, Hiller WD, Smith RA, Sisk TD. Overuse injuries in ultraendurance triathletes. Am J Sports Med. 1989;17(4):514–8.

30. Andersen CA, Clarsen B, Johansen TV, Engebretsen L. High prevalence of overuse injury among iron-distance triathletes. Br J Sports Med. 2013;47(13):857–61.
31. Ireland MLML. Triathletes: biographic data, training, and injury patterns. Ann Sport Med. 1987;3:117–20.
32. Korkia PK, Tunstall-Pedoe DS, Maffulli N. An epidemiological investigation of training and injury patterns in British triathletes. Br J Sports Med. 1994;28(3):191–6.
33. Collins K, Wagner M, Peterson K, Storey M. Overuse injuries in triathletes. A study of the 1986 Seafair triathlon. Am J Sports Med. 1989;17(5):675–80.
34. Williams MHJ, Black R, et al. Injuries amongst competitive triathletes. New Zealand J Sports Med. 1988:2–6.
35. Migliorini S. An epidemiological study of overuse injuries in Italian national triathletes in the period 1987–1990. J Sports Traumatol Rel Res. 1991;(13):197–206.
36. Vleck VE, Garbutt G. Injury and training characteristics of male elite, development squad, and Club triathletes. Int J Sports Med. 1998;19(1):38–42.
37. Zwingenberger S, Valladares RD, Walther A, Beck H, Stiehler M, Kirschner S, et al. An epidemiological investigation of training and injury patterns in triathletes. J Sports Sci. 2014;32(6):583–90.
38. Egermann M, Brocai D, Lill CA, Schmitt H. Analysis of injuries in long-distance triathletes. Int J Sports Med. 2003;24(4):271–6.
39. Manninen JS, Kallinen M. Low back pain and other overuse injuries in a group of Japanese triathletes. Br J Sports Med. 1996;30(2):134–9.
40. Burns J, Keenan AM, Redmond AC. Factors associated with triathlon-related overuse injuries. J Orthop Sports Phys Ther. 2003;33(4):177–84.
41. Jacobs SJ, Berson BL. Injuries to runners: a study of entrants to a 10,000 meter race. Am J Sports Med. 1986;14(2):151–5.
42. Messier SP, Martin DF, Mihalko SL, Ip E, DeVita P, Cannon DW, et al. A 2-year prospective cohort study of overuse running injuries: the runners and injury longitudinal study (TRAILS). Am J Sports Med. 2018;46(9):2211–21.
43. Gosling CM, Forbes AB, McGivern J, Gabbe BJ. A profile of injuries in athletes seeking treatment during a triathlon race series. Am J Sports Med. 2010;38(5):1007–14.
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. Issurin VB. New horizons for the methodology and physiology of training periodization. Sports Med. 2010;40(3):189–206.
46. Bosch F, Klomp R. Running: biomechanics and exercise physiology applied in practice: Elsevier Churchill Livingstone; 2005. 424 p.
47. Verchoshankij, J. The end of "periodisation" of training in top-class sport. New Studies in Athletics. 1999;14(1):47–55.
48. Skiba PF. The Triathlete's guide to training with power. 1st ed. PhysFarm Training Systems: Neptune, NJ; 2008.
49. Matveyev L. About the construction of training. Teoriya I Praktika Fizichesckoi Kultury. 1991;12
50. Mujika I, Halson S, Burke LM, Balague G, Farrow D. An integrated, multifactorial approach to periodization for optimal performance in individual and team sports. Int J Sports Physiol Perform. 2018;13(5):538–61.
51. 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.
52. Issurin VB. Benefits and limitations of block Periodized training approaches to Athletes' preparation: a review. Sports Med. 2016;46(3):329–38.
53. Hellard P, Avalos-Fernandes M, Lefort G, Pla R, Mujika I, Toussaint JF, et al. Elite Swimmers' training patterns in the 25 weeks prior to their Season's best performances: insights into periodization from a 20-years cohort. Front Physiol. 2019;10:363.
54. Solli GS, Tonnessen E, Sandbakk O. Block vs. traditional periodization of HIT: two different paths to success for the World's best cross-country skier. Front Physiol. 2019;10:375.
55. Clemente-Suarez VJ, Ramos-Campo DJ. Effectiveness of reverse vs. traditional linear training periodization in triathlon. Int J Environ Res Public Health. 2019;16(15):2807.
56. Coyle EF, Feltner ME, Kautz SA, Hamilton MT, Montain SJ, Baylor AM, et al. Physiological and biomechanical factors associated with elite endurance cycling performance. Med Sci Sports Exerc. 1991;23(1):93–107.
57. Magel JR, Foglia GF, McArdle WD, Gutin B, Pechar GS, Katch FI. Specificity of swim training on maximum oxygen uptake. J Appl Physiol. 1975;38(1):151–5.
58. Tanaka H, Costill DL, Thomas R, Fink WJ, Widrick JJ. Dry-land resistance training for competitive swimming. Med Sci Sports Exerc. 1993;25(8):952–9.
59. Viru A. Molecular cellular mechanisms of training effects. J Sports Med Phys Fitness. 1994;34(4):309–22.
60. Bowen L, Gross AS, Gimpel M, Li FX. Accumulated workloads and the acute: chronic workload ratio relate to injury risk in elite youth football players. Br J Sports Med. 2017;51(5):452–9.

32 Weightlifting

Johnel Mayberry and Terry L. Nicola

32.1 Overview

Weight training is a form of strength training, also called resistance training, where the athlete utilizes weights to work a specific group of muscles around a joint or multiple joints. The use of weighted resistance is an increasingly popular conditioning technique, competitive sport, and recreational activity.

As a means to measure strength and power, weightlifting was practiced both by ancient Egyptian and Greek societies. It developed as an international sport primarily in the nineteenth century and is one of the few sports featured at the 1896 Athens games [1].

At least 45 million Americans participate in regular resistance training [2, 3]. The US Consumer Product Safety Commission studied a cohort of Emergency Departments and identified 25,335 weight training injuries, which they extrapolated to an average 970,801 total emergency department visits every year [4]. This number likely underestimates the total number of injuries as surveys have shown that 54% of strongmen athletes rely on self-treatment, and only a modest percentage of these experienced weightlifters seek treatment from medical professionals [5].

Heavy lifting is a well-known injury risk in the general population, and one can assume that risk of injury is high in weight training athletes as they require coordination and balance while attempting to lift maximum loads [6]. However, when compared to other sports, the injury incidence in weight training sports is near that of non-contact sports and lower than contact sports. Aasa et al. [6] found the injury incidence in weight lifters to be 2.4–3.3 injuries/1000 h of training and 1.0–4.4 injuries/1000 h of training in powerlifters. Comparatively, Jacobsson et al. [7] showed an incidence of 3.56 injuries/1000 h of training in track and field athletes while Westin et al. [8] found an incidence of 1.7 injuries/1000 h of training in alpine skiing. In American football, the injury incidence is 9.6/1000 h of training.

Weight training uses the force of gravity acting upon a given resistance, including the exerciser's own body weight or specialized forms of equipment such as barbells, dumbbells, and resistance training machines to target specific muscle groups and joint actions. While many people who regularly exercise and perform weight training along with cardiovascular or flexibility exercise for overall health benefit, several athlete groups also compete in weight training as their primary

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Fig. 32.1 Finishing position of the clean and jerk

form of training and/or competition. These sports include weightlifting, powerlifting, bodybuilding, strongman, and Highland Games [9].

Competitive weightlifting requires the lifter to lift maximal loads for one repetition. There are two events in weightlifting competitions, the clean and jerk (Fig. 32.1) and the snatch (Fig. 32.2). As these exercises require the barbell to be lifted explosively from the floor to an overhead position, they may produce the greatest power outputs of any human activity [10].

Powerlifting is similar to weightlifting, with lifters attempting to lift the maximum loads for one repetition. However, in powerlifting competitions, there are three events, the squat, bench press, and deadlift (Figs. 32.3, 32.4, and 32.5).

The Scottish Highland Games and strongman competitions—the most similar form of weight training competition to that performed in ancient or medieval times, found in the sports traditionally performed as tests of "manhood" in many countries [9]. These tests of "manhood" typically had farming and/or military applications and

Fig. 32.2 Showing the starting position and finishing position of the snatch, respectively

Fig. 32.3 Depiction of the squat

Fig. 32.4 Depiction of the bench press

Fig. 32.5 Depiction of the deadlift

involved the lifting or throwing of a variety of natural and man-made objects that have been available for hundreds or thousands of years. Specifically, strongman events utilize a variety of heavy implements such as stones for lifts and carries, tires for flipping, logs and stones for overhead pressing, and trucks or sleds for pulling [9]. While some of these strongman events are similar to weightlifting and powerlifting, with the athletes attempting to lift the heaviest load for one repetition, many of the events are timed, with the winner being the fastest athlete to complete the task. The Highland Games events are further ancient/medieval examples of tests of "manhood" and involve a range of heavy throwing events such as the caber, stone put, hammer throw, or sheaf toss, as well as weight for height and distance. The variety of weight-for-distance events are simply much heavier versions of many of the throwing events currently seen in regular track and field competitions [9].

Bodybuilding differs from the other weight-training sports in that it is not judged on the weight lifted or the time taken to complete an event, but rather on the esthetics of the athlete.

These weight-training sports have annual world championship events for male and female athletes, with some of these sports also offering various bodyweight or age (junior, open, and masters) classes. However, weightlifting is the only one of these sports currently included in the Olympic Games, although powerlifting (bench press only) is also a part of the Paralympics [9].

32.2 Weight Training-Related Injuries

Improper training and poor technique can often lead to musculoskeletal injury and time away from training. Risk factors for injury include technical errors, fatigue, overloading, and dropping the weights. The shoulder, lower back, knee, and elbow are the most commonly injured anatomical locations across weight-training sports.

Sprains and strains are the most common type of injury and account for 46.1% of all resistance training injuries [11]. Additionally, most injuries are acute in nature (60–75%) but may vary in type and severity [4, 1]. Chronic weightlifting injuries are associated with overuse and make up the remaining 30% of injuries. These chronic-type injuries are more common in aging athletes who suffer from increased rates of tendinopathy, tendon rupture, and degenerative joint disease [8].

32.3 Risk Factors

Keogh and Windwood [9] looked at risk factors that may affect the injury incidence and pattern in weight-training athletes. Intrinsic factors included sex, competitive standard, age, and weight class. In general, intrinsic factors had relatively little effect on the injury epidemiology of the weight-training sports with the following exceptions. Female lifters had significantly lower overall injury rates, 1.3 vs 2.1 injuries per lifter per year. Females also had a significantly lower rate of chest and thigh injuries but had a considerably higher rates of knee injuries which appears consistent with some findings for other sports and activities. Elite lifters had a substantially lower rate of injuries, both acute and chronic than non-elite lifters. Elite lifters also had significantly fewer chest and shoulder injuries but significantly more thigh injuries than non-elite lifters. Competition injuries were reported among younger (≤30 years old) strongman athletes. This suggests that experience plays a key role in injury prevention. Higher rate of injury was also found in heavyweight (>105 kg) than lightweight strongman competitors (≤105 kg). This can be attributed to the heavier loads that athletes in a higher weight class will attempt to lift.

Extrinsic factors such as coaching, rules of the sport and training environment could be related to injury in weight-training sports. However, no experimental studies have been conducted to examine this possibility.

Wang et al. [12] found that weight lifters attributed 60% of their injuries to fatigue, while 31% were associated with technical errors and 21% with excessive overload. Bodybuilders studied by Xiaojun and Taotao [13] felt that 21% of their injuries were caused by fatigue and poor recover, 18% by training with overly heavy loads, and 14% attributed to insufficient warm-up. Bodybuilders in Eberhart et al. [14] study felt injuries were a result of improper warm-up, 42%, too vigorous exercise, 35%, and lack of spotter during training, 25%.

32.4 Shoulder Injuries

Shoulder injuries account for the highest percentage of weight-training sports injuries in a 2016 meta-analysis [9]. This may reflect the frequent use of heavy loads and exercises such as the bench press and overhead press, as seen in the snatch and clean and jerk.

Golshani et al. [15] found that 36% of injuries in weightlifting population occur at the shoulder complex. The susceptibility of the shoulder complex to weight-training injuries is partly due to the high compressive loads these exercises apply to a traditionally non-weight-bearing joint [16]. The risk of shoulder injury is increased when the shoulder joint is abducted and externally rotated, which is a position that can be seen in competitive weightlifters during the snatch and clean and jerk [17].

Studies have found more shoulder injuries in competitive powerlifters in comparison to competitive weightlifters. The bench press may predispose powerlifters to rotator cuff injuries because of the unfavorable position of the rotator cuff during the lift and the pursuit of higher one-repetition maximum lifts. Furthermore, there is a rapid alternation between eccentric and concentric muscle contraction [18].

Weight-training athletes usually focus on exercises that emphasize large muscle groups for strength, hypertrophy, and esthetics. They often neglect smaller muscles, like the rotator cuff muscles responsible for upper extremity joint stabilization. The combination of repetitive loading, unfavorable positioning, and biased exercise selection creates joint and muscle imbalances that increase the athlete's risk of labral tears, labrocapsular junction dysfunction, and shoulder instability, which can precipitate rotator cuff disease.

While most studies only classified injuries based on anatomical location or types of injuries without a specific diagnosis, Van der Wall et al. [17] studied shoulder injuries in weight lifters by using scintigraphy. Soft tissue damage of the rotator cuff, the biceps tendon, and the capsular and ligamentous insertion were shown.

32.4.1 Rotator Cuff Tendonosis and/or Tears (Partial or Full Thickness)

Rotator cuff injuries may present with weakness and deep aching pain over the deltoid muscle especially with lifts such as the overhead/military press, seated shoulder press, or bench press. As mentioned previously, these lifts predispose lifters to rotator cuff injuries because of the unfavorable position of the rotator cuff. Other lifts can also place the rotator cuff in an unfavorable position, including the popular barbell squat, especially the low bar squat (Fig. 32.6) in which the shoulders are maximally externally rotated while holding the barbell in place.

Treatment for rotator cuff disease initially involves rest and avoiding activities that will exacerbate symptoms. Rehabilitation can be initiated with a focus on strengthening the core muscles of the shoulder girdle, including the scapular stabilizers, levator scapulae, rhomboids, and trapezius muscles. Strengthening can then progress to the rotator cuff muscles [19–20]. The lifter's technique should also be closely evaluated, such as ensuring scapular retraction during the bench press. This provides a stable base on the bench for the lifter and also prevents the acromion from moving inferiorly on the rotator cuff during the lift. The athlete should avoid incline bench presses while injured as there is more stress placed on the shoulder girdle compared to the traditional bench press. Shoulder flys, performed in abduction and forward flexion can also further injure the rotator cuff. Typically this exercise is performed with the forearm in pronation and the shoulder in internal rotation which can lead to impingement. If shoulder flys are performed, they should be done with the shoulder externally rotated through a full over-head arc. Symptoms may persist and worsen if the athlete is not counseled on activity modification to avoid such technique errors.

Fig. 32.6 Depicts the bar position during the low bar squat

32.4.2 Distal Clavicle Osteolysis (DCO)

Distal Clavicle Osteolysis (DCO) is an uncommon injury that is strongly associated with weight training. Cahill et al. [21] reported on 45 male athletes with DCO, and 44 of those 45 patients were weightlifters. Roedl et al. [22] found that 6.5% of young people, aged 14–19, who presented with shoulder pain, were found to have imaging findings consistent with DCO. Of the patients, 63% endorsed weight training as part of their physical activity. DCO occurs secondary to repetitive stress leading to disruption of articular cartilage, subchondral cyst formation, and underlying osteoclastic activity. Patients will typically present with pain, described as dull or achy, over the acromioclavicular joint (ACJ). Anterior to posterior X-rays with 10–15° cephalad should be obtained to evaluate the ACJ. Imaging findings are normal early in the disease course but with time progress to loss of subchondral bone with a widening of the ACJ. The acromion is spared of lytic changes [21]. Athletes typically respond well to conservative treatment, including activity modification. However, weight-training athletes may opt to for distal clavicle resection which has good-to-excellent results.

32.4.3 Biceps Tendinopathy/Rupture

The proximal biceps tendon is placed under the similar stresses as the rotator cuff muscles during lifts, such as the bench press, which can lead to degeneration, tear, and/or rupture. Squats and overhead shoulder/military presses can also strain the proximal biceps tendon [23]. The repetitive motion during these lifts can lead to degeneration of the long head of the biceps as it moves through the bicipital groove and the subacromial space [15]. Ninety percent of biceps tendon ruptures occur proximally in the long head, often within the bicipital groove [24]. With bicipital tendinopathy, the athlete will present with anterior shoulder pain and tenderness over the groove. In cases of a rupture, the athlete may present with a "Popeye sign", bulging of the biceps muscle. Proximal biceps tendon ruptures can be acute or chronic. In acute cases, the athlete may perceive a pop followed by bruising. In older patients, associated rotator cuff disease is typically present. Ruptures may result in 20% loss of forearm supination strength and 8% loss in elbow flexion strength with little functional compromise [20]. Proximal biceps tendinopathy and ruptures in those over 40 years are typically treated conservatively, with a similar therapy program as seen in rotator cuff disease. Those younger than 40 years old with a proximal biceps tendon rupture may opt for surgical intervention, with options including a tenotomy or tenodesis. Associated lesions of the rotator cuff and labral pathologies must also be addressed [19–20].

The distal biceps tendon is most at risk for acute rupture from the radial tubercle [25] during eccentric contraction with a weight of >68 kg [26]. This type of motion occurs when performing biceps curls or when lifting weights from the floor in lifts such as the deadlift and clean and jerk [25]. Treatment of a distal biceps brachii tendon rupture consists of surgical repair [12]. When considering nonoperative treatment, the risk of residual pain, muscle spasm, and the cosmetic appearance of the arm must be discussed with the patient. In younger patients, most surgeons advocate for operative treatment of distal biceps tendon ruptures using either one or two-incision technique. Chronic ruptures may require allograft reconstruction of the tendon [27].

32.4.4 Pectoralis Muscle Strains

Pectoralis muscular strains are common injuries also seen in weight-training athletes. This injury is more likely to occur in powerlifters as the bench press is one of their competitive events. It may also be seen commonly in bodybuilders as their goal is to cause micro-trauma to increase the size and definition of their pectoralis muscles. The incidence of pectoral injuries has increased over the last two to three decades because of the rise in recreational weightlifting and competitive events participation [33]. Pectoralis muscle injuries may go un-noticed as these athletes expect some degree of pain in this region following chest workouts and mistake injuries for delayed onset muscle soreness. De Castro et al. [34] reported that 47–70% of reported pectoralis major injuries were a result of bench pressing. According to Kakwani et al. [35], pectoralis tendon rupture is a relatively rare injury, with 50% of cases occurring during weight training. This injury may be secondary to the shoulder being forced into abduction at the time of maximal eccentric contraction of the pectoralis major during the pressing motion. Fatigue, improper warm-up, and/or overloading may also contribute to injury.

Partial tears or strains are treated conservatively, initially with protection, rest, ice, compression, elevation (PRICE) followed by a strengthening program over 4–6 weeks. In general, surgical treatment is recommended for all complete ruptures of the distal insertion and myotendinous junction. Some authors suggest that results are improved when surgery is performed during the acute phase [33].

32.4.5 Glenohumeral Capsular Injury

Repetitive loading of the shoulder complex can also cause capsular strain leading to instability and persistent pain. Proper technique is the mainstay for the avoidance of "progressive" capsular injury. Athletes with anterior shoulder instability or labral injuries require a handoff from/to a spotter for un-racking and re-racking [36]. They should also avoid the incline bench press and any exercises that place the arm in the "high five" position [37]. Athletes with posterior shoulder instability should increase their grip width over two times the biacromial width; this maximizes the glenohumeral articulating surface area [38]. The bench press places heavy stress on posterior stabilizers, and attempts at a one-rep max should be avoided because of the significant strain on shoulder capsule and labrum [39]. The overhead shoulder press should never be performed with the bar being lowered behind the neck. The physiologic effects of this exercise can be replaced by specific posterior deltoid muscle exercises such as rear deltoid reverse flies, seated rows, or dumbbell rows. The shoulder press can be modified by starting at ear level and ending in the overhead position or replaced with isometric exercises. Furthermore, utilization of machines, such as the seated shoulder press or smith machine, will limit horizontal (AP) translation to protect the anterior glenohumeral ligament [15]. Also, when performing latissimus dorsi pulldowns in front of the neck, the weightlifter should recline to approximately thirty degrees to protect the shoulder [29].

32.4.6 Technique Modifications

A handgrip of 1.5 times the shoulder width during barbell exercises aligns the clavicle, pecs, and biceps brachii into a mechanically advantageous position to maximize shoulder flexion. (Fig. 32.7) It allows the long head of the biceps to move smoothly through the bicipital groove [28]. Also, an overhand grip moves the biceps tendon out of the subacromial space, by internally rotating the humerus, which limits damage to the proximal biceps tendon from impingement. Fees et al. [29] looked at additional modifications to protect the shoulder complex during weight training for patients with rotator cuff tendinitis, impingement syndrome, acromioclavicular problems, shoulder instability, and glenoid labral/biceps anchor pathology. A narrow bench press grip decreases shoulder torque and minimizes shoulder adduction and extension [30]. A higher touchpoint on the chest above the xiphoid process also decreases shoulder torque [31]. Additionally, alternating

Fig. 32.7 Depicting the military press/overhead shoulder press with hand grip 1.5 times shoulder width

grip patterns can offload specific entities: a pronated grip places the supraspinatus under the acromion, [18] while a supinated grip places the long head of the biceps under the acromion [32].

32.5 Low Back Injuries

Low back injuries has been reported in 30–50% of weightlifters [40]. Brown and Kimball [41] also found that low back pain accounts for 50% of injuries in adolescent powerlifting competitors. Muscle strains accounted for 61% of these back injuries.

Lumbar strain to the intrinsic lumbar musculature, tendons, and/or ligaments is believed to be the result of overuse, acceleration-deceleration, trauma, and/or repetitive loading which is common in many lifts such as the barbell squat and deadlift. Treatment for lumbar strains generally includes activity modification but avoidance of bed rest. Therapy should focus on pelvic mobility, including hamstring flexibility, and lumbar core stabilization [19].

Another common low back-related injury in weight training athletes is acute lumbar radiculopathy. It typically occurs when the contents of the nucleus pulposus are extruded through the annulus fibrosis into the spinal canal leading to irritation of the nerve root. The irritation of the nerve root can be secondary to mechanical compression and /or chemical irritation by the disc nucleus material. The L5-S1 disc herniation is the most common, with the L4-L5 being second most common [19–20]. During lifts such as the deadlift, there can compression forces averaging >17,000 N in elite powerlifters [10]. Athletes will typically present with acute low back pain and radicular leg symptoms. Symptoms can be thought to be secondary to delayed onset muscle soreness in some cases, as pain symptoms may occur following lifts that focus on lower extremity or lumbar strength training. Sensory or motor symptoms in the relative dermatomes or myotomes may accompany pain symptoms. Similar to a lumbar strain, treatment initially includes relative rest with avoidance of bed rest. Patient should be educated on the natural history of disc herniations. A majority will resolve in 3–6 months as the herniated disc material is resorbed. Two-year outcomes for surgical v. nonoperative interventions are the same [19]. Physical therapy can be prescribed with gentle spine mobility, primarily in extension. A transforaminal epidural corticosteroid injection can be helpful for radicular leg pain symptoms if symptoms are debilitating or occur at rest. Surgery can be considered if the athletes fail conservative management or have worsening neurological symptoms [19–20].

32.5.1 Technique Modifications

Maintaing a neutral lumbar spine is key to advoiding injuries of the low back. Athletes should engage their core and pivot at the hips when performing lifts such as the squat and deadlift. Athletes often wear weight belts (Fig. 32.8) during heavy lifts to support the lumbar spine in hopes of injury prevention. It is believed that the weight belt compresses the abdominal contents and increase intra-abdominal pressure, which in tum reduces the compressive forces acting on the lumbar spine: this relief of the spine and spinal

Fig. 32.8 Front and back of a weight belt

muscles has been verified during the squat lift [42]. The weight belt may have disadvantages: if used invariably, it may prevent the development of maximal strength in the abdominal and back muscles; it is also not practical during some lifts. The high intra-abdominal pressure may also impede blood return to the heart. It has suggested the use of weight belts during heavy [>80% of 1 repetition maximum (1 RM)] lifts in exercises which its use is practical and loosening the belt between sets [42].

Fig. 32.9 Depicts bar position in the high bar squat

32.6 Knee and Thigh Injuries

Knee injuries are found to be more common in competitive weightlifters [6] compared to other resistance training athlete. Koegh and Winwood [9] suggest the reason for this might be that the weightlifters perform high bar squats. (Fig. 32.9) During high bar squats the barbell rests on the upper parts of the trapezius muscle with a vertical back position, which results in higher torque around the knee joints during the lift.

High-bar and front squats (Fig. 32.10) also produce a larger knee resistance moment arm and smaller hip/lower back resistance moment arm than the low bar squat. Such differences in resistance moment arms suggest that these lifts may require greater knee extensor torque and produce higher mean compressive patellofemoral forces than the low-bar squat [9].

Fig. 32.10 Depicts the bar position in the front squat

32.6.1 Quadriceps

The quadriceps is a key muscle group in weight-training athletes for strength and esthetics. In weightlifters and powerlifters, quadriceps strengthening programs are a focus when seeking to increase their 1RM in lifts such as the snatch and barbell squat.

Weight training athletes commonly incur quadriceps injuries. These injuries likely result from the amount of knee extensor torque required during competitive lifts, coupled with the importance of quadriceps strengthening. Authors agree that quadriceps injuries occur with forceful, repetitive quadriceps concentric and eccentric contraction, which is seen during exercises such as weighted knee extensions, lunges, and barbell squats [5, 9, 11].

Quadriceps strains may also go under-diagnosed as weight-training athletes expect some degree of pain following lower extremity training sessions. Strains are more common in the rectus femoris compared to the other quadriceps muscles, as it passes over two joints. Athletes may present with local pain, tenderness, pain with stretching, and/or resisted knee extension. It is important to check the athlete's strength on onset as this will guide return to activity. Initial treatment includes PRICE. Grade II & III strains should be immobilized, and muscle activation should be initiated after 3–5 days [19–20]. A therapy program should focus on restoring range of motion and strength, beginning with isometrics with progression to eccentrics.

32.6.2 Collateral Ligament Sprains

Reeves et al. [3] postulated that medial and lateral collateral ligament sprains may occur during squats, leg presses, and lunges with high loads and/or improper lower extremity placement. Complete disruption of the collateral ligaments is rare in weight-training athletes. Injury to the MCL occurs when there is valgus stress on a partially flexed knee [20]. Treatment includes bracing with rehabilitation program focused on correcting underlying biomechanical defects that lead to injuries. Injuries to the LCL are less common in comparison to MCL injuries. Isolated injuries as also rare as a higher force is required to injure the LCL which would lead to injuries of nearby structures of the posterolateral corner [19].

32.6.3 Quadriceps and Patellar Tendon Injuries

Quadriceps tendons injuries are discussed more in the current literature as an injury site in powerlifting and weightlifting due to the deep loaded flexed knee required in the clean and jerk, snatch, and squat. Due to the repetitive nature of bodybuilding, patellar tendonitis of the knee can be assumed to be a common occurrence as well. Anabolic steroid use has been associated with ruptures of the patellar tendon [43].

32.6.4 Knee Osteoarthritis (OA)

It has also been suggested that the squat could increase the risk of knee OA. In powerlifting and weightlifting, deep squats are included in daily training and competition which exposes the knee to high shear forces. The prevalence of osteoarthritis in the patellofemoral or tibiofemoral joint in former weightlifters was found to be as high as 31% [44].

32.6.5 Meniscal Injuries

Meniscal injuries are uncommon in weight training athletes, but medial meniscus tears have been associated with knee flexion exercises, such as hamstring curls and deadlifts [45].

32.6.6 Technical Modifications

Technique is vital for injury prevention, especially during compound exercises such as the squat and deadlift. Evidence-based guidelines exist for the squat, and these include foot stance of shoulder width or wider, maintaining a flat foot on the ground and toes pointing outward, no more than 10 degrees [46–49]. Also, the knee should track over the toes without knee displacement medially or laterally. Improving ankle mobility and strength is recommended along with heel lifts or weightlifting shoes (Fig. 32.11) to decrease varus or valgus movement during the squat [48].

Fig. 32.11 Depicts typical weightlifting shoes with raised stiff heel

32.7 Elbow Injuries

32.7.1 Medial/lateral Epicondylitis

Elbow injuries occurred equally in competitive powerlifters and Olympic weightlifters, accounting for 11% of all injuries in these athletes [9, 47]. Due to the repetitive movement seen in weight-training athletes, it can be assumed that medial and/or lateral epicondylosis accounted for a large majority of these injuries. Compared to throwing sports, where ligamentous injuries to the elbow are common, there is little-to-no varus or valgus force on the elbow during weight training exercises.

Common exercises in improving forearm esthetics and grip strength are weighted wrist extension and flexion exercises. Other exercises including the bench press, pull-ups, and biceps curls also stress the medial epicondyle. Some lifters also perform biceps curls with a pronated grip in an effort to work the forearm extensor muscles and the biceps simultaneously.

Athletes may present with medial or lateral elbow pain that radiates down into the proximal forearm. There will be tenderness to palpation over the common flexor tendon or common extensor tendon. There will also be pain with passive wrist extension or resisted wrist flexion in medial epicondylosis. There is typically pain with passive wrist flexion and/or resisted wrist extension in lateral epicondylosis. Initial treatment typically involves rest, ice, and/or anti-inflammatories, topical or oral. Athletes can use counterforce proximal forearm bracing with activities, including weight training. Lifters should be encouraged to alternate between pronated/supinated grip to offload the affected area. Lifting mechanics should be evaluated, and an overview of the athletes training schedule should be performed if there is a concern for overtraining. Physical therapy can be initiated with a focus on correcting underlying flexibility deficits. If symptoms do not resolve with conservative treatments, percutaneous tenotomy with or without platelet-rich plasma can be considered [19–20].

32.8 Paralympic Weightlifting Injuries

Weightlifting was added to the Paralympics games in 1964. The sport originally catered to male athletes with spinal cord injuries. Powerlifting replaced weightlifting in 1984 and now include other impairment groups. There are currently 20 divisions, 10 males and 10 females, depending on weight class who compete in the bench press. Athletes are given three attempts with the winning athlete lifting the largest amount of weight [50]. There is a scarcity of literature regarding injuries in athletes participating in Paralympic powerlifting. Willick et al. [51] performed a prospective study during the 2012 Paralympic Games and found powerlifting to have the second highest injury incidence, following football. There were 38 injuries in 38 different athletes, the overall injury incidence rate was 33.3 injuries/1000 athlete days. There was no significant difference between male and female competitors. Athletes in weight class above 75 kg suffered more injuries compared to those in weight classed below 75 kg. This may be attributed to these participants lifting heavier loads. Like powerlifters and weightlifters, the most common area of injury was the shoulder/clavicle which accounted for 31.6% of the injuries. The second most commonly affected areas were the chest and elbow at 13.2% each. The upper arm and the foot/ankle injuries accounted for, 7.9% each while 5.3% of injuries were to the cervical spine. One athlete's injuries were classified as multiple body locations which accounted for 2.6% of injuries. The remaining 18.4% of injuries were undefined. In comparison to able-bodied weight training athletes where most injuries tend to be acute, 86.8% of the injuries reported were chronic overuse injuries or acute on chronic injuries. This is likely secondary to these athletes using their upper extremities for mobility with the use of assistive devices such as wheelchairs and walkers. Due to the importance of upper extremity health for mobility and activities of daily livings in this population of athletes, injury recognition, treatment, and prevention are crucial.

32.9 In Summary

Common injuries across the weight training sports occur in the shoulder, low back, knee, and elbow. The incidence of these injuries does differ between sports. Weightlifters had more knee injuries which can be attributed to the deep squat that occurs with the snatch and clean and jerk. Shoulder injuries were more common in powerlifters. This finding can be attributed to stress caused by the bench press on the shoulder complex [6].

In comparison to bodybuilders, weightlifters, powerlifters, and strongman have muscle stains as their most common injury type. This can be attributed to these competitive lifters lifting weight close to their 1RM. In contrast, bodybuilders who typically lift at a lower percentage of 1RM experience more tendon injuries [9].

Fatigue has previously been implicated as an inciting factor to sporting injuries. Therefore, lifters should perform the most demanding, challenging, and high-risk exercise during the initial part of the training session.

Warm-up and stretching are advocated. When an athlete is going to attempt to lift maximal or near-maximal amounts of weight, they should start with less weight and progress to those they will use for their near max or maximal lift. Stretching at the end of the lifting session is also recommended [51, 52].

Proper technique is essential when performing weight training exercises. Good technique should also be used when moving weights, racking and un-racking weights. Proper technique requires the use of a good grip; a stable lifting position, with the weight kept close to the body. The athletes should use their legs, not the back, to do the lifting [53].

Athletes should consider a spotter for some free weight exercises, such as the bench press, to avoid injuries. During the lifting of large amounts of free weights, athletes should also consider wearing a weight belt when feasible. In general, it appears that machines are safer than free weights, especially for inexperienced users [53].

To avoid the syncope or near-syncope that may result from Valsalva while lifting, the athlete should breathe out during the exertion phase and inhale during the relaxation phase [51].

32.10 Guidelines for Strength Training

The American College of Sports Medicine (ACSM) guidelines on strength training include training 2–3 times a week for healthy adults. Older adults or those that have been sedentary should start at two times a week and choose light-intensity exercises. The recommended time between sessions is 48 h. The ACSM also suggests performing 8–12 repetitions of 8–10 multijoint exercises that stress major muscle groups. Exercises should be performed with good form with a controlled motion, suggesting a 2-s count for the concentric and eccentric portion of the exercise [54].

References

1. Lavallee ME, Balam T. An overview of strength training injuries: acute and chronic. Curr Sports Med Rep. 2010;9:307–13.
2. CDC. Trends in strength training-United States, 1998-2004. MMWR Morb Mortal Wkly Rep. 2006;55:769–72.
3. Reeves RK, Laskowski ER. Smith J. onald K. Reeves, Edward R. Laskowski & jay Smith (1998) weight training injuries. Phys Sportsmed. 1998;26(2):67–96.
4. Risser WL, Risser JM, Preston D. Weight-training injuries in adolescents. Am J Dis Child. 1990;144:1015–7.
5. Winwood PW, Hume PA, Cronin JB, Keogh JW. Retrospective injury epidemiology of strongman athletes. J Strength Cond Res. 2014;28:28–42.
6. Aasa U, Svartholm I, Andersson F, Berglund L. Injuries in among weightlifters and powerlifters: a systemic review. British J Sports Med. 2017;51:211–20.
7. Jacobsson J, Timpka T, Kowalski J, et al. Injury patterns in Swedish elite athletics: annual incidence, injury types and risk factors. Br J Sports Med. 2013;47:941–52.
8. Westin M, Alricssson M, Werner S. Injury profile of competitive alpine skiers: a five-year cohort study. Knee Surg Sports Traumatol Arthrosc. 2012;20:1175–81.
9. Keogh JWL, Winwood PW. The epidemiology of injuries across the weight-training sports. Sports Med. 2017;47:479–501.
10. Cholewicki J, McGill SM, Norma RW. Lumbar spine loads during the lifting of extremely heavy weights. Med Sci Sports Exerc. 1991;23(10):1170-1186.
11. Kerr ZY, Collins CL, Comstock RD. Epidemiology of weight training-related injuries presenting to United

States emergency departments, 1990 to 2007. Am J Sports Med. 2010;38:765–77.
12. Wang D, Joshi NB, Petrigliano FA. Trends associated with distal biceps tendon repair in the United States, 2007–2011. J Shoulder Elb Surg. 2016;25:676–80.
13. Xiaojun Z, Taotao Li, Sport injury law and preventing method of Chinese elite bodybuilding players. J Shenyang Inst Phys Educ 2008:2794075-2794077
14. Eberhardt A, Dzbanski P, Fabirkiewicz K, et al. Frequency of injuries in recreational bodybuilding. Phys Educ Spor. 2007;51:40–4.
15. Golshani K, Cinque ME, O'Halloran P, Softness K, Keeling L, Macdonell JR. Upper extremity weightlifting injuries: diagnosis and management. J Orthop. 2017;15(1):24–7.
16. Kolber MJ, Beekhuizen KS, Cheng MS, et al. Shoulder injuries attributed to resistance training: a brief review. J Strength Cond Res. 2010;24960:1696–704.
17. Van der Wall H, McLaughlin A, Bruce W, et al. Scintigraphic patters of injury in amateur weight lifters. Clin Nucl Med. 1990;24:915–20.
18. Gross ML, Brenner SL, Esfrormes I, et al. Anterior shoulder instability in weight lifters. Am J Sports Med. 1993;21:599–603.
19. Brukner P, Khan K. Brukner & Khan's clinical sports medicine. In: 5ed, vol. 1. Syndey: McGraw-Hill Publishing; 2012.
20. Harrast M, Finnoff J. Sports medicine study guide and review for boards. 2ed. In: New York: springer publishing; 2017.
21. Cahill BR. Atraumatic osteolysis of the distal clavicle. A review. Sports Med. 1992;13(3):214–22.
22. Roedl JB, Nevalainen M, Gonzalez FM, Dodson CC, Morrison WB, Zoga AC. Frequency, imaging findings, risk factors, and long-term sequelae of distal clavicular osteolysis in young patients. Skelet Radiol. 2015;44(5):659–66.
23. Hanna CM, Glenny AB, Stanley SN, Caughey MA. Pectoralis major tears: comparison of surgical and conservative treatment. Br J Sports Med. 2001;35:202–6.
24. Refior HJ, Sowa D. Long tendon of the biceps brachii: sites of predilection for degenerative lesions. J Shoulder Elb Surg. 1995;4:436–40.
25. Schamblin ML, Safran MR. Injury of the distal biceps at the musculotendinous junction. J Shoulder Elb Surg. 2007;16:208–12.
26. Pearl ML, Bessos K, Wong K. Strength deficits related to distal biceps tendon rupture, report r A c. Am J Sports Med. 1998;26:295–6.
27. Hamer MJ, Caputo AE. Operative treatment of chronic distal biceps tendon ruptures. Sports Med Arthrosc. 2008;16:143–7.
28. Cools AM, Witvrouw EE, Mahieu NN, Danneels LA. Isokinetic scapular muscle performance in overhead athletes with and without impingement symptoms. J Athl Train. 2005;40:104–10.
29. Fees M, Decker T, Snyder-Mackler L, Axe MJ. Upper extremity weight-training modifications for the injured athlete. A clinical perspective Am J Sports Med. 1998;26:732–42.
30. Wagner LL, Evans SA, Weir JP, Housh TJ, Johnson GO. The effect of grip width on bench press performance. Int J Sport Biomech. 1992;8:1–10.
31. Elliott BC, Wilson GJ, Kerr GK. A biomechanical analysis of the sticking region in the bench press. Med Sci Sports Exerc. 1989;21:450–462.
32. Thompson WO, Debski RE. Boardman N.D. a biomechanical analysis of rotator cuff deficiency in a cadaveric model. Am J Sports Med. 1996;24:286–92.
33. Balazs GC, Brelin AM, Donohue MA, Dworak TC, Rue J-PH, Giuliani JR, Dickens JF. Incidence rate and results of the surgical treatment of pectoralis major tendon ruptures in active-duty military personnel. Am J Sports Med. 2016;44(7):1837–43.
34. de Castro Pochini A, Ejnisman B, Andreoli CV. Pectoralis major muscle rupture in athletes: a prospective study. Am J Sports Med. 2010;38:92–8.
35. Kakwani RG, Matthews JJ, Kumar KM, Pimpalnerkar A, Mohtadi N. Rupture of the pectoralis major muscle: surgical treatment in athletes. Int Orthop. 2007;31(2):159–63.
36. Wilk KE, Arrigo CA, Andrews JR. Current concepts: the stabilizing structures of the glenohumeral joint. J Orthop Sports Phys Ther. 1997;25:364–79.
37. Kolber MJ, Beekhuizen KS, Cheng MS, Hellman MA. Shoulder joint and muscle characteristics in the recreational weight training population. J Strength Cond Res. 2009;23:148–57.
38. Provencher MT, Bhatia S, Ghodadra NS, et al. Recurrent shoulder instability: current concepts for evaluation and management of glenoid bone loss. J Bone Joint Surg Am. 2010;92(Suppl. 2):133–51.
39. Soriano MA, Suchomel TJ, Marin PJ. The optimal load for maximal power production during upper-body resistance exercises: a meta-analysis. Sports Med. 2017;47:757–68.
40. Raske Å, Norlin R. Injury incidence and prevalence among elite weight and power lifters. Am J Sports Med. 2002;30(2):248–56.
41. Brown EW, Kimball RG. Medical history associated with adolescent powerlifting. Pediatrics. 1983;72:636–44.
42. Lander JF, Simonton RL, Giacobbe JKF. The effectiveness of weight-belts during the squat exercise. Med Sci Sports Exerc. 1990;22:117-124.
43. Chen SK, Lu CC, Chou PH, Gu LY, Wu WL. Patellar tendon ruptures in weight lifters following local steroid injections. Archives Orth and Trauma Surg. 2009;129:369–72.
44. Kujala UM, Kettunen J, Paananen H, et al. Knee osteoarthritis in former runners, soccer players, weight lifters, and shooters. Arthritis Rheum. 1995;38:539–46.

45. Brady TA, Br C, Bodnar LM. Weight training related injuries in the high school athlete. Am J Sports Med. 1982;10(1):1–5.
46. NSCA. N.S.C.a. POSITION PAPER: the squat exercise in athletic conditioning: a position statement and review of the literature. Strength Cond J. 1991;13:51–8.
47. Kulund DM, Dewey JB, Brubaker CE, et al. Olympic weightlifting injuries. Phys Sportsmed. 1978;6:111–8.
48. Myer GD, Kushner AM, Brent JL, Schoenfeld BJ, Hugentobler J, Lloyd RS, Vermeil A, Chu DA, Harbin J, McGill SM. The back squat: a proposed assessment of functional deficits and technical factors that limit performance. Strength Cond J. 2014;36:4.
49. Comfort P, Kasim P. Optimizing squat technique. Strength Cond J. 2007;29:10–3.
50. International Paralympic Committee (IPC). About the Sport. 2020. Available at https://www.paralympic.org/powerlifting/about (accessed July 1, 2020)
51. Willick SE, Cushman DM, Blauwet CA, et al. The epidemiology of injuries in powerlifting at the London 2012 Paralympic games: an analysis of 1411 athlete-days. Scand J Med Sci Sports. 2016;26(10):1233–8.
52. Mazur LJ, Yetman RJ, Risser WL. Weight-training injuries. Sports Med. 1993;16:57–63.
53. Baechle TR, Groves BR. Weight and training: steps to success. Leisure press. In: Champaign IL; 1992.
54. Fiatraone-Singh M, Hackett D, Schoenfield B, Vincent HK, Wescott W. American College of Sports Medicine (ACSM) Guidelines on Strength Training. Featured Download. July 31, 2019. Available at https://www.acsm.org/blog-detail/acsm-certified-blog/2019/07/31/acsm-guidelines-for-strength-training-featured-download (accessed July 1, 2020)

33 Wrestling

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33.1 Introduction

The origin of wrestling as a sport can be traced back to the Sumerians 5000 years ago. In ancient Egypt drawings in the tomb of Beni-Hassan show 400 pairs of wrestlers [1]. For the Greeks, wrestling trained men for war, and drawings of Greek wrestling style resemble contemporary Freestyle wrestling [1]. In 708 BC, wrestling was incorporated as a major discipline in the Olympic Games [1, 2]. Greek wrestling was called pankration ("all power"), combined boxing and wrestling techniques, and the pankratists were skilled at throws, takedowns, joint locks, and choke holds using the maneuvers in present day wrestling, and also now forbidden in Olympic Freestyle and Greco-Roman wrestling, but allowed in, for example, judo, Brazilian JuJitsu, and mixed martial arts. Trainers focused on developing strength, endurance, and skills best suited to each fighter. The only rules were that fighters were not allowed to bite or gouge out the opponent's eyes, but these were permitted in Sparta. The Olympic Pankration competition 332–331 BC is depicted on an amphora. Pankration was used on the battlefield by the Spartan hoplites and the Macedonian phalanx in the army led by Alexander the Great. Roman pancratium competitions took place in the Colosseum [3]. Wrestling and pancratium continued in many countries, and wrestling was reintroduced into the Olympic Games in 1896 [4]. Freestyle wrestling was included in the 1904 Olympic Games [2], Greco-Roman wrestling in the 1908 Olympic Games [5], and females were included in the World Championship organized by the International Federation of Associated Wrestling Styles (FILA) in 1987, to became an Olympic sport in the 2004 Olympic Games [4]. Wrestling is at present a very popular sport. It allows the participation of individuals matched according to their weight. Two different styles are Olympic sports: Freestyle wrestling, where the legs can be used to throw and grab an opponent, and the attacks can be brought below the waist; and Greco-Roman wrestling, which allows holds and attacks only above the waist, and only the upper limbs can be used to attack the opponent [4, 6]. Wrestling causes injuries found in other contact sports. Physicians must know how to manage such injuries, and must be able to

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assist athletes in different problems such as diet and weight loss, skin infections, and ear injuries.

33.2 Epidemiology

Wrestling injury rates are second only to American football. The average injury rate of male wrestlers is 16.3 per 1000 athletes [7–11]. Accident rates differ in the type of competition and age of the wrestlers. The injury rates of high school wrestlers were 5.8 per 100 athletes [8, 11], that of the Olympic wrestlers were 5.6 per 1000 athletes [9, 10], and that of the college wrestlers were 35.3 per 1000 athletes [7, 12]. A study of 6- to 16-year-old wrestlers provided a rate of 15.4 per 1000 athletes [13]. The injury rate of female wrestlers is on average 37.9 per 1000 female athletes [9]. Most injuries occur during takedowns, with rates of 74% [14] and 44% [15].

33.3 Location of Types of Injuries

33.3.1 Head and Neck Injuries

Head and neck injuries account for about 20% of all injuries [16–22]. Injuries to the head occur by head-to-head or head-to-knee collisions during takedowns. Concussions are also produced by contact with the wrestling mat or the floor surrounding the wrestling mat area. The fact that slamming an opponent to the mat is illegal makes serious head injury less likely via this mechanism. Rechel estimated that there were 6671 injured wrestlers in the United States each year [23], and Myers estimated 432 injuries in the age group between 7 and 11 years and 5320 in those aged 12–17 years nationwide [24]. A common mechanism for a neck injury occurs during a takedown. In this, the wrestler drives into their opponent with their neck, hyperextending it while "shooting." This can cause sprains, strains, and neurologic trauma such as stingers. Non-catastrophic injuries to the neck are common despite generally extremely well-developed neck muscle strength among wrestlers. The cumulative effects of neck injuries seen in some wrestlers are evidenced by an increased incidence of degenerative changes on radiographs and by long-term cervical pain and cervicobrachialgia reported by ex-wrestlers.

33.3.2 Cervical Strain/Sprain

A cervical strain is a tear of one of the musculotendinous units in the neck. The spectrum of injury ranges from mild to moderate, with actual muscle rupture being extremely rare. These injuries all result from mechanical overloading when the force is too great for the anatomical structure to withstand. Sprain to ligaments and capsular structures of the cervical spine are frequently seen concomitantly, making it difficult to differentiate between the two. The usual mechanism for sprains/strains is a hyperextension twisting injury. These account for approximately 50% of neck injuries in wrestling.

33.3.3 Stingers

Stingers occur in wrestling second only in frequency to American football. Most commonly, because of traumatic stretching or compression of the brachial plexus or nerve roots, they occur almost exclusively during takedowns [25]. The most common mechanism is forced hyperextension-ipsilateral flexion when a wrestler "shoots" a takedown with their neck extended, striking their opponent's chest or thigh with their forehead. Oblique neck extension results in narrowing of foramina, the mechanism of the compression-type stinger seen in wrestlers [26]. The vast majority are neurapraxic lesions. The clinical picture of these injuries is typical: severe, electric, shock-like pain occurs at the time of impact and extends from fingertips to shoulders. These momentary dysesthesias are replaced by a dull ache and numbness and weakness of the arm and hand lasting from a few seconds to a few minutes. The wrestler keeps the arm hanging limply by their side or with a flexed posture of the cervical spine, elevating the shoulder of the involved side. Both these maneuvers alleviate tension and pressure on the neurologic structures [27]. Often the wrestler shakes the wrist and rubs the affected arm. Tenderness is noted in the ipsilateral paraspinous muscles and trapezius. The range of motion tends to be decreased in all planes and guarding occurs. Changes in neck range of motion, particularly lateral bending, are persistent. Ipsilateral lateral bending and extension coupled with axial loading reproduce the pain.

Neurological deficits are most commonly found in the upper brachial plexus distribution (C5–C6), but, in contrast to American football, low brachial plexus lesions (C7–T1) are not uncommon. The lower trunk of the brachial plexus or the lower cervical nerve roots are more vulnerable to injury with the arm abducted overhead. This corresponds to the position that the wrestler adopts during the takedown and the clinical findings of more frequent lower brachial plexus injury among wrestlers than of American football players. Deficits in motor strength, particularly of the biceps, deltoid, and external rotators of the shoulder, are the rule, but these usually normalize within 5 min of the injury.

Occasionally, weakness persists for extended periods, and less often becomes chronic with noticeable muscle atrophy. Changes in sensation and deep tendon reflexes are also noted only transiently. If complete recovery without symptoms occurs within 1–2 min, immediate return to action is permitted. If any neurologic abnormality exists after that time, the wrestler is restricted from participation. If the wrestler returns to action before pain, tenderness, and range of motion have returned to normal, the likelihood of recurrence is extremely high. However, wrestlers who return with no symptoms or radiographic changes have no obvious increased risk over their teammates. Repetitive acute injuries in the same season will often lead to persistent rather than just transient weakness. Repeated trauma appears to induce osteophyte formation and resultant foraminal narrowing. Several wrestlers develop severe radiographic changes and permanent neurologic loss on this basis. Despite stopping the repetitive trauma of wrestling, 28–37% of ex-wrestlers with a history of neck injury report continued symptoms. Electrodiagnostic testing is generally not needed because of the relatively short duration of symptoms. Rehabilitation initially focuses on range of motion and reducing irritation of the neural structures. Next, strength considerations are important as these are the dynamic stabilizers of the cervical spine, and thus will allow complete restoration with reduced risk [25].

33.3.4 Cervical Cord Neurapraxia

Axial loading with hyperflexion or hyperextension causes cervical cord neurapraxia or transient quadriplegia. Symptoms are seen in the upper and lower extremities bilaterally, and can consist of sensory or motor components, or both. Symptoms typically last 10–15 min. Return to play depends on the mechanism of the injury, the anatomical site, imaging findings, and the time to full recovery.

33.3.5 Ear Injuries

The classic problem of the wrestler's ear is auricular hematomas. The percentage of injuries to the ears varies from 0.9 to 23.4% [28]. The auricular hematoma or "cauliflower ear" results from blunt trauma to the ear, either on direct impact with another wrestler's head or knee, or by abrasive, friction-causing forces, as when wrestlers "tie up" head-to-head. The anterior or lateral surface of the auricle with its multiple depressions and eminences has skin which is thin and tightly adherent to the underlying fibrocartilage. Conversely, the posterior medial skin is thicker and more mobile. Hematomas form anteriorly where the skin is most adherent and rarely extend posteriorly. The skin does not "slide," which results in high shear forces in response to blunt trauma. Blood vessels tear and blood accumulates in the space separating the cartilage and perichondrium, forming a hematoma [29–31]. An auricular hematoma typically presents as a tender, tense, and fluctuating swelling on the anterior surface of the ear, with mild to moderate throbbing pain [32]. The hematoma typically fills the hollow between the helix and the antihelix (scapha) and extends forward into the fossa triangularis. Less frequently, the hematoma may occupy the concha or the area in and around the external auditory meatus. An auricular hematoma may also occur on the posterior surface of the ear, or possibly on both surfaces, although this is less common [33]. The risk of necrosis is greater if hematomas are present on both anterior and posterior surfaces [34]. The overlying skin may have normal color, or may be erythematous or ecchymotic [32]. As the blood supply to the perichondrium is cut off by the expanding hematoma, the cartilage undergoes necrosis, and in time is replaced by fibrocartilaginous scar tissue, producing the typical "cauliflower ear" [35]. Treatment goals include restoring the normal appearance and pliability of the ear, early return to wrestling activity, and low recurrence rate. Acute evacuation under sterile technique to minimize risk of infection is required for all auricular hematomas [36, 37]. Needle aspiration or incision and drainage can be performed by or under direct supervision of a physician. It is important for this to occur as quickly as possible so that the cartilage does not become necrotic. An alternative approach is required if the hematoma is more than 7 days old. Such hematomas will often be more organized and more difficult to drain. There may also be ulceration and/or necrosis of the skin, in which case the patient should be referred to an otorhinolaryngologist or plastic surgeon [32]. The aspiration technique includes anesthesia with 1% lidocaine, betadine sterilization, insertion of 18-gauge needle connected to 10 mL syringe inserted into the largest area of the hematoma; aspiration while milking the hematoma with the finger and index finger; application of pressure for 3–5 min to the hematoma; if a blood clot remains, a hemostatis inserted in the hematoma after making a small incision to break up the clot; a pressure dressing is applied once the entire clot has been evacuated; the ear is checked in 24 h to evaluate for fluid reaccumulation.

Incision and drainage technique: anesthesia with 1% lidocaine; sterilization of the area with betadine; using a no. 15 scalpel blade, the hematoma is incised parallel to the natural skin folds; the hematoma is completely evacuated, and the area is irrigated with normal saline; an antibiotic ointment is applied to the incision; using a 4–0 nylon suture, the opposing skin edges of the incision are brought together, suturing through the cartilage passing the suture around a dental roll. Using an incision and drainage approach with application of dental rolls carries the lowest incidence of recurrence when compared with aspiration or incision and drainage alone [34]. Many methods have been described to prevent reaccumulation of fluid. Contemporary techniques use different types of suturing over materials including dental rolls and different silicone materials [38–41]. An area with little blood supply is vulnerable to infection. It is recommended that all patients receive 7–10 days of prophylactic antibiotics [37].

Protective gear should be worn by athletes at risk of receiving a direct blow to the ear. Quick and complete drainage of an auricular hematoma can prevent abscess and scar formation. Follow-up with the patient 24 h after drainage is recommended to assess for reaccumulation of the hematoma [31]. Athletes can return to play immediately after evacuation of the hematoma if protective gear is worn, and care is taken to minimize the risk of infection [34]. The traditional wrestler's headgear, which covers the ears, is designed to minimize the risk of these injuries. While most often hematomas result when wrestling headgear is not being worn, hematomas can and do happen with headgear on. Once the wrestler begins to sweat, the headgear can slide and cause the hematoma itself by abrading the external ear. Several design features contribute to this problem—shallow depth of the earpieces, inadequate number of straps for fixation, and construction with plastic materials which allow sliding of the headgear [40].

33.3.6 Knee

Knee is the single most commonly injured anatomical region. In prospective studies, knee injuries have ranged from 7.6 to 44% of all wrestling injuries [10, 28]. In the only study with the percentage of knee injuries below 10%, Lorish et al. described injuries in tournaments to wrestlers aged 6–16 years [13]. More number of injuries to knee occurred in attack position [16]. Knee injuries are more frequent in older athletes, and they are also correlated with the duration of athletic practice; there seems to be no correlation with the athlete's weight and category [16]. Takedowns are involved in most knee injuries of all types. Meniscus injuries occur most commonly during takedowns through a twisting injury to a weight-bearing extremity. Collateral ligament sprains occur when a varus or valgus force is applied to the weight-bearing extremity of the defending wrestler. These mechanisms far more commonly cause injuries than application of holds that intentionally apply twisting forces: these techniques are considered illegal, and are penalized. The most common knee injuries are sprains, which constitute 30–65% of all knee injuries. Meniscal injuries are also common, with a relatively high proportion of lateral to medial meniscus tears [42]. Wroble et al. reported that the most frequent knee injuries included prepatellar bursitis, lateral and medial collateral ligament sprains and meniscal tears [13]. Ligament and muscular sprains and strains were highly prevalent in a recent prospective study [42]. Meniscus injuries are the most common knee injuries leading to surgery, and another very common injury is prepatellar bursitis. On the other hand, wrestling has a very low incidence of ACL and other catastrophic knee injuries. Nonetheless, we are aware of two knee dislocations occurring in wrestling matches.

33.3.6.1 Ligament Injuries

The most common knee injuries are sprains, which constitute 30–65% of all knee injuries [42]. Three studies provided epidemiological data [16, 18, 20], with a weighted average of 17.3% of all injuries.

Two studies reported 1747 ligament tears and cartilage injuries, with weighted averages of 11.7% and 0.85/1000 AE of all injuries [8, 43]. The medial and lateral collateral ligaments are the most commonly affected [44]. The cruciate ligaments are injured less often, in contrast to American football. Not surprisingly, most injuries occur during takedowns with the defending wrestler being injured most often. Some wrestlers may function reasonably well in the short term with anterior cruciate-deficient knees because the demands of the sport are such that the knee is uncommonly in a position where tibial subluxation could occur. Nonetheless, most wrestlers will not have satisfactory function in their sport. However, given the peripheral and core muscle control, and the intrinsic proprioception capabilities of wrestlers, it is not unreasonable to implement a period of 6–12 weeks of intensive rehabilitation to ascertain whether the athlete can function without an anterior cruciate ligament. If this fails, we recommend ACL reconstruction with return to wrestling at about 6 months. Functional braces are of no or at best questionable value. Psychologically, it seems to cause a big disadvantage to the wrestler, making them more aware of their own disability. They also know that their opponent is aware of it and may try to exploit it. In addition, we are skeptical that a brace will afford any substantial protection. On the other hand, those wrestlers who do use functional braces must have them taped and padded for matches and practice. Wrestling rules dictate that any device must be protected so that it cannot cause injury to the opponent by sharp corners or exposed metal, etc. The rules also state that braces may not limit motion.

33.3.6.2 Meniscus Injuries

Meniscal injuries are common, with a relatively high proportion of lateral to medial meniscus tears [42]. One study reported 3 torn menisci (3% of total injuries) [45]. Lateral meniscus injuries were 46% of the total number of meniscal injuries, and there were 45% lateral versus medial meniscectomy in a study on 56 meniscectomies in wrestlers [10, 46]. Lateral meniscus injuries were 58.3% of all meniscal injuries in a recent study [42].

33.3.6.3 Prepatellar Bursitis

Prepatellar bursitis is associated with frequent recurrences, occasionally requires surgery, and accounts for substantial time loss. Prepatellar bursitis can be caused by a single traumatic event (e.g., forceful impact of the knee to the mat) or by chronic repeated trauma. In both, takedowns are frequently implicated. Mysnyk et al. documented 28 cases of prepatellar bursitis, representing 21% of all knee injuries [47], with 50% being recurrent injuries, at times with a septic component. Prepatellar bursitis represented 16.88% of all lesions in a recent study [42]. Of these, 46.15% were recurrent, and there were no cases of septic bursitis. The diagnosis of prepatellar bursitis usually is not difficult. There is usually a history of trauma, even though no exact inciting incident can be recalled. Swelling occurs superficial to the patella, and effusion may not be present. The range of motion is relatively painless, even in septic cases, except with maximum flexion. Conservative management is difficult since repeated irritation is inherent to the sport.

Aggressive initial treatment is indicated in wrestlers with prepatellar bursitis. This is important, not only because the initial episode is disabling but also because early aggressive management gives the best chance to prevent progression to chronic bursitis. In chronic bursitis, the bursal wall is thickened and becomes irritated by minimal trauma. Excision of the bursa is generally required. When a wrestler presents with typical symptoms and signs, iontophoresis with 5% hydrocortisone and a non-steroidal anti-inflammatory drug (NSAID) can be used. Wrestling is rarely curtailed, and wrestlers can return to training and competition with a Neoprene knee sleeve with extra Neoprene anteriorly. Petroleum jelly is applied to the anterior aspect of the knee before the sleeve is worn, decreasing friction between the skin and the sleeve, thereby reducing the irritation imposed on the prepatellar bursa. For the few wrestlers who do not respond to this routine and develop a recurrence, the same process will be repeated, imposing a rest period. If a second recurrence arises, bursectomy is recommended. Bursectomy can be performed easily using arthroscopic techniques, minimizing the time off the mat. Intrabursal corticosteroid injections are not used as they do not have any effect on recurrence or time loss. At times, septic bursitis, most commonly caused by *Staphylococcus aureus*, may present with typical local evidence of infection [48]. Most cases likely result from direct penetration through the skin, even though a wound is rarely obvious. Continuing blunt knee trauma and minor mat burns may allow bacterial seeding to occur. Because the systemic or even local symptoms are often lacking, early septic bursitis is more difficult to diagnose than septic arthritis. However, since the prepatellar bursa is a closed space, not communicating with the joint, complications of septic bursitis are rare. Aspiration, Gram stain, and culture should be performed in all wrestler with a bursitis, regardless of whether they appear infected. The aspirate is not always grossly purulent. Once the diagnosis is established, and even before if clinical suspicion is high, oral antibiotics, preferably a first-generation cephalosporin or a penicillinase-resistant penicillin, should be started if the infection is not severe. The wrestler may need parenteral treatment if the infection is severe or if oral treatment fails. Incision and drainage with irrigation using local anesthesia are necessary only if the bursa appears loculated, and clinical improvement does not occur with repeated aspirations. Wrestlers, particularly those with a history of prepatellar bursitis, are encouraged to use kneepads [44].

33.3.7 Foot and Ankle Injuries

The most common ankle injuries are inversion injury and the lateral ligament sprain, which most often occurs during takedowns. Two specific mechanisms have been identified. First, when a wrestler attempts to throw his opponent and rises onto his toes and twists, a momentary loss of balance may cause him to roll over his ankle into an inverted position. The second occurs to the defensive wrestler during the takedown. When his opponent lifts one of his legs, his support remains on a single foot. As his opponent attempts to bring him to the mat by various combinations of rapid changes in direction or trips, inversion stress can occur. Most often, these sprains are mild. In prospective studies, ankle injuries have ranged from 3.9% to 9.7% of total wrestling injuries [28]. Standard wrestling shoes afford little protection to the ankle: the rubber soles cause very high friction with the mat and have virtually no give. Although they extend above the ankle, the uppers consist of nylon or soft leather, and offer little support.

33.3.8 Shoulder Injuries

Shoulder injuries account for 3.5–24% of all wrestling injury [28]. The shoulder is injured in three main ways. When being thrown to the mat from a standing position, a wrestler may attempt to brace his fall with his extended arm (Fig. 33.1). Alternatively, the fall may be taken directly on the shoulder. The third mechanism of shoulder injury occurs during takedown maneuvers. When the wrestler attacks his opponent's legs and gets caught in a position with the body overextended, the head down, and the arm elevated above the head, the defender's body lies above the attacker's shoulder. As the opponent throws his hips back and increases the weight upon the wrestler's shoulder, hyperflexion, and external rotation ensues, causing anterior subluxation. The most common problems were anterior shoulder instability (subluxation/ dislocation), acromioclavicular (AC) sprain, and muscle-tendon strain [18, 43].

All wrestlers are subject to forces that tend to stress or stretch the anterior structures of the shoulder, such as the half nelson technique, and all those activities which place the shoulder in abduction and external rotation (Fig. 33.2). The most frequent surgeries are SLAP repair followed by Bankart lesion, bursectomy, and muscle repair [49].

Shoulder instability necessitates that the wrestler modifies his technique, to avoiding maneuvers that may sublux the shoulder. The overextended takedown position described previously is a good example. The wrestler is taught to use other options: using the other arm, using the upper body rather than attempting leg attacks, or employing a defensive or counter strategy.

Acromioclavicular (AC) sprains are nearly as common as anterior instability.

They occur almost exclusively from a fall on the unprotected shoulder. This happens when a wrestler, taken down by his opponent, is brought to the mat with his arm trapped. Occasionally, the force is transmitted medially along the clavicle and causes simultaneous sprains of the ipsilateral sternoclavicular joint.

Almost all AC injuries are type I or mild type II.

Should a type I or II AC sprain occur during a match, the wrestler is allowed to continue as long as he can tolerate the pain. After an AC injury

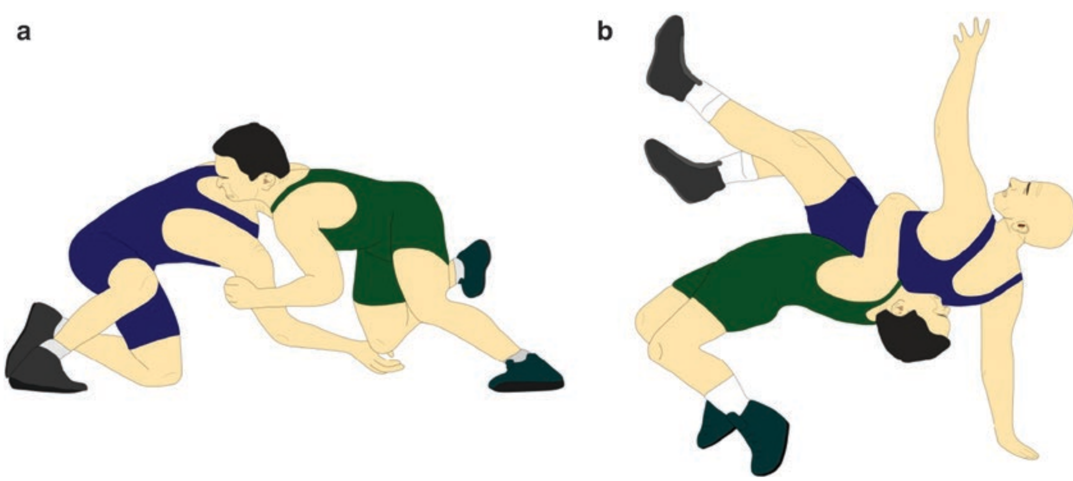

Fig. 33.1 Shoulder injury mechanisms. (**a**) Fall taken directly on the shoulder; (**b**) Fall with his extended arm

Fig. 33.2 All wrestlers are subject to forces that tend to stress or stretch the anterior structures of the shoulder, such as the half nelson technique, and all those activities which place the shoulder in abduction and external rotation

resulting in time loss, early return to competition is the goal. The probability of repetitive direct trauma and multiple aggravations by direct traction on the affected arm makes early return problematic at times. In type I or mild type II injury, the wrestler returns to training and competition according to his symptoms, typically in 2–3 weeks in mild type II injuries. In more severe type II and all type III injuries, deformity is accepted, early range of motion is allowed as soon as tolerated, and scapula and shoulder rotational strengthening is implemented. Return criteria include no tenderness, 90% strength of deltoids and external rotators, and no pain with downward traction of the adducted arm. Laxity and deformity persist in many wrestlers, and occasionally wrestlers develop posttraumatic osteolysis of the distal clavicle, causing persistent low-level pain.

An almost unique injury to wrestling is the rupture of the pectoralis major muscle. This occurs by sudden forceful overload to the maximally contracted pectoralis muscle (Fig. 33.3). The shoulder is usually in adduction, internal rotation, and forward flexion. A sudden pop may be heard and is accompanied by severe pain and weakness. Swelling and bruising develop rapidly. A painful defect accentuated by strong adduction against resistance as well as weakness in adduction, internal rotation, and flexion is diagnostic [50, 51]. The outcome of surgical treatment is better than conservative management [52].

33.3.9 Elbow Injuries

Elbow injuries account for 1.0–7.9% of all wrestling injuries [28]. The most common elbow injury is the hyperextension–abduction sprain affecting the ulnar collateral ligament (UCL) and the anterior capsule [11, 18]. Elbow injuries most commonly occur when the wrestler is forcibly brought down to the mat with an arm extended or "posted." At low-to-moderate force levels, hyperextension–abduction sprains occur. With higher forces, an elbow dislocation can result. All these

Fig. 33.3 Rupture of the pectoralis major muscle. The shoulder is usually in adduction, internal rotation, and forward flexion

injuries present with medial pain, with or without concomitant anteromedial pain. Elbow dislocation may be complicated by entrapment of the brachial artery. Care should be taken to monitor radial pulse and capillary refill [53]. If an ulnar neurapraxia has occurred, the wrestler may describe paresthesiae or numbness in the ulnar two fingers. On examination, tenderness is found along the ulnar collateral ligament (UCL) and anterior capsule. Pain increases with passive extension and valgus stress. Neurological findings may include weakness of the intrinsic muscles of the hand, decreased sensation to light touches, and a positive Tinel's sign in the ulnar groove. In a match, the wrestler may continue if tenderness is minimal, range of motion is full, and moderate valgus stress does not produce much pain. If symptoms and findings are more severe, he is withdrawn from competition. In the presence of a dislocated elbow, if the clinician is certain of the diagnosis, gentle closed reduction should be attempted on the mat before muscle spasm and swelling are severe. Detailed neurovascular assessment must be performed before and after reduction. Furthermore, the wrestler must be sent for immediate radiographic evaluation. Treatment of these injuries follows the RICE algorithm with addition of a sling or hinged elbow brace in more severe cases. The brace is initially locked in an angle comfortable to the wrestler (about 60°–90°). Mobilization begins immediately with mild sprains, and after about 7 days with elbow dislocations. Motion should be resumed early. Prolonged immobilization invariably leads to limited range of motion and difficult rehabilitation. Return to competition occurs when range of motion is full and pain-free, strength is equal to the contralateral arm, and tenderness and pain with valgus stress are absent. Phased return to wrestling after elbow dislocation occurs at about 4–6 weeks with careful monitoring of symptoms and findings. Mild sprains may result in only a few days time loss. Ulnar collateral ligament (UCL) reconstruction may be required in wrestlers with chronic insufficiency, which is manifested by numerous recurrent sprains. After UCL reconstruction, wrestling is curtailed for at least 6 months and prognosis for return to wrestling at the preinjury level is guarded [54].

33.3.10 Lumbar Spine

Lower back injuries commonly take place during takedowns. While sparring for position, wrestlers push against each other with the lumbar spine in slight hyperextension. This extension, coupled with twisting, results in injuries. Extension against resistance, as in lifting an opponent off the mat, and hyperflexion, as in rolling, are also mechanisms that account for low back sprain or strain. On the other hand, low back injuries may result not only from a single episode but also from repetitive use. Low back injuries are less frequent, and are generally less severe than corresponding cervical injuries. Low back injuries involve 1.2–18.6% of total wrestling injuries in prospective and retrospective studies [28]. The most frequent types of injuries are sprains and strains [44].

Wrestlers with back pain often have spondylolysis or spondylolisthesis [18, 55]. There appears to be a defined association between back pain and low extensor muscle strength [56], so a personalized muscle strengthening and reconditioning program should be performed. When sprains or strains of the lower back occur, the athlete usually reports a feeling of "something twisting" or that their back "comes out." The localization of pain can be at the level of the muscular abdomen or at the insertion site, unilateral or bilateral and maximum at about 24–48 h. Physical examination evidences a reduced range of motion of the lumbar spine, a normal neurological examination, and signs of sciatica tension. Anterior–posterior and lateral radiographs of the lumbosacral spine are obtained if the symptoms are severe enough to bring the wrestler to the clinic. The oblique views better show the lesions of the pars interarticularis and are obtained if the low back pain is persistent despite treatment. A bone scan is useful if plain radiographs are normal and there is a high suspicion of pathology.

The management of the acute injury of the lumbar area is divided into three general areas: rest, pain and spasm reduction, and patient education. Relative rest allows conditioning and strengthening, weight training exercises in which a significant load is transferred through the lumbar spine (e.g., military press or cleaning) are eliminated, and wrestling is not allowed. The modalities used for pain control include ice, massage, ultrasound, TENS, NSAIDs, immobilization, and epidural steroid injections (if root symptoms are present). An anti-lordotic brace or elastic lumbar support may be useful to control pain during the activity modification period, but it is not practical when the wrestler returns to competition. Most injuries are self-limiting, with return based on full range of motion and strength criteria. Particular attention should be paid to the prevention and management of back injuries in athletes. A personalized conditioning and stretching program should be carried out. A stretching program should include not only stretching the lower back but also the hip flexors and the hamstring muscle group.

33.3.11 Rib and Chest Injuries

Rib cage injuries are a consequence of direct trauma during takedowns when the opponent's head or shoulders hit the chest of the defending wrestler. The most common mechanism is when a wrestler brings down his opponent by gripping him from the waist, so that the ribs of the defender rest on the arm of the attacking wrestler. When the opponent lifts or throws the wrestler while in a bear hug, the force produced by the opponent's grip may be sufficient to cause injury to the chest wall. Rib and chest injuries account for 3.6–14.3% of the total injuries suffered by wrestlers in prospective studies [28]. Most of these injuries are bruises or costochondral sprains, and rib fractures are relatively infrequent. Injury to the costochondral junction ranges from contusion to sprain to dislocation. The lesions are located on the anterior margin of the ribs, about 7–10 cm lateral to the sternum, where the ribs articulate with the rib cartilages. Clinically, the wrestler reports anterior chest pain, accentuated by coughing and sneezing. Physical examination evidences local tenderness and swelling at the costochondral junction. Direct pressure on the injured area, on the same rib in the axillary line and sternal pressure cause pain. If a subluxation or dislocation is present, a palpation or click may be perceived on palpation. Radiographs are non-contributory unless a fracture of the bone rib has occurred lateral to the costochondral junction. Symptomatic management is instituted with ice, NSAIDs and TENS used for pain control. A few days of rest (usually 7–10 days) are necessary until the symptoms have subsided. Immediately after the injury, even deep breathing alone can be painful, which makes conditioning difficult. Taping and splinting are ineffective and tend to draw the opponent's attention to the injured rib cage. However, it is possible to try a circumferential compressive dressing around the trunk with an elastic tape, an elastic band, or a corset for symptomatic relief. Rarely local anesthetic injections are necessary. Occasionally, the injury is complicated by the late development of calcifications at the costochondral junction, which produce a painless mass on only cosmetic importance [57].

33.3.12 Dermatological Conditions

Skin infections are very common in wrestling. The risk factors are repeated physical contact, superficial skin cuts and abrasions, the heat and humidity of the typical wrestling training gymnasium, and the general inattention to the hygiene of the wrestlers. These conditions provide an ideal environment for the infectious skin flora. The etiology of skin infection in wrestling includes bacterial, viral, and fungal agents. Early recognition of common dermatological problems is needed to provide timely and adequate treatment. Proper management accelerates the resolution of skin lesions, and protects teammates and opponents from exposure. Wrestling rules recommend that a doctor be present at the weights in all tournaments and examine competitors for communicable diseases. Referees perform this function when doctors are unavailable. Disqualification of an individual from competition can occur if, in the opinion of the attending physician, the presence of a communicable disease makes participation inadvisable. In most studies of wrestling injuries, skin diseases are not reported or rarely investigated in detail. As a percentage of the total injuries reported, skin problems occurred from 5% to 21.6% [28].

33.3.13 Injury Severity

In a study of the US National Centre for Catastrophic Sports Injury 1981–1999, 17 nonfatal, 17 serious, and one fatal (brain hemorrhage) catastrophic injuries were reported. The 17 serious injuries involved 11 cases of quadriplegia, one of paraplegia, and six multiple neurological deficits from spinal cord injury; there were also three severe head injuries. Twenty-two wrestlers required surgery, typically a cervical vertebral fusion, 12 athletes made a full recovery, and of these six resumed wrestling. Twenty-six injuries occurred during takedown and nine in a kneeling or in ground defense position [14]. A study of an overlapping and shorter period of the US National Catastrophic Sports Injuries database 1982–1988 found 23 catastrophic injuries in high school wrestlers, reporting them in a different format from Boden [14]. Fifteen injuries were from trauma to the spinal cord or the head, of which 2 were fatal, and 10 occurred during wrestling matches but were not due to wrestling (four heart failure, four "recorded as natural causes," and two "exact cause unknown") [58]. One study from Iran identified nine catastrophic injuries, all of which were heart attacks or strokes in veteran wrestlers with known cardiac conditions [59, 60].

33.4 Clinical Outcome/Residual Disability

Wrestling produces a high rate of wear on the joints and the spine. Former wrestlers have neck problems almost three times more frequently than non-athletes, and knee problems almost four times more frequently [61].

33.5 Physical Characteristics

Injuries are related to the weight and age of the wrestlers. The heavier wrestlers, being able to generate greater forces, cause more damage. Younger wrestlers are less strong and muscularly immature. Wrestling with its weight classes ensures homogeneity among the wrestlers, reducing the risk of injuries. However, the rapid and drastic weight loss performed to “make the weight” can itself be a risk factor for injury, given the adverse physiological changes induced by dehydration and fasting [60]. However, a wrestler who loses fat can improve relative power and stamina. This suggests that the wrestler who practices rapid weight loss may compete with a relatively weaker opponent. This in turn may be reflected in an increase in injury rates in the weaker of the two wrestlers [62]. In high school wrestlers, those with the lowest body fat tend to be more likely to succeed than those who struggle with higher body fat [63]. Catastrophic injuries occur most often in inexperienced wrestlers, as they may find themselves in precarious situations that predispose them to serious injuries [14].

33.6 Motor/Functional Characteristics

Fatigue does not appear to be correlated with increasing injury rates. Injuries did not increase in subsequent rounds and tournaments [10, 64].

33.6.1 Exposure

Injury rates increase with the level of competition, as the intensity of competition increases. The match accident rates are always higher than those in training. The more explosive and collision-oriented maneuvers involved in the moves also result in higher accident rates. A major risk factor is assuming a defensive role in an upright position, when the defending wrestler was injured 60% of the time [44]. In some studies, the position of the wrestler at the time of injury was analyzed. Injury rates are higher for disadvantaged or disadvantaged wrestlers in varsity and high school wrestlers [44, 66].

33.6.2 Training Conditions

Inadequate wrestling technique can increase the risk of injury. A good example is how shoulder injuries occur during taking downs. Anterior subluxations or sprains commonly occur if a wrestler is caught in an overextended position with the shoulder flexed fully forward and turned outward.

Dermatological problems arise from close contact with the opponent’s body, which is obviously integral to the sport, in combination with heat, humidity, sweaty training clothes, and contact with the mat. Contact with an opponent with a cold sore or a rash is also a risk factor [57].

33.6.3 Environment

The general training pattern can affect injury rates. Injuries occur in the first half of the wrestling season [44, 67]. The poor conditions of wrestling mat are a risk factor for accidents, and they can increase the rates of prepatellar bursitis [47]. The use of older and deconditioned wrestling mats during the off season is a potential reason. If the mats are in poor condition, their ability to absorb shocks can deteriorate, and therefore increase the risk of injury when wrestlers land on them. Another element is the state of cleanliness of the mats. Without daily disinfection, the count of microorganisms on the mat increase, and therefore increases the chance of transmission of dermatological infections from the mat to the wrestler. Unpadded walls, obstacles such as columns or steps, inadequate space, and extreme heat or humidity are obviously harmful [57, 65].

33.6.4 Protective Equipment

Normally, wrestlers do not wear much in terms of protective gear. Not wearing headgear is a risk factor in auricular hematoma, though these injuries may also occur with headgear [40] when it does not fit properly. The role of knee pads and shoes has not been assessed, but in other sports they have been effective in preventing injuries.

Mouthguards are not necessary in wrestling, but considering that American football, ice hockey, and rugby players have seen a drastic reduction in dental injuries with the use of mouthguards, wrestlers may benefit from their use [57].

33.6.5 Injury Prevention

Wrestling is an aggressive contact sport in which it is impossible to avoid potential injury situations. However, it may be possible to prevent them. Few wrestling studies are rigorous enough to draw definitive conclusions about risk factors, but it is still possible to suggest measures that reflect the best and most up-to-date information in the literature. Obviously, these suggestions should be confirmed by well-controlled prospective studies. Preventive measures are suitable for warm-up, muscle strengthening, and cardiovascular conditioning program, improve and correct the technique, no gross discrepancy in practice partners, limit weight loss, improve hygiene, ensure adequate supervision by the coach qualified, use appropriate headgear and mouthguards, ensure adequate space and appropriate temperature/humidity, use quality mats with frequent disinfection, guarantee the presence of qualified medical personnel during competitions, validated emergency protocols and precise treatment protocols, and appropriate post-injury rehabilitation [57].

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References

1. United World Wrestling. History of wrestling. https://unitedworldwrestling.org/organization/history
2. The International Olympic Committee. Olympic games, freestyle wrestling. https://www.olympic.org/wrestling-freestyle
3. https://en.wikipedia.org/wiki/Pankration
4. García-Pallarés J, López-Gullón JM, Torres-Bonete MD, Izquierdo M. Physical fitness factors to predict female Olympic wrestling performance and sex differences. J Strength Cond Res. 2012;26(3):794–803.
5. The International Olympic Committee, Olympic Games, Greco-Roman Wrestling. https://www.olympic.org/wrestling-greco-roman
6. Chaabene H, Negra Y, Bougezzi R, Mkaouer B, Franchini E, et al. Physical and physiological attributes of wrestlers: an update. J Strength Cond Res. 2017;31(5):1411–42.
7. Estwanik JJ, Bergfeld J, Canty T. Report of injuries sustained during the United States Olympic wrestling trials. Am J Sports Med. 1978;6(6):335–40.
8. Pasque CB, Hewett TEA. Prospective study of high school wrestling injuries. Am J Sports Med. 2000;28(4):509–15.
9. Shadgan B, Feldman BJ, Jafari S. Wrestling injuries during the 2008 Beijing Olympic games. Am J Sports Med. 2010;38(9):1870–6.
10. Strauss RH, Lanese RR. Injuries among wrestlers in school and college tournaments. JAMA. 1982;248:2016–9.
11. Yard EE, Comstock RD. A comparison of pediatric freestyle and Greco-Roman wrestling injuries sustained during a 2006 US national tournament. Scand J Med Sci Sports. 2008;18:491–7.
12. Kersey RD, Rowan L. Injury account during the 1980 NCAA wrestling championships. Am J Sports Med. 1983;11(3):147–51.
13. Lorish TR, Rizzo TD Jr, Ilstrup DM, Scott SG. Injuries in adolescent and preadolescent boys at two large wrestling tournaments. Am J Sports Med. 1992;20(2):199–202.
14. Boden BP, Lin W, Young M, Mueller FO. Catastrophic injuries in wrestlers. Am J Sports Med. 2002;30(6):791–5.
15. Hoffman HS, Powell JW. Analysis of NATA high school injury data on wrestling. Athletic Training. 1990;25(2):125.
16. Agarwal S, Mann E. Knee Injuries in wrestlers: a prospective study from the Indian subcontinent. Asian J Sports Med. 2016;7(4):e35000. https://doi.org/10.5812/asjsm.35000.
17. Estwanik JJ, Rovere GD. Wrestling Injuries in North Carolina high schools. Phys Sports Med. 1983;11(1):100–8. https://doi.org/10.1080/00913847.1983.11708438.
18. Estwanik JJ III, Bergfeld JA, Collins HR, Hall R. Injuries in interscholastic wrestling. Phys Sports Med. 1980;8(3):111–21.
19. Kordi R, Akbarnejad A, Wallace WA. Catastrophic injuries in the Olympic styles of wrestling in Iran. Br J Sports Med. 2010;44(3):168–74. https://doi.org/10.1136/bjsm.2008.046870.
20. Lők R, Yücetürk G. Injuries in wrestling. J Sport Med. 1974;2(6):324–8.
21. Otero JE, Graves CM, Bollier MJ. Injuries in collegiate wrestlers at an elite division I NCAA Wrestling

program: an epidemiological study. Iowa Orthop J. 2017;37:65–70.
22. Powell JW. National athletic injury/illness reporting system: eye injuries. Int Ophthalmol Clin. 1981;21(4):47–58.
23. Rechel JA, Yard EE, Comstock RD. An epidemiologic comparison of high school sports injuries sustained in practice and competition. J Athlet Train. 2008;43(2):197–204.
24. Myers RJ, Linakis SW, Mello MJ, Linakis JG. Competitive wrestling-related injuries in school aged athletes in U.S Emergency Departments. West J Emerg Med. 2010;1(5):442–9.
25. Kuzma SA, Doberstein ST, Rushlow DR. Postfixed brachial plexus radiculopathy due to thoracic disc herniation in a collegiate wrestler: a case report. J Athl Train. 2013;48(5):710–5.
26. Feinberg JH, Nadler SF, Krivickas LS. Peripheral nerve injuries in the athlete. Sports Med. 1997;24(6):385–408.
27. Feinberg JH. Burners and stingers. Phys Med Rehabil Clin N Am. 2000;11(4):771–84.
28. Hewett TE, Pasque C, Heyl R, Wroble R. Wrestling injuries. Timothy Med Sport Sci. 2005;48:152–78.
29. Giffin CS. The wrestler's ear (acute auricular hematoma). Arch Otolaryngol. 1985;111(3):161–4.
30. J T, Renner GJ. Injuries of the external ear. Otolaryngol Clin North Am. 1990;23(5):1003–18.
31. Giffin S, Charles S. Wrestler's ear: pathophysiology and treatment. Ann Plasr Surg. 1992;28:131–9.
32. Ingvaldsen, Aam C, Tønseth, Alexander K. Auricular haematoma. Tidsskrift for Den norskelegeforening. 2017;137(2):105–7.
33. Shakeel M, Vallamkondu V, Mountain R, et al. Open surgical management of auricular haematoma: incision, evacuation and mattress sutures. J Laryngol Otol. 2015;129:496–501.
34. Eagles K, Fralich L, Stevenson JH. Ear trauma. Clin Sports Med. 2013;32:303–16.
35. Kelleher JC, Sullivan K, Baibak G, et al. The wrestler's ear. Plast Reconst Surg. 1967;40:540–6.
36. Jones SE, Mahendran S. Interventions for acute auricular haematoma. Cochrane Database Syst Rev. 2004;2:CD004166.
37. Malloy KM. Assessment and management of auri- cular hematoma and cauliflower ear. UpToDate- versjon 9.9. 2015. www.uptodate.com/contents/assessment-and-management-of-auricular-hematoma-and-cauliflower-ear.
38. Dimeff RJ, Hough DO. Preventing cauliflower ear with a modified tie-through technique. Phys Sports Med. 1989;17(3):169–73.
39. Eichel BS, Bray DA. Management of hematoma of the wrestler's ear. Phys Sports Med. 1978;6(1):87–8.
40. Schuller DE, Dankle SK, et al. Auricular injury and the use of headgear in wrestlers. Arch Otolaryngol Head Neck Surg. 1989;115(6):714–7.
41. Kelleher JC, Sullivan JG, et al. The wrestler's ear. Plast Reconstr Surg. 1967;40:540–6.
42. Agarwal S. Ekta Mann. Knee Injuries in wrestlers: a prospective study from the Indian subcontinent. Asian J Sports Med. 2016;7(4):e35000.
43. Agel J, Ransone J, Dick R, Oppliger R, Marshall SW. Descriptive epidemiology of collegiate men's wrestling injuries: National Collegiate Athletic Association Injury Surveillance System, 1988–1989 through 2003–2004. J Athl Train. 2007;42(2):303–10.
44. Wroble RR, Mysnyk MC, Foster DT, Albright JP. Patterns of knee injuries in wrestling: a six year study. Am J Sports Med. 1986;14(1):55–66.
45. Snook GA. Injuries in intercollegiate wrestling. Am J Sports Med. 1982;10(3):142–4.
46. Baker BE, Peckham AC, Pupparo F, Sanborn JC. Review of meniscal injury and associated sports. Am J Sports Med. 1985;13(1):1–4.
47. Mysnyk MC, Wroble RR, Foster DT, Albright JP. Prepatellar bursitis in wrestlers. Am J Sports Med. 1986;14(1):46–54.
48. Roland GC, Beagley MJ, Cawley PW. Conservative treatment of inflamed knee bursae. Phys Sportsmed. 1992;20(2):66–77.
49. Otero JE, Graves CM, Bollier MJ. Injuries in collegiate wrestlers at an elite division I NCAA Wrestling program: an epidemiological study. Iowa Orthop J. 2017;65-70(56):37.
50. Berson BL. Surgical repair of pectoralis major rupture in an athlete: case report of an unusual injury in a wrestler. Am J Sports Med. 1979;7(6):348–51.
51. Bak K, Cameron EA, Henderson IJP. Rupture of the pectoralis major: a meta-analysis of 112 cases. Knee Surg Sports Traumatol Arthrosc. 2000;8(2):113–9.
52. Pavlik A, Csépai D, Berkes I. Surgical treatment of pectoralis major rupture in athletes. Knee Surg Sports Traumatol Arthrosc. 1998;6(2):129–33.
53. Halloran L. Wrestling injuries. Orthop Nurs. 2008;27(3):189–92.
54. Bruce SL, Wsielewski N, Hawke RL. Cubital tunnel syndrome in a collegiate wrestler: a case report. J Athl Train. 1997;32(2):151–4.
55. Rossi F, Dragoni F. Lumbar spondylolysis: occurrence in competitive athletes. Updated achievements in a series of 390 cases. J Sports Med Phys Fitness. 1990;30:450–2.
56. Iwai K, Nakazato K, et al. Physical characteristics of university wrestlers with low back pain. Jpn J Phys Fitness Sports Med. 2002;51:423–36.
57. Wallace WA, Wroble RR, Maffulli N, Kordi R. Combat sports medicine. London: Springer; 2009.
58. Mueller FO, Cantu RC. Catastrophic injuries and fatalities in high school sports, fall 1982-spring 1988. Med Sci Sports. 1990;22(6):737–41.
59. Kordi R, Ziaee V, Rostami M, Wallace WA. Indirect catastrophic injuries in olympic styles of wrestling in Iran. Sports Health. 2011;3(1):29–31.
60. Thomas RE, Zamanpour K. Injuries in Wrestling: systematic review. Physician and Sportsmedicine. 2018;46(2):168–96.
61. Mysnyk MC, Albright JP. Relative risk and long-term impact of injuries from amateur football and wrestling

competition. In: Hawkeye sports medicine symposium. Iowa City: Iowa; 1988.
62. Horswill CA. Applied physiology of amateur wrestling. Sports Med. 1992;14:114–43.
63. Wroble RR, Moxley DP. Weight loss patterns and success rates in high school wrestlers. Med Sci Sports Exerc. 1998;30:625–8.
64. McGuine TA. Injury frequency during a one-day collegiate wrestling tournament. Athl Train. 1989;24:227–9.
65. Jarret GJ, Orwin JF. Dick RW .Injuries in collegiate wrestling. Am J Sports Med. 1998;26(5):674–80.
66. Requa R, Garrick JG. Injuries in interscholastic wrestling. Phys Sports Med. 1981;9(4):44–51.
67. Patacsil J. An analytical survey of the incidents of injuries sustained in intercollegiate and interscholastic wrestling. Thesis, Purdue University, 1955.

Part III

Special Aspects in Sports Injuries

34 Mental Health Concerns in Athletes

Margot Putukian

34.1 Introduction

Participation in competitive sport can be associated with several benefits including general health and wellness, as well as the development of skillsets such as goal setting, teamwork, and resilience. In addition to the risks for injury, there are mental health (MH) concerns that occur in sport that are important to recognize and address. Several recent publications have addressed MH concerns in sport [1–7]. Whether certain MH issues are more or less common in our athletic population is unclear. Certain issues such as the psychological response to injury and the issues related to transition out of sport are unique stressors for elite athletes [1, 2, 6, 8–16]. In addition, some issues such as sexual abuse, hazing and bullying, post-traumatic stress related to concussion or trauma and eating disorders (ED) may be more common in the sport setting [1–3, 5, 6, 17]. Minimal data is available for paralympic athletes [7, 11].

The MH concerns that are frequently seen in the athletes include anxiety/stress, depression and suicide, overtraining, disordered eating/eating concerns, response to injury, sleep disorders, Attention Deficit/Hyperactivity Disorder ADHD, substance use, and substance abuse [18–22]. For the purposes of this chapter, elite athlete will include those competing at professional, Olympic, or collegiate levels. The reader is directed to other comprehensive reviews in terms of personality issues, hazing/bullying, sexual misconduct, as well as ADHD, some of which include specific treatment guidelines [1–3, 5, 6, 23–25]. This chapter will not address the complexity of MH issues in their entirety but instead will focus on select issues including sleep disorders, anxiety and stress, depression and suicide, overtraining, substance use/abuse, and the psychological response ton injury. The purpose of this chapter is to briefly review select mental health concerns commonly encountered in athletes, with a focus on what might be unique in their presentation, and how the medical staff can incorporate early recognition strategies to support the health and wellness of elite athletes.

34.2 Epidemiology of Mental Health (MH) Concerns

There is limited literature regarding the prevalence of MH concerns in athletes compared to the general population, with methodological limitations (e.g., different definitions of both "athlete," lack of comparison groups) as well as in differentiating reported MH symptoms from the

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physician-diagnosed MH disorders. There are several tools that screen for MH symptoms, such as the Generalized Anxiety Disorder (GAD) screen for anxiety [26], the Patient Health Questionnaire [27] for depression, and the Alcohol Use Disorders Identification Test-Concise (AUDIT-C) [28] for alcohol. These screens ask about symptoms over a short period of time and are vastly different than the criteria used to make clinical diagnoses, such as those in the Diagnostic and Statistical Manual (DSM) of mental disorders [29] or the World Health Organization [18]. Among University aged athletes, the prevalence of depression and ED has been reported to occur in 10–25% [30–35]. In elite sport, anywhere from 5% to 45% of male athletes participating in team sports suffer from burnout and alcohol misuse as well as anxiety and depression [36–45]. MH disorders, including ED, have also been reported in female athletes [3, 46–50]. Whether participation in sport is protective against the development of MH concerns, or whether there are issues that are more likely seen in elite athletes compared to their nonathletic peers, due to the stressors or risk factors as well as the psychological response to injury has been reviewed [1–3, 5, 14–16, 38, 51, 52]. For different MH considerations, there may be different considerations that either protect athletes or put them at particular risk.

34.3 Athletic Culture and Personality Factors

There are environmental and athletic culture factors that can also impact MH, including hazing and bullying, sexuality and gender issues, sexual misconduct, and transition out of sport, and these are discussed in greater detail in other reviews [1, 3, 5, 23]. Similar to the impact on MH disorders themselves, the culture of the sporting environment can be either supportive or negative. Some individual personality traits may enhance athletic performance and potentially be protective against the development of MH symptoms, yet other traits may actually increase the risk for developing MH symptoms [53–58]. The personality traits evaluated most frequently in relationship to MH issues include conscientiousness, autonomy, perfectionism, and athlete identity [54, 55, 59–63]. Some of these will be discussed in the individual sections below. Finally, though the landscape is changing, recognizing that MH issues are common in athletes and team physicians and other medical providers must understand that there may be barriers in seeking care that are unique to elite athletes. Including screening tools during pre-participation physicals and physician visits, especially in situations where an athlete is injured, out of sport, or considering retirement is essential in both normalizing health-seeking behaviors as well as for early recognition and management of MH concerns when they occur.

34.4 Depression and Suicide

The DSM-5 criteria for depression are provided in Table 34.1 [29]. There are reports of depression in athletes that range anywhere from 4% [64] to 68% [42]. Though the data is limited, the prevalence of depression in athletes appears to be similar to that of the general population [34, 65–68]. At the collegiate level, 23.7% reported depressive symptoms over a 5-year period [34] similar to that reported in their nonathletic peers. In the general population females tend to report symptoms more often than males, and in a study of athletes, female athletes appear to have a two-fold risk for reporting symptoms of depression compared to their male counterparts, and within a collegiate cohort, freshman are more likely to report symptoms of depression when compared to upperclassmen [35]. A recent study of female German football (soccer) players reported symptoms of depression similar to the general population, and mild to moderate and severe symptoms using the Center for Epidemiological Studies Depression Scale (CES-D) of 16.6% and 14.1%, respectively [66].

There have been additional links to depression in individuals that have low self-esteem, as well as those with marijuana use and those with body image issues [59, 69, 70]. In addition, there has

Table 34.1 DSM-5 diagnostic criteria for a major depressive episode [29]

Diagnosis: at least five[a] of the following symptoms for at least 2 weeks[b] (Symptoms cannot be explained by another medical condition, medication or substance, or another MH disorder)	
Symptoms	Comments
• Depressed mood • Decreased interest or pleasure in most activities, for the majority of each day (anhedonia) • Significant change in weight or appetite • Sleep disturbance (insomnia or hypersomnia) • Fatigue or loss of energy • Change in activity: Agitation or psychomotor retardation • Feelings of worthlessness or excessive or inappropriate guilt • Diminished ability to concentrate, focus, or think; indecisiveness • Recurrent thoughts of death or suicide, with or without a plan for committing suicide	• In children may see irritability, or feeling sad or empty, or report by others that they appear tearful or other observations • In children, may see failure to achieve expected weight gain

[a]MUST include either depressed mood or decreased interest or pleasure in doing things

[b]The symptoms cause significant distress or impairment in social, occupational, or other important areas of functioning

been a link between depression symptoms and self-report of alcohol abuse [71]. In addition, not surprisingly, depression in athletes has been linked to the psychological response to injury [8, 19, 71–80].

There appear to be specific risk factors for depression unique to athletes. In one study, four specific risk factors were identified: (1) sustaining a significant time loss injury, (2) the increased time demands of sport, (3) performance expectations from themselves or others, and (4) an identity linked to athletic performance [81]. In other reports, athletes in individual sports such as diving or track and field reported a higher rate of clinical depression than those athletes participating in team sports [34, 64]. For elite athletes, a unique stressor can be transition from sport and retirement, especially if the athlete is dissatisfied with their career [20, 39, 82]. In one study of male rugby players, dissatisfaction with their career was associated with more distress, sleep disturbance, use of nicotine, and adverse nutrition [39]. Athletes considering retirement report higher rates of depressive symptoms [36], and in one study 20% of athletes terminated from their team to force retirement reported significant traumatic stress 3 and 8 months later [83]. Depression in athletes may also present slightly differently than in nonathletes, with a "lack of focus" as well as anger, insomnia, hypersomnia, fatigue, or a change from prior level of function or behavior at home or school as common signs of symptoms of depression in athletes [1]. Being aware of these atypical presentations in athletes is important for early recognition of depression.

Suicide accounted for 7.3% of the deaths in college-aged athletes in the USA [84], with male American football players at increased risk. The rate of suicide in this study was 0.93/100,000 athletes per year, which compares favorably to the rate of suicide in the general population of the same age in the USA at that time of 11.6/100,000 per year [84]. The risk factors for suicide are multifactorial, including history of childhood trauma, agitation, impulsivity, anxiety, aggression, hopelessness, conflict physical injury/illness, sleep disturbance, drug and alcohol use, and prior suicide attempts [5, 77, 84–89].

Screening tools for depression and suicide validated in a nonathlete population include the Beck Depression Inventory (BDI), Centers for Epidemiological Studies Depression (CES-D), the Patient Health Questionnaire 2 and 9 (HQ-2 and PHQ-9) [27, 90], and in athletes, the Baron Depression Screener for Athletes (BDSA) [91] has been shown to be effective [91, 92] (see Table 34.2). There can be barriers for athletes to seek help for MH issues and in particular depres-

Table 34.2 Baron Depression Screener for Athletes (BDSA) (modified from [91])

Baron depression screener for athletes
Self-report survey; How an athlete has felt over the past 2 weeks.
Scoring: 0 for never, 1 for some of the time, and 2 for most of the time.
1) I feel sad even after a good practice session or successful competition.
2) I rarely get pleasure from competing anymore and have lost interest in my sport.
3) I get little or no pleasure from my athletic successes.
4) I am having problems with my appetite and weight.
5) I do not feel rested and refreshed when I wake up.
6) I am having problem maintaining my focus and concentration during training and competition.
7) I feel like a failure as an athlete and person.
8) I cannot stop thinking about being a failure and quitting sports.
9) I am drinking alcohol or taking supplements to improve my mood.
10) I have thoughts of ending my life.
A score of >5 indicates the need for referral to a mental health-care professional.

sion, as they may not aware that they are depressed or acknowledge these feelings. In addition to these obstacles, the stigma of MH can make it challenging for athletes to seek help [16, 93, 94]. The unique challenges to recognizing and providing MH services to elite athletes will be discussed in more detail in the following.

34.5 Overtraining

It can be challenging to differentiate major depressive disorder from overtraining, which occurs when training loads are excessive, recovery periods are insufficient, and decreases in performance as well as depressed mood or other mood disturbances occur [95–97]. Overtraining syndrome does not have any specific diagnostic criteria and has been considered an extreme form of non-functional overtraining (NFO), lasting typically longer than 2 months and with associated psychological and/or neuroendocrinological features [97–99]. It has been described as occurring in anywhere from 10 to 64% of elite athletes [97], and an increased volume of training with inadequate recovery is clearly contributive [100–102]. One study of swimmers over 11 years demonstrated an increase in training correlating with decreased mood which improved after the training load decreased [103]. Several features of overtraining overlap with depression including weight loss, insomnia, decreased concentration, and fatigue [104]. Along with depressed mood, other risk factors that have been described include other psychosocial stressors (e.g., coach, team, school, work), poor nutrition, illness, sleep disturbances, and illness [97, 102]. Though there has been a lot of work to determine a diagnostic approach to overtraining, including measuring heart rate, hormones, or other catecholamines, defining and treating OTS remain elusive [96, 100].

Several measures have been used to screen for the psychological response that occurs with OTS including the Recovery Stress Questionnaire for Athletes (REST-Q sport) [105], and the Training Distress Scale [106] appears to be the most useful, with the Profile of Mood State (POMS) being an earlier tool used that is nonspecific to athletes [107]. The utility of these scales in evaluating OTS in athletes has been reviewed [97].

34.6 Anxiety/Stress-Related Disorders

Generalized Anxiety Disorder (GAD) is defined by the DSM-5 criteria [29] are listed in Table 34.3. The symptoms of anxiety cause distress and impair function and cannot be explained by other medical conditions. GAD and the associated anxiety disorders of social anxiety disorder, obsessive-compulsive disorder, and panic disorder (see Table 34.4) are common in athletes [108]. In elite athletes the reported range of GAD from 6% to 14.6% is confirmed by a physician [64] or by self-report [41, 66, 68]. The incidence of GAD in athletes appears to be similar to that seen in the general population [66, 109, 110], and as in the general population more common in female athletes compared to their male counterparts [30, 35, 48, 75, 111, 112]. The incidence of

Table 34.3 DSM-5 diagnostic criteria for generalized anxiety disorder [29]

Diagnosis: Excessive anxiety and worry about a number of events or activities, with the individual finding it difficult to control the worry. Occurs most days for at least 6 months At least three additional symptoms must be present	
Additional symptoms	Comments
• Restlessness or feeling keyed up or on edge • Easily fatigued • Irritability • Impaired concentration, "mind going blank" • Difficulty sleeping (difficulty falling or staying asleep, or restless, unsatisfying sleep) • Increased muscle aches or soreness	
The anxiety, worry, or physical symptoms cause significant distress or impairment in important areas of functioning Symptoms are not better explained by another medical condition, including another mental disorder, or by a substance	

Table 34.4 DSM-5 diagnostic criteria for selected other anxiety and related disorders [29]

Panic disorder	Social anxiety disorder	Obsessive-compulsive disorder
• Recurrent unexpected panic attacks; abrupt surges of intense fear or discomfort that reach a peak within minutes, and during which several accompanying symptoms occur, including "impending doom"	• Marked fear or anxiety about 1 or more social situations in which the individual is exposed to possible scrutiny by others	• Presence of obsessions, compulsions, or both: – Obsessions: Recurrent and persistent thoughts, urges, or impulses that are intrusive and unwanted, and that typically cause marked anxiety or distress – Compulsions: Repetitive behaviors or mental acts that the individual feels driven to perform in response to an obsession or according to rules that must be rigidly applied
• At least 1 attack is followed by 1 month or more of persistent concern about additional panic attacks or their consequences, and/or a significant maladaptive change in behavior related to the attacks (e.g., avoidance of certain situations)	• The individual fears that they will act in a way or show anxiety symptoms that will be negatively evaluated	• Obsessions or compulsions are time-consuming or cause clinically significant distress or impairment in important areas of functioning
• Symptoms are not better explained by another medical condition, including another MH disorder, a medication or substance	• The social situations almost always provoke fear or anxiety • The fear or anxiety is out of proportion to the actual threat posed by the social situation and to the sociocultural context • Symptoms are persistent, typically lasting 6 months or more • The fear, anxiety, or avoidance causes significant distress or impairment in important areas of functioning • Symptoms not better explained by another medical condition, including another MH disorder, or a medication or substance	• Symptoms are not better explained by another medical condition, including another MH disorder, a medication or substance

panic disorder has been noted to be 2.2% in the general population and in elite French athletes, the lifetime prevalence of panic disorder has been noted to be 2.8% [64]. Stress-related disorders include post-traumatic stress disorder (PTSD) as well as acute stress disorder, with the latter being differentiated from PTSD in that the symptoms are less than 1 month. The other stress-related disorder that is reported in athletes includes performance anxiety [60, 69].

Common features for athletes at risk for anxiety and stress-related disorders are similar and include perfectionism [53, 113], low self-esteem [56, 58], and injury/fear of re-injury [38, 41, 72, 114–118]. Other athlete-specific risks include competition and the fear of failure, social judgment, perceived negative consequences [1]. In addition, in current as well as former elite football (soccer) players, history of injury as well as prior surgery, recent life events, lower levels of social support, and career dissatisfaction have been associated with anxiety [38, 82].

The Generalized Anxiety Disorder-7 (GAD-7) is a validated screening tool that has been used to recognize anxiety [26]. Though not specific to athletes, it can be very useful in evaluating anxiety in this population [64, 66, 68]. Anxiety has been shown to negatively affect performance [119, 120] and has been associated with impaired cognitive performance and overall function [1, 121, 122]. Athletes that struggle with anxiety can often end up in a vicious cycle where their performance struggles, they start to worry more such that their cognitive activity starts to fail and their ability to engage and perform continues to decrease [1].

34.7 Sleep Disorders

There has been an increased amount of literature regarding sleep disorders in athletes and their relationship with MH concerns [1, 5]. Less than 7 hours of sleep is considered insufficient for a healthy adult [123, 124], and an increased need for sleep (e.g., 9–10 hours) has been recommended for adolescents and young adults [123, 125]. Poor sleep quality has been associated with an increased risk (2.4 times) for depression, anxiety, and somatoform disorders in collegiate athletes [69], and also associated with daytime sleepiness and poor-quality sleep in 50–83% of competitive athletes [37, 126]. At the collegiate level in the USA, 50% of athletes self-report getting less than 7 hours of sleep when they are in season, and overall 79% report getting 8 hours or less of sleep each night [127]. At the Olympic level, the numbers are similar, with 49% of Olympic athletes self-reporting as “poor sleepers,” especially the night before competition [37, 128]. Poor quality of sleep has been reported in athletes with disabilities during the 2008 Paralympic Games [129]. In athletes with insomnia, defined as persistent difficulty initiating or maintaining sleep at least three times per week for at least 3 months, difficulty falling asleep and waking up in the middle of the night were reported more frequently in females compared to their male counterparts [64]. Insomnia symptoms and poor sleep quality have been associated with depression, irritability, and a lack of concentration [129]. Sleep dysfunction is a common comorbidity with several other MH concerns including hazing and bullying, depression, anxiety, bipolar disorder, overtraining, and ED [1, 5, 130–132].

Obstructive sleep apnea has been linked to mood disorders as well as cognitive impairment and has been described in the athlete population most commonly with American football and other sports with an increased body mass index [126, 133, 134]. In American football, symptoms consistent with sleep apnea are reported in 8% of college players [135], and at the professional level the reported rates of sleep apnea as 5–11 times that seen in the general population [136].

In several studies, sleep dysfunction has been noted to have an adverse effect on performance in several sports including soccer, golf, tennis, and basketball [137–143]. The disruption to circadian rhythms and in particular the effect of traveling (e.g., jet lag) and home advantage on sports performance has been discussed previously [128, 142]. The effect of early morning practices on performance suggests that these should be minimized [144, 145]. There is evidence to support the concepts of sleep education, sleep optimization and nutrition in improving sleep function, self-reported vigor and decreased fatigue as well as improved overall mood and athletic performance [78, 138, 146, 147].

There are tools to screen for sleep dysfunction, including the Insomnia Severity Index and the Stop-BANG Questionnaire, which are reliable and validated in the general population [148, 149], and the Athlete Sleep Screening Questionnaire (ASSQ), which is a validated athlete-specific screening tool [150]. Given the impact on performance, the early recognition of sleep dysfunction in elite athletes is important. The treatment of sleep dysfunction should be individualized, and several options exist including behavioral interventions [21, 98, 151–153]. A comprehensive review of treatment options in athletes is provided elsewhere [1, 5, 141, 154].

34.8 Disordered Eating (DE)/ Eating Disorders (ED)

DE occurs on a spectrum, with at the mildest energy restriction and pathogenic weight control behaviors and at the most severe meeting the criteria of ED defined in the DSM-5 as anorexia nervosa (AN), bulimia nervosa (BN), binge eating disorder (BED), and other specified eating or feeding disorders [29]. A pre-occupation with weight and experimentation with pathogenic behaviors such as the use of diet pills, laxatives, excessive exercise, and self-induced vomiting should be differentiated from the diagnosis of ED which should be made by a MH care provider or a primary care physician with competencies in MH [3]. The hallmark of all ED is a distorted body image [29]. Common signs, symptoms, consequences, risk factors, and effects on performance of DE and DE are provided in Table 34.5. Comorbidities with abuse or depression are common [1, 3, 17]. Across all age groups and genders, athletes have an increased prevalence of both DE and ED compared to their nonathlete counterparts [22, 155]. In the sports medicine literature, the conditions discussed that present the spectrum of DE to ED are the Female Athlete Triad and Relative Energy Deficiency in Sport (RED-S) [17, 156]. The Female Athlete Triad, initially described in 1997 and researched for several decades, is described as the relationship between energy availability, menstrual dysfunction, and bone health [156]. RED-S has been described more recently to describe similar pathophysiology, yet includes male athletes, and relates the impact of energy deficiency on not only bone health and endocrine function but also metabolic rate, immunity, protein synthesis, and cardiovascular health [17]. Risk factors for DE/ED include participation in weight-restricted and esthetic sports, female gender, and athlete-specific risk factors including performance pressure, team weigh-ins, and injury [157]. Other risk factors associated with an increased incidence of DE/ED include perfectionism, a history of hazing, sexual abuse, and as a response to injury [6, 7, 13, 71, 92, 145, 158–162].

Effective screening tools to evaluate for the presence of DE/ED have been developed and validated with many specific to athletes, including the Brief ED in Athletes Questionnaire (BEDA-Q), the Athletic Milieu Direct Questionnaire (AMDQ), the Compulsive Exercise Test-Athlete Version (CET-A), the Female Athlete Screening Tool (FAST), and the Psychologic Screening Test (PST) [163, 164]. These tools were developed using the DSM-4 criteria and may not be applicable to male athletes and/or incorporate the RED-S criteria. Screening measures that are not athlete-specific but are validated and can be applied to males and females include the SCOFF [165] and the Eating Disorder Examination interview [166]. Algorithms for care and risk stratification for athletes with ED are presented in Figs. 34.1 and 34.2. Treatment decisions must be individualized and take into account current medical issues, risk factors, sport, coexisting, and preexisting medical issues [2, 17, 156]. The Team Physician has the ultimate responsibility for making the return to play decision [167, 168]. Having a multi-disciplinary treatment team that includes a physician, athletic trainer/physiotherapist, psychologist, psychiatrist, or other MH care provider and dietitian can be very useful in the management of the spectrum of disordered eating and ED that can occur in elite athletes [1, 5, 17, 156].

Table 34.5 Common presentation of eating disorders/disordered eating in elite athletes (modified from [80])

Symptoms	Signs	Consequences	Risk factors	Performance issues
Distorted body image ("feeling fat" despite being thin)	Pre-occupation with weight	Electrolyte abnormalities as a result of purging (e.g., vomiting, laxatives, diuretics)	Perfectionism, high achievers	Energy deprivation leads to negative effects on VO2 max, and running speed
	Excessive exercise	Abnormal bone health (e.g., low bone mineral density, premature osteoporosis)	Pressure to lose weight	Dehydration and malnutrition as a result of decreased energy intake and/or pathogenic behaviors can lead to decreased performance
Anxiety or depression	Dehydration	Stomach ulcers, pancreatitis, gastric rupture	Revealing uniforms and subjective rating of performance (e.g., skating, gymnastics, swimming)	Intense dieting can negatively affect VO2 max and running speed.
Difficulty concentrating	Menstrual dysfunction			
Abdominal pain	Stress fractures			
Cold intolerance	Significant weight loss			
Muscle cramps, weakness, or fatigue	Dental or gum problems			
	Excessive use of restroom			
	Avoidance of eating and eating situations			

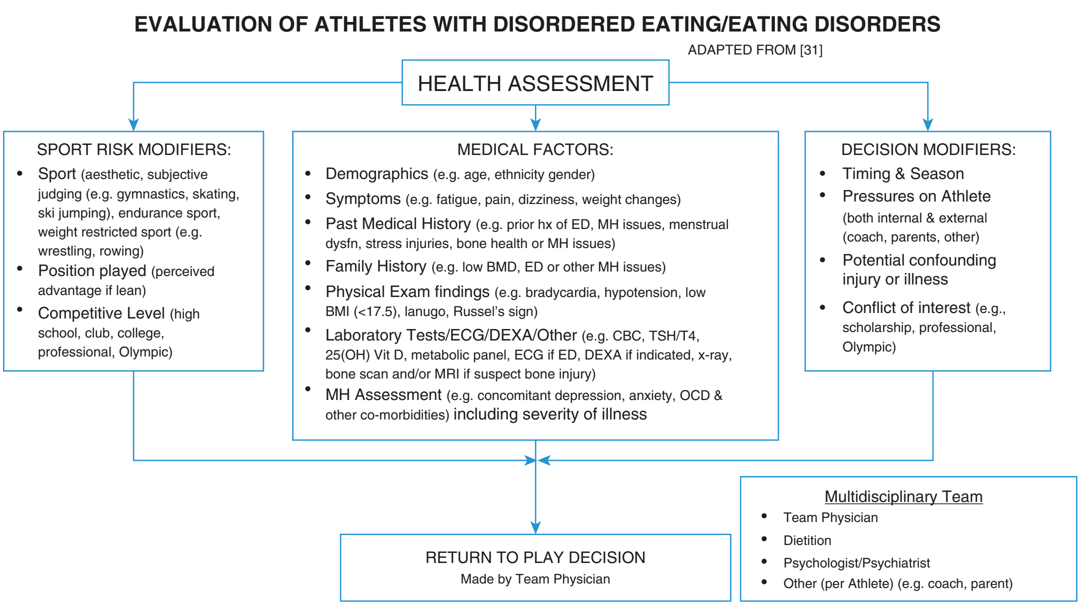

Fig. 34.1 Evaluation of athletes with disordered eating/eating disorders. Adapted from [31]. Key: *ED* eating disorders, *MH* mental health, *dysfn* dysfunction, *BMD* bone mineral density, *DEXA* dual energy X-ray absorptiometry, *CBC* complete blood count, *TSH/T4* thyroid stimulating hormone/Thyroxine, *25(OH) Vit D* 25-hydroxyvitamin D, *MRI* magnetic resonance imaging, *OCD* obsessive-compulsive disorder

RISK ASSESSMENT FOR PARTICIPATION ADAPTED FROM [122]

Low Risk Individuals

- Energy availability & expenditure matched, sustain healthy growh & development, response to training
- Healthy eating and training habits
- Normal metabolic and hormonal function
- Healthy Bone Mineral Density (BMD) (based on age, gender, ethnicity)
- No musculoskeletal issues

Moderate Risk Individuals

- Energy availability below expenditure, attenuation of expected growth & development (adolescent), and inadequate response to training
- Abnormally low % body fat for prolonged period of time
- Substantial weight loss (5–10% body mass in 1 month)
- Abnormal menstrual cycle in female (e.g. amenorrhea, oligomenorrhea) as well as delayed menarche (>16 years) or abnormal hormonal profile in male
- Reduced BMD (Z score < –1 SD), or decrease in BMD from prior measurement
- One or more stress fractures, not felt to be related to training errors
- Lab or other (Electrocardiogram) abnormalities, or confounding MH disorders, including Disordered Eating

High Risk Individuals

- Energy availability well below expenditure, associated with extreme weight loss and serious medical consequences
- Anorexia Nervosa and other serious Eating Disorders [1]
- Dehydration, hemodynamic instability, arrhythmias, electrolyte disturbances related to extreme weight loss techniques

Fig. 34.2 Risk assessment for participation. Adapted from [122]

34.9 Substance Use Disorders

Substance use in athletes and in particular elite athletes is a significant concern, with several overlapping conditions and MH disorders. Substance use occurs as a spectrum from casual use, to abuse, to the DSM-5 Substance Abuse Disorders, and the most common substances include alcohol, caffeine, cannabis/cannabinoids, nicotine, opioids, and anabolic-androgenic steroids [5, 6, 169–175]. Alcohol remains a significant issue, cannabis/cannabinoid use has increased, and the use of opioids has sparked significant concerns, especially in the United States (USA) where opioid use is considered “a crisis” [125, 176]. In the USA, at the collegiate level 23% of elite athletes reported using a prescription pain medication in the past year and another 6% reported use without a prescription, a pattern also seen in adolescent athletes [158, 173, 175, 177, 178]. Whether the use and misuse of substances is less or more common in athletes is controversial, and varies based on the substance, sport, gender, country, sexual orientation, and ethnicity [1, 5, 6, 116, 127, 159, 169–172, 174, 177, 179–186]. For a complete review of this topic, the reader is referred elsewhere [1, 5, 6, 170, 172, 186, 187].

Substance use is commonly associated with hazing, bullying, transition out of sport as well as sexual abuse [1, 23]. In addition, substance use symptoms or disorders are commonly listed as part of the differential diagnosis for several MH disorders including generalized anxiety, obsessive-compulsive disorder, social anxiety disorder, post-traumatic stress disorder, bipolar disorder, depression, ED, ADHD/LD, and Sport-Related Concussion (SRC) [6, 160, 174, 186, 188]. The use and misuse of substances is a common comorbidity of several MH concerns, with their use as a means to relieve stress, handle emotions or cravings, manage pain, or in some situations in an attempt to increase performance [1, 3, 5, 171, 173]. A common trigger for substance use and misuse is as a response to trauma and injury [5, 181, 188, 189], and not unexpectedly there are often several comorbidities as a response to trauma and injury including depression, anxiety, ED, and poor coping behaviors.

34.10 Psychological Response to Injury

Injury is a risk factor for several MH conditions including substance use, disordered gambling, depression and suicide, trauma-related disorders, anxiety, overtraining, ED, as well as issues related to transition/retirement from sport [6, 32, 35, 42, 72, 73, 81, 86, 114, 118, 190, 191]. In addition, injury and illness can also cause several MH symptoms. The interface between injury, MH, and performance in elite athlete is complex, and it is an area of interest [2, 3, 6, 15, 16]. There may be stressors specific to participation in elite sport that increases the risk for injury or illness that includes MH disorders [8, 19, 74, 79, 192]. The psychological response to injury can also potentially unmask underlying MH disorders and complicate recovery from injury [8, 12, 74, 75, 79, 85, 176, 192]. A higher level of symptoms of both depression and anxiety are reported in injured athletes compared to their noninjured peers [41]. Lower injury rates have been reported in athletes that have resiliency and "mental toughness," with lower rates of depression, stress, anxiety, and obsessive-compulsive symptoms [32, 42].

Risk factors for injury include psychological and sociocultural factors [79, 80, 193]. Psychological risk factors include anxiety/worry, perfectionism, hypervigilance, poor body image or low self-esteem, limited coping resources, life event stress (e.g., stress associated with major life event stressors such as the death of a family member or starting at a new school), risk-taking behaviors, or low mood state [8, 19, 74, 75, 79, 192]. Sociocultural risk factors include a lifetime history of sexual or physical abuse, social pressures, limited social resources, organizational stress (i.e., how the athlete feels about the structure and function of their team), stress related to a negative self-assessment of their academic or athletic performance, coaching quality (how they perceive their relationship with their coach), and the culture of their sport/team (e.g., the mindset of the team; win at all costs versus continued team growth) [2, 79, 194, 195].

Three athlete "responses" identified in a systematic review that are most important in determining outcome in sport injury and rehabilitation include the cognitive response, the emotional response, and the resultant behavioral response [193]. The cognitive response is how the athlete understands their injury or illness. The emotional response can include sadness, anger, frustration, changes in sleep or appetite, and lack of motivation. The cognitive response leads to an emotional response, and both can be either "normal" or "problematic" [2], and both can affect behavioral responses such as goal setting, motivation, and compliance with treatment [193]. Improved injury recovery has been shown in athletes that have more positive cognitive emotional and behavioral responses to injury including higher levels of optimism and self-efficacy [8–10, 196]. Useful strategies that can aid in recovery include (1) using modeling techniques (e.g., videos, peer athletes) to decrease fear of re-injury, (2) support athlete autonomy (e.g., explain purpose for rehabilitative exercises), (3) increase confidence (e.g., goal setting, functional tests), (4) provide social support, (5) find a role for the athlete within their sport (e.g., take stats, help coach), and (6) stress inoculation training (if the injury requires surgery to avoid the need for pain medication) [13, 197].

One of the most common injuries that is particularly challenging is SRC [160], which requires excluding substance use when diagnosed [160], and has several symptoms listed in its assessment tool [198] which overlap with symptoms of MH disorders [199, 200]. There is minimal data that supports an increase in substance use after concussion, though there is data to suggest that athletes with SRC may develop MH symptoms and that athletes may use substances in order to cope with these emotions [171, 186]. Symptoms of depression, anxiety, and impulsivity are commonly noted in male collegiate athletes after SRC [174]. In one study of collegiate athletes, 20% reported symptoms of depression after SRC, with predictors for reporting depression symptoms including baseline reporting of depression symptoms as well as ethnicity (non-white), the number of games missed after injury (the more games, the more symptoms), and the number of years they had been involved in sport (more symptoms if fewer number of years in sport) [200]. In addition, the development of MH symptoms, preexisting history of MH or mood disorders or family history for MH disorders, and high-stress life events have all been related to a longer recovery time after SRC in most studies [73, 76, 161, 191, 201, 202]. MH symptoms, and fear of re-injury is commonly cited after anterior cruciate ligament reconstruction [9, 10, 42, 196, 203, 204], and is also associated with persistent symptoms after SRC [176].

The most recently discovered human Severe Acute Respiratory Syndrome (SARS) coronavirus is called COVID-19 [205], and in December 2019, COVID-19 created a worldwide pandemic that is ongoing. It has brought elite sports to a halt and postponed the upcoming summer Olympic Games. Though the literature regarding COVID-19 and its health impacts are still evolving, the psychological effects of COVID-19 are evident. In a survey of >37,000 college aged athletes, a majority reported high rates of mental distress, with more than 1 in 4 reported feeling sadness and a sense of loss and 1 in 12 reporting "feeling so depressed it has been hard to function" at least "constantly" or "most every day" [162]. 1 in 3 reported sleep dysfunction and 80% reported having barriers to training including fear of exposure to COVID-19 (43%), lack of motivation (40%), feeling of stress or anxiety (21%), and sadness or depression (13%) [162]. This demonstrates the need for providing MH resources and other areas where screening and recognition are most important.

34.11 Screening Tools

Throughout this chapter, several different screening tools have been discussed for MH disorders seen in elite athletes. As a follow-up to the IOC Consensus Statement on MH in elite athletes [5], a task force was developed to create the IOC Sports MH Assessment Tool (SMHAT) [206]. This Tool includes as its first triage component the Athlete Psychological Strain Questionnaire (APSQ). The APSQ was developed for and has been validated in elite athletes [207, 208]. If an athlete scores above specific cut-off points, additional screening tools such as the PHQ-9, GAD-7, and AUDIT-C can then be performed. Screening tools such as the APSQ and SMHAT are useful in identifying athletes that may be in significant psychological distress. Given that there are barriers to seeking MH care for elite athletes [7, 16, 94, 164, 209], utilizing screening tools during sports pre-participation physicals or during regular physician visits, especially in situations where athletes have sustained a season or career ending injury, where the return to participation status is unclear (e.g., SRC), or where there is any sense that the athlete may have underlying risk factors for MH disorders, should be considered. In addition, asking an athlete to complete a short questionnaire related to mental well-being during their sports physical examination underscores the importance of MH and supports health-seeking behaviors such that an athlete may feel more comfortable reaching out to their health-care provider to discuss a MH concern.

34.12 Paralympic Athletes

There is a paucity of literature focusing on MH issues for disabled athletes and very few on elite paralympic athletes [7]. There may be specific and unique stressors for elite athletes including chronic pain, overtraining, complex medical situations, stressors related to logistics (e.g., travel to competition sites) sleep conditions, equipment and technology issues, negative coaching behaviors (e.g., comments made that are demeaning), issues related to classification in disability categories, or retirement issues [7, 11, 129, 210, 211]. More prospective literature is needed to answer important questions related to the MH needs of disabled elite athletes.

Take-Home Messages

1. MH issues are common in athletes.
2. Team physicians and other medical providers must understand that there may be barriers in seeking care that are unique to elite athletes.
3. Screening tools can help normalize health-seeking behaviors and improve early recognition of MH concerns.

References

1. Chang C, Putukian M, Aerni G, et al. Mental health issues and psychological factors in athletes: detection, management, effect on performance, and prevention: American medical Society for Sports Medicine Position Statement – executive summary. Br J Sports Med. 2020;54:216–20.
2. Herring SA, Kibler WB, Putukian M, et al. Psychological issues related to illness and injury in the athletes and the team physician: a consensus statement – 2016 update. Med Sci Sports Exerc. 2017;49(5):1043–54.
3. Inter-association Consensus Document: Mental Health Best Practices. Understanding and Supporting Student-Athlete Mental Wellness. 2016. Updated 2020. https://ncaaorg.s3.amazonaws.com/ssi/mental/SSI_MentalHealthBestPractices.pdf. Accessed April 1, 2020.
4. Moesch K, Kentta G, Kleinert J, et al. FEPSAC position statement: mental health disorders in elite athletes and models of service provision. Psych of Sport & Exerc. 2018;38:61–71.
5. Reardon CL, Hainline B, Aron CM, et al. Mental health in elite athletes: International Olympic Committee consensus statement (2019). Br J Sports Med. 2019;53:667–99.
6. Rice SM, Purcell R, De Silva S, et al. The mental health of elite athletes: a narrative systematic review. Sports Med. 2016;46:1333–53.
7. Swartz L, Hunt X, Bantjes J, et al. Mental health symptoms and disorders in Paralympic athletes: a narrative review. Br J Sports Med. 2019;53:737–40.
8. 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;47:1120–6.
9. Gignac MA, Cao X, Ramanathan S, et al. 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 Science, Medicine, and Rehab. 2015;7:4. http://www.biomedcentral.com/2052-1847/7/4
10. Glazer DD. Development and preliminary validation of the injury-psychological readiness to return to sport (I-PRRS) scale. J Athl Train. 2009;44:185–9.
11. Guerrero M, Martin J. Para sport athletic identity from competition to retirement: a brief review and future research directions. Phys Med Rehabil Clin N Am. 2018;29:387–96.
12. Nippert AH, Smith AM. Psychological stress related to injury and impact on sport performance. Phys Med Rehabil Clin N Am. 2008;19(2):399–418.
13. Podlog L, Banham SM, Wadey R, et al. Psychological readiness to return to competitive sport following injury: a qualitative study. The Sport Psychologist. 2015;29:1–14.
14. Putukian M, Echemendia RJ. Psychological aspects of serious head injury in the competitive athlete. Clin Sports Med. 2003;22:617–30.
15. Putukian M. How being injured affects mental health. In: Brown GT, Hainline B, Kroshus E, Wilfert M, editors. Mind, body and sport: understanding and supporting student-athlete mental wellness. Indianapolis, IN: NCAA Press; 2014. p. 72–5.
16. Putukian M. The psychological response to injury in student athletes: a narrative review with a focus on mental health. Br J Sports Med. 2016;50:145–8.
17. Mountjoy M, Sundgot-Borgen J, Burke L, et al. 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.
18. World Health Organization. International Classification of Disease for mortality and morbidity statistic (ICD-11) 2018. Available: http://icd.who.int.
19. Ivarsson A, Johnson U, Lindwall M, et al. Psychosocial stress as a predictor of injury in elite junior soccer: a latent growth curve analysis. J Sci Med Sport. 2013:S1440-2440(13)00473-8.
20. Van Ramele S, Aoki H, Kerkhoffs G, et al. Mental health in retired professional football players: 12-month incidence, adverse life events and support. Psych of Sport and Exerc. 2017;28:85–90.
21. Wu JQ, Appleman ER, Salazar RD, et al. Cognitive behavioral therapy for insomnia comorbid with psychiatric and medical conditions: a meta-analysis. JAMA Intern Med. 2015;175:1461–72.
22. Herring SA, Kibler WB, Putukian M, et al. Female athlete issues for the team physician: a consensus statement – 2017 update. Med Sci Sports Exerc. 2018 May;50(5):1113–22.
23. Mountjoy M, Brackenridge C, Arrington M, et al. International Olympic Committee consensus statement: harassment and abuse (non-accidental violence) in sport. Br J Sports Med. 2016;50(17):1019–29.
24. Putukian M, Kreher JB, Coppel DB, et al. Attention deficit hyperactivity disorder and the athlete: an American medical Society for Sports Medicine Position Statement. Clin J Sports Med. 2011;21(5):392–401.

25. White RD, Harris GD, Gibson ME. Attention deficit hyperactivity disorder and athletes. Sports Health. 2014;6:149–56.
26. Spitzer R, Kroenke K, Williams JBW, et al. Arch Intern Med. 2006;166:1092–7.
27. Kroenke K, Spitzer RL, Williams JB. The PHQ-9. Validity of a brief depression severity measure. J Gen Intern Med. 2001;16(9):606–13.
28. Bush K, Kivlahan DR, McDonell MB, et al. The AUDIT alcohol consumption questions (AUDIT-C): an effective brief screening test for problem drinking. Ambulatory care quality improvement project (ACQUIP). Arch Intern Med. 1998;158(16):1789–95.
29. American Psychiatric Association. *Diagnostic and statistical manual of mental disorders* (DSM-5). Washington, DC: American Psychiatric Publishing; 2013.
30. Brand R, Wolff W, Hoyer J. Psychological symptoms and chronic mood in representative samples of elite student-athletes, deselected student-athletes and comparison students. School Ment Health. 2013;5:166–74.
31. Greenleaf C, Petrie TA, Carter J, et al. Female collegiate athletes: prevalence of eating disorders and disordered eating behaviors. J Am Coll Heal. 2009;57:489–95.
32. Proctor SL, Boan-Lenzo C. Prevalence of depressive symptoms in male intercollegiate student-athletes and nonathletes. J Clin Sport Psychol. 2010;4:204–20.
33. Rao AL, Hong ES. Understanding depression and suicide in college athletes: emerging concepts and future directions. Br J Sports Med. 2015;50:136.
34. Wolanin A, Hong E, Marks D, et al. Prevalence of clinically elevated depressive symptoms in college athletes and differences by gender and sport. Br J Sports Med. 2016;50:167–71.
35. Yang J, Peek-Asa C, Corlette JD, et al. Prevalence of and risk factors associated with symptoms of depression in competitive collegiate student athletes. Clin J Sport Med. 2007;17:481–7.
36. Beable S, Fulcher M, Lee AC, et al. SHARPSports mental health awareness research project: prevalence and risk factors of depressive symptoms and life stress in elite athletes. J Sci Med Sport. 2017;20:1047–52.
37. Drew M, Vlahovich N, Hughes D, et al. Prevalence of illness, poor mental health and sleep quality and low energy availability prior to the 2016 summer Olympic games. Br J Sports Med. 2017;2017:47–53.
38. Gouttebarge V, Backx FJ, Aoki H, et al. Symptoms of common mental disorders in professional football (soccer) across five European countries. J Sports Sci Med. 2015;14:811.
39. Gouttebarge V, Kerkhoffs G, Lambert M. Prevalence and determinants of symptoms of common mental disorders in retired professional Rugby union players. Eur J Sport Sci. 2016;16:595–602.
40. Gouttebarge V, Castaldelli-Maia JM, Gorczynski P, et al. Occurrence of mental health symptoms and disorders in current and former elite athletes: a systematic review and meta-analysis. Br J Sports Med. 2019;53:700–6.
41. Gulliver A, Griffiths KM, Mackinnon A, et al. The mental health of Australian elite athletes. J Sci Med Sport. 2015;18:255–61.
42. Hammond T, Gialloreto C, Kubas H, et al. The prevalence of failure-based depression among elite athletes. Clin J Sport Med. 2013;23:273–7.
43. Li H, Moreland JJ, Peek-Asa C, et al. Preseason anxiety and depressive symptoms and prospective injury risk in collegiate athletes. Am J Sports Med. 2017;45:2148–55.
44. Nixdorf I, Frank R, Hautzinger M, et al. Prevalence of depressive symptoms and correlating variables among German elite athletes. J Clin Sport Psychol. 2013;7:313–26.
45. Schuring N, Kerkhoffs G, Gray J, et al. The mental wellbeing of current and retired professional cricketers: an observational prospective cohort study. Phys Sportsmed. 2017;45:463–9.
46. Gerber M, Holsboer-Trachsler E, Pühse U, et al. Elite sport is not an additional source of distress for adolescents with high stress levels. Percept Mot Skills. 2011;112:581–99.
47. Hulley AJ, Hill AJ. Eating disorders and health in elite women distance runners. Int J Eat Disord. 2001;30:312–7.
48. Junge A, Feddermann-Demont N. Prevalence of depression and anxiety in top-level male and female football players. BMJ Open Sport Exerc Med. 2016;2:e000087.
49. Prinz B, Dvorak J, Junge A. Symptoms and risk factors of depression during and after the football career of elite female players. BMJ Open Sport Exerc Med. 2016;2:e000124.
50. Sundgot-Borgen J, Torstveit MK. Prevalence of eating disorders in elite athletes is higher than in the general population. Clin J Sport Med. 2004;14:25–32.
51. Gouttebarge V, Aoki H, Lambert M, et al. A history of concussions is associated with symptoms of common mental disorders in former male professional athletes across a range of sports. Phys Sportsmed. 2017;45:443–9.
52. Kilic O, Aoki H, Goedhart E, 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;26:946–54.
53. Haase AM, Prapavessis H, Owens RG. Perfectionism, social physique anxiety and disordered eating: a comparison of male and female elite athletes. Psych Sport and Exerc. 2002;2002:209–22.
54. Hendawy HMF, Awad EAA. Personality and personality disorders in athletes. In: Baron DA, Reardon CL, Baron SH, editors. Clinical sports psychiatry: an

international perspective. Oxford: Wiley-Blackwell; 2013. p. 53–64.
55. Nia ME, Besharat MA. Comparison of athletes' personality characteristics in individual and team sports. Procedia Soc and Behav Sci. 2010;5:808–12.
56. Samadzadeh M, Abbasi M, Shahbazzadegan B. Comparison of sensation seeking and self-esteem with mental health in professional and amateur athletes, and non- athletes. Procedia Soc Behav Sci. 2011;15:1942–50.
57. Shariati M, Bakhtiari S. Comparison of personality characteristics athlete and non-athlete student, Islamic Azad University of Ahvaz. Procedia Soc Behav Sci. 2011;30:2312–5.
58. Smith RE, Smoll FL, Cumming SP. Effects of a motivational climate intervention for coaches on young athletes' sport performance anxiety. J Sport Exerc Psych. 2007;29(1):39–59.
59. Brewer BW, Van Raalte JL, Linder DE. Athletic identity - Hercules muscles or Achilles heel? Int J Sport Psychol. 1993 Jan;24(2):237–54.
60. Frost RO, Marten P, Laharat C, et al. The dimensions of perfectionism. Cognit Ther Res. 1990;14(5):449–68.
61. Hill AP, Hall HK, Appleton PR, et al. Perfectionism and burnout in junior elite soccer players: the mediating influence of unconditional self-acceptance. Psychol Sport Exerc. 2009;9:630–44.
62. Linville PW. Self-complexity as a cognitive buffer against stress-related illness and depression. J Pers Soc Psychol. 1987;52(4):663–76.
63. Soenens B, Elliot J, Goossens L, et al. The intergenerational transmission of perfectionism: parents' psychological control as an intervening variable. J Fam Psychol. 2005;19(3):358–66.
64. Schaal K, Tafflet M, Nassif H, et al. Psychological balance in high level athletes: gender-based differences and sport-specific patterns. PLoS One. 2011;6:e19007.
65. Gorczynski PF, Coyle M, Gibson K. Depressive symptoms in high-performance athletes and non-athletes: a comparative meta-analysis. Br J Sports Med. 2017;51:1348–55.
66. Junge A, Prinz B. Depression and anxiety symptoms in 17 teams of female football players including 10 German first league teams. Br J Sports Med. 2019;53(8):471–7.
67. Sarac N, Sarac B, Pedroza A, et al. Epidemiology of mental health conditions in incoming division I collegiate athletes. Phys Sportsmed. 2018;46:242–8.
68. St Leger Dowse M, Whalley B, Waterman MK, et al. Diving and mental health: the potential benefits and risks from a survey of recreational scuba divers. Diving Hyperb Med. 2019;49(4):291–7.
69. Crisp A, Sedgwick P, Halek C, et al. Why may teenage girls persist in smoking*?* J Adolesc. 1999;22:657–72.
70. Stice E, Hayward C, Cameron RP, et al. Body-image and eating disturbances predict onset of depression among female adolescents: a longitudinal study. J Abnorm Psychol. 2000;109:*43844*.
71. Miller BE, Miller MN, Verhegge R, et al. Alcohol misuse among college athletes: self medication for psychiatric symptoms? J Drug Educ. 2002;32(1):41–52.
72. Armstrong S, Omen-Early J. Social connectedness, self-esteem, and depression symptomatology among collegiate athletes versus non-athletes. J Am Coll Heal. 2009;57(5):521–6.
73. Guskiewicz KM, Marshall SW, Bailes J, et al. Recurrent concussion and risk of depression in retired professional football players. Med Sci Sports Exerc. 2007;39(6):903–9.
74. Ivarsson A, Johnson U, Anderson MB, et al. Psychosocial factors and sport injuries: meta-analyses for prediction and prevention. Sports Med. 2017;47:353–65.
75. Ivarsson A, Johnson U, Podlog L. Psychological predictors of injury occurrence: a prospective investigation of professional Swedish soccer players. J Sport Rehabil. 2013;22(1):19–26.
76. Kerr ZY, Marshall SW, Harding HP, et al. Nine-year risk of depression diagnosis increases with increasing self-reported concussions in retired professional football players. Am J Sports Med. 2012;40(10):2206–12.
77. Smith AM, Milliner EK. Injured athletes and the risk of suicide. J Athl Train. 1994;29(4):337–41.
78. Van Ryswyk E, Weeks R, Bandick L, et al. A novel sleep optimisation programme to improve athletes' Well-being and performance. Eur J Sport Sci. 2017;17(2):144–51.
79. 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(Suppl 2):103–11.
80. Wiese-Bjornstal DM, Smith AM, Shaffer SM, et al. An integrated model of response to sport injury: psychological and sociological dynamics. J Appl Sport Psychol. 1998;10:46–69.
81. Wolanin A, Gross M, Hong E. Depression in athletes: prevalence and risk factors. Curr Sports Med Rep. 2015;14(1):56–60.
82. Gouttebarge V, Aoki H, Ekstrand J, et al. Are severe joint and muscle injuries related to symptoms of common mental disorders among male European professional footballers. Knee Surg Sports Traumatol Arthrosc. 2015;24(12):3934–42.
83. Wippert PM, Wippert J. Perceived stress and prevalence of traumatic stress symptoms following athletic career termination. J Clin Sport Psych. 2008;2:1016.
84. Rao AL, Asif IM, Drezner JA, et al. Suicide in National Collegiate Athletic Association (NCAA) athletes: a 9-year analysis of the NCAA resolutions database. Sports Health. 2015;7:452–7.
85. Iverson GL. Chronic traumatic encephalopathy and risk of suicide in former athletes. Br J Sports Med. 2014;48:162–4.

86. Lindqvist AS, Moberg T, Ehmborg C, et al. Increased mortality rate and suicide in Swedish former elite male athletes in power sports. Scand J Med Sci Sports. 2013:1–6.
87. Perlis ML, Grandner MA, Chakravorty S, et al. Nocturnal wakefulness as a previously unrecognized risk factor for suicide. J Clin Psychiatry. 2016;77:e726–33.
88. Pigeon WR, Conner K, Pinquart M. Meta-analysis of sleep disturbance and suicidal thoughts and behaviors. J Clin Psychiatry. 2012;73:e1160–7.
89. Silverman MM, Meyer PM, Sloane F, et al. The big ten student suicide study: a 10-year study of suicides on midwestern university campuses. Suicide Life Threat Behav. 1997;27:285–303.
90. Smarr KL, Keffer AL. Measures of depression and depressive symptoms. Arthritis Care. 2011;63:S454–66.
91. Baron DA, Baron SH, Tompkins J, et al. Assessing and treating depression in athletes. In: Baron DA, Reardon C, Baron SH, editors. Clinical sports psychiatry: an international perspective. Chichester, West Sussex: Wiley; 2013. p. 65–78.
92. Polat A, Cakir U, Karabulut U, et al. Reliability and validity of Turkish form of Baron depression screener for athletes. Bull Clin Pharmacol. 2015;25:S134.
93. 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.
94. Gulliver A, Griffiths KM, Christensen H. Barriers and facilitators to mental health help-seeking for young elite athletes: a qualitative study. BMC Psychiatry. 2012;12:157.
95. Kellmann M, Bertollo M, Bosquet L, et al. Recovery and performance in sport: consensus statement. Int J Sports Physiol Perform. 2018;13(2):240–5.
96. Le Meur Y, Hausswirth C, Natta F, et al. A multidisciplinary approach to overreaching detection in endurance trained athletes. J Appl Physiol. 2013;114:411–20.
97. Meeusen R, Duclos M, Foster C, et al. Prevention, diagnosis, and treatment of the overtraining syndrome: joint consensus statement of the European College of Sport Science and the American College of Sports Medicine. Med Sci Sports Exerc. 2013;45:186–205.
98. Halson SL, Jeukendrup AE. Does overtraining exist? An analysis of overreaching and overtraining research. Sports Med. 2004;34:967–81.
99. Roy BA. Overreaching/overtraining: more is not always better. ACSM's Health and Fitness Journal. 2015;19:4–5.
100. Cadegiani FA, Kater CE. Hormonal aspects of overtraining syndrome: a systematic review. BMC Sports Sci Med Rehabil. 2017;9:14.
101. Carfagno D, Hendrix J. Overtraining syndrome in the athlete: current clinical practice. Curr Sports Med Rep. 2014;13(1):45–51.
102. Schwellnus M, Soligard T, Alonso JM, 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;50:1043–52.
103. Morgan WP, Brown DR, Raglin JS, et al. Psychological monitoring of overtraining and staleness. Br J Sports Med. 1987;21:107–14.
104. Armstrong LE, VanHeest JL. The unknown mechanism of the overtraining syndrome: clues from depression and psychoneuroimmunology. Sports Med. 2002;32:185–209.
105. Kellmann, M & K. Wolfgang Kallus. "Recovery-stress questionnaire for athletes: User manual." 2001. Human Kinetics.
106. Raglin JS, Morgan WP. Development of a scale for use in monitoring training-induced distress in athletes. Int J Sports Med. 1994;15:84–8.
107. McNair D, Lorr M, Doppleman L. POMS manual for the profile of mood states. In: San Diego. Educational and Industrial Testing Service: CA; 1971.
108. Rice SM, Gwyther K, Santesteban-Echarri O, et al. Determinants of anxiety in elite athletes: a systematic review and meta-analysis. Br J Sports Med. 2019;53:722030.
109. Bandelow B, Michaelis S. Epidemiology of anxiety disorders in the 21st century. Dialogues Clin Neurosci. 2015;17:327–35.
110. Goodman WK, Grice DE, Lapidus KA, et al. Obsessive-compulsive disorder. Psychiatr Clin North Am. 2014;37:257–67.
111. Lancaster MA, McCrea MA, Nelson LD. Psychometric properties and normative data for the brief symptom Inventory-18 (BSI-18) in high school and collegiate athletes. Clin Neuropsychol. 2016;30:338–50.
112. Weber S, Puta C, Lesinski M, et al. Symptoms of anxiety and depression in young athletes using the hospital anxiety and depression scale. Front Physiol. 2018;9:1–12.
113. Stirling AE, Kerr GA. Perfectionism and mood states among recreational and elite athletes. *Athletic Insight - The Online*. Journal of Sport Psychology. 2006;8(4):13–27. https://pdfs.semanticscholar.org/351a/f85fd509070caa6eb1d0e9c7ec7b0c8d0c0d.pdf (accessed 04 June 2020)
114. Beals KA, Manore MM. Disorders of the female athlete triad among collegiate athletes. Int J Sport Nutr Exerc Metab. 2002;12(3):281–93.
115. Kellman M. Preventing overtraining in athletes in high-intensity sports and stress/recovery monitoring. Scand J Med Sci Sports. 2010;20(Suppl 2):95–102.
116. Lindqvist AS, Moberg T, Eriksson BO, et al. A retrospective 30-year follow-up study of former Swedish-elite male athletes in power sports with a past anabolic androgenic steroids use: a focus on mental health. Br J Sports Med. 2013;47:965–9.

117. Staufenbiel SM, Penninx BW, Spiiker AT, et al. Hair cortisol, stress exposure, and mental health in humans: a systematic review. Psychoneuroendocrinology. 2013;38(8):1220–35.
118. Weigand S, Cohen J, Merenstein D. Susceptibility for depression in current and retired student athletes. Sports Health. 2013;5(3):263–6.
119. Guillén F, Sánchez R. Competitive anxiety in expert female athletes: sources and intensity of anxiety in national team and first division Spanish basketball players. Percept Mot Skills. 2009;109:407–19.
120. Halvari H, Gjesme T. Trait and state anxiety before and after competitive performance. Percept Mot Skills. 1995;81:1059–74.
121. McKnight PE, Monfort SS, Kashdan TB, et al. Anxiety symptoms and functional impairment: a systematic review of the correlation between the two measures. Clin Psychol Rev. 2016;45:115–30.
122. Moran TP. Anxiety and working memory capacity: a meta-analysis and narrative review. Psychol Bull. 2016;142:831–64.
123. Hirshkowitz M, Whiton K, Albert SM, et al. National Sleep Foundation's updated sleep duration recommendations: final report. Sleep Health. 2015;1:233–43.
124. Watson NF, Badr MS, Belenky G, et al. Recommended amount of sleep for a healthy adult: a joint consensus statement of the American Academy of sleep medicine and Sleep Research Society. Sleep. 2015;38:843–4.
125. St-Onge M-P, Grandner MA, Brown D, et al. Sleep duration and quality: impact on lifestyle behaviors and cardiometabolic health: a scientific statement from the American Heart Association. Circulation. 2016;134:e367–86.
126. Swinbourne R, Gill N, Vaile J, et al. Prevalence of poor sleep quality, sleepiness and obstructive sleep apnea risk factors in athletes. Eur J Sport Sci. 2016;16(7):850–8.
127. NCAA: NCAA GOALS Study, 2015 Indianapolis, IN, NCAA; 2016.
128. Roberts SSH, Teo WP, Warmington SA. Effects of training and competition on the sleep of elite athletes: a systematic review and meta-analysis. Br J Sports Med. 2019;53(8):513–22.
129. Silva A, Queiroz SS, Winckler C, et al. Sleep quality evaluation, chronotype, sleepiness and anxiety of Paralympic Brazilian athletes: Beijing 2008 Paralympic games. Br J Sports Med. 2012;46(2):150–4.
130. Baglioni C, Riemann D. Is chronic insomnia a precursor to major depression? Epidemiological and biological findings. Curr Psychiatry Rep. 2012;14:511–51.
131. Baglioni C, Battagliese G, Feige B, et al. Insomnia as a predictor of depression: a meta-analytic evaluation of longitudinal epidemiological studies. J Affect Disord. 2011;135:10–9.
132. Spiegelhalder K, Regen W, Nanovska S, et al. Comorbid sleep disorders in neuropsychiatric disorders across the life cycle. Curr Psychiatry Rep. 2013;15:364.
133. Haddock N, Wells ME. The association between treated and untreated obstructive sleep apnea and depression. Neurodiagn J. 2018;58(1):30–9.
134. Stranks EK, Crowe SF. The cognitive effects of obstructive sleep apnea: an updated meta-analysis. Arch Clin Neuropsychol. 2016;31(2):186–93.
135. Dobrosielski DA, Nichols D, Ford J, et al. Estimating the prevalence of sleep-disordered breathing among collegiate football players. Respir Care. 2016;61(9):1144–50.
136. George CF, Kab V. Sleep-disordered breathing in the National Football League is not a trivial matter. Sleep. 2011;34(3):245.
137. Benton ML, Friedman NS. Treatment of obstructive sleep apnea syndrome with nasal positive airway pressure improves golf performance. J Clin Sleep Med. 2013;9:1237–42.
138. 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.
139. Nédélec M, Halson S, Abaidia AE, et al. Stress, sleep and recovery in elite soccer: a critical review of the literature. Sports Med. 2015;45:1387–400.
140. Schwartz J, Simon RD. Sleep extension improves serving accuracy: a study with college varsity tennis players. Physiol Behav. 2015;151:541–4.
141. Simpson NS, Gibbs EL, Matheson GO. Optimizing sleep to maximize performance: implications and recommendations for elite athletes. Scand J Med Sci Sports. 2017;27:266–74.
142. Smith RS, Efron B, Mah CD, et al. The impact of circadian misalignment on athletic performance in professional football players. Sleep. 2013;36:1999–2001.
143. Strand LB, Laugsand LE, Wisløff U, et al. Insomnia symptoms and cardiorespiratory fitness in healthy individuals: the Nord-Trøndelag health study (HUNT). Sleep. 2013;36:99–108.
144. Edwards BJ, Waterhouse J. Effects of one night of partial sleep deprivation upon diurnal rhythms of accuracy and consistency in throwing darts. Chronobiol Int. 2009;26:756–68.
145. Racinais S, Connes P, Bishop D, et al. Morning versus evening power output and repeated sprint ability. Chronobiol Int. 2005;22:1029–39.
146. Halson SL. Sleep in elite athletes and nutritional interventions to enhance sleep. Sports Med. 2014;44(1):13–23.
147. Mah CD. Extended sleep and the effects on mood and athletic performance in collegiate swimmers. Sleep. 2008;31:A128.
148. Chiu HY, Chen PY, Chuang LP, et al. Diagnostic accuracy of the Berlin questionnaire, STOP-BANG, STOP, and Epworth sleepiness scale in detecting obstructive sleep apnea: a bivariate meta-analysis. Sleep Medicine Rev. 2017;36:57–70.

149. Gagnon C, Bélanger L, Ivers H, et al. Validation of the insomnia severity index in primary care. Am Board Fam Med. 2013;26(6):701–10.
150. Bender AM, Lawson D, Werthner P, et al. The clinical validation of the athlete sleep screening questionnaire: an instrument to identify athletes that need further sleep assessment. Sports Med Open. 2018;4(23) https://doi.org/10.1186/s40798-018-0140-5. (accessed 28 Mar 2020)
151. Perlis ML, Aloia M, Kuhn BR. Behavioral treatments for sleep disorders: a comprehensive primer of behavioral sleep medicine interventions. London: Elsevier; 2011.
152. Perlis ML, Smith MT, Cacialli DO, et al. On the comparability of pharmacotherapy and behavior therapy for chronic insomnia: commentary and implications. J Psychosom Res. 2003;54:51–9.
153. Smith MT, Perlis ML, Park A, et al. Comparative meta-analysis of pharmacotherapy and behavior therapy for persistent insomnia. Am J Psychiatry. 2002;159:5–11.
154. Byrd KL, Gelaye B, Tadesse MG, et al. Sleep disturbances and common mental disorders in college students. Health Behav Policy Rev. 2014;1(3):229–37.
155. Joy E, Kussman A, Nattiv A. 2016 update on eating disorders in athletes: a comprehensive narrative review with a focus on clinical assessment and management. Br J Sports Med. 2016;50(3):154–62.
156. 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.
157. Arthur-Cameselle J, Sossin K, Quatromoni P. A qualitative analysis of factors related to eating disorder onset in female collegiate athletes and non-athletes. Eat Disord. 2017;25(3):199–215.
158. Veliz P, Epstein-Ngo QM, Meier E, et al. Painfully obvious: a longitudinal examination of medical use and misuse of opioid medication among adolescent sports participants. J Adolesc Health. 2014;54(3):333–40.
159. Sagoe D, Molde H, Andreassen CS, et al. The global epidemiology of anabolic-androgenic steroid use: a meta-analysis and meta-regression analysis. Ann Epidemiol. 2014;24:383–98.
160. McCrory P, Meeuwisse W, Dvorák 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.
161. Corwin DJ, Zonfrillo MR, Master CL, et al. Characteristics of prolonged concussion recovery in a pediatric subspecialty referral population. J Pediatr. 2014;165:1207–15.
162. NCAA: NCAA Student-Athlete COVID-19 Well Being Study. http://www.ncaa.org/about/resources/research/ncaa-student-athlete-covid-19-well-being-study (accessed 1 Jun 2020).
163. Knapp J, Aerni G, Anderson J. Eating disorders in female athletes: use of screening tools. Curr Sports Med Rep. 2014;13(4):214–8.
164. Plateau CR, Arcelus J, Meyer C. Detecting eating psychopathology in female athletes by asking about exercise: use of the compulsive exercise test. Eur Eat Disord Rev. 2017;25(6):618–24.
165. Morgan JF et al. The SCOFF questionnaire: assessment of a new screening tool for eating disorders. BMJ 1999 4;319(7223):1467–8.
166. Fairburn CG, Cooper Z, O'Connor ME. Eating disorder examination (edition 16.0D). In: Fairburn CG, editor. Cognitive behavior therapy and eating disorders. New York: The Guilford Press; 2008. p. 265–308.
167. Herring SA, Kibler WB, Putukian M, et al. Team physician consensus statement: 2013 update. Med Sci Sports Exerc. 2013;45(8):1618–22.
168. Herring SA, Kibler WB, Putukian M, et al. The team physician and the return-to-play decision: a consensus statement – 2012 update. Med Sci Sports Exerc. 2012;44(12):2446–8.
169. Brisola-Santos MB, Mello e Gallinaro JG, Sampaio-Junior B, et al. Prevalence and correlates of cannabis use among athletes: a review. Am J Addict. 2016;25(7):518–28.
170. Gil F, Guerra de Andrade A, Castaldelli-Maia JM. Discussing prevalence, impacts and treatment of substance use disorders in athletes. Int Rev Psychiatry. 2016;28(6):572–8.
171. McDuff DR, Baron D. Substance use in athletics: a sports psychiatry perspective. Clin Sports Med. 2005;24:885–97.
172. Morse ED. Substance use in athletes. In: Baron DA, Reardon CL, Baron SH, editors. Clinical sports psychiatry: an international perspective. Oxford, UK: Wiley; 2013.
173. NCAA student-athlete substance use study: executive summary August 2014. NCAA.org http://www.ncaa.org/about/resources/research/ncaa-student-athlete-substance-use-study-executive-summary-august-2014 (accessed 21 Mar 2020).
174. Rice SM, Parker AG, Rosenbaum S, et al. Sport-related concussion and mental health outcomes in elite athletes: a systematic review. Sports Med. 2018;48:447–65.
175. US Department of Health and Human Services. HHS Acting Secretary Declares Public Health Emergency to Address National Opioid Crisis. October 26, 2017. https://www.hhs.gov/about/news/2017/10/26/hhs-acting-secretary-declares-public-health-emergency-address-national-opioid-crisis.html (accessed 28 Mar 2020).
176. Anderson MN, Womble MN, Mohler SA, et al. Preliminary study of fear of re-injury following sport-related concussion in high school sports. Develop Neuropsych. 2019;44(6):443–51.

177. Veliz PT, Boyd CJ, McCabe SE. Competitive sport involvement and substance use among adolescents: a nationwide study. Subst Use Misuse. 2015;50:156–65.
178. Veliz P, Boyd CJ, McCabe SE. Nonmedical prescription opioid and heroin use among adolescents who engage in sports and exercise. Pediatrics. 2016;138(2):e20160677.
179. Ford JA. Alcohol use among college students: a comparison of athletes and nonathletes. Subst Use Misuse. 2007;42:1367–77.
180. Ford JA. Nonmedical prescription drug use among college students: a comparison between athletes and nonathletes. J Am Coll Heal. 2008;57:211–20.
181. Ford JA, Pomykacz C, Veliz P, et al. Sports involvement, injury history, and non-medical use of prescription opioids among college students: an analysis with a national sample. Am J Addict. 2018;27:15–22.
182. Kondric M, Sekulic D, Mandic GF. Substance use and misuse among Slovenian table tennis players. Subst Use Misuse. 2010;45:543–53.
183. Momaya A, Fawal M, Estes R. Performance-enhancing substances in sports: a review of the literature. Sports Med. 2015;45:517–31.
184. O'Brien KS, Ali A, Cotter JD, et al. Hazardous drinking in New Zealand sportspeople: level of sporting participation and drinking motives. Alcohol. 2007;42:376–82.
185. Pereira HMG, Sardela VF. Stimulant doping agents used in Brazil: prevalence, detectability, analytical implications, and challenges. Subst Use Misuse. 2014;49:1098–114.
186. Reardon C, Creado S. Drug abuse in athletes. Subst Abus Rehabil. 2014:95–105.
187. Diehl K, Thiel A, Zipfel S, et al. Substance use among elite adolescent athletes: findings from the GOAL study. Scand J Med Sci Sports. 2014;24:250–8.
188. Wenzel T, Zhu LJ. Posttraumatic stress in athletes. In: Baron DA, Reardon C, Baron SH, editors. Clinical sports psychiatry: an international perspective. New York, NY: Wiley; 2013. p. 102–14.
189. Zhou J, Heim D, O'Brien K. Alcohol consumption, athlete identity, and happiness among student sportspeople as a function of sport-type. Alcohol. 2015;50:617–23.
190. Huang J-H, Jacobs DF, Deverensky JL, et al. Gambling and health risk behaviors among U.S. college student-athletes: findings from a national study. J Adolesc Health. 2007;10:390–7.
191. Kerr ZY, Thomas LC, Simon JE, et al. Association between history of multiple concussions and health outcomes among former college football players: 15-year follow-up from the NCAA concussion study (1999-2001). Am J Sports Med. 2018;46:1733–41.
192. Ivarsson A, Johnson U. Psychological factors as predictors of injuries among senior soccer players. A prospective study. J Sports Sci Med. 2010;9:347–52.
193. Forsdyke D, Smith A, Jones M, et al. Psychosocial factors associated with outcomes of sports injury rehabilitation in competitive athletes: a mixed studies systematic review. Br J Sports Med. 2016;50:537–44.
194. Johnson U, Ivarsson A. Psychological predictors of sport injuries among junior soccer players injury prediction in Swedish soccer. Scand J Med Sci Sports. 2011;21:129–36.
195. Pensgaard AM, Ivarsson A, Nilstad A et al: Psychosocial stress factors, including the relationship with the coach, and their influence on acute and overuse injury risk in elite female football players. BMJ Open Sport & Exercise Medicine 2018;4e000317.doi:https://doi.org/10.1136/bmjsem-107-000317.
196. 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;7:224–30.
197. Hainline B, Turner JA, Caneiro JP, et al. Pain in elite athletes: neurophysiological, biomechanical and psychosocial considerations: a narrative review. Br J Sports Med. 2017;51:1259–64.
198. Echemendia RJ, Meeuwisse W, McCrory P, et al. The sport concussion assessment tool 5th edition (SCAT5): background and rationale. Br J Sports Med. 2017;51:848–50.
199. Kontos AP, Covassin T, Elbin RJ, et al. Depression and neurocognitive performance after concussion among male and female high school and collegiate athletes. Arch Phys Med Rehabil. 2012;93:1751–6.
200. Vargas G, Rabinowitz A, Meyer J, et al. Predictors and prevalence of postconcussion depression symptoms in collegiate athletes. J Athl Train. 2015;50:250–5.
201. Lange RT, Iverson GL, Rose A. Depression strongly influences postconcussion symptom reporting following mild traumatic brain injury. J Head Trauma Rehabil. 2011;26
202. Putukian M, Riegler K, Amalfe S, et al. Pre-injury and post-injury factors that predict sport-related concussion and clinical recovery. Clin J Sports Med. 2018; https://doi.org/10.1097/JSM.0000000000000705. [Epub ahead of print]
203. Czuppon S, Racette BA, Klein SE, et al. Variables associated with return to sport following anterior cruciate ligament reconstruction: a systematic review. Br J Sports Med. 2014;48:356–64.
204. Garcia GH, Wu HH, Park MJ et al: Depression symptomatology and anterior cruciate ligament injury: incidence and effect on functional outcome-a prospective cohort study. Am J Sports Med 2015, published online Dec 1 DOI: https://doi.org/10.1177/0363546515612466.
205. World Health Organization. Novel Coronavirus 2019. https://www.who.int/emergencies/diseases/novel-coronavirus-2019 (accessed 31 May 2020).
206. Gouttebarge V: Overview of the IOC consensus statement on mental health in elite sports. 18th

Biennial Congress of the South African Sports Medicine Association (SASMA). Cape Town, South Africa, 2019.
207. Rice S, Olive L, Gouttebarge V, et al. Mental health screening: severity and cut-off point sensitivity of the athlete psychological strain questionnaire in male and female elite athletes. BMJ Open Sport Exerc Med. 2020;6(1):e000712.
208. Rice SM, Parker AG, Mawren D, et al. Preliminary psychometric validation of a brief screening tool for athlete mental health among male elite athletes: the athlete psychological strain questionnaire. Int J Sport Exerc Psychol. 2019;(6):1–16.
209. Castaldelli-Maia JM, Gallinaro JGDME, Falcao RS, 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;53:707–21.
210. Campbell E, Jones G. Sources of stress experienced by elite male wheelchair basketball players. Adapt Phys Act Q. 2002;19:82–99.
211. Macdougall H, O'Halloran P, Shields N, et al. Comparing the Well-being of Para and Olympic sport athletes: a systematic review. Adapt Phys Act Q. 2015;32:256–76.

35 Radiological Assessment of Sports Injuries

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35.1 Introduction

Sports performance is governed by an athlete's innate and neuromotor training skills, anatomical and physiological factors as well as psychological balance. In clinical practice, this efficiency is projected by the prompt and coordinated reaction of the musculoskeletal system to overcome the physical demands that take place during a game or fight, such as changing direction during soccer dribbling, handball throwing, tennis serving, basketball jumping, or when the fighter dodges a punch. It is important to remember that each sport has a specific physical demand and, as a result, specific sports-related injuries may occur.

In order to better investigate and establish an accurate diagnosis, the sports medicine physician has to initially assess sports trauma considering three key points: athlete's history of trauma, physical examination, and radiological evaluation.

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35.2 History of Trauma

The athlete's history of trauma must be explored as accurately as possible to identify the mechanism of injury and damages to anatomical structures in sports trauma. Contact sports can produce direct and indirect trauma, while in non-contact sports, indirect traumas are the main mechanism of injury.

Generally, direct traumas range from minor ones, such as cuts and abrasions of the skin, to major ones involving fractures. On the other hand, indirect trauma produces ligament stress resulting in ligament injuries in common joint sites such as the ankle and knee.

The anatomy of the human body has topographic differences according to the anatomical site. For example, the thigh has a considerable muscle mass while the bone prominences are easily palpable in front of the knee, as well as in the abdomen and the face.

35.3 Initial Clinical Signs

The severity of tissue damage is clinically manifested by the edema, ecchymosis, hemorrhage, joint effusion, bone crepitus, bone deformity,

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athlete's inability of moving or remaining standing, loss of consciousness, and so on. All these signs in conjunction with the mechanism of observed or reported trauma guide the physical assessment.

In general, sports trauma results from lower energy of trauma and causes small injuries such as skin cuts and abrasions. In the facial region, these types of trauma can also cause hemorrhage, especially in the nose, a well-vascularized site. However, most of the time, the physical assessment in the field allows establishing the diagnosis and treatment: bandage, local compression, and in case of skin cut cleaning of the wound, suture, and dressing. In contrast, the higher energy of trauma causes more significant tissue damage such as fractures, ligaments injuries, brain damage, and so on.

This chapter explores the most common anatomical sites of sports injury and discusses the strategy of radiological evaluation as well as the contribution of each exam to establish an accurate diagnosis of the athlete's sports injury.

35.4 Radiological Assessment

The improvement and advances in the methods of diagnostic imaging has come to the forefront of the sports medicine field, helping and guiding physicians, physiotherapists, physiatrists, physiologists, physical trainers, and other health professionals improve the management of sports injuries, monitoring of injuries, and contribution to the decision-making on return to play.

Although knowing the principles of each method is fundamental, there are still frequent doubts about the most appropriate imaging method for the study of an anatomical structure or investigation of a specific clinical suspicion.

35.5 How Do We Manage It?

Hypothetically, this condition may be compared to the moment you deliver your car to your trusted mechanic for a tune-up. If you do not report any complaints, your mechanic will make a routine analysis on various items of your car. On the other hand, if you make a specific complaint, for example, informing about a noise and its probable location, your mechanic will able to perform a guided analysis, considering the reported "symptoms."

Therefore, the knowledge of the diagnostic hypothesis and an adequate anamnesis will allow the radiologist to choose the most appropriate imaging method. Besides using specific techniques and sequences for a more accurate diagnosis, tomography and magnetic resonance imaging (MRI) increase the predictive value of these methods and enable a more detailed description of anatomical and pathological findings.

35.6 Types of Radiological Exams

Radiological exams may be grouped according to the use of ionizing radiation. Conventional radiography and computed tomography are methods based on the use of X-ray beams to obtain the images, i.e., the use of ionizing radiation is involved, and specific contraindications, such as pregnancy, should be researched. The interpretation of these tests is based on the analysis of the five basic radiographic densities, from the most radiotransparent to the most radiodense (air, fat, soft tissues, bone and metal) (Fig. 35.1).

35.7 Conventional Radiography

Conventional X-ray is still the modality of choice in the initial evaluation of any musculoskeletal complaint. It has a high resolution for bone structures, wide availability, and low cost, but low resolution for soft tissues. It should be always requested before further indication of any other imaging methods. The information obtained with the analysis of conventional radiography, even if normal, is fundamental for the technical programming and interpretation of other imaging methods and should be always forwarded or made available to radiologists.

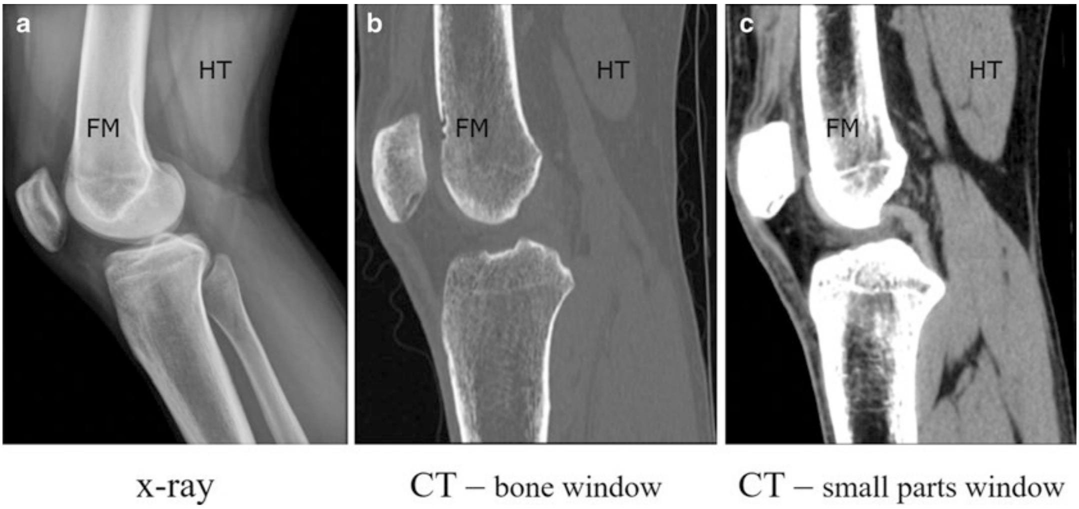


Fig. 35.1 Knee. (**a**) Lateral X-ray; (**b**) CT sagittal reconstruction (bone window); (**c**) CT sagittal reconstruction (small parts window). *—Fat densities (infrapatellar fat pad). HT—Densities of small parts (hamstring muscles); FM—Bone densities (femoral metaphysis)

35.8 Computed Tomography (CT)

When there are important clinical complaints and apparently normal conventional X-ray examinations from trauma sites, the athlete should undergo a complementary radiological assessment by computed tomography. It allows a multiplanar study and plays a fundamental role in the evaluation of places where there are large overlaps of bone structures, such as the shoulder girdle, hips, and axial skeleton (skull, face, spine, ribs, sternum, sacrum, and coccyx) that could hide a fracture. Although it is an excellent method for assessing calcifications, bone, and cortical avulsions, it is not the method of choice for the evaluation of bone marrow and bone-related soft tissues. In the evaluation of the other segments of the appendicular skeleton, computed tomography should be used when a more detailed description of fracture lines, fragments, and when intra-articular extensions are required.

35.9 Magnetic Resonance Imaging (MRI)

MRI is the method of choice for the evaluation of ligament, tendinous, bursal, muscular, and disc injuries related to the musculoskeletal system, and it is very useful in the analysis of the bone marrow, avoiding complications related to non-detection of fractures such as collapses, acetabular protrusions, and fragmentations.

Thus, the knowledge of the basic sequences has become fundamental for all those involved in the treatment of sports injuries. Knowledge of the main MRI sequences is based on two basic principles: the type of used sequence and the presence of fat saturation. There are two groups of basic sequences used in MRI, sensitive and non-sensitive to water.

In water-sensitive sequences, the structures that have free water or are hydrated present hypersignal (shine) and, in non-water-sensitive sequences, the liquid has hyposignal (does not shine). The main water-sensitive sequences are T2, PD, and STIR, while the main non-water-sensitive sequence is T1. **The fatty tissue always presents hypersignal in the T1 and T2**, PD **sequences**. However, when there is fat saturation, regions with adipose tissue show a sign equal to or less than the muscle (isosignal or hyposignal).

Knowing these two concepts in musculoskeletal radiology is important since most traumatic injuries cause edema in the bones, tendons, muscles, and ligaments, and water-sensitive sequences have a greater sensitivity in detecting this edema and these changes. **Fat saturation**

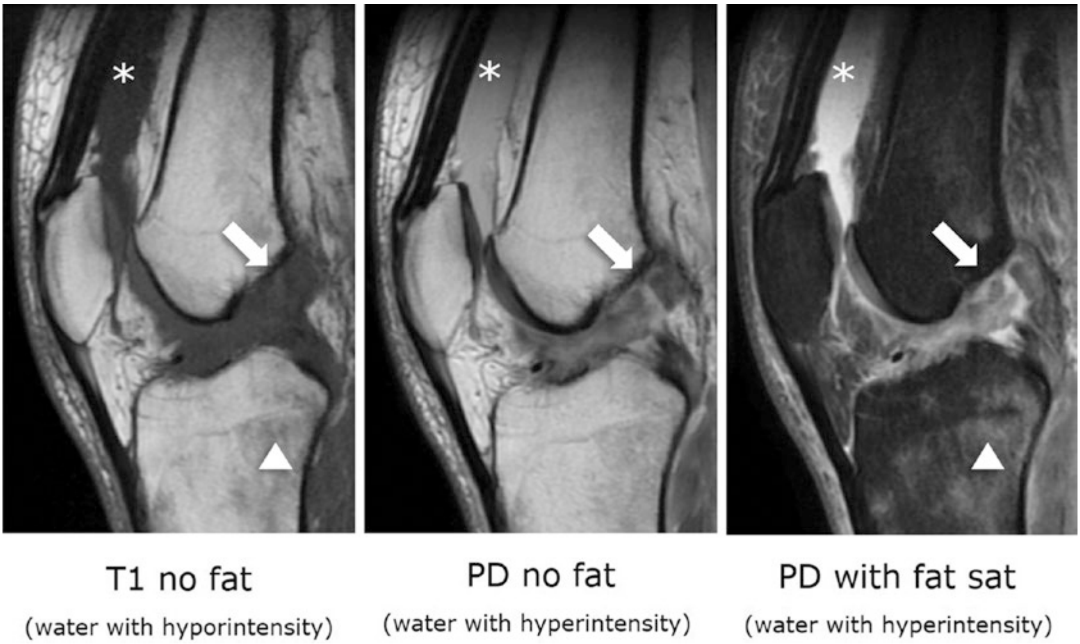


Fig. 35.2 Sagittal MRI images of the knee with ACL rupture (arrow), joint effusion (star), bone bruise (triangle). (**a**) T1 no fat (water with hypointensity—joint effusion and bone bruise with hypointensity). (**b**) PD no fat (water with hyperintensity—joint effusion with hyperintensity and bone bruise with the same signal of the bone marrow). (**c**) PD with fat sat (water with hyperintensity—joint effusion with hyperintensity and bone bruise with hyperintensity)

(fat signal cancelation) **facilitates the detection of edema in places** where fat hypersignal may be confused with edema hypersignal in cases of bone contusion (Fig. 35.2).

35.10 Comparing Computed Tomography with MRI

In a didactic way, let us imagine a glass with wine, if our intention is to evaluate the wine, we should request MRI, whereas if we want to analyze the glass, we should request computed tomography (Fig. 35.3). Thus, there is a concept that computed tomography is an excellent method to evaluate the continent, and magnetic resonance imaging is a useful method to assess the content of bone structures.

The principle of ultrasonography is based on the use of mechanical waves and has great importance as it is transportable and allows a dynamic evaluation of structures. However, most times, it does not have the spatial resolution and the ability to multiplanar reconstruction of MRI.

Thus, in order to guide the request for radiological examinations, we discuss, in the next pages, the radiological assessment of the most common sites of injuries reported in elite and recreational sports:

35.11 Knee

The knee joint stability is achieved primarily by ligaments, muscles act as secondary stabilizers, while bone structures have little participation in it. Moreover, this joint is not surrounded by a great muscle mass differently from the thigh, resulting in low protection against direct trauma. Therefore, these anatomical particularities and its biomechanical role in sports practice make the knee a prevalent site of sports injuries.

The knee is a structure that offers mobility and supports the body's weight distribution

Fig. 35.3 Glass with wine

and is essential to endure speed gain and braking. Due to this, its injuries are commonly associated to high-speed motion competitive and repetitive sports that involve running and jumping such as soccer, American football, athletics, basketball, volleyball, and street running, where the force on the knees can reach up to 10 times the body weight [1]. These injuries are mostly related to indirect and direct trauma, which can cause from bony lesions, such as fractures, to soft tissues injuries like ligament rupture, meniscal tears and tendinopathy, and tendon rupture.

35.11.1 Fractures

In fractures around the knee, direct trauma is the main mechanism of injury, and knee X-rays are used to define the diagnosis in most cases. However, the physician must be aware of all radiographic views adapted to each clinical condition. An example of that is the case of a longitudinal patellar fracture that is not seen in AP and lateral views, but it is clearly observed in the axial view.

In cases of depression fracture on the proximal tibia or distal femur, the CT will offer more details of the bone depression, such as split or compression fractures, compared to the X-rays, helping the physician to choose the best treatment management for each case.

35.11.2 Fatigue Fracture

Fatigue fractures (also known as overuse fractures) are a type of stress fracture due to abnormal stresses on normal bone. They should not be confused with an insufficiency fracture, which occurs due to normal stresses on abnormal bone. An unbalance between the ability of a healthy bone structure regenerating itself and repetitive stress charges may result in a fatigue fracture by an increase of osteoclast recruitment [2].

In athletes, this kind of fracture usually occurs in the proximal tibia and is a progressive lesion due to its growing stress. On the earliest stages, it may be seen as a subtle chondral irregularity or subchondral lucency on plain radiograph, and generally it may be unnoticed. Bone edema can be easily detected on early stages as a medullar hyperintense signal on MRI, and as the lesion advances due to continuous charge overload, and a hypointense fracture line on all sequences, commonly associated to bone edema, can be detected [3] (Fig. 35.4).

35.11.3 Avulsion Fracture

Avulsion fractures or injuries occur when an aggressive force pulls a tendon or ligament and rips out the cortical bone near their insertion, establishing instability of the joint. Therefore,

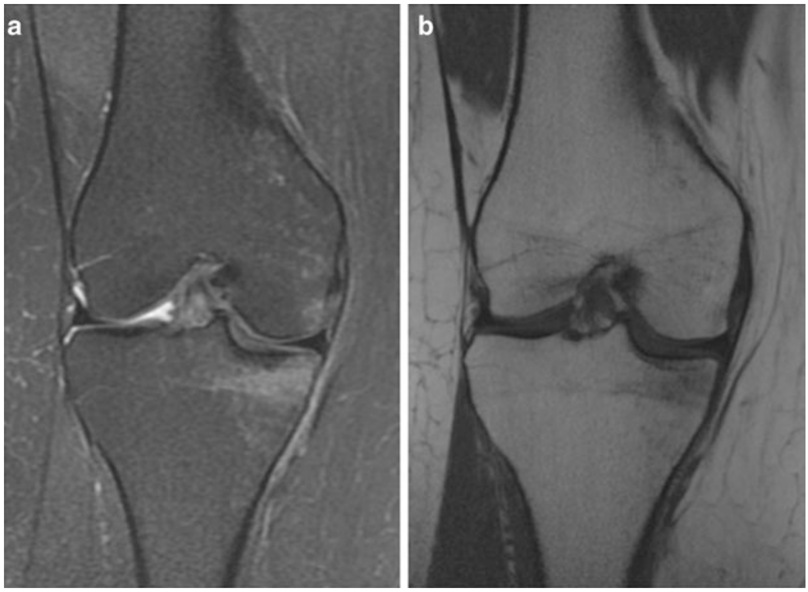


Fig. 35.4 Coronal T2 Fat Sat (**a**) ant T1 (**b**) MRI of the knee. Linear bone edema on medial tibial condyle showing hyperintense signal on T2 and hypointense signal on T1 sequences (fatigue fracture)

Table 35.1 Most common avulsion fractures around the knee and the associated injuries

	Associated injury
Pelligrini–Stieda	MCL and ACL
Segond fracture	LCL, ACL, lateral meniscus
Reverse Segond fracture	MCL and PCL
Medial tibial spine	ACL
Posterior tibia	PCL
Fibular head	LCL, biceps femoral tendon, and indirect sign to knee dislocation

the sports physician must be aware that some avulsion fracture may present an indirect sign of ligament injury such as PCL tibial avulsion fracture.

Table 35.1 shows the most common avulsion fractures around the knee and the associated injuries [4, 5].

The little bony avulsion fragments and smaller intra-articular bone fragments can be seen on plain radiographs but are better characterized on CT. MRI analyzes related lesions on soft tissue injuries, such as meniscal tears, ligament tears, muscle rupture, and bone edema.

Second fracture occurs when a mechanism of internal rotation with a varum stress of the knee causes the pulling of the external cortical of the lateral tibial plate, next to Gerdy's tubercle. This fragment can be easily seen in plain radiograph, but as most consider this type of lesion being pathognomonic of ACL tear, an MRI study is highly recommended.

35.11.4 Osteochondral Lesions

Acute traumatic osteochondral injury includes bone bruises and chondral, subchondral, and osteochondral fractures (Fig. 35.5). In general, these injuries are more common in young active patients and are usually the result of high-impact force applied to a normal bone that has sustained an acute injury. Patients present an acute onset of pain and clear history of preceding trauma [6].

Osteochondritis dissecans (OCD) is a focal idiopathic alteration of subchondral bone with a risk for instability and disruption of an adjacent articular cartilage that may result in premature osteoarthritis (Fig. 35.5). A chronic repetitive stress in active children and high-level athletes are always reported. The process originally OCD starts deep underneath the articular surface [7] and subsequently involves the articular cartilage at the peripheral border of the lesion.

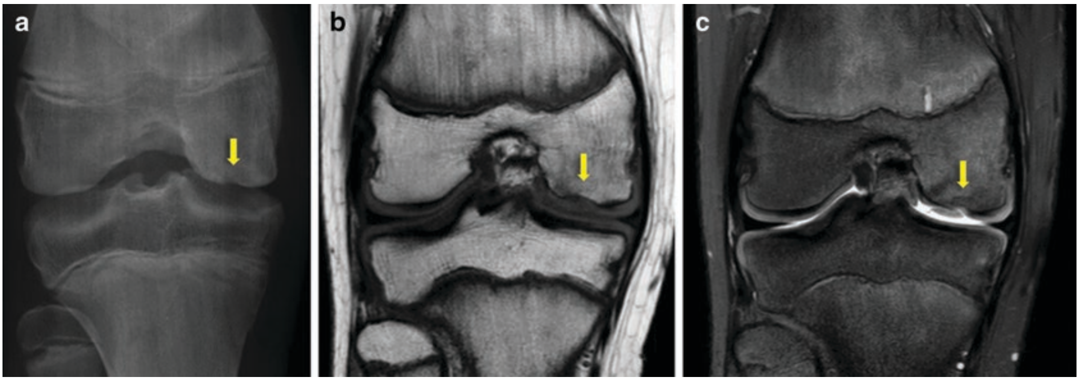


Fig. 35.5 (**a**)–X-ray, (**b**)—Coronal T1 MRI, and (**c**)—Coronal PD Fat Sat MRI. Arrow: irregularity on the medial condyle of the femur, suggesting Osteochondritis Dissecans

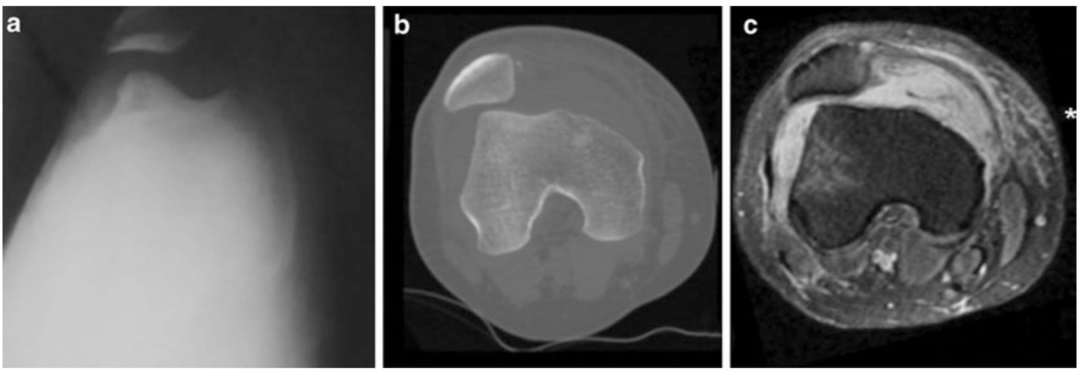


Fig. 35.6 (**a**) X-ray: Lateral displacement of the patella. (**b**) Axial CT of the knee: (bone window) with lateral patellar displacement and avulsion fracture on the patellar medial corner. (**c**) Axial MRI T2 Fat Sat with lateral patellar displacement, high signal on the medial corner of the patella and on the lateral femoral condyle, due to its impaction of one on another *(bone bruises)*

35.11.5 Ligamentar Lesions

35.11.5.1 Traumatic Patellar Dislocation

Lateral patellar displacement can be caused by a valgus-flexion-external rotation by compromising the medial patellar retinaculum, presenting high signal on the medial corner of the patella and on the lateral femoral condyle due to its impaction of one on another. There can also be avulsion fracture on the patellar medial corner right beside the retinaculum insertion. The soft tissue findings are depicted on MRI [8] (Fig. 35.6).

35.11.5.2 Anterior Cruciate Ligament

The anterior cruciate ligament (ACL) disruption is most commonly in sports activities that involve blunt starting, stopping, and pivoting. When teared, there are some indirect signs that can be identified on plain radiograph, such as the lateral femoral notch that occurs when the knee suffers hyperextension and represents an impaction on the lateral femoral condyle's surface, which can be seen on the lateral view as a depression, usually bigger than 1.5 mm. MRI is the method of choice in which the ACL bundles can be appreciated with low signal in all sequences. The tears can be characterized as total or partial, and can also have direct and indirect signs (Table 35.2) [9] (Fig. 35.7).

35.11.5.3 Posterior Cruciate Ligament

The posterior cruciate ligament (PCL) lesions are highly associated to sports practice and usually come along with ACL and medial meniscal tears. The PCL tear usually happens near the middle of its entire piece or near its distal part and can be accompanied or not by avulsion fracture of the tibial insertion. A plain radiograph can suggest PCL tear if the avulsion injury is spotted, as well if a posterior drawer of the tibia can be seen.

Similar to the ACL tears, the PCL injuries can be presented as total or partial tears, better evaluated on MRI method. In the case of complete tear of the PCL, its fibers will appear as ill-defined or simply will not appear at all in the acquired images. In regard of the PCL incomplete tears, they are easier to identify than the ACL partial tears and can be spotted as a longitudinal high-intensity lesion through its fibers. Some signs can come along with these findings, such as tibial bone edema in case of direct trauma, or tibial with femur bone edema if hypertension of the knee is the factor of injury [10] (Fig. 35.8).

35.11.5.4 Medial Collateral Ligament

A medial collateral ligament (MCL) knee sprain is a prevalent injury in athletic populations that may result in significant time lost to injury [11]. Its tears mostly occur in the proximal region and, as in the case of most soft tissues, the MRI is the method of choice to detect them (Fig. 35.9). ACL and medial meniscal tears are often seen in contact sports such as soccer, basketball, and rugby caused by the mechanism of pivot shift [12]. After a few weeks, it may appear as a Pellegrini-Stieda lesion (Fig. 35.9), a post-traumatic/post-avulsion calcification of the proximal medial collateral ligament [13].

35.11.6 Lateral Collateral Complex

The lateral collateral complex is the primary varus stabilizer of the tibiofemoral joint. Diagnosing an injury to these structures can be challenging in the setting of multiligamentous trauma; however, failure to recog-

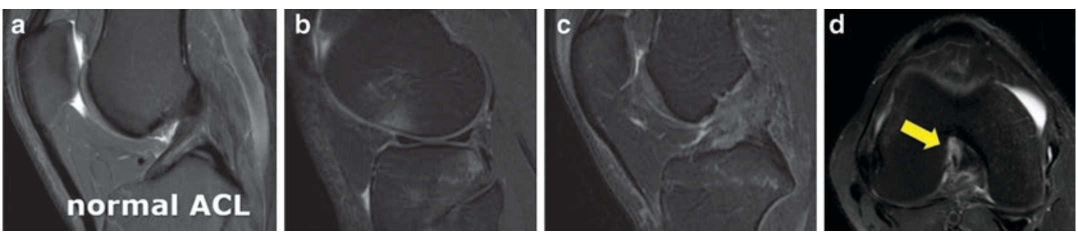


Fig. 35.7 T2 Fat Sat MRI sagittal views of the Knee. (**a**) Normal ACL, (**b**) bone contusions in the lateral compartment, (**c**) complete tear of the ACL, and (**d**) Empty notch sign in the femoral origin of the ACL (yellow arrow)

Table 35.2 Direct and indirect radiological signs of ACL injury

Radiological sign of ACL injury	
Direct	Indirect
The non-visualization of hyperintensity of the ACL fibers that represents edema or bleeding	**Anterior translation of the tibia** (more than 5 mm)
Horizontalization of the ACL (a more proximal tear)	**Bone contusions on the lateral compartment** of the knee
Loss of linearity or curling of its fibers	**Lesion on the medial collateral ligament**
Empty notch sign in which the fibers of the ACL's femoral attachment are missing while its space is filled with fluid signal	Anterior verticalization of the ACL (**anterior drawer**).

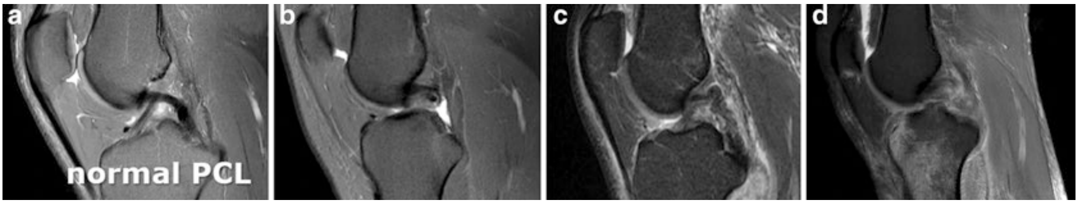


Fig. 35.8 MRI sagittal views of knee posterior cruciate ligament injuries. (**a**) Normal PCL, (**b**) PCL distal tear, (**c**) PCL tortuosity and intra-substantial injury, and (**d**) PCL complete tear (sagittal PD Fat Sat MRI)

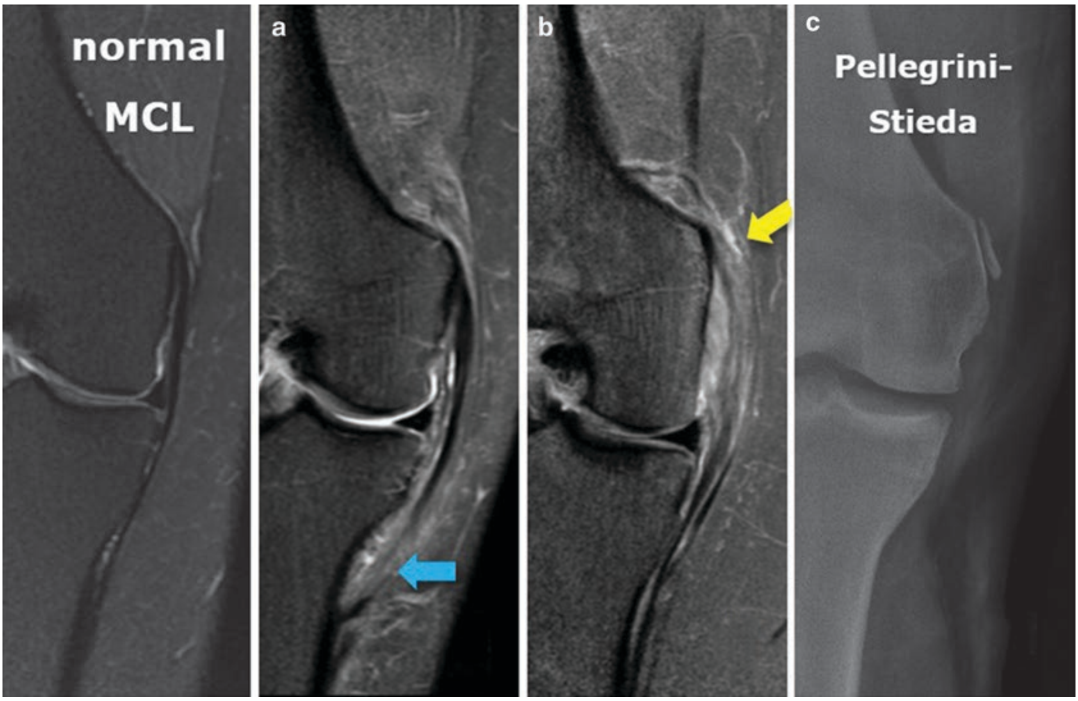


Fig. 35.9 Coronal PD Fat Sat MRI of the right knee. (**a**) Distal tear of the MCL (blue arrow). (**b**) Proximal tear of the MCL (yellow arrow). (**c**) (X-ray): Pellegrini-Stieda lesion. Ossified post-traumatic lesions at (or near) the medial femoral collateral ligament adjacent to the margin of the medial femoral condyle

nize these injuries can result in instability of the knee and unsatisfactory outcomes after cruciate ligament reconstruction. The findings are better evaluated on MRI and stress X-rays (Fig. 35.10) [14].

35.11.7 Meniscal Lesions

Menisci are fibrocartilaginous structures that cover the knee joint, facilitating its congruence and supporting the charge distribution while helping to stabilize the knee. Due to this, meniscal injury is usually associated to sports that involve running, speeding, braking, and jumping, such as soccer, basketball, skating, and athletics among others [15].

They normally appear as a low signal bow tie image in all sequences on MRI that has a 90% accuracy rating. Meniscal tears usually are accompanied in 72% of ACL tears in athletes and can have various presentations depending on its location and direction, such as horizontal, radial, vertical longitudinal, horizontal flap, vertical flap, and complex. The horizontal tears are the most common lesion (30%), are normally seen

Fig. 35.10 Knee X-rays. (**a**) no varus stress and (**b**) with varus stress. Enlargement of the lateral femorotibial joint space

without trauma and their posterior horns are usually the most affected. Radial tears can be seen with or without a previous trauma incident, and the vertical longitudinal tears almost always come along with an acute trauma event [15].

A meniscal tear is represented by an abnormal intrameniscal signal intensity on T2 or PD images extending to an articular surface on at least two sections (Fig. 35.11a–d). In addition, lesions such as bucket-handle tears can show a meniscal flap on the intercondylar notch, giving the impression of the existence of two PCLs (Double PCL sign—Fig. 35.11e) (Oei et al., 2003).

35.11.8 Tendons and Other Ligaments

Rupture of patellar tendon and quadriceps tendon occur almost invariably when in the setting of trauma (Fig. 35.12).

35.11.8.1 Rupture, Tendinitis, and Tendinosis

Tendinitis and tendinosis are two most common sport-related lesions associated with tendons around the knee. The first one is associated to activities that involve jumping, and that is why it is known as the Jumper's Knee or Patellar Tendinitis, although the structure involved is the patellar ligament, and corresponds to a chronic insertional injury of the posterior and proximal fibers of the patellar tendon near the inferior pole of the patella. The other one is the Quadriceps Tendinosis, a similar pathology to the former, but on the quadriceps tendon, near the superior patellar pole and associated to sports that involve kicking [16].

Both can be evaluated on ultrasonography and MRI, and the latter provides more information. They can be seen as thickening due to swelling and hyperechoic or hyper signal on the affected region of the tendons. As the lesions progress, they can evolve to tears [17, 18].

35.12 Hip

The hip is a joint that offers great support to the body, allowing a wide range of motion. Therefore, injuries to the hip are relatively common in athletes who practice soccer, tennis, athletics, weightlifting, rugby, Olympic gymnastics, martial arts, and CrossFit®.

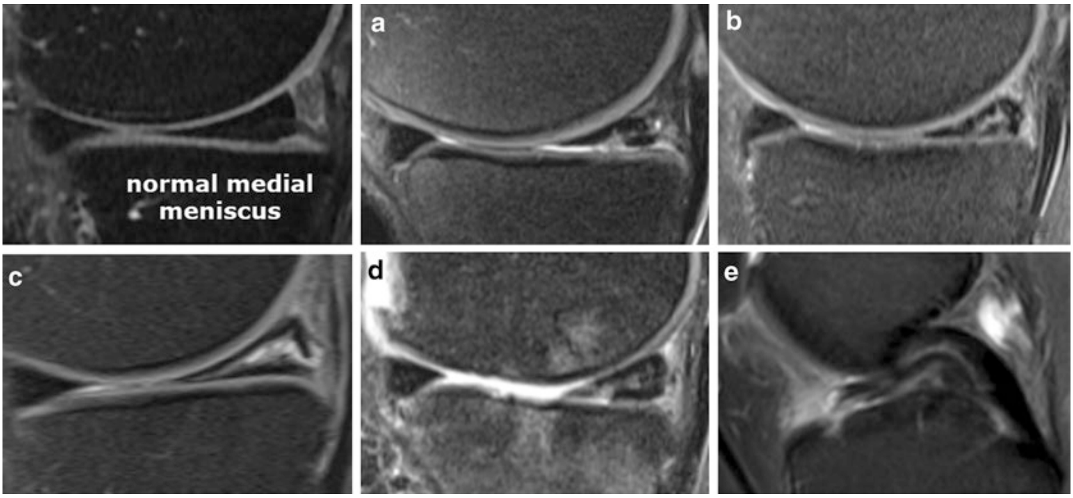


Fig. 35.11 Sagittal T2 Fat Sat MRI of the knee. (**a**–**e**) Different types of meniscal tears. (**e**) Bucket-handle meniscal tear (“**double PCL sign**”)

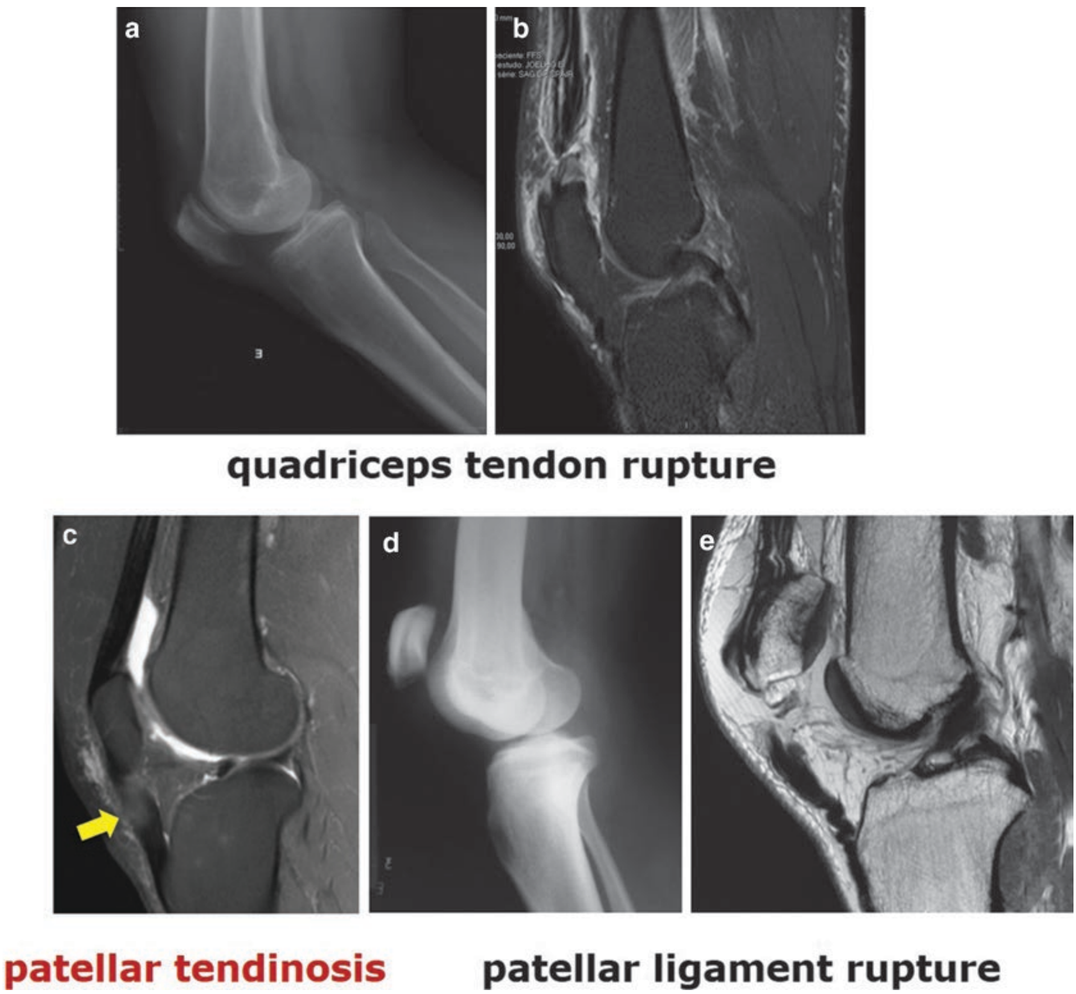
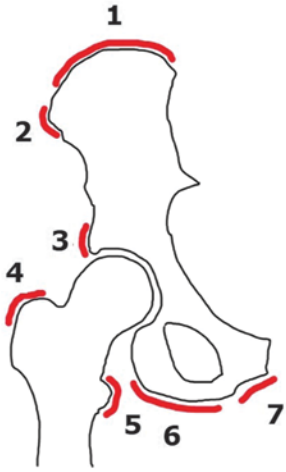


Fig. 35.12 Knee sagittal views of Quadriceps tendon complete rupture in (**a**) X-ray and (**b**) MRI; (**c**) Patellar tendinosis in MRI, and Patellar ligament complete rupture in (**d**) X-ray and (**e**) MRI

Table 35.3 Avulsion Fractures Around the Hip



ANATOMICAL SITE	ASSOCIATED INJURIES
Iliac crest (1)	Abdominal muscle
Anterosuperior iliac spine (2)	Sartorius, tensor fascia lata
Anteroinferior iliac spine (3)	Rectus femoris
Great trochanter (4)	Hip rotators
Lesser trochanter (5)	Iliopsoas
Ischial tuberosity (6)	Hamstrings
Body of pubis and inferior pubic ramus (7)	Adductors, gracilis

Most sports injuries are extra-articular, such as muscle injuries, bursitis, and tendinopathies. Among bone injuries, avulsion or stress fractures and femoroacetabular impingement are injuries that commonly occur due to overloads (Table 35.3). Similar to differential diagnoses in cases of pubalgia, we should think about conditions such as osteitis pubis and muscle-tendon injuries in the adductor compartment and rectus abdominis.

35.12.1 Avulsion Fractures

Avulsion fractures are most frequently seen in sprinters and hurdlers. In other cases, avulsion fractures in runners occur during speed training, or when there is a maximum effort toward the end of the race. Soccer and gymnastics are the most common sports-related causes of apophyseal avulsion fractures [5, 19].

Apophyseal avulsion fractures are usually the result of a sudden forceful concentric or eccentric contraction of the muscle attached to the apophysis. Avulsion fractures of the pelvis are most common in young athletes due to the discordance between increased muscle strength and an unfused physis at the muscle-tendon-bone unit [19].

35.12.1.1 Greater Trochanteric Pain Syndrome

It refers to pain that originates from the lateral hip region. Greater trochanteric bursitis (or inflammation of one or more of the peritrochanteric bursae), gluteus medius and/or minimus insertional tendinopathy and/or tears and repetitive friction between the greater trochanter and ITB have been considered the main sources of lateral hip pain [20] (Fig. 35.13). Ultrasound may be useful in some cases, but MRI is the imaging exam of choice for the evaluation of this region.

35.12.1.2 Athletic Pubalgia (or "Sports Hernia")

Athletic pubalgia is either a musculotendinous or osseous injury that involves the insertion of abdominal muscles on the pubis and the upper aponeurotic insertion of the adductor muscles [21].

Patients with MR imaging findings of athletic pubalgia are predominantly male and generally under the age of 40 years. The most commonly observed injury is along the lateral border of the rectus abdominis, just cephalad to its pubic attachment, or at the origin of the adductor longus. The most specific finding is a hyperintense T2WI signal involving the anteroinferior aspect of the pubic symphysis (Fig. 35.14) [20, 21].

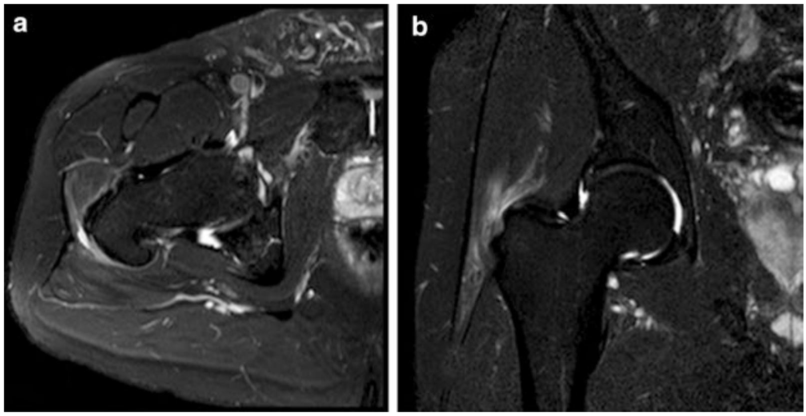


Fig. 35.13 Axial (**a**) and coronal (**b**) PD Fat Sat MRI of the hip. Trochanteric bursitis, gluteus medium, and minimum tendinopathy and peritendinitis

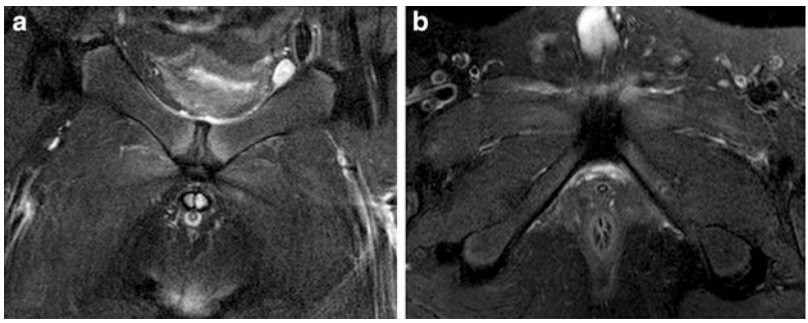


Fig. 35.14 Coronal (**a**) and axial (**b**) PD Fat Sat MRI of the pubic symphysis. Rectus abdominis muscle insertion signal change, fiber discontinuity on the left and pubic symphysis osteitis

35.12.1.3 Femoroacetabular Impingement

Femoroacetabular impingement (FAI) is a clinical syndrome of limited motion and painful hip resulting from certain types of underlying morphological abnormalities in the femoral head/neck and/or surrounding acetabulum. It is now recognized that FAI can cause serious joint damage among young athletes, even in their second and third decades [22].

There are three types of impingement: pincer, cam, and combined. **Pincer impingement** is caused by *an excessive overcoverage of the acetabulum*. General overcoverage is associated with a deep acetabulum, due to coxa profunda or protrusio acetabuli. Focal overcoverage is caused by a retroverted acetabulum or a prominent posterior acetabular wall. **Cam impingement** refers to the cam effect caused by a *non-spherical femoral head rotating inside the acetabulum,* mostly located at the lateral or anterosuperior aspect of the femoral head–neck junction just lateral to the physis scar, with subsequently decreased head–neck offset. This cause wear and tear of the labrum and cartilage anterosuperiorly. Combined impingement is a mixture of both [23, 24].

A plain radiograph is very useful to reveal this condition. The pistol grip deformity is considered a typical sign of cam impingement. The shape of the proximal femur in this deformity is reminiscent of a flintlock pistol. Since the visual aspect only provides a qualitative assessment of the deformity, several attempts at quantification have been made for use with conventional two-plane radiographs. In the pincer type, the anterior acetabular rim projecting laterally to the posterior rim which is called "crossover sign." The lateral center-edge angle, extrusion index, or acetabular index may be measured to confirm acetabular overcoverage. Focal acetabular overcoverage can be assessed using posterior wall sign [25–27]. CT or MRI volume imaging with secondary radial (oblique) reformats along the axis of the femoral neck can also be used. Both are more reliable to locate and quantify the cam deformity because of their three-dimensional character [28]. Degenerative changes of the lateral ace-

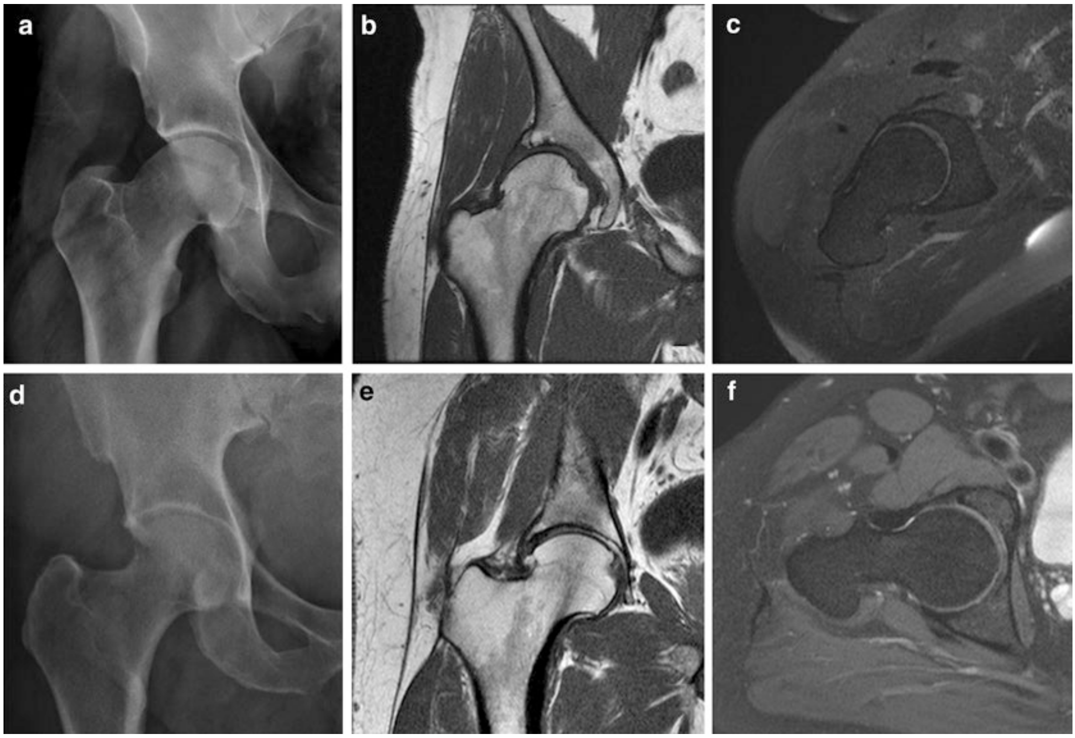


Fig. 35.15 (**a**–**c**): Cam impingement (Bone prominence in the femoral head/neck transition, cartilage damage, and labral tears). (**d**–**f**) Pincer impingement (increased acetabular bone coverage). **a** and **d** (X-ray), **b** and **e** (Coronal T1 MRI), **c** and **f** (Axial-oblique PD Fat Sat MRI) of the hip

tabular margin and the hip joint may be seen. Labral tear may also be visualized on MRI or CT arthrography. MRI is the most accurate imaging study to diagnose cartilage damage and labral tears (Fig. 35.15) [29].

35.12.2 Fatigue Fractures

Sports activities, including endurance and military activity, are frequently responsible for fatigue fractures. Long-distance runners are more predisposed to the long bone and pelvic fractures [30].

The most affected location on the hip is the superolateral femoral neck. Fracture line visualization is a rare radiology sign in fatigue fractures. **X-Rays may more often describe indirect signs** [30] while the CT is much more sensitive to detect the bone loss that precedes fracture; however, MRI is the most sensitive and specific modality for the diagnosis of fatigue fractures. The use of MRI for fatigue fracture is helpful for predicting recovery time (important especially for athletes) (Fig. 35.16) [3].

35.13 Elbow

Elbow injuries are common in athletes participating in sports that require overhead movement of the arms. They are common among weight-training sports, golf, tennis, baseball, football, gymnastic artistic, and judo [31–35].

35.13.1 Biceps Brachii Rupture

This type of rupture occurs at either proximal or distal attachment. The distal part injury is rare, seen in young people and related to trauma. Rupture of the distal biceps causes weakness when supinating the forearm and is associated with significant functional loss. MRI and ultrasound can play a role in this diagnosis (Fig. 35.17) [36, 37].

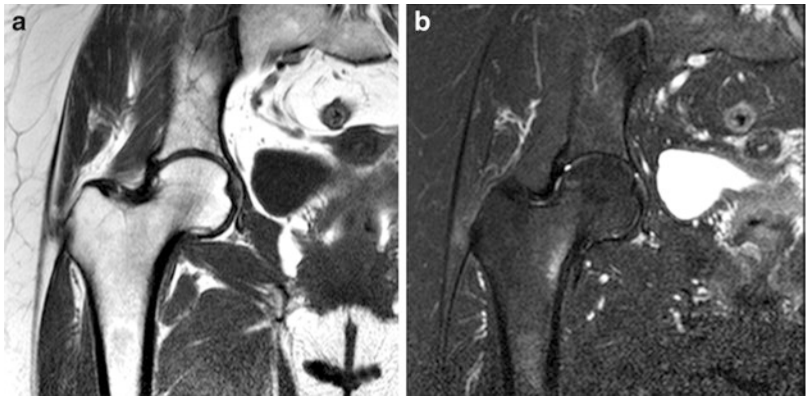


Fig. 35.16 Coronal T1 and PD Fat Sat MRI of the hip. Subcortical bone edema in the femoral calcar region, which may be related to fatigue fracture

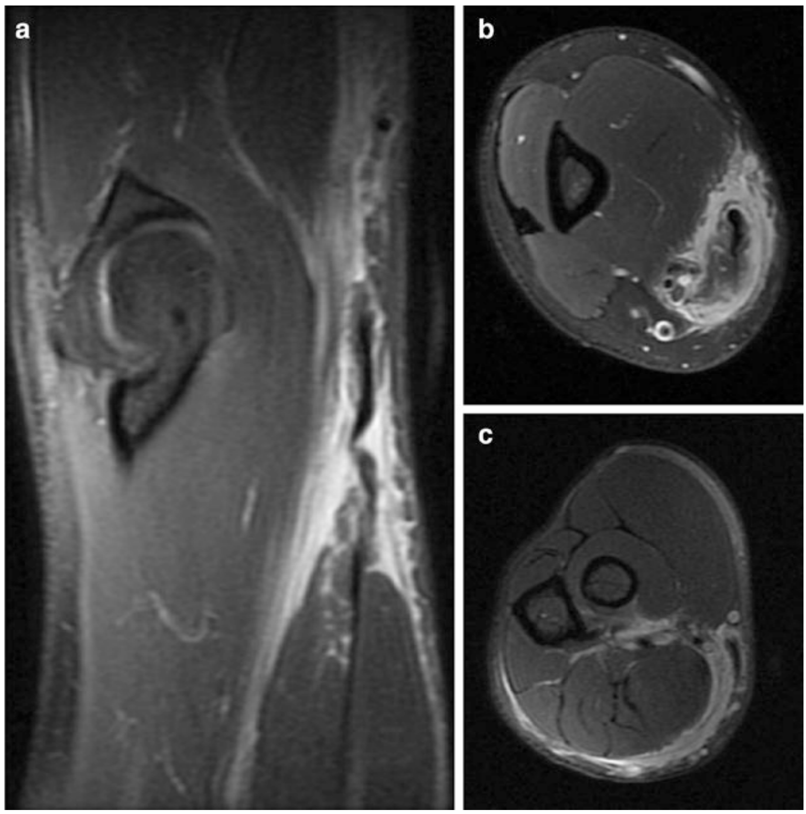


Fig. 35.17 Sagittal (**a**) and Axial (**b** and **c**) PD Fat Sat elbow MRI. Complete rupture of the distal bicipital tendon insertion with proximal retraction

35.13.2 Epicondylitis

It is a tendinosis (overuse syndrome) of the common flexor-pronator tendon group of the elbow (medial) or extensor tendon (lateral). MR imaging and ultrasound may be performed for diagnosis [38].

35.13.3 Ligament Injuries

The two main causes of elbow ligament injuries are trauma and overuse. Lateral collateral ligament injuries are generally associated with trauma, especially fracture or dislocation, and activities such as falls or forced twisting of the arm. Medial collateral ligament injuries are generally more associated with overuse.

35.13.4 Fractures

Recognizing an elbow joint effusion on lateral radiographs is a key finding that should be used to search for a fracture or an occult fracture in the elbow joint (sail sign). Avulsion fractures are common in young sports athletes (Fig. 35.18).

35.14 Foot and Ankle

The foot and ankle are commonly injured at all levels of sport and are particularly vulnerable in agility sports. Routine radiographic series, ultrasound, and MRI take a place in the imaging evaluation of the foot. MRI of the foot to address the specific area of clinical concern, ideally, should be focused to the region of interest, whether the hindfoot, midfoot, or forefoot, so that protocols can be optimized to permit small field of view and high-resolution imaging. Clinical information is crucial to choose the best protocol [39, 40].

35.14.1 Fatigue Fracture

The most common stress fracture in athletes occurs in the tarsal navicular and metatarsal bones.

Conventional radiographs are relatively insensitive to detect fatigue fracture. MRI allows early visualization of stress-related marrow edema, which may be accompanied by parosteal soft tissue edema (Fig. 35.19) [3, 40–42].

35.14.2 Ligament Injuries

The ankle joint is one of the most important and mobile joints in the human body. Sports involving rapid and frequent changes of direction, jumping, and contact with opposing players represent a greater risk.

Sprains and ligament injuries of the ankle and foot are among the most frequent of all injuries. The lateral ligament complex of the ankle is affected in 85% of cases. Other less frequent lesions are fractures, contusions, fascitis, tendon injuries, dislocations, and bursitis.

Lisfranc ligament complex sprain injuries in the athlete are relatively common, particularly among those who practice football, basketball,

Fig. 35.18 (**a**) Lateral radiograph of the elbow. The anterior fat pad becomes elevated due to joint effusion appearing as a triangle. This has been named the sail sign. X-ray (**b**), Coronal PD Fat Sat MRI (**c**), and Coronal T1 MRI (**d**) of the elbow with avulsion fracture of the medial epicondyle

and gymnastics; they can be a cause of substantial time off in the sport [43–45]. T2-weighted images on MRI show injured ligament thickened with increased internal signal (Fig. 35.19) [46].

35.14.3 Morton Neuroma

A symptomatic Morton neuroma is relatively common in athletes and can mimic plantar plate injuries of the metatarsals. Ultrasound in Morton

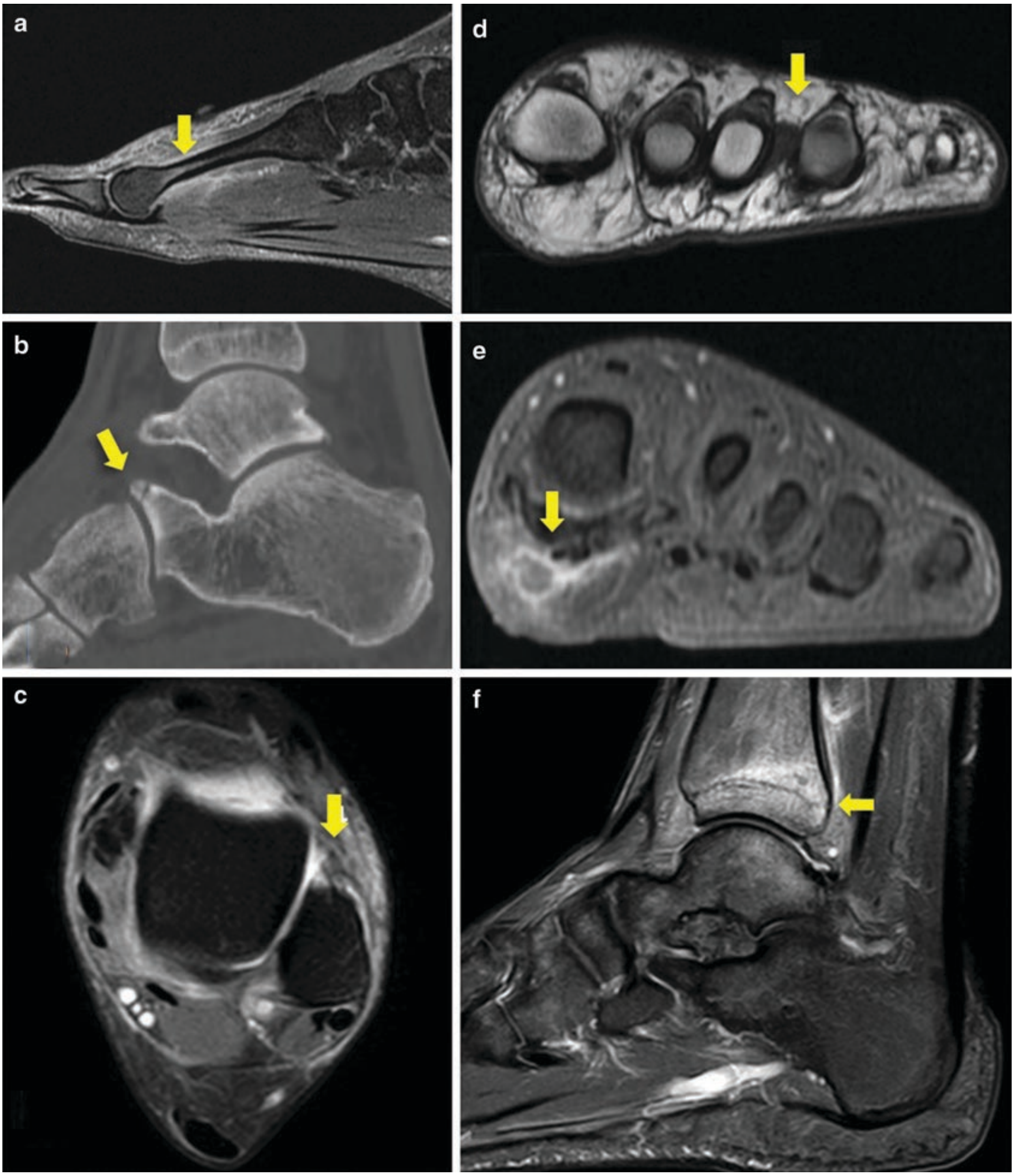


Fig. 35.19 Ankle and foot injuries (arrows). (**a**) fatigue fracture. (**b**) Anterior process of calcaneus fracture. (**c**) Anterior talofibular ligament injury. (**d**) Morton's neuroma. (**e**) Adventitious bursitis. (**f**) medullary fracture in the distal metaphysis of the tibia and edema in the body of the talus (fatigue fracture)

neuroma is typically a fusiform-ovoid hypoechoic thickening along the line of the common plantar digital nerve toward the distal margin of the interdigital space. On MRI, Morton neuroma is often most conspicuous on the short-axis T1-weighted sequence (Fig. 35.19) [47].

35.14.4 Metatarsalgia

Capsuloligamentous injury, plantar plate degeneration tear, adventitious bursitis, sesamoiditis, and synovitis are best evaluated through MRI (Fig. 35.19).

35.14.5 Osteochondritis Dissecans (OCD)

This condition is the end result of the aseptic separation of an osteochondral fragment with a gradual fragmentation of the articular surface. Direct trauma is associated with 96% of lateral lesions and 62% of medial lesions in the talus. It results in an osteochondral defect, and it is often associated with intra-articular loose bodies. Early findings in plain radiographs include subtle flattening or indistinct radiolucency about the cortical surface. As the process progresses, more pronounced contour abnormalities, fragmentation, and density changes (both lucency and sclerosis) become evident. If an osteochondral fragment becomes unstable and displaced, then donor site and intra-articular fragment may be seen. CT has the advantage of sectional imaging through the joint and multiplanar reformats. Findings are similar to those seen on plain radiographs. MRI is the test of choice, with high sensitivity (92%) and specificity (90%) in the detection of separation of the osteochondral fragment. This is essential in determining management (Fig. 35.20) [48].

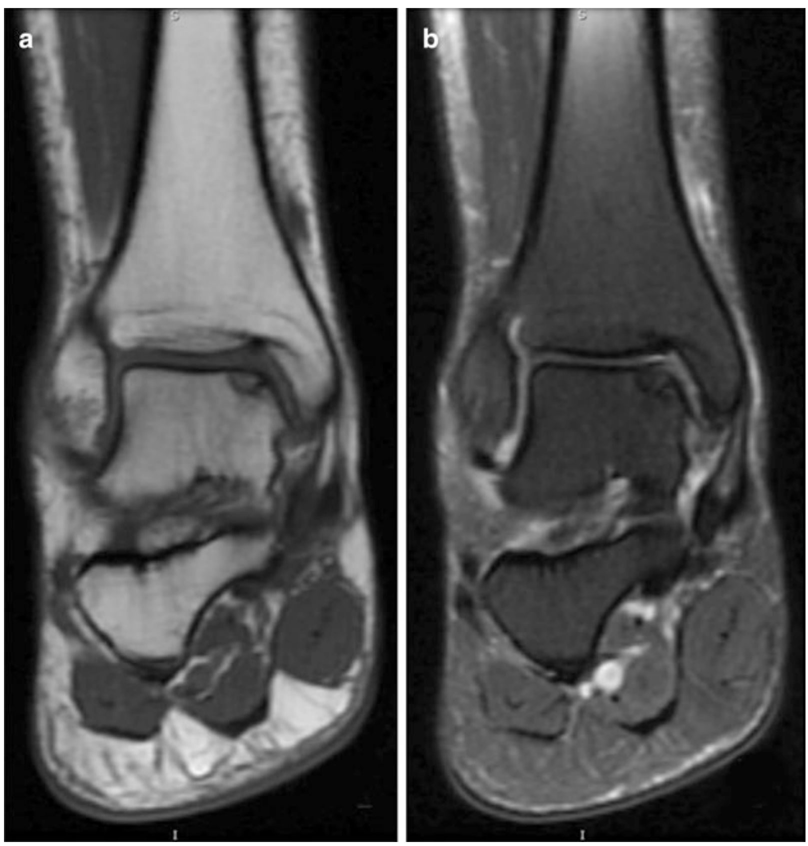


Fig. 35.20 Osteochondral defect in talar domus. Coronal T1 (**a**) and coronal PD Fat Sat (**b**) MRI of the ankle

35.14.6 Achilles Tendon Tear and Tendinosis

Achilles tendon tears are the most common ankle tendon injuries and are most commonly seen secondary to sports-related injury. The spectrum of tears ranges from microtears, interstitial tears, partial tears, and eventually to complete tears. Tears can be acute or chronic, with repeated minor trauma. At the mildest end of the spectrum all that may be present is peritendonitis.

Plain radiographs may show soft tissue swelling and obliteration of pre-calcaneal fat pad (Kager's triangle). Ultrasound can be useful to differentiate between a partial and full-thickness tear. Partial-thickness tears show an enlargement of the tendon. Full-thickness tears often show separation of the torn ends with a contour change of the tendon, and there is acoustic shadowing at the margins of the tear. Appearances can vary on MRI. A full-thickness tear often shows a tendinous gap filled with edema or blood. Retraction of tendon ends are seen when there is a complete rupture. Partial-thickness or interstitial tears may show high signal on T2WI (Fig. 35.21) [49].

35.14.7 Plantar Fasciitis

Plantar fasciitis refers to the inflammation of the plantar fascia of the foot. It is reported in different sports, mainly in running and soccer athletes. It is considered the most common cause of heel pain. Passive dorsiflexion of the toes may exacerbate discomfort [50].

The plantar fascia extends from the calcaneus to the distal part of metatarsophalangeal joints of each toe and is divided into central, medial, and lateral sections. The central section is the strongest component [51].

Plain radiograph features are unspecific. They may show an associated plantar calcaneal spur. The thickness of the fascia can be increased to more than 4.5 mm. Ultrasound is often the initial imaging modality of choice as increased thickness and a hypoechoic fascia can be seen on it. Positive findings on MRI are signal changes of the affected tissues. Fluid sensitive sequences are highly sensitive in the detection of both fascial and perifascial edema, which appear as poorly marginated areas of high-signal intensity. Other MRI features include plantar fascial thickening, increased T2/STIR signal intensity of the proximal plantar fascia, edema of the adjacent fat pad

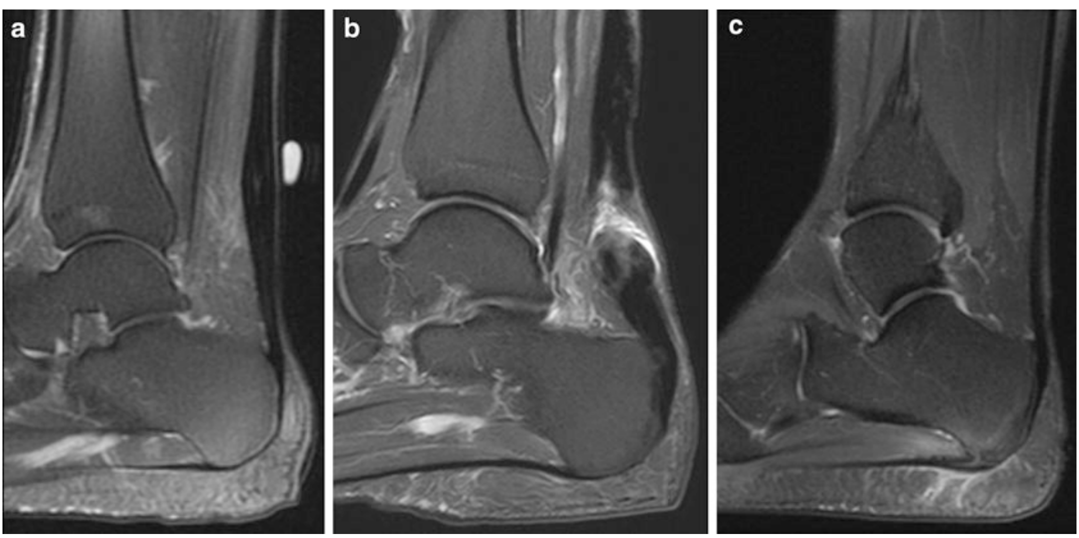


Fig. 35.21 T2-weighted sagittal MRI images. (**a**) Aquilles tendinosis. (**b**) Aquilles complete rupture. (**c**) Plantar fasciitis

and underlying soft tissues and limited marrow edema within the medial calcaneal tuberosity (Fig. 35.21) [52, 53].

35.15 Shoulder

The shoulder is the joint that articulates the upper arm with the thorax, involving broad types of movements and a wide range of mobility. Therefore, it is required in many sports modalities with overhead activities such as basketball, baseball pitching, cricket bowling, volleyball, swimming, and weightlifting.

Its lesions are related to traumatic events or repetitive movements that result in rotator cuff injuries, impingement, dislocation, labral lesions, instability, and nerve entrapments.

35.15.1 Rotator Cuff Injuries

Right before tearing, the tendons of the rotator cuff may pass through tendinopathy or a tendinosis process unleashed by traumatic events or repetitive stress associated to impingement. On ultrasonography, rotator cuff tendinopathy can appear as a thickened and hyperechoic image of the tendons, representing its edema. MRI can bring more information showing thickening of the tendons and high signal, but lower than fluid signal, on T2 Fat Sat or DP Fat Sat sequences.

As the lesions progress, they can end up as tears that are commonly classified as partial-thickness or full-thickness tears, depending on if the lesion affects the full width of the tendon. They can be seen on ultrasonography as hypoechoic lesions on the tendons and as a high signal similar to joint effusion or intermediary signal that stands for granulation tissue on MRI.

Full-thickness tears can be appreciated on MRI as a discontinuity of the attachment of the tendons that can be accompanied of tendon retraction and/or muscle womb atrophy (Fig. 35.22).

35.15.2 Instability

The shoulder is one of the most unstable joints and is responsible for 50% of all cases of subluxation in the body. The glenohumeral instability is often subdivided in anterior, posterior, or multidirectional. It can be also separated according to its mechanism: TUBSr, Traumatic, Unidirectional instability and Bankart lesion, or AMBRI, Atraumatic, Multidirectional, Bilateral instability [54].

35.15.3 Traumatic Instability

Most of the shoulder dislocations occur anteriorly (95% of the cases), usually disrupting in chronic instability due to capsule-labral compromise due to forced abduction, external rotation, and extension. This can occur especially if one of the main shoulder stabilizers, the inferior glenohumeral ligament, is lesioned, triggering chronic instability.

In athletes, they are by far more prevalent in young adult men who sustained high-energy

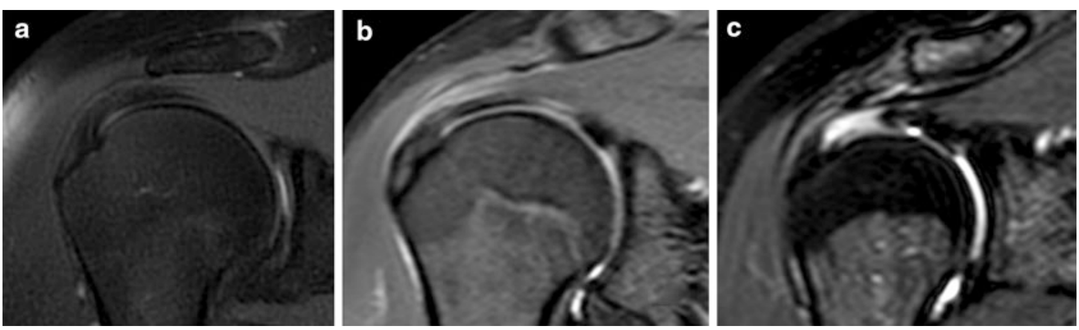


Fig. 35.22 Coronal T2 Fat Sat MRI of the supraspinatus tendon. (**a**) Normal. (**b**) Tendinosis. (**c**) Full-thickness tear

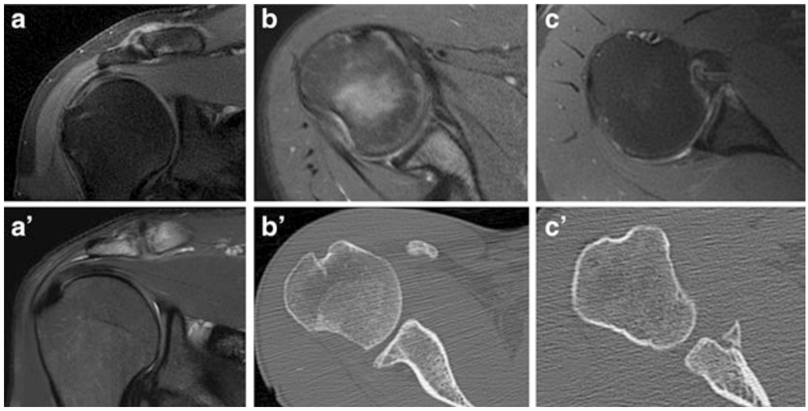


Fig. 35.23 Shoulder injuries. (**a**) Acromioclavicular injury. (**a′**) Distal clavicular osteolysis. (**b**) Hill-Sachs lesions (MRI). (**b′**) Hill-Sachs lesions (CT). (**c**) Osseous Bankart lesion (MRI). (**c′**) Osseous Bankart lesion (CT)

shoulder lesions; plain radiography can usually show some fractures or indicatives of anterior subluxation, but in some cases the MRI or MRI arthrography shows us more detailed information [55].

35.15.4 Bankart and Other Labral Lesions in Instability

Labral tears and other lesions are commonly seen in athletes exposed to repetitive overhead activities, increasing the risk of fibroligamentous injuries in which 39% show instability symptoms.

Bankart lesions are the most common labral injury, usually associated to trauma, that causes instability. The classic Bankart lesions are characterized by anteroinferior fibrocartilaginous labral injuries, mostly seen in association with Hill-Sachs lesions, a posterolateral humeral head compression fracture. There is also the Osseous Bankart when the same labral lesion exists with an avulsed inferior glenoid fragment.

The glenoid labrum cannot be seen on plain radiograph or CT, but it can suggest labral lesion if there is a fracture at the anteroinferior aspect of the glenoid. MRI will show frank displacement of the anterior glenoid labrum or a linear high-signal T2/PD indicating tear (Fig. 35.23).

Regarding Hill-Sachs lesion, it can be hard to see on plain radiograph, but it is better detected in internal rotation after shoulder relocation as a sclerotic line on the top of the humeral head toward the shaft, as a wedge defect if there is a large lesion. It is well seen on CT or MRI, both very sensitive, showing a region of flattening or wedge-shaped on the posterolateral humeral head (Fig. 35.23) [56, 57].

35.15.5 Superior Labral Anterior Posterior Lesion

The Superior Labral Anterior Posterior (SLAP) tears are superior glenoid labrum injuries, where the long head of the bicep tendon inserts, but it can extend to anterior or posterior. It is caused by the fall onto outstretched arm mechanism (FOOSH), and it can be seen on MRI arthrography as fluid, extending into the superior labrum and through, sometimes entering the bicep tendon [58, 59].

35.15.6 External Impingement Syndromes

External impingement syndromes that result from abnormal contact between the humeral head and other extra-articular structures like the acromion and the coracoid process is divided into two types [60].

Subacromial impingement is the most common type of external impingement syndrome. They can be caused by normal variants of acromioclavicular diseases, related to acromial shape types III (hooked) and keeled acromion (spur formation) and can disrupt on rotator cuff tears. Os acromiale is believed to contribute with impingement, tending to move inferiorly with deltoid contraction. The acromioclavicular joint apparently does not contribute to impingement since their osteoarthritic changes are common, and 65% of the cases are asymptomatic [60]).

The normal variants can be easily seen on plain radiograph; other commemoratives like subacromial bursitis, supraspinatus tendinopathy, and tears that can be present in impingements are distinctive on ultrasonography or MRI [61].

35.15.7 Acromioclavicular Injury

It is defined as a lesion of the acromioclavicular joint and adjacent structures, varying from sprain to complete disruption, almost always due to trauma after direct blow of FOOSH mechanism.

These lesions can be seen on plain radiograph as superior displacement of the distal clavicle widening and as a coracoclavicular increased distance. Swelling can accompany them, and it is always important to remember that comparing the lesion to the contralateral side can help (Fig. 35.23) [62].

35.15.8 Distal Clavicular Osteolysis

Distal clavicular osteolysis involves the acromioclavicular joint and is an important cause of shoulder pain, especially after trauma or repetitive microtrauma in sports like weightlifting.

It can be seen as osteopenia on plain radiograph in the distal clavicle, losing the articular cortical margin; swelling may be present, too [63].

A shoulder coronal view on MRI T1 sequence, showing a degenerative acromioclavicular joint with initial losing of the clavicular margin, characterizes distal clavicular osteolysis (Fig. 35.23).

35.15.9 Spine Injuries

The spine problems are relatively frequent in athletes. Chronic spine problems are much more common compared to acute injuries. Acute injuries are more common in high-speed and full contact sports and are traumatic in origin. Most of the acute injuries are minor, but some can be severe and catastrophic. Imaging is essential to the diagnosis and care of spinal trauma. Plain radiographs and CT are used for detecting vertebral fracture, and MR imaging for evaluating muscular, ligamentous, discal, and spinal cord injury [64–67].

35.15.10 Degenerative Spine Disease and Disc Herniations

Participation in sports can be a risk factor for the development of disc degeneration at an early age [56]. Degenerative changes are more common in athletes than in non-athletes in the general population, and this relationship seems to be more evident in professional or high-level athletes [64]. There is a consensus diagnostic terminology, category and subcategory classification, and reporting of imaging studies in lumbar disc herniation (Fig. 35.24) [68].

Spondylolysis.

Spondylolysis is a condition characterized by defects in the pars interarticularis of the posterior elements of the vertebrae. If the defect is bilateral and complete, spondylolisthesis can occur. Young athletes have a significantly higher risk of spondylolysis. Sports like gymnastics, weightlifting, and football are associated with a higher degree of spondylolysis. In many cases, there are no clinical symptoms in the early phase of the disease (Fig. 35.25) [69, 70].

Acute traumatic injuries.

Evaluating conventional images of the spine not only consists of assessing the height of the individual vertebrae but also the alignment of the spine. Any distortion or unexpected disruption of these contours should raise suspicion of injury. Small avulsion lesions anteriorly or posteriorly with otherwise normal alignment of the vertebrae can indicate underlying ligamentous or other soft tissue injuries (Fig. 35.25).

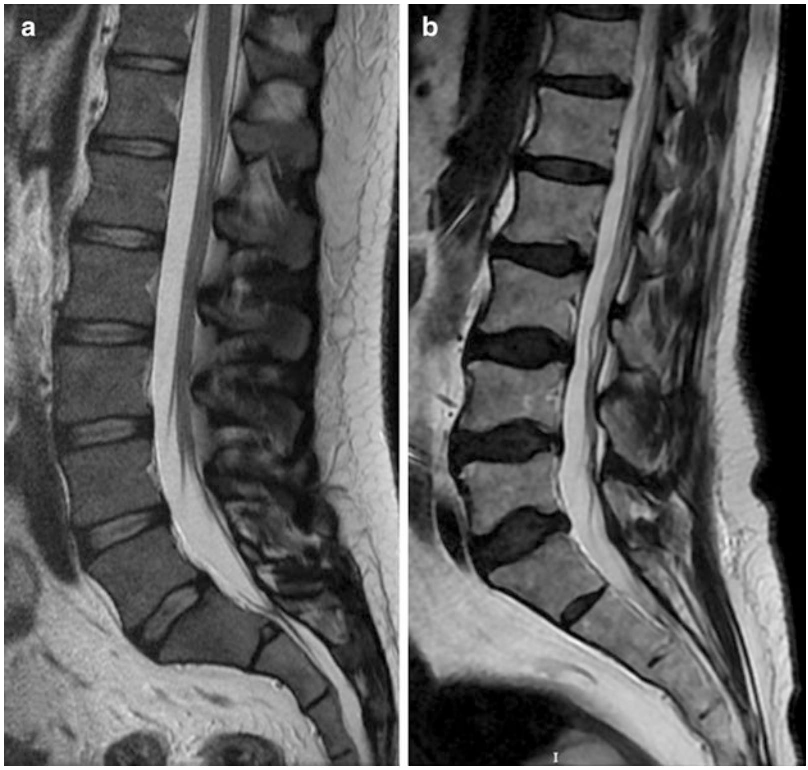


Fig. 35.24 Sagittal T2 MRI of the lumbar spine. (**a**) Disc hypersignal (normal disc hydration). (**b**) Disc hyposignal—degenerative disc disease (disc hypohydration)

Fig. 35.25 Lumbar spine. (**a**) Sagittal CT (L2 fracture). (**b**) Sagittal and (**b′**) Axial CT (bilateral L5-S1 spondylolysis)

35.16 Face and Head Trauma

Plain radiograph has poor sensitivity for detection facial fractures. CT is fast, and despite the large overlap of bone structures in this region, it is highly accurate to detect fractures. CT is also used for the first evaluation of intracranial lesions which may be present in sports injuries [71].

35.17 Muscle Injury

35.17.1 Muscle Injuries

Muscle injuries are among the most frequent lesions in the sporting and athletic population. It still remains a concern for athletes, coaches, managers, and sports physicians, leading to a loss of training time and withdrawal from competitions. Despite of their frequency and recurrence, there is still a lack of uniformity in the categorization, description, and grading of muscle injuries [72].

An adequate classification of muscle injury is essential for a full understanding of the injury and optimization of its treatment and return-to-play process. Determining when a player is ready to return to play after a muscle injury is challenging because the recovery from injury is highly variable [73]; premature return to play may be a factor in the observed high reinjury rates and prolonged time loss [74, 75].

Most classification systems classify lesions into three degrees, and there is still a lack of diagnostic accuracy as these systems provide limited prognostic information to the clinician [76].

An ideal classification system should include non-ambiguous terms, be easily applied, and describe objective findings that are clearly demonstrable and have clinical value and prognostic validity for clinicians, trainers, and athletes [77, 78].

In 2012, 15 international experts in muscle injuries and sports medicine organized a consensus meeting with the endorsement of the International Olympic Committee (IOC) and the Union of European Football Associations (UEFA and produced the "Munich Muscle Injury Classification") [79]. This system has the valuable merit of having been clinically validated in terms of prognostic value for specific injuries and was the first classification validated by a large research on muscle injuries [80].

At the end of 2013, the Italian Society of Muscle, Ligament and Tendons (ISMuLT) released the "ISMuLT Guidelines for muscle injuries" [81], combining the Munich Classification with the anatomical location of the injury in the case of structural injuries.

The British Athletics Muscle Injury Classification proposed a new system, in 2014 [82], which should provide a diagnostic base for therapeutic decision-making and prognostication. Injuries are graded 0–4 based on MRI features.

Finally, in 2015, the medical team of FC Barcelona, Aspetar, and the FIFA Medical Centre for Excellence (Duke Sports Science Institute) proposed an original comprehensive system named the "MLG-R Classification" [83]. This system describes injuries based on the direct "D" or indirect "I" *mechanism* (M), proximal "p," middle "m," or distal "d" *location* (L) in the case of direct injuries, and involvement of tendon "T," muscle-tendon junction "J," or muscle periphery "F" in the case of indirect injuries (followed by proximal "p" or distal "d" location). The severity of the injury is also evaluated through a 0–4 *grading scale* (G) of cross-sectional area involvement in MRI images, ideally at 24–48 h following injury. Nonstructural grade 0 injury has been associated with quicker return to sport [84, 85]. Muscle-tendon junction may be associated with more prolonged and different rehabilitation requirements than a peripheral myofascial injury, and injury within the tendon is associated with a poorer prognosis [86, 87]. Finally, the first or *recurrent condition* (R) is described as first episode "R0," first reinjury "R1," second reinjury "R2," and so on. A recurrence is defined as an injury of the same type and location occurring during the first 2 months after return to full competition. Reinjury is an important predictor for a longer recovery period compared with first-time injury [88]. With MLG-R, these authors offer the possibility of describing the injury, its location, grading, and chronological evolution.

Muscle Injury Classification—Adapted from [82, 83]:

Mechanism (M)

1. DIRECT (D)
   Proximal location (**p**); Middle location (**m**); Distal location (**d**)
2. INDIRECT (I)
   STRETCHING TYPE (subindex "s") and SPRINTING-TYPE (subindex "p").
3. **Structure** Involved
   (a) Tendon Involvement (T)
   Proximal location (**Tp**); Distal location (**Td**).
   (b) Muscle-Tendon Junction Involvement or within the Muscle Belly (J)
   Proximal location (**Jp**); Distal location (**Jd**);
   (c) Muscle Periphery Fibers—Myofascial (F)
   Proximal location (**Fp**); Distal location (**Fd**).

Grading Scale (G)

- **Grade 0: Muscle soreness**.
- **Grade 0a: Focal neuromuscular injury with normal MRI.**
- **Grade 0b**: **Generalized muscle soreness with normal MRI or MRI characteristic of DOMS** (generalized, patchy high-signal change affecting several muscles).

Grade 1: Small muscle tears

- **Grade 1a:**
- **High-signal changes in myofascial or within muscle periphery fibers**
  - No greater than 10% of the muscle cross-sectional area.
  - Longitudinal length of less than 5 cm within the muscle.
- Evidence of fiber disruption of less than 1 cm.
- Intermuscular fluid/hematoma on MRI may be evident within the fascial planes over a greater distance.
- **Grade 1b**
- **High-signal changes in muscle-tendon junction or within the muscle belly**
  - No greater than 10% of the muscle cross-sectional area at its maximal site.
  - Longitudinal length of less than 5 cm within the muscle.
- Evidence of fiber disruption of less than 1 cm.

Grade 2: Moderate muscle tears

- **Grade 2a**
- **High-signal changes in myofascial or within muscle periphery fibers**
  - Measure between 10% and 50% of the muscle cross-sectional area.
  - Longitudinal length between 5 and 15 cm within the muscle.
- Evidence of fiber disruption will be less than 5 cm.
- **Grade 2b**
- **High-signal changes in muscle-tendon junction or within the muscle belly**
  - Measure between 10% and 50% of the muscle cross-sectional area.
  - Longitudinal length between 5 and 15 cm within the muscle
- Evidence of fiber disruption will be less than 5 cm.
- **Grade 2c**
- **High-signal changes into the tendon**
  - Injury within the tendon is evident over a longitudinal length of less than 5 cm.
  - Injury within the tendon is evident less than 50% of the maximal tendon diameter on axial images.

Grade 3: Extensive muscle tears

- **Grade 3a**
- **High-signal changes in myofascial or within muscle periphery fibers.**
  - Greater than 50% of the muscle cross-sectional area.
  - Greater than 15 cm in length.
- Architectural fiber disruption which is likely to be greater than 5 cm.
- **Grade 3b**
- **High-signal changes in muscle-tendon junction or within the muscle belly**
  - Greater than 50% of the muscle cross-sectional area.
  - Greater than 15 cm in length.
- Architectural fiber disruption which is likely to be greater than 5 cm.
- **Grade 3c**
- **High-signal changes into the tendon**
  - Injury within the tendon is evident over a longitudinal length greater than 5 cm.

– Injury within the tendon is evident greater than 50% of the maximal tendon diameter on axial images.
- There is no evidence of a complete defect, but there may be loss of the usual straight margins and tendon tension suggesting some loss of the tendon integrity.

Grade 4: Complete muscle tears.
- **Injuries are complete tears to the muscle** (grade 4).
- **Injuries are complete tears to the tendon** (grade 4c).
- A palpable gap will often be felt.

ReInjuries (R)
- 0 (zero): First **episode.**
- 1 (one): First **reinjury.**
- 2 (two): Second **reinjury.**
- 3 (three): Third **reinjury.**
- 4 (four): Fourth **reinjury.**

Take-Home Message
- Although in a vast majority of sports-related injury, clinical evaluation allows establishing the diagnosis and strategy of treatment, in such cases, the radiological assessment is vital in elucidating confirming the diagnosis of involved structures in the injury.
- Conventional X-ray is still the modality of choice in the initial evaluation of any musculoskeletal complaint. It has a high resolution for bone structures, wide availability and low cost, but low resolution for soft tissues.
- Computed tomography allows a multiplanar study and plays a fundamental role in the evaluation of places where there are large overlaps of bone structures.
- Regarding a glass with wine, if our intention is to evaluate the wine, we should request MRI, whereas, if we want to analyze the glass, we should request computed tomography.
- MRI is the method of choice for the evaluation of ligament, tendinous, bursal, muscular, and disc injuries related to the musculoskeletal system, and it is very useful in the analysis of the bone marrow.

References

1. Nicolini AP, de Carvalho RT, Matsuda MM, Filho JS, Cohen M. Common injuries in athletes' knee: experience of a specialized center. Acta Ortop Bras. 2014;22:127–31. https://doi.org/10.1590/1413-78522014220300475.
2. Berger FH, de Jonge MC, Maas M. Stress fractures in the lower extremity. The importance of increasing awareness amongst radiologists. Eur J Radiol. 2007;62:16–26. https://doi.org/10.1016/j.ejrad.2007.01.014.
3. Marshall RA, Mandell JC, Weaver MJ, Ferrone M, Sodickson A, Khurana B. Imaging features and management of stress, atypical, and pathologic fractures. Radiographics. 2018;38:2173–92. https://doi.org/10.1148/rg.2018180073.
4. Boles CA, Lee JA. The knee: breaking the MR reflex. Semin Roentgenol. 2005;40:338–57. https://doi.org/10.1053/j.ro.2005.01.017.
5. Stevens MA, El-Khoury GY, Kathol MH, Brandser EA, Chow S. Imaging features of avulsion injuries. Radiographics. 1999;19:655–72. https://doi.org/10.1148/radiographics.19.3.g99ma05655.
6. Gorbachova T, Melenevsky Y, Cohen M, Cerniglia BW. Osteochondral lesions of the knee: differentiating the Most common entities at MRI. Radiographics. 2018;38:1478–95. https://doi.org/10.1148/rg.2018180044.
7. Barrie HJ. Osteochondritis dissecans 1887-1987. A centennial look at König's memorable phrase. J Bone Joint Surg Br. 1987;69:693–5.
8. Diederichs G, Issever AS, Scheffler S. MR imaging of patellar instability: injury patterns and assessment of risk factors. Radiographics. 2010;30:961–81. https://doi.org/10.1148/rg.304095755.
9. Brandser EA, Riley MA, Berbaum KS, El-Khoury GY, Bennett DL. MR imaging of anterior cruciate ligament injury: independent value of primary and secondary signs. Am J Roentgenol. 1996;167:121–6. https://doi.org/10.2214/ajr.167.1.8659355.
10. Rodriguez W, Vinson EN, Helms CA, Toth AP. MRI appearance of posterior cruciate ligament tears.

AJR Am J Roentgenol. 2008;191:1031. https://doi.org/10.2214/AJR.07.2921.
11. Roach CJ, Haley CA, Cameron KL, Pallis M, Svoboda SJ, Owens BD. The epidemiology of medial collateral ligament sprains in young athletes. Am J Sports Med. 2014;42:1103–9. https://doi.org/10.1177/0363546514524524.
12. Bollier M, Smith PA. Anterior cruciate ligament and medial collateral ligament injuries. J Knee Surg. 2014;27:359–68. https://doi.org/10.1055/s-0034-1381961.
13. Somford MP, Janssen RPA, Meijer D, Roeling TAP, Brown C, Eygendaal D. The Pellegrini-Stieda lesion of the knee: an anatomical and radiological review. J Knee Surg. 2019;32:637–41. https://doi.org/10.1055/s-0038-1666867.
14. Miller TT, Gladden P, Staron RB, Henry JH, Feldman F. Posterolateral stabilizers of the knee: anatomy and injuries assessed with MR imaging. Am J Roentgenol. 1997;169:1641–7. https://doi.org/10.2214/ajr.169.6.9393184.
15. Naraghi AM, White LM. Imaging of athletic injuries of knee ligaments and menisci: sports imaging series. Radiology. 2016;281:23–40. https://doi.org/10.1148/radiol.2016152320.
16. Khan KM, Bonar F, Desmond PM, Cook JL, Young DA, Visentini PJ, et al. Patellar tendinosis (jumper's knee): findings at histopathologic examination, US, and MR imaging. Radiology. 1996;200:821–7. https://doi.org/10.1148/radiology.200.3.8756939.
17. El-Khoury GY, Wira RL, Berbaum KS, Pope TL, Monu JUV. MR imaging of patellar tendinitis. Radiology. 1992;184:849–54. https://doi.org/10.1148/radiology.184.3.1509078.
18. McLoughlin RF, Raber EL, Vellet AD, Wiley JP, Bray RC. Patellar tendinitis: MR imaging features, with suggested pathogenesis and proposed classification. Radiology. 1995;197:843–8. https://doi.org/10.1148/radiology.197.3.7480766.
19. 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;30:127–31. https://doi.org/10.1007/s002560000319.
20. Klauser AS, Martinoli C, Tagliafico A, Bellmann-Weiler R, Feuchtner GM, Wick M, et al. Greater trochanteric pain syndrome. Semin Musculoskelet Radiol. 2013;17:43–8. https://doi.org/10.1055/s-0033-1333913.
21. Litwin DEM, Sneider EB, McEnaney PM, Busconi BD. Athletic Pubalgia (sports hernia). Clin Sports Med. 2011;30:417–34. https://doi.org/10.1016/j.csm.2010.12.010.
22. Byrd JWT. Femoroacetabular impingement in athletes: current concepts. Am J Sports Med. 2014;42:737–51. https://doi.org/10.1177/0363546513499136.
23. Jäger M, Wild A, Westhoff B, Krauspe R. Femoroacetabular impingement caused by a femoral osseous head-neck bump deformity: clinical, radiological, and experimental results. J Orthop Sci. 2004;9:256–63. https://doi.org/10.1007/s00776-004-0770-y.
24. Tannast M, Siebenrock KA, Anderson SE. Femoroacetabular impingement: radiographic diagnosis—what the radiologist should know. Am J Roentgenol. 2007;188:1540–52. https://doi.org/10.2214/AJR.06.0921.
25. Nouh MR, Schweitzer ME, Rybak L, Cohen J. Femoroacetabular impingement: can the alpha angle be estimated? Am J Roentgenol. 2008;190:1260–2. https://doi.org/10.2214/AJR.07.3258.
26. Prioreschi T, Abdullah W, Sala L. Della. Tecniche di radiologia convenzionale e TC nell'impingement di anca, guidate da uno studio biomeccanico applicato. J Biomed Pract. 2018:2. https://doi.org/10.13135/2532-7925/2712.
27. Prioreschi T, Sala L. Della, Agresti M, Coriasco M. Diagnostica radiologica dell'articolazione coxofemorale: la corretta tecnica per una diagnosi efficace. Case report. J Biomed Pract. 2019:3. https://doi.org/10.13135/2532-7925/3377.
28. Dudda M, Albers C, Mamisch TC, Werlen S, Beck M. Do normal radiographs exclude Asphericity of the femoral head-neck junction. Clin Orthop Relat Res, vol 467, Springer New York. 2009;467:651–9. https://doi.org/10.1007/s11999-008-0617-5.
29. Kassarjian A, Yoon LS, Belzile E, Connolly SA, Millis MB, Palmer WE. Triad of MR arthrographic findings in patients with cam-type femoroacetabular impingement. Radiology. 2005;236:588–92. https://doi.org/10.1148/radiol.2362041987.
30. Changstrom BG, Brou L, Khodaee 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;43:26–33. https://doi.org/10.1177/0363546514562739.
31. Alizai H, Engebretsen L, Jarraya M, Roemer FW, Guermazi A. Elbow injuries detected on magnetic resonance imaging in athletes participating in the Rio de Janeiro 2016 summer Olympic games. J Comput Assist Tomogr. 2019;43:981–5. https://doi.org/10.1097/RCT.0000000000000929.
32. Dines JS, Bedi A, Williams PN, Dodson CC, Ellenbecker TS, Altchek DW, et al. Tennis injuries: epidemiology, pathophysiology, and treatment. J Am Acad Orthop Surg. 2015;23:181–9. https://doi.org/10.5435/JAAOS-D-13-00148.
33. Goodman AD, Raducha JE, DeFroda SF, Gil JA, Owens BD. Shoulder and elbow injuries in NCAA football players, 2009–2010 through 2013–2014. Phys Sportsmed. 2019;47:323–8. https://doi.org/10.1080/00913847.2018.1554167.
34. Keogh JWL, Winwood PW. The epidemiology of injuries across the weight-training sports. Sport Med. 2017;47:479–501. https://doi.org/10.1007/s40279-016-0575-0.
35. Zouzias IC, Hendra J, Stodelle J, Limpisvasti O. Golf injuries: epidemiology, pathophysiology, and treatment. J Am Acad Orthop Surg. 2018;26:116–23. https://doi.org/10.5435/JAAOS-D-15-00433.

36. Falchook FS, Zlatkin MB, Erbacher GE, Moulton JS, Bisset GS, Murphy BJ. Rupture of the distal biceps tendon: evaluation with MR imaging. Radiology. 1994;190:659–63. https://doi.org/10.1148/radiology.190.3.8115606.
37. Lobo LDG, Fessell DP, Miller BS, Kelly A, Lee JY, Brandon C, et al. The role of sonography in differentiating full versus partial distal biceps tendon tears: correlation with surgical findings. Am J Roentgenol. 2013;200:158–62. https://doi.org/10.2214/AJR.11.7302.
38. Walz DM, Newman JS, Konin GP, Ross G. Epicondylitis: pathogenesis, imaging, and treatment. Radiographics. 2010;30:167–84. https://doi.org/10.1148/rg.301095078.
39. Linklater JM. Imaging of sports injuries in the foot. Am J Roentgenol. 2012;199:500–8. https://doi.org/10.2214/AJR.12.8547.
40. Umans HR. Imaging sports medicine injuries of the foot and toes. Clin Sports Med. 2006;25:763–80. https://doi.org/10.1016/j.csm.2006.06.006.
41. Stanitski CL, McMaster JH, Scranton PE. On the nature of stress fractures. Am J Sports Med. 1978;6:391–6. https://doi.org/10.1177/036354657800600615.
42. Kiuru MJ, Niva M, Reponen A, Pihlajamäki HK. Bone stress injuries in asymptomatic elite recruits: a clinical and magnetic resonance imaging study. Am J Sports Med. 2005;33:272–6. https://doi.org/10.1177/0363546504267153.
43. Meyer SA, Callaghan JJ, Albright JP, Crowley ET, Powell JW. Midfoot sprains in collegiate football players. Am J Sports Med. 1994;22:392–401. https://doi.org/10.1177/036354659402200316.
44. Mulcahy H. Lisfranc Injury: Current Concepts. Radiol Clin North Am. 2018;56:859–76. https://doi.org/10.1016/j.rcl.2018.06.003.
45. Shapiro MS, Wascher DC, Finerman GAM. Rupture of Lisfranc's ligament in athletes. Am J Sports Med. 1994;22:687–91. https://doi.org/10.1177/036354659402200518.
46. Perrich KD, Goodwin DW, Hecht PJ, Cheung Y. Ankle ligaments on MRI: appearance of normal and injured ligaments. Am J Roentgenol. 2009;193:687–95. https://doi.org/10.2214/AJR.08.2286.
47. Zanetti M, Strehle JK, Zollinger H, Hodler J. Morton neuroma and fluid in the intermetatarsal bursae on MR images of 70 asymptomatic volunteers. Radiology. 1997;203:516–20. https://doi.org/10.1148/radiology.203.2.9114115.
48. Tol JL, Struijs PAA, Bossuyt PMM, Verhagen RAW, Van Dijk CN. Treatment strategies in osteochondral defects of the talar dome: a systematic review. Foot Ankle Int. 2000;21:119–26. https://doi.org/10.1177/107110070002100205.
49. Schweitzer ME, Karasick D. MR imaging of disorders of the achilles tendon. Am J Roentgenol. 2000;175:613–25. https://doi.org/10.2214/ajr.175.3.1750613.
50. League AC. Current concepts review: plantar fasciitis. Foot Ankle Int. 2008;29:358–66. https://doi.org/10.3113/FAI.2008.0358.
51. Benjamin M. The fascia of the limbs and back - a review. J Anat. 2009;214:1–18. https://doi.org/10.1111/j.1469-7580.2008.01011.x.
52. Berkowitz JF, Kier R, Rudicel S. Plantar fasciitis: MR imaging. Radiology. 1991;179:665–7. https://doi.org/10.1148/radiology.179.3.2027971.
53. Cardinal E, Chhem RK, Beauregard CG, Aubin B, Pelletier M. Plantar fasciitis: Sonographic evaluation. Radiology. 1996;201:257–9. https://doi.org/10.1148/radiology.201.1.8816554.
54. Thomas SC, Matsen FA. An approach to the repair of avulsion of the glenohumeral ligaments in the management of traumatic anterior glenohumeral instability. J Bone Jt Surg - Ser A. 1989;71:506–13. https://doi.org/10.2106/00004623-198971040-00005.
55. Robinson G, Ho Y, Finlay K, Friedman L, Harish S. Normal anatomy and common labral lesions at MR arthrography of the shoulder. Clin Radiol. 2006;61:805–21. https://doi.org/10.1016/j.crad.2006.06.002.
56. Farrar NG, Malal JJG, Fischer J, Waseem M. An overview of shoulder instability and its management. Open Orthop J. 2013;7:338–46. https://doi.org/10.2174/1874325001307010338.
57. Waldt S, Burkart A, Imhoff AB, Bruegel M, Rummeny EJ, Woertler K. Anterior shoulder instability: accuracy of MR arthrography in the classification of anteroinferior labroligamentous injuries. Radiology. 2005;237:578–83. https://doi.org/10.1148/radiol.2372041429.
58. Burkhart SS, Morgan C. Slap lesions in the overhead athlete. Orthop Clin North Am. 2001;32:431–41. https://doi.org/10.1016/S0030-5898(05)70212-1.
59. Modarresi S, Motamedi D, Jude CM. Superior labral anteroposterior lesions of the shoulder: part 1, anatomy and anatomic variants. Am J Roentgenol. 2011;197:596–603. https://doi.org/10.2214/AJR.10.7236.
60. Chang EY, Moses DA, Babb JS, Schweitzer ME. Shoulder impingement: objective 3D shape analysis of acromial morphologic features. Radiology. 2006;239:497–505. https://doi.org/10.1148/radiol.2392050324.
61. Cowderoy GA, Lisle DA, O'Connell PT. Overuse and impingement syndromes of the shoulder in the athlete. Magn Reson Imaging Clin N Am. 2009;17:577–93. https://doi.org/10.1016/j.mric.2009.06.003.
62. Girish G, Lobo LG, Jacobson JA, Morag Y, Miller B, Jamadar DA. Ultrasound of the shoulder: asymptomatic findings in men. Am J Roentgenol. 2011;197:W713. https://doi.org/10.2214/AJR.11.6971.
63. Seymour EQ. Osteolysis of the clavicular tip associated with repeated minor trauma to the shoulder. Radiology. 1977;123:56. https://doi.org/10.1148/123.1.56.
64. Barile A, Limbucci N, Splendiani A, Gallucci M, Masciocchi C. Spinal injury in sport. Eur J Radiol. 2007;62:68–78. https://doi.org/10.1016/j.ejrad.2007.01.017.

65. De Jonge MC, Kramer J. Spine and sport. Semin Musculoskelet Radiol. 2014;18:246–64. https://doi.org/10.1055/s-0034-1375568.
66. Maxfield BA. Sports-related injury of the pediatric spine. Radiol Clin N Am. 2010;48:1237–48. https://doi.org/10.1016/j.rcl.2010.07.006.
67. Mhuircheartaigh NN, Kerr JM, Murray JG. MR imaging of traumatic spinal injuries. Semin Musculoskelet Radiol. 2006;10:293–307. https://doi.org/10.1055/s-2007-972000.
68. Fardon DF, Williams AL, Dohring EJ, Murtagh FR, Gabriel Rothman SL, Sze GK. Lumbar disc nomenclature: Version 2.0 Recommendations of the combined task forces of the North American Spine Society, the American Society of Spine Radiology and the American Society of Neuroradiology Spine J 2014;14:2525–2545. https://doi.org/10.1016/j.spinee.2014.04.022.
69. Belfi LM, Ortiz AO, Katz DS. Computed Tomography Evaluation of Spondylolysis and Spondylolisthesis in Asymptomatic Patients. vol. 31. n.d.
70. Randall RM, Silverstein M, Goodwin R. Review of pediatric Spondylolysis and Spondylolisthesis. Sports Med Arthrosc. 2016;24:184–7. https://doi.org/10.1097/JSA.0000000000000127.
71. Piedade SR, Carvalho LM, Mendes LA, Possedente M, Ferreira DM. Facial Trauma. In: Rocha Piedade S, Imhoff AB, Clatworthy M, Cohen M, Espregueira-Mendes J, editors. Sport. Med. Physician, Cham: Springer International Publishing; 2019, p. 261–273. https://doi.org/10.1007/978-3-030-10433-7_20.
72. Freckleton G, Pizzari T. Risk factors for hamstring muscle strain injury in sport: a systematic review and meta-analysis. Br J Sports Med. 2013;47:351–8. https://doi.org/10.1136/bjsports-2011-090664.
73. Ekstrand J, Healy JC, Waldén M, Lee JC, English B, Hägglund M. Hamstring muscle injuries in professional football: the correlation of MRI findings with return to play. Br J Sports Med. 2012;46:112–7. https://doi.org/10.1136/bjsports-2011-090155.
74. Malliaropoulos N, Isinkaye T, Tsitas K, Maffulli N. Reinjury after acute posterior thigh muscle injuries in elite track and field athletes. Am J Sports Med. 2011;39:304–10. https://doi.org/10.1177/0363546510382857.
75. Verrall GM, Slavotinek JP, Barnes PG, Fon GT, Esterman A. Assessment of physical examination and magnetic resonance imaging findings of hamstring injury as predictors for recurrent injury. J Orthop Sports Phys Ther. 2006;36:215–24. https://doi.org/10.2519/jospt.2006.36.4.215.
76. Grassi A, Quaglia A, Canata GL, Zaffagnini S. An update on the grading of muscle injuries: a narrative review from clinical to comprehensive systems. Joints. 2016;4:39–46. https://doi.org/10.11138/jts/2016.4.1.039.
77. Tol JL, Hamilton B, Best TM. Palpating muscles, massaging the evidence? An editorial relating to terminology and classification of muscle injuries in sport: the Munich consensus statement. Br J Sports Med. 2013;47:340–1. https://doi.org/10.1136/bjsports-2012-091849.
78. Warren P, Gabbe BJ, Schneider-Kolsky M, Bennell KL. Clinical predictors of time to return to competition and of recurrence following hamstring strain in elite Australian footballers. Br J Sports Med. 2010;44:415–9. https://doi.org/10.1136/bjsm.2008.048181.
79. Mueller-Wohlfahrt HW, Haensel L, Mithoefer K, Ekstrand J, English B, McNally S, et al. Terminology and classification of muscle injuries in sport: the Munich consensus statement. Br J Sports Med. 2013;47:342–50. https://doi.org/10.1136/bjsports-2012-091448.
80. Ekstrand J, Askling C, Magnusson H, Mithoefer K. Return to play after thigh muscle injury in elite football players: implementation and validation of the Munich muscle injury classification. Br J Sports Med. 2013;47:769–74. https://doi.org/10.1136/bjsports-2012-092092.
81. Maffulli N, Nanni G. ISMuLT skeletal muscles injuries Guidelines. Muscles Ligaments Tendons J. 2013;3:240. https://doi.org/10.11138/mltj/2013.3.4.240.
82. Pollock N, James SLJ, Lee JC, Chakraverty R. British athletics muscle injury classification: a new grading system. Br J Sports Med. 2014;48:1347–51. https://doi.org/10.1136/bjsports-2013-093302.
83. Valle X, Alentorn-Geli E, Tol JL, Hamilton B, Garrett WE, Pruna R, et al. Muscle injuries in sports: a new evidence-informed and expert consensus-based classification with clinical application. Sport Med. 2017;47:1241–53. https://doi.org/10.1007/s40279-016-0647-1.
84. Fournier-Farley C, Lamontagne M, Gendron P, Gagnon DH. Determinants of return to play after the nonoperative Management of Hamstring Injuries in athletes. Am J Sports Med. 2016;44:2166–72. https://doi.org/10.1177/0363546515617472.
85. Kerkhoffs GMMJ, van Es N, Wieldraaijer T, Sierevelt IN, Ekstrand J, van Dijk CN. Diagnosis and prognosis of acute hamstring injuries in athletes. Knee Surgery, Sport Traumatol Arthrosc. 2013;21:500–9. https://doi.org/10.1007/s00167-012-2055-x.
86. Connell DA, Schneider-Kolsky ME, Hoving JL, Malara F, Buchbinder R, Koulouris G, et al. Longitudinal study comparing sonographic and MRI assessments of acute and healing hamstring injuries. Am J Roentgenol. 2004;183:975–84. https://doi.org/10.2214/ajr.183.4.1830975.
87. Cross TM, Gibbs N, Houang MT, Cameron M. Acute quadriceps muscle strains: magnetic resonance imaging features and prognosis. Am J Sports Med. 2004;32:710–9. https://doi.org/10.1177/0363546503261734.
88. Ekstrand J, Hägglund M, Waldén M. Epidemiology of muscle injuries in professional football (soccer). Am J Sports Med. 2011;39:1226–32. https://doi.org/10.1177/0363546510395879.

36 Patient-Reported Outcomes Tailored to Sports Medicine

Sérgio Rocha Piedade, Mark R. Hutchinson, and Nicola Maffulli

36.1 Introduction

In all areas of Medicine, physicians must be aware of patients' voice and opinions because patients' observations about their treatment play an essential role in developing and refining a management plan.

Adequate tools should allow to collect data on the treatment received from the patient's point of view. These tools allow to analyze and compare outcomes, boost changes and improvements in treatment plans and are named Patient-Reported Outcome Measures—PROMs [1–4].

In the literature, different PROMs have been validated and used in clinical practice. *However, it is critical to recognize the apparent void in a condition-specific PROM in the field of sports medicine. If the demands and expectations of the athletic population are uniquely different than those of the general population, then a more targeted patient-reported instrument may be necessary to assess the valid outcomes in this population* [5].

This chapter aims to discuss PROM in Sports Medicine, starting by its definition, contributions in Orthopedics, the lack of PROM in Sports Medicine, differences between athletes and high-active sports practitioners versus ordinary people, the development and implementation of a new PROM, the need of a PROM tailored for Sports Medicine, benefits of PROM designed specifically for Sports Medicine, and, finally, our philosophy of a PROM tailored for Sports Medicine.

36.2 The Definition of PROM

Historically, the concept of patient-reported outcomes was very simple assessments that included a strong potential of clinician's bias in the interpretation of the outcomes. Do you believe your outcome was poor, average, good, or excellent? Are you better than you were before surgery? Are you satisfied with your result and would you do it again? Over time, these assessments have become more complex, tested for validity, and better represent an unbiased patient's perspective. According to the National Quality Forum, patient-reported outcomes are "any report of the status of a patient's health condition that comes directly from the patient, without interpretation of the patient's response by a clinician or anyone

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else" [6]. A key goal of patient-reported outcomes is to remove the potential bias that a clinician might misinterpret patient's feedback and assess it as better or worse than the patient had intended. Patient-reported outcomes can be used in concert with physiologic, mechanical, and imaging measures to provide a more holistic assessment of success for the patient.

A patient-reported outcome measure (PROM) is the tool or instrument used to measure patient-reported outcome. The instruments are often completed independently by the patient to assess and evaluate functional status, health-related quality of life, symptoms and symptom burden, personal experience of care, and health-related behaviors such as anxiety or depression [7]. PROMs can be broad and general, or more focused and disease/condition-specific. Indeed, the use of both a broad and a focused PROM can be complementary. The broader PROM can allow comparisons across conditions which allow for an assessment of the impact of treatments on a health-care system. More focused or condition-specific PROMs are designed to assess more targeted symptoms and functions related to a specific condition or body part. This gives disease-specific PROMs more face validity and credibility but lower ability to compare across diseases. Examples of broad-based PROMs include the EuroQol EQ-5D, which measures health-related quality of life, the SANE which measures treatment outcomes, the Veteran's RAND 12, the PROMIS, SF-12, and the SF-36. Examples of disease-specific PROMs in orthopedic surgery [8] include the following:

1. **Foot and Ankle**
   - Foot and Ankle Ability Measure (FAAM)
   - Foot and Ankle Disability Index (FADI)
2. **Knee (Anterior Cruciate Ligament)**
   - International Knee Documentation Committee (IKDC) Subjective Knee Form (Pedi-IKDC)
   - Marx Activity Rating Scale
3. **Knee (Osteoarthritis)**
   - Knee Injury and Osteoarthritis Outcome Score (KOOS)
   - Knee Injury and Osteoarthritis Outcome Score Jr. (KOOS Jr.)
4. **Hip (Osteoarthritis)**
   - Hip Disability and Osteoarthritis Outcomes Survey (HOOS)
   - Hip Disability and Osteoarthritis Outcomes Survey Jr. (HOOS Jr.)
5. **Shoulder**
   - American Shoulder and Elbow Surgeons Standardized Shoulder Assessment Form (ASES)
   - Oxford Shoulder Score (OSS)
6. **Shoulder (Instability)**
   - American Shoulder and Elbow Surgeons Standardized Shoulder Assessment Form (ASES)
   - Western Ontario Shoulder Instability Index (WOSI)
7. **Elbow**
   - Disabilities of the Arm, Shoulder, and Hand Score (DASH)
   - Quick-DASH
8. **Wrist**
   - Disabilities of the Arm, Shoulder, and Hand Score (DASH)
   - Quick-DASH
9. **Hand**
   - Disabilities of the Arm, Shoulder, and Hand Score (DASH)
   - Quick-DASH
10. **Spine**
    - Oswestry Disability Index (ODI)
    - Neck Disability Index (NDI)

36.3 The Importance and Contribution of PROM in Orthopedics

All clinicians who care for patients have taken some variation of the Hippocratic Oath, which is founded on the core concept that a physician will first be focused on the needs and benefits of the individual patient. The assessment of the benefits to patients is critical to guide which treatment and its length, or which treatment modality best accomplishes this goal.

Historically, outcomes have largely been assessed from the clinician's perspective. Our internal medicine physician colleagues can measure the therapeutic level of a drug without assessing functional improvement, side effects, and overall satisfaction of the patient. They might measure the survivorship of a cancer drug without assessing the struggles the patient had to go through to get there.

For orthopedic surgeons, outcome assessment includes numerous physical and mechanical measurements including the range of motion, muscle strength, ligament/joint stability, and interpretation of images. For example, success or failure after a total knee arthroplasty can be documented by measurements of overall alignment or radiographic loosening. The success after an anterior cruciate ligament reconstruction can be measured by instrumented testing of the ligament with a KT-1000 device.

While important contributors to functional outcome of a musculoskeletal injury, they may not always represent patient's satisfaction or outcomes from a patient's perspective. Even the measures of successful return-to-work or return-to-play may not *express* the pain, effort, or satisfaction that patients may express in accomplishing those goals. Over time, based on patient's demands, the need for better quality assessment, and guidelines from government and payor organizations, there has been a redirection of how we measure success in the field of orthopedics and musculoskeletal Medicine. Lately, the central role of patient-reported assessment using validated measures has been acknowledged, contrasting with the assessment, and potential bias, performed solely by clinicians.

36.4 The Lack of a PROM Tailored for SPORTS Medicine

In clinical practice, outcome measures are commonly used as helpful tools to assess the risks and gains of treatment; however, these tools focus mainly on performance-based test, and not on capturing data of general aspects of patient's life that could be impacted by injury and treatment [9]. Moreover, the status of health has a broad definition and must involve physical, mental, and social well-being instead of only an illness or disability condition [10].

It would be important to use and implement a ***tailored*** *instrument* ***to capture the athletes and highly active sports practitioners' perception*** *of the whole treatment* process that allows a more comprehensive approach of how injury and treatment may affect their **lives** such as family and social roles, daily living and sports activities, that is, the patient-report outcome measures (PROM) **in Sports Medicine.**

Moreover, PROMs may have considerable contributions in Sports Medicine, collecting data to clinical practice, research, and health-care policies, presenting a precise mechanism to manage treatment and decision-making.

Although PROMs have come to the forefront in the evaluation of the results of treatment for musculoskeletal ailments [11], recently a systematic review showed that the PROMs presently available have not been useful to evaluate, for example, postoperative outcomes in athletes and highly active sports practitioners. Moreover, this review showed that there is no uniformity on the type of scores commonly used to evaluate the postoperative outcomes of the same clinical problem, namely ACL injury [5].

36.5 Athletes and Highly Active Sports practitioner's Population Versus Ordinary People

"*One dream, one soul, one prize, one goal*," this explains athletes' thinking regarding their aims, demands, and expectations which differ from the ones of the general population. Therefore, since individual patients do not have uniform characteristics such as the same physical and psychological demands, why should PROMs consider these specific issues related to athletes and highly active sports practitioners? Athletes are not ordinary people, and, therefore, a more patient-targeted report instrument is necessary to assess valid outcomes in this population of athletes and highly active sports practitioners. Moreover, depending on the sports practiced, different physical demands, as well as anatomical sites, must be considered due to specific sports-related injuries—the pattern and type of injury differ [12, 13].

All of these points reinforce the fact that PROM questionnaires should be designed from the patients' perspective, enabling them to provide a clear answer to the posed question, providing the clinician and the researcher the opportunity to interpret and score the answers, taking into consideration all aspects that may change the physical and psychological expectations [14, 15].

36.6 Why Do We Need a PROM Tailored for Sports Medicine If There Are Already a Considerable Number of PROM Developed to Orthopedics?

The patients' perception of outcomes of treatment of sports injuries has become the focus of recent research efforts, in both medical and social sciences. Patient-reported outcome measures (PROMs) had become an integral part of the process of evaluation of outcomes in our field, as medical management should ultimately benefit patients and should take into account patients' perspectives.

In this respect, PROMs should allow giving a snapshot of patients' point of view on all aspects of clinical care, including surgery. In this way, global, disease-specific, and joint-specific outcomes can be explored.

36.7 Which Will Be the Benefits of a Designed-PROM for Sports Medicine?

A variety of PROMs routinely used in Orthopaedic Sports Medicine, including IKDC (knee ligament injuries), DASH (upper extremity), FAOS (foot and ankle), EQ-5D (health-related quality of life), Lysholm (knee ligament injury and TKA), KOOS (Total Knee Replacement), and HAAS (High-Activity Arthroplasty Score), have been scientifically validated and cross-culturally adapted [8]. However, they are anatomically specific, focus on a given joint or anatomical location, and do not take into account the needs of athletic populations. Indeed, the physical expectations and psychological goals sporting and physically active individuals differ from those of the general population, and the various sports require different physical needs [5, 16].

We, therefore, need there a PROM for athletes and high-performance athletes, taking into account their perception of the injury, their expectations of treatment; their evaluation of postoperative care and treatment received, and the outcomes compared to their pre-injury status [15–17].

36.8 Our Philosophy of a PROM Tailored for Sports Medicine

Recognizing the apparent void in a disease-specific PROM in the field of sports medicine, we have started working on the development of a PROM for Sports Medicine in the ISAKOS Sports Medicine Committee. The research line that has been adopted to develop the concept of a PROM for Sports Medicine was based on a target population, tool applicability, and PROM tailored for Sports Medicine.

36.8.1 Target Population

Our PROM was developed to evaluate the characteristics of athletes and highly active sports practitioners as well as physical and psychological specificities expectations *to record their opinion on the treatment received more accurately*.

36.8.2 Applicability

Our PROM aims to be a **reproducible, accessible, validated** tool to assess the outcomes of athletes and high-performing practitioners who suffered a sport injury. It includes the principles of patient-centered care, offering *clear information* which is easy to understand and to fill out, *to this target population.*

36.8.3 The Structured Design of 4-Domain PROM in Sports Medicine

Our PROM consists of 11 questions organized in four domains of two questions each. The first domain approaches the pre-injury status, the second the post-injury status, the third handles the athlete's expectations of treatment, and, finally, the fourth deals with their evaluations of postoperative care and outcomes.

The critical point is that there are three other questions to capture reported information on the level of sports practice of athletes and highly active sports practitioners, main physical demands in sports practice, and complaints after injury. It defines the personal baseline that will be used to answer eight questions graded from 0 to 10.

Moreover, this structured design allows its application to different sports injuries regardless of the anatomical site, **shoulder, elbow, wrist, hand, hip, knee, foot, and ankle—a universally applicable approach to sports injuries.**

Although this 4-Domain PROM does not give a final score, it allows to group and analyze athletes according to their sports modality, type of surgical procedure, and to compare the treatment outcomes, regardless of the injury site. Our PROM tailored for Sports Medicine is presented in the following.

36.8.4 Our PROM Tailored for Sports Medicine

36.8.4.1 First Domain: Uninjured Baseline Status (Patient's Report)

1. **Does sport activity influence your quality of life?**

0	1	2	3	4	5	6	7	8	9	10
None										High influence

2. Considering your **sports** modality, **what is your level of competition**?

Recreational	Regional	National	International
(1)	(2)	(3)	(4)

3. How **motivating** (**positive** activity) is **sport activity for you**?

0	1	2	3	4	5	6	7	8	9	10
None										High influence

4. What are **the main physical demands** in your sports activity?

Running	Kicking	Jumping	Changing direction	Acceleration/deceleration	Throwing	Others
(1)	(2)	(3)	(4)	(5)	(6)	(7)

Second DOMAIN—injury status (quality of life and sports performance).

5. How **much** did this **injury influence your quality of life**?

0	1	2	3	4	5	6	7	8	9	10
None										High influence

6. **Considering** the **main physical demand reported**, how much did this injury influence your **sports performance**?

0	1	2	3	4	5	6	7	8	9	10
None										High influence

7. What **were** your **most important physical complaints** (symptoms) after this injury?

Pain	Joint stability	Decrease of range of motion	Loss of strength
(1)	(2)	(3)	(4)

Third DOMAIN—patient's expectations.

8. After talking (**discussing**) with your doctor, did **you really understand** your injury?

0	1	2	3	4	5	6	7	8	9	10
No way										No doubt

9. Did you **expect to return to the same level of sport activity**?

0	1	2	3	4	5	6	7	8	9	10
No										I'm very confident

Fourth DOMAIN—Treatment and postoperative results.

10. How did **you analyze your postoperative care**?

0	1	2	3	4	5	6	7	8	9	10
Very bad										Excellent

11. **Regarding** your injury, how is **your feeling** (psychological status) **at the end of treatment** (final postoperative results)?

0	**1**	**2**	**3**	**4**	**5**	**6**	**7**	**8**	**9**	**10**
Very bad										**Excellent**

36.8.4.2 Final Considerations

In this chapter, we presented and discussed the philosophy and basis of our PROMs. However, we **may wish to acknowledge that this PROM tailored for Sports Medicine is a project that is a pending peer-reviewed publication** [18] (in press).

With this structured design PROM, the sports physician will have a comprehensive evaluation of athletes' care and outcomes before implementing adequate measures to produce continuous improvement in patient-centered care. Future advancements in patient care will need to be more attentive to the patient's perspective. We, as clinicians and providers, should take this challenge on simply: not only it is the right thing to do, but government agencies and payors will most assuredly insist we use both general and disease-specific PROMs to ensure that the treatments implemented in our athletes are both clinically effective and cost-efficient, optimizing patient outcomes, and satisfaction.

References

1. Ruzbarsky JJ, Marom N, Marx RG. Measuring quality and outcomes in sports medicine. Clin Sports Med. 2018;37(3):463–82.
2. Kyte DG, Calvert M, van der Wees PJ, ten Hove R, Tolan S, Hill JC. An introduction to patient-reported outcome measures (PROMs) in physiotherapy. Physiotherapy. 2015;101(2):119–25.
3. Dawson J, Doll H, Fitzpatrick R, Jenkinson C, Carr AJ. The routine use of patient reported outcome measures in healthcare settings. BMJ. 2010;c186:340.
4. Higginson IJ, Carr AJ. Measuring quality of life: using quality of life measures in the clinical setting. BMJ. 2001;322(7297):1297–300.
5. Rocha Piedade S, Hutchinson MR, Maffulli N. Presently PROMs are not tailored for athletes and high-performance sports practitioners: a systematic review. J ISAKOS Joint Disorders Orthop Sports Med. 2019;4:248–53.
6. FDA Guidance for Industry. Patient-reported outcome measures: use in medical product development to support labeling claims. 2009. Available at: http://www.fda.gov/downloads/Drugs/Guidances/UCM193282.pdf. Accessed September 25, 2013.
7. Weldring T, Smith SMS. Patient-reported outcomes and patient-reported outcome measures. Health Serv Insights. 2013;6:61–8.
8. https://www5.aaos.org/CustomTemplates/landingPage.aspx?id=4294968282&ssopc=1
9. Black N. Patient reported outcome measures could help transform healthcare. BMJ. 2013;f167:346.
10. Valderas JM, Kotzeva A, Espallargues M, Guyatt G, Ferrans CE, Halyard MY, et al. The impact of measuring patient-reported outcomes in clinical practice: a systematic review of the literature. Qual Life Res. 2008;17(2):179–93.
11. Davis JC, Bryan S. Patient Reported Outcome Measures (PROMs) have arrived in sports and exercise medicine: why do they matter? J Sports Med. 2015;49(24):1545–6. https://doi.org/10.1136/bjsports-2014-093707.
12. Franklyn M, Oakes B. Aetiology and mechanisms of injury in medial tibial stress syndrome: current and future developments. World J Orthop. 2015 Sep 18;6(8):577–89.
13. Murphy C, O'Connell JE, Kearns G, Stassen L. Sports-related maxillofacial injuries. J Craniofac Surg. 2015;26(7):2120–3.
14. Hutchings HA, Alrubaiy L. Patient-reported outcome measures in routine clinical care: the PROMise of a better future? Dig Dis Sci. 2017;62:1841–3.
15. Snyder AR, McLeod TCV, Sauers EL. Defining, valuing, and teaching clinical outcomes assessment in professional and post-professional athletic training education programs. Athl Train Educ J. 2007;2(2):31–41.
16. Valier AR, Jennings AL, Parsons JT, Vela LI. Benefits of and barriers to using patient-rated outcome measures in athletic training. J Athl Train. 2014;49(5):674–83.
17. Piedade SR, Ferretti M, Ferreira DM, Sullitel DA, Patnaik S, Samitier Maffulli N. PROMs in sports medicine. In: Rocha Piedade S, Imhoff A, Clatworthy M, Cohen M, Espregueira-Mendes J, editors. The sports medicine physician. Cham: Springer; 2019. https://doi.org/10.1007/978-3-030-10433-7_50.
18. Piedade SR, Hutchinson MR, Ferreira DM, Ferretti M, Maffulli N. Validation and Implementation of 4-Domain PROM tailored for Orthopedic and Sports Medicine. Int J Sports Med. 2021;42:1–6.