Spinal Conditions in the Athlete

A Clinical Guide to Evaluation, Management and Controversies

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I dedicate this book to my children, Savannah, Jaiden, Evie, and Eamon. Your curiosity and inquisitiveness drive my passion for research and the search for solutions to clinical problems.

To my wife and work/life partner, Erin, you have made and will continue to make everything possible in my life.

Wellington K. Hsu

To my Lord and Savior, Jesus Christ, for His infinite blessings. To Ashley, my wife, for her unending love and encouragement. To my children, Luke and Elizabeth, who inspire me and give me endless joy.

Tyler J. Jenkins

Preface

The purpose of this textbook is to provide a user-friendly guide that marries the strongest literature evidence with expert recommendations to provide a comprehensive overview of spine care in the elite athlete. While the data around sports science and performance outcomes has experienced a rapid growth in recent years, healthcare treatment and outcomes in athletes have lagged. Numerous external variables make performance of well-designed clinical trials challenging in this population. Despite these challenges, the rigorous demands of high-level performance have led athletes and physicians to question the typical management, outcomes, and return to play for spinal conditions and search for more individualized treatment.

In this book, we have gathered world leaders in the management of spine and brain injuries in the elite athlete and asked them to combine their practical experience with evidence-based research to fill a gap in the team physician's knowledge (spine or sports). Medicine has always been a combination of knowledge and art to achieve the highest individualized outcomes. The following pages will aid the treating physician in walking this line to provide the best patient-centered spine care.

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Part I
Team Physician Management of Spine and Brain Injuries

Chapter 1
On-Field Assessment and Management of Spine Injuries

Shawn Sahota and Bryan Kelly

Introduction

Spine injuries are relatively rare in sports, and a multifaceted approach should be utilized to further decrease rates of injury. Prevention via sport-specific regulations, coaching, and proper athletic technique is critical in lowering the number of adverse events. Still, when spinal injuries ensue, they can be devastating due to the potential for significant and long-standing morbidity. It is imperative to have a plan guiding management of the injured athlete, and preparing for these clinical scenarios starts long before the on-field assessment occurs.

Data from the National Spinal Cord Injury Statistical Center between 2010 and 2017 ranks sports/recreation activities as the fourth most common cause of spinal cord injury (SCI), with 8.9% of SCI happening during these events [1]. Perhaps more concerning is that SCI in athletes generally occurs at a younger age than other leading causes and sports activities are the second leading cause of SCI in patients under the age of 30 [2]. These acute sports-related traumatic injuries tend to occur in the cervical spine, where decreases in life expectancy and increases in total cost after injury are noted [1]. While sport-specific degenerative conditions (disk degeneration, herniation, spondylolysis, and others) are seen in the thoracolumbar spine, traumatic injuries in these locations are much less common and rarely seen in sports secondary to the innate stability provided by surrounding structures [3]. Therefore, the primary focus is toward assessing, stabilizing, and protecting the cervical spine; yet many of the same principles discussed directly apply to the thoracic and lumbar spine as the goals of care remain the same.

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While American football has certainly been in the spotlight when considering SCI in the athlete, other sports have also noted relatively high rates of injury to the spine. Hockey, rugby, skiing/snowboarding, diving, gymnastics, and equestrian sports also have spinal injuries, many with higher incidences than in American football [4–7]. However, the number of football players nationally is significantly higher than these other sports; thus it largely carries the stigma of SCI [8]. Changes in the rules by respective national sporting organizations have led to a reduction in the number of catastrophic injuries. The elimination of spear tackling (hitting an opponent headfirst with the head lowered creating substantial axial pressure on a slightly flexed cervical spine) in the National Football League (NFL) in 1976 has significantly decreased the number of spinal cord injuries [9]. In a 10-year span after the implementation of this rule, cervical quadriplegia decreased by over 75% [10, 11]. Likewise, the National Hockey League (NHL) has prohibited checking an opponent from behind as this high-risk activity had potential to lead to SCI when players are unexpectedly pushed head first into the ice rink boards with their head in a forward flexed position. In 2010, the NHL prohibited illegal checks to the head (lateral or blind side hits to an opponent where the player's head is targeted). This international rule change has helped decrease spinal cord injuries throughout hockey [12]. USA Hockey has keenly recognized the need for rule changes as well in order to protect young players. In 2011, USA Hockey changed the age of legal body checking from 12 to 14. Similar to the NFL and NHL, it is important that all sports create and maintain health advisory commissions to monitor, evaluate, and critically analyze sporting activities to find ways to alleviate risk of injury to participating athletes, particularly in regard to SCI. With the heightened awareness regarding concussion, committees such as these are being developed with the goal of preserving the integrity of respective sports while maintaining safety of the athlete.

Despite efforts to mitigate injuries through prevention and equipment innovation, injuries can still occur. Physicians, athletic trainers, coaches, and other personnel should employ a systematic approach to the injured athlete. Efficient, effective, and safe on-field evaluation and management is critical and can prevent further harm to players. The goal of the chapter will be to outline components of a successful pregame emergency plan as well as discuss on-field evaluation, stabilization, management, and transfer of the spine injured athlete.

Pregame Preparation

Planning for spine injuries in athletics starts well before the game. Given the severity and potential long-term ramifications of spine injuries, medical personnel must have developed protocols in place to ensure quality assessment, delivery of care, and transfer. An injury plan should detail who is responsible for care of the injured athlete, what supplies are needed and available in the event of an injury, and where tools (including sport-specific tools) to care for the injured athlete can be found, and

it should explicitly identify what the escalation of care policy is with emergency medical services as well as when escalation of care should occur.

In this capacity, planning starts with identifying medical personnel that will respond in the event of an injury. Oftentimes a team of six to eight people is needed to safely maneuver the injured athlete while maintaining spinal precautions. The head athletic trainer or team physician is frequently the designated leader of the response group. While a team effort is needed in appropriately caring for the athlete, a chain of command should be established, and the team leader should direct all communication to ensure safety and efficiency in providing care to the injured athlete. Practice runs should be performed routinely with the entire team giving each member a thorough understanding of his/her responsibility [13]. It is imperative to review protocols, procedures, and equipment when new members are added to the medical team to ensure consistency of approach should an emergency occur.

Medical personnel should be familiar with general and sport-specific injury patterns of the spine because this can help guide care when injuries happen. Additionally, knowledge and in-depth understanding of player equipment is required as this may have implications on delivery of care. Player equipment can routinely change, and equipment type, manufacturer, and technology are often not uniform across a team. It is the job of medical staff to be comfortable in handling and maneuvering all possible equipment options the players may have. Consequently, it is necessary to have proper instruments for an appropriate response to spine injuries. Necessary tools include those needed for spine stabilization (spinal backboard, C-collar, foam pads, towels, tape), airway management and life support (oral/nasal airway kits, automated external defibrillator (AED)), removal of equipment (trauma shears, screw driver, power tools, 18-gauge needle, wire cutters), and physical exam (reflex hammer, needle to check sensation, light source). All of these should be readily accessible to medical personnel at the time of injury.

The final step in preparation is creating an emergency action plan for escalation of care. This plan should be explicit and include logistical considerations such as identifying routes for emergency personnel, stretchers, and ambulances as it pertains to the specific event venue. The team medical staff should have a predetermined hospital that will be available if transport for emergency and/or definitive care of an injured athlete is required. Team physicians must be in communication with this hospital such that the hospital is aware of events and properly staffed to treat spinal cord or neurologic injuries. Ideally, when an injury occurs, a member of the on-field medical team is assigned to accompany the injured athlete during transport and in the hospital where more definitive care is determined. This provides clear and concise transfer of information to apprise medical staff not present at the time of injury. Familiarity of all medical personnel, from those on field to those in hospital, is valuable. At minimum, team medical staff, EMS, and hospital staff should meet annually to go over the emergency action plan. Furthermore, on-site medical personnel (trainers, physicians, and EMS) should briefly meet prior to each event and discuss the emergency action plan. This enables all participants in medical care to understand the plan as well as promotes communication among members of the team. Lastly, an important component of the emergency action plan is to

ensure athletes have updated emergency contacts who may be reached in the event of an injury [3]. While not always possible, it is also prudent to introduce players and families to all medical personnel to help alleviate angst in the event of an injury.

Pregame preparation is perhaps the most critical component to successful on-field management of the injured athlete as it dictates the response. It requires diligence and foresight to put a plan in place that will promote efficiency and safe care for the injured athlete.

On-Field Assessment

The initial injury assessment begins with visual and auditory observations of the injury event and the injured athlete. The early focus is on determining cardiorespiratory status and level of consciousness as these two factors guide care. Seeing the injury aids in development of a differential diagnosis as particular mechanisms are often associated with typical injury patterns. Subsequently, looking at body position, spontaneous or independent limb motion, and chest rise and listening for the athlete's ability to communicate or breathe will help in determining the level of acuity. Prior to any movement, it is crucial that a differential diagnosis is considered and spine injury is ruled out since moving the spine-injured athlete has the potential of harming the patient [14]. Communication with the athlete by asking simple questions such as "can you hear me?" or "are you ok?" is the first step when approaching. Any verbal response identifies a patent airway. Furthermore, even if the response is vague, it will help identify level of consciousness and give grounds for starting this assessment. In the event that the athlete is unconscious or altered, has bilateral neurological symptoms, and has midline spine pain or spinal deformity is present, medical personnel should assume a spine injury has occurred and take appropriate precautionary measures [3, 13, 15]. Even if these are not present, examiners should avoid focusing on a certain diagnosis prematurely and must remain diligent on the global assessment to ensure thorough evaluation. To this extent, it is recommended that the potential spine-injured athlete be treated using the Basic Life Support/Advanced Trauma Life Support (BLS/ATLS) protocol after preliminary immobilization of the spine [8], and assessment should systemically follow the "ABCDE" (airway, breathing, circulation, disability, and exposure) structure. Of note, this evaluation may be more difficult in sports requiring significant protective equipment, such as the helmeted athlete. The key is having appropriate tools and being facile in equipment removal to safely, efficiently, and effectively provide access to the airway and chest if need be. This will be discussed in more detail later. Regardless of the circumstances, when conducting the evaluation, it is important to maintain stability to the cervical spine.

During the initial moments of the exam, determining cardiorespiratory status is critical. If there is compromise, addressing airway, breathing, or circulation takes precedence, and emergency medical service personnel should be activated. An AED should also be immediately brought onto the playing surface. If the airway is not

patent, maneuvers to obtain and maintain an airway should be performed. The head tilt maneuver has been shown to alter alignment and force the spine into extension [16, 17]; therefore, a jaw thrust or oral/nasal airway is preferred to head tilt. Ventilation is often best accomplished using bag mask ventilation techniques. If needed, CPR must begin immediately as well. In the athlete with protective equipment, chest pads should be removed prior to CPR. Cutting the laces or straps on the chest pad with trauma shears, either at the sternum or axilla, will allow medical personnel the ability to spread pads open and gain access to the chest for manual compressions or AED placement. Consideration for an advanced airway (orotracheal intubation) while maintaining in-line cervical spine stabilization is reasonable, if necessary and if trained personnel are present. Airway establishment in this manner may provide less spinal manipulation than previously mentioned if done by skilled medical staff [18, 19]. Lastly, on-field physicians should be comfortable with surgical airways (tracheotomy) in the event that all other options fail. While performing respiratory and circulatory evaluations under the ATLS guidelines is a priority, all maneuvers should be done with diligence toward spinal precautions.

Once the initial evaluation is complete and normal cardiorespiratory status is confirmed, further assessment is determined based on level of consciousness, which can be established with the GCS assessment (evaluating eye opening, verbal response, and motor response) (Table 1.1). The GCS score is a useful, reliable, and objective way of monitoring neurologic status during the examination and has prognostic value. Patients with lower scores have a higher incidence of spinal injury [8, 13, 20]. A score of less than or equal to 7 is associated with a serious injury such as a cervical spine injury; conversely, a score greater than or equal to 11 is largely indicative of a good prognosis [21, 22]. If the athlete has an altered level of consciousness or is frankly unconscious, then spine stabilization and transfer using emergency services is the practical course of action. Finally, if the athlete maintains normal mentation and cardiorespiratory status, the examiner should perform a brief yet comprehensive history and physical exam to further evaluate disability and uncover neurologic or musculoskeletal injury. Spine stabilization should be instituted if neck pain, spinal column tenderness, spinal column deformity, neurologic deficits, or bilateral symptoms are present. Further management addressing symptoms is dictated by specific diagnosis, which will be discussed in later chapters. Having an algorithm to focus questions and exams is beneficial as they allow for rapid triage of the player with a spine injury. One such algorithm by Banerjee et al. is shown in Fig. 1.1.

Table 1.1 Glasgow coma scale

Score	Eye	Verbal	Motor
1	No response	No response	No response
2	Eyes open to pain	Incomprehensible/moaning	Extension response to pain
3	Eyes open to voice	Inappropriate	Flexion response to pain
4	Spontaneous eye opening	Confused/disoriented	Withdraws to painful stimuli
5		Oriented	Purposeful
6			Obedient/follows command

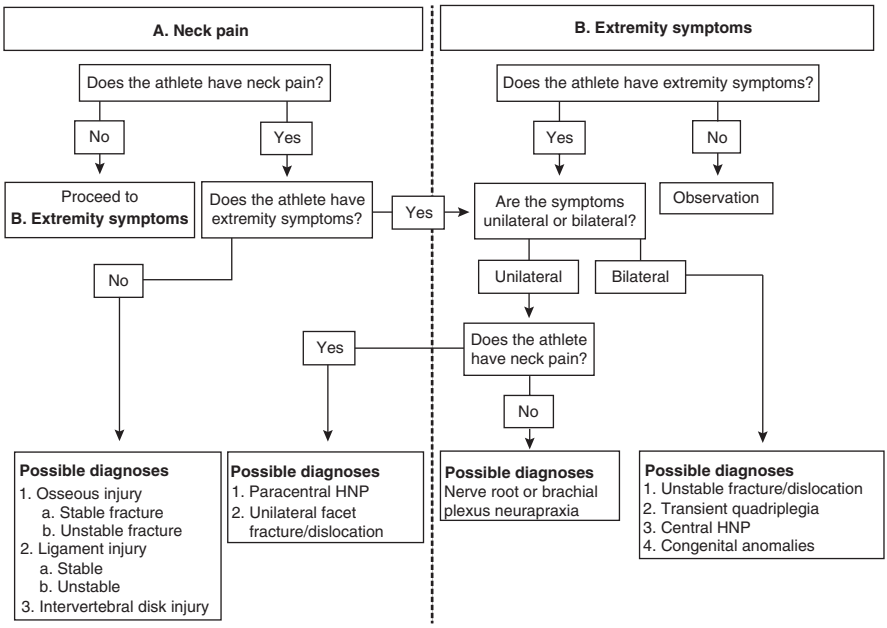

Fig. 1.1 On-field algorithm during evaluation of cervical spine injury based on neck pain or extremity symptoms. *HNP* herniated nucleus pulposus. (From: Banerjee et al. [42]. Reprinted with permission from Sage Publications)

Spine Stabilization and Transfer

Spine stabilization is essential to prevent further damage and starts during ATLS protocol after a potential spine injury has been identified. To accomplish this the spine is kept in neutral position and alignment with minimal motion. If the athlete's spine is not in a neutral position when initially approached, it is reasonable to gently move the athlete's head to create a more anatomic orientation; however, this change in position should cease if the athlete has increased pain or change in neurologic status during movement [13]. One should resist the urge to use distraction or traction as a means of reduction as this has been shown in several studies to lead to further damage [23, 24]. During the on-field evaluation, stabilization in a neutral alignment can be preliminary held manually by medical personnel. This is most effectively done by kneeling above the athlete while facing them and placing both hands under the head, thereby cupping the occiput with the palms and allowing the fingers to grasp over the mastoid processes [25]. This basic management of stabilization should be transitioned to a more definitive stabilization as soon as possible. Use of a hard cervical collar (c-collar) to maintain alignment is routine as it can quickly and easily be applied with minimal motion to the spine.

In the event that transport of the injured athlete is needed, transfer to a spine board is required. Multiple members of the medical team working in unison are

needed to maintain stability to the spine and minimize motion. There are two primary techniques of transferring a patient onto a spine board: lift and slide or logroll. For the supine athlete, the National Athletic Trainers' Association recommends the lift-and-slide technique [26] as it imparts less motion in the axial and coronal planes to the spine [27]. While this technique is preferred, it is not always logistically possible as it requires eight people: one person to take the lead (physician or trainer) and be at the head of the patient; three people should be on each side of the patient at shoulder, pelvis, and leg; and one person must be available to slide the board under the player. The leader will direct the remainder of the team to lift the athlete, in concert, approximately 6 inches. above the ground allowing the final person to slide the spine board beneath the athlete. Subsequently the athlete is lowered to the board in unison at the direction of the leader [28]. To perform the logroll technique, the leader (physician or trainer) should stand at the head of the patient, immobilize the spine as previously discussed, and direct all communication. The remaining assistants (at least three depending on the size of the athlete) should be on the side where the athlete is to roll to. They should reach across the body and securely grab the contralateral side of the athlete. Arms of the assistants should be crossed, thus allowing more stable points of fixation for turning. The leader should give a verbal count to allow for a smooth roll with all participants turning in unison. After the spine board is placed, the athlete may be rolled back onto it at the leader's direction. In both techniques, it is critically important that all participants take cues from the leader as the team must act in unison when performing the lift or logroll so as not to compromise stability of the spine. Once on the spine board, combinations of supportive straps from the board, foam padding, and blocks should be used to secure the head and shoulders to ensure minimal motion during transport.

Though injuries to the thoracolumbar spine occur with less frequency, the same principles apply during transfer. Limiting motion of the thoracic or lumber spine will mitigate risk for further injury. These areas of the spine should be secured in the spine board such that minimal motion in any plane is allowed. This can be accomplished by appropriately fastening two to three cross body straps around the spinal board. Additional use of towels, foam pads/blocks, and tape can aid in securing the torso.

Considerations for the Injured Athlete in the Prone Position

The injured athlete in the prone position requires unique considerations for evaluation and spinal stabilization. In the prone athlete, the logroll technique should be performed to flip the athlete supine. This allows access to the airway and will enable the examiner to proceed with the evaluation. Ideally in this scenario, logroll can be performed onto a spinal backboard to eliminate the need for additional movement later as this may inevitably be required. However, if the athlete is mentally altered, is unconscious, or has cardiorespiratory compromise, then logroll and assessment should not be delayed if the backboard is not readily available. Conversely, if the

athlete is conscious and communicating appropriately with medical personnel, it is reasonable to give pause prior to logroll allowing time for a spinal board to be brought onto the field and appropriately placed.

The logroll technique should be performed as previously described, with some modifications for the prone athlete. The leader should similarly stand at the head of the patient and direct all communication. In the prone athlete, the leader's arms will start crossed such that when the athlete is flipped supine, the arms will be uncrossed and can maintain stability of the cervical spine in a neutral position throughout the roll [29]. The remaining assistants should follow the same protocol as previously described. All participants should exclusively follow the direction of the leader as the logroll in the prone athlete requires a turn of up to 180° as opposed to 90° in the supine athlete for board placement, creating the opportunity for more motion during movement.

To diminish risk of iatrogenic injury from the roll, terms of the roll should be established prior to moving the athlete. It is our recommendation that turning begins at the command of the leader and is paused at 90°. This will allow a moment for the medical personnel to safely readjust their bodies as needed and ensure smooth, simultaneous transition to the supine position while maintaining spinal alignment. Once supine, the remainder of the evaluation can proceed as previously described.

Considerations for the Athlete in Protective Equipment

The athlete in protective equipment (helmet, face mask, shoulder pads, etc.) also requires special attention, as there may be obstacles to comprehensive assessment. Initial evaluation of airway, breathing, and circulation may be difficult in the helmeted athlete. Again, medical staff must have the appropriate tools and be facile in equipment removal to safely and efficiently provide access to the airway and chest if need be; preparation is paramount as proficiency of medical staff is most important [30–32]. An in-depth understanding of the equipment and implications of equipment removal is also required. Medical personnel should be take inventory prior to each season and ensure familiarity with all types of equipment and manufactures for player equipment.

If there is concern for SCI, the spine must be immediately stabilized by medical personnel in the same manner as if the helmet and other protective equipment were not present. The face mask should be promptly removed using appropriate tools that minimize or eliminate motion to the cervical spine. This mandates one person remains focused on maintaining cervical spine alignment, while a separate individual focuses on face mask removal. Studies have shown a cordless power screwdriver is the most efficient way to remove a face mask from a helmet and also reduces the amount of motion at the spine as compared to other tools [33, 34]. Medical personnel must be aware that there may be multiple points of fixation for the face mask; commonly, the use of a screwdriver for screw removal as well as a cutting tool for attachment loops is required. The helmet and shoulder pads should be left in place

as this combination can aid in maintaining spinal stability [35]. Situations that require removal of protective equipment are rare and only indicated if equipment is inhibiting adequate exposure in a timely manner, the spine is inadequately immobilized with the equipment in place, or the equipment is prohibitive to transport for further care. If removal is required, then both helmet and shoulder pads should be removed simultaneously as removing one can negatively affect alignment in the supine athlete as well as cause unnecessary motion to the spine [36–38]. For example, removing a helmet while leaving shoulder pads will enable the head to extend or hyperextend in a resting state; conversely, removing the shoulder pads while keeping the helmet can induce flexion or hyperflexion at the cervical spine.

Once the athlete in protective equipment is on a spinal board, immobilization remains necessary. In this instance, using foam pads, rolled towels or blankets, and tape will aid in restricting motion of the spine as spinal board straps will oftentimes not accommodate the head and shoulders with equipment in place. The use of hard cervical collars are not advised in the helmeted athlete as these rarely fit appropriately over the protective equipment and may cause excessive motion to the spine when applying [39].

Considerations for the Spine-Injured Pediatric Athlete

The pediatric athlete with a possible SCI requires certain nuances in evaluation. Factors related to pregame preparation or initial on-field assessment remains unchanged. Being aware of different types and brands of athletic equipment is important, particularly as there is often even less uniformity in this than older athlete cohorts. Having knowledge of advanced airways in the pediatric population is prudent. A key difference in the pediatric population is in regard to transfer on the spine board. Relative to the adult, children have a larger head to body ratio. This is of particular concern when considering resting position on a backboard during spine stabilization. Without modification to the standard adult spinal board, a child's head would rest in a flexed position at the cervical spine given this relative mismatch of head to body ratio [40]. Therefore, it is necessary to elevate the body relative to the position of the head. In response to this, pediatric-specific spine boards are available that provide a depression for the occiput or a padding that creates an elevation for the shoulders, thoracic spine, and lumbar spine. These boards are recommended for children 8 years of age and less [41].

Conclusion

Though the incidence of SCI in athletics remains low, these injuries can be catastrophic with significant morbidity and long-term disability. Prevention of these injuries must be the primary focus through sport-specific education, innovation in

protective equipment, and rule modifications. Medical personnel must be thoughtful and have a plan in place well before the injury occurs to effectively make an appropriate response. This plan must identify the "who, what, where, and when" questions to efficiently and completely care for the athlete with a spinal injury. For those sports that involve protective equipment, the plan must also account for this, and medical personnel should understand the equipment and be facile in its safe removal. Further, creating an emergency action plan and confirming that all contributors are actively engaged with the plan will help expedite care and save valuable time. Communication is vital to the success of the plan, and annual rehearsals will confirm that all members have a complete understanding of what to do when injuries transpire. Using a team approach will be needed, but the team leaders should take charge and direct the remaining medical personnel to ensure safety in delivery of care to the athlete and to prevent iatrogenic secondary injuries from occurring. Prioritizing spinal stabilization during on-field assessment should be standard, and careful consideration must be given to maintaining spinal stability during maneuvers necessary for resuscitation. While each injury is unique and circumstances surrounding the event change, a well-organized, well-rehearsed, and systematic response will help ensure safe and efficient care for the spine-injured athlete and will mitigate risk of further injury.

Expert's Opinion

Covering sporting events as a team physician can be exciting, exhilarating, and stressful all at the same time. There is a tremendous amount of pressure on the physician to make decisions regarding diagnosis and treatment following an injury. While the player, coaches, and parents may not have the athlete's health as a priority, it is the job of the team physician to exercise superior clinical judgment, knowledge, and ability. Team physicians must look out for the well-being of their patients (athletes) and place the needs of the patient first while providing evidence-based care.

Though experience is critical to understanding team doctoring, there are certainly things that can assist the team physician along the way. First, get to know the players before the game. The only way to do this is to spend time with the team. Understanding personalities in addition to having a comprehensive knowledge of each player's medical history, physical exam, and pertinent imaging findings will help make educated decisions in a timely manner when injuries happen. Second, be prepared. When an injury occurs, events move quickly, and time is of the essence. Practice makes perfect, and the entire team must be present during the practice. Having a rehearsed plan in place with the team allows for shared responsibility in ensuring effective, efficient care is provided for the injured athlete. Knowing where supplies are, who is assigned with certain tasks, and who will lead the encounter can be very helpful in decreasing valuable time lost. Third, be a team physician on the sideline, not a spectator. Team physicians must watch a game anticipating or look-

ing for possible injuries to occur. Often, this means continuing to watch the play after the whistle, taking a step back and more globally observing the game as opposed to watching the ball/puck, identifying environmental or sport-specific factors that may place players at risk and being mindful of those. Even in the exciting final moments of a close game, it is imperative that the team physician put emotions aside and perform the necessary tasks. Fourth, ask for help when needed. Given the heightened awareness of injury in sport, there are often multiple physicians or trainers at any particular event. While one person may be the head physician or trainer, each person has unique skills and experience that may benefit the injured athlete. Using combined knowledge and experience when appropriate can be beneficial. Likewise, each team member on the medical staff should feel comfortable in contributing to and addressing issues. It is everyone's job on the medical staff to play a role in the care of the injured athlete, and communication strengthens the ability to provide quality care. Lastly, do no harm. To be a team physician is to do the best you can for your patient in the time you have, with what you have.

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Chapter 2
Considerations for Spinal Cord Injury in the Athlete

Joseph E. Molenda, Brian T. David, and Richard G. Fessler

Introduction

In 1969, Frankel and colleagues first attempted to define spinal cord injuries [1]. In 1982, this was expanded by the American Spinal Injury Association (ASIA) with the addition of a 0–5 motor scale of 10 predefined motor groups, representing specific motor distributions. Today, the ASIA scale is the preferred method of choice utilized as a neurologic examination tool in the diagnosis of acute SCI [2] (Tables 2.1 and 2.2).

A catastrophic cervical spine injury occurs when there is a structural distortion of the cervical spinal column associated with actual or potential damage to the spinal cord [3]. In the cervical spine, sports-related injuries are grouped into three separate categories. This classification has the additional utility to aid decisions

Table 2.1 ASIA impairment scale

A – Complete	No sensory or motor function is preserved in the sacral segments S4–5
B – Sensory incomplete	Sensory but not motor function is preserved below the neurologic level and includes the sacral segments S4–5, with no motor function preserved more than three levels below the motor level on either side of the body
C – Motor incomplete	Motor function is preserved below the neurologic level, and more than half of key muscle functions below the neurologic level of injury have a muscle grade less than 3
D – Motor incomplete	Motor function is preserved below the neurologic level, and at least half of key muscle functions below that level have a strength grade greater or equal to 3
E – Normal	If sensation and motor testing are normal but the patient had deficits on prior evaluations

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Table 2.2 Key muscle group tested in ASIA evaluation

C5	Elbow flexors	L2	Hip flexors
C6	Wrist extensors	L3	Knee extensors
C7	Elbow extensors	L4	Ankle dorsiflexors
C8	Long finger flexor	L5	Great toe extension
T1	Small finger abduction	S1	Ankle plantar flexion

regarding the safe return to play for the athlete [4]. When a type 1 injury occurs, the athlete sustains a permanent SCI. A permanent SCI encompasses those with complete paralysis as well as incomplete SCI syndromes. In an athlete with normal radiographic studies, but deficits which completely resolve within minutes to hours, a type 2 injury is diagnosed. Finally, type 3 injuries include those with radiographic abnormalities without associated neurologic deficits.

Prehospital Immobilization and Transportation

It is critical that athletes with a SCI be assessed and managed in the immediate period of injury on the field as any standard trauma patient. This involves a systematic approach to rapidly assess the extent of injuries and begin life-preserving therapy established in the Advanced Trauma Life Support (ATLS) protocol, which emphasizes addressing airway, breathing, and circulation status. After initial stabilization by medical personal on the field, the athlete can be transported off the field while maintaining strict immobilization of the spine.

Neurologic deficits can develop after treatment has begun if proper immobilization is not utilized. In a 1983 publication, Podolsky and colleagues reported that up to 25% of spinal cord injuries had been caused by or worsened under medical care [5]. While this number may be an overestimate, it emphasizes the importance of safe transport and initial stabilization of the athlete with a possible SCI. On the field, the athlete is immobilized with a cervical collar and a spine backboard, and the head is secured. It is important to note that although a cervical collar can effectively stabilize most cervical injuries, with complete ligamentous disruption, the collar has minimal effect, emphasizing the importance of manual stabilization in these instances [6]. Patients with a SCI should be transferred immediately to a center that specializes in SCI, which has been linked to better neurologic outcomes, reduced length of stay, fewer complications, and reduced mortality [7, 8]. Upon arrival at the hospital, the helmet and shoulder pads should be removed, if they are still in place, before radiographic examination. Of note, logroll maneuvers should be avoided with employment of a lift-and-slide technique preferred, given that they create less motion of the injured segment [9]. After initial resuscitation and radiographic evaluation, decisions can be made regarding the management of the injury.

Unstable spine injuries should be initially reduced and temporarily stabilized with cervical traction (Gardner-Wells tongs or halo device). In cases where relevant,

early cervical traction for reduction of cervical fractures/dislocations is recommended to optimize alignment and minimize compression of the spinal cord [10, 11]. It is of critical importance to obtain a contrast-enhanced CT or MRI prior to reduction to ensure the absence of a herniated disc which can worsen a SCI upon attempted reduction in this setting.

Adjunct Treatment/Pathophysiology of Spinal Cord Injury

To understand the currently investigated adjunct treatment options, a basic understanding of the pathophysiology of SCI is essential. An acute SCI can be thought of as an initial traumatic primary injury with a secondary injury that follows as a result of the progressive cascade of events that results in tissue destruction and systemic autonomic consequences.

The primary injury results from a mechanical insult to the spinal cord most commonly a result of failure of the integrity of the spinal column, leading to compressive and often sustained forces on the spinal cord. The result is disruption of neuronal axons, blood vessels, and cell membranes [12, 13]. This triggers a cascade of processes that define the secondary injury phase.

During the secondary injury phase, necrosis results from mechanical disruption of cellular membranes, with simultaneous upregulation of cytokines and release of glutamate, which may reach excitotoxic levels [14]. Ongoing hemorrhage with increasing edema continues with ischemia resulting from local effects (i.e., thrombosis, vasospasm, microvascular disruption) as well as from systemic autonomic effects on the cardiovascular system caused by the SCI itself. The resultant hypoxia leads to impaired neuronal homeostasis and further cell death [14]. The cellular inflammatory response, driven predominantly by macrophages, is thought of as the primary mediator of the progressive secondary injury. Through regulation of perfusion pressure and the potential addition of a neuroprotective agent/strategy, the early stages of the secondary injury are thought to be critical areas where medical intervention can benefit the patient.

Depending on the level of injury, SCI can be complicated by respiratory and cardiovascular dysfunction. Innervation to the muscles of inspiration and expiration may be compromised leading to decreased forced vital capacity and peak expiratory flow rate [15, 16]. This can lead to insufficient oxygen delivery to the spinal cord, which can be further worsened by systemic hypotension resulting from traumatic disruption of the descending vasomotor pathways of the spine. These carry supraspinal innervation to the preganglionic sympathetic neurons in the intermediolateral cell column between T1 and L2. Hypotension results from decreased sympathetic supply to the peripheral vascular system, and bradycardia may occur due to unopposed parasympathetic supply to the heart through the intact vagal nerve [17]. Lehmann and colleagues found that patients with severe cervical SCI are more likely to have bradycardia, hypotension, and cardiac dysrhythmias than patients with mild cervical SCI or thoracolumbar injury [18]. In addition to the aforemen-

tioned mechanisms of impaired ventilation, any pulmonary injury itself may be present and leads to poor gas exchange and decreased lung compliance. Furthermore, painful chest wall injuries may decrease ventilation.

It is recommended that hypotension be corrected as soon as possible with a goal mean arterial blood pressure maintained between 85 and 90 mmHg for the first 7 days following an acute SCI [19]. If a pressor is needed, norepinephrine is favored with dobutamine as second line when increased cardiac output is desired. Phenylephrine should be avoided in patients with a SCI level above T6 due to its proclivity to trigger reflex bradycardia as it is purely a peripheral vasoconstrictor.

Corticosteroid Administration

The 2013 American Association of Neurological Surgeons and the Congress of Neurological Surgeons (AANS/CNS) Guidelines for the Management of Acute Cervical Spine and Spinal Cord Injury included the level 1 recommendation that the administration of methylprednisolone sodium succinate (MPSS) is not recommended [20]. MPSS has been the most extensively studied steroid in the medical management of acute SCI and is thought to work by its anti-inflammatory effects and halting peroxidation of neuronal membrane lipids [21–23]. The most frequently cited studies in the use of MPSS in acute SCI are the three National Acute Spinal Cord Injury Study (NASCIS) trials [24–26]. In all of the primary analyses, no significant difference was detected in motor, sensory, or functional recovery. However, post hoc analyses of NASCIS II data demonstrated that those receiving MPSS (30 mg/kg bolus at admission followed by 5.4 mg/kg/h for 23 h) within 8 h of injury improved significantly in both sensory and motor functions [26]. These differences remained significant 1 year post-injury. Additional post hoc analyses of NASCIS III data showed significantly greater motor recovery if a 48-h MPSS protocol (30 mg/kg bolus at admission followed by 5.4 mg/kg/h for 47 h) was used instead of a 24-h protocol, when treatment was started within 3–8 h [26]. The results from the third study also demonstrated no benefit to extending treatment past 24 h if MPSS was administered within the first 3 h after SCI. However, the 48-h MPSS protocol did show an increased incidence of severe pneumonia and severe sepsis ($p = 0.02$ and $p = 0.07$, respectively). High-dose MPSS has also been associated with increased prevalence of wound infections and death due to respiratory complications. Despite increased morbidity, there is no demonstration of increase in mortality with MPSS use [27].

Of note, there also appears to be a relationship between surgical timing and the safety of MPSS in acute SCI. In a multivariate analysis performed by Fehlings and colleagues, the primary data from STASCIS demonstrated that the 24-h MPSS protocol in combination with early surgery predicted significantly improved neurologic recovery at 6 months [28]. Particular consideration should also be given to the athlete with a cervical SCI. In this population, improvement in motor function is likely to have the greatest impact [29].

Hypothermia

The early induction of hypothermia has also been anecdotally reported to be beneficial in acute SCI. The mechanism for the neuroprotective benefit of systemic therapeutic hypothermia has yet to be elucidated [30]. It is hypothesized to result from reductions in cellular apoptosis [31], inflammation [32], glutamate excitotoxicity [33], edema, and other additional factors. In a phase I trial in patients with acute SCI, 14 patients were treated with 48 h of 33 °C intravascular hypothermia [34]. At the 1-year follow-up, 6/14 (42.9%) converted from complete SCI to incomplete. This is favorable considering the commonly reported value of 20% reported in the literature [35]. This underpowered study gathered enough data to garner support for further studies.

Medications

Vast research has been conducted on potential pharmacologic agents that aid in neuroprotection; unfortunately few therapeutic benefits have been realized from these studies. The three agents with the most current literature are GM-1 ganglioside, riluzole, and minocycline [36]. GM-1 ganglioside is an endogenous substance found in the mammal central nervous system and has shown to be anti-cytotoxic and anti-apoptotic. Preclinical animal trials demonstrated improvement in motor score at 3–5 days post-injury. Phase II trials in humans have shown to improve ASIA motor score at 1-year post-injury. However, phase III randomized control trial (RCT) showed no difference in motor scores at 52-week follow-up. Riluzole has been another highly investigated pharmacologic agent; it is a sodium channel blocker currently used in amyotrophic lateral sclerosis (ALS). Preclinical and phase I/II studies have shown improvement in ASIA motor score with its administration. It is currently undergoing a multicenter phase III RCT. Minocycline is a tetracycline antibiotic with anti-inflammatory properties. Preclinical and phase I studies have demonstrated improved motor scores, and phase II/III studies are currently underway. In addition to the above agents, newer neuroprotective and neuroregenerative therapies continue to be studied. As SCI research expands, providers will need to remain up to date with developing evidence-based standards.

Surgical Timing

Evidence exists that persistent compression of the spinal cord is a reversible form of secondary injury [37]. The Surgical Timing Acute Spinal Cord Injury Scale (STASCIS) was an international, multicenter prospective cohort study designed to determine whether early decompression (within 24 h) versus late (after 24 h) was

more beneficial after traumatic cervical SCI. An improvement of two or more grades of the ASIA Impairment Scale (AID) was seen in 19.8% of early surgery patients compared to 8.8% in the late surgery patients [28]. This consensus for early decompression has been demonstrated beneficial in the thoracolumbar region [38] and cauda equina syndrome [39]. From these studies it can be concluded that when feasible, early decompression is desirable.

Expert Opinion

- Acute SCI is an initial traumatic injury with a secondary injury due to a biochemical cascade. Since the initial injury has already occurred, most modalities of management focus on reducing the secondary injury cascade.
- Initial care should concentrate on removing mechanical compression of the cord and maintaining spinal cord perfusion.
- Mechanical compression on the cord can be removed via reduction techniques and/or surgery. There is evidence to support better outcomes with early surgical intervention.
- Avoidance of spinal cord hypoperfusion is of the utmost importance and should be emphasized as soon as the SCI is diagnosed. Current recommendations would suggest maintaining a mean arterial pressure (MAP) greater than 85–90 mmHg for 7 days post-injury.
- Although the NASCIS trials demonstrate complications with steroid administration, there is also evidence of neurologic improvement. Given that most athletic SCIs are likely to be isolated injuries, without the same comorbidities of the general trauma population, they may be a population ideally suited for high-dose steroid administration. Since the benefits outweigh the risks, we would recommend athletes with an isolated SCI receive IV steroids for acute SCI. This assumes administration of the steroids within 8 h of injury.
- Pharmacologic agents focused on neuroprotection and regeneration remain in their infancy. Further research is warranted before these promising modalities can be utilized in standard practice.

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Chapter 3
Rehabilitation of the Athlete's Spine

Robert Watkins IV and Michael Kordecki

Introduction

The goal of the elite athlete with a spine injury is a pain-free return to sport without decline in performance outcome. Evidence has shown that a structured and graduated rehabilitation protocol is crucial to return to play. Our experience in treating numerous professional athletes with spine injuries has allowed us to develop a specific and regimented rehabilitation protocol. The goal of this program is the same whether the patient is an elite athlete, recreational athlete, or injured worker: restore normal movement patterns and strength to the hips, legs, and spine, which will restore the highest level of function with the least amount of pain. Communication among a multispecialty team (physician, physical therapist, patient) during this process is paramount.

Evaluation

The first step to proper rehabilitation is an accurate diagnosis. Often spinal injuries are the result of poor movement patterns and muscle imbalance that has been present for years. The pathologic postures and movement left uncorrected will not only lead to more acute injuries but will also cause chronic pain and deterioration of the lumbar spine [1, 2].

As with any condition, history and comprehensive physical exam are the first steps in diagnosis. The history is used to help determine whether the problem is acute or chronic. If the patient complains of back pain that is focal to a specific

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area, a facet joint could be the problem. If a patient complains of pain that is more "belt-like," the problem could be more muscular in nature. If the patient complains of leg, buttock, or thigh pain that "shoots or radiates," the problem is more likely to involve the neurologic structures of the spine. If the patient wakes up in the morning feeling stiff and sore and the pain is alleviated with movement, the pain may be muscular in origin. Conversely, if a patient wakes up feeling better and experiences more pain as the day goes by, especially in the buttocks and legs, the pain is usually neurogenic in nature. For axial back pain, the disc is the most likely source of pain.

The physical examination is used to evaluate the patient's movement patterns. Basic biomechanics dictate that human beings are designed to ambulate and move primarily from the hips, knees, and ankles rather than the lumbar spine. The spine is meant for cushioning, shock absorption, and stability. In many cases, due to rapid growth in the younger population or lack of general exercise in the older patient, the hip flexors and hamstrings become tight. The gluteal muscles and abdominal muscles become weak, and the individual loses the ability to move normally. Once this happens, hip, knee, and ankle motion is limited, and the spine is forced into a resting position of hyperextension. As this occurs, hip motion is substituted by excessive motion at the lumbar spine which puts significant pressure on the facet joints and discs leading to facet and disc pathology.

A simple gait analysis will reveal typical patterns seen in patients with low back pathology. They often demonstrate loss of true heel strike due to a tight gastrosoleus complex. The legs are externally rotated due to tight hip flexors and rotators, with accompanying weakness in the gluteal muscles. The patients will pull themselves along using the hamstrings rather than pushing themselves forward by using the gluteal muscles and extending the hips. The pelvis is maintained in an anterior tilt due to lack of abdominal strength, tight hip flexors, and weak gluteal muscles. In turn the lumbar spine is in a position of extreme hyperextension locking the facet joints and overloading the posterior aspect of the disc.

Range of motion measurements are taken to determine the patient's basic flexibility. When assessing flexibility, care must be taken to maintain a neutral spine throughout the examination. A modified straight leg raise is used to test the hamstrings. Results greater than 15° short of neutral indicate a positive test. The quadriceps are compared from a supine position (heel to buttock) to a prone position. Any discrepancies between the two positions indicate tightness in the quadriceps. The patients' hip flexors should be assessed using the Thomas test. Motion less than 5° below neutral is indicative of a positive test. The hip rotators and piriformis are tested in supine with the hip flexed to 50° to determine their length. Normally the hip should rotate at least 30° and adduct 40° for a normal test. The Obers test is performed in the side-lying position to test the length of the IT band and tensor fasciae latae. Normally the hip should adduct to 45° with the knee straight without rotating the spine. The final step in the physical exam is the manual muscle test. Close attention should be paid to strength of the hip flexors, gluteal muscles, hamstrings, and abdominals. The gluteal muscles should be tested as both hip external rotators and hip extensors.

Cervical

Return to play after cervical spine injury depends upon the healing of anatomic structures, neurologic recovery, and rehabilitation. If the cervical injury does not require surgery, the rehabilitation program can be started after pain has become tolerable. The key is to avoid exercises that increase intra-discal pressure. The program begins with trunk stabilization and chest-out posture as these exercises reinforce ideal posture, thereby increasing intervertebral foraminal height, increasing the thoracic outlet, and decreasing the effective weight of the head.

If a cervical surgery has been performed, then rehabilitation can begin when the incision/soft tissues have healed, and pain is controlled. The focus is on building strength while maintaining good posture and alignment. In general, after a posterior cervical foraminotomy, the paraspinal muscles and facet capsule can be considered healed at 6 weeks. If a posterior discectomy has also been performed, soft-tissue healing takes approximately 8–12 weeks due to the annular defect. We allow 3–6 months for a fusion to heal. It is important to note that surgical "healing" does not equal return to play. The athlete does not become released until rehabilitation is completed.

The first goal of rehabilitation is to establish an ideal posture and to eliminate the rounded shoulder, forward head posture, and create a neutral cervical spine. This requires the lumbar spine and thoracic spine to be rehabilitated with the cervical spine. The regional alignment of the lumbar spine will carry over to the thoracic and cervical spine (lumbar hyperextension → thoracic kyphosis → cervical hyperlordosis). Excessive tightness across the chest with associated weak parascapular muscle can also primarily induce excessive thoracic kyphosis [3].

Neck rehabilitation starts with trunk stabilization exercises in the back program, such as dead bug, quadruped, and prone exercises (see next section). The goal is to build trunk strength to support the head in an ideal postural position. By starting in the lumbar spine and working up to the neck muscles, the anatomic structures of the healing cervical spine are less likely to be stressed.

Building postural muscle strength is balanced with stretching abnormally contracted musculature, typically the anterior chest wall. Exercises that stretch the chest can be done in the standing position. While the patient uses a doorway, the shoulders are held at 90° of abduction and 90° of external rotation. The patient then performs a pelvic tilt and while keeping the spine neutral slowly walks forward to stretch the chest and pectoralis minor. Specific strength exercises that target the rhomboids, middle trapezius, and latissimus are critical elements in restoring scapular position [4]. Proper scapular position, in turn, improves the inclination of the thoracic spine [3]. A neutral thoracic spine allows the cervical spine to realign itself with the help of isometric exercises. Rehabilitation at this stage focuses on proper performance of the strength exercises.

Patients who demonstrate scapular weakness often have a difficult time moving the scapula to the retracted position properly. Often a patient will create retraction in the scapula by extending the shoulder. Extending the shoulder causes the humerus

to push the scapula back toward the spine. If this occurs, the muscles of scapular retraction do not engage and therefore will remain weak. When done properly, the scapular moves first, followed by movement of the arm. The most basic scapular exercise is the "scapula set." The scapular set can be done in standing and then progressed to the side-lying position and finally prone. The patient is taught to properly retract the shoulder blades without moving the humerus in the glenoid. A more advanced exercise can be done while standing using exercise bands to provide resistance to the motion of scapular retraction and shoulder extension. If this exercise is done properly, the scapula will move first to the fully retracted position, and then the shoulder will move secondarily. Once proper scapular control is established, common scapular strengthening exercises can be used to strengthen the parascapular muscles [5]. When done correctly, these exercises will also help strengthen the extensor muscles of the cervical and thoracic spine. These exercises can first be undertaken in the prone position with a pillow under the thorax while the patient performs a pelvic tilt. Using this position keeps the cervical spine from being forced into hyperextension, while at the same time, allowing the scapula to move against gravity carrying the weight of the arm as resistance. These can then be progressed to the prone position over an exercise ball as long as the patient is pain-free and can perform the exercises correctly.

Lumbar

As mentioned previously, lumbar rehabilitation is the foundation for spinal injury rehabilitation. The rehabilitation program focuses on establishing proper functional movement and coordinated core strength. The first objective of lumbar spine rehabilitation is the establishment of a pain-free neutral position. Then balance and coordination are added into the program with endurance exercises. By building endurance strength centered on a neutral pain-free position, post-injury and post-surgical rehabilitation can begin relatively early because it avoids extreme and painful ranges of motion.

The protocol is initiated immediately once the injury or post-surgical pain is controlled. Every postoperative patient is encouraged to ambulate after surgery. The goal is to walk several times a day for a comfortable distance. Dedicated rehabilitation is initiated when soft tissues have adequately healed, symptoms have sufficiently stabilized, and stability of the anatomic structures is acceptable.

The stretching portion of the program can begin as soon as the surgical incisions are healed typically 14 days after surgery and almost immediately after any injury. The stretching exercises are all performed in a neutral spine position slowly and deliberately without pain, to protect the healing tissue. Each stretch is held for 10 full seconds and repeated seven to ten times twice per day. The stretching portion of the program will be maintained throughout the entire rehabilitation process and continue after discharge.

To properly stretch the muscles of calf, the stretch is performed in the standing position without shoes. Care is taken so the lumbar spine is not allowed to fall into

a position of hyperextension. The patient stands with their back against the wall and slowly bends at the knees and ankles while the heels stay firmly planted on the ground. This ensures the motion takes place at the ankle and not the arch of foot. The spine stays neutral throughout.

Stretching the hamstrings is done in supine with the leg raised off the ground and supported by a doorway. Performing the stretch in supine and keeping the contralateral leg flat on the ground will ensure the proper position of the pelvis in a neutral position. The leg being stretched is gently extended (by straightening the knee) until a slight tension is felt in the hamstring and the knee is in full extension. If the knee cannot extend fully or the contralateral leg comes off the ground, the patient is too close, and they must back away to proper position. Attempting to stretch the hamstrings in standing or long sitting will only cause excessive flexion in the lumbar spine, placing stress on the discs and spinal extensors. Stretching the quadriceps should initially be done in the prone position with a firm pillow under the pelvis to help maintain a neutral spine. The heel is gently pulled toward the buttock until a gentle stretch is felt in the front of the thigh.

The hip flexor stretch is performed in the high-kneeling position. The patient shifts their weight forward and tightens the buttock muscle of the leg being stretched. The shoulders and the pelvis move together as the stretch is performed, moving the hip toward extension. Care must be taken to maintain the lumbar spine in a neutral position throughout the exercise. Using the high-kneeling position and hyperextending the spine by throwing the shoulders back will only lock the facet joints and place unwanted stress on the healing tissues.

In testing of professional golfers, major league baseball players, and other athletes, it has been well demonstrated that it is the coordination of trunk muscles that produces maximum control of the spine [6]. Coordinated strength is more effective than uncoordinated strength. Each of the trunk muscles fires in an exact sequence to perform optimal movement. This coordinated strength protects the spine from injury and produces the desired athletic result.

Watkins' rehabilitation program is a five-level program that gradually increases in endurance, proprioception, and strength demands (Fig. 3.1). Level 1 starts in a neutral pain-free position with isometric exercises that train the core muscles to protect the spine and specific stretching exercises for the legs and hips (Fig. 3.2). In the acute post-injury and postoperative period, motion through the spine can cause mechanical trauma and exacerbate symptoms. Patients are taught to strictly maintain a neutral pain-free position while performing the basic stretching and core stabilization exercises [1]. The program accelerates to increasing intensity and compromised positions with balance and coordination exercises, if the patient can maintain a pain-free state (Fig. 3.2). If an exercise exacerbates symptoms, the exercise is modified, decreased, or discontinued. Patients may proceed to the next level more rapidly in some exercises rather than others. The average level (1–5) accomplished is used to determine core strength and ability to integrate other physical activities including cardiovascular exercise, sport-specific training, and activities of daily living.

By focusing on a neutral spine technique, the rehabilitation program can be started 2–4 weeks after a single-level lumbar laminotomy or discectomy, 4–6 weeks

Fig. 3.1 Watkins' lumbar rehabilitation program

after a multilevel laminotomy or laminectomy, 6–8 weeks after an artificial disc replacement, and 6–12 weeks after a fusion. Restoring normal mobility in the ankles, knees, and hips, while maintaining a neutral spine, will help reduce mechanical stress from the bony and muscular structures in the lumbar spine. This in turn will help reduce pain and improve function as quickly as possible [7].

Specifically, the program allows the athlete to progress through seven different exercises rated one through five in difficulty (Fig. 3.1). The entire program starts with finding a neutral pain-free position for the spine and strictly holding it in that

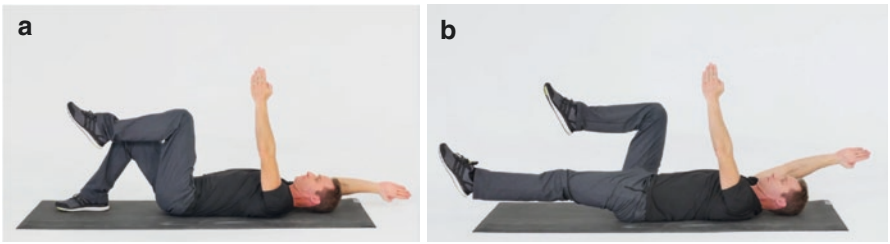

Fig. 3.2 Dead bug exercise. (**a**) Level 1 of the exercise. The spine is kept in a neutral tucked position, and feet are marched in place with one foot constantly on the floor. The goal is to perform this movement consistently without pain for 2 min. (**b**) Levels 2–5 of the exercise. Here the spine is kept in a neutral tucked position, and the opposite foot/arm is extended in an alternating pattern. The goal for level 2 completion is to perform the exercise for 3 min. Likewise, the goals for levels 3, 4, and 5 are to complete the exercise for 7, 10, and 15 min, respectively. Weights may be added to the hands to increase the difficulty. (Instructional photo by Watkins Spine Inc.)

position while performing the exercises. The entire program can be performed with relatively simple exercise equipment: exercise balls, hand weights, and pulleys.

The key to the rehabilitation program is to learn the proper technique. Proper technique is simply maintaining the neutral spine position. This is accomplished using the pelvic tilt maneuver. By properly tilting the pelvis using the abdominal and gluteal muscles, the patient places the lumbar spine in a neutral position. Once the patient learns the neutral position, they are taught to maintain this position while performing all levels of the rehabilitation program.

A proper pelvic tilt has three components. First and foremost, the abdominal muscles must fire correctly. Most patients will tend to fire the abdominals using the "draw-in" maneuver (DIM). It has been shown that the DIM is a very poor technique used to establish abdominal control. With the DIM, the patient is elevating the rib cage away from the pelvis and stretching the rectus abdominis rather than causing a contraction. The transverse abdominis and oblique muscles do very little. A correct technique to engage the entire abdominal muscle group is called the abdominal bracing technique (ABT). Using the ABT creates a pushing out maneuver that draws the pelvis up toward the rib cage using the rectus abdominis, the transverse abdominis, as well as the internal and external oblique groups [8]. The second component and the third component occur together. The gluteal muscles fire and pelvis tips in a posterior direction. By performing the pelvis tilt correctly, the lumbar spine will be held in a neutral position.

Once the patient demonstrates the ability to perform and hold a proper tilt, the exercises can begin. No movement of the spine is allowed. By doing this not only will one avoid exacerbations in the early post-injury and postoperative period but develop proper timing through proprioceptive feedback of the core muscles. The goals during both the stretching and strengthening phases are as follows: stability through the spine, slow and deliberate execution with all levels of exercise, and building of endurance. Feedback insuring no motion of the spine is critical. At first

feedback is tactile and eventually becomes internal as patient advances to higher levels. It is not a matter of brute strength; it is a matter of doing the technique properly.

The therapist's objective is to teach the patient how to do the exercises correctly. Regardless of how advanced the exercise, strict spine stability must be maintained. Often one category will advance faster than another category. Patients may be doing the level 3 in dead bug exercises, yet only level 2 in prone exercises. The therapist will advance the patient quicker in some exercises as long as they are able to perform the specific exercise correctly without pain. Key to the program is strictly maintaining the neutral position with all exercises. The exercises challenge the patient in different planes of motion: anterior and posterior sagittal plane, right and left frontal plane, and right and left transverse plane. If a patient has difficulty with a certain exercise category, determine what plane of motion is the suspect. This will help direct the stabilization progression. Core strengthening is neurological retraining as much as it is physiological strengthening [8, 9]. Precise application will enhance results.

Ball exercises provide a platform that requires a higher level of coordination and proprioceptive control to maintain strict stability [10]. Initially the therapist may need to provide tactile feedback to achieve this. The leg press begins with just a simple balancing exercise, rolling on the ball, and maintaining control of the ball throughout the motions (Fig. 3.3). Prone exercises of superman's, swimming, and shoulder abduction challenge abdominals and gluteals to prevent hyperextension. Prayer exercises and push-ups demand upper abdominal control to maintain stability. Always start slowly to insure a stable spine position.

The wall slide exercises can begin with a gentle flexion of the knees and with no real lower extremity or back strain (Fig. 3.4). This is an easy exercise, initially, that can be begun in the immediate post-injury and postoperative period. Quadriceps strength is directly proportional to the ability to work in a bent-forward position in a lifting job, and most importantly, the quadriceps exercises reflect the ability of a

Fig. 3.3 Ball/knee press exercise. (Instructional photo by Watkins Spine Inc.)

Fig. 3.4 Wall slide exercise. (Instructional photo by Watkins Spine Inc.)

patient to use their legs for bending and lifting, rather than their back. Patients with weak quads and tightness of the hamstrings and calf muscles lock their knees and bend at the waist, which is exactly the opposite of what we want for a back-pain patient. The wall slide progresses through a full 90°, with longer periods of holding. The addition of weights and extended arms increase the difficulty of the maneuver. The transition from the initial stage of identifying neutral position and maintaining that proceeds through a series of unsupported arm and leg motion exercises. Actively having the patient activate the abdominals and gluteals to maintain neutral spine position enhances quadriceps function and provides desired closed chain proprioceptive feedback.

Quadruped positions offer unique challenges in that there is less tactile feedback. The patient needs to develop better internal proprioceptive feedback mechanisms to strictly maintain the neutral spine position [10, 11]. The patient must learn to hold this position while progressing from more simple leg or arm lifts to alternate arm and leg lifts without and then with weights. Use of a stick lying across the pelvis will give feedback if lost position occurs on the frontal or transverse plane; however, feedback in the sagittal plane must be monitored internally by the patient or assisted by therapist hand feedback.

Aerobic exercise is important for general conditioning. Choosing the right type of exercise is important. An effective strategy is diversification. Those who rely only on running/jogging may be predisposed to strains and sprains. Pool walking is an excellent solution for many patients and can commence as soon as 3 weeks post-operatively. More complex and sophisticated types of aerobic conditioning must be

approached carefully. Nordic track and swimming for the untrained patient can result in an exacerbation of their condition. A diversified approach to aerobic conditioning will be less likely to produce overuse syndromes. Be aware of proper technique and make sure equipment is fitted appropriately. With the versaclimber and stairmasters, the key is to have the appropriate height step. We use the versaclimber with a very narrow step. The aerobic conditioning is there without getting the pelvic tilting that you get with too high of a step. The same is true with the exercycle. The seat should be low enough that the feet are not reaching down for the pedals, producing rocking of the pelvis on the seat. Running is a stiffening exercise, prone to development of contractures and weaknesses in isolated areas that are not used. If running technique is poor, the likelihood of compensatory dysfunction is high. Taking time to review running technique is worthwhile. Skipping rope is an excellent technique for trunk strength. The slight bent-forward flexion posture, locking the back in a neutral position, maintaining trunk control, while producing the aerobic exercise, can produce very tight trunk control while getting aerobic conditioning.

Once the athlete has established proper technique at level 1 of the program, they are advanced through the five increasingly difficult levels. The trunk stabilization program is categorized into levels, which helps the patient, therapist, trainer, doctor, etc. stay on the same page in regard to return to activity. Level 1 consists of establishing neutral pain-free position. Upon completing level 2, most patients can return to low-impact exercises such as bicycle, elliptical, and swimming. After level 3, most patients can begin incorporating sport-specific exercises including running, skating, throwing, shooting, and weight-lifting. Competitive athletes should complete level 4 before returning to sport-specific exercises that involve significant force and extremes of motion, including practicing with team. Professional athletes should maintain level 5 before and during return to play.

Return to play depends on:

1. Achieving the proper level of the stabilization program:
    - Level 3 for recreational
    - Level 4 for college
    - Level 5 for professional
2. Obtaining good aerobic conditioning
3. Performing the sport-specific exercises
4. Returning gradually to the sport (i.e., non-contact > contact).
5. Continuing the stabilization exercise once the athlete returns to sport

Resources

Our rehabilitation program is available for free on Apple and Android application stores under the name “Back Doctor.”

Expert Opinion

- It is critical that elite athletes complete a comprehensive rehabilitation program prior to return to play.
- The key to treating a spinal injury is an accurate diagnosis; all treatment should begin with a detailed history and physical exam.
- Both cervical and lumbar injuries should begin with stabilization of the neutral lumbar spine. Our lumbar program is our treatment of choice to accomplish this goal.
- Watkins' lumbar rehabilitation program is a system of exercises that produces functional movement patterns and coordinated core strength for the hips, abdominal, and low back muscles.
- After completing level 3 of our program, sport-specific exercises can be incorporated. Competitive athletes can return to sport upon completion of level 4; however, professional athletes should complete level 5 prior to return to play.
- After recovery from the injury, athletes should incorporate the program into their maintenance exercises.
- In addition to the lumbar rehabilitation program, cervical spine injuries require specific rehabilitation consisting of chest-out posture exercises that decrease the effective weight of the head, open the intervertebral foramen, and open the thoracic outlet.

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Chapter 4
Diagnosis and On-Field Management of Sports-Related Concussion

Lucas T. Buchler and Martin Boublik

Introduction

Sports-related concussions (SRCs) remain a challenging problem for the medical and public health communities as they are difficult to define, diagnose, treat, and prevent. They represent a significant injury burden on the athletic community with an estimated 1.6–4.0 million SRCs occurring annually in the USA [1]. Other studies estimate that between 1.0 and 1.8 million SRCs occur each year in the 0–18 years age group with roughly 400,000 SRCs in high school athletes alone [2]. These injuries account for approximately 75% of all traumatic brain injuries occurring in the USA and have been labeled a "silent epidemic" by The Centers for Disease Control and Prevention (CDC) [3]. Over the past several years, sports-related head trauma and traumatic brain injury (TBI) have garnered significant, and appropriate, public attention due to the concern for the short-term and long-term impacts of these injuries [3]. There has been an associated increase in the evaluation and treatment of SRC in emergency departments as well as the development of specific state laws governing the management of sports-related safety for school-age athletes in all 50 states [4–7].

An abundance of medical literature has been published in an effort to better understand SRC. A PubMed search for "concussion" in all the published literature over just the past 10 years returns over 6100 results, and the systematic reviews undertaken in preparation for the 2016 Berlin International Consensus Meeting on Concussion in Sport required the examination of nearly 60,000 articles [8]. The summary and interpretation of those reviews formed the Consensus Statement on Concussion in Sport [9] – a valuable resource for anyone responsible for the care of athletes. Additionally, nearly every representative or governing body of physicians, athletic trainers, and any other health professionals involved in the care of athletes has released some sort of position statement on the SRC in the past 10 years [6, 9–16].

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The CDC has recommended the adoption of the term *mild traumatic brain injury (mTBI)*, rather than *concussion* or *minor head injury*, as this description has become more popular over recent years [14]. In this chapter, however, we refer to this injury as sports-related concussion, or SRC, because this is the term mostly widely used in the available sports medicine literature. While a clear definition of concussion remains fairly elusive, one of the most comprehensive descriptions of SRC comes out of the Consensus Statement from the 2016 International Conference on Concussion in Sport [9]:

- Sports-related concussion is a traumatic brain injury induced by biomechanical forces. Several common features that may be [utilized] in clinically defining the nature of a concussive head injury include:
  - SRC may be caused either by a direct blow to the head, face, neck, or elsewhere on the body with an impulsive force transmitted to the head.
  - SRC typically results in the rapid onset of short-lived impairment of neurological function that resolves spontaneously. However, in some cases, signs and symptoms evolve over a number of minutes to hours.
  - SRC may result in neuropathological changes, but the acute clinical signs and symptoms largely reflect a functional disturbance rather than a structural injury, and, as such, no abnormality is seen on standard structural neuroimaging studies.
  - SRC results in a range of clinical signs and symptoms that may or may not involve loss of consciousness. Resolution of the clinical and cognitive features typically follows a sequential course. However, in some cases symptoms may be prolonged.
  - The clinical signs and symptoms cannot be explained by drug, alcohol, or medication use, other injuries (such as cervical injuries, peripheral vestibular dysfunction, etc.), or other comorbidities (e.g., psychological factors or coexisting medical conditions).

As implied by the definition above, it is important to remember that concussion is only one of a number of neurological injuries that may occur as a result of sport-related head and neck trauma. When evaluating athletes who have sustained head and/or neck trauma, it is important to first rule out catastrophic injuries, such as an intracranial hemorrhage and/or cervical spine injury. Additionally, the effect of sub-concussive head trauma – that is those head and/or brain impacts with forces that fall below the individual athlete's concussion threshold and do not result in the identifiable clinical signs/symptoms of concussion [17] – is largely unknown, but should not be ignored. Though the direct causative link has not been clearly identified, many authors suggest that sub-concussive impacts occur more frequently than concussive impacts and can also cause damage to the central nervous system that may contribute to long-term complications, such as chronic traumatic encephalopathy (CTE) [18–29].

The importance of recognizing sports-related head traumas cannot be overstated given the deleterious, and potentially fatal, impact of subsequent trauma, and second

impact syndrome [30–36]. Ultimately, the immediate recognition of a potentially injurious blow to the head, whether direct or indirect, can be lifesaving, and the athlete's safety must always be held paramount. Recent studies have begun to shed light on the potential long-term impacts of SRC, further underscoring the importance or diagnosing and treating these injuries appropriately [29, 37]. Additionally, recent literature has shown that immediate removal from activity may limit symptom severity and duration after SRC [38]. The difficulty remains in that no definitive, objective, diagnostic test has yet been developed for identifying SRCs, and, thus, clinical evaluation remains the gold standard [9]. Additionally, it is important to recognize and remember that the medical and scientific understanding of SRC is continually evolving and we are constantly learning more and more. In this chapter we describe a systematic approach to the clinical evaluation for, and on-field management of, sports-related concussion and other related head and neck traumas.

Preparation and Planning

Adequate preparation and planning are vital in the optimal management of SRC. It is important to note that this portion takes a significant amount of time and cooperative effort including everyone involved with the athletes from the medical staff to coaches to the athletes themselves. This is no small endeavor and should be started as early in the preseason as possible, or even immediately after the end of the previous season. We advocate for first, meeting as a medical staff – athletic training, physicians, emergency responders, etc. – and establishing a well-defined head trauma evaluation and management policy as a part of the team's emergency action plan. Additionally, an educational program for the athletes, coaches, management, parents, etc. should be laid out. Each of these items should be formally evaluated and updated on an at least annual basis. As mentioned above, due to the general lack of understanding in regard to concussion, recommendations and best practices change frequently, and it is important to remain up to date with the current literature.

Designing and Instituting a Head Trauma Evaluation and Management Policy

The management of head trauma and injuries should be included in the emergency action plan put into place by the sports medicine team. This plan should be discussed among the sports medicine staff and formalized prior to the start of any official team activities. It should include a plan for evaluating and managing a variety of potentially significant injuries – airway trauma, cardiac events, spinal cord injury, etc. – in addition to head trauma; however, we will focus on head injuries in

this chapter. While a concussion protocol outlining a standardized assessment for evaluation of athletes having sustained head trauma should be a part of this policy, the head trauma evaluation and management policy is more comprehensive and includes clear definitions of the role that each individual member of the sports medicine team will play.

Mostly, this policy should outline who will be responsible for what when responding to an athlete who has sustained any form of head trauma. Some important questions to consider, as highlighted by Broglio and Guskiewicz, are listed below [39]:

- Who will be responsible for the on-field response?
- Who will conduct the emergency assessment and handle communication if advanced help is needed?
- Who will observe the athlete on the sideline following injury?
- Who will make the diagnosis, especially in the absence of a physician?
- Who will communicate the diagnosis and prognosis with the parents, [family], and coaches?

While professional and high-level collegiate teams typically have clearly defined injury management policies and protocols, the more limited a team or athletic department's resources are, the more important this process becomes. The head trauma evaluation and management policy should be written such that each individual's role and each step of the evaluation and management process are clearly defined. This policy should be disseminated to any and all involved with the care of the athletes at hand and should be immediately available for reference at all times. This includes maintaining an up-to-date copy of the protocol in the training room, on the sidelines (especially at away games), at practices, etc. Additionally, the policy should clearly distinguish any changes to roles as it relates to home and away events. The formulation of a Pregame Checklist can also help to ensure that vital details are not overlooked – especially for away events. The Pregame Checklist should include an abbreviated – preferably one-page – version of the head trauma evaluation and management policy as well as establishing where vital resources such as a spine board, automated external defibrillator (AED), and emergency medical personnel are located. Reviewing this information with the sports medicine team prior to each event will help to ensure that pertinent information is communicated well in advance. In the event that an injury occurs requiring such resources, prior communication can help prevent any delay in appropriate management after an injury occurs.

Concussion-Specific Testing and Baseline Data Collection

A significant portion of forming a comprehensive head trauma evaluation and management policy, as it relates to managing concussion, is the determination of which, if any, concussion-specific evaluation tools will be used for assessment

after a suspected concussive injury has occurred. Several assessment tools have been developed over the years, but unfortunately, no definitive diagnostic test, tool, lab, or criteria exist for the diagnosis of SRC [9]. A variety of clinical tools are available to help aid in the evaluation for, and diagnosis of, SRC, and it is important to establish a consensus among the sports medicine team as to which tools or protocols will be used in the evaluation of an athlete with a suspected head trauma. As no perfect diagnostic criteria exist for SRC, a variety of tools may be considered. We will cover the details of several available tools later in this chapter, but the decision as to which tools a given team will employ must be made in the preseason. And, ideally, baseline testing data should be obtained for all athletes prior to practice or competition. While many concussion evaluation tools can be employed without comparative baseline data, the majority of these tools are best compared to a baseline.

Education and Relationship with the Athletes

As is the case with most of sports medicine, there is no replacement for established trust and a good relationship with the athletes under one's care. When it comes to SRC, the signs and symptoms of which are often subtle, knowing the injured athlete well can significantly improve the ability to perceive changes in personality and/or affect. Additionally, we know from the available literature that certain factors may impact an athlete's risk of concussion – history of previous concussions, learning disabilities, age, and sex to name a few [40–43]. This highlights the importance of establishing a relationship with the athletes under your care and having a detailed understanding of not only their concussion history but also their general medical history, personality, and affect.

Equally important, however, is educating others with regard to SRC and other sports-related head and neck trauma. Ideally, this takes place as a formal portion of preseason preparations with not only the medical staff but also the players, coaches, and management as well. It is important to educate any and all personnel who may be involved with the athletes regarding the importance of recognizing concussions and head trauma and also the potential serious long-term ramifications of ignoring these injuries. Additionally, some athletes may try to hide concussions and other head injuries in an effort to prevent removal from play [44, 45]. It is our obligation as the medical staff for these athletes to place an emphasis on education and to engage the team, as well as each individual athlete, in the management of SRCs. Some authors advocate the development of a Concussion Statement, signed by each athlete, confirming that he or she understands the signs and symptoms of concussion and his or her individual responsibility to report suspected concussions [39]. This could certainly be a valuable component of concussion education, but it is important to remember that a signed statement of understanding does not obviate the responsibility of the sports medicine team to ensure adequate education and understanding. The National Athletic Trainers' Association Position Statement: Management

of Sport Concussion, 2014 Update also includes excellent educational information and handouts for athletes/parents in its appendices [11].

The steps noted above should be undertaken for all athletes regardless of the likelihood of sustaining head trauma; however, we know from epidemiologic studies that certain sports place athletes at a greater risk. While football garners the most societal attention, likely owing to the popularity of the National Football League (NFL), it is important to remember that it is not the only sport in which athletes are at risk for sustaining SRCs. Epidemiologic data from the National Athletic Treatment, Injury and Outcomes Network (NATION) demonstrated that, in high school student-athletes, football had the highest rate of SRC followed by boys' lacrosse, girls' soccer, boys' wrestling, and girls' lacrosse [46]. Though SRC occurred most commonly in these sports, it is important to note that SRC was reported in nearly every high school sport, including many traditionally considered noncontact, such as cross-country and crew [46]. Additionally, the overall rate of concussion was higher in female high school athletes than male high school athletes – 2.64 vs. 1.69 per 10,000 athlete-exposures, respectively [46].

Evaluation and Diagnosis

The evaluation and management of traumatic head injuries sustained during practice and/or competition should simply be an exercise of executing a carefully thought out plan that has been reviewed and rehearsed in its entirety. The availability of a detailed manual can also be helpful, especially in the setting of a less organized medical staff as is often the case in youth or high school athletics. Many times, the on-field management will occur under the guidance of the team's athletic trainer, but in other settings, a team physician may be responsible for the evaluation. When a head trauma is recognized – whether by the medical staff, an official, a coach, a player, a parent, etc. – it is imperative that the athlete be immediately removed from competition. If the initial evaluation is to take place on the field, play must be stopped, and the safety of the athlete held paramount.

A variety of technologies have been employed in an effort to more systematically recognize concussive impacts in high-risk sports. Significant research efforts have been undertaken in an attempt to identify an objective characterization of a concussive impact using helmet-based accelerometers to measure a variety of biomechanical variables and communicate impact data in real time. Though many studies have been published based upon this approach, no study has been able to successfully identify a set of criteria or combination of variables to objectively define a concussive impact [27, 47–60]. Further, no definitive biomechanical technology, biomarker, or neuroimaging study has been developed with the ability to reliably diagnose SRC in real time. As such, we must rely on the athletes and any other observers to report potentially injurious impacts. Once such an impact has been recognized, we use a systematic, multimodal approach to clinical exam and evaluation in an effort to identify SRCs.

Sports-related concussion is largely a diagnosis of exclusion. That is, one may settle on the diagnosis of SRC if some form of concerning subjective (symptom) or objective (sign) neurologic disturbance is noted that cannot be attributed to some other neurologic injury or pathology. Broadly, we divide the head/neck trauma evaluation into the primary survey (on-field evaluation), secondary survey (sideline evaluation), and tertiary survey (locker room/training room evaluation). Each step of the evaluation must be completed carefully and purposefully to ensure that an SRC, or more ominous injury, is not missed. And, though it should go without saying, if any concern for a concussion or other significant head or neck injury arises at any point, the athlete should be withheld from returning to play.

Primary Survey (On-Field Evaluation)

It is important to remember the basics first when initially evaluating an injured athlete: airway, breathing, and circulation. An isolated concussion, in and of itself, is not an immediately life-threatening injury; however, one must not assume any sort of diagnosis after a head impact or trauma. Though catastrophic head and neck injuries are not common in sports-related injuries, they do occur, and it is important to first rule out immediate life- or limb-threatening injuries. The primary survey will often take place on the field of play, and the main function of this evaluation is not to establish a definitive diagnosis, but rather to rule out any injuries that may prevent the athlete from safely being removed from the field of play. This aim should be kept in mind, and the goal should be to move the athlete to a protected environment as soon as safely possible. This evaluation should be considered a triage evaluation rather than a diagnostic evaluation. It is not the time for extensive testing, but rather for a focused and succinct evaluation to ensure that the athlete is not in any immediate danger. The bulk of the true diagnostic evaluation(s) will take place during the secondary and tertiary surveys as detailed below.

Some injuries to consider during this evaluation are airway emergencies, cardiac events/injuries, spine injuries, intracranial hemorrhage, skull fractures, and severe neurovascular injury. And, though significant alterations in consciousness are not all that common, the Glasgow Coma Scale (Table 4.1) can be a useful tool for quickly assessing for signs of traumatic brain injury on the field [61]. Additionally, the following are generally considered "red flags" and may indicate a significant and/or evolving neurologic injury that requires immediate attention: loss of or fluctuating level of consciousness, decreasing level of consciousness, increasing confusion, increasingly irritable/restless/combative, neck pain or tenderness, upper or lower extremity numbness or paresthesia, unequal pupil size, double vision, vomiting, seizure or convulsion, slurred or altered speech, worsening headache, and inability to recognize people or places (Table 4.2) [11, 62]. The NFL, for example, identifies loss of consciousness (including impact seizure and/or "fencing posture"), confusion, amnesia, and gross motor instability (determined to be neurologically caused) as "no-go" events, and if any of these are observed at any point, the athlete is deemed

Table 4.1 Glasgow Coma Scale

Test	Response	Score
Eye opening	Spontaneous	4
	To speech	3
	To pain	2
	None	1
Verbal response	Oriented	5
	Confused	4
	Inappropriate	3
	Incomprehensible	2
	None	1
Motor response	Obeying	6
	Localizing	5
	Withdrawal	4
	Flexing	3
	Extending	2
	None	1

Table 4.2 Red flags during the on-field survey[a]

Fluctuating level of consciousness	Inability to recognize people or places
Increasing confusion	Increasingly irritable/restless/combative
Neck pain or tenderness	Persistent/worsening extremity numbness or paresthesia
Unequal pupil size	Double vision
Vomiting	Seizure or convulsion
Slurred or altered speech	Worsening headache

[a]Warrants transfer to emergency center immediately

ineligible for return to play [6, 63]. Finally, assessment of vital signs, including pulse and blood pressure, should be considered. Persistent widened pulse pressure associated with relative bradycardia, for example, may indicate elevated intracranial pressure and may represent an early sign of significant intracranial injury [39]. The presence of one or more of these at any time point during the evaluation is highly concerning and warrants immediate attention and, when necessary, resuscitation, proper immobilization, and ultimately transportation to an emergency department for formal workup and evaluation.

Secondary Survey (Sideline Evaluation)

If an athlete requires an on-field assessment for any reason, after sustaining an impact or injury to the head and/or neck area, a thorough secondary survey is warranted once the athlete reaches the sideline. Once a clinical evaluation for cervical spine trauma and other life-threatening injuries has been completed, the athlete

should be moved to a safe area for a complete sideline evaluation. At this point, the evaluating clinician should repeat the primary survey and complete a more comprehensive evaluation. Again, the emphasis must be on identifying any evolving cervical spine or intracranial pathology first and diagnosing concussion, or other injury, second. Each individual practitioner will develop his or her own system for evaluation, but we recommend the inclusion of at least the following components: observation and history, focused neurological exam including a cervical spine evaluation, and functional testing.

Observation and History

The first step in evaluating an athlete who has sustained trauma to the head and neck region is establishing the mechanism of injury and the sequence of events leading up to and immediately following the injury. Often the evaluating clinician will have witnessed the injury and may have an established mechanism of injury in mind based on his or her observation of the incident. Nonetheless, it is worthwhile obtaining a history from the athlete directly as this may provide added insight in regard to mechanism and offer an opportunity to assess any evidence of change in affect, aphasia, or amnesia – anterograde or retrograde [64]. Additionally, collateral information from other athletes, coaches, medical staff, or other observers can be very helpful. At higher levels of competition, such as collegiate or professional sports, video may be available for review as well. As is the case with most of medicine, any additional information can be helpful.

Standard orientation questions – person, place, and time – have been found to be unreliable in the setting of sport and the injured athlete. Rather, the recommended assessment of orientation uses some variation of the following five questions (Maddocks Questions) [65, 66]: What venue are we at today? Which period (quarter, half, etc.) is it now? Who scored last in this match? What team did you play last week/game? Did your team win the last game? These questions can, and should, be modified as necessary to more appropriately reflect the sport and/or competition at hand.

According to the Consensus Statement from the 2016 International Conference on Concussion in Sport [9], the diagnosis of SRC may include disturbances in the following clinical domains:

(a) Symptoms: somatic (e.g., headache), cognitive (e.g., feeling like in a fog), and/or emotional symptoms (e.g., lability)
(b) Physical signs (e.g., loss of consciousness, amnesia, neurological deficit)
(c) Balance impairment (e.g., gait unsteadiness)
(d) Behavioral changes (e.g., irritability)
(e) Cognitive impairment (e.g., slowed reaction times)
(f) Sleep/wake disturbance (e.g., somnolence, drowsiness)

Thus, one should specifically look for and/or ask about these signs and symptoms. Several resources for evaluating for such signs and symptoms are avail-

able, but the following are two commonly utilized tools: Concussion Recognition Tool 5 from the Concussion in Sport Group [67] and The Graded Symptom Checklist (GSC) [11, 68]. Additional symptom scales include the Acute Concussion Evaluation (ACE), Concussion Symptom Inventory (CSI), Health and Behavior Inventory (HBI) (primarily for pediatric patients), Post-Concussion Symptom Inventory (PCSI) (primarily for pediatric patients), Post-Concussion Symptom Scale (PCSS) (primarily studied in adolescent athletes), and Rivermead Post-Concussion Symptoms Questionnaire (RPCSQ) among several others [69, 70].

The Graded Symptom Checklist (GSC) is the mostly widely discussed symptom scale, at least in the sports medicine literature, and its use is recommended by the National Athletic Trainers Association [11, 68]. Additionally, one may choose to ask the athlete being evaluated to grade his or her symptoms on a scale of 0–6 (0, not present; 1, mild; 3, moderate; 6, most severe) and then calculate a total symptom score. When used in this manner, the evaluation is referred to as the Graded Symptom Scale (GSS).

Most of disturbances noted as a result of concussion are symptomatic in nature, and one must rely on subjective assessment and reporting to detect these changes. This underscores the importance of the observation and history portion of the evaluation, and thus emphasis should be placed on this assessment.

Focused Neurological Examination Including Cervical Spine Evaluation

The focused neurological examination should start with the bony structures of the face and cranium to assess for any obvious evidence of craniofacial fracture and, next, palpation of the cervical spine for any sign of midline tenderness or pain with range of motion of the cervical spine. If these are normal, provocative exam maneuvers, such as Spurling's maneuver, may be considered. If the athlete demonstrates midline tenderness to palpation and/or pain with range of motion, he or she should be placed into a cervical collar as these findings may represent findings of an unstable cervical spine injury and further evaluation with advanced imaging (typically a CT scan) may be warranted [71]. Additionally, an assessment of sensory and motor function in at least the major upper (C5-T1) and lower extremity (L2-S1) dermatomes and myotomes should be included. Finally, a brief cranial nerve examination, including an assessment of eye movement/extraocular muscles as well as pupillary function, should be completed.

Functional Testing

Presuming the athlete demonstrates no positive signs/symptoms of head injury or concerning physical exam findings, assessment of functional activities should be the final step in the sideline evaluation. This includes evaluation of speech pattern and gait. If walking gait is found to be normal, the evaluation should progress to

running and then athletics movements such as pivoting or cutting. It is important to evaluate for not only signs of ataxic movement or difficulties with balance but also to question the athlete about the development of any symptoms with exertion.

If the secondary survey, or sideline evaluation, elicits any positive findings concerning for SRC or other neurological injury, the athlete should be escorted to the locker room or training room for a complete tertiary survey and withheld from returning to play until the full evaluation is completed [72]. If the full secondary survey is found to be negative, the medical staff may should then make a clinical decision as to if the athlete may safely and appropriately return to play. It is essential for all involved parties, especially athletes and the coaching staff, to recognize that a sideline evaluation does not mean that a player is to be automatically removed from competition. The hope in such a design, in combination with extensive preseason and continuing athlete education, is to minimize the desire for athletes to hide head injuries and/or avoid formal evaluation.

Tertiary Survey (Locker Room/Training Room Evaluation)

If the secondary survey (sideline evaluation) is deemed to reveal any positive findings or found to be indeterminate for SRC or other head/neck injury, the athlete should be taken to the locker room, training room, or other quiet, controlled environment for a comprehensive examination. This examination should include, at minimum, a complete neurologic examination and some form of a multimodal concussion-specific assessment.

Complete Neurological Examination

Though components of a comprehensive neurological examination have been performed in the primary and secondary surveys, those portions should be repeated in evaluating the athlete in the training room or locker room. The signs and symptoms of SRC are often subtle, and this level of redundancy helps to ensure that mild disturbances are recognized. Though a variety of physical exam maneuvers may be incorporated into a complete neurological examination, the details of these are beyond the scope of this chapter. Briefly, at least the following components should be included: spine evaluation (palpation, range of motion, upper and lower extremity motor function, full sensory examination, reflex assessment); complete cranial nerve evaluation (including assessment of eye motion and extraocular muscles, pupillary reactivity to light, and pupillary accommodation); and cerebellar function testing including assessment of balance and coordination. The goal of this evaluation is to rule out some other form of neurologic injury – such as spinal cord injury, brainstem injury, intracranial hemorrhage, etc. – and to assess for subtle neurologic disturbance that may be attributed to SRC.

Application of Concussion-Specific Tests and Tools

A number of concussion-specific assessment tools have been developed and are available for use, but it is important to recognize that none of these tools have demonstrated adequate sensitivity and specificity to be considered an optimal diagnostic tool. Additionally, most concussion assessment tools are susceptible to influence of underlying mental health conditions, such as depression or anxiety [73]. Thus, most authors recommend a multimodal approach toward diagnostic evaluation using an assessment that evaluates for some combination of the following: symptoms of concussion, orientation, short-term/immediate recall, concentration, balance, and delayed recall.

The Sports Concussion Assessment Tool, 5th Edition (SCAT5©), was developed by the Concussion in Sport Group and represents a systematic, multimodal assessment. The SCAT5© is a resource that is readily available to all sports medicine practitioners and includes an approach that is based on the best available evidence. It incorporates Maddocks Questions, the Glasgow Coma Scale (GCS), a cervical spine assessment, a symptom evaluation tool, the Standardized Assessment of Concussion (SAC), a neurological screen, and the Modified Balance Error Scoring System (mBESS) [9, 61, 62, 65, 74, 75]. The Standardized Assessment of Concussion (SAC) was developed as a quick, reliable assessment tool for screening for signs and symptoms of concussion and has been validated with reasonable sensitivity and specificity when administered immediately post-injury [66, 74, 76, 77].

Balance and postural stability deficits following concussion have been well-documented; however, early research in this domain focused on objective, quantifiable assessments using sophisticated force-plate systems for evaluation. The Balance Error Scoring System (BESS) was developed in an effort to create a practical and objective sideline assessment of balance and postural stability [75]. To perform the tests, athletes are asked to maintain three positions (double-leg stance, single-leg stance, tandem stance) with their hands on their iliac crests and their eyes closed for a period of 20 s each while an evaluator counts errors (hands lifted off iliac crests, opening eyes, step/stumble/fall, move hip into more than 30° of flexion or abduction, lift forefoot/heel, remaining out of testing position for more than 5 s) and scores the exam [75]. In its initial design, the series of three tests was completed once on a firm surface and then repeated on a piece medium-density foam, but it has since been modified (mBESS) to be performed as a single series on one surface – preferably the same surface upon which baseline testing was completed. For sports using special footwear, such as cleats, this testing should be completed in competition footwear.

Neuropsychological Testing

Neuropsychological testing, such as the Immediate Post-Concussion and Cognitive Testing (ImPACT), represents another tool that can be useful in the evaluation of SRC. ImPACT is a computerized injury assessment tool and is most commonly

applied using a desktop version. The difficulty with this approach, however, is that the testing can be fairly cumbersome and time-consuming, and the assessments must be applied and interpreted by trained neuropsychologists and/or technicians [78]. This limits the usefulness of such testing in the on-field evaluation and management of SRC; however, the information garnered from neuropsychological testing may be useful in assessing recovery progression when compared to baseline. As is the case with most diagnostic tools for SRC, the psychometric properties of ImPACT are insufficient for such an assessment to be considered diagnostic on its own [79, 80].

The King Devick (K-D) test is another neurocognitive test that incorporates visual pathways into the assessment. This test entails a 2-min rapid number naming exercise in which the athlete quickly reads numbers from a screen or card. This activity involves eye movements (saccades, convergence, accommodation), attention, and language function – all of which may be affected by SRC [81]. As is the case with ImPACT testing, results of K-D testing can be susceptible to variation due to underlying conditions, such as learning disability and/or attention disorders, such as attention deficit hyperactivity disorder [41, 82, 83]. This underscores the role of baseline testing and also the importance of considering the results of such testing on an individual basis. While further research is necessary, there is some hope that a rapid neurocognitive assessment, such as the K-D test, may eventually serve as a primary sideline diagnostic tool.

Other Emerging Diagnostic Technologies

The lack of a definitive diagnostic test for SRC has been the focus of much research and innovation. Significant efforts have been placed toward evaluating serum biomarkers indicative of intracranial trauma and/or hemorrhage. While these studies show some promise in aiding the diagnosis of concussion in the hospital setting, they offer little value to the sideline clinician in their current states. If one of these lab tests reaches clinically significant sensitivity/specificity for evaluation, there is some hope that a finger stick, point-of-care test could later be developed [84, 85]. Other studies suggest that alterations in cerebral blood flow may be responsible for some of the neurologic disturbances associated with concussion, and thus, multiple brain MRI protocols have been developed to assess cerebrovascular reactivity after concussion [86, 87]. Assessment of ocular movements and pupillary reflex is a common part of several recommended examination protocols for evaluation for SRC. Some researchers have found that the incorporation of automated binocular pupillometry may help to more objectively measure pupillary function and may be useful diagnostically [88]. Brain Network Activation assesses changes in brain activity and functional connectivity after concussion using high-density, multichannel electroencephalogram mapping of event-related potentials (EEG-ERP) [89]. While all of these technologies are exciting and show promise in terms of potential future applications, none demonstrates the appropriate sensitivity and specificity to serve as a tool for on-field management and diagnosis of SRC at this time.

Removal from Competition and Next Steps

After completion of the full evaluation, including the primary/on-field, secondary/sideline, and tertiary/locker room portions when necessary, one must make the critical decision as to if the athlete may or may not return to play. The goal of the systematic and comprehensive assessment is to detect any signs of injury and/or subtle neurologic disturbance. At this point, the clinician is in charge of decision-making and must determine if the athlete may return to play. With that being said, a properly designed head trauma evaluation and management policy will result in a significant number of athletes being identified as having suffered an at-risk impact and indicated for a formal assessment, but found to have a completely normal evaluation. It should go without saying that the most prudent advice when making this decision is to error on the side of caution and protecting the athlete. The consequences of second impact syndrome are significant, and, if there is any question, the athlete should be held out from play [30–36]. Finally, it is important to recognize that a concussion is an evolving pathophysiologic process and continued evaluation and monitoring are necessary [9]. Thus, any athlete who is indicated for a formal assessment, even if he or she has a completely normal evaluation, should receive follow-up evaluation after the event. The athlete should also be re-evaluated at some scheduled interval within the next several days to ensure that an evolving SRC has not been missed.

Return to Sport and the Management of Post-Concussive Syndrome

Return to sport after concussion and the management of post-concussive syndrome are beyond the scope of this chapter. The consensus statements from several national and international organizations are, however, in agreement with some variation of the following stepwise approach: (1) medical assessment, (2) rest, (3) symptom-limited activity, (4) light exercise, (5) sports-specific exercise, (6) noncontact training, (7) medical clearance, (8) full-contact practice, and (9) return to sport [9, 90]. An SRC-specific return to sport protocol should be designed and implemented as a portion of the head trauma evaluation and management policy for each specific team, sport, competition, etc. While protocols vary, most are centered around a principle of gradually returning athletes to exertional exercise and eventually competition in a stepwise and controlled manner. It is important to note that each individual athlete will progress through a stepwise return to sport protocol at a different rate and the athlete should not be permitted to progress to the next step in the protocol until able to complete the previous step entirely symptom-free.

Post-concussive syndrome can vary widely from athlete to athlete, and it is important to ensure that recovery and treatment are individualized to fit each player and each injury. Additionally, it is helpful if the same person, whether it be a physician, athletic trainer, or otherwise, evaluates the athlete on a regular basis as he or

she recovers from an SRC. This allows for consistent evaluation of progress and helps to ensure that the athlete is not inappropriately allowed to move to the next phase of the protocol without successfully completely the previous step. It is also important to recognize that the rate of recovery can vary drastically from athlete to athlete. In the case of children and adolescents, recovery may take up to 4 weeks, compared to the typical 1–2 weeks in adults [91].

Expert Opinion

Despite the significant effort and resources directed toward the study of SRC over the past 50 years, we are, unfortunately, still left with more questions than answers with regard to how this condition is best managed. This renders SRC an incredibly difficult entity to understand, recognize, and treat. For all the valuable information included in the Consensus Statement from the 2016 International Conference on Concussion in Sport, the most telling portion of the document may be its final line: "…the science of concussion is incomplete and therefore management and return-to-play decisions lie largely in the realm of clinical judgement on an [individualized] basis" [9]. This speaks to the importance of continued learning and maintaining an up-to-date understanding on the current literature regarding SRC.

Our experience in the diagnosis and management of SRC, at least over the past several years, has been primarily with professional football players. The National Football League Head, Neck and Spine Committee's Concussion Diagnosis and Management Protocol is reviewed and updated prior to each season and the 2017–2018 season version is published and available for reference as an example of a comprehensive concussion program in a high-level collision sport [6]. This protocol is one example of a succinct, summary document that can be provided to all involved personnel to guide the management of head and neck injuries, including SRC. Additionally, the National Football League's "Concussion Game Day Checklist" is included in this protocol [63, 92]. We recommend that each and every sports medicine team create a similar written policy for the management of head and neck injuries as well as a well-defined game day evaluation checklist. This will make practice and game day roles clear and the assessment of these injuries systematic. Taking such an approach will help to ensure that head and neck injuries are appropriately evaluated and properly managed.

Additionally, we recommend an all hands – or, more appropriately, all eyes – on deck approach. Any trainer, physician, coach, player, official, parent, etc. who observes a concerning impact should not only be allowed, but encouraged, to speak up and ensure that the player receives an appropriate evaluation. Developing this culture takes a significant effort in terms of education and communication, but, ultimately, helps ensure that each and every athlete is properly cared for. Also, comprehensive educational efforts can help encourage athletes to advocate for themselves. We know from the literature that athletes hiding head injuries and SRCs is not uncommon [44]. In our experience this has become less of an issue with our

professional athletes as the potential long-term effects of these injuries have come to light and we have improved our efforts with regard to education.

Until we as a medical community are able to develop and scientifically validate a reliable sideline diagnostic tool, SRC will remain a clinical diagnosis. In that same vein, any and all available resources should be utilized in gathering information to inform clinical decision-making. At the professional level, video review of the injury or impact at hand can be incredibly helpful in understanding the mechanism and/or severity of the impact sustained [93].

Finally, we encourage anyone evaluating and managing these injuries to take the conservative approach. If there is any doubt whatsoever, hold the athlete out for further evaluation. The safety and long-term health of the athlete must always be held paramount.

Recommended Reading

This chapter serves as a brief overview of the available literature on SRC and our approach to the on-field evaluation and management of this injury. For those interested in a deeper dive into the literature, we recommend reviewing the June 2017 issue (volume 51, issue 11) of the British Journal of Sports Medicine. This issue contains the Consensus Statement on Concussion in Sport, the 5th International Conference on Concussion in Sport held in Berlin, October 2016, as well as the systematic reviews on varying SRC-related topics undertaken in preparation for this meeting [9].

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Chapter 5
Return to Play After Sports Concussion

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Sports-related concussion (SRC) is defined as a traumatic brain injury induced by biomechanical forces [1]. Due to the complex nature of the injury, multiple common features may help with clinically defining SRC. Critically, concussions result from a direct blow to the head, neck, or elsewhere in the body resulting in an impulsive force transmitted to the head [1–4]. Concussions often result in rapid onset of short-term impairment of neurological function, though symptoms may evolve over minutes to hours [1]. Symptoms of SRC result from functional disturbances in the brain rather than structural damage; therefore traditional neuroimaging studies are often inconclusive [1, 4, 5]. Importantly, concussion may take place with or without loss of consciousness, and the majority of athletes remain conscious through the inciting event [1]. Symptoms typically resolve following a sequential course but may be prolonged in certain circumstances [1, 4, 6]. Additionally, signs of concussion cannot result from alcohol, drug use, or comorbidities that may explain symptoms [1].

Sport-related concussion affects between 1.6 and 3.8 million Americans annually with the majority being youth or adolescent athletes [5, 7]. Expectedly, there is greater risk of concussion in contact sports such as football, ice hockey, and lacrosse

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when compared to noncontact sports such as basketball, baseball, and tennis [5, 7–9]. Generally, there is between a 10% and 100% increase in concussion rate in female athletes compared to their male counterparts in the same sport [5, 7–10]. According to a national study on collegiate athletes, football has the highest number of reported concussions with 4404 reported between 1988 and 2004 according to the NCAA Injury Surveillance System, but the highest rate occurs in women's ice hockey with 0.91 concussions per 1000 athletic exposures [9]. The rate of SRC across all collegiate sports rose by 7% per year from 1988 to 2004, and data suggests similar trends in the NFL. This trend likely represents an increased awareness and recognition of concussion rather than a true increase in incidence of the injury [8, 9, 11].

Biomechanics and Pathophysiology

SRC is a traumatic brain injury resulting from linear and rotational acceleration to the brain [1, 4]. It is often caused by a direct blow to the head, neck, or face. There is currently no known threshold for a concussive injury [4]. Herring et al. note that modifying factors (e.g., concussion history, neck strength, anticipatory reaction time, frequency of head trauma, and locations of impact) contribute to a minimum threshold for SRC, but there is still no consensus throughout the sports community [4].

It is also contested whether SRC should be considered in the TBI spectrum or better classified as a reversible physiologic change [1, 4]. Animal models have shown acute metabolic changes following concussive injuries that are thought to occur in humans [4]; these metabolic changes include alterations in the concentration of intracellular/extracellular potassium, calcium, and glutamate as well as a relative mismatch between cerebral blood flow (decreased) and an increased cerebral glucose requirement [4]. This mismatch between glucose requirement and cerebral blood flow observed in the acute post-concussion setting has been theorized to contribute to injured cells' increased vulnerability to a second insult and the development of second impact syndrome [4].

Numerous studies have characterized head impact-exposure patterns in collision athletes. These studies use instrumented helmets to report collision characteristics including impact location, frequency, and injury outcomes [4]. The results of these studies vary considerably, with significant differences in the reported mean peak linear and rotational acceleration in concussed athletes [4]. Furthermore, the accelerations detected by the helmet-based sensors do not necessarily reflect the impact imparted on the brain itself; as previously mentioned there are a number of modifying factors that affect the minimal threshold for SRC [4]. Thus, expert consensus does not support the use of helmet-based sensors in the clinical diagnoses of concussion.

Prevention Strategies

Prevention of SRC is challenging due to the unpredictable nature of sports, but preseason strategies exist which can help reduce the number of SRCs. It is recommended that a detailed concussion history is taken from each athlete before the

season begins [12–15]. Critically, this history should include more than number of concussions but also symptoms and recovery time from prior SRCs [1, 3, 15]. There is value to recording other head, face, and cervical injuries which may have masked the diagnosis of concussion [1]. Additional family history of mood and migraine disorders, attention-deficit hyperactivity disorder, dementia, and long-term complications of concussions should be taken [16].

Preseason education targeting athletes, families, coaches, officials, and teachers is a leading strategy in the prevention of SRC [1, 3]. Ideally, the educational program should be carried out by the physician, but may be carried out by athletic trainers or school officials who have been trained by licensed health-care professionals [3–5]. A discussion regarding risk factors, symptoms, and severity of concussions is critically important as SRC is generally underreported by athletes [3, 17]. In the case of youth and adolescent athletes, educating the family is of utmost importance, as the parent or guardian occupies a unique position to help in the recognition of SRC [3–5]. Ideally, physicians should disseminate information regarding the prevention of concussions to schools and sporting leagues [3, 5].

The role of preseason baseline testing remains unclear after the most recent meeting of the *Concussion in Sports Group* in 2016, where they declared that baseline testing was not mandatory but potentially beneficial in the post-injury diagnosis of concussion [1, 6]. There are four areas of baseline testing including baseline symptom scores, sideline evaluation testing, balance testing, and computerized neuropsychological (NP) testing [1, 18]. Commonly used baseline tests include the Sport Concussion Assessment Tool (SCAT 3/5) and the Immediate Post-Concussion Assessment and Cognitive Testing (ImPACT) [19, 20]. The reliability of baseline results compared to post-injury data varies depending on the test [21]. More research is needed to determine which baseline tests are most beneficial in the diagnosis of concussion [1, 21]. Fundamentally, baseline testing requires an honest effort on behalf of the athlete, as some athletes believe that scoring poorly on preseason tests will allow them to play through concussions during the season [21]. Physicians may also hold out an athlete following baseline testing if signs and symptoms point toward diagnosis of concussion [22].

The final prevention strategy is the development of a plan of evaluation following SRC [3, 4, 6]. For teams that do not always have a physician present at events, a concussion management protocol should be in place, whereby certified athletic trainers, school officials, and coaches are provided with instructions on when and how to contact emergency response personnel [4].

Management of the Concussed Athlete

On-Field Assessment

Signs and Symptoms

Concussion should be suspected during any play resulting in a direct hit to the head or neck region or any contact resulting in a whiplash-type motion of the head [2–4]. While it is difficult for physicians to observe every player on each play, the

Table 5.1 Symptoms following a concussion

Physical	Cognitive	Emotional	Sleep
Headache	Feeling like "in a fog"	Irritability	Drowsiness
Nausea or vomiting	Difficulty concentrating	"Don't feel right"	Sleep more than usual
Dizziness	Difficulty remembering	Sadness	Sleep less than usual
Loss of consciousness	Feels slowed down	Nervous or anxious	Hard to fall asleep
Seizures or convulsions	Forgets recent events	More emotional	
Neck pain	Confusion	"Pressure in head"	
Sensitivity to light	Repeats questions		
Sensitivity to noise	Answers slowly		
Balance problems	Amnesia		
Blurred vision			
Fatigue or low energy			
Stunned or dazed			
Numbness or tingling			

availability of video review at higher levels of athletics has dramatically improved our ability to recognize athletes at risk of concussion [2]. Importantly, most concussions occur without the loss of consciousness; as a result, physicians should rely on mechanism of injury and presenting symptoms [1, 3]. Symptoms of concussion fall under four clinical domains – physical, cognitive, emotional, and sleep – as shown in Table 5.1 [1–4]. There is no perfect diagnostic marker of concussion, and symptoms can change rapidly in the acute phase of injury; as such, athletes suspected of concussion should be removed from the game and be regularly reevaluated [1].

Evaluation Tools

Physicians should use the "Recognize and Remove" concussion signs from the SCAT3 or SCAT5 tests to determine when an athlete should be removed from a game and evaluated [2].

Immediate Management

In the case of an athlete lying motionless on the field, physicians should first assess for adequate airway, breathing, and circulation [2, 4, 16]. Next, a physical examination and focused neurological assessment should be performed to rule out cervical spine or severe brain injury and assess mental status [2, 6]. Level of consciousness should be evaluated followed by determination of whether to transport to the emergency room or assess and treat on the sideline [2, 4, 6]. If the player is to be taken to the hospital, the head and neck should be immobilized, and no attempt to remove equipment should be made until the ambulance arrives [2, 4, 6]. Table 5.2 summarizes the red flags which would prompt ambulance transport to a

Table 5.2 Concussion red flags

Red flags – call an ambulance		
Neck pain or tenderness	Severe or increasing headache	Deteriorating conscious state
Double vision	Seizure or convulsion	Vomiting
Weakness or tingling/burning in arms or legs	Loss of consciousness	Increasingly restless, agitated or combative

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local emergency department according to the Concussion Recognition Tool 5 [23]. There should be no same-day return to play for any athlete diagnosed with a concussion [1, 2, 4–6].

Sideline Assessment

Concussion diagnosis can be difficult in an athletic environment due to time pressures and noise level [2, 16]. Physicians should never feel rushed to make a decision even at the explicit wish of the athlete or coaching staff [16]. Ideally, a quiet room should be used, but this may not be possible and is not required [1]. The key tenet of sideline assessment for concussion is the use of rapid screening rather than definitive diagnosis [1]. Rapid testing paradigms used on the sideline are not designed to replace a comprehensive neurological evaluation; however, studies have shown increasing the number and types of tests increases both the sensitivity and specificity of SRC diagnosis [1, 24]. Regardless, physicians should err on the side of caution acknowledging the possibility of sitting out an athlete who does not actually have a SRC [1–3, 6].

Evaluation tools for sideline assessment are designed to evaluate physical, cognitive, somatic, and affective symptoms of concussion [2–4]. Prior to formal concussion assessment, a physical exam should be performed including an evaluation of cranial nerves to rule out more severe spinal and brain injuries which would require immediate treatment [2, 4, 6]. If physical exam findings are negative, neurological assessments including the SCAT3 (or newly released SCAT5) test should be performed to assess attention and memory function [1]. This test contains the Maddocks questions which evaluate for orientation to time and place [25, 26]. Standard orientation questions are ineffective in the sporting context compared with memory assessment [25, 26]. The SCAT3/5 also contains the Standardized Assessment of Concussion (SAC) which evaluates orientation, immediate memory, concentration, and delayed recall [27]. The sensitivity of these tests increases when used in combination with symptom inventory and balance tests [28]. The Balance Error Scoring System (BESS balance test) is an assessment of balance and postural control clinically supported in the diagnosis of concussion due to the temporary balance deficits found in concussed athletes [28–33]. It is helpful to compare sideline results to baseline scores if available, but this is not required [1,

5, 6]. Baseline scores for sideline tests vary significantly based on age, sport, sex, and comorbidities making evaluation without personal baseline data challenging [6, 10, 34–36]. Similarly, there are multiple confounding factors in the diagnosis of concussion as sideline tests have been shown to vary with maturation, mood, and fatigue [6]. For this reason, appropriate baseline testing is recommended before each season particularly in youth and adolescent athletes who are still maturing.

Athletes suspected of having a concussion are not to be left alone until a diagnosis has been made [4]. If a concussion is diagnosed, a disposition decision should be made to send the athlete home with family or keep them on site [4]. If they remain on site, it is advised to take a required piece of playing equipment from the athlete to prevent inadvertent return to play as there is no same-day return to play following SRC [1, 4–6]. Before sending the athlete home, plans should be made with parent/guardian for follow-up evaluation with a physician [4, 6]. The family should also be given take-home instructions including signs of worsening symptoms which warrant taking the athlete to the emergency room [4, 6]. Even if sideline screening is negative and the athlete returns to play, they should undergo follow-up evaluation after the game as some patients experience delayed-onset of concussive symptoms [1].

Clinical Assessment

Physicians from a variety of specialties are qualified to treat patients with SRC including pediatricians, primary care, emergency medicine, internal medicine, physiatrists, neurologists, neurosurgeons, and orthopedic surgeons [3]. Regardless of specialty, the evaluation should be treated similar to other musculoskeletal injuries despite the complexity of SRC [2]. The components of a complete clinical assessment include the BESS balance test, vestibular/vision assessment, Rivermead Post-Concussive Symptom Questionnaire (RPQ), physical evaluation of the cervical spine, SCAT3/Child SCAT3 (or newly released SCAT5/Child SCAT5), ImPACT, and King-Devick tests [2, 37] (Table 5.3).

Balance Error Scoring System (BESS Balance Test)

The BESS balance test is also used during sideline evaluation of athletes with suspected concussion [31]. The test involves having the athlete perform a double-leg stance for 20 s, a single-leg stance on each leg for 20 s, and a tandem stance for 20 s [2]. This test is useful for determining balance deficits in concussed athletes but has a relatively short window of sensitivity as most balance deficits resolve within 24 h [2]. Regardless, this makes a useful part of the clinical assessment to rule out long-term balance issues which are possible [2].

Table 5.3 Comparison of concussion assessment tools

Test	Advantages	Disadvantages
PCSS	Large battery of concussion-related symptoms tests	Subjective self-reported questionnaire; possible wide variability in results
SAC	Ease of administration (paper and pencil); high sensitivity and specificity	Cannot be used for continued monitoring due to rapid return to baseline (usually within 48 h post-concussion)
SCAT3	Wide variety of symptoms tested (including all symptoms in PCSS); separate version for children	Not a comprehensive neuropsychological test and therefore cannot be used alone
ImPACT	Comprehensive test with high sensitivity and specificity; can be used as a standalone test; can identify athletes attempting to hide symptoms; can be used for longer-term monitoring; separate version for children	Athletes more forthcoming with symptoms may display more normal behavior and decrease sensitivity of test
CRI	Highly sensitive and resistant to retest effects	Cannot be used for longer-term monitoring (many false positives on later tests)
CogSport	High correlations with paper and pencil neuropsychological tests	Reportedly high variability in sensitivity and specificity
KD	Easy to administer; tests eye movement and brainstem functions that other tests do not; able to identify events in athletes without symptoms of concussion (unrecognized concussions)	Not a comprehensive neuropsychological test; does not test many of the classic concussion symptoms

Table taken from Dessy et al. [38] (Open Access)
PCSS Post-Concussion Symptom Scale; *SAC* Standard Assessment of Concussion; *SCAT3* Standard Concussion Assessment Tool; *ImPACT* Immediate Post-Concussion Assessment and Cognitive Testing; *CRI* Concussion Resolution Index; *KD* King-Devick

Vestibular/Vision Assessment

The vestibular and vision assessment is comprised of two separate tests. The smooth pursuit test examines the ability to follow an object smoothly without effort, strain, or nystagmus. The convergence test evaluates the ability to follow a converging object without effort, strain, or diplopia [39, 40]. Both are useful in the evaluation of concussion as concussed patients may experience diplopia or other vision impairments [1, 2, 41].

Rivermead Post-Concussive Symptom Questionnaire (RPQ)

This test evaluates the clinical symptoms of concussion through a scaled questionnaire. The modified version separates scoring into two parts which allows the RPQ to be used clinically as a subjective test-retest assessment tool for concussion symptoms in patients experiencing symptoms over a more prolonged period of time [42].

Physical Evaluation of the Cervical Spine

Assessment of the cervical spine should be performed to rule out potential injuries which may be signs of a more severe injury [2, 43].

SCAT3/Child SCAT3

The SCAT3 is to be used for patients age 13 and over, while the Child SCAT3 is used in patients between the ages of 5 and 12 [44, 45]. As described in the sideline evaluation portion of this chapter, the SCAT3 incorporates the SAC and Maddocks questions as well as other assessments to provide a more sensitive and specific diagnostic tool for SRC [44, 45].

SCAT5/Child SCAT5

Following the 5th International Conference on Concussion in Sport, the *Concussion in Sport Group* released the SCAT5 and Child SCAT5 after systematically reviewing the most recent literature on SRC [19]. This version has several important changes to note including its duration, as the complete SCAT5 can no longer be performed in under 10 min. The new iteration also includes an immediate/acute assessment section which provides indications for emergency management [19]. Other modifications include the addition of questions comparing athlete's post-injury presentation with preinjury behavior, a lengthened SAC immediate and delayed word recall, additional versions of the Digits Backwards test, and emphasis that the physical and cognitive rest should typically only last 24–48 h [19]. Additionally, the SCAT5 includes "Recognize and Remove" criteria to assist health-care professionals in determining when to remove an athlete from the game [19]. These include neck pain, double vision, weakness or tingling in the arms and legs, severe or increasing headache, seizure, loss of consciousness, deteriorating consciousness, vomiting and increasing restlessness, or becoming agitated or aggressive [19]. Although there are no formal validation studies supporting the SCAT5 test as a diagnostic tool for SRC, the authors feel comfortable recommending its use as a diagnostic tool in place of the SCAT3 given the rigorous and systematic nature of its development [19].

Diagnostic Testing

Imaging

Plain skull radiographs, head computed tomography (CT), and magnetic resonance imaging (MRI) have limited value as diagnostic tests for SRC as SRC typically develops due to functional brain disturbances rather than structural changes

[1, 4, 5]. Indications for head imaging include concern for intracranial bleed, cerebral edema, or skull fracture. These clinical entities can present as worsening concussion symptoms, decreasing level of consciousness or persistent focal neurologic deficit; therefore imaging should be performed when experiencing any of these symptoms [4, 5]. Functional magnetic resonance imaging (fMRI), diffusion tensor imaging, and magnetic resonance spectroscopy are currently being used as research tools and may 1 day be clinically useful in the diagnosis of concussion [4].

Neuropsychological Testing

Neuropsychological testing (NP) by qualified neuropsychologists has clinical value in the evaluation of SRC but is not a requirement [4, 46–51]. Additionally, NP testing should not be used as a stand-alone tool in diagnosing concussion as symptoms do not necessarily correlate with NP scores [4]. As discussed in the sideline assessment portion of this chapter, NP testing data is more valuable in correlation with baseline test results for a specific athlete and is most valuable in conjunction with balance and symptom checklists [4]. Computerized NP testing may be used to test multiple athletes at once which is particularly helpful when performing baseline tests [4].

Biomarkers

Several blood biomarkers are being evaluated for their role in SRC including S-100 proteins, neuron-specific enolase, and tau proteins, but these are currently inconclusive in clinically diagnosing concussion [1, 4].

Event- and Evoked-Related Potentials

Electrophysiologic research has also been found to be inconclusive for the clinical management of concussion. Research is being conducted to determine the diagnostic utility of evaluating brain activity with quantitative electroencephalography (qEEG) and event- and evoke-related potentials for athletes suspected of suffering SRC [1, 4].

Genetic Testing

Specific genetic markers may contribute to increased risk of initial injury, prolonged recovery, or decreased long-term neurological health following SRC, but this research is not yet clinically relevant [1].

Referral Strategy

All athletes who suffer a SRC should be referred to a physician with experience managing athletic head injuries [16]. In some cases, it can take up to 72 h following a concussion for symptoms to develop; thus it is important to monitor injured athletes with serial exams for the first 48–72 h to ensure that appropriate diagnoses and follow-up is made [2]. Concussed athletes should not return to physical activity without being evaluated and cleared by a physician [16]. In general, there are three categories of SRC physician referrals: urgent referral, same-day referral, and post same-day concussion referral.

Patients presenting with red flag symptoms indicate a need for an urgent referral. Urgent referrals require immediate spinal immobilization with a cervical collar and transportation to the nearest trauma hospital by ambulance for further evaluation [6]. Red flag symptoms include but are not limited to Glasgow Coma Scale (GCS) score less than 13, mental status change from baseline, prolonged loss of consciousness, decreased neurological function or neurological deficit, persistent vomiting, seizure, severe or worsening headache, slurred speech, visual changes, unequal or unreactive pupils, confusion, or agitation.

A same-day referral is recommended when an athlete's symptoms meet the criteria for a same-day referral in the absence of red flag symptoms [2]. Berrigan et al. outline criteria for same-day referrals, which includes the following: no loss of consciousness, normal neurological exam, mild nausea, mild memory issues, headache that does not increase in intensity, and no other urgent indicators as listed above. It is important to note that an athlete should be monitored for at least 4 h before making the decision whether to refer as a same-day or post same-day concussion referral [2].

In the absence of symptoms meeting urgent or same-day referral criteria, it is recommended that an athlete be assessed by a physician for a post same-day concussion referral within 3–7 days of the concussive injury [2]. Post same-day concussion referral criteria include mild headache with the resolution of all other associated symptoms within 15 min of the initial injury. The goal of this visit is to monitor for symptom improvement, new signs or symptoms, or a change in the severity of symptoms [4].

Home Care

Athletes diagnosed with a concussion should be carefully monitored for the first 24–72 h following a SRC injury in order to identify an evolving issue. Concussed athletes should be provided with education on the signs and symptoms of concussion, symptom management strategies, and the risks of returning to sport without medical clearance [3]. The National Athletic Trainers Association recommends using an agreed-upon standard concussion home-instruction form for all concussed

patients [16]. It is also recommended to give oral and written home care instructions to both the patient and a responsible adult who will be observing the patient in the acute post-concussion setting [52, 53].

Physicians caring for athletes in the acute post-concussion setting should review the patient's home medications and adjust them accordingly; there is Level C evidence supporting the discontinuation of all medications other than Tylenol in athletes suffering a concussive injury [54, 55]. Athletes should also be instructed to eat a well-balanced diet and drink fluids to stay hydrated [53]. In the home care setting, athletes should be counseled to avoid mental or physical exertion that exacerbates their concussive symptoms [4, 31, 56].

Any acute change in a concussed athletes' symptom severity or deterioration in neurological status requires immediate physician evaluation [2]. Symptoms meeting the criteria for immediate physician evaluation in the post-concussion setting include but are not limited to the following: loss of consciousness, severe headache, increasing neck pain, dizziness, diplopia, repeated vomiting, focal weakness, confusion, seizure, slurred speech, or gait disturbances [2].

Special Considerations: Mood Disorders

SRC is often a challenging injury for student athletes; unlike most musculoskeletal injuries, the timeline for return to full activity can be difficult to project. In addition, athletes reportedly have unpredictable psychological responses to concussive injury [18]. There have been multiple reports of athletes experiencing emotional distress when they are kept out of competition for prolonged periods of time following a concussion injury [16]. Thus, it is important to monitor concussed athletes for signs and symptoms of depression. Health-care providers must also keep in mind that prolonged concussion symptoms may present similarly to mood disorders [57]. The importance of depression screening in this patient population cannot be emphasized enough, as passive management of a concussive injury in a truly depressed patient can be counterproductive and delay treatment [18, 58].

Initial treatment for SRC typically involves prescribed cognitive and physical rest [18]. The consensus statement from the Conference on Concussion in Sport (2016) recommends a brief period of rest during the acute post-concussion phase (24–48 h), followed by progressively increasing an athletes' activity level while ensuring to stay below the cognitive and physical symptom-exacerbation level [1]. There is emerging evidence from randomized controlled trials that targeted approaches for treating SRC in certain populations may, in fact, be more beneficial than previously believed [1, 46, 48, 59]. In fact, some evidence reports that prolonged rest following a concussive injury may lead to adverse effects including low self-esteem, academic difficulties, physical deconditioning, social isolation, and anxiety [18, 60, 61]. Given the potential adverse effects of prolonged rest in concussed patients, researchers are advocating for targeted treatments

that match an athlete's concussion symptoms (migraine, vestibular, oculomotor, cognitive function, etc.) [58, 62].

Recovery

Clinical recovery from concussion is defined in functional terms as a return to normal activities including work, school, and sport. This includes a resolution of post-concussion-related symptoms as well as a return to normal cognitive functioning status and balance ability. It has been well established in the literature that concussive injuries can have adverse effects on an athlete's balance and cognitive functioning for the first 24–72 h after injury [1, 31]. With this in mind, it is important to note that the severity of an athlete's initial symptoms has been proven to be the strongest predictor of a slower recovery time from concussion [1, 31]. Not surprisingly, having mild symptoms the day after a concussive injury is a favorable prognostic indicator [1, 31]. Risk factors for the development of persistent post-concussion symptoms lasting more than 1 month include concomitant migraine headaches or depression [1, 31]. Young adults with a pre-injury history of mental health problems have also been shown to be at a greater risk of developing persistent symptoms [1, 31]. Conversely, athletes with ADHD or learning disabilities have not been shown to be at a greater risk of persistent concussion symptoms [1, 31].

Establishing recovery from a concussive injury is a difficult task for physicians. To date, there is no gold standard for defining a physiological window of time for recovery from a concussion [1, 31]. Furthermore, determining recovery can also be clouded by subjective symptom scores and nonspecific clinical and neuropsychiatric testing. Often, physicians are tasked with making a difficult return to play decision with limited data to guide decision-making. In addition, recent literature suggests that physiological recovery time from a concussive injury may outlast clinical recovery [1, 31]; the consequence of these findings is currently unknown, but in theory, allowing athletes with ongoing brain dysfunction to return to play may expose them to additional risk.

Return to Sport

Graduated stepwise rehabilitation is the current recommended strategy for return to sport participation after a concussive injury. Of note, all current return-to-play guidelines for SRC are consensus based and have not been validated with evidence-based studies [5]. Most return-to-play protocols resemble the guideline presented in the Consensus Statement of Concussion in Sport; an example of this protocol is outlined in Table 5.4 [1]. However, an individualized approach for return-to-play is appropriate when managing SRC [18]. In these cases concussive symptomatology as well as modifiers that may prolong recovery, such as a history of prior concussions, migraine, or mood disorders, must be considered and return-to-play protocol modified

Table 5.4 Stepwise return to play protocol

Stage	Aim	Activity	Goal of stage
1	Symptom-limited activity	ADLs that do not provoke symptoms	Gradual reintroduction of work/school activities
2	Light aerobic exercise	Walking or stationary bike at slow to medium pace. No resistance training	Increase heart rate
3	Sport-specific exercise	Running or skating drills. No head impact activities	Add movement
4	Noncontact training drills	Harder training drills (e.g., passing drills) May start progressive resistance training	Exercise, coordination, and increased thinking
5	Full-contact practice	Normal training activity participation pending medical clearance	Restore confidence and assess function status by coaching staff
6	Return-to-sport	Normal game play	

accordingly [18]. Thus, the protocol outlined in the consensus statement should serve as a general guideline for return-to-sport following a SRC, but ultimately an experienced physician should make the final determination on timeline.

As mentioned earlier, initial management of SRC should include a brief period of rest (24–48 h) [1]. Athletes may not return to play the same day they suffer a concussive injury [1, 5, 16, 18, 31]. Concussed athletes should not return to sport-related activity for at least one calendar day following a concussion injury [18]. As stated earlier, all athletes should be evaluated by a health-care provider with experience managing SRC injuries before returning to sport. Once the athlete's symptoms have resolved and they return to baseline cognitive function, then return-to-play progression can be started [18].

Activity during rehabilitation is symptom-limited; while progressing through stepwise recovery, athletes must stay below their cognitive and physical symptom exacerbation threshold [1, 31]. Athletes must remain asymptomatic throughout a stage of recovery in order to advance to the next stage [18]. If at any point during recovery, an athlete becomes symptomatic, they should return to their previous level of activity [18]. The general outline of return-to-play progression typically begins with light aerobic exercise and then progresses to noncontact sport-specific activity, followed by contact sport-specific activity, and finally return to normal game play [1, 18]. Medical clearance is ultimately determined by the team physician or athletic trainer in consultation with team physician [1, 18].

Targeted Therapy

Specific cases of athletes with SRC have been reported to benefit from targeted treatment therapies. Athletes with prolonged concussive symptoms have been shown to benefit from progressive aerobic activity [63]. There is also evidence

supporting the use of vestibular therapies targeting proprioception, postural control, and deficits in gaze stability in patients with this clinical presentation [39, 64]. Vision therapies have been reported to be effective in treating concussed athletes with vision and ocular motor symptoms [65, 66]. It is unknown what effects targeted post-concussion treatments have on the underlying pathophysiology of concussion [18]. There is currently no empirical data to recommend timing or intensity of targeted therapy for treatment of patients with SRC [18]. Thus, guidelines for targeted treatment of concussive injuries should serve as an evolving framework for treating athletes.

Special Considerations: The Child and Adolescent Athlete

There are no evidence-based recommendations for treating age groups of children with SRC differently from adults [1]. Furthermore, no studies have assessed whether or not the signs and symptoms of concussion differ in children than in adults [1]. Concussion symptoms are expected to last up to 4 weeks in children [1, 31, 61]. Predictors of prolonged recovery from concussion in children and adolescents have not been well established [1, 31, 61]. Current recommendations for resumption of activity following concussion in children and adolescents are based on expert consensus. Similar to adults, recommended initial management of concussion in children and adolescents includes a brief period of physical and cognitive rest followed by symptom-limited activity progression. It is also recommended that children and adolescents not return to sport before successfully returning to school.

Premature Return-to-Play and Second Impact Syndrome

There are significant short-term risks associated with premature return to sport following a concussion. Athletes with persistent concussion symptoms that return to sport are at an increased risk of suffering recurrent or more severe concussions as well as developing prolonged symptom duration [6]. Second impact syndrome has also been reported as a consequence of premature return-to-play [6]. Not surprisingly, second impact syndrome is reported to occur when a patient suffers a second head injury before the symptoms of their first head injury resolve [67, 68]. The pathophysiology of second impact syndrome is thought to involve a loss of cerebral blood flow autoregulation resulting in vascular engorgement, increased intracranial pressure leading to brain herniation, and possibly coma or death [6, 67, 68]. There currently is debate as to whether the pathophysiology of second impact syndrome is related to a patient's first head injury or if it is its own form of malignant brain edema [69–71]. Interestingly, animal models have reported diffuse cerebral swelling after first impact [6, 72, 73]. Furthermore, case reports of second impact

syndrome are limited [6]. Whether or not second impact syndrome exists, its association with repeated concussion injuries is a compelling argument why athletes shouldn't return to play before their concussion symptoms resolve.

Post-concussion Syndrome

The majority of SRC injuries resolve within 1–2 weeks; in some rare cases, concussion symptoms may persist for months to years beyond the initial injury [6]. Post-concussion syndrome is defined as signs and symptoms of concussion that persist for months after the initial injury [34, 74, 75]. The development of post-concussion syndrome after an initial concussion injury is ill-defined and poorly understood [6]. Commonly reported post-concussion symptoms include insomnia, headache, dizziness, cognitive intolerance, exercise intolerance, depressed mood, anxiety, irritability, memory loss, poor concentration, fatigue, noise, and light sensitivity [6, 57]. There is no proven or accepted cause of post-concussion syndrome [6]. However, concussion severity has not been correlated with a higher likelihood of developing post-concussion syndrome [6]. Proven risk factors for the development of post-concussion syndrome include female sex, increasing age, or non-sports-related concussion [6].

Management of post-concussion syndrome revolves around time. Post-concussion syndrome recovery is a long, slow process that typically is frustrating for patients. Therapies that have proven to be beneficial in the treatment of post-concussion syndrome include supervised progressive exercise programs, cognitive therapy, and neurorehabilitation [74, 76, 77]. As with treatment of concussion, athletes with post-concussion syndrome should stay below their physical and cognitive symptom threshold when progressing back to normal activity [6].

Return to Academics

Return to academics after a SRC follows the same general principals as return-to-sport [78, 79]. Like return-to-play, return-to-learn should be managed in a stepwise fashion with special consideration for the individualized needs of the student athlete [78, 79]. Return-to-learn requires multidisciplinary support and should include physicians, coaches, athletic trainers, neuropsychologists, administrators, and academic representatives [18]. Like return-to-play guidelines, there is no evidence-based recommendation for return-to-learn following a SRC; all current guidelines are based on expert consensus [18].

Expert consensus recommends return-to-learn progression begin with a period of relative cognitive and physical rest [18]. Athletes must be counseled to minimize cognitive stressors, which include but are not limited to video games, school work, reading, texting, and watching television [18]. It may be beneficial for collegiate

athletes to avoid going to class, for at least 1 day following a SRC [18]. The timeline for return-to-learn should be individualized and based on concussion symptomatology. Like return-to-play, return-to-learn progression should be based on the absence of concussion symptoms following cognitive activity [18]. Student athletes who cannot tolerate light cognitive activity should remain at home until their symptoms resolve [78, 79]. If a student athlete becomes symptomatic during light cognitive activity, the team physician should be notified and the athlete's cognitive activity reassessed.

Typically, student athletes who suffer a concussive injury will not need a detailed return-to-learn plan because full recovery occurs, in most cases, within 2 weeks [18]. Some student athletes may require minor alterations to their academic schedule in the first 2 weeks following a concussive injury, but these adjustments can often be made without significant curriculum or testing alterations. Academic adjustments for student athletes with persistent symptoms lasting longer than 2 weeks should be determined by a multidisciplinary team that may include the team physician, coach, academic advisor, athletic trainer, teacher or professor, dean, and psychologist. Academic accommodations for student athletes can typically be accessed through an institution's disability services office.

Retirement from Sports

Currently, there are no evidence-based guidelines for retiring athletes after multiple concussion injuries. However, multiple authors have proposed criteria for considering disqualification of an athlete from sport; these include multiple lifetime concussions, persistent decreased academic performance, persistent concussion symptoms, structural abnormality on neuroimaging, prolonged recovery course, or perceived lower threshold of suffering recurrent concussions [80–82]. There is no agreed-upon lifetime number of concussions sustained before absolute disqualification from sport is necessary [6]. As with management of concussion, an individualized, multidisciplinary approach, including the student athlete, coach, team physician, and athletic trainer, is essential in determining retirement from sport due to concussion.

Residual Effects and Long-Term Sequelae

There is inconsistent literature on the long-term sequelae of exposure to repetitive brain trauma [1]. However, there is increasing concern that recurrent concussions are associated with the development of long-term neurobehavioral pathology including chronic traumatic encephalopathy (CTE) [1, 6]. CTE is a neurodegenerative disease characterized histologically by the accumulation of tau proteins in the brain [6]. CTE is a post-mortem diagnosis that is confirmed by histopathological

findings in the context of a patient developing typical CTE symptoms of depression, memory impairment, poor impulse control, and executive dysfunction preceding death [83–86].

CTE is a distinct entity independent of post-concussion syndrome or acute concussion; CTE typically develops decades after exposure [83–86]. Interestingly, not all athletes diagnosed with CTE have a reported history of concussion or multiple concussions, thus raising the question as to whether sub-concussive head injuries contribute to the development of CTE [6]. The incidence of CTE in the athletic populations is unknown [6, 87]. However, there are a far greater number of athletes participating in contact sports than reported cases of CTE; this fact likely indicates that there are, yet to be described, predisposing factors that play a role in the development of CTE outside of repetitive brain trauma [6, 87]. Researchers have not yet been able to demonstrate a cause-and-effect relationship between concussion and CTE [1]. Further research on CTE is needed to understand and clarify risk and protective factors, the incidence and prevalence of CTE in athletic populations, and clinical diagnostic criteria.

Prevention

Concussion prevention strategies can help reduce the severity and number of concussions in athletics. The evidence supporting the protective effect of helmets in contact sports is inconclusive as mandatory helmet regulations make study design difficult [1]. There is, however, adequate evidence supporting the use of helmets in reducing head injuries in skiing/snowboarding populations [1]. Mouthguard use has not been shown to be protective in preventing athletic head injuries [1]. Successful concussion prevention strategies that have consistently proven to reduce the frequency of athletic head injuries include limiting contact in youth football practices and body checking in youth hockey [1]. Individual concussion prevention strategies that address risk factors for concussion (e.g., tackling technique training) have not been shown to reduce the risk of concussion [1].

Expert Opinion

1. SRC is a traumatic brain injury that results in functional but rarely structural changes; thus, take caution when ordering and interpreting advanced imaging.
2. Education surrounding SRC should be implemented in preseason training by licensed health-care professionals.
3. As there is limited evidence as to which clinical measurement tool is best, health-care professionals should develop a protocol for their team with baseline and on-field assessments appropriate to their athlete population. This protocol should be updated regularly.

4. We recommend managing a SRC with a supervised progression of activity, focusing on an absence of symptom recurrence.
5. Exact return to sport timelines should be individually based, depending on symptoms and progression through a structured rehab program.
6. Considerations for retirement continue to evolve as long-term sequelae are still being investigated.

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Chapter 6
Persistent Post-concussion Symptoms and Long-Term Sequelae

Jacqueline Turner and Cynthia R. LaBella

Introduction

The majority of athletes recover from a concussion within a typical time frame. However, approximately 10–30% of athletes who sustain a concussion will experience a prolonged recovery [1]. There is no universal standard definition for persistent post-concussion symptoms. The Berlin Consensus statement defines persistent symptoms following a concussion as those lasting longer than 2 weeks in adult patients and longer than 4 weeks in pediatric patients, while the Centers for Disease Control and Prevention defines prolonged post-concussion symptoms as lasting longer than 1–3 months after the injury [2, 3]. Prolonged symptoms are not thought to be caused by a single pathological process within the brain, but rather a collection of a variety of symptoms that are often complicated by pre-existing and noninjury factors [3]. As such, athletes with prolonged recovery benefit from a multidisciplinary treatment approach to address their various symptoms [4].

Risk Factors for Prolonged Recovery

No two concussion recoveries are the same, even in the same individual; therefore, predicting precise recovery time is challenging. Nonetheless, there are some factors that have been shown to increase the risk for longer recovery including:

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history of previous concussion, neurological or psychological disorder, learning difficulties, family and social stressors, and increased concussive symptoms at baseline (pre-concussion) [2, 3]. Post-injury symptoms that have been associated with increased likelihood of prolonged recovery include dizziness, headaches or migraines, initial severity of cognitive deficits, deficits in oculomotor function, and development of depressive symptoms [3]. Risk factors for development of depressive symptoms and other mood disorders in elite and professional athletes include performance expectations, worries over career security, contracts, privacy of condition, and retirement from sport [5]. Some studies also found that women report symptoms for a longer period compared to men, and adolescents are at higher risk for prolonged recovery compared to other age groups [4]. Of all of these factors, pre-existing anxiety and high symptom load immediately after injury have been shown to be the most consistent predictors of prolonged recovery across all studies [3, 4].

How a concussion is initially managed may also influence recovery time. Athletes who stop playing immediately after the injury seem to recover faster than those who continue to play immediately after the injury [6]. Similarly, those who initiate physical and cognitive rest immediately after the injury have shorter recovery times than those who delayed rest for 1–7 days after the injury [7]. It is important to note that prolonged strict rest (more than a few days) after a concussion is associated with longer recovery times and therefore is not recommended [4]. An undiagnosed and therefore untreated concussion can also lead to prolonged symptoms [3]. Suffering another blow to the head while recovering from a concussion can increase the risk for prolonged symptoms. The brain has an increased vulnerability following an initial head injury which can result in more severe and prolonged concussion symptoms and worsened metabolic changes when a second injury is sustained [4]. In addition, athletes who receive a second blow to the head while still recovering from a concussion are at risk for "second impact syndrome." Although very rare and mostly documented in individuals of high school age and younger, second impact syndrome results in cerebral vascular congestion, diffuse edema, and death [8]. This is why current consensus guidelines emphasize no return to play on the day of injury, and that athletes must be completely recovered and back to their pre-concussion baseline before they can safely return to contact sports and other activities that pose a risk for head injury [3, 4, 8].

Treatment

Even in a patient with a known concussion, it is important to obtain a comprehensive history and physical exam looking for other etiologies of symptoms. Athletes reporting migraines or headaches should be evaluated for pre-existing or underlying disorders and have a thorough evaluation of their cervical spine for cervicogenic cause [4]. Due to the myriad of symptoms experienced by athletes during

their recovery, often a variety of treatments are needed to address their persistent symptoms. Treatment is therefore symptom-focused and can include cervical spine rehabilitation, sub-symptom threshold exercise training, vestibular and visual therapy, cognitive behavioral therapy, academic adjustments, lifestyle changes involving sleep, nutrition, and hydration, and rarely, pharmacologic treatments [3].

Cervical Spine Rehabilitation

It is not uncommon for the cervical spine to be injured at the time of a concussion, especially with whiplash type injuries. Missed diagnoses of other injuries inevitably lead to persistent symptoms from misdiagnosis. Dysfunction in the upper cervical spine can cause cervicogenic headaches leading to prolonged concussion headaches [9]. Physical therapy programs targeted at cervical spine dysfunction and have been shown to assist recovery in those with persistent post-concussion symptoms [4]. Cervical spine rehabilitation should include stretching and soft tissue massage to improve cervical muscle flexibility and reduce myofascial trigger points, as well as strengthening exercises and posture training. Modalities such as electrical stimulation and ultrasound may also be helpful.

Sub-symptom Threshold Exercise

An active rehabilitation program that includes limited aerobic exercise is a safe and effective treatment for athletes experiencing persistent symptoms [3, 4]. A unique feature of athletes with persistent concussion symptoms is that they often demonstrate physiologic differences when undergoing aerobic exercise, compared to non-concussed patients. Specifically, athletes with persistent symptoms report higher levels of perceived exertion and have lower heart rates during exercise [1]. Individuals who experience symptoms at a lower heart rate during sub-symptom threshold exercise testing take longer to recover compared to individuals that are able to tolerate exercising at higher heart rates [5]. An athlete has exercise intolerance when they cease exercise due to increase in symptoms at a submaximal intensity and have not yet reached exhaustion or maximum age-predicted heart rate [10]. The most studied test for measuring post-concussion exercise tolerance is the Buffalo Concussion Treadmill Test (BCTT). After establishing an athlete's aerobic tolerance using the BCTT, a structured aerobic exercise program, such as the Buffalo Concussion Exercise Treatment protocol, can be used to progressively increase their aerobic tolerance [1, 4]. For athletes who may not have access to skilled provider who can administer the BCTT and exercise protocol, healthcare providers can advise athletes to engage in light aerobic exercise for 20 min and to cease exercise sooner if their symptoms increase more than two points from baseline (on a

ten-point scale) [10]. In addition to facilitating recovery, sub-symptom threshold exercise training programs can also minimize physical deconditioning, while the athlete is resting from his/her sport [1].

Vestibular and Visual Therapy

Vestibulo-ocular dysfunction has been shown to be prevalent in patients with prolonged concussion symptoms [1]. Vestibular-Ocular Motor Screening (VOMS) is a clinical tool to evaluate oculomotor function and symptom provocation with ocular movements. VOMS assessment includes smooth pursuits, horizontal and vertical saccades, vestibular ocular reflex, near point of convergence, and visual motion sensitivity [4]. Mucha et al. provide a detailed description of how to administer VOMS in the setting of concussion [11]. Patients with persistent symptoms usually have difficulty with one or more of these ocular movements or experience symptom provocation during VOMS testing [4]. Individualized visual therapy is often helpful for these patients and can include exercises to retrain visual skills such as convergence, focus, tracking, and eye teaming.

Vestibular deficits may also be seen in patients with persistent symptoms. These may include difficulty with tandem walking, positive Romberg test, motion sickness, or symptoms of dizziness or nausea with positional changes. Vestibular therapies expose impairments with symptom provoking maneuvers in a controlled manner and direct the athlete's rehabilitation with focus on dynamic movements that cause an increase in symptoms [12]. Physical therapy programs targeted at vestibular dysfunction have been shown to facilitate recovery, specifically improving balance and reducing dizziness, in those with persistent post-concussion symptoms.

Academic Adjustments

Concussions can have effects on cognition including attention, memory, and executive function difficulties. Particularly in collegiate and high school athletes, memory difficulties, slower processing speed, and executive dysfunction interfere with learning and can negatively impact academic performance. These difficulties are often distressing for the student and can lead to significant anxiety which can compound the symptoms further. An individualized return-to-learn plan can help athletes with persistent symptoms continue to learn and succeed academically during their recovery [4]. It is important for the treating physician to get specific details from the athlete about which cognitive tasks they are struggling with so that an individualized academic plan can be developed and shared with the school and teachers. Athletes may have difficulty advocating for themselves when communicating with teachers, so providing this support for them and communicating with

school administrators and teachers can be essential in reducing their anxiety about falling behind academically.

Sleep Hygiene and Nutrition/Hydration

Athletes with persistent symptoms often experience one or more symptoms related to sleep such as difficulty falling asleep, daytime sleepiness, or sleeping more or less than usual. This can be problematic as getting adequate sleep is important to concussion recovery. As such, athletes should be counseled in sleep hygiene, including maintaining consistent sleep and wake times each day, limiting naps to 20–30 min to avoid interrupting nighttime sleep, and turning off electronics at least an hour before bedtime. Proper nutrition and hydration are frequently overlooked by athletes during concussion recovery, especially when they are not training intensively for their sport every day as they typically do when they are uninjured. Headaches and dizziness can be exacerbated by dehydration and low blood sugar, so they must pay careful attention to adequate hydration and eat small frequent meals that include protein, complex carbohydrates, and fruits and vegetables and are low in added sugar and unnecessary additives/preservatives.

Psychological Counseling and Cognitive Behavioral Therapy

Patients with prolonged symptoms are often frustrated that they have not yet recovered and may develop symptoms of anxiety and depression. Referral to a clinical psychologist can be very helpful as they can provide stress management strategies, relaxation techniques, and supportive counseling. They can also screen for more severe mood symptoms and refer to a psychiatrist for pharmacologic management if warranted. Cognitive behavioral therapy (CBT), a form of psychotherapy which aims to modify dysfunctional thinking and beliefs, has recently been shown to help patients with prolonged recovery manage their symptoms [4].

Pharmacologic Treatments

There is insufficient evidence to support the use of pharmacologic treatment of concussion symptoms [3]. A systematic review found no convincing evidence to support use of peripheral nerve blocks, amitriptyline, and amantadine in the treatment of persistent symptoms [1]. It is important to note that if an athlete is prescribed a medication for a persistent concussion symptom, they should be weaned off the medication and be symptom-free prior to returning to their sport [3]. Medication-overuse headaches can be caused by long-term use of

nonsteroidal anti-inflammatory medications and acetaminophen, and therefore chronic use of over the counter analgesics should be discouraged [8].

There has been no research on humans to show that any nutritional supplements can prevent or treat concussion symptoms [4, 8]. Additionally, they are not FDA regulated and therefore have potential for harm or contamination. As a result, using supplements in the treatment of concussion is not recommended [4].

Prevention of Persistent Symptoms

Participation in both physical and cognitive activities should be limited in the first few days following a concussion. Then the athlete's usual activities should gradually be resumed as tolerated as long as they do not increase the number or severity of symptoms. This includes engaging in light aerobic activity as tolerated. In fact, early individualized sub-symptom threshold exercise training may reduce chances of prolonged recovery from concussion in athletes [13]. Prolonged strict rest can increase symptom burden [2].

Retirement from Sport

There are no evidence-based recommendations to guide physicians and athletes regarding retirement or extended breaks from contact sports in the setting of concussion. Current recommendations are based on expert opinion. Consideration for retirement or an extended break from contact sports should not be based solely on the number of concussions, but rather on the quality of recovery from each concussion. As such, factors that should prompt a discussion and consideration of extended time off from contact sports include persistent post-concussive symptoms, worsening symptoms with each subsequent concussion, repeated concussions occurring with lesser and lesser force, persistent deficits on neurologic exam or neuropsychiatric testing, certain traumatic structural injuries or abnormalities on neuroimaging (e.g., large arachnoid cyst, intracranial hemorrhage, or Chiari malformation), and decline in academic or athletic performance. An athlete's readiness to return to sport should also be considered after each injury [4, 5]. Any decision regarding retirement or extended break rest from contact sports should be evaluated on an individual basis and include a comprehensive discussion with the athlete and multidisciplinary medical team of the long- and short-term benefits and risks of continued participation in contact sports.

Long-Term Sequelae

Recently, there has been significant concern about the potential long-term effects of repeated concussions on cognitive function and emotional health [8]. Small autopsy studies of mostly former professional contact-sport athletes who had

sustained numerous concussions have demonstrated deposition of hyperphosphorylated tau (p-tau) protein around small blood vessels of the cortex, typically at the sulcal depths. This has been labeled, "chronic traumatic encephalopathy" (CTE). CTE can only be diagnosed on a postmortem autopsy. Currently, there is no way to conclusively diagnose CTE in a living individual [8]. Additionally, a cause and effect relationship between CTE found on autopsy and behavioral changes, mood disorders, or cognitive difficulties during life has not been demonstrated [3, 4]. Further research is needed to determine what factors may predict or predispose an individual to permanent or long-term sequelae after a concussion [3, 4, 8].

Expert Opinion

- Approximately 10–30% of athletes with concussion will experience symptoms that last beyond the expected recovery time period (2 weeks for adults and 4 weeks for children).
- The strongest risk factors for prolonged recovery are pre-existing anxiety and high symptom load immediately after the injury.
- Risk for prolonged recovery may be reduced by removing the athlete from play immediately after the injury, initiating relative physical and cognitive rest for the first few days and then gradually resuming usual activities as tolerated. Resumption of activities should include sub-symptom threshold aerobic exercise which can help facilitate recovery.
- Prolonged, strict rest after a concussion is no longer recommended as it has been shown to increase the risk for persistent symptoms. Treatment for athletes with persistent symptoms is symptom-targeted and requires a multidisciplinary approach.
- Athletes with persistent symptoms can benefit from cervical spine rehabilitation, sub-symptom threshold exercise training, vestibular and visual therapy, cognitive behavioral therapy, and counseling with regard to sleep hygiene, nutrition, and hydration.
- More research is needed to understand the potential long-term effects of one or more concussions on cognitive function and emotional health, who is at risk, and how this risk might be mitigated.

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Part II
Injuries to the Cervical Spine

Chapter 7
Evaluation of Athletes with Neck or Arm Pain

Michael H. McCarthy, Joseph A. Weiner, and Todd J. Albert

Introduction

Physicians treating injured athletes have a unique and difficult role in both triaging a wide range of injuries and identifying life-threatening conditions. High-intensity athletic activity has the potential to place athletes at a significant risk for musculoskeletal injuries, which can range from sprains, tendon and ligamentous ruptures, and even fractures. Providers must keep in mind mechanisms of injuries resulting from acute trauma in addition to overuse and/or repetitive injuries when triaging neck and shoulder injuries. Although upper extremity injuries are common among athletes, cervical spine injuries (CSIs) [1] are rare, with 2.4% of athletic-associated hospitalizations related to CSIs [2, 3]. Pathologies of the cervical spine and upper extremity commonly have overlapping symptoms and exam findings, posing a diagnostic challenge to treating physicians. The confounding variables present among athletes with neck and arm pain can lead to confusion among providers underscoring the importance of a broad differential diagnosis.

It is important for providers to have a high suspicion for CSI in any athlete with either arm and/or neck pain, since 9.2% of all spinal cord injuries, within the United States, are sustained during athletic activity [4]. Although the most common sports-related CSIs are strains and sprains to muscles and ligaments supporting the spinal column, neurologic and bony injuries are the most concerning and should prompt immediate medical attention. The most common fractures are compression, burst, transverse process, and spinous process fractures [5]. Neurologic injuries can range

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from complete and incomplete spinal cord injuries, transient quadriparesis, to stingers/burners, representing the wide spectrum of severity associated with these injuries. Stingers/burners are a relatively common injury characterized by unilateral burning pain radiating down the neck to the arm and hand. Stingers/burners represent a neuropraxia of a cervical root or brachial plexus and typically resolve within seconds to hours and rarely persist beyond a 24 h period [6]. Transient quadriparesis, also known as cervical cord neuropraxia, is characterized by paresthesia and/or weakness in more than one extremity with rapid and complete resolution of symptoms and normal physical exam within 48 h after injury. Complete and incomplete spinal cord injuries are devastating insults which can lead to permanent neurologic injury and even death.

Management of CSIs among athletes requires a thorough working knowledge of the neurologic exam in addition to a heightened suspicion of potential underlying catastrophic injury. Symptoms may present as acute or chronic in nature, in conjunction with a specific mechanism, or as a vague complaint of pain. The diagnostic challenge is addressed by formulating a broad differential diagnosis and utilizing the physical exam and history to achieve a correct diagnosis.

Epidemiology

The mechanism of injury in cervical spine and shoulder injuries varies based on the specific sport being played, as well as the sport-specific position. The growing number of participants in interscholastic sports increases providers' likelihood of treating sports-related injuries among young athletes. The CDC estimates that high school sports alone account for approximately 2 million injuries, 500,000 physician visits, and 30,000 hospitalizations every year [7]. Between 2017 and 2018, nearly 8 million high school athletes in the United States competed in interscholastic sports, with the highest participation in football, track and field, basketball, and soccer [8].

During the 1960s and 1970s, the advent of the spear tackle in American football, tackling with the crown of the head, correlated with an increase in the number of CSIs among these athletes [9]. Improvement in helmet design decreased the number of head injuries but was thought to encourage the use of the device as a battering weapon. Biomechanical studies during this time demonstrated the impact of axial loads on the cervical spine; in 1976, spear tackling was banned, leading to a marked decrease in the number of CSIs in football [10]. The ruling decreased the number of deaths related to spinal cord injuries from 40 between 1965 and 1974 to 5 such fatalities between 1985 and 1994 [11].

Football is one of the most popular sports among American male high school students and is one of the leading causes of sports-related injuries, followed by wrestling, girls' basketball, and girls' soccer [12]. Shoulder injuries account for 11% of all injuries among high school athletes, whereas cervical spine injuries account for less than 1% [12, 13]. Among football players, injuries to the axial skeleton most commonly occur in the cervical spine; however, less than 1% of cervical

spine injuries result in cervical spine fractures or spinal cord injuries [14]. An annual survey between 1977 and 2012 found 327 CSIs, defined as cervical spine fractures with concomitant neurologic symptoms, reported in football players. Of the 327 CSIs, 8 occurred in players below high school age, 266 occurred in high school players, 38 occurred in college players, and 15 occurred at the professional level [15]. In a study of high school and college football players between 2005 and 2006, CSIs comprised 4.1% of all reported injuries with 41% of these CSIs attributed to muscle strains and 25.7% associated with nerve injury [16]. Although CSIs are relatively rare, more common neurologic sequelae, such as stingers/burners, have been reported in 50–65% of football players [6].

As the data above illustrates, devastating CSIs are rare, but CSIs are still common injuries among athletes. Schroeder et al. reviewed the records of 2965 athletes attending the National Football League combine from 2003 to 2011 and found 4.8% of these athletes were diagnosed with a cervical spinal disorder, pathology, or injury, with the three most common diagnoses being cervical spondylosis, cervical stenosis, and cervical herniated disk [17]. These injuries can have a significant impact on athletic performance, return to play, and career longevity.

Upper extremity injuries, specifically shoulder pathology, can have a significant impact on athletes. Overlapping symptomatology between the neck and upper extremities can pose as a challenge to providers, which necessitates a thorough understanding of injury specific incidence. Three of the top 15 musculoskeletal diagnoses made in the NFL combine between 1987 and 2000 were related to shoulder pathology [18]. Specifically, these shoulder injuries included stingers/burners, shoulder instability, and acromioclavicular joint (AC) injury. Almost 50% of football players in the 2004 NFL combine reported previous shoulder injuries during their career, and of those, 34% required surgical management [19]. Understanding the intersection between CSIs and upper extremity injuries is vital in establishing the diagnosis and formulating an appropriate treatment plan.

Patient History

The initial focus of both on- and off-field evaluations should center around the prompt recognition of any potentially catastrophic injuries. Proper management ensures that movement does not exacerbate any initial injury to the spinal cord. Further on-field assessment and management are covered later in this chapter.

Following the initial on-field examination, or in the evaluation of the athlete in the office, a comprehensive history and examination should be completed. A comprehensive history includes evaluating the presenting symptoms in terms of onset, location, character, severity, radiation, exacerbating and alleviating factors, and prior treatments. It is critical to also understand the medical history of the patient, including underlying medical conditions, medications, and prior surgical interventions. Patients should always be questioned about previous neck pain, radicular symptoms, extremity pain, and other neurologic symptoms. Many athletes may

have prior imaging which can reveal the presence of congenital cervical stenosis, a condition which can greatly increase the risk for neurologic injury [20].

A thorough knowledge of common cervical spine injuries in athletes will allow providers to formulate an accurate differential diagnosis. Strains and sprains to the paraspinal muscles and cervical spine ligaments are the most common types of injury [5]. These injuries generally cause axial neck pain. However, axial neck pain may also be associated with cervical spine fractures or other more serious injuries [5]. Neurologic injuries can involve the spinal cord, nerve roots, brachial plexus, and peripheral nerves. Apart from catastrophic spinal cord injury, common cervical neurologic injuries include stingers/burners and transient quadriparesis. The focus of the patient history should be the differentiation between axial neck pain, radicular pain, spinal cord injury, and other common masqueraders of cervical pathology. Each of the common cervical pathologies will be discussed in detail in this chapter.

Physical Examination

Once catastrophic spinal cord injury and cervical instability have been ruled out through the initial on-field evaluation, a more comprehensive cervical spine examination should be performed in the hospital or office setting. The chief complaint of athletes with neck pathology will generally be pain; however, it is essential to investigate associated neurologic symptoms such as weakness and numbness. Pain of cervical origin generally falls into one of the following categories: axial pain, referred pain, radicular pain, or myelopathic pain. The physical examination should serve to identify the source of pain and neurologic deficit.

Cervical Exam

The exam should be systematic to differentiate between non-neurologic and neurologic pain from compression of the neural elements. The examination should always include a concise general physical examination. This is important to evaluate for systemic conditions that can present with neck pain as a primary or secondary symptom, such as meningitis. The first step in the neck examination is observation; note the patient's head position, overall posture, arm position, prior surgical incisions, and external signs of trauma. Patients with cervical muscle spasm will often hold the head in a rigid position and have reduced neck motion. Patients with cervical radiculopathy may hold the head tilted away from the affected side or hold the arm abducted to decrease tension on the affected nerve root.

Next, the range of motion of the cervical spine should be assessed. Evaluation of cervical motion includes both passive and active range of motion. Normal range of motion in young healthy controls has been described as 60° of flexion, 75° of extension, 45° of side bending, and 80° of rotation [21]. When assessing older ath-

letes, it is important to note that range of motion predictably diminishes with age [21]. Observe for limited motion and measure the degree of limitation.

Palpation of the cervical spine can be useful to identify localized muscle tenderness or bony tenderness. Palpation should focus on the spinous processes, the paraspinal muscles, and the underlying facet joints. While this can be somewhat useful to localize general anatomic regions of pain, palpation has overall poor sensitivity and specificity for identifying specific cervical pathology [22].

Neurological Exam

After completion of the local cervical examination, a comprehensive neurological examination must be completed, including a detailed motor examination, assessment of reflexes, and a sensory examination. Motor evaluation must include both upper and lower extremities with evaluation of each muscle group supplied by its respective nerve root (Table 7.1). Weakness in the distribution of a single upper extremity nerve root is indicative of radiculopathy, while bilateral weakness, lower extremity dysfunction, or involvement of multiple roots is concerning for spinal cord dysfunction. Assessment of both upper and lower extremity reflexes helps to localize the neurologic injury (Table 7.2). Diminished reflexes in the distribution of a single nerve root indicate a lower motor neuron (nerve root) injury. On the contrary, hyperreflexia indicates upper motor neuron (spinal cord) dysfunction. Finally, a sensory examination, including assessment of sharp, light touch, vibration, and temperature, should be completed to outline the dermatomal distribution of any sensory disturbances.

Table 7.1 Myotomes

Nerve root	Myotome
C5	Shoulder abduction/elbow flexion
C6	Elbow flexion/wrist extension
C7	Elbow extension/wrist flexion
C8	Finger flexion
T1	Finger abduction
L2	Hip flexion
L3	Knee extension
L4	Ankle dorsiflexion/Hip adduction
L5	Hallux extension/Hip abduction
S1	Ankle plantarflexion

Table 7.2 Reflexes

Nerve root	Reflex
C5	Biceps
C6	Brachioradialis
C7	Triceps
L4	Patellar
S1	Achilles

Spurling's Test

Spurling and Scoville first described the Spurling neck compression test, also known as the foraminal compression test, in 1944 [23]. The test is a highly sensitive and specific maneuver for detecting cervical radiculopathy validated by magnetic resonance imaging [1] and electrodiagnostic studies [24–26]. It involves moving the neck into extension and lateral bending toward the affected arm and then applying axial compression. The purpose of this maneuver is to compress the neural foramen; a positive result is the reproduction of radicular pain and paresthesia.

Shoulder Abduction Test

The shoulder abduction test, first described by Spurling in 1956, is a helpful ancillary test for evaluating neck and arm pain. This test is performed by actively or passively abducting the ipsilateral shoulder so that the hand rests on top of the head. Resolution or reduction of ipsilateral radicular symptoms is a positive test. The overall sensitivity has been described between 43% and 50% and specificity from 80% to 100% [27]. Abduction of the shoulder leads to a decrease in tension on the affected nerve root, thus reducing radicular symptoms.

Lhermitte's Sign

In addition to special physical exam maneuvers for radiculopathy, several provocative tests have proven useful for the diagnosis of myelopathy. Lhermitte's sign consists of "electric-like" sensation down the spine or in the extremities with passive cervical flexion. This occurs with cervical spinal cord pathology from a variety of conditions, including multiple sclerosis, spinal cord tumor, and cervical spondylotic myelopathy [28]. Few studies exist regarding the diagnostic validity of Lhermitte's sign, with most demonstrating a poor sensitivity from 3% to 17% [29].

Hoffman's Sign

In the presence of upper motor neuron dysfunction, such as cervical myelopathy, several pathologic reflexes can be elicited. Hoffman's sign is elicited by supporting the patient's hand so that it is completely relaxed and the fingers partially flexed. The middle finger is firmly grasped and partially extended, and the distal phalanx is snapped by the examiner's thumb. The sign is present if flexion of both the thumb

and index finger is observed. Presence of Hoffman's sign has been reported to have 58% sensitivity and 78% specificity for cervical spinal cord compression [30].

Upper Extremity Exam

Many non-neurologic conditions can mimic cervical pathology, and physicians must always consider alternative diagnoses. A full upper extremity exam assessing passive and active range of motion, strength, and sensation can be helpful in differentiating cervical pathology from extremity pathology. This should include focused examination of the shoulder, elbow, wrist, and hand. Rotator cuff disease, a common condition among athletes, may cause referred pain to the cervical spine along with shoulder pain radiating to the upper arm. This pain is generally worse with shoulder elevation and never radiates below the elbow. Patients will generally have evidence of impingement with positive Neer's and Hawkin's signs on physical exam.

Another common mimic of cervical pathology is peripheral nerve entrapment. This commonly creates sensory deficits, pain, and weakness that may be in similar distributions to a cervical radiculopathy. Carpal tunnel syndrome, which is caused by compression of the median nerve at the wrist, presents with decreased sensation in the radial digits. This is similar to a C6 radiculopathy; however, carpal tunnel syndrome should not impact wrist extension strength nor cause pain proximal to the elbow. Similarly, cubital tunnel syndrome or compression of the ulnar nerve at the elbow can often mimic a C8 radiculopathy, which is discussed further in this chapter. Peripheral nerve compression should be considered and ruled out in all patients with radiating extremity pain using a systematic physical exam and electromyogram, if necessary. In patients with upper limb pain, it is important to exclude local structural limb and joint pathology. This includes use of passive and active ROM of the shoulder, elbow, and wrist as well as careful palpation over the acromioclavicular joint and the ventral aspect of the shoulder for bicipital tendinitis. Consider rotator cuff pathology if attempts to abduct the shoulder result in pain.

Diagnostic Imaging

Diagnostic imaging in the evaluation of cervical spine injuries has become nearly universal across the United States. However, many have questioned whether routine imaging is necessary or merely a response to the medicolegal environment [31]. Two large studies addressed the need for imaging in cervical spine trauma: the National Emergency X-ray Utilization Study (NEXUS) and the Canadian Cervical-Spine Rule Group (CCR). The NEXUS study outlined that imaging is not necessary if the patient has no midline cervical tenderness, no focal neurological deficit,

Table 7.3 NEXUS low-risk criteria

No posterior midline cervical spine tenderness
No evidence of intoxication
Normal level of consciousness
No focal neurologic deficit
No painful distracting injury

normal level of alertness, no intoxication, and no painful distracting injury (Table 7.3) [32]. The Canadian Cervical-Spine Rule suggests that if a patient has any high-risk factors (age >65, a defined dangerous mechanism or paresthesias in the arms or legs), then they require c-spine imaging. If a patient has neck pain and does not meet any of the high-risk factors, yet cannot meet low-risk criteria (sitting position in the ED, ambulatory at any time, delayed neck pain, no midline tenderness, simple rear-end motor vehicle collision), they require c-spine imaging. If the patient with neck pain has no high-risk factors but meets one low-risk criteria, then it is safe to assess whether the patient can rotate their neck 45°. If successfully able to rotate the neck 45° in either direction, then they do not require further imaging. Otherwise, c-spine images are indicated (Fig. 7.1) [33].

Plain Radiography

Radiographs of the cervical spine can be a quick and cost-effective initial study in athletes with neck pain. In the setting of trauma, the utility of radiographs has largely been replaced by computed tomography. Adequate radiographs should include all seven cervical vertebrae, including the C7–T1 disk space. Multiple orthogonal views should be obtained: AP, lateral, open-mouth odontoid view, and oblique views to evaluate for foraminal stenosis. A swimmer's view can aid in assessing the cervicothoracic junction. If there is concern for dynamic instability, flexion/extension views can be beneficial.

Computed Tomography

Computed tomography (CT) has largely superseded plain radiographs as the study of choice for the evaluation of cervical spine trauma. CT is the most efficient modality for detecting skeletal injuries and is almost 40% more sensitive than a single lateral radiograph [34]. In 2007, the American College of Radiology formulated an algorithm for cervical spine trauma: if NEXUS or CCR criteria indicate low risk, then no imaging should be performed. If NEXUS or CCR criteria indicate imaging, then a CT of the cervical spine with sagittal and coronal reformations is highly recommended [35]. CT imaging is relatively quick and provides more granular information of injury morphology; thus, many trauma centers opt for CTs of the cervical spine rather than initial plain radiographs.

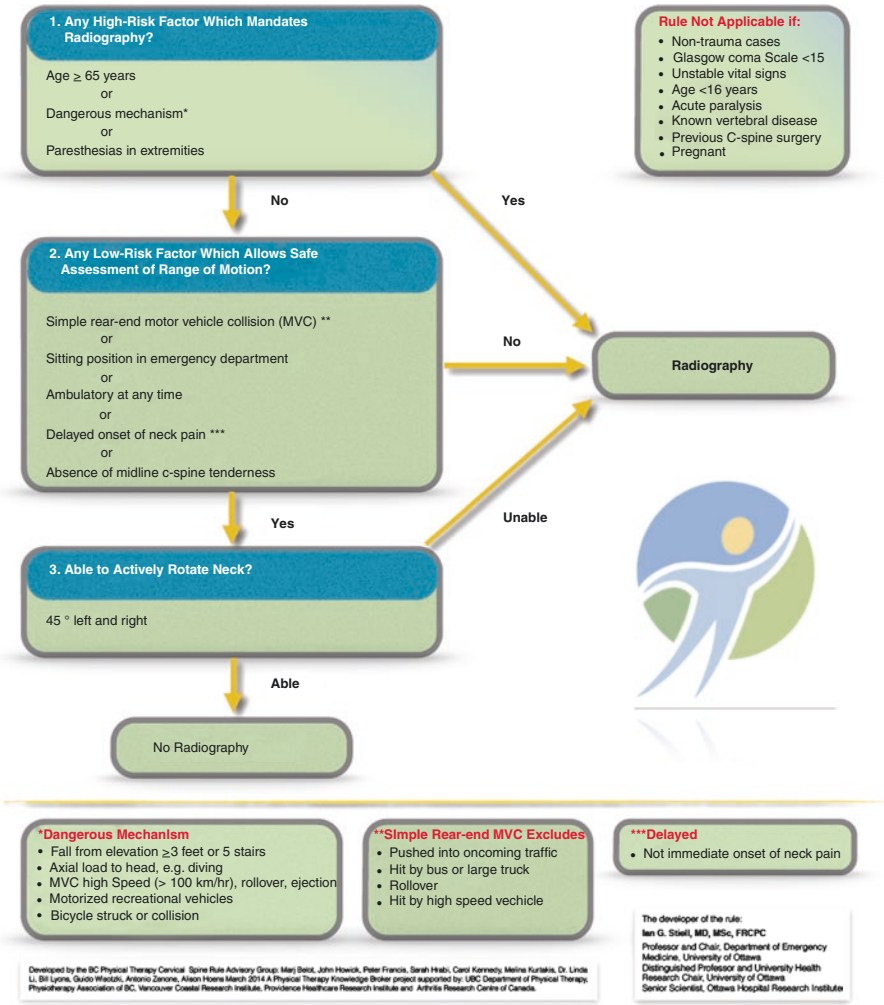

Fig. 7.1 Canadian spine rule algorithm. (Source: The University of British Columbia, Department of Physical Therapy. The Canadian C-Spine Rule Project. https://physicaltherapy.med.ubc.ca/physical-therapy-knowledge-broker/the-canadian-c-spine-rule-project/. Accessed 15 May 2019. Reprinted per terms of Creative Commons Attribution-ShareAlike 4.0 International License)

Magnetic Resonance Imaging

Magnetic resonance imaging [1] is a noninvasive imaging technique that can differentiate between various soft tissues and bone based on water content. MRI is an excellent imaging modality for evaluating the neural elements, ligamentous structures, and intervertebral disks. Generally, MRI is a useful modality for evaluating athletes with neck pain and neurologic symptoms in the outpatient

setting. MRI is highly sensitive at detecting degenerative changes, such as disk bulges and cervical spondylosis. However, in the general population, lifetime prevalence of degenerative changes in the cervical spine on MRI in asymptomatic patients younger than 40 years old is 25% [36]. Athletes who suffer repeated microtraumas to the cervical spine are at greater risk for degenerative disk disease compared to the general population [37]. Therefore, diagnostic imaging may frequently identify asymptomatic pathology and should be considered only as an adjunct to history and clinical examination.

Non-contrast MRI is the imaging modality of choice in athletes with clinically diagnosed radiculopathy that fails to improve with conservative therapy. If history and physical examination reveal concern for cervical myelopathy, MRI should be obtained to evaluate for the source of spinal cord compression. If an MRI is contraindicated (i.e., metallic implants), CT myelogram should be considered.

The role of MRI in acute cervical trauma is still debated. The American College of Radiology does not support the routine use of MRI for all trauma patients with neck pain. They recommend MRI in the setting of trauma only if NEXUS or CCR criteria are met, and there are clinical findings of myelopathy and neurologic deficit, clinical or imaging findings to suggest ligamentous injury, or clinical concern for an unstable spine [35].

Initial Management/Treatment

The initial on-field evaluation of an athlete with a suspected cervical spine injury is typically initiated by athletic trainers and/or a trained group of personnel. Basic equipment, such as a stretcher, spine board, and tools to remove protective gear, should be readily available and identified during pregame preparations. Assessing cardiopulmonary status, following basic/advanced life support protocol, and maintaining cervical immobilization are of the utmost importance during initial triage of significant cervical spine trauma. Athlete's helmets and pads should remain in place during the triage period due to the support and alignment provided to the injured spine [38]. Immobilization of the cervical spine should be maintained, and removal of face mask might be necessary for airway control. Athletes with neck pain/tenderness, limited cervical motion, neurologic symptoms, or altered mental status should be promptly transferred to the nearest trauma center. Helmets should remain in place until multiple trained healthcare workers are available to aid in removal [39].

Any subacute presentation or ambulating athlete presenting with tenderness, pain, or decreased range of motion after trauma should undergo a full spine and neurologic exam in addition to any specific musculoskeletal exam. During the initial triage, if there is suspicion of any structural or neurologic injury, the athlete's cervical spine should be promptly immobilized until full evaluation at the nearest trauma center.

After the athlete is transferred from the field to the nearest trauma center, primary and secondary surveys, in addition to full neurological examination, should be completed. Any complaints of shoulder or upper extremity pain should prompt a thorough musculoskeletal exam to assess for possible fractures, dislocations, or ligamentous/tendon injuries. Cervical radiographs, including anterior-posterior, lateral, and open-mouth series, are helpful in assessing for obvious bony or ligamentous injuries. CT imaging provides a more granular assessment of subtle injury patterns and should be utilized in cases where radiographs are inadequate. Triaging providers should promptly involve a consulting spine surgeon once initial imaging is obtained. Lastly, if the patient is stable and has concomitant signs or symptoms suggestive of a spinal cord injury, a cervical MRI is warranted.

Specific treatment of cervical spine injuries depends on existence of ligamentous disruption, dislocations, fractures, or spinal cord injury. Although unstable cervical spine injuries are rare among athletes, surgical intervention is necessary for these injury patterns. Stable cervical injuries are more common among this population and can be treated conservatively with short periods of immobilization followed by rehabilitation programs encouraging range of motion and strengthening exercises.

The majority of these rehabilitation protocols start with isometric exercises, followed by concentric exercises and gradual increasing range of motion [6]. Stretching exercise should be avoided during the acute inflammatory phase, and strengthening should only be initiated once painless range of motion is achieved [40]. Factors such as bony healing and achieving painless range of motion impact athlete's rehabilitation. Vaccaro and Kepler proposed nine absolute contraindications for athletes with previous cervical spine fractures from participating in sports: occipital-cervical arthrodesis, atlantoaxial instability, spear tacklers spine, residual subaxial spine instability, substantial sagittal malalignment, narrowing of the spinal canal as a result of retroposed fragments, residual neurological deficits, loss of cervical spine range of motion, and arthrodesis of three or more disk levels [41]. Once athletes demonstrate full strength and capabilities comparable to preinjury state, the topic of return to play should be broached. The decision to return to play is predicated on the individual athlete, previous injury, specific treatment, and the requirements of the sport.

Diagnostic Dilemmas

There are numerous diagnostic dilemmas when evaluating patients with neck pain with or without concomitant upper extremity pain and/or weakness. The significant overlap of symptoms generated from upper extremity and cervical spine pathology can create a perplexing diagnostic situation. It is important for providers to create a broad differential when approaching patients with cervical spine and upper extremity pain while utilizing well-executed and focused exams to refine potential diagnosis.

Transient Spinal Cord Injury

Transient spinal cord quadriparesis, also known as cervical cord neuropraxia, is traditionally seen in contact sports, such as football or hockey, where collisions can impart sufficient force, leading to spinal cord injury. Transient paralysis in these situations is characterized by paresthesia +/− weakness in affected extremities, typically resolving within 30 min but may last up to 24–48 h. The most common pattern of this injury results in a combination of weakness, quadriplegia, and/or sensory deficits in all four extremities (~80% of cases) [42]. Numerous mechanisms have been proposed, including spinal cord concussion, failure of neural transmission, and selective vascular attenuation, which likely result from structural insults such as fractures/dislocations, ligamentous infolding, instability, syrinx, and/or herniated nucleus pulposus.

Athletes with congenital cervical stenosis and acquired cervical stenosis, typically secondary to degenerative osteophyte formation, are defined as having a spinal canal diameter of 14 mm or less. These athletes have a notably high incidence of spinal cord and peripheral nerve injuries, and return to play or clearance for athletic participation must entail a thorough discussion with the athlete/family/agent as to the risks of catastrophic injury. Return to play for athletes with known cervical stenosis is a controversial topic, but surgeons will generally broach the topic of return to play after full neurologic recovery and without ongoing insult. However, all parties must have a thorough understanding of the inherent risks.

Cervical Sprain/Strain vs Cervical Spondylosis

The athlete with a cervical sprain will primarily complain of neck soreness and tightness. It often occurs in association with a traumatic event. There are typically no neurologic manifestations, and paraspinal muscle tenderness is the hallmark finding.

Whereas, cervical spondylosis will not usually be associated with a traumatic event, the athlete will primarily complain of neck pain and stiffness. It often is not associated with a traumatic event. It will tend to be symptomatic in older athletes. Neurologic manifestations can be present from osteophyte compression on cervical roots or the spinal cord. Paraspinal muscle tenderness will be present less frequently than with a sprain/strain.

Axial Neck Pain/Referred Pain vs Cervical Radiculopathy

Axial neck pain is a common complaint among athletes, especially those participating in contact sports. The cervical spine is a multifaceted region composed of neural elements, discoligamentous complexes, bone, facet and uncovertebral joints, and

paraspinal musculature. The interaction of these complex cervical components can represent a spectrum of disorders with each specific component serving as a potential site of pathology. Instability, malalignment, intervertebral disk degeneration, disk herniation, and spinal stenosis can all present as neck pain [43]. The characteristic clinical findings associated with axial neck pain are deep-seated aching pain, painful range of motion, and pain with palpation of paraspinal musculature.

Cervical radiculopathy secondary to traumatic events results from traction, compression, laceration, or ischemia of the involved nerve [44]. Patients with cervical radiculopathy can exhibit loss of motor function within specific myotomes in addition to inconsistent sensory deficits at the affected level. Clinically, this can manifest as unilateral or bilateral numbness, paresthesia, pain, and loss of motion function within specific nerve distributions. Classically, patients report improvement in radicular symptoms with shoulder abduction or what is known as the hand on head position or Bakody's sign. Patients can report worsening pain with distinct movement or positioning of the cervical spine. It is important to note that when differentiating radiculopathy from musculoskeletal neck pain, radicular symptoms should outweigh axial symptoms.

Rotator Cuff Tears vs Glenoid Labrum Injuries

Rotator cuff injuries present chronically as progressive pain and weakness with overhead activity; however, acutely, these injuries are more likely to be associated with dislocations or subluxations. Paresthesia and numbness are typically not present, although patients can present with radiating neck pain usually exacerbated with active shoulder motion. Weakness is apparent with initiation of shoulder abduction and further elucidated with strength testing using the Jobe's test and resisted internal and external rotation of the shoulder. Further C5 weakness, specifically biceps and deltoid function, is absent in isolated rotator cuff tears.

Glenoid labrum injuries are often associated with shoulder instability. Patients may have a history of shoulder dislocation. The typical pain can be reproduced with abduction and external rotation (anterior instability) or shoulder adduction and axial loading (posterior instability). SLAP tears are associated with pain with overhead activity and are characterized by a deeper pain when compared to rotator cuff pathology.

C5 Radiculopathy vs Suprascapular Nerve Entrapment vs Traumatic Upper Brachial Plexus Injury

C5 radiculopathy can result from traumatic disk herniations, facet dislocations, fractures, or chronic degenerative changes [38]. Acute and rapid rotation of the cervical spine and chronic axial loads serve as the most common mechanisms of injury

associated with C5 radiculopathies among athletes. Weakness and atrophy can be clinically evident when assessing the biceps, supraspinatus/infraspinatus, or deltoid. Shoulder range of motion has limited association with cervical pathology; however, as previously mentioned, shoulder abduction (hand on head position) typically relieves symptoms.

Suprascapular nerve entrapment typically involves isolated weakness and atrophy of the supraspinatus and/or infraspinatus depending on the site of compression, i.e., suprascapular notch. Other muscles innervated by C5 are spared, and radicular pain does not follow classic C5 dermatomal patterns. EMG and nerve conduction velocity tests can be helpful in assessing sites of distal compression.

Brachial plexus injuries most commonly present as burners/stingers and signify a relatively benign insult on the spectrum of traumatic brachial plexus injuries. Athletes with severe brachial plexus injuries present after forceful lateral deviation away from the affected upper extremity or after a traumatic fall onto the shoulder, neck, or head. Typically, upper brachial plexus injuries have preserved function of long thoracic nerve (i.e., lack of scapular winging) and variable involvement of the suprascapular nerve depending on the level of insult.

C6 and C7 Radiculopathy vs Median Nerve Entrapment (Pronator Syndrome/Carpal Tunnel Syndrome)

C6/C7 radiculopathy can result from rotational cervical spine injuries or axial loads (i.e., spear tackling technique) leading to weakness in muscles innervated by C6 and C7. Paresthesias and numbness follow dermatomal distributions of C6 (thenar eminence and thumb) and C7 (middle finger). Assessing thenar strength can be a differentiating exam maneuver since motor innervation is primarily provided by C8; thus, it should be normal with an isolated C6/C7 insult.

C8 Radiculopathy vs Ulnar Nerve Compression

C8 radiculopathies result from flexion and compression forces across the cervicothoracic junction. At the C7 to T1 junction, the mobile and lordotic cervical spine rapidly transitions to the rigid and kyphotic thoracic spine. Initial radiographic triage of injuries at this level has been largely replaced by CT imaging in addition to MRI. Patients present with weakness in finger flexion in addition to paresthesias in the fifth digit and along the medial border of the forearm. One clinical pearl is to assess opposition and abduction of the thumb (medial nerve) which distinguishes C8 radiculopathy from an ulnar nerve compression (i.e., cubital tunnel syndrome) [45]. Ulnar nerve compression can be elicited by compressing the medial side of the elbow (Tinel's sign) with resulting paresthesias. Paresthesias from compression at

the elbow are evident in the hand but not in the forearm due to innervation of the medial antebrachial nerve, which serves as another distinguishing clinical exam finding.

Expert Opinion

The presenting symptoms of cervical spine and upper extremity injuries often overlap, which requires providers to distinguish between the two in order to accurately diagnose. The essential components in achieving an efficient and precise diagnosis are utilization of a broad differential, a focused history and physical exam, advanced imaging, and diagnostic studies when necessary. Differentiating between cervical spine and upper extremity pathologies involves a strong understanding of the neurologic exam, peripheral exam findings, and myotomal/dermatomal findings correlative to cervical spine levels. The most common dilemma encountered by providers assessing athletes with neck and arm pain is differentiating radicular symptoms from cervical pathology and musculoskeletal injuries. The importance of distinguishing the source of the patient's chief complaint cannot be underscored enough since treatment plans vary widely depending on the etiology.

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Chapter 8
Transient Brachial Plexopathy (Stingers/Burners)

James B. Carr II and Joshua S. Dines

Introduction

Transient brachial plexopathies, also referred to as "stingers" and "burners," are common athletic injuries, especially in collision athletes. Stingers present as a temporary episode of unilateral upper extremity pain and/or paresthesias with or without weakness. There are multiple proposed etiologies of stingers with trauma to the upper brachial plexus or cervical nerve roots being the most important causative factor. Certain anatomical features of the nerve roots and upper brachial plexus leave this region particularly vulnerable to direct or indirect trauma. Knowledge of upper brachial plexus anatomy, along with associated innervated muscles, will help guide the examiner in making the appropriate diagnosis.

Athletes with a stinger typically present with weakness or inability to move the involved upper extremity after a forceful collision. Along with upper extremity weakness, the athlete often experiences intense ipsilateral paresthesias or "burning pain," consistent with the name of "stinger" or "burner." It is paramount that signs and symptoms of more serious, potentially permanent, cervical spine injuries are quickly identified and managed appropriately.

Management of an isolated stinger is often supportive with minimal intervention required. Return to play criteria is dependent on clinical examination and previous history of stingers or other neurological injuries. Clearing an athlete for competition after numerous stringers can be controversial, and this decision is influenced by multiple factors, including severity of symptoms, number of previous stingers, and degree of cervical spine stenosis.

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Epidemiology

Stingers are a common but often underreported injury in contact athletes. Many athletes choose not to report their symptoms to the medical staff due to the very transient nature of symptoms and fear of missing time from competition. Therefore, ascertaining the exact incidence of stingers can be difficult.

In general, stingers occur most frequently in American football players but have also been reported in wrestling, hockey, boxing, rugby, basketball, and weight lifting [1–6]. A stinger is the most common symptomatic upper extremity nerve injury reported among college football players with up to 65% of collegiate football players reporting at least one stinger during their college careers [6]. Recurrence rates can also be high in collegiate football players with up to 87% of athletes with a prior history reporting a recurrent stinger during their careers [7]. Stingers are also the most common peripheral nerve injury in high school collision athletes [4].

An updated epidemiology report using the National Collegiate Athletic Association Injury Surveillance Program (NCAA-ISP) reported a relatively lower stinger injury rate in American football players [3]. The 6-year surveillance study from 2009 to 2014 reported 229 stingers, resulting in an injury rate of 2.04/10,000 athlete exposures. Most stingers (55.5%) were reported during competitions and resulted in less than 24 hours of playing time loss (63.8% of cases). Nearly one in five stingers (18.8%) were recurrent injuries. The overwhelming majority of stingers were due to player contact (93%) with tackling (36.7%) and blocking (25.8%) being the most common mechanisms. Defensive ends and linebackers were most commonly affected (25.8%) followed closely by offensive linemen (23.6%). In general, stingers occur most frequently in defensive players, likely due to an aberrant tackle or unplanned collisions. In another similar study, among football players sustaining at least one burner, 30% were defensive backs, 18% were defensive lineman, 17% were offensive linemen, 11% were offensive backs, 5% were wide receivers, and 2% were punters [8].

Rugby is also a common cause of stingers both in North America and internationally. An epidemiology study of 569 elite high school and college rugby players during a full season of competition reported that 33.9% of players reported a history of stinger prior to the season, and 20.9% of players had at least one stinger during the season (34.2 events/1000 player-hours of match exposure) [2]. The reinjury rate for stingers was 37.3%. The mean severity of injury was 2.9 days with 79.3% of players not losing any time from competition. A prolonged recovery of more than 14 days was reported in 5.8% of cases. A history of previous stingers and a stinger with more than three symptoms, especially motor weakness, correlated with the severity of injury.

In general, medical personnel caring for contact athletes should be aware that the incidence of stingers may be higher than reported rates. Athletes may not fully understand the importance of reporting transient symptoms. They also may be afraid of reporting their symptoms due to fear of being held out of competition. It is

important for medical personnel to educate coaches and athletes on the importance of recognizing and reporting stingers so that affected athletes can receive appropriate care.

Mechanism of Injury

Chrisman et al. first described a mechanism for stingers in 1965 [9]. They reported a "lateral-flexion neck injury" that resulted from "a blow on the side of the head, with immediate pain from the base of the neck to the hand, prickly paresthesia of the whole arm, and inability to move the extremity." They hypothesized that cervical nerve roots were stretched along with the surrounding soft tissues. Two years later, Bateman described a similar mechanism of falling onto the shoulder with resultant movement of the head and neck in the opposite direction, resulting in a stretch injury [10]. He also was the first to suggest that direct trauma across the neck and shoulder region could lead to similar symptoms. The injury pattern was further defined by Robertson et al. in 1979 when they noted a high rate of brachial plexus injuries in defensive football players [5]. They used EMG to localize the injury to the upper brachial plexus. Clancy et al. further classified stingers as grade 1 or grade 2 brachial plexus nerve injuries based on Seddon's nerve injury grading scale [11]. Grade 1 injuries were neuropraxia injuries that resolved within 3 weeks, and grade 2 injuries were axonotmesis injuries that took up to 10.5 months to resolve. This injury pattern was frequently referred to as "cervical pinch syndrome," which was further simplified to "stinger" or "burner" by athletes and athletic trainers.

Since Chrisman's original description, several mechanisms of injury have been elucidated. Broadly, these include both indirect and direct mechanisms with trauma to the upper brachial plexus or cervical nerve roots at or near Erb's point as the common denominator. Proposed mechanisms for a stinger or burner include (1) brachial plexus stretch or traction, (2) compression of cervical nerve roots in the neural foramen, and (3) a direct blow to the brachial plexus.

A traction injury to the brachial plexus usually occurs from a direct blow to the head that results in simultaneous contralateral lateral neck flexion and ipsilateral shoulder depression. This is arguably the most classic mechanism for a stinger, and it frequently occurs in football or rugby players during a block or tackle. This mechanism is also thought to be more common in younger athletes who lack experience, have poorer tackling form, and have weaker neck and shoulder musculature in the absence of cervical spine stenosis [12]. Of note, this is the same mechanism of injury also believed to occur in newborns that develop an Erb's brachial palsy at birth as the relatively larger shoulder girdle is forcibly depressed through a narrow pelvic outlet during delivery.

Compression of cervical nerve roots can also lead to a stinger. This mechanism occurs during forced lateral neck flexion with extension with or without an axial load, which causes acute narrowing of the contralateral neural foramen. This is a mechanism similar to Spurling's exam maneuver for cervical radiculopathy. Nerve

root compression is thought to be a more common etiology of stingers in collegiate and professional football players who have a higher prevalence of cervical spine stenosis [6, 13, 14].

Finally, a forceful, direct blow to Erb's point, often from an opponent's helmet, can result in a stinger. The superficial location in the supraclavicular region makes Erb's point particularly vulnerable to direct trauma. The use of appropriate shoulder pads and protective equipment may help reduce, yet likely does not eradicate, stingers from this mechanism.

Applied Anatomy

Anatomically, stingers are thought to involve the upper trunk (C5 and C6 nerve roots) of the brachial plexus. Therefore, the muscles most commonly affected from a stinger include the deltoid, supraspinatus, infraspinatus, teres minor, teres major, biceps, brachialis, brachioradialis, and supinator. Involvement of the C5 and C6 nerve roots was further confirmed in a classic report by Rockett where surgical exploration after a stinger demonstrated scarring of these nerve roots to the medial and anterior scalene muscles [15].

The intricate anatomy of the cervical spine and brachial plexus helps explain the various mechanisms of injury for stingers. In general, cervical nerve roots are at higher risk for both a tensile and a compressive injury compared to the brachial plexus for several reasons [16, 17]. The roots originate off the spinal cord with a linear orientation. Conversely, the brachial plexus has a plexiform orientation, which makes it more flexible and tolerant of tensile forces compared to the linearly oriented nerve roots. The plexus is also surrounded by more compliant soft tissues, which increases force absorption and flexibility. Conversely, cervical nerve roots exit through rigid neuroforamen, which may be narrowed secondary to degenerative changes, such as osteophytes, disc space narrowing, or facet joint hypertrophy. Furthermore, the nerve roots are anchored by the dural dentate ligaments, which create a countertraction force when the brachial plexus is placed under tension. The nerve roots also relatively lack perineurium and epineurium, which reduces their elasticity.

Specifically, the C5 nerve root is believed to be the most vulnerable nerve root for multiple reasons. It exits through the C4/5 neuroforamen, which becomes particularly narrowed with neck lateral bending, rotation, and extension. It is also the shortest nerve root, and it is in direct alignment with the brachial plexus. These factors make the C5 nerve root particularly vulnerable to traction and compressive forces, which likely explains why muscles innervated by the C5 nerve root are most commonly affected with a stinger. Additionally, the ventral motor root lacks the dampening effect of the dorsal root ganglion, which may explain why motor symptoms tend to predominate over sensory symptoms [18].

Despite these various anatomical features that place the cervical nerve roots at particular risk, the literature still generally favors brachial plexus stretch as

the most common mechanism of injury. This is likely because most stingers occur in younger patients who have not developed cervical stenosis and typically experience indirect trauma that results in a brachial plexus stretch mechanism of injury.

Cervical spinal stenosis is also a very important anatomical consideration. The relationship between cervical spinal stenosis and stingers was first reported in 1986 by Torg et al. [19] and has been consistently shown in multiple studies since this time [6, 7, 20]. The Torg ratio, also known as Pavlov's ratio [21], was a commonly used radiographic measurement to calculate cervical stenosis before the regular availability of advanced imaging (Fig. 8.1). A Torg ratio of less than 0.8 indicates a stenotic cervical canal, and this has been associated with as much as a threefold increase in sustaining a stinger in collegiate football players after cervical spine extension-compression injuries [6]. While the Torg ratio can be a quick tool to calculate cervical stenosis on initial radiographic examination, many physicians consider this method outdated and less reliable compared to modern advanced imaging, such as magnetic resonance imaging (MRI) or computed tomography (CT) myelogram scans. Since its initial description, the Torg ratio has been found to have poor predictive value and misleading results in the setting of hypertrophied vertebral bodies in high-level athletes [22, 23]. Regardless of the method for calculation,

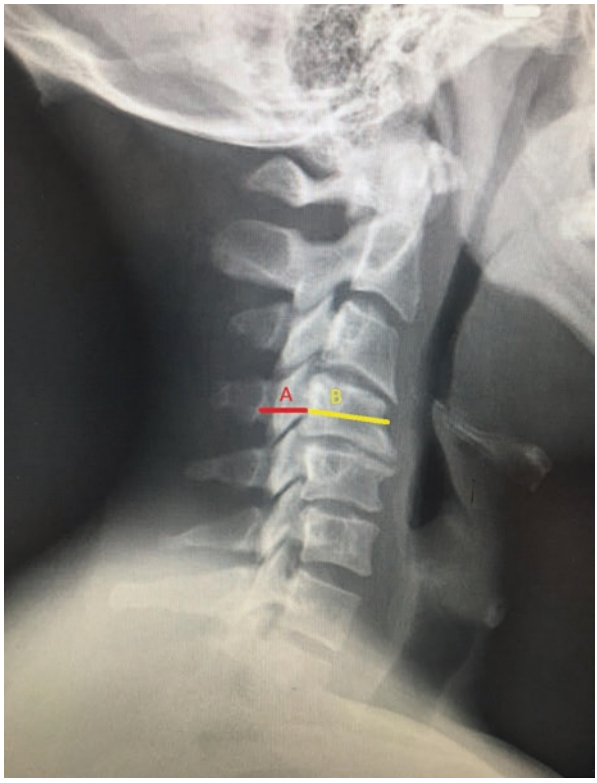

Fig. 8.1 Pavlov-Torg ratio: A/B <0.8 indicative of congenital stenosis

cervical spinal stenosis must be considered and identified in athletes with multiple stingers, which will be discussed in more detail in the management and return to play sections of the current chapter.

Presentation and Evaluation

The diagnosis of a stinger is a clinical diagnosis that is made by history and physical examination. Most athletes will experience one of the three aforementioned mechanisms of injury and then present to the medical staff immediately after the offending play. If the player is able to leave the field of play under his or her own strength, then a sideline evaluation is appropriate. If the player is unable to leave the field of play, then evaluation should proceed on the field. Regardless of where the initial evaluation is performed, the most important and critical aspect of the evaluation should be recognizing any red flag symptoms that suggest a more serious spinal cord injury. In general, it is exceedingly rare for a stinger to affect more than one extremity. When a player presents with pain, weakness, or paresthesias in multiple extremities, then the evaluating medical staff should suspect a spinal cord injury until proven otherwise. Additional red flag symptoms include cervical spine pain, tenderness to palpation along the cervical spine, pain with any attempted movement of the cervical spine, abnormal reflexes, deformity, localized swelling, difficulty breathing, or changes in vision or mentation. These symptoms should prompt an emergent neurological evaluation along with full spine precautions. The player should be placed on a spine board with removal of any face mask to allow for airway management if needed. The player should be immediately transferred to the emergency room via ambulance for imaging of the entire spine and further care whenever a more serious spinal cord injury is suspected. However, in the setting of isolated upper extremity symptoms without any red flag symptoms, the initial diagnosis of a stinger may be presumed.

Athletes with a stinger will often grab the affected extremity or hold it in an elevated position with the opposite arm in order to reduce tension on the cervical nerve roots. The athlete will commonly report a burning pain or sensation that starts around Erb's point and shoots down the entire arm. Paresthesias and sensory loss may or may not be present and may present in either a circumferential or dermatomal pattern. Isolated upper extremity weakness is often present in various degrees, yet weakness can also present in a delayed fashion or not at all, depending on the severity of injury. The athlete will generally present with full and painless cervical spine range of motion without tenderness to palpation.

Once the diagnosis of a stinger is made, a neurological examination should be performed on the sideline. The examination should include palpation of the cervical spine followed by strength testing of all muscle groups, sensory evaluation of all dermatomes, and assessment of deep tendon reflexes. The unaffected extremity can be used as a point of reference to help detect any subtle weakness. The examiner should pay special attention to weakness with arm abduction, shoulder external

rotation, and elbow flexion as these actions are largely controlled by the C5 and C6 nerve roots. A shoulder examination should also be performed to assess for shoulder instability or any other abnormality to the clavicle, acromioclavicular joint, or sternoclavicular joint. Erb's point can also be percussed, which may elicit radiating pain.

If the patient denies any neck pain and has no tenderness to palpation of the cervical spine, then neck range of motion may be assessed in flexion, extension, lateral bending, and rotation. Range of motion should only be tested when a more serious spine injury has been sufficiently ruled out as outlined above. Lastly, a Spurling's maneuver may be performed. This test has been found to be positive in up to 70% of patients with a stinger [7].

Serial examinations are very important in the assessment of a player with a stinger because the symptoms tend to be transient and evolve over time. Symptoms from a stinger may last for only a few minutes or up to days, weeks, or even months. The majority of stingers will resolve within minutes or hours. Though less common, weakness may present in a delayed fashion after normal strength on the initial examination, which further heightens the importance of serial assessments. Additionally, if serial examinations reveal worsening neurological signs or symptoms, then the evaluating staff should re-evaluate for the presence of any red flag symptoms and act accordingly.

Management and Work-Up

Initial management requires removal from competition, rest, and pain control. A sling may be used for comfort in the setting of persistent upper extremity weakness. Treatment of a stinger is largely symptomatic, and most stingers will resolve quickly without intervention. First-time stingers that resolve rapidly do not require any further management or treatment. However, when an athlete has persistent symptoms that preclude return to play, a comprehensive rehabilitation program should be initiated. The program should focus on cervical spine and upper extremity range of motion, appropriate posture, proprioception, and lastly muscle strengthening. Neuromuscular coordination is also a very important component of the rehabilitative process following a stinger.

The role of cervical spine imaging and electrodiagnostic studies after a stinger is controversial. Routine cervical spine radiographs following every stinger are typically not recommended. Players who sustain a first-time stinger with rapid resolution of symptoms do not require cervical spine imaging or diagnostic tests. Commonly suggested indications for imaging work-up include persistent symptoms beyond 1 h, concomitant neck pain, symptoms localized to only one nerve root, or recurrent stingers [16, 24, 25]. The initial test of choice is cervical spine radiographs because they are obtained quickly and provide valuable diagnostic information. Specifically, radiographs can identify fracture, foraminal stenosis, or instability on flexion and extension views. The next imaging study obtained should be a MRI,

especially in the setting of persistent weakness [24]. A cervical spine MRI can further define any neuroforaminal stenosis, nerve root injury, spinal cord edema, disc herniation, or disc-osteophyte complexes that may be contributing to neuroforaminal stenosis. MRI is especially important in the evaluation of chronic or recurrent stingers since these are both frequently associated with foraminal narrowing and cervical disc disease.

Electrodiagnostic testing can be considered in the athlete with persistent symptoms, though the information obtained typically does not guide treatment options. In general, electrodiagnostic testing is more helpful in evaluating muscle weakness than sensory symptoms. It can also help localize the site of injury by differentiating a cervical nerve root injury from a brachial plexus injury. Additionally, electrodiagnostic testing can help differentiate a neuropraxic injury from an axonal injury, which can help guide prognosis and time frame for recovery.

In general, electrodiagnostic testing should rarely be considered within 3 weeks of the injury because signs of denervation are not present before this time point. Furthermore, findings on electrodiagnostic testing can remain abnormal even after the athlete has sustained a full clinical recovery of strength [26]. Therefore, these tests should only be performed in patients with persistent weakness on exam at least 3 weeks after the injury. Any radicular pattern of injury seen on electrodiagnostic tests should also be further evaluated with an MRI if one has not been previously obtained.

Return to Play

Return to play criteria following a stinger can be controversial. The decision to withhold an athlete from competition is largely dependent on the examination, on history of previous stingers, and sometimes on results of advanced imaging [16, 17, 25, 27, 28]. No athlete should be allowed to return to play until he or she has complete resolution of neurological deficits, including return of full strength and sensation. Any degree of persistent neurological deficit is an absolute contraindication to return to play. Additionally, the athlete should have full, pain-free cervical spine range of motion, no neck tenderness to palpation, and no suspicion for underlying cervical injury. If symptoms resolve rapidly and the athlete meets the above criteria, he or she may be considered for return to play in the same game in the setting of a first-time stinger.

A first-time stinger with persistent symptoms is a contraindication to return to play in the same game. When symptoms from a stinger persist beyond approximately 1 h, then cervical spine imaging should be obtained. Any evidence of neck pain also precludes return to play in the same game and requires the patient to undergo further imaging work-up. Absolute contraindications for return to play include persistent weakness, cervical anomalies or pathology on advanced imaging, continued pain, evidence of cervical myelopathy, and reduced cervical range of motion.

While return to play after a first-time, rapidly resolving stinger is generally accepted as safe, return to play in the setting of a recurrent stinger can be quite con-

troversial and is largely dependent on the timing and severity of the recurrence. A second stinger that occurs in a separate season with rapid resolution and a normal examination is an indication for return to play in the same game. A recurrent stinger in the same game or season, even with rapid resolution of symptoms, precludes return to play in the same game. The decision to return to play for the following game depends on the persistence of symptoms. After a second, rapidly resolving stinger in the same game or season, the athlete may return the following game with or without cervical spine imaging. However, if symptoms are persistent after a second stinger, then the player should be withheld from physical activity, and cervical spine imaging should be obtained.

A third stinger, regardless of timing, is a contraindication to return to play. These athletes must undergo cervical spine imaging if it was not obtained after the second stinger [29]. Management of an athlete with a third stinger is also controversial but typically involves removing the athlete from competition for the remainder of the season. These athletes have a high prevalence of cervical spine stenosis and other anomalies that place them at a higher risk for future spinal injuries [6, 7, 13, 14]. The treating medical staff should have extensive discussions with the athlete and consider restricting the athlete from future participation in contact sports, especially when advanced imaging reveals cervical spine anomalies.

Advanced imaging and electrodiagnostic testing usually play a supportive role when making return to play decisions. These tools can be helpful when evaluating an athlete's risk for future stingers, but they should not be used in isolation when determining return to play status for an athlete. Even if cervical spine imaging is within normal limits, the athlete should not return to competition if any neurological symptoms persist. Furthermore, cervical spinal stenosis has been associated with a higher risk of experiencing a stinger, but it is also quite prevalent in elite football and rugby players who have not obtained stingers [23].

Similarly, abnormal electrodiagnostic findings should not preclude progression of rehabilitation or return to play in the setting of a normal neurological examination. In fact, abnormal findings on electrodiagnostic tests may persist even after full clinical neurological recovery [18]. Therefore, advanced imaging is most helpful within the context of an athlete's history and current physical examination findings.

A summary of return to play recommendations can be found in Table 8.1.

Treatment and Prevention

Because stingers are generally self-limited, treatment beyond supportive care and physical therapy is rarely needed. Some physicians may prescribe B vitamins after a persistent stinger because of the role of B vitamins in other peripheral neuropathies. However, this is generally not recommended because there is minimal evidence to support its use in treating various peripheral neuropathies, much less in stingers [24]. Prescribers should not exceed the recommended upper limit of B

Table 8.1 Return to play guidelines following a stinger

No contraindication[a]
First-time stinger that is transient (less than 1 h) with full resolution of symptoms
Second-time stinger not in the same game or season that is transient (less than 1 h) with full resolution of symptoms
Relative contraindication[b]
Persistence of symptoms beyond 1 h
Second stinger in the same game or season
Absolute contraindication
Unresolved neurological deficits on clinical exam
Symptoms in bilateral or multiple extremities[b]
Neck pain and/or lack of full cervical range of motion[b]
Third-time stinger regardless of timing

[a]Athlete may return to play the same day without imaging work-up
[b]Athlete should not return to play the same day. Imaging work-up should be considered, and return to play is possible in the same season pending resolution of symptoms and results of imaging work-up

vitamins as this can be fraught with iatrogenic complications, including worsening peripheral neuropathy and depletion of other B vitamins.

When conservative measures are exhausted and the patient continues to have persistent symptoms with significant spinal stenosis or disc herniation, then other options may be considered. For example, treatment may include fluoroscopically guided epidural steroid injections for pain relief. Surgical decompression of a narrowed foramen or spinal fusion in the setting of continued or progressive weakness is exceedingly rare.

A variety of preventative measures have been proposed with little data to support their efficacy. A cervical collar is a piece of protective equipment used to decrease the risk of recurrent stingers. They are designed to decrease neck extension and lateral flexion, yet it is unknown if they actually reduce the rate of stingers [30–32]. Furthermore, the cervical collar has multiple disadvantages. Reducing cervical spine range of motion may reduce a player's performance on the field and ability to maintain protective vision. More importantly, by reducing neck extension, it may have the undesired effect of placing the player's neck into a more flexed position, which could place the athlete at increased risk for catastrophic cervical spine injury. The cowboy collar is another piece of protective equipment that provides additional padding over Erb's point. This may help prevent direct trauma to the brachial plexus, but data to support its use is also lacking. Lastly, straps that connect the helmet to the shoulder pads are outdated, hazardous, and strongly recommended against since they do not allow appropriate compensatory neck range of motion during contact.

One of the most important prevention measures is teaching appropriate tackling form, which should focus on keeping the defensive player's head up while minimizing dropping the shoulder and arm tackling [1]. This helps the player avoid a vulnerable position that could allow forceful cervical spine extension. Every effort should be made to teach young athletes the proper tackling form early in their playing careers.

Conclusions

Stingers often present with a classic presentation and are a relatively common injury in collision athletes. They are usually transient and self-limiting with minimal time missed from competition. Conservative management almost invariably results in complete resolution of symptoms. Physical therapy and other modalities can be used when symptoms are persistent. Imaging is indicated in cases of persistent symptoms or recurrence. Return to play is guided by complete resolution of symptoms, restoration of a normal physical exam, and benign imaging in the setting of recurrent stingers. When a player has a third stinger, the treating medical staff should remove the athlete from competition for the season and consider permanent removal from contact sports.

Expert Opinion

When an athlete presents with a transient stinger, as defined by symptoms lasting less than 1 h, return to play is safe after resolution of all symptoms and a normal neurological examination. We do not allow players to return to the game after a recurrent stinger in the same game or the same season or if the player has persistent symptoms beyond 1 h. We prefer to obtain cervical spine imaging after a second stinger in the same game or season; however, we typically do not obtain imaging after a second stinger that occurs in a different season, as long as symptoms are transient.

We recommend obtaining both cervical spine radiographs and a cervical spine MRI when performing imaging work-up of a player with a stinger. We have found MRI to be invaluable in further assessing neuroforaminal stenosis and additional cervical spine pathology, such as disc herniation. After a third stinger at any time point, we initiate discussions with the athlete about possibly abstaining from contact sports, especially in the setting of cervical spine stenosis. Many extraneous factors can potentially influence this conversation, including financial and career motives, yet we firmly believe that the physician should always place the patient's safety ahead of these factors when having such conversations.

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Chapter 9
Cervical Cord Neurapraxia

Frank H. Valone III and Kiehyun Daniel Riew

Background

Cervical cord neurapraxia (CCN) is a transient neurological deficit following cervical cord trauma. CCN occurs at a rate of 1.3–6 per 10,000 athletes, with the highest rates of CCN having been found in football players, where it is estimated to be as high as 7.3 per 10,000 participants [1]. The mechanism of injury in CCN involves hyperflexion, hyperextension, or an axial load to the cervical spine causing a temporary derangement in the axonal permeability of the spinal cord [2, 3]. The pincer mechanism was described by Penning, wherein extension causes the spinal cord to become pinched between the posterior inferior aspect of the superior vertebral body and the anterior superior aspect of the inferior lamina. Contrariwise, in flexion the cervical spine becomes compressed between the lamina of the superior vertebrae and the posterior superior aspect of the inferior vertebral body [3, 4].

Torg tested these theories in the laboratory by utilizing the giant squid axon and measured membrane permeability and cytosolic free calcium concentrations. The study demonstrated that rapid stretch resulted in calcium influx, resulting in hyperpolarization, followed by a prolonged period of depolarization. During this depolarization period, the axon was no longer excitable. The rise in the calcium concentration was directly proportional to the rate and amount of tension applied to the axon, whereas neurologic recovery was inversely proportional to the rise in the calcium concentration [3].

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Symptoms

Torg developed the primary classification systems for CCN as defined by the duration of symptoms, i.e., Grade 1 (<15 min), Grade 2 (15 min–24 h), and Grade 3 (>24 h), and the type of neurological deficit: Type (1) "plegia" with complete paralysis, Type (2) "paresis" with motor weakness, and Type (3) "paresthesia," only sensory changes without motor involvement. Lastly, CCN can be defined by anatomic location of the symptoms: "quad" involving all four extremities, "upper" involving both arms, "lower" involving both legs, and "hemi" involving an ipsilateral arm and leg [1, 5].

Neck pain and loss of cervical range of motion are not frequently experienced in adults at the time of the injury [6]. The largest adult case series was presented by Torg in 1986 of 110 patients, wherein the majority of episodes were Grade 1 (74%), resolving within 15 min, Grade 2 (15%), and Grade 3 (11%). Additionally, the incidence of plegia was 40%, paresis 25%, and paresthesia 35%. Additionally, the anatomic location was "quad" in the majority of cases (80%), "upper" extremity in 15%, "lower" in 2%, and "hemi" in only 3% [7]. Symptom location and duration change when evaluating CCN in children. The anatomic location is most commonly upper extremity paresis (38%), quadriparesis (31%), hemiparesis (23%), and lower extremity paresis (8%). Additionally, symptoms are present for significantly longer; the mean was 26 h, lasting as long as 5 days in one case; and 77% of pediatric patients experienced neck pain and decreased cervical range of motion at the time of their injury [8].

Risk Factors for CCN

Risk factors for CCN were evaluated in a multi-cohort study by Torg. The study had five cohorts: Cohort 1 ($N = 227$), college football players who did not have a history of CCN; Cohort 2 ($N = 97$), professional football players who did not have a history of CCN; Cohort 3 ($N = 45$), high school, college, and professional football players with at least one episode of CCN; Cohort 4 ($N = 75$), individuals with permanent quadriplegia following a football injury; and Cohort 5 ($N = 105$), a control group of nonathletes without a history of CCN. Cohort 3 had a significantly smaller ratio of diameter of spinal canal to vertebral body (Torg ratio), as well as a significantly smaller mean diameter of the cervical spinal canal. The results of this study suggest an association between stenosis and CCN [9].

Cervical spinal stenosis can be determined by absolute numbers as well as ratios to surrounding tissues. A study of 1066 human cadaveric specimens from the Hamann-Todd collection at the Cleveland Museum of Natural History found an

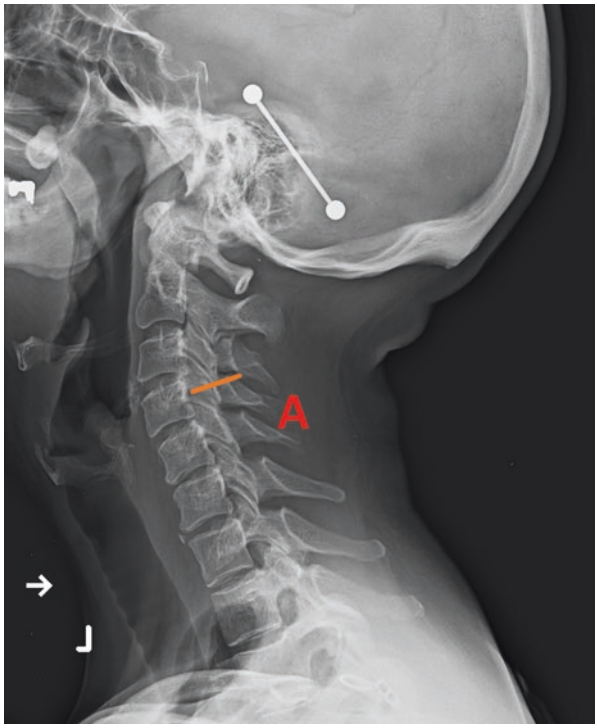

Fig. 9.1 Space available for the cord

absolute sagittal value of <13 mm was strongly associated with cervical spinal stenosis at all levels [10]. Wolf et al. established normative values for cervical spine by measuring from the posterior vertebral body to spinolaminar line (Fig. 9.1). Wolf found the average: 22 mm at C1, 20 mm at C2, and 17 mm from C3 to C7. Sagittal diameters from C3 to 7 >15 mm were normal, and stenosis was found with sagittal diameters <13 mm [11]. The Torg ratio is based on the ratio between the spinal canal diameter and the vertebral body diameter at the C3–C7 levels (Fig. 9.2). A Torg ratio of <0.8 is considered evidence of congenital stenosis. The benefit of a ratio is that it is independent of magnification factors; however, it does not take into account disproportionate differences in vertebral body size in some patient populations [2].

By utilizing these standard radiographic techniques, it can be shown that cervical spinal stenosis is common and can be found in 7.6–29 cases per 100 football players [9, 12]. Additionally, magnetic resonance imaging is able to demonstrate both bone and soft tissue encroachment on the spinal canal, allowing for a more precise evaluation of the space available for the cord. “Functional reserve” can be determined if there is cerebrospinal fluid signal surrounding the spinal cord, or “functional stenosis” if there is not (Fig. 9.3) [13, 14].

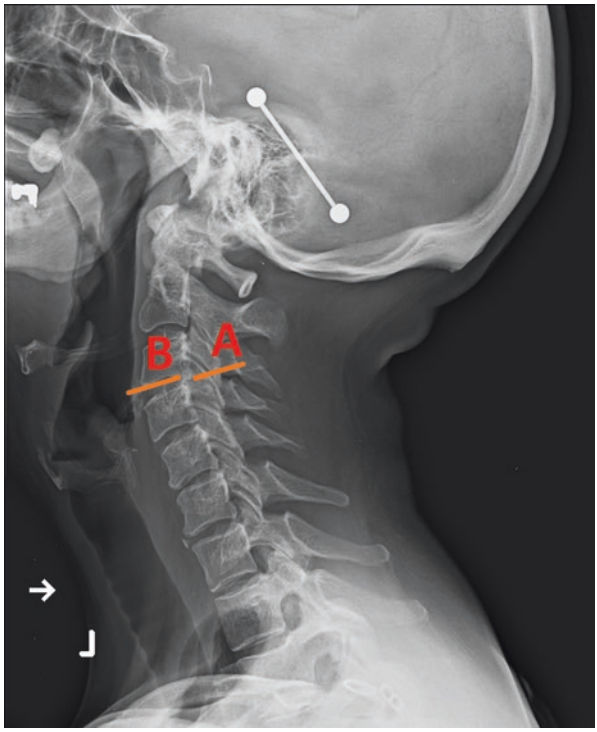

Fig. 9.2 Torg ratio – (**a**, **b**)

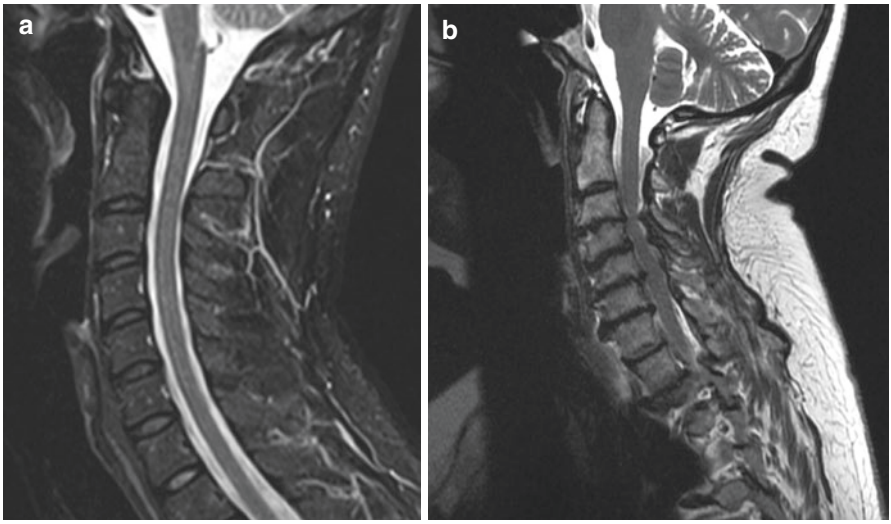

Fig. 9.3 (**a**) T2-weighted sagittal MRI demonstrating no evidence of functional stenosis. The spinal cord has adequate spinal fluid surrounding it. (**b**) T2-weighted sagittal MRI demonstrating functional stenosis. The spinal cord does not have adequate spinal fluid surrounding it

Treatment and Return to Play

Initial management for the athlete with an episode of CCN includes immobilization and clinical, standard radiographic, as well as advanced radiographic examination. None of the MRIs in Torg, nor Maroon's study, that had experienced CCN demonstrated any posttraumatic cord swelling, deformity, or syrinx; however, it is important to rule out focal lesions with cord compression or instability [1, 5]. CCN must be differentiated from the more common symptoms following brachial plexus stretch injuries or radiculopathy.

The majority of patients that experience CCN will be treated non-operatively with supportive care. However, in two series, 8.5% of 142 patients underwent surgery for cord compression or spinal instability [1, 7]. Additionally, in a series of five professional athletes who underwent anterior cervical decompression and fusion for focal cord compression after an episode of CCN, all five athletes returned to their prior level of sport [5].

Return to play criteria following CCN remains controversial [15, 16]. A review of 170 articles demonstrated current research lacks prospective randomized trials regarding return to play criteria following CCN [17]. Therefore, return to play is primarily based on expert opinion, case series, and retrospective reviews. Generally agreed-upon basic requirements for returning to athletics include normal strength, painless range of motion, and vertebral stability [16].

Additionally, with the more frequent use of MR imaging, it is suggested that the players have "functional reserve" around the cord. In MR imaging of ten athletes that experienced episodes of CCN, all subjects had stenosis between 7 and 12 mm over three levels on MRI, but only three had no functional reserve at those levels. The three athletes with no functional reserve voluntarily retired, and the other seven returned to their sport without subsequent episodes of CCN [18, 19].

A recommended relative contraindication for return to play is the presence of T2 hyperintensity on MR imaging [14, 17, 20, 21]. However, a recent study of five patients who were treated operatively concluded that if postoperatively the contact athlete is symptom-free, T2 hyperintensity may not be a contraindication to return to play [22]. However, the numbers within this study are small, and it is unknown if this study's conclusions regarding T2 hyperintensity are reproducible in the athlete that does not undergo surgical intervention. In a study of four athletes that had one episode of CCN, MR imaging demonstrated maintained functional reserve. None of these athletes had recurrent episodes of CCN once they returned to their sport [23]. Additionally, dynamic flexion and extension cervical magnetic imaging would further evaluate for stenosis and the presence of the pincer mechanism; however, this is of limited availability.

Torg's study of 110 adult athletes demonstrated 57% of the subjects returned to sports participation at their previous level of competition. Among this group there was no significant difference between the group that returned to play and the group that did not in regard to age, sex, sport, CCN clinical grade, or radiologic findings. However, of the athletes that returned to contact sports, 56% ($N = 35$) experienced

a second episode of CCN with an average of 3.1 ± 4 episodes. Subjects who experienced recurrence had smaller disc-level canal diameter and had less space available for the cord diameter compared with those with no recurrence ($p = 0.05$). Therefore, it is important to counsel athlete's with CCN that there is a 50% chance that it may occur again and specifically take these factors of stenosis into account during counseling.

Predictors for recurrence are different in the pediatric population. In the single case series of pediatric CCN and return to sport, Torg ratios were calculated and noted to be above 0.8 for all patients, and no instability was seen on flexion and extension radiographs. Furthermore, MR imaging was obtained for each patient and demonstrated no evidence of extraneural pathology or stenosis. Among the ten subjects with long-term follow-up in the study, each had returned to his or her sport without a subsequent episode of CCN. This leads to the conclusion that the relative hypermobility of the pediatric cervical spine is an inciting factor, rather than preexisting stenosis [8].

Risks of return to sport can be catastrophic. Cantu reported on a case involving a high school athlete who described an episode of CCN. Radiographs demonstrated a vertebral canal space of 12 mm, consistent with spinal stenosis, and Torg ratios of 0.48 at C4 and 0.5 at C5. The athlete returned to football and during a tackle incurred a spinal cord injury with right-sided hemisensory loss and a flaccid left side. MR imaging after this tackle demonstrated a disc herniation and displacement of the spinal cord. Surgery was performed; however, the patient remained with permanent neurologic deficits [13].

Recommended absolute contraindications for return to play are a single event with evidence of cord injury, multiple neurapraxic events, ligamentous instability, or neuropraxic symptoms lasting greater than 36 h [6]. Additional factors to consider for return to play are the specific sport, likelihood of contact, mechanism of contract, length of future career, and anatomic features specific to the athlete; as a single episode of CCN does not substantially increase the risk of permanent spinal cord injury, there remains a small but nevertheless present risk of permanent spinal cord injury [19].

Summary

Cervical cord neurapraxia is a transient neurological deficit resulting from trauma to the cervical spine. The majority of symptoms resolve in adults within 15 min and, however, may last much longer in children. The association of cervical cord neurapraxia with cervical stenosis has been shown in many series in adults but has not been well demonstrated in children.

Our recommendations include initial management consisting of immobilization and clinical and standard and advanced radiographic examination. We believe that absolute contraindications to return to play include instability or focal cord compression that cannot be resolved with surgical intervention as

well as any residual weakness in a major motor group, imbalance, loss of dexterity, or other cord-related neurologic deficits.

Return to play criteria is greatly based on expert opinion and retrospective case reviews. The literature is without any large series that would allow for recommendations based upon meaningful and accurate epidemiological results. Adults that do return to their sport have at least a 50% risk of recurrence, whereas the risk of recurrence in children has not yet been established.

Expert Opinion

The treatment of CCN remains controversial, as there is no definitive Class I data and, with the difficulties of performing a randomized controlled study, there may never be one. Therefore, only expert opinions and small case series exist to guide the treatment.

Most players who experience transient quadriplegia will not become permanently quadriplegic and most who become permanently quadriplegic have never experienced transient quadriplegia. This was consistent with Torg's finding that, of the 117 players who became permanently quadriplegic, none had a history of transient quadriplegia in the past [7]. This is used as evidence that transient quadriplegia does not predict permanent quadriplegia and therefore, in the absence of repeated or prolonged episodes, CCN should not preclude a return to play.

The problem with this reasoning is that the number of players who become quadriplegic is small. The lack of a prior history in such a small population may not be an accurate indicator of the actual risk of CCN being a risk factor for permanent quadriplegia. This may be analogous to concluding that myelopathy is not a risk factor for quadriplegia after evaluating 117 patients with quadriplegia who never had symptoms of myelopathy. It is well-recognized that myelopathy can progress on to quadriplegia and that myelopathy is indeed a risk factor for quadriplegia, even though the vast majority of quadriplegic patients in the USA never had myelopathy. This is because, in a country like the USA, it would be rare for someone to progress on to quadriplegia without getting proper care. However, in third-world countries where access to medical care is often unavailable, it is not uncommon to see patients present with quadriplegia due to spondylotic cord compression in the absence of significant trauma.

Similarly, professional athletes who either have a prolonged episode of CCN or repeated episodes are likely to be diagnosed with stenosis and be advised to quit or become so frightened that they will voluntarily quit playing. The evidence for this is in Torg's series of 110 players with CCN, 43% of whom gave up the game following their initial episode [7]. It is reasonable to suspect that the 43% who quit had the worst or the most prolonged episode(s) of CCN and may have been the ones at greatest risk for permanent quadriplegia due to their anatomy. By quitting, they may have eliminated the players most at risk for permanent quadriplegia, biasing the results of Torg's study on the 117 players [1]. Unfortunately, there is no data on the

risk of permanent quadriplegia following a single or multiple episodes of CCN. In Torg's series, only 35 had recurrent episodes, and none ended up a permanent quadriplegic. If the risk of quadriplegia is one out of 36 in such a group, a sample size of 35 could easily have missed the one quadriplegic. Most nonprofessional athletes would choose to avoid an activity that is associated with a risk of permanent quadriplegia as low as 1 in 36 or even 1 in 100. In fact, we would most likely recommend surgery to any patient with condition that poses a 1% risk of quadriplegia, since the risk of surgery causing quadriplegia is lower than that.

Twenty years ago, concussions were considered inconsequential, and team doctors routinely cleared patients for play following a brief time out. We now recognize the serious consequences of repetitive trauma to the brain, with injury to the microvasculature and neural tissue. But we still do not know the consequences of similar trauma to the spinal cord. It is not difficult to imagine that similar micro-damage may occur with repetitive trauma to the spine.

Therefore, we believe that absolute contraindications to return to play include the following: instability, focal cord compression not amenable to surgical intervention, or residual neurologic deficits. In addition, we recommend several additional relative contraindications to return to play following CCN:

1. Greater than 24 h of Grade 4 motor loss (able to resist but not normally)
2. Greater than 60 min of Grade 3 motor loss (able to move against gravity but not against resistance)
3. Greater than 30 min of Grade 2 motor loss (able to move but not against gravity)
4. Greater than 15 min of Grade 1 or 0 motor loss (minimal to no motion)
5. Any episode causing respiratory arrest
6. Second episode of CCN of any degree
7. T1 or T2 cord signal change on MRI
8. Any nonprofessional athlete after their first episode of CCN

The above is our opinion and not based on science, since there is no definitive study to guide us. Because scientific evidence is lacking, the time intervals are arbitrary and should only serve as relative guidelines. It is likely that most players who choose to ignore the above guidelines will not suffer permanent quadriplegia. But we warn our patients about the potential for permanent deficits if they return to play. This is similar to warning a patient with mild myelopathy about the risks of progression and counseling them to avoid risky activities.

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Chapter 10
Congenital Cervical Anomalies in Athletes

Jason L. Pittman, Chong Weng, Steven Theiss, and Andrew M. Cordover

Introduction

Congenital and developmental abnormalities of the cervical spine present a difficult and special situation for the healthcare provider charged with evaluating and caring for athletes. There are few guidelines in place to provide concise and evidence-based criteria for clearing an athlete to participate in sporting activities; hence expert opinion is often the only source of guidance. These guidelines are limited in athletes without congenital abnormalities and mostly nonexistent for those with abnormalities of the cervical spine. This chapter discusses common developmental and congenital abnormalities of the cervical spine and focuses on findings which are important for the athletic healthcare provider. Additionally, criteria for the evaluation and counseling of special needs athletes will be discussed.

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Congenital Failure of Fusion in the Upper Cervical Spine (C1 Ring Anomalies, Os Odontoideum)

The odontoid process and the atlas originate from the first cervical sclerotome, whereas the body, lateral masses, and posterior arch of the axis arise entirely from the second cervical sclerotome. Normally, the atlas has three primary ossification centers, which develop during the 7th week of gestation [1]. Two lateral ossification centers extend posteromedially to form the posterior arch at 3–5 years of age. Ossification of the anterior arch involves one or two ossification centers, which extend posterolaterally to fuse with the lateral masses between 5 and 9 years of age. The odontoid process separates from the atlas between the 6th and 7th week of intrauterine life and moves caudally to join the body of the axis [2].

C1 Ring (Atlas) Anomalies

Malformations of the atlas include both clefts and dysplasias [3–5]. Senoglu et al. report that the incidence of clefts and dysplasias of the anterior arch is as low as 0.09% [6]. Clefts and dysplasias of the posterior arch are also rare but are well-described by Currarino [7]. Currarino et al. classified the posterior arch anomalies into five types, with the extent of the absence of the posterior arch and the presence of the posterior tubercle as the basis of the distinction (Fig. 10.1). Ninety-seven percent of defects are Type A, a small failure of fusion of the posterior ring. Type E defects, a complete absence of the entire posterior arch and posterior tubercle, are estimated to be present in 0.18% of the general population [7, 8]. After reviewing 160 normal CT scans of the craniocervical junction in adolescents (age ≤4 years old), Menezes et al. determined that the ring of the atlas should be completely fused by 3 years of age. In order to completely evaluate a defect of the C1 ring, a combination of CT scan and MRI is ideal. The authors also suggest that a bony arthrodesis is recommended for patients older than 4 years of age where atlantoaxial instability is still present due to failure of fusion [9]. Weng et al. reported a case in which a 13-year-old female with clefts of the anterior arch and dysplasia of the posterior arch of the atlas combined with an os odontoideum presented with quadriplegia after suffering a minor trauma [10].

Following an injury to the cervical spine, radiographic evidence of C1–C2 hypermobility with an anterior dens interval of 4 mm or greater and the presence of cervical myelopathy in the patient's clinical history or physical examination are absolute contraindications for return to play (RTP) [11]. Considering the high potential for neurological injury and the potential jeopardy to the upper cervical spinal cord, it is highly suggested that unstable anomalies of the C1 ring be an absolute contraindication for return to play. Isolated Type A defects are inherently stable; therefore athletes with this particular defect would be allowed to participate in sporting activities without restriction. Stability should still be documented in these patients with dynamic imaging. Additionally, any neurologic symptom or deficit would preclude athletic participation.

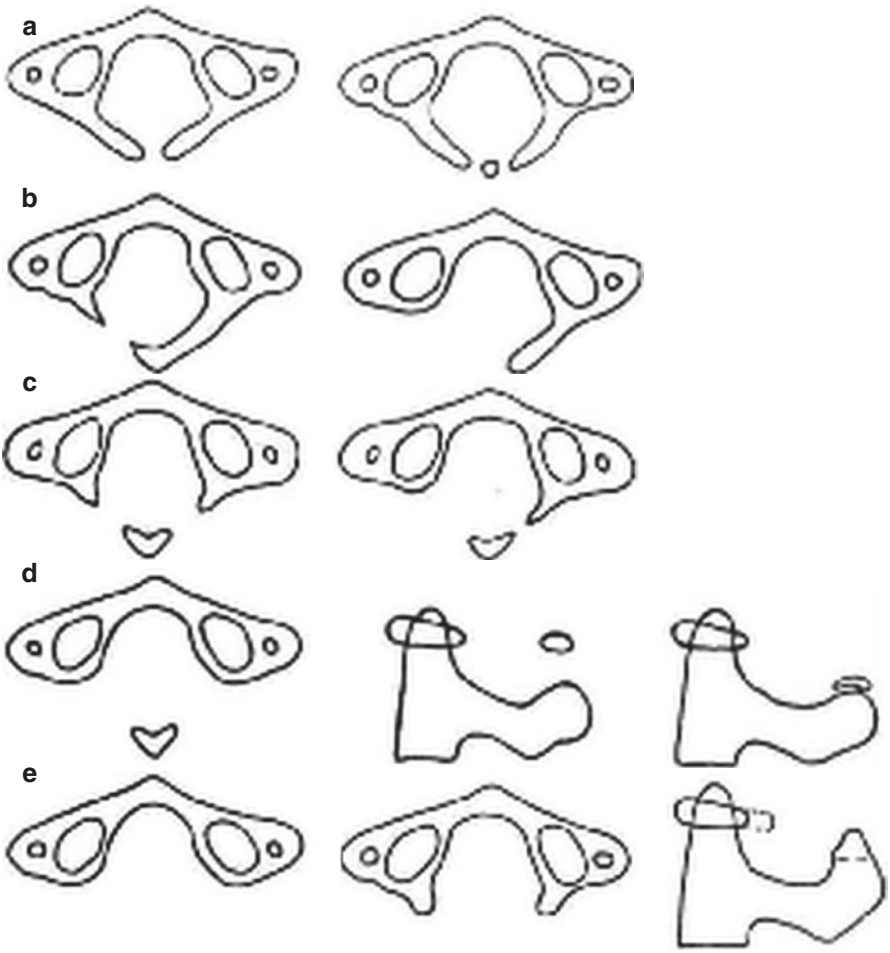

Fig. 10.1 Classification system for posterior C1 ring anomalies. (**a**) Failure of posterior midline fusion of the two hemiarches. (**b**) Unilateral clefts. A defect is present in one of the two arms of the posterior arch ranging from a small gap to a complete absence of the half arch including the posterior tubercle. (**c**) Bilateral clefts. A bony defect is present in the lateral aspect of the arch bilaterally with preservation of the most dorsal part of the arch. (**d**) Absence of the posterior arch with persistent posterior tubercle. (**e**) Absence of the entire arch including posterior tubercle. The entire posterior arch is missing, but occasionally one or both roots of the arch near the lateral masses are preserved. (From: Currarino et al. [7]. Reprinted with permission from American Society of Neuroradiology)

Os Odontoideum

Os odontoideum is an oval or round-shaped ossicle of variable size with a smooth cortical border which may be located in the position of the odontoid process (orthotopic) or near the base of the occipital bone in the region of the foramen magnum

(dystopic), without bony connection to the body of the axis [12]. The etiology of os odontoideum has been mainly attributed to embryological (congenital) or traumatic (acquired) causes [13–15]. Os odontoideum is considered to be a segmental anomaly resulting from a failure of fusion between the dens and the body of the axis. The congenital theory has two potential explanations. Firstly, the incomplete ossification of the intervertebral disc that separates the odontoid process from the base of the axis has been thought to be the reason for the formation of the residual ossicle. Secondly, it is felt that there is a familial component with several cases of os odontoideum within a family group having been reported. Wang et al. reported a familial example of os odontoideum with three occurrences [13].

However, current evidence now suggest that there is a traumatic etiology in the majority of cases [16, 17]. This hypothesis is supported by a history of trauma being present in several patients found to have radiographic evidence of an os odontoideum. There are also a large number of patients where it is difficult to draw a clear connection to a traumatic cause due to there being a delay in the clinical diagnosis and the traumatic event. At the time of injury, it is believed that the alar ligaments contract resulting in a distraction of the fractured odontoid fragment away from the base of the axis. As the fractured ossicle migrates rostrally toward the occiput and the fracture remodels, the smooth circumferential cortical margins remain. This remodeling process in an adolescent or juvenile patient makes os odontoideum different from a geriatric Type II odontoid fracture. Babak et al. agree that the os odontoideum is likely caused by a traumatic event [16]. They felt that a traumatic cause of os odontoideum is likely due to the gap between the os odontoideum and the remnant of the odontoid process characteristically appearing above the level of the superior facets of the axis. According to the congenital theory, the failure of fusion should be observed at the level of the neurocentral synchondrosis.

Plain radiographs are used to diagnose os odontoideum and to assess C1-C2 stability. The sensitivity and specificity of plain radiographs for this diagnosis remain to be reported. Radiographic evaluation should include open mouth odontoid, AP, and lateral views of the cervical spine in flexion and extension. Flexion and extension radiographs can add valuable information for the diagnosis; however neck pain can result in limited excursion between views and potentially cloud the diagnosis of instability (false negative). Caution is advised when obtaining flexion and extension views in an athlete complaining of neck pain or with neurological deficits on physical exam as pathologic motion can result in potential neurological consequences.

Acquisition of a CT scan and/or an MRI of the craniocervical junction is at the discretion of the healthcare provider and can be considered if plain radiographic evaluation is felt to be inadequate [18]. CT scans and MRI scans are useful for the more detailed evaluation of osseous abnormalities, vertebral arteritis, and spinal cord compression [16]. Hughes et al. suggested that dynamic MRI may have an advantage in visualizing the instability of the joints and surrounding soft tissues [19]. The os odontoideum should be clearly differentiated from ossiculum terminale, which refers to the nonunion of the apex at the secondary ossification center. Ossiculum terminale is rarely associated with atlantoaxial instability and usually does not require surgical treatment [16].

The presence of os odontoideum may be completely asymptomatic or present with isolated neck pain or potentially myelopathy. For the athlete with an asymptomatic os odontoideum, it is commonly an incidental finding with the athlete being completely neurologically intact; however, flexion-extension radiographs may show evidence of atlantoaxial instability. For the symptomatic patient, neck pain, especially occipitocervical pain, is a common presentation. Dai et al. reported that the most common neurologic finding was myelopathy with associated radicular symptoms (22 patients) [20].

The initial presentation of os odontoideum can be insidious in nature. Os odontoideum with atlantoaxial dissociation may develop acutely or chronically and cause compression of the cervicomedullary junction, vascular compromise, and cervical pain if left untreated [21]. Untreated, this may cause symptoms including transient myelopathy to tetraplegia, central cord syndrome, and even death. Clinically, patients with os odontoideum experience severe neck pain and myelopathy resulting from craniocervical instability and spinal cord compression. A close correlation between the extent of hyperintensity in T2-weighted images and the severity of neurological deficits, either on admission or at the last follow-up, has been reported. Zhang et al. suggested that a prophylactic surgery should be considered for patients at risk of developing myelopathy and to avoid the associated neurological deterioration [21]. A level III recommendation raised by Curtis et al. also suggests that clinical and radiographic surveillance or posterior C1-C2 fixation and fusion is recommended for patients with asymptomatic os odontoideum [18]. For the symptomatic os odontoideum patient, posterior fixation and fusion is recommended [18].

There are reports of major neurologic complications after minor trauma as the initial presentation of patients with previously undiagnosed os odontoideum. In one report, a 13-year-old girl with an os odontoideum, a bifid atlas, and a cyst around the odontoid tip, with consequent severe atlantoaxial and craniovertebral instability, developed quadriplegia after a minor injury to the head [10]. Tory et al. also report a case with quadriplegia after a minor trauma and recommended that an odontoid anomaly be considered an absolute contraindication for sporting activity [22].

According to the criteria of White et al. [23], radiographic evidence of C1–C2 hypermobility is indicated by an anterior dens interval of 4 mm or greater, and we would consider this to be an absolute contraindication to return to play. Even after atlantoaxial cervical fusion, it is still an absolute contraindication to participate in contact sports. In contrast, an ossiculum terminale is not a contraindication to athletic activity and can be considered an incidental finding in isolation. Figure 10.2 is a treatment algorithm that we have used to guide clinical treatment of os odontoideum.

Klippel-Feil Syndrome

First described in 1912 by Maurice Klippel and Andre Feil in France, Klippel-Feil syndrome (KFS) is commonly associated with a low posterior hairline, a short neck, and limited cervical range of motion [24–26]. To be diagnosed with KFS, the failure

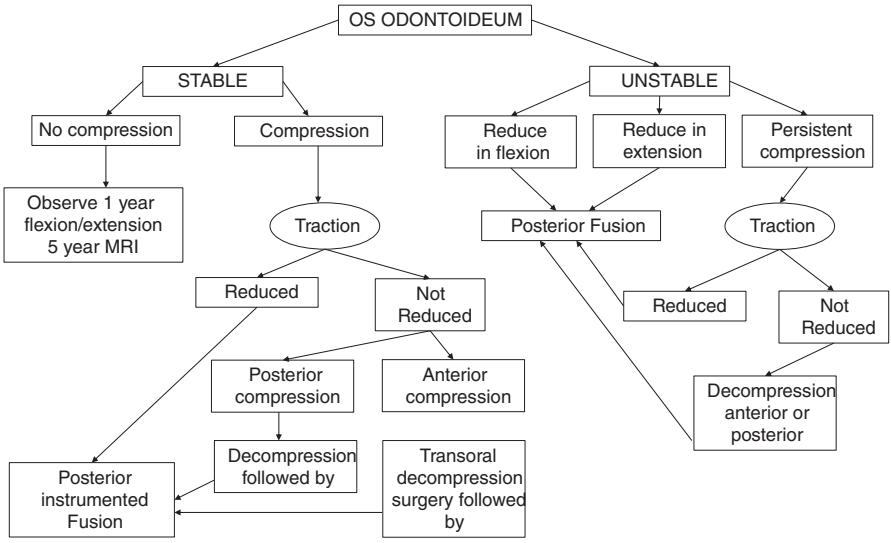

Fig. 10.2 An algorithm for the management of a patient with os odontoideum

of segmentation of cervical motion segments, noted as congenitally fused vertebrae, must be present [24, 27]. When evaluating the athlete for the presence of KFS, the presence of the classically described clinical triad cannot be relied upon. In a retrospective review of 31 adolescent patients (12 male, 19 female), with an average age of 9.7 years at initial evaluation, Samartzis et al. found that limited cervical range of motion was the most common finding present in patients with KFS [28]. In 35.5% of the patients evaluated by Samartzis, none of the expected clinical findings were present, while one of the expected findings was present in 38.7%, two in 16.2%, and three in 9.7% of the cohort [28]. While the classically taught clinical triad represents the major clinical findings of KFS, there are multiple other associated findings that are important to consider when evaluating an athlete for sport eligibility. Major anomalies associated with KFS syndrome are small stature, thoracic kyphoscoliosis, lumbar scoliosis, Sprengel deformity, restricted mouth opening, and bilateral sensorineural hearing loss [29–32]. Minor associations are mild face asymmetry, high-arched palate, rhino scoliosis, high nasal bridge, renal agenesis, persistent trigeminal artery, thoracic bifurcation of the common carotid artery, aortic coarctation, anomalous course of the subclavian artery, and agenesis of the internal carotid artery [25, 27, 29, 32].

Klippel-Feil syndrome can be classified into three subtypes: Type I is defined as a single congenitally fused cervical segment; Type II is composed of multiple non-contiguous, congenitally fused segments; and Type III is multiple contiguous, congenitally fused cervical segments [24, 27]. The most commonly identified subtype is the Type II fusion with the most commonly fused segment being C2-C3, followed by C5-C6 [33]. The age at which complete fusion of the affected segments (C2-T1) occurs is 77.8% at ≥10 years of age, 87.5% at ≥15 years of age, 91.7% at ≥16 years

of age, 95.7% at ≥17 years of age, 86.5% in skeletally mature patients, and 100% in adults [33]. Ultimately, in a review of 28 patients, Samartzis et al. found 36% of KFS patients to have axial symptoms, predominantly associated with Type I patients [24]. Type II and Type III patients predominantly presented with radiculopathy and myelopathy [24].

The impact of KFS on the diameter of the cervical spinal canal has been evaluated by Samartzis et al. in a prospective evaluation of 29 patients with KFS [34]. Congenital fusion of the vertebral bodies within KFS may also result in an alteration of appositional bone growth of the affected vertebral bodies. In this study, it was noted that not only were the diameters of the fused vertebral body segments affected but also the diameters of the cranial and caudal vertebral bodies. This results in an increased space available for the cord [34]. Therefore, the likelihood of congenital cervical stenosis associated with the presence of KFS is decreased in the pediatric and adolescent athlete. As the age of the athlete increases, it may be necessary to begin considering the potential presence of stenosis due to degenerative changes at the levels adjacent to the fused segments.

Patients with KFS have been noted to be at increased risk for neurologic injury from minor trauma [27, 35]. Vaidyanathan et al. reported on a 51 years of age patient that suffered an incomplete tetraplegia following a slip and fall to the ground where the patient struck his head [27]. Of note, the patient had a similar incident approximately 15 years prior which resulted in a transient numbness and paresis of the lower extremities. This patient was found to have congenital fusions of C2-C3 and C4-C5 (Type II KFS) [27]. The authors of this report recommend that patients with KFS "should be made aware of the increased risk of sustaining transient neurologic deterioration after minor trauma if there is associated radiographic evidence of spinal stenosis" [27]. Pizzutillo et al. in a study of 111 patients with KFS noted that those with hypermobility of the upper cervical spine were at increased risk of neurologic sequelae when compared to a similar control population [35]. If an injury resulting in airway compromise on the track, field, or court was to occur, it is important for the healthcare provider to know that the shortened neck, decreased cervical range of motion, or kyphoscoliosis present in some patients with KFS can make emergent airway management difficult [36].

While the clinical and radiographic presentations of Klippel-Feil syndrome are quite varied, recommendations regarding athletic participation of the athlete with subaxial Klippel-Feil can be based on basic principles. A Klippel-Feil patient with noted hypermobility (greater than 3.5 mm of horizontal displacement or 11 degrees rotation difference to the adjacent level on flexion-extension radiographs) of the cervical spine should be restricted from participating in contact or other high-risk sports [23]. The patient with cervical stenosis in the setting of Klippel-Feil syndrome should be advised that they are at increased risk of transient neurologic injury from minor trauma, but this should not necessarily preclude athletic participation. In general, the Type I KFS athlete should be treated as any other athlete; neurologic symptoms and stenosis should guide sport participation. In our opinion, Type II and type III KFS patients should be contraindicated from participating in contact sports.

Assimilation of the Atlas and Klippel-Feil Syndrome

The assimilation of the atlas is caused by the failure of segmentation between the fourth occipital sclerotome and the first spinal sclerotome. It may present with focal, segmental, unilateral, or bilateral fusion. It is usually observed in patients with Klippel-Feil syndrome. Ultimately, this deformity may lead to atlantoaxial instability and basilar invagination [9].

An occipital-C1 assimilation is a relative contraindication to return to participation in contact sports. The patient and family should understand that recurrent injury is a possibility and the degree of risk is uncertain. When C1 assimilation is combined with Klippel-Feil syndrome, basilar invagination, or atlantoaxial instability, it is an absolute contraindication for return to play [11].

Congenital Cervical Stenosis

Congenital cervical stenosis (CCS) is a complex multifaceted topic with implications for all athletes, especially those who participate in contact sports. Cervical stenosis can be congenital, degenerative, or acquired from accumulated trauma or a combination of each. For a detailed understanding of these topics and related clinical issues related to athletes, the reader is encouraged to refer to the appropriate chapters of this text. It is important to have a thorough understanding of CCS so that more objective return-to-play recommendations can then be made.

A significant number of athletes have congenital cervical stenosis. Some are diagnosed after the onset of neurologic symptoms, others following the onset of pain, and some are discovered incidentally during the evaluation of a brachial plexus neuropraxia. Over the last few decades, with the accessibility of MRI scans, the topic of spinal cord signal change and functional stenosis (amount of cerebrospinal fluid around the cord) have become increasingly studied [37–39], because not only are the bony dimensions of the canal important but also the role of the discs, ligaments, and osteophytes in creating extrinsic cord compression can be more easily and precisely evaluated. Additionally, the cord diameter [40] and cross-sectional area vary greatly [41].

There is not always a consensus of what is considered stenosis as there are numerous methods to measure this, and radiographic standards, both technically and anatomically, are not consistent. While there are varied definitions of cervical stenosis, most agree that with a static sagittal canal diameter >14 mm, the diagnosis is excluded. The Torg ratio (canal diameter-vertebral body diameter) has been used extensively, but with more advanced imaging, further evaluation can now be undertaken and the process further understood [37].

Bajwa evaluated 1066 American cadavers and defined congenital cervical stenosis as a geometric canal area 2 standard deviations below a standard measurement [42]. The values for CCS were defined at each vertebral level and ranged from 1.82 cm^2 (C3/4) to 1.89 cm^2 (C6/7). When this was correlated with sagittal canal

diameter (SCD) and interpedicular distance (IPD), they concluded that values of SCD <13 mm and IPD <23 mm were strongly associated with the presence of CCS at all levels.

Attempting to predict the increased risk of potential neurologic sequelae in athletes with CCS has gained interest in the literature. Presciutti [43] determined that a space available for the cord (CSF surrounding the cord) of 5 mm in American football players resulted in a sensitivity of 80% and a negative likelihood ratio of 0.23 for potential neurologic sequelae. If the cutoff was decreased to 4.3 mm, the sensitivity is increased to 96%, and the negative likelihood ratio is increased to 13.25, respectively. Hence, a space available for the cord of 5 mm was determined to be a good test for screening athletes at risk, and a measurement of 4.3 mm added additional confidence as a confirmatory test.

Herzog et al. [44] radiographically evaluated asymptomatic professional football players to determine radiographic parameters in these athletes. They determined that the correlation coefficient was higher for CT scan than it was for MRI measurements. The diameter of the canal on plain radiographs in the subaxial spine, when corrected for magnification, ranged from 14.8 to 15.4 mm, with the cervical-6 level being the most capacious. These closely approximated a control group [45].

Nouri [39] studied cervical cord-canal mismatch. This accounts for both cord and canal size by measuring a spinal cord occupation ratio (SCOR). When the SCOR was found to be ≥70% on midsagittal or ≥80% on axial imaging, a mismatch was identified, and the potential concern of an increased risk of spinal cord injury was determined to be present. The importance of this ratio is that canal size and cord size are both important factors in determining the risk of potential injury. A narrow canal as an independent factor may not be as predictive of increased risk of injury. Nouri found that the smallest average spinal canal diameter is at the cervical-5 to cervical-6 levels [39].

Cord size was found to vary independently of canal size in an MRI study by Kato et al. They determined that morphologically, the male spinal canal sagittal diameter was 11.2 mm and 9.5 mm at the cervical-5 mid-body and the cervical-5–6 disc level, respectively [41]. In reviewing similar studies, they noted that there were different findings of normal spinal parameter measurements and hypothesized that different MRI magnet strengths and different imaging modalities used (CT scans and plain radiographs) could potentially be the source of these observed differences. Hence, any individual imaging study must be taken in context, further complicating this topic. The cervical stenosis in this chapter lends additional information on diagnosis, management, and return-to-play.

Marfan Syndrome

Marfan syndrome is a relatively uncommon autosomal dominant disorder that results from a mutation of the fibrillin-1 gene. Musculoskeletal manifestations include scoliosis and ligamentous laxity [46]. Bony and ligamentous abnormalities of the

cervical spine include increased atlantoaxial translation, basilar invagination, and focal kyphosis, but clinically symptomatic cases are rare [47]. Clinically symptomatic cases usually present with neck pain and may initially be managed with physical therapy, cervical traction, and non-operative management. Arthrodesis is an option for stabilizing the cervical spine should gross instability be present [48, 49]. For the subaxial cervical spine, kyphosis and subluxation can also be observed, and symptomatic cases may warrant arthrodesis [50, 51]. Preoperative evaluation and consequent care with intubation and positioning of these patients may be necessary.

Neck pain in the patient with Marfan syndrome should alert the physician to evaluate the patient for cervical instability. When a patient diagnosed with Marfan syndrome presents with a cervical spinal cord abnormality on MRI, asymptomatic ligamentous laxity (i.e., greater than 11 degrees of kyphotic deformity as compared with the cephalad or caudal vertebral functional spinal unit or more than 3.5 mm movement on lateral flexion-extension radiographs) or C1–C2 hypermobility is present with an anterior dens interval of ≥4 mm; it is an absolute contraindication to return to contact sports [11].

Basilar Invagination

Basilar invagination (BI) is a congenital abnormality of the craniovertebral junction in which the odontoid process prolapses into the foramen magnum. It is a radiographic finding with possibly significant clinical findings that can be the result of hypoplasia of the clivus, incomplete formation of the C1 ring with displacement of the C1 lateral masses, achondroplasia, or atlanto-occipital assimilation. Basilar invagination is often confused with basilar impression, platybasia, and cranial settling. Basilar impression results from the secondary acquired form of invagination, due to conditions such as rheumatoid arthritis (RA), tumor, Marfan syndrome, or other diseases. Platybasia is defined as an abnormal basilar angle when measured from the plane of the clivus to the plane of the anterior skull base and is often associated with BI. Cranial settling refers to a form of basilar impression caused by RA.

Basilar invagination is often associated with other craniovertebral abnormalities, including assimilation of the atlas, remnants of the occipital vertebrae, blocked vertebrae, short neck, or other vertebral anomalies. Between 25% and 35% of BI cases are also associated with a Chiari I malformation, syringohydromyelia, syringobulbia, and hydrocephalus [52]. Caetano et al. reported 66 cases of basilar invagination and showed that the most common clinical symptoms included weakness in the lower limbs (68%), unsteady gait (56%), and headache (53%) [53]. Goel et al. reported a surgical study on 190 BI patients in which 80 of the patients had no evidence of an associated Chiari I malformation. Among these 80 patients, the most common symptoms included weakness (100%), neck pain (59%), and posterior column dysfunction (39%). Physical exam findings included torticollis (69%), restricted neck movement (59%), low hairline (48%), webbed neck (47%), and short neck (41%). Trauma was a major factor that influenced the acute development

of symptoms in these previously asymptomatic patients. However, in the second group of patients with associated Chiari malformations, symptoms progressed slowly without any antecedent trauma. Goel et al. postulated that the symptoms and signs in group 1 were related to brainstem compression by the odontoid process, whereas in group 2 they were related to the crowded neural structures at the foramen magnum. In group 2 patients with associated Chiari malformations, 44% are presented in the third decade of life, and the duration of symptoms was slowly progressive, and the most common symptoms included weakness (94%), paresthesia (79%), and posterior column and spinothalamic tract disturbance (56%). Localized findings are similar to those observed in the patients included in group 1 [54].

Many measurements have been designed to gauge the degree of BI. The commonly used measurements include the Chamberlain line, McGregor line, or McRae line (Fig. 10.3). The Chamberlain line is drawn from the posterior portion of the hard palate to the posterior edge of the foramen magnum. The tip of the odontoid usually lies below or at the Chamberlain line. Compared with plain radiographs, CT scans and MRI offer more detailed information for the measurements of BI and permit assessment of the soft tissues, including neural structures, vascular structures, and ligaments. Advanced imaging techniques are also critical for operative planning when needed. Basilar invagination is diagnosed if the tip of the odontoid protrudes greater than 5 mm past the Chamberlain line. The McGregor line originates from the posterior margin of the hard palate to the lowest point of occiput. It is considered abnormal when the tip of the odontoid protrudes greater than 7 mm past the McGregor line. The McRae line is defined by the anterior and posterior rim of the foramen magnum. The tip of the odontoid typically lies below this line.

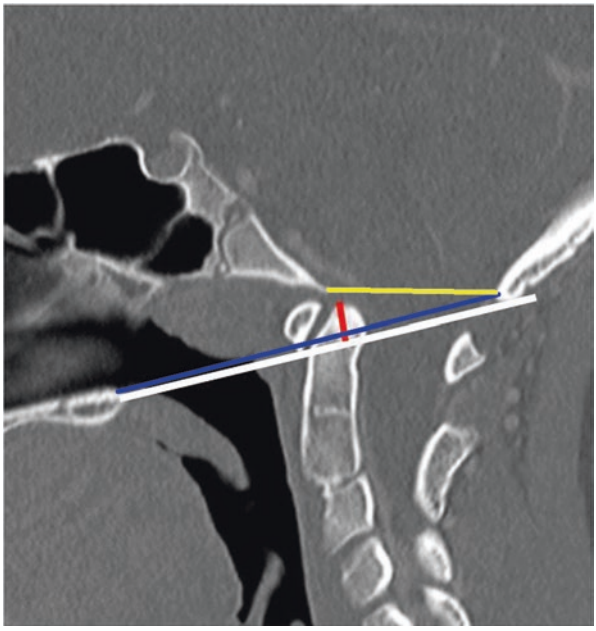

Fig. 10.3 Basilar invagination as demonstrated by a sagittal reconstruction of a CT scan. The red line indicates that the tip of the odontoid process extends 6.6 mm above the McGregor line (white). Chamberlain line (blue); McRae line (yellow). (Adapted from: Donally CJ III, Varacallo M. Basilar Invagination. StatPearls [Internet]. 2019. Distributed under terms of the Creative Commons Attribution 4.0 International License [http://creativecommons.org/licenses/by/4.0/.] Lines indicating McRae and Chamberlain Lines were added by Cordover et al.)

Basilar invagination results in a progressive neurologic deficit if left untreated. Vaccaro et al. summarize the criteria for clearing the cervical spine of an athlete for participation in competition. There were three categories: no contraindications, relative contraindication, and absolute contraindications to participation. Clinical history or physical examination findings of cervical myelopathy and spinal cord abnormality noted on MRI are absolute contraindications to participation [11]. Goel et al. also reported that BI patients without Chiari malformation may develop neurological deficits after minor trauma [54]. Therefore, radiographic evidence of BI is an absolute contraindication to return to play considering that many BI patients have or may develop neurologic symptoms.

Cervical Spine Clearance in the Special Needs Athlete

One organization that is involved with athletes that often have not only special needs but also congenital anomalies of the cervical spine is the Special Olympics. Due to the nature of their athletic population, the Special Olympics Inc. has created guidelines for clearance of the cervical spine and participation by athletes with cervical spinal abnormalities. Due to the non-specific nature of plain radiographs and the cost of more advanced imaging studies, only athletes with symptomatic spinal cord compression or atlantoaxial instability are evaluated further. Specifically, symptoms such as significant neck pain, radicular pain (localized neurological pain), weakness, numbness, spasticity (unusual "tightness" of certain muscles) or change in muscle tone, gait difficulties, hyperreflexia (highly reactive deep tendon reflexes), change in bowel or bladder function, or other signs or symptoms of myelopathy (injury to the spinal cord) are evaluated for by the examining physician. Once the presence of symptoms has been confirmed by the examining physician, the athlete must be evaluated further by a specialist who is able to fully evaluate the nature of the athlete's condition. After this evaluation is completed, the athlete will be allowed to participate if the parent or guardian signs a waiver stating understanding of the athlete's condition and associated risks [55].

Expert Opinion

The evaluation of the athlete in the setting of congenital cervical spinal abnormalities is complicated by both a lack of conclusive evidence and limited recommendations on treatment or restrictions regarding athletic participation. It is the opinion of the authors of this chapter that:

- The symptomatic, otherwise, healthy athlete be evaluated with a careful eye to the presence of instability.
- In the setting of an incidental identification of a congenital abnormality in the asymptomatic and otherwise healthy athlete, attention must be turned to evaluating the athlete for the presence of instability or congenital stenosis.

- Treating the symptomatic athlete is guided by identifying the nature of the abnormality and determining if instability is present and whether or not surgical treatment is indicated.
- In the special needs athlete, no focused cervical evaluation is indicated in the asymptomatic participant.

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Chapter 11
Cervical Disc Herniation in Athletes

Shalin S. Patel, Brett David Rosenthal, and Wellington K. Hsu

Introduction

Cervical disc herniations account for approximately one-quarter of all disc herniations in elite athletes [1]. In the general population, there is a consensus that the criteria for return to recreational sport are full, painless cervical range of motion, absence of residual neurologic deficits, absence of neural compression on advanced imaging, and bridging bone formation after fusion [2]. Historically patients sustaining a cervical spine injury had been recommended to avoid contact and collision sports, such as football, hockey, rugby, wrestling, and martial arts [2, 3]; however, recent experience from professional athletes, particularly those participating in high-risk sports, has led to the development of criteria for return to play recommendations.

Only a few peer-reviewed studies have addressed cervical disc herniation management in elite athletes and how it impacts their ability to return to play. To date, pragmatic considerations have limited consensus regarding optimal treatment strategies for high-level athletes and when they can return to play. Many authors have also pointed out that spine surgeons are often pressured by various parties other than the athlete, including family members, coaching staff, and organizational staff during the management of these spine injuries [4]. Finally, given the limited data regarding this unique patient population, recommendations are often arbitrary and based on the judgment of each individual surgeon [4]. For these reasons, return to play decision-making for athletes with cervical disc herniations has historically been challenging [5, 6]. In this chapter, we will discuss the current

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literature regarding cervical disc herniation management in elite athletes, outcomes, and how these injuries affect players' ability to return to play.

Epidemiology

Injuries to the axial skeleton comprise 7% of all injuries to professional athletes [7]. The National Football League (NFL) Sports Injury Monitoring System reported that 275 players suffered a disc herniation between 2000 and 2012 [1] with offensive lineman as the most common position and 76% in the lumbar spine. Cervical disc herniations (CDH) accounted for 23% of all disc pathology and were mostly sustained by linebackers, defensive backs, and linemen [1, 8]. CDH ranked second only to spinal cord injuries in causing the most mean number of days lost, including training, practice, and games in NFL athletes [1].

Non-operative Management

In the general population, there is a clear consensus that the initial course of management for cervical disc herniations should be non-operative, conservative care. Particularly, in younger patients, even for those with associated radicular symptoms, the majority will spontaneously resolve with time. In the interim, patients can be managed with symptomatic control afforded by rest, nonsteroidal anti-inflammatory drugs, physical therapy, and potentially corticosteroid injections.

Similarly, in the elite athlete, non-operative management for cervical disc herniations should be the first-line treatment when indicated. Avoiding the risks of surgery while still regaining full, painless cervical range of motion without associated radiculopathy has proven to be successful in many patients [8]. In a paper from our research group with the largest cohort of players, Hsu et al. found that 21 of 46 American football players (45.7%) were able to successfully return to play after non-operative management [9]. A study of Major League Baseball (MLB) pitchers with CDH demonstrated that of the 11 pitchers included, 3 were treated non-operatively, and of those, 1 returned to professional play [10]. Despite the limited evidence examining conservative treatment of elite athletes with cervical disc herniations, the data suggests athletes can return to contact and high-demand professional sports.

Surgical Management

When non-operative management is unsuccessful in treating a cervical disc herniation, or when there are other associated conditions such as transient paresis or spinal cord signal change on MRI, surgical management may be indicated. There have

been several small studies looking at outcomes of elite athletes with cervical disc herniations treated with either an anterior cervical discectomy and fusion (ACDF), total disc replacement (TDR), and/or posterior foraminotomies.

Anterior Cervical Discectomy and Fusion

In one of the earliest studies of professional athletes with a CDH [8], three American football athletes underwent ACDF after failing non-operative treatment with spinal cord signal changes on MRI, and only one successfully returned to play [8]. Subsequently, a single-surgeon case series of 15 athletes with cervical disc herniations from both the NFL and professional wrestling demonstrated that 13 of 15 athletes (86.7%) were able to return to professional play after a single-level ACDF [11]. This experience led Maroon and colleagues to conclude that athletes were able to return to play in contact sports after ACDF if neurologically intact and asymptomatic and had evidence of a solid fusion on postoperative imaging [11].

Data from our research group suggests that surgical treatment has just as good of a prognosis for return to play in collision sports when compared to conservative care [9]. Although the patients treated with surgery returned to play at higher rates than those treated conservatively, selection bias may have affected these results. Notably, there were risk factors for a poor outcome such as position (defensive back) and/or age at treatment [9].

In a follow-up retrospective multicenter study, Mai and colleagues stratified cervical disc herniations in the elite athlete population into upper (C2-4) and lower (C4-T1) cervical segments [12]. Historically speaking, because of the implications of adjacent segment degeneration, there has been a disagreement as to the implication of the level of ACDF surgery on the potential to RTP to a collision sport. Out of 40 NFL athletes in the cohort, 15 had upper cervical disc herniations, and of these, 10 (66.6%) were able to return to play with no significant difference in their performance metrics, which was comparable to those with lower cervical disc herniations. The authors concluded that collision athletes may have a higher percentage of upper cervical disc herniations compared to the general population, but the level may not preclude return to professional sport or compromise the athlete's performance level [12].

In a study of Major League Baseball pitchers with cervical disc herniations, 8 out of 11 were treated surgically [10]. Seven of these patients underwent ACDF, and one underwent a cervical TDR with 87.5% successfully return to play and maintaining a stable performance level after returning to sport [10]. In a series of professional rugby players, 19 athletes underwent ACDF after failing conservative management. 89.5% of players noted symptomatic improvement, and 68.4% of players were able to return to professional play [13]. Of the 13 players who returned to rugby after ACDF, 2 suffered a recurrent cervical disc herniation.

One retrospective case series identified professional athletes of the NBA, NFL, NHL, and MLB who had undergone surgical treatment [14]. Using sport-specific

performance metrics, it was determined that athletes of the NBA, NFL, and NHL did not experience decreases in their performance after surgical intervention (of any type), while MLB athletes had a 14.3% reduction in their performance after ACDF. Overall, after ACDF, return to play occurred at a rate of 70.9% and at a mean of 367 days [14].

Continued controversy exists over whether a two-level ACDF should be considered a contraindication for returning to play collision sports. While studies have suggested that this is a contraindication [8], reports of safe return to play have been published in contact sports [13]. Certainly, the determination of whether an athlete should be allowed to return to play must be dependent upon the expected rigors of the sport, presence of persistent symptoms, physical examination, and success of fusion. Additional studies are needed to shed light on the controversy surrounding two-level anterior fusions.

Alternative Treatments

Because cervical TDR has only been performed recently in elite athletes, little data exists. Reinke and colleagues [15] reported clinical outcomes in 2 professional lugers and 20 semiprofessional athletes who underwent cervical TDR. Subjectively, these athletes experienced symptomatic improvement, primarily noted by the absence of radicular symptoms, and returned to their sport with no loss in performance. To date, there is no data available that would guide the decision-making process when applying to contact sports (Fig. 11.1).

Posterior cervical foraminotomies (PF) are occasionally explored as a motion-sparing option for treatment of cervical radiculopathy secondary to a cervical disc herniation. In a retrospective case series comparing ACDF, PF, and TDR in athletes performed by Mai et al., it was determined that the PF cohort had a significantly greater return to play rate (92.3%) compared to athletes who had an ACDF (70.9%) [14]. Additionally, the PF athletes returned to play sooner (mean = 238 days) compared to those who had an ACDF (mean = 367 days). The PF athlete cohort, however, had a significantly higher risk of reoperation (46.2%) compared to ACDF (5.8%). These outcomes are helpful in setting an athlete's expectations during preoperative counseling [14] (Fig. 11.2).

Expert Opinion

While some authors have opined that an acute disc herniation is an absolute contraindication for return to play for professional athletes engaging in contact sports [16], most of the peer-reviewed studies available in the current literature would suggest otherwise. Kang et al. endorses that there is a strong consensus among the current literature that return to contact sports after single-level ACDF are safe and feasible [2]. Some have even proposed that spinal cord signal change on MRI is not

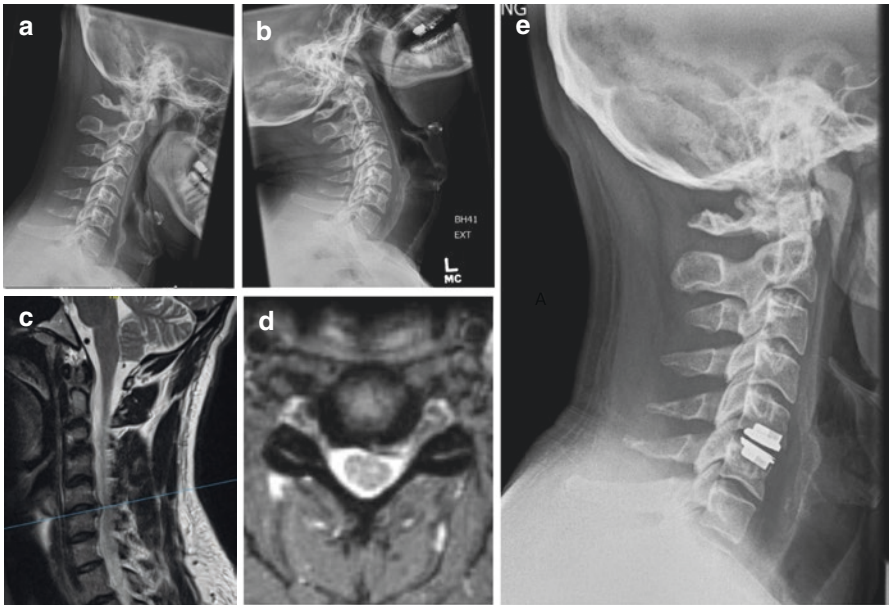

Fig. 11.1 This is a case presentation of a 21-year-old elite baseball player who presented to our clinic with 8 weeks of left C6 radiculopathy. He had no weakness on examination. He was unable to participate in his off-season workout despite extensive conservative treatment. Panels (**a**, **b**) show lateral flexion/extension radiographs demonstrating full range of motion with minimal spondylosis. Panels (**c**, **d**) show a T2-weighted cervical MRI demonstrating a soft paracentral disc herniation on the left at C5-6. After extensive discussion with the patient, he elected to move forward with a CDA for surgical treatment. Panel (**e**) shows the post-op radiograph at 6 weeks demonstrating the C5-6 CDA. The patient was released to return to play at 3 months when the patient had full range of motion, no pain, and no persistent neurologic symptoms

a contraindication for return to play in contact sports, as long as the player is asymptomatic and neurologically intact [11]. Overall, when accounting for the results from all of the currently available studies of cervical disc herniations in elite athletes, more than 50% of players were able to return to all professional sports [17], which is probably equivalent regardless of operative vs. non-operative treatment. Notably, these types of injuries have a much different prognosis than those associated with myelopathy or even transient neuropraxia.

While single-level ACDF has been extensively studied as the primary surgical treatment for athletes with cervical disc herniations, there remain significant controversy and limited evidence regarding return to play to contact sports after 2- and 3-level ACDF, TDR, and with posterior approaches, including posterior laminectomy and fusion, posterior laminoforaminotomy, and laminoplasty [2]. In a study of active military personnel, Tumialan et al. found that posterior laminoforaminotomy allowed for faster return to unrestricted full duty and was more cost-effective than ACDF [18].

Ultimately, further investigation is needed to address the optimal treatment strategies for elite athletes with cervical disc herniations. At present, the available data

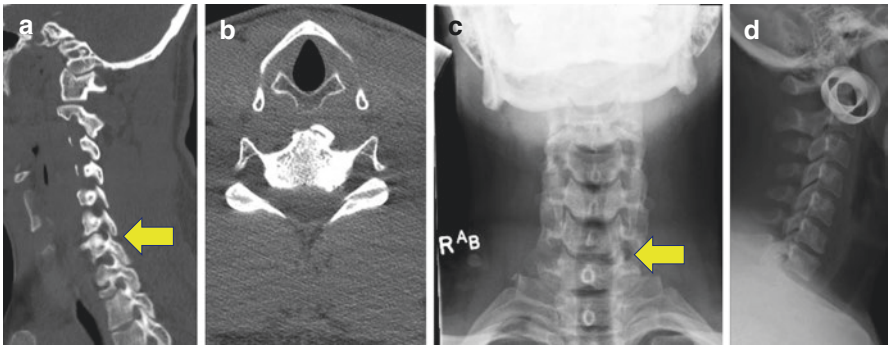

Fig. 11.2 This is a case presentation of a 26-year-old professional body builder. He presented to our clinic with complaints of left arm pain and triceps weakness that had progressed over several months. He did demonstrate weakness in his left triceps compared to his right side. He had received to two prior ESIs which temporarily provided relieve. Panel (**a**) shows a parasagittal CT scan showing the left foramen. The arrow points to the left neuroforamen and demonstrates a bony osteophyte extending from the superior articulating process causing stenosis. Panel (**b**) shows a CT scan axial image of the C6-7 neuroforamen. The left neuroforamen is stenotic compared to the contralateral side. MRI (not shown) again demonstrated neuroforaminal stenosis with a disc herniation. We had a discussion with the patient and elected to move forward with a left-sided C6-7 laminoforaminotomy. Panel (**c**) demonstrates an AP radiograph at 6 months post-op. The yellow area shows the decompressed foramen. Panel (**d**) is a lateral radiograph at 6 months post-op demonstrating maintained lordosis with minimal spondylosis. The patient was able to return to weight lifting at 3 months post-op

suggest that athletes can be successfully treated non-operatively and return to play at high rates. It is acceptable to allow athletes to return to contact sports if they are asymptomatic, neurologically intact, and have full, painless cervical range of motion. For those who fail conservative treatment, it is both safe and feasible to return to contact sports after cervical disc surgery, if the previously mentioned criteria, along with a solid fusion mass, are met. Further studies are needed to address return to contact sports after TDR, multiple-level ACDF, and posterior surgical approaches, including laminoforaminotomy with discectomy, laminectomy and fusion, and laminoplasty. Due to the small population of elite athletes and highly individualized nature of these injuries in players who depend on their athletic careers, it is likely that treatment of cervical disc herniations in athletes will remain a controversial topic. Personalized treatment plans and shared decision-making between the athlete and surgeon are critical to exploring the benefits and risks of surgical intervention and returning to play.

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Chapter 12
Cervical Stenosis in the Elite Athlete

Tyler J. Jenkins, John M. Rhee, and John G. Heller

Introduction

Cervical stenosis has been studied extensively in the general population; however there is a paucity of evidence on treating the elite athlete with cervical stenosis. While many of the treatment principles from the general population can be applied to the elite athlete, the rigors of their sport require a nuanced, case-by-case approach. The presentation of cervical stenosis can range from an asymptomatic radiographic finding to quadriparesis [1]. Return to play (RTP) for professional athletes with cervical stenosis is a controversial topic [2]. Most athletes can successfully RTP after a single-level anterior cervical discectomy and fusion (ACDF), and players have also RTP after multilevel procedures [3]. However, subtle differences in range of movement (ROM) after fusion can lead to altered performance in the elite athlete depending on sport and position-specific demands [3]. This chapter will review the best literature available and apply our personal clinical experience to help guide decision-making for athletes with cervical stenosis.

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Cervical Stenosis

Cervical stenosis (CS) can present with a range of symptoms. When this diagnosis is found incidentally, there is little guidance in the evidence-based literature for RTP recommendations. This often leads to challenges among team physicians as to the relative risk of neurological deficit with repeated collision events. We do know that critical CS increases an athlete's risk for SCI after spine trauma [4–6]. However, many associated factors can affect the risk, including the clinical symptoms, severity of stenosis, and cord pathology. We believe that four critical factors should be considered in the determination of RTP in athletes with CS: (1) clinical history, (2) physical exam. (3) imaging characteristics, and (4) sport played. Each of these aspects adds additional information to the current state of neurological dysfunction and future risk to the spinal cord.

Clinical History

Clinical history should assess symptom chronology, traumatic events, and interrogation of current neurological complaints [7]. Transient quadriparesis is diagnosed when a player suffers symptoms similar to a spinal cord injury immediately after head/neck trauma that recovers over time. This condition can sometimes be confused with a "burner/stinger," acute radiculopathy, or a concussion. Delineating the exact symptoms and history surrounding trauma is important in identifying risk of future injury. It is helpful to have members of the training staff available for questions as the player may not be able to give a detailed history. Any videos of the injury, or time period after the injury, can also provide important clues. The more detailed the history of prior events, the more insight the spine surgeon will have when providing a recommendation.

While most experts agree that athletes who have had multiple episodes of transient quadriparesis from cervical stenosis should not be allowed to return to contact sports [4, 8–13], a player's fate after a single episode has been debated [2]. Guidelines offered by Torg and Ramsey-Emrhein suggest that a single episode with critical stenosis should serve as a relative contraindication to RTP [9, 12, 13]. However, there is disagreement on this subject, and most argue for a nuanced approach that considers the incident, exam, and severity of CS [10, 14]. For example, an isolated episode of cervical cord neurapraxia (CCN) with mild symptoms that recover within seconds may not necessarily preclude an athlete from RTP. On the other hand, an episode of paralysis that results in hospitalization requires hours to recover and/or, with residual symptoms, should serve as a contraindication to return to play [2].

Physical Examination

Physical examination identifies the degree of neurologic dysfunction. Findings such as hyperreflexia, weakness/numbness of the hands, subtle gait abnormalities, and loss of hand dexterity lead to a diagnosis of cervical myelopathy that precludes RTP

in contact sports [2, 7]. These signs indicate a threatened spinal cord that would be at high risk by the forces imparted in contact and collision sports. Other findings such as cervical range of motion, focal neurologic deficits, or pain with manipulation are also important to consider for return to play decision-making.

Imaging

Many times, the player has recovered from the neurologic insult prior to spine surgeon consultation. In these instances, imaging may guide recommendations for return to play. The imaging interpretation of cervical stenosis has evolved over time. One of the initial methods used for interpreting CS was the Pavlov-Torg ratio method, using the relative length of the vertebral body and spinal canal distance measured on plain radiographs [15]. However, this method has been proven to be unsatisfactory due to inherent magnification errors and poor predictive value [13, 16, 17]. MRI has become the imaging modality of choice, and with it the reproducibility of measurement, as well as the definition of CS, has evolved. The most common method utilized in the literature is to measure the sagittal diameter of the cervical spinal canal on a midsagittal T2 MRI image. Ladd and Scranton first defined stenosis as a diameter of the cervical spinal canal in the sagittal plane <15 mm [18] with "critical" stenosis as <12 mm. Since then, there has been some disagreement as to the exact thresholds of a stenosis diagnosis ranging from 10 to 14 mm, which has been based mainly on expert opinion [5, 12, 19–21]. These values provide some framework for an absolute definition, but none of them are based upon clinical symptoms or injuries, which greatly limit their use.

Jenkins et al. defined congenital cervical stenosis (CCS) as a canal diameter of less than 10 mm measured on midsagittal MRI at two or more sub-axial cervical levels in a patient population <50 years of age and concluded that CCS was a posterior-based anatomic anomaly [22]. Based on our personal experience in this patient population, it appears that this may be a more common condition in symptomatic high-level athletes than the general population. Recent expert opinion suggests that the presence of CS should not prevent an asymptomatic athlete or an athlete with no prior episodes of TQ from participation in a contact sport [2].

Schroeder et al. described a case series of professional American football players ($n = 10$) who had successful careers with a cervical spinal canal diameter of <10 mm at one or more levels measured on MRI without spinal cord injury [5]. None of these athletes were symptomatic, nor were there prior episodes of transient quadriplegia (TQ) [5]. Aebli and colleagues studied the risk of spinal cord injury after minor trauma in the general population and concluded that a canal diameter of 8 mm or less in diameter had the highest predictive value for SCI compared to canal diameters of 8.5, 9.0, and 9.5 mm [6]. It is our opinion that if the absolute diameter at one of the sub-axial cervical levels is less than 8 mm, then the athlete should not RTP.

Due to inherent variations in cord anatomy, experts have been using a more qualitative assessment of the spinal canal, defined as "functional stenosis." Defined as a

lack of cerebrospinal fluid (CSF) around the spinal cord on MRI or CT myelography, functional stenosis has been used as a contraindication to RTP [12, 16, 20, 23–26]. The concept is that those with functional stenosis do not have a cushioning CSF buffer between the spinal cord and the other elements of the spinal canal. Bailes reported on a case series of three athletes who were diagnosed with functional stenosis that had experienced severe episodes of transient quadriparesis, and all were recommended to retire from sport [12]. Subsequently, Paulus and Kennedy stated that most cases of cervical cord neurapraxia happen in the context of functional stenosis [27]. Cantu et al. showed that the only documented cases of athletes with quadriplegia without a spine fracture in the National Center for Catastrophic Sports Injury Research database all had functional stenosis [25]. Based on the current literature, we believe that athletes who have functional stenosis should not RTP in collision sports.

There is a fair amount of controversy surrounding the significance of signal changes in the cervical spinal cord, or myelomalacia, in the professional athlete population [28, 29]. Since this finding may indicate swelling and/or damage to the cord [30], some experts have opined that this should prevent an athlete from returning to a contact sport [2]. Many physicians would tolerate return to play to contact activities if the cord changes resolved after successful treatment. A more difficult scenario is the athlete in which myelomalacia persists despite the player having a full recovery, normal neurological exam, and no pain after cervical insult. Warren et al. published a series of single-level ACDF in three NFL players who had cord signal change as a result of cervical disc herniation (CDH) [31] and cleared all three to RTP after surgery. It is our opinion that the finding of persistent myelomalacia alone should not preclude an athlete from returning to collision sports as long as there is adequate area for the spinal cord and no neurological symptoms, which is in agreement with other surgeons [32]. However, myelomalacia with any degree of functional stenosis should preclude return to play for the athlete.

Consideration of Sport

Athletes who participate in noncontact (golf, swimming, tennis, etc.), contact (basketball, baseball, soccer, etc.), and collision sports (American football, hockey, lacrosse, cheerleading, etc.) should be evaluated differently because of the relative physical demands required to participate at a high level for each respective activity. Collision sports athletes are at greatest risk for head/neck trauma on a routine basis, while noncontact sports mainly avoid this risk [33, 34]. The relative requirements of the spinal canal in these situations differ based on the demands of the specific sport. Collision sport athletes should have an extra degree of caution applied to any return to play recommendations: clearly, the player should have no functional stenosis, normal neurologic exam, and full pain-free range of motion prior to clearance.

Treatment

The criteria for non-operative versus operative management do not vary significantly from the general population. In general, a spinal cord injury with correlative abnormal imaging warrants surgical intervention. The presence of myelopathic signs also favor toward operative management. Mild myelopathy may be observed in the general population, but athletes wishing to RTP will need operative intervention, particularly if they are involved in contact or collision sports. In the non-collision athlete without critical stenosis and a normal neurologic exam, non-operative management is clearly an option. Once again, clinical decisions should be nuanced and individualized to the patient, presentation, and sport.

Athletes can RTP after both non-operative and operative treatments once they become asymptomatic [10, 19]. The primary goals of surgical intervention for CS are to decompress the spinal cord and to provide a stable spine, which can be accomplished with either anterior or posterior approaches. In general, one- or two-level pathology favors an anterior approach (ACDF, CDA), whereas 2+ levels of pathology merit consideration of posterior surgery (e.g., laminoplasty if appropriate). Warren et al. cleared 11/13 professional football players to RTP after conservative treatments [31], and multiple studies across a variety of sports, such as football, rugby, wrestling, and baseball, have displayed that a single-level ACDF can be a safe treatment in professional athletes [3, 35–38].

Hsu et al. examined NFL athletes who underwent single-level spine surgery for CDH and found that players treated surgically experienced RTP at a higher rate than those treated non-operatively (72% compared to 46%) [3]. Among the operative group, NFL defensive backs experienced significantly shorter careers after treatment compared to other positions, which may be explained by the position's unique physical demands, such as the instinctive reactions to a football requiring uninterrupted cervical ROM. Not surprisingly, age was also found to be a factor, with older athletes experiencing significantly shorter careers after CDH.

Mai et al. compared RTP in professional athletes who underwent either single-level ACDF ($n = 86$) or posterior foraminotomy (PF) ($n = 13$) and found that players who underwent PF returned to play at a significantly higher rate (92.3% vs. 70.9%) and in a significantly shorter amount of time (238 vs. 367 days) [34]. However, the reoperation rate for PF was significantly higher (46.2% vs. 1.2%) within that time period [34]. While foraminotomy is used for radiculopathy and is not a treatment option for cervical stenosis, these studies illustrate that a majority of athletes can RTP after a single-level ACDF.

While the use of cervical disc arthroplasty (CDA) has grown in the general population over the past decade [39–41], its widespread applicability to professional athletes remains unknown [42]. Small case series of professional lugers and baseball players who underwent a one-level CDA and successfully RTP have been reported [34, 43]. Data on return to active duty in the military population after CDA suggest successful outcomes in the elite athlete [44, 45]. In 2010,

Tumialan and colleagues compared clinical outcomes in military personnel who underwent single-level CDA and ACDF [46]. The CDA group, which included seven Navy SEALs and one marine, all returned to unrestricted active duty and in a significantly shorter amount of time (10.3 vs. 16.5 weeks) compared to the ACDF group. Long-term outcomes after CDA in professional athletes are currently unknown [2].

Many surgeons believe that a two-level ACDF is considered a contraindication to return to play for collision sports [1, 9, 10, 19, 25, 27, 31, 47, 48]; however, there is a paucity of objective data supporting this notion [2]. Other surgeons have advocated for RTP in American football after two-level fusion [2]. A professional rugby player [38] and two military servicemen have been reported to return to full active duty after a two-level fusion [49]. We believe that the criteria for RTP after such an operation should depend on several factors, including collision vs. contact sports, position, symptoms, and physical examination. For example, a two-level ACDF may not be compatible with successful RTP for a defensive back but may be for an offensive lineman in American football. Further study is clearly required before definitive recommendations can be made.

Congenital CS often occurs over more than two levels of the cervical spine, and a posterior approach is often employed to treat this pathology because of its ability to enlarge the entire canal without the need for corpectomy. In the elite athlete, because a multilevel posterior cervical fusion would likely be career ending, laminoplasty is an attractive motion-sparing posterior alternative. We do have experience in treating elite athletes with laminoplasty who present with congenital CS (Fig. 12.1). The ideal candidate would be a patient that participates in a noncontact sport. Collision sports should be contraindicated from RTP in our opinion. We have treated a contact athlete (NBA basketball player) who RTP after laminoplasty, but extensive education was performed. In addition, the following qualifications had to be met prior to RTP: healing of laminoplasty on CT scan, MRI to document adequate space available for the cord, cervical flexion/extension radiographs with no instability, and return of pain-free ROM and strength.

While ACDF has been a highly effective treatment in athletes, even in the best hands, pseudarthrosis can occur. There is no consensus as to how to approach a nonunion after ACDF in the professional athlete. Some experts feel that the risk after contact is too high, while others believe that pseudarthrosis after a one-level cervical fusion does not impart additional risk to the spinal cord. One important consideration is the presence of a stable “fibrous union” in which there is documented stability at the index segment without full bony bridging. This finding contrasts with an unstable pseudarthrosis which can manifest as screw loosening or breakage, local kyphosis, or spondylolisthesis. Our opinion is that RTP protocols should differ depending on sport played, physical examination findings, nature of the nonunion, and full informed consent. It is reasonable that a stable “fibrous union” may be compatible with repetitive contact activities, while an unstable cervical pseudarthrosis is not. More data is required to address this controversy.

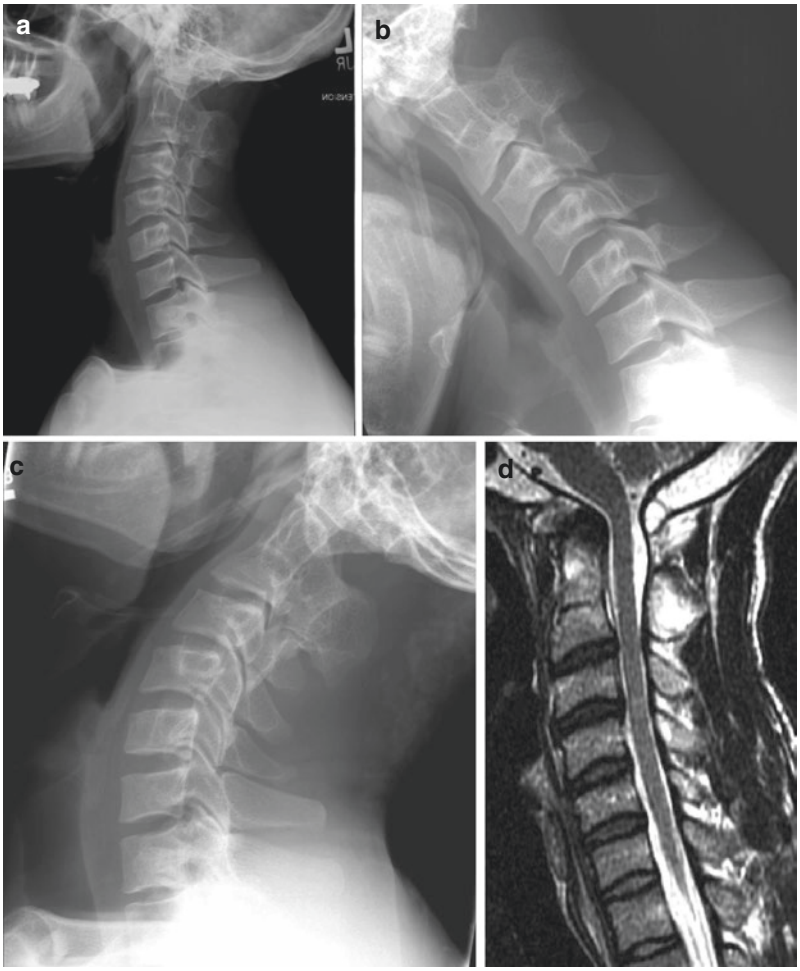
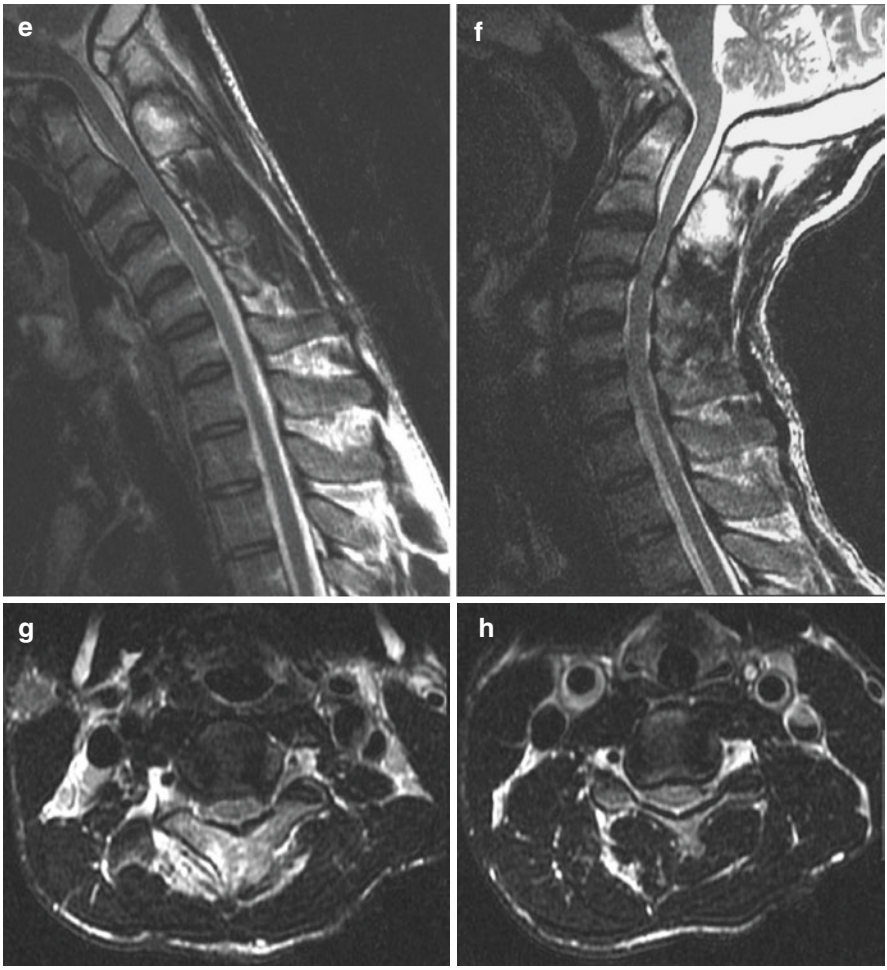


Fig. 12.1 This is a case presentation of a 30-year-old professional basketball player. He presented to our clinic after an in-game hyperextension injury. He had experienced an episode of transient paralysis which lasted around 5–7 min. After ICU monitoring, he experienced a full return of motor strength. He presented to our clinic, because other providers had told him he would no longer be able to participate in contact sports. Panels (**a**–**c**) demonstrate standard upright lateral and flexion/extension cervical radiographs. No dynamic instability is identified. Even on this imaging, one can appreciate the degree of congenital stenosis. Panels (**d**–**f**) show T2-weighted sagittal MRI cuts in both neutral, flexion, and extension. The extension radiograph shows that the stenosis decreases below the C5-6 interspace. This is important in selecting the levels needed to be decompressed. Panels (**g**–**j**) show axial T2-weighted MRI images at C2-3, C3-4, C4-5, and C5-6, respectively. They demonstrate severe congenital stenosis. Panels (**k**–**m**) are sagittal CT c-spine images demonstrating an assimilation of the C1 and C2 arch. After an extensive discussion, we recommended a partial laminectomy of C2 and a C3-5 laminoplasty. We felt that this was the smallest surgery that could provide a complete decompression. It also allowed him the motion needed to return to play. Panel (**n**) shows the postoperative lateral radiograph. The player was able to return to play after a CT scan demonstrated healing of the laminoplasty fracture site and a MRI demonstrated adequate space available for the cord. In addition, he had to have no neurologic symptoms, full range of motion, and no pain. He was ultimately successful in his return to the NBA and played in multiple subsequent seasons



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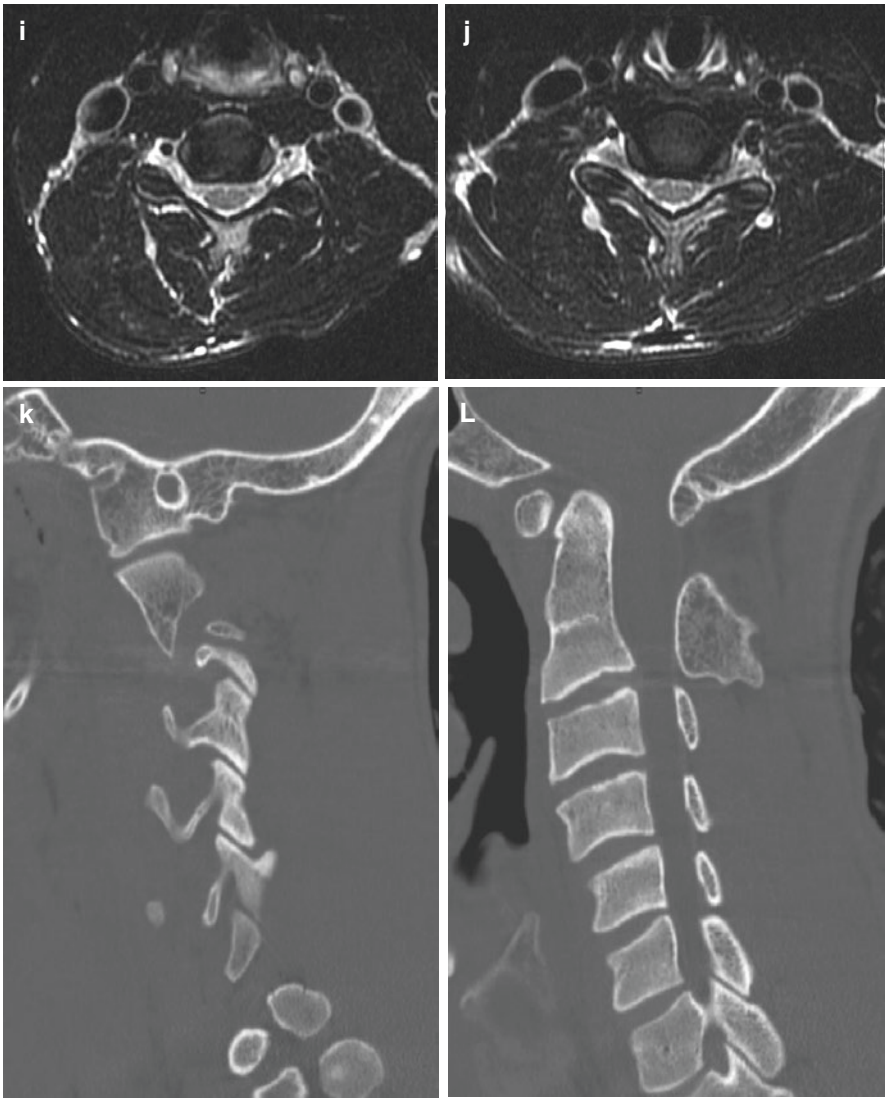

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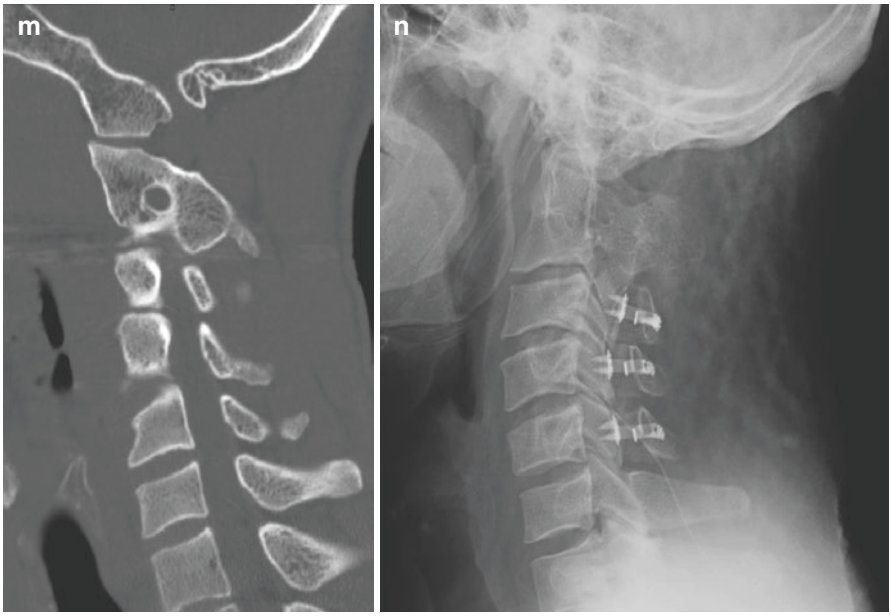

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Expert Opinion

There are many considerations that deserve attention when making decisions regarding cervical stenosis for an elite athlete's career. While many of the treatment principles from the general population can be applied to the elite athlete, the unique demands of elite athletes require an individualized approach. Clinical history, physical exam, imaging characteristics, and sport played are the four critical factors to consider when providing recommendation to the elite athlete with CS. While each situation is unique, our experience suggests the following: absolute canal diameter less than 8 mm and/or evidence of functional stenosis should not return to play without a surgical intervention to decrease spinal cord injury risk. However, persistent myelomalacia alone should not preclude an athlete from returning to collision sports, if there is adequate area for the spinal cord and a normal, symptom-free, neurologic exam. When providing surgical counseling to elite athletes, good evidence exists that RTP should be the expectation for a one-level anterior cervical operation; a two-level operation is not an absolute contraindication to RTP. RTP protocols should differ depending on sport played, physical examination findings, and full informed consent. Ultimately, the athlete must make a well-informed decision based on their tolerance of risk and individual presentation.

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Chapter 13
Cervical Spine Injuries in Athletes

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Introduction

The proportion of cervical spinal cord injuries (SCIs) has increased in recent decades relative to injuries at the other vertebral levels [1]. Sports-related SCIs have decreased since the 1970s, and while the average age of athletes sustaining such injuries has increased slightly since that time, most athletes who suffer cervical spine injuries are younger than 30 years of age [2, 3].

While cervical SCIs are rare, they have been reported in noncontact sports and numerous recreational activities where one would not typically expect to see this injury pattern. Despite the rarity of cervical spine injury, 8.2% of SCIs in the USA from 2015 to 2017 were sustained during athletic activity, and 2.4% of athletic hospitalizations have been associated with SCIs [4, 5]. The potentially devastating consequences of a cervical spine injury necessitate that all athletes suspected of having a cervical spine injury be treated with spinal precautions and taken to the nearest SCI or trauma center.

Epidemiology

Sports most commonly associated with cervical spine injuries include American football, rugby, wrestling, and gymnastics. The mechanism of injury can vary widely depending on the sport or type of action leading to cervical spine injury

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[2, 3]. In football, cervical spine injuries are the most common injury to the axial skeleton, occurring far more frequently than lower back injuries or rib fractures. Fortunately, fewer than 1% of cervical spine injuries result in a fracture or SCI. Serious SCIs are relatively rare but associated with potentially devastating consequences and long recovery times [6]. In the USA, 327 SCIs sustained from American football were reported between 1977 and 2012. The vast majority (81%) of these injuries occurred in high school athletes. The number of football-related cervical spine injuries has decreased since its peak in the 1960s when equipment changes led many players to use the spear tackling technique, which involves leading with the crown of the head [7]. Due to the highly dangerous nature of this technique for both offensive and defensive players, it was banned in 1976. Following this rule change, the number of serious cervical spine injuries in football players has greatly decreased [8].

Schroeder et al. report that cervical spinal disorder, pathology, or injury was diagnosed in 4.8% of athletes who attended the NFL Combine from 2003 to 2011 [9]. Of the 2965 athletes who attended the combine during this time period, the most common diagnoses were cervical disk herniation, spondylosis, and stenosis (congenital and/or acquired).

A Canadian study of ice hockey players reported 311 spinal injuries and SCIs between 1943 and 2005. Cervical injuries accounted for 82.8% of these. This high percentage of cervical injuries and increased prevalence in males younger than 30 years of age is consistent in other regions [3, 7, 10]. The most common mechanism of injury was impact with the boards (64.8%) which is most often the result of being checked from behind (35%) [3]. Since 2001 there has been a dramatic (69%) decrease in the incidence of cervical spinal injuries in ice hockey. This is likely a result of rule changes which ban checking from behind, as well as improved player and coach education [3].

Rugby is another sport with a high incidence of cervical spine injuries; however, many of these injuries occur outside of structured athletic events, occurring more commonly during recreational matches with nonprofessional athletes. Similarly, cervical spine injuries that result from diving most often occur during recreational activity rather than during swimming and diving meets [11–14]. Cycling is another activity in which cervical spine injuries are more common during recreational outings than during competitive events [15].

On-Site Management

Extreme caution must be exercised by physicians in the initial evaluation of a suspected cervical spine injury. Tenderness, pain, and decreased range of motion are red flags that warrant a full neurologic examination in athletes who have sustained trauma to the cervical spine region. Subsequently, if a structural or neurologic injury (such as fracture or SCI) is suspected, specific precautions should be taken. The basics of trauma response including airway, breathing, and circulation

(ABC) management should be performed first. The athlete's cervical spine should be immobilized with a rigid cervical collar, and the athlete should be placed on a rigid backboard and transported to the nearest SCI or trauma center for further examination. The potentially catastrophic consequences of SCI necessitate the use of these precautions and outweigh potential risks of using a rigid backboard [16]. American football represents a unique challenge in that the athlete wears a helmet with a face mask. In this situation, the face mask should be removed to allow access to the airway; however, the rest of the helmet should remain in place in order to limit unnecessary movement of the head and neck. The helmet can be removed once the athlete is in a controlled environment with multiple healthcare professionals [8, 17–19].

Cervical Spine Fractures and Dislocations

Upper Cervical Spine Fractures

Upper cervical spine fractures—defined as injuries the occiput to C2—are uncommon even in sports with the highest incidence and prevalence of cervical spine injury, such as mountain biking [15]. A study by Dodwell et al. reported that 17.7% of cervical injuries observed in mountain bikers affected the upper cervical spine, compared to another study by Boden et al. who reported this to be true in 4.6% in a cohort of American football players [15, 20].

Injuries to the upper cervical region are usually associated with the head on collisions when the neck is slightly flexed. Mechanisms for this injury pattern include impact with other players or the playing surface or any other high-energy impact to the crown of the head during slight neck flexion [18, 20].

Highly unstable upper cervical injuries (usually atlanto-occipital or atlanto-axial) must be treated with surgical stabilization. Most single-level injuries without ligament damage, however, can be treated nonoperatively. For example, many odontoid fractures can be treated nonsurgically with a hard cervical collar [21]. On the other hand, if an odontoid fracture or any high cervical injury is associated with neurological symptoms or is displaced with risk for nonunion, surgical intervention should be undertaken.

Injury to the Posterior Tension Band

The posterior tension band, or posterior capsuloligamentous complex, is composed of the supraspinous ligament, interspinous ligaments, ligamentum flavum, and articular facet capsules. Injuries to this region are associated with a flexion-distraction mechanism and can be highly unstable. Nearly all athletes diagnosed with injuries to the posterior tension band require surgical stabilization [22].

The most severe injury associated with disruption of the posterior tension band is bilateral facet dislocation. MacLean et al. found SCIs to be significantly more common in rugby players with bilateral facet dislocations than with other types of cervical spine injury [22]. The study reported significant spinal cord injury in 90% of rugby players with bilateral facet dislocation compared to only 58% of players with other types of cervical spine injury [22].

Injury to the Anterior Tension Band

The anterior tension band of the spine consists of the anterior longitudinal ligament, posterior longitudinal ligament, intervertebral disks, and vertebral bodies [18]. Injuries affecting these structures are generally caused by an applied force causing neck extension and intervertebral distraction. This results in failure of the anterior osteoligamentous complex and, often, disruption of the outer annulus of the intervertebral disk [23]. These are considered "extension-distraction"-type injuries.

Nearly all anterior osteoligamentous injuries are unstable; thus, it is recommended that the vast majority of these injuries be treated surgically. It is not uncommon in anterior ligament injuries for the rostral vertebral body to translate posteriorly, causing cord compression and resultant myelopathy or acute SCI [24]. In simple injuries such as cervical sprain, athletes may experience symptoms limited to the neck—such as neck pain, local tenderness, or limited range of motion—which may indicate sub-catastrophic damage to the anterior column [18].

Compression Fractures

Compression fractures result from an axial load applied to a neck that is neutrally aligned or slightly flexed [25]. These injuries demonstrate vertebral body height loss but maintain ligamentous integrity, which confers a low risk for neurologic injury. The height loss can result in focal post-traumatic kyphosis at the level of injury. Treatment of these injuries can usually be accomplished with a hard cervical collar, although exceptions to this treatment plan may be made. For example, injuries associated with other fractures or simultaneous disruption of the posterior tension band may require early surgical intervention [25].

Burst fractures are a subtype of compression fracture in which the axial load is delivered directly perpendicular to the spinal column, leading to structural failure of the vertebral body [18]. These fractures carry an increased risk of neurologic injury compared to other types of compression fractures due to the possibility of displaced retropulsed fragments into the spinal canal which can cause acute SCI [26]. Similar to other compression fractures, burst fractures in young athletes have been shown to

produce kyphosis. These injuries must be followed closely with periodic radiographs in order to determine the need for surgical intervention.

Return to Play Following Cervical Spine Fractures and Dislocations

Ideally determination of return to play following cervical spine fracture should be tailored to both the patient and their sport, with the main priority being the goal of future safety. There is a paucity of literature regarding return to play criteria for athletes with cervical spine injuries. A study by Morganti et al. found no significant consensus regarding return to play even in high-risk sports [27]. Kepler et al. have recommended athletes not return to athletic activity until fracture union is achieved, full painless range of motion is obtained, and normal strength of the surrounding musculature is restored [28]. Athletes who experience persistent pain or neurological changes should not return to athletic activity until resolution of these symptoms.

Athletes who have undergone operative fixation of a cervical spine injury may in some instances return to play. A meta-analysis of athletes who underwent anterior cervical discectomy and fusion (ACDF) reported a high return to play rate of 73.5% after ACDF [29]. This meta-analysis identified ACDF as a successful treatment modality for athletes who experience greater stresses on the cervical spine than the general population, especially those who participate in contact sports. A list of relative and absolute contraindications for athlete return to play following cervical spine injury is illustrated in Table 13.1 [26].

Table 13.1 Return to play criteria for athletes with cervical spine injury

Injury	Contraindication	Return to play criteria
Fracture associated with residual neurologic sequelae	Absolute	None
Fracture in a patient with congenital cervical stenosis	Absolute	None
Injury requiring occipital fusion	Absolute	None
Injury requiring C1-C2 fusion	Absolute	None
Injury requiring fusion of ≥3 vertebral segments	Absolute	None
Spear tackler's spine	Absolute	None
Minimally displaced C1 ring fracture	Relative	Solid union demonstrated on CT; no residual instability on flexion/extension radiographs; complete, painless range of motion of the cervical spine; and pre-injury muscle strength in the neck

(continued)

Table 13.1 (continued)

Injury	Contraindication	Return to play criteria
C2 compression fracture	Relative	Solid union demonstrated on CT; no residual instability on flexion/extension radiographs; complete, painless range of motion of the cervical spine; and pre-injury muscle strength in the neck
Upper cervical spine fracture requiring surgical stabilization (excluding C1-C2 arthrodesis)	Relative	Solid union or arthrodesis demonstrated on CT; no bony elements narrowing the spinal canal; no residual instability on flexion/extension radiographs; complete, painless range of motion of the cervical spine; and pre-injury muscle strength in the neck
Isolated compression fracture of subaxial cervical spine	Relative	Solid union demonstrated on CT; no residual instability on flexion/extension radiographs; complete, painless range of motion of the cervical spine; and pre-injury muscle strength in the neck
Isolated stable burst fracture of subaxial cervical spine	Relative	Solid union demonstrated on CT; no retropulsion of the fracture; no substantial sagittal malalignment (>11° relative to noninjured segments); surgical fusion of <3 segments; no residual instability on flexion/extension radiographs; complete, painless range of motion of the cervical spine; and pre-injury muscle strength in the neck
Fracture disrupting the lateral mass, articular processes, or posterior tension band (excluding spinous or transverse processes) of subaxial cervical spine	Relative	Solid union or arthrodesis demonstrated on CT; no bony fragments in the canal; no substantial sagittal malalignment (>11° relative to noninjured segments); surgical fusion of <3 segments; no residual instability on flexion/extension radiographs; complete, painless range of motion of the cervical spine; and pre-injury muscle strength in the neck
All other upper cervical fractures treated nonoperatively	Relative	Solid union demonstrated on CT; no bony elements narrowing the spinal canal; no residual instability on flexion/extension radiographs; complete, painless range of motion of the cervical spine; and pre-injury muscle strength in the neck
Subaxial spinous process fracture	None	Complete, painless range of motion of the cervical spine and pre-injury muscle strength in the neck
Subaxial transverse process fracture	None	Complete, painless range of motion of the cervical spine and pre-injury muscle strength in the neck

Based on data from Schroeder and Vaccaro [30]

Other Conditions in the Athlete's Cervical Spine

Cervical Spine Stenosis

Cervical spine stenosis occurs when the spinal canal is narrowed, which leads to compression of the thecal sac and increased pressure on the spinal cord [31]. Patients with cervical stenosis are at a higher risk of a neurologic injury with a cervical fracture, so it is critical to have an understanding of this when evaluating patients with a cervical spine fracture. The accepted etiology is repetitive contact stresses on athletes, which leads to degenerative osteophyte formation; ultimately, these osteophytes narrow the spinal canal and eventually lead to acquired stenosis [8]. Stenosis may also be congenital in nature. A narrowed spinal canal diameter of 14 mm or less places athletes at an increased risk of spinal cord from cervical trauma [8, 32].

The best way to characterize and screen for cervical spine stenosis has been a topic of controversy [32]. The Torg ratio—the ratio of the width of the spinal canal to that of the vertebral body (measured on a lateral radiograph)—is one indicator used to screen for spinal stenosis [32, 33]. A ratio less than 0.8 is indicative of spinal stenosis; however, Herzog et al. have shown that since athletes tend to have larger vertebral bodies, 41% of asymptomatic professional football players had a Torg ratio of less than 0.8. This finding severely limits the Torg ratio as a screening tool for stenosis in athletes [32, 34]. Cantu et al., on the other hand, propose that using axial MRI slices to measure cerebrospinal fluid surrounding the spinal cord (known as the "functional reserve") or screening for the presence of cord deformation is a valid way to screen for stenosis [35]. Unfortunately, performing an MRI on every athlete as a screening tool is not a cost-effective approach and leads to unnecessary concerns from incidental but clinically insignificant imaging findings [32].

One of the main concerns for athletes with cervical spine stenosis is the risk of transient quadriparesis or cervical cord neurapraxia (CCN). This occurs in a wide range of sports including football, rugby, hockey, and basketball [36]. Symptoms include paresthesias (i.e., burning, numbness, and tingling)—which can occur in the ipsilateral leg, bilateral legs, or all extremities—and motor symptoms ranging from paresis to plegia [37]. Torg et al. compared athletes with a previous episode of CCN with those without CCN and found that athletes with CCN had significantly smaller spinal canals and lower Torg ratios [33]. Bailes et al. and numerous others have reported cervical stenosis in all athletes with prior CCN [38]. The literature suggests previous episode of CCN may increase risk of recurrence; however, this finding seems to correlate with the type of athletic activity and unique spine anatomy involved. Torg et al. showed no recurrent episodes in athletes with prior CCN that returned to play, while Bailes et al. found this to be true in three of four athletes studied [38, 39]. Furthermore, Brigham et al. observed athletes who returned to play after undergoing ACDF for stenosis and found that two of these participants experienced a second episode of CCN [40].

For athletes with congenital or acquired cervical spine stenosis, the safety of play is of paramount concern. Schroeder et al. observed that zero of ten athletes drafted to the NFL with absolute spine stenosis sustained a spinal cord injury; however this sample size is extremely low [9]. Aebli et al. compared the size of spinal canals in 53 patients who sustained a cervical spine injury and associated spinal cord injury, with that of 184 patients with a cervical spine injury and no spinal cord injury, and discovered that patients with a SCI had significantly smaller spinal canals [41]. Furthermore, the authors demonstrated a midsagittal intervertebral disk space diameter of ≤8 mm as a strong predictor of SCI after minor cervical spine trauma (positive predictive value of 84%, likelihood ratio of 15.6) [41]. Regarding return to play, it is imperative to keep in mind that athletes, even those with asymptomatic cervical spine stenosis, are at an increased risk of sustaining a spinal cord injury and, thus, must be counseled on the dangers of participation. We strongly recommend that any athlete with absolute cervical spine stenosis defined by a SAC of <10 mm, and either neurologic deficit or sensory disturbance following any trauma, be discouraged from returning to play. Based on the substantial rate of recurrence of symptoms observed, we recommend against return to play for athletes who experience CCN.

Spear Tackler's Spine

Spear tackler's spine is a condition often diagnosed in athletes who play American football, specifically players who tackle with the top of the head, applying repetitive axial loads to the semi-flexed cervical spine during high-impact collisions [32]. Torg et al. provided four key radiographic findings that characterize this diagnosis: (1) stenosis of the cervical canal, (2) persistent straightening or reversal of normal cervical lordosis, (3) concomitant radiographic evidence of previous cervical spine abnormalities, and (4) history of using the spear tackling technique [42].

Although American football has banned the use of spear tackling, spear tackler's spine is a serious condition that puts athletes at significant risk for catastrophic head and neck injuries—risk physicians should be aware of [42]. Common signs and symptoms include stiffness or tenderness of the neck, muscle spasms, numbness, and weakness or paralysis of the shoulder, arm, or hand [43]. As a result, athletes are encouraged to keep their heads up and to the side of the ball carrier and lead the tackle with the chest, shoulders, or arms [44]. Ultimately, it is recommended that athletes diagnosed with spear tackler's spine be excluded from participation in athletic activities due to the increased risk of severe spinal cord injury [32, 42].

Conclusion

Cervical spine fractures in athletes can be devastating injuries, and patients with underlying stenosis are at an increased risk of a spinal cord injury. Early recognition and treatment are paramount in these injuries. Fortunately, the number of

catastrophic injuries has decreased over the last few decades. Return to play after a cervical spine injury is determined on a player-by-player basis. This decision is dependent upon the nature of the injury, the patient's underlying spinal anatomy, the patient's neurological symptoms and examination, and the effects of surgical or nonsurgical treatment on the stability and mobility of the spine.

Expert Opinion

1. Athletes with the highest risk of cervical spine injuries include American football, rugby, wrestling, and gymnastics.
2. On the field evaluation should begin by identifying whether the player in question demonstrates potential signs of an unstable spine injury – including tenderness, pain, and decreased range of motion, all of which are red flags for this particular injury pattern.
3. For American football players, the player should be removed from the playing field using cervical spine precautions. In our experience, equipment managers are vital to safe and efficient removal of the helmet and other athletic gear. Team physicians should keep this in mind because the inability to remove a helmet safely can lead to catastrophic outcomes in cervical spine injuries.
4. We recommend removing the face mask and performing the trauma ABCs as necessary. Definitive removal of the helmet is best carried out in a safe and controlled environment with a multidisciplinary care team.
5. After removal of necessary equipment appropriate radiographic workup should be performed following standardized cervical trauma protocols.
6. Cervical spine injuries in the athlete may be subtle, and scrutiny of imaging is paramount.
7. Return to play after a cervical spine injury is determined on a player-by-player basis. Basic tenants include full painless range of motion, normal muscular strength, normal neurologic examination, and evidence of fracture union.

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Part III
Injuries to the Thoracic and Lumbar Spine

Chapter 14
Evaluation of Athletes with Back or Leg Pain

Robert L. Brochin, Zoe B. Cheung, and Andrew C. Hecht

Introduction

Low back pain (LBP) is a ubiquitous health problem in the general population that has detrimental effects on functional status, ability to work, and quality of life [1, 2]. Furthermore, it constitutes a significant socioeconomic burden and represents the fifth most common reason for physician visits in the United States with annual costs estimated to range from $84 to $624 billion [3, 4]. In epidemiologic studies, the reported prevalence rate of LBP has ranged widely from 58% to as high as 85% [2, 5, 6].

The relationship between LBP and physical activity has been well studied [7, 8]. While increased physical activity may be protective against LBP, some evidence shows it may also be a potential risk factor. For example, frequent lifting, bending, and twisting have been found to be risk factors for LBP [9]. Most athletic activities involve such movements, with the amount of strain on the back varying depending on the type of sport, level of competition, as well as the intensity, duration, and frequency of training. Therefore, it is not unexpected that athletes may be at higher risk for developing LBP. However, it remains unclear if the incidence of LBP is higher in athletes than the general population.

The reported prevalence of LBP in athletes ranges widely and is not the same across all sports. In a study of intercollegiate athletes across 17 varsity sports over a 10-year period, the incidence of LBP was 7% [10]. The highest incidence of LBP was found in football and gymnastics – 17% and 11%, respectively [10]. Acute muscle strain was also found to be the most frequent cause of LBP [10]. A study of LBP among retired wrestlers and heavyweight lifters found a lifetime prevalence of LBP of 59% among wrestlers compared with 23% among heavyweight lifters [11]. Sports that require repetitive hyperextension may be associated with a high incidence

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of LBP [12]. For example, the incidence of spondylolysis in competitive gymnasts and divers is 32% and 63%, respectively [13]. Aside from hyperextension, sports requiring repetitive trunk rotation have also been associated with high rates of LBP, for example, professional golfers have been reported to have a 29% incidence of LBP [14]. In comparison, runners have a comparatively low incidence of LBP, ranging from 1% to 22% [13].

Similar to the general population, LBP in athletes can have detrimental effects on functional status and quality of life. LBP in athletes can lead to impaired performance, lost playing time, as well as high treatment costs. In the evaluation of athletes with LBP, it is important to consider not only psychosocial factors but also mechanical factors that can be modified by alterations in technique and training. Furthermore, the drive and desire to return to play despite pain should not be overlooked when evaluating and treating athletes with LBP.

Low Back Pain with Associated Leg Pain

In both athletes and the general population, LBP often presents with associated radicular leg pain. The prevalence of LBP with associated leg pain in the general population has been well studied and ranges from 1% to 43% [15]. However, few studies have reported on the prevalence of LBP with associated leg pain specifically in athletes. The presence of radicular leg pain is suggestive of nerve root involvement from direct mechanical compression or chemical irritation. This should be taken into consideration in the evaluation and treatment of athletes with LBP as well as associated leg pain.

Evaluation

History

The first step in the evaluation of LBP in an athlete is a thorough history. The onset of symptoms must be elucidated, including the mechanism of injury during the inciting event. Specifically, the position of the spine, as well as the direction and amount of force applied to the spine at the time of injury, should be noted. Other pertinent considerations include the location, rate of onset, duration, and quality of LBP. Additionally, any movements that improve or exacerbate the pain should be noted. In athletes, it is also important to explore and understand the types of sports played, as well as the duration, frequency, and intensity of training and competition. Any history of LBP or back injuries must be considered, as well as any previous treatments by other health-care providers, athletic trainers, physical therapists, or chiropractors. A family history of LBP should also be investigated. Finally, a thorough review of systems should be conducted to assess for systemic problems

Table 14.1 Red flag symptoms in athletes with low back pain

Symptoms	Rule out
Fever, malaise, unintentional weight loss	Infection
Fever, malaise, unintentional weight loss, night pain, history of cancer	Cancer
Neurologic deficits, bowel or bladder dysfunction	Cauda equina
Abnormal behavior, mood swings	Drug or steroid abuse

leading to LBP. Red flag symptoms should also be solicited, such as fever, malaise, unintentional weight loss, neurologic deficits, bladder or bowel incontinence, urinary retention, night pain, history of cancer, long-term steroid use, and parenteral drug use (Table 14.1).

Obtaining a thorough and accurate history is essential in order to narrow the differential diagnosis and guide toward a correct diagnosis. The location of pain is an important distinguishing feature. LBP without radiating leg pain is often nonspecific and includes etiologies such as mechanical back pain, facet joint pain, muscle strain, and muscle spasm. LBP that localizes to a single midline point suggests an etiology involving a single motion segment, whereas diffuse LBP without leg pain more likely involves multiple motion segments and often suggests muscular pain. In contrast, LBP with radiating leg pain is indicative of nerve compression or irritation. The onset of pain is also indicative of the etiology of LBP. Sudden onset of LBP suggests an acute etiology, such as fracture or disc herniation. In contrast, an insidious onset is more suggestive of disc degeneration, spondylolysis, or stress fracture. Aggravating and alleviating factors also provide clues toward the correct diagnosis. Disc-related LBP is often aggravated by forward bending, whereas LBP arising from the posterior elements (e.g., facet joints, pars interarticularis) is worsened with lumbar extension and alleviated by forward flexion.

A positive family history can be suggestive of inflammatory rheumatologic disorders, such as ankylosing spondylitis and reactive spondyloarthropathies. Constitutional symptoms that are noted on review of systems may be indicative of systemic processes, such as connective tissue disorders (e.g., systemic lupus erythematosus) and endocrine disorders (e.g., hyperparathyroidism). The presence of red flag symptoms necessitates immediate further workup in order to evaluate for potentially serious problems such as cauda equina syndrome, infection (e.g., epidural abscess, vertebral osteomyelitis, discitis), and malignancy (e.g., multiple myeloma, lymphoma, metastases).

Physical Examination

The physical examination of an athlete with LBP begins with the inspection of static posture to assess for the presence of scoliosis, thoracic hyperkyphosis, or loss of lumbar lordosis. Posterior asymmetry in the shoulders, trunk, or pelvis should be noted with the athlete facing away from the clinician. Lateral inspection should then

be performed to assess spinal curvature, including normal cervical lordosis, thoracic kyphosis, and lumbar lordosis. Next, palpation of the entire spine should be performed. The spinous processes and paraspinal muscles should be systematically palpated for tenderness. Point tenderness over a single spinous process suggests an etiology involving a single motion segment, whereas multilevel tenderness over the paraspinal muscles suggests muscle strain or spasm.

Range of motion in the lumbar spine should be evaluated. Flexion, extension, and rotation through the lumbar spine should be observed while ensuring that the pelvis remains in a fixed position in order to isolate lumbar spine motion. While assessing lumbar spine range of motion, it is important to distinguish between limited range of motion that is painless from limited range of motion that is painful. Painless limitation in range of motion suggests loss of spinal flexibility, whereas painful limitation in motion suggests that an underlying pain generator is being loaded. Painful forward bending is indicative of disc-related pain. Painful lumbar extension is more suggestive of pain arising from the posterior elements.

Provocative tests for lumbar spine, sacroiliac joint, and hip pathology should be performed. The straight leg raise test should be performed in the supine position. Reproduction of leg pain and/or paresthesia at 30–70° of hip flexion suggests tension on the lumbosacral nerve roots. The contralateral straight leg raise test can also be performed. In addition, a modification of the straight leg raise that can be performed is the Bragaad's test, in which the straight leg raise is aggravated by forced ankle dorsiflexion [16]. The femoral nerve stretch test (FNST) should be performed in the prone position by passive extending the hip, while the knee is fully passively flexed and is positive with radiating pain to the anterior thigh [17]. A positive FNST is indicative of nerve root pain from thigh high lumbar roots (L1-L3). A single-legged hyperextension test can be performed to evaluate for a pain generator in the posterior elements. LBP arising from the sacroiliac joint can be elicited with the flexion, abduction, external rotation (FABER), compression, and Gaenslen's tests. LBP generated from hip pathology can be assessed with the flexion, adduction, internal rotation (FADIR) and Thomas and Ober tests (Fig. 14.1) [18].

A thorough neurologic examination of both the upper and lower extremities must be performed, including a detailed assessment of motor strength, sensation, and reflexes. Neurologic deficits in specific dermatomes can guide the clinician toward correctly identifying the specific level of injury. Pathological signs of an upper motor neuron lesion include the Hoffman sign, Babinski sign, inverted radial reflex, and more than four beats of sustained clonus (Figs. 14.2 and 14.3). The presence of these pathological signs necessitates further workup with advanced imaging of the cervical, thoracic, or lumbar spine. Lastly, a gait assessment should be performed. A Trendelenburg gait from weakness in the gluteus medius may indicate L5 nerve root pathology. Difficulty with walking on the toes and heels may suggest subtle lower extremity weakness due to lumbosacral nerve root involvement.

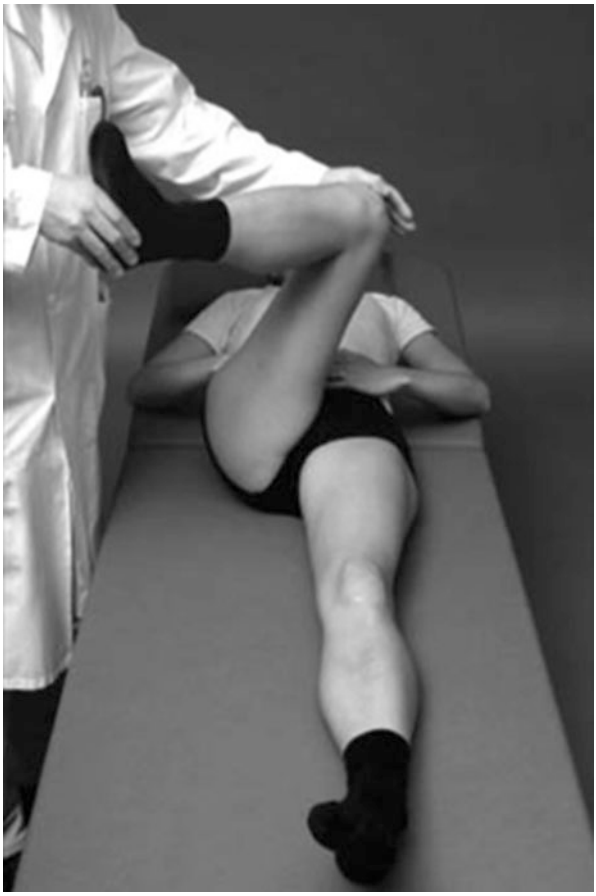

Fig. 14.1 The flexion, adduction, internal rotation (FADIR) test to evaluate low back pain originating from hip pathology. (Reproduced with permission from Sierra et al. [18])

Fig. 14.2 The Hoffmann reflex in which flipping the distal phalanx of the patient's middle finger downward results in spontaneous flexion of the ipsilateral thumb and/or index finger. (Reproduced with permission from Emery [49])

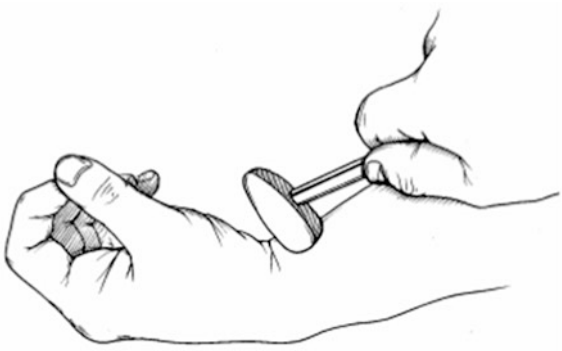

Fig. 14.3 The inverted radial reflex in which tapping the distal brachioradialis tendon results in abnormal finger flexion. (Reproduced with permission from Emery [49])

Laboratory Testing

Routine laboratory testing is typically not necessary in the initial evaluation of LBP in athletes. However, if the history and physical examination are concerning for infection or tumor, then laboratory testing should be performed, including a complete blood cell count, C-reactive protein, and erythrocyte sedimentation rate [19]. Further laboratory testing should be determined as clinically warranted on a case-by-case basis.

Imaging

In an athlete with LBP lasting more than a few weeks in the absence of red flag symptoms, plain radiographs of the lumbar spine should be performed. Although plain radiographs are often low yield with no remarkable findings, they can show spinal alignment abnormalities, such as thoracic hyperkyphosis, loss of lumbar lordosis, and spondylolisthesis. In addition, radiographs can also show evidence of acute or chronic fractures. Flexion and extension views are recommended and can demonstrate dynamic instability or spondylolysis that is not readily apparent on neutral standing radiographs. Oblique views may be helpful in visualizing the pars interarticularis and assessing for spondylolysis. However, recent studies have found no improvement in the sensitivity and specificity of diagnosing spondylolysis with oblique views [20]. Therefore, the additional cost and radiation exposure associated with oblique views may outweigh the lack of associated diagnostic benefit.

If LBP persists beyond 6 weeks without improvement after initial conservative treatment, advanced imaging should be obtained. The initial advanced imaging of choice is magnetic resonance imaging (MRI). MRI is useful in evaluating the intervertebral discs, neural elements, as well as soft tissues. MRI can also provide information regarding stress reactions, occult fractures, or spondylolysis. A CT scan can also be helpful in defining bony anatomy when plain radiographs do not provide

sufficient detail. However, given the significant amount of radiation exposure associated with CT, it should be reserved for only those cases in which MRI is contraindicated or inconclusive in establishing a diagnosis.

In cases of spondylolysis, which will be discussed in more depth later in this chapter, CT has been considered the gold standard for detecting the bony defect. However, emerging evidence suggests that MRI is comparable to CT in terms of accuracy in detecting early stress reactions of the pars interarticularis without a fracture [21]. Single-photon emission computed tomography (SPECT) has been shown to be superior to plain radiographs and bone scan in detecting spondylolysis [21]. However, similar to CT, SPECT is limited by high radiation dose [21]. Furthermore, SPECT has been associated with high rates of false-positive and false-negative results [21].

Compared to spondylolysis, the detection of a stress reaction prior to fracture is more difficult. Stress reactions are not detectable on plain radiographs or standard CT. MRI or bone scan can be helpful in detecting stress reactions, but SPECT is the most sensitive test for diagnosing stress reactions [22, 23]. Early detection of a stress reaction is important in order to implement early treatment and prevent progression to a frank stress fracture.

An important consideration in the discussion of advanced imaging for LBP in athletes is that degenerative changes in the lumbar spine may be present in asymptomatic athletes. That is, the presence of both LBP and degenerative changes in the lumbar spine does not imply causality. For example, in a study of former national level athletes, the intensity of LBP symptoms was not significantly correlated with degenerative findings on MRI [24]. Multiple other studies have demonstrated a higher rate of degenerative changes on MRI in athletes compared to non-athletes, but no correlation between these MRI changes and the prevalence of LBP [25–27]. Therefore, care must be taken in establishing a causal relationship between clinical symptoms and imaging findings when evaluating athletes with LBP.

Differential Diagnosis

This section will focus on the most common etiologies of LBP with and without leg pain in athletes. Back pain is the most common reason for competitive athletes to lose playing time [28], and as such, a detailed knowledge of the causes of LBP is crucial for clinicians who take care of athletes. While the focus will remain on spinal conditions, it is important that the clinician also considers involvement of other systems and keeps their differential diagnosis broad initially. Among orthopedic etiologies of LBP, pain resulting from hip pathology may be referred to the back. The clinician must perform a thorough history and physical examination in order to rule out intra-articular hip issues (e.g., labral tears, femoroacetabular impingement) and extra-articular issues (e.g., trochanteric bursitis, muscle tears). Among non-orthopedic etiologies, the clinician should consider intra- and retropelvic conditions, such as those of the genitourinary system including renal and ovarian

pathologies that may cause LBP with or without radiating leg pain. A full discussion of these conditions is out of the scope of this text; it is important for the clinician to keep a broad differential initially and consider further non-spine-related workup when indicated.

Muscle and Ligamentous Injury

Thoracolumbar musculo-ligamentous injuries are a common cause of LBP in athletes [28]. These occur most commonly in athletes participating in high speed and/or full contact sports when the trunk is exposed to significant rotational and flexion/extension forces. Athletes will typically not experience immediate symptoms. The onset of pain is often delayed 12–24 h, which has been attributed to the timing of the inflammatory cascade [17]. Patients typically complain of LBP that can be either diffuse or localized without radiation. On physical examination, there may be paravertebral spasm, diminished thoracolumbar range of motion, and pain with resisted rotation or flexion/extension. Since these injuries do not involve the spine itself, there should be no neurological deficits or associated radicular symptoms.

In general, musculo-ligamentous injury is a clinical diagnosis, and advanced imaging is not necessary. Radiographic findings are typically normal immediately after injury. If initial radiographs are normal and pain persists, flexion and extension views should be obtained after 2–3 weeks to rule out dynamic ligamentous instability that may not have been noticeable on static radiographs [17]. If there remains further concern, CT and/or MRI can be obtained to rule out occult fracture and ligamentous injury, respectively.

Treatment of musculo-ligamentous injury should focus on managing pain and inflammation. Modalities such as ice for inflammation reduction and heat for muscle relaxation may be effective initially. Nonsteroidal anti-inflammatory drugs (NSAIDs) and muscle relaxants may be used as adjuncts as necessary for symptomatic management. Rehabilitation should begin when pain is tolerable and focus on range of motion and strengthening, with the goal of returning to pre-injury level of function. Bracing has been noted to possibly be helpful in the acute setting but should not be used as a long-term treatment. If pain persists beyond 3 weeks despite conservative treatment, advanced imaging as discussed above should be pursued [29].

Degenerative Disc Disease

Lumbar degenerative disc disease (DDD) refers to the process of disc space narrowing secondary to loss of disc hydration leading to facet arthropathy from abnormal loading of the facet joints. This has been described in a three-phase process [30]. In the initial phase, pain is produced from synovitis of the facet joints or annular tears

of the intervertebral disc. This is followed by a second phase of segmental instability due to diminished functionality of the facet capsule and annulus. In the final phase, the facet and discovertebral joint restabilize [31].

Although historically, it was thought that increased loading such as that encountered by athletes was a predisposing factor for degenerative disc disease, several studies have also demonstrated a strong genetic component [32, 33]. However, there remains evidence that elite athletes have not only a higher incidence of early lumbar degenerative changes but also more severe changes when compared to the general population [34, 35].

The presentation of lumbar degenerative disc disease is nonspecific. In general, discogenic pain will occur with activities that load the intervertebral disc. Most typically, an athlete will complain of LBP that is worse with movements that stress the low back. On physical examination, increased pain with flexion and relief with extension of the lumbar spine is often seen.

Standard anteroposterior (AP) and lateral radiographs of the lumbar spine may demonstrate disc space narrowing, subchondral cysts, facet degeneration, and osteophytes. In cases of isolated degenerative disc disease, flexion and extension views will not yield any further information. MRI can confirm the diagnosis and will demonstrate loss of disc signal intensity on T2-weighted images, as well as changes in vertebral body end plate and bone marrow changes as described by the Modic classification, which describes the severity of vertebral body end plate changes [36]. Table 14.2 summarizes the Modic classification; type 1 represents bone marrow edema and inflammation, type 2 represents the conversion of normal red hematopoietic bone marrow into yellow fatty marrow secondary to marrow ischemia, and type 3 represents subchondral bony sclerosis.

The treatment of degenerative disc disease in athletes is primarily conservative. Symptoms can initially be managed with NSAIDs and refraining from play. It is important to reiterate to patients that this is generally a self-limiting process. Although high quality literature is sparse, it appears physical therapy that focuses on core strengthening is effective in treating LBP in athletes [37]. Other nonsurgical options include epidural spinal injections and bracing, neither of which have been conclusively shown to be successful interventions.

Surgery for degenerative disc disease is rarely indicated. Strict surgical indications have been proposed and include mechanical LBP associated with a single-

Table 14.2 Modic classification of signal changes on MRI in the vertebral body

Type	MRI	Vertebral body changes
I	T1-weighted	Decreased signal
	T2-weighted	Increased signal
II	T1-weighted	Increased signal
	T2-weighted	Increased signal
III	T1-weighted	Decreased signal
	T2-weighted	Decreased signal

Reproduced with permission from Hsu and Jenkins [36]

level degenerative disc on imaging, physical examination findings of midline tenderness that corresponds to the diseased level, and failure of 6 months of nonsurgical treatment [36]. Discectomy with spinal fusion is the standard treatment when surgery is indicated, but as in the general population, results are less reliable when compared to surgery performed for radicular symptoms [31].

Disc Herniation

Lumbar disc herniation results from annular injury caused by axial loading and rotation of the flexed lumbar spine that allows nuclear material to escape into the epidural space. While disc herniation is associated with trauma, its overall incidence in athletes is unknown, although some authors note it is more common in obese athletes who are at increased risk for premature disc degeneration [17]. Herniated nuclear material irritates adjacent nerve roots through both direct mechanical compression and chemical inflammation [36].

The presentation of a herniated disc is typically sudden onset lower extremity radicular symptoms with or without LBP. Disc herniation occurs most commonly at levels L4-L5 and L5-S1, constituting up to 95% of disc herniations [38]. Athletes aged 20–35 years old are most likely to have a symptomatic disc herniation [39]. Physical examination findings will correspond with the nerve root affected by the disc herniation and will typically be unilateral. The L5 nerve root is most commonly affected in an L4-L5 disc herniation. Physical examination will reveal weakness of ankle dorsiflexion and great toe extension, and sensory changes over the lateral leg and dorsum of the foot. The S1 root is most commonly affected in an L5-S1 disc herniation. Physical examination findings demonstrate weakness with foot eversion and plantar flexion and sensory changes over the lateral foot. The most sensitive physical examination finding for lumbar disc herniation is a straight leg raise performed either with the patient supine or sitting. This maneuver stretches the affected nerve root over the herniated disc. A contralateral straight leg raise reproducing symptoms on the affected side is highly specific for disc herniation [40]. Although encountered less commonly, disc herniation between the levels L1 and L4 may be evaluated with femoral nerve stretch testing, which is performed by extending the hip, which will produce anterior thigh radiculopathy. Lumbar disc herniations can cause cauda equina syndrome when the terminal nerve roots of the cauda equina are compressed. Cauda equina syndrome is a complex of symptoms that can variably include bowel and bladder dysfunction, bilateral leg pain, saddle anesthesia, and autonomic nervous system dysfunction. Cauda equina syndrome is a surgical emergency that should be ruled out due to superior outcomes with expedient intervention.

Diagnostic studies for suspected lumbar disc herniation should begin with plain lumbar radiographs to rule out bony pathology. These may demonstrate nonspecific findings. MRI is the imagining modality of choice for disc herniation. Due to a high rate of asymptomatic disc herniations, the level of disc herniation must match physical examination findings if intervention is to be considered.

Initial treatment for symptomatic lumbar disc herniation is conservative. The great majority of both the general population and professional athletes with symptomatic disc herniations have symptomatic resolution with nonsurgical treatment [41]. Nonsurgical treatment typically consists of oral NSAIDs, tapered oral corticosteroids, and physical therapy focusing on core strengthening and flexibility. When initial conservative treatments fail, epidural steroid injections may be performed. If conservative management fails, then should surgery be considered. Surgical options include open both open and microscopic laminotomy with discectomy.

Spondylolysis and Spondylolisthesis

Spondylolysis is a defect in the pars interarticularis believed to be caused by repetitive microtrauma. Repetitive torsion and extension of the spine, such as that experienced in gymnastics, ballet, weight lifting, and football, among other sports, are thought to predispose athletes to spondylolysis [42]. Spondylolysis may be associated with spondylolisthesis, which is the anterolisthesis, or forward slipping, of a vertebral body relative to the subjacent vertebral body. Most cases of spondylolysis occur at L5 (85–95%), followed by L4 (5–15%) [43]. Isthmic spondylolisthesis refers to anterolisthesis of the spondylolytic vertebra in relation to the subjacent vertebra. Therefore, isthmic spondylolisthesis in athletes is most common at L5-S1. The incidence of spondylolysis has been reported as 8% in the general population [44], whereas the incidence in athletes has been variably reported as being consistent with that of the general population in some studies and ranging from 15% to 47% in other studies [36, 45].

Patients with symptomatic spondylolysis typically present with midline LBP that is exacerbated by activity. The most common neurologic symptom is L5 radiculopathy caused by irritation of the L5 nerve root if the degree of spondylolisthesis causes foraminal stenosis. Although rare, a step-off may be appreciated on palpation of the lumbar spinous processes in the setting of severe spondylolisthesis. Concomitant hamstring tightness may be present and cause a characteristic “stiff-legged” gait. Provocative examination maneuvers include exacerbation of pain with lumbar hyperextension and the straight leg raise.

Standing AP and lateral lumbar spine radiographs should be the initial imaging study performed when spondylolysis is suspected. Lateral upright radiographs can demonstrate pars disruption and associated spondylolisthesis if present. Historically it was common practice to obtain right and left oblique radiographs of the lumbar spine in addition to standard AP and lateral views. However, as previously mentioned, studies have not demonstrated a difference in sensitivity and specificity between two- and four-view studies [20]. Spondylolisthesis can be graded according to the Meyerding classification of spondylolisthesis, which is calculated based on the percentage of the overhanging aspect of the superior vertebral body relative to the subjacent vertebral body (Fig. 14.4). Flexion and extension views should also be performed to assess the stability of the spondylolisthesis.

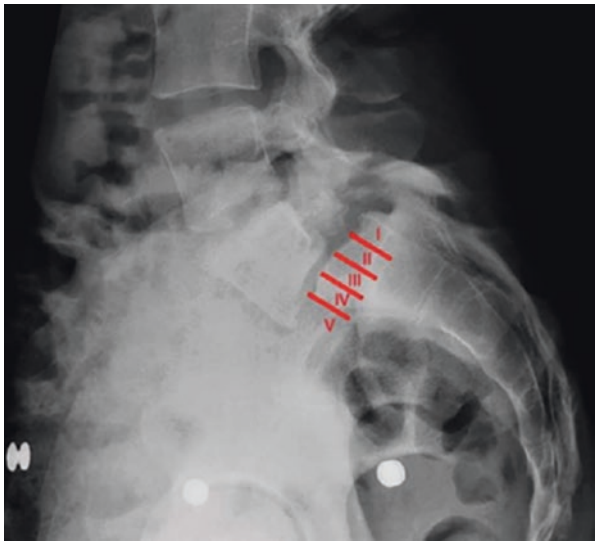

Fig. 14.4 Lateral lumbar radiograph demonstrating the Meyerding classification of spondylolisthesis. The ratio of the length overhanging superior vertebral body to the inferior adjacent body is as follows: Grade I 0–25%, Grade II 25–50%, Grade III 50–75%, Grade IV 75–100%, Grade V >100% (spondyloptosis). (Reproduced with permission from Hsu and Jenkins [36])

When plain radiographs are inconclusive but there is high clinical suspicion for spondylolysis or spondylolisthesis, advanced imaging should be pursued. Some authors advocate thin-sliced MRI as the advanced diagnostic imaging of choice to limit patients' radiation exposure [36]. Others advocate the use of CT to best delineate bony anatomy, as spondylolysis is sometimes missed on MRI [46]. If spondylolysis is diagnosed on MRI, a lumbar spine CT should still be considered in order to have a comparison for future CT scans if needed. As previously discussed, SPECT is also useful in diagnosing impending pars fractures and stress reactions but involves similar risks of radiation exposure as standard CT.

The management of acute spondylolysis is typically conservative, beginning with activity modification, temporary sport cessation, bracing, and physical therapy [47]. Bracing primarily serves to provide symptomatic relief and prevent lumbar extension. NSAIDs can be used to provide pain relief.

Surgical intervention is generally reserved for failure of 6 months of nonsurgical management. Other indications include progressive neurologic symptoms and unstable spondylolisthesis. Surgical options include direct pars fixation if no neurologic symptoms are present. If significant spondylolisthesis is present, then fusion – typically L5-S1 – is necessary. If neurologic symptoms are present, then decompression may need to be performed concomitantly with fusion.

Sacral Stress Fracture

Sacral stress fractures are an overall uncommon cause of LBP in athletes and almost exclusively found in long-distance running athletes, such as marathoners and cross-country runners [28]. Although its prevalence is unknown, sacral stress fracture is

likely the least common etiology of LBP of all the diagnoses presented in this chapter. These fractures are more common in women and have been found to be associated with an increase in impact activity, abnormal menstrual history, dietary deficiencies, and low bone mineral density [48].

The clinical presentation of sacral stress fractures is usually asymmetric low back or gluteal pain that progresses over weeks to months with no history of acute injury [28]. Sacral stress fractures are typically unilateral. Physical examination findings include point tenderness over the affected side of the sacrum/sacroiliac joint, pain with crossing the leg of the affected side (i.e., FABER test), and pain with single leg stance of the affected side.

Imaging is necessary for diagnosis. Plain radiographs are usually negative. MRI has been found to be sensitive for the diagnosis of sacral stress fractures [48]. Treatment consists of rest and protected weight bearing, with gradual return to play when symptoms resolve [29].

Expert Opinion

LBP with or without radicular leg pain is a common condition in athletes with a broad differential. The most common causes of LBP in the athlete remain musculoligamentous injury, DDD, LDH, spondylolysis, spondylolisthesis, and stress fractures. Careful history taking and physical examination are crucial in narrowing the differential diagnosis. Red flag symptoms such as fever, malaise, unintentional weight loss, neurologic deficits, bladder or bowel incontinence, urinary retention, night pain, history of cancer, long-term steroid use, and parenteral drug use should be targeted in the history. Knowledge of appropriate imaging is essential in order to establish the correct diagnosis and appropriate treatment.

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Chapter 15
Lumbar Disk Herniation and Degenerative Disk Disease in the Athlete

Tyler J. Jenkins and Rick C. Sasso

The most frequent reason for missed playing time in competitive athletes is low back pain (LBP), with a prevalence as high as 30% [1, 2]. Lumbar disk herniation (LDH), degenerative disk disease (DDD), and spondylolysis are the most common lumbar conditions that cause symptoms in this patient population [1, 3]. These conditions have been studied extensively in the general population, but generalizing this data to the elite athlete is problematic. The rigorous demands of high-level performance have led athletes and physicians to question the typical management, outcomes, and return to play for lumbar conditions. Although the literature is still evolving, recent data has provided new insight into the management of lumbar conditions in this patient population.

Lumbar Disk Herniation

LDH occurs when the nucleus pulposus ruptures through the outer annulus of the intervertebral (IV) disk. Athletes may be predisposed to LDH due to the rigorous demands of elite sports [2]. After skeletal development, IV disks receive a limited nutrient supply by diffusion through the vertebral vasculature. Irreparable damage accumulates due to the inherent avascularity of the disk. The damaged disk can ultimately herniate with repetitive athletic maneuvers, such as torsion and axial loading. These repetitive maneuvers are required for elite athletic performance and underscore the risk for LDH in athletes.

After herniation, the nucleus pulposus can irritate an adjacent nerve root, producing the symptoms associated with LDH. Two intertwined pathways drive nerve root

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irritation: chemical inflammation and mechanical compression. The inflammatory cascade leads to a priming of the nerve root and secondary hypersensitivity from mechanical compression, which causes local ischemia and inflammation, escalating the cascade. LDH should be considered in any athlete presenting for the evaluation of radiating leg and/or back pain.

Clinical Presentation

LDH results in dermatomal radicular pain associated with paresthesia and weakness in the lower extremities. The pattern of pain experienced by the patient depends on the level and location of the herniation (Table 15.1). Up to 95% of LDHs occur at the L4–L5 and L5-S1 levels [4]. Commonly, axial back pain and sclerotomal pain also are present. These symptoms of low back, buttock, and posterior thigh pain occur secondary to the irritation of local mesodermal tissue, such as muscle and ligaments. Age can provide a clue for diagnosis, because athletes aged 20–35 years have the highest risk of symptomatic LDH [5].

More specific signs for LDH include dermatomal distribution of symptoms, the predominance of leg pain over back pain, reduced reflexes, and pain that increases with the Valsalva maneuver. A positive ipsilateral straight leg raise is sensitive but not specific for LDH, whereas a positive contralateral straight leg raise test is more specific but less sensitive. Alternatively, the femoral nerve stretch test may be used for herniations affecting the L1–L4 nerve roots. A reproduction of anterior thigh radiculopathy is considered a positive test.

LDH evaluation also should exclude two surgical urgencies: cauda equina syndrome and conus medullaris syndrome. Both conditions cause saddle anesthesia, autonomic nervous system dysfunction, (i.e., overflow incontinence and impotence), and leg pain. Concern for either diagnosis should lead to prompt advanced imaging. The urgency of these two conditions is justified largely by the poor outcomes associated with delays in surgical decompression.

Table 15.1 Clinical signs of lumbar disk herniation

Level	Nerve root/ dermatome	Motor weakness	Sensory distortion	Reflex loss
L1–L2, L2–L3	L2,L3	Hip flexors	Anterior thigh	None
L3–L4	L4	Quadriceps	Medial calf	Patellar
		Tibialis anterior	Medial foot	
L4–L5	L5	Extensor hallucis longus	Lateral calf	None
		Peroneals	Dorsum of foot	
L5-S1	S1	Posterior calf	Gastrocnemius	Ankle
		Plantar foot		
S2–S4	S2, S3, S4	Bowel/bladder dysfunction	Perianal	Cremasteric

Plain lumbar radiographs can be used to rule out any associated pathology and may show nonspecific findings for LDH, including loss of lordosis, loss of disk height, or vacuum phenomena. Non-contrast MRI is the imaging study of choice for LDH diagnosis. Asymptomatic LDH occurs at a high rate and illustrates the importance of correlating presenting symptoms and physical examination findings to the pathology observed on MRI. If MRI is contraindicated, then a CT myelogram may be performed to visualize neural element compression.

Management

Nonsurgical

In the general population, >90% of patients with LDH improve within 6 weeks of symptom onset after nonsurgical treatment [2]. Similarly, in a study of 342 professional basketball, American football, baseball, and hockey players diagnosed with LDH, 82% were able to return to their sport after treatment [2]. The natural history of LDH is not altered by nonsurgical treatment options; however they provide symptomatic relief, while the radiculopathy resolves naturally.

Anti-inflammatory medications reduce the production of inflammatory cytokines. Oral corticosteroids long have been advocated to treat acute radiculopathy, but a recent randomized clinical trial showed that they provided no benefit over placebo [6]. Psychological support is used to establish an expectation for recovery and to reaffirm the rehabilitation process. Short-term management with narcotics is reserved for patients with severe pain that limits rehabilitation. The treating physician must weigh the potential for abuse and complication associated with these drugs.

Commonly, physical therapy also is prescribed during the nonsurgical management of LDH. Therapy regimens mainly focus on core and back muscle strengthening and flexibility. A phased rehabilitation protocol for athletes with LDH has been described by Watkins and is discussed in the rehabilitation chapter in this book [7].

In patients with severe symptoms, epidural steroid injections provide an alternative to surgical treatment. Improvement in symptoms may enable surgery to be avoided in up to 50% of patients [8]. A study of 17 National Football League athletes with LDH treated with epidural steroid injections showed a return-to-play rate of 89% and an average loss of only 0.6 games played [8].

Surgical

Surgical management of LDH typically is considered after failure of a 6-week course of nonsurgical management. The surgical treatment of choice for LDH is laminotomy with diskectomy (Fig. 15.1). High-level evidence to support specific treatment options for LDH in elite athletes is currently lacking because numerous external variables

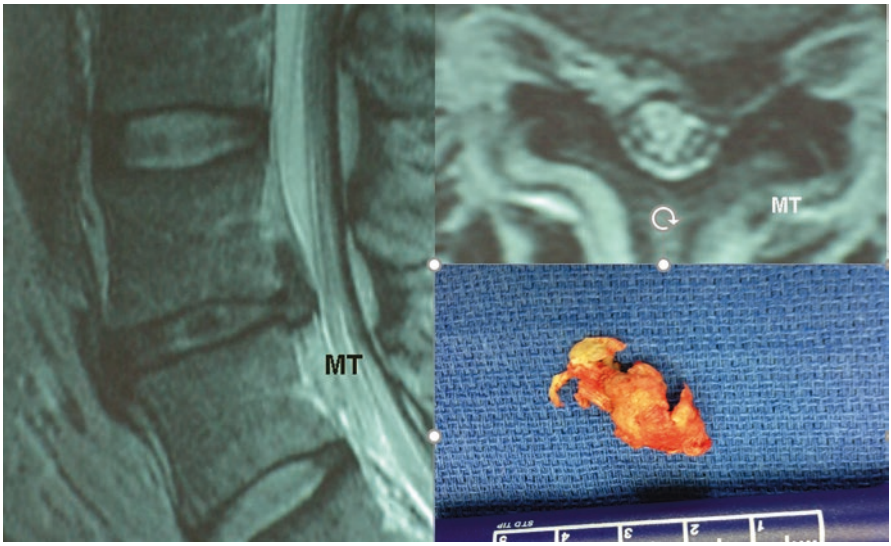

Fig. 15.1 This is a 21-year-old division 1 defensive lineman presenting with 5 days of severe left buttock pain and numbness extending into the top of his foot. He had 4/5 strength and is left EHL and peroneals. Imaging in the left and top right panel are a T2 sagittal and axial MRI showing a large L4–L5 left paracentral disk herniation. The timing of the injury was 2 months prior to the start of his season. One epidural steroid injection was attempted with some relief of pain for a day, but his strength had not improved 1 week after the injection. Continued nonoperative treatment was offered, but the player wanted to have a chance to return for the upcoming season, and the motor deficit was concerning to the patient. A microdiscectomy was performed and the removed disk herniation is shown in the bottom right panel. The 2 weeks' postoperative visit showed a healed wound, improvement in motor strength, and no pain. Functional rehabilitation was performed, and in this motivated patient he was back to practice at the 2 months' postoperative timepoint. He was able to return to play later that same season

make performance of a well-designed clinical trial challenging. In this chapter, we present the highest level of evidence currently available. In a systematic review of elite athletes undergoing lumbar diskectomy, Nair et al. [5] reported that 75–100% of elite athletes successfully return to play 2.8–8.7 months after surgery. Athletes who successfully returned to play had a career longevity that ranged from 2.6 to 4.8 years [5]. Similarly, Hsu et al. concluded that no differences in return to play, career games, and years played were observed after surgical or nonsurgical management for LDH in professional athletes. Certain subgroups, such as players who were younger and had more game experience, had better performance-based outcomes after treatment [2].

Inherent differences in specific sports and sport positions should be considered when treating elite athletes. For example, linemen in American football are considered to have a higher risk of LDH than that of players in other sports, because of the flexion and axial loads experienced by the spine during position-specific movements. A cohort study of NFL linemen demonstrated that surgical treatment yielded a significantly higher rate of return to play (81%) than that of the nonsurgical cohort (29%) [9]. Of note, 7 of 52 of the surgically treated linemen (13%) required revision

decompression; however, 6 of the 7 patients (86%) successfully returned to play [9]. Conversely, professional baseball players with LDH treated surgically have considerably longer recovery times (8.7 months) than those players treated nonsurgically (3.6 months) [10]. Furthermore, career length in patients treated surgically was shorter than in those treated nonsurgically (233 games versus 342 games, respectively; $P = 0.08$) [10]. One potential explanation for these differences is the daily rotational torque demands of throwing and hitting that are unique to baseball [11]. In National Hockey League athletes, 82% returned to play after treatment for LDH; no differences were seen between the surgical and nonsurgical cohorts with regard to games played or statistical performance [12].

Lumbar Degenerative Disk Disease

Lumbar degenerative disk disease (DDD) refers to the progressive degenerative changes seen in the IV disk. Lumbar DDD is characterized by the loss of disk hydration, disk space narrowing, and annular tears, ultimately culminating in ankylosis of the lumbar segment. Altered biomechanics underlie the observed pathologic changes. The loss of nucleus pulposus hydration causes the disk to become fibrotic, leading to abnormal loading of the facet joints. This process in turn facilitates the development of facet arthropathy and a further deterioration of normal biomechanics. The etiology of pain associated with lumbar spondylosis has been associated with nerve root irritation, claudication, or IV disk and/or facet pain.

A physical loading model was thought to be the predominant risk factor for lumbar DDD, but this theory has not been substantiated for the general population [13]. High-level evidence supports the notion that the most important risk factor for lumbar DDD is genetic predisposition [14], although aging, occupational hazards, and smoking also have been associated with its development [14]. In a cross-sectional study, Patel et al. [14] demonstrated that patients with a first-degree or third-degree relative with DDD have a markedly elevated risk for DDD. Similarly, in a cohort study of twins, Battié et al. [13] reported that, despite substantial differences in adult physical loading activities, no differences were observed in the incidence or severity of DDD. Smoking did predispose patients to DDD across all spinal levels, but the effect appeared to be minimal. The authors concluded that DDD is influenced largely by genetics, with minor contributions from environmental factors [13].

Despite limited evidence of physical loading as a risk factor for DDD in the general population, elite athletes are subjected to a higher level of physical activity. Intense training regimens begun at early ages may leave the adolescent spine at risk. In these patients, the spine experiences daily repetitive loads greater than those of most manual laborers [15]. Hangai et al. [15] compared 308 university athletes with 71 nonathlete university students and noted a considerably higher incidence of early lumbar degenerative changes in the athletes. These findings suggest that the physical demands of elite athletes may play an additive role in the development of DDD, especially in the adolescent spine.

Clinical Presentation

The history and physical examination of patients with isolated lumbar DDD is often nonspecific. The typical description is a deep, aching LBP. Discogenic pain is exacerbated by movements that load the disk and is relieved with rest and supine positioning. Age can once again provide a clue for diagnosis; in a study of 100 adolescent athletes and 100 adult athletes with LBP, only 11% of the adolescent athletes had disk pathology, compared with 48% of adult athletes [16].

Standard lumbar radiographs are the initial radiographic assessment for DDD and evaluate for disk space narrowing, subchondral cysts, facet degeneration, and osteophytes. Flexion and extension radiographs can be obtained to assess mobility, but they provide little information in cases of isolated lumbar DDD. MRI has a much higher sensitivity for detecting disk pathology and degenerative changes. MRI findings consistent with DDD include a loss of signal intensity on T2-weighted images, annular tears, and associated bone marrow/vertebral end plate changes. These findings do not necessarily correlate with the incidence of LBP, however, because one study showed that they can be seen in more than one third of asymptomatic patients [17]. A 7-year follow-up to this study showed that degenerative changes also did not predict the development of LBP, confirming that the correlation between imaging and symptoms is crucial in the management of DDD [18]. Conversely, in more severe cases, Modic changes that affect the bone marrow of the vertebral body have been described (Table 15.2, Fig. 15.2) [19]. These radiographic signs recently have been shown to correlate positively to the presence of symptomatic LBP [19].

Management

Nonsurgical

Nonsurgical management is the standard of care for lumbar DDD in the elite athlete. Physical therapy combined with anti-inflammatory medications is prescribed routinely. Many physical therapy protocols exist for long-term treatment of LBP in athletes, but the combination of core strengthening, lumbar mobilization, and biopsychosocial support yields good outcomes [20]. A staged rehabilitation protocol involving early protected mobilization, stabilization exercise, and a maintenance

Table 15.2 Modic classification of signal changes on MRI in the vertebral body

Stage	MRI	Vertebral body changes
Type I	T1-weighted	Decreased signal
	T2-weighted	Increased signal
Type II	T1-weighted	Increased signal
	T2-weighted	Increased signal
Type III	T1-weighted	Decreased signal
	T2-weighted	Decreased signal

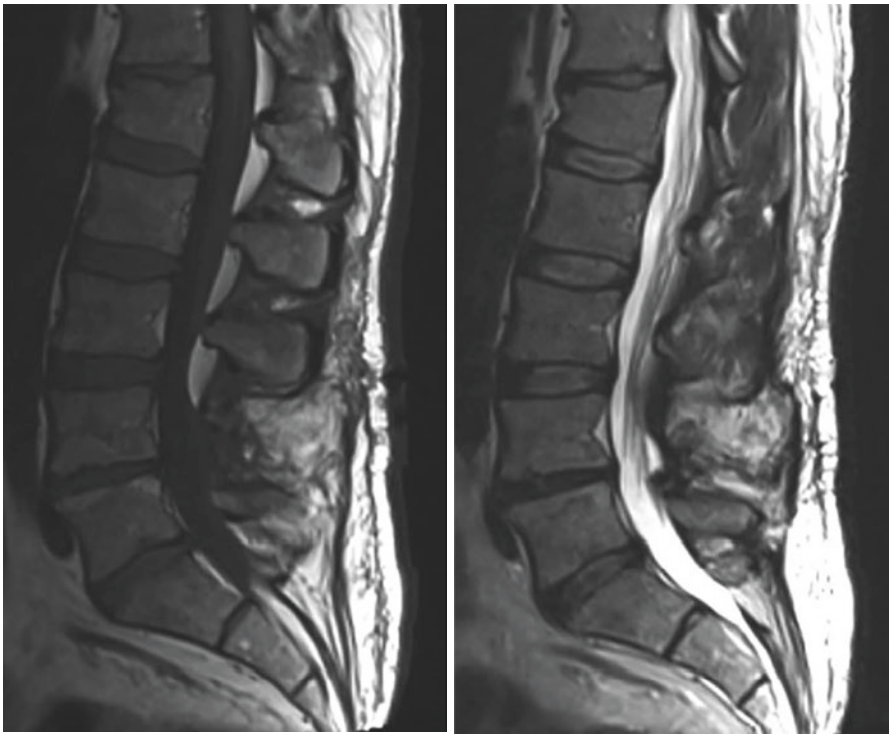

Fig. 15.2 Sagittal lumbar MRI showing degenerative disk disease at L4–L5 and type II Modic changes in a 37-year-old athlete. Left panel: T1-weighted imaging shows increased signal in the L4–L5 vertebral bodies. Right panel: T2-weighted imaging shows increased signal in the L4–L5 vertebral bodies

program is used to aid in the return to play. The duration of rehabilitation is patient-specific and symptom-specific.

Evidence is lacking to support epidural spinal injections and facet joint injections as therapeutic interventions for lumbar DDD [21]. A Cochrane review of 18 trials involving 1179 patients found insufficient evidence to support using injection therapy to manage subacute and chronic LBP [21]. However, they can be trialed for the elite athlete, especially in season. Alternative methods of treatment, including acupuncture, chiropractic care, massage therapy, traction, and behavioral therapy, can be trialed, but little evidence exists to support the efficacy of such modalities.

Surgical

Surgical management of lumbar DDD should be used only in select patients in whom nonsurgical management has failed and in those who cannot return to sport. Strict surgical indications include mechanical LBP with evidence of a single-level

degenerative disk on imaging studies, failure of at least 6 months of nonsurgical treatment, and localized midline spinal tenderness that corresponds to the radiographic level of disease. Narcotic abuse, smoking, and unrealistic patient expectations are relative contraindications to surgical treatment. Even when using selective indications, the clinical success of surgical management of DDD is unpredictable. Provocative diskography, which previously has been used widely to enhance surgical decision making for DDD, can lead to considerably faster progression of degenerative findings than that seen in control subjects [22]. Therefore, the American Pain Society recently published guidelines recommending against using provocative diskography [23].

Surgical management of DDD involves removal of the diseased disk and subsequent fusion or arthroplasty. Lumbar fusion is the standard treatment, but concerns over adjacent segment disease led to an increased interest in total disk arthroplasty (TDA) (Fig. 15.3). Symptomatic adjacent segment disease, possibly caused by increased stresses next to the fused segment, is estimated to occur in as many as 36% of patients at 10 years post-fusion [24]. Consequently, TDA treatments have been studied and compared directly with fusion [25]. These studies have not demonstrated a reduction in the incidence of adjacent segment disease in short-term and midterm follow-up after TDA versus fusion for the management of lumbar DDD [25]. Lumbar fusion outcomes can be affected by workers' compensation claims, chronic narcotic abuse, smoking, and the number of levels fused, which are negative predictors for the successful surgical management of lumbar DDD [26].

Few studies have investigated the outcomes following the surgical treatment of athletes with DDD. Siepe et al. [27] reported that, of 39 athletes treated with lumbar TDA for the treatment of LBP, 37 (95%) resumed their sporting activity and 33 (85%) were completely satisfied with the surgery, based on patient surveys [27]. After surgery, 69% of patients returned to play within the first 3 months, and the average time to peak fitness was 5.2 months, based on patient surveys [27]. Minor subsidence was observed in 13 patients (33%) within the first 3 months, with no further implant migration observed in 12 of these patients [27]. Of note, only two patients were involved in contact sports (karate, wrestling) and neither was able to return to play [27]. Tumialán et al. [28] compared the outcomes of lumbar fusion with those of TDA for LDH or DDD in military personnel [28]. A total of 10 of 12 TDA patients (83%) returned to unrestricted full duty compared with 8 of 12 lumbar fusion patients (67%), leading to the conclusion that TDA is comparable with fusion for treatment of active patients. In a study of eight professional hockey players who underwent single-level fusion [12], all the players returned to play and still were active after 4 years. The study also showed no substantial differences in the number of games played or performance scores before or after the procedure, but definitive conclusions were limited by the small sample size [12]. Similarly, Schroeder et al. [29] reported productive careers in two National Football League players after undergoing lumbar fusion; one player had a two-level fusion and had a 7-year National Football League career postoperatively [29]. Further studies are needed to identify the appropriate indications and long-term outcomes of surgically treated DDD in elite athletes, but the current literature does show that lumbar fusion does not necessarily contraindicate a return to play.

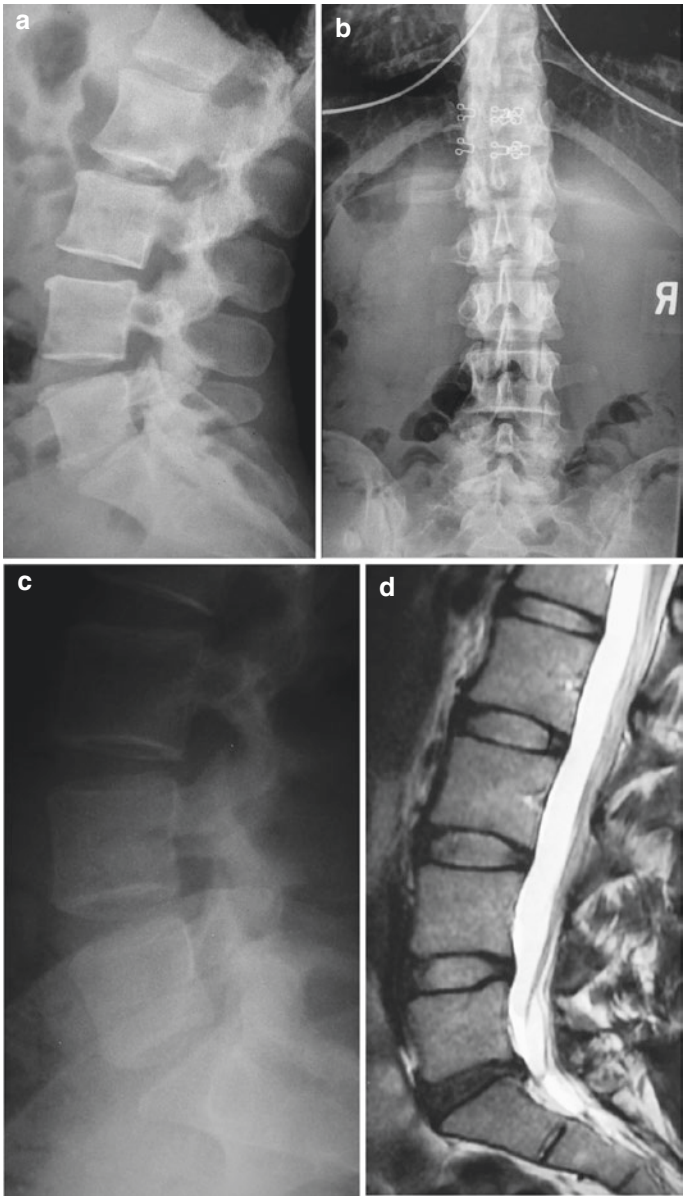

Fig. 15.3 This is a 20-year-old female division 1 basketball player with a history of chronic debilitating back pain. It had caused her to miss numerous games over her high school and college career. She had tried multiple nonoperative modalities and was interested in surgery to alleviate the pain. Images (**a**–**c**) show disk height loss and spondylotic changes at L5–S1. Images (**d** and **e**) are MRI findings illustrating disk degeneration at L5–S1 without neurologic compression. After extensive counseling, she elected to move forward with a single-level lumbar fusion. The surgical technique aimed at maximizing fusion with minimal muscle disruption, hence a L5-S1 ALIF was performed in conjunction with percutaneous facet fusion. She had to redshirt a season and was able to return to basketball the subsequent season. Images (**f** and **g**) are 1-year postoperative lumbar radiographs that show a fusion across L5-S1

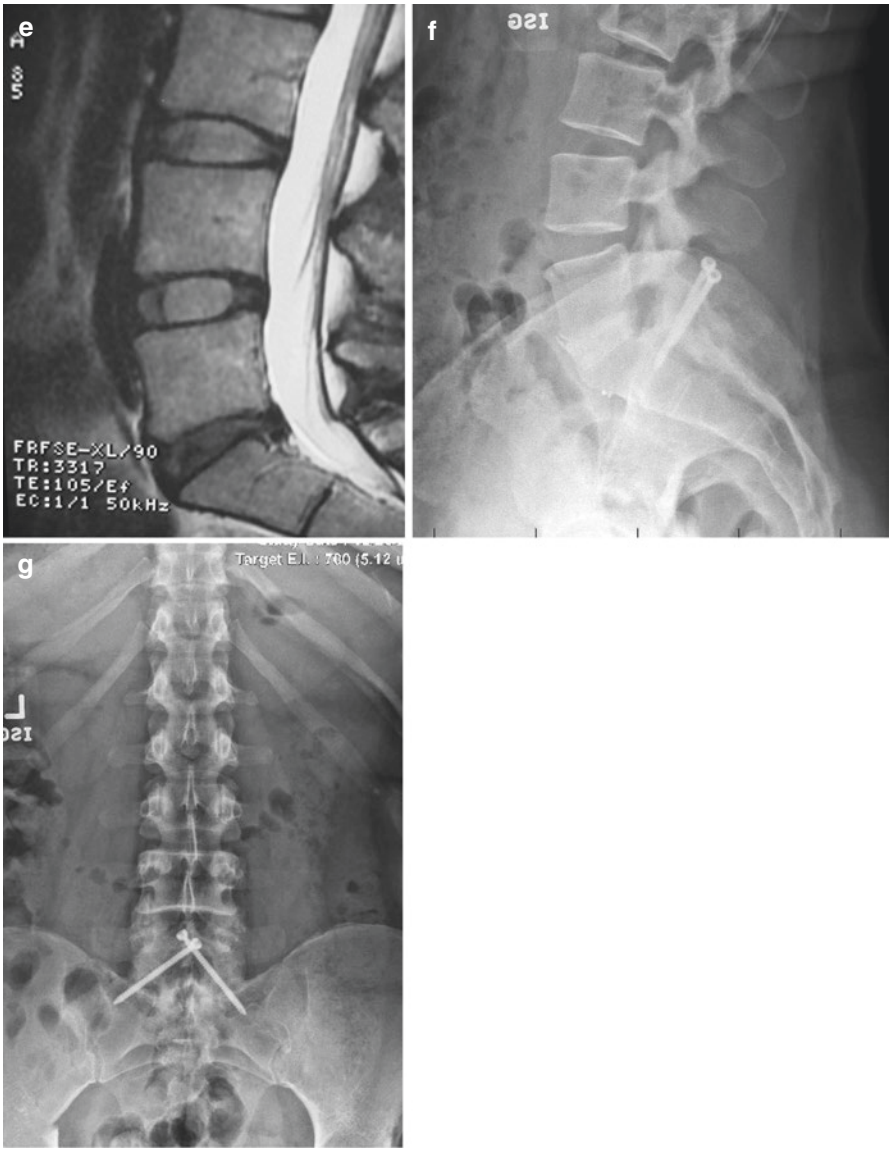

Fig. 15.3 (continued)

Return to Play

Return to play should be determined on a case-by-case basis, with consideration of the athlete, the injury, and the sport. The guidelines for return to play are similar to those for other lumbar conditions and generally depend on the athlete demonstrating resolution of symptoms and full preinjury range of motion and strength, as well as the successful completion of a structured rehabilitation program [7, 30]. Structured protocols have been found to improve recovery time [31].

Expert Opinion

In elite athletes, most lumbar conditions can be managed nonsurgically with excellent outcomes. Surgical treatment is a viable option for athletes in whom nonsurgical treatment has failed. Many elite athletes can return to play even after surgical treatment, especially those who require lumbar diskectomy. High-level evidence that elite athletes with surgically treated LDH return to play in 75–100% of cases [5]. Return to play is assessed on an individual basis. It is our opinion that regardless of nonsurgical versus surgical management, the key components to return to play are as follows: pain control and progression through a physical therapy protocol. We prefer the therapy protocol as described by Watkins et al. in this book. There is no specific time table to return to play because it is more dependent on how quickly the athlete moves through the physical therapy protocol. Some athletes may progress rapidly (weeks) and others may take longer (months). Stand-alone anterior lumbar interbody fusion may be a better surgical option in athletes with multiple recurrent disk herniations at a single-level, rather than performing continual revision discectomies.

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Chapter 16
Lumbar Disc Herniation in the Adolescent Athlete

Andrew M. Cordover, Jacob B. Cordover, Glenn S. Fleisig, and Jesse A. Raszewski

Introduction

While participation in sports has been an important part of culture and childhood development, injuries to the lumbar spine may preclude participation with devastating social, psychological, and physical consequences. Injuries to the lumbar spine can range from minor strains and sprains to career ending requiring surgery. The treatment goal is to quickly return the patient to daily activities, school, and sports with satisfactory outcomes.

While a known cause of missed playing time, lumbar pathology is often overlooked by other health-care providers resulting in a delayed or missed diagnosis. The diagnosis of pediatric and adolescent disc herniation is difficult to make initially, as the symptoms are not always typical of adult patients with disc pathology. The presenting symptoms, natural history of the disease, and pathologic process all contribute to a different presentation from the adult lumbar disc herniation (LDH).

Unlike the cervical and thoracic spine, devastating neurologic sequelae are rare with lumbar pathology. Return to sport in skeletally mature patients with lumbar pathology has been studied, and returning to play has become the expectation [1, 2]. However, fewer return to play guidelines and recommendations exist for skeletally immature patients.

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Surgically treated LDH in the skeletally immature was reported by Wahren in 1945, in his treatment of a 12-year-old gymnast [3]. After 1 month of symptomatic treatment with bed rest and heat, the symptoms remained intractable [3]. She underwent surgery, and the herniation was described as more mucous compared to the adult counterpart [3]. She had an uneventful postoperative course and within 1 month was asymptomatic. Lumbar disc herniation requiring surgery has also been reported in children as young as 6 years of age [4].

Epidemiology and Risk Factors

The epidemiology of adult lumbar disc disease has been well-studied. Roughly 5% of lumbar disc disease occur in patients younger than 18 years old (y/o) [5]. Dang et al. found the incidence of adolescents and children who are hospitalized for LDH to be 0.1–0.2% [6]. Kumar et al. reported the incidence of LDH in the pediatric and adolescent population to be 3.5% in patients less than 20 years of age (YOA) [7]. When lumbar discectomy as a procedure is looked at, the surgical treatment of children and adolescents comprises between 0.5% and almost 4% [8–13].

The distribution of skeletally immature patients with LDH by gender varies from study to study. Some find a slight male predilection [10, 14], while Slotkin et al. found a 2:1 female-to-male ratio, and Celik et al. found 56% of his patients were female [5, 15]. Papagelopoulos et al. reported a 1.25:1 male-to-female ratio, and Sarma et al. showed 81% of his patients were male [16, 17]. Kumar et al. found 84% of his adolescent discectomy patients were male [7].

Many authors have found that radiographic anatomical variants and abnormalities are more often found with LDH in the skeletally immature. These include transitional anatomy, abnormal facet tropism, stenosis, spina bifida occulta, narrowed disc space, and spondylolisthesis [11, 13, 14].

In the adult population, it is accepted that genetic factors are a significant risk factor for disc disease, as found in the twin studies [18–20]. The studies evaluated twins with discordant lifestyles and found that while environmental influences may play some role, genetics, including anthropometric factors, such as body habitus, cross-sectional area of the muscle, and strength, play a substantial influence on the heredity of lumbar disc degeneration [18–20]. The twin studies found that although there was extraordinary discordance between twin siblings in occupational and leisure time physical loading conditions throughout adulthood, a similar amount of degeneration was observed [18]. The study group also found that anthropometric factors have an effect. Similarly, Videman et al. found that genetics played the largest factor in disc degeneration [19].

Figure 16.1 demonstrates different anthropometric morphologies. Figure 16.1a demonstrates a nonathlete and Fig. 16.1b an elite NFL player. Perhaps these anthropometric factors are what give elite athletes performance advantages and are of paramount importance in their disc degeneration, as seen in Fig. 16.1b.

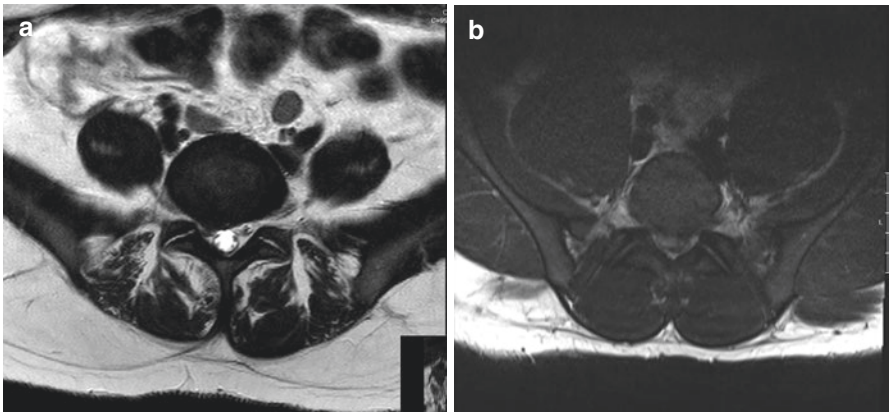

Fig. 16.1 (**a**) Axial MRI images of a 38 y/o nonathletic patient with a L5-S1 HNP. (**b**) Axial MRI images of 19 y/o elite athlete (who is currently an elite NFL player) with a L5-S1 HNP. Note the difference in anthropometric factors (cross-sectional area of the psoas, paraspinal muscles, and other anatomic structures) compared to the nonathlete in (**a**)

Kumar et al. found a positive family history for lumbar disc disease in 24% of pediatric and adolescent patients with an LDH [7]. Grobler et al. also found 24% of adolescence with a surgically treated lumbar herniated nucleus pulposus (HNP) have a positive family history for disc disease [13]. Zamani and MacEwen found a positive family history in 17% of their patients with a symptomatic LDH [21]. In totality, these studies demonstrate the influence of genetics on lumbar disc degeneration.

Pathophysiology

The report of trauma as a precursor to adolescent and pediatric disc herniation is varied. Sarma et al. found this was a significant factor in 57% cases, and Ozgen et al. found a positive history of trauma or intense sports activities in 83% of adolescent cases [10, 17]. Epstein et al. found 52% of teenaged children reported antecedent trauma prior to symptomatic LDH [11]. Slotkin et al. opined that pediatric disc herniations are much more likely to be the result of an acute injury rather than a degenerative condition [5]. Kumar et al. believed that gross trauma is not necessarily a contributory factor in adolescent LDH since it was only present in 8% of their patients, but repetitive microtrauma may be a predominant factor [7]. Papagelopoulos et al. reported 60% of the patients in their series had an antecedent trauma, which included sports injuries [16].

In a study on surgical treatment of adolescent intervertebral disc herniations, Grobler et al. found trauma was a significant factor in 59% of the cases [13]. In patients under 16 y/o, 61% were female, while in patients 16–20 y/o, 63% were

male [13]. They suggested this occurred secondary to earlier skeletal maturity in females [13]. Cahill et al. found that 64% of adolescent LDH patients were competitive athletes [22].

In our experience, we have found younger athletes often have a traumatic prodrome to their LDH. In particular, athletic trauma due to training, such as dead lifts, squats, and power cleans, is often documented.

Ring Apophysis

Many symptoms of adolescent disc herniations are morphologically found to be fractures of the ring apophysis rather than the herniated nuclear material typically found in the adult population. It has been emphasized that recognition of these fractures is essential for appropriate treatment, preoperative planning of surgery, if indicated, and prognosis. Since this is an osseous/cartilaginous lesion, it is more readily appreciated on computed tomography (CT) scan than magnetic resonance (MRI) imaging. Epstein et al. found only one third of limbus vertebrae fractures were identified on the MRI scan, while 100% were appreciated on CT scan [23]. CT scanning is considered the optimal modality for imaging these types of fractures [24].

Bick et al. was the first to histologically evaluate the ring apophysis (RA) in detail, after Schmorl defined it [25]. Bick et al. described the RA as a region with increased amounts of traction due to the branching fibers of the long intervertebral ligament inserting into the individual vertebrae [25]. Calcification begins at approximately 6 y/o, ossification begins at 13 y/o, and fusion with the vertebral body at around 17 y/o [25]. By 18 years, fusion is complete, and at 20 y/o, the ring cannot be histologically identified [25].

The RA can separate from the caudal aspect of the cephalad vertebra or the cephalad aspect of the caudal vertebrae before skeletal maturity causing mass effect similar to, or in conjunction with, a disc herniation. The exact mechanism of the fracture is not understood, but many theories exist. In a study by Epstein et al., approximately half of the patients, mostly athletes, had a trauma as the inciting event [23]. Moreover, if there is neural compression, a radiculopathy may present.

Takata et al. described fractures of the RA and then morphologically classified them as type I, II, or III, as seen in Fig. 16.2 [26]. Type I fractures are a separation of the posterior margin or rim of the vertebra with no osseous defect [26]. It is described on CT scan to have an arcuate structure in the spinal canal. Type II fractures are an avulsion fracture of the posterior rim of the vertebral body [26]. There

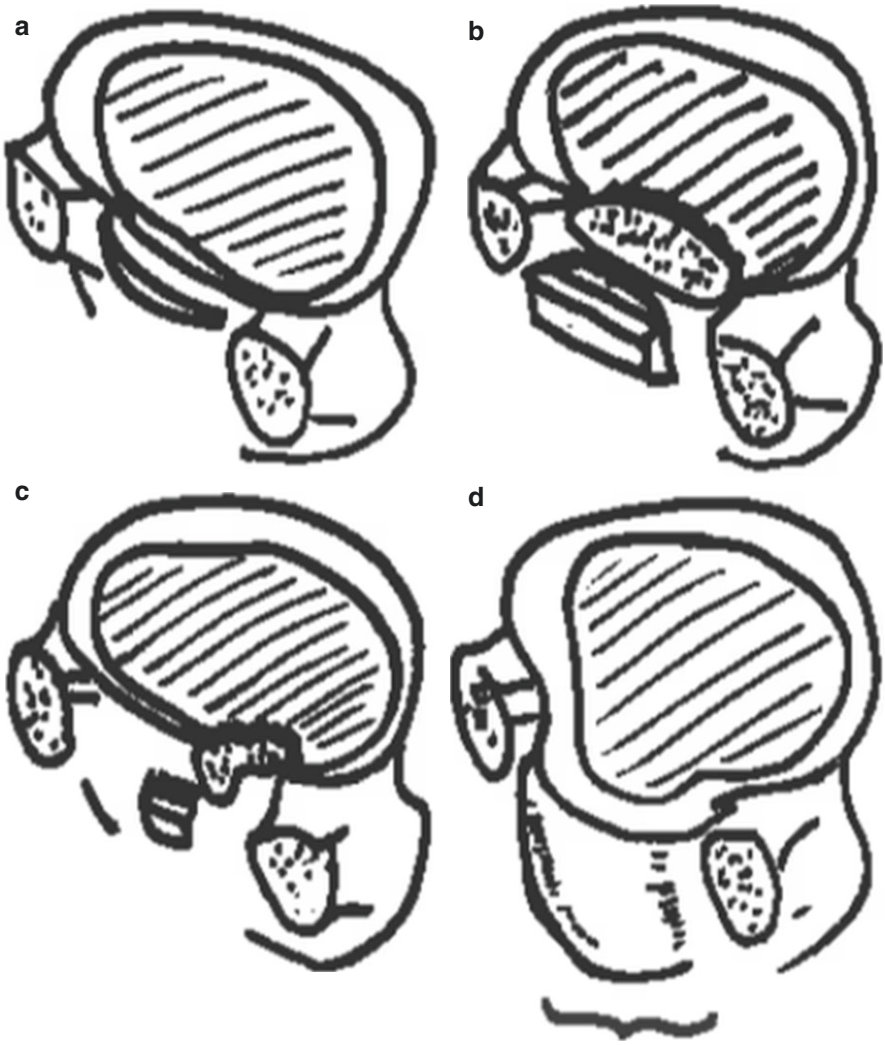

Fig. 16.2 Schematic diagram of fractures of the vertebral limbus. (**a**) Type I: simple separation of the entire posterior vertebral margin. (**b**) Type II: avulsion fracture of some of the substance of the vertebral body, including the margin. (**c**) Type III: more localized lateral fracture of the posterior margin of the vertebral body. (**d–g**) Type IV: fracture that extends both beyond the margins of the disc and the full length of the vertebral body between the end plates. The Type IV fracture effectively displaces bone posteriorly, filling the floor of the spinal canal with a combination of reconstituted cortical and cancellous bone accompanied in part by scar formation

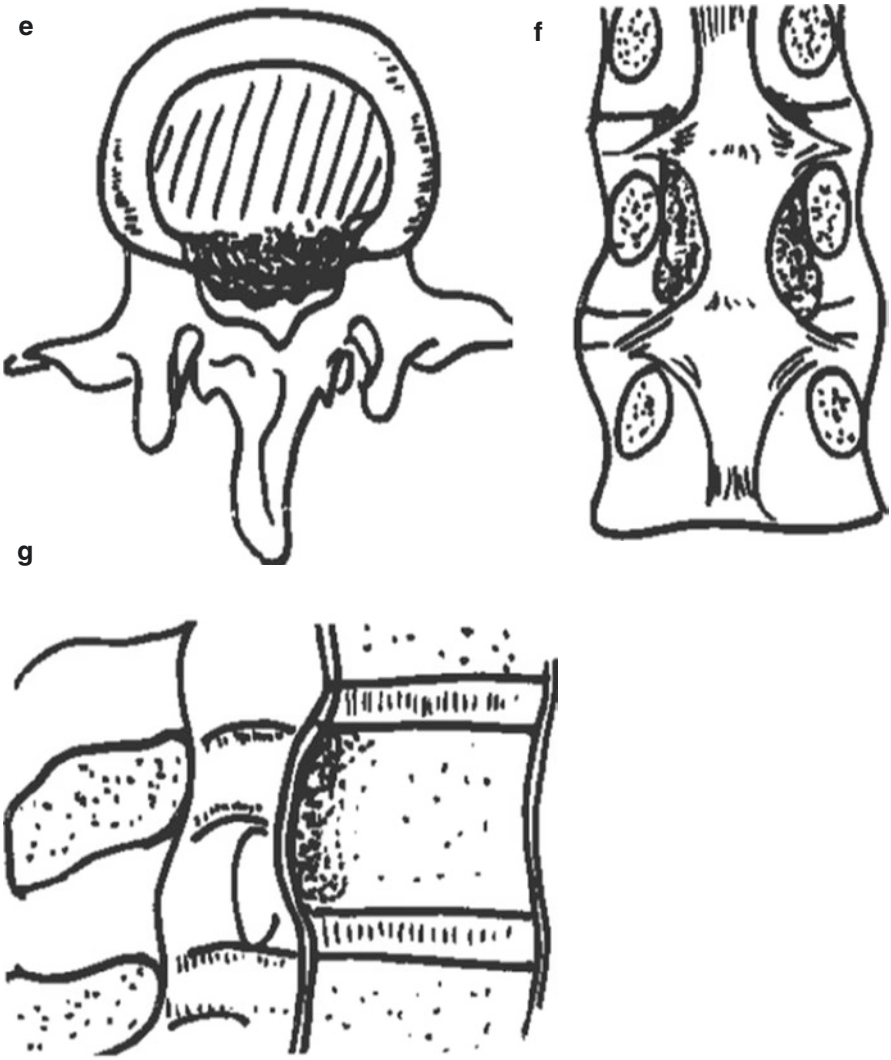

Fig. 16.2 (continued)

are cortical and cancellous bone fragments with a portion of the vertebral body that include the annulus. Type III fractures are smaller, more localized lateral chip fractures with a bone defect adjoining the fracture site [26]. Type I fractures tend to occur in younger adolescent patients, while type III are more commonly found in the skeletally mature [26]. Takata et al. looked at 29 patients, 66% were male. Of those with symptoms, 84% had a positive straight leg raise [26]. All surgical patients in the study had a good outcome [26]. Epstein et al. described a type IV fracture, where the lesion spans the entire length and breadth of the posterior vertebral margin between the end plates as seen in Fig. 16.2 [23].

Grobler et al. found that 38% of pediatric LDHs had a RAF [13]. They reported on 29 surgical patients with a RAF and found that trauma was a significant factor in 59% of cases. Moreover, 78% of these were caused by sports [13].

Wu et al. conducted a literature review on RAF with LDH that included 366 patients and discussed the importance of preoperative planning. The surgical treatment with excision of the apophyseal fragment was somewhat different than a more typical LDH. Wu et al. concluded that the surgical outcomes in patients were equally beneficial for the posterior RAF and LDH patients, but due to the shortcomings in the literature, no definitive consensus on treatment modality could be established [27]. Wu et al. demonstrated that there was a 2.85:1 male-to-female ratio, and 7.9% of RAF occurred at vertebral levels other than L4-L5 and L5-S1 which were similar to LDH patients [27].

Epstein et al. evaluated 27 patients with a RAF with a mean age of 32 years [23]. Fifty-nine percent of patients were male [23]. Trauma, which included sports-related injuries, was related to more than half the patients [23]. Lavelle et al. found that 28% of adolescent disc herniations involve apophyseal fractures, and these have a higher rate of surgical intervention [28].

Singhal et al. evaluated CT scans of 42 patients less than 18 y/o that were evaluated for LDH [29]. Fifty-five percent of patients with a LDH had a traumatic etiology [29]. Of the 42 patients, 38% of the pediatric patients did have an associated RAF [29]. Of their LDH patients, 55% of the males and 20% of the females were found to a RAF [29]. RAF was also associated with central herniations [29].

The literature on RAF shows a clear association with LDH in the adolescent. This trend is especially notable in adolescent males likely due to their delayed skeletal maturity in comparison to females. Recognition of this lesion preoperatively and maintaining a level of suspicion intraoperatively are essential when surgical treatment on an adolescent is performed. Failure to adequately resect the lesion can lead to persistent radiculopathy. Figure 16.3 is a case example of a failure

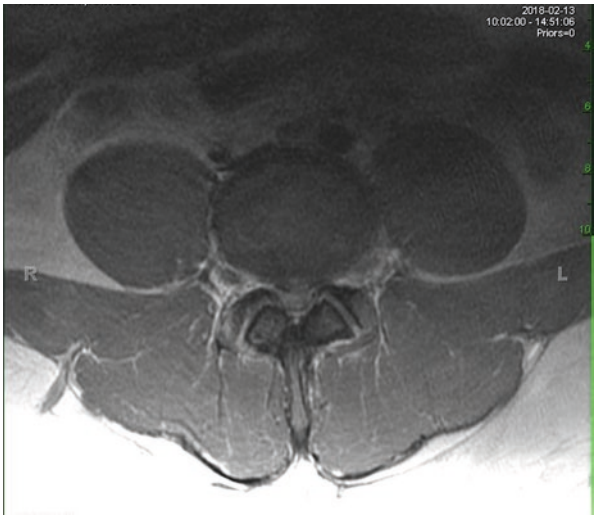

Fig. 16.3 Postcontrast MRI in a 16-year-old male demonstrating a persistent RAF that was not resected at the time of initial micro-decompressive surgery. See text for further discussion

to resect a RAF. The figure shows a postcontrast MRI in a 16 y/o male demonstrating a persistent RAF that was not resected at the time of initial micro-decompressive surgery. Postoperatively, the radiculopathy persisted, and he was unable to participate in all sporting and leisure activities. The severity of symptoms also precluded him from returning to school, and home schooling was necessary. Two years postoperatively, his parents sought another opinion. Within 2 weeks after revision decompressive surgery where a RAF was removed, he had returned to school and 3 months after revision surgery, was competing in sports.

Disc Degeneration in Adolescent Athletes

Alyas et al. evaluated MRI findings in 33 asymptomatic elite adolescent tennis players with a mean age of 17.3 and found disc degeneration, desiccation, and bulging in 39% of these athletes [30]. Only 15% of those studied had a normal MRI exam of the lumbar spine [30]. Additionally, 27% had a pars lesion and 70% had early facet arthropathy [30]. Most pathology was exclusively displayed at the most caudal two motion segments [30]. This study, as with others in the spine literature, underlies the importance of correlating abnormal imaging to the patient's symptoms. Abnormal imaging alone does not indicate a diagnosis necessitating treatment.

Gerbino et al. found degenerative disc disease (DDD), facet degeneration, and low back pain increased in football players with their years of participation [31]. Bono discussed that while the prevalence of disc degeneration in athletes is higher than in nonathletes, it remains unclear whether it correlates with a higher rate of low back pain [32].

Sarma et al. found that 29% of skeletally immature patients with a disc herniation had a single level of disc degeneration, and 29% had multilevel disc degeneration [17]. Kumar et al. found only 16% of LDH patients had underlying degenerative changes [7].

Bartolozzi et al. looked at training and the occurrence of disc lesions with training overload in volleyball athletes. They found training regimens are a more important risk factor than player age and overall period of athletic activity [33]. Volleyball players who followed appropriate training procedures had positive MRI findings 21% of the time, and those who trained with exercises creating significant functional overload had positive findings in 62% of cases [33]. Their control group of swimmers displayed positive MRI findings in only 20% of cases [33].

Ong et al. looked at Olympic athletes with low back pain and/or sciatica. They showed 58% had an element of disc displacement, most of which were disc bulges [34]. Elite athletes have a greater prevalence and degree of disc degeneration than their age-matched controls [34]. In total radiographic disc degeneration does appear to occur at a slightly higher rate in elite athletes; however, it is unclear if these changes cause long-term pain or disability. Figures 16.4 and 16.5 demonstrate morphologic differences in the nonathletic population compared to elite athletes.

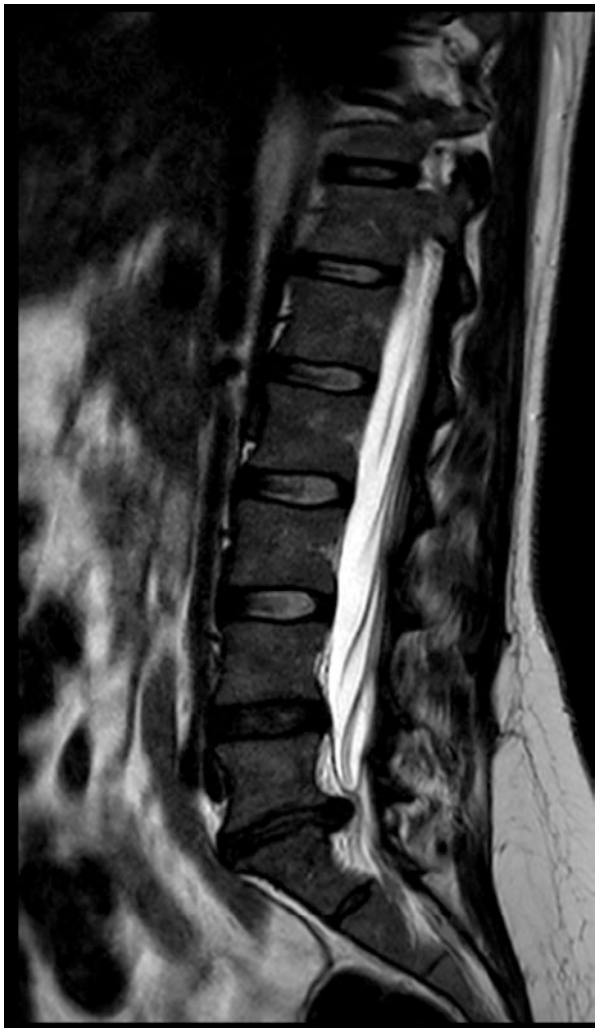

Fig. 16.4 Sagittal MRI of a lumbar disc herniation at the L5-S1 level with age-appropriate changes

Symptoms

While a significant portion of adolescent athletes experience episodic low back pain, only a small percentage have disc herniations, leading the diagnosis to often be delayed or misdiagnosed. The inability of children or adolescents to fully articulate their symptoms further compounds the issue of diagnosis. The initial differential diagnosis often includes strains, sprains, stress fractures, spondylolisthesis, DDD, Scheuermann's disease, scoliosis, discitis, tumors, or other neoplastic lesions. These processes may all mimic LDH. It is also important to rule out an extraspinal pathology.

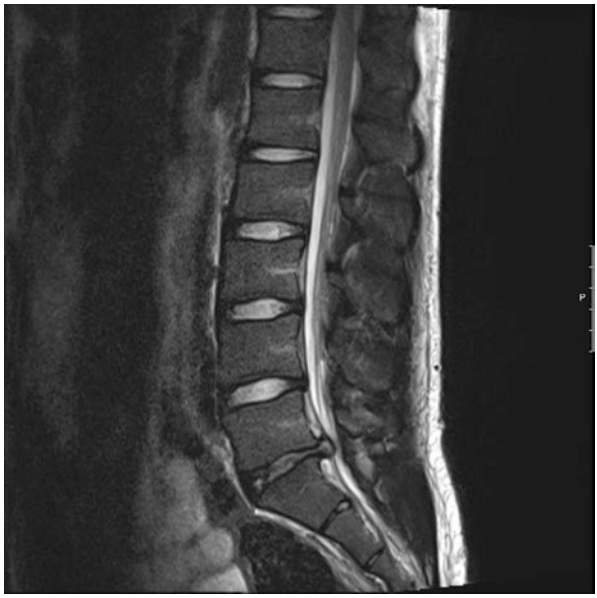

Fig. 16.5 Sagittal MRI of a 19-y/o elite athlete with a disc herniation and advanced degenerative changes at the L5-S1 level

In the skeletally immature patient, there is a trend toward more back pain and fewer radicular complaints, although this varies between studies. Grobler et al. looked at the surgical treatment of adolescent intervertebral disc herniations and determined that back pain was a major chief complaint in all cases surgically treated [13] where sciatica was the primary symptom in only 55% of the cases [13].

In a series published by Kumar et al., all patients with LDH presented with low back pain and 68% had radiculopathy [7]. Nerve tension signs vary and significant motor deficits are not common. In a study by Ozgen et al., 88% of patients presented with low back pain, 41% of patients had a positive straight leg raise, 35% presented with radiating sciatica, 47% of patients had scoliosis, and the median duration of symptoms from the onset to time of presentation was 7.7 months [10]. Papagelopoulos et al. found a 6.4-month period between the onset of symptoms and initial surgery, and all of the patients presented with sciatica [16].

Borgesen et al. found that lumbar pain and sciatica were both present in 96% of patients less than 20 y/o [9]. Motor and sensory findings were far less common in this study. Scoliosis was present in 52% of cases [9]. In contrast to other studies, Borgesen et al. opined trauma as an unlikely etiology in adolescent lumbar disc herniation and found this in only 16% of those in their series [9]. This is the same as their adult control group; hence they concluded that disc degeneration is the primary cause, while trauma is only a precipitating factor [9]. A good or excellent surgical outcome was found in 98% of patients [9].

Grobler et al. found restricted forward flexion was found in 76% of patients, and all patients had a positive straight leg raise and 3.8 months of conservative treatment prior to surgery [13].

Pinto et al. demonstrated that scoliosis was one of the presenting findings secondary to an antalgic position, and this resolved with successful removal of the inciting

disc herniation [35]. Cahill et al. reported a reactive scoliosis was present in 18% of the 87 patients [22]. In a study by Sarma et al., 31% of adolescents with a LDH had scoliosis upon presentation [17]. Zhu et al. evaluated 26 patients whose scoliotic posture was the initial symptom in adolescents with LDH and found a pattern of a short lumbosacral curve accompanied with a long thoracic or thoracolumbar curve toward the opposite side [36]. Fifty-eight percent of these that had low back pain, and only 69% had a positive straight leg raise exam finding [36]. There were 88.5% that had a trunk shift greater than 2 cm toward the side opposite of the disc herniation and had poor coronal plane balance [36]. All patients had a straight sagittal profile, and all had marked improvement in the curve after excision of the offending disc herniation [36].

Ozgen et al. evaluated 17 adolescent patients who required surgery for LDH [10]. Fifty-nine percent of the patients were male [10]. Eighty-two percent of patients were involved trauma or intense sports activities [10]. Eighty-eight percent of the patients had low back pain as the most common complaint [10]. The straight leg raise test was only positive in 41% of patients [10]. All of the cases were at the L4-L5 and L5-S1 vertebral levels [10].

As seen above, most studies show that adolescent LDH differ from adult presentation with a preponderance of back pain compared to leg symptoms. In addition, a possible reactive scoliosis should trigger the clinician that a disc pathology may be present.

Treatment

Conservative treatment options vary, including nonsteroidal anti-inflammatory drugs (NSAID), other medications as indicated, physical therapy, interventional pain management with blocks, activity modification, orthosis, and observation of the natural history of disc resorption. However, the natural history of disc regression may not be the same in adolescents and children as in adults. Many of the older case studies recommend nonoperative treatment with prolonged initial bed rest, traction, activity limitations, and a body cast [3, 21]. Early imaging with an MRI is sometimes prudent and is mandatory for any neurologic findings, systemic issues, or atypical findings.

Prior to surgical intervention, MRI is the study of choice, allowing for visualization of edema, compressive neural lesions, tumors, stenosis, disc degeneration and associated changes, pars fractures/stress reactions (edema), etc. CT scanning can be helpful for preoperative evaluation of pars lesions, RAF, and facet pathology.

According to the experience of the senior author, in well over 60 adolescent discectomies, we have found that the herniated material is frequently contained and far more tenuous than in their adult counterparts. All of the studies that were reviewed agree with this premise and found fewer extrusions and more contained disc material as seen in Fig. 16.6. Generally, it is felt that these highly elastic disc herniations respond poorly to conservative treatment [5, 6, 22]. The pathophysiology of LDH and hence healing occurs because an inflammatory response is elicited by the body secondary to the nucleus pulposus extruding from the annulus. This inflammatory

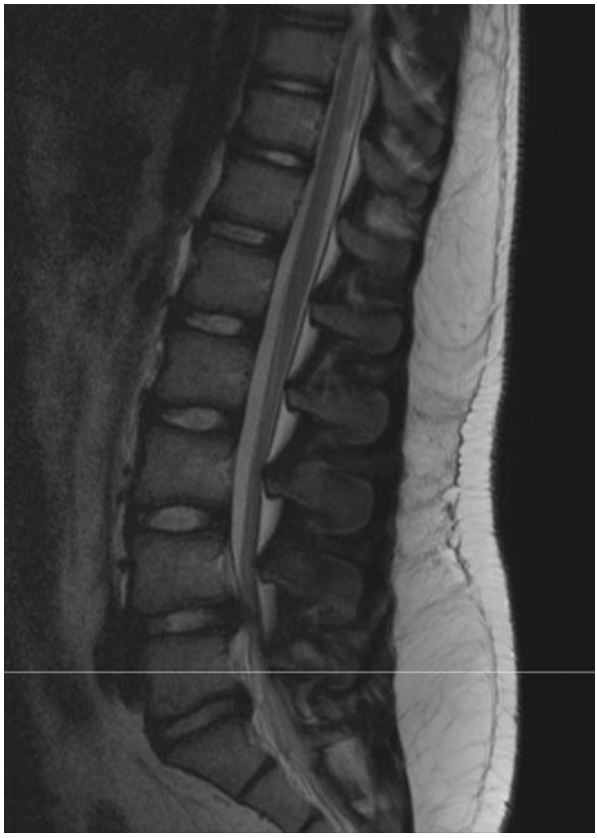

Fig. 16.6 T2 sagittal MRI demonstrating a contained HNP in an adolescent athlete. He did not respond to nonoperative treatment and underwent successful microdiscectomy

cascade over time allows the body to absorb the extruded disc material. In adolescents, the contained disc protrusion is unable to elicit the same inflammatory response. Despite this, other authors have found that only a minority of patients, around 10%, required surgery [21].

When treated surgically, the tenuous disc material often necessitates the use of reverse currets and tamps to impact the compressive lesion, sometimes including RAF's into the disc space. The material may then be resected with a pituitary from the disc space. Also, Kerrisons, osteotomes, drills, or other instruments can be used to extract the offending lesion and provide satisfactory neural decompression. Identification of the pars interarticularis is necessary, and preservation of this structure is necessary to prevent iatrogenic instability. Consequently, this procedure may be more technically demanding than an adult microdiscectomy.

We have found that a number of patients have an osseous-cartilaginous end plate fracture, and the surgeon must be prepared to address this. While not all authors advocate removal of the bony fragment in a RAF, removal of the bony fragment is indicated for decompression when it creates neural impingement. Successful outcomes depend on the neural decompression and require meticulous microsurgical techniques. Because of this, the surgeon must have a level of awareness for the RAF pathology.

We have found, in our experience, approximately 65% of athletes, 21 y/o or younger, are able to return to sports at the collegiate or high school level. However, for each patient after the convalescence of surgery, because of their age, returning to sport would have been at a higher level (i.e., junior varsity to varsity or high school to college level), and some chose not to participate; hence, the actual return to play is still not fully understood. For example, only 6.9% of high school football players go on to play at the NCAA level and far less at the Division I level (see Table 16.1).

Table 16.1 Estimated probability of competing in college athletics

	High school participants	NCAA participants	Overall % HS to NCAA	% HS to NCAA Division I	% HS to NCAA Division II	% HS to NCAA Division III
Men						
Baseball	491,790	34,980	**7.1**	2.1	2.2	2.8
Basketball	550,305	18,712	**3.4**	1.0	1.0	1.4
Cross country	266,271	14,350	**5.4**	1.8	1.4	2.2
Football	1,057,382	73,063	**6.9**	2.7	1.8	2.4
Golf	141,466	8,527	**6.0**	2.1	1.7	2.2
Ice hockey	35,210	4,199	**11.9**	4.8	0.6	6.5
Lacrosse	111,842	13,899	**12.4**	2.9	2.3	7.1
Soccer	450,234	24,986	**5.5**	1.3	1.5	2.7
Swimming	138,364	9,691	**7.0**	2.7	1.1	3.1
Tennis	158,171	7,957	**5.0**	1.6	1.1	2.3
Track and field	600,136	28,595	**4.8**	1.8	1.2	1.7
Volleyball	57,209	2,007	**3.5**	0.7	0.7	2.0
Water polo	21,286	1,013	**4.8**	2.7	0.7	1.3
Wrestling	244,804	7,175	**2.9**	1.0	0.8	1.1
Women						
Basketball	430,368	16,532	**3.8**	1.2	1.1	1.5
Cross country	226,039	15,966	**7.1**	2.6	1.8	2.7
Field hockey	60,549	6,066	**10.0**	3.0	1.3	5.7
Golf	75,605	5,372	**7.1**	2.9	2.1	2.2
Ice hockey	9,599	2,355	**24.5**	8.8	1.2	14.5
Lacrosse	93,473	11,752	**12.6**	3.7	2.7	6.2
Soccer	388,339	27,638	**7.1**	2.4	1.9	2.8
Softball	367,405	19,999	**5.4**	1.7	1.6	2.1
Swimming	170,797	12,684	**7.4**	3.3	1.2	2.9
Tennis	187,519	8,736	**4.7**	1.5	1.1	2.1
Track and field	494,477	29,907	**6.0**	2.7	1.5	1.8
Volleyball	444,779	17,387	**3.9**	1.2	1.1	1.6
Water polo	20,826	1,159	**5.6**	3.4	0.9	1.3

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Nonoperative treatment of skeletal mature LDH in athletes has also been studied and is a viable option for some. Iwamoto found 79% of athletes returned to play at an average of 4.8 months after the start of conservative treatment. In the Professional Athlete Spine Initiative (PASI), Hsu et al. found that 82% of athletes returned to play, and there was no statistically significant difference between the surgical and the nonoperative cohorts [1]. However, the cohorts may have been different. It remains to be seen at what level these data can apply to the adolescent athlete.

Outcomes

Papagelopoulos et al. reviewed the long-term outcomes of lumbar discectomy in 72 patients who were 16 y/o or younger at the time of surgery [16]. The mean duration of follow-up was 27.8 years [16]. Seventy-two percent of patients did not require a reoperation [16]. At the time of the last follow-up, 92% had no pain or occasional pain related to strenuous activities, and 98% could participate in daily activities with no or mild limitations [16]. Repeat surgery was required for 1% of patients within the 1st year, and 20–30% required surgery later in life [16]. Papagelopoulos et al. found reactive scoliosis resolves with successful treatment [16].

Grobler et al. reported that all of their adolescent microdiscectomy patients had complete relief of their symptoms in their immediate postoperative period, and 89% had excellent or good results at 5.3 years with surgical treatment [13]. Dang and Liu concluded that pediatric patients respond less favorably than adults do to nonsurgical management [6]. At the 10-year follow-up, the outcomes of surgery remained satisfactory, but the results deteriorated slightly [6].

Singhal et al. reported on posttreatment follow-up, including both surgical and nonoperative treatment [29]. They found after treatment, 58% of all patients with a RAF were symptom-free, while 68% of patients without a RAF were asymptomatic [29]. While the surgical outcomes of both groups did favorably, the non-RAF patients had a more favorable outcome with regard to residual symptoms then the RAF patients after surgery [29]. Overall, 100% of the treatment groups had partial or complete relief of symptoms [29]. There were 32% of patients that had a partial recovery [29]. They also observed worse outcomes in the nonsurgical group, of which, only 55.6% completely recovered [29].

Ishihara et al. followed 11 patients less than 16 y/o for an average of 9 years postoperatively [8]. Eight of the patients underwent posterior discectomy, two patients who had a central herniation underwent extraperitoneal anterolateral discectomy, and one with hypermobility underwent anterior fusion [8]. All patients had favorable outcomes [8]. The posterior discectomy patients had quicker relief, with a mean of 5.5 weeks to recover, versus the other procedures [8]. The mean time for straight leg raise recovery was 7.8 months [8]. In the posterior discectomy patients, there was initial narrowing of the disc space at 3–6 months, and then widening occurred [8]. No patient required repeat surgery [8].

Celik et al. compared the surgical outcomes of 32 pediatric patients and 32 adult patients after lumbar microdiscectomy [15]. They concluded that after 3 years of

follow-up, all parameters were significantly better in the pediatric population with no recurrence of the disease [15]. Follow-up MRIs were performed during the second month postoperative visit, and all pediatric patients had significantly less epidural scarring [15]. Celik et al. opined possible reasons for this were the elastic component of disc in this group, low rate of mechanical back pain, no compensation or litigation issues, and rarity of psychological components, as well as the belief that better overall prognosis may limit surgical intervention [15]. Postoperative recovery was also quicker in the pediatric population [15]. There were four recurrent herniations in the adult group (and none in the pediatric), two of which required further surgery [15]. The clinical preoperative symptomatology was similar in both the adult and pediatric patients in this series [15].

In a study evaluating discectomy outcomes by Lagerback et al., 86% of patients 18 y/o or younger were satisfied with outcomes, whereas 78% of those 19–39 y/o and 76% of those 40 y/o or older were satisfied [12]. Adolescents had a longer period of back and leg pain preoperatively than the older age groups [12]. The number of repeat operations was 2%, 5%, and 4% in the adolescent, younger adult, and the older adult groups, respectively [12].

Cahill et al. evaluated the safety of 87 microdiscectomies by a single surgeon in patients 12–18 y/o [22]. Of these, 60% were female [22]. The mean duration of symptoms from onset until surgical treatment was 12.2 months [22]. Motor changes were present in 26%, sensory changes in 41%, and positive straight leg raise in 95% of cases [22]. There were 6% of the patients that needed repeat lumbar surgery, and 1% of patients had either a cerebrospinal fluid leak, new postoperative neurologic deficit, or infection [22]. They concluded the procedure was safe with a low complication rate in the pediatric population [22]. Similar to other studies, the vast majority of surgery was at the L4-L5 and L5-S1 levels [22]. Epidural injections were used in 32% of patients, with a mean of 2.1 injections [22]. However, Cahill et al. did not look at how often patients successfully responded to injections and did not require surgery [22]. They concluded that pediatric patients may not respond as well to nonsurgical treatment as adults, but conservative treatment should be attempted [22]. More than 70% of patients referred to the author of this series were treated nonsurgically, and less than 5% had a free fragment disc herniation [22].

Bradford et al. reported on 36 children and adolescents with a LDH whom underwent surgical treatment [14]. There were 61% of the patients that were male, and 53% had antecedent trauma [14]. Low back pain progressing to sciatica was observed in 60% of cases, or 25 patients [14]. There were 16 of the patients that underwent concomitant fusion, reason unspecified, and their outcomes were not different from those who did not undergo fusion [14]. The mean onset of symptoms until treatment was 10.7 months, and the median was 6 months [14]. Follow-up was 3–10 years [14]. There were 42% of the patients that had intermittent back discomfort after surgery; this was mild in 79% of cases [14].

Sarma et al. evaluated 32 patients, with a mean of 15.64 YOA [17]. There were 81% males [17]. Trauma was significant etiologic factor in 57.14% of cases [17]. Vertebral anomalies were present in 35.7% of patients [17]. Multilevel DDD was present in 28.6% of patients [17]. At long-term follow-up, 71.4% of patients were symptom-free [17]. The most common level treated was lumbar 4–5 [17]. Kumar

et al. found simple discectomy offers a good result in 92% patients and states trauma may be a predisposing factor in adolescent HNP [7]. However, only 16% had gross degenerative changes [7].

Currently, fusion for a primary LDH in the adolescent with or without RAF is not indicated without spondylolisthesis or other anatomic anomalies creating instability. While the studies on this topic are limited, the evidence illustrates that an overall positive recovery can be expected with surgical intervention after adolescent LDH. However, despite the satisfactory results of discectomy and RAF resection, one must not forget about the initial injury to the disc and consequent internal damage. Therefore, while many studies have good medium-term follow-up, few have followed the patients for decades. Hence, the consequence of this pathology over a lifespan is unknown.

Return to Play

In the elite athlete population, the return to play results are promising. A study found NFL linemen return rate was 81% after surgically treated HNP but only 29% after nonop treatment [37]. Watkins reported that 89% of professional athletes return to play after lumbar discectomy [2]. At 3, 6, 9, and 12 months postoperative, 50%, 72%, 77%, and 84% had returned [2]. Wang et al. found that 90% of elite athletes return to play at a high level of competition [38]. Two-level disc disease may be associated with a less favorable result [38]. A meta-analysis looking at return to play after lumbar microdiscectomy found that of 558 athletes, 83% returned to play [39]. Overley et al. found no difference in return to play rate of nonsurgical treatment, similar to the Professional Athlete Spine Initiative conducted by Hsu et al., although the two groups may have had clinically significant differences [1, 39].

While none of these studies looked at pediatric or adolescent athletes, the results seem promising. In evaluating the outcomes of 50 microdiscectomies in athletes 21 years of age or younger, we have found in our experience that 65.3% return to sport. However, as the young athlete ages, there is a normal attrition of their participation in competitive sports, and this needs to be considered in the outcomes (Table 16.1). Only 6.9% of high school football players go on to play at the college level, and only 1.6% of college players play at the professional level, as seen in Tables 16.1 and 16.2.

Postoperative restrictions vary with little empirical evidence. As the demands and expectations of returning to sport and the conditioning necessary for such elite athletes are higher than the general population, rehabilitation protocols have been developed specifically for elite athletes. Return to play after lumbar microdiscectomy varies on a case-by-case basis. Often the focus has been rehabilitation protocol advancement levels such as the Watkins protocol [2].

The senior author has discussed return to play with other surgeons who are actively involved in the care of elite athletes, and the consensus is approximately

Table 16.2 Collegiate to professional advancement. Estimated probability of competing in professional athletics

	NCAA participants	Approximate # draft eligible	# Draft picks	# NCAA drafted	% NCAA to major pro	% NCAA to total pro
Baseball	34,980	7,773	1215	735	**9.5**	–
M basketball	18,712	4,158	60	50	**1.2**	19.3
W basketball	16,532	3,674	36	34	**0.9**	4.9
Football	73,063	16,236	253	253	**1.6**	1.9
M ice hockey	4,199	933	217	60	**6.4**	–
M soccer	24,986	5,552	88	78	**1.4**	–

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3 months. There is also concern of late sequelae and deterioration of results postoperatively in athletes, despite an initial short-term benefit, especially when considering the increased demands of athletics on the body. The disappointment of a recurrent HNP cannot be overstated. Sport-specific issues also exist; for example, baseball pitching and batting create a repetitive torque that may have a negative impact on longer-term prognosis [40].

Expert Opinion

As we reviewed the material for this chapter, we found tremendous variation in the literature regarding the epidemiology and risk factors for lumbar disc herniations in the adolescent patient as well as in athletes. As we discussed in the chapter, the symptoms in the adolescent population are often atypical, and patients are often referred to our senior author by hip arthroscopists and other subspecialists after the diagnosis has been missed for a protracted time period. The patients, and more often the parents, are frustrated as the adolescent has often missed out on a year or more of high school sports and other activities. As specialists, we need to continue to educate our colleagues on the diagnosis and emphasize that a level of suspicion is necessary.

Our senior author practices in a large orthopedic group with an emphasis on sports medicine and has performed over 50 discectomies in adolescent athletes ranging from recreational to the elite level. The vast majority felt they could have returned to sport at their pre-injury level. However, because of the natural attrition of athletes to higher levels of play as they advance in years, compounded by many of our patients being high school juniors or seniors, it is difficult to discern the actual numbers of careers that have been shortened by this injury.

It cannot be emphasized enough that while the surgical approach is the same as in the adult population, the epidural exploration is typically far different. Our senior

author has found the adolescent discectomy cases tend to be more technically demanding as large free fragments are the exception, and the contained neuro-compressive nuclear disc material is quite tenacious, requiring annulotomy, and at times, it can be difficult to remove. As discussed in the chapter, the surgeon must be aware of neuro-compression by a RAF and be ready to excise this lesion when necessary. Intraoperatively, we usually use Kerrisons and/or reverse curettes to resect the neuro-compressive lesion, while a highly qualified assistant retracts the nerve root and thecal sac.

Pushing the compressive lesion ventrally into the disc space and then retrieving it is often a good technical option.

Whether it be a disc herniation, RAF, or a combination of both, the surgical prognosis and return to sport are initially promising, but the long-term follow-up and effects on returning to all activities later in life are not fully understood.

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Chapter 17
Spondylolysis and Spondylolisthesis in Athletes

Koichi Sairyo, Toshinori Sakai, Yoichiro Takata, Kazuta Yamashita, Fumitake Tezuka, and Hiroaki Manabe

Etiology

Lumbar spondylolysis is the most common pathology identified in adolescents with chronic back pain. It has been widely accepted that lumbar spondylolysis is a stress fracture of the pars interarticularis that commonly appears in children and adolescents [1–3]. Alternatively, genetic predisposition is another theory in the etiology of lumbar spondylolysis. In 1975, Wiltse et al. stated that a pars fracture (spondylolysis) is a fatigue fracture based on a strong hereditary basis [4]. We have seen evidence of this genetic predisposition in our own practice. Figure 17.1 demonstrates three separate cases of lumbar spondylolysis from three brothers [5].

In 1978, Haukipuro et al. reviewed the pedigrees of spondylolysis families and concluded that inheritance of lumbar spondylolysis is autosomal dominant [6]. Finally, Cai et al. [7] found a possible gene associated with spondylolysis. Future studies are likely to identify specific genetic alleles that predispose patients to pars fractures.

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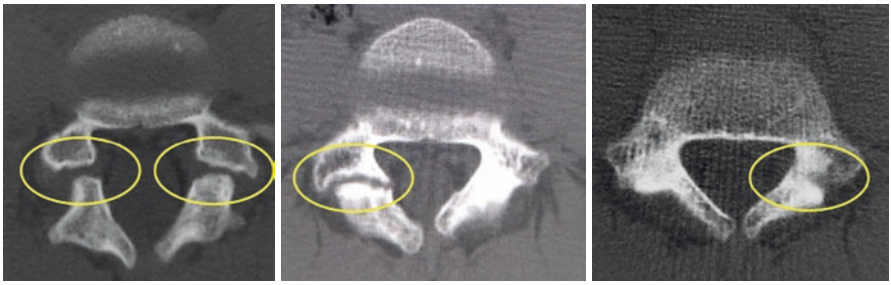

Fig. 17.1 Three cases of spondylolysis from one family

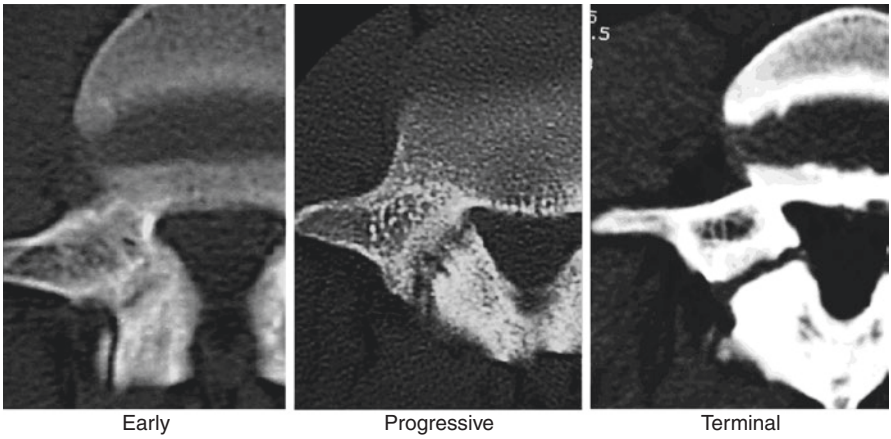

Fig. 17.2 CT stages of the pars fracture

CT Stage Classification

A key component to the diagnosis and treatment of lumbar spondylolysis in our practice relies on CT stage classification. Figure 17.2 demonstrates the CT stages of lumbar spondylolysis [8, 9]. The heart of this classification relies on a baseline understanding of fracture healing. As the pars fracture develops, it will ultimately undergo changes that will lead to union or non-union. These unique stages in healing will lead to varying clinical presentations and treatments in patients with spondylolysis. Bone absorption is seen in the early stage and is demonstrated as an incomplete fracture on sagittal reconstruction CT scan. The progressive stage shows evidence of a complete fracture of the pars without sclerotic fracture margins. The terminal stage is equivalent to a pseudoarthrosis and demonstrates sclerotic fracture margins and blunting of the fracture edges.

In our classification schematic early- and progressive-stage defects are designated as acute pars fractures. These acute fractures still have the opportunity to

form a bone union under the correct biomechanical circumstances. The terminal CT stage is classified as a chronic pars fracture, since it is a pseudoarthrosis. Once a fracture is in this stage, it will never progress to a union, and this influences management.

Early Diagnosis of the Pars Fracture

It is very difficult to diagnose the early stage using plain radiographs. For the accurate diagnosis of the early-stage defects, we have proposed two hallmark findings: bone marrow edema of the adjoining pedicle on MRI [3] and bone absorption at the caudal aspect on the sagittal reconstructed CT scan [10]. Figure 17.3 demonstrates a CT scan and T2-weighted MRI for a patient with the early-stage defect. Even though the fracture is not clear on CT (left panel), bone marrow edema in the adjoining pedicle is clear on MRI (right panel). We have found that assessment of these early-stage defects on CT scan is more readily identifiable on the sagittal reconstructed CT scan. In Fig. 17.4, we present three cases of the early-stage defects. As you can see, the pars fracture is most identifiable at the caudal aspect of the pars interarticularis. This area should be scrutinized on an adolescent presenting with back pain and a CT scan. This inferior aspect of the pars is especially vulnerable to stress fracture development due to the high concentration of mechanical stress during lumbar motion, which has been proven using the finite element analysis [10]. Technetium (Tc-99 m) single-photon emission CT often is used to identify acute lesions in athletes for whom the clinician has a high suspicion for spondylolysis in the setting of negative results on plain radiography, but this imaging modality can expose patients to high levels of radiation.

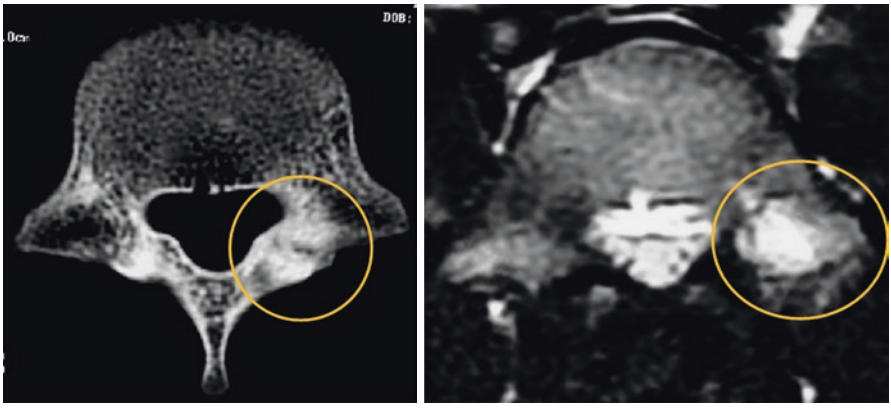

Fig. 17.3 Early-stage defects with pedicle marrow edema

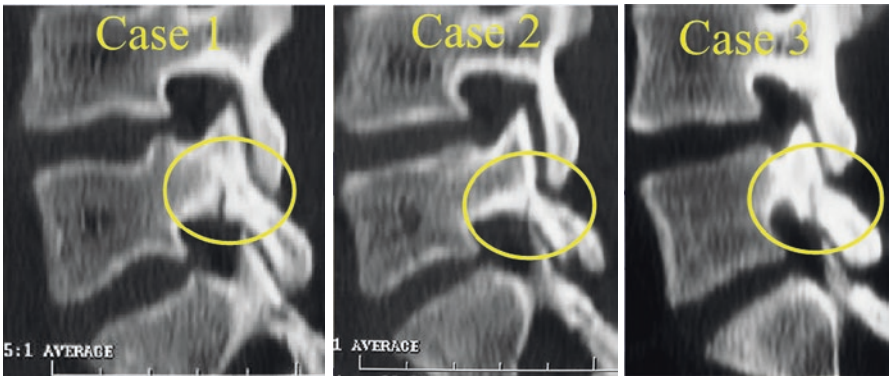

Fig. 17.4 Sagittal reconstructed CT scans in the early-stage defect

Although MRI historically has not been recommended for detecting pars defects, more recent evidence suggests that specific sequences can enable successful detection in up to 98% of patients with pars defects. In totality, this information has led us to recommend MRI as the first-line imaging in a patient suspected of spondylolysis. Diagnosis of progressive and terminal stages of pars fractures is readily identified on advanced imaging, either MRI or CT scan. If the suspicion is still high for a pars fracture after a negative MRI, then a bone scan should be ordered.

Pain Mechanism

For each stage, the pain mechanism is different. Therefore, the goal of conservative treatment is also different. For the early and progressive stages, pain is due to an acute fracture, which is obvious on STIR-MRI as marrow edema and/or extra-osseous bleeding (edema) (see Fig. 17.3).

Figure 17.5 presents two cases that plainly illustrate the difference in the stage of fracture healing and therefore pain mechanism. The left pars in Case 1 and right pars in Case 2 can both be classified as the progressive stage. The CT scan shows a complete fracture without overt blunting of the fracture margins. The associated MRI findings are in the right panels. Once again, the left pars in Case 1 and right pars in Case 2 demonstrate marrow edema and extra-osseous edema consistent with the progressive stage. This is in direct contrast to the right pars in Case 1 and the left pars in Case 2 which demonstrate the radiographic characteristics of the terminal stage. In these pars, the fracture edges are clearly blunted on CT scan, and there is no marrow edema or extra-osseous bleeding. These images clearly show that although each patient has bilateral spondylolysis, the classification of each particular pars fracture can be unique. In the progressive stage, the edema indicates a more acute fracture that stands a chance at union. In the acute fracture, inflammation is

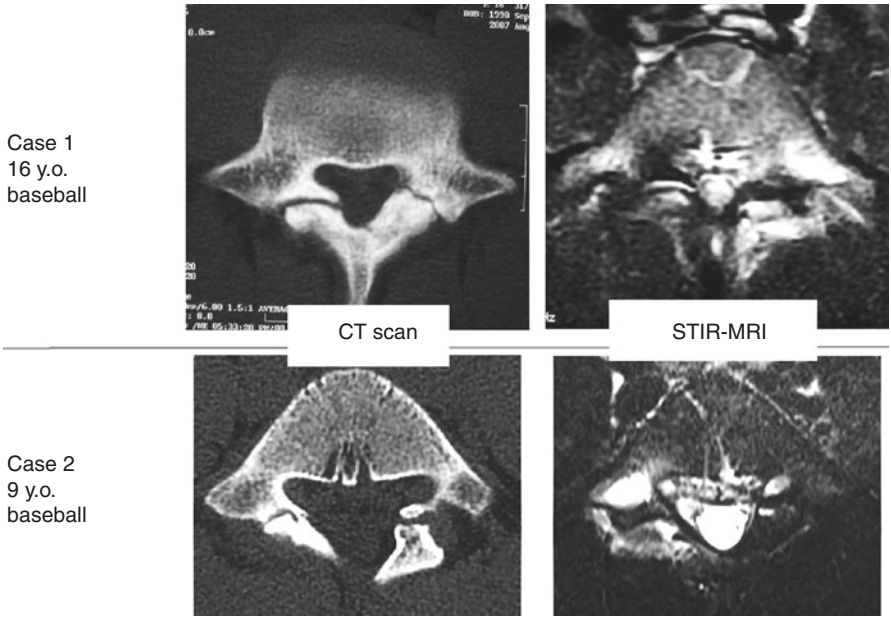

Fig. 17.5 Painful defects on STIR-MRI [11, 12]

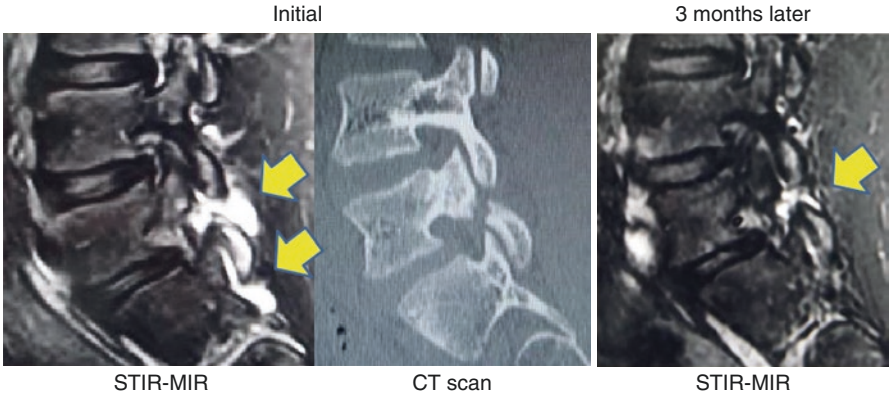

Fig. 17.6 Communicating synovitis of facet joint (14-year-old soccer player, male)

the pain generator. The terminal stage has no edema, and the pain generator is communicating synovitis from pseudoarthrosis [13].

Figure 17.6 demonstrates the typical MRI findings of communicating synovitis in terminal-stage spondylolysis. Effusion is obvious in the defect and adjoining facet joints (yellow arrows). With conservative management, low back pain can be decreased, and the effusion due to synovitis can subside. The decreased effusion is obvious in the STIR-MRI taken 3 months after conservative treatment.

Slippage Mechanism

Regarding slippage in spondylolytic spines (spondylolisthesis), it has been well reported that slippage is very common in children and adolescents and very rare after the skeletal maturation [14–16]. Seitsalo et al. followed 272 children with spondylolysis and found that in age groups of early puberty (girls, 9–12 years; boys, 11–14 years), slippage was likely to progress [15]. Our data is in good agreement with them [16]. We followed 46 pediatric patients aged under 18 years. The mean follow-up period was 6 years. We evaluated correlation between their skeletal age and progression of slip. As shown in Fig. 17.7, skeletal age of the spine can be evaluated by the condition of the secondary ossification center (SOC) of the vertebral body. In the cartilaginous (C) stage of the spine (Fig. 17.7, left panel), SOC is cartilage and cannot be seen on a plain radiograph. The SOC is ossified and is visible at the apophyseal (A) stage (Fig. 17.7, middle panel). Finally, the ossified SOC is fused to the vertebral body; and this defines the epiphyseal stage (Fig. 17.7, right panel). We reviewed the progression of spondylolisthesis to skeletal maturation. The most prevalent stage regarding slippage was found to be the C stage. From stage C to A, 80% of patients showed slip progression. On the contrary, after maturation, there were no slip progressions. Thus, surgeons should be cautious of slip progression in patients in the cartilaginous stage; this corresponds roughly to elementary school age.

The pathomechanism associated with slippage in the immature spine was analyzed by Sairyo and co-workers using calf [17, 18] and rat models [19, 20]. The growth plate in the immature spine is located between the vertebral body and the SOC. This area is a weak point and fails under the biomechanical stress experienced after spondylolysis. Figure 17.8 demonstrates the separation of the growth plate and

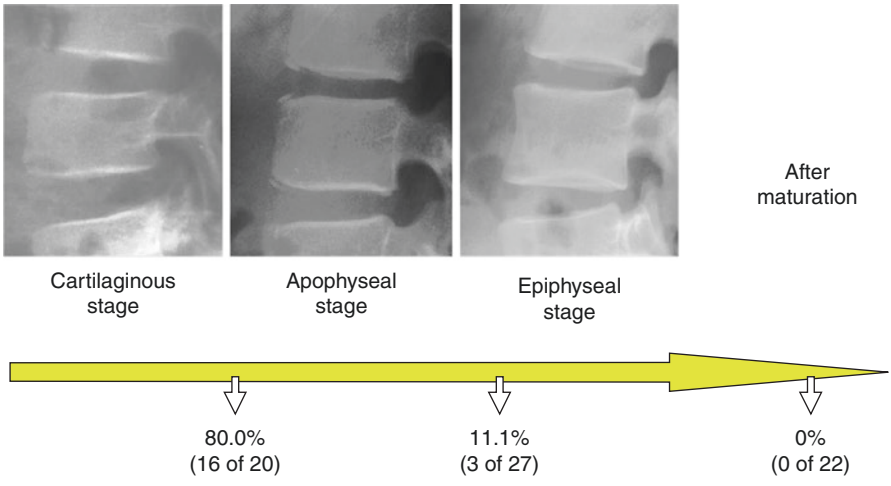

Fig. 17.7 Slippage with the skeletal age

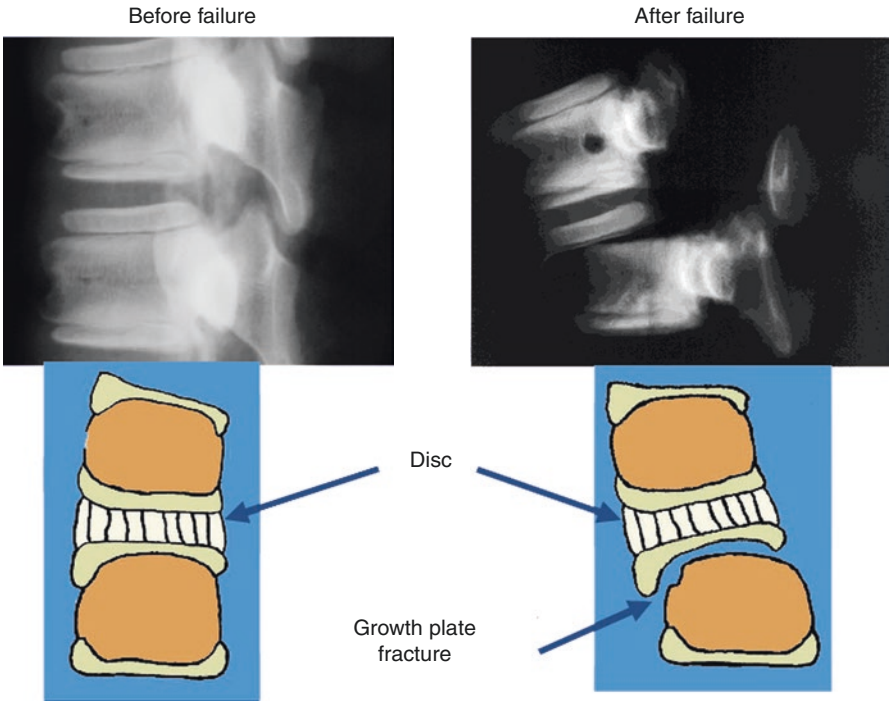

Fig. 17.8 Calf spine slippage model

the location of pediatric spondylolisthesis pathoanatomy. This explains why progression of the slip is common in children and adolescents. After skeletal maturation, the growth plate disappears; and the weak point for slippage also disappears.

Treatment Strategy for Elite Athletes in Children and Adolescents

There are two goals of conservative treatment. For the acute pars fractures (early and progressive stages), bone union is still possible. We therefore attempt to achieve bone healing with the use of a hard brace. Figure 17.9 demonstrates a case where bone union was achieved after 6 months of conservative treatment. We have shown that in general, it takes 3 months for the early stage and 6 months for the progressive stage to achieve bone healing as shown in Fig. 17.10 [9].

For the chronic pars fracture (terminal stage), pain management is the goal because there is no possibility of bone union for pseudoarthrosis. Again, the pain mechanism at this stage is synovitis of the pars defects and adjoining facet joints. Treatment for this pain is focused on anti-inflammation of synovitis. Lumbosacral soft brace is usually used to prevent extension during performance. When pain persists, steroid infil-

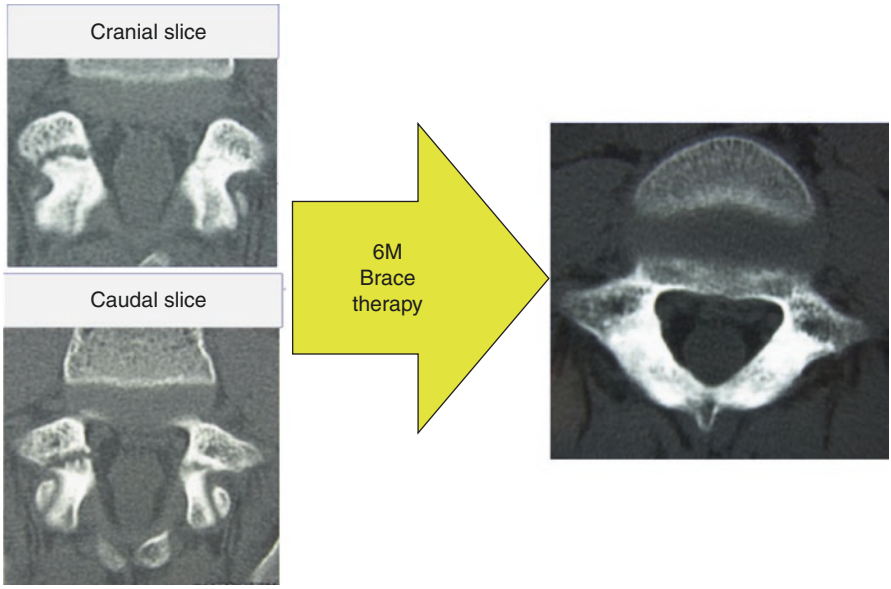

Fig. 17.9 Bone union with the conservative treatment

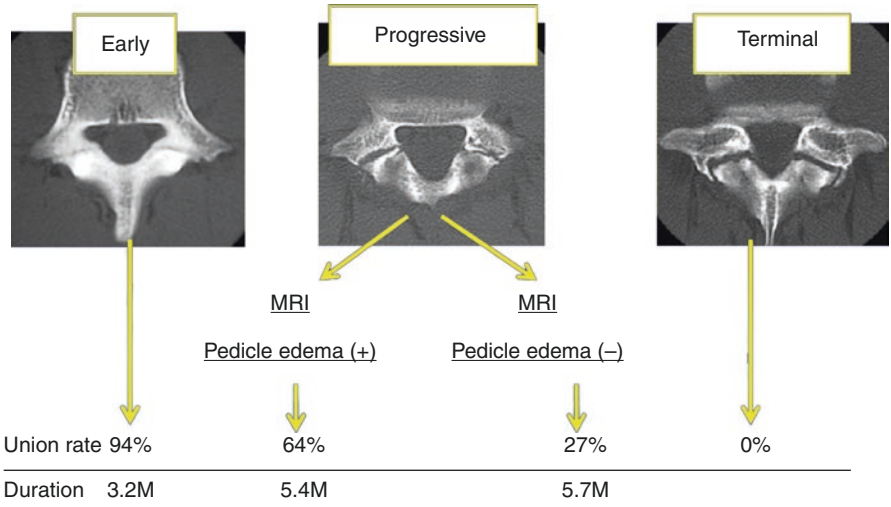

Fig. 17.10 Bone union with the conservative treatment

tration in the defects and intake of NSAIDs are other options. Almost all pediatric patients can return to the baseline activity with such conservative care.

The concern when pediatric patients with pars fracture return to the original activity is slip progression. Progression of the slippage should be carefully checked in the pediatric immature spine [14–16]. We recommend performing and evaluating lateral plain radiographs two to three times a year to check the status of the slippage, deformity, and skeletal age until the spine reaches maturation.

Treatment Strategy for Adult Elite Athletes

Acute pars fractures tend to occur in children and adolescents. An acute adult pars fracture would be considered very rare. Thus, most of cases of spondylolysis in adult athletes are chronic (pseudoarthrosis). The chronic pars fractures in adults are mostly painless. Rarely, the chronic pars fracture can become a pain generator, which would be due to synovitis at pseudoarthrosis. Similar to pediatric patients, steroid infiltration in the defects and intake of NSAIDs are effective for such pathology.

We have experienced 11 cases of acute pars fractures in adults [21], and all of them were very active athletes. Basically, pain management is the treatment strategy regardless of the stage. The biggest difference from pediatric patients is that the adult elite athlete cannot be expected to endure long-term conservative treatment due to short career lengths and reliance on performance for salary.

Figure 17.11 demonstrates a male case of bilateral pars fractures in the adult (20 years old). Although the left pars showed a progressive stage and it had a possibility of bone healing with conservative treatment, we selected just pain management. We decided the time required to achieve a union was not appropriate for this elite athlete. One month later, the pain disappeared, and thereafter he participated in the Olympic Games 2012 in London.

Recurrence (Refracture After Union) and Prevention

Sakai et al. [22] reviewed 63 pediatric cases with lumbar spondylolysis. Results showed that in the very early stage, the bone healing rate was 100%; in the early stage, it was 93.8%; and it was 80.0% in the progressive stage. Surprisingly, the recurrence (re-pars fracture) rate was 26.1%. They stated that physical therapy before return to the sport may prevent and/or decrease recurrence.

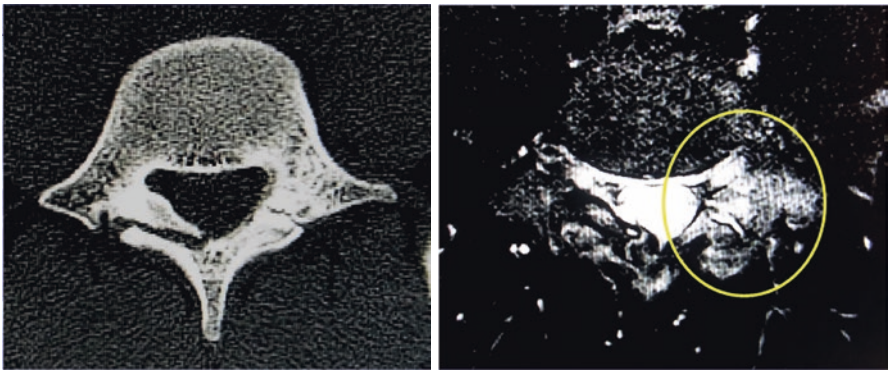

Fig. 17.11 Adult-onset acute pars fracture: 20-year-old male, track and field. RT: chronic (terminal stage). LT: acute (progressive stage)

We believe that the joint-by-joint theory is the most effective physical therapy in reducing the mechanical stress experienced by the lumbar spine during sport [23]. This theory relies on mobilization of the hip joint and thorax to prevent recurrence. Thus, we recommend stretching of the hamstrings, quadriceps, and thoracic spines. For the purpose of efficient stretching, we propose active stretching utilizing reciprocal inhibition [24] and stabilization of the trunk core muscles [25].

Operative Management

Spondylolysis is clinically benign, and more than 90% of the athletes with the disorder can return to the original activity with conservative treatment [26]. However, in certain cases pain management is not effective with conservative treatment, and surgical intervention is required. In general, three surgical methods have been reported: direct repair, segmental fusion, and decompression. For athletes, the direct repair of the pars fracture is favorable. There have been a variety of maneuvers for direct repair such as Scott wiring [27], Buck screwing [28], pedicle screw hook rod [29, 30], V-rod [31], and smiley face rod method [32].

For very active athletes, we have been recommending minimally invasive "smiley face" rod method [32] using percutaneous pedicle screw system [30]. Figure 17.12 demonstrates radiographs of the direct repair surgery using the "smiley face" rod method for a professional tennis player. First a 4–5 cm midline skin incision is made; then, removal and decortication of pseudoarthrosis are performed. Two small skin incisions are made bilaterally as shown in the figure. The percutaneous two pedicle screws are inserted under the guidance of the fluoroscope. Via the midline skin incision, a U-shaped rod is inserted underneath the spinous process,

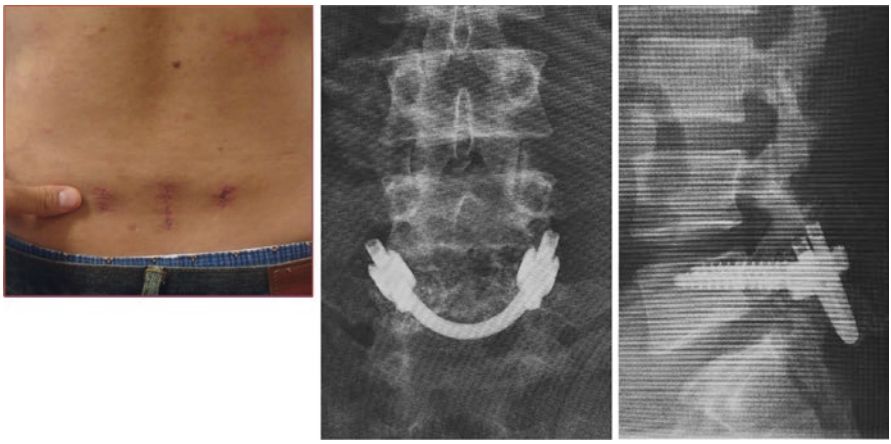

Fig. 17.12 Direct repair of the pars fracture using the smiley face rod method: 30-year-old professional tennis player

and the end of the rod is secured with the screw heads; then, stabilizing the loose lamina, finally on-lay bone graft is made.

Usually, 6 months after the surgery, moderate sports are allowed; however, complete return to the field would be 1 year after the surgery.

Conclusion

Here, we described how to manage spondylolysis and spondylolisthesis in athletes. The skeletal age and CT stage of spondylolysis dictate treatment strategy. Before returning to sports, effective physical therapy is recommended based on the joint-by-joint theory.

Expert Opinion

We believe that classification of the pars fracture based on CT stage yields important information on the etiology of the pain mechanism and thus drives treatment. Early defects are best found on MRI with assessment for edema in the adjacent pars. Slippage is the long-term sequela to be avoided. Evidence has shown that the immature spine is more prone to slippage, and therefore routine surveillance is required. In general, lumbar spondylolysis is a benign disease, and pain management in the athlete is an effective strategy. In the rare case that requires operative intervention, we favor techniques that lead to direct pars union and spare spinal segment fusion in young active individuals.

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Chapter 18
Traumatic Lumbar Injuries in Athletes

Elizabeth P. Davis, James E. Showery, Mark L. Prasarn, and Shah-Nawaz M. Dodwad

Introduction

The incidence of lumbar injuries in athletes is highly variable and dependent on the specific demands of the sport involved. Within the National Collegiate Athletic Association (NCAA), the rate of lumbar spine injuries varies from 24.62 injuries per 1000 athletic exposures in men's football to less than 2 injuries per 1000 athletic exposures in women and men's cross-country swimming [1]. Certain weight lifting movements have been found to increase intervertebral disc pressure beyond the normal physiologic range and may be associated with increased rates of degenerative disc disease [2–4]. Athletes that participate in contact sports are frequently subjected to sudden violent forces transmitted to the spine in a multitude of directions. Football linemen, for example, experience high-magnitude shear and compression forces in excess of thresholds necessary to cause pathologic changes to the intervertebral disc and pars interarticularis [5]. High-velocity extreme sports such as skiing, high diving, and motocross racing can result in devastating spinal cord injuries in the setting of poly-trauma [6–9]. A thorough history and physical exam are critical to identifying both acute and chronic injuries in athletes. When appropriate, obtaining advanced imaging is often necessary to make an accurate diagnosis [10–12]. A multidisciplinary approach is critical to treatment compliance and success as each athlete will have unique expectations, performance goals, and outside pressure.

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Lumbar Anatomy

When evaluating an athlete for spine pathology, comprehensive knowledge of anatomy is necessary to obtain and interpret imaging and diagnostic tests. By understanding at-risk structures, the location of neurologic elements, and components of instability, one can avoid unnecessary imaging and arrive at an accurate diagnosis. Injuries can be isolated to the bony anatomy including the joints but can also involve the surrounding muscles and soft tissues, specifically discoligamentous injuries. When an excessive force is placed across the spine in an inopportune vector, injury can occur. More significant pathology occurs when a discoligamentous injury occurs, for example, the posterior ligamentous complex (PLC) is disrupted which includes the facet joints, the interspinous ligament, the ligamentum flavum, the supraspinous ligament, as well as the thoracolumbar dorsal fascia. Injury to the PLC can result in spinal instability and neurologic deficits. The thoracic spine is inherently more stable than the lumbar spine due to the rib cage. The thoracolumbar junction is a transition zone from the rigid thoracic spine to the mobile lumbar spine and is therefore more susceptible to injuries. Solitary anterior column injuries are generally stable injuries, injury to the PLC has significant instability, and fracture dislocations can result in devastating injuries with neurologic deficits.

Evaluation

Chronic low back pain is associated with both very high and low activity and load bearing [13, 14]. The incidence of mechanical low back pain in high-level athletes ranges significantly from 24% to 81% depending on the sport in question [15–17]. Additionally, the total time participating in a specific sport or activity as well as the unique demands of each sport should be carefully considered for each athlete presenting with thoracolumbar back pain. Activities that involve hyperflexion, hyperextension, or axial rotation under load such as gymnastics, American football, soccer, baseball, and hockey have been associated with spondylolysis, intervertebral disc herniations, and early degenerative changes on imaging studies of the thoracolumbar and lumbar spine [12, 18–23]. Furthermore, contact sports in particular appear to be associated with degenerative disc disease, facet arthropathy, and chronic low back pain in a dose-dependent relationship [20–23]. When evaluating an athlete after acute injury, consideration of the injury mechanism as well as the athlete's position during the injury will facilitate the differential diagnosis and guide diagnostic testing.

As with any patient presenting with back pain or injury, obtaining a detailed history is fundamental to arriving at an accurate diagnosis. Athletes with acute back pain should be assessed for preexisting back pain and history of other musculoskeletal injuries. Extremity injuries, muscular imbalance, and pain can disrupt the

kinetic chain and lead to poor trunk control, predisposing athletes to lumbar pain and injury [24]. Psychosocial factors should also be considered in athletes with back pain and injury. Careful questioning regarding strength deficits during athletic activity can help elucidate subclinical weakness from nerve root compression or deconditioning.

It is important to take a detailed history regarding the mechanism of injury, symptoms, alleviating and aggravating factors, as well as treatments the patient has tried. Mechanism of injury can alert the physician to structures that are injured. Extension-type mechanisms stress the posterior elements such as the pars interarticularis, flexion-type mechanisms stress the anterior column as well as place tension on the posterior ligamentous complex, rotational injuries can strain the facet joints or musculature, and focal posterior pain over the sacroiliac (SI) joint can indicate SI joint pain. Red flags during examination are saddle anesthesia, progressive neurologic deficits, urinary retention, and fecal incontinence as these can indicate cauda equina syndrome, which is a surgical emergency. Rapid weight loss, night pain, and nonmechanical back pain are concerning for infection or neoplasm. While screening for positive red flag findings should be performed, interpretation should be within the context of the patient's entire clinical picture, and further testing should be carefully selected [25–27].

Physical Exam

Athletes sustaining lumbar injuries may be initially evaluated at the site of injury on the playing field, in the emergency room or hospital setting, or in clinic. Each setting necessitates a different approach. The goals of an on-field evaluation should be to determine if a serious injury has occurred and if removal from the sporting event is necessary.

Initial evaluation on the scene should begin with advanced trauma life support (ATLS) protocol. Airway, breathing, and circulation should be assessed first. Next, the patient should be carefully positioned supine with uniform and helmet left in place. Neck and back pain should be assessed by performing brief neurologic examination assessing for weakness, decreased sensation, and paralysis of the extremities and trunk. If the patient is found to have neurologic symptoms or severe neck or back pain, they should be emergently transferred to the nearest trauma center equipped to evaluate and manage spine trauma. Full spine precautions with cervical spine immobilization and careful transportation with rigid backboard should be maintained during transport. When dealing with a critically injured football player on field, not requiring CPR/airway control, the helmet and padding should generally be left in place, and a seven-member team, with one person maintaining cervical alignment, should lift the player onto the moving board/stretcher [28–31]. If the athlete is neurologically intact and has no axial neck or back pain, they may be allowed to return to play. If the athlete endorses mild neck or back pain without neurologic symptoms, they should be removed

from the event and obtain prompt evaluation in a clinical setting without distractions. Depending on the mechanism of injury, the patient should be screened for concussion by a qualified athletic trainer or team physician [32].

In the clinical setting examination should include inspection, palpation of the entire spine, and observation for asymmetry, swelling, ecchymosis, step offs, or gaps. It is important to know of any baseline preexisting deformity such as kyphosis or scoliosis. Palpation can help localize the injury as well as identify muscle spasm of the paraspinal musculature. Patients with high-energy thoracolumbar spine trauma and disruption of the posterior ligamentous complex will often have noticeable swelling over the area of injury, and step-off or gaps between spinous processes may be identified. A complete neurological examination including extremity motor strength, sensation to light touch, proprioception, and deep tendon reflexes should be performed. Pathologic reflexes such as Hoffman sign and Babinski reflex should be assessed. Straight leg raise and contralateral straight leg raise can help diagnose intervertebral disc herniation with nerve impingement.

Imaging

Athletes with generalized paraspinal muscular lumbar pain without neurologic symptoms generally respond to nonsteroidal anti-inflammatory drugs (NSAIDS) and physical therapy focused on core strengthening. In this setting, plain AP and lateral X-rays are sufficient. Routine anterior-to-posterior (AP) and lateral views can alert the physician of any disc height loss, spondylolisthesis, compression/burst fractures, and other sagittal or coronal malalignment (Fig. 18.1). Oblique views can be obtained to assess the pars interarticularis, and flexion-extension views can be done to assess dynamic instability. Any signs of fracture or instability on plain X-rays warrant advanced imaging with CT and/or MRI. Injury in one level of the spine necessitates imaging of the entire spine as 17–20% of these patients will have non-contiguous spine fractures [34–37].

When back pain does not improve after treatment with rest, NSAIDS, and physical therapy, or if presenting with neurologic deficit or following significant traumatic situations, then advanced imaging is warranted. Also, when dealing with thoracolumbar pain/injury in high-level athletes, advanced imaging is usually required. Advanced imaging includes computed tomography (CT) and/or magnetic resonance imaging (MRI). CT is best for detailing bony anatomy; MRI is best for detailing soft tissue and neural anatomy. CT is best for identifying pars defects and fracture as it allows for high-fidelity evaluation of complex three-dimensional bony anatomy of the spine. In an athletic injury patient, MRI is useful to evaluate for neural compression, posterior ligamentous complex injury, or an occult stress fracture. CT and MRI can both be used to determine if the patient has a pars injury or pars defect [19, 38]. A 1.5T MRI using short tau inversion recovery (STIR) and T2-weighted imaging can show bony edema or impending fracture/stress reaction.

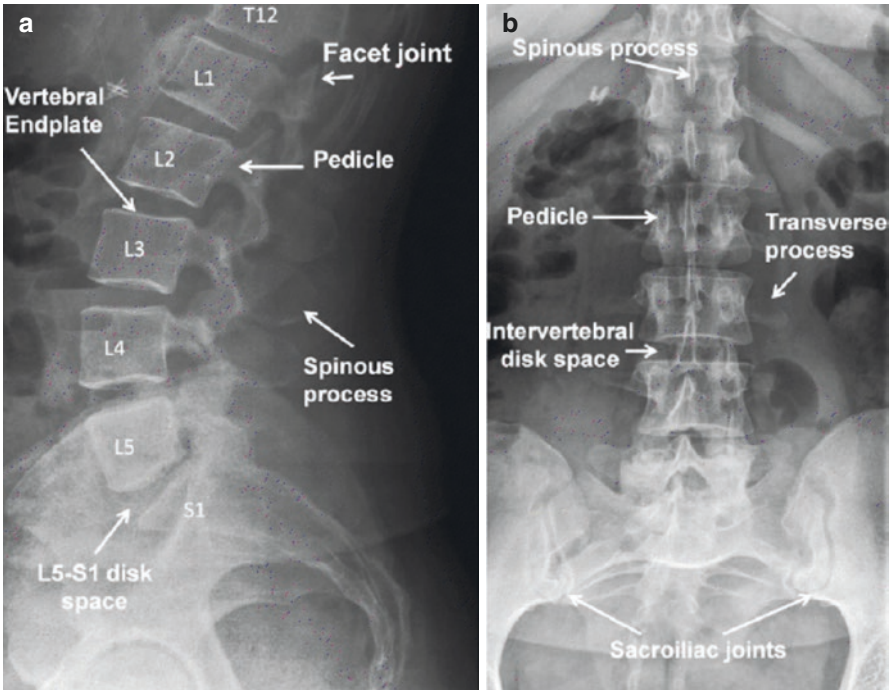

Fig. 18.1 Lateral and AP radiograph of the lumbar spine with relevant anatomy labeled. (From: Valladares-Otero [33]. Reprinted with permission from Springer Nature) (**a)** X-Ray Lateral View Lumbar Spine. (**b)** X-Ray Anterior-Posterior View Lumbar Spine

Lumbar Injuries

The most common lumbar injury in the athlete is a simple lumbar strain, accounting for 54% of reported lumbar spine injuries in collegiate athletes [1]. This can range from a simple bruise to a traumatic tearing or shearing of muscle fibers in the paraspinal muscles or core musculature that supports the thoracolumbar spine. The patient will usually have posterior paraspinal tenderness that is non-radiating. Pain is worsened with activity and range of motion and improves with NSAIDs and rest. Contact injuries to the paraspinal musculature may result in similar symptoms but are frequently associated with bruising or abrasions overlying the injury. Athletes that sustain injuries with a hyperflexion moment may injure the supraspinous and interspinous ligaments and will have midline pain over the spinous processes of the affected area. Splaying of the spinous processes can be seen on routine lateral XR if a significant posterior ligamentous complex injury has occurred.

Athletes with a blow to the gluteal region can develop inflammation of the sciatic nerve that is independent of lumbar disc pathology. Patients are tender to palpation deep in the gluteal region over the piriformis muscle, and pain can be recreated by

forced internal rotation of the thigh or with resisted abduction and external rotation. These tests put the piriformis muscle on tension and irritate the sciatic nerve. MRI will demonstrate no lumbar disc pathology despite radicular symptoms from piriformis aggravation.

The lumbar intervertebral discs are commonly injured in football players as well as those undergoing repetitive axial loading of the spine [3, 4, 20–23]. The annulus fibrosis and nucleus pulposus are affected by rotation and axial loading, respectively. An acute traumatic injury to the spine or chronic repetitive microtrauma can damage the annulus fibrosis. Small rents in the annulus fibrosis may develop allowing the nucleus pulposus to herniate, potentially causing nerve root compression and radiculopathy. Most commonly, herniation of the nucleus pulposus occurs in the posterolateral aspect of the disc where the posterior longitudinal ligament thins. Low back pain with radicular pain can indicate a herniated disc. Most symptomatic disc herniations occur at L4-L5 or L5-S1. It is imperative to rule out cauda equina syndrome when large lumbar disc herniations are identified. L4-L5 posterolateral disc herniations affect the L5 nerve root and can cause pain and sensory deficits to the lateral leg and dorsum of the foot with weak great toe extension. L4-L5 far lateral disc herniation affects the L4 nerve root and causes anterior shin pain and numbness with weakness to ankle dorsiflexion. L5-S1 posterolateral disc herniations affect the S1 nerve root and cause radicular pain and sensory deficits on the posterior aspect of the leg into the plantar aspect of the foot with weakness to plantar flexion. L5-S1 far lateral disc herniations affect the L5 nerve root and present the same as stated above. Toe-off weakness and slapping gait are subtle findings of motor deficit that may be difficult to detect on physical exam in very strong athletes.

Isolated facet injuries are rare and typically occur in the lumbar spine. Increased motion in this region as well as the sagittal orientation of the facet joints makes these structures more vulnerable to injury compared to the thoracic spine. Facet fractures are usually the result of violent rotational forces, such as those that occur in contact and extreme sports. Most athletes report axial and paraspinal pain that is made worse with flexion, extension, and rotation. Fortunately, isolated facet injuries rarely result in neurologic injury or instability, respond well to conservative treatment, and are unlikely to cause long term pain or disability.

Spondylolysis is a common cause of lumbar back pain in athletes. Traumatic spondylolysis may occur with forced hyperextension with axial loading and/or rotational forces and will typically result in acute low back pain and point tenderness over the area of injury. In contrast isthmic spondylolysis typically occurs in growing adolescent athletes as a result of repetitive microtrauma from hyperextension and rotational forces as well other genetic and environmental factors [39–43]. Additionally, McClellon et al. found 63% of adolescents with a new diagnosis of symptomatic spondylolysis had deficient or insufficient serum vitamin D levels [44]. For this reason, we recommend routine testing for vitamin D deficiency and supplementation for all patients with symptomatic pars interarticularis defects.

The posterior elements are the primary restraint to anteriorly directed shear forces in the lower lumbar spine [39, 42, 43]. Defects in the pars intraarticularis disrupt the osseous anchor between the effected vertebra and caudal levels and may

lead to instability and anterolisthesis of the effected level. Although most symptoms will improve with time and most patients will follow similar course of degenerative changes as the general population, some patients will experience persistent instability and progressive anterolisthesis [45]. Significant instability will frequently result in back pain that is made worse with extension activities as well as stranding from a seated position. Additionally, patents with high grade anterolisthesis, congenital narrowing of the spinal canal, or hypertrophic extraneural tissue from degenerative processes may report symptoms of neurogenic claudication or radiculopathy of the exiting nerve roots. These patients may also have associated paraspinal muscle spasm and posterior chain contracture that alters kinematics an contributes to disability.

Acute fractures of the lumbar spine are rare, potentially devastating injuries. As with long bones, classically described morphologic patterns of these injuries are dependent on the mechanism of injury. Additionally, the three-column theory of spine stability is a useful conceptual framework for lumbar and thoracolumbar trauma (Fig. 18.2). Compression fractures of the superior end plate typically occur with low energy axial loading in flexion [46, 47]. High energy axial loading in neutral or extended alignment typically results in burst fractures of the vertebral body

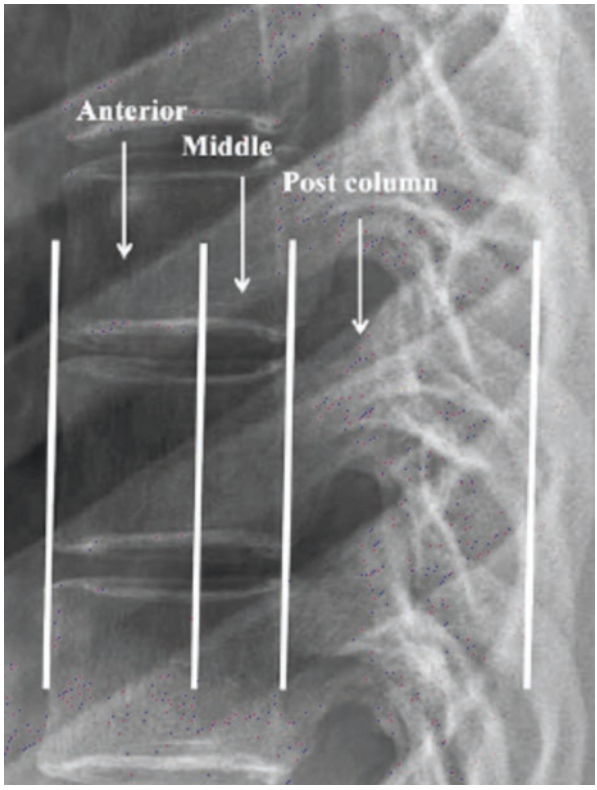

Fig. 18.2 Magnified view of the thoracolumbar spine with lines demarcating the three-column theory of spinal division. (From: Valladares-Otero [33]. Reprinted with permission from Springer Nature)

[46–49]. Violent flexion or extension mechanisms accompanied by distraction and/or torsional forces tend to produce three column injuries that are highly unstable [46]. Most fractures can be treated conservatively with bracing and monitoring, particularly if there is low concern for injury of the posterior ligamentous complex. Fractures that demonstrate instability on advanced imaging or upright plain radiographs as well as those with associated neurologic deficits usually warrant operative fixation with or without decompression and or fusion [50]. The senior authors routinely use the AO Spine Thoracolumbar Spine Injury Classification System (TLICS) to help guide operative decision making for lumbar and thoracolumbar fractures in this population. In general, when high energy lumbar spine injuries occur, standard trauma principles should be applied to decompress neural elements when necessary and provide adequate fixation correct and prevent deformity.

At times, athletes may present with chronic sacroiliac (SI) joint pain. These patients present with low back and posterior hip pain that is worse with activity and improves with rest. Any athlete or adolescent patient presenting with these symptoms should be evaluated for hip pathology as well. Adolescents that are overweight or have certain genetic and metabolic disorders are at risk for developing slipped capital femoral epiphysis (SCFE) and AP and frog-leg lateral imaging should be obtained of both hips to rule out this diagnosis. Additionally, patients with pain elicited by hip flexion, adduction, and internal rotation (FADIR) are likely to have intra-articular hip pathology whereas hip flexion, abduction, and external rotation (FABER) stresses the ipsilateral SI joint. SI joint pain in general and arthritis in particular may be the first symptom of ankylosing spondylitis (AS), psoriatic arthritis, or other inflammatory spondyloarthropathies and these patients should be referred to primary care or rheumatology for a complete rheumatologic workup. Sacral stress fractures should also be included in the differential diagnosis of caudal low back pain, particularly when the female athlete triad is suspected [51–54].

Management

Nonoperative Treatment

Most soft tissue injuries respond well to conservative treatment. Rest, ice, and non-steroidal anti-inflammatory medications should be initiated immediately after an injury has occurred to control the acute inflammatory response. Patients with neurogenic pain and radiculopathy due to nerve root compression will often benefit from gabapentenoid medications such as pregabalin or gabapentin, where pregabalin appears to be more effective [55–59]. Although gabapentin and pregabalin are effective for some patients, approximately 10-20% of patients discontinue therapy due to medication related somnolence. Additionally, treatment with gabapentinoid medications should continue for several weeks to

reach maximum benefit and patients should be monitored for medication related depression and anxiety [56, 57, 59]. Corticosteroids are commonly used to reduce inflammation, control symptoms, and improve short term function in patients with acute radiculopathy secondary to intervertebral disk herniation. A short oral corticosteroid taper or single IV or IM administration may modestly improve neurogenic pain. However, these medications do not improve pain or functional outcomes long term [60–63]. Athletes with axial back pain or paraspinal muscle strain may be treated with non-habit forming muscle relaxants such as cyclobenzaprine, tizanidine, or methocarbomol [64]. Physicians should avoid prescribing opiate pain medications for non-surgical or non-fracture related pain due to high risk dependence and addiction. If necessary, providers should always prescribe the lowest potency and lowest dose pain medication to achieve functional pain relief.

If adequate pain relief is not achieved or if the athlete reports chronic low back pain, referral to interventional pain management may be warranted. Many different treatment options including epidural steroid injections, facet joint injections, and radiofrequency ablation of facet joint innervation may be utilized to achieve symptomatic control. The specific indications and relative efficacy of these treatment modalities are highly variable and outside the scope of this text.

Once the acute inflammatory phase of soft tissue injury has passed, the athlete should be started on a structured physical therapy program. Maintaining strong anterior abdominal and paraspinal musculature is critical to postural control. Additionally, deconditioning can occur very quickly after injury and poor core strength may predispose athletes to reinjury [65]. Physical therapy protocols should begin with truncal stability and core strengthening exercises [66]. With time, athletes may be progressed to more sports specific activities as symptoms resolve and adequate truncal stability is achieved [67]. Lastly, many physical therapists and athletic trainers are trained in a myriad of alternative physiotherapy modalities such as targeted transcutaneous electrical nerve stimulation, therapeutic massage, and dry needling that may improve myofascial pain after injury [68–72].

Operative Treatment

Most injuries and pathology of the lumbar spine are best treated with conservative means as described above. Lumbar disc herniations causing radiculopathy and significant weakness typically warrant operative intervention. In athletes, microdiscectomy may be performed with predictably good results with approximately 80% of patients returning to competitive play after surgery [73, 74]. Cauda equina syndrome, which may result from large central disc herniations, causes urinary retention, fecal incontinence, and saddle anesthesia with a variable pattern of weakness and radiculopathy is a surgical emergency and should be decompressed

expediently [75, 76]. High energy spinal column injuries resulting in instability or neurologic comprise should be urgently decompressed and stabilized to optimize neurologic outcomes and prevent deformity [50]. Any fusion that is performed at a high motion segment is at risk for non-union and many athletes requiring fusion or longer segment instrumentation should refrain from contact sports until fusion or fracture healing is confirmed on radiography.

Rehabilitation and Return to Play

Most athletes are principally concerned with return to play and achieving their pre-injury level of performance, particularly at higher levels of competitive play. Additionally, athletes experience significant social and, at times, financial pressure to return to play as quickly as possible. It is important to establish clear return to play criteria and develop a treatment plan jointly with the physical therapists, coaches, athletic trainers, and the athlete. Failure to utilize a multidisciplinary approach and communicate clearly with all parties involved may lead to treatment failure and ultimately delay or prevent the athlete from returning to their pre-injury level of performance. Regardless of the treatment chosen, a progressive trunk stability and core strengthening rehabilitation program should be completed with the athlete demonstrating sufficient truncal control to safely meet the demands of their sport on formal return to play testing before they are released to resume unrestricted athletic activity [66, 67, 77–80].

Controversies and Expert Opinion

When dealing with the elite athlete, there is an incredible amount of pressure from the coach and trainers to return the athlete to play regardless of whether the athlete is ready to return. It is the responsibility of the physician to maintain their beliefs and recommendations in the best interest of the athlete despite this pressure. Although some surgeons believe in operating on acute pars defects in the thoracolumbar spine, we treat these patients conservatively initially. Only if they develop spondylolisthesis with radiculopathy do we entertain surgical intervention. Vertebral augmentation is a valid treatment option for ligamentously stable compression/burst fractures that surgeons should be more comfortable offering to athletes for them to potentially mobilize and return to play in an expedited fashion. Vertebral augmentation, such as kyphoplasty, should be in a surgeon's armamentarium in treating compression/burst fractures in the athlete population. Unstable lumbar fractures that require operative intervention are the result of a high-energy mechanism. These patients should be evaluated and treated as any other acute spinal injury presenting to a trauma center. Care should be focused on safe transport and evacuation from the playing field.

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Chapter 19
Thoracic Pathology in Athletes

Joseph P. Maslak and Jason W. Savage

Introduction

The thoracic spine, which is composed of 12 motion segments (vertebral bodies with corresponding intervertebral discs) and anchored to the rib cage and sternum, is characterized by significant biomechanical stability, contributing to the comparatively rare occurrence of thoracic spine pathologies. Nonetheless, thoracic spine injuries are feared because of the potential for catastrophic neurological injury. The athletic population is unique due to their relatively young age, low body fat percentage, lack of associated comorbidities, and the frequent possibility of high-energy impact. Approximately 15% of all spine injuries occur in sport-related activities, making athletics the fourth most common mechanism behind motor vehicle collisions, violence, and falls [1]. Violent sports such as American football and rugby often receive the most attention; however sports with repetitive loading of the spine can cause severe injuries, including rowing, dancing, and gymnastics. With the emergence in popularity of extreme sports, such as snowboarding, motocross, and base-jumping, higher-energy injuries may present at increasing frequency. This chapter will discuss common thoracic pathologies in the athletic population, diagnostic approaches, and both nonoperative and operative treatment strategies.

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Anatomy

Twelve thoracic vertebrae make up the thoracic spine, with ten articulating ribs and the 11th and 12th "floating" ribs. The thoracic vertebrae are characterized by their small size and distinctive articulations with the ribs. T1 is a transitional vertebra with a slightly quadrangular-shaped body and an uncovertebral process, and both T4 and T5 have a small impression from the aorta on the left lateral aspect of their vertebral bodies [2]. The sternum is a long flat bone that forms the front of the ribcage and articulates with the top seven ribs at the sternocostal joints. Normal thoracic alignment is 20–45°of kyphosis. In contrast to the cervical spine, the thoracic spinous processes are long and considerably caudally oriented, and the articular processes are vertically oriented. The thoracic facet joints transition from coronal orientation cranially to sagittal orientation caudally, and they allow six degrees of freedom: side bending, twisting, and flexion/extension.

Thoracic pedicles are thicker medially than laterally, and T4–6 have the narrowest pedicles, on average [3]. Pedicle length decreases, on average, from T1 to T4 and then increases caudally. The transverse pedicle angle varies from 10° in the mid-thoracic spine to 25° at T1, and the sagittal pedicle angle is approximately 15° cephalad for the thoracic spine.

The costovertebral joint is comprised of the two mobile diarthrodial costocentral and costotransverse articulations (Fig. 19.1). Although there are costocentral joints at all thoracic levels, costotransverse joints are not present at the floating ribs of T11 and T12. The articulating surfaces are separated by the neck of the rib with the rib head medially and the bony prominence of the rib tubercle posteriorly. The costo-

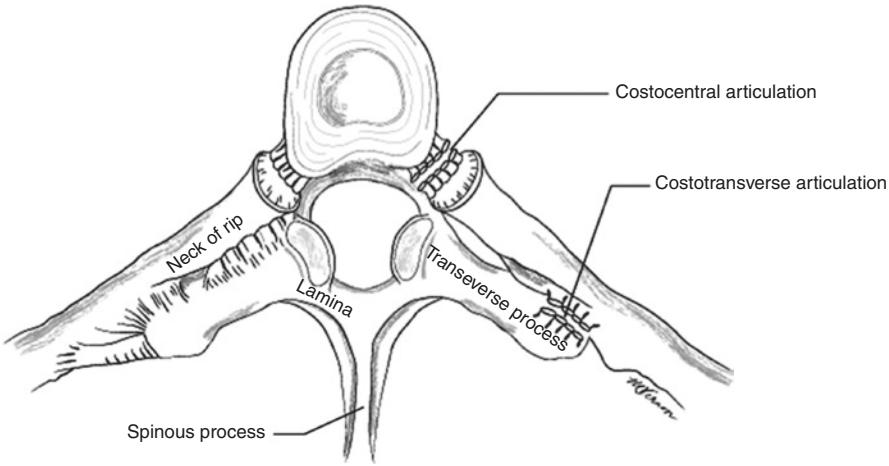

Fig. 19.1 Pertinent anatomy of costovertebral articulations. Axial figure of costovertebral joint shows costocentral and costotransverse articulations, normal on the right and disrupted on the left. (From: O'Brien and Bui-Mansfield [41]. Reprinted with permission from Wolters Kluwer)

central articulation involves a broad attachment with the intervertebral disc and the posterolateral portion of the vertebra of the same level and the level above. Stabilizers of the costovertebral joint include the thin synovial capsule, interarticular ligaments (not present at the 1st, 10th, 11th, and 12th ribs), and the radiate ligament.

The true ribs are those that articulate directly, through their costal cartilage, with the sternum. The 8th, 9th, and 10th ribs are false ribs that articulate with the sternum indirectly, through the costal cartilage from other ribs. Ribs 11 and 12 are called "floating" ribs because they are shorter and do not articulate with the sternum at all, only with their corresponding vertebrae. The floating ribs have a single articular facet on the rib head, with no neck or tubercles.

The erector spinae muscles function to extend the trunk and provide support to the axial skeleton. They are located dorsal to the vertebral column and are innervated by the dorsal rami of spinal nerves. The spinalis is located most medial and connects spinous process to spinous process. The iliocostalis is most lateral and originates from the ilium and ribs, inserting on the ribs and transverse processes. In between the two, the longissimus links transverse process to transverse process.

The stability of the spine is often considered in the context of the three column spine theory, as popularized by Denis [4]. The anterior column includes the anterior longitudinal ligament and the anterior 2/3 of the vertebral body and annulus. The middle column includes the posterior longitudinal ligament and posterior 1/3 of the vertebral body and annulus. The posterior column includes the pedicles, lamina, facets, ligamentum flavum, spinous process, and posterior ligamentous complex. Instability is often dependent on the involvement of the middle column, as evidenced by widening of the interpedicular distance on the AP radiograph and/or loss of height of the posterior cortex of the vertebral body on the lateral radiograph. Furthermore, the additional stability provided by the rib cage and sternum has often been referred to as a "fourth column" of spinal stability [5]. In fact, the sternum has been shown to contribute over 40% stability to thoracic spine range of motion [6].

Presentation

Athletes presenting with complaints in the thoracic region should be evaluated promptly. A senior athletic trainer is often responsible for on-the-field triage and making the initial diagnosis. Team physicians become involved when further testing is required or urgent medical attention is warranted. A comprehensive history should include characterization of the chief complaint (pain, stiffness, dysesthesia, weakness, etc.), timing (acute vs. subacute vs. chronic), associated symptoms (fever, chills, traumatic injuries, infections, dyspnea, chest pain), and prior treatments (activity modification, medications, physical therapy, injections, procedures). A corroborative history via trainer, coach, teammate, or parent is helpful, when possible.

Physical Exam

Initial general observations should include evaluation of mental status, muscular tone, presence of Horner's syndrome, pupil symmetry, and use of accessory respiratory muscles. When inspecting the thoracic spine specifically, note any skin lesions (rashes, erythema, discoloration, hair patches, etc.), focal edema, abnormal curvatures, and prior incisions. Thorough superficial and deep midline and paraspinal palpation should be performed to delineate any tenderness to palpation, pathologic step-offs, and masses. Range of motion and any associated pain in the coronal, sagittal, and axial (rotation) planes should be evaluated in patients with stable injuries. Patients should also be examined for joint hypermobility, with the Beighton score (0–9) often used as an objective measure. Associated muscular flexibility should be evaluated, such as the hamstring and hip flexors. In the event of a suspected spinal cord injury, a comprehensive neurologic exam should be performed in accordance with the recommendations by the American Spinal Injury Association (Fig. 19.2). A thorough neurologic exam includes motor, sensory, reflex, and rectal examination to assess tone, sacral nerve root function, and the presence or absence of the bulbocavernosus reflex.

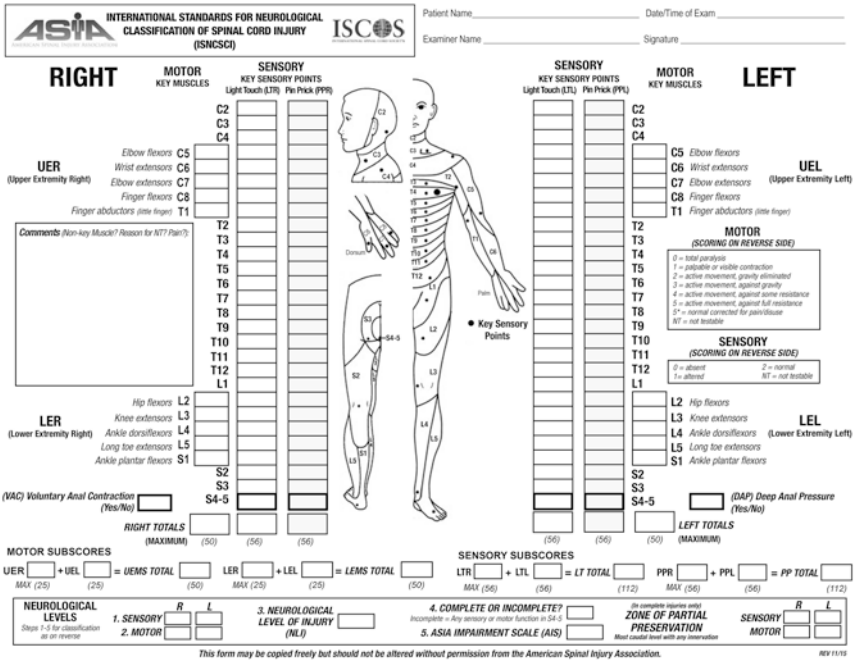

Fig. 19.2 Neurologic examination recommended by the American Spinal Injury Association for each patient sustaining a spinal injury. (International Standards for Neurological Classification of SCI) (Copyright © 2011 American Spinal Injury Association. Reprinted with permission)

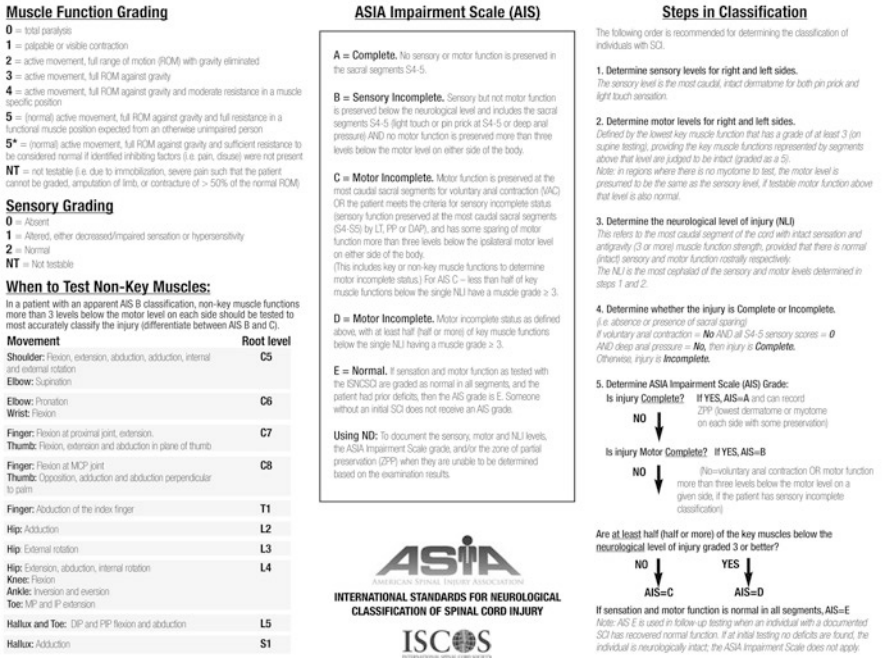

Fig. 19.2 (continued)

Imaging

Anteroposterior and lateral thoracic standing spine radiographs should be performed at the physician's discretion if there is concern for spinal pathology. Chest radiograph should be performed if "anterior" pathologies are in the differential diagnosis, including rib fracture, pneumothorax, and costochondral separation [7]. Advanced imaging with computed tomography (CT) is used to further assess bony pathologies, while magnetic resonance imaging (MRI) is reserved for elucidation of neurologic, ligamentous, and soft tissue disorders.

Pathologies

Muscular and Ligamentous Injuries

Thoracic "myofascial" pathology may result from both acute trauma and with chronic, overuse mechanisms. High-energy acute injuries are caused by rotational or bending forces similar to whiplash injuries in the cervical spine. Pain and tenderness often present 12–24 h after injury secondary to the inflammatory cascade. The

neurologic exam and radiographs are normal in purely myofascial injuries, and any signs of neurologic dysfunction should prompt further evaluation. An MRI is helpful in evaluating the neurologic and ligamentous elements of the thoracic spine, and CT can be useful in ruling out occult fracture.

Acute myofascial injuries are treated with conservative management, including hiatus from aggravating activities, physical therapy, and anti-inflammatory medication. Muscle relaxants and selective injections are often used as adjunctive therapies. Chronic overuse injuries from sustained, high-repetition activities can result in debilitating, long-lasting pain. The pull of the rhomboids, latissimus dorsi, and erector spinae at the T4–T7 junction is particularly susceptible to strains and stress fractures [1]. Physical therapy should focus on the kinematics of the thoracic spine, rib cage, and shoulder girdle. The therapist should use both passive and active modalities, if possible. Passive modalities do not rely on muscle activation and include hot-cold compress, ultrasound, and massage. These therapies may alleviate acute symptoms and facilitate transition to active therapy modalities focusing on postural mechanics, core strengthening, and trunk stabilization.

Fractures

Fractures to the thoracic spine are rare given the relative stability of this region of the spine. Nonetheless, myriad fracture morphologies are possible, ranging from minor injuries such as isolated transverse or spinous process fractures to major injuries such as unstable fracture/dislocations with high potential for associated spinal cord injury. Stress fractures may occur due to overuse activities and may affect the spine or the ribs. Clay-shoveler fractures are an overuse avulsion-type fracture of multiple lower cervical and/or upper thoracic spinous processes. They are caused by shear forces over the dorsal aspect of the neck, and several case reports include paddlers, baseball, and wrestling [8, 9]. Yamaguchi et al. reported on both a high school wrestler and baseball player, and acute posterior neck pain with an associated “pop” was reported by both patients. Both patients were treated conservatively and returned to play at 4 months.

Higher-energy activities such as snowboarding, rugby, American football, and motocross may cause higher-energy fractures. Anterior compression fractures should be considered in athletes with repetitive axial loading with flexion of the thoracic spine, including gymnasts, extreme sport athletes, and basketball players. Compression fractures are defined by anterior column failure with maintenance of the posterior elements of the spine, appearing as anterior wedging on lateral radiograph. Thoracic compression fractures rarely require operative management due to this isolated involvement of the anterior column and the stability inferred by the ribs and sternum. External orthoses such as a thoracic lumbar sacral orthosis (TLSO) or a Jewett brace are often used for comfort in patients with fractures involving T6 or below, although there is no data to support their use. Above T6, inclusion of the cervicothoracic junction is recommended with a Minerva brace. Elattrache et al. reviewed a professional football player who sustained T8 and T9 compression frac-

Table 19.1 Thoracolumbar Injury Classification and Severity Score (TLICS)

Injury characteristic	Qualifier	Points
Injury morphology		
Compression	–	1
	Burst	+1
Rotation/translation	–	3
Distraction	–	4
Neurologic status		
Intact	–	0
Nerve root	–	2
Spinal cord, conus	Incomplete	3
	Complete	2
Cauda equina	–	3
Posterior ligamentous complex integrity		
Intact	–	0
Indeterminate	–	2
Disrupted	–	3

From: Patel and Vaccaro [11]. Reprinted with permission from Wolters Kluwer
+1: Additional point given to morphology

tures after a football tackling injury [10]. There were no neurologic deficits, radiographs demonstrated 40% loss of vertebral height, and CT scan demonstrated isolated anterior column involvement. He was successfully treated with an extension thoracolumbar spinal orthosis for 12 weeks and successfully returned to play once radiographs confirmed no increased vertebral collapse or kyphosis.

Major thoracic trauma may result in unstable thoracic fracture patterns that have significant potential for associated spinal cord injury. These include burst, translation-rotation, and flexion-distraction fracture patterns or patterns with greater than single column involvement. Burst fractures involve failure of the anterior and middle columns, and retropulsion of fragments into the canal is often elucidated with a CT scan. Indicators of instability include loss of disc height, local kyphosis, PLC injury, and neurologic deficits. Treatment is aimed at facilitation of fracture healing, preservation of neurologic function, preventing progression of deformity, and restoration of range of motion, if possible. Burst fractures are mechanically stable injuries but are sometimes associated with neurologic deficits (neurologically unstable). Operative intervention is reserved for patients who have neurologic symptoms in the setting of an acute thoracic burst fracture. These injuries are typically treated with a posterior decompression and stabilization. Anterior decompression is considered if there is a large retropulsed bone fragment causing spinal cord compression.

Translation-rotation and flexion-distraction fractures often involve all three vertebral columns and commonly result in compromised spinal alignment involved, and subluxation may occur. The Thoracolumbar Spine Trauma Classification and Severity Score is an algorithm that integrates fracture morphology, neurologic status, and PLC integrity in an effort to guide nonoperative versus operative treatment (Table 19.1) [11]. Operative treatment is recommended with a

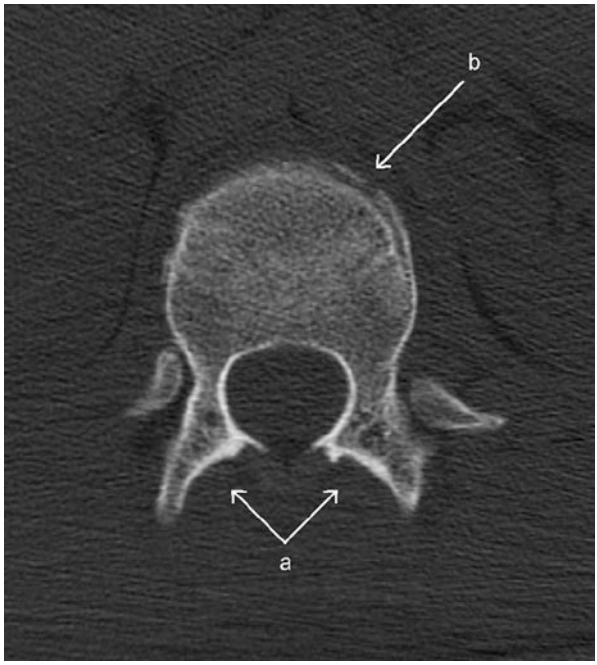

Fig. 19.3 Chance fracture in an 18-year-old bareback rider. Transaxial computed tomography image of T12 vertebral body showing superior articular processes of T12 without normal articulation of T11. (a) Findings represent a "naked-facet" sign and signify distraction of posterior elements. (b) Anterior compression or "sandwich" sign of the vertebrae. In addition, note the absence of the spinous process which was fractured and unable to be seen in this image. (Reprinted from: Boham and O'Connell [12]. Journal of Athletic Training is an open access publication)

score greater than 4. These types of injuries are unstable and are often associated with spinal cord injury. Treatment is typically with a posterior decompression and stabilization. Fracture dislocations necessitate a posterior reduction prior to stabilization.

Boham and O'Connell reported on an 18-year-old bareback rodeo athlete who sustained a T12 chance when his horse bucked, slamming him into a wall and forcing thoracolumbar hyperflexion [12]. He felt an immediate "pop" with intense pain, was able to dismount from the animal, and had a normal physical exam save for tenderness to palpation. He was found to have 70% loss of T12 vertebral body height with 20° of local kyphosis and fracture extending through the bilateral pedicles into the lamina and spinous process of T12 (Fig. 19.3). He underwent T11-L1 posterior spinal fusion with instrumentation, and, despite discouragement from returning to competition, he was able to return to sport at 3 months post-op with the aid of a padded vest for protection.

Chronic overuse activities can be associated with stress fractures. Elite rowers have an 8–16% rib stress fracture rate over the course of a career due to continuous, repetitive motion [13, 14]. The posterior ribs of such athletes are at risk for stress fractures due to the pull of the serratus anterior muscle, and they often occur after time off from sport when endurance is suboptimal. Costal cartilage fractures have also been case reported in an amateur rugby player [15].

Disc Herniation

The intervertebral disc functions to absorb axial stress and provide flexibility. Traumatic herniation of the nucleus pulposus through the annulus is caused by axial load and rotation on a flexed spine. In the general population, the incidence of thoracic disc herniation is estimated between 1 in 1000 and 1 in 100,000 [16]. While reported at every thoracic level, 75% occur below T8, with T11–T12 being the most common level due to increased spinal mobility and weakness of the posterior longitudinal ligament [17]. Thoracic disc herniation are rare injuries, although there have been case reports in elite soccer, football, and baseball athletes [18–20]. Symptoms often manifest as axial pain with possible associated radiculopathy and/or myelopathy. Radiculopathy is described as a band-like discomfort in a dermatomal distribution. Myelopathy is indicative of cord compression and is evidenced by both upper and lower motor signs such as gait disturbance, spasticity, clonus, weakness, and positive Babinski reflex. Upper extremity symptoms may predominate in high thoracic herniations, while lower herniations may mimic lumbar disc disease due to involvement of the lower extremities.

Radiographs are not usually useful in diagnosing disc herniation, but they should be obtained to rule out other osseous pathologies. MRI is the imaging modality of choice to diagnose thoracic disc herniation, and findings must be correlated clinically. As with cervical and lumbar disc herniations, the majority of thoracic disc herniation cases are successfully treated with conservative management, including rest, oral steroids, NSAIDs, and physical therapy. Directed injections can also be used both diagnostically and therapeutically in select cases.

However, if the symptoms persist after at least 6 weeks of conservative management, or there is progressive myelopathy or neurologic deficits, operative intervention is recommended. Preoperative CT and MRI imaging is helpful in identifying the herniation's location, size, intradural extension, and associated calcifications. Surgical treatment often consists of a minimally invasive or open discectomy, with the approach dictated by the location of the herniation and adjunctive fusion reserved for cases with associated instability [16]. While elite athletes undergoing single-level lumbar microdiscectomy return to play 83.5% of the time, there are no current studies effectively powered to evaluate thoracic disc herniation return to play after discectomy [21].

Spinal Cord Injury

Over 17,000 new spinal cord injuries (SCI) are diagnosed per year (54 per million) in the United States alone, changing countless lives and costing millions of dollars over a patient's lifetime [22]. An estimated 282,000 people are currently

living with SCI in the United States, and males account for 80% of newly diagnosed cases. Incomplete and complete paraplegia account for 41.3% of diagnoses since 2010. While motor vehicle crashes are the most common cause, sporting injuries are the fourth leading cause behind falls and acts of violence. Sports most frequently cited include diving, horseback riding, American football, and skiing [1].

Initial evaluation in the case of suspected SCI should include comprehensive neurologic exam, including rectal exam and evaluation of the bulbocavernosus reflex. Spinal shock is a transient period of hyporeflexia and autonomic dysfunction following spinal cord injury, with the return of the bulobocavernosus reflex marking the end of spinal shock. Conversely, neurologic shock results from the loss of sympathetic tone and is characterized by hypotension and bradycardia. Current guidelines suggest maintenance of mean arterial blood pressure (MAP) greater than 85 mmHg may predict favorable functional outcomes in patients with SCI, although these guidelines are evolving [23]. The use of high-dose steroids (NASCIS Trials) is controversial but may have a role in young healthy patients who are treated within 8 h of injury [24–26]. Appropriate immobilization and spinal precautions are vital to prevent further injury during initial management. Operative decompression and stabilization may be necessary depending on the associated osseous injury, and emerging evidence suggests that early surgery may improve neurologic outcomes, particularly with incomplete SCI, and may reduce non-neurologic complications and health care resource utilization [27].

SCI is a multisystem pathology with low likelihood for complete recovery. Harrop et al. found in their review of thoracic and thoracolumbar spinal cord injuries that thoracic, specifically T4–T9, had the least likely potential for neurologic improvement [28]. As such, multidisciplinary management is vital in the long-term treatment of patients with SCI, as 30% of persons with SCI are rehospitalized one or more times during any given year following their injury, most often for genitourinary issues followed by integumentary, digestive, circulatory, and musculoskeletal causes [22].

Deformity

Spinal deformity represents a spectrum of pathologies affecting up to all three geometric planes (coronal, sagittal, axial) that commonly begin in childhood or adolescence and may have lifelong consequences. Idiopathic scoliosis occurs in 2–3% of the population, while excessive kyphosis occurs in up to 8% [29]. These rates vary in certain athletic populations, with Warren et al. noting a significantly increased prevalence (24%) of a minor curve in young ballet dancers and Tanchev et al. noting a tenfold increase in scoliosis frequency among rhythmic gymnasts [30, 31]. Regarding the sagittal profile, both thoracic kyphosis and lumbar lordosis increase during the adolescent growth spurt. These may be accentuated further in sports that require repetitive activities, such as hyperkyphosis in swimming

and hyperlordosis in gymnastics. Plausible explanations for this include the repetitive asymmetric forces across the growth cartilage of the spine, particularly in prepubertal and pubertal athletes. Swimming, throwing, and serving have also been associated with asymmetric torque forces contributing to a functional scoliosis [32].

The decision on type and frequency of radiographic evaluation is based on pubertal status and physical examination. The forward bend Adams test evaluates thoracic rotation with a scoliometer. If the scoliometer reaches 7° in an immature child, a PA radiograph from C7 to the iliac crest is obtained. For coronal curves less than 25° of radiograph, observational treatment is recommended with the frequency dependent on puberty. A prepubertal athlete with a curve 10–14° may be reevaluated in 1 year, whereas a curve from 15 to 19° should be evaluated again in 3–6 months with a history and scoliometer examination. In prepubertal athletes with a curve of 20–24°, a repeat radiograph is performed in 3 months. A curve greater than 30° or one that manifests a progression of more than 5° should be considered for bracing. Bracing may include the Boston brace, worn 18–23 h per day, or a nighttime brace such as the Charleston or Providence brace. Curves that reach 40–45° in immature patients should be considered for surgical stabilization.

For Scheuermann's kyphosis, the treatment is largely conservative, with surgery indicated in rare cases of progressive deformity and persistent symptoms. For curves of less than 50°, conservative management with stretching of the anterior chest wall/shoulders/hamstrings, extension-based strengthening, and core stabilization exercises can be effective. Bracing may be recommended for those with a curve between 50° and 70° and growth remaining. The type of brace is dependent on the level of the apex, with the Milwaukee brace indicated for an apex above T7 and thoracic-lumbar-sacral orthosis (TLSO) for an apex below T7. Ideally, the brace should be worn 16–18 h per day.

Nonetheless, while scoliosis and kyphosis may be aggravated by certain sports, the causal association is minimal, and athletes with scoliosis are treated similar to the general population. Athletes should be encouraged to continue sports participation. Brace wear is commonly prescribed for scoliosis and kyphosis, and many sports can be played with the brace. Even with sports that cannot practically be played with the brace, most bracing protocols have enough time out of the brace during the day to allow for continued participation. Appropriate therapy and exercises should be continued to enhance flexibility or strengthening of muscles about the spine.

Costovertebral Dislocation

Dislocation of the costovertebral articulation requires significant energy and is very rare. They often present with subtle radiographic findings and can portend a more serious injury and need for further workup. Often both radiographs and computed

tomography (CT) are necessary for diagnosis. On anterior-posterior (AP) radiographs, asymmetric rib interspace narrowing above and widening below the level of injury can be seen. Another radiographic finding is the displacement of the costal head from the pedicle. On CT, the finding of a "naked transverse process" sign, or a transverse process with its associated rib displaced, is identified on the axial view [33]. The need for operative intervention is dependent on the presence of neurologic compromise and regional instability.

Degenerative Disease

Degenerative thoracic spine pathology is rare in the athlete population, likely due to the relatively young age of this population and stability of the thoracic region of the spine. Nonetheless, literature is sparse and suggests certain athletic population may be at higher risk than others. A study of athletes from the 2016 Olympic Summer Games demonstrated no athletes with moderate or severe degenerative disease of the thoracic spine [34]. However, a retrospective cohort study of adolescent motocross riders identified a significant increased risk of degenerative changes in the thoracic spine of riders compared to age-matched controls [35]. Furthermore, a study of 24 elite male gymnasts demonstrated a significantly higher incidence of reduced thoracolumbar disc intensity on MRI compared to age-matched controls [36]. While the long-term consequences are unknown, this risk should be acknowledged by parents and participants in select athletic subgroups, some of which have yet to be identified.

Rare Conditions

Physicians evaluating athletes with thoracic complaints should be aware of rare spinal pathologies that may present in the presence or absence of trauma. Conditions such as congenital thoracic stenosis, synovial cysts, spinal epidural lipomatosis, tumor, and infection can often be confirmed with a complete history, physical, and advanced imaging. These disorders may also predispose an athlete to a worse outcome in the setting of a traumatic thoracic injury.

Rare anterior thoracic musculoskeletal conditions may also be misdiagnosed as spinal pathology. "Slipping rib" syndrome is a rare condition often misdiagnosed and can lead to months of unresolved symptoms, as reported in a collegiate swimmer [37]. The condition arises from hypermobility of the anterior ends of the false rib costal cartilages, which often leads to slipping of the affected rib under the superior adjacent rib. This movement can lead to an irritation of the intercostal nerve, strain of the intercostal muscles, sprain of the lower costal cartilage, or general inflammation of the area. Surgical resection of the abnormal cartilaginous rib attachment was shown to provide a successful result and return to sport for the collegiate swimmer.

Table 19.2 Time lost from play by location of spinal injury

	Days lost			Practices lost			Games lost		
Location	Mean	Median	SD	Mean	Median	SD	Mean	Median	SD
Cervical	93	69	82	113	24	333	15	7	36
Thoracic	189	179	64	72	80	31	17	15	4
Lumbar	51	14	70	39	8	110	11	2	33
All	63	23	76	55	10	183	12	2	33

From: Gray et al. [19]. Reprinted with permission from Wolters Kluwer
Thoracic disc herniations in NFL players led to a mean of 189 days (SD +/− 64), 72 practices (SD +/− 31), and 17 games lost from play (SD +/− 4)
SD indicates standard deviation

Return to Play

Given the rare nature of thoracic spine injuries, outcome data used to construct return-to-play recommendations are sparse, and no consensus opinions currently exist. General return-to-play criteria include full strength without neurologic deficit and painless full range of motion [38]. Athletes sustaining acute fractures with associated instability or neurologic symptoms should not participate in athletics. After surgical stabilization, furthermore, there are few proposed guidelines for return to play. Burnett and Sonntag recommend that spinal fusions bypassing or terminating at transition zones in the cervicothoracic or thoracolumbar region are an absolute contraindication to participation in contact sports [39].

There exist multiple retrospective reviews evaluating return to play after thoracic injuries in specific athletic populations. In a retrospective review of spine and axial skeleton injuries over 12 NFL seasons (2000–2012), Gray et al. found thoracic disc herniations to have the greatest impact on return to play (189 days/injury), worse than cervical fracture, cervical disc herniation, and lumbar disc herniation (Table 19.2) [19]. Tackling and blocking were the two most frequently noted injury mechanisms, and offensive linemen were the most likely to suffer a spinal injury. A large retrospective review of American high school athletes demonstrated thoracic/abdominal rate of injury 4.9 per 100,000 athletic exposures (AEs), with contusion and muscle strain accounting for over 60% of the injuries [40]. Football and wrestling were found to have the highest rate of injury, and only 57.7% of athletes were able to return to play within 1 week. As such, the likelihood and timeline for return to play is dependent on the type of injury, characteristics of the athlete, and availability of appropriate resources for expeditious recovery.

Expert Opinion

An understanding of the anatomy and biomechanics of the thoracic spine is critical in evaluating thoracic injury patterns. In the context of athletics, thoracic spinal injuries are less common than cervical or lumbar injuries but may lead to relatively

prolonged absences from sport and have the potential for life-changing catastrophic neurologic injury. Nonetheless, the most common athletic spinal pathologies are most frequently successfully treated with conservative management, and further studies may help elaborate return-to-play standards and optimal recovery protocols.

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