Contemporary Pediatric and Adolescent Sports Medicine
Series Editor: Lyle J. Micheli

Lyle Micheli
Cynthia Stein
Michael O'Brien
Pierre d'Hemecourt *Editors*

Spinal Injuries and Conditions in Young Athletes

Springer

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The Micheli Center for Sports Injury Prevention

The mission of the Micheli Center for Sports Injury Prevention is at the heart of the *Contemporary Pediatric and Adolescent Sports Medicine* series.

The Micheli Center uses the most up-to-date medical and scientific information to develop practical strategies that help young athletes reduce their risk of injury as they prepare for a healthier future. The clinicians, scientists, activists, and technologists at the Micheli Center advance the field of sports medicine by revealing current injury patterns and risk factors while developing new methods, techniques, and technologies for preventing injuries.

The Micheli Center had its official opening in April 2013 and is named after Lyle J. Micheli, one of the world's pioneers in pediatric and adolescent sports medicine. Dr. Micheli is the series editor of *Contemporary Pediatric and Adolescent Sports Medicine*.

Consistent with Dr. Micheli's professional focus over the past 40 years, The Micheli Center conducts world-class medical and scientific research focused on the prevention of sports injuries and the effects of exercise on health and wellness. In addition, the Micheli Center develops innovative methods of promoting exercise in children.

The Micheli Center opens its doors to anyone seeking a healthier lifestyle, including those with medical conditions or illnesses that may have previously limited their abilities. Fellow clinicians, researchers, and educators are invited to collaborate and discover new ways to prevent, assess, and treat sports injuries.

Dr. Lyle J. Micheli, Series Editor

Dr. Lyle J. Micheli is the series editor of *Contemporary Pediatric and Adolescent Sports Medicine.* Dr. Micheli is regarded as one of the pioneers of pediatric and adolescent sports medicine, a field he has been working in since the early 1970s when he co-founded the USA's first sports medicine clinic for young athletes at Boston Children's Hospital.

Dr. Micheli is now director of the Division of Sports Medicine at Boston Children's Hospital, and Clinical Professor of Orthopaedic Surgery at Harvard Medical School. He is a past president of the American College of Sports Medicine and is currently the Secretary General for the International Federation of Sports Medicine. Dr. Micheli co-chaired the International Olympic Committee consensus on the health and fitness of young people through physical activity and sport.

In addition to many other honors, Dr. Micheli has served as Chairperson of the Massachusetts Governor's Committee on Physical Fitness and Sports, on the Board of Directors of the United States Rugby Football Foundation, as Chairman of the USA Rugby Medical and Risk Management Committee, and on the advisory board of the Bay State Games. He has been the Attending Physician for the Boston Ballet since 1977 and is Medical Consultant to the Boston Ballet School.

Dr. Micheli received his undergraduate degree from Harvard College in 1962 and his medical degree from Harvard Medical School in 1966. As an undergraduate student, Dr. Micheli was an avid athlete, competing in rugby, gridiron football, and boxing. Since graduating, Dr. Micheli has played prop for various Rugby clubs including, the Boston Rugby Football Club, the Cleveland Blues Rugby Football Club, Washington Rugby Club and Mystic Valley Rugby Club where he also served as team coach.

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Anatomy and Development of the Young Spine

1

Brian A. Kelly and Brian Snyder

Anatomic changes during the growth and development of the spine put the young athlete at risk for specific injuries of the axial skeleton. As such, a basic understanding of embryology and changes in spine anatomy during growth can inform the clinician regarding mechanisms of injury and specific pathological conditions that affect the structure and function of the spine in the young athlete. Knowledge of the changing anatomy of the spine during growth and development can also provide the clinician with insight as to the range of normal variation as well as an appreciation of the differences between the pediatric and the adult spine and how this can impact interpreting radiographic images of the spine. The goal of this chapter is to review the embryologic development of the spine and pertinent anatomy of the spine in children and adolescents as it changes during normal growth including the identification of common anatomic variants. This anatomic information will serve as the basis for evaluating the structure and function of the young athlete's spine in health and disease.

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Embryology

Orientation of the developing embryo begins early in gastrulation. The primitive streak begins to define the longitudinal axis of the embryo on about day 15 of development; during the third week of gestation, cells migrating from this area form the three germinal layers: the endoderm, mesoderm, and ectoderm (Fig. 1.1) [1]. The neural tube also forms at this time, beginning as an infolding of ectodermal tissue that eventually will form the neural elements of the spinal cord. Neural tube defects result from incomplete closure of these in-folding cells. Incomplete closure at the cranial end leads to disorders such as anencephaly, while incomplete closure at the caudal end leads to the spectrum of spina bifida. A group of specialized cells migrate from the cranial portion of the primitive streak and give rise to the notochord, which lies ventral to the developing neural tube. The notochord is the precursor of the vertebral column; it eventually develops into the nucleus pulposus comprising the intervertebral discs and the apical and alar ligaments [2, 3].

The mesodermal cells differentiate into the paraxial, intermediate, and lateral mesoderm (Fig. 1.2). During the fourth and fifth week of gestation, 42–44 pairs of somites form from the paraxial mesoderm on both sides of the notochord. These somites develop in a cranial to caudal fashion to form the skeletal elements and musculature of the face, spine, and thorax. Each somite further differentiates into the sclerotome, which develops into the spinal elements, and the

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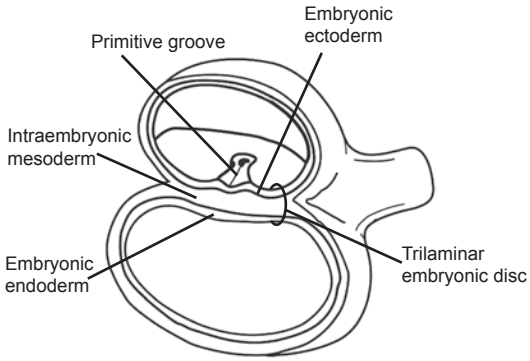

Fig. 1.1 Gastrulation. The primitive streak appears on the bilaminar germ disc on approximately day 15. During the third week, migrating cells from this area become the definitive endoderm and mesoderm

dermatomyotome, which develops into skin and muscle [1].

During the fourth week of gestation, cells from the most cranial sclerotomes begin to migrate and envelop the adjacent notochord. These sclerotomes then divide into a cranial and a caudal half, which will fuse with the adjacent-level sclerotome to form the provertebrae, completing a process known as metameric shift (Fig. 1.3). There are 4 occipital, 8 cervical, 12 thoracic, 5 lumbar, 5 sacral, and 8–10 coccygeal sclerotomes. These fused segments undergo chondrification during the fifth and sixth weeks of gestation in response to signals from the surrounding tissues to derive the bony elements of the spine [1, 2, 4, 5].

Formation of the spinal elements is a complex, highly regulated process. Any perturbation (i.e., infection, trauma, mutagenic effect of drugs or radiation) during the segmentation and reformation of the sclerotomes can result in abnormal spine anatomy and is frequently associated with abnormalities of other organs such as the heart or kidneys forming simultaneously. The spinal abnormalities consist of either a failure of formation, which produce hemivertebrae, or a failure of segmentation, which produce segmental bony fusions between adjacent vertebrae known as bars [5]. These anomalies can occur in isolation or at multiple levels in various combinations to produce a congenital scoliosis.

Developmental Anatomy

Understanding the pattern of progressive ossification and sequential fusion of synchondroses during the growth and development of the spine is required to properly differentiate apparent discontinuities in vertebrae imaged radiographically from true injuries (Fig. 1.4) [6]. Spinal segments distal to C2 exhibit a similar pattern of ossification and fusion. C1 and C2 are unique in their development and therefore are considered separately.

The C1 vertebra, or atlas, develops from the fourth occipital and first cervical sclerotomes. Three distinct ossification centers develop: the anterior arch and two bilateral posterior neural arches [7]. Twenty percent of children have ossification of the anterior arch at birth, and 50 % undergo ossification of the anterior arch by 1 year of age [4]. The paired posterior neural arches fuse in the midline by 3–4 years of age. The neurocentral synchondroses, between the anterior and posterior neural arches, persist longer and fuse between 6 and 8 years of age [8].

Development of C2, the axis, is considerably more complex. The multiple ossification centers can be confusing when interpreting cervical spine radiographs in children and may be mistaken for fractures [9]. C2 develops from the first and second cervical sclerotomes as five distinct ossification centers. The odontoid process itself begins as two separate ossification centers divided vertically. Fusion of these halves usually occurs by the time of birth, but can persist as a "dens bicornis." As with the atlas, the neural arches fuse posteriorly by 2–3 years of age [4, 8]. Additionally, the tip of the odontoid process, the os terminale, can appear separately in children aged 3–6 years until it fuses at approximately 12 years of age [10]. As this ossification center is located at the insertion of the apical ligament, it can be mistaken for a type I avulsion fracture of the dens [11, 12].

The dentocentral, or basilar, synchondrosis exists between the body of C2 and the base of the odontoid process. The synchondrosis itself exists inferior to the level of the articular process

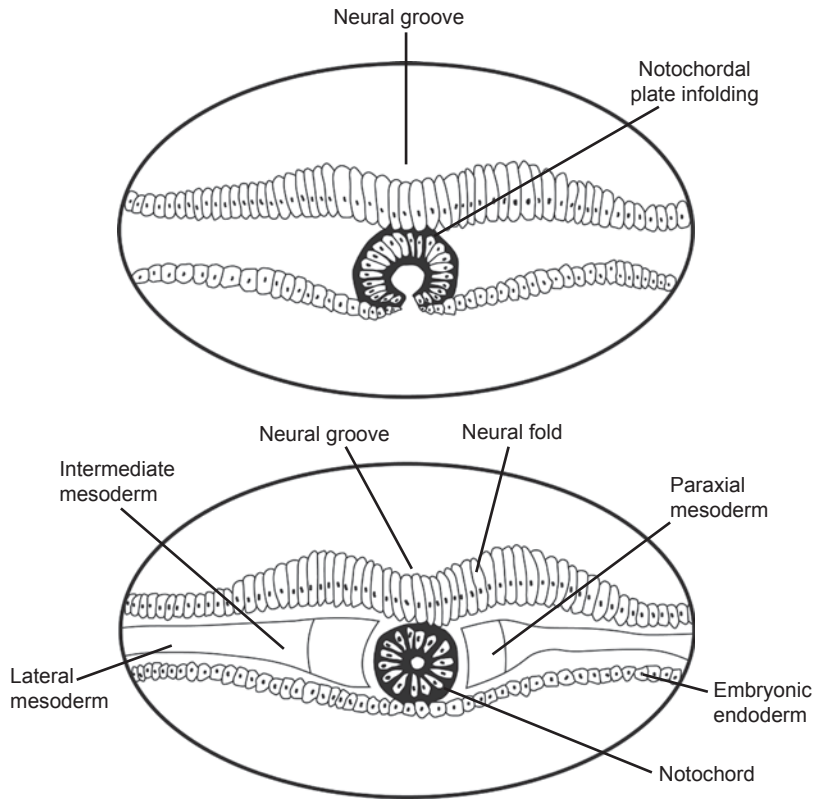

Fig. 1.2 During the third week of gestation, the notochord forms and mesodermal tissue on either side differentiates into paraxial mesoderm. The paraxial mesoderm further differentiates into somites in a cranial to caudal fashion, and 42–44 pairs of somites will form by the end of the fifth week

of the atlas and has a "cork in bottle" appearance on anteroposterior (AP) plain radiographs. Because this synchondrosis fuses later, it can be confused with a dens fracture. The synchondrosis is present in 50% of children 4–5 years old, and is fused in most children by the age of 6 years [4]. On radiographs, the physeal scar remains visible as a sclerotic line in children up to age 11.

Cervical vertebrae C3 through C7 can be considered together as their growth is similar. Reminiscent of the atlas, these vertebrae begin as three separate ossification centers. The neural arches fuse posteriorly between 2 and 3 years of age. The neurocentral synchondroses fuse between 3 and 6 years of age [8]. The subaxial vertebrae also exhibit secondary ossification centers at either the tips of the transverse processes or the tip of the spinous process that can persist until the third decade of life and can be mistaken for an avulsion on imaging studies [11]. The thoracic and lumbar vertebrae follow much of the same pattern as the subaxial cervical vertebrae.

The sagittal alignment of the spine begins as a single primary kyphotic curve involving the entire length of the spine [3, 13, 14]. As the fetus develops and muscle forces act on the growing spinal column, the secondary lordotic curves of the cervical and lumbar spinal levels begin to develop [15]. This process accelerates when the child begins to load the spine axially during sitting, standing, and walking. These secondary curves continue to progress throughout childhood and adolescence [16, 17].

The spinal canal reaches adult diameters by the age of 6–8 years [18, 19]. Early in gestation, during formation of the spinal cord, the neural elements occupy the entire length of the spinal column. Differential growth between the neural and the vertebral elements causes the terminal aspect of the cord, the conus medullaris, to migrate cranially during fetal development. By 2 months of age, the conus medullaris terminates at approximately the L1–L2 level. After this point in time, growth of the spinal cord and the vertebral

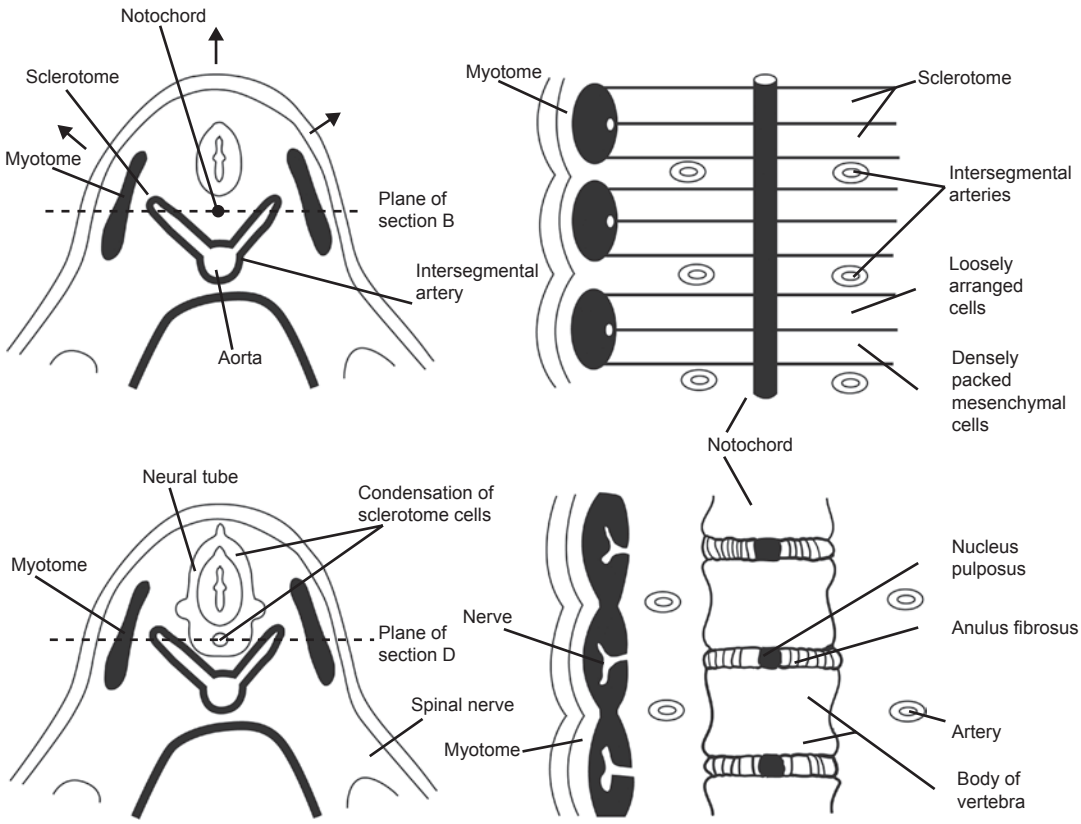

Fig. 1.3 Metameric shift. Starting in the fourth week of gestation, sclerotomes will divide into a cranial and caudal portion and recombine with the adjacent sclerotome. This occurs while segmental nerves grow out to innervate the myotomes

column is relatively symmetric and the conus medullaris remains at the upper end of the lumbar spine [20]. Tethering of the spinal cord during asymmetric growth between the spinal cord and the vertebral column can cause neurologic deficits and scoliosis.

The blood supply to the vertebrae arises segmentally from the intercostal arteries or from nearby arteries in the cervical and lumbar spine. The thoracolumbar spinal cord is supplied by the great anterior radicular artery and paired posterior arteries [18]. In 80% of individuals, the great anterior radicular artery arises from the left side off an inferior intercostal artery and enters the intervertebral foramina accompanying one of the ventral roots T9–T12 [21]. To limit injury to this artery which supplies the inferior two-thirds of the spinal cord, ligature of vessels during anterior approaches to the spine should be avoided close to the foramen. Innervation of the intervertebral disc arises primarily from the sinuvertebral nerve, a branch off the dorsal root ganglion [22, 23]. A normal vertebra is illustrated in Fig. 1.5.

Cervical Spine

Up to 80% of pediatric spine injuries involve the cervical spine, underscoring the importance of the anatomy of this region [8]. Nearly 87% of cervical spine injuries that take place in children under the age of 8 years occur at the C3 level or above (Fig. 1.6) [2]. This is markedly different from the pattern of adult spine injuries, where fewer injuries occur to the cervical spine and the majority of cervical spine injuries that do occur, involve C5 or below. There are several anatomic

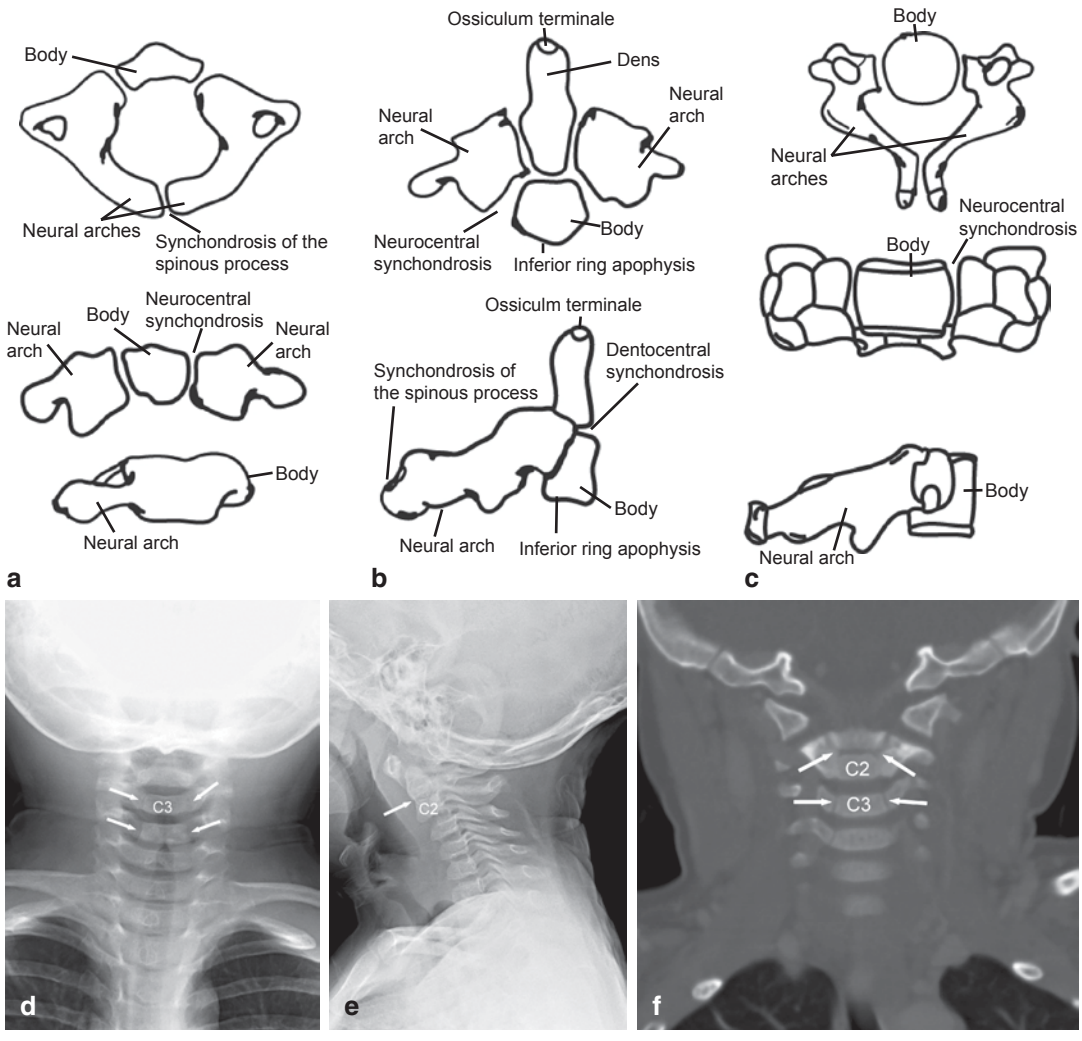

Fig. 1.4 Ossification of cervical vertebrae. Ossification centers and synchondroses of **a** C1, **b** C2, and **c** C3–7. Thoracic and lumbar vertebrae follow a similar pattern as the subaxial vertebrae. Synchondroses of the cervical spine (*arrows*) as seen on **d** AP plain radiograph, **e** lateral plain radiograph, and **f** coronal CT scan

features that account for the differential pattern of spine injuries affecting children and adults [12, 24].

The cervical spine of children differs anatomically from adults in several important ways that contribute to increased motion between spinal segments. Incomplete ossification of both the axis and the dens leads to increased physiologic motion between C1 and C2. At subaxial levels, the facet joints of the cervical spine are shallower and more horizontally oriented at birth, leading to increased translational motion. Early in development, the facets are oriented approximately 30° from the horizontal, but by adolescence, the depth of the facets joint increases and the orientation of the facet increases to 60–70° in the upper cervical spine and 55–70° in the lower cervical spine [25]. Additionally, the uncinate processes, which serve to limit lateral translation and rotation between adjacent cervical vertebrae, are underdeveloped in children. The uncinate processes do not form until approximately age 7 [25].

In addition to specific differences in cervical spine anatomy, children have a proportionally larger head than adults with relatively more weight being supported by the neck. This is

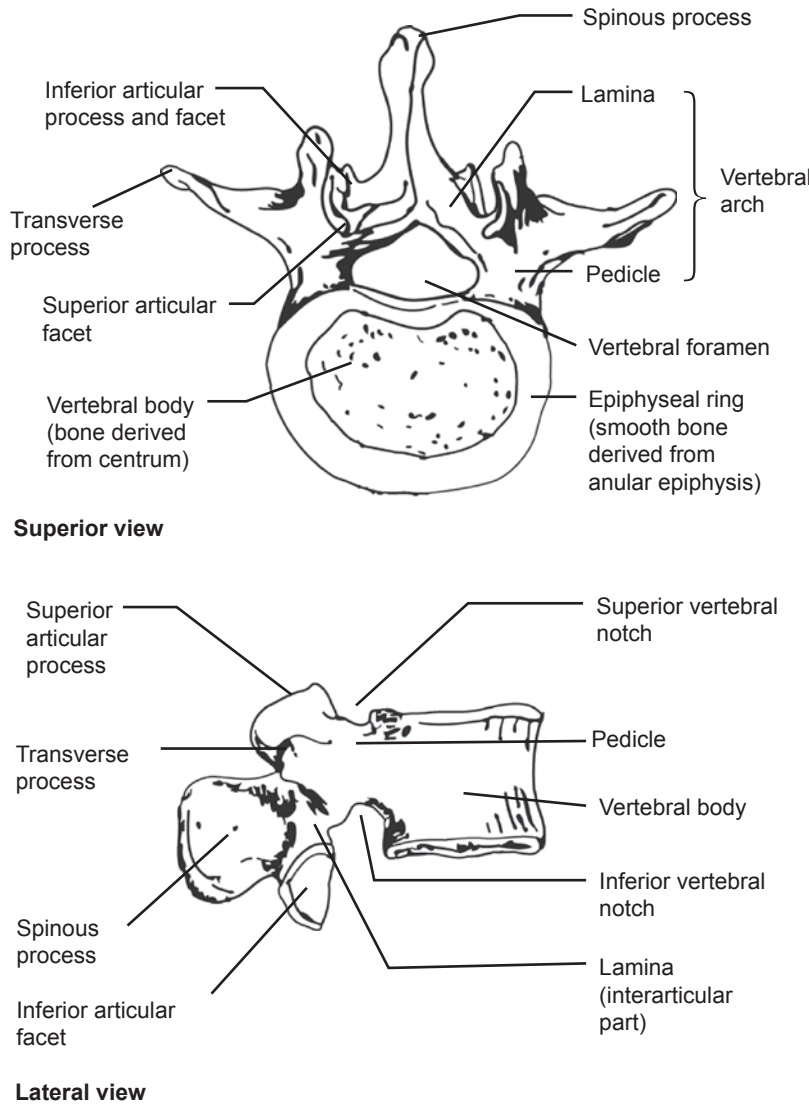

Fig. 1.5 Normal vertebra

paired with smaller and less developed musculature which makes head control and stabilizing the neck more difficult for younger children [11]. The increased elasticity of the supporting soft-tissue structures, particularly the interspinous ligaments, posterior capsule, and cartilaginous end plates in the growing child, contributes to the mechanical instability observed in the upper cervical spine and increased propensity for injury to this area [8]. Furthermore, with growth and development there is a change in the kinematic motion of the cervical spine as a consequence of a shift in the instantaneous center of rotation inferiorly. Early in childhood, the instantaneous center of rotation for flexion–extension exists at the C2–C3 level. Changes in the relative size of the head and the structural properties of the vertebrae, surrounding soft tissues, and musculature during growth alter the mechanics of the cervical spine. By the age of 8–10 years, the instantaneous center of rotation shifts inferiorly to the C5–C6 level, where it remains during adulthood [2, 8, 11, 25]. Therefore, the transformation in static and dynamic mechanical properties of the cervical spine that transpire during growth explains the pattern of cervical spine injuries seen in children with a higher incidence of upper cervical spine injuries occurring in children younger than

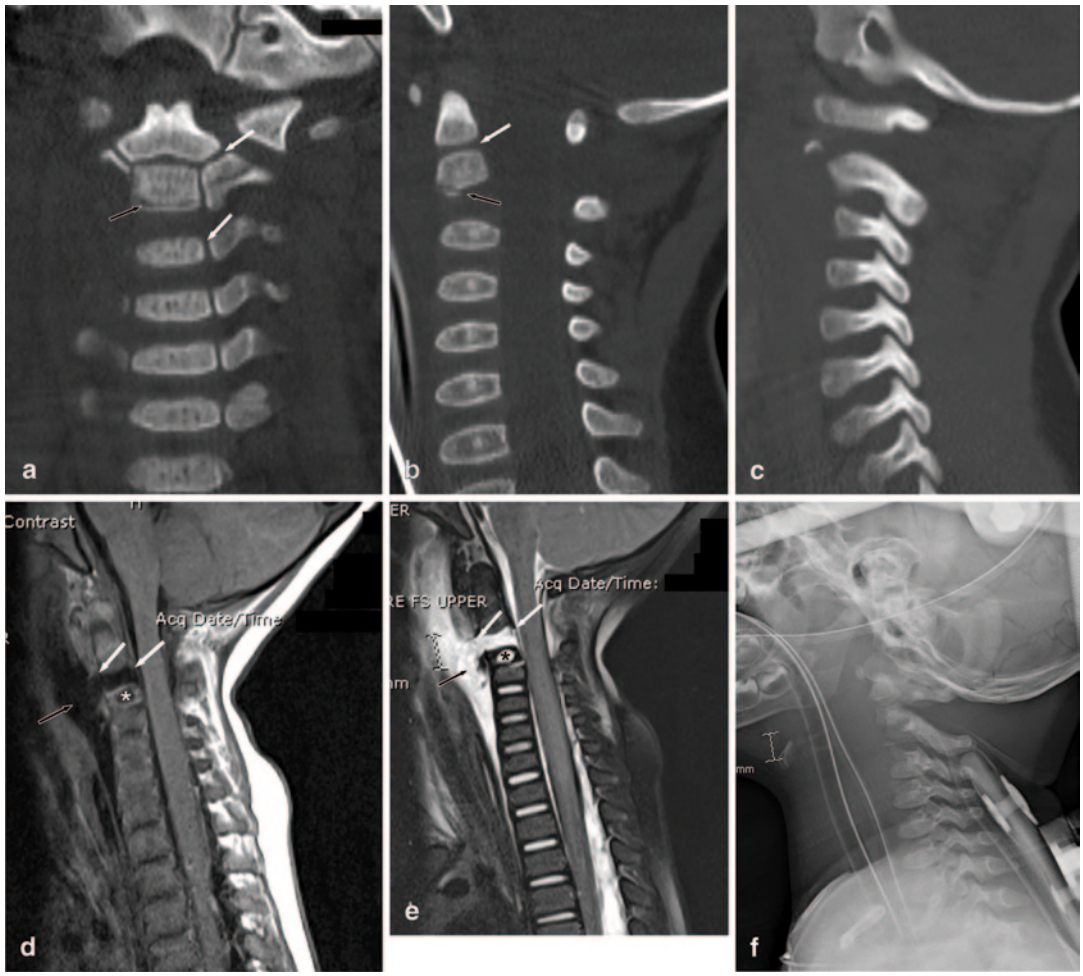

Fig. 1.6 Upper cervical spine injury in a 15-month-old child. The patient suffered a fracture of the inferior end-plate apophysis of C2 in a rollover motor vehicle collision. On coronal CT (**a**) and sagittal CT (**b**), note the differences between normal synchondroses indicated by *white arrows* and fracture of the end-plate apophysis indicated by *black arrows*. Sagittal CT through the facet joints (**c**) illustrates the normal shallow architecture of the joints which contributes to the pattern of upper cervical spine injury in young children. The same injury on T1 (**d**) and T2 (**e**) sagittal MRI, which again demonstrate the fracture, and also the widened disc space (*star*), hematoma (*black arrow*), and injury to ligaments (*white arrows*). Contrast these images with lateral plain radiograph after halo stabilization (**f**) and note the difficulty in interpreting the extent of the injury in a young patient. Many of the concepts discussed later in the chapter can be seen in these images

8 years and lower cervical spine injuries, resembling adults, in children older than 8 years.

Thoracolumbar Spine

Injuries to the thoracic and lumbar spine are less common in children than in adults. Similar to cervical spine injuries, the sagittal contours of the thoracic and lumbar spine segments change during growth affecting the degree of thoracic kyphosis and lumbar lordosis. This shifts the instantaneous center of rotation for these regions of the spine which alters spine kinematics and influences the location of thoracic and lumbar spine injuries in children relative to adults. In children, the most frequent locations for thoracic spine injuries are T6 and T7 and for lumbar spine injuries

L1 and L2, whereas in adults, thoracic fractures occur at T7 and T8, and lumbar fractures occur at the thoracolumbar junction, T12 and L1 [26].

The thoracic spine is unique due to its association with the rib cage. The articulations of the vertebrae with the ribs impart particular rigidity to the thoracic spine. The head of each rib articulates with the vertebral body anterior to the pedicles and the neck articulates with the transverse process. While these joints serve to increase rigidity and stability of this segment of the spine, they also serve to limit motion in the thoracic spine due to the articulation of the ribs with the sternum anteriorly. Motion of the spine is permitted through the costal cartilage as well as through the articulation of the ribs with the sternum [3]. Conversely, motion of the ribs and the chest wall can be affected by the anatomy of the spine. It should, therefore, be emphasized that spine, chest wall, and lung growth are interrelated. Anatomic changes to the ribs or to the spine, whether through injury, congenital abnormality, or iatrogenic causes, can affect chest wall growth and function and therefore respiration [27–29].

The anatomic relationship of the spine to the pelvis has implications to the sagittal balance of the spine as well as to the development of abnormal conditions that can occur in childhood and adolescence. The sacrum has a fixed anatomic relationship to the pelvis, requiring compensation of the spine in the sagittal plane to maintain balance and upright posture [30]. When the sacrum is angled relatively anteriorly relative to the pelvis, the lumbar spine must compensate with increased lordosis to keep the spine balanced and the trunk upright. Increased lumbar lordosis increases the susceptibility of the young athlete to spondylolisthesis, which is a relative translation of one vertebral body relative to another and is most common at the L5–S1 level [31].

Other anatomic variants can predispose the young athlete to lower lumbar conditions. Spondylolysis is a defect or abnormality in the region of the vertebra between the superior and inferior facet joints known as the pars interarticularis. This condition is most commonly seen in the young athlete undergoing repeated hyperextension of the lumbar spine in activities such as gymnastics and rowing [32, 33]. Spina bifida occulta is a common variant in the spectrum of spinal dysraphism in which there is incomplete closure of the posterior bony elements of the spine without herniation of intraspinal contents [34, 35]. This defect occurs in almost 2% of the population and is associated with spondylolysis [36]. Transitional lumbar vertebrae, in which the lower lumbar segments share features of the sacral spine or associate with the sacrum through overgrown transverse processes (e.g., Bertolotti's disease), are a recognized cause of back pain in children and may also be associated with spondylolysis. Transitional vertebrae are thought to be present in 4–8% of the population [37, 38].

Radiographic Variants in the Pediatric Spine

Essential to the proper evaluation of the pediatric spine is an understanding of the radiological anatomy of the growing spine (Table 1.1). When viewing radiographic images of the pediatric spine, it is important to consider the child's age and stage of spinal development to prevent misinterpreting a normal synchondrosis for a fracture. Synchondroses occur in predictable anatomic locations and have smooth, rounded edges with a sclerotic bone border. Fractures present radiographically as irregularly shaped lucencies with non-sclerotic borders in locations atypical for synchondroses [11].

The atlanto-dens interval (ADI) is measured on a lateral radiograph of the cervical spine and represents the distance from the posterior aspect of the anterior arch of C1 to the anterior aspect of the dens of C2 (Fig. 1.7). An increase in this interval might indicate disruption of the ligamentous structures supporting the atlantoaxial joint. In adults, this distance should be less than 2–3 mm, whereas in children up to 8 years of age, an ADI of up to 5 mm can exist with an intact transverse ligament [11, 39, 40]. Up to 20% of children have an ADI of 3–5 mm [41].

Table 1.1 Summary of normal radiographic variants in the pediatric spine

Finding	Children	Adults
ADI	Up to 4–5 mm	<3 mm
Pseudo-Jefferson	Displacement of lateral masses of C1 relative to C2 < 6 mm to age 4–7	No displacement
Wedging	3 mm, most common in C3 body	None
Odontoid epiphysis	Open until as late as 6 years, scar can be seen until age 11	Closed
Pseudosubluxation	Up to 2–3 mm of anterolisthesis, C2 on C3 most common	None
Cervical lordosis	Absent in neutral up to age 16	Present
Overhanging anterior arch C1	Up to two-thirds of arch above dens	None

ADI atlanto-dens interval

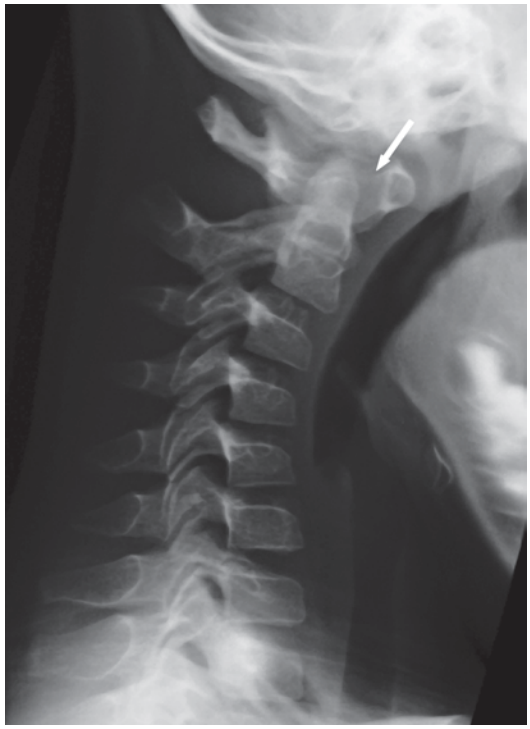

Fig. 1.7 Increased atlanto-dens interval (ADI, *arrow*). ADI can be increased in children when compared to adults, with an upper limit of 5 mm up to age 8; 20% of children will have an ADI between 3 and 5 mm

When viewing AP odontoid views of C1 and C2, the relationship of the lateral masses of C1 relative to the dens of C2 is different in children compared to adults. In children up to the age of 4–7 years, the displacement of the lateral masses of C1 relative to the articular surface of C2 can be up to 6 mm (Fig. 1.8) [42, 43]. The apparent offset of the lateral masses (pseudospread) can

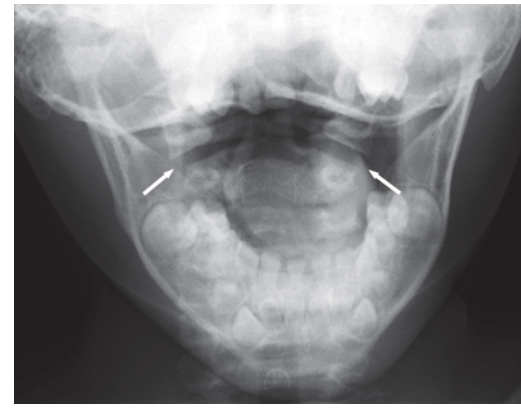

Fig. 1.8 Pseudospread of C1 in a 3-year-old child. On AP odontoid view, the lateral masses of C1 can be seen overhanging the articular surface of C2 (*arrows*). This overhang can be as much as 6 mm in children

be misinterpreted as a Jefferson fracture of the atlas caused by axial compression that disrupts the ring of C1 [44, 45]. In the pediatric patient, excessive lateral offset does not necessarily represent a fracture or ligamentous injury (pseudo-Jefferson), but is due to incomplete ossification of the dens and lateral masses. Owing to difficulty in obtaining a quality open-mouth view in children, as well as difficulty in interpreting the radiograph, it has been recommended not to obtain this projection in children under the age of 5 [11, 46].

To facilitate interpreting lateral radiographs of the cervical spine for subluxation or listhesis (i.e., anterior or posterior translation of one adjacent vertebral body relative to the other), the spinolaminar line is formed by connecting the anterior portion of consecutive spinous processes to

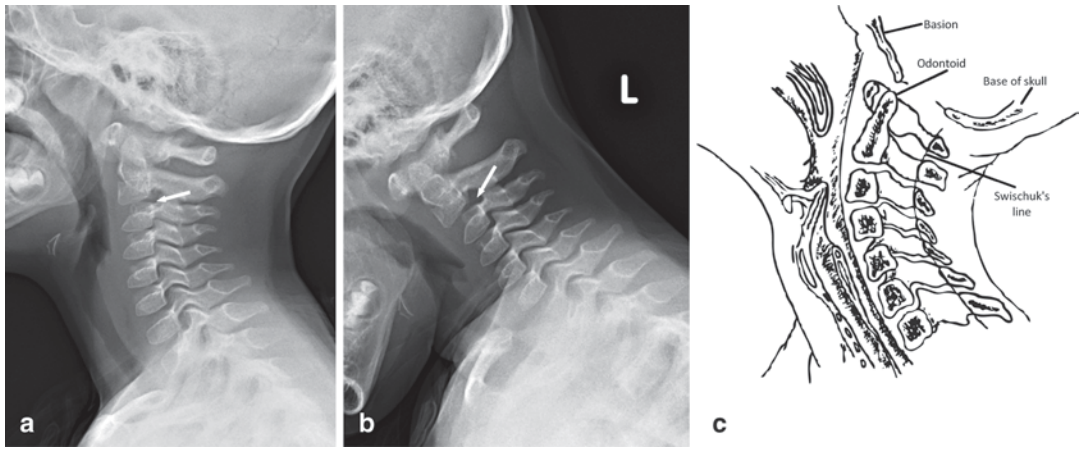

Fig. 1.9 Pseudosubluxation. Appearance of pseudosubluxation (*arrows*) on lateral radiograph in both **a** neutral and **b** flexion. A normal physiologic listhesis can be seen in the c-spine of children, with C2–3 the most common location. This is typically less than 2–3 mm. Further, note the disproportionate increase in distance with flexion between the C1 and C2 spinous processes. **c** The spinolaminar line (Swischuk's line) can be used to differentiate pseudosubluxation from true injury

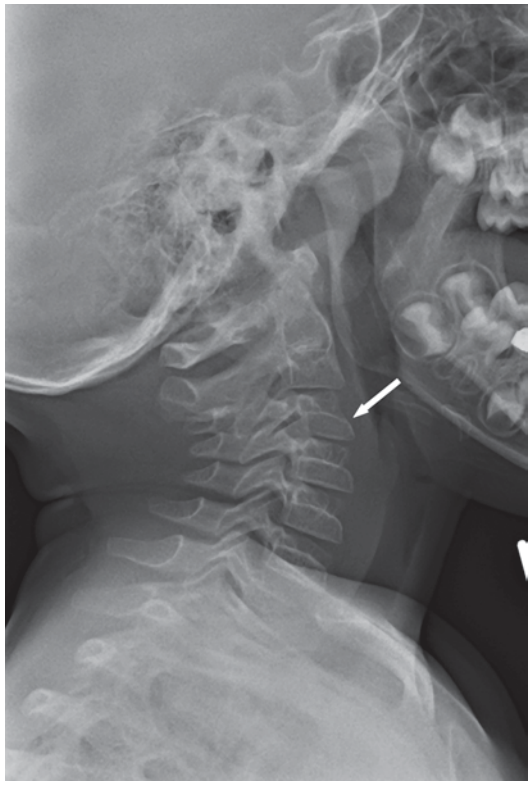

Fig.1.10 Wedging of C3 (*arrow*). A common finding on lateral plain radiographs

create a smooth, unbroken line that passes within 1.5 mm of the spinous process of C2 [47, 48]. A value greater than this is supra-physiologic; it implies listhesis or subluxation. A spinolaminar line distance less than 1.5 mm distinguishes "pseudosubluxation" from true instability patterns in children [11, 39, 49, 50]. Pseudosubluxation is most common at the C2–C3 level followed by the C3–C4 level and can appear as a relative listhesis of up to 2–3 mm between adjacent vertebral bodies (Fig. 1.9) [39]. Pseudosubluxation results from a number of factors, including incomplete ossification of the vertebral bodies, physiologic laxity of ligamentous structures, and facet joint morphology and orientation.

Before the spine becomes completely ossified, the vertebral bodies can appear to be abnormal in shape when imaged using plain radiography. This is most pronounced at C3 where the anterior portion of the body appears to be wedge-shaped on lateral radiographs and is often confused with a wedge compression fracture (Fig. 1.10). Up to a 3 mm difference between the anterior and posterior heights of the vertebral body can be considered physiologic [11]. As ossification of the vertebral body progresses, the vertebrae will take on their

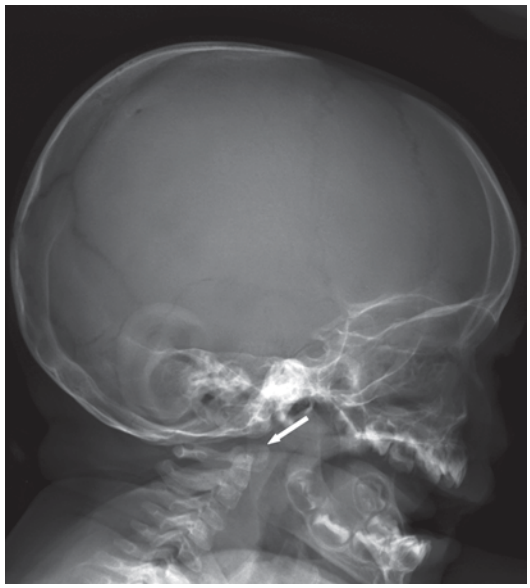

Fig. 1.11 Overriding arch of C1 on lateral radiograph of an 18-month-old child (*arrow*). Up to two-thirds of the arch of C1 can project above the dens in children. Further, note the large head relative to the spine in this younger child

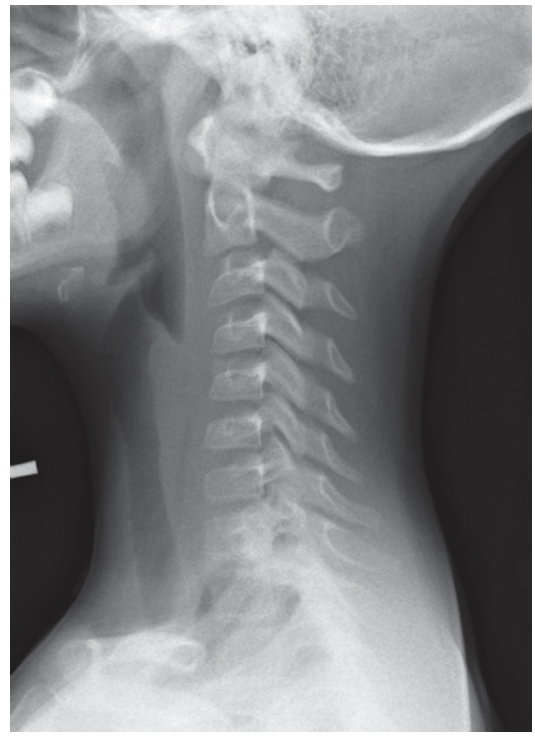

Fig. 1.12 Lack of cervical lordosis in an 8-year-old child

normal rectangular appearance. Complete ossification occurs in a majority of children by 7 years of age, but some mild residual wedging can persist into adolescence [51].

In the adult, the anterior arch of C1 projects anterior to the odontoid process of C2 when imaged in the lateral projection on plain radiographs. As a consequence of incomplete ossification, this relationship can appear abnormal in a child: because the tip of the dens tends to ossify later than the anterior arch of C1, the arch appears to sit superior to and override the dens (Fig. 1.11) [49]. Up to 20% of normal children aged 1–7 years may have up to two-thirds of the anterior arch of C1 project superior to the dens [2, 11].

Cervical spine lordosis normally develops over time. Thus, in children up to age 16, absence of cervical spine lordosis imaged radiographically in the lateral projection with the neck in neutral position may not be indicative of injury (Fig. 1.12) [52]. As a general rule, the distance between consecutive spinous processes should not exceed 1.5 times the interspinous distance of the level above or below. Measurements that exceed this distance might indicate a true flexion-type injury. If flexion and extension lateral radiographs are obtained, it should be noted that in children the posterior occipitocervical ligaments are relatively tighter than the interspinous ligaments, and the distance between C1 and C2 on the flexion view may increase disproportionally [49].

Evaluation of the soft tissues on plain radiographs can be useful in evaluating the cervical spine for injury. Swelling, hemorrhage, or inflammation can increase the projected width of the anterior soft-tissue density observed on lateral radiographs and alert the clinician as to the possibility of an occult injury. In the pediatric patient, the retropharyngeal soft-tissue density should be less than 7 mm, and the retrotracheal space should be less than 14 mm (Fig. 1.13); however, these values can be falsely increased in the screaming or crying child [2, 53].

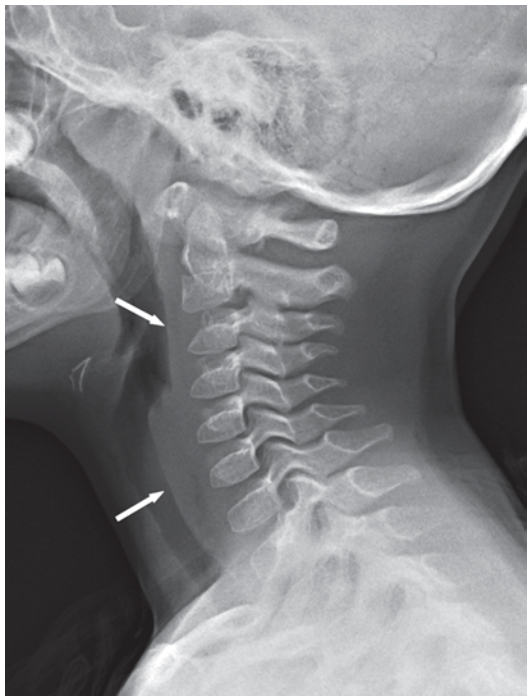

Fig. 1.13 Normal appearance of prevertebral soft tissues on lateral radiograph (*arrows*). Retropharyngeal tissues should not exceed 7 mm while the retrotracheal tissues should not exceed 14 mm

Spinal Cord Injury Without Radiographic Abnormality

A condition unique to the pediatric population is an entity known as "spinal cord injury without radiographic abnormality," or SCIWORA [54]. This is defined as a neurological deficit following trauma in the absence of any identifiable bony or ligamentous injury to the spinal column observed on imaging studies. This phenomenon is a consequence of the differential elasticity of the vertebral column relative to the spinal cord that normally exists in growing children. The ligamentous elements of the spine can stretch up to 5 cm, while the spinal cord can only tolerate approximately 0.5–1 cm of distraction before suffering serious injury or rupture. SCIWORA comprises 18–38% of cervical spine injuries in children. Magnetic resonance imaging (MRI) is the best imaging modality to define the location and extent of spinal cord trauma in SCIWORA-related injuries [55, 56].

Os Odontoideum

Os odontoideum is a condition that the clinician should be aware of when evaluating the young athlete. It appears as a separate ossicle with smooth cortical margins (Fig. 1.14) [57]. The os is typically seen above the level of the facets, and therefore above the level of the dentocentral synchondrosis. Os odontoideum is often found incidentally on plain radiographs of the cervical spine, frequently without a history of antecedent trauma. It is important to recognize this entity both because it does not represent an acute fracture and because of implications for potential instability during contact sports [58]. It should be distinguished from a nonunion of the os terminale, which is not necessarily associated with spinal instability.

Several theories regarding the etiology of this entity have been proposed, including congenital failure of fusion of the odontoid process or avascular necrosis resulting from trauma. There is now general agreement that os odontoideum is likely secondary to a traumatic process, although a specific incident of cervical spine trauma may be remote or not identified [59].

Summary

Knowledge of the normal development and anatomy of the growing spine is essential for the proper evaluation of the young athlete's spine. Children over the age of 8–10 years tend to experience spine injuries similar to those observed in adults. However, children under 8 years of age are at particular risk for injuries to the upper cervical spine as a result of their relatively larger head size and changes in the structural properties of the vertebrae, surrounding soft tissues, and musculature during growth that alter the static and dynamic mechanical properties of the spine. When viewing radiographic images of the pediatric spine, it is important to consider the child's age and stage of spinal development to prevent misinterpreting a normal synchondrosis or unossified portion of the vertebra as a fracture or injury pattern.

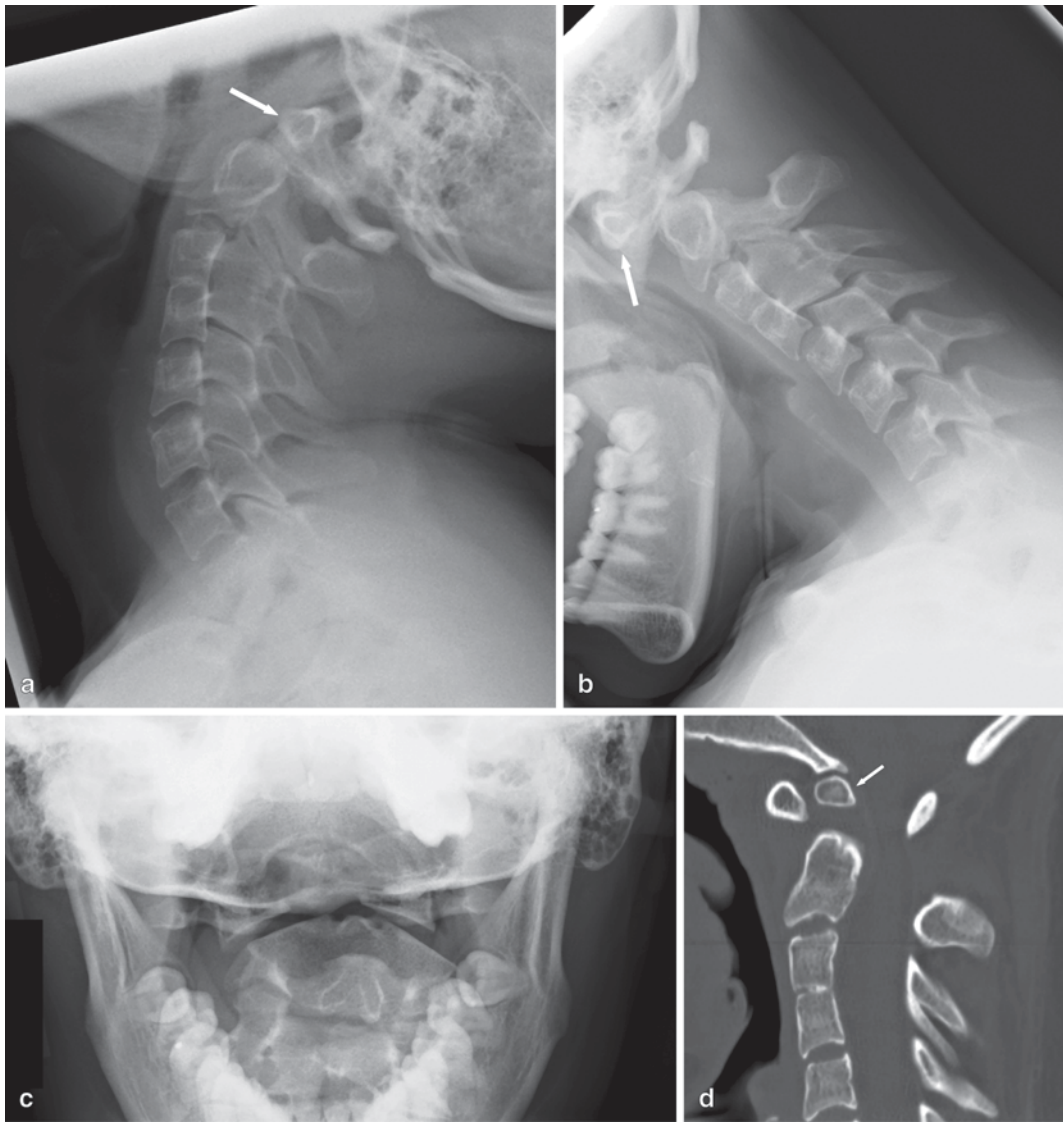

Fig. 1.14 Appearance of os odontoideum in an 18-year-old patient. **a** Lateral extension plain radiograph (*arrow*, anterior arch of C1). **b** Flexion radiograph (*arrow*, anterior arch of C1). **c** Open mouth odontoid view. **d** Sagittal CT scan (*arrow*, os odontoideum)

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The Young Athlete's Spinal Mechanics

2

Robert A. Donatelli and Michael S. Thurner

Introduction

The young athlete who engages in exercise and sport has distinct health benefits. However, the dangers associated with participation in sports at a young age are high. Cupisti et al. [1] reported that 30% of adolescents have experienced some form of back pain and this is a frequent complaint among young athletes, especially in girls. The spine, in the youth athlete, is still in the developmental process of fully maturing into the adult spine. The immature spinal muscles, ligaments, tendons, and fascia tissue may lag behind bone growth [2]. The young athlete's musculoskeletal system may be at a disadvantage secondary to asymmetries and soft tissue developmental deficits. Although the young athlete's spine is still developing, the musculoskeletal structures are required to perform complex spinal movement patterns that involve bending and twisting at high speeds during sporting activities. Injuries to the spine increase significantly from 12 to 17 years of age as a child undergoes rapid growth and development causing physiological changes as the anatomical structures evolve to skeletal maturity [3]. The purpose of this chapter is to present the biomechanics of the adolescent spine and describe how the articulations of the axial skeleton provide stability and movement in the youth athlete. A focus of emphasis is on development, functional anatomy, biomechanics, and kinematics as it relates to movements of the spine during sporting activities. An understanding of the normal anatomical architecture of the spine and its associated biomechanical properties is important for gaining an appreciation of pathomechanics and recognize abnormal movement patterns or postures caused by trauma, overuse, or developmental abnormalities. In addition, the interaction of the passive, active, and neural subsystems, as described by Panjabi, is introduced to conceptually illustrate the intricate stabilization system of the spine.

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Special Considerations in the Young Athlete's Spine

Development and Spinal Postures of the Young Athlete

There are several factors of the spinal developmental process that have an influence on mechanics and associated injuries in the young athlete. Portions of the pediatric spine will not completely ossify well into adolescence [4]. In addition, the young spine has greater elasticity of the disc and surrounding ligaments which allows for greater flexibility. The growing athlete also demonstrates musculature immaturity resulting in reduced strength. The combination

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of increased flexibility, muscle weakness, and incomplete ossification may result in altered mechanics and greater potential for injury [4]. Growth spurts during early puberty can lead to changes in spinal mechanics in the young athlete's spine secondary to asymmetries, such as leg length discrepancies, growth plate trauma, sprains and strains to the soft tissue structures, and muscle damage [2]. Young athletes who participate in aggressive competitive sports, such as football and gymnastics, have a greater risk of developing poor, unnatural spinal postures which may alter spinal mechanics. In particular, due to the high impact loading and repetitive extension-biased movement patterns commonly associated with gymnastics, excessive lumbar lordosis and injuries such as spondylolysis and spondylolisthesis are prevalent. High-speed impact collisions occur frequently as is the nature of the sport in American rules football increasing the risk of traumatic spinal and concussion type injuries. Also note that pediatric and preadolescent athletes are more susceptible to traumatic spinal injury, especially in the premature cervical spine in contact sports that expose these young athletes to excessive high-speed impact forces. The high-impact sports referred to include, but not limited to, American rules football and gymnastics. Due to the repetitive movements, excessive forces, and heavy tension placed on joints that occur during competitive sports, 50–85% of young athletes participating in the vigorous sports previously noted are at an increased risk of spinal injury [2, 5]. Sports that require repetitive motion predominantly in one direction subsequently puts the spine at risk of developing pathological curvatures of the spine, such as scoliosis [6]. Sward has reported scoliosis in up to 80% of youth athletes participating in sports that promote asymmetrical loading to the trunk and shoulders [6]. The referenced sports that involve overhead throwing athletes include, but not limited to, tennis, baseball, and javelin.

The curvature of the vertebral column is naturally engineered through development and adaptation to the normal forces of gravity as we mature to upright bipedal locomotion. The human vertebral column is comprised of four reciprocating curvatures in the sagittal plane (Fig. 2.1). In

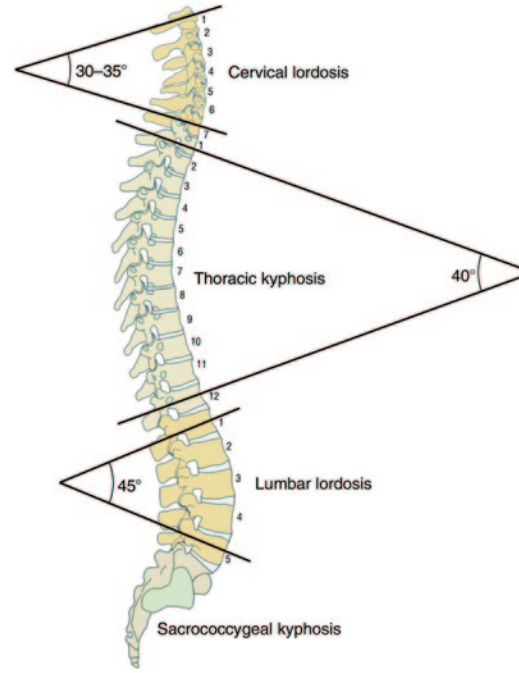


Fig. 2.1 Normal curvatures of the spine

context from a sagittal view, the cervical spine is concave posteriorly, the thoracic spine is convex posteriorly, the lumbar spine is concave posteriorly, and the sacrum/coccyx is convex posteriorly. In a neutral erect standing posture, the forces of gravity act to maintain these natural curvatures [7] (Fig. 2.2). The term "neutral spine" refers to these natural anatomical curvatures in a healthy spine. The structural integrity of these spinal curvatures allows for load transmission and dispersion of forces due to its ability to "give" slightly under a load [7].

Orientation of the spine and the ensuing posture varies with age, gender, and weight of the subject. A physiologically healthy aligned spine is an essential component in the function of the human body [7]. Cil A. et al. [8] observed the development of sagittal segmental alignment of the spine in 151 children (72 girls and 79 boys) between the ages of 3 and 15 years without musculoskeletal abnormalities. Sagittal spinal alignment was found to be continuously changing as the child grows. Cil A. et al. [8] reported that, as the young athlete's age increases, there is a noteworthy change in the total curvature of thoracic kyphosis and lumbar lordosis. In addition,

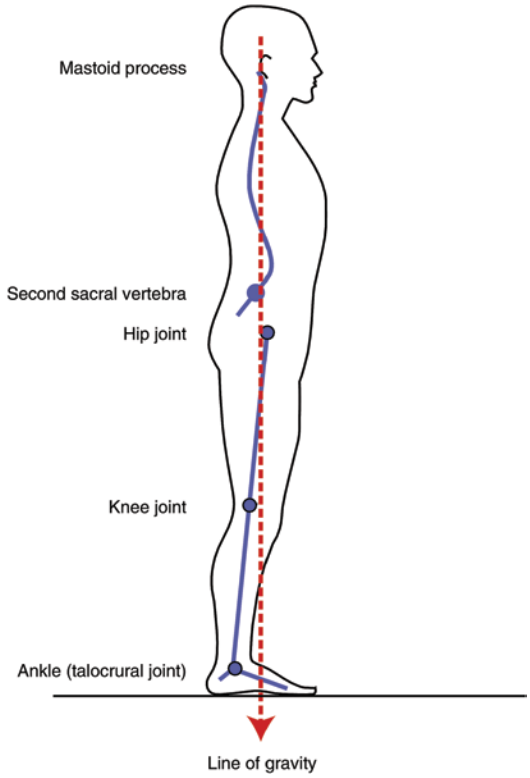


Fig. 2.2 Line of gravity passing through the body

the above study reported a statistically significant difference among different age groups, especially evident at the cervicothoracic, thoracolumbar, and lumbosacral junctions.

Functional Anatomy and Spinal Mechanics of the Youth Athlete

The following section reviews functional anatomy integrated with currently accepted concepts of biomechanics. This section includes the craniocervical, thoracolumbar, and lumbosacral regions of the vertebral column.

Craniocervical Region

The craniocervical region consists of the atlanto-occipital, atlanto-axial, and intracervical apophyseal joints. The atlanto-occipital joints articulate from the convex occipital condyles and the reciprocally concave superior articular facets of the atlas. Movement primarily occurs about a frontal axis producing motion of flexion and extension in the sagittal plane [7]. The atlanto-axial joint is noted for its unique articulation consisting of a vertically protruding odontoid process from the axis projecting through an opening formed by the anterior arch of the atlas and the transverse ligament. Approximately one-half of cervical rotation occurs at the C1–C2 joint about a nearly vertical axis [7]. The intracervical apophyseal, or facet joints from C2 to C7, are aligned between the horizontal and frontal planes on a 45° slope [7]. This orientation provides freedom of movement in all three planes of motion allowing for considerable range of motion characteristic of the cervical spine. If combined, the craniocervical region may allow up to 120–130° of flexion/extension, 65–75° of axial rotation, and 35–40° of lateral flexion [7, 9]. Evidence of lower cervical spinal coupling patterns exists and is purported as a simultaneous ipsilateral coupling between rotation and lateral flexion [10]. A normal, healthy mature cervical spine naturally exhibits a lordotic curvature of approximately 30–35° representative of its anatomical architecture [7].

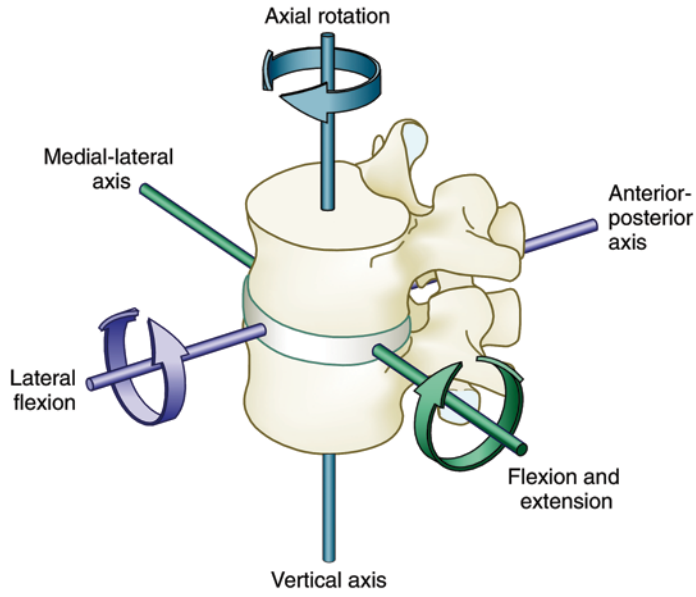
Anatomical, biomechanical, and kinematic changes in the cervical spine facet joints occur throughout the growing years [11, 12]. The cervical spine ossification centers, commonly referred to as growth plates, are beginning to fuse, but are incompletely ossified at the age of 8–12 years [3]. The complete fusion of the end plates occurs at 21–25 years of age [3]. The facet joints establish a mature configuration at 8 years of age, but an oblique mature adult pattern is not fully developed until the age of 15 [3]. The facet joint is formed by two adjacent vertebrae consisting of the inferior facets of the superior vertebra articulating with the superior facets of the inferior vertebra [7, 13]. The pediatric and preadolescent cervical spine has a unique anatomy and associated biomechanics compared to its adult counterpart. The fulcrum of motion in the cervical spine in the young athlete is at the C2–C3 level, while in adults it is at the C5–C6 level [11, 12]. Younger children exhibit more anteriorly wedge-

shaped vertebrae, most notably in the upper cervical spine [11, 12]. The orientation of the articular surfaces of the facet joints is more horizontal and shallow when compared to the adolescent and adult cervical facets in the cervical and upper thoracic regions, which enables a greater degree of mobility, including spinal coupling to occur [11, 12, 14]. The joint capsules, ligaments, and cartilaginous end plates in the pediatric and preadolescent cervical spine are much more elastic than those of adults secondary to the viscoelastic properties, which further add to the cervical spine instability in the young athlete [15, 16]. In essence, the young spine will stretch before it breaks, thus rendering the immature spine more predisposed to soft tissue stretching injuries such as sprains, dislocations, growth plate separation, which may include Salter–Harris type fractures, or elongation injury to the spinal cord [17]. A more cephalic cervical fulcrum, underdeveloped curvature due to the anterior wedge shape of the vertebrae, and shallow orientation and horizontal alignment of the facets, along with ligamentous laxity characteristic in the premature spine, contribute to high torque and shearing forces acting on the C1–C2 segments. Furthermore, incomplete ossification or calcification of the odontoid process and the relative size of the torso and cervical spine to the comparatively large head along with relatively weak and underdeveloped neck muscles predispose the pediatric cervical spine to instability and injury [15, 16]. All of the dynamic maturation changes noted above occur during development throughout the pediatric and preadolescent years, 3–15 years of age.

Thoracolumbar and Lumbosacral Regions

The thoracic vertebrae are inherently stable in nature due to its anatomical relationship to the encompassing rib cage. The thoracic region consists of 12 vertebrae T1–T12 and the corresponding ribs bilaterally, 24 in all. A hallmark of the thoracic spine, the structural stability protects the underlying organs, provides a stable base for muscles to precisely control the mobility of the craniocervical region (equilibrium and vision), and is designed with enough pliability for the expansion of the chest cavity to allow for breathing. The intrathoracic apophyseal or facet joints from T1–T12 are aligned predominately in the frontal plane oriented on a small slope from vertical approximately 15–25° [7]. Variance of the anatomical architecture within the thoracic vertebral column is representative of the transition of structural characteristics to the adjacent cranial and caudal regions, cervical and lumbar spine respectfully. Segmental mobility in this region is relatively small due to the stability provided by the attachment sites of the ribs at the costoporeal and costotransverse joints of the corresponding vertebrae [7]. However, as a cumulative summation of segmental motion, the thoracic spine allows for approximately 50–65° of flexion/extension, 30–35° of axial rotation, and 25–30° of lateral flexion [7, 9]. In a neutral, erect standing posture, a reciprocating curve with respect to adjacent regions of approximately 40–45° of kyphosis is naturally exhibited in the mature thoracic spine [7].

The articular facets of the lumbar vertebrae are aligned vertically predominately in the sagittal plane [7]. This orientation advocates substantial sagittal plane flexion and extension about a frontal axis while allowing only limited rotation and lateral flexion within the lumbar spine segments (Fig. 2.3). The five lumbar vertebrae allow up to 55–70° of flexion/extension while only 10–15° of rotation are inherently available of all the lumbar segments combined [7, 9]. The lumbosacral junction of L5–S1 typically transitions to a more frontal plane alignment [7]. The sacrum angles away from the caudal lumbar vertebrae in an anterior and inferior direction approximately 40° from the horizon [7]. The lumbar spine exhibits a natural lordotic curvature of approximately 45° before transitioning to a reciprocal kyphotic curvature of the sacrum and coccyx [7]. It is important to note that the curvature of the lumbar vertebral column is dynamic in nature inherently influenced by the integrated movement of the lumbar spine and the pelvis known as lumbopelvic rhythm. The segments of the sacrum and coccyx are permanently fused early



Terminology Describing the Osteokinematics of the Axial Skeleton

Common Terminology	Plane of Movement	Axis of Rotation	Other Terminology
Flexion and extension	Sagittal	Medial-lateral	Forward and backward bending
Lateral flexion to the right or left	Frontal	Anterior-posterior	Side bending to the right or left
Axial rotation to the right or left*	Horizontal	Vertical	Rotation, torsion

*Axial rotation of the spine is defined by the direction of movement of a point on the *anterior side* of the vertebral body.

Fig. 2.3 Osteokinematics of the axial skeleton

in life and thus structurally have no ability for movement to occur between segments. Limited movement may be possible between the sacrum and innominate bones, but this represents a trace amount and controversy exists as to the presence of movement occurring at all.

In the lower thoracic and lumbar regions of the spine, the facets gradually become more vertically oriented, which limits the mobility of the spine in both lateral bending and rotation [18]. However, this decrease in flexibility protects the intervertebral discs and spinal cord from non-physiological kinematic and kinetic exposures that could create pathological conditions and potentially result in injury [19]. Voutsinas and MacEwen [20] reported a gradual and relatively small overall change in the magnitude of the cumulative total of thoracic kyphosis and lumbar lordosis during growth and development (36.7° of thoracic kyphosis in children 5–9 years of age to 38.5° in young adults 15–20 years of age and 52.2° of lumbar lordosis in children 5–9 years of age to 56.6 in young adults 15–20 years of age). As the young athlete grows, the spinal curvatures demonstrate maturation changes until development is complete. During growth spurts, different forces that result from the changes of maturation to the spinal curves may potentially be destructive to the bone itself, surrounding musculature and soft tissue structures.

Cil A. et al. [8] determined that the position of the sacrum (inclination and translation) and spatial orientation change during growth. The pelvis has been described to rotate and translate about the axis of the hip [21, 22]. The sacral inclination angle inherently influences the extent of hip extension and pelvic rotation (Fig. 2.4). In a study by Cil A. et al. [8], the sacral inclination angle measurements revealed that subjects in the pediatric age group had lower sacral inclination

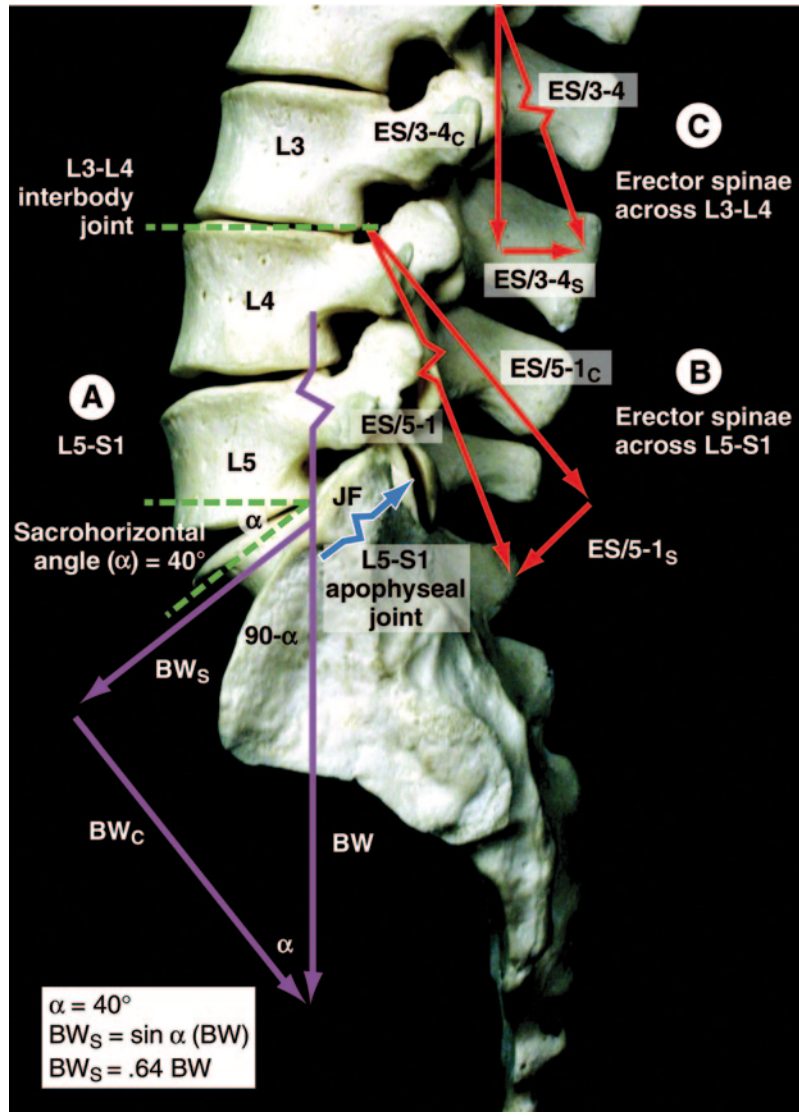


Fig. 2.4 Sacrohorizontal angle

angles than adolescents and adults. As the lumbar lordosis increases with age, sacral inclination also increased. The increased sacral inclination resulted in a more horizontal sacrum in which the sacropelvis rotates anterior to the hip axis resulting in decreased standing hip extension [8]. Reduction of hip extension in the young athlete may subsequently promote greater extension in the lumbar spine during sporting activities such as the golf swing, tennis serve, and baseball pitching. The compensatory hyperextension of the lumbar spine may be a factor in the etiology related to the high incidence of mechanical low back injuries in the young athlete. Due to the vigorous demands and repetitive nature of the sport, gymnastics has also been identified as a sport associated with lumbar extension injuries. The traumatic forces imposed on the pediatric spine during the repetitive motions in gymnastics may result in spondylolysis, a stress fracture of the pars interarticularis of the vertebral arch usually occurring in the lower lumbar region. In severe cases, the traumatic stress exceeds the structural integrity of the vertebral arch resulting in displacement of a vertebra relative to adjacent vertebrae, referred to as spondylolisthesis.

Mac-Thiong JM. et al. [23] reported that pelvic tilt and lumbar lordosis are two parameters, interdependent on each other, that increase with age to avoid inadequate anterior displacement of the bodies' center of gravity to maintain an adequate sagittal balance during growth and development. In summary, the youth athlete participating in competitive sports may be subject to excessive forces or abnormal loading on the musculoskeletal system causing changes or adaptations of spinal and pelvic–sacral posture.

Current Concepts and Biomechanical Considerations

Study of the anatomical architecture of the zygapophyseal, or facet joints of the vertebral column, and its relationship to the biomechanical behavioral characteristic predictability have become the focus of recent attention. The pediatric and adult spines alike were once thought of as following similar predictable biomechanical patterns by rule of physics law. The facet joints are located at the junction of each vertebral level from the cervical to lumbar spine on the posterior lateral aspect of each motion segment [13]. The orientation of the apophyseal joints directly influences the kinematics, or possibly better described as the directional movement tendencies characteristic of the distinct, yet integrated regions of the vertebral column [7, 13]. Current evidence suggests an integrative role of anatomical structural properties of an array of surrounding tissues and neuromuscular control mechanisms contributing to spinal coupling patterns [13, 24–27]. Contrary to previous thought, this concept proposes a potential of variance at each segment or region, most notably among individuals and would be especially evident throughout development as maturation changes occur. Recent reviews of the literature highlight the inconsistencies of spinal coupling behavior questioning the previously accepted biomechanical "laws" of spinal motion based upon the concave–convex rule [24–27].

Spinal coupling is defined as a kinematic phenomenon consistent of one motion of rotation or translation of a vertebral body about or along an axis associated with another motion about or along a second axis [7, 24, 25]. Currently, there is no definitive study that concludes a consistent coupling pattern or the mechanism by which these coupled motions can be explained. A review of the literature by Legaspi and Edmond [26] reports that the concept of coupled motion in the lumbar spine has been studied extensively with little consensus as to its presence and direction. Sizer et al. [27] report similar findings in a review of the literature on coupled motion of the thoracic spine. Cook et al. [27] report consistent coupling patterns in the lower cervical spine with variable patterns of the upper cervical spine coupling in a recent review of the literature. References most commonly cited in the literature on spinal coupling are based on the previous work of Lovett and Fryette's "laws" of spinal motion. These so-called biomechanical laws of motion lack sufficient evidence as to their efficacy in the adult or adolescent spine, as previously discussed.

Review of the Stabilizing System of the Spine

Panjabi [28] describes three subsystems as the foundational premise of the spinal stabilizing system. The interaction of the passive, active, and neural subsystems conceptually acts to provide stability to the spine to meet the demands of postural changes, static and dynamic loads placed upon it in an ever-changing environment. The *passive system* consists of vertebrae, facet articulations, intervertebral discs, spinal ligaments, joint capsules, and the passive mechanical properties of the surrounding musculature [28]. The *active system* includes the musculature and associated tendons surrounding the vertebral column [28]. The passive and active system complement one another by providing static stability at end range of motion and dynamic stability during movement, respectively. The *neural and feedback system* as described by Panjabi modulates the active, muscular system based on the internal and external demands placed upon the body through a complex network of afferent and

efferent neural feedback and responses, referred to as neuromuscular control [28, 29]. The efficiency of these systems may be compromised during growth and development as the soft tissue and skeletal structures mature at different rates inadvertently requiring continuous adaptation of the neuromuscular control system consequently affecting the integral balance of the integrated subsystems potentially rendering the youth athlete at a higher risk of injury.

In conclusion, the pediatric spine undergoes developmental changes as the skeletal system evolves into the mature spine. An understanding of the functional anatomy, biomechanics, and kinematics of the mature adult spine is important for appreciating differences in comparison to the pediatric immature spine and the potential injury risk in the youth athlete. Current evidence suggests an integrative role of the anatomical architecture, structural properties of surrounding tissues, and neuromuscular control mechanisms contributing to spinal coupling patterns. The stabilizing subsystems of the spine continuously attempt to adapt to the changes that occur throughout the maturation process to counteract the developmental instability characteristic of the pediatric spine.

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Acute Thoracic and Lumbar Injuries

3

Michael P. Glotzbecker and Daniel J. Hedequist

Introduction

Low back pain is very common in the general population. Nearly 30% of athletes will experience low back pain at some point in their career [1–3], and spine injuries account for 9–15% of all athletic injuries [4]. Acute injuries are often more prevalent than overuse injuries [5]. Most injuries to the thoracic and lumbar spine represent muscle strain or contusion and are self-limited [5]. However, it is important to identify more serious conditions that may require treatment.

A recent review of 4,790 collegiate athletes participating in 17 varsity sports demonstrated a back injury rate of 7 per 100 participants [5]. In a review of injuries found in National Football League (NFL) players, 7% affected the spine or axial skeleton; 44.7% of these injuries involved the cervical spine, with the lumbar spine being the second most commonly affected (30.9%), and the thoracic spine/ribs was relatively less involved (3.9%) [6]. Thoracic injuries were more specifically broken down into muscle injury or sprain (75.6%), fracture (11.6%), disc injury (4.7%), other (4.7%), rib (2.3%), and nerve injury (1.2%) [6]. Lumbar injuries were more specifically broken down into muscle injury or sprain (46%), disc injury (28%), other (8.8%), contusion (7.9%), fracture (6.6%), and nerve injury (2.6%) [6].

Sports that demonstrate increased risk for back injuries include gymnastics and football, and a majority (80%) of spinal injuries occur during practice, rather than during competition [5]. For football players, thoracic spinal injuries are most commonly caused by tackling (19.8%), blocking (18.6%), or being tackled (18.6%) [6]. Lumbar spine injuries occur most commonly during noncontact activity (20.8%) followed by blocking (18.6%) [6]. Other sports that have an increased incidence of low back injury include wrestling, rowing, rugby, skiing and ski jumping, hockey, ballet, swimming, diving, weight lifting, running, baseball, and golf [2–4, 7–9]. Risk factors for lumbar injury and back pain include prior back injury, decreased range of motion, improper technique, abrupt increases in training, and poor conditioning [3, 10, 11]. Those with previous back injuries are three times greater to suffer a back injury than those who did not have previous injury [10].

While the incidence of injuries to the thoracic or lumbar spine may be relatively lower than other musculoskeletal injuries, the morbidity including time missed from these injuries can be significant. In a study of NFL players, axial skeleton injuries resulted in an average of 21.3 practices missed and 4.4 games missed [6]. Other studies have shown significant lost playing time at some point in a collegiate football player's career (30%) [12] and 38% of professional tennis

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players have missed at least one tournament secondary to low back pain [8].

General Evaluation

The evaluation of a patient with an acute lumbar or thoracic spinal injury starts with a thorough history and exam [3]. This includes understanding the mechanism of injury. It is important to understand duration, location, and severity of symptoms. Previous back injuries or surgeries should be documented. The type of sport and level of sport should be understood, and previous treatment/evaluation of the back should be considered.

In the acute on-field injury, the patient should be assessed for the airway, breathing, and circulation (ABCs). The neck should be immobilized and the patient placed on a backboard if there is a suspected spinal injury [4]. Assessment for weakness and numbness as well as loss of consciousness or altered mental status should be documented [4].

In the less acute situation, the physical examination should include an assessment of posture and gait. Palpation of the spine can identify more specific areas that may be affected. Palpation of the ribs can isolate a rib injury. Range of motion of the spine should be assessed. Any pain with flexion or extension should be documented, as pain with forward flexion may indicate pain originating from the disc, while pain with extension may indicate pain related to the posterior elements of the vertebrae. A full neurologic exam should be performed, including strength and sensation testing, as well as testing of lower extremity reflexes. A straight leg test can assess for sciatic or nerve root irritation from a herniated disc. Hip range of motion and a flexion, abduction, external rotation (FABER) maneuver can be performed to rule out hip/sacroiliac joint pathology masquerading as lower back pathology. An abdominal exam should also be performed to rule out any associated or confounding intra-abdominal injuries.

Imaging in a young athlete with acute onset back pain often includes anterior–posterior (AP) and lateral views of the affected area as these radiographs will demonstrate up to 90% of bony fracture and alignment issues [4]. If ligamentous injury is suspected, flexion/extension radiographs can be used to identify dynamic instability, but a patient must be comfortable enough to participate. Computed tomography (CT) can be helpful if an occult fracture is suspected, but is not routinely required. It is the optimal study to assess bony morphology of a suspected injury, but it does expose the patient to ionizing radiation which is of particular concern in the pediatric population. Magnetic resonance imaging (MRI) is particularly helpful in assessing the disc, nerve roots and spinal cord, and other soft tissues. It should be reserved for patients with neurologic findings, a history highly suspicious of a disc herniation, or to assess occult injury that is not identified on plain radiography. Bone scan can also be utilized to identify an injury as it has a high sensitivity but identifying a clear diagnosis may be difficult given the low specificity of the test. If a fracture is identified, the rest of the spine should be carefully screened as up to 32% of patients may have noncontiguous injuries [13, 14].

Specific Injuries

Muscle Injury/Strain

The majority of low back injuries represent a muscle injury [2, 5, 6]. It can be caused by direct trauma (contusion) or when there is excessive stretch of the muscles (strain). The back pain may be significant and associated with spasm, but in general is not as severe as the pain associated with more significant pathology. The diagnosis of a muscle strain is generally made in the absence of other concerning signs or symptoms, and associated with a normal radiographic examination (i.e., diagnosis of exclusion). Specifically these patients will not have "red flags" such as radiating symptoms to the legs or neurologic findings on exam. The spine may be tender to palpation, and in general there is pain with flexion, extension, and lateral rotation [4, 15]. Further, the back pain generally is self-limited; the severity is often

greatest in the first 24–48 h and improves quickly with a period of rest [3].

Well-known principles of a brief period of rest, anti-inflammatory medications (nonsteroidal anti-inflammatory drugs, NSAIDs), and physical therapy modalities such as ice, heat, massage, and other modalities generally are used with good effect to reduce the pain and inflammation. As anti-inflammatory medicines are often used as a first-line approach to treat injuries of the thoracic and lumbar spine, athletes should be cautioned about possible gastrointestinal and renal risks [16]. A physical therapy program can be initiated once symptoms are improved enough to allow participation, which includes stretching, postural training, and core-strengthening exercises [2, 3]. With prolonged rest and/or inactivity, muscle imbalance leads to further mechanical disruption, muscle wasting, and persistent pain [15].

Most recommend return to play (RTP)/activity as the patient's symptoms have abated [2, 15, 17]. More specifically, Cooke et al. recommend that athletes may return to play once they have obtained full painless range or motion and can maintain a neutral spine position during sport-specific exercises, which is associated with return of muscle strength, endurance, and control [2, 17]. In general, recovery within approximately 2 weeks can be expected for a soft tissue injury, with 90% achieving full resolution within 2 months [4, 15].

Fractures

Presentation

Acute fractures of the lumbar and thoracic spine are rare injuries and are associated with high-velocity trauma. All patients who sustain an acute fracture need to be appropriately managed on the field and during transport by emergency services to the hospital. The use of logroll techniques on the field and backboards during transfer can minimize further injury and help prevent any neurologic demise which may be seen in highly unstable fracture patterns.

The majority of fractures present with significant pain over the involved area. Initially the patient should be turned using logroll techniques and an inspection of the area of injury should be done followed by palpation of the midline and paraspinal regions. A thorough palpation should be done of the entire spine as contiguous fractures may exist. Once inspection and palpation have been done the patient should be logrolled and removed from the backboard as prolonged time spent on the backboard may lead to pressure sores or skin breakdown. Paramount to the examination is the accurate documentation of the patient's neurologic status. This should include accurate assessment and documentation of motor strength, sensation, and reflexes including spinal reflexes such as the bulbocavernosus reflex when an associated spinal cord injury has occurred.

Frequently, the provider taking care of the athlete becomes responsible for the care of the spinal injury. However, the overall status of the patient must be thoroughly evaluated given the potential associated injuries of the chest or abdomen which may be seen with spine trauma.

Imaging

The mainstay of imaging following an athletic injury with significant tenderness to the spine is plain radiographs. Plain radiographs may accurately diagnose fractures in the face of a suspected injury. The use of CT for the evaluation of bony injuries to the spine is superior to other imaging techniques and when a fracture is suspected or diagnosed then an accurate documentation of the injury may be done via CT. CT scans are associated with a radiation exposure to the patient and the ordering physician should be specific about the area to be scanned to improve accuracy and reduce the scope of the anatomic area exposed. This can be done via a thorough examination of the back and initial review of the radiographs. The majority of spine fractures can be adequately classified via CT scans and the need for an additional imaging test such as an MRI is case dependent. MRI is a superior imaging modality when evaluating the soft tissues of the spine such as discs, ligaments, and neural elements. Any spine fracture associated with a neurologic injury

needs to be studied with an MRI to look at the neural elements and any associated compression that may be present from either disc material or bony fragments. MRI is also a very good imaging choice if there is potential concern regarding a disc separation with an endplate fracture or if there is concern about ligamentous injury.

Treatment

The treatment of spine fractures in athletes varies according to the specific injury patterns discussed below. Treatment for certain stable fractures with minimal associated pain is focused on rehabilitation, and time away from athletic activity may be as short as 6 weeks. Treatment for more significant fractures such as axial compression fractures or relatively stable burst fractures includes a period of bracing for comfort for 6 weeks followed by intensive core strengthening for 6 weeks and return to sports not before 3 months. Occasionally surgery is warranted in cases of unstable injuries or spinal fractures associated with neurologic deficit. Fortunately, these are rarely seen with sports injuries and the surgical treatment, rehabilitative recovery, and RTP guidelines are not within the scope of this textbook.

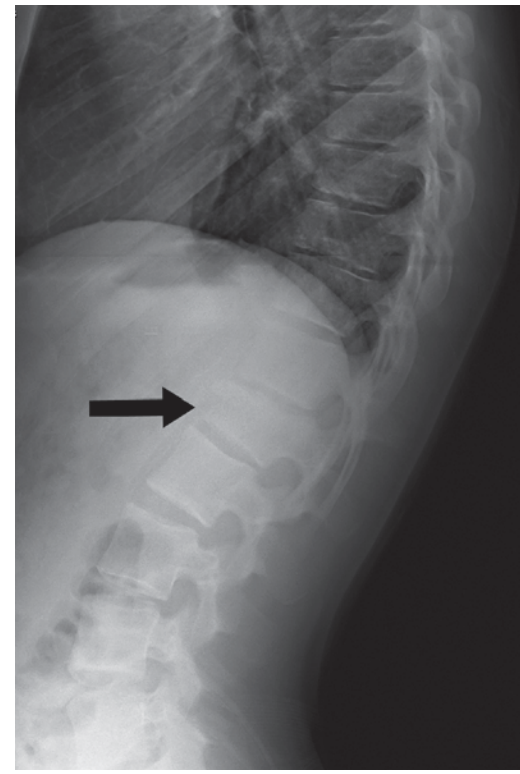

Fig. 3.1 Lateral radiograph of a patient with a stable compression fracture depicted by the *arrow*

Return to Play

There are no series in the literature with regard to RTP guidelines after thoracic and lumbar spine injuries. As a general rule, RTP guidelines would be predicated by the fact that athletes must have reached their pre-injury fitness level, must be pain free, and must have an injury pattern which does not place them at an increased risk for further injury. Athletes with stable compression fractures, burst fractures associated with no significant global alignment issues or neurologic compression, and stable spinous process/transverse process injuries may return to play safely once rehabilitated. As a general rule, fractures heal with strength consistent with the pre-injury level. There are no clear guidelines regarding RTP after operative fixation and fusion of a spine fracture. Most series or reviews in the literature are based on authors' experience and expertise. Individual guidelines are based on injury severity, extent of operative intervention, and the risks associated with the sport in question. Once again, any RTP should be prohibited until the athlete has rehabilitated to their pre-injury fitness level and has a painless spinal range of motion in the absence of any symptoms.

Compression Fractures

Compression fractures are usually caused by an axial load to the spine. They are characterized by loss of vertebral body height and usually there is anterior wedging to a variable degree of the vertebral body (Fig. 3.1). The mechanism seen in sports is due to an axial load, many times after landing or falling from a height during ski jumping or skate boarding, for instance. An isolated compression fracture is not typically associated with neurologic deficit. Additionally, it is uncommon to have concomitant internal organ injuries; however, it would not be unusual to sustain inju-

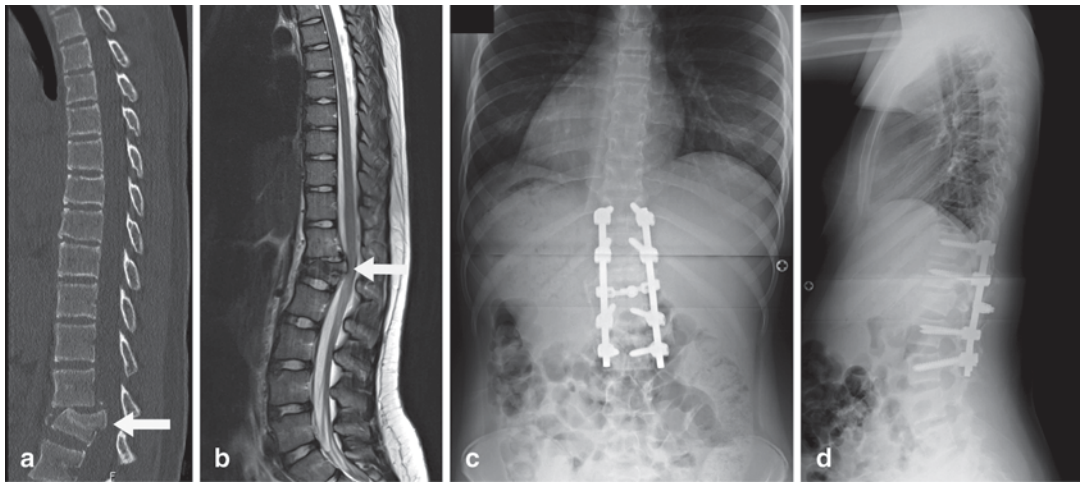

Fig. 3.2 **a** CT scan of a patient who sustained a burst fracture at L1 (*arrow*) during skiing. **b** MRI scan of the same patient depicting the fracture and neurologic compression (*arrow*). **c**, **d** Plain radiographs of the same patient after instrumented reduction and fusion for the fracture

ries to the feet or ankles (in particular, calcaneus fractures) during the fall from a height. These in general are stable spine fractures and can be treated with a spinal orthosis for 4–6 weeks followed by rehabilitation for 6 weeks. Return to sports may happen as early as 3 months. In the pre-adolescent patient, compression fractures are commonly seen and usually will involve two to three continuous levels of the spine. These are not associated with an appreciable amount of vertebral compression and need to be braced only if significantly painful. Return to sport for these patients is typically not before 6 weeks.

Burst Fractures

Burst fractures are high-velocity injuries and involve circumferential injury to the spine. There is associated bony collapse to a varying degree with retropulsion of bone fragments into the spinal canal. These can be associated with partial or complete injury to the spinal cord and/or exiting nerve roots. Burst fractures are usually seen with motor vehicle sports such as motocross but can also be seen with sports such as skiing/snowboarding or hockey. The treatment of burst fractures is related to the amount of vertebral collapse, the amount of spinal canal compromise, and the neurologic status of the patient. Well-aligned fractures with no associated neurologic injury can be treated with a thoracolumbar orthotic [18]. Fractures with partial or complete neurologic deficits are treated with neurologic decompression and instrumented fusion of the spine (Fig. 3.2).

Transverse Process/Spinous Process Fractures

These are stable fractures which cause significant pain and many times are due to a direct blow. Transverse process fractures caused by direct trauma may also be associated with renal or splenic injuries and appropriate evaluation must be done. The fractures are usually readily identified on plain radiographs and do not require further imaging if they are isolated injuries (Fig. 3.3). Treatment is symptomatic and usually focused on anti-inflammatories, occasional bracing for discomfort, and a rehabilitation program. RTP is usually at 6 weeks if the patient is back to full strength and motion.

Apophyseal Ring Fractures

Apophyseal ring injuries are due to injuries with significant velocity which cause a separation of the ring apophysis from the vertebral endplate. They commonly occur at the thoracolumbar

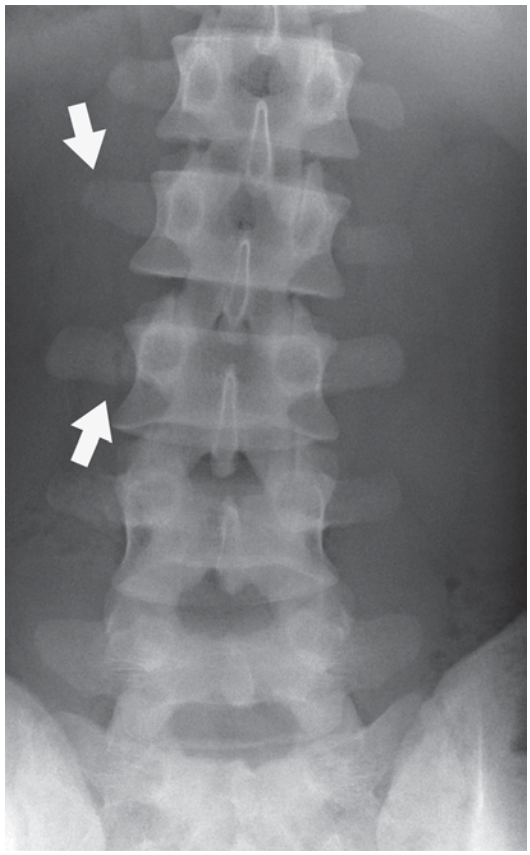

Fig. 3.3 Plain radiograph of the lumbar spine with *arrows* depicting fractures of the lumbar transverse processes

junction given the transition between the rigid thoracic spine and more mobile lumbar spine. These may present with significant pain after injury or may present with neurologic signs and symptoms if there is extrusion against the neural elements. Treatment is focused around activity modification, bracing, and rehabilitation with expectation of resolution of symptoms. Patients with neurologic signs from an endplate injury may require surgical decompression with removal of the disc and fusion of the involved segment (Fig. 3.4). Given the size and extent of the injury and endplate separation, removal of the disc with fusion is usually required rather than just a simple discectomy.

Lumbar Disc Herniation

Presentation

A disc herniation is characterized by an injury to the annular fibrosis which allows the escape of the gelatinous core (nucleus pulposus). This material may compress and/or cause chemical inflammation around the nerve roots, leading to symptoms [3, 16]. Either an acute injury or more commonly repetitive injury can weaken the annular fibers. An axial load on the disc is associated with increased pressure within the disc, and escape of the nucleus pulposus is possible through the weakened annulus [4]. Lumbar disc herniation is common among football players, particularly in offensive and defensive linemen [2, 19–21]. This is largely due to higher body mass indexes (BMIs), consistent play in the squatting and crouching positions, frequent high-velocity trauma, and intense weight training [21]. However it is encountered in other sports such as baseball, hockey, basketball, rugby, and rowing [20, 22–25].

A symptomatic lumbar disc herniation commonly presents with back pain that is worsened by lumbar flexion, sitting intolerance, and associated with shooting pain or paresthesias down the legs. Initially, back pain may be the primary symptom, as the nerve fibers associated with the annulus are irritated. Bending forward or performance of the Valsalva maneuver, such as coughing or bearing down, is likely to exacerbate the symptoms [3]. Leg symptoms predominate later, through direct compressive and inflammatory mechanisms. Leg symptoms commonly involve pain (radiculopathy), but numbness and/or weakness that correlate with the compressed nerve root also may be found. It is important to understand that neurologic deficits are less common in the pediatric and adolescent population when compared to adults [3, 26]. An important distinction with discogenic pain is the presence of symptoms that radiate beyond the knee to the lower leg; whereas, with symptoms from other etiologies, the patient may describe pain that involves thigh or gluteal region that does not extend below the

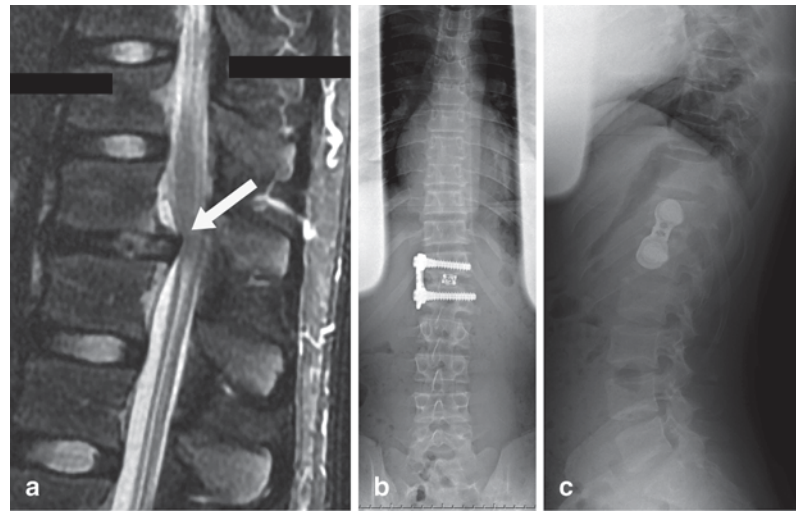

Fig. 3.4 **a** MRI depicting an endplate fracture in a 14-year-old boy who was playing football. The *arrow* depicts the acute herniation of the endplate into the spinal canal with neurologic compression. **b**, **c** Plain radiographs after the patient had undergone removal of the herniated endplate with decompression and anterior fusion with instrumentation

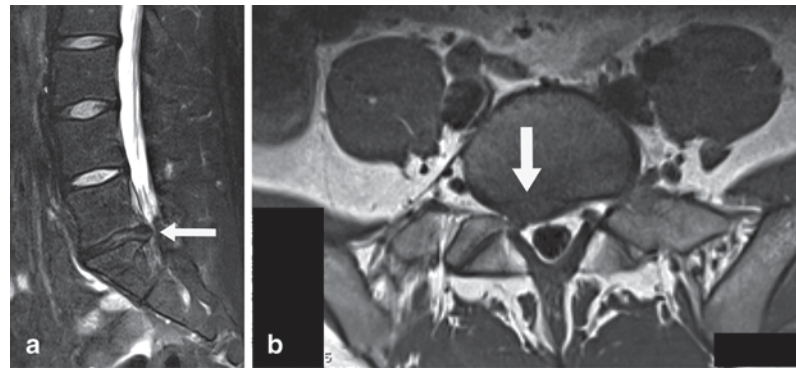

Fig. 3.5 **a** Sagittal cuts of an MRI in a patient with a herniated disc depicted by *arrow*. **b** Axial view on the MRI of the same patient showing nerve compression (*arrow*) as a result of disc herniation

knee, and which may not represent true lumbar radiculopathy.

A complete neurologic exam of the lower extremities is warranted. While L4/L5 and L5/S1 represent the majority of disc herniations [4], careful testing of all nerve roots of the lower extremities is important. With an L5/S1 herniation, the most common root affected is S1, which would present as numbness on the lateral aspect of the foot and weakness in foot eversion (peroneals) and plantar flexion (gastrocnemius/soleus). The Achilles tendon reflex (S1) will be diminished or absent. With an L4/L5 herniation, the disc most frequently affects the L5 nerve root, which manifests as weakness in great toe extension (extensor hallucis longus) and numbness over the lateral leg and dorsum of the foot. In the less common far lateral disc herniation, the L4 nerve root will be affected at the L4/L5 level and the L5 root will be affected at the L5/S1 level [4]. Special tests include straight leg testing (Lasegue's sign) which will exacerbate symptoms by pulling tension on the irritated root and reproduce radicular symptoms (sensitivity 91%, specificity 26%). For cross straight leg raising (pain with flexion of contralateral leg), the sensitivity is 29% and specificity is 88% [27]. For the test to be positive, pain should be reproduced with flexion before 70°; dorsiflexion of the foot should exacerbate symptoms and knee flexion should relieve symptoms [27]. Hip range of motion and a FABER maneuver can be performed to rule out hip/sacroiliac joint pathology masquerading as lower back pathology [28].

Imaging

In older patients with disc degeneration that precedes the disc herniation, narrowing of the disc

space may be appreciated on plain radiographs. However, in the young athlete, a disc herniation will often not be associated with degenerative changes, and therefore radiographs are likely to be normal. With a high index of suspicion (leg symptoms, pain worse with lumbar flexion, positive straight leg raise or contralateral straight leg raise, neurologic finding on exam), MRI can be used to confirm diagnosis and identify the anatomy of the disc herniation (Fig. 3.5). It is paramount that changes on MRI be correlated to physical exam findings. Cheung et al. demonstrated that 40% of individuals under 30 years of age had lumbar intervertebral disc degeneration increasing progressively to over 90% by 50–55 years of age [29]. Similarly, Jensen et al. demonstrated that 35% of asymptomatic individuals between the ages of 20 and 39 have disc degeneration evident on MRI evaluation [30].

Treatment/Outcomes

Initial treatment is often conservative, with good results in up to 90% of patients [31]. A period of rest and anti-inflammatory (NSAIDs) medication is required. In general the natural history is favorable; with disc resorption, the nerve irritation abates. Occasionally a corticosteroid dose pack is useful in reducing the acute inflammation; however no studies have demonstrated benefit over placebo [3, 16, 32, 33]. Epidural steroid injections (ESIs) may have a moderate short-term effect in the management of low back pain with radiculopathy, although studies have been inconclusive based on several systematic reviews of the literature [34]. In general ESI are used when radicular symptoms are persistent and/or nonresponsive to initial oral therapy and in general are considered better for chronic rather than acute symptoms [3, 16]. Use of lumbar corsets and braces is not supported by the literature [16, 35].

If symptoms persist despite optimal nonoperative management, operative discectomy may be indicated. Absolute surgical indications include cauda equina syndrome, progressive neurologic deficit, or a profound neurologic deficit [15]. Spinal fusion is indicated for disc herniation if there are multiple recurrences or coexisting spinal instability [15].

The Spine Patient Outcomes Research Trial (SPORT), a prospective observational cohort study of 743 patients, demonstrated significant improvement in bodily pain and physical function scales of the Short Form 36 (SF-36) and the Oswestry Disability Index (ODI) for patients treated with open discectomy when compared to those who were treated nonoperatively at 3 months, 1 year, and 2 years [36]. A subsequent randomized trial did not confirm these findings, although there was a significant amount of crossover between the two groups, and there was a nonsignificant trend towards larger improvements in the surgically treated group [37]. As these studies examined the general population, it is not clear whether this information can be applied specifically to the young athlete.

In general, several studies have demonstrated a high rate of success after surgical and nonsurgical treatment in most athletes attempting to return to sports after a lumbar disc herniation. Some studies favor operative intervention. In a study of 71 consecutive athletes with a mean age of 21 and a symptomatic disc herniation, 78.9% returned to original sport at an average of 4.7 months after start of conservative treatment and sustained activities for at least 6 months [25]. Watkins et al. reviewed RTP in 59 professional and Olympic athletes who had microscopic lumbar discectomy, with 88% returning on average 5.2 months after surgery; however, no performance-based outcomes were assessed [9]. This timeline is similar to the findings seen in other studies [19, 23].

Hsu et al. [19] examined outcomes of 137 NFL players after treatment of a lumbar disc herniation. RTP was similar in 34 nonoperative and 96 operatively treated patients, with 78% returning, which is a return to work rate that is similar to the general population [2, 19, 36, 37]. The recurrence rate of 8% in this series after discectomy is similar to that of the general population [38].

In a second study, Hsu et al. examined 342 professional athletes from the NFL, National Basketball Association (NBA), Major League Baseball (MLB), and National Hockey League (NHL) with lumbar disc herniation. Eighty-two percent of patients returned for at least one game

after treatment. There was no statistically significant difference in outcome in surgical versus nonoperative cohorts. The average career length after injury was 3.4 years. Players in MLB had the highest rate of return, while those from the NFL were least likely to return [20].

Other studies have demonstrated a high rate of return in MLB players; Earhart reported 97% of baseball players successfully returned to play at an average of 6.6 months after diagnosis [23], and Roberts et al. also demonstrated a high rate of RTP at an average of 7.3 months after diagnosis [24].

Anakwenze et al. compared RTP in 24 NBA players who had surgical discectomy to a matched control group. RTP rates were equal between the two groups (75% and 88% respectively), suggesting that those who require lumbar discectomy are able to achieve the same level of performance as NBA athletes who did not require surgery [22].

One study favors surgical treatment. Weistroffer et al. examined RTP of 66 NFL linemen after operative and nonoperative treatment. Those treated with surgery (81%) had a higher rate or RTP than those treated nonoperatively (29%). Those treated operatively averaged 33 games over a 3-year period, whereas those treated nonoperatively averaged 5.1 games over a 0.8-year period. Seven patients required revision surgery, and six out of those seven returned to play [21].

For those who had surgery, multiple-level disease has a bad prognosis. Wang et al. assessed 14 collegiate athletes who were treated for disc herniations, and 90% of athletes who underwent single-level open discectomy returned to play at the varsity level while none with two-level disease returned to sports because of ongoing symptoms [39].

Return to Play

Variable rates for RTP exist. Eck et al. allow patients to return to sport once they have sufficient pain relief and range of motion. Those who have had microdiscectomy typically return to activity at 6–8 weeks and remain out of contact sports for at least 3 months [15]. Alba conducted a survey of 523 members of North American Spine Society (NASS) to assess return to golf after lumbar spine surgery, and the majority allowed golf 4–8 weeks after microdiscectomy [40]. Cahill et al. reviewed 87 pediatric patients who had lumbar microdiscectomy (64% were athletes), and full sports participation was allowed at 8–12 weeks postoperatively [41].

Watkins et al. allow return to sport when patients complete trunk stabilization program, achieve aerobic conditioning, and can perform sport-specific stretching and strengthening exercises [42]. The average time it took operative patients to return to their sport in a follow-up study was 5.8 months. Progressive return data for surgically treated patients showed the percentage of athletes who returned increased from 50% at 3 months to 72% at 6 months to 77% at 9 months and 84% at 12 months [43].

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Acute Cervical Spine Injuries

4

Robert V. Cantu and Robert C. Cantu

Presentation

Most cervical spine injuries in athletics are relatively mild involving a muscle strain, ligament sprain, or contusion. With this type of injury an athlete typically reports localized pain and stiffness without neurologic symptoms, and symptoms typically last for days to weeks. The athlete can return to the competition when he or she is free of neck pain with and without axial compression, cervical range of motion is full, and neck strength is normal. More severe injuries involving fracture, ligamentous disruption, or spinal cord or nerve root injury can occur. With these injuries, neurologic signs and symptoms may be present. Such injuries require further evaluation and imaging before considering return to competition. Although rare, severe spinal injuries are among the most devastating in sports, often leading to long-term permanent impairment. As such, accurate on-the-field assessment of the athlete with suspected cervical injury, appropriate initial treatment and triage, and prompt definitive medical/surgical care are important.

Imaging

Imaging of the young athlete with suspected cervical spine injury traditionally began with plain radiographs including anteroposterior (AP), lateral, and open-mouth odontoid views. Interpreting cervical spine radiographs in children can be challenging due to the developing anatomy where pseudosubluxation of C2 on C3 and occasionally of C3 on C4 can occur (see Fig. 4.1). In one retrospective review of 138 pediatric trauma patients, a 22 % incidence of pseudosubluxation of C2 on C3 was found [1]. One way to differentiate pseudosubluxation from true injury is to assess the spinolaminar (as known as Swischuk's) line on the lateral c-spine x-ray. In cases of pseudosubluxation, the spinolaminar line should pass within 1 mm of the anterior cortex of the posterior arch of C2 (see Fig. 4.2). When this line passes >1.5 mm from the anterior cortex of the posterior arch of C2, acute injury is likely (see Fig. 4.3). The atlantodens interval (ADI), the distance from the anterior aspect of the dens to the posterior aspect of the anterior ring of the atlas, can show more variation in children than adults. The ADI in adults is usually ≤ 3 mm; however in children <8 years of age an ADI of 3–5 mm is

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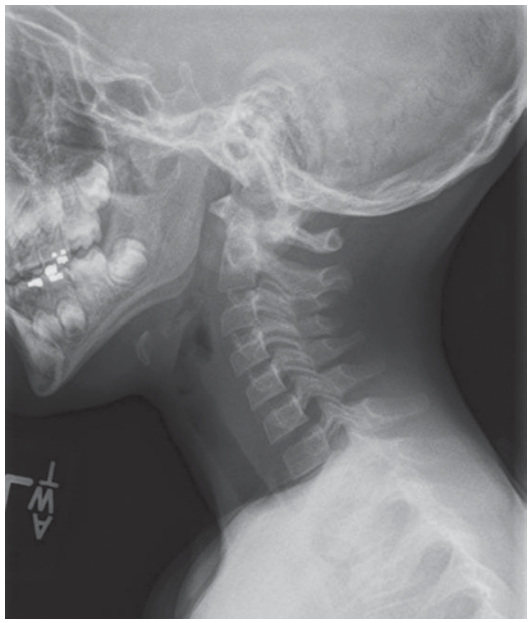

Fig. 4.1 Lateral cervical spine radiograph demonstrating mild pseudosubluxation of C2 on C3

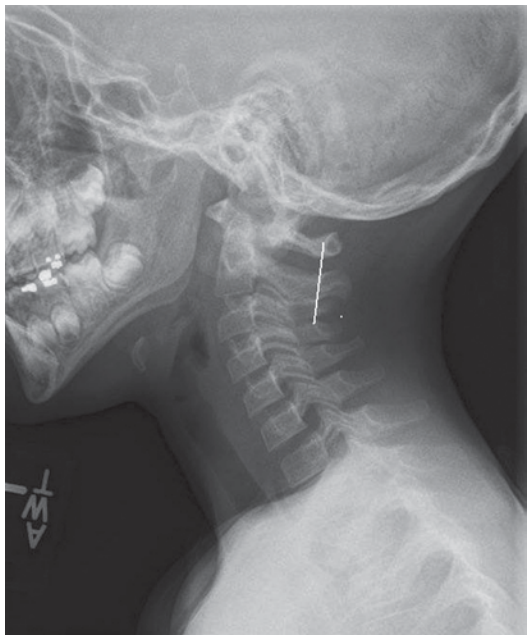

Fig. 4.2 Same x-ray as seen in Fig. 4.1 with Swischuk's (spinolaminar) line showing normal alignment

seen in about 20% of patients [2]. Understanding normal physes in children is another challenge not encountered when interpreting adult radiographs. Unfused C1 ring apophyses, apical odontoid epiphysis, and secondary centers of ossification of the spinous processes can all be mistaken for fractures. In general, normal physes are smooth structures with sclerotic subchondral lines, whereas fractures are more irregular and lack sclerotic lines. In children under the age of 8 years, anterior wedging of the vertebral bodies up to 3 mm is within normal limits (see Fig. 4.1). Wedging can be most pronounced at C3 due to hypermobility of the pediatric cervical spine with increased motion especially at C2–C3.

For adult trauma patients, computed tomography (CT) has largely replaced or is used in conjunction with conventional radiography due to its higher sensitivity for detecting injury [3]. There is concern, especially in pediatric patients, regarding the radiation dose from CT scanning. A missed cervical spine injury, however, can have life-long devastating consequences and therefore CT scan is often used when clinical suspicion for injury is high. Although multiple reports have recommended CT scan as the preferred screening tool for adult trauma patients, demonstrating quicker time to diagnosis and a shorter stay in the trauma resuscitation area compared to plain films, there remain some questions with pediatric patients [4–8]. A recent review of 1,307 pediatric trauma patients compared CT scan to plain x-rays in diagnosing cervical spine fractures [3]. The study found CT scan had a sensitivity of 100% and a specificity of 98% while x-rays had a sensitivity of 62%. The authors concluded that "CT scans should be the primary modality to image a cervical spine injury." The study also looked at flexion/extension views and the authors stated that "flexion/extension views did not add to the decision making for C-spine clearance after CT evaluation and are probably not needed" [3]. CT scan is likely most effective in older children and adolescents, where injury patterns are similar to adults. Younger children, however, are more prone to purely ligamentous or soft tissue injury, which may not be appreciated on CT scan. In these patients, magnetic resonance imaging (MRI) may be the modality of choice to identify injury. One study of MRI in 64 pediatric cervical spine patients found that MRI demonstrated

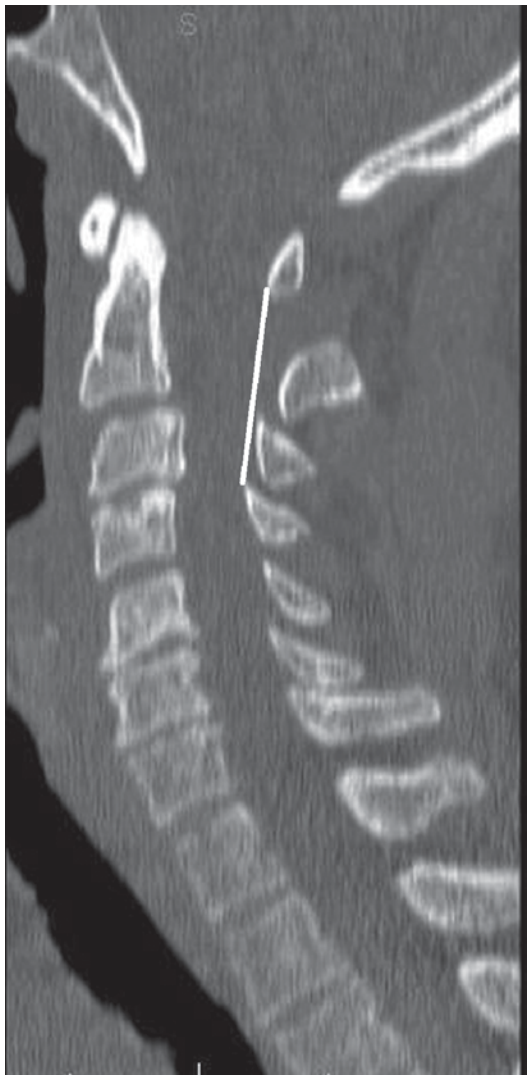

Fig. 4.3 Sagittal image of computed tomography (CT) scan showing abnormal spinolaminar line representing injury at C2–C3

injury in 24 % of patients whose x-rays were normal and allowed for spine clearance in three children whose CT scan was equivocal [9].

The question of radiation exposure from CT scan in pediatric patients merits consideration. One study prospectively examined radiation exposure in pediatric patients undergoing CT versus conventional radiographs [10]. The authors found the effective radiation dose with CT scan was 1.25 times higher than with plain x-rays. Another study comparing CT scan to x-rays found a higher radiation dose with CT for patients with a Glasgow Coma Scale >8, but for those with a GCS <8 the doses were equivalent due to the higher need for repeated radiographs [8]. Although CT scans produce more radiation than plain radiographs, there are ways to shield patients and by following these protocols, CT exposure can be reduced by 30–50 % in pediatric patients compared to adults with no loss in the quality of images [11, 12].

Fractures

Cervical spine fractures are relatively rare in athletic events, especially in younger children. Most pediatric cervical fractures result from motor vehicle accidents or falls. In children aged ≤8 years, it is very uncommon to see a subaxial fracture from athletics. The atlantoaxial complex is most at risk in younger children, with the majority of serious injuries involving a ligamentous disruption rather than fracture. Axial compression with extension can potentially cause a fracture of the ring of C1, but the forces required to do so are rarely seen in youth sports. Similarly, odontoid fractures can occur, usually from a rapid deceleration with flexion mechanism, but the forces in youth sports rarely are high enough to cause such an injury. When fractures of the odontoid occur, they tend to happen through the synchondrosis of C2 at the base of the odontoid. These fractures tend to displace anteriorly and reduction can usually be accomplished through immobilization of the cervical spine in extension. In adolescents, the cervical anatomy approaches that of adults and subaxial injuries are occasionally seen, the most common being a compression fracture. More severe burst patterns can also occur. When these compression/burst injuries happen they tend to occur between C5 and C7, a result of the increased forces on this portion of the spine as the anatomy matures.

Neurapraxias

Neurapraxias are more common than fractures in youth sports. A stinger injury is the most common neurologic injury and involves a temporary

burning sensation and/or weakness in a single upper extremity. Collision sports such as football and rugby pose the highest risk for stingers. In younger athletes, a stinger most commonly results from a forced stretching of the head and neck away from the involved limb, resulting in traction to the brachial plexus typically affecting the C5 and C6 nerve roots. In older adolescents and adults, the stinger more commonly results from a forced compression of the head and neck toward the involved limb. The shoulder may simultaneously be forced upward causing a momentary narrowing of the cervical foramen and resulting in a pinching or compression of the nerve root and transient radiculopathy. Kelly et al. have shown that some children have congenital narrowing of the cervical foramen, placing them at increased risk of sustaining a burner [13]. Athletes who have sustained a stinger typically report immediate burning and weakness in the involved extremity and report a "dead arm" sensation. Both sensory and motor function typically return to normal within seconds to minutes and as a rule full recovery should occur by 10 minutes. With repetitive injury, permanent damage can occur and rarely, with a severe cervical pinch mechanism, a nerve root(s) can be severed.

Cervical cord neurapraxias are important to differentiate from stingers. Stingers are limited to a single upper extremity whereas cervical cord neurapraxias typically present with transient quadriplegia and either loss of sensation in all four extremities or a burning or tingling sensation. Motor function usually recovers within minutes, but sensory changes can last longer. Athletes with a congenital narrowing or stenosis are thought to be at increased risk of cervical cord neurapraxias. Although the determination of cervical stenosis is an area of some controversy, general consensus holds that between C3 and C7, the AP spinal canal heights are normal above 15 mm and spinal stenosis is present below 13 mm. Resnick et al. have stated that CT and myelography are more sensitive than plain x-rays in determining spinal stenosis [14]. They note that x-rays fail to appraise the width of the spinal cord and cannot detect when stenosis results from ligamentous hypertrophy or disc protrusion. Ladd and Scranton state that the AP diameter of the spinal canal is "unimportant" if there is total impedance of the contrast medium [15]. For all these reasons, spinal stenosis requires more than just bony measurements. "Functional" spinal stenosis, defined as the loss of the cerebrospinal fluid around the cord or in more extreme cases deformation of the spinal cord, whether documented by contrast CT, myelography, or MRI, is a more accurate measure of stenosis [16]. The term functional is taken from the radiographic term "functional reserve" as applied to the protective cushion of cerebrospinal fluid (CSF) around the spinal cord in a normal spinal canal.

Cervical spinal stenosis in an athlete may be a congenital/developmental condition or may be caused by acquired degenerative changes in the spine. For an athlete with severe stenosis and no CSF around the spinal cord on MRI, it is this author's opinion that collision sports such as football should be avoided. The athlete with spinal stenosis is at risk for neurologic injury during hyperextension of the cervical spine [17]. When the neck is hyperextended, the sagittal diameter of the spinal canal is further compromised by as much as 30 % by infolding of the interlaminar ligaments. Matsuura et al. studied 42 athletes who sustained spinal cord injury and compared them to 100 controls [18]. They found that "the sagittal diameter of the spinal canals of the control group were significantly larger than those of the spinal cord injured group." Eismont et al. have stated that "the sagittal diameter of the spinal canal in some individuals may be inherently smaller than normal, and … this reduced size may be a predisposing risk factor for spinal cord injury" [17]. The idea that spinal stenosis predisposes to spinal cord injury is not new, with multiple authors as far back as the 1950s reaching the same conclusion including Wolfe et al. [19], Penning [20], Alexander et al. [21], Mayfield [22], Nugent [23], and Ladd and Scranton [15] who stated that "patients who have stenosis of the cervical spine should be advised to discontinue participation in contact sports." More recent support for this stand comes from the National Center for Catastrophic Sport Injury Research, where cases of quadriplegia have been seen in athletes with cervical

stenosis but without fracture or dislocation. In athletes with a normal-size canal, quadriplegia has not been seen without fracture/dislocation of the spine. In addition, most importantly, full neurologic recovery has been observed in 21% of athletes who were rendered initially quadriplegic after fracture/dislocation with normal size cervical canals, while complete neurologic recovery has not been seen in any athlete after fracture/dislocation and quadriplegia when spinal stenosis was documented by MRI.

Instabilities

Children have a relatively larger head size to torso ratio compared to adults, putting increased stresses on the upper cervical spine. Cervical instability due to ligamentous disruption may prove challenging to diagnose immediately after injury in a youth or adolescent athlete. As previously mentioned, some degree of laxity can be normal in children and muscle spasm following injury may prevent initial subluxation of the cervical spine. Atlantooccipital dislocation is a serious injury in children, with a mortality rate of approximately 50% [24]. Fortunately, this injury is quite rare in sports and typically results from distraction forces seen in high-speed motor vehicle collisions (see Fig. 4.4). The atlantooccipital joint is less stable than the lower cervical joints, with the alar ligaments, joint capsule, and the tectorial membrane serving as the primary stabilizers. The basion-dental interval (BDI) or distance from the basion to the tip of the odontoid as seen on a lateral radiograph can be used to assess an atlantooccipital dislocation (see Fig. 4.5). A BDI >12.5 mm is indicative of injury, although this measurement is not as reliable in children <5 years [25, 26]. Atlantoaxial injury can occur as the C1–C2 articulation is also relatively less stable than the lower cervical joints. The transverse ligament runs posterior to the odontoid and limits its anterior translation of C1. The apical and two alar ligaments serve to limit rotation around the odontoid. The ADI or distance from the anterior aspect of the odontoid to the posterior cortex of the C1 anterior ring should measure <5 mm in

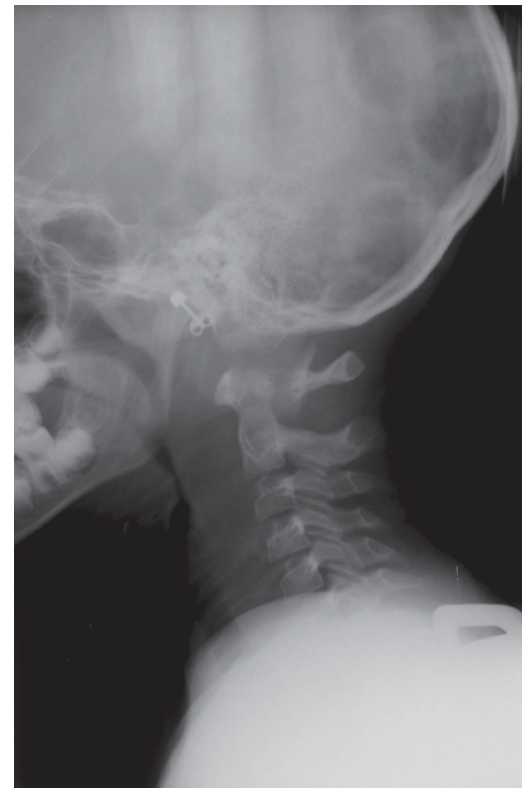

Fig. 4.4 Lateral c-spine x-ray showing atlantooccipital dislocation

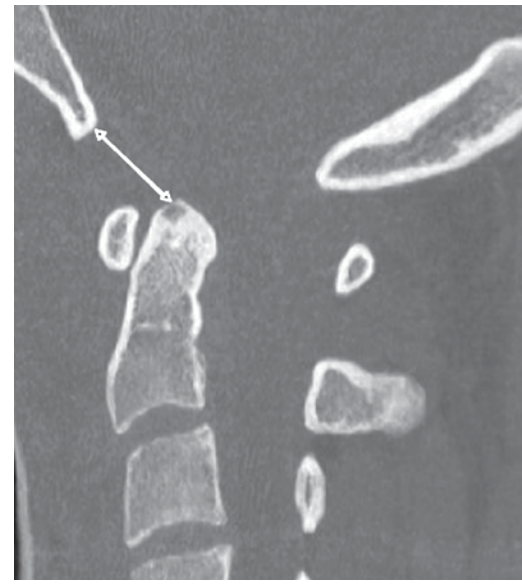

Fig. 4.5 Representation of abnormal basion-dens interval as seen on computed tomography (CT) scan

children less than 8 years of age and <3 mm in older children and adolescents [27]. With injury to the transverse ligament the ADI will increase, causing a decrease in the space available for the cord, but provided the apical and alar ligaments are intact, translation will usually be limited and spinal cord compression is rare.

Atlantoaxial rotatory subluxation involves a rotational deformity of C1 on C2. This condition can be seen with trauma or secondary to infection such as Grisel's syndrome. A young athlete with atlantoaxial subluxation will present with the neck flexed to one side and rotated toward the other. The odontoid view x-ray will show asymmetry of the lateral masses; the lateral mass that is more anterior will appear wider and closer to midline. CT scan usually provides the most complete view of the injury, including the degree of facet subluxation.

An athlete with Down syndrome merits special consideration. Individuals with Down syndrome have increased mobility at the occipitocervical and atlantoaxial articulations. Whether to perform radiographic screening of the child or adolescent athlete with Down syndrome is a matter of debate. Many of the Special Olympic organizations require lateral flexion and extension radiographs for athletes in high-risk sports such as diving, equestrian, and soccer [28]. Athletes with normal radiographs may participate without restrictions, but those with an increased ADI should avoid high-risk sports. For athletes in low-risk sports with normal neck and neurologic exam, radiographic screening is generally not recommended. As Herman has stated, "for many of these special athletes, the value of participation in safe and well-supervised sports and recreational programs outweighs the potential risks of injury related to cervical hypermobility" [28].

Treatment

On-the-field management of an athlete suspected of having a cervical spine injury begins with the airway, breathing, and circulation (ABCs) of acute trauma care. If the patient is prone and there is concern for the airway, the athlete should be carefully logrolled into the supine position with one person in charge of maintaining cervical alignment. In sports such as football or hockey, where helmets and shoulder pads are worn, they should remain in place during evaluation. This occurs, provided the facemask can be quickly removed to allow access to the airway. A study by Swenson et al. on ten healthy individuals showed that if the helmet is removed and the shoulder pads remain in place, an increase in cervical lordosis results [29]. Although young children have an increased head-to-torso size, removing just the helmet still results in an increased lordosis [30]. For children ≤6 years of age, a backboard with a cutout for the helmet is recommended to maintain neutral alignment. If the helmet has to be removed then the shoulder pads should also be taken off, following the generally accepted "all or none" policy. A recent study has shown that some of the newer football helmets, with increased protection around the mandible, can make basic airway maneuvers such as chin lift more difficult [31] (see Fig. 4.6). Participants attempting to perform bag mask ventilation on 146 college athlete volunteers reported the helmet as a cause of difficulty in 10.4% of athletes wearing a modern hockey helmet, and in 79% of athletes wearing a football helmet [31].

Fig. 4.6 Example of football helmet with increased protection around the base of jaw, making airway maneuvers such as chin lift more difficult

Definitive treatment of cervical injuries depends on the type and level of involvement. Athletes who have sustained a stinger can generally return to competition when all motor and sensory

symptoms have cleared and they have full painless neck range of motion. In rare cases, the motor and sensory symptoms of a stinger last more than a few minutes. In these cases, MRI of the spine should be considered to look for a herniated disc or other compressive pathology. If symptoms persist more than 2 weeks, then electromyography (EMG) can allow for an accurate assessment of the degree and extent of injury. Transient quadriplegia or any bilateral motor or sensory symptoms after injury necessitate removal of the athlete from competition and further diagnostic evaluation. CT scanning can identify subtle fractures or malalignment, but may not show ongoing extrinsic cord compression or intrinsic cord abnormalities; MRI is the most sensitive study to evaluate these conditions. Somatosensory evoked potentials may prove useful in documenting physiological cord dysfunction. Definitive treatment depends on the pathology identified.

Treatment of cervical spine fractures also depends on the type and level of injury. Some bony injuries, such as spinous process fractures or unilateral laminar fractures, may require no treatment or only immobilization in a cervical collar. Others, such as the bilateral pars interarticularis fracture of C2 ("hangman's fracture"), may be treated with a cervical collar or halo vest immobilization. Unstable injuries such as fracture dislocations should initially be reduced and temporarily stabilized with cervical traction using Gardner-Wells tongs or a halo ring device. Surgical treatment may subsequently be required for severely comminuted vertebral body fractures, unstable posterior element fractures, type 2 odontoid fractures, incomplete spinal cord injuries with canal or cord compromise, and in those patients with progression of their neurologic deficit [32].

Treatment of the spinal cord-injured patient depends on the underlying injury. Injury to the spinal cord involves an initial mechanical disruption of axons, blood vessels, and cell membranes which is then followed by a secondary injury involving further swelling and inflammation, ischemia, free-radical production, and cell death. Only prevention can limit the initial injury and treatment is focused on preventing secondary damage. In a review of 57 rugby players who sustained an acute spinal cord injury, most commonly due to facet dislocations, five out of eight who underwent reduction of the injury within 4 h had compete neurologic recovery, whereas 0 out of 24 who underwent reduction beyond 4 h had complete recovery [33].

Return to Play

The return to play decision depends largely on the type and extent of injury. An athlete with a cervical ligament sprain or muscle strain/contusion, with no neurologic or osseous injury, can return to competition when he or she is free of neck pain with and without axial compression, has full range of motion, and neck strength is normal. Cervical radiographs should show no subluxation or abnormal curvature. It is preferable that the athlete is asymptomatic and can perform at his preinjury ability prior to returning to competition.

The athlete who has sustained a stinger-type injury should be held out until motor and sensory symptoms have resolved and there is full and painless cervical range of motion. If residual symptoms are present or if there is concern for neck injury, return to play should be deferred. Athletes with brachial plexus injuries may be considered healed and safe for return to play when their neurologic examination returns to normal and they are symptom free. An athlete with a permanent neurologic injury should be prohibited from further competition.

An athlete who has sustained transient motor or sensory symptoms (neuropraxia), bilaterally or in an arm and leg, must have a cervical spine MRI to rule out a spinal cord injury or a condition that puts the spinal cord at risk. If the cervical MRI is normal, the athlete can return to competition when free of neurological symptoms, free of neck pain with and without axial compression, has full range of motion with normal neck strength, and the neurologic exam is normal. Even with complete resolution of symptoms and a normal exam, having had such an event would be considered by some a relative risk for return

to play. Having had three such events should be considered an absolute contraindication to return.

For an athlete who has sustained a cervical spine fracture, return to play is deferred at least until the fracture has healed. In general, stable fractures managed nonoperatively, such as those involving a spinous process or a unilateral lamina, that have healed completely will allow the player to return to competition by the next season. Athletes with a healed fracture who required halo vest or surgical stabilization as part of the treatment are considered to have insufficient spinal strength to safely return to contact sports, unless formal testing demonstrates it has returned to normal. Even after the fracture has healed and strength has returned, the altered biomechanics in surrounding spinal segments may produce an increased risk of further sports-related injury. If there is a one-level anterior or posterior fusion for a fracture, athletes are usually allowed to go back when neck pain is gone, the range of motion is complete, muscle strength of the neck is normal, and the fusion is solid. When there are multilevel fusions or a fusion involving C1–C2 or C2–C3, return to contact or collision sports is contraindicated. The athlete could return to a noncontact sport with a low risk of neck injury, such as golf or tennis.

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Concussion in Youth Sports

5

Cynthia J. Stein and William P. Meehan III

Introduction

Given the mechanism of many sport-related spine injuries, athletes who sustain these injuries are also at risk of concomitant concussions. Concussion, a type of traumatic brain injury, most often results from trauma to the head. However, it may also follow injury to the spine or trunk without direct impact to the head, especially if the injury involves a rapid rotation of the head or a whiplash-type movement of the neck. Concussion itself may be an unrecognized problem, a short-lived impairment, or a devastating injury. Clinicians who treat athletes with spine injuries should also be comfortable with diagnosis and management of sport-related concussion.

Definition

Concussion is a type of traumatic brain injury. It is defined as a complex pathophysiological process affecting the brain, induced by traumatic biomechanical forces. It results from a rapid rotational acceleration of the brain caused by impact to the head or by the transmission of force to the head without direct contact [1].

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Concussion is a disturbance of normal brain function, as opposed to a structural injury. It has been suggested that this disturbance is due to intra- and extracellular changes, including shifts in cellular ions, neuronal depolarization, axonal injury, alterations in glucose metabolism, and changes in cerebral blood flow patterns. [2] The resulting mismatch between energy demand and the available energy supply may leave cells vulnerable to additional injury [3, 4].

Although prior teaching often focused on loss of consciousness (LOC), LOC is an uncommon occurrence in sport-related concussion, occurring in less than 10% of cases [5–7]. Headaches, dizziness, and many other symptoms of concussion are much more common. In general, the symptoms of concussion are temporary and resolve spontaneously, usually in a matter of days or weeks. For some, however, the effects of concussion are prolonged.

Epidemiology

Estimates suggest that as many as 3.8 million traumatic brain injuries occur during sports each year in the United States [8]; the vast majority are concussions. Twenty-five to 30% of all mild traumatic brain injuries seen in the emergency department in 5–14-year olds are bicycle-related or sport-related injuries [9, 10], and concussion accounts for almost 15% of all sport-related injuries in high school athletes [11].

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The incidence of concussion tends to be highest in collision and contact sports [8, 12]. One study of 12 high school sports found the overall rate of concussion to be 17 per 100,000 athlete exposures (AEs), with the highest rate in football (33/100,000 AEs) [7]. Another study, which evaluated concussion rates in 20 high school sports, found the highest incidence rates in football, boys' ice hockey, and boys' lacrosse [13]. As expected, the total number of concussions occurring per year in a given sport is affected by the number of athletes participating. Popular sports that have high participation rates, such as football, girls' soccer, boys' wrestling, and girls' basketball [13], also have large numbers of athletes sustaining concussions. The incidence of concussion is high in boys' ice hockey, and several studies have shown that concussion accounts for a larger proportion of injuries in ice hockey than in other sports [13, 14]. However, fewer athletes participate in ice hockey when compared to sports like soccer and basketball; therefore, it accounts for a lower total number of sport-related concussions nationally. Comparing sports played by the same rules for both male and female athletes, the risk of concussion appears to be higher in females [13].

Concussion rates are generally higher in competition than in practice, except in cheerleading where the reverse is true [7]. Player–player contact is the most common mechanism of injury, which accounts for 70% of concussions, followed by player–surface contact, which accounts for 17% [13].

Second Impact Syndrome

In rare cases, additional injury following concussion can lead to second impact syndrome and death [15]. First described in 1984 [16], second impact syndrome occurs when an athlete, who has not completely recovered from a concussion, suffers another blow, often minor, which causes a loss of cerebrovascular autoregulation, followed by cerebral edema, cerebral herniation, and death [16–18]. Although some authors argue that the first concussion is not necessary, and that only a single blow to the head is required for such a catastrophic outcome [19, 20], animal models have shown a window of increased vulnerability during which the brain is more susceptible to additional injury [21, 22]. Therefore, to prevent additional injury, it is critical that athletes should not be returned to their sports until they have fully recovered from concussions.

Risk Factors

A history of prior concussions, participation in contact sports, and comparatively low body mass index may increase the risk of concussion [7]. Athletes who have suffered a prior concussion are at increased risk of sustaining subsequent concussions [6, 7]. In one study, football players with a history of three or more prior concussions were three times more likely to have an in-study concussion than players without previous concussions [23]. The reasons for this increased risk are unclear. There may be something inherent in individual athletes that predisposes them to injury. Certain playing styles may increase the risk. It may simply be a matter of playing time; athletes who play more are at greater risk than those athletes on the bench, or there may be a change that occurs to the brain at the time of the initial insult that places the athlete at risk for additional injury [24, 25].

In some sports, for which the rules are the same for male and female athletes, females appear to be at higher risk for concussion [12, 13, 26]. Again, the reasons for this are unclear. Lower head and neck mass, decreased neck strength, smaller head-to-ball size ratio, and increased honesty in reporting concussions symptoms have all been proposed as possible explanations for the discrepancy in concussion incidence between male and female athletes [26].

Symptoms

Some of the most common symptoms of concussion are listed in Table 5.1 [27]. Concussion symptoms vary by individual and by injury.

Table 5.1 Symptoms of concussion

Headache
Dizziness
Nausea or vomiting
Difficulty balancing
Sleep disturbance
Vision changes
Photophobia
Phonophobia
Feeling "out of it" or "dazed"
Difficulty concentrating
Tinnitus
Drowsiness
Sadness

These symptoms are also not specific to concussion. Frequently reported symptoms, such as headache, dizziness, nausea, fatigue, confusion, difficulty with concentration and memory, sensitivity to light or noise, and emotional changes can result from other injuries, illnesses, and/or medications; however, they have been noted more commonly after concussion than after other types of injuries [28].

The most common concussion symptom, both acutely and chronically, is headache [23, 27]. The pathophysiology of many posttraumatic headaches remains unclear [29], but most appear to be migraine or tension-type headaches [30].

Some authors recommend grouping concussions into symptom clusters: neuropsychiatric (depression, irritability, anxiety), somatic (headache, dizziness, light or noise sensitivity), cognitive (difficulty with concentration or memory, feeling of fogginess), and sleep disturbance (hypersomnia, insomnia, interrupted sleep) [31]. This allows treatments to be tailored to the individual athlete's specific needs, addressing the cluster from which the athlete experiences the most troublesome symptoms.

Diagnosis

Since there is no overt structural damage, concussion may be overlooked, especially in the setting of multiple traumatic injuries. Although dramatic when present, most sport-related concussions do not involve LOC or convulsion [5–7, 27, 32–34]. Therefore, athletes, coaches, parents, athletic trainers, and team physicians must maintain a high index of suspicion for sport-related concussions. In order to assist in diagnosing a concussion, sideline tools have been developed, such as the Sport Concussion Assessment Tool version 2 (SCAT2), which is free and available online and as an appendix to the consensus statement from the Third International Conference on Concussion in Sport [1].

Concussion is diagnosed by medical history and physical examination, aided by various instruments developed specifically for the assessment of sport-related concussions, such as symptom inventories, balance evaluations, and neurocognitive assessments. One of the most common symptom inventories was developed by the International Conference on Concussion in Sport and is available as part of the SCAT2. Similarly, a modified version of a common balance assessment, the balance error scoring system (BESS) [35], and some of the more common sideline cognitive assessments are also available as part of the SCAT2. Given the substantial variability of balance and cognitive performance among athletes, these assessments are best used when compared to a baseline measure taken at the start of the season, before sport-related injuries occur.

In addition, neuropsychological testing and computerized assessments of neurocognitive function are becoming more widely used to diagnose and monitor recovery from sport-related concussions [12, 27, 36]. These assessments provide standardized and objective measures, which can be analyzed and followed over time, and, thus, represent an important part of concussion evaluation. Scores, however, can be affected by a variety of factors, and as with balance and sideline assessments of cognition, there is substantial variability between individuals. Therefore, preseason baseline measurements are ideal; proper interpretation of the results is essential.

Athletes are known to play through pain [37], and they often experience pressure to return to play, both external pressure from coaches, teammates, and parents, and internal pressure from their own self-image and values. Therefore, there is often significant motivation to down-

play or deny any ongoing symptoms after injury. Even after an athlete feels truly asymptomatic, neuropsychological testing may still show deficits [38–40] that suggest incomplete recovery. While some professional athletes may return to full sports participation rapidly without negative consequences, more conservative management is called for with younger athletes, especially those under the age of 18 [1].

Since concussion is a functional problem, without a clear structural injury, current imaging techniques typically cannot detect concussive brain injury. Although recent studies suggest newer imaging techniques show some promise in detecting concussion [41], their use at this point remains investigative. Therefore, routine imaging is not recommended. However, imaging is useful for ruling out other potential etiologies of an athlete's signs and symptoms, and should be used for that purpose when indicated.

Treatment Guidelines

In an effort to advance understanding and treatment of sport-related concussion, multiple guidelines have been developed. The most recent consensus statement from the Third International Conference on Concussion in Sport in Zurich (2008) [1] replaced the earlier recommendations from those in Vienna (2001) [42] and Prague (2004) [43]. Although useful during their time, earlier grading systems (I/II/III) and classifications of concussion (simple/complex) have been superseded by a more individualized approach to concussion management, based on ongoing evaluation of symptoms, balance, and cognitive function, followed by a graduated return-to-play protocol.

Athletes suspected of sustaining a concussion should be immediately removed from play and evaluated. During recovery, they should remain out of all activities that place them at risk of direct or indirect injury to the head. The mainstays of concussion management are physical and cognitive rest. Because physical rest is a key element in treatment of concussion [44], athletes should initially avoid all strenuous aerobic activities and resistance training. In addition, athletes should decrease strenuous cognitive activities, which involve concentration, reasoning, and memory tasks. These activities include reading, schoolwork, video games, text messaging, working online, and playing games which require concentration, such as crossword puzzles and chess. These activity limitations often require home and school accommodations. For scholar athletes, school attendance and academic workloads may need to be adjusted [45]. The need for physical and cognitive rest (including the degree and duration of this rest) varies among athletes and should be determined after full evaluation of all contributing factors such as age, duration and intensity of symptoms, and scholastic/occupational demands.

Once athletes are symptom-free at rest, they are gradually returned to both athletic and cognitive activity as tolerated by their symptoms. The consensus statement from the Third International Conference on Concussion in Sport in Zurich recommends a graded return to physical activity, in a stepwise fashion. If athletes develop symptoms as they progress through these return-to-play stages, they drop down to the previous level at which they were symptom free before attempting to progress again [1].

The majority (85–90 %) of athletes sustaining sport-related concussions recover within the first few weeks after injury [12, 23, 27], with over 90 % of high school athletes recovering within a month [12, 27]. Thus, physical and cognitive rest are often the only treatments required. For those unfortunate athletes who suffer from prolonged symptoms which negatively impact their quality of life, other therapies may be appropriate [46]. In particular, insomnia, posttraumatic headaches, cognitive dysfunction, vestibular problems, and emotional difficulties may be treated to some degree with medications and/or physical therapy. Many of these medications are experimental, or their use in concussion is considered "off label" and not necessarily approved by the Food and Drug Administration (FDA). Therefore, they are best considered by clinicians experienced in the assessment and management of sport-related concussion or traumatic brain injury in general.

Recovery Time

As noted above, most athletes will recover quickly, within days to weeks, from their sport-related concussions [12, 23, 27]. Certain athletes, however, may be predisposed to longer recoveries. For example, female athletes report more symptoms and show worse performance on neuropsychological testing following concussion than do male athletes [47]. Those athletes with a history of previous concussions may take longer to recover than athletes with no history of prior concussion [23]. In addition, studies conducted in younger athletes show longer mean recovery times than those conducted in older athletes [40, 47].

Although frightening when it occurs, brief LOC is not a reliable predictor of a prolonged recovery from concussion [48]. However, LOC, especially prolonged LOC (>30 seconds), may be a sign of structural brain injuries, such as intracranial hematomas [49]. Therefore, all athletes with a LOC should be evaluated immediately by a medical professional.

Many other factors may be associated with a longer recovery, including previous concussions [7, 23, 50], amnesia at the time of injury [23, 39, 51], and decreased computerized neurocognitive test scores after injury [52, 53]. At present, however, there is no reliable way of predicting which athletes will suffer prolonged recoveries from concussion.

Because duration of symptoms, and thus time away from sports, schoolwork, and other activities can be highly variable, athletes and their families should be educated on the natural course of concussion. Individual athletes may need help managing expectations and dealing with a potentially frustrating and prolonged process of recovery.

Stages of Return

Once athletes becomes completely asymptomatic at rest, they may start to advance their physical activities. Six stages have been described [1]:

1. No activity
2. Light aerobic exercises
3. Sport-specific exercises
4. Noncontact training drills
5. Full contact practice
6. Return to play

The objective of the stepwise return is to gradually increase the athlete's activity level and monitor for any return of symptoms. As described in the guidelines, each stage should take at least 24 hours. However, each stage may take longer, especially with younger athletes and those who have had prolonged symptoms. With any return of postconcussion symptoms, the athlete should drop back to the last asymptomatic level, rest for a minimum of 24 hours, and reattempt an advance to the next stage.

Cumulative Effects

Once thought to be a benign and self-limited injury, scientific and medical research has revealed long-term effects from sustaining multiple concussions [23, 49, 54–57]. In addition to an increased risk of additional concussions and slower recovery [23], some of the long-term effects include: decreased information-processing speed [49], cognitive impairment, and memory difficulties [54]. Athletes, particularly those participating in contact and collision sports, should be made aware of the risks and counseled each time they sustain a sport-related concussion. Particularly after multiple concussion or prolonged recovery, decisions about return to high-risk sports should be made with guidance from those experienced in concussion management.

Prevention

While helmets and mouth guards are vital in the prevention of certain types of injury, and should be recommended to athletes where appropriate, they have not been shown to consistently reduce the risk of concussion. Other products have also been developed and advertised to prevent concussion, but to date none have proven effective in

reducing or preventing the rotational force to the brain that leads to concussion [58].

Some studies have suggested that strengthening of the cervical musculature can reduce the risk of concussion. In addition, concussion more commonly occurs when an athlete does not anticipate a blow to the body [59, 60]. Thus, a constant awareness of what is happening around them may reduce athletes' risk of concussion.

Future Research

Additional research is needed in the areas of prevention and treatment of concussion. Many questions need to be addressed: Are there methods to better identify individuals at increased risk of concussion or prolonged recovery through personal history, family history, or genetic markers? Can new or existing protective equipment be used to decrease the risk of concussion? Does muscular training have a role in prevention of concussion? Can rule changes effectively reduce the number of concussions? Are there sideline tools that can be used to predict the severity of concussion? Which medications and rehabilitation strategies reduce the severity and/or duration of symptoms? Are there ways to prevent or mitigate the long-term effects of multiple concussions?

Conclusion

Concussion is a traumatic injury to the brain that affects millions of athletes each year. Concussion is a functional impairment, which, for most, is self-limited and of short duration. However, the symptoms can be severe, and many athletes suffer through a prolonged process of recovery. Our understanding of concussion and treatment options continues to expand, and additional research and education is needed to optimize prevention, diagnosis, and management strategies.

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Adolescent Overuse Spine Injuries

6

Michael O'Brien and Pierre d'Hemecourt

Introduction

Until relatively recently, back pain was considered rare in pediatrics. Certainly, this is true until approximately the age of 6–9. However, between the ages of 10 and 18, the prevalence of back pain in young people is similar to that seen in adults [1]. At least 15 % of pediatric and adolescent athletes complain of back pain [2]. Furthermore, the prevalence of back pain varies with sports participation. Back pain in football has been reported at 50 %, while gymnasts have reported back pain as frequently as 86 % [3, 4]. This increase in back pain is related to the increased overall duration of sports participation and training as well as an increased sports specialization [5], which exposes the young athlete to the repetitive motions of a single sport that are often experienced year round.

Back pain may be secondary to acute trauma or overuse microtrauma, as well as inflammation, spinal deformity, infection, and tumors. It is important to understand the red flags that should initiate a workup for nonmechanical causes of pain. These would include night pain, neurologic deficits, persistent morning stiffness, age less than 8 years, fevers, night sweats, use of immunosuppressants, and a history of cancer. This chapter focuses on overuse injuries, while other causes will be discussed elsewhere in this text. Spinal overuse injuries may be divided into anterior and posterior element injuries. Anterior injuries involve the disc and adjacent vertebrae and end plates. Posterior elements refer to the posterior arch including facet joints, pars interarticularis, and spinous processes.

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Risk Factors

Risk factors for spinal overuse injuries in sports include the adolescent growth spurt, duration of sports participation, anthropomorphic factors, gender, and the biomechanics of the individual sports.

Growth Spurts The adolescent growth period involves several factors affecting the back. First, during this growth period, lumbar lordosis increases naturally and is further increased with intensifying hours of sports participation [6]. This increased lordosis combined with extension-based sports increases posterior element stress. Second, linear growth precedes bone mineralization during the adolescent growth spurt, which increases susceptibility to fracture [7]. Finally, growth cartilage is more vulnerable to injury than the bone or ligament structures, especially at the cartilaginous vertebral end plate and ring apophysis [8].

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Anthropomorphic Factors Anthropomorphic factors include muscular weakness and inflexibilities. Nader demonstrated that weakness of the gluteal musculature was a risk factor for back pain in the collegiate athlete [9, 10]. Tight hip flexors and weakened lower abdominal muscles are associated with lordosis, which has been seen as a risk factor for adolescent back pain [11]. Additionally, lumbar extensor weakness is also associated with adolescent back pain [12].

Gender Gender differences were previously believed to be a factor in spondylolysis, but this is no longer thought to be the case [13]. However, spondylolisthesis is more prevalent in the female athlete [14, 15]. Stress fractures such as sacral stress fractures are more common in the female athlete. Some of this is due to a caloric imbalance with disordered eating which contributes to decreased bone density, a condition which is more common in the female athlete. Finally, scoliosis prevalence is higher in the overall female population. However, scoliosis is not usually associated with back pain. Additionally, some increased spinal asymmetry has been noted in certain unbalanced spinal loading sports such as rhythmic gymnastics [16]. The asymmetry attributable to sports is usually minor.

Sports that emphasize extension and rotation are typically associated with posterior element stress. Athletic pursuits that emphasize spinal-loaded flexion may be more of a concern for anterior disc involvement. Other sports-specific risk factors will be discussed in other chapters.

Anterior Element Injury

Between each vertebra is an intervertebral disc. It has a central core called the nucleus pulposus (NP). The NP is encased by the annulus fibrosus (AF), which consists of layers of collagen arranged in concentric sheets called lamellae. The lamellae of the AF are arranged in a crisscross pattern that allows the disc to resist forces in axial and rotational planes [17]. The NP is comprised of proteoglycan and water. The water content of the NP in youth is about 70%, and it decreases with age. As this desiccation occurs, disc height is lost and the disc can become more prone to tearing or herniation [18]. Cranial and caudad to the disc are the vertebral end plates of each adjacent vertebral bodies.

The discs are largely avascular and disc nutrition is achieved largely from diffusion from the end plates. One theory about the beginning of degenerative disc breakdown is that it is triggered by diminished blood supply in this area beginning in the second decade of life [19].

The region of the vertebral end plate is innervated by divisions of the gray rami of the sympathetic and sinuvertebral nerves [20]. These nerves travel with blood vessels and have been noted in all anatomical locations within the vertebra except in zones deeper than the outer 1/3 of the annulus or in the NP [21]. Interestingly, degenerative discs and adjacent end plates have more extensive innervation with nociceptive properties when compared to asymptomatic discs [22, 23]. In addition, levels of inflammatory mediators are elevated in degenerative and herniated discs as compared to their healthy counterparts [24, 25].

Disc Degeneration

Disc degeneration is likely a multifactorial process with several risk factors including family history, body habitus (elevated body mass index, BMI), and type of activity or sport. For instance, it likely begins with repetitive microtrauma from shearing forces causing small, circumferential tears in the AF. These tears can coalesce into larger radial tears, which may or may not lead to herniation. This process, in addition to progressive disc desiccation, disrupts the disc [26]. Disc height is lost and the disc's connection with the adjacent vertebral end plates is compromised. This segmental dysfunction results in instability and subsequently leads to lateral recess and foraminal narrowing with nerve root impingement. It also causes local muscular weakness and instability of the posterior elements, promoting facet degenerative changes over time. Ultimately, the mechanical changes and progressive instability affect the levels above and below the original

segmental degeneration, resulting in multilevel degenerative changes and stenosis. The eventual formation of scar tissue, osteophytes, and joint surface irregularities results in loss of motion, which theoretically could allow restabilization, and often a decrease in pain [24, 27, 28].

Herniated Disc

Disc herniations result when axial loads are sufficient to force NP material past the AF. The disc protrusion or herniation may result in NP material that mechanically compresses an adjacent nerve root, though direct compression may not be necessary for significant back pain or radiculopathy [28]. Disc material has been implicated as a causative agent for chemically induced low back pain (LBP) due to the irritative nature of the NP when it comes in contact with structures other than the AF [29].

Clinically significant herniated nucleus pulposus (HNP) is most common in the general population in patients aged 30–55 years, occurring in approximately 2% of the general population. It is also common in elite athletes aged 20–35 years [30]. It rarely occurs in young children, with a reported incidence of approximately 0.9% [31]. In young athletes with back pain, one study demonstrated disc involvement in 10% of cases [32]. Gymnasts, weightlifters, American football players, and rowers may present a higher risk [33, 34], presumably from repetitive axial loading or lumbar stress in a flexed position. Classic pain from a herniated disc may present with LBP and possibly radicular symptoms. Pain is worse with flexion or with coughing and the Valsalva maneuver. Radicular pain can be in the sciatic distribution.

On physical examination, the combination of weakness, sensory loss, and diminished or absent reflexes may indicate nerve root impingement [35]. Special tests are also helpful to demonstrate dural tension, such as straight leg raise (SLR), crossed SLR, the slump test, and ankle dorsiflexion with SLR (Braggard's test) [36]. This is in contrast to other types of back pain, such as spondylolysis, where pain is worsened by extension of the spine and there is typically no radiculopathy or dural stretch signs. The most commonly affected levels are L4–L5 and L5–S1, which together account for 90% of symptomatic disc herniations [37]. Like the adult, the young athlete with a herniated disc will often complain of leg pain and sitting intolerance [36]. Symptoms that would be immediately of concern include bowel or bladder incontinence or retention and saddle paresthesias, raising suspicion of the cauda equina syndrome, which, if present, would necessitate emergent treatment.

Imaging

After ruling out emergent etiologies with history and physical examination, empiric treatment may be started even without imaging. Routine imaging for those with nonspecific acute LBP, brief in duration and without neurologic compromise, is not recommended [38]. While patients may expect or even insist on lumbar radiographs [39, 40], they are often unnecessary and have not been shown to lead to better outcomes [39, 41]. The decision about imaging is important since it is imperative to avoid unnecessary radiation exposure, especially in the female population, where gonads are not shielded with typical lumbar radiographs [36]. Obtaining plain films is appropriate if there is a history of trauma, chronic steroid use, evidence of instability, or spondylolysis. In addition, they may also be considered if LBP persists beyond 6 weeks despite conservative treatment, which typically includes relative rest, physical therapy, and a trial of nonsteroidal anti-inflammatory drugs (NSAIDs) [36]. Beyond standard radiographs, magnetic resonance imaging (MRI) is the test of choice for evaluating symptoms that fail to respond to conservative treatments after 4–6 weeks or to evaluate symptoms that may indicate neurologic compromise, infection, or tumors [42]. In addition, it is useful to assess disc morphology and helpful in the planning of interventional procedures such as epidural injections or surgery [39, 43]. MRI should be ordered with care and the results reviewed in reference to the history and physical examination. Studies have estimated that between 35 and 64% of asymptomatic patients under the age of 60 may have

degenerative or bulging discs [44–46]. Interestingly, the size or number of herniations seen on MRI does not correlate to the patient's symptoms or examination [47]. It is essential to have good clinical confidence that an abnormal disc is truly responsible for the patient's pain, particularly if invasive procedures such as injections or surgery are being considered.

Treatment

Treatment efficacies are poorly documented in the adolescent with a herniated disc [48]. Relative rest is encouraged, but complete bed rest, which can promote physical deconditioning, should be avoided [36]. Effective muscle control, specifically lumbar multifidi and transverse abdominis, can provide segmental stability by controlling the motion of the spine [49, 50]. Therapy that targets retraining of the stabilizing spinal musculature and peripelvic musculature has been shown to result in less LBP recurrence compared to therapy that does not include specific exercise training [51, 52]. This type of therapy, called motor control, has been shown to be more effective than medical management and education in chronic, nonspecific lumbar pain [53], although radiculopathy from nerve irritation or compression may not respond as vigorously.

Epidural steroid injections (ESIs) have been used to provide short-term clinical relief for patients with ongoing discogenic pain [54, 55], but there is a paucity of studies done with a placebo control. When ESI is compared with placebo, the results are conflicting, but the general consensus is that ESIs are reasonable for acute radicular pain when other conservative measures have failed and while waiting for the natural healing process to occur [28, 48].

When conservative treatment fails, a lumbar discectomy can be considered. The indications for surgery include the presence of the cauda equina syndrome, progressive or profound neurologic deficits, and persistent symptoms despite conservative treatment. In one study of surgical outcomes for herniated discs in the pediatric and adolescent population, lumbar discectomy was found to be relatively safe and successful, with a return to full athletic activities in 8–12 weeks after surgery [56]. Ranges for return to sports after surgery vary widely however, from 7 weeks to 12 months [57]. With conservative treatment, athletes typically return to sport in 3–6 months, with an average of 4.7 months quoted in Iwamoto's study [58].

Apophyseal Ring Fracture

In the skeletally immature population, forces that create disc herniation can create an associated apophyseal ring fracture. Because the fibers attaching the apophyseal ring to the AF are stronger than the fibrocartilage junction of the apophysis, an injury through the growing cartilage is possible [59]. The vertebral ring apophyses are located outside the epiphyseal plates of the vertebrae both cranially and caudally [60, 61] and begin to calcify at about 6 years of age. They start to ossify at about 13 years of age and begin to fuse with the vertebral body at about 17 years of age [59, 62, 63]. The ring apophyses do not add to the longitudinal growth of the vertebral body but act more like traction apophyses [60, 62, 64].

Apophyseal injuries are typically caused by trauma or overload in physically active individuals, particularly in sports such as wrestling and gymnastics [60]. Radiographic evidence of apophyseal injuries is generally not seen in nonathletes [60]. Several mechanisms have been proposed for these injuries. One likely etiology involves compression overload of the disc resulting in intravertebral disc herniation [60, 65–70]. A separate proposed mechanism is failure in tension–shear, analogous to the Osgood–Schlatter avulsion at the knee [60].

A physical examination with anterior apophyseal ring injuries is similar to patients with disc herniation and may include positive results from an SLR test, or back pain with forward flexion. A plain radiograph may show a triangular bony projection at the caudal or cranial anterior end plate [71]. In addition to X-ray findings, single-photon emission computed tomography (SPECT) bone scans, computed tomography (CT), or MRI can help identify the injury. Evidence-based treatment protocols are scant for this relatively

uncommon injury (although one study showed that this problem may occur in up to 28% of adolescents with a herniated disc, as evaluated by CT scan so care should be taken to ensure it is not overlooked) [72]. Treatment includes relative rest with avoidance of impact or spine flexion activities. In addition, neutral spine bracing has been used for symptomatic cases.

Lumbar Scheuermann's Disease (Atypical Scheuermann's)

Scheuermann's disease of the thoracic spine is discussed in the chapter on spinal deformity. This is associated with thoracic kyphosis. When similar end plate deformities occur during the growth period with Schmorl's nodes and end plate irregularities at the lower thoracic and upper lumbar regions (Fig. 6.1), the condition is often referred to as atypical Scheuermann's disease [73]. Although more frequently seen in males, it is not uncommon to see this in the female athlete with sports of extreme spinal motion such as gymnastics [74]. This injury can be quite painful and often presents with more of a flexion-based complaint. Examination will often reveal a flat back on forward flexion and at times even a kyphotic deformity of the lower thoracic and upper lumbar spine. A lateral radiograph will often show compressions of the end plate such as Schmorl's nodes and lumbus vertebrae.

Treatment is conservative with relative rest and anti-inflammatory medication. Temporary lordotic bracing is often helpful. Athletes in greater pain with a kyphotic deformity may find it challenging to return to sports.

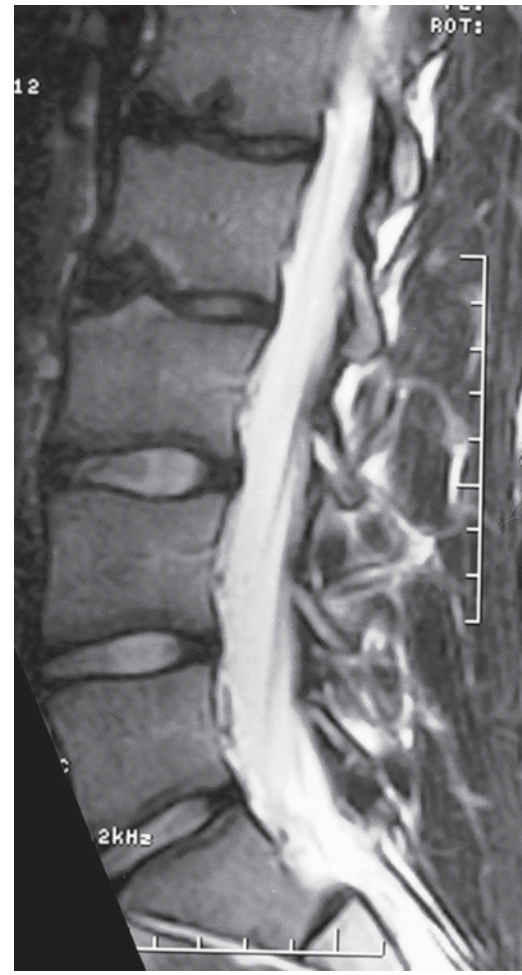

Fig. 6.1 Lumbar Scheuermann's

Posterior Element Injury

Spondylolysis and Spondylolisthesis

Back pain etiology differs in the adolescent versus the adult population. Spondylolysis is the most common cause of identified back pain in the adolescent athletic population reported as high as 47% in the young athlete in contrast to a disc etiology in 48% of adults [32]. Spondylolysis represents a stress fracture to the pars interarticularis between the inferior or superior articular processes of the facet joints (Fig. 6.2). This injury is secondary to repetitive cyclic loading of the pars from the facet process above while in lumbar hyperextension [75]. It is most common at L5 and is bilateral in 80% of cases. It may be multilevel in 4% of patients [76]. Spondylolysis is commonly seen in sports such as ballet, diving, gymnastics, football, and rugby. However, it is also seen in the general population. In one study, it was shown to occur in 4.4% of first graders and 6% of adults with no increased spinal morbidity after being followed up for 45 years [77]. How-

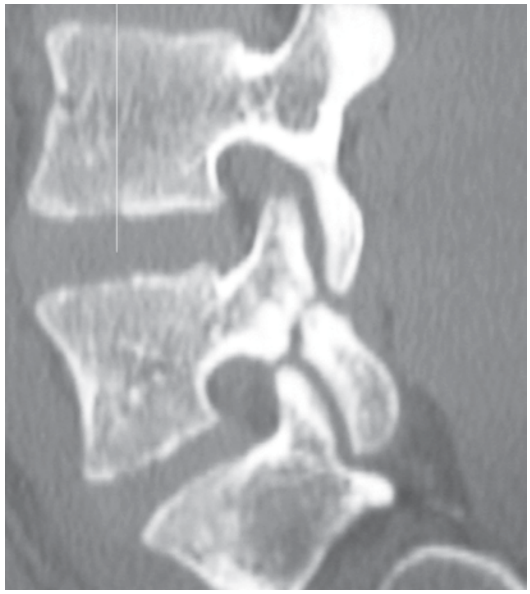

Fig. 6.2 Spondylolysis

ever, spondylolysis that occurs during the adolescent growth period is commonly associated with sports and is often painful. One meta-analysis of spondylolysis demonstrated that 84% of athletes did well with nonoperative treatment in a 1-year time period and success was not associated with bony union [78].

These athletes usually present with activity related pain. It is uncommon to experience pain while sitting unless the fracture involves a more anterior pedicle component of the stress fracture. There is usually no night pain or sitting intolerance. On examination, the athlete will demonstrate pain on lumbar hyperextension and single leg extension. Dural tension signs such as an SLR are usually absent.

Spondylolisthesis occurs with forward slippage of one vertebra on the caudal segment. This is most often seen at L5–S1. The Wiltse Classification is the classic characterization of the slip by cause. The types are: type I dysplastic, type II isthmic, type III degenerative (commonly L4–L5), type IV traumatic, and type V pathologic. Type II is the common athletic injury with only 4% of progression. Dysplastic types demonstrate a much higher progression at 32% [79].

Spondylolysis may be detected with plain radiographs but this modality is very insensitive to identifying early fractures. The most sensitive method is a SPECT bone scan [80]. Nonetheless, an MRI scan has demonstrated good sensitivity for detecting acute lumbar spondylolysis, particularly with the demonstration of pedicle edema representing an acute phase fracture [81, 82], and this modality imparts no radiation exposure. The CT scan is best to demonstrate details of the fracture as early, progressive, or terminal [83]. However, there is ionizing radiation exposure with CT, and it is often reserved unless there is a problem with healing such as a painful nonunion.

Treatment of the athlete with an acute spondylolysis is directed toward eliminating pain and returning the athlete to full function. One must recognize that although bony union may be desired, it is not necessarily related to a successful, pain-free, clinical state. A bony union may occur in most unilateral, half of bilateral, and no chronic fractures [84]. Treatment of spondylolysis is controversial. An initial period of sports restriction is needed. Bracing with a rigid thoracolumbar orthosis has not been demonstrated to improve outcomes but has been shown to allow the athlete a shorter return to sports activity, often in 4–6 weeks with continued brace wear for 3–4 months [85]. The length of bracing is also controversial. However, Sairyo demonstrated that healing was complete in 3.2 months if the fracture was early (by CT criteria) with a high signal on MRI, while it took more than 5 months to heal if it was a progressive fracture and lower in the MRI T2 signal [86]. The same author also demonstrated that predictability of bony union was 77% if the MRI scan showed high signal initially [87]. The purpose of bracing is not to achieve immobilization but to limit the lumbar hyperextension believed to be the injuring factor.

When using the Boston Brace protocol, the athlete is placed in the brace and started on antilordotic physical therapy for the first phase of 4–6 weeks. Athletic activity is limited to freestyle swimming and biking. After the initial period of sports limitation, the athlete who is pain free and compliant with brace wear and therapy is allowed a gradual return to full activity while

continuing brace wear. However, the brace will not allow full participation in some sports such as gymnastics. The duration of bracing is maintained for about 3–4 months. In the case where there was only pedicle edema seen, a 3-month period is used.

Full spinal stabilization must be achieved before returning the athlete to play. Physical therapy is also intended to strengthen the erector spinae and multifidi which are invariably shut down in the initial phases of activity restriction and deconditioning. It is also very useful to look at the biomechanics of the athlete before returning them to play. For instance, the dancer may demonstrate a weak gluteus maximus and therefore hyperextend at the lumbosacral juncture to compensate.

For those athletes who are still painful after 4 months of treatment, one must first determine if there are other comorbidities such as disc disease for sacro-iliac instability. If the pain generator is the original spondylolysis, there is consideration for the utilization of both growth stimulators [88]. In chronic refractory cases, there is evidence that this may enhance healing with either a bony union or stable fibrous union. Surgical intervention is rarely needed for symptomatic unilateral lesions. However, painful spondylolisthesis in more advanced grades may require surgical stabilization. Lesions at L5 are usually treated with fusion. L4 pars defects may be addressed with direct repair of the fracture.

Spinous Process Apophysitis

A less well-described injury to the spinous processes, also seen exclusively in the adolescent athlete, is spinous process apophysitis. This may be associated with increased lordosis, often referred to as lordotic LBP. This condition is not well described in the literature but is most commonly seen in athletes with repetitive hyperextension of the lumbar spine, such as gymnasts, figure skaters, and ballet dancers. As with calcaneal apophysitis, spinous process apophysitis is due to repetitive impact (from spinous process and soft tissue impingement during lumbar hyperextension) and axial loading.

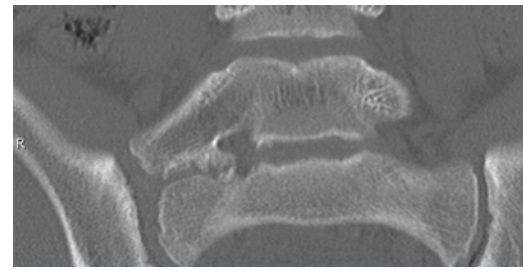

Fig. 6.3 Transitional pseudarthrosis

Typically this condition involves the lower lumbar spine, but it may extend to the lower thoracic levels as well. Physical examination may mimic spondylolysis (discussed elsewhere in this chapter) where pain is worsened by lumbar extension or single leg extension. However, the patient's pain is characteristically worsened by direct palpation or percussion over the spinous processes, a finding which is not typical with spondylolysis. Pain may also be worsened by resisted active extension of the spine [89]. Imaging patterns also differ from spondylolysis. X-rays and CT scans typically do not show fracture or defects in this atraumatic, overuse injury. However, SPECT scans would be expected to show diffusely increased uptake in the affected spinous process, rather than in the pars interarticularis, as is seen with spondylolysis.

Prognosis and recovery times are better than those for spondylolysis [86], and bracing is not typically required unless it is used for pain relief in refractory cases. Adjusting activity to avoid lumber extension and impact is typically sufficient. As with other forms of apophysitis, 6 weeks of relative rest is usually effective but, on occasion, activity modifications may need to be extended to 3 or 4 months.

Bertolotti's Syndrome

This occasionally painful syndrome in the athlete is manifested by a transitional vertebra, usually an enlarged transverse process of L5 that articulates with a pseudarthrosis at the sacral ala (Fig. 6.3). This congenital anomaly has been reported from 4 to 30% of the population but is

usually symptom free [90]. However, the athlete performing repetitive lumbar hyperextension may aggravate this pseudarthrosis and present with extension-based pain [91]. The pseudarthrosis may also limit motion and predispose some disc degeneration at the level above [92].

Treatment is conservative with relative rest, anti-inflammatory medication, possible corticosteroid injection, and intense attention to spinal biomechanics and stability to limit injury to this region. Surgical resection of the pseudarthrosis and fusion has been described but is rarely done [93].

Sacro-Iliac Pain

The sacro-iliac joint (SIJ) is the point of force transfer from the lower to the upper extremities. The SIJ has demonstrated only minimal motion of about 2–6 degrees in reference to the ileum [94]. However, this minimal motion is important for force transfer. The stable position for the sacrum is forward nutation (flexion) relative to the ilium, or commonly called the "closed pack position" [95]. It is the multifidi and erector spinae that ensure this motion. These are the muscles that are often inhibited with spinal injuries. This nutated position is important on impact with the ground such as running. The antagonistic motion is counter-nutation or posterior tilting of the sacrum relative to the ilium. This is primarily accomplished by the biceps femoris action on the sacrotuberous ligament which lies between the ischial tuberosity (biceps femoris attachment) and the lower sacrum. The counter-nutated position is the relaxed position. Asymmetric landing on one leg as well as ligamentous laxity and incomplete rehabilitation of extensor muscles may predispose the athlete to instability of the SIJ.

SIJ pain is often elicited on provocation testing. The sensitive tests include the thigh thrust where the hip and knee are flexed at 90 degrees and a downward force is applied to the knee. The sacral thrust is useful with a direct compression of the sacrum in the prone athlete. Other tests include lateral compression and distraction of the pelvis. One must always consider infectious and inflammatory processes in the SIJ as well as stress fractures in the track athlete.

Treatment involves joint mobilization, active release therapy, and a well-designed exercise program to address the lumbar extensors in a neutral zone, the gluteus maximus, and all core muscles. These muscle groups encourage the stable nutated position of the SIJ. A sacro-iliac belt may be useful to improve symptoms related to instability while the exercise program is initiated.

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Throwing Sports and Injuries Involving the Young Athlete's Spine

7

Peter Kriz

Introduction

In throwing or overhead sports, the generation of force and energy that is ultimately transferred to a ball, spear, disc, or other projectile object is a result of a complex, coordinated sequence of events involving elements of the kinetic chain. Each of the "links"—the individual body segments and joints of the athlete's body—requires stability, strength, stamina, mobility, and neuromuscular control to execute an effective, efficient transfer of energy along the kinetic chain. A disturbance in force generated at any link translates into an increased load on the next link in the chain. Consequently, injury may result to the body segment or joint distal to the weak link [1].

In the pediatric and adolescent throwing athlete, the thoracolumbar spine and core musculature collectively are key links in the transmission of force resulting in limb acceleration. However, inherent to this subset of the athletic population are periods of rapid growth, which contribute to loss of flexibility and muscle–tendon imbalance. Strength and flexibility imbalances on opposite sides of more proximal body segments such as the hip/pelvis (e.g., tight, overactive erector spinae and hip flexors and weak, inhibited gluteus medius and rectus abdominus musculature) contribute to asymmetries which can lead to overuse injuries and improper mechanics which affect more distal joints such as the shoulder or elbow in the throwing or overhead youth athlete.

In this chapter, we will review the functional anatomy and biomechanical interaction of the pelvis, thoracolumbar spine, and scapulothoracic articulation in the throwing/overhead athlete; the presentation of thoracolumbar spinal injuries in throwing/overhead youth athletes; and the treatment and return to play issues pertaining to various throwing and overhead sports.

Anatomy and Biomechanics of a Throwing Athlete

The throwing athlete generates most of his or her throwing power through a complex sequence of muscle activation, which begins in the lower limbs and translates through the hips and trunk (core musculature) into the arm, with eventual release of energy through the fingers. The kinetic chain in throwing includes the following motion: stride, pelvis rotation, upper torso rotation, elbow extension, shoulder internal rotation, and wrist flexion [2]. These motions have been further integrated into phases of the overhand throw, described slightly differently by Andrews and Jobe and their respective colleagues (Fig. 7.1) [3–6]: (1) *wind-up* (Andrews, Jobe); (2) *stride* (Andrews) or *early cocking* (Jobe); (3) *arm cocking* (Andrews) or *late cocking* (Jobe); (4) *arm acceleration* (Andrews) or *acceleration*

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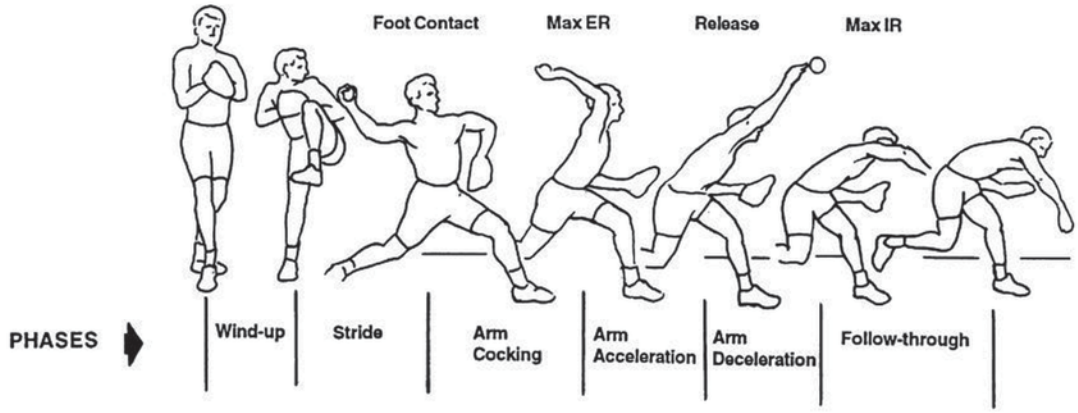

Fig. 7.1 Phases of the overhand throw. (Reprinted from Fleisig et al. [40], Copyright 1999, with permission from Elsevier)

(Jobe); (5) *arm deceleration* (Andrews); and (6) *follow-through* (Andrews, Jobe). During the wind-up phases of the throwing motion, leg and torso muscles generate potential energy by raising the center of gravity. A change from potential to kinetic energy occurs during the arm cocking and acceleration phases, which are "controlled falling phases" [7]. As each joint rotates forward, the subsequent joint completes its rotation back into a cocked position, allowing the connecting segments and musculature to be stretched and eccentrically loaded [2].

External rotation of the shoulder, which reaches a maximum value of approximately 180°, is a combination of glenohumeral rotation, trunk hyperextension, and scapulothoracic motion. At the time of maximum shoulder external rotation during arm cocking/late cocking phases of throwing, eccentric contraction of shoulder and elbow musculature produces shoulder internal rotation torque and elbow varus torque. At this position, both the shoulder and the elbow are susceptible to injury. At the time of ball release, significant energy and momentum have been transferred to the ball and the throwing arm. Following ball release, a kinetic chain is used to decelerate the rapidly moving arm with the entire body. By producing large compressive forces, shoulder and elbow muscles resist joint distraction [2].

The pelvis and the lumbar spine are key in providing a stable, level foundation during the throwing motion [8]. Rotation of the larger base segments of the pelvis and upper torso about the longitudinal vertebral axis results in transfer of a substantial amount of force and energy to the more distal segments of the kinetic chain [2]. If spine motion, strength, and stability cannot be maintained, loss of control, dissipation of energy, and altered shoulder biomechanics throughout the throwing motion are bound to occur [8].

Hip flexor tightness, a common finding in adolescent athletes, can contribute to several biomechanical flaws in the throwing motion, ultimately increasing the likelihood of shoulder and elbow injury. Hip inflexibility can lead to decreased stride length and excessive hip external rotation affecting femur and foot positioning during throwing or serving motions in pitchers and tennis players [8, 9], leading to an open shoulder position, which has been associated with increased humeral internal rotation torques and elbow valgus loads in baseball-pitching-biomechanical studies [10].

"Controlled" lumbar lordosis during the arm cocking phase of throwing is accomplished in part by sustained eccentric contractions of the abdominal musculature. If this supportive abdominal musculature is weak and prone to fatigue, there is greater reliance on the static stabilizers of the trunk such as the iliofemoral ligament and the posterior elements of the spine to provide passive restraint [8].

The thoracolumbar fascia plays an important role in the biomechanics of lumbar motion by:

(1) stabilizing the spine in forward flexion and (2) enhancing coactivation with the posterior extensor group (e.g., latissimus dorsi, erector spinae, multifidi, and gluteus maximus) through its anterior attachments of the transversus abdominis and internal oblique fibers. Protective hydrostatic intra-abdominal pressure is produced by this coactivation, resulting in the absorption of compressive forces [11].

Moving more cephalad up the kinetic chain, the scapula plays an integral role in throwing kinetics by facilitating force transfer from the core to the hand. Given the relatively limited bony attachment of the scapula to the thorax and the humerus, scapulothoracic articulation is largely dependent on muscle activation of the upper and lower trapezius, rhomboids, serratus anterior, and to a lesser extent, the pectoralis major/minor and latissmus dorsi muscle groups, to provide dynamic stability [12]. Optimal energy transfer to the upper extremity is achieved when the scapular stabilizers are as equally developed as the hip and trunk musculature.

Later phases of the throwing/pitching motion, most notably arm deceleration and follow-through, place significant stresses upon the thoracolumbar and scapulothoracic segments of the young athlete's spine. Approximately 85 % of the muscle activation to slow the forward-moving arm is generated in the periscapular and trunk muscles, rather than the rotator cuff [13]. Trunk flexion during follow-through allows energy to be absorbed by the large musculature of the trunk and legs, reducing stress on the throwing arm by transferring weight and momentum of the body to the lead leg. Large loads are produced to decelerate the rapidly moving arm and prevent distraction at the shoulder and elbow. Shoulder posterior force is produced by activation of infraspinatus, supraspinatus, teres major and minor, latissimus dorsi, and posterior deltoid. Additionally, serratus anterior, middle trapezius, and rhomboids are activated to decelerate scapular protraction [2, 14]. Most overuse throwing injuries at the elbow and shoulder are believed to occur during arm cocking and arm deceleration.

Another concept paralleling the kinetic chain and throwing motion in overhead athletes is one of torque transfer, known as the serape effect [15]. Like the Mexican shawl that drapes loosely and crosses over the front of the body in an "x" shape, the serape muscles—rhomboids, serratus anterior, external and internal obliques—are a chain of muscles diagonally related to each other that connect the hip to the contralateral shoulder and undergo a coordinated, concentric activation. During high-velocity throwing, a well-sequenced, coordinated contraction of the serape muscles transfers internal forces from larger body segments such as the trunk, pelvis, and lower extremities to a relatively smaller segment—the throwing upper limb. The result is a greater force generation and projectile velocity than would be produced from the upper segment alone [8].

Presentation of Thoracolumbar Injuries in Throwing/Overhead Athletes

Thoracolumbar injuries in young throwing and overhead athletes can be categorized into muscular, bone-related, and discogenic.

Muscular

Acute strains of core muscles such as the internal and external obliques, transversus abdominus, and rectus abdominus lumborum are common in young throwing/overhead athletes. In baseball and javelin throwing, oblique muscle strains typically present with a sharp, sudden onset of side pain after throwing, swinging, or twisting movements. Location is near or on the rib cage and is associated with localized tenderness. Reinjury rates of core muscle strains are relatively high, particularly among professional baseball players. Predisposing factors for reinjury are thought to include lack of complete healing and failure to modify training techniques, form, or preparation [16]. Core muscle strains are also fairly common in tennis players and are sustained during hitting overhead strokes and serves (rectus abdominus), as well as during changes in service motion or in ground strokes (obliques) [17].

Upper segment–lower segment strength imbalances in the pediatric and adolescent throwing athlete can also contribute to injury. Young pitchers demonstrate increased trunk and leading hip rotation velocity from cocking to acceleration phases compared with adult pitchers, likely due to a decreased capacity to generate lower core force. Consequently, this upper–lower trunk rotational disassociation results in a tendency for young pitchers to "open up" with their throwing arm positioned behind their trunk with increased anterior loads across the shoulder and increased valgus loads across the medial elbow [13, 18].

Bone-Related

The adolescent spine has areas of growth cartilage and immature ossification centers that are susceptible to compression, distraction, and torsional injury. In the skeletally immature athlete, these areas are often the weakest link of energy transfer [19, 20]. Consequently, injuries to the anterior column (vertebral body, intervertebral disc) and the posterior column (pedicles, facet joints, pars interarticularis, spinous process) of the spine can manifest in throwing and overhead athletes, due in part to the sheer volume and intensity of repetitive pitches, serves, and throws performed.

Vertebral end-plate injuries are common among young throwing and overhead athletes. Risk factors include the vulnerable growth period of adolescence, trauma, and overload. Examples include: (1) ring apophyseal abnormalities (including Schmorl's nodes), which have been reported in tennis players [17], and (2) lumbar spine (L2–L5) vertebral osteophytes in shot putters and discus throwers [21]. Typically, such injuries present with localized pain in the thoracolumbar spine associated with flexion, extension, or axial loading of the thoracolumbar spine.

Injuries to the posterior elements of the lumbar spine related to rotation, hyperextension, and flexion during repetitive activities such as pitching and bat swinging are relatively common injuries in baseball players as they are in many overhead/throwing sports. Stress reactions and fractures of the pars interarticularis (spondylolysis) and the pedicles may present in the adolescent throwing/overhead athlete as extension-based low back pain. A recent study by Sakai et al. showed the incidence of lumbar spondylolysis in Japanese professional baseball players was 30%—more than five times the incidence of lumbar spondylolysis in the general Japanese population (5.9%) [22]. Likewise, young tennis players are also susceptible to posterior element injuries, likely due to poor biomechanics during serving, including the inability to achieve adequate knee flexion to gain extension of the trunk. Consequently, trunk extension is achieved in the lumbar spine at the expense of the posterior elements. Aylas et al. performed an observational study involving 33 asymptomatic elite adolescent tennis players with a mean age of 17.3 years. Magnetic resonance imaging (MRI) of the lumbosacral spine was performed in 18 male and 15 female players. Twenty-eight of 33 players (84.8%) had an abnormal examination, with nine players demonstrating 10 pars lesions (one player had two-level involvement) predominately at the L5 level; three out of ten were complete fractures (all grade 1–2 spondylolisthesis). Twenty-three patients showed signs of early facet arthropathy at the L4–L5 and L5–S1 levels [23].

Overhead/throwing sports that distribute an asymmetric load on the trunk and shoulders—such as javelin and tennis—have been associated with an increased reported incidence of scoliosis. However, the rotational curves of these athletes appear to be small (Cobb angles < 15°) and asymptomatic. It is not uncommon to encounter one-sided hypertrophy of back and shoulder muscles in overhead/throwing athletes, which may result in a falsely positive Adams forward-bend scoliosis screen [8, 24, 25].

Discogenic

Disc-related disease, while less common in pediatric and adolescent throwing/overhead athletes than in adult competitors, is prevalent among young athletes compared to their sedentary peers, particularly over the age of 12 [26, 27]. While

disc injuries among collision sport and weightlifting athletes are often cited in the literature [11, 24, 27, 28], throwing and overhead athletes are also susceptible to disc injury due to axial loading, hyperextension/-flexion, and rotational forces involved both in training/conditioning as well as the biomechanics of throwing, swinging, and serving. Pain worse with forward flexion is typically located in the low back, buttock, posterior thigh, and/or leg as the L4–L5 and L5–S1 discs represent 92 % of cases of herniated nucleus pulposus injuries in adolescents [27, 29]. In a study of elite adolescent tennis players performed by Aylas et al., 13 of 33 players showed mild-to-moderate disc degeneration predominantly at the L5–S1 level, followed by the L4–L5 level [23]. While there are no available studies assessing the prevalence of degenerative disc disease in adolescent baseball players, a study of college athletes found baseball players to be at the highest risk of lumbar disc degeneration compared to other groups of athletes, at a rate greater than three times that of nonathlete controls [30, 31]. Baseball players are known to develop lumbar disc herniations, comprising 13–35 % of reported cases in studies of elite athletes [32, 33].

Treatment of Thoracolumbar Spinal Injuries in Throwing/Overhead Athletes

Assessment of core, peripelvic, and lumbar dynamic stabilization is a key tenet to the evaluation of a throwing/overhead athlete with a thoracolumbar spinal injury, as well as the development of an appropriate treatment program. Single-leg bridge and single-leg-squat testing are clinical assessments of the lower abdominal and hip abductor musculature as well as trunk neuromuscular control that can be performed rapidly during a clinical evaluation. Additionally, postural assessment (e.g., anterior pelvic tilt, lumbar lordosis) and flexibility testing (Thomas testing for hip flexor tightness, popliteal angles for hamstring tightness) can provide clues to muscular imbalances which may be predisposing a young throwing/overhead athlete to injury anywhere along their kinetic chain.

Rehabilitation of thoracolumbar injuries in the young throwing/overhead athlete involves provision of therapeutic exercises to correct identifiable muscle imbalances that result from deficits in flexibility, strength, endurance, and balance [34]. Konin and colleagues have established a performance enhancement and injury prevention program for throwing athletes that addresses the kinetic chain as well as Serape muscles, and includes the following components: (1) lower-extremity flexibility (ipsilateral hip extension, hip internal/external rotation), (2) core training (prone cobra progressions), (3) lower-extremity balance/proprioception (standing pulls with resistance tubing addressing gluteal weakness), and (4) and lower-extremity functional strength training (wind-up, lunge, and step-up exercises combined with trunk rotation). This comprehensive program provides multiplanar, functional exercises that incorporate the lumbopelvic-hip core complex and utilize proprioceptive and balance inputs to stabilize and strengthen body segments along the kinetic chain, specifically identifying and correcting biomechanical errors such as open shoulder/trunk position due to limited hip internal rotation, hip/shoulder "dropping" due to poor pelvic control/gluteal weakness, and reducing deceleration injuries to the shoulder by improving lunge/lower-extremity functional strengthening [35]. Lumbopelvic control has been correlated with overall performance enhancement in elite-level pitchers. Chaudhari et al. demonstrated that pitchers with stable pelvic positions in a pitching stance had significantly fewer walks plus hits per inning as well as significantly more innings pitched during a minor-league season than pitchers with less stable pelvic positions [36].

Return To Play Issues Involving Thoracolumbar Injuries in Throwing/Overhead Young Athletes

Pain-free range of motion, stabilization of the lumbopelvic-hip core complex, and progression through sport-specific phases of rehabilitation

are the prerequisites for a throwing/overhead athlete to return to play following a thoracolumbar injury. To some extent, injury type and severity dictate the amount of time loss and recovery. Abdominal muscle strains take several weeks for recovery, particularly in baseball pitchers. For athletes with lumbar spondylolysis, return to full competition has been accomplished within 4–6 weeks of treatment initiation with a lumbar orthosis and pain-free extension [37]. Athletes who undergo surgical treatment (e.g., single-level spinal or lumbosacral fusion, microdiscectomy) and are not participating in collision sports are typically allowed to return to sport 6–12 months post-operatively [38, 39].

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Spine Injuries in Collision/Heavy Contact Sports

8

Deborah I. Light and Hamish A. Kerr

Introduction

Collision sports involve play in which athletes routinely collide with other players and playing surfaces. Injuries to the spine can occur in all of these types of sports, and while some patterns of spinal injury are more common in one collision sport than in another, similar mechanisms can lead to injury in all collision sports. The majority of the available data in young athletes comes from the study of such sports as American football, rugby, and hockey. This chapter will focus on spinal injuries and injury mechanisms that are common to young participants of collision sports, with specific discussion of the current understanding of injury patterns and mechanisms as well as prevention. The issue of participation in collision sports in adolescents with congenital or acquired spinal abnormalities is also discussed.

Epidemiology and Prevention

Data on the incidence of spinal injury in both the adolescent and the adult population come largely from national injury registries in the USA and Canada (e.g., the National Football Head and Neck Injury Registry, National Center for Catastrophic Sports Injury Research, Canadian Committee on Prevention of Spine and Head Injuries Due to Hockey [now SportSmart Canada]). No similar large spinal injury registries exist for rugby, though data from several countries have been reported in the literature, all with different collection methods. Efforts continue in the New England area to create a large and inclusive rugby injury registry [1]. Table 8.1 summarizes recent available data regarding the overall incidence of spinal injuries in American football, rugby, and ice hockey.

Morbidity and mortality from spine injuries are less common than that from traumatic brain injury in pediatric athletes [2, 3], but the incidence of severe injury increases with age and playing level, presumably due to increasing levels of contact and speed in these participants [2, 4]. Catastrophic spinal injury resulting in permanent impairment or death is a dreaded event in collision sports and, as such, has received much attention in the analysis of injury registry data.

An understanding of the mechanisms that lead to spinal injuries is critical in the ongoing effort to prevent them. In both American football and ice hockey, the primary mechanism of catastrophic spine injury has been shown to be axial loading in the setting of either a hyperextension or a hyperflexion injury [5–8]. A recent review of football injuries recorded by the National Center for Catastrophic Sports Injury Research between

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Table 8.1 Incidence and severity of spinal injury in contact/collision sport

Sport	Study	Age	Study design	Overall incidence	Game incidence	Practice incidence	Severity
Rugby (all spine)	Fuller, Brooks, and Kemp, Clin J Sports Med 2007, 17:10–16	Adult	Prospective cohort of 546 male professional players (UK)	–	10.90 (95% CI: 9.43–12.60) per 1,000 player match hours	0.37 (0.29–0.47) per 1,000 player training hours	No CSI, 3 players sustained career-ending injuries
Rugby (neck)	Swain, Pollard, and Bonello, Chiropractic & Osteopathy 2010, 18:18–:1–12	Adult	Prospective cohort of 262 amateur male players (Aus)	–	2.9 (95% CI: 2.3–3.6) injuries/1,000 player hours	–	69.3% neck injuries were minor, 17% mild, 6.8% moderate, and 6.8% severe
Rugby (all injuries)	King and Gissane, Clin J Sports Med 2009;19:277–281	26 ± 4.5 years	Prospective cohort study of 53 player matches with a risk exposure of 951 playing hours	238.2 (95% CI 188.4–301.0) head/neck injuries/1,000 player hours, 37.4 (95% CI 20.7–67.6) chest/back injuries/1,000 player hours	–	–	–
Football (CSI)	Cantu and Mueller, Neurosurgery, 2003;53:358–363	High school and adult	Retro 233 CSI 1977–2001	0.52/100,000 high school participants, 1.55/100,000 college participants	–	–	Catastrophic injury
Football (CSI)	Mueller and Cantu, Annual Survey of Catastrophic Football Injuries, 2008	High school and college	Annual Survey of Catastrophic Football Injuries registry	(based on estimated 1,800,000 participants) 0.72 injuries/100,000 players	–	–	Incomplete neurological recovery
Football (stinger/cervical neurapraxia)	Charbonneau, McVeigh, and Thompson, Clin J Sport Med 2012;0:1–6	College	Prospective cohort of 244 players from the Canadian Atlantic University Sport football league	26% (64 of 244; 95% CI: 21–32%)	–	–	–

Table 8.1 (continued)

Sport	Study	Age	Study design	Overall incidence	Game incidence	Practice incidence	Severity
Football (all spine)	Mall, Buchowski et al., Am J Sports Med 2012;40:1755	Adult (NFL)	Retrospective descriptive epidemiological study over a period of 11 years in the NFL, including 987 spine injuries	0.93 axial spine injuries/1,000 athlete exposures	3.55 injuries per 1,000 athlete exposures	0.41 injuries per 1,000 athlete exposures	44.7% cervical spine, 30.9% lumbar spine, 3.9% thoracic spine and ribs, 0.6% spinal cord, 9.8% unclassified
Football (CSI)	Boden, Tacchetti et al., Am J Sports Med 2006;34(8):1223–1232	High school and college	Retro 196 CSI reported to NCCSIR over 13 years	1.1/100,000 high school participants, 4.71/100,000 college participants	146 injuries (75.6%)	47 (24.4%)	76 had quadriplegia, 5 athletes had Brown-Sequard-like syndrome, 1 made a full recovery
Ice hockey (all spine)	Rishiraj, Lloyd-Smith, Lorenz et al., J Sports Med Phys Fitness 2009;49:159–66	College	Prospective study of 46,215 player exposures	0.35 per 1,000 athlete exposures	-	-	Average of 4.3 sessions lost due to injury

CSI catastrophic spine injury, *NCCSIR* National Center for Catastrophic Sports Injury Research

1989 and 2002 showed an annual incidence of catastrophic football injuries of 1.1 per 100,000 high school athletes, and 4.72 per 100,000 college athletes [9]. Similar statistics have been reported in rugby [10]. These injuries were three times more likely to occur during games than in practice, and the highest percentage occurred to defensive backs, who are routinely exposed to axial loading forces during tackles [9]. Likewise in hockey, most spinal cord injuries occur during competitive games, and occur in the setting of axial loads to the helmet from striking the boards [11, 12]. In rugby, the scrum, spear tackle, and the ruck and maul have likewise been identified as major causes of catastrophic cervical spine injury in youth and adult players, secondary to axial loading mechanisms on the flexed cervical spine [10, 13] (see Fig. 8.1).

In both American football and Canadian ice hockey, the incidence of catastrophic spine injury was noted to increase after the introduction of improved helmet designs, leading several authors to conclude that players were using their heads as the initial point of contact due to a false sense of invulnerability. In football, the practice of using the helmet as the initial contact point in blocking and tackling became known as "spearing," and has been banned by the National Collegiate Athletic Association (NCAA) and the National Federation of State High School Athletic Associations since the mid-1970s. Similarly in hockey, the practice of checking from behind has been shown to result in axial loading injuries of the flexed cervical spine during collision with the boards or other players, and penalties for this practice were instituted in the mid-1980s in Canada [12, 14].

Enforcement of these safety rules may still remain an issue in some venues, particularly for adolescent participants. Heck reported that the incidence of spearing was not significantly different in 1975 compared to 1995 for one high school team, when the video was reviewed, despite the ban on spear tackling [15, 16]. This was attribut-

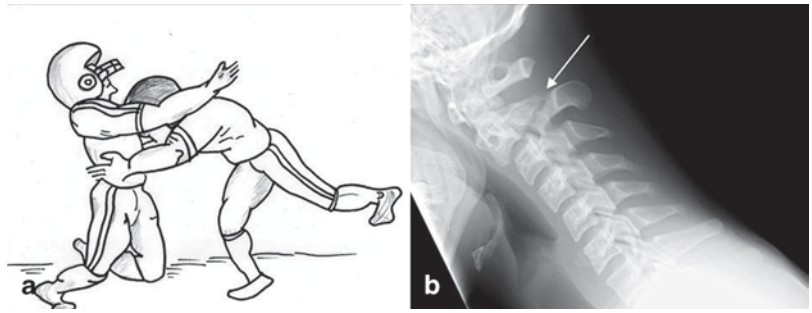

Fig. 8.1 Axial loading mechanism of injury on a flexed cervical spine. During a spear tackle, the athlete impacts with the vertex of the helmet (**a**). When the neck is slightly flexed, there is loss of the cervical lordosis, (**b**) predisposing to fracture/dislocation (*arrow*) in the setting of an axial load. (Image (**a**) courtesy of George Isaacson)

ed to lack of enforcement and lack of understanding of spearing on the part of officials [17]. Nevertheless, there was a decrease in the incidence of severe cervical spine injury in football during a similar period, from 7.72 per 100,000 high school athletes (30.33 per 100,000 collegiate athletes) to 2.31 per 100,000 high school athletes (10.66 per 100,000 collegiate athletes) in 1987 [18]. In Canadian ice hockey, a 69% decrease in spinal injuries was reported in the period 2001–2005 compared to that before 2001 [12].

Perhaps one of the best examples of injury prevention due to modification of playing rules and tactics comes from New Zealand, where a nationwide injury prevention program was implemented in 2001. Called RugbySmart, it was aimed at educating players and coaches about safer playing techniques and limiting exposure to axial loading injuries. Quarrie and colleagues analyzed the incidence of spinal injury in rugby in New Zealand from 1976 through 2005, and have shown that the implementation of this program has coincided with a decreased incidence of severe spinal injury, from 2.7/100,000 players between 1996 and 2000 to 1.3/100,000 during 2001–2005 [19].

Sprains and Strains

Sprains of the ligamentous structures and strains of the muscular elements of the spine can occur in isolation or with other spinal injuries. The most common cause of cervical sprain is motor vehicle accidents, though Versteegen and colleagues reported that almost 25% of cervical sprains presenting to an emergency room over a 25-year period were a result of sports injuries, with the highest incidence occurring in adolescents [20]. Patients may present with pain or limitation of movement, and may have an appreciable muscle spasm on examination. The presence of bony tenderness or any neurological deficits should prompt consideration of more serious spinal injury, and requires further investigation. Ligamentous injury involving a complete tear may result in cervical spine instability. For patients in whom acute fracture or neurological injury has been ruled out, flexion and extension radiographs can be helpful in ruling out abnormal cervical spine alignment.

Adolescents with an isolated sprain or strain of the cervical or thoracolumbar spine should be managed individually. Symptoms may be addressed with analgesics and anti-inflammatories, and some patients may benefit from physical therapy. Athletes can return to play when they are asymptomatic, and have full painless range of motion with normal strength.

Fractures and Dislocations

Fractures and dislocations are the most common cause of catastrophic cervical spine injury in collision sports [21]. As described above, these injuries are often caused by axial loading with flexion, as can occur in rugby during a scrum collapse, during spear tackling in football, or checking into the boards in hockey. The most

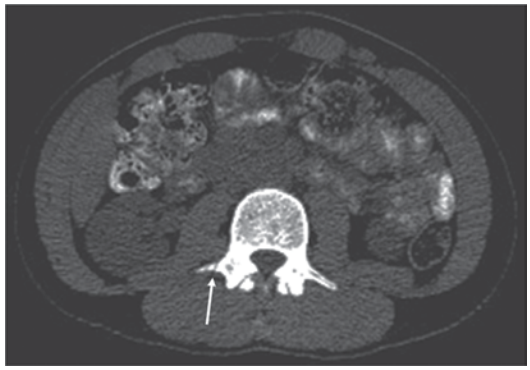

Fig. 8.2 Fracture of the transverse process (*arrow*) in a rugby player

common cause of spinal cord injuries in rugby is facet dislocation [21]. Fractures of the cervical spine and thoracolumbar spine can occur in the vertebral body, spinous processes (Fig. 8.2), or posterior arch. The most frequently fractured cervical vertebrae are C4–C6 [18]. Hyperflexion mechanisms can result in odontoid fractures and atlantoaxial dislocations [22], as well as compression fractures of the vertebral bodies. Hyperextension can result in fractures of the atlas, and fractures of the posterior spinal elements, including the posterior arch and spinous processes. Avulsion fractures of the spinous process of C7 or T1, also known as clay-shoveler fractures, are seen more frequently in football linemen and weightlifters [3].

Fractures of the thoracolumbar spine can also occur in collision sports which place axial loads on the spine. In adolescents, sports injuries have been reported to account for up to 21% of thoracolumbar fractures [23]. Compression fractures as a result of axial loading injuries, such as teardrop and burst fractures, are commonly seen in rugby players [13]. Teardrop fractures are defined by the presence of a fragment of the anteroinferior corner of the vertebral body, while a burst fracture describes a comminuted fracture of the vertebral body. These fractures are considered unstable, and can be associated with neurological compromise if the vertebral body fragment translates posteriorly toward the spinal canal.

Full spinal precautions and prompt transportation to a facility equipped to manage spinal injury are required for any athlete in whom a spinal fracture or dislocation is suspected. This should include any player with focal neurological signs or symptoms that are not consistent with a simple burner or stinger (discussed later in this chapter), neck or back pain with point tenderness after an appropriate mechanism of injury. It has been shown that the time to reduction of fracture–dislocation injuries in rugby players is directly related to neurological outcome [21]. Prompt recognition and transport is therefore critical.

Return-to-play decisions for athletes who have sustained a spinal fracture depend greatly on the type and location of the fracture. It is generally agreed that high cervical fracture or fusion is an absolute contraindication to return to play [24]. Some authors have argued that injuries requiring multilevel fusion should also constitute a relative or absolute contraindication to continued participation, though little data are available regarding recurrent injuries in collision sports in such patients [24].

Specific Cervical Spine Injuries

Cervical spine injuries have been estimated to occur in approximately 10% of football players [25]. Most cervical spine injuries in collision sports involve flexion or extension injuries with an axial load. Injury can occur to the bony structures, supporting ligaments and muscles, or the spinal cord and exiting nerve roots. Flexion injuries can cause compression of the anterior spinal structures, which can lead to compression fractures of the vertebral bodies. Simultaneously, muscle and ligamentous injury can be caused by stretching of the posterior ligaments and muscles. Common mechanisms of flexion injuries include spearing in American football, hockey players hitting the boards or the ice with the top of the helmet, and scrum collapses or spear tackles in rugby. Hyperextension injuries can cause compression of the posterior spinal structures, resulting in injury to the posterior elements of the spine, including hangman fractures (bilateral fractures of the C2 pedicles), and posterior arch fractures [22]. Hyperextension can also pinch the

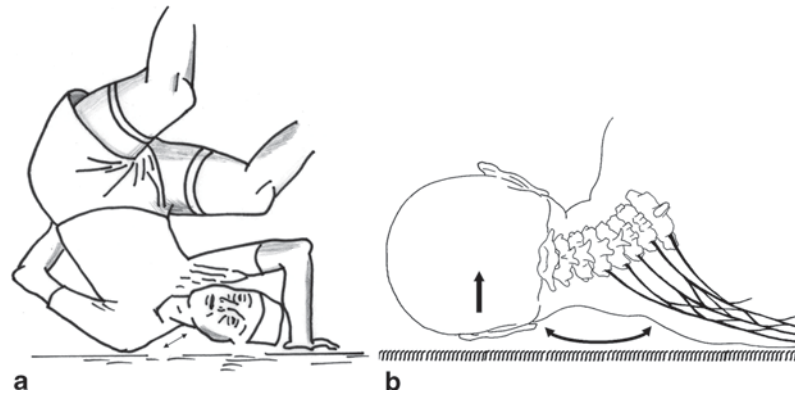

Fig. 8.3 Injury mechanism of a burner or stinger. A downward force on the shoulder with simultaneous lateral neck flexion to the contralateral side (**a**) causes traction injury to the cervical nerve roots or the brachial plexus (**b**). (Images courtesy George Isaacson (**a**) and William H. Light, MA, PhD (**b**))

spinal cord and lead to neurological injuries such as cervical cord neurapraxia.

Transient Neurological Injuries

Stingers and Burners

Stingers and burners are relatively common injuries in collision sports, and Wilson et al. [26] reported that up to 65 % of collegiate-level football players sustain such an injury during the course of their career. In high school and college-age patients, stingers with prolonged symptoms are the most common reason for evaluation of the cervical spine in the emergency department setting [27]. These injuries present as transient neurological deficits involving the brachial plexus of a single upper extremity. Patients may complain of burning pain or numbness and tingling, which may or may not be accompanied by motor weakness. Symptoms typically resolve within 24 hours, though some athletes may experience symptoms for several weeks. Symptoms are almost always unilateral, and the presence of bilateral symptoms or lower extremity involvement should prompt consideration of spinal cord pathology.

Two mechanisms of injury have been proposed. The first is a traction injury of the brachial plexus or cervical nerve roots, caused by forcible downward movement of the shoulder with simultaneous lateral neck flexion to the opposite side (see Fig. 8.3). This mechanism is more common in adolescent athletes [28].

A compressive mechanism has also been described, whereby the cervical nerve roots are compressed within the neural foramina during ipsilateral head rotation in the setting of an axial loading force [29].

Players who have complete resolution of their symptoms can generally be returned to play the same day. However, coaches and physicians should be aware that athletes who have sustained a stinger in the past are more likely to have another one. It has been shown that the relative risk of sustaining a recurrent stinger is twice that of sustaining a first stinger [27]. Cervical canal stenosis, discussed in detail below, is a possible risk factor for stingers as well [27, 30, 31].

Cervical Cord Neurapraxia (Transient Quadriplegia)

Cervical cord neurapraxia, sometimes termed transient quadriplegia or quadriparesis, was described by Torg et al. in 1986 [32], and refers to transient functional disruption of the spinal cord. Players may experience numbness, tingling, and motor weakness below the level of the injury, which lasts from several minutes to 48 hours. In a study of high school and college football players, the risk of cervical cord neurapraxia increased from high school to collegiate levels of play, with an annual incidence of 0.17 per 100,000 and 2.05 per 100,000, respectively [9]. Torg previously reported an overall prevalence of 7 per 10,000 athletes [32]. Based on review of data between 1990 and 2001, Castro reported an incidence of incom-

plete neurological recovery of 0.4 per 100,000 and 1.2 per 100,000 high school and collegiate players, respectively [33].

The commonly accepted mechanism of such injuries was first described by Penning in 1962, and is thought to involve hyperextension of the cervical spine, which causes folding of the dura mater and ligamentum flavum, thickening of the spinal cord, and approximation of the posterior inferior edge of the vertebral body with the anterior superior aspect of the lamina of the inferior vertebra [33–35]. This causes a decrease in the diameter of the spinal canal, with transient compression of the spinal cord. Hyperflexion mechanisms, theoretically with similar transient narrowing of the anteroposterior (AP) diameter of the spinal canal, have also been reported, particularly in young athletes [36]. It seems logical that in patients with preexisting cervical spinal stenosis (congenital or acquired), this mechanism may be amplified, and this has been shown to be a risk factor for recurrence [37].

There is debate as to whether players who have had an episode of cervical cord neurapraxia should be allowed to return to play. Torg reported a 56% recurrence rate in football players who returned to play after an episode of cervical cord neurapraxia [37], but also found no evidence that these players were at increased risk of sustaining a permanent neurological deficit. This is discussed in detail below in the section on preexisting injuries.

Specific Thoracolumbar Injuries

Spondydololysis and Spondylolisthesis

Spondylolysis is a defect of the pars interarticularis that is seen most commonly in adolescents, and is the most common cause of low back pain in adolescent athletes [38]. The incidence in the general population is about 4.4% by age 6, and 6% by age 14 [39, 40], but athletes have a higher risk of developing spondylolysis, with an incidence as high as 14% [41, 42]. Spondylolysis in athletes is thought to result most commonly from chronic stress secondary to repetitive loading onto the pars interarticularis by the inferior articular facet of the adjacent superior vertebra [43]. This can occur in sports that require repetitive loading in the extended position, including diving, weight lifting, gymnastics, and football, particularly among linemen. Other risk factors include family history and preexisting developmental spine defects, such as spina bifida occulta [43]. The defect most commonly occurs at the level of L5 [3], and can progress to spondylolisthesis, a forward slippage of the vertebral body relative to the adjacent inferior vertebra, particularly when pars defects are bilateral.

Patients will often present with low back pain that is exacerbated with extension. They may have reproduction of pain with single leg extension, and are often found to have evidence of hamstring tightness [39, 43, 44]. The diagnosis can sometimes be made with plain radiographs alone. In cases where clinical suspicion is high but plain films are negative, computed tomography (CT), magnetic resonance imaging (MRI), or bone scan may be indicated. Figure 8.4 shows typical radiographic findings of spondylolysis that can be seen on MRI and CT scan. Patients with spondylolysis should be referred to a specialist for management, which may include bracing, and they should not return to play until they can participate without pain.

Disc and Apophyseal Ring Injury

Acute Disc Injury

Intervertebral disc injury is less common in adolescents than in adults, and when present may lack the classic radicular symptoms often seen in adults [45, 46]. Micheli and Wood [46] found that disc herniation accounted for 11% of cases of low back pain in a cohort of 100 adolescents, compared to 48% in a cohort of 100 adults. While the most common cause of disc herniation in adults is chronic degeneration, in children and adolescents trauma is the most common etiology [47]. Injury to the intervertebral discs may result from acute trauma or from repetitive microtrauma to the annulus fibrosis. It is thought that athletes such as football linemen are at increased risk of

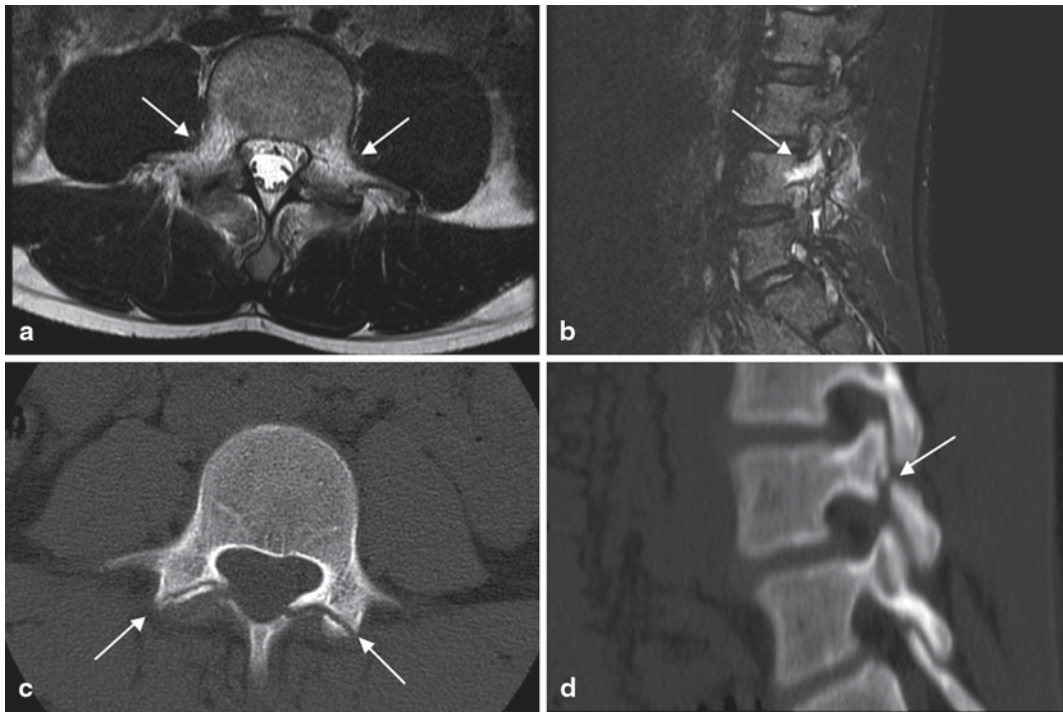

Fig. 8.4 Spondylolysis of the lumbar spine. MRI of the lumbar spine of a soccer player with bilateral spondylolysis (**a** and **b**) demonstrates increased T2 signal of the pars interarticularis (*arrows*). CT scan (**c** and **d**) shows clear bilateral pars defects of L4 (*arrows*)

such injuries due to the repetitive loading stresses on the lumbar spine from blocking [38]. Patients may present with low back pain or radicular pain, though adolescents with disc herniations may complain of buttock or hamstring pain rather than classic radicular pain. Patients will be more likely to have pain with flexion rather than extension, and there may be neurological deficits in a radicular distribution.

Plain radiographs may show narrowing of the disc space, but MRI is considered the gold standard for diagnosis. Many patients will improve with conservative management including rest, anti-inflammatories, muscle relaxants, and physical therapy. In some adolescents with acute disc herniations and associated neurological symptoms or severe pain, a steroid pulse can be considered. Patients should be referred for surgical evaluation in the presence of any severe or progressive radicular symptoms, or failure to respond to conservative management. Patients with disc injury who present acutely with neurological compromise, suggestive of spinal cord compression or cauda equina syndrome, require immediate imaging and surgical evaluation.

Apophyseal Ring Avulsion/Fracture

Ossification of the ring apophysis of the vertebral body occurs during adolescence, with fusion occurring around age 18. Before fusion occurs, the junction between the vertebral body and the apophyseal ring is weaker than the junction between the ring and the annulus fibrosis. As a result, forces on the spine can lead to an avulsion of the apophysis. These injuries are almost exclusively reported in adolescents and young adults, most commonly occurring at the L4–L5 level [43]. They may be associated with intervertebral disc herniations [48, 49]. An acute compressive force or repetitive hyperflexion and hyperextension movements with compressive loading, such as those that occur in football, are thought to place adolescents at increased risk of this injury [22, 43].

Patients may present with back pain with or without radicular signs and symptoms. Subtle changes may be revealed on plain radiographs, such as irregularities of the endplate and narrowing of the disc space, however CT is more sensitive for detecting avulsion fractures of the apophyseal ring. Adolescents with apophyseal ring fractures should be referred to a specialist, and may require surgical management [50].

Pre-existing Injuries and Developmental Abnormalities of the Spine

Developmental Abnormalities of the Spine

While there are many types and degrees of developmental spinal abnormalities, not all of them represent contraindications to participation in collision sports. For example, scoliosis is a common finding among adolescents, but does not necessarily preclude participation [51]. Some congenital abnormalities, however, are considered to be absolute contraindications to participation in collision sports which place the spine at risk of injury.

Anomalies of the Cervical Vertebrae

Atlantooccipital fusion and abnormalities of the odontoid (e.g., odontoid agenesis, odontoid hypoplasia, os odontoideum) are generally agreed to be absolute contraindications to participation in contact sports, due to the inherent instability of the cervical spine, which in the presence of a traumatic mechanism could result in catastrophic structural failure [52]. Patients with Down syndrome are known to have an increased laxity of the atlantoaxial ligaments, and it is recommended that their participation in collision sports be restricted if the distance between the anterior dens and the posterior arch of the atlas is greater than 4.5 mm, as this could predispose to subluxation [53]. Klippel–Feil anomaly refers to a congenital fusion of two or more cervical vertebrae. A multilevel fusion with limited range of motion is considered a contraindication to participation in contact sports [26, 54].

Cervical Stenosis and Cervical Cord Neurapraxia

Cervical stenosis has been shown by some authors to be a risk factor for permanent neurological injury in contact sports [55–57]. The diagnosis of cervical stenosis was previously made by measuring the canal diameter on a cervical plain film. However, this method is subject to error due to magnification artifact, which may be present to different degrees on different films. To correct for this, Pavlov and Torg [58] proposed evaluating the width of the canal by comparing it with the vertebral body width on the same film. The Torg ratio (or Pavlov ratio) is calculated by dividing the distance between the midpoint of the posterior aspect of the vertebral body to the nearest point on the spinolaminar line, by the sagittal diameter of the vertebral body (see Fig. 8.5). In a retrospective cohort study, Torg found that a ratio of 0.8 or less was 93 % sensitive for predicting an episode of cervical cord neurapraxia [59]. However, it was found to be a poor predictor of true functional spinal stenosis, with a positive predictive value of only 12 % [60].

Torg and colleagues found no association between cervical cord neurapraxia and permanent neurological injury in football players [61, 62]. However, a single case was subsequently reported of a player with a permanent quadriplegic injury who had a history of an episode of cervical cord neurapraxia, raising concern as to the safety of continued participation after such an injury [33, 63]. Given the sensitivity of the Torg ratio for predicting cervical cord neurapraxia, patients with an abnormal Torg ratio should be considered for further evaluation of functional spinal stenosis, such as MRI or CT myelography [28].

It is unclear whether the Torg ratio can be applied similarly to the younger adolescent athlete. There have been few studies to determine whether there are age-related differences in the association of the Torg ratio with the risk of transient

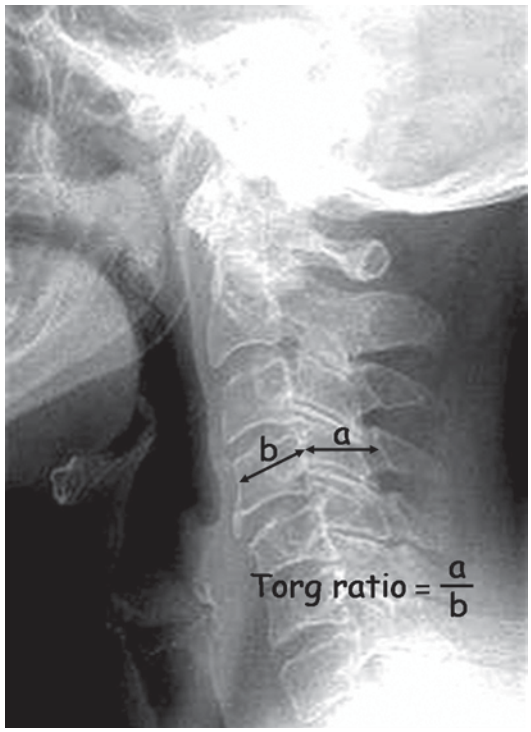

Fig. 8.5 Torg ratio. The distance between the midpoint of the posterior aspect of the vertebral body to the nearest point on the spinolaminar line (**a**) is divided by the sagittal diameter of the vertebral body (**b**)

or permanent neurological injury. Boockvar et al. [36] reported on 13 children aged 7–15 years with cervical cord neurapraxia. None of these children had an abnormal Torg ratio, and the authors concluded that neurapraxia in younger patients could be attributed to the greater mobility in the pediatric spine, rather than to developmental narrowing. It has not been directly investigated, however, whether the same cutoff value for the Torg ratio is a valid predictor of cervical spinal stenosis in these younger patients. The sagittal diameter of the cervical spinal canal reaches its adult size by about age 13 [33, 64], though the vertebral bodies continue to grow [65]. Given that the denominator in the Torg ratio is the vertebral body width, it is conceivable that as a young adolescent matures, the Torg ratio might decrease as the vertebral body grows with no corresponding change in canal diameter. The usefulness of the Torg ratio for predicting cervical spinal stenosis in young adolescents therefore remains unclear.

As no clearly agreed-upon guidelines exist to guide decisions regarding participation for asymptomatic athletes with functional cervical stenosis, decisions must be made on an individual basis.

Acquired Spinal Abnormalities

Spear Tackler's Spine

This entity was first described by Torg in 1993 based on an analysis of 15 athletes from the National Football Head and Neck Injury Registry; four of whom sustained permanent neurological injury, and 11 of whom had transient neurological symptoms [35]. All of these players were found to have a history of employing spear-tackling techniques, loss of the normal cervical lordosis on X-ray, evidence of post-traumatic changes on X-ray, and developmental narrowing of the cervical spine based on a Torg ratio of less than 0.8. Based on these findings, it was recommended that spear tackler's spine be considered an absolute contraindication to participation in collision sports, or any sport which places the athlete at risk of axial loading of the cervical spine [35].

Protective Equipment

No protective equipment has been definitively shown to prevent concussion or severe spinal injury in collision sports, though it may still be of benefit for preventing other injury types. For example, neck rolls or cowboy collars (molded collars that make contact with the chest, shoulders, and neck) used in football do limit cervical spine hyperextension and lateral flexion, and have been shown to decrease the incidence of stingers and burners [26, 66, 67]. While all protective equipment should be worn according to the standards in place for each sport, player and official education regarding appropriate training and safe contact techniques is more likely to prevent catastrophic injury. Ongoing efforts to further understand and ameliorate spinal injury mechanisms in collision sports are therefore of great importance.

Summary

Adolescents who participate in collision sports are at risk for several mechanisms of spinal injury, including hyperflexion, hyperextension, and rotational and axial loading forces. The majority of catastrophic spine injuries in collision sports result from hyperflexion or hyperextension of the cervical spine in the presence of an axial loading force. Rule changes and educational programs focused on limiting the exposure of athletes to these injury mechanisms have been shown to decrease the incidence of severe spinal injuries. A wide range of non-catastrophic injuries to the adolescent spine can also occur during participation in collision sports, and the physician who cares for adolescent athletes should be familiar with the differences in these injury patterns and presentations between adolescents and adults. Return-to-play decisions should be considered individually based on injury type, severity, and symptoms. The presence of underlying developmental or acquired abnormalities of the spine should be considered when making decisions about participation in collision sports.

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Spine Injuries in the Aesthetic Athlete

9

Bridget J. Quinn

Introduction

Aesthetic athletes are a unique combination of physical skill, athleticism, and artistry. They begin their training at a young age, and perform at a high level by adolescence. They are susceptible to spine injuries based on the physical and aesthetic demands of their art/sport and errors in technique and training. Dancers, gymnasts, and figure skaters place their spine in extreme ranges of motion, with repetitive, high-impact maneuvers. There is emphasis on spine hyperextension, flexibility, and axial loading associated with jumping and landing. Furthermore, these athletes are at risk for metabolic and nutritional disorders that can affect their rate of injury and quality of life [1–5].

Dance

Spine injuries constitute 7–18 % of all dance injuries and are found in 60–80 % of ballet and modern dancers [6]. They are typically overuse in nature but can be associated with trauma [6, 7]. The lumbar spine is the area most affected [8]. Male dancers are predisposed to spine issues that result from partnering, which usually involves lifting female dancers overhead. There are many contributors to back injuries, including the dancer's alignment, range of motion, strength and flexibility, technique, dance style, and training errors. Shoe wear, nutrition, and floor surface may also be implicated [5, 7].

The foundation of dance technique is a balanced core and turnout. Ballet dancers utilize five different positions of their feet. The goal is to achieve 180° of turnout along the longitudinal axis of the feet. This allows the dancer to execute complicated maneuvers while still facing the audience [9]. Turnout originates from the hip, knee, and foot. Ideally, dancers would achieve 70° of hip external rotation, 5° of tibia external rotation, and 15° of foot external rotation, for a total of 90° [9]. However, it has been shown that in most dancers the hip joint can only obtain about 60° of turnout [10, 11]. As a result, it is not uncommon to see dancers trying to enhance their turnout by performing various stretches of the hip. These dancers are targeting ligaments and muscle–tendon units. The bony alignment of a dancer may be influenced by cautious and specific training before the age of 11 but typically not thereafter [6, 12].

The dancer with decreased hip external rotation will compensate in order to achieve the ideal turnout. One common form of compensation is to increase lumbar lordosis (Fig. 9.1). Dancers also forcefully plant their foot and externally rotate the knee ("screwing the knee") and/or force the foot into pronation ("rolling in"). As a result of these compensations, the pelvis tilts anteriorly allowing for increased external rotation due to the

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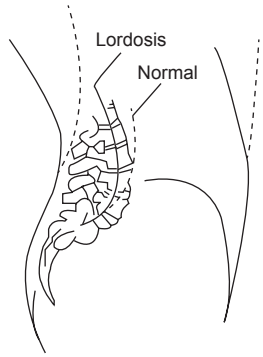

Fig. 9.1 Lumbar lordosis

deeper and more capacious posterosuperior acetabulum and relaxation of the iliofemoral ligament. While the dancer may have achieved enhanced turnout, the resultant lordotic posture increases the stress placed on the posterior aspects of the lumbar spine. Hyperlordosis originates not only from poor turnout technique but also from muscle imbalances in the lumbopelvic region, such as weak abdominals, tight lumbodorsal fascia, tight hip flexors, and weak gluteals.

Many dance movements involve lumbar "extension," including combré back (back arch), arabesque, and attitude effacée derrière. Keep in mind that these are maneuvers performed in turnout. Dancers rely heavily on their abdominal and paraspinal muscles in executing extension [7]. However, the hip extensors, including gluteus maximus and the hamstring, do help stabilize the hip in extension and may assist in decreasing lumbar lordosis [6, pp. 85–86]. The abdominal muscles include the rectus abdominus, external oblique, internal oblique, and transversus abdominus. The lordotic posture elongates and stretches this musculature. The lengthened muscles struggle to maintain strength. Eccentric contraction of the abdominal muscles provides control during return from a back arch, and isometric contractions provide stability during a lift. If the chronically elongated muscles have not been adequately strengthened, a vicious cycle ensues as the dancers are unable to activate the abdominal muscles to decrease their lordosis and appropriately execute their maneuvers.

Solomon [6] advocates use of the iliopsoas to decrease lordosis, both through eccentric and concentric strengthening and flexibility. An overly tight iliopsoas has also been implicated in increased lordosis. This muscle, referred to as the "dancer's" muscle, links the lumbar spine, pelvis, and hip (Fig. 9.2). It is responsible for hip flexion and possibly some hip external rotation. Overuse of the iliopsoas can tighten the muscle and result in anterior pelvic tilt.

A variety of other factors contribute to lordosis and spine injury. Dancers with benign joint hypermobility and genu recurvatum (or, "sway-back" of the knees) have increased lumbar lordosis. Growth is another risk factor for lumbar spine injury. As the bones accelerate in length, there is a tightening of the muscle–tendon units. This can result in tightened lumbodorsal fascia, hamstrings, and anterior hip muscles, resulting in increased lumbar lordosis. Heeled shoes have also been implicated in increased lordosis.

Gymnastics

A large review of female gymnasts found that 12.2% incurred injuries to the spine [13]. These athletes repetitively load the spine and perform high-impact maneuvers that contribute to significant axial loading and shear force on the spine. Gymnasts participate in multiple events. The women compete in the floor, vault, uneven bars, and beam. Their male counterparts compete in the floor, vault, horizontal bar, parallel bars, rings, and pommel horse. Rhythmic gymnasts utilize elements of ballet (with a focus on 180° of turnout), dance, and gymnastics, while manipulating clubs, hoops, balls, and ribbons. These athletes are judged for their aesthetic effect—leaps, balance, flexibility, and turns—in addition to the handling of their apparatus.

The floor routine in gymnastics involves maneuvers similar to dance, such as turns and leaps, but also includes aerial maneuvers, such as saltos or flips. The beam also incorporates dance elements, including leaps and jumps with the legs in a 180° split, as well as acrobatic skills and dismount. The vault includes a variety of styles,

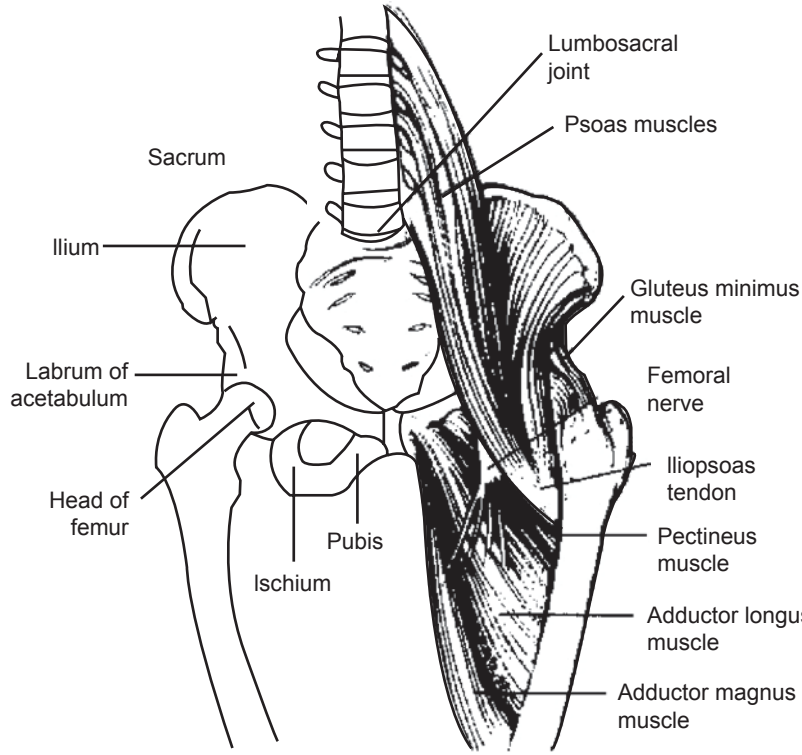

Fig. 9.2 The iliopsoas, also known as the "dancer's" muscle, links the lumbar spine, pelvis, and hip

such as handsprings and turns, with the end goal of landing with both feet planted. The dismount involves a transfer of force through the spine from the upper to the lower extremities, with the athlete often landing in a lordotic posture. There are also extremes of hyperextension and traction while being suspended from the rings or parallel bars and swinging on the high bar or uneven bars [14, 15]. These different events are responsible for a variety of stresses on the spine. Large vertical and lateral impact force to the spine occurs with the handspring, vault, and walkover. Hyperextension is produced with the back handspring and back walkover, and accentuated with bridging, front handsprings, and vaulting [14]. In addition to the mechanical stresses placed on the spine by these maneuvers, the athlete may have an imbalance in the core, pelvic, and lower extremity strength and flexibility that can contribute to spine injuries and pain. As was discussed in the dance section, any combination of a tight iliopsoas, weak lower abdominals, tight thoracolumbar fascia, and weak gluteals can result in anterior tilt of the pelvis and increased stress on the posterior elements on the spine.

Figure Skating

Figure skating is a combination of artistry and athleticism on ice. There are four disciplines in figure skating: singles skating, pairs skating, ice dance, and synchronized skating. These athletes perform in a stiff, leather-heeled boot, with a blade. In singles skating, an individual skater executes jumps, spins, and free skating. Pairs skating is similar to a pas de deux, or partnered dance, in ballet. A male and female skater perform jumps and spins both individually and in tandem, and the male frequently lifts his partner overhead. Ice dance has a strong emphasis on intricate footwork, speed, carriage, and rhythm. Synchronized skating involves a team of 8–24 skaters moving simultaneously. The sport has increased in technical difficulty to include complicated maneuvers like quadruple jumps by men

and triple axels by women. Similar to the other aesthetic sports, training begins at a young age, and figure skaters often reach their competitive peak by their teens to early 20s. They train both on and off the ice several hours per day, often 5–7 days per week, throughout the year [16].

While many of the same principles of movement in dance apply to figure skating, one of the major differences is in footwear. In ballet, both men and women wear soft-soled slippers, but women dance mainly in pointe shoes. Gymnasts compete barefooted. As previously noted, figure skaters use stiff skater's boots with a heel. The increased rigidity of the boot limits the range of motion at the ankle and knee. To compensate, the skater will increase hip flexion and back extension, often with hyperextension at the knees [16]. Furthermore, skaters often exhibit the same iliopsoas tightness, tight thoracolumbar fascia, anterior pelvic tilt, and weak gluteals as their dance and gymnast counterparts. They often perform repetitive unilateral loads during jump landings. They also perform propulsion, spin, and lift movements involving repetitive axial and torsional loading with lumbar hyperextension. There is a large unilateral shear stress placed on the sacroiliac joint due to repetitive jump landings on one leg and missed landings [17].

Patterns of Injury and Their Rehabilitation

The lumbar spine is the most common site for injury in the aesthetic athlete. As discussed above, dancers, gymnasts, and figure skaters place their spine in extreme range of motion both repetitively and during high-impact maneuvers. Lumbar lordosis also contributes to many spine injuries in the aesthetic athlete. This can emanate from poor technique and compensation for inadequate turnout in dance or restricted ankle and knee movement in figure skating. Furthermore, weak lower abdominals, tight iliopsoas and thoracolumbar fascia, and weak gluteals may play a major role, especially during periods of growth. This lordotic or hyperextended alignment places a large stress on the posterior elements of the spine, including the pars interarticularis, pedicles, and facet joints. Defects of the pars interarticularis, also known as spondylolysis, are commonly associated with the aesthetic athlete (Fig. 9.3). The incidence of spondylolysis in the general population has been reported to be 3–6%, with a higher incidence in the young athletic population, especially in dancers, gymnasts, and figure skaters [13, 18–22].

Spondylolisthesis is a forward displacement of one vertebral body over the next. The most common location for this injury is at L5, with L4 being the next most common [23]. The athlete presents with extension-based back pain: pain with arabesque in the dancer, back handspring in the gymnast, and layback spin in the figure skater. Clinical examination reveals limitations in forward flexion and pain with hyperextension of the spine. Stork testing, or unilateral hyperextension of the spine with the athlete standing on one foot, strongly indicates a pars fracture on the ipsilateral side (Fig. 9.4) [7, 23]. However, this maneuver often places stress on structures other than pars, so the overall clinical picture must be assessed. The clinician may also be able to reproduce pain with torsional rotation (Fig. 9.4).

Diagnostic imaging includes plain radiography, nuclear bone scan, magnetic resonance imaging (MRI), and/or computed tomography (CT) scan. The subtle changes of stress to the pars interarticularis may not be appreciated on plain film radiographs; thus, their interpretation should be made with caution. Lateral oblique views of the lumbar spine are used to detect lucent lesions in the region of the pars interarticularis. This has the appearance of a collar on the "Scotty dog" (Fig. 9.5). The orientation of the plane of the defect may limit its visualization on plain film radiography, and subtle lesions of stress change without cortical disruption may similarly be missed. Radionuclide imaging using single photon emission computed tomography (SPECT) can detect pars lesions before they appear on plain radiography or in the case of false negative plain films (Fig. 9.6). Furthermore, when pars lesions are detected on plain radiography, SPECT can be useful in differentiating symptomatic from asymptomatic lesions by uptake in the defect. CT isolated to the affected level is used as a supplementary

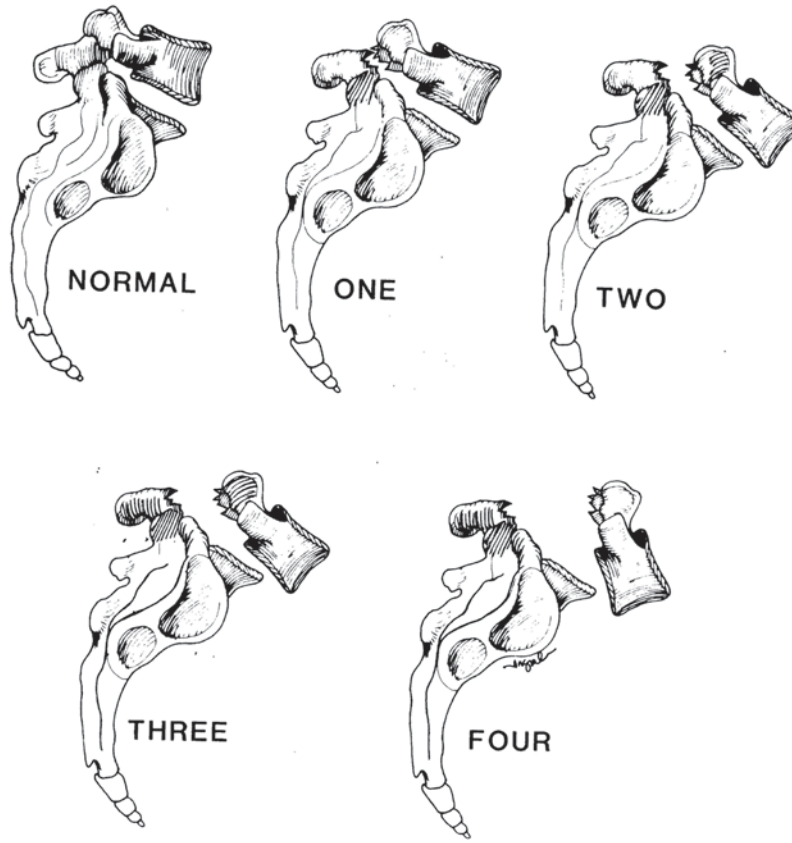

Fig. 9.3 Spondylolysis and spondylolisthesis. Spondylolysis is a defect of the pars interarticularis. Progression of the defect can lead to spondylolisthesis, a forward displacement of one vertebral body over the next

test in evaluating the anatomic and physiologic state of the bone, as well as healing (Fig. 9.7). MRI offers several advantages in that it involves no ionizing radiation and allows visualization of structures within the spine. MRI is not as sensitive at detecting pars defects as SPECT bone scan [24], but has been enhanced by increasing slice thickness up to 3 mm on T1 and 4 mm on T2 sequences (Fig. 9.8) [23].

Treatment approaches vary with regard to bracing, ranging from the rigid Boston Overlapping Brace, to a soft brace, to no bracing, but should involve relative rest and avoidance of aggravating activity. A rehabilitation program involving abdominal muscle strengthening, hip flexor and hamstring flexibility, and antilordotic exercise should be initiated under the guidance of a physiotherapist. Activity begins in the brace and is gradually increased until all activities can be resumed without pain. A strong emphasis on technique and decreasing lordosis is also initiated.

Sacroiliac joint (SIJ) dysfunction is a common source of pain localized where the sacrum and ilium meet. The pelvis is a central base between the spine and the lower extremities. This base has three bones articulating: the sacrum and ilium at the SIJ, lumbar spine and sacrum via the lumbar disc and L5–S1 facets, and the femoroacetabular joint. Repetitive and/or high-energy loads with asymmetric force transmission (as seen in asymmetric landings) is a common source of injury to the SIJ. Thoracolumbar lumbar fascia transfers load from the lower extremity through the pelvis, lumbar spine, and abdominal muscles. Furthermore, the muscles around the lumbar spine, pelvis, and hip affect motion at the SIJ [25]. Patients report low back pain and pain over the sacral sulcus with transitional movements, weight bearing on the affected side, and unsupported sitting. Figure skaters frequently land one leg (the right leg in particular) in a stiff boot with their pelvis anteriorly tilted. Young dancers are often

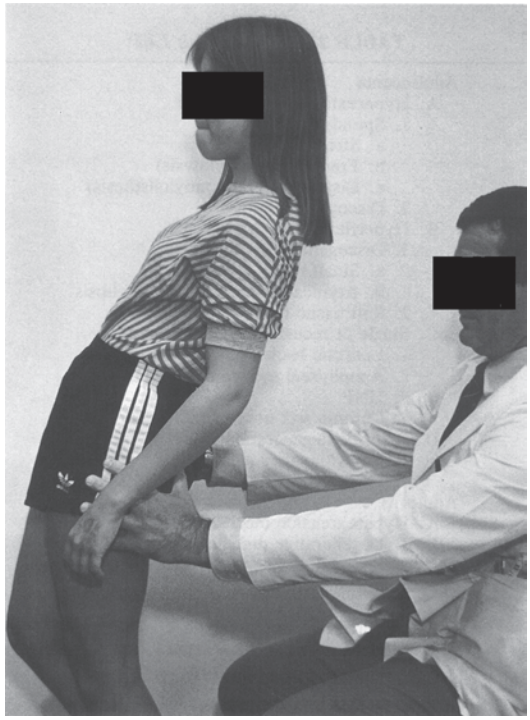

Fig. 9.4 Stork testing, or unilateral hyperextension of the spine with the athlete standing on one foot, strongly indicates a pars fracture on the ipsilateral side

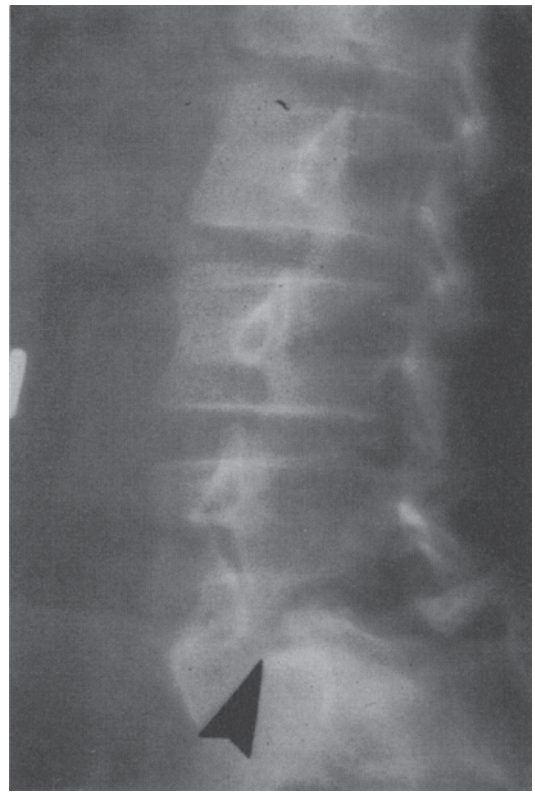

Fig. 9.5 Lateral oblique views of the lumbar spine are used to detect lucent lesions in the region of the pars interarticularis. This has the appearance of a collar on the "Scotty dog"

cued to decrease their lordosis by "tucking" their pelvis under and drawing in through their core. In an effort to perform, the posterior tilt dancers will clench their gluteals with their feet planted, creating a posteriorly tilted pelvis and forward locked ilium, increasing the strain across the SIJ. Gymnasts also create shear across the SIJ with high-impact asymmetric landings. The clinician should investigate functional or anatomic leg length discrepancies in these athletes. Pathology of the lumbar spine can change the mechanics of that anatomic unit and increase stress to the SIJ [20]. Stress fractures can also occur at this joint and can present with symptoms similar to SIJ dysfunction.

Examination of these athletes includes plain film radiographs to evaluate for sacroilitis and sclerosis, as well as MRI to investigate inflammatory arthropathy and stress fractures [26]. Supine examination maneuvers, including the flexion/abduction/external rotation (FABER)/Patrick's test, posterior shear, Gaenslens, and pelvic compression, place stress and shear force across the SIJ. In the Gaenslens test, the patient is supine with the hip joint maximally flexed on one side and extended on the other. The examiner simultaneously applies slight pressure to and increases flexion of the extended hip. Further diagnostics may include intra-articular SIJ blocks to look for SI dysfunction as a pain generator [26]. Treatment involves peripelvic strengthening and stabilization, educating the dancer on appropriate technique, and addressing leg length discrepancy [26].

Posterior element overuse syndrome presents similarly to spondylolysis and SIJ pain in the aesthetic athlete. It stems from repetitive extension and rotation of the spine. Tight thoracolumbar fascia accentuates lordosis and places pressure on the posterior elements, such as the facet joint and its joint capsule. As with spondylolysis,

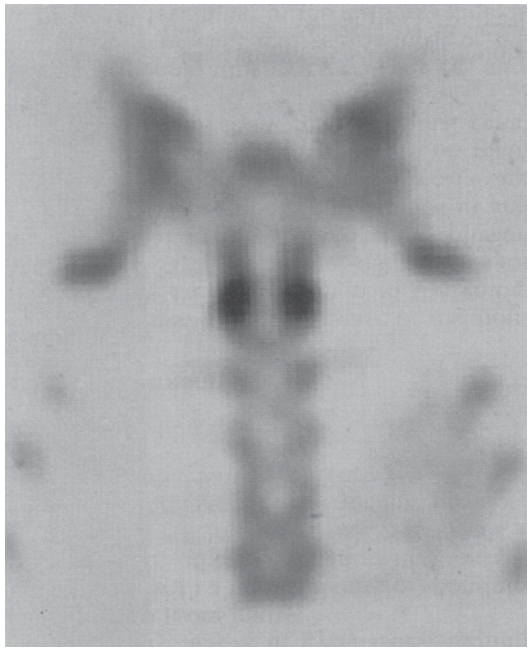

Fig. 9.6 SPECT bone scan showing increased uptake at the L4 pars region indicative of spondylolysis

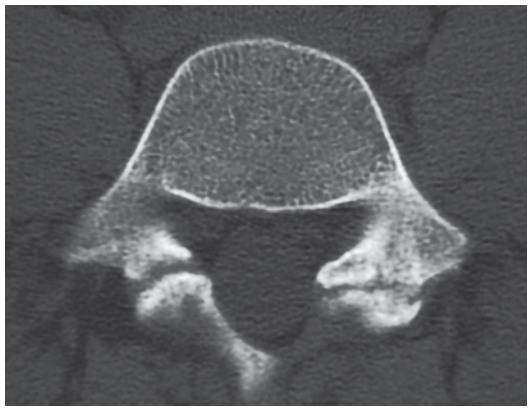

Fig. 9.7 Axial CT scan depicting defects through the pars interarticularis indicating spondylolysis

pain is reproduced clinically with extension and rotation. Imaging to rule out spondylolysis is prudent. Facet arthrosis can be detected on plain films, MRI, and CT scan. Treatment involves abdominal strengthening, antilordotic exercises, and hamstring and thoracolumbar stretching. Antilordotic bracing can provide short-term relief. Relative rest and avoidance of extension-based

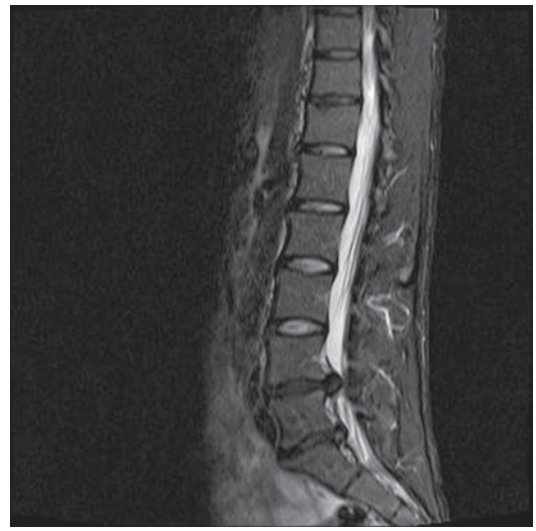

Fig. 9.8 T2-weighted MRI showing L4–5 and L5–S1 disc protrusion

maneuvers is also recommended. As with all injuries, the aesthetic athlete should be evaluated for incorrect technique and alignment.

The injuries discussed thus far have focused predominantly on extension-based pathology. The aesthetic athlete also utilizes flexion-based maneuvers. Therefore, injuries to the anterior and middle aspects of the thoracolumbar spine should be considered. These include Schmorl's nodes, apophyseal ring abnormalities, and vertebral wedging, as seen in Schuermann's kyphosis of the thoracic spine. The combination of torsion and axial loading also induces stress on the lumbar discs [27].

Activities that involve repetitive flexion can lead to Schuermann's kyphosis of the thoracic spine and atypical Schuermann's at the thoracolumbar juncture. Athletes with atypical Schuermann's may present with more of a flatback appearance. These have been seen in aesthetic sports such as gymnastics [28]. This is the result of anterior vertebral body endplate wedging. Postural kyphosis is reversible with overhead arm extension, but Schuermann's is not. Schuermann's is defined as a kyphotic angle $>45°$ with 5° or more of wedging in at least three adjacent vertebrae. Treatment involves upper trunk and postural exercises. Bracing for Schuermann's

with a thoraco-lumbar-sacral orthosis (TLSO) brace begins at kyphotic curves of > 50° in individuals with continued potential for spinal growth [29].

An atypical form of Schuerman's, known as Lumbar Schuermann's, is seen in sports that involve rapid flexion and extension, such as gymnastics [24]. There are endplate and disc changes at the thoracolumbar juncture. These athletes present with back pain and a flat back due to decreased thoracic kyphosis and lumbar lordosis and tight thoracolumbar fascia [30]. Treatment involves core stabilization and stretching of the thoracolumbar fascia. Bracing in 15° of lordosis may also be considered [24].

Discogenic back pain is less common in the younger aesthetic athlete; prevalence increases with age. The annulus fibrosus can be stretched and torn with rotational load. The lower levels receive the greatest repetitive axial loads. This can lead to acute and chronic changes in the discs. With the protective shock absorption, offered by the discs compromised, the facet joints are more exposed to strain and may develop arthrosis or degenerative changes. In ballet, partnering and lifts place a significant axial load on the spine. Due to specific demands of the choreography or incorrect technique, male dancers may initiate and execute lifts with a lordotic back. Furthermore, the dancer is often leaning forward or turning during the lift [7]. Gymnasts also demonstrate disc changes both symptomatic and asymptomatic [31]. Athletes often present with flexion-based pain, sitting intolerance, and radiating pain with nerve root inflammation. There is often associated muscle back spasm, hamstring tightness, and occasionally deep buttock pain. Physical examination findings are variable, including limitations in flexion, positive straight leg raise sign (Lasègue test), and/or decreased reflex on the affected side. Plain film radiographs of the spine are obtained to rule out bony injury. MRI is reserved for progressive or refractory symptoms, to evaluate the degree of disc damage and look for nerve root impingement. Conservative treatment results in improvement in approximately 90% of patients. Physical therapy is based on an extension-based stabilization program. Pain can be managed via nonsteroidal anti-inflammatories (NSAIDs), muscle relaxers, oral steroids, and, when necessary, epidural corticosteroid injections. Surgery is reserved for cauda equina syndrome, progressive neurologic deficit, and symptoms refractory to conservative management. Return to art/sport is initiated once full pain-free range of motion and strength has been achieved and the athlete has been progressed through dance/sport-specific activities. In pairs skating and dance, proper lifting technique should be emphasized.

Aesthetic athletes are also at risk for mechanical back pain. This occurs when no definite anatomic cause can be elucidated, to include muscle strains and ligament sprains of the cervical, thoracic, and lumbar spine. These can occur acutely or chronically with repetitive maneuvers, and are commonly associated with lordotic posture. Mechanical back pain is a diagnosis of exclusion once other specific causes of pain have been ruled out. Treatment focuses on correct technique and physical therapy [6].

The prevalence of scoliosis is increased in both ballet dancers and rhythmic gymnasts [32, 33]. Warren et al. proposes a delay in menarche and prolonged intervals of amenorrhea as risk factors due to hypoestrogenism [32]. Tanchev et al. also implicate delayed maturity, as well as asymmetric spinal loading and joint laxity [33].

The clinician should always have a high index of suspicion for atraumatic causes of back pain. These include infections, tumors, and inflammatory arthropathies. Intra-abdominal pathology can also refer to the spine.

Summary

This chapter has focused on dance, gymnastics, and figure skating. All three disciplines begin their training at a young age. Each discipline requires maneuvers that involve repetitive application of extreme ranges of spine motion, often with an emphasis on hyperextension. Common technical faults include lordosis due to weak lower abdominals, tight thoracolumbar fascia, weak gluteals, and faulty technique. A basic knowledge of the requirements of these athletes is essential to understanding the possible pathologic contributions to their back pain. A thorough

investigation is often warranted in these athletes prior to making the diagnosis of mechanical back pain. Treatment involves sport-specific physical therapy focused on the athlete's areas of weakness, as well as correction of their technical faults. Return to sport is initiated only after pain-free sport-specific range of motion is achieved. While micro- and macro-trauma are the biggest risk for spine injuries, other factors to take into account are dancer fatigue resulting from long hours of training, malalignment (including leg length discrepancy), footwear, growth, nutrition, and hormonal balance. In order to treat these unique athletes appropriately, the proper diagnosis must be made [34].

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Swimming and the Spine

10

Erika B. Persson and Merrilee Zetaruk

Introduction

Many children and adolescents participate in swimming, either at a recreational or at a competitive level. A few select youth reach excellence and represent their country at international competitions such as the Olympic Games. Nearly 300,000 high school athletes participated in swimming in the USA during the 2011–2012 season, ranking swimming 8 and 10 for female and male sport participation [1]. In Canada, one in four children is involved in swimming [2], with even higher rates noted in the USA, Australia, and the UK [3–6].

Swimming as a Sport

Swimming venues vary from indoor and outdoor pools to open water such as lakes, rivers, oceans, and ponds [7]. Competitive or club swimming usually begins at age 8 [8] and involves four main stroke types: freestyle or crawl, backstroke, butterfly, and breaststroke. Swimmers can be single-stroke specialists or participate in multiple strokes including the medley where all four strokes are performed [7, 8]. Within competitive swimming, athletes are divided by gender into age cohorts with events in sprint, middle, and long distances [7, 8]. Many highly competitive club swimmers swim daily for up to 20 h per week [9], including pool and dry land training (strength and conditioning). They may swim distances of 10–20 km per day [7, 10]. Traditional training regimens involve pre-swim stretching, a warm up (dry land and pool), training or competition session in the pool, and cool down (dry land and pool) [7]. Most stroke training is in freestyle despite the athlete's stroke specialty [11].

Swimming Biomechanical Risk Factors for Spinal Injury

Swimming requires significant power and endurance and has been thought of as both an upper-extremity- and spine-intensive sport [12]. Of the four strokes, three (freestyle, backstroke, and butterfly) are similar in the arm positions and can be broken into two main phases: pull and recovery [13–15]. During the pull phase which occurs under water, the arm(s) move from an abducted and externally rotated position to an adducted and internally rotated position [13–15]. In the recovery phase, the arm(s) move out of the water to a fully extended position in abduction and external rotation, with assistance of the body roll [13–15]. The body position and spinal motion differ among the strokes with freestyle and

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backstroke characterized by rolling, while butterfly and breaststroke have an undulating rhythmic motion. The roll of the torso in freestyle and backstroke is created by the paraspinal and abdominal muscles to provide much needed power via increased force generation and reduced drag [13–15]. The body roll should be smooth with the shoulders and hips moving as one unit [12], which allows for reduced segmental rotation and torque forces in individual spinal segments [12]. In the butterfly stroke, swimmers should have an effective spinal undulation motion with repetitive and rhythmic flexion and extension of the lumbar spine [12]. There has been a shift in breaststroke body movement with a more undulating motion and less gliding and body roll motions [12, 14, 16, 17]. This change has increased the relative lumbar extension and stress to the facet joint and thus increased the risk of spinal injury and pain. The various kicks are utilized to maximize body roll and resultant power and propulsion[13, 14]. All swimmers, regardless of stroke, need to maintain the correct body position in the water to ensure a streamlined swim, to maintain balance, to correct stroke technique, and to reduce drag [7, 15, 18, 19]. If there are any technique errors within the stroke, including arm position, body roll or undulation movement, kick, and head position for breath, these can predispose to spinal injury.

Injury in Youth Sport

Rates of injury sustained by athletes in various sports are available, although data for younger athletes are often limited [20]. This is partially due to a lack of formalized monitoring programs at various levels of participation [20]. Due to the retrospective nature of most studies, along with a lack of calculations of athlete exposures (AEs), much of the literature describing sport injury rates among youth has been of poor quality [21]. This is particularly true for swimming where studies have involved low athlete numbers [21]. Spinal involvement in athletic injuries varies by sport from a low of 1% in basketball to 43% in gymnastics (Table 10.1) [21].

Table 10.1 Spinal injury rates by sport. (Adapted from [21])

Sport	Spinal injury rates (% of all injuries)
Track and field	3.2–16.7
Baseball	2.0–40
Basketball	1.3–14.6
Gymnastics	7.5–43.6
Soccer	3.0–33
Swimming	3.0–37

Overview of Swimming Injuries

Injuries in swimming commonly affect the shoulder, knee, or spine [21–27]. The shoulder is most frequently injured with 12–87% of athletes affected at some point in their season, followed by the knee with 6–28% affected. Reported rates of low back injuries among swimmers of all ages range from 4 to 37% [11, 12, 21, 22]. Specific strokes are associated with particular injury patterns such as the medial knee in breaststroke and the lumbar spine in breaststroke and butterfly [21]. More injuries are seen in non-freestyle swimmers [9, 11, 27]. Swimming injuries are more commonly chronic and overuse rather than acute [7, 21], occurring in practice rather than in competition settings [21, 27]. Increasing rates of overuse injuries and morbidity are being seen in pediatric swimmers due to more intensive training in an organized team setting [28]. Increased training volume and intensity accompanied by an increased competitive level and the growth spurt creates a period of risk for the adolescent swimmer [7, 21]. Other risk factors for injury in swimmers include incorrect stroke techniques and repetitive motions during the stroke [7, 13, 17]. Injury prevention can include attention to periodization, utilization of correct stroke technique, and a gradual increase in training intensity and volume [7].

Table 10.2 Risk factors for spinal injury in swimmers

Risk factor for spinal injury	Mechanism of injury
Sport-specific biomechanical forces	Repetitive microtrauma with shearing, rotatory, tensile, and compressive forces applied to the spine from increased training volume or repetition of a single movement type
Growth	Reduced resistance of vertebral growth plates to forces compared to ligament or bone
	Reduced flexibility and increased joint tightness during periods of rapid growth
Spinal alignment	Altered force transfer from legs though the pelvis and spine due to structural abnormalities
Muscular weakness	Weakness of the abdominals and tight hip flexors that predispose to increased lumbar lordosis and greater spinal compression
Training issues	Changes in stroke technique, increased swimming volume and associated fatigue, incorrect weight-training technique, use of equipment (e.g., fins, kickboards, and pull buoys), and sudden changes in training
Coexisting injury	Altered motions at the cervicothoracic and thoracolumbar zones due to injury at a peripheral joint (e.g., shoulder, hip, and knee)

Spinal Injury in Swimming: Specific Injuries

Low back pain is very common in swimmers, with 16% of all swimmers complaining of pain in the lower back over the course of a season [8]. Swimmers who train in breaststroke and butterfly often suffer more spinal and back injuries compared to those who do not [21]. Injury to the spine ranges from 3 to 37% of all swimming injuries [21] and has been reported to affect 17% of adolescent swimmers [26]. Risk of injury to the spine is increased due to the specific body positions an athlete must adopt in order to create maximal effective forces [13]. In addition, rule changes to allow an underwater component of each stroke up to the first 15 m of the race and a change in the breaststroke to allow the head to submerge and the heels to break the water surface have increased the forces applied to the spine and thus have increased risk of injury [13, 16]. Specific risk factors for spinal injury in swimmers include overuse, growth, spinal alignment abnormalities, muscular weakness, training issues, and coexisting injury (Table 10.2) [8, 9, 11–13, 28]. Spinal tissues that are at risk of injury in the young athlete include the paraspinal musculature, myofascial tissues, vertebral growth plates, intervertebral discs, lumbosacral and sacroiliac ligaments, and the facet joints [23].

Training-specific aspects of the history that are important when assessing a swimmer who presents with a spinal injury include years of training, weekly practice hours including dry land, breath side, known technique errors, recent stroke changes or additions, and training volume changes [10]. Injury prevention programs have focused on improving core strength and scapular stabilization as well as correcting stroke errors [22].

Spondylolysis and Spondylolisthesis

Spondylolysis is a common cause of lower back pain in the young swimmer with up to 10% of swimmers developing this injury to the pars interarticularis; however, up to a third are asymptomatic [21]. The injury is most commonly found at L5 and can be uni- or bilateral in nature and may have an associated anterior slip (spondylolisthesis) [28]. Increased stress to the pars interarticularis from butterfly and breaststroke, along with repetitive lumbar extensions during the dolphin kick and flip turns, set the stage for spondylolysis [9, 12, 22]. Symptoms are often insidious in onset with focal lumbar discomfort that is aggravated by extension. They occasionally radiate to the buttock and posterior thigh [9, 17, 28]. Rarely, athletes may present only with a sense of hamstring tightness and increased anterior pelvic tilt [9, 17]. Physical examination findings include pain with lumbar extension and single leg extension test, tight hamstrings, and tenderness over the affected side. In the case of spondylolisthesis,

there may be a palpable step off at the level of the lesion [28]. The diagnostic test of choice after completing plain films of the spine is a single-photon emission computed tomography (SPECT) bone scan [28]. Treatment is usually nonsurgical except in the cases of high-grade spondylolisthesis or failure of conservative measures [28]. Strokes associated with spinal extension such as butterfly should be avoided until symptoms have settled. Rehabilitation is designed to improve posture and strengthen core muscles and hip abductors [28]. Some experts advocate bracing in the treatment of spondylolysis [28].

Intervertebral Disc Pathology

Disc degeneration and pathology is more prevalent in swimmers, especially those who have increased intensity and volume of training compared to their peers [25, 26, 29]. All strokes place the discs under some degree of stress [26] due to repetitive flexion, extension, and rotational movements that occur during the stroke [28, 29]. When compared to nonswimmers, youth who swim at high volumes have a greater proportion of degenerative disc changes at multiple levels [11]. The adolescent growth spurt is a high-risk period for developing changes to the disc as a result of swimming [28]. The mechanism of adolescent disc changes involves injury to the cartilaginous end plates and an associated avulsion with the attached disc annulus or herniation of the disc into the vertebral body at the growth plate [28]. Swimmers can also experience nerve root impingement due to repetitive hyperextensions of the lumbar spine in freestyle and butterfly strokes leading to narrowing of the neural foramina and the spinal canal [12]. The most commonly affected levels are L4–5 and L5–S1 [25, 26, 28, 29]. The presenting symptoms often include focal back discomfort, pain in the upper thigh or buttocks, and reduced hamstring flexibility [28]. It is rare for the young swimmer to experience true radicular symptoms [28]. Physical examination findings include pain with spinal flexion and valsalva, a positive straight leg raise, and occasionally a positional scoliosis away from the lesion [28]. The diagnostic investigation of choice is magnetic resonance imaging (MRI), which can identify disc pathology and associated nerve root impingement [28]. In the young swimmer, treatment is primarily nonsurgical, including avoidance of aggravating activities ("relative rest"), physiotherapy, use of nonsteroidal anti-inflammatories, and a gradual return to sport [28]. Although rarely required in the young swimmer with a disc injury, steroid injections and rigid bracing may be considered [28].

Scoliosis and Kyphosis

Scoliosis is a torsion of the spine associated with a lateral curvature of greater than 10° [30]. Structural scoliosis has associated fixed changes and rotatory vertebral bony abnormalities. Functional scoliosis is due to muscular imbalances [12, 13, 30] and postural changes that can be resolved with bending to the opposite side or lying down [30]. Adolescent swimmers have an increased incidence of both functional and structural scoliosis. Studies have found a rate of idiopathic scoliosis of 6.9% in swimmers compared to 2–3% in the general population [13]. Of swimmers with functional curves, 100% of the curves are directed to the dominant side and are thought to be the result of increased peak forces on the dominant arm side pulling the spine [12, 13, 30]. To address this issue and to develop more symmetrical swimmers, alterations in training methods have been made. These include alternation of breath sides and creation of even arm strokes and trunk rotations [12, 13, 30].

A number of young swimmers who have presented with thoracic back discomfort related to the butterfly stroke have been found to have underlying Scheuermann's kyphosis [22, 23, 31]. Scheuermann's kyphosis is a thoracic kyphosis in which three or more consecutive vertebral segments are anteriorly wedged 5° or more [32]. Other radiographic findings include Schmorl's nodes, caused by herniation of the intervertebral disc through the vertebral end plates and irregularity of the vertebral end plates [33, 34]. It is unclear if the forceful contraction of the anterior

chest wall and abdominal muscles during the butterfly stroke contributes to the deformity or if the repetitive muscular contractions are an aggravating factor [28]. Young swimmers with Scheuermann's kyphosis will often complain of back fatigue at the end of practice or discomfort in the upper back with prolonged sitting or walking [22, 23, 31]. The kyphosis is often evident on physical examination and confirmed with plain film radiographs. Treatment is nonsurgical involving bracing, physiotherapy to improve posture and upper back flexibility and strength, and avoidance of the butterfly stroke [22, 23, 31]. Any young swimmer with reports of thoracic back pain and a clinical kyphosis should have imaging done to look for early evidence of this condition [22].

Cervical Spine Trauma and Overuse

Trauma to the cervical spine is often due to diving into unsafe water or into a shallow pool, but swimmers can also suffer trauma due to direct collision with the wall in the backstroke or with a steep angle entry dive into a relatively shallow pool [7, 10, 22]. Risk mitigation during block starts and backstroke include a minimal depth of 5 ft for all pools using block starts as well as overhead warning flags at 5 m from the pool wall [7, 22, 35]. Coaches teach safe block entry dives with the shallow dive technique [22, 35] and avoidance of the pike dive entry [16]. Swimmers who have a known cervical instability should not be permitted to dive into the pool [8]. In addition, all pools should have appropriate equipment and appropriately trained personnel who can address potential c-spine injuries sustained in the pool area [35].

Cervical spine overuse injuries are less common than lumbar spine injuries, but are encountered in swimmers who have poor breathing techniques such as unilateral or head forward postures [13]. Specific strokes also predispose to cervical spine overuse. The backstroke requires significant head stability in order to reduce drag and allow for the breath. This often causes overuse of the cervical spinal muscles and excessive flexion of the cervical spine [13]. In the breaststroke and butterfly, if the breath is not timed correctly, the athlete often will have hyperextension of the cervical spine to breathe and this can lead to cervical facet joint compression [13, 14].

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Spinal Injuries in Combat Sports

11

Merrilee Zetaruk

Introduction

Combat sports attract participants of all ages, with more than 6 million children involved in martial arts in the USA [1] and more than 18,000 youths under 19 years of age registered with USA Boxing [2]. Some styles such as judo have approximately 75% representation by children under 15 years of age, while youth participation in other martial arts such as karate has doubled over the past decade [3].

Combat sports include traditional martial arts with diverse cultural origins but a shared philosophical approach to training (e.g., Shotokan karate, aikido, wushu (kung-fu), traditional taekwondo) as well as disciplines that focus primarily on sport or competition (e.g., Olympic-style taekwondo, boxing, kickboxing, judo.) Combat sports can be grouped according to the level of contact (noncontact, light contact, or full contact) or by the type of techniques (grappling/throwing versus striking/blocking). Judo, aikido, and wrestling exemplify the former, while karate, wushu, and taekwondo focus primarily on strikes and blocks. Some combat sports (e.g., aikido, wushu) also utilize weapons in training or competition. This diversity among combat sports accounts for the different rates and types of injuries sustained.

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Injury Rates

Complaints of low-back pain among adolescents are on the rise, with a greater likelihood of associated structural pathology identified in younger athletes than among adults [4, 5]. Among highly competitive athletes, as well as those with high demands on their lumbar spine such as wrestlers, judoka (judo athletes), and gymnasts, the risk of low-back injury is even greater [4–6]. Incidence of low-back pain in youth sports ranges from 10 to 15% [4, 7]. These active youth are just over one and a half times as likely to report a history of low-back pain compared with nonsporting youth, while young judoka are more than twice as likely as their nonsporting peers to report having ever experienced low-back pain [8].

Injuries to the cervical spine are a concern in combat sports, particularly among participants of grappling and throwing sports (wrestling, judo, aikido). In a 2005 study by Zetaruk et al, comparing injury rates in different martial arts, 32% of aikidoka (aikido athletes) reported injuries to the head and neck region in the preceding year [9], with no concussions reported.

Combat Sports

Aikido

Aikido is a grappling style that shares its origins with judo [10]. Very limited data are available in the literature regarding spine injuries in aikido,

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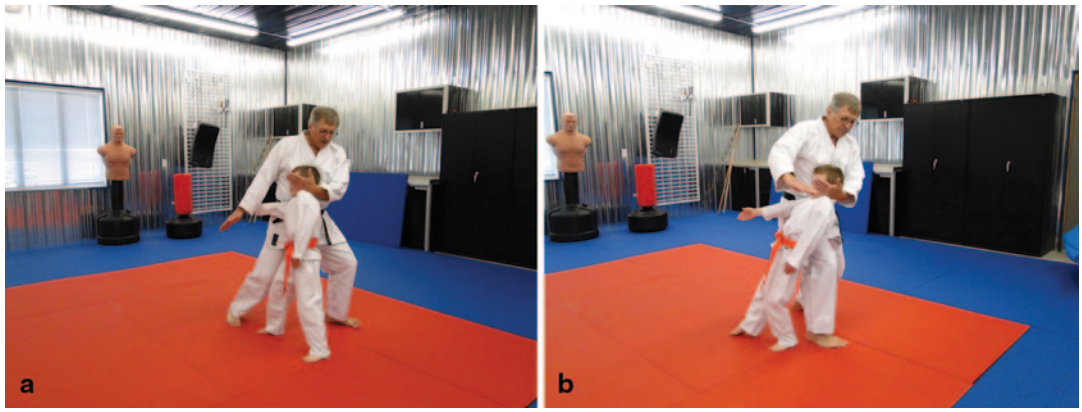

Fig. 11.1 **a, b** *Irimi nage,* or entering throw, requires careful practice and attention to proper technique to avoid injury to the cervical spine when using the head/neck as a control

and even less that pertains specifically to young athletes. Throws in aikido often involve the wrist, elbow, and shoulder to execute correctly [10]. These throws take advantage of the opponent's momentum [10]. Some throws, such as *irimi nage* (entering throw), are performed by controlling the head (Fig. 11.1a, b). In Kurland's study of aikido injuries [10], the mechanism of injury for the single case of cervical strain was the thrower holding the subject's head too long during a throw. Injury to the lumbar discs may occur in circular throws when the receiver of the throw (*uke*) does not perform with good technique [11].

Judo

Among young martial artists presenting to emergency departments, judoka (practitioners of judo) are more likely to sustain neck injuries than karateka (karate athletes) or taekwondo athletes [12]. Judoka are more likely to sustain injury from being thrown/flipped, which may account for the greater risk of neck injury [12] particularly if techniques are poorly executed by *uke* or *tori*(the one who performs the throw) [13]. Minor sprains and strains of the cervical region are common in judo [14]. Though rare, serious cervical spine injury, such as unilateral C5–C6 facet fracture dislocation and discoligamental rupture, has been reported in judo and jujitsu [13, 15]. In martial arts, the headlock keeps the opponent's neck immobile and under traction. The inexperienced martial artist may attempt to break free from a headlock using rotational head movements that are generally ineffective and pose a risk to the facet joints as well as to the stability of the vertebrae [15]. Application of correct techniques in this scenario (induce a fall or apply a lever hold) [15] may prevent some of these injuries.

In a study of young adult judoka recruited from a collegiate judo club, up to 41% reported nonspecific low-back pain, while more than 90% in middle and heavyweight categories had radiologic abnormalities of the lumbar spine [6]. The practice of judo requires abrupt rotation of the hips and lumbopelvic region for leverage in order to execute throws [16]. An association between low-back pain in judoka and reduced hip range of motion has been identified [16]. In a study of young judoka (15–23 years) with at least 4 years of experience, those with reduced range of motion, particularly in their nondominant limb, were more likely to report a history of nonspecific low-back pain [16]. It is postulated that the reduction in hip range of motion leads to an increased load on lumbopelvic structures due to a compensatory increase in lumbopelvic rotation [16]. It is also possible that the lumbar symptoms cause adaptations at the hip secondary to altered movement. The authors of this study recommend further research to determine if improving hip flexibility and range of motion helps prevent some cases of low-back pain in young judoka [16].

Most back injuries in judo result from repeated falling, lifting, stretching, and twisting [17]. While lumbar disc pathology may be frequently encountered among older judoka [14], judo training appears to predispose the younger athlete to spondylolysis. Sakai [18] noted an incidence of spondylolysis ranging from 12.5% among adolescent judoka to 33.3% among highly trained judoka in a review of Japanese-language medical literature. This was substantially higher than the rate of 5.9% seen in the general Japanese population. The repetitive flexion of the spine may result in Scheuermann's disease in young judoka as well [14].

Wrestling

Wrestling is an ancient combat sport that originated approximately 5,000 years ago [19]. It shares some common elements with judo: it is a grappling sport that employs takedowns; points are scored by holding or pinning the opponent on his or her back; and striking techniques are not permitted. Wrestling has been identified as one of the higher-risk sports for cervical spine injuries [20]. In fact, the neck is the second most frequently injured body region in the sport [19]. Takedowns are responsible for many of the injuries to the neck [19]. The mechanism of injury may be landing on the head while the arms are caught up in a hold and unable to provide protection. Injuries also occur when the wrestler, attempting a roll, is landed upon by an opponent resulting in twisting or hyperflexion of the neck.

Repetitive hyperextension of the lumbar spine appears to be associated with greater risk of spondylolysis [21]. Wrestlers push against each other with the lumbar spine in slight hyperextension. This lumbar stress is then compounded by twisting motions used in takedowns [19]. In an older study of wrestlers with low-back pain, one-quarter had injuries to the pars interarticularis (spondylolysis or spondylolisthesis) [22]. Deficits in extensor strength have been identified in wrestlers with low-back pain; therefore, strength and conditioning training that addresses this weakness should be incorporated into a preventative program [19].

Taekwondo

Olympic-style taekwondo, a full-contact, free-sparring sport, rewards points for head contact using kicking techniques. In a 2004 study on injuries sustained during a Canadian National Taekwondo Championship, Kazemi and Pieter [23] reported that 17% of injuries occurred to the neck, upper back, lower back, or coccygeal region. Among youth under 16 years of age, 2–7% of tournament injuries reported were to the neck region (sprains, strains, contusions) with no other specific spine injuries identified in the data [24, 25]; however, between 10 and 28% of the injuries were to the head region, prompting the authors to recommend against blows to the head for children participating in taekwondo [24, 26]. The very forceful kicks demonstrated in taekwondo could place the cervical spine at risk if contact to the head or inadvertent contact to the neck were to occur.

Karate

Shotokan karate, one of the most widely practiced traditional karate styles, is considered "non-contact" with attacks theoretically pulled short of contact to the opponent. This is particularly true of strikes to the head and neck; however, light contact to the body frequently occurs [27]. During supervised training, the risk of spine injury among young karateka is very low [27, 28].

The front stance is commonly employed in karate training. When properly executed, the core muscles support the low back, eliminating excessive stress on the posterior elements of the lumbar spine. In the author's experience, excessive lumbar lordosis and poor abdominal strength are frequently encountered in younger, less experienced karateka. This places undue stress on the posterior elements of the lumbar spine, including the facet joints and pars interarticularis. Over time, these athletes may develop lumbar

Fig. 11.2 Weapons are commonly used in wushu

extension pain due to posterior element overuse syndrome or spondylolysis [3].

Wushu

Wushu is the Chinese word for all styles of Chinese martial arts, including kung-fu. Kung-fu may have originated as early as the sixteenth century B.C. [29]. Unlike many combat sports, wushu training may draw from hundreds of weapons, although only a small number are used in competition (Fig. 11.2) [30]. Wushu uses very low stances (Fig. 11.3a, b). Many kung-fu styles imitate the fighting styles of animals. Fighting in kung-fu involves strikes, kicks, and blocks, as well as takedowns. Choreographed forms (Taolu) often include elements of ballet and gymnastics, in addition to martial arts [30]. These forms involve both hyperextension and flexion of the spine, often within the same technique (Fig. 11.4a, b, c). The repetitive flexion, hyperextension, and twisting movements can predispose the young athlete to posterior element injury, as well as injury to the apophyseal rings of the vertebral bodies.

Rules

Many combat sports have rules in place to protect younger participants from serious injury. Rules may limit the amount of contact or force applied during striking techniques, or may prohibit strikes to the head for younger or less experienced participants. The risk of injury is greater during martial arts competitions; therefore, prevention of spine injuries is dependent upon the vigilance and ability of match referees to enforce the rules. Judo Canada, in its Long-Term Athlete Development model, stresses the importance of learning to fall and roll safely before tackling more complex techniques. Certain techniques such as headlocking with a grip over or around the neck, as well as potentially more dangerous throws, are prohibited in the younger age groups. Wrestling bans moves that twist the neck or back as well as headlocks that do not include an arm [19]. If a wrestler executes an illegal technique in a competition, the referee must intervene to break the dangerous hold and possibly penalize the athlete or award points to the opponent [19]. Prevention of catastrophic cervical spine injuries in wrestling relies on stringent penalties for intentional slams or throws [20] as well as emphasis in training on safe, legal techniques and proper rolling techniques [20].

Specific Injuries

Cervical Sprains and Strains

Injuries to the muscle–tendon units of the neck (strains) as well as sprains of the ligaments and capsules often occur in the setting of hyperextension and twisting of the neck. This mechanism of injury occurs primarily in grappling sports, with sprains and strains occurring commonly in judo [14] and accounting for approximately half of all neck injuries in wrestling [19].

Athletes with soft-tissue injuries to the neck may present with pain posteriorly and on both sides of the neck. They may have difficulty lifting their head the day following the injury [14]. Treatment includes physiotherapy, nonsteroidal anti-inflammatories, and avoidance of movements or techniques that aggravate symptoms. Strength exercises for the neck muscles are an integral part of grappling sports for prevention of injury.

Fig. 11.3 Very low stances of wushu. **a** Bow stance, similar to front stance of karate and taekwondo. **b** Drop stance with extreme flexion of the spine

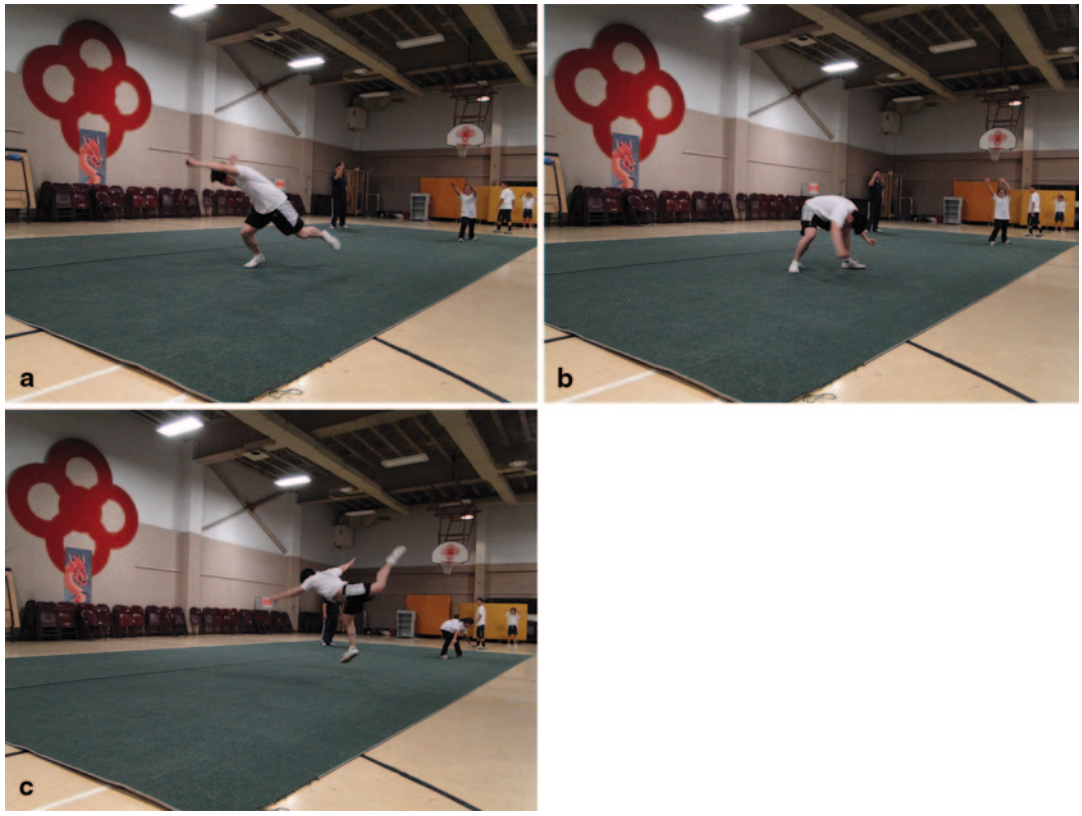

Fig. 11.4 Butterfly kick in wushu. **a** Preparation for take-off. **b** Spine flexed with chest on knees. **c** Continued rotation with jump, high kicks, spine extended

Burners/Stingers

Burners or stingers are reversible injuries to cervical nerve roots or the brachial plexus that result from either compression or traction forces [14, 19]. They are a common occurrence among wrestlers, with this sport being second only to American football in terms of frequency [19]. When the neck is subjected to forced hyperextension and ipsilateral flexion during a takedown, a compression-type neurapraxia leads to classic symptoms of burning pain extending from the shoulder down to the fingertips [19, 31] followed by weakness [31], particularly of the biceps, deltoid, and external rotators of the shoulder [19]. The athlete will often rub or shake the affected arm which hangs limply by his or her side [19, 31]. In the presence of bilateral upper extremity symptoms, persistent neck pain or neurologic symptoms, or limited range of motion of the neck, a more serious cervical spine injury must be ruled out [31]. Cervical spine radiographs with flexion/extension views, as well as magnetic resonance imaging (MRI) may be indicated [31]. Athletes may return to play following a stinger once all neurologic symptoms and signs have resolved and full range of motion and strength have been regained [31].

Catastrophic Cervical Spine Injuries

Catastrophic cervical spine injuries are structural distortions of the cervical spinal column associated with actual or potential damage to the spinal cord [32]. The spectrum of catastrophic cervical spine injuries in sports includes unstable fractures and dislocations, cervical cord neurapraxia (transient quadriplegia), and intervertebral disc herniation [32].

The risk of fracture or dislocation increases when an axial load is placed on a flexed neck. This posture aligns the spine into a segmented column that no longer benefits from the force dissipation of paravertebral muscles and vertebral ligaments [32]. Such a mechanism of injury may occur during incorrectly executed throws in judo [13, 14]. Appropriate on-field management including institution of spinal precautions must be initiated in any athlete who has sustained a potential injury to the cervical spine.

Posterior Element Overuse Syndrome

Certain biomechanical factors can contribute to injuries of the posterior elements of the lumbar spine in combat sports. The posterior elements refer to muscle–tendon units, ligaments, joint capsules, and facet joints of the lumbar spine. Common to several different striking styles (karate, wushu, taekwondo) is the front stance [3]. Experienced martial artists learn to stabilize the trunk and reduce lumbar lordosis through flexibility, strength, and technique training. Younger participants with inadequate abdominal strength and poor stance posture place additional stress on these posterior spinal elements (Fig. 11.5a, b). This increases the risk of overuse injury to this area [3]. This is more likely to occur in styles that employ a longer and lower front stance (Shotokan karate) or bow stance (wushu) versus a shorter and higher stance (Goju-ryu karate, Uechi-Ryu karate).

Repetitive stress on the posterior elements can lead to pain on hyperextension of the spine with focal tenderness over the affected level just lateral to the midline [3]. If secondary muscle spasm is present, there may be some tightness and discomfort on trunk flexion as well. Neurologic signs and symptoms are typically absent in Posterior Element Overuse (PEO) syndrome. Risk factors for this condition, including increased lumbar lordosis, weak core muscles, and tight hip flexors, may be identified on physical examination. Raising both legs off the table, a few centimeters in a supine position (supine stress test), provokes pain in the lumbar region as it loads the posterior elements [3].

In the absence of any “red-flag” symptoms, such as fever, malaise, weight loss, or night pain, conservative treatment may be instituted. Treatment has three main components: reduction of pain and inflammation, promotion of healing, and modification of risk factors. Regular application of ice and nonsteroidal anti-inflammatories can help settle symptoms. Relative rest

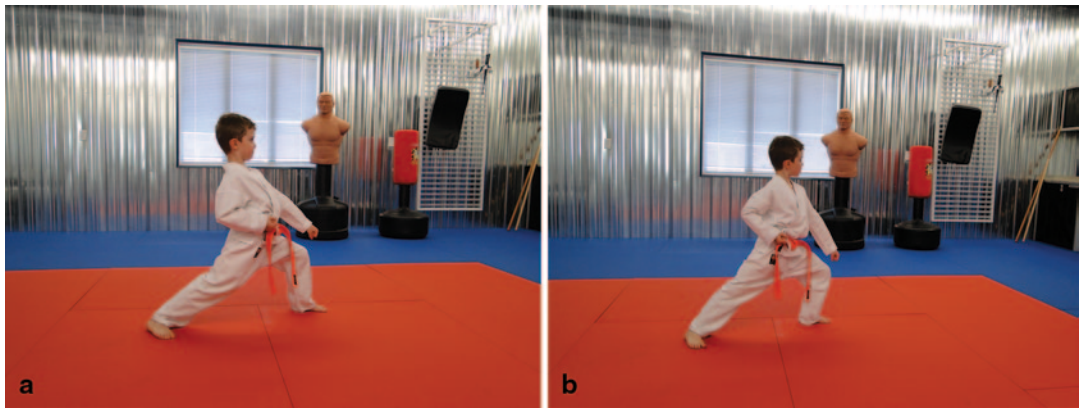

Fig. 11.5 **a** Incorrect posture in a karate front stance can lead to chronic overloading of the posterior elements of the lumbar spine. **b** Correct posture with good core stabilization

(avoidance of techniques that produce pain) and use of a flexible lumbar support brace help limit movements and activities that may interfere with healing. The brace, which is often used for up to 6 weeks, may also help reduce pain and muscle spasm associated with the injury [33] and facilitate return to training [34].

Underlying risk factors for lumbar spine injury must be identified and corrected whenever possible. Tight muscles, weak core strength, and hyperlordotic posture must be addressed through a home exercise program or with the assistance of a physiotherapist. Incorrect karate techniques and posture must be corrected. Proper stabilization of the trunk and limitation of lordosis in the front stance/bow stance will help unload the posterior elements and reduce the risk of reinjury [3]. Judoka must maintain good flexibility and core strength and stabilization to reduce stress on the posterior elements.

Young athletes with lumbar pain that persists for at least 3 weeks should undergo further investigations to rule out other more serious causes of low-back pain [7]. Plain radiographs including oblique views, screening bloodwork, or single-photon emission computed tomography (SPECT) bone scan may help identify underlying pathology such as spondylolysis or rheumatologic conditions. The SPECT scan, which can detect occult spondylolytic lesions not visualized on plain films, should be considered if there is no improvement after 4–6 weeks of treatment [3]. MRIs are being used with increasing frequency with the benefit of looking at soft tissue and bony stress without radiation exposure.

Spondylolysis and Spondylolisthesis

Stress fractures of the pars interarticularis, also known as spondylolysis, present like posterior element overuse syndrome: pain in the lower lumbar region that increases with extension of the spine. L5 is the level most commonly affected [34], followed by L4 and much less frequently L3. The onset of symptoms is typically insidious, with symptoms persisting for months before presenting for evaluation [3]. Unilateral spondylolysis may lead to increased stress and possible fracture of the contralateral side [35]. With little restraint to forward translation of one vertebral segment over the next caudal segment, spondylolisthesis may ensue. The degree of spondylolisthesis is graded I–IV depending on the percent of surface area that one segments "slips" with respect to the adjacent segment. Grade I is defined as 0–25 %, grade II as 25–50 %, grade III as 50–75 %, and > 75 % is classified as grade IV [36].

Physical examination findings of spondylolysis are similar to those observed in posterior element overuse syndrome. Pain is typically worse with hyperextension of the spine and unilateral pars lesions will have maximal pain with stork

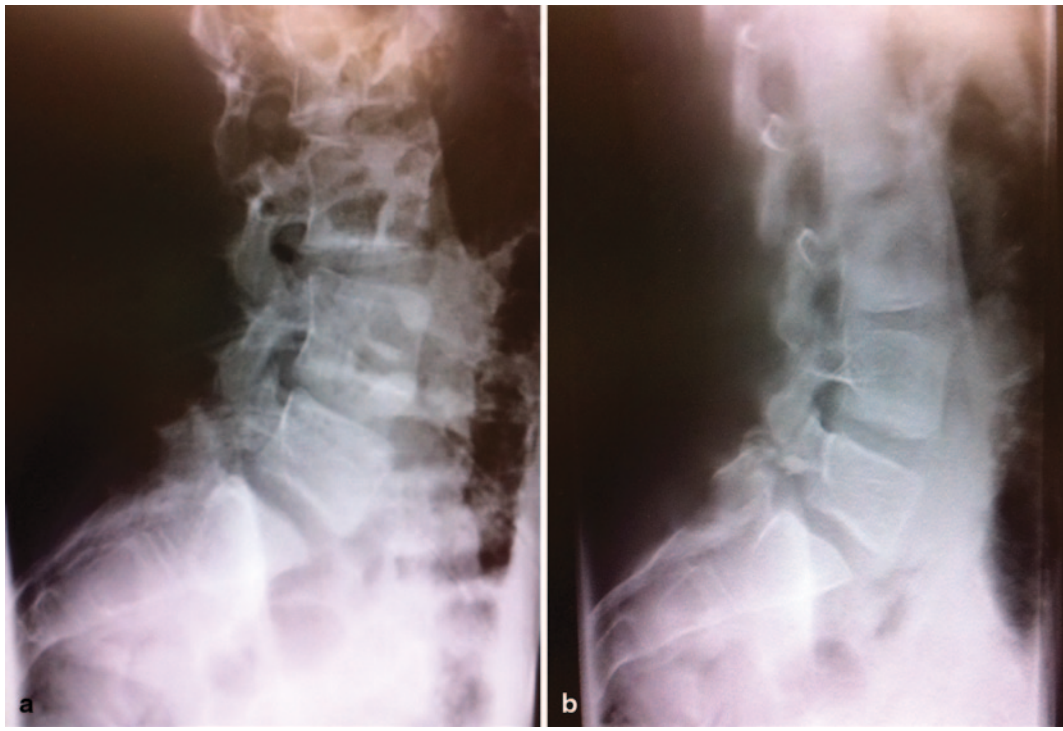

Fig. 11.6 **a** Conventional lateral radiograph, coned to level of symptoms. **b** Volume RAD lateral (same patient) is more sensitive for identifying spondylolysis

testing on the affected side. Secondary spasm of the paraspinal muscles frequently accompanies spondylolysis, producing some tightness or discomfort on flexion of the spine. Focal tenderness over the lesion assists in determining the affected level of lumbar vertebra. Loading the posterior elements in a supine position provokes pain and may reveal the presence of weak core muscles or poor trunk stabilization. Although not typical features of spondylolysis, nerve root signs such as positive straight leg raise test may be present, particularly in the setting of spondylolisthesis [3].

Young athletes with low-back pain of at least 3 weeks duration and exacerbated by extension of the spine should be investigated further. Anteroposterior and lateral views are classically supplemented with oblique views to better delineate lesions of the pars interarticularis. Oblique views will identify only a third of lesions due to orientation of the beam with respect to the fracture [37]; therefore, some authors have advocated against routine use of oblique radiographs [7]. A coned lateral view directed toward the suspected level of spondylolysis may be more sensitive than regular lateral films [38].

More recently, Volume RAD (tomosynthesis) images have been used to identify spondylolytic lesions. These are low-dose radiographs with multiple high-resolution slice images. In the author's experience, this imaging modality is more sensitive than a conventional plain film series of the spine in detecting spondylolysis (Fig. 11.6a, b).

If spondylolysis is still suspected but plain films or volume RAD images are negative, Technetium-99 SPECT bone scan should be considered. This imaging modality can identify early stress reactions that have not yet progressed to a frank fracture. If plain films or volume RAD images do detect pars lesions, SPECT scan will differentiate between active bony injuries that have a potential for healing [39] and older lesions that may have already healed with a fibrous union. If a young athlete is not responding to treatment, computed tomography (CT) scanning may be indicated. CT through the level of the spondylolytic

lesion may identify degree of bony healing which helps guide further management.

MRI is increasingly used in the evaluation of spondylolysis. It has the advantage of identifying structural defects while differentiating between active lesions and chronic, inactive lesions of the pars interarticularis. Unlike SPECT scan, MRI is able to detect other significant pathology of the spine (e.g., juvenile disc herniation) without the potentially deleterious effects of ionizing radiation [40]. Campbell et al. [41] concluded that MRI accurately demonstrates normal pars, acute complete defects, and chronic, established defects [41]; however, MRI was limited in its ability to correctly grade early lesions (stress reactions and incomplete lesions) when compared with a combination of CT and SPECT imaging [41]. Of note, MRI was still able to detect abnormalities in 39 of 40 pars defects identified in the study [41]. Despite this limitation, several authors [40, 41] advocate for MRI as a first-line imaging modality in young athletes with suspected spondylolysis.

Initial management of spondylolysis is similar to that of posterior element overuse syndrome: relative rest (avoidance of painful maneuvers such as arching or running), daily home exercises (core strength, stretching of hamstrings/hip flexors/lumbodorsal fascia, antilordotic exercises), and ice. A custom thoracolumbar orthosis helps prevent some movements (spine extension) that can aggravate spondylolysis and may facilitate a safe return to modified training in some combat sports [3].

Scheuermann's Disease (Atypical or Lumbar)

Rapid alternating flexion and extension of spine, often within a single technique like the wushu butterfly kick or the kick-up, can cause stress injuries to the vertebral end plates around the thoracolumbar junction in adolescents [4, 42]. Unlike classic Scheuermann's thoracic kyphosis, atypical Scheuermann's is associated with a very flat back. Anterior wedging of the vertebral bodies is absent, but other radiographic features of classic Scheuermann's may be present: end plate fractures, Schmorl's nodes, and apophyseal avulsions [4].

Athletes with Scheuermann's usually note pain on flexion of the spine and may have tightness of the thoracolumbar fascia and hamstrings [4, 42]. Plain lateral radiographs confirm the clinical suspicion.

Treatment should include an exercise program that focuses on spinal stabilization and extension exercises as well as hamstring and thoracolumbar fascia stretches [4, 7]. A brace with at least 15° of lordosis may facilitate return to training within a few months [7, 42].

Summary

The key to the diagnosis and management of spine injuries in the young athlete in combat sports is to have a clear understanding of the unique demands of each discipline. Acute injuries in grappling and throwing sports, often the result of throws or takedowns, may result from landing in a vulnerable position on the mat or from rotational motions during contact with an opponent. In the striking sports, risk of acute injury will vary depending on the degree of contact that is permitted. Variations in training and fighting stances among disciplines may also contribute to overuse injuries. The two unifying themes for prevention of injuries to the spine in combat sports are careful attention to correct technique and strict adherence to rules of safety in place in each sport.

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Principles of Rehabilitation

12

Michellina Cassella, Carl Gustafson
and Pierre d'Hemecourt

History and Theory of Spine Stabilization

Back pain is common in the nonathletic population with up to 80% of the population being affected at some point [1]. The athletic population also complains of back pain with varying prevalence by sport. Back injuries in football linemen have been reported with an incidence as high as 50%, while in rhythmic gymnasts, 86% of participants reported pain [2, 3]. Consequently, there has been a lot of attention given to spinal stability. Since the 1970s, there has been an interest in nontraumatic spinal injury, which was felt to be secondary to instability with tissue overload and joint degeneration [4]. Bergmark first defined the local and global stabilizers [5]. The lumbar spine has the direct attachments of the multifidi, transverses abdominis (TA), and internal oblique fibers, which act as the local stabilizers. These attachments maintain posture and stiffness of the spine, particularly the multifidi [6]. The global stabilizers include the rectus abdominus, external obliques, erector spinae, and quadratus lumborum muscles that transfer forces across multiple segments to the pelvis and thoracic spine.

Subsequently, Panjabi proposed three interrelated systems of segmental stability [7]. These include the passive system of the boney vertebrae, intervertebral discs, and ligaments. The active system involves the spinal musculature and tendons. The third system is the neural innervation that monitors the first two systems. Inherent to this is the defined neutral zone. This is the limit of spine motion with minimal resistance from the osseous and ligamentous restraints [8]. This neutral zone will increase with injury that may be compensated with improving the strength of the supporting musculature. Spinal stability is now often referred to as the ability to maintain or return to this neutral position.

The TA and the multifidi have been shown to have incomplete recovery after lumbar injury and may be associated with pain and thus became the focus of rehabilitation [9, 10]. A number of smaller studies demonstrated some utility of specific exercises for these muscles. Richardson and Jull emphasized an exercise regimen addressing these deep stabilizers clinically [11]. O'Sullivan confirmed the clinical utility of these exercises in a study of spondylolysis and spondylolisthesis with patients with chronic back pain [12]. This was based on the concept that these muscles could be isolated. However, McGill showed that these muscles work in synchrony and are clinically difficult to differentiate [13].

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This deep local stabilizer group is often what is referred to as the core stability. However, the utility of core stabilization as a treatment is still controversial with a number of reviews demonstrating a lack of superiority for core exercises to conventional active exercises [14–16], although in some there appeared to be a slight increased effectiveness with chronic low-back pain (LBP). Conversely, Cairns demonstrated in a randomized controlled study that there was no significant improvement in outcomes of specific spinal stabilization over general active exercise and mobilization [17]. However, most of these studies do not reflect working with the athlete.

The clinician dealing with LBP in the athlete must focus on the muscle activating patterns to work in synchrony with the full closed kinetic chain similar to the treatment of other musculoskeletal injuries. For example, it is well understood that multifidi and TA weakness may perpetuate in the athlete with a back injury. As such, these deficiencies are addressed for restoration of bulk and decreased symptoms [9]. However, the integration with other muscular activation must be addressed. The rehabilitation of an athlete must follow a logical progression to reestablish proper neuroactivation of these muscle groups.

Athletic Concerns in Rehabilitation

Injury Pattern

The actual injury pattern is the first consideration in athletic rehabilitation. As described in other chapters of this text, there are a number of injuries specific to the athlete. Football, gymnastics, and diving have a higher incidence of posterior spinal injury such as spondylolysis [18]. An anterior element injury to the disc is also recognized in certain sports such as gymnastics, crew, and weight training with repetitive flexion, extension, and shearing [19]. The anterior element injuries often present with more flexion-based pain while spondylolysis presents with more extension-based pain. Certainly, there is crossover. However, understanding the pattern is important for two reasons. First, the direction of initial mobilization is often in the opposite direction of injury. Second, with the lack of motion in the direction of injury, weakness may develop from incomplete rehabilitation. For instance, with spondylolysis and chronic pain, there is often extensor weakness that must be addressed as healing proceeds [12].

Patterns of Muscular Activation and Lumbopelvic Motion

The basic core is comprised of the diaphragm, pelvic floor, abdominals (including rectus abdominis, TA, external oblique, and internal obliques), and the back musculature. The abdominals act primarily as lumbar flexors [20]. The back extensors are primarily composed of the multifidi, longissimus and iliocostalis, and quadratus lumborum [21]. The multifidi span only a few segments and act to stabilize posture as well as add some rotation and extension. The erector spinae in the lumbar region are represented by the longissimus and more lateral iliocostalis. These span multiple segments from their attachments to the sacrum and iliac crest and act concentrically to extend the spine and eccentrically during lumbar flexion. The quadratus lumborum also acts for lateral stabilization [20]. The psoas acts to stabilize the spine anteriorly with a compressive force here.

The peripelvic muscles of the gluteus maximus and medius muscles play a strong role in spinal motion. The gluteus maximus has strong attachments to the iliac crest and the caudal portion of the thoracolumbar fascia (TLF) [22]. This muscle may directly transfer forces from the lower extremity to the upper extremity with this connection as well as acting to extend and externally rotate the hip. The gluteus medius acts in motions to abduct as well as flex and rotate the hip. At the cephalad portion of the TLF, the latissimus dorsi is attached receptive to transfer of forces. As such, Vleeming proposed the four slings that transfer forces across the spine [23, 24]. In the posterior oblique sling, the contralateral gluteus maximus is attached to the latissimus dorsi via the TLF. The transfer here is of obvious importance for a throwing athlete transferring forces from the

ground up and placing the scapula in the proper position. The second sling is the anterior oblique sling from the external oblique muscle through the abdominal fascia to the contra lateral internal oblique and adductor tendon. The third sling is the longitudinal sling from the erector spinae through the sacrum and sacrotuberous ligament to the biceps femoris and into the calf peroneal muscles. Finally, the lateral sling maintains the lateral stabilization from the gluteus medius muscles via the TLF to the quadratus lumborum and lateral stabilizers. These interconnections of force transfer are important in the athlete and are often disrupted with spinal injuries.

The hip and lower extremity interaction is also pertinent as lower extremity injuries and back pain have been correlated with pelvic weakness [25]. Adductor dominance over abductor strength is common in athletes. Addressing this abductor weakness is useful in treating back and lower extremity pain [26, 27].

The lumbopelvic boney and ligamentous connections must also be considered in the athlete in several regards. The first reflects the position of the hip and pelvis. In the athlete attempting to maximally turnout or externally rotate the hip, the iliolumbar ligament is relaxed with an anterior pelvic tilt and concomitant lumbar lordosis [28]. This may be a manifestation of poor form in the ballet dancer forcing turnout at the expense of increased lumbar posterior impingement [29]. The second pelvic interaction is at the sacroiliac joints. The motion here represents only 2–6 degrees of motion but is involved in some force transfer [30]. When the sacrum flexes forward in relation to the ileum, this is referred to as nutation, and when the sacrum extends relative to the sacrum, this is referred to as counternutation. The stable or closed pack position is the nutated sacrum and is activated prior to heel impact via the erector spinae [31]. Subsequent to this there is a relaxed counternutation activated by hamstring activation through the sacrotuberous ligament. Although minimal in motion, dysfunction of this action may be associated with pain in the sacroiliac joint. This dysfunction may include weakness of the erector spinae often seen in adolescent spine injuries.

Pediatric Concerns for Spinal Rehabilitation

During the adolescent growth spurt, lumbar lordosis increases, which presumptively increases posterior element stress. Additionally, high levels of athletic activity in hours per year have also been shown to be a factor in increasing lumbar lordosis, particularly in the gymnast. Since posterior element stress injury such as spondylolysis is prevalent in the young athlete, this relatively sudden increase in lordosis may be a factor [32]. Many young athletes will continue growth while in rehabilitation and this needs consideration in returning to activity. Additionally, weakness of static lumbar extensor endurance has been identified as an association of LBP in the adolescent [33].

Athlete Evaluation

The major principle of rehabilitation is to safely restore function and prevent future injury. The goals of the rehabilitation program are to reduce and control inflammation, decrease pain, promote healing, restore function, and safely resume activities.

The rehabilitation program includes a detailed patient history and comprehensive assessments of posture, joint range of motion, muscle strength, neuromuscular system, and function. Postural deviations and muscle weakness and/or tightness can often lead to serious imbalances that can exacerbate symptoms and delay a full recovery.

History

The diagnosis as determined in other chapters must first be confirmed. The specific issues pertinent to rehabilitation must be determined by asking the following:

- Is the injury acute or chronic? The answer will reveal if the suspected injured tissue is still in a normal time frame of healing or beginning to progress into a more chronic myotendinous decompensation.

- What postures or positions make the patient feel better or worse (sitting/flexing vs. standing/ extension vs. transition). This would reflect more anterior or posterior element injury. A disc herniation will often present with sitting intolerance and possibly with radiation. A spondyloysis reaction may present with more standing and activity-based pain. There is certainly crossover in all of this. What movement patterns make the patient feel better (sitting vs. standing)? This is usually the opposite direction of what symptoms bring on the pain. This will start the directional preference category for rehabilitation [34].
- Is the pain greater in the morning or evening? Morning stiffness may indicate inflammatory pain if it is prolonged. Evening pain is often due to muscle spasm and fatigue.
- Assessing for red flags involves asking about the presence of searing night pain, fever, chills, night sweats, or any bowel or bladder symptoms.

Physical Exam

Posture A comprehensive posture assessment is essential to identify postural deviations and/or malalignment that can have a negative impact on body mechanics. The posture assessment can also help to identify joint limitations and muscle impairments (Table 12.1).

Standing Testing lumbar flexion and extension for pain reproduction. This assessment is paramount in assessing the direction of spinal rehabilitation in the athlete. The diagnosis itself is important in noting the motions that should be avoided initially. For instance, a disc herniation may preclude excessive flexion activity while a spondylolysis will usually preclude extension activity initially. However, the exam should confirm that pain occurs in flexion or extension respectively as there is crossover and the direction of pain on spinal motion will influence the initial direction of rehabilitation. Heel and toe walking provides an initial neurologic screening exam.

Seated Manual resistance of toe, foot, knee, and hip musculature bilaterally. A positive slump test may reveal some dural tension.

Supine and Prone

1. Thomas test (Fig. 12.1a, b): Measures tightness in the iliopsoas muscle. Restricted flexibility in this muscle can cause an increase in lumbar hyperlordosis, decreased hip extension, and an increase in knee hyperextension.
2. Straight leg raise (Fig. 12.2a, b): Measures hamstring tightness. Restricted hamstring flexibility will negatively affect low back, pelvis, hip, and knee alignment.
3. Ober test (Fig. 12.3a, b): Measures tightness in the tensor fascia latae specifically the iliotibial band (ITB). Restricted flexibility often promotes malalignment of the hip as well as lateral tracking of the patella. Both can have a negative effect on the spine especially the low-back region.
4. Ely test (Fig. 12.4a, b): Measures tightness in the rectus femoris muscle. Restricted flexibility in this muscle not only has a negative effect on the knee but also contributes to hip flexor muscle tightness that will add to malalignment of the lumbar spine and increase lumbar hyperlordosis.

Global Hip, Lower Extremity and Core Stability Tests:

The following tests are helpful in assessing pelvic and core stability. First, the single leg squat that may demonstrate weakness of the gluteal muscles has been demonstrated by Nadler to be an associating factor with LBP [35]. This may be assessed with the single leg squat while the athlete holds the hands on the hips [36]. The abnormal findings would include dropping the pelvis on the non-weight bearing leg with gluteus medius weakness and valgus collapse at the knee with gluteus maximus weakness.

Second, lumbar endurance strength is a marker of weakness with back pain [37]. This may be shown with a lumbar extension endurance test (see Fig. 12.5). Abdominal strength may be as-

Table 12.1 Posture evaluation form

Posture Evaluation Form

Name:_____Medical Record NO._____D.O.B._____Sex____
Diagnosis_____Surgical Procedure/Date_____
Precautions_____

Posterior View			**Left**	**Right**
Head	Centered	Tilt		
Shoulders	Level	Elevated		
Scapulae	Level	Elevated		
Spine	Aligned	Shifted		
Waist folds	Symmetrical	Increased		
Pelvis	Level	Elevated		
Knees	Aligned	Varus		
		Valgus		
Heels	Aligned	Varus		
		Valgus		
Anterior View			***Left***	**Right**
Head	Centered	Tilt		
Neck folds	Symmetrical	Increased		
Breasts	Symmetrical	Prominent		
Arm length	Equal	Longer		
Pelvis	Level	Elevated		
Knees	Aligned	Varus		
		Valgus		
Forefoot	Aligned			
	Pronated			
	Supinated			
Lateral View			**Left**	**Right**
Head	Aligned		Forward	Backward
Cervical (anterior) curve	Normal		Increased	Decreased
Shoulders	Level		Forward	Backward
Scapulae	Aligned		Protracted	Retracted
Thoracic (posterior) curve	Normal		Increased	Decreased
Lumbar (anterior) curve	Normal		Increased	Decreased
Pelvis	Aligned		Anterior Tilt	Posterior Tilt
Adams Forward Bend Test			**Left**	***Right***
Thoracic	Negative		Rib hump	Rib hump
Lumbar	Negative		Increased m. bulk	Increased m. bulk
Knees	Aligned		Hyperextension	Hyperextension
Ankles	Aligned		Increased DF	Increased DF
			Increased PF	Increased PF
Line of Gravity (plumbline)	Aligned	Shifted Forward		
		Shifted Backward		

sessed with an abdominal brace (see Fig. 12.6). Overall core stability is assessed with a single leg bridge (see Fig. 12.7).

Neuromuscular System This involves testing the athlete's muscle strength, coordination, proprioception, agility, and gait. Impairments in any of these areas will not only have a negative effect on the spine but will also impact the success of the rehabilitation program. This will often involve having the athlete perform a sport-specific maneuver. For a dancer this may be a sustained arabesque and for the gymnast a back scale. For a pitcher, a video analysis of the pitch may be needed.

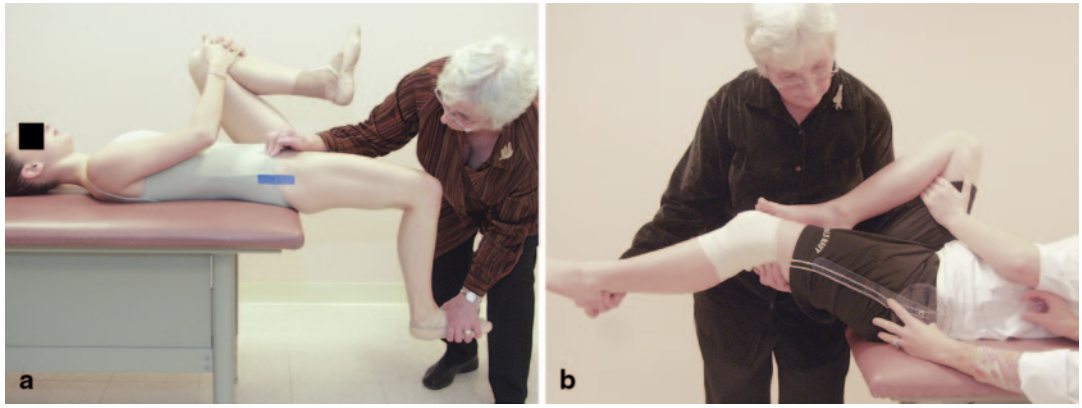

Fig. 12.1 **a**, **b** Thomas test

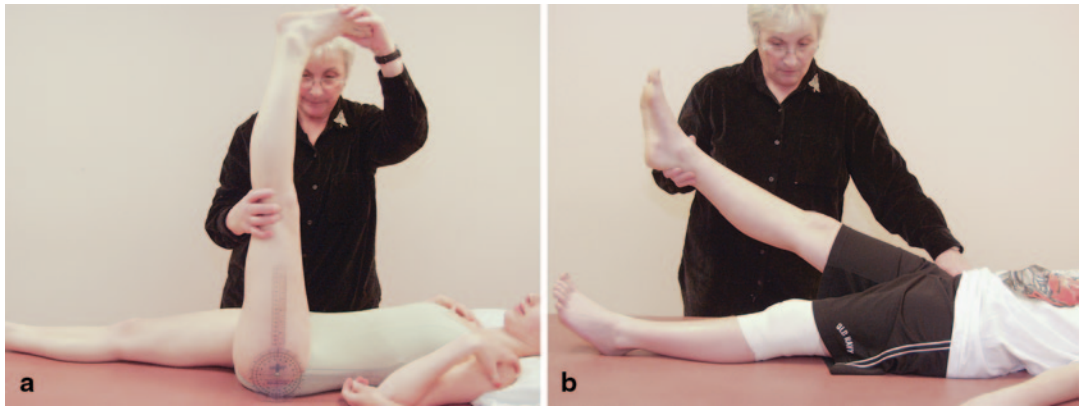

Fig. 12.2 **a**, **b** Straight leg raise

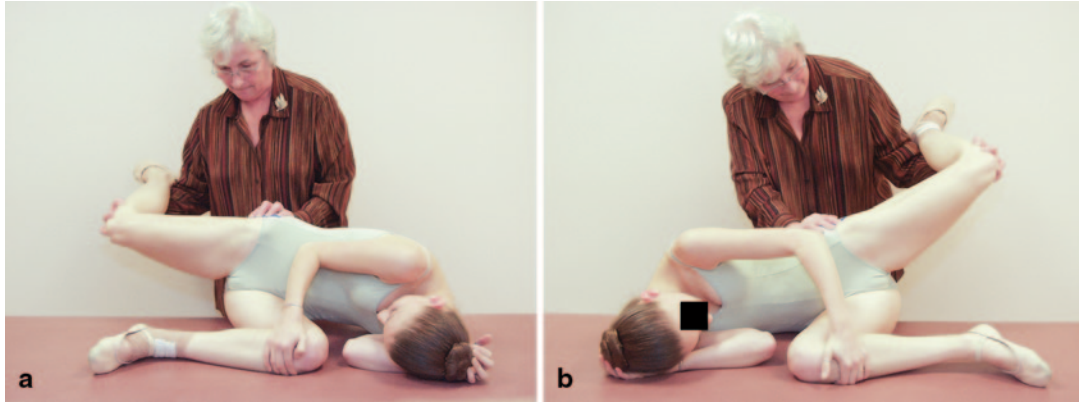

Fig. 12.3 **a**, **b** Ober test

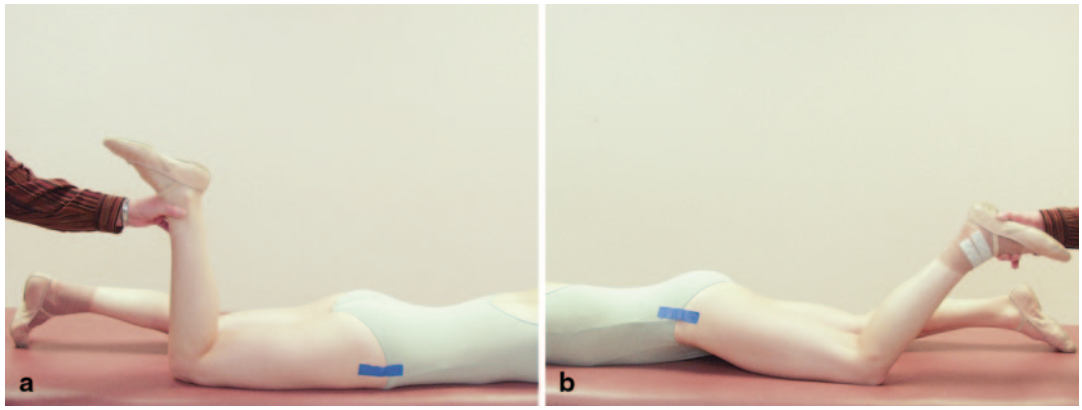

Fig. 12.4 **a**, **b** Ely test

Fig. 12.5 Lumbar extension endurance test

Fig. 12.6 Assessing abdominal strength with an abdominal brace

Rehabilitation

The clinician must understand the stages of rehabilitation as well as the directional preference before advancing to the next phase.

Acute Stage Tissue has been injured within 24–48 hours and is in the initial healing stage. This may require rest and immobilization.

Subacute Stage Although the tissue is healing, the patient is able to begin to move the injured part in multi-joint patterns. Modalities may be helpful here.

Rehab Stage This is the longest part of rehabilitation. The patient is able to add resistance training, flexibility, and cardiovascular training. Cardiovascular conditioning is very important as the muscles of spinal endurance are mostly the type I fiber types. We will usually recommend at least 150 min per week of cardiovascular fitness and the athlete needs to demonstrate endurance equal to the level demanded on their return to sports.

Fig. 12.7 Assessing over-all core stability with a single leg bridge

Functional or Sports-Specific Stage This phase is unfortunately often skipped in getting the athlete back to play. This is a critical phase that involves training recently rehabilitated muscles to work in synchrony with the musculoskeletal system. It is often referred to as neuromuscular reeducation. The patient is able to add graded functional movements which progress to sports-specific movement patterns in to resume their activity or sport.

Figure 12.8a–d illustrates the program of rehabilitation from the acute stage to the sports-specific stage, in this example when focusing on the muscle group.

Directional Preference The directional preference model is most helpful during the acute, subacute, and rehab stages. Once the patient returns to the functional or sports-specific stage, you would expect full range of motion.

Flexion Bias These patients will have sustained a lumbar extension injury or have extension pain. Thus, lumbopelvic flexion is initiated and lumbar hyperextension is avoided but activation of the extensors is often possible with maneuvers such as bridging in the early phases.

Extension Bias These patients will have sustained a lumbar flexion injury or have lumbar flexion pain. These patients will often have sustained a disc or apophyseal injury. Thus, exercises are in the neutral to extension direction. Lumbar flexion is avoided but abdominals may be activated to avoid atrophy.

Neutral This is an entity which is not uncommon. Disc injuries in particular may present with pain on both lumbar flexion and extension. Patients will prefer to do exercises in the mid-range. The patient can exercise their lumbar/abdominal flexors, extensors, rotators, and lateral flexors in this mid-range.

Directional preference is applicable for strength, aerobic training, and flexibility. Aerobically, a spondyolysis will often tolerate a bike better than running type of activity. Swimming free style is usually an excellent neutral zone activity.

Example of Common Adolescent Spine Injury: Spondylolysis

Acute and Subacute Phase The plan of care includes posture alignment both in and out of the orthosis; instruction in proper body mechanics and a series of safe stretching and strengthening exercises that must be "pain free."

The short-term goals are to decrease pain, improve posture, and increase and maintain muscle flexibility.

The long-term goal is for the athlete to safely return to his or her sport and/or performing arts activities. The following is a suggested list of exercises:

1. Posterior pelvic tilt with lumbar flexion to reduce lumbar hyperlordosis. This exercise should be done both in and out of the orthosis.
2. Low-back stretches to be done out of the orthosis.
3. Abdominal muscle strengthening exercises, out of the orthosis.
4. Anterior hip flexor stretch with brace on; performing this exercise with the brace on helps to maintain lumbar flexion with a posterior pelvic tilt, thus gaining a greater stretch of the iliopsoas muscle. Continued tightness in this muscle has a negative effect on the lumbar spine.

Fig. 12.8 **a–d** Program of rehabilitation from the acute stage to the sports-specific stage

Ancillary Interventions: When an athlete is unable to progress from one phase to the next, there are often other interventions that may be useful in quieting the pain to allow better stabilization. This may include mechanical measures such as bracing and taping. Some injuries such as spondylolysis will often utilize a brace. Other interventions may include oral anti-inflammatory medication and at times steroid injections. Additionally, active release therapy, myotherapy, and acupuncture may be very helpful.

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Congenital Spine Malformations and Sports Implications

13

Kristin S. Livingston and John B. Emans

Classification

Congenital spinal anomalies can be classified according to location (cervical, cervicothoracic, thoracic, thoracolumbar, lumbar, or lumbosacral), pattern of deformity (sagittal plane kyphosis, coronal plane scoliosis or kyphoscoliosis in combination), and type of malformation, which may be a failure of formation, failure of segmentation, or a combination (Fig. 13.1).

A failure of formation results when the vertebra forms incompletely, and this can occur in the coronal or sagittal plane. In coronal plane deformity, if both pedicles form but one side grows asymmetrically, this results in a wedge vertebra. If only one pedicle develops and there is complete failure of formation on one side, this results in a hemivertebra. Likewise, sagittal plane deformity may involve a wedge vertebra if the anterior vertebral body forms partially, or a hemivertebra if only posterior elements exist. A wedge vertebra or hemivertebra may be nonsegmented, partially segmented, or fully segmented depending on whether the vertebra has no associated intervertebral disc, one associated intervertebral disc, or two associated intervertebral discs, respectively. A similar phenomenon occurs with ribs, which may be fused or absent, representing failure of segmentation or formation. Rib anomalies may occur on their own or in conjunction with vertebral anomalies and can also lead to "thoracogenic" curvature of the spine (Fig. 13.2).

Any of these abnormalities can result in angular deformity of the spine and can cause deformity in multiple planes, with sagittal plane abnormalities leading to kyphosis (or lordosis) and coronal plane abnormalities leading to scoliosis. A patient may have an isolated vertebral anomaly or many, and they may be all the same type, or a combination of types.

Natural History

Congenital spine malformations are often noticed by the parent or pediatrician at an early age or may be an incidental finding. Some may present with pain or neurologic findings of insidious or traumatic onset as an adolescent or even later. Regardless of whether a curve exists or not, the presence of any vertebral anomalies warrants a thorough workup and long-term monitoring. There are certain geometric patterns that have a higher propensity to worsen over time. For example, in congenital scoliosis, a hemivertebra with a contralateral bar has the greatest potential for curve progression (>10 degrees per year), particularly during the first 3 years of life and during the adolescent growth spurt [2]. For this reason,

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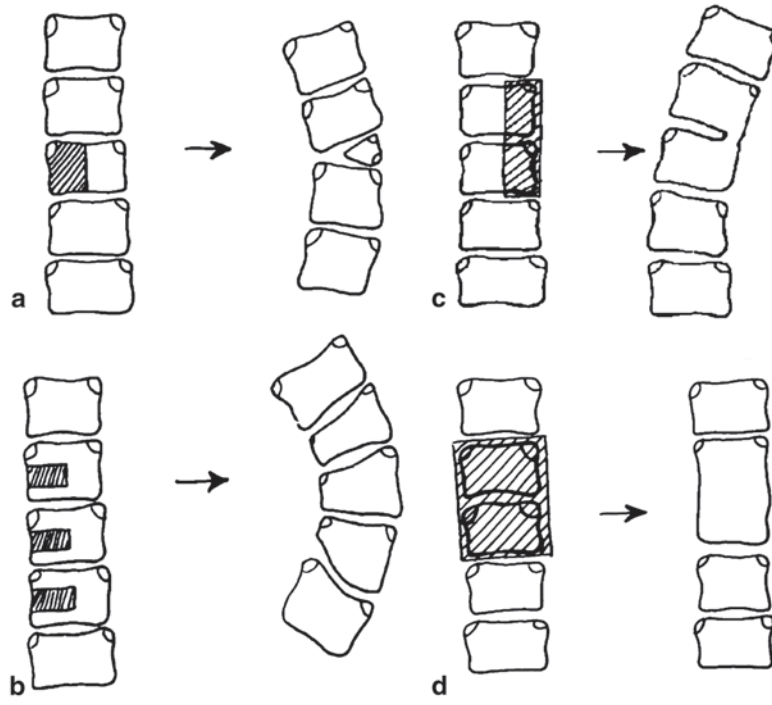

Fig. 13.1 Failure of formation resulting in either a hemivertebra (**a**) or a wedge vertebra (**b**). Failure of segmentation resulting in unilateral bar (**c**) or block vertebrae (**d**)

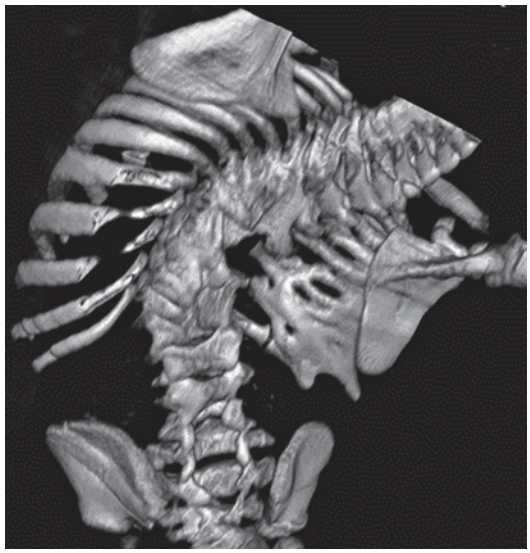

Fig. 13.2 Congenital scoliosis with rib fusions

it is recommended that most congenital spine abnormalities be followed with serial plain radiographs during growth, more frequently for curves at higher risk of progression.

Associated Anomalies and Workup

In addition to being associated with many named syndromes, vertebral anomalies are commonly associated with both neural axis and visceral abnormalities due to the temporal proximity of the development of the spine and other organs during the fetal period. This frequent association requires vigilance to ensure that the patient with congenital spinal malformations receives the appropriate workup [3]. Neural axis anomalies (e.g., diastematomyelia, tethered cord, or Chiari malformation) may be present in up to 38% of patients with congenital scoliosis and even more frequently in congenital kyphosis [31]. The presence of cutaneous manifestations (e.g., hair patches, sacral dimpling) should heighten suspicion and a thorough neurologic examination should always be performed. In addition to spinal cord abnormalities, visceral abnormalities, most commonly of the heart or urogenital system, are commonly present in patients with congenital vertebral abnormalities. A combination of anomalies of the spine, anus, heart, trachea, es-

ophagus, kidneys, and/or limbs may be seen with VACTERL association. Due to the high likelihood of concomitant morbidity, patients with congenital spine malformations should have whole-spine radiographs and often magnetic resonance imaging (MRI), cardiac echo, and renal ultrasound for screening purposes, and should be referred to the appropriate specialist in case of abnormal results. Up to one quarter of patients with congenital spinal malformations will have an associated cardiac abnormality, most commonly ventricular septal defect (VSD) [1]. Congenital heart disease may or may not preclude a patient from participating in athletics, and a cardiologist should help in determining the appropriate activity level for each patient. Furthermore, around one-third of these patients will have renal abnormalities, most commonly unilateral kidney [1]. Urologic injury is not very common in athletics, though cycling, winter sports, and contact sports may place the patient at risk [4]. Although a unilateral kidney may not preclude participation in such sports, it is important that the patient be appropriately counseled by a urologist.

Functional Issues Common to Congenital Vertebral Anomalies

Loss of Spinal Motion

Loss of flexibility of the spine can be a significant issue with vertebral malformations as the absence of normal discs and facet joints between involved vertebrae decreases the number of motion segments. Depending on how extensive the abnormalities are, the type and amount of loss of motion will vary. Upper cervical spine anomalies at C1–2 will tend to cause changes in axial rotation, whereas lumbar spine anomalies will cause loss of flexion–extension. Intersegment motion in the thoracic spine is relatively modest at an individual level, but as a whole the total capacity for motion in rotation, bending, and flexion/extension is significant [5]. Thus, single vertebral malformations in the thoracic spine do not often limit flexibility, but a large area of malformations will. Associated congenital rib fusions will also tend to decrease torso motion along with chest excursion. This loss of motion may be particularly troublesome for an athlete participating in gymnastics, diving, wrestling, football, golf, or other sports which involve significant spine rotation but may be well tolerated by athletes of sports that primarily involve running straight ahead.

Adjacent Segment Hypermobility and Degeneration

Many congenital anomalies lack normal mobility in the segment of spine involved. In some circumstances, this rigid segment begets an immediately adjacent region of excessive stress with compensatory hypermobility above or below the congenital segment. An isolated preserved motion segment between rigid congenital segments may be particularly prone to excessive stress. Adjacent segment disease (ASD) involves radiologic degeneration with associated clinical symptoms of the intervertebral disc levels immediately above or below a fusion mass. This can develop as a late complication after surgical spinal fusion due to increased intra-discal pressure, increased facet loading, and increased mobility below the fused site [6].

In congenital vertebral malformations with segmentation defects, patients may develop adjacent segment disease regardless of whether or not they have had surgery, as they already lack motion segments. More fused segments will result in less overall flexibility with more stress concentration. More fusion levels thus increase the chance of long-term chronic degeneration, though even a single-level fusion in the lumbar spine can cause preferential transfer of motion to the disc level below the site of fusion (Fig. 13.3) [13].

This focal degeneration may be made symptomatic by participation in vigorous athletics. Symptoms may include axial pain or radicular symptoms such as paresthesias and weakness. ASD can be demonstrated by MRI. There are no studies that have identified specific risk factors among patients with congenital vertebral malformations that lead to ASD. The progression of

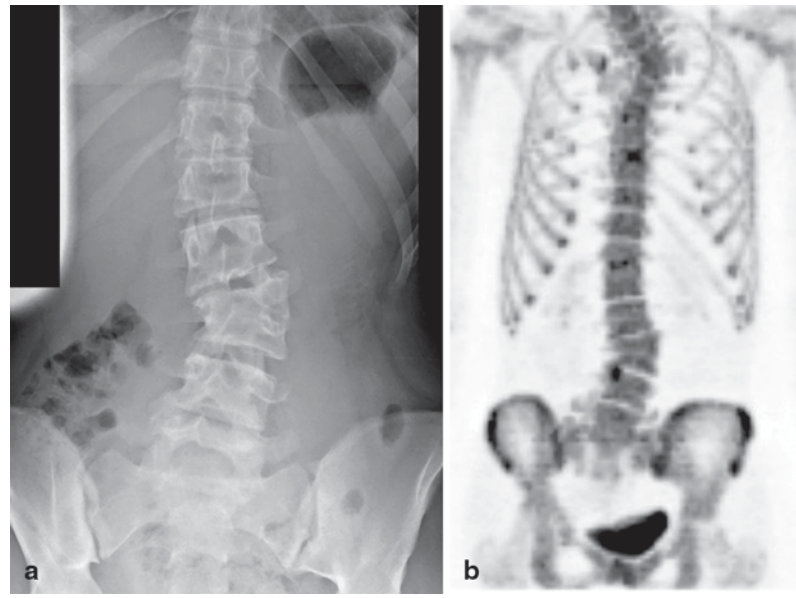

Fig. 13.3 A 14-year-old girl with congenital scoliosis and back pain in the lower lumbar spine (**a**). Bone scan of same patient demonstrates increased uptake immediately adjacent to abnormal vertebra suggesting hypermobility and degenerative change (**b**)

ASD may happen over many years with increasing degeneration and progressive stenosis and instability. Patients with congenital cervical anomalies and Klippel–Feil syndrome (see below) are particularly at risk and should be counseled about the potential need for radiographic follow-up beyond maturity.

Stenosis and Instability

Stenosis of the vertebral canal can develop in children with spinal anomalies due to abnormal growth patterns. While the ring apophyses are responsible for much of the vertical growth of the spine, the neurocentral cartilage located in the posterior two-thirds of the vertebra is responsible for the peripheral growth and development of pedicles and posterior elements. If these posterior elements are not formed correctly, or are iatrogenically compressed with fusion, this may lead to vertebral stenosis. More commonly, stenosis may result over time from ligamentous or facet joint hypertrophy or disc degeneration, hypertrophy, or herniation due to increased motion at segments adjacent to the congenital fusion mass. Increased motion at segments at the ends of or between fusion segments may lead to eventual local spinal instability (Fig. 13.4).

The combination of stenosis and instability increases the risk of acute neurologic injury with even minor trauma. Chronic instability and stenosis may lead to insidious neurologic injury and myelopathy. Unstable segments should be tracked with serial dynamic radiographs or possibly MRIs to monitor progression of disease.

Pulmonary Function

Thoracic deformity, whether idiopathic or congenital, can be associated with significant restrictive lung disease. The distorted thorax has less volume and moves less effectively than a normal chest wall. Early-onset spinal deformity (significant spinal deformity before age 5) can have a particularly profound effect on the developing thorax and is associated with thoracic insufficiency syndrome (TIS), defined by Campbell as the inability of the thorax to support normal respiration or lung growth [7]. TIS is particularly problematic when vertebral anomalies are compounded by rib fusions, further stiffening the chest wall. Thoracic deformity leads to diminished lung volume with loss of functioning alveolar-capillary units which in turn causes abnormal gas exchange, abnormal respiratory muscle function, and secondary hemodynamic and

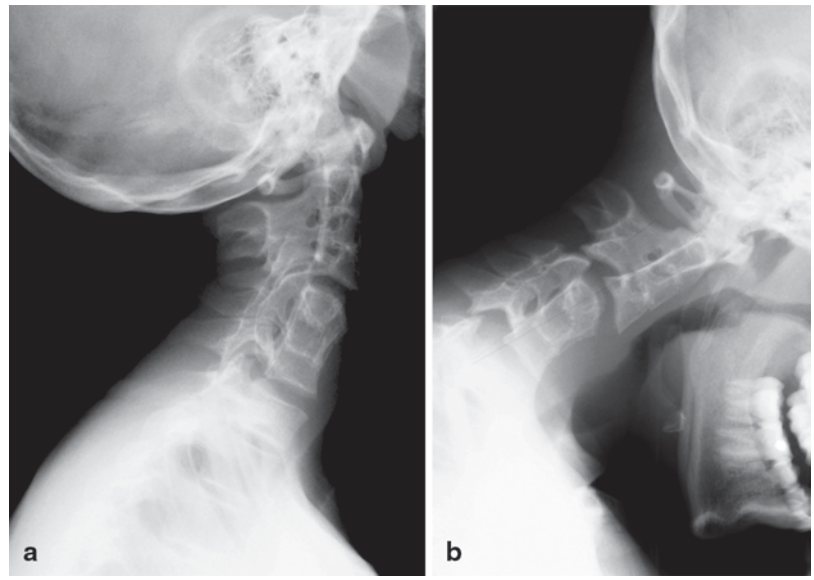

Fig. 13.4 Patient with congenitally fused segments in neck and instability between two fused segments. X-rays demonstrate significant mobility between extension (**a**) and flexion (**b**)

cardiac dysfunction [8]. It has been demonstrated that vital capacity is inversely correlated with Cobb angle, vertebral rotation, and thoracic lordosis [9]. The ventilatory pattern in these patients is that of low tidal volume with high respiratory rate and increased work of breathing. The associated increase in energy required for breathing detracts from the total available oxygen available for the rest of the musculoskeletal system. This extrinsic pattern of restrictive lung disease can lead to hypoxemia due to decreased diffusing capacity. Ultimately, these alterations can lead to pulmonary hypertension and cor pulmonale [9]. Patients with extensive congenital spine and thoracic deformities are thus at risk for developing exercise intolerance and should be followed up by a pulmonologist with physical examination, radiographs, and pulmonary function tests. The pulmonologist may be helpful with decisions regarding sports participation.

Common Treatment Strategies for Congenital Spine Deformity During Growth

Because there is such a wide range of extent and type of congenital spine deformity, treatment varies widely and must be tailored to the individual. Most congenital spine anomalies need no treatment at all, but for some there are common themes of treatment.

Physical Therapy

Although exercises alone are unlikely to benefit the natural history of a congenital deformity during growth, a tailored physical therapy program with targeted strengthening of spinal musculature and appropriate extremity flexibility may greatly diminish the stresses and hence the symptoms associated with adjacent segment disease. Passive stretching or repetitive motion-based exercises intended to increase flexibility in a congenitally stiff area of the spine may have the unintended effect of increasing dangerous instability or hastening degeneration. All exercises applied to congenital deformity should be tailored to the individual anomaly.

Bracing

Although bracing plays a large role in the management of idiopathic spinal asymmetries, congenital vertebral anomalies are usually not flexible and therefore much less successfully treated with bracing [10]. In some cases, 18–20-hour

bracing may be used in order to prevent secondary curves from developing or worsening. As in idiopathic scoliosis, the patient/athlete may wish to use their athletic time as their "off hours," out of the brace, especially if they participate in sports such as swimming, wrestling, or gymnastics where brace wear is not possible during practice or competition [11].

Surgery

Surgery is often needed for progressive congenital deformity and may take many forms. Fusion and instrumentation may involve a short or long segment of spine with different functional consequences created by the congenitally and surgically stiff segment of spine.

Almost invariably, the question arises regarding when the child can return to athletic activity after surgery. There are no universally accepted rules for return to sport after a spinal surgery and the stakes are high, as a trauma after a spinal fusion, particularly in the cervical spine, could result in catastrophic consequences including paralysis. Ultimately, the decision regarding return to sport should be that of the treating surgeon together with the patient and parents. This may depend on the number of motion segments remaining and whether there is enough residual flexibility and potential for dissipation of force proximally and distally to the fusion [12]. A survey of scoliosis surgeons about activity levels after idiopathic scoliosis surgery revealed that 38% of surgeons allow patients to return to gym class at 6 months, 46% allow noncontact sports at 6 months, and 34% allow them at 1 year. Eleven percent of surgeons advise against any contact sports after spinal fusion, but 61% allow contact sports such as soccer and basketball at 1 year. More aggressive contact sports such as American football, wrestling, and ice hockey are allowed by 32% of surgeons at 1 year, while 36% advise against them and 24% forbid them. Football and gymnastics were the two most common sports to be forbidden after fusion for scoliosis [13].

No studies have specifically examined return to sport after fusion for congenital scoliosis, but there are data regarding return to sport after posterior spinal fusion for idiopathic scoliosis, which should be applicable to patients with congenital scoliosis in the absence of other comorbidities. In one study, 59.5% of patients returned to their preoperative level of sport at an average of 7 months and these patients had higher postoperative SRS-22 scores [14]. Importantly, none of these patients had a sport-related complication. Return to athletic activity was significantly correlated with distal level of fusion with a stepwise decline in percentage of patients who returned to athletics from fusions ending at T11 to those ending at L4. Thus, the ability to spare distal motion levels may be an important conversation with a patient who has their heart set on returning to sport. This also indicates that patients with congenital vertebral abnormalities of the lumbar spine may have less likelihood of return to sport after surgical treatment than those whose vertebral anomalies are located in the thoracic spine. Furthermore, the type of sport to which the athlete wants to return is also an important factor. After spinal fusion, patients may find it more difficult to return to a sport that requires significant truncal flexibility (namely gymnastics, cheerleading, and ballet) whereas participating in sports that involve only forward or lateral motion (i.e., running, swimming, basketball etc.) may be more attainable.

Recently, more and more patients with congenital scoliosis are being treated with "growth-friendly" techniques, namely growing rods or vertical expandable prosthetic titanium rib (VEPTR). These techniques are predominantly used in younger children for whom a lot of spine growth is still remaining. These patients may undergo many lengthening surgeries over many years prior to having a final spinal fusion and instrumentation. Rod fractures are common in growing rods, and device anchor point migration is common in VEPTR, but rarely are these events dangerous. Most can be repaired or reinserted at the time of the next elective surgical device lengthening. In spite of the risk of implant fracture or migration, most children are allowed to participate fully in those sports compatible with a relatively immobile spine.

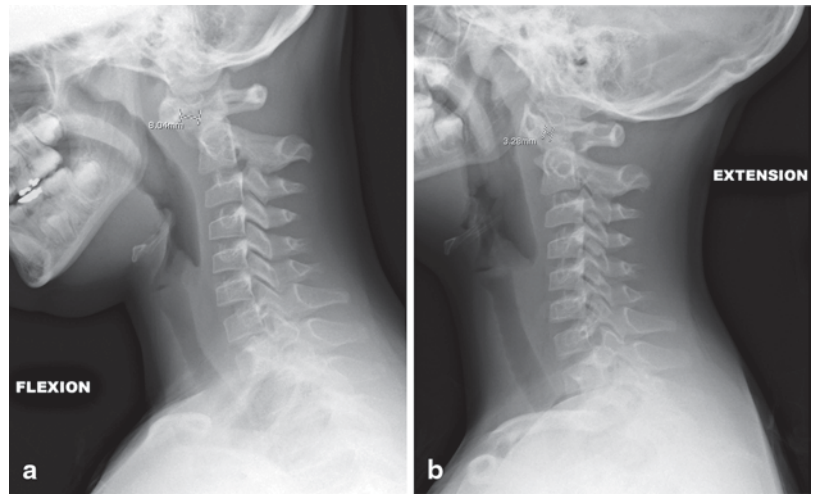

Fig. 13.5 Patient with os odontoideum. X-rays in flexion (**a**) and extension (**b**) demonstrate instability between C1 and C2, with resulting risk of spinal cord injury from subluxation of C1 on C2

Specific Disorders

In this section, we will focus on each separate region of the spine and discuss specific congenital spine processes that may be found in adolescent athletes. We will not address the many spinal anomalies with such severe morbidity as to make it unlikely for those affected to participate in sports, but rather we will focus on the more common conditions that are more likely to appear in young athletes.

Upper Cervical

Osseous anomalies of the upper cervical spine may occur as part of a syndrome or as isolated anomalies. They are important entities due to their fundamental risk of neurologic sequelae, which result from the anomalous vertebra(e) impinging on the space available for the brain stem or spinal cord, with ≤13 mm being abnormal. Compared with adults, children's heads are relatively large and their muscles and ligaments relatively weak, leading to larger acceleration/deceleration forces across the cervical spine and less ability to resist them. Fusion masses may cause hypermobility, instability, disc herniation and stenosis adjacent to the fused segments. It is important to note as well that many of these patients are asymptomatic as children, and may only present in their third or fourth decade of life [22]. Neck pain and stiffness may be the presenting complaint, particularly after a mild hyperextension injury. Neurologic signs and symptoms, such as headache, neck pain, visual and auditory symptoms, weakness and numbness of the extremities, long tract and posterior column signs, ataxia, and nystagmus have also been observed in young patients with craniovertebral anomalies [22]. There are even reports of central cord syndromes in wrestlers and football players with preexisting spinal stenosis [15, 16]. Any of these symptoms should prompt clinical and radiographic evaluation. The presence of stenosis or the potential for instability should prompt strict activity restriction and may require surgical stabilization.

Os Odontoideum

Os odontoideum refers to a dens that is incompletely fused to the body of C2. The exact cause of os odontoideum is controversial, as some believe it is due to errors in the fetal development [17], while most believe it is a result of unrecognized early trauma that causes an avulsion fracture and subsequent nonunion of the odontoid [18]. Whatever the cause, this nonunion can result in atlantoaxial instability, as the superior fragment of the odontoid moves with the anterior arch of C1, without boney connection to C2. Instability is indicated by excessive movement of the fragment in relationship to the body of C2 on lateral flexion–extension X-rays (Fig. 13.5) [19].

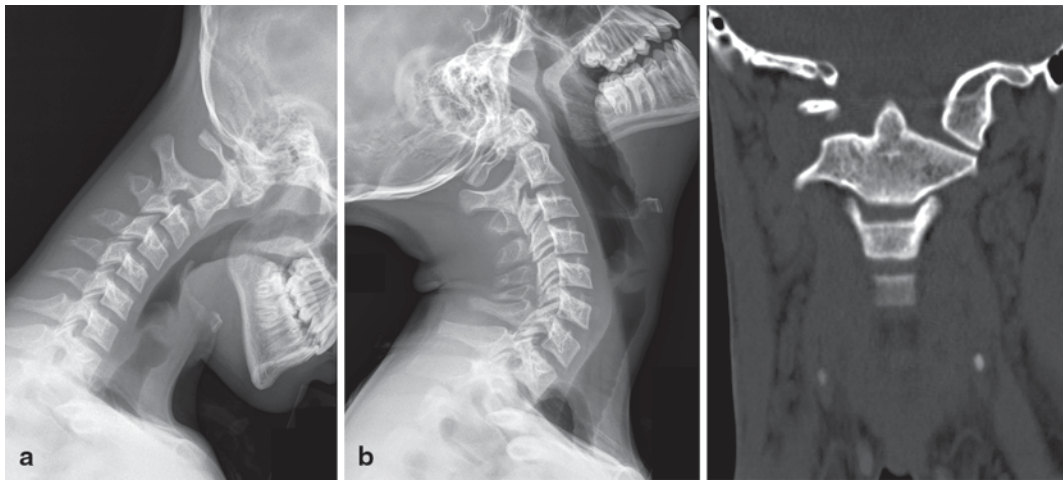

Fig. 13.6 Flexion (**a**) and extension (**b**) X-rays of a 14-year-old boy with congenital occipitoatlantal fusion and compensatory hypermobility at C1–C2, diagnosed after a transient episode of quadriparesis and respiratory distress following a football tackle. The patient regained full motor function after 30 min. CT scan (**c**) demonstrates unilateral osseous fusion of occiput to C1. Transient spinal cord injury occurred at the hypermobile C1–C2 level

Due to the high possibility of C1–C2 instability and spinal cord injury, it is usually recommended that os odontoideum be an absolute contraindication to collision or contact sports and that these patients should be followed up for development of later instability. Odontoid hypoplasia or aplasia can result in similar instability and should similarly be an absolute contraindication [20]. Typical treatment of os odontoideum is C1–C2 fusion.

Occipitoatlantal Fusion

Congenital occipitoatlantal fusion is a failure of segmentation between the fourth occipital and first cervical somites that leads to a partial or complete osseous fusion between the occiput and the atlas anteriorly, posteriorly, laterally, or a combination. The diagnosis is confirmed by lateral radiographs that show no evidence of motion between the occiput and atlas between flexion and extension views. This lack of motion can lead to instability and hypermobility at C1–C2. Occasionally there can be an associated C2–C3 fusion that places added strain on the atlantoaxial articulation [21]. Symptoms, usually presenting during the third or fourth decade, are due to posterior cord compression by the posterior lip of the foramen magnum. While symptoms are usually insidious in onset, sudden death or quadriparesis has been reported. Occipitoatlantal fusion is thus an absolute contraindication to participation in contact sports if the hypermobile segments have not been surgically fused (Fig. 13.6).

Cervicothoracic

Klippel–Feil Syndrome

The unifying feature of Klippel–Feil syndrome (KF) is congenital fusion of some or all of the cervical and/or thoracic vertebrae that results in restricted neck motion, and represents a failure of segmentation during fetal development. KF classically presents as the triad of short neck, low posterior hairline, and limited range of motion in the neck; however, this constellation of symptoms is present in only about half of the patients and the spectrum of disease is large [22]. There is a significant association with congenital scoliosis, documented in as many as 60–78 % of KF patients [23]. Congenital neurologic, cardiac, and renal abnormalities may also occur with KF and thus screening MRI, echocardiogram, and renal ultrasound are warranted.

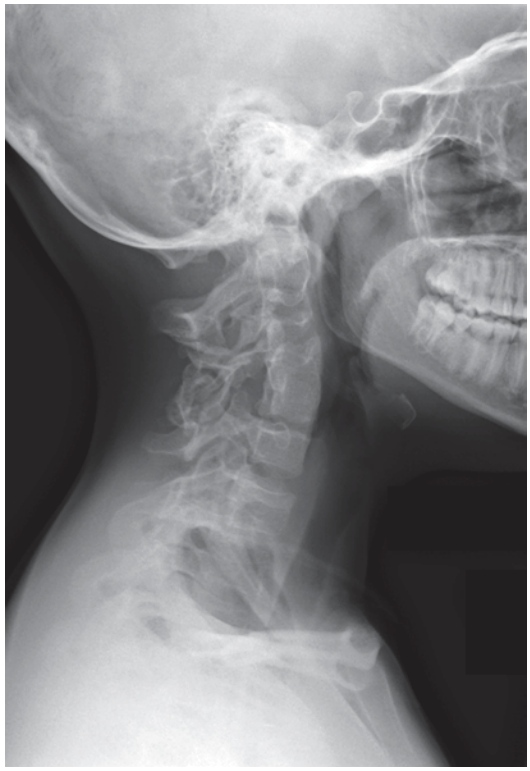

Fig. 13.7 Patient with congenital fusion of C2–C5, type 3 KF. Also see Fig. 4a, b demonstrating type 2 KF with instability between two fused segments

The morphology of and risks associated with KF vary greatly. There are three general patterns of malformations in KF [23]. Type 1 lesions involve a single level of cervical fusion. C2–C3 or C5–C6 is the interspace most commonly fused. Type 2 lesions demonstrate multiple noncontiguous congenitally fused segments. Type 3 lesions have multiple contiguous congenitally fused segments (Fig. 13.7).

Regardless of the number of fusion levels, many of these patients may not have symptoms at all (64% in a series by Samartzis), but the patients who do become symptomatic tend to present with symptoms in the late teenage years [23].

KF has the potential to cause devastating neurologic consequences after minor trauma due to the potential for unstable patterns of deformity. There have been reports, for example, of central cord syndrome resulting from falls during skiing in patients with KF [24]. Even without trauma, neurologic deficits, such as weakness, hyperreflexia, paresthesias, and bladder dysfunction, may occur due to central spinal stenosis, disc herniation, osteophytes, stenosis of intervertebral foramina, associated abnormalities of the central nervous system, or hypermobility of adjacent segments [25, 26]. Potentially fatal subluxations may occur at these hypermobile levels in the setting of trauma or sports collisions.

Type 2 or 3 KF is an absolute contraindication to most contact sports that might involve neck flexion due to the severely abnormal mechanics adjacent to or between large fused segments. Type 1 lesions are an absolute contraindication to collision sports in the setting of high cervical fusions (above C3) with associated instability, occipitocervical abnormalities, disc disease, degenerative changes, or spinal canal stenosis [21, 26]. On the other hand, type 1 lesions involving fusion of one interspace at or below C3 in a patient with full range of motion and no instability, disc disease or degenerative changes may have minimal increased risk of catastrophic injury and should not be considered an absolute contraindication to contact or collision sports [21, 27].

Thoracolumbar

Congenital Scoliosis

With an incidence of 1 in 1,000 births, congenital scoliosis is the most common of all congenital spine malformations (Fig. 13.8).

While vertebral malformations may be an isolated finding, congenital heart diseases, such as ventricular or atrial septic defects, Tetralogy of Fallot, and transposition of the great vessels, occur in up to 26% of patients with congenital scoliosis. Likewise, urologic abnormalities, such as unilateral renal aplasia (most common), horseshoe kidney, duplicate ureters, ureteral obstruction, or renal ectopia, may be found in up to 34% of patients [31].

There is a wide range of severity among patients with congenital scoliosis. Patients with a focal area of mild malformation (for example, a single block vertebra) or a symmetric abnormality (hemimetameric shift) may be asymptomatic. On the other hand, patients with a sharp and rigid

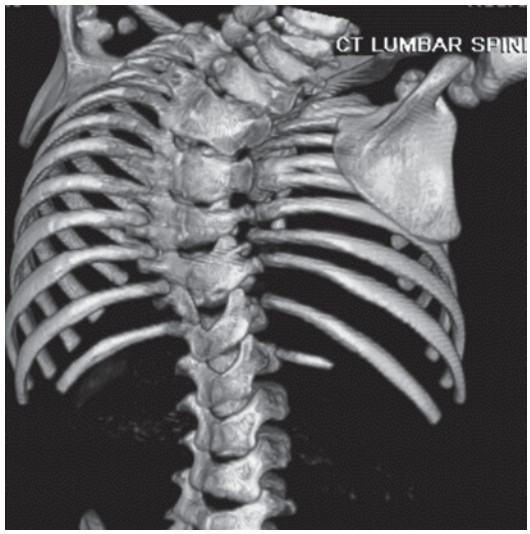

Fig. 13.8 Congenital scoliosis with multiple abnormal thoracic segments demonstrating both failures of formation and segmentation

curve will most likely require surgical treatment, often at a young age. Surgical options to control deformity include in situ fusion to prevent curve progression, hemiepiphysiodesis or hemiarthrodesis to slowly improve deformity, hemivertebra or wedge vertebra resection with fusion and instrumentation to acutely correct deformity, and growth-sparing surgery with growing rods for cases without rib fusions and VEPTR placement for cases requiring expansion thoracostomy for rib fusions.

Long-term follow-up studies have compared congenital scoliosis patients treated with early spinal fusion to their healthy peers and found that patients with congenital scoliosis had shorter spines, worse pulmonary function, and more pain. They also had lower physical functioning and physical summary values as compared with healthy kids. That said, follow-up data at 16 years showed no significant differences vs. normal [28].

Congenital Kyphosis

Congenital kyphosis, like congenital scoliosis, results from either failure of segmentation or failure of formation of vertebral bodies, though the failure of formation or segmentation tends to occur anteriorly or anterolaterally leading to deformity in the sagittal plane (Fig. 13.9). Sagittal plane deformity usually occurs along with coronal plane deformity creating kyphoscoliosis, though kyphosis may occur on its own. The apex of deformity is most commonly between T10 and L1 [29].

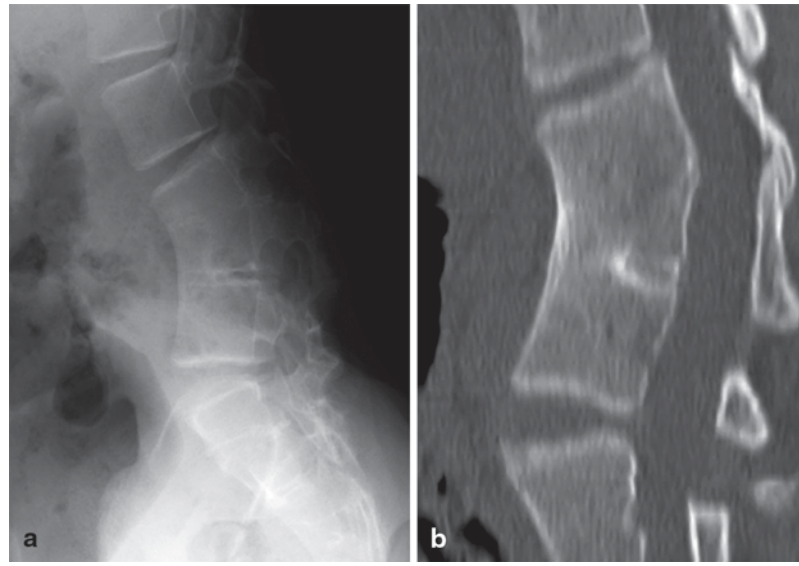

Fig. 13.9 X-ray (**a**) and CT (**b**) of a patient with congenital kyphosis with low lumbar pain due to abnormal mechanics at the hyperextended L5–S1 segment below the fusion mass

Congenital kyphosis is much less common than congenital scoliosis, but does have similar associations with cardiac and renal anomalies. One study found congenital kyphosis to have a higher incidence of intraspinal anomalies than congenital scoliosis [30]. Three types of congenital kyphosis are classically described. Type 1 is an anterior failure of vertebral body formation, type 2 is an anterior failure of vertebral body segmentation, and type 3 is a mixture of the two. Type 1 and type 3 are particularly concerning because cord compression and paraplegia may ensue. These types should be considered a contraindication to contact or collision sports prior to fusion. As with congenital scoliosis, brace treatment is usually ineffective and surgical treatment is often indicated, particularly before the kyphosis exceeds 50 degrees. Patients who have been surgically fused are no longer at significant risk for paralysis but should be subject to post-fusion restrictions on activity.

Lumbosacral

Spina Bifida Occulta

Spina bifida comes in two main types, aperta and occulta. Spina bifida aperta (commonly called "spina bifida" or "myelomeningocele") involves a protrusion of the meninges, with or without neural elements, and can cause profound neurologic deficits distal to the level of the defect and is usually associated with hydrocephalus. Spina bifida aperta will not be covered in this chapter. Spina bifida occulta (SBO), the mildest form of spina bifida, involves a small congenital defect in the posterior elements of the vertebral column but without any protrusion of neural elements, and thus no neurologic deficit and no risk of damage to the spinal cord (Fig. 13.10).

SBO does not appear to cause pain or other symptoms and is usually found incidentally on radiographs. In a study that examined high school and college athletes with SBO, the incidence of back pain was not significantly different from those with no radiologic abnormality [31]. Another study evaluated 120 elite skiers, 26% of whom had SBO. Of those 26% (31 of 120), 10% experienced back pain, which was not significantly different from those elite skiers who did not have SBO (13%) [32]. There is evidence, however, that suggests a higher incidence of posterior disc herniation with SBO [33]. SBO should not be a contraindication to athletic activity and requires no special precautions.

Lumbosacral Transitional Vertebrae

These vertebral anomalies are present in 3–21% of people and involve an abnormal connection between the transverse process of the lowest lumbar vertebra and the sacrum, which may contribute to the development of low back pain [34]. Low back pain associated with this anomaly is called Bertolotti's syndrome (Fig. 13.11).

Lumbosacral transitional vertebrae (LSTVs) are characterized according to the classification system by Castellvi. In type I, there is a large transverse process at the lowest lumbar vertebra (a, unilateral; b, bilateral). In type II, there is a diarthrodial joint between the transverse process and the sacrum (a, unilateral; b, bilateral). In type III, there is a true boney union between the transverse process and the sacrum (a, unilateral; b, bilateral). And in type IV, there is a type II malformation on one side and a type III malformation on the other [35].

The abnormal lumbosacral connection can lead to hypermobility and abnormal stresses at the superior disc space and the articulation between the transitional transverse process and the sacrum. These abnormal stresses can subsequently lead to disc degeneration and pain, particularly with repetitive flexion and extension activity [36, 37]. Thus, the presence of a transitional vertebra, while posing no contraindication to sporting activities, may make it more likely for the young athlete to develop early degenerative changes at or around the level of the transitional vertebra, particularly in sports such as gymnastics and wrestling, which may place increased strain and torsional stress through the lower lum-

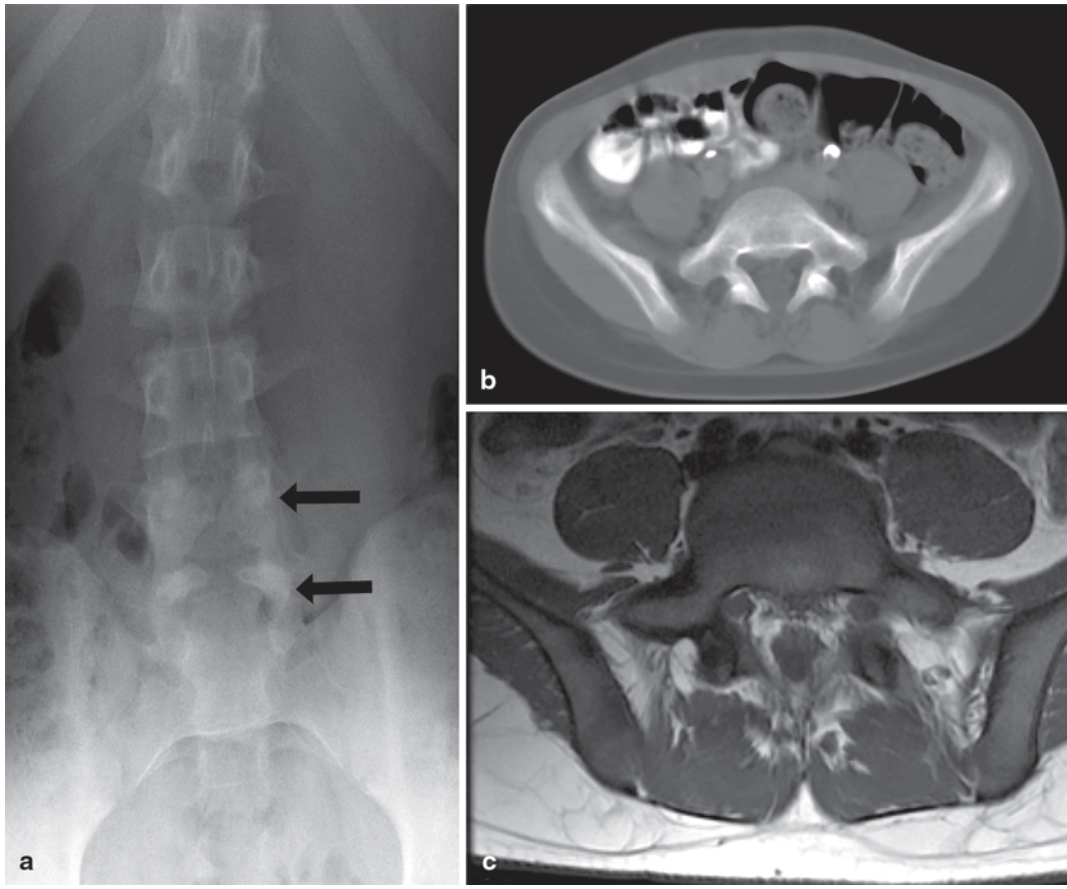

Fig. 13.10 AP lumbar spine of patient with posterior element defect at L4 and L5 (*arrows*) (**a**). Corresponding axial CT scan (**b**) and typical MRI (**c**). The SBO is asymptomatic and needs no specific treatment or restrictions

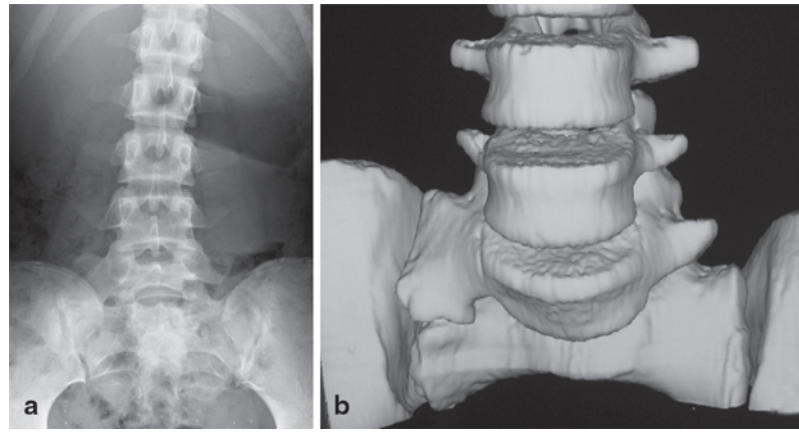

Fig. 13.11 A 19-year-old girl who complains of right-sided low back pain. Transitional vertebra demonstrated on right side at L5 on X-ray (**a**) and corresponding 3D CT (**b**)

bar spine. Treatment of Bertolotti's syndrome may include the use of a lumbosacral orthosis, physical therapy to stretch and strengthen the spine, and sport-specific training to avoid or limit exposure to the mechanism of injury [37].

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The Young Athlete with Down Syndrome

14

Benjamin J. Shore

Background

In 1866, J. Langdon Down first published his observations on a series of children with the syndrome that bears his name [1]. With an incidence of 1 in 660 live births, Down syndrome (DS) is the most common chromosome abnormality among live-born infants [2] and is the most frequent form of intellectual disability (mental retardation) caused by a microscopically demonstrable chromosomal aberration [3]. The risk of having a child with DS increases with maternal age, from 1 in 5,000 live births for mothers between 15 and 29 to 1 in 50 live births for those older than 45 years [4].

The diagnosis of DS is made by chromosomal analysis, either prenatally or after birth. Currently, screening begins with a blood test that is offered to all pregnant women, followed by cytogenic diagnosis with chorionic villus sampling or amniocentesis, if needed. After birth, a karyotype can be performed with either a blood or a tissue sample [5]. Trisomy of chromosome 21 is the most common cause of DS (95%). The genes that code for collagen IV are located on chromosome 21 and an alteration in type IV collagen is believed to be responsible for joint/ligamentous laxity seen in children with DS [4]. Children with DS exhibit a variable phenotype with multiple malformations, including some degree of mental impairment (average intelligence quotient (IQ) 50), generalized ligamentous laxity, hypotonia, and a characteristic facial appearance, with upwardly slanted palpebral fissures, epicanthal folds, and a round flat face (Table 14.1) [6].

Sports for the Physically and Mentally Disabled

Participation opportunities for physically and mentally challenged athletes have increased over the past several decades, with organized games such as the Paralympics and Special Olympics. The Paralympic games include athletes with spinal cord injuries, limb amputations, cerebral palsy, blindness, and other visual impairments. Athletes with intellectual disabilities (IQ < 70) participate in the Special Olympics.

There are an estimated 3 million Special Olympic athletes worldwide, with many more involved in recreational activities [7]. The most popular sports for these athletes are track and field events, soccer, basketball, bowling, and aquatics [8]. The primary goals of the Special Olympics are to: (1) promote a healthy competitive spirit, (2) develop leadership and self-esteem, (3) facilitate physiologic health through the improvement of the strength and endurance

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Table 14.1 Common phenotypic features in Down syndrome [2]

Facial features
Flat nasal bridge
Epicanthanl folds
Upward-slanting palpebral fissures
Open mouth
Hand abnormalities
Small finger hypoplasia
Family finger clinodactyly
Single, deep palmar crease (simian crease)
Characteristic pelvis with lateral flare of iliac wings
Joint hypermobility
Ligamentous laxity
Hypotonia
Short stature
Mental impairment

of the neuromuscular and cardiovascular systems, (4) nurture positive mental attitudes, and (5) encourage a life-long habit of physical activity to improve overall quality of life [9, 10].

Classifying athletes with disabilities helps level the playing field so that athletes with similar functional abilities can compete with each other [11]. According to the American Association of Intellectual and Developmental Disabilities (AAID), intellectual disability "is a disability characterized by significant limitation both in intellectual functioning and in adaptive behavior as expressed in conceptual, social and practical adaptive skills" [12]. Sensory, motor, communication, or behavioral factors should also be appropriately considered in cognitive assessment and interpretation of results of cognitive tests [12]. In addition to medical doctors, athletic trainers and specially trained certified classification specialists are responsible for applying this classification [13].

Athletes with DS participate in sports under the umbrella of Special Olympics. Hearing and visual impairments are prevalent in persons who have intellectual disability [14, 15, 16]. In general, persons who have intellectual disability have been shown to score lower than those who do not have intellectual disability on measures of strength, endurance, agility, balance, running speed, flexibility, and reaction time [17, 18]. Persons who have intellectual disability also tend to have lower peak heart rate and lower peak oxygen uptake (VO_2 peak) than those who do not have intellectual disability. All of these medical conditions and unique medial characteristics must be carefully examined on a patient's preparticipation examination (PPE).

The Role of the Physician in Supporting Sports Participation in DS—The PPE

PPE is an essential component of injury and illness prevention in athletes [19]. Recommendations for the elements of the PPE for able-bodied athletes have been developed by a number of organizations, including the American Academy of Family Physicians, American Academy of Pediatrics, American Medical Society for Sports Medicine, American Orthopaedic Society for Sports Medicine, and the American Academy of Orthopaedic Surgeons [20]. However, the critical elements of the PPE for athletes with a physical or mental disability are less clearly delineated. As a result, many athletes with DS are cleared for participation by a personal physician who is not specifically trained to care for an athlete with the disorders associated with DS [3].

The incidence of sports-significant abnormalities found on screening history and physical examination in the nondisabled, noncognitively delayed population is 1–3 %. On the contrary, the incidence may be as high as 40 % in the Special Olympics population [9]. In general, the PPE for the physically challenged athlete should be tailored to the individual's disability and to his or her specific condition. The PPE is used not only to identify athletes at risk for injury and comorbidity but also to assist in establishing equal competition among athletes. A detailed medical history is the cornerstone of any PPE. In athletes with disability, a team of medical professionals who are involved in the longitudinal care of these athletes and who understand baseline physical and cognitive levels of functioning are most qualified to complete PPE for athletes with disabilities [8]. These athletes should be examined in an office setting, and the mass station method

for PPE completion should be avoided. Examiners should be cognizant of the disability-specific medical issues for the DS athlete when completing the PPE.

Common Medical Problems to Consider in DS

Important factors relevant to sports participation in children with DS include hearing loss, cardiovascular disorders, pulmonary hypoplasia, obesity, and immunologic deficiency. Screening for these conditions, managing associated medical problems, and educating families can significantly improve their level of function [21]. Children with DS meet motor milestones later than children without DS do; for example, children with DS often have poor coordination and on average walk a year later than children without DS do. Children with DS have impaired IQ (range 25–50), with variable developmental delay; however, they perform better socially than expected for their mental age [5]. Medical management requires an organized approach to the initial and ongoing evaluation and monitoring for associated abnormalities and prevention of common disorders. (Table 14.2)

Table 14.2 Medical conditions in patients with DS [2, 5]

Condition (frequency %)
Cardiac abnormalities
Congenital heart disease (50)
Atrioventricular septal defect (45)
Ventricular septal defect (35)
Patent ductus arteriosus (7)
Tetralogy of Fallot (4)
Leukemia
Leukemia (1)
Acute lymphoblastic leukemia (0.33)
Acute myeloid leukemia (0.33)
Otolaryngologic abnormalities
Hearing loss (75)
Otitis media (50–75)
Obstructive sleep apnea (50–75)
Ophthamologic disorders
Refractive errors (35–75)
Strabismus (27–57)
Congenital cataracts
Gastrointestinal disorders
Gastrointestinal atresias (12)
Celiac disease (7)
Hirschsprung disease (1)
Skin disorders (87)
Neurologic and psychiatric disorders
Mental impairment
Seizures (8)
Disruptive behavior disorders (17)
Endocrine disorders
Diabetes mellitus (1)
Hypothyroidism (15)

Cardiac Disease

Approximately 50 % of individuals with DS have congenital heart disease that is surgically treated in infancy or childhood and it is important to obtain a detailed cardiac history during PPE [22]. All patients with DS should be evaluated for congenital heart disease in consultation with a pediatric cardiologist. An echocardiogram is recommended to detect abnormalities that may not be symptomatic or apparent on physical examination. Continued clinical cardiac evaluation is needed because of the high risk of mitral valve prolapse and aortic regurgitation in adolescents and young adults [2]. Specific cardiac follow-up is tailored according to the patient's cardiac defects and the follow-up schedule is established by treating cardiologist [23].

In those persons who have pulmonary hypertension, right to left shunting, and/or chronic hypoxia, the physician's advice to pursue athletic endeavors or sports that do not require high cardiac outputs and potentially increase the degree of hypoxia is critical for safe participation in sport for that individual. Physical examination and echocardiogram are useful screening tools to identify significant underlying pathology. In general, 20–30 min of aerobic exercise (walking, running, swimming, biking, and cross-country skiing) three times a week is recommended. A number of studies have looked at the cardiorespiratory capacity of individuals with DS and found that these individuals have lower cardiovascular fitness levels than their peers without

DS, including peers who have forms of mental retardation other than DS [17, 24]. These studies show that individuals with DS have a reduced peak VO_2 and lower peak heart rates when compared to the general population and others with mental retardation. Specifically, peak heart rates in those with mental retardation (not DS) are 10 % lower than the general population (adjusted for age); however, the peak heart rate for those with DS is 15 %, which is lower than that found for non-DS population with mental retardation [17, 24]. Despite these limitations, a systematic literature review suggests that there is evidence that individuals with DS can improve their cardiovascular fitness through exercise program [25] and the current American College of Sports Medicine (ACSM) guidelines recommend cardiovascular training programs for individuals with DS.

Hearing and Ophthalmologic Disorders

Approximately 75 % of children with DS suffer from congenital hearing loss. Therefore, all newborns should undergo a newborn hearing screen with brainstem auditory evoked response (BAER) [2]. Children with DS should have repeat hearing screening every 6 months until 4 or 5 years of age and then yearly. In addition, many children suffer from otitis media (50–75 %), which can further compromise hearing function. Impaired hearing can also relate to vestibular dysfunction and balance problems that may limit a child's ability to climb, jump, dive, and tumble [23].

Children with DS have a 50 % risk of developing refractive eyesight error between the ages of 3 and 5 years [23]. Unaffected children with DS should be examined annually before age 5 to detect refractive errors that may occur during childhood. Additionally, examination is recommended every 2 years after age 5 (every 3 years after age 13) to screen for disorders, including keratoconus and lens opacities that may develop in adolescents or adults [22]. Uncorrected vision impairment may result in increased clumsiness and poor performance during sporting events.

Obstructive Sleep Apnea

Children with DS have an increased risk of obstructive sleep apnea because of soft tissue and skeletal alterations that lead to upper airway obstruction. During the PPE, symptoms related to sleep apnea should be questioned including the presence of snoring, heavy breathing, restless sleep, altered sleep position, daytime sleepiness, apneic pauses, and frequent night awakening [23]. There is poor correlation between parent report and polysomnogram results [26]. Therefore, referral to a pediatric sleep laboratory for a sleep study of polysomnogram for all children with DS by 4 years of age is recommended [23, 27]. It is important to discuss the association and risk factor of obesity and sleep apnea with all parents of DS children [27].

Common Orthopedic Considerations in DS

A number of musculoskeletal abnormalities are seen in patients with DS. Many of these are related to generalized ligamentous laxity and joint hypermobility. Persons with DS have abnormal collagen that results in increased ligamentous laxity and decreased muscle tone [21, 28, 29]. Areas of concern include cervical spine, hip and patellofemoral instabilities, and foot deformities.

Cervical Spine Instability

The major cervical spine concern in DS is instability and should be carefully assessed during the PPE. Atlantoaxial instability (AAI) has been reported in 15 % of persons with DS, as determined by radiographic evidence on a lateral X-ray of an atlantodens interval (ADI) of 4.5 mm or more [30] (Fig. 14.1a-c). This increase in distance signifies an excess of ligamentous laxity of the transverse and alar ligaments of the atlantoaxial joint [28]. Unfortunately, the instability cannot be detected by examination or clinical criteria alone [31]. Approximately 2 % of persons with AAI may be symptomatic because of sublux-

ation. Unfortunately, it is possible that a patient's first presentation of AAI can be catastrophic with quadriplegia and death from undiagnosed AAI after a severe hyperflexion/hyperextension injury [32]. There are at least 13 cases of acute posttraumatic neurologic deficit related to AAI in DS in the literature [29, 32, 33, 34].

Although most patients with radiographic evidence of AAI have no neurologic defects, symptoms may occur as the space available for the spinal cord becomes < 13 mm [13]. Symptomatic AAI patients often present between ages 5 and 15 and may report symptoms of easy fatigability, abnormal gait, neck pain, incoordination, sensory deficits, and bladder control problems [5, 6, 9, 22, 28, 35, 36, 37]. On physical examination, a variety of clinical signs can be present including wide-based gait, torticollis, decreased neck range of motion, incoordination, weakness, spasticity, hyperreflexia, clonus, extensor plantar reflex (abnormal Babinski reflex), and other upper motor neuron and posterior column signs [5, 22, 28, 31, 38, 39]. Any of these complaints or physical signs are concerning and the child should be urgently referred to a pediatric orthopedic surgeon or pediatric neurosurgeon for management. In addition, urgent evaluation of the spinal cord with magnetic resonance imaging (MRI) to detect signal changes within the cord is also critical. Generally, a neural MRI is sufficient to document signal changes within the cord; however, in certain circumstances, a flexion/extension MRI is necessary to demonstrate the appropriate pathology [6, 39].

Most children with DS and instability at C1–C2 are asymptomatic. Long-term studies demonstrate that only minor changes in the ADI develop over time in most patients with atlantoaxial abnormality [28]. Historically, it has been common practice to obtain lateral cervical spine radiographs in flexion, extension, and neutral positions to screen for asymptomatic AAI. However, the correlation of the radiographic diagnosis of AAI with neurologic abnormalities is not well established [40]. Historically, the observation of radiographic abnormalities in individuals with DS resulted in overtreatment and unnecessary physical restrictions based solely on radiographic findings [35].

Once asymptomatic AAI has been discovered (ADI > 4.5 mm), the treatment involves close observation and adherence to the restrictions for contact sports instituted by the Special Olympics [9, 41]. Even this suggestion of activity restriction is controversial as there are no reported cases in the literature of patients with isolated radiographically documented asymptomatic AAI sustaining a neurologic injury secondary to sports participation [42, 43 44]. In asymptomatic children whose ADI is greater than 9 mm, the space available for the cord is compromised and surgical stabilization is recommended even in the neurologically normal child [39]. For symptomatic AAI with abnormal radiographic evaluation, surgical fusion is warranted [45, 46, 47] (Fig. 14.1d).

Upper cervical spine instability in DS can occur not only at the atlantoaxial joint (C1–C2) but also at the occiput–C1 junction. Wills and Dorman [48] found that true occiput–C1 instability was underappreciated on most plain radiographs in DS; however, the authors recognized that the true incidence of instability at this level is unknown. Instability at the occiput–C1 level in patients with DS likely results from laxity of the atlantooccipital joint capsule, the tectorial membrance, and the anterior and posterior atlantooccipital membranes [49]. Although Power's ratio is a standard method for assessment of occiput–C1 instability in skeletally mature patients, Karol et al. [50] found that in DS, this measurement was not reliable and recommended MRI to confirm instability in children and adolescents with DS. Power's ratio is calculated by drawing a line from the Basion (B) to the posterior arch of the atlas (C) and a second line from the Opisthion (O) to the anterior arch of the atlas (A). The length of line BC is divided by the length of line OA. A ratio greater than 1.0 demonstrates anterior atlantooccipital dislocation (Fig. 14.2) [48]. Once instability is identified, treatment is similar to AAI instability with cervical occiput fusion.

Significant controversy exists with regard to the need for screening for cervical spine instability in children with DS. The primary concern with cervical spine screening is the lack of defined

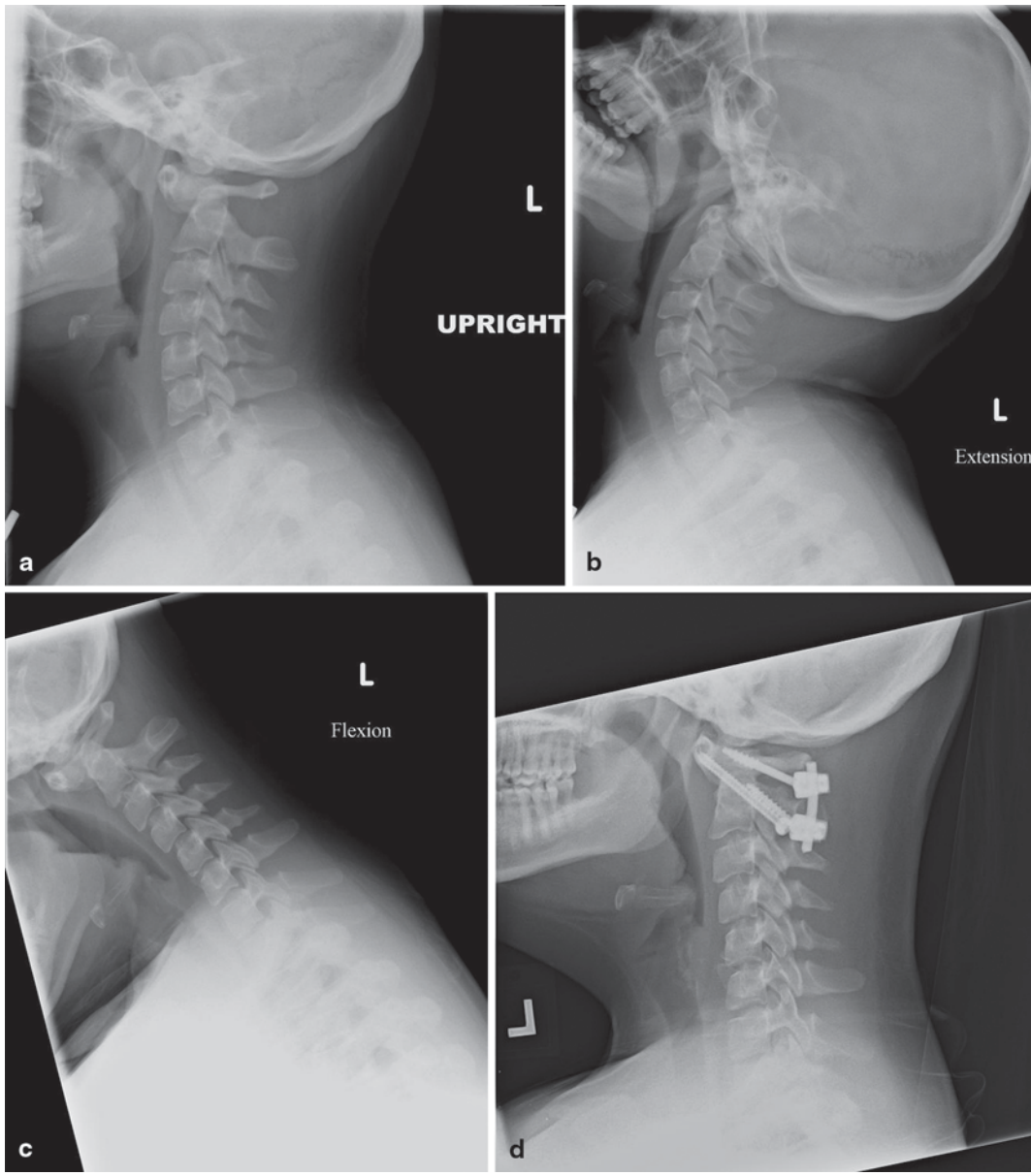

Fig. 14.1 Neutral **a** extension **b** and flexion **c** radiographs of a 16-year-old girl with DS, demonstrating severe AAI that was symptomatic. AAI is measured from the posterior border of the anterior *arch* of C1 to the anterior *border* of C2 as indicated by the *red arrows*. An atlantodens interval (ADI) greater than 5 mm is considered abnormal. **d** Postoperative lateral cervical radiograph of the same 16-year-old girl demonstrating in-situ fusion

efficacy and questionable cost effectiveness of this intervention within the DS population. Recommendations span the spectrum from no screening, to screening on an annual basis. In 1995, the American Academy of Pediatrics (AAP) retired its 1984 recommendation to obtain a screening cervical spine radiograph before sports participation [29]. The members argued that lateral radiographs have low reproducibility, leading to decreased sensitivity and specificity in detecting instability. Furthermore, the authors found that effective, low-risk treatment to prevent progres-

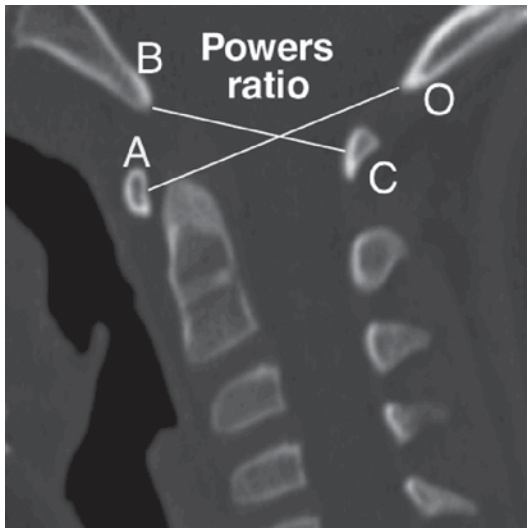

Fig. 14.2 Power's ratio. Calculated by drawing a line from the basion *(B)* to the posterior arch of the atlas *(C)* and a second line from the opisthion *(O)* to the anterior arch of the atlas *(A)*. The length of line BC is divided by the length of line OA. A ratio greater than 1.0 demonstrates anterior atlantooccipital dislocation

sion from asymptomatic to symptomatic cervical instability was not available [29]. Their current opinion is that a yearly examination with inquiry about neurologic symptoms or with detection of signs of myelopathy on clinical examination is more useful and predictive of progressive myelopathy or neurologic injury than a screening radiograph is. In asymptomatic patients, the role for lateral flexion and extension radiographs is limited to those cases where signs or symptoms of instability are detected on history or physical examination.

Although the AAP has published suggested guidelines for cervical spine screening in DS, not all experts agree with their position statement [43]. Pueschel [51] favors screening cervical spine radiographs in all children with DS. He identified several reasons for continued radiographs, including increased frequency of instability within the population and risk of progression of neurologic symptoms. Furthermore, the Special Olympics requires that all athletes with DS be screened by lateral cervical spine radiographs in full flexion and full extension prior to participation in sports considered to have potential risk (diving, pentathlon, high jump, equestrian, soccer, gymnastics, and certain swimming events) [41]. Interestingly, this restriction can be waived by an acknowledgement of the risks signed by either an adult athlete, or his/her parent or guardian if the athlete is a minor, and if two physicians, excluding the examining physician of record, give written certification [10].

In addition, individuals with DS who are scheduled to undergo otolaryngologic surgical procedures or general anesthesia are thought to be at similar risk for spinal cord injury to those persons who wish to participate in high-risk sports [52, 53]. Neither evidence-based guidelines nor a consensus statement exists in the anesthesia literature regarding the inclusion of cervical spine radiographs in the preoperative assessment of patients with DS. Litman et al. [54] surveyed 117 pediatric anesthetists and found that 18% obtain preoperative radiographs and 9% obtain subspecialty consultation in asymptomatic children with DS. While for symptomatic DS children, 64% would obtain a radiograph and 74% would seek preoperative subspecialty consultation prior to proceeding with surgery. Hata and Todd [55], upon searching the literature, found eight possible cases of cervical spine injury associated with anesthesia in patients with DS, and recommended a thorough preoperative assessment by the anesthesiologist, who should look specifically for signs and symptoms of cervical spine compression.

Hip Instability

The rate of hip instability in DS is between 2–7% [36, 56] and considered to be multifactorial, related to ligamentous laxity with associated femoral anteversion and acetabular retroversion [57]. Unlike patients with developmental dysplasia of the hip, patients with DS who have dislocation typically have stable hips before walking age; hip instability appears spontaneously between the ages of 2 and 10. The natural history of established hip instability is progression from acute dislocation to recurrent dislocation, habitual dislocation, and fixed dislocation [58, 59]. Initially painless,

secondary bone changes develop (primarily acetabular) and then the dislocation can become painful. As the life expectancy of patients with DS increases, the incidence of painful arthritis in adulthood is also rising [58].

Hip instability in DS is divided into acute and habitual dislocation groups. Athletes often present in the acute dislocation phase at age 7 or 8 with refusal to weight bear or limp [5]. These hips can often be reduced under general anesthesia with a closed reduction maneuver. Children with habitual hip dislocation have hips that dislocate without trauma and spontaneously reduce with or without a period of limp and hip pain.

Definitive management of hip instability is surgical, with combinations of femoral and pelvic osteotomies and associated capsular plication. In patients with a normal appearing acetabulum, femoral varus derotation osteotomy has been advocated to improve stability [56, 60]. Acetabular dysplasia and deficiency can be addressed through a variety of pelvic osteotomies with good medium-term follow-up [61, 62, 63]. After appropriate postoperative rehabilitation, resumption of full sports activity is a reasonable goal.

Patellofemoral Instability

Approximately 20% of patients with DS suffer from patellofemoral instability [64, 65]. Hypermobile patella resulting in subluxations and dislocations of the patellofemoral joint has been attributed to a generalized laxity of tissues and hypotonia of the muscles that restrain the patella within the intercondylar groove [65]. Most DS children with patellar instability may not complain of pain, but will demonstrate gradual gait disturbance. Pain in children with DS is under reported, yet children with DS are not insensate to pain. It should be noted, however, that they do express pain more slowly and less precisely than the general population [66]. Pain does appear to be related to the degree of patellar instability. In prior studies, it was noted that children with mild instability were found to have no measureable pain, whereas 25–60% of patients with dislocated patella had pain [65, 67, 68]. History may reveal frequent falls associated with acute knee swelling, progressive patellar abnormality, and unsteadiness with walking.

Table 14.3 A classification of patellofemoral instability [67]

Grade	Patellofemoral instability
Grade 1	Normal laxity
Grade 2	Subluxates >50% of patellar width—no dislocation
Grade 3	Dislocatable
Grade 4	Dislocated but reducible
Grade 5	Dislocated and irreducible

In 1986, Dugdale and Renshaw [67] presented a classification of patellofemoral instability in DS patients (Table 14.3). Frank patellar dislocation (Grade 4 and 5) is found in 2–10% of patients with DS [64, 65, 67, 69]. Treatment of patellofemoral instability in patients with DS begins with nonsurgical management. Mendez et al. [65] found that nonsurgical management including physical therapy, orthotics, and braces was effective in maintaining or improving ambulation in most patients who were ambulatory before treatment. However, in patients whose ambulation was already deteriorating, nonsurgical management did not improve ambulation for these patients. The current standard for nonsurgical management includes a neoprene knee sleeve or a soft patellar tendon orthosis, activity modifications, and physical therapy including vastus medialis strengthening and functional electrical stimulation.

The deformities associated with long-term untreated patellar dislocations have been well documented and most commonly involve a combination of genu valgum, external tibial torsion, knee flexion contractures, and variable degenerative arthritis [65, 70–72]. There are only a handful of studies, which report the surgical results for the treatment of patellar dislocation in DS; unfortunately, each study reports a different surgical technique with variable outcome [65, 67, 68, 73] (Fig. 14.3a, b).

In DS, Mendez et al. [65] was first to report a graded surgical approach to patellar instability

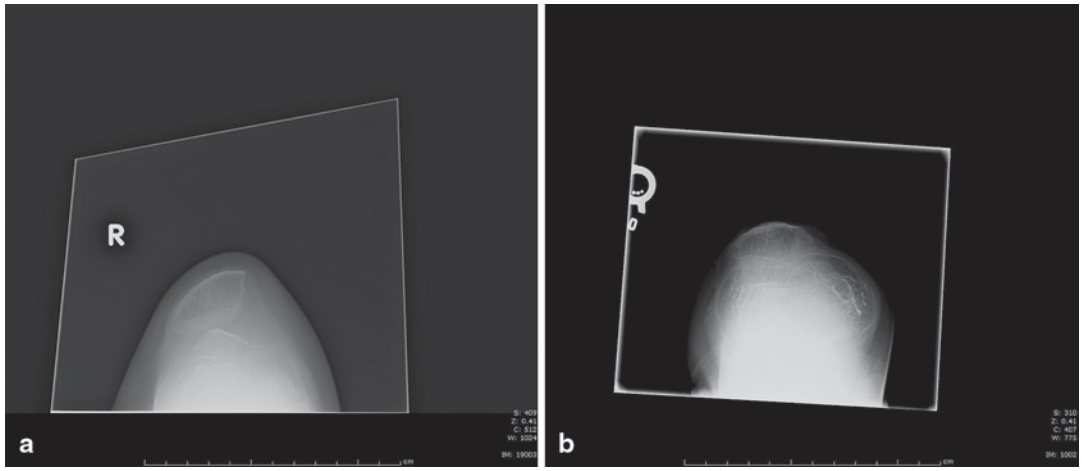

Fig. 14.3 Preoperative **a** and postoperative **b** radiographs of a 12-year-old boy with DS demonstrating symptomatic patellofemoral instability with a dislocated lateral patella. His patella was stabilized with a combination of Roux–Goldthwait reconstructions, lateral release and medial plication. He is asymptomatic and pain-free at the current time

using the Dugdale classification. They recommended a lateral release and medial plication for Grade 2 and 3 patellar instability, while reserving a Roux–Goldthwait type procedure for those children with Grade 4 and 5 instability. Bettuzzi et al. [68] reported that for children with DS, a double stabilization procedure was necessary using both a distal Roux–Goldthwait and a proximal checkrein using a strip of the medial capsule sutured around the quadriceps tendon as described by Campbell [74]. Addition of a tibial tubercle osteotomy in mature patients has been advocated by several authors to address patellar instability in adult patients with DS [65, 67, 69, 74, 75, 76].

Recently, in 2012, Kocon et al. [73] reported the results of eight children (ten knees) with DS treated with a quadriceplasty and Galeazzi augmentation for patellar instability. The authors report good results in seven knees, while three knees had early recurrence at 9 months. The authors conclude that quadriceplasty provides satisfactory results but addition of the Galeazzi is warranted when children are older (over 8 years of age). Overall, children with DS achieve successful results with surgery for patellofemoral instability and often participate in sports after a careful rehabilitation program.

Guidelines for Sports Participation in Children with DS

The AAP classifies sports as contact or noncontact. Contact sports involve collision (boxing, football, wrestling) or impact (Basketball, diving, gymnastics). Noncontact sports are strenuous (aerobics, crew, swimming), moderately strenuous (badminton, table tennis), or nonstrenuous (archery, golf, riflery). From this review, it would seem that children with DS should avoid collision sports. A detailed pre-participation history and physical examination are critical in facilitating safe and healthy sports participation in children and adolescents with DS.

Currently, the Special Olympics requires all children with DS to undergo a cervical spine radiologic examination prior to their participation in the Special Olympics. To date, there has not been a reported case of a cervical spine injury during participation in the Special Olympics in a patient who had either known or unknown atlantoaxial instability or any other underlying cervical spine issue [9, 35, 45, 47]. It is possible that that this lack of an injury is secondary to the fact that the screening has been mandated by the Special Olympics. Although current flexion and extension cervical spine radiographs have flaws,

they are currently the most reasonable objective parameters available for assessment of cervical spine instability.

Summary

Approximately 20 % of all patients with DS have some associated musculoskeletal problem. Personnel familiar with the specific medical and orthopedic conditions associated with DS should complete an athlete's PPE. A specific focus on cervical spine instability and cardiac anomalies should be included in the PPE. Given appropriate management and monitoring, children with DS should be able to participate actively in and derive benefits from sporting activities.

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Spinal Deformity: Presentation, Treatment and Return to Sport

15

M. Timothy Hresko

Introduction

Major spinal deformities often become evident in the preteen years due to the growth of the spine and rib cage. The presentation of spinal deformities coincides with the child's emergence into sports as a young athlete. The spinal deformity may influence the performance of the young athlete or may be a factor for the young athlete and their family to consider in the decision to pursue a competitive athletic program. The spinal deformity may limit the range of motion of the spine which would make it difficult for the young athlete to advance through the challenges of competitive sports as the level of skill and endurance become more demanding in the teenage years and later [1]. In addition, the appearance of the spinal deformity may indicate an underlying medical condition, with the spinal deformity being the presenting symptom. It is estimated that 5% of adolescents in a scoliosis clinic have an underlying systemic disease. In particular, adolescents with scoliosis must be screened for neurofibromatosis and Marfan syndrome. Similarly, an adolescent with excessive thoracic kyphosis (TK) may have an underlying Chiari malformation or a heritable connective tissue disorder such as Ehlers–Danlos or Marfan syndrome.

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Scoliosis

Presentation

The most common deformity of the spine is scoliosis. Images of persons with the distorted torso, asymmetric shoulders, and waistline which are typical of scoliosis can be found throughout history. "Scoliosis" is the term used to describe a coronal plane deformity with lateral curvature of the spine that is 10° or greater on an upright radiograph of the spine. Advanced imaging has shown that the uniplanar analysis of a radiograph is a simplification of three-dimensional spinal deformity. For idiopathic scoliosis, the lateral deviation of the thoracic spine is associated with hypokyphosis or true lordosis in the sagittal plane and rotation in the axial plane to create the chest wall and trunk deformity.

Characterization of the scoliosis based on etiology is useful. Congenital scoliosis describes deformity due to a spinal anomaly present which is identified on imaging of the spine. The anomaly is present at birth although often not evident until later years. The congenital deformity occurs in uteri due to failure of formation, segmentation defects, or combined conditions which cause asymmetric growth with progressive spinal deformity and trunk imbalance. The lack of normal lengthening of the thorax with growth may lead to reduced chest volume and to respiratory deficiency in later life. Neuromuscular scoliosis is associated with central nervous system conditions as seen with asymmetric muscle function in cerebral palsy or in the peripheral neuromuscular

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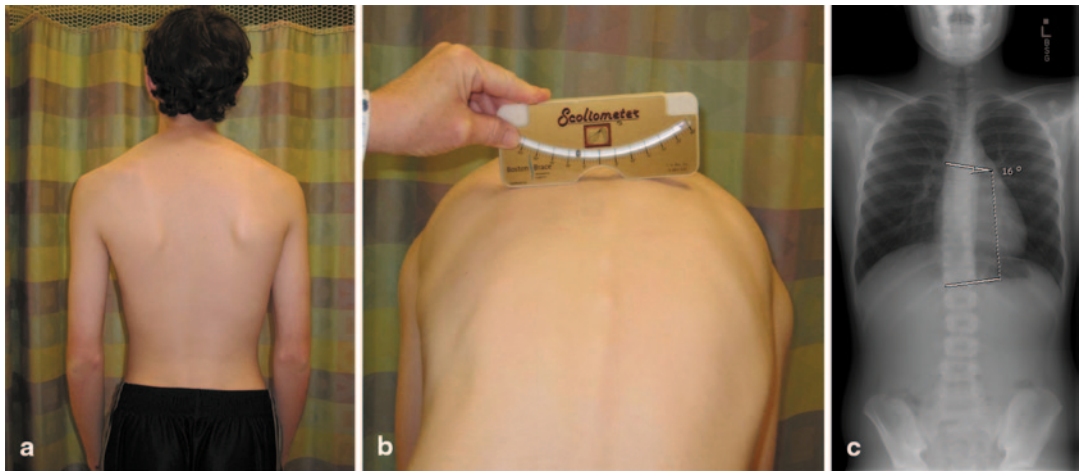

Fig. 15.1 Case presentation of adolescent athlete with mild scoliosis. **a** Clinical photo of standing posture: note shoulder elevation, scapular prominence. **b** Forward bend Adams test with inclinometer measurement. **c** Radiographs of spine with Cobb angle measurement of thoracic scoliosis

unit as with muscular dystrophy, or to combined sensory and motor dysfunctions of Chiari malformation with syringomyelia or neurofibromatosis. Collagen-based conditions such as Marfan or Ehlers–Danlos syndrome have a high incidence of scoliosis. The tall stature and long limbs of the Marfan patient are particularly beneficial for athletes in net sports such as basketball and volleyball. A thorough physical examination for the stigmata of collagen disease is mandatory for all children with scoliosis. Common physical characteristics of collagen diseases are tall stature, long narrow fingers (arachnodactyly), valgus foot deformity, and long arm span that exceeds height. In adolescents and young athletes with scoliosis, the vast majority of patients do not have of a recognized etiology for the spine deformity. The diagnosis by exclusion is "idiopathic scoliosis."

Although idiopathic scoliosis may have a varied age of onset, the vast majority of patients have the initial presentation during the peak growth velocity prior to the start of puberty. For girls, peak growth velocity often is between ages 11 and 13 or the year prior to menarche while peak growth in boys is 1–2 years later. The screening examination for participation in a sport or the routine physical examination by the primary care physician may discover the chest and trunk asymmetry of scoliosis. School screening programs consistently identify asymmetric torso beyond an acceptable level in 2–4% of screened children [2]. Detailed physical examination remains as the most useful tool in the diagnosis of idiopathic scoliosis and elimination of other causes of scoliosis. The classic findings on examination are shoulder, scapular, or waist asymmetry, and unilateral paraspinal or rib prominence on forward flexion (Adams test) (Fig. 15.1a, b). The use of an inclinometer at the apex of deformity helps to quantitate the findings and aids in the determination of the need for radiographic imaging. Depending on the age and the physique of the patients, an inclinometer reading of greater than 5–7° has been used as an indication for spinal radiographs [3]. The recommended view is a standing posterioanterior (PA) radiograph of the spine from C7 to the femoral heads with digital imaging enhancement. Radiation exposure to thyroid and breast tissue can be reduced with the PA view. The severity of the scoliosis is measured by the Cobb angle. The Cobb method of measurement is the angle formed by a line parallel to the most cephalad vertebrae in the spinal curve and a line parallel to the inferior vertebrae (Fig. 15.1c). In general terms for the deformity, a Cobb angle measure of less than 10° is normal, 10–25° is mild, 25–45° is moderate, and greater

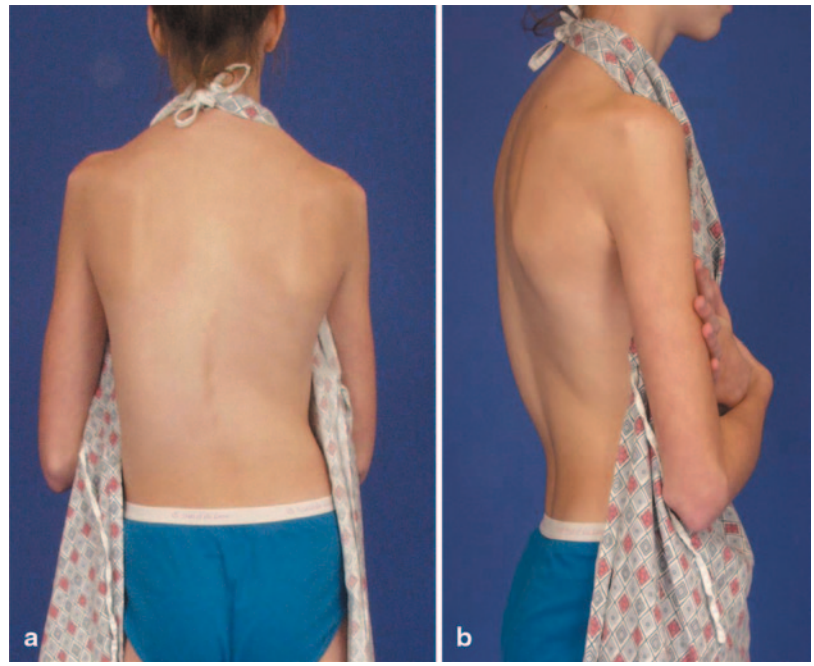

Fig. 15.2 Clinical photograph of left thoracolumbar scoliosis. **a** Coronal view: note trunk shift to left, pelvic prominence on the right, shoulder elevation asymmetry. **b** Sagittal view: note lumbar paraspinal prominence

than 45° is severe scoliosis in the growing child (Fig. 15.2a, b).

Severe pain in an athlete should not be attributed to mild scoliosis as other causes of pain should be investigated. Painful scoliosis occurs with spondylolisthesis, spinal column tumors, infection, Chiari 1 malformation with syrinx, and tethered spinal cord. Spondylolisthesis as a cause of the scoliosis with back pain can be assessed by a lateral image of the spine. The spondylolisthesis is associated with hamstring tightness and paravertebral spasm to limit the Adams bend test validity. Also, asymmetric forward translation of the L5 vertebrae on the sacrum may create true lumbar scoliosis. Spondylolysis and spondylolisthesis are common in athletes especially in repetitive activity which require lumbar hyperextension, most notably gymnastics (discussed in Chap. 9).

Treatment

Many nonoperative treatment options such as physical therapy, surface electrical stimulation, and chiropractic treatment have been advocated based on case series but have not been supported by higher level of evidence studies. Nevertheless, most patients with mild scoliosis (10–25°) do not progress. The condition may be safely monitored by the primary care physician or in conjunction with an orthopedist. Repeated clinical exams at specific intervals based on the adolescent growth velocity may be sufficient for the mild curve while radiographs are performed when clinical concern arises [4].

The aesthetic appearance of the body is the main disability with idiopathic scoliosis; slight at first but progressive with growth. Therefore, the immature patient with progressive scoliosis of greater than 25–30° is considered a candidate for nonoperative treatment. An additional concern of progressive scoliosis is restrictive pulmonary disease with reduced vital capacity which can occur later in life with progression of thoracic curves of greater than 45–50° in a skeletal immature patient [5]. The age of onset of scoliosis is also an important prognostic indicator as the presence of a large thoracic deformity before age 5 (infantile or juvenile scoliosis) places a patient at high risk for cardiopulmonary restrictive disease and secondary cor pulmonale. The risk for progression of late onset idiopathic scoliosis is also related to the age of onset as patients with a 30 degree

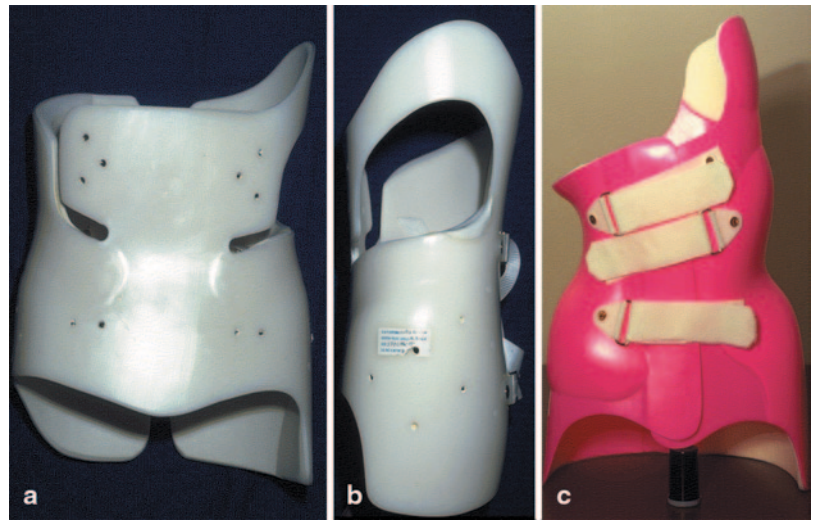

Fig. 15.3 Thoracolumbar orthosis used for treatment of scoliosis Boston Brace **a** anterior. **b** lateral. **c** Charleston brace

curvature prior to the puberty have a high risk for progression [6].

Brace treatment of the spinal curve is the preferred method to arrest or reduce the rate of progression of the scoliosis in the growing child through adolescence. The indication to initiate brace treatment is progressive scoliosis in a child or adolescent with a curve magnitude of 25–45° with significant growth remaining. Bracing with a rigid thoracolumbar orthosis (TLSO) has a long clinical history which began with the Milwaukee brace and has evolved with the use of thermoplastics into a lightweight, underarm orthosis that conforms to the body shape. The best rates of success in bracing (defined as less than 6° of progression) are obtained with daytime ambulatory bracing. Many designs have been proposed based on regional preferences—Boston, Cheneau, Lyon, and Wilmington to name a few (Fig. 15.3). All ambulatory bracing programs require a skilled orthotist to construct a comfortable but effective brace, a strong supportive family, and a compliant patient. At best, the goal of brace treatment is to arrest the progression of scoliosis below the level of surgical treatment. Optimal results from bracing are obtained when brace wear is a minimum of 18–20 h/day. TLSO success in preventing progression of the scoliosis has varied from 80% in the most compliant subgroup of a compliance study on bracing to less than 50% in a retrospective study that included all patients who were prescribed brace including those who were noncompliant [7, 8]. Noncompliance and a high rate of failure have led some clinicians to recommend against bracing with a focus on early surgery for progressive deformity. The uncertainty about bracing treatment led to a multicenter clinical trial sponsored by the National Institute of Arthritis and Musculoskeletal and Skin Diseases (NIAMS) called "The Bracing for Adolescent Idiopathic Scoliosis Trial" (BrAIST). Enrollment in BrAIST is completed and results are expected in late 2013 [9]. Physical therapy programs to supplement the brace program have been developed. The intensity of the physical therapy varies from a generalized flexibility and strengthening program to intensive physical therapy programs to teach asymmetric chest expansion and spinal elongation/derotation techniques such as the Schroth technique may be beneficial for specific patients [10].

Operative treatment is recommended for patients who progress despite bracing or have scoliosis of greater than 45–50° with significant growth remaining. Patients who are under age 10 who fail orthotic management are candidates for growth preserving surgery. Nonfusion surgical techniques to maintain spinal growth have been developed to delay definitive spinal fusion until adolescence for continued chest growth and lung volume expansion [11]. Growth modulation by the use of a mechanical tether on the convexity

of the spine has been proposed and is undergoing preliminary study at this time. However, at this time nonfusion growth modulation has been primarily with a posterior-based growing spinal instrumentation. The technique involves placement of anchors at the cephalad and caudal aspect of the curve with an expandable rod-sleeve mechanism to distract the spine for reduction of the scoliosis. Surgical expansion of the rods occurs every 6 months until the child reaches preadolescence which is usually at least at the age of 10 years in girls and 12 years in boys. This treatment limits spinal motion in the instrumented segments.

Operative treatment of adolescent scoliosis entered the modern era in the 1960s with the introduction of internal fixation by Dr. Paul Harrington. The original technique involved placement of a stainless steel rod to distract the spine into a straighter position. Over time, the technique and implants have been refined to improve the correction and safety of the procedure. Important improvement in safety has occurred with intraoperative neuromonitoring (IONM), anesthetic management, blood conservation, and infection control. Implant design has become a major industry with implants designed to allow segmental control of the spine and allow early return to function. Spinal fusion over the instrumented segment has remained part of the surgical technique so the inherent trade-off for the correction of the spinal curvature with stabilization of the spine is a loss of spinal motion. In the thorax, the restriction of rotation of the fused segment is well tolerated and many patients return to all preoperative activities after surgery. Spinal fusion of the thorax will restrict trunk rotation which is an important component to many sports may be detrimental to the competitive athlete. Nevertheless, a patient with short segmental spinal fusion can still succeed in competitive sports at the highest level [12]. Spinal fusion extending into the lumbar spine is of greater concern as it has been associated with reduced flexion and extension of the spinal range of motion, reduced activity levels, and later degeneration arthritis. The original Harrington rod instrumentation in the lumbar spine produced a loss of normal lumbar lordosis with subsequent painful "flatback" syndrome. Modern segmental spinal instrumentation has addressed the three-dimensional deformity by allowing modulation of the spine to improve the scoliosis while restoring the lumbar lordosis. Most modern spinal implant systems are designed for a dual-rod, posterior segmental spinal fixation system with stainless steel or titanium implants which allows multiple anchor types to fit the local anatomy and size of the deformed spine while maintaining an anatomic sagittal posture. The "ideal" fixation strategy which provides the strength and stability necessary to stabilize the spine while reducing the cost of implant and safety of surgery has yet to be precisely determined. Nevertheless, the improved surgical technique has reduced the perioperative morbidity and recovery period for the adolescent patient with scoliosis. The typical surgical patient with idiopathic scoliosis now usually spends 4–5 days in the hospital and will be able to return to school 1–1.5 months after the procedure.

Return to Play

Children and adolescents with mild idiopathic scoliosis of 10–25° may participate in all sports without restriction. The biomechanical effect of scoliosis at this magnitude on the spine is minimal. To my knowledge, no sports-specific activities have been identified to be associated with an increased risk of progressive scoliosis although deformation of the spine has been demonstrated in weight lifting.

Patients who are actively being treated with a brace may need to do additional core strengthening and flexibility stretching of the lumbar spine. Brace treatment regimens may vary but most physicians allow for removal of the orthosis during sport and dance. Prolonged exercise program, such as long-distance running or swimming, may conflict with the brace time prescription. Nighttime bracing in an "anti-scoliosis" position with a bending brace (Charleston) or in a neutralization brace (Providence) is available but the success in preventing progression has not been studied to the extent of daytime bracing programs.

Surgically treated patients will have reduced spinal motion. Fusion of the thoracic spine will reduce trunk rotation while fusion of the lumbar spine will reduce spinal flexion and extension. In addition, increased strain can be expected in the nonfused segment of the spine. A retrospective study of sports participation in 42 athletes who had spine fusion for scoliosis showed average time to return to play to be 7.4 months, and 59.5 % of patients returned to athletics at an equal or higher level of participation [13]. The most common reasons for a decline in activity level at 5.5-year follow-up were loss of flexibility, back pain, and deconditioning. A stepwise decline in the activity level was seen with more distal fusion levels, with 73 % of patients with a T12 distal fusion level returning to their previous activity compared to 20 % of patients with a L4 fusion level.

There are no published guidelines for the length of recovery after surgery before return to sport. A survey of members of the Scoliosis Research Society by Rubery and Bradford found that 43 % of surgeons recommended low-impact, noncontact sports at 6 months and 61 % allowed contact sports at 1 year postoperatively [14]. Collision sports, including wrestling, football, hockey, and gymnastics, were only permitted by 32 % of respondents at 1 year. Sixty percent of surgeons recommended against or forbade return to collision sports after scoliosis fusion. The most common sports forbidden after surgery were football, gymnastics, collision, skydiving, and trampoline. Distal fusion level did not influence decision-making on return to sports for the majority of surgeons. However, with modern segmental instrumentation quicker patient recovery and rehabilitation makes return to sport at earlier times more likely.

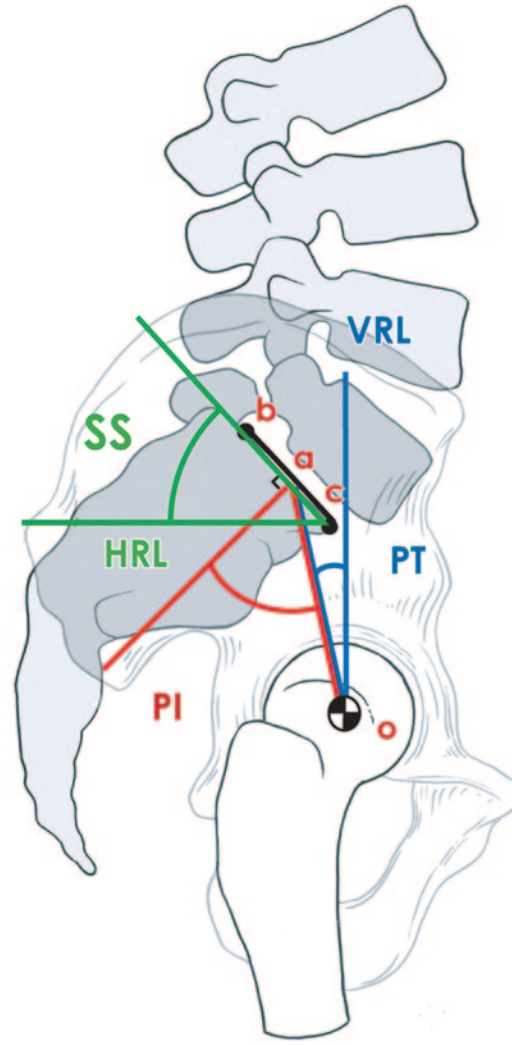

Fig. 15.4 Sagittal pelvic radiographic alignment is measured in degrees of arc. Pelvic incidence (*PI*) is an anatomic parameter, sacral slope (*SS*) and pelvic tilt (*PT*) are positional parameters which will vary in a reciprocal manner based on each individuals standing posture such that SS + PT = PI. (Images provided by the Orthopaedic Research and Education Foundation)

Kyphosis and Lordosis

Presentation

There is significant variation in the sagittal plane of the spine. "Normal" kyphosis of the thoracic spine and lordosis of the lumbar spine is dependent on the shape of the pelvis and inclination of the pelvis on the hip. The term pelvic incidence (PI), which is measured in degrees of arc, describes an anatomic relationship between the sacral end plate and the center of the femoral head [15]. "Sacral slope" (SS) describes the angulation in degrees of the sacral plate to a horizontal reference line while "pelvic tilt" describes the inclination of the pelvis on the femoral heads

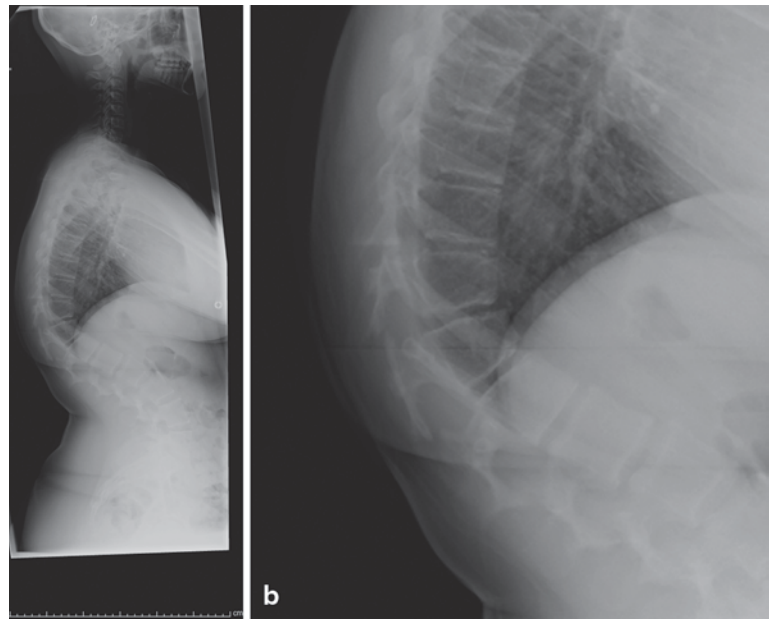

Fig. 15.5 The standing lateral radiograph is characterized by thoracic kyphosis and lumbar lordosis. **a** Scheuermann's hyperkyphosis. **b** "Coned down" view showing wedged apical vertebrae

to a vertical reference line. SS and pelvic tilt (PT) are positional parameters that will vary with posture such that PI = SS + PT (Fig. 15.4). Similarly, the amount of lumbar lordosis (LL) necessary for a balanced posture is related to the SS and PI with LL = PI ± 10º [16]. To have a "normal" balance thorax, TK is usually TK = LL −20º. Ranges of normal values are PI = 45º–55º, SS = 35º–45º, PT = 5º–15º, LL = 35º–65º, and TK = 15º–45º.

Abnormal TK mandates that the LL and/or SS increase by a proportional amount. Dorsal spine muscle fatigue and pain can occur when the compensatory balance is not achieved. Scheuermann kyphosis is a developmental condition characterized by excessive TK (greater than 50°) with anterior wedging of three or more vertebrae (Fig. 15.5). The consecutive wedged vertebrae create a short sharp arc which is most evident on physical exam while the patient reaches for toe touch, a maneuver which eliminates the LL. The TK remains even when the patient attempts hyperextension of the thoracic spine. Young patients tolerate the excessive kyphosis with an exaggerated LL but as the spine ages into adulthood mild lumbar facet orthosis is common and the ability to compensate for excessive kyphosis is lost. In contrast to the rigid kyphosis seen with Scheuermann's disease, postural TK is a benign condition without vertebral wedging which is commonly seen in adolescents with ligament laxity and hyperlordosis. Core strengthening, pectoral stretching, and thoracic extension strengthening can maintain or improve postural kyphosis.

As with scoliosis, sagittal plane deformity may be associated with generalized medical conditions. Heritable connective tissue disorders such as Marfan or Ehler–Danlos syndrome should be considered during physical examination. Referral to a geneticist may be warranted.

Treatment

Postural deformity is amendable to physical therapy programs that emphasize balance, strengthening, and postural awareness. For thoracic postural kyphosis, the physical therapy program should emphasize dorsal paraspinal and scapular strengthening with anterior chest wall and pectoral stretching. The flexible hyperlordotic patient will benefit from abdominal, gluteal, and hamstring strengthening with hip flexor stretching to promote reduced pelvic tilt. This exercise program should be part of all gymnastic training for injury prevention due to the high incidence of lumbosacral overuse syndromes in elite gymnasts.

Brace treatment for kyphosis is considered in the skeletal immature patient when progressive deformity greater than 60° or pain is a significant factor. The Milwaukee brace, a cervical–thoracic–lumbosacral orthosis, is biomechanically effective but compliance to wear is difficult during daytime activities of daily life; therefore, the Milwaukee brace is now primarily a nighttime brace. Low-profile underarm orthosis with an anterior sternal extension achieves better daytime compliance. The kyphosis bracing program is accompanied by a physical therapy regimen as described above. The effectiveness of brace programs for prevention of progression of kyphosis has not been established by prospective clinical studies.

Surgical treatment for painful or progressive kyphosis involves segmental instrumentation over the regional kyphosis deformity with consideration of global sagittal balance as defined by the PI and sagittal balance. In teens and young adults, correction is usually achieved by a posterior approach with posteriolateral vertebral (Ponte) osteotomies to shorten the posterior elements and compression forced applied with the segmental instrumentation. The proximal and distal extent of the fusion and instrumentation needs to take into accord the global sagittal balance to restore harmony to the spine.

Return to Play

Patients with flexible painless hyperkyphosis or hyperlordosis may participate in all sports. The concept of injury prevention in the immature athlete comes into play in the selection of which sport to pursue and in avoiding overuse syndromes. Conditioning programs for pelvic and spinal muscles strengthening should be encouraged. Athletes with a rigid TK may be at risk of lumbar spine overuse syndromes or stress fractures (spondylolysis) with repetitive hyperextension and flexion activity. An assessment of the requirement of the sport and proper spinal mechanics will be an important element in continued participation of the athlete with Scheuermann kyphosis.

Recovery after surgery is similar to patient with idiopathic scoliosis

Summary

Spinal deformities emerge during the preadolescent phase of growth. The detection of a spinal deformity is often during a routine evaluation by the primary care physician, a school screening program, or casual observation of a parent or coach. Early detection of the spinal deformity is beneficial in the identification of the child at risk for progression of the deformity. Most adolescent athletes will be able to continue in their sports programs but may need alteration in the training regimen if the treatment regimen for the spinal deformity requires orthotic or surgical treatments.

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Intrinsic Spinal Cord Abnormalities in Sport

16

Edward R. Smith and Mark R. Proctor

Introduction

The diagnosis of lesions within the spinal cord or major arteries supplying the central nervous system can be a source of significant concern for both athletes and the physicians involved in their care. This concern is magnified by the consequences associated with spinal cord injury and the dearth of information surrounding the proper management of many of these conditions. This chapter seeks to review the literature relevant to athletic activity with regard to congenital conditions (intradural cysts, Chiari malformations, syringomyelia, tethered spinal cord), vascular lesions (vertebral artery dissections, arteriovenous malformations and fistulae, cavernous malformations), and neoplasms. Each section offers an overview of the condition, recommendations for evaluation, and expected impact on athlete participation. Plans of action will be derived from published data, expert consensus, and examples of actual practice patterns reflecting "common sense" approaches.

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Congenital Conditions: Chiari Malformations, Syringomyelia, Intradural Cysts, and Tethered Cord

Chiari Malformation

Overview

The term Chiari malformation has accrued multiple meanings over time. Formally, this includes various groupings of hindbrain dysmorphism, with four numbered malformations (I–IV) which describe different cerebellar configurations. Importantly, the numbers do not imply a grading system—a CM I cannot progress to a CM IV, for example. Chiari I represents the vast majority of lesions that will present in common practice.

For the sake of simplicity: type I is displacement of the tonsils below the plane of the foramen magnum; type II is a far more complex malformation that includes displacement of the hindbrain, fourth ventricle, and cerebellar tonsils and vermis below the foramen magnum as well as a more diffuse constellation of brain anomalies with varying degrees of severity found in association with myelomeningocele and therefore always evident and diagnosed at birth; and type III is similar to type II but is found in association with a suboccipital or high cervical encephalocele and generally not compatible with life. Type IV is cerebellar hypoplasia and likely a completely separate process from the other three—linked only by name. In addition, some groups have described a so-called and somewhat controversial

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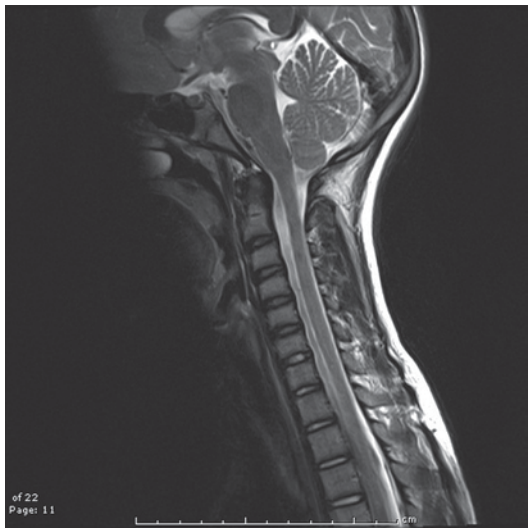

Fig. 16.1 Mild Chiari malformation. Often, a mild Chiari malformation is detected incidentally on MRI scans, and may be described as cerebellar tonsil ectopia in the radiology report. In this healthy athlete with neck pain after trauma, the cerebellar tonsils are rounded, and just a few millimeters below the foramen magnum. This is not of clinical concern, and he responded to conservative therapy

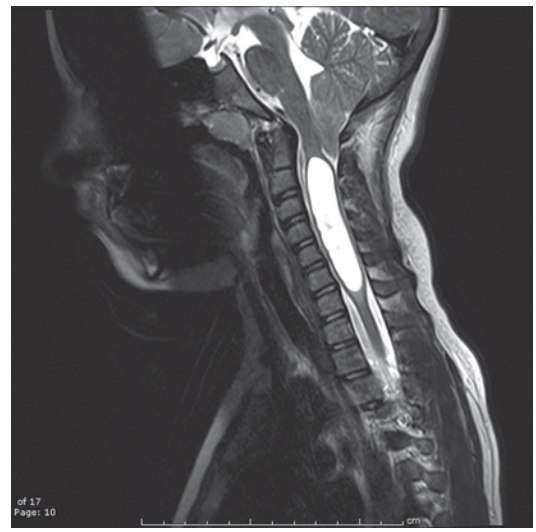

Fig. 16.2 Chiari malformation with syrinx. This adolescent presented with significant spinal curvature. MRI scan reveals a tight Chiari malformation with approximately 15 mm of tonsillar herniation. There is a large cervical syrinx which resolved after surgery

Chiari 0, in which there are symptoms of brainstem compression or foramen magnum outlet obstruction without tonsillar herniation seen on imaging, presumably secondary to other anatomic structures such as arachnoid bands or scar [1].

This chapter focuses on the Chiari I malformation (CM I), as it is the most commonly encountered type. There is a great deal of controversy surrounding the diagnosis and treatment of Chiari I, including the degree of displacement required for diagnosis (with most requiring at least 5 mm of herniation below the foramen magnum, but some accepting 0–2 mm) and what constitutes a pathologic Chiari I. Some experts have called for renaming the condition to Chiari "anomaly", as it is thought to be present in about 0.75–3 % of the population [1, 2, 3, 4] (Fig. 16.1).

The vast majority of Chiari I malformations are asymptomatic and do not need intervention [3]. When symptomatic, Chiari I malformations often present with sudden-onset suboccipital headaches classically aggravated by activities that invoke a valsalva maneuver. Less commonly, patients can present with lower cranial nerve dysfunction (especially dysphagia or sleep apnea), cerebellar dysfunction (e.g., ataxia), or spinal cord dysfunction secondary to an associated hydromyelia (Fig. 16.2) (weakness and scoliosis). Treatment (when necessary) is predicated on surgical decompression of the cervicomedullary junction.

Recommendations for Evaluation

Many patients will be asymptomatic on examination if the lesion is found incidentally. However, a detailed neurologic examination and history are always important. Attention should be paid to evidence of neurologic dysfunction in the history.

- Headache: usually occipital and tussive, which can often be replicated on examination; need to assess whether the pain is related to flexion or extension of the neck
- Lower cranial nerve dysfunction: apnea (infants), snoring, dysphonia, dysphagia, trapezius weakness
- Spinal cord dysfunction: weakness in the arms or legs (especially the hands with signs of muscle wasting), long tract signs (with hyperreflexia Babinski, "cape" sensory loss distribution), scoliosis

The radiographic diagnosis of a CM I is relatively straightforward, with the difficulty being in distinguishing asymptomatic patients from those who are appropriate surgical candidates. Furthermore, careful history taking is important to determine if things such as lumbar puncture or **intracranial pressure** (ICP)-elevating medications (such as retinoic acid) may be contributing to the radiographic findings. Lastly, given the often subjective nature of complaints in CM I, the clinician must carefully evaluate the patient for other causes that may explain presenting symptoms.

Magnetic resonance imaging (MRI) is the imaging modality of choice in the evaluation of CM I. If possible, studies of the brain should be obtained to exclude the possibility of mass lesions or hydrocephalus as proximate, treatable causes of CM I. The use of high-resolution MRI, such as fast imaging employing steady state acquisition (FIESTA), may be useful in assessing for the possibility of obstruction to the outflow of the fourth ventricle. Contrast is usually not needed.

In addition to the objective measurement of tonsillar herniation, there are subjective measures that can be assessed using MRI to help determine the extent of compression. These subjective findings include the presence of "peg-like" tonsils, obliteration of cerebrospinal fluid (CSF) spaces at the cervicomedullary junction, the presence of a syrinx (sometimes requiring dedicated spinal imaging), and the quantification of CSF flow.

Expected Impact

One of the greatest challenges in CM I is appropriate selection of surgical candidates. "The Chiari malformation is, in fact, one of the few conditions for which the American Association of Neurological Surgeons (AANS) issued a position statement regarding the inappropriate use of surgery (AANS Position Statement on the Use of Cervical Decompression for Chronic Fatigue Syndrome, March 2000). When a patient presents with two common conditions, there is always going to be some degree of coincidental overlap, and surgeons must be careful not to perform surgery in patients in whom there is little chance that the Chiari malformation is symptomatic" [2].

In general, operation is indicated in patients with associated symptoms (see above) and clear radiographic evidence of disease (> 5 mm herniation, ± syrinx). For those with atypical symptoms (frontal headache, fatigue, etc.) and minimal radiographic findings, a conservative approach with a referral to pain management might be appropriate. Surgery for asymptomatic patients (including incidentally found lesions) remains controversial and has been justified on the basis that this lesion has the potential to become symptomatic or could place the patient at greater risk of spinal cord injury if left untreated, although this has not been substantiated.

One of the factors to support surgical intervention for asymptomatic Chiari includes prevention of an exacerbation after trauma. Development of symptoms related to a Chiari after a minor traumatic event has occurred, with reports indicating approximately 13 % of previously asymptomatic CM I developing symptoms after trauma (including isolated case reports of sudden death in severe cases) [5, 6]. However, no prospective series has ever shown a risk of catastrophic injury in an athlete with a known Chiari malformation, and the risks of participation are likely quite low [7].

Athletes with Chiari malformation present additional challenges in the determination of appropriate management for an incidentally found Chiari. Numerous athletes have been found to have Chiari malformation on imaging obtained after suffering a concussion, and athletes with known Chiari experiencing drop attacks have been reported. The implication of these associations is unclear, and it has yet to be determined whether or not athletes with asymptomatic Chiari who play contact sports are at a greater risk for catastrophic injuries. A known Chiari with clear associated symptoms is often considered a contraindication to contact sports. Asymptomatic Chiari malformation may be a relative contraindication due to increased risk of injury based on anecdotal evidence. Although the literature has yet to define the exact degree of concern that a sport medicine clinician should have in this situation, most neurosurgeons do not advocate prophylactic surgery for asymptomatic Chiari malformations in the competitive athlete [8, 9].

A careful discussion with the athlete and family is important and it is generally prudent to include a neurosurgeon in these deliberations. Most athletes can return to sports after undergoing a surgical Chiari decompression.

Syringomyelia

Overview

Syringomyelia and *hydromyelia* are often used interchangeably to describe the presence of fluid (presumably CSF) within the substance of the spinal cord. The spinal cord normally has a tiny central canal running along its entire length, which can enlarge in both normal and pathological situations. Syringomyelia can be found in isolation, after injury, or in association with pathologic conditions such as Chiari malformations, tumors, previous infection, or tethered spinal cord [4]. One of the major difficulties faced by clinicians is the rising incidence of discovered, dilated CSF spaces within the spinal cord secondary to improved MRI technique and increased frequency of imaging, many of which appear to be normal, non-pathological variants (Fig. 16.3).

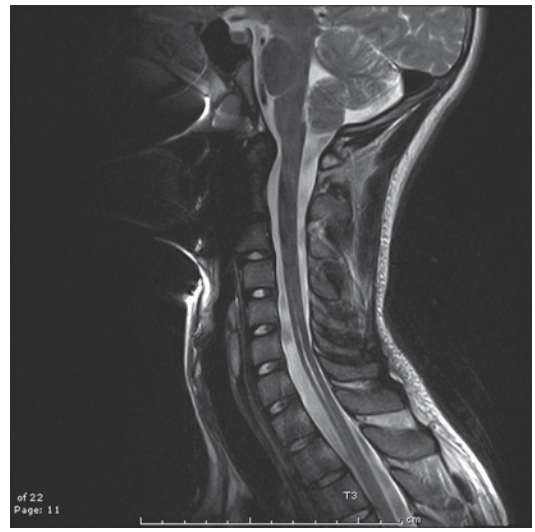

Fig. 16.3 Dilation of central canal. This teenage athlete underwent an MRI scan due to back pain and was referred for a spinal syrinx. This is a mild dilation of the central canal, without Chiari malformation or other associated pathology. No treatment or restrictions are necessary

Recommendations for Evaluation

The vast majority of patients with syringomyelia—especially if the collection is less than 2 mm in diameter on axial imaging—have normal clinical examinations and most likely have a normal anatomic variant of no consequence. In a pathologic setting (such as Chiari or tumor), the syrinx will often enlarge and symptoms can occur. On physical examination, syringomyelia can cause symptoms by chronic injury to pain and temperature fibers (which cross centrally in the spinal cord to produce a classic "suspended" sensory loss), compression of anterior horn motor neurons leading to lower motor neuron weakness (typically in the hands), compression of corticospinal tracts leading to upper motor neuron weakness with spasticity (typically in the legs), and possible scoliosis from weakness of the axial musculature [10]. The complaint of back pain in isolation—without other neurologic or radiographic findings—is usually not cause for concern when found in association with syringomyelia (and is not considered to be caused by the syrinx) [11].

If a syrinx is discovered (particularly if greater than 2 mm in axial diameter), MRI of the whole spine (and possibly brain) should be considered to exclude surgically treatable causes, such as Chiari malformation, tumor, or tethering [11].

Expected Impact

Because of the high prevalence of syringomyelia in the population, it is common to see athletes with this finding identified after imaging for back pain. Most cases of syringomyelia have no clinical significance and do not require further imaging or treatment, with expected full activity without restriction for the athlete [11]. In the case in which an associated, causative pathologic condition is identified (Chiari malformation, tumor, etc.), the outcome is dependent on treatment of the underlying pathologic cause. In these cases, referral to a neurosurgeon is warranted.

Intradural Cysts

Overview

Intradural cysts are generally defined by their contents and location. The most common are collections of CSF, including arachnoid cysts, perineural cysts (enlarged nerve root sleeves, the so-called "Tarlov" cyst), or extensions of the dural sac ("ballooning" of the meninges) called meningoceles (often located in the distal thecal sac within the sacrum) [1, 2, 3]. Nearly all of these CSF-containing cysts are congenital (with the exception being post-traumatic or post-surgical meningoceles) and are usually asymptomatic. Rarely, symptoms can present from cyst expansion (such as myelopathy from cord compression by an arachnoid cyst), nerve root pain (from herniation of a nerve root within a perineural cyst or meningocele), or "tailbone" pain, with or without bowel and bladder problems (constipation, incontinence, or urgency) from sacral cysts.

Other cysts may contain epidermis or admixed tissue types (epidermoid or dermoid). These cysts may be congenital (sometimes found in association with dermal sinus tracts; see the section on tethered spinal cord) or acquired (after lumbar puncture or previous surgery). Symptoms can arise from focal compression (with growth), leakage of contents (a sterile "chemical" meningitis), or—in the case of sinus tracts—bacterial meningitis.

Recommendations for Evaluation

Most cysts will be identified with MRI. If found, detailed imaging in the region of the cyst, including high-resolution multiplanar studies (such as FIESTA sequences in axial, coronal, and sagittal planes) can be helpful, including diffusion weighted imaging (DWI), which can often distinguish between CSF cysts and dermoid/epidermoid. Discussion with a radiologist prior to obtaining studies may be helpful to ensure that proper sequences are obtained.

In addition to MRI, careful physical examination is important. In particular, inspection of the spine for midline dimples, sinus tracts, or other surface pathology may help to identify possible areas of concern. Dimples can be associated with an associated patent tract that puts the athlete at risk for localized infection or meningitis.

Expected Impact

The impact of an intradural cyst on an athlete is hard to predict, as there is tremendous variation in size, location, and reason for discovery. In general, identification of one of the aforementioned cysts should prompt a referral to a neurosurgeon. For lesions that are clearly associated with related symptoms, surgical treatment may be warranted. Asymptomatic lesions can often be observed, with the exception of those that have dermal sinus tracts or evidence of growth on serial imaging. In the majority of cases, athletes can expect a full return to play.

Tethered Cord

Overview

The definition of a tethered spinal cord can be confusing, as clinicians sometimes use the same term to describe different conditions. A radiographic tethered cord is a finding on imaging in which an abnormal attachment between the spinal cord and the meninges or associated soft tissues exists—with or without clinical symptoms. In contrast, tethered cord syndrome is a constellation of demonstrable signs and symptoms resulting from tension on the spinal cord due to an area of tethering. Symptoms are generally considered to be a result of local ischemia secondary to tugging on neural and vascular structures, although it is possible that some deficits may be reflective of primary defects in cord development. An athlete can have a radiographic tethered cord without symptoms.

Tethered cord is most commonly associated with the presence of congenital anomalies, such as spinal lipomas, fatty fila (Fig. 16.4a, b), or dermal sinus tracts. In some of these cases, findings on the skin—usually in the midline of the back—are present, such as dimples, patches of hair, hemangiomas, skin tags, or a fatty prominence. However, in addition to these congenital lesions, tethering can also result from scarring, as might occur after infection (meningitis), previous spinal cord surgery, or trauma.

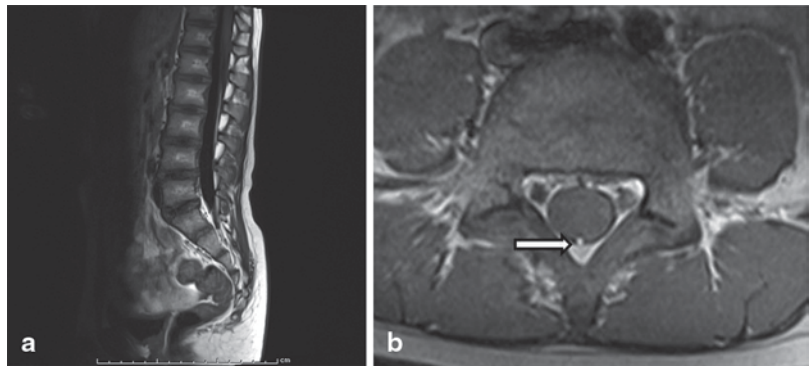

Fig. 16.4 Fatty filum. This soccer player had progressive leg pain and urinary frequency. The sagittal **a** and axial **b** MRI images show a fatty filum terminale, the cause of spinal tethering. Symptoms improved after sectioning the filum

Recommendations for Evaluation

Clinical symptoms depend on the site and extent of tethering. Presentation in symptomatic patients commonly includes lower extremity weakness, spasticity/hyperreflexia, and bladder dysfunction, but pain in the back and/or legs can be an isolated finding [12, 13]. Cutaneous stigmata of an underlying congenital defect (as described above) or lower limb/foot deformities are also common and should be kept in mind during the examination of an athlete [13]. However, diagnosis of spinal cord tethering is often difficult; symptoms are usually insidious in onset, can mimic more common, sports-related problems and the classic findings on physical examination may not always be present. The persistence of back pain despite routine conservative treatment, any of the neurological symptoms noted here—or evidence of clear abnormalities on neurologic examination—should trigger consideration of spinal cord imaging.

The preferred imaging method for spinal tethering is MRI, with the extent of imaging dictated by the likely level of cord involvement based on history and examination (frequently the lumbosacral levels). Findings can include a thickened fatty filum (generally considered concerning if greater than 2 mm in axial diameter), low conus (below L2), syrinx, or evidence of other lesional tissue (lipoma, dermal sinus tract, etc.). Other studies may include plain X-ray films to assess spinal curvature or look for bony defects. If imaging is positive, referral to a neurosurgeon should then be considered. Other ancillary studies might include urodynamics or rectal manometry to more accurately assess for bladder and bowel difficulties, but likely, this decision should be made in conjunction with the specialist.

Expected Impact

If an athlete is found to have a tethered spinal cord, the effect on activity can vary greatly. Asymptomatic lesions, especially in older, skeletally mature athletes, may not require any treatment and will have no effect on activity at all. Other findings, like complex terminal lipomas or sinus tracts, may require surgery and can ultimately limit athletic pursuits, although many children can return to full activity without restriction [14]. Some children can continue sports, but may need routine monitoring during periods of growth. The wide range of tethering lesions, coupled with the often complex nature of symptoms (which may be related to tethering in some cases and which may be totally unrelated in others), mandates close consultation with specialists to chart the best course of care for individual athletes.

Vascular Lesions (Arterial Dissections, Arteriovenous Malformations, Fistulae, and Cavernous Malformations)

Vertebral Artery Dissections

Overview

Arterial dissections are potentially life-threatening injuries to vessels. The vertebral artery supplies the brainstem and spinal cord and can present with spinal symptoms. They can also occur in the carotid arteries, although these will pres-

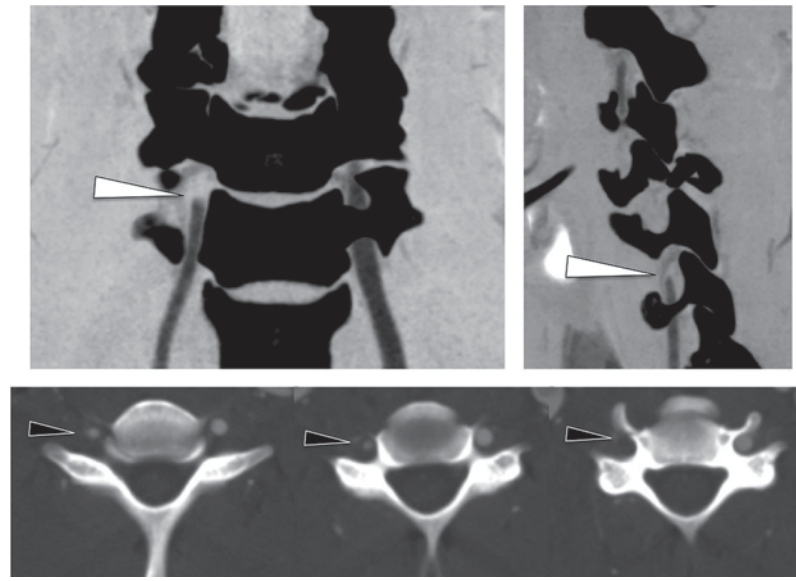

Fig. 16.5 Imaging of vertebral dissection: Series of CT angiogram (CTA) studies demonstrating right vertebral dissection after spine fracture. Upper left image is coronal CTA with arrowhead marking proximal dissection, with upper right image showing same area on sagittal study. Lower panel of three images show progressive loss of contrast (black arrowheads) from lower to upper vertebral levels, indicating region of dissection on axial studies

ent with findings in the brain, not spinal cord, as the carotids have no significant spinal cord blood supply. Dissections can occur after relatively innocuous trauma and are more commonly found in patients with underlying connective tissue disorders, but may also be seen in association with fractures of the spine (Fig. 16.5). While some patients can be asymptomatic, others can present with severe neck pain or neurologic deficits, ranging from brainstem problems (lateral medullary syndrome, with tongue weakness, swallowing problems, and ataxia) to sudden death. They often occur where the artery crosses bony canals (such as C1) and can produce injury from ischemia (if the lumen is narrowed) or emboli (if fragments of clot travel through the vessel).

Recommendations for Evaluation

The diagnosis of vertebral artery dissection can be difficult. Patients will commonly present with severe, unilateral neck pain after trauma. Symptoms may include dysarthria, swallowing problems, upper-motor neuron problems (weakness, hyperreflexia) or cerebellar problems (ataxia, slurred speech, etc.). If suspected, MRI with fat-saturated sequences on magnetic resonance angiogram (MRA) may be helpful, as well as DWI sequences, to look for stroke in the brainstem or cerebellum. Catheter angiography may also be necessary, depending on the MR findings.

Expected Impact

Immediate treatment is predicated on minimizing the risk of stroke and often includes some form of anticoagulation to open the artery and reduce the possibility of embolic/thrombotic events. This requires the consultation of an experienced stroke neurologist or neurosurgeon. In some cases, surgical or endovascular obliteration of the vessel will be necessary. If identified, vertebral artery dissection is an absolute contraindication to most sports in the immediate period. Often several months (or longer) are needed to allow for healing of the vessel wall, as monitored by serial imaging. Return to play is individualized based on the extent of injury and type of sport, but many contact or high-risk activities will likely be permanently prohibited.

Structural Vascular Lesions: Arteriovenous Malformations, Fistulae, and Cavernous Malformations

Overview

Nearly all structural vascular lesions of the spine (arteriovenous malformations, AVMs; arteriovenous fistulae, AVFs; and cavernous malformations, CMs) will be discovered after symptomatic hemorrhage or as an incidental finding when imaging for other reasons. The symptomatic lesions

are readily addressed and treated as needed with referral to neurosurgery/interventional neuroradiology. AVMs often present with catastrophic neurologic deterioration, while AVFs may manifest as a more progressive loss of function secondary to cord swelling/ischemia. CMs typically present with an acute deterioration after hemorrhage, then demonstrate slow improvement as swelling subsides, although usually not back to baseline. In general, these lesions should be treated if at all possible. AVMs can be resected or radiated, AVFs can be clipped or embolized, and CMs can be resected (or—in some cases—observed). Treatment is predicated on the risk of injury with natural history balanced against the risk of injury from therapeutic intervention.

Recommendations for Evaluation

The typical onset of symptoms from AVM or CM is acute, with symptoms referable to the location of the lesion within the spine and the presentation manifesting over minutes. AVFs may be more chronic, with progressive swelling/ischemia producing symptoms such as pain or weakness over weeks or months. In all cases, MRI is the initial study, followed by catheter angiography in the case of AVM or AVF (and not indicated in the setting of CM). If AVM, AVF, or CM is identified, evidence of multiple lesions (if present) should prompt consideration of genetic causes, including RASA-1 mutations, hereditary hemorrhagic telangiectasia (HHT), or anomalies of the KRIT genes [15, 16].

Expected Impact

The presence of an AVM, AVF, or CM should prompt referral to a neurosurgeon. Long-term return-to-play potential is dependent on extent of initial injury (if the lesion was found after hemorrhage) and treatment. For those patients with complete obliteration of vascular lesions (resection of AVM, removal of CM, etc.), it is likely that they can resume full sporting activity, assuming that there are not residual deficits from the initial presentation or treatment. For patients with lesions that are to be observed, return-to-play guidelines need to be individualized based on the lesion pathology, location, and sport-specific risks.

Neoplasms

Overview

The finding of an intradural spinal cord tumor in an athlete is extremely rare, although it certainly can occur [17]. The more common situation is that of a lesion of the skeleton or surrounding soft tissues, a topic discussed elsewhere in this book. Intradural tumors can range from slow-growing meningiomas (arising from the dura) or schwannomas (arising from nerves, sometimes passing through bony foramina in the spine) to faster, more aggressive cord lesions, such as ependymoma or disseminated tumor from elsewhere in the central nervous system. In general, any intradural tumor is rare, with the exception of athletes who have a history of known tumor-related conditions, such as neurofibromatosis type I (NF I).

Recommendations for Evaluation

Findings that might trigger evaluation for a spinal cord tumor include a history of pain which is especially worse at night due to swelling when laying flat, clear neurologic deficits on examination (loss of pain or temperature sense, decreased proprioception, or altered extremity reflexes), and progressive scoliosis or gait problems that worsen with time. If reported or observed on examination, then referral to a neurologist or neurosurgeon should be considered, possibly with an MRI of the spine (with and without contrast).

Expected Impact

It is impossible to make any blanket statements regarding the return-to-play capacity of athletes with intradural spinal cord tumors. The overall prognosis of the tumor type, the potential need for adjuvant therapy (radiation or chemotherapy), and the impact of surgery (with bone removal,

neurologic deficits, and long-term spinal stability) will all influence the overall outcome of future athletic activities.

Conclusions

The identification of a spinal cord abnormality is often a source of concern for both athlete and physician. In general, tumors, vascular lesions, tethered cords, and cysts should prompt referral to a neurosurgeon or neurologist for an individualized treatment plan that may ultimately allow return to sports. In contrast, many small Chiari malformations (so-called cerebellar ectopia less than 5 mm below the foramen magnum without syrinx), isolated syringomyelia, or incidentally discovered lesions may not interfere with activity and may be managed by the sports physician. Return-to-play criteria and indications to treat remain largely consensus-driven decisions and future work increasing the role of evidence based-study in this area is a great need.

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Metabolic Spinal Disorders in the Young Athlete

17

Naomi J. Brown and Kathryn E. Ackerman

Introduction

The most important clinical consequences of metabolic spinal diseases in children and adolescents include bone deformations, decreased linear growth, and non-traumatic fractures leading to bone pain and disability. The abnormal bone morphology, mineralization, or microarchitecture of vertebrae can lead to poor mechanical properties and bone failure under loads experienced during athletics. In this chapter, we review basic bone physiology and growth, and discuss various metabolic bone diseases in children. These include primary and secondary osteoporosis, hormonal abnormalities, nutritional deficiencies, and other genetic bone diseases. When a patient presents with spinal pain, a detailed history and physical exam are an essential component of the workup to lead to a correct diagnosis. The presenting visit is also an excellent time to screen for potential risk factors for overall poor skeletal health; thus, reviewing physical activity, nutritional requirements, and hormonal balance is important.

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Bone Composition and Regulation

Water makes up approximately 5% of bone weight, while the rest is mostly bone matrix. The matrix is comprised of organic and inorganic material. The organic material includes collagen, but also proteoglycans and non-collagen proteins, as well as osteoblasts, osteocytes, and osteoclasts. Osteoblasts, derived from mesenchymal cells, are matrix-forming cells. Osteocytes originate from osteoblasts and function to preserve and maintain bone, likely sensing bone deformation and providing signals for adaptive remodeling. Osteoclasts are derived from hematopoietic stem cells and break down bone. Osteoblasts and osteoclasts are stimulated by different hormones, nutritional factors, and weight-bearing activity. The inorganic material is comprised mostly of hydroxyapatite, a compound made of calcium and phosphate [1].

Growth and mineralization of the spine involve modeling, the resorption of bone from one location via osteoclasts, and the replacement of bone in another by osteoblasts. This process is the sculpting of the skeleton that occurs until full development. Childhood and adolescence are times of substantial skeletal growth, with greater than 90% of total peak bone mass (PBM) being achieved by age 18 years [2]. In a longitudinal study by Baily et al., peak height velocity occurred on average at age 11.8 years in girls and 13.4 years in boys, with peak bone mineral

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content (BMC) velocity occurring at age 12.5 years in girls and 14.1 years in boys [3]. About 85% of the human skeleton is cortical bone and 15% is trabecular bone. Trabecular bone is strongly affected by hormonal and metabolic factors associated with puberty, while cortical bone consolidation is slower. Overall, PBM of the axial skeleton (predominantly trabecular bone) is achieved around the end of the second decade and PBM of the appendicular skeleton (predominantly cortical bone) occurs later [4]. Studies in women suggest that the size and density of vertebrae peak soon after sexual and skeletal maturity. Vertebral cross-sectional area (CSA) in women appears to stay constant from ages 15–90 years [5]. Data on men are more conflicting, with some researchers reporting no change in CSA shortly after peak height, while others have reported that CSA continues to increase in men throughout adulthood [5, 6].

After skeletal maturity, bone remodeling occurs, which involves osteoclasts' resorption of old bone followed by osteoblasts' replacement with new bone in the same location. Approximately 10% of the skeletal mass of an adult is remodeled each year. Throughout life, the work of osteoblasts and osteoclasts is coupled together via paracrine cell signaling, but bone is regulated by a variety of mechanisms. Bone mass remains constant when the rate of deposition equals the rate of breakdown. Increased bone deposition can occur when osteoblasts are more active during growth, with weight-bearing activity, with fluoride exposure, and when osteoclasts are less active. Inhibition of osteoclasts by estrogen and androgens, calcitonin, and adequate vitamin D and calcium intake all increase bone deposition as well. Osteoblasts' bone deposition is inhibited by chronic malnutrition, chronic disease, normal aging, hypercortisolism, alcoholism, lack of weight-bearing activity, and more active osteoclasts. Osteoclasts are stimulated by antigravity, hyperparathyroidism, hypercortisolism, hyperthyroidism, estrogen deficiency, testosterone deficiency, acidosis, myeloma, lymphoma, and inadequate calcium intake [1].

Minerals and Hormones Affecting Bone Regulation

The regulation of calcium and phosphate is critical to healthy bone. Both are necessary for bone mineralization, but have other functions as well. Calcium is required for skeletal muscle, nerve function, and cardiac conduction, while phosphate is important for nearly all metabolic functions. Ninety-nine percent of the body's stored calcium and 85% of the body's stored phosphate are found in the bone. Parathyroid hormone (PTH), 1,25-dihydroxyvitamin D (calcitriol), and calcitonin are the three main hormones that affect bone metabolism. PTH is located in the chief cells of the parathyroid glands, with its release stimulated by low serum calcium and dependent on adequate plasma magnesium. PTH binds to osteoblasts, which stimulate osteoclasts to resorb bone, and signals the kidney to increase calcium resorption at the expense of phosphate, with the net effect of decreased bone mineralization, increased serum calcium, and decreased serum phosphate. Calcitriol is the active form of vitamin D and is synthesized in the kidney. It is upregulated by low serum calcium, low serum phosphate, and elevated PTH. It functions by stimulating the intestines to increase absorption of calcium and phosphate and by inhibiting PTH production, with a net effect of increased serum calcium. Calcitonin is a peptide produced by the parafollicular cells (C cells) of the thyroid and has the opposite effect of PTH, but to a much smaller degree. Calcitonin's release is stimulated by elevated serum calcium. It increases renal calcium excretion and directly inhibits osteoclast activity, with a net effect of transient decreased serum calcium [7].

A number of other hormones, cytokines, and growth factors affect bone formation and remodeling. Endogenous circulating estrogens and androgens exert independently positive effects on bone growth, development, and mineral acquisition among both female and male adolescents. Later in puberty, estrogens decrease bone turnover by inhibiting bone resorption and are needed for epiphyseal closure in females and males [8]. Patients with hormonal deficiencies or

receptor abnormalities demonstrate delayed bone age and epiphyseal closures as well as lower than expected bone mineral density (BMD) [9]. Growth hormone (GH), insulin-like growth factor-1 (IGF-1), transforming growth factor beta (TGF-β), and other bone morphogenic proteins, fibroblast growth factors (FGFs), prostaglandins, and nitric oxide all exert positive effects on BMD [10, 11]. Hyperthyroidism, hyperparathyroidism, hypercortisolemia, various cytokines, and leukotrienes all directly impair bone, while hyperprolactinemia and hypopituitarism negatively impact bone indirectly via their effects on the sex steroids [12]. Decreased energy availability in adolescent athletes is associated with decreased fat mass and BMD, as well as alterations in the adipose-derived hormones leptin and adiponectin and the appetite-regulating hormones ghrelin and peptide YY [13]. While these hormones are correlated with bone turnover markers and quantitative bone measurements, further work is needed to elucidate the mechanistic effects of such hormonal alterations on bone health.

Assessment of Bone Mineral Density

In a young athlete with multiple stress fractures, or a vertebral fracture, BMD assessment is certainly warranted. Measurement of BMD via dual-energy X-ray absorptiometry (DXA) is now widely available, with both pediatric and adult reference databases. In children and young adults, only Z-scores, which compare BMD with age- and sex-matched peers, should be used. The diagnosis of osteoporosis should not be made on the basis of densitometry alone. Terms such as "low bone density for chronologic age" or "below the expected range for age" may be used if the Z-score is lower than −2.0. Use of the term "osteopenia" is discouraged. In children and young adults, the term "osteoporosis" is reserved for those with a low BMD (Z-score <-2) and evidence of fragility fractures. Because of the variations in bone with puberty and sexual development, it is often important to obtain a hand radiograph to assess bone age in those 15 years or younger. The bone age should be used to determine BMD if it significantly differs from chronologic age. In general, involvement in high-impact or odd-impact loading sports during childhood and adolescence enhances BMD and bone geometry. Thus, lower BMD than expected for age is of particular concern in a young athlete [14].

Table 17.1 Primary osteoporosis. (Reprinted from Bianchi [3], with permission from Elsevier)

Idiopathic juvenile osteoporosis
Heritable disorders of connective tissue
Osteogenesis imperfect
Ehler-Danlos syndrome
Bruck syndrome
Marfan syndrome
Osteoporosis pseudoglioma syndrome
Homocystinuria

This table lists only the most frequent diseases according to recent literature

DXA has a very low radiation dose and has good precision and reproducibility. However, it is unable to determine volumetric BMD; hence, it underestimates the BMD of small bones and overestimates the BMD of large ones. DXA also cannot distinguish between cortical and trabecular bone and has inconsistent correlations with fractures in the pediatric population. Central and peripheral quantitative computerized tomography (QCT and pQCT) provide volumetric measurements in addition to cortical and trabecular differentiation, but are primarily used in research settings, with the central QCT supplying a much higher radiation dose to patients. High-resolution-pQCT can assess bone microarchitecture, but is only available in research centers. Quantitative ultrasound has no radiation, but is much less accurate than DXA. Finally, magnetic resonance imaging (MRI) is used currently for research purposes only. Thus, for now, DXA is our best BMD clinical tool [15].

Causes of Osteoporosis

The full differential for childhood and adolescent osteoporosis is extensive and beyond the scope of this chapter, but many common and some rare primary and secondary etiologies of pediatric osteoporosis are listed in Tables 17.1 and 17.2. The most common causes in a young athlete presenting with back pain will be discussed below.

Table 17.2 Main causes of secondary osteoporosis. (Reprinted from Bianchi [3], with permission from Elsevier)

Neuromuscular disorders
Cerebral palsy
Duchenne muscular dystrophy
Prolonged immobilization
Chronic diseases
Leukemia
Diffuse connective tissue diseases
Cystic fibrosis
Inflammatory bowel diseases
Malabsorption syndromes (celiac disease)
Thalassemia
Primary biliary cirrhosis
Nephropathies (nephrotic syndrome)
Anorexia nervosa
Organ transplants
HIV infection
Endocrine diseases
Delayed puberty
Hypogonadism
Turner syndrome
Growth hormone deficiency
Hyperthyroidism
Juvenile diabetes mellitus
Hyperprolactinemia
Cushing syndrome
Inborn errors of metabolism
Protein intolerance
Glycogen storage diseases
Galactosemia
Gaucher disease
Iatrogenic
Glucocorticoids
Methotrexate
Cyclosporine
Heparin
Radiotherapy
Anticonvulsant drugs

This table lists only the most frequent diseases according to recent literature

Hormonal Abnormalities

Female Athlete Triad

The female athlete triad is the interrelatedness of energy availability, menstrual function, and bone health [16]. Energy availability is the amount of dietary energy remaining for other bodily functions after that which is needed for exercise. In athletes with restrictive eating or who overexercise, caloric intake may not meet the needs of the individual's normal function, and thus the reproductive axis is suppressed. Gonadotropin-releasing hormone (GnRH) pulsatility is diminished, leading to decreased gonadal steroids, including estradiol, which is important to prevent excess bone resorption. In addition, other anabolic hormones, such as IGF-1, are decreased [17]. Thus, athletes with decreased energy availability and subsequent menstrual dysfunction are at higher risk for decreased BMD, stress fractures, and fragility fractures [16].

Because female athlete triad is a diagnosis of exclusion, in a female athlete with menstrual dysfunction, it is important to rule out other non-energy availability causes. These include a prolactinoma, hypo- and hyperthyroidism, polycystic ovarian syndrome, and other hypogonadal states. In general, if hypothalamic amenorrhea or female athlete triad is diagnosed, patients often need a multidisciplinary approach involving a physician, nutritionist, and sports psychologist [18]. But if pathologic behavior has led to extremely delayed menses (primary amenorrhea) or years without cycles after experiencing menarche (secondary amenorrhea), peak bone mass may never reach normal levels [19].

Hyperparathyroidism

Hyperparathyroidism can occur from either primary or secondary causes. Classic symptoms of hyperparathyroidism are symptoms of hypercalcemia, such as constipation, nausea, abdominal pain, confusion, and in severe cases stupor and coma. However, milder hyperparathyroidism may initially be asymptomatic, and may first be identified secondary to routine screening of serum calcium. Primary hyperparathyroidism causes include solitary parathyroid adenoma, multiple gland hyperplasia disease, and parathyroid carcinomas [20]. The incidence of primary hyperparathyroidism is approximately 1 in 1,000 people. Asymptomatic primary hyperparathyroidism can be discovered incidentally through

routine screening of serum calcium. Other causes of hyperparathyroidism include vitamin D deficiency, hypocalcemia, familial hypocalciuric hypercalcemia, malignancy such as lung carcinoma overproducing PTH, and multiple endocrine neoplasia (MEN) syndromes [1].

Bone loss in hyperparathyroidism occurs as PTH stimulates osteoclasts to reabsorb bone to release more calcium [7]. Despite the fact that hyperparathyroidism exerts a more negative effect at sites with a large proportion of cortical bone versus trabecular bone (e.g., the forearm and hip), there still may be an increased risk for vertebral fractures with hyperparathryroidism [21]. Hyperparathyroidism is suspected when there is an inappropriately normal or elevated serum PTH level in the setting of hypercalcemia. With primary hyperparathyroidism, urinary calcium excretion is either normal or elevated and vitamin D levels are often normal or elevated as well. Other tests such as laboratory markers of bone turnover, DXA, renal imaging, and adenoma localization studies can be considered but are not required for the diagnosis. Spinal imaging may or may not demonstrate diffuse osteopenia [22].

The mainstay for curative treatment for symptomatic primary hyperparathyroidism is parathyroidectomy. Those who are asymptomatic should be monitored closely and if there is evidence of low BMD or the patient becomes symptomatic, then surgery should be considered [23]. Treatment for secondary hyperparathyroidism includes dietary phosphate restriction, phosphate binders, vitamin D supplementation, and calcium supplementation, but obviously varies depending on etiology.

Thyroid Disorders

Thyroid hormone stimulates bone resorption directly through osteoclast activation as well as via osteoblasts, which mediate osteoclast bone resorption. Overt hyperthyroidism is associated with increased bone turnover, decreased BMD, and increased fracture risk [24]. Hyperthyroidism is usually a very correctable etiology of osteoporosis. In order to reduce the risk of low BMD, hyperthyroidism should be aggressively treated with antithyroid drug therapy, radioactive iodine, or thyroidectomy followed by replacement thyroid hormone. Rarely, hypothyroidism can lead to joint aches and pains, as well as stiffness, but bone pain related to thyroid disease is much more correlated with hyperthyroidism [25].

Iatrogenic Steroid Use

Systemic corticosteroids, when taken chronically, have an overall net effect of decreased BMD secondary to inhibition of osteoblast production, reduced calcium absorption, down-regulated synthesis of calcitriol, and decreased gene expression of calcium-binding protein [26]. Corticosteroids increase calcium secretion in the kidney which leads to elevated PTH levels [27]. With chronic glucocorticoid therapy, osteoporosis can occur in up to 50 % of patients. Treatment includes adequate calcium and vitamin D intake and sometimes bisphosphonates. Because of the long-term half-life and effects of bisphosphonates and potential teratogenic effects, bisphosphonate use in pre-menopausal women and young men should be carefully considered [28].

Abnormalities in Vitamin and Mineral Availability and Metabolism

Vitamin D Deficiency

As previously mentioned, vitamin D is essential to calcium and phosphorus homeostasis and helps regulate bone formation and maintenance. Vitamin D can be ingested in the form of vitamin D_2 (ergocalciferol) or vitamin D_3 (cholecalciferol), and can also be produced in the skin secondary to sunlight exposure. It is stored in and released from fat cells. Vitamin D is converted to 25-hydroxyvitamin D [25(OH)D] in the liver. This is the major circulating form of vitamin D that is measured to determine vitamin D status. 25(OH)D is biologically inactive and must be converted in the kidneys to the biologically ac-

tive 1,25-dihydroxyvitamin D [1,25$(OH)_2$D or calcitriol]. Serum calcium, phosphorus, FGF-23, and other factors can modify the renal production of calcitriol [29]. Vitamin D deficiency is seen in 30–50 % of young, healthy Americans. Winter months, higher latitudes, malabsorption (e.g., celiac disease), use of anticonvulsants that impair the P450 enzymes, dark skin, sunscreen use, and less exposure to sunlight are risk factors for nutritional deficiency [30]. Vitamin D deficiency can lead to severe disorders of bone mineralization related to abnormalities in calcium metabolism, such as hypocalcemia and tetany, hypophosphatemia, osteomalacia, and in those with open growth plates, even rickets [1]. In the young athlete population, low vitamin D levels are often asymptomatic and discovered because of fractures. Diagnosis of vitamin D deficiency is made by obtaining a serum 25(OH)D level, with a level greater than 30 ng/mL considered normal [31].

If the 25(OH)D level is low, supplementation with 2,000 international units (IU) of vitamin D daily or 50,000 IU weekly for 6–8 weeks is suggested, until a normal level is obtained. To maintain a normal vitamin D level, the American Academy of Pediatrics recommends 400 IU daily of vitamin D supplementation for all infants and those children and adolescents who do not have adequate dietary intake [32]. However, many bone experts and the Endocrine Society suggest up to 1,000 IU may be necessary to maintain appropriate levels [33]. In those with malabsorption, obesity, or those on medications that may increase vitamin D catabolism, doses may need to be much higher [15].

Hypocalcemia

There are many reasons one may have hypocalcemia, such as primary or secondary hypoparathyroidism, drug-induced hypocalcemia, hypomagnesemia, and genetic disorders. Etiologies of childhood hypocalcemia include hypoparathyroidism, autosomal dominant pseudohypoparathyroidism (parathyroid resistance), hypomagnesemia, and vitamin D deficiency [34]. Acute hypocalcemia presents very differently from chronic hypocalcemia. With acute hypocalcemia, the hallmark clinical finding is tetany or neuromuscular irritability, as seen with Trousseau's sign and Chvostek's sign. Seizures, papilledema, hypotension, and psychiatric changes may also occur. Chronic hypocalcemia is quite rare, and when associated with hypoparathyroidism can cause cataracts, as well as basal ganglia calcifications that may lead to Parkinsonism or dementia. Skeletal abnormalities are not directly associated with hypocalcemia but with the underlying etiology. Post-surgical hypoparathyroidism may have increased BMD [35] but patients with congenital hypoparathyroidism may have craniofacial abnormalities and osteosclerosis [36]. Another finding is a prolonged QT interval as seen on electrocardiography (ECG). Radiographs of the wrists or knees could be obtained to evaluate for rickets.

Ideally, treatment of hypocalcemia should be with oral calcium replacement, but if the patient is not stable, then intravenous (IV) supplementation is required. The adequate calcium intake varies by age and is related to pubertal status, lactation, and pregnancy status. Children of ages 1–3 years require 500 mg/day of calcium, children of ages 4–8 years require 800 mg/day of calcium, and children of ages 9–18 years require 1,300 mg/day of calcium. Adults ages 19–50 require 1,000 mg/day of supplementation, but as one continues to age the requirement increases to 1,200 mg/day [37].

Hypophosphatemia

Hypophosphatemia can be a result of other conditions such as vitamin D deficiency, chronic diarrhea, chronic ingestion of antacids, starvation, or alcoholism [38]. Common symptoms of hypophosphatemia include bone pain, confusion, and muscle weakness [39]. In some cases, hypophosphatemia can be caused by an inborn error of metabolism leading to hypophosphatemic rickets, also known as vitamin D-resistant rickets. The most common inborn error is X-linked hypophosphatemic rickets, in which there is a loss-of-function mutation in the PHEX gene that

encodes for a metalloprotease enzyme that allows for leakage of phosphorus at the renal tubular level. Laboratory results include low serum phosphate, an inappropriate normal serum level of $1,25(OH)_2D$, and a normal calcium level. Patients with hypophosphatemic rickets will develop bowing of the legs but not tetany. There is a less common autosomal form of hypophosphatemic rickets that has a variable age of onset from childhood to adulthood. Another separate form of hypophosphatemic rickets is an autosomal recessive form with hypercalciuria, leading to renal stone disease [40].

Other Metabolic Causes of Spinal Pain

Idiopathic Juvenile Osteoporosis

Idiopathic juvenile osteoporosis (IJO) is a rare form of primary osteoporosis and is a diagnosis of exclusion. IJO typically presents at an average age of 7 ± 6 years, in previously healthy children. Fractures of weight-bearing bones are commonly seen, especially of the vertebrae. Kyphosis and fractures of the knee and ankle may also be seen. There is no consensus for treatment for IJO and typically there is spontaneous remission after puberty. Growth and mineralization are usually restored, but in some cases kyphoscoliosis or collapse of the rib cage is permanent [41]. It is important to differentiate IJO from osteogenesis imperfecta (OI).

Osteogenesis Imperfecta

OI or "brittle bone disease" is a spectrum of rare genetic diseases that results from a collagen defect that affects the quantity and/or quality of collagen produced. It should be considered in a child with a history of multiple fractures. Most forms of OI are autosomal dominant. Classic findings are blue or gray sclera secondary to abnormal collagen in the eye, and teeth that are opalescent secondary to decreased opaque dentin and increased transparent enamel, termed dentinogenesis imperfecta [27]. Other associated clinical manifestations include short stature, scoliosis, skull deformities, hearing loss, increased laxity, and easy bruising.

A familial history of OI is seen in about 65 % of cases. A person with mild OI may have as few as ten fractures in their lifetime, versus a patient with severe OI, who may endure several hundred fractures [41]. The prevalence of OI is thought to be 1 in 10,000 individuals. Most individuals with OI have a defect in either COL1A1 or COL1A2, which encode the two chains of type I collagen. There are nine types of OI described in the literature, but four main types of OI described by Sillence: type I is a mild phenotype with quantitatively deficient normal collagen and typically presents with bone fragility, blue sclera, and near-normal stature; type II is lethal in the perinatal period with either abnormal collagen or severe quantitative deficiency; type III is a severe, progressive deforming type involving abnormal collagen production and presents with severe growth restriction; and type IV involves abnormal collagen production and has a mild-to-moderate presentation with low-to-normal stature [27, 42].

Aside from the symptomatic treatment of fractures associated with OI, the mainstay medical treatment for severe OI is cyclical administration of IV bisphosphonates, such as pamidronate, as well as calcium and vitamin D supplementation. Osteopetrosis is a complication of bisphosphonates and thus caution is required with treating type I OI with these medicines, in addition to the risks previously mentioned, relating to the long half-lives of these drugs [27]. Recent studies have demonstrated mixed results. A study by Ward, et al. demonstrated that treatment orally with the bisphosphonate alendronate for OI types I, III, and IV led to significantly decreased bone turnover and increased spinal BMD, but no associated improvement in fracture incidence, height of the vertebral bodies, cortical thickness, mobility, or bone pain [43]. Larger, longer duration studies are needed. Treatment of scoliosis associated with OI can be very challenging and bracing may be ineffective [27].

Oncologic Processes

Less than 10% of bone tumors are primary spinal tumors, which occur much less frequently than metastatic lesions. However, many cancers metastasize to the spine. Often, the first indication of a pathologic fracture is pain at the fracture site. Lack of trauma should heighten suspicion for a pathologic fracture as well. Screening laboratory tests should be obtained if there is a suspicion for tumor, including complete blood count, erythrocyte sedimentation rate, C-reactive protein level, alkaline phosphatase, lactate dehydrogenase (LDH), and uric acid [44, 45]. Cancers that commonly metastasize to the spine include breast, kidney, thyroid, and lung in adult patients, and neuroblastoma in children. Multiple myeloma is the most common primary neoplasm of the spine, although this tumor occurs in older adults [46]. In younger patients, primary malignant tumors include chordoma, chondrosarcoma, and malignant fibrous histiocytoma. Primary benign tumors include osteoid osteoma and osteoblastoma, which should be considered in the differential diagnosis of a young patient with axial pain, painful scoliosis, or radicular pain. Vertebral hemangiomas are not uncommon benign spinal lesions, with an overall incidence of 10% in the population, but less than 1% of hemangiomas are believed to be symptomatic. Osteochondroma, aneurismal bone cysts, eosinophilic granulomas, and giant cell tumors are other benign lesions of the spine [44].

Conclusion

Spinal pain in the young athlete may not simply represent a muscle strain, but may indicate a more worrisome underlying disease. Clinicians must have an understanding of the various etiologies of bone pain to recognize less common but significant disorders. These include hormonal abnormalities, vitamin and mineral availability and metabolic abnormalities, and other less common causes such as IJO, OI, and oncologic processes. Imaging and a screening laboratory assessment may be necessary when a patient's history and examination indicate a bony abnormality. Interpretation and understanding of these results can help minimize lifelong complications with growth and bone health.

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Infectious and Inflammatory Diseases Affecting the Young Athlete's Spine

18

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Abbreviations

ESR	Erythrocyte sedimentation rate
CBC	Complete blood count with differential
PVO	Pyogenic vertebral osteomyelitis
PADI	Posterior atlanto-dens interval
AADI	Anterior atlanto-dens interval
JIA	Juvenile idiopathic arthritis
JRA	Juvenile rheumatoid arthritis
RA	Rheumatoid arthritis
SpA	Spondyloarthritis
IBD	Inflammatory bowel disease
ESSG	European Spondyloarthropathy Study Group
JSpA	Juvenile spondyloarthritis
CRP	C-reactive protein level
MRI	Magnetic resonance imaging
CT	Computed tomography
WBC	White blood count
MDR TB	Multidrug resistant tuberculosis
PCR	Polymerase chain reaction
AFB	Acid-fast bacillus
PPD	Purified protein derivative

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Introduction

The pediatric athlete is a unique patient and disorders of the spine in the pediatric athlete can be challenging for practitioners. The spine is vulnerable to a variety of insults which can be localized or systemic, acute or chronic, and which may preferentially affect patients of a pediatric or adolescent age. Back pain is common in the pediatric athlete, and correctly diagnosing infectious or inflammatory causes is critical for optimal medical care, for the growth and development of the patient as well as for their future participation in sports. Sports medicine specialists and orthopedists must be familiar with infectious and inflammatory spinal pathologies that may affect the young athlete. This ensures the recognition of spinal complaints and associated systemic symptoms, while expediting appropriate workup, initial management, and prompt referral of affected patients. This review is intended to aid healthcare practitioners in treating the young athlete, but is not intended as an exhaustive review.

Spine Infections and Implications for the Pediatric Athlete

Spine infections comprise bacterial, fungal, mycobacterial, and parasitic infections that occupy the vertebral body, disc or bony posterior elements, the paravertebral soft tissues, and/or the epidural space of the spinal axis and can occur by direct insult, extension from a nearby infec-

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Table 18.1 Demographics of spinal infections

Type of infection	Age distribution	Association with immunocompromise	Risk factors	Frequency of surgical treatment	Risk of late deformity	Frequency of neurological deficit
PVO	Older	Somewhat	IVDU, HIV, immunocompromise	Moderate	Moderate	Moderate to frequent
Pott	Children, any age	Yes, but not always	Endemic exposure	Moderate	Frequent	Moderate
Brucella	Men > 40	No	Animal exposure	Low	Low	Low
Fungal	All	Yes, except coccidiomycosis and histoplasmosis	Immunocompromise, endemic areas	Low	Rare	Low

PVO pyogenic vertebral osteomyelitis, *IVDU* intravenous drug use

tion, or hematogenous seeding of spinal tissue through arterial or venous routes. Although spine infections are no more common in the pediatric athlete than in the adult, back pain is common, and differentiating back pain from infection can be troublesome for the clinician [24]. Delay in diagnosis may have devastating consequences for the patient; however, promiscuous workup of back pain in the pediatric athlete is expensive and impractical. Spine infections affect different demographics of patients as demonstrated in Table 18.1. In addition, infectious diseases of the spine can occur at increased frequency in pediatric athletes with risk factors for infection due to immune-compromised health or a specific high-risk exposure. In the USA, the most common spinal infection is pyogenic vertebral osteomyelitis (PVO), but in other regions of the world, brucellosis and tuberculosis are more common etiologies [44]. Many athletes are involved in international competitions and thus a variety of organisms must be included in the differential diagnosis for spine infection.

Pediatric Discitis and Pyogenic Vertebral Osteomyelitis

Pediatric discitis and PVO can affect young athletes, although there is no evidence that these infections have any predilection for the young athlete. Experts generally conclude that discitis is an isolated bacterial infection of the intervertebral disc and adjacent endplates. It is a mild manifestation of infectious spondylitis in contrast to vertebral osteomyelitis with epidural or paraspinal abscess at the other end of the continuum [14]. These entities differ strikingly in their presentation, epidemiology, and prognosis [18].

The anatomy of the pediatric blood supply explains both the pathoetiology and self-limited nature of discitis. In the immature spine, the circulation to the avascular disc traverses arterial channels in the cartilaginous endplate. These channels, which persist from fetal life until the ring apophyses fuse in the third decade of life, allow direct inoculation of the relatively immunoisolated disc tissue during episodes of bacteremia. Meanwhile, until about age 15 the vertebral body enjoys a rich anastomotic vascular network, which suppresses growth of interosseous septic emboli and extension of disc infection in most cases. Thus, vertebral body involvement is somewhat rare in children. In cases of pediatric discitis, the infection may be self-limited as the vertebral body's rich blood supply protects it from spread of the infection and sustains a sufficient response to quell the neighboring disc infection even without antibiotics [14, 18].

Discitis classically has a bimodal pediatric age distribution with peaks in early childhood and adolescence [13]. Vertebral osteomyelitis is generally thought of as a disease affecting adults but there is also a peak in incidence among adolescents [18, 47]. Fernandez et al. highlight the epidemiologic differences between patients presenting with discitis versus those presenting with vertebral osteomyelitis. They note that vertebral

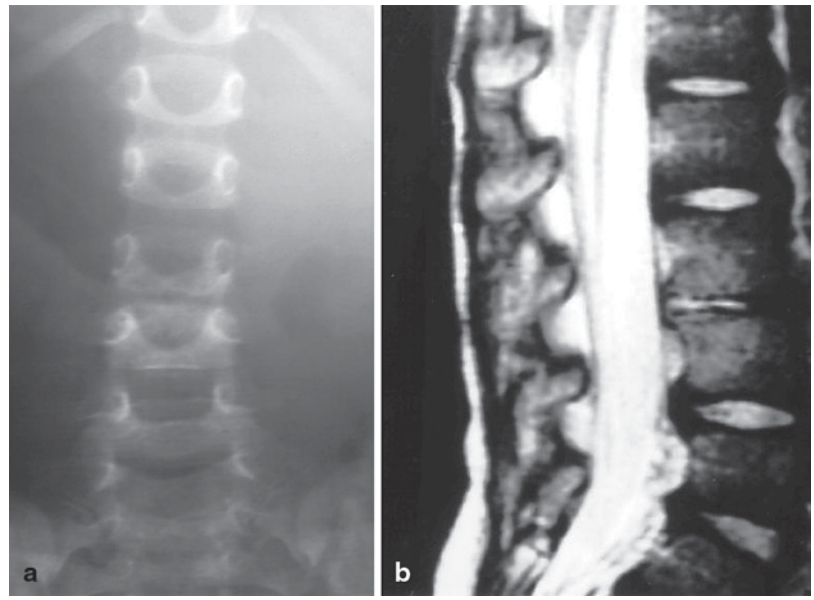

Fig. 18.1 **a** XR pediatric discitis. Posteroanterior radiograph of a 3-year-old girl with 2-week history of irritability and refusal to walk for 2 days. The disc space narrowing at L3/4 is consistent with discitis. **b** T2 MRI Pediatric discitis. Sagittal T2-weighted MRI demonstrating loss of normal signal intensity and disc height at L3/4 and mild signal increase at adjacent vertebral bodies, consistent with discitis. (© 2003 American Academy of Orthopaedic Surgeons. Reprinted from [14], pp. 413–420, with permission)

osteomyelitis was infrequently encountered in children younger than 3 years and discitis was rare in children older than 8 years, a finding that differs somewhat from that of Cushing et al. Furthermore, discitis patients were far less likely to present with a history of fever than PVO patients (28 % vs. 79 %). Only three discitis patients had fevers exceeding 101 °F whereas all of those PVO patients with fever exceeded 102 °F. Importantly, they note that radiographs were insensitive for PVO and even in cases with strong clinical signs of spinal axis infection, discitis and PVO were not radiographically distinguishable without magnetic resonance imaging (MRI).

The incidence of discitis is estimated to be between 3 and 6 cases per 100,000, typically affecting children younger than 5 years of age [13, 44]. Suspicion of pediatric spine infection is raised in patients with progressive refusal to sit, crawl, or walk without symptoms that localize to either lower extremity, whether fever is present or not. Symptoms will vary depending on age and may include abdominal pain, limp, or generalized irritability [14] and may have persisted for several weeks prior to presentation. Although most patients with discitis are otherwise healthy, a history of antecedent infection such as otitis media or urinary tract infection is not uncommon [14]. The initial workup includes a full set of vital signs and physical examination of the spine and lower extremities including a spine-focused neurologic examination. Evaluation of the limping child must include examination of the spine [14].

Laboratory workup includes complete blood count with differential (CBC), erythrocyte sedimentation rate (ESR), C-reactive protein level (CRP), and at least one peripheral blood culture. Imaging must include posteroanterior (PA) and lateral radiographs of the spine with dedicated views of the lumbar spine or other regions of suspected infection (Fig. 18.1a). We advocate a low threshold for obtaining a spinal MRI containing T1-weighted axial and sagittal images and T2-weighted sagittal images (Fig. 18.1b). MRI is helpful in cases of atypical clinical or radiographic presentation of discitis and in ill-appearing children where progression to PVO is a possibility. MRI is essential in cases with a neurologic abnormality, a poor clinical response to empiric discitis treatment, or when a presentation is worrisome for noninfectious spinal pathology. Tc 99 bone scan is of limited utility in older children as it is nonspecific, but in a child too young to localize symptoms, it can help to distinguish spinal pathology from that of the sacroiliac joints, hips, and lower extremities [14].

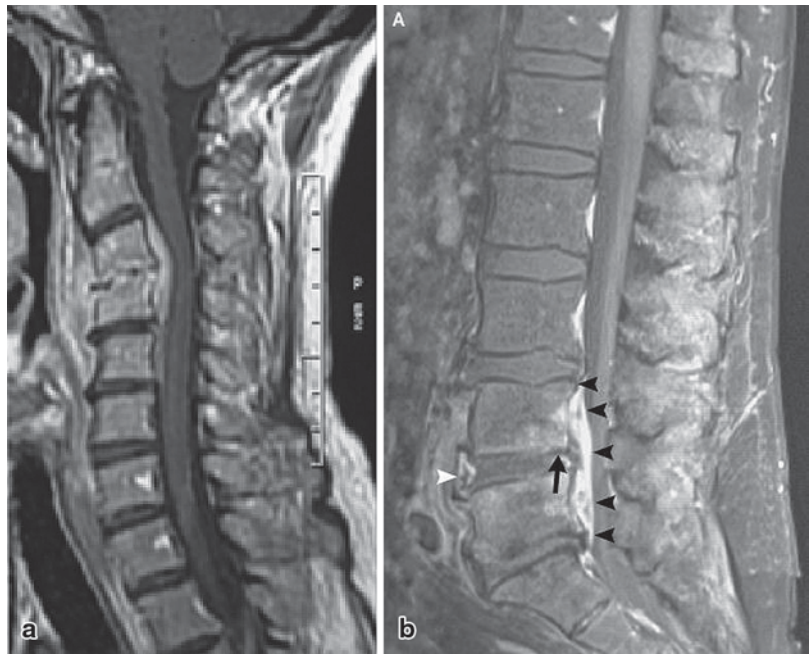

Fig. 18.2 **a** T1 MRI cervical osteomyelitis. Sagittal T1-weighted MRI demonstrating cervical osteomyelitis at L3/4 with epidural abscess (© 2004 American Association of Neurological Surgeons. Reprinted from Neurosurgery Focus [3], p. 2, used with permission). **b** XR *E. coli* Vertebral Osteomyelitis. Fat-suppressed, gadolinium-enhanced, T1-weighted MRI of 57-year-old man with PVO caused by *E. coli*. *Black arrowheads* point to epidural abscess, *white arrowhead* depicts small paravertebral abscess, *black arrow points* to enhancement in the bone of the involved vertebral bodies. (© 2010 Massachusetts Medical Society. Reprinted from [58], used with permission)

Workup of suspected discitis or osteomyelitis merits inpatient admission whether a firm diagnosis is made or not. Antibiotics are the mainstay of treatment and are empirically targeted at staphylococcal organisms in most cases. Obtaining blood cultures prior to starting antibiotic therapy is preferable, but antibiotics should not be withheld from a toxic child. CRP should fall soon after initiation of treatment. An early increase in CRP indicates inadequate coverage and a late increase indicates inadequate duration of therapy or residual sequestrum. As with all pediatric musculoskeletal infections, immobilization is primarily a comfort measure; the efficacy of bracing to prevent deformity is controversial. When an infection is unresponsive to treatment and blood cultures have not been useful, computed tomography (CT)-guided percutaneous biopsy or even open biopsy may be required [14, 18]. Follow-up radiographs at regular intervals for 12–18 months should be evaluated to rule out recurrence or late deformity [14].

There is limited literature on the long-term results of nonoperative treatment for pediatric spondylodiscitis. One study retrospectively reviewed 25 patients, 75% of whom had radiographic and clinical follow-up a minimum of 10 years after treatment. The study found that although 20% of patients at follow-up had restricted spine mobility and mild pain and 100% had evidence of permanent radiographic changes, ranging from decreased disc space to ankylosis of adjacent vertebrae, there were no serious functional or neurologic deficits in any of the patients [31].

Vertebral osteomyelitis is an infection of the vertebral body and intervertebral disc, associated with epidural abscess in 17% (Fig. 18.2a, b) of cases and paravertebral abscess in 26% of cases. Most cases result from hematogenous seeding from distant infection or transient bacteremia as is common in intravenous drug abusers or immune-compromised hosts. Direct inoculation can result from penetrating trauma [36] and open fractures, or from prior operative or percutaneous procedures [17]. Direct extension from a retroperitoneal or retropharyngeal infection can also lead to vertebral osteomyelitis [26]. PVO, secondary to intrapelvic infections, due to seeding

of the spine along Batson's plexus has been reported [26]. When hematogenous seeding of the vertebra is suspected, the primary site of infection is only found in half of the cases. Importantly, multifocal disease must also be ruled out [58].

Patients present with pain along the spinal axis in 86–92% of cases [40, 44]. Back pain is commonplace in athletes; however, pain located in the midline rather than paraspinal pain, severe pain waking patients from sleep, pain not proceeded by training or trauma, and pain accompanied by history of fever are worrisome symptoms that should not be dismissed as routine. Although history of fever, night sweats, and anorexia are common at presentation for PVO, initial clinical evaluation may reveal fever in less than half of the cases [58]. Neurologic compromise occurs in about one-third of patients [40].

Uncontrolled PVO can lead to systemic infection and sepsis, spinal instability, or neurologic compromise resulting from either instability or epidural abscess [8]. When PVO is suspected, workup includes vital signs, a full-extremity neurologic examination, with documentation of rectal tone and perianal sensation, and laboratory work including CBC, ESR, CRP, and peripheral blood cultures. Elevated ESR and CRP have been reported in 98 and 100% of PVO cases, respectively [58]. CRP is also a sensitive marker of the clinical response to treatment and can indicate resolution or treatment failure when followed longitudinally. At least two peripheral blood cultures should be drawn prior to antibiotic administration and sent for routine culture as well as specialized culture for acid-fast bacilli (ACB) and Brucella species in patients suspicious for those exposures [58]. Mylona et al. found 58% of blood cultures positive in their meta-analysis of clinical variables in PVO. Imaging for suspected PVO includes full-length standing anteroposterior (AP) and lateral X-rays of the spine as well as dedicated views of the suspected region. MRI with gadolinium of the entire spine will show the extent of disease and detect epidural abscess, but cannot reliably predict structural stability of the spine. CT scan is more sensitive for determining the structural damage to the spine and may augment preoperative planning. Treatment involves intravenous antibiotics, medical and nutritional optimization, and evaluation by an infectious diseases expert and spinal surgeon to determine whether operative debridement is required. Mylona et al. indicated a relapse rate of 8% and a mortality rate of 6%.

Spinal Brucellosis

Brucellosis is a systemic zoonotic bacterial infection caused by various facultative intracellular bacteria of genus Brucella. The infection occurs worldwide with the highest frequency seen in the Mediterranean, the Middle East, and Central and South America. Brucellosis is rare in the USA with only about 200 cases reported annually [50]. Humans contract the infection by contact with domesticated animals. Working and middle-aged men are at highest risk, while children and elderly people are at lower risk. Raw milk is a common cause of infection in children [1]. Although spinal involvement may only occur in 6–58% of cases [59], Brucellosis remains a frequent cause of spine infection in endemic areas evidenced by a recent study from Greece citing it as the infectious organism in one-third of spinal infections [46].

Musculoskeletal manifestations of infection include spondylitis, sacroiliitis, arthritis, osteomyelitis, tenosynovitis, and bursitis [9]. The knee and the sacroiliac joints are more frequent sites of infection in children and young adults. However, spinal involvement can occur and the spine is the most frequent location of musculoskeletal Brucellosis in older adults. Brucellar spondylitis presents most commonly in the lumbar spine (60%), followed by the thoracic (19%), and cervical spine, respectively (12%) [9]. The location of infection in the spine can be localized or diffuse, with multilevel involvement at presentation in about 6–14% of cases [2, 9, 59]. In local disease, the infection has a predilection for the anterior superior vertebral endplate (Fig. 18.3) and the subjacent intervertebral disc and can be mistaken for degenerative disc disease or erosive osteochondrosis in light of its predominant lumbar distribution.

MRI and post-contrast CT imaging can demonstrate associated paravertebral, psoas, and epi-

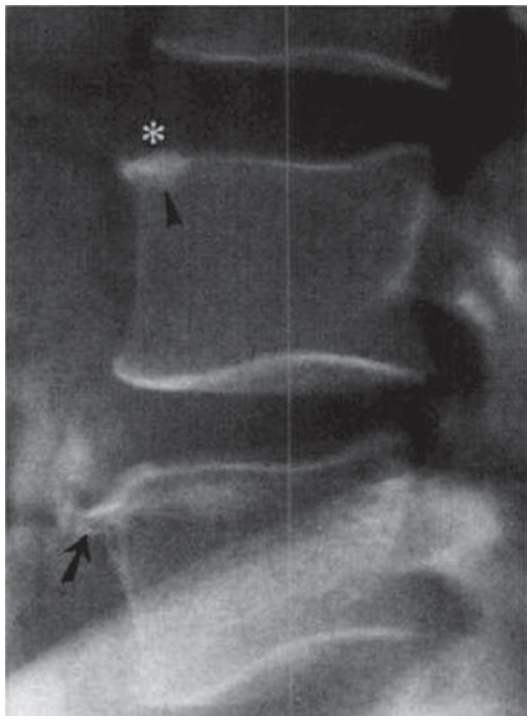

Fig. 18.3 Brucellar spondylitis anterior osteophyte and disc gas. Focal Brucellar spondylitis with an anterior osteophyte (*arrow*), bony sclerosis (*arrowhead*), and disc gas (*asterisk*). (© 1994 Radiological Society of North America. Reprinted from [2], used with permission)

dural abscesses as well as early disc and endplate changes not evident on plain films [9]. Brucellar spondylitis also has similarities in presentation and radiographic appearance to tuberculous spondylitis (hence the term pseudo-Pott's disease) and pyogenic spondylitis, which can make diagnosis difficult in the absence of positive cultures. Treatment with an anti-brucellar antibiotic regimen and supportive therapy is effective in the majority of cases. Unless tissue is needed to confirm the diagnosis, surgery is rarely indicated in the absence of neurologic deficit [51]. Complications of late spinal deformity and neurologic compromise are rare [9, 51].

Tuberculosis and Pott's Disease

Tuberculosis of the spine, known as Pott's disease, is an infection of the spine and paraspinal soft tissues by Mycobacterium tuberculosis or a similar mycobacterial organism. The incidence of Pott's disease varies widely and correlates both to the endemic incidence of tuberculosis (TB) and to the endemic incidence of malnutrition and co-morbid diseases such as human immunodeficiency virus (HIV). In areas with a higher endemic burden of TB, there is a higher incidence of Pott's disease in pediatric populations [50]. In the USA, TB has an incidence of 3.8 cases per 100,000 persons, but has been reported up to 50 times higher in inner cities as recently as the 1990s. The proportion of spinal involvement varies; pediatric patients and HIV-positive patients are more likely to have extrapulmonary and spinal diseases [20]. Some reports estimate greater than 60% extrapulmonary involvement in HIV-positive patients with TB [27, 57]. Approximately 50% of musculoskeletal TB involves the spine [27].

Outside the lungs, the spine is the second most common location of TB, following only lymph node involvement. The pathoetiology of spinal TB differs from that of PVO in that nearly all cases of Pott's disease result first from hematogenous spread from the lung, despite a substantial incidence of pulmonary involvement that remains subclinical. Hematogenous inoculation of the vertebral body is followed by extra corporeal spread along the path of the anterior longitudinal ligament, to adjacent as well as distant vertebral bodies. The posterior elements are often involved and are the sole area of involvement in 2–10% of lesions, a risk factor for paraplegia [27]. In serious cases, spinal TB can lead to severe kyphotic deformity, as shown in Fig. 18.4, or neurologic compromise [28].

The suspicion of Pott's disease increases when back pain is accompanied by chronic malaise, weight loss, night sweats, or intermittent fevers in patients with a history of exposure to TB [50]. Pott's disease can present with or without active pulmonary infection [50]. Laboratory workup includes white blood count (WBC), ESR, CPR, and imaging with full-length standing spinal X-rays and a full spine MRI with gadolinium (Fig. 18.5) and also includes PA and lateral chest X-rays and the placement of a purified protein derivative

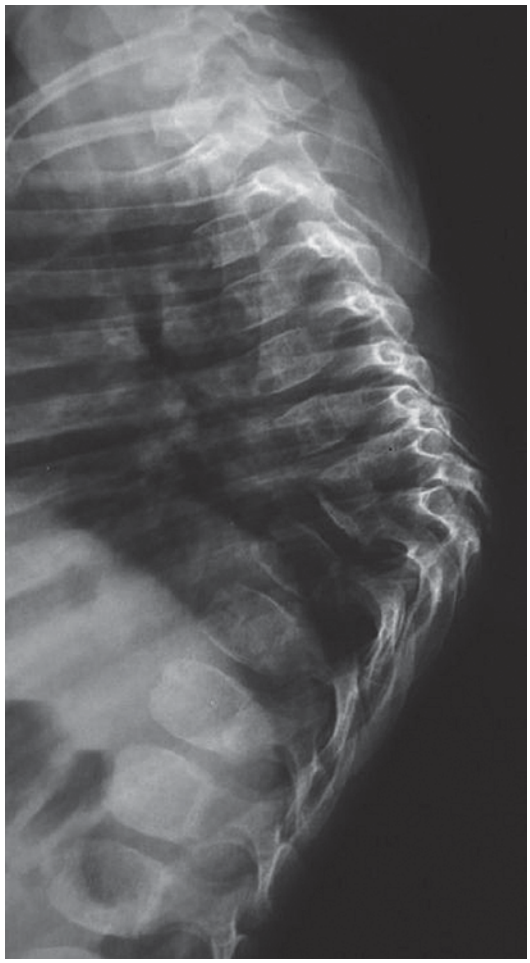

Fig. 18.4 XR spinal TB kyphosis. A lateral radiograph of the thoracic spine of a 3-year-old child showing involvement of T7–T10 with severe kyphosis. (© 2010 British Editorial Society of Bone and Joint Surgery. Reprinted from [27], used with permission)

(PPD). CT-guided biopsy of the lesion followed by polymerase chain reaction (PCR)-based rapid diagnosis and culture should be used to determine anti-mycobacterial sensitivities. If empiric therapy is used, multidrug resistant (MDR) TB should be suspected if there is no significant improvement in 3 months. These patients should still be regarded as potentially contagious through an airborne route as positive sputum AFB cultures in chest X-ray-negative patients with spinal TB have been reported [48].

Although treatment is typically nonoperative, surgical indications include (1) neurologic compromise, (2) unresponsiveness to anti-tubercular treatment or MDR TB, (3) kyphosis greater than 60° in an adult, less in a growing child (Fig. 18.4), (4) panvertebral lesion, (5) intradural or intramedullary involvement, and (6) debilitating residual pain [27]. Debridement, stabilization, and corrective osteotomy have an important role in management of the progressive kyphosis typically associated with Pott's disease. With ongoing anti-tubercular treatment, spinal hardware infection is unlikely [27, 41]. Although "healed status" can be designated after 2 years without clinical or radiographic recurrence, late progression of kyphosis and associated neurological deficit remain concerns [27].

Fungal Infections of the Spine

Fungal infection of the spine in pediatric or adult patients is typically the result of systemic fungemia, which is rare in immunocompetent hosts. When patients presenting with fungemia or known localized fungal infections complain of back pain, suspicion of fungal spondylitis should be raised. These infections tend to be indolent; thus, delay in diagnosis is common. Pain typically increases in proportion to local involvement as well as associated compression fractures and paravertebral abscesses [32, 50]. Fungal organisms frequently reported to affect the spines of immunocompromised hosts include *Candida albicans*, other *Candida* species, *Nocardia asteroids*, and *Aspergillus fumigatus* [19]. Vinas et al. reviewed 39 published cases of spinal aspergillosis finding only monomicrobial infections that predominated in lumbar spine (54%), in a 78% male population with an average age of 40 years [56].

Disseminated *Coccidioides immitis*, and rarely *Histoplasma capsulatum* and related species, infections can affect the spine in immunocompetent hosts. Coccidiomycosis is a fungus, endemic to California, Arizona, and the southwestern USA as well as northern Mexico that can cause systemic infection even in healthy patients including young athletes. Infections may involve the axial and/or appendicular skeleton in 20–50% of

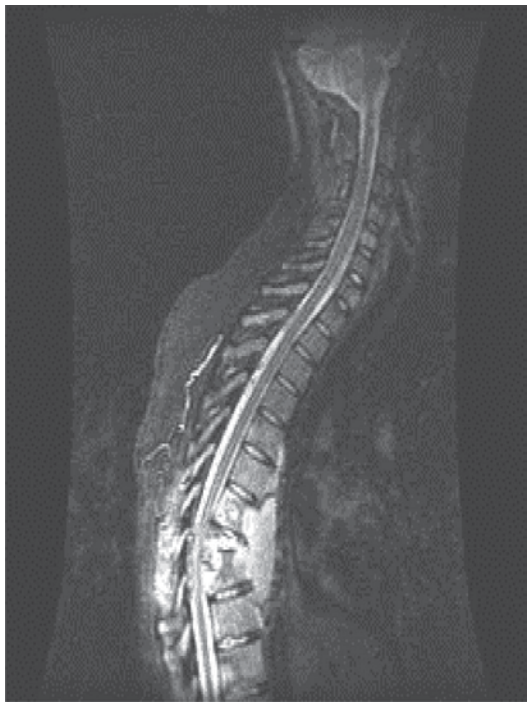

Fig. 18.5. MRI spinal TB. MRI showing spinal tuberculosis with destruction of vertebral bodies. (© 2006 Elsevier. Reprinted from [35], used with permission)

disseminated cases [33]. There are an estimated 100,000 cases worldwide per annum [12]. Frazier et al. reviewed their experience in treating spinal fungal infections and found that 10 of 11 patients required surgical treatment in addition to antifungals [19], though this retrospective study of surgical patients most likely overestimates the true proportion of cases that require surgical treatment.

Risks of Spinal Infections in the Pediatric Athlete

Post-infectious instability and deformity can result from any spinal infection and are a cause of late morbidity that may impair the pediatric athlete. In addition to routine radiographic follow-up for up to 24 months, regular physical examination during the recovery period should include a spine examination with particular attention to progressive kyphosis and neurologic changes. Return to play after a spinal infection should be guided by a physical therapist after an orthopedist or spine surgeon has determined participation to be safe. Any pain that is new, severe, or does not improve and any new neurological symptoms are red flags, as are fevers and constitutional symptoms. If any question remains about the fitness of an athlete's spine, prompt evaluation by a spine surgeon is indicated.

Inflammatory and Autoimmune Diseases of the Spine

Inflammatory disorders often have initial presentations in childhood and adolescence. Given that the initial presentation may be pain in many circumstances, the diagnosis in a pediatric athlete can be confusing. Inflammatory disease is a well-established cause of spinal dysfunction. Vertebral ligament and capsular insertions and the discovertebral junction are targets of autoimmunity in seronegative spondyloarthropathies, whereas the synovial joints are the initial targets of rheumatoid arthritis (RA) and similar autoimmune conditions. Inflammatory diseases affecting the pediatric spine in particular include enthesitis-related arthropathy, psoriatic arthritis, and undifferentiated arthritis. Some inflammatory diseases affect the adult spine more often but may have early presentation in the pediatric population and include RA, ankylosing spondylitis (AS), and the other seronegative spondyloarthropathies listed in Table 18.2 [10].

Juvenile Spondyloarthritis

Juvenile spondyloarthritis (JSpA) usually manifests as peripheral enthesitis and arthritis. Of patients with spondyloarthritis (SpA), 10–20% have symptoms that begin in childhood and JSpA accounts for 15–20% of the arthritis in children. JSpA rarely has axial involvement at presentation but it can manifest in later stages of the disease process. Diagnostic criteria and definitions of the types of JSpA are evolving as new science reveals the subtle differences between the various

Table 18.2 Forms of spondyloarthritis recognized in adults [10]

1. *Ankylosing spondylitis*
a. Defined according to modified New York criteria: definite disease requires radiographic sacroiliitis, either bilateral grade II–IV or unilateral grade III–IV, plus inflammatory back pain, limitation of lumbar motion, or decreased chest expansion
b. No lower age limit defined
2. *Undifferentiated spondyloarthritis*
a. Defined by ESSG13 or Amor criteria, which factor in (but do not require) signs and symptoms of axial arthritis together with other features SpA, including gastrointestinal symptoms, psoriasis, and manifestations of psoriatic arthritis; MRI is not used in either classification system
b. ESSG and Amor criteria are usually used in adults, but no lower age limits have been defined and these criteria have been applied to children
c. "Axial spondyloarthritis" might now be considered a form of undifferentiated SpA
3. *Reactive arthritis*
a. Peripheral arthritis with onset typically within 6 weeks after certain infections of the gastrointestinal or genitourinary tracts
b. Classical extra-articular manifestations include rash (keratoderma blennorrhagicum and circinate balanitis), oral ulcers, conjunctivitis, enthesitis, and dactylitis
4. *Arthritis associated with IBD*
a. Coexisting arthritis and IBD, typically Crohn disease or ulcerative colitis; also referred to as enteropathic arthropathy
b. Arthritis is most commonly peripheral
c. Most frequently classified as SpA when sacroiliitis or spondylitis is present
5. *Psoriatic arthritis*
a. Coexisting arthritis and psoriasis, most often in the absence of rheumatoid factor
b. Frequently associated with nail pitting or onycholysis, dactylitis, and enthesitis
c. Like IBD-related arthritis, most frequently considered a form of SpA when sacroiliitis or spondylitis is present

ESSG European Spondyloarthropathy Study Group, *IBD* inflammatory bowel disease, *SpA* spondyloarthritis

forms of arthritis and enthesitis; there are several classification schemes. Currently the most widely recognized system divides the syndromes into differentiated (seronegative enthesopathy and arthropathy syndrome, enthesitis-related arthritis) and undifferentiated (juvenile ankylosing spondylitis, psoriatic arthritis, reactive arthritis, arthritis associated with inflammatory bowel disease (IBD)) forms.

Spinal manifestations can develop 5–10 years after onset of juvenile ankylosing spondylitis but are rare in the initial presentation. Most forms of JSpA are treated initially with nonsteroidal anti-inflammatory drugs (NSAIDs), corticosteroid injections, and short courses of oral or intravenous corticosteroids for symptom management. The disease can achieve remission with NSAIDs alone. Treatment with disease-modifying anti-inflammatory drugs (DMARDs) is also highly effective. Sulfasalazine is preferred over methotrexate. Biologic agents are recommended for patients with axial involvement or when standard therapies have failed. Midterm results regarding the efficacy of biological agents (6–8-year follow-up) are promising with high remission rates and minimal or no complications.

In 2011, the American Council on Rheumatology published treatment guidelines for juvenile idiopathic arthritis (JIA) that can be applied to JSpA. Overall, the disease course and outcomes are variable for JSpA with overall remission between 17 and 44 % and many patients progress to development of AS. These results are skewed by the fact that biologic agents have not been a part of long-term treatment strategies until recently.

Rheumatoid Disease of the Cervical Spine

RA is a particularly common inflammatory disorder affecting young adults. The related pediatric autoimmune disorder was previously known as juvenile rheumatoid arthritis (JRA) but is now

known as JIA. JIA is a T-cell-mediated autoimmune disease that manifests as a symmetric polyarthropathy affecting the joints of the axial and appendicular skeleton at the hands, wrists, feet, ankles, elbows, shoulders, hips, knees, and spine. Despite extensive study, the pathogenesis of JIA is incompletely understood but likely shares similarities with RA.

In the USA, 1 % of persons have RA, with females affected approximately three times as often as males. Spinal involvement is less frequent than is seen in AS and primarily involves the cervical spine, with clinically symptomatic disease affecting 40–80 % of patients [7]. Cervical spine disease ranges in severity from referred pain and radiculopathy to frank nonambulatory myelopathy, brainstem compression, and even sudden death.

RA-mediated inflammation is primarily manifested in synovial joints. Clinically important serological markers of disease include rheumatoid factor (RF) and anti-cyclic citrullinated peptide (anti-CPP) antibodies. RF-positive serology is 69 % sensitive and 85 % specific for RA making it a key part of the diagnostic workup. Anti-CCP-positive serology is 67 % sensitive and 95 % specific for RA. In RA, cytokines tumor necrosis factor (TNF)-alpha and interleukin-1, which sustain the inflammatory cascade, have surfaced as targets of most biologic immune-modulating drugs as shown in Table 18.3. Inflammation leads to the synovial cell production of metalloproteinases, which destroy articular cartilage. Synovial cell and lymphocyte-driven differentiation of osteoclast precursor cells leads to bony erosion by active osteoclasts [21]. The long-term effect of inflammation is widespread osteoarticular damage.

Among the most serious musculoskeletal sequelae of RA is cervical spine instability, particularly of the exclusively synovial articulation of C1–C2. RA-associated inflammation leads to articular cartilage destruction, capsular and ligamentous laxity, and bony erosion. Cervical spine involvement is prevalent in the juvenile RA population, in particular in patients with polyarticular involvement [25]. Juvenile patients will exhibit early spine involvement which may not be detectable on plain radiographs but is evident on MRI [42]. The C1–C2 articulation is stabilized primarily by capsular and ligamentous structures, as the facet surfaces lie in the axial plane and lend minimal AP stability from their bony articulation. For the same reason, the atlanto-occipital articulation is subject to instability. Furthermore, subluxation can be seen in the subaxial cervical spine, which is a source of pain and neurologic compromise. The resulting deformities in the cervical spine are: (1) subluxation of C1 on C2, usually anteriorly; (2) pseudobasilar invagination, which involves encroachment of the dens into the foramen magnum with impingement on the brainstem caused by erosion at the atlanto-occipital and/or atlantoaxial articulations; and (3) subaxial subluxation caused by facet erosion and capsuloligamentous laxity, which can involve multiple levels [39].

Symptoms of cervical instability are occipital headache, sensation of movement of the head on the neck with changing head position, and neurological symptoms such as syncope, myelopathy, Lhermite sign, weakness, vertigo, dysphagia, and cranial nerve involvement [7]. Neurologic abnormalities are present in one-tenth to one-third of patients with RA [7, 39]. The Ranawat classification, shown in Table 18.4 [39], is frequently used to describe the severity of cervical myelopathy in RA and is used to measure improvement after surgical intervention.

Radiographic evaluation is indicated in any patient with RA and neck pain or neurological symptoms. Radiographic abnormalities are present in 34–86 % of patients with RA; this rate differs between reported cohorts secondary to differing study populations and radiographic techniques and criteria used. Radiographs should be adequate to evaluate the standardized radiographic criteria shown in Table 18.5 [5], and should include a PA view, a lateral series with flexion and extension views, and an open-mouth odontoid view of the cervical spine [39]. Standard radiographic measurements include the anterior atlanto-dens interval (AADI), posterior atlanto-dens interval (PADI, Fig. 18.6a), the McGregor line, and the Ranawat (Fig. 18.6b) and Redlund-Johnell (Fig. 18.6c) measurements.

Table 18.3 Drug therapy for inflammatory spinal disorders [7]

Drug (chemical class)	Trade name	Pill size (mg)	Maximum dose (mg/day or week)	Frequency (per day)
Salicylates				
Aspirin	Bayer	81, 325	5,200	4–6
	Ecotrin	325	5,200	
Substituted salicylates				
Diflunisal	Dolobid	250, 500	1,500	2
Propionic acid				
Ibuprofen	Motrin	200, 400, 500, 800	4,500	4–6
Naproxen	Naprosyn	220, 375, 500	1,500	2–3
Flurbiprofen	Ansaid	50, 100	300	2–3
Ketoprofen	Orudis	25, 50, 75, 200	300	1–4
Pyrole acetic acid				
Sulindac	Clinoril	150, 200	450	2–3
Indomethacin	Indocin	25, 50, 75SR	225	2–3
Benzeneacetic acid				
Diclofenac	Voltaren	25, 50, 75, 100SR	225	2–3
Diclofenac/Misoprostil	Arthrotec	50, 75, 200	225	2–3
Oxicam				
Piroxicam	Feldene	10, 20	20	1
Pyranocarboxylic acid				
Etodolac	Lodine	200, 300, 400XL, 500XL	1,600	2–4
Naphthylalkanone				
Nabumetone	Relafen	500, 750	2,000	2
COX-2 inhibitors				
Celecoxib	Celebrex	100, 200	400	2
Meloxicam	Mobic	7.5, 15	15	1–2
Disease-modifying antirheumatic drugs				
Hydroxychloroquine	Plaquenil	200	400	1–2
Sulfazalazine	Azulfidine	500	3,000	1–3
Penicillamine	Cupramine	125, 250	125–750	1–2
Leflunomide	Arava	10, 20, 100	20	1
Methotrexate	Rheumatrex	2.5, 5, 7.5	25	1/week
Azathioprine	Imuran	50	50–300	1
Etanercept	Enbril	25, 50	100	1/week
Adalimumab	Humira	40	40	2 weeks
Infliximab	Remicaide	3–10 mg/kg	10 mg/kg	8 weeks

An AADI of up to 2.5 mm in women, 3.0 mm in men, and 4.5 mm in children is considered normal. As pseudobasilar invagination and anterior subluxation are of chief concern with increasing atlantoaxial instability, the PADI, which is the distance from the posterior aspect of the odontoid process to the anterior-most aspect of the posterior arch of the axis, should also be measured. A study by Boden et al. demonstrated that a PADI < 14 mm had a sensitivity of 97 % in predicting neurologic deficit, and that 94 % of patients with a PADI > 14 mm were neurologically intact [6].

Addition of MRI is indicated in the presence of new or progressive neurologic compromise as radiographs do not appreciate synovial pannus, which can exert additional mass effect on the brain stem and spinal cord. An MRI of the C-spine should be obtained for patients presenting with myelopathy, pseudobasilar invagination,

Table 18.4 Ranawat classification of neurological deficit [39]

Classification	Clinical criteria
Class I	Pain without neurologic deficit
Class II	Subjective weakness, hyperflexia, dysesthesias
Class III	Objective weakness, long tract signs
Class IIIA	Class III, ambulatory
Class IIIB	Class III, nonambulatory

Table 18.5 Roentgenologic criteria for rheumatoid arthritis of the cervical spine [5]

1. Atlantoaxial subluxation of 2.5 mm or more
2. Multiple subluxations of C2–C3, C3–C4, C4–C5, C5–C6
3. Narrow disc spaces with little or no osteophytosis
a. Pathognomonic at C2–C3 and C3–C4 b. Probable at C4–C5 and C5–C6
4. Erosions of vertebrae, especially vertebral plates
5. Odontoid, small, pointed; eroded loss of cortex
6. Basilar impression (odontoid above McGregor's line)
7. Apophyseal joint erosion; blurred facets; narrow spaces
8. Osteoporosis, generalized

or instability. MRI can also evaluate for myelomalacia and better characterize active lesions. The midsagittal cervical MRI slice can be used to measure the cervicomedullary angle, which is normally 135–175°, but can be less than 135° in cases of instability or pseudobasilar invagination. The presence of hand deformity correlates closely with cervical involvement in RA. In cases where instability is suspected, radiographic evaluation should be completed with CT of the cervical spine, which is much more sensitive in characterizing bony erosions and quantifying subluxation at the atlantoaxial junctions. Although CT scan is more sensitive and specific for determining the AADI, it is not able to demonstrate dynamic, reducible instability and is not routinely indicated when high-quality radiographs are within normal limits in a neurointact patient.

The goals of management when RA affects the cervical spine are to avoid the development of an irreversible neurologic deficit or sudden death from undermanaged or unrecognized cervical instability [39]. If significant basilar invagination or cervical instability is documented or suspected in a young athlete, the athlete should be restricted from sports until evaluated by a spine surgeon. Nonsurgical management of RA is targeted to interrupt the inflammatory cascade driving joint destruction and bony erosions with the goal of permanent disease remission. Table 18.3 lists the medications commonly used to treat RA in the USA. Providers should be cognizant of bone fragility in RA patients, which results both from the disease process and from its treatments [38]. Various treatments including calcium and vitamin D supplementation and bisphosphonates therapy are efficacious in reducing bone loss in RA [38, 53]. Resistance training for patients with RA improves muscle strength and physical function; however, improvements in bone density have not been as evident [22].

Surgical indications for cervical spine disease in RA are controversial in children and adults, especially with asymptomatic instability. Strong indications are progressive myelopathy, severe pain refractory to nonoperative management, and symptomatic instability at the upper or lower cervical spine. Given instability and impaired fusion biology, surgical stabilization of C1–2 should be instrumented and can be performed posteriorly by Gallie or Brooks wiring, screw and rod constructs, and by various plating options when fusion is extended to the occiput. With significant proximal migration of the dens, extension of fusion to the occiput should be considered [39].

Many athletes of various ages and skill levels are impaired by RA. In the pediatric population, pain after athletic activities may be the first presenting symptom of JIA [30]. The goals of care are to control the disease progression before cervical instability severely limits activities from pain and the threat of neurological injury. Athletics, in particular resistance training, may have benefits for maintaining strength and function and should be encouraged within safe limits. In patients with cervical spine instability, activities should be modified to minimize fall risk, and contact sports are contraindicated [52]. Activity limitations after surgical stabilization should be discussed with the operating spinal surgeon.

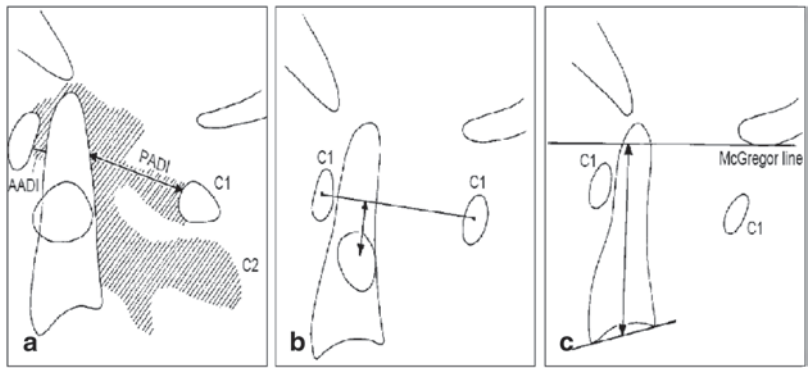

Fig. 18.6 Depiction of measurements for atlantoaxial instability. **a** Measurement of anterior atlanto-dens interval (AADI) and posterior atlanto-dens interval (PADI). **b** The Ranawat method for measuring vertical settling. **c** The Redlund-Johnell method for measuring vertical settling. (© 1997 American Academy of Orthopaedic Surgeons. Reprinted from [39], used with permission)

Table 18.6 Modified New York diagnostic criteria for ankylosing spondylitis [54]

Clinical criteria	Radiographic criteria
Lower back pain and stiffness for > 3 months that improves with exercise but is not relieved with rest	Bilateral sacroiliitis > Grade 2
Limited lumbar spine motion in frontal and sagittal planes	Unilateral sacroiliitis > Grade 3
Limitation of chest expansion	

Definite ankylosing spondylitis: one clinical criterion plus one radiographic criterion
Probable ankylosing spondylitis: three clinical criteria and no radiologic criteria or one radiologic criterion and no clinical criteria

Ankylosing Spondylitis

Although it generally presents in adulthood, AS can manifest in juveniles. Juvenile AS is defined as a diagnosis prior to age 16; however, given that the average time of delay from the first presenting symptom to diagnosis is between 4 and 9 years, the true incidence of juvenile onset AS is unknown [11]. AS is the prototype of a group of autoimmune diseases called the seronegative spondyloarthropathies and can cause significant functional disability through its effects on the sacroiliac joints and axial skeleton [7]. AS is defined by the constellation of symptoms, signs, and radiographic findings, which meet the modified New York criteria [54] as demonstrated in Table 18.6. Definite disease involves sacroiliitis of grade II–IV bilaterally or grade III–IV unilaterally in the presence of inflammatory back pain (Table 18.7) as well as either limitation of lumbar motion or decreased chest expansion. Harper et al. offer a diagnostic approach to the evaluation of inflammatory back pain in Fig. 18.7 [23]. The biological hallmarks of AS are its nonassociation with RF (an antibody to the Fc portion of one's own circulating immunoglobulinG (IgG) antibodies) and its association with human leukocyte antigen (HLA) B27. HLA B27 is a major histocompatibility complex (MHC) class I molecule involved in antigen presentation, expressed by more than 90% of affected patients [49], far more prevalent than the 8% of non-affected persons carrying the gene [7].

Table 18.7 Characteristics of inflammatory back pain [23]

Age of onset < 35 years
Insidious onset
Morning stiffness lasting longer than 30 min
Improvement with exercise but not with rest
Alternating buttock pain
Awakens in second half of night

Despite its strong association with HLA B27, less than 5% of patients with this gene will de-

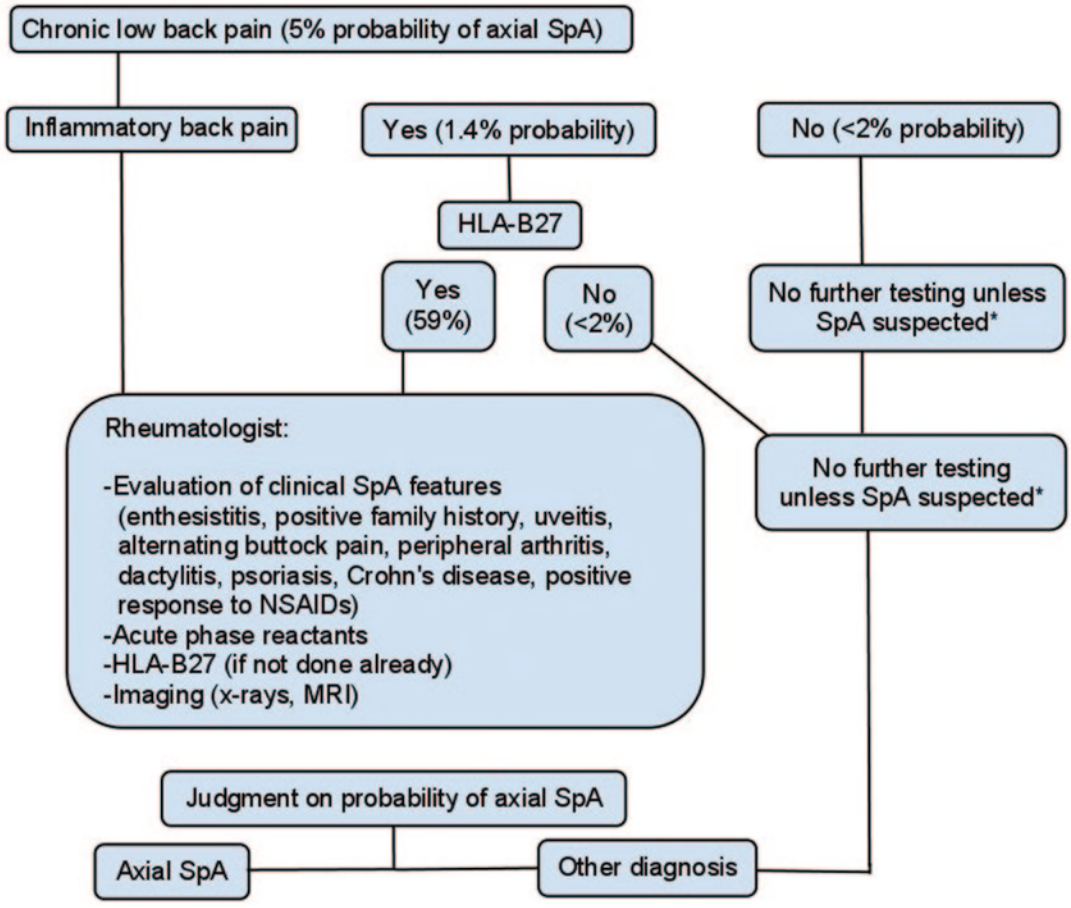

Fig. 18.7 Axial spondyloarthritis (SpA) determination. A diagnostic approach to the patient with low back pain. (© 2009 LWW. Reprinted from [23], used with permission)

velop symptomatic AS. Incidence differs among the various subtypes of the B27 allele [49]. It is unclear what events might trigger autoimmunity in AS; however, theories about a traumatic unveiling of previously immunoisolated antigens and various infectious triggers, including *Klebsiella*, have circulated [15]. After the inciting event, inflammatory lesions appear at entheses throughout the axial skeleton followed by new bone formation at sites of prior inflammatory lesions, thus the characteristic autofusion of sacroiliac joints and the spinal motion segments with characteristic marginal syndesmophytes.

Although the exact prevalence of AS is not known owing to misdiagnoses and its often milder course in women, it is thought to affect approximately 1 % of Caucasians, with a 3:1 male-to-female distribution [7]. At presentation, 90–95 % of patients have intermittent inflammatory pain in the lumbar spine, described in Table 18.7. Juvenile AS more commonly involves the peripheral joints compared to adult AS; however, the axial skeleton may be involved in juveniles as well.

As with adult AS, patients with juvenile AS often have a precipitating trauma or period of heavy activity just prior to initial symptoms; therefore, pain may be confused with athletic injury and diagnosis may be further delayed [34]. Physical examination reveals pain with percussion of the sacroiliac joints and a positive flexion, abduction, and external rotation (FABER) or Gaenslen test. Spinal sagittal alignment shows varying degrees of lumbar hypolordosis, thoracic kyphosis, and cervical hypolordosis. Reduced

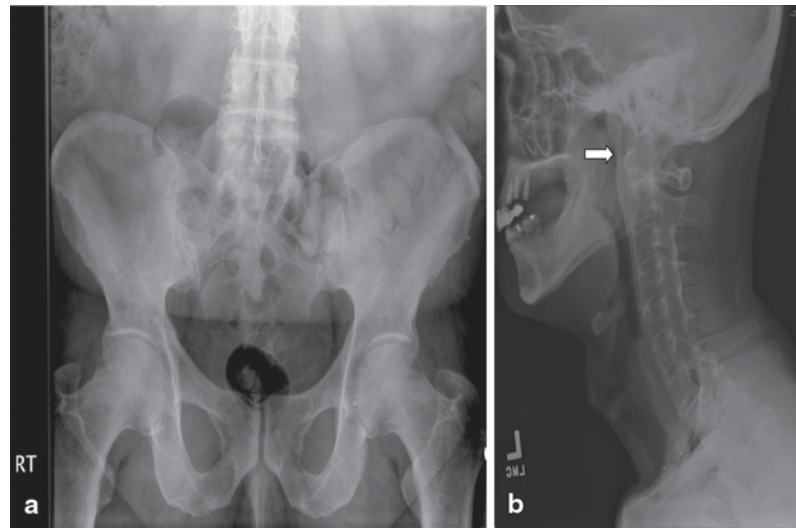

Fig. 18.8 **a** XR bilateral sacroiliitis. Bilateral grade III sacroiliitis with sclerosis, erosions, and joint space narrowing of bilateral SI joints. **b** XR cervical ankylosis. Fusion of C2–C7 with fracture of dens (*arrow*). (© 2009 Lippincott, Williams and Wilkins. Reprinted from [23], used with permission)

motion in the lumbar spine is demonstrated on Schober's test with limited separation of lumbar surface landmarks with forward flexion. As the disease also affects the costovertebral joints of the thoracic spine, reduced chest expansion with inspiration is a common finding. Laboratory tests reveal elevated ESR in 80% of AS patients and CRP elevation is frequent, with a median highly sensitive CRP level of 4.8 mg/L in one cross-sectional study [43]. RF and antinuclear antibody are typically negative.

Radiographic changes in AS include symmetric sclerotic sacroiliitis (Fig. 18.8a) in more than 85% of patients with predominant lumbar involvement in the first 20 years of the disease course, after which lumbar and cervical involvement are equal (Fig. 18.8b) [31]. Complete spine involvement is seen in 28% of patients 30–40 years into the disease process and in 43% of patients after their 40th year with AS [29]. Ossification begins at the vertebral rim and extends across the annulus fibrosus in line with and perpendicular to the spinal axis. Vertebral corner inflammatory lesions (CILs) are observed as hyperintensities at the anterosuperior and anteroinferior corners of vertebral bodies on sagittal T2 and Short Tau Inversion Recovery (STIR) MRI sequences as demonstrated in Fig. 18.9a, b. CILs signify active disease, as there is a four- to fivefold increased probability of new syndesmophyte formation at the site of these lesions within 24 months of their appearance on MRI [29]. The term Bamboo spine describes the appearance of the vertically oriented syndesmophytes, calcified annulus fibrosus, and ossified anterior and posterior longitudinal ligaments on lateral radiographs.

AS causes progressive autofusion of the spine, which begins in the sacroiliac joints and lumbar spine and typically progresses proximally. The costovertebral joints are also affected, as shown in the MRI in Fig. 18.9b, leading to a progressive extrinsic restrictive lung disorder is some patients. A rigid thoracic kyphosis in conjunction with lumbar and cervical hypolordosis leads to the characteristic chin-in-chest deformity, which is painful, functionally limiting and only partially compensated by obligate knee flexion and hip hyperextension.

Surgical management of AS is required in cases of fracture, fracture-associated epidural hematoma, neurological compromise, and most commonly painful or function-limiting deformity. Spinal fracture from minor trauma and low-energy falls can occur secondary to the long inflexible lever-arm of fused spinal segments. Additional risk of vertebral fracture may be attributable to fragility that results from chronic inflammation, abnormal biomechanics, and drug-related osteopenia. Trauma in the athlete with AS

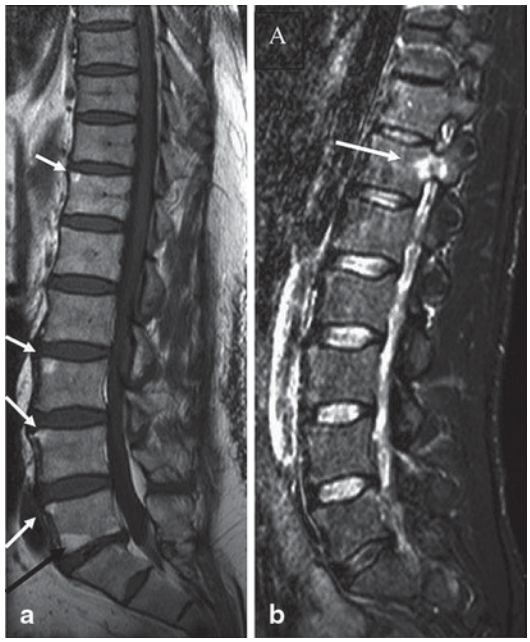

Fig. 18.9 **a** Multiple FRLs, corner inflammatory lesions. Example of multiple corner inflammatory lesions (*white arrows*) with one end-plate lesion (*black arrow*). **b** Axial Spondyloarthropathy, posterior element T12 lesion. Inflammatory posterior element lesions on sagittal STIR MRI sequence, depicting posterior element/costovertebral inflammatory lesion at T12. (© 2010 Oxford University Press. Reprinted from [4], used with permission)

Table 18.8 Extra-articular features of ankylosing spondylitis (AS) [49]

Organ system	Manifestations
Ophthalmic	AAU
Cardiovascular	Aortitis
	Aortic valve insufficiency
	Thickening of aortic valve leaflets
	Subaortic fibrosis
	Conduction abnormalities
	Mitral valve insufficiency
	Left ventricular dysfunction
Gastrointestinal	Inflammatory bowel disease
Neurological	Myelopathy and radiculopathy
	Cauda equina syndrome
	Vertebrobasiar insufficiency
	Peripheral neuropathy
Pulmonary	Interstitial fibrosis
	Restrictive thoracopathy
Renal	Amyloidosis
	Immunoglobulin A nephropathy

AAU acute anterior uveitis

and acute or delayed neck or back pain deserves close observation as the diagnosis of a potentially neurologically devastating epidural hematoma associated with even radiographically subtle fractures can be delayed by days or weeks. MRI evaluation of the spine is imperative in all suspected spinal fractures in patients with AS, as X-rays are notoriously insensitive for subtle fractures particularly in the lower cervical spine, the most frequently involved region. Special attention to the stabilization and transport of an AS patient with a C-spine injury should focus on maintaining the individual's native neck alignment which may be more flexed than a healthy counterpart [37]; acknowledging this factor has been shown to reverse an acutely progressing neurological deficit. There is high in-hospital morbidity and mortality in AS patients with C-spine injuries likely owing to preexisting restrictive pulmonary disease and AS-associated cardiopulmonary disease, as listed in Table 18.8 [7]. Compared to RA, few AS patients experience atlantoaxial subluxation (21 % in a recent large series, and of these patients, just 23 % required surgical stabilization [45]).

The mainstays of nonoperative treatment of AS include medications to target pain and inflammation in addition to physical therapy to combat impending deformity during autofusion. Medications are categorized into NSAIDs, DMARDs, and biologic immune-modulating drugs designed to block specific targets in the immune cascade (Table 18.3) [7]. With regard to physical activity in patients with AS, the orthopedist should be cognizant of the documented benefits of exercise therapy and aerobic exercise balanced by the increased risk of low-energy spinal fracture and possible cardiac disease associated with AS (cardiac disease risks are listed in Table 18.8) [49]. Physical therapy and ergonomics include extension-directed spine exercises, as listed in Table 18.9 [23], deep breathing exercises, and avoidance of spine flexion with pillow positioning [16]. Swimming and water therapy are cost-effective adjuncts to medical treatment and standard physical therapy [16, 55]. Corset style braces should be avoided as their efficacy is un-

Table 18.9 Exercises for spondyloarthritis [23]

Chest expansion	Single knee to chest	Leg slides out
Chin tuck	Double knee to chest	Leg rotation
Neck rotation	Trunk rotation	Back extension
Neck side bending	Shoulder stretch	Trunk side bending
Hamstring stretch	Shoulder blade stretch	"Cat and camel"
Pelvic tilt	Bridging	Kneeling stretch
Hip flexor stretch	Trunk side bending	Trunk rotation
Calf stretch	Wall calf stretch	Corner stretch

proven and potential adverse effects on respiratory capacity may be detrimental.

Biologic treatments for AS held hope for prolonged disease remission; however, early trials have somewhat tempered enthusiasm for these drugs. AS manifests in the appendicular skeleton as an asymmetric, large joint, pauciarticular arthritis in 30% of patients. Biological therapies have been effective in halting the progression of peripheral arthritis and although they aid in providing symptomatic relief of back pain, they are not completely effective in preventing spinal ankylosis [7].

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Spine Tumors in the Young Athlete

19

Megan E. Anderson

Abbreviations

MRI	Magnetic resonance imaging
CT	Computed tomography
NSAIDs	Non-steroidal anti-inflammatory drugs
SPECT	Single-photon emission computed tomography
RFA	Radiofrequency ablation
ABC	Aneurysmal bone cyst
RANK	Receptor activator of nuclear factor kappa B

Introduction

Spine tumors are infrequently encountered in the young athlete population. However, when they are not identified in a timely manner, the consequences can be significant: spinal deformity, neurologic deficits, long-standing pain, etc. It is thus important for all clinicians caring for athletes, to keep neoplasia in their differential when they are evaluating an athlete with a spine complaint.

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In this chapter, clues in presenting symptoms and appropriate imaging will be discussed. Specific tumor types that are most commonly encountered in this population and their treatment will also be included. This is not intended as an exhaustive review of spine tumors in general, but focused on those that are more commonly identified in the young athlete.

Presentation

Most athletes with a spine tumor present similarly to those with overuse syndromes or traumatic injuries—with pain. Pain that persists beyond the normal time frame for an injury or overuse problem, or that is of a much more severe intensity should prompt further investigation [14]. Pain associated with signs of nerve root compression, myelopathy, or spinal deformity should trigger a more urgent evaluation. Pain that is worse at rest or that wakes a patient from sleep can be a worrisome feature as well.

Athletes may also present with a tumor as an incidental finding when they are being evaluated after trauma or overuse. Many of these lesions are benign, but consultation with a clinical spine tumor specialist and/or radiologist should be considered so that the appropriate workup and biopsy, if necessary, can be performed.

Physical exam should be directed to the area of concern (upper extremities for cervical spine, for example), checking for radicular nerve root or cord compression findings. The athlete should be

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evaluated for spinal deformity as well. An acute curve towards the side of pain may be a spastic scoliosis due to inflammation from a tumor such as an osteoid osteoma. An acute kyphosis may indicate vertebral body collapse secondary to involvement of the vertebral body with tumor or Langerhans cell histiocytosis (LCH). Any of these findings warrant further evaluation with imaging studies.

Imaging

Plain radiographs are rarely diagnostic in the evaluation of a patient with a spine tumor. They may indicate clues, though, that should prompt further evaluation. Spinal deformity would be evident on full spine X-rays. The absence of the bony landmark of the pedicle, the "winking owl sign," may indicate that a pedicle is involved with the tumor. Either of these findings would then necessitate further three-dimensional imaging studies.

Magnetic resonance imaging (MRI) is extremely helpful in evaluating a patient with a spine tumor. The normal anatomy of the neighboring cord, cauda equina, and/or exiting nerve roots is demonstrated, along with their relation and proximity to the tumor. Soft-tissue tumors, both intra- and extra-dural, are best delineated with this modality. Bone tumors are also nicely assessed with MRI where internal characteristics of certain tumors can be identified (these will be discussed with specific tumor types below). Computed tomography (CT) is better at evaluating the intraosseous extent and fracture risk associated with a spine tumor with bony involvement. CT can also identify internal osteoid, bone, or cartilage matrix formation within a tumor [33].

Bone scan is useful when multiple lesions are suspected, to evaluate the whole skeleton for involvement, or to localize the site of origin of an athlete with concerning spine pain of a more diffuse nature. Otherwise, bone scan typically does not add to what may be gleaned from MRI and CT. It is however, part of the staging process for a malignant primary tumor of bone.

Specific Tumors and Tumor-Like Conditions

Enostosis

Also known as a bone island, enostoses are almost always incidental findings. They are more of a developmental abnormality than a true neoplasm, a result of incomplete bone resorption during bone maturation. They appear as dense areas of lamellar compact bone within cancellous bone and typically have a slightly brushed or spiculated border on CT. They demonstrate universally low signal on all MRI sequences and can have uptake on bone scan. Treatment is observation. A suspected enostosis that enlarges more than 25% in 6 months should be evaluated further [1].

Osteoid Osteoma

Osteoid osteomas are very small lesions (less than 1.5 cm) that incite a tremendous inflammatory response. They are located in the spine in only 10% of cases where most are lumbar, followed by cervical, thoracic, then sacral, in location, typically in the posterior elements [1]. Patients are usually less than 30 years of age and more likely to be male (2:1) [3]. Osteomas classically cause sharp pain that is worse at night, unrelated to activity, and better with nonsteroidal anti-inflammatory drugs (NSAIDs) or aspirin. The pain may be very long-standing, lasting from weeks to years. Painful scoliosis is a frequent association as noted above (Fig. 19.1a).

Osteoid osteomas are so small that they usually cannot be identified on plain radiographs when they are located in the spine. CT is the modality of choice [19], where the small radiolucent nidus is often surrounded by a significant amount of sclerotic reactive bone (Fig. 19.1b). If it is difficult to localize the lesion, bone scan can help identify the area of concern and then a CT can be performed of that area in detail. These lesions typically have a significant amount of uptake on a bone scan (Fig. 19.1c). Alternatively, single-photon emission computed tomography (SPECT) can be even more sensitive and specif-

ic, combining the two modalities together [13]. On MRI, the lesion is often obscured by the tremendous surrounding edema, but is usually low to intermediate in signal on T1 and variable on T2 (Fig. 19.1d).

Treatment ranges from long-term treatment with NSAIDs to surgical resection. The symptoms do tend to "burn out" over time, but this may take several years and the side effects of chronic NSAID use lead many to seek surgical intervention. Observation is often considered for osteoid osteomas in locations that are difficult to access, such as the spine. A small recent series noted that NSAID use may actually accelerate spontaneous healing of osteoid osteomas [16]. The painful scoliosis often resolves as the pain does, but may persist if the symptoms continue for over 15 months [15]. Radiofrequency ablation (RFA) is an appealing technique as it is minimally invasive with little recovery time and good success. In this procedure, a small probe is inserted through a cannula placed with imaging guidance into the lesion. This probe emits radiofrequency waves that cause thermal necrosis of the tumor, thereby eradicating the symptoms. RFA must be used with caution in the spine due to the proximity of neural elements which may suffer thermal injury in the process, but several series have pointed to the safety and efficacy of using this technique for spine locations [24, 29, 34, 41]. Surgical excision usually provides immediate pain relief, but must be balanced against the risks of the surgical approach, the structural elements destabilized, and the need for and type of reconstruction [10]. Intraoperative bone scan can assist in localizing and thus performing a complete resection in these cases (Fig. 19.1e) [6].

Osteoblastoma

Osteoblastomas are very similar to osteoid osteomas pathologically, but often differ in their presentation. While patients frequently complain of long-standing pain, they often describe it more as a dull pain, which is typically not relieved by NSAIDs. They are less often associated with scoliosis, but patients with osteoblastoma are more likely to have symptoms of nerve root or cord compression than those with osteoid osteoma [3]. Osteoblastomas involve the spine 30–40% of the time and they can be locally aggressive [1, 3, 33]. On imaging, they are larger than 2 cm and can be purely radiolucent or demonstrate internal osteoid matrix. They typically demonstrate significant uptake on bone scan. The CT shows a geographic lesion that may expand and thin the cortex significantly. Most are either in the posterior elements or in the posterior elements with extension into the body. MRI shows a lesion that is hypointense on T1 and hyperintense on T2 sequences other than the mineralization which is hypointense universally. They enhance after injection of contrast material. They often have a marked surrounding inflammatory response, termed the flare phenomenon, which may lead to overestimation of the size of the lesion and confusion with the appearance of osteosarcoma [3, 33]. Needle biopsy in advance of open surgical approaches can be indicated in these cases, but pathologic distinction between osteoblastoma and low-grade osteosarcoma is challenging and consultation with a subspecialist in bone pathology may be necessary [3].

The preferred treatment is complete resection but often curettage and bone grafting is the only possibility due to the risk of significant neurologic injury associated with the former [5]. Recurrence rates are high, ranging from 10 to 20%, and higher with intralesional procedures [5, 15]. Second surgical procedures for recurrence can be quite difficult in the spine, leading some centers to utilize postoperative radiation in cases where en bloc excision could not be performed [8]. Radiation in children, however, must be carefully balanced against the risk of secondary malignancy and growth disturbance.

Aneurysmal Bone Cyst

Aneurysmal bone cysts (ABCs) involve the spine in 3–20% of cases with the majority in the posterior elements, some expanding into the vertebral body [1, 33]. They may be primary tumors (70%) or secondary to another lesion (30%), such as osteoblastoma, giant cell tumor, and

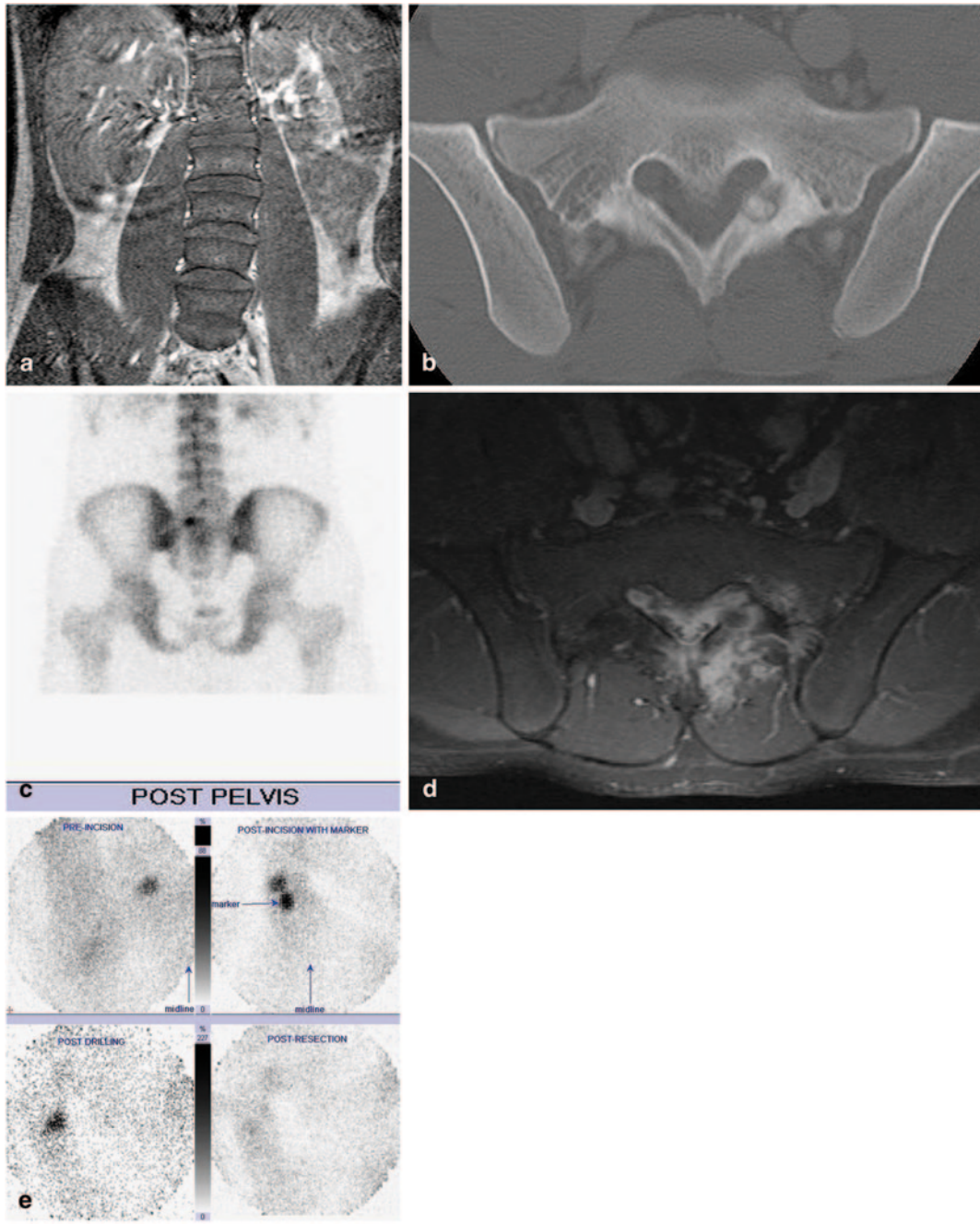

Fig. 19.1 Osteoid osteoma of the S1 lamina. **a** Inflammatory scoliosis noted on the coronal scout during the patient's MRI. **b** Axial CT image through the area of concern. Note the radiolucent nidus with central matrix mineralization surrounded by reactive sclerotic bone. **c** Bone scan demonstrating significant uptake in the S1 lamina recorded from a posterior projection. **d** Axial MRI image T1 with fat saturation after contrast administration demonstrating the significant inflammatory change around the lesion. **e** Bone scan images acquired during surgical resection of the S1 osteoid osteoma

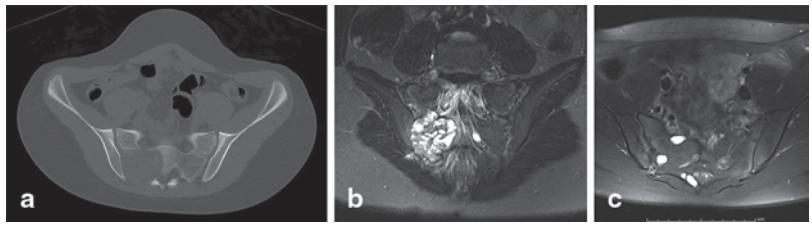

Fig. 19.2 Aneurysmal bone cyst of the right sacral ala. **a** Axial CT image demonstrating thin cortices and expanded appearance of the bone. **b** Coronal T2 MRI image through the lesion showing multiple fluid–fluid levels. **c** Appearance of the same sacral aneurysmal bone cyst 2 years after three sclerotherapy injection procedures on an axial MRI image T2 sequence with fat saturation

others [31]. Patients tend to be in the second decade of life and more commonly female than male [25]. These lesions are purely radiolucent on radiographs and CT and have a bubbly, expanded appearance with a very thin cortical shell (Fig. 19.2a). On MRI, fluid–fluid levels are common within large spaces in the lesion (Fig. 19.2b) [1, 33]. These are due to the blood in these spaces separating into solid and fluid components. While they are almost always present in ABCs, they can also be seen in other lesions, most concerning of which is telangiectatic osteosarcoma. Biopsy is, thus, frequently performed either prior to a surgical procedure or at the start of a procedure with frozen section to confirm the diagnosis before definitive treatment.

Treatment depends on the location of the lesion. In areas that can be approached and removed surgically, excision is the treatment of choice and is associated with a lower recurrence rate. Curettage and bone grafting is associated with a higher recurrence rate and may be associated with significant blood loss intraoperatively, prompting many surgeons to advise preoperative embolization. A surgical approach is the treatment recommendation for an ABC associated with fracture and/or neurologic involvement [15, 30, 44, 45]. Radiation is also utilized for more aggressive ABCs and/or recurrences with alone or as adjuvant therapy with surgical approaches [25]. Like osteoblastoma, though, radiation in children must be considered carefully for fear of radiation-induced malignancies and growth issues.

Recent series have pointed to success with multiple injection procedures for these lesions, especially in lesions that are difficult to approach, such as the spine. Selective embolization and/or sclerotherapy has been associated with good healing responses and low risks when performed by a subspecialized team (Fig. 19.2c) [11, 15, 22]. A recent study suggests embolization should be the initial approach for all spinal ABCs due to the optimal cost-to-benefit ratio, high rates of successful healing, and possibility for other treatments should they be necessary [2].

Hemangioma

Hemangiomas are the most common benign bone tumor in the spine and are frequently found incidentally. They can be solitary or multiple and most involve the vertebral body. When they are large and/or involve the pedicle, they can present a risk for fracture. This is particularly the case for transitional vertebral body levels [42]. On CT, the coarse striations of trabecular bone between the irregular vascular spaces are evident and give a honeycomb or polka dot appearance depending on whether they are imaged in line with the trabeculae or en face. On MRI, they have a high signal intensity on T1- and T2-weighted images due to the fatty tissue within them and fluid, respectively [33].

If hemangiomas are an incidental finding and are reasonably small, then no treatment is necessary. If they are large, growing in size, present risk for pathologic fracture, or have fractured, they usually need to be treated surgically with excision or curettage and reconstruction, the magnitude of which depends on the vertebral level

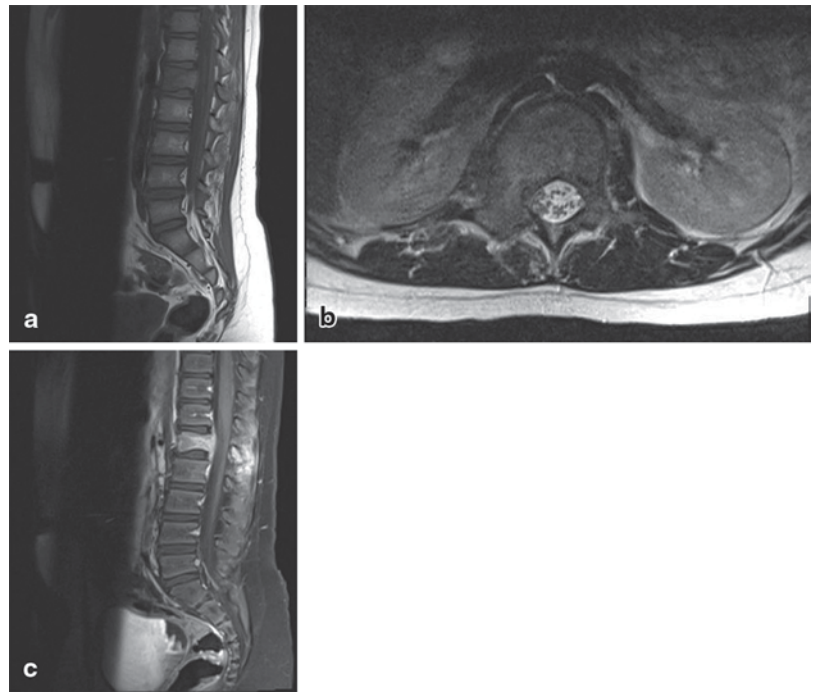

Fig. 19.3 MRI images of Langerhans cell histiocytosis involving the first lumbar vertebral body and pedicle. **a** There is subtle replacement of the fatty bone marrow signal and slight loss of height in the L1 vertebral body on this sagittal T1 sequence. **b** Involvement of the pedicle with permeative extension into the soft tissues and epidural space is evident on this axial T2 image. **c** The lesion is more apparent and appears aggressive on this sagittal T1 sequence with fat saturation after contrast

involved and the extent of involvement of the body [21, 37]. Rarely, some hemangiomas of the spine can be painful; minimally invasive methods of treatment, such as vertebroplasty, embolization/sclerotherapy, and radiation (typically in older adults only), can be quite effective and are less morbid than open surgical approaches [9, 17, 18, 27].

Langherhans Cell Histiocytosis

LCH is the abnormal proliferation of histiocytes from the reticuloendothelial system and is not a true neoplasia [4]. Bone involvement is common, and spine location is notable in 10–15% of cases [15]. It tends to affect a younger athletic population, in the first and second decades most commonly [37]. While vertebral collapse into vertebra plana is common, symptoms are usually fairly mild and neurologic symptoms are uncommon. Plain radiographs demonstrate a radiolucent lesion, most commonly in the body of the vertebra, and vertebra plana in about 40% [37]. Bone lesions may be solitary or multiple [20, 22] and so skeletal survey and bone scan are recommended at initial diagnosis; some lesions may be missed if only one of these modalities is utilized. MRI is helpful to identify any associated soft-tissue mass or worrisome features that may suggest something more aggressive such as Ewing sarcoma or lymphoma. If the classic appearance of vertebra plana with no soft-tissue mass is identified, these lesions can be followed [40]. Biopsy may injure the residual apophysis which is necessary to restore height in these vertebral bodies [15]. In earlier stages when the lesions are radiolucent with local destruction, biopsy is necessary to rule out more aggressive and malignant lesions (Fig. 19.3). Often, this is the only treatment necessary, with or without cortisone injection, as the bone lesion can heal in after biopsy only [32]. At first presentation with LCH, consultation with an oncologist is indicated to evaluate the child for multisystem involvement, in which case systemic therapy would likely be recommended [4].

Giant Cell Tumor of Bone

Giant cell tumor of bone involves the spine in roughly 10% of cases with the majority involving

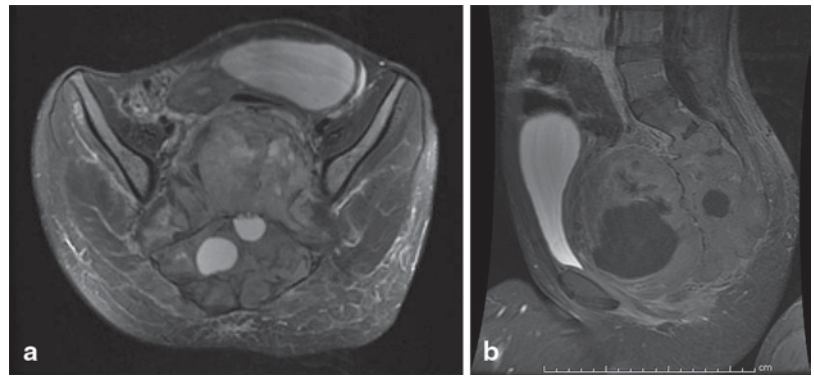

Fig. 19.4 MRI images of a very large giant cell tumor of the sacrum in a patient who presented with no pain, but obstipation and urinary frequency. **a** Axial T2-weighted image. **b** Sagittal T1-weighted image with fat saturation after contrast administration

the sacrum [1]. Patients, mostly young adults and adolescents, usually present with long-standing pain and occasional signs of nerve root impingement. Some tumors may reach a large size before diagnosis (Fig. 19.4). Imaging shows a radiolucent lesion with cortical thinning and expansion. They can cause enough local destruction to also cause pathologic fracture. MRI shows heterogeneous signal on all imaging sequences. Giant cell tumors can rarely metastasize to the lungs, with perhaps a slightly higher incidence for primary location in the spine [12]. Chest imaging with CT is reasonable in these cases, particularly in recurrent tumors. Giant cell tumor can also degenerate into a malignant giant cell tumor in 1–3 % of cases [38].

Treatment is based on the size and location of the lesion. If en bloc resection of the lesion is possible, it is preferred and is considered to be curative. Often, however, these lesions are difficult to approach and remove entirely due to their location and significant vascularity. Radiotherapy is an option with good tumor control, but with risks of secondary malignancy and infertility particularly in women with sacral giant cell tumors [38]. Denosumab (Amgen, Thousand Oaks, CA), the receptor activator of nuclear factor kappa B (RANK) ligand inhibitor, is currently under investigation as an effective agent for unresectable and metastatic giant cell tumor of bone [39]. This may prove to be a monumental swing in the treatment of patients with giant cell tumors of the spine. Further investigation is necessary as many questions about this therapy remain unanswered, in particular its effect on the growing skeleton, long-term effects, and length of therapy [38].

Malignant Tumors

Primary malignant tumors of the spine are rare, but certainly can present in the young athlete patient population. A high index of suspicion is necessary to diagnose these conditions, though, as most patients present with nonspecific pain. The pain, however, tends to be deep and unresponsive to medications, present independent of activity, worse at night, even waking the patient from sleep, and/or persistent for longer than traumatic or overuse syndromes. Patients with Ewing sarcoma and lymphoma may also have systemic symptoms of fever, chills, malaise, and night sweats. This can make the presentation similar to a youngster with infection, so great care must be taken in the workup to elucidate the differences. In children, adolescents and young adults, the most common entities are osteosarcoma and Ewing sarcoma, but lymphoma and leukemia can also rarely present with spine involvement in this age group.

In addition to pain, many patients may have signs of neurologic compression due to extension of tumor out of the bone and/or involvement of the epidural space. In these situations, spinal cord compression requires emergency management, ideally by a multidisciplinary team with experience in spine tumor treatment. Decompression most often requires surgery, but can be achieved medically in tumors that are chemosensitive [43]. While steroids are frequently used in the acute setting of an athlete with suspected traumatic cord compression, they should not be used in the situation where a malignancy is suspected prior to diagnosis as some cases of leukemia or lymphoma

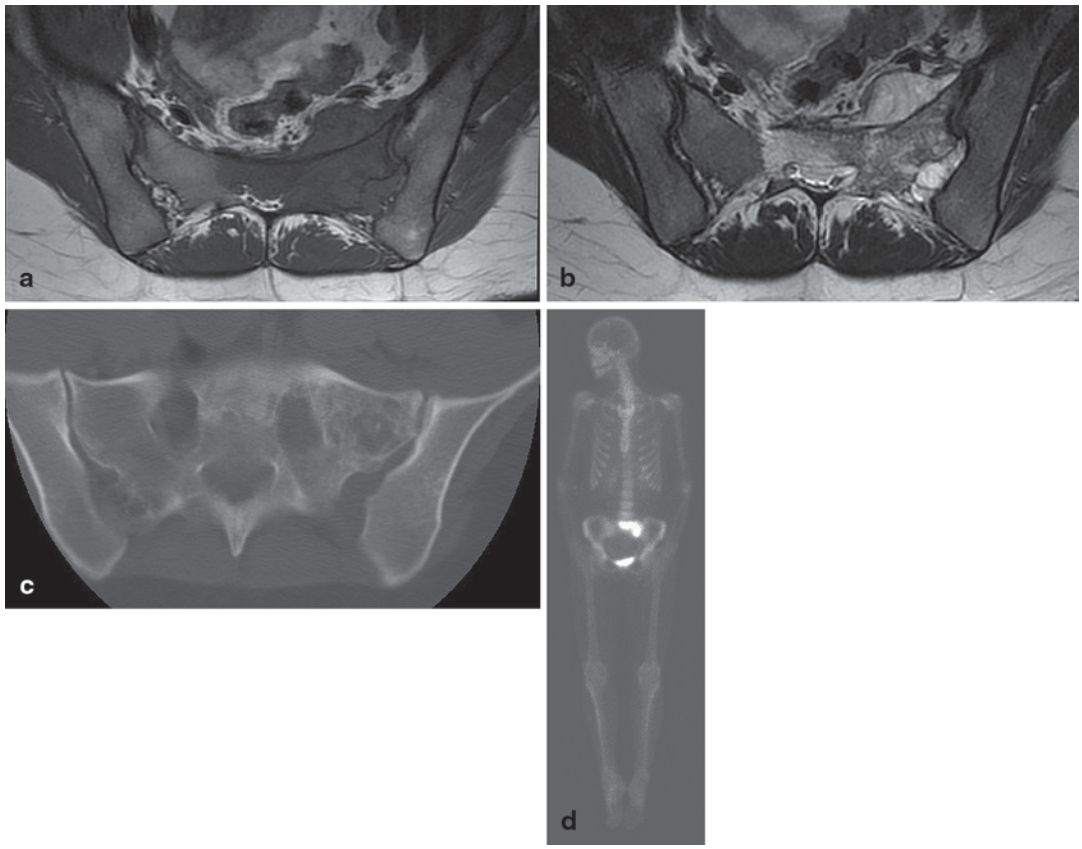

Fig. 19.5 High-grade osteosarcoma of the sacrum in a 25-year-old woman who presented with pain and radicular symptoms. The MRI images demonstrate a marrow-replacing process with extension of the T1 hypointense (**a**) T2 hyperintense (**b**) tumor into the soft tissues. The lesion has a permeative appearance on CT (**c**) with areas of bone destruction mixed with subtle areas of matrix mineralization. There is intense activity in the lesion on bone scan (**d**) with no sites of bony metastasis

may resolve entirely with steroid administration, making diagnosis and staging impossible. Similarly, biopsy of a primary spinal neoplasm with aggressive features must be approached carefully so as to not eliminate the opportunity for surgical resection, should that prove necessary. Again, it is best undertaken by a multidisciplinary team with experience in these tumors.

Osteosarcoma involves the spine in less than 5% of cases where it tends to arise in the vertebral body most commonly [23, 36]. Imaging demonstrates an aggressive tumor with destruction of normal bone and matrix mineralization within the tumor to a variable extent (Fig. 19.5). In cases where the tumor is very heavily mineralized, it may appear as an "ivory vertebra." Treatment involves chemotherapy and total spondylectomy or wide local excision, sometimes with the use of radiation as an adjuvant when total resection is not possible. Spine location carries a poor prognosis compared to nonspine and nonaxial osteosarcomas with a higher rate of metastasis at presentation [23, 36]. A few recent studies have shown survival benefit to en bloc excision over nonsurgical treatment [28] and intralesional excision [35]. However, newer techniques in radiation therapy, such as carbon ion radiotherapy, may have a role in unresectable cases [26].

Ewing sarcoma more commonly involves the spine in the setting of metastatic disease as opposed to presenting as a primary tumor (3–10% of all Ewing sarcomas [1, 23]). Boys are affected

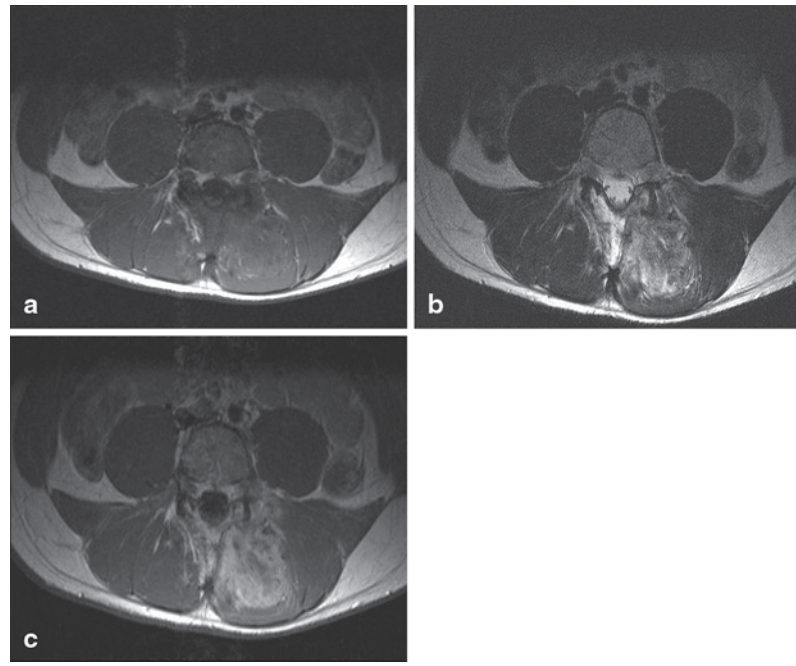

Fig. 19.6 Ewing sarcoma of the posterior elements of L4 in a 17-year-old male who presented with pain in his lower back radiating down his left leg. The MRI demonstrated a permeative and destructive mass of the posterior elements of L4 with extension into a large soft-tissue mass: **a** axial T1-weighted image, **b** axial T2-weighted image, **c** axial T1-weighted image with fat saturation after the administration of gadolinium

more often than girls and white populations more than African and Asian. It most commonly involves the bone (Fig. 19.6), but can also arise in the soft tissues adjacent to the spine. Soft-tissue masses from tumors that arise in bone primarily can be quite large (Fig. 19.6) and so neurologic impairment and cord compression are common. These tumors tend to be purely radiolucent in bone with enough bone destruction to cause vertebral body collapse and even vertebra plana in some cases. Most common locations within the spine are lumbar levels and sacrum [23]. Treatment involves chemotherapy systemically and most commonly radiation therapy for local control. Surgical resection, especially in the rare case the tumor can be removed en bloc, may provide more durable local control [7, 28]. Though slightly better than osteosarcoma of the spine, Ewing sarcoma of the spine carries a poorer prognosis [23].

Primary *lymphoma* of bone is rare and is most commonly a non-Hodgkin's diffuse large cell type. Occasionally the spine is the primary site, arising in the bone, epidural space, or retroperitoneal lymph nodes. Much like Ewing sarcoma, when it arises in bone, the soft-tissue component can be very large and can result in cord compression. Most lesions are radiolucent, but some can be mixed or sclerotic. Lymphoma can also be sclerotic enough to cause the "ivory vertebra" appearance; these cases tend to be Hodgkin's lymphoma [23]. Treatment frequently involves laminectomy and biopsy for decompression and diagnosis, followed by systemic chemotherapy. Radiation is used in some cases of spinal lymphoma to consolidate therapy, more so in adults than in children.

When *leukemia* involves the spine, it tends to be acute lymphoblastic leukemia [1]. Often, several vertebral bodies are involved, usually with radiolucent areas. Compression fractures can also be seen. The involved bone marrow has a lower signal on T1 and higher signal on T2 imaging by MRI. Patients may have systemic symptoms such as fatigue, weight loss, fever, and/or lymphadenopathy. Chemotherapy is the mainstay of treatment.

Soft-tissue masses in the tissues adjacent to the bony spine or within or near the spinal cord itself may also rarely present in the athlete. These are very rare and beyond the scope of this chapter.

Conclusion

Spine tumors in the young athlete are rare but should be included in the differential diagnosis of any spine patient with persistent or severe pain. Presenting symptoms are often nonspecific, so a high index of suspicion is needed to diagnose these lesions in a timely fashion. Early recognition and referral to an appropriate multidisciplinary spine tumor center can greatly improve prognosis for these challenging cases.

Disclosures None.

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Return to Play After Spinal Surgery

20

Robert G. Watkins, III and Robert G. Watkins, IV

Introduction

Successful return to sport after spinal surgery depends on several factors: accurate diagnosis, proper choice of surgery, effective surgery, and comprehensive rehabilitation program. The same exercise program is used post surgery as with post-injury nonoperative care.

When a young athlete presents for treatment, it is the obligation of the clinician to properly demonstrate the lesion to the patient and the parents and devise an appropriate treatment program for the patient's complaints. Making the correct diagnosis is the key to initiating an appropriately aggressive diagnostic and therapeutic plan (Table 20.1) [1, 2].

The chief indications for surgery in the athlete are the indications for surgery in any patient:

1. Sufficient morbidity to warrant surgery
2. Failure of conservative care
3. An anatomic lesion that can be corrected with a safe, effective operation
4. A proper, fully developed postoperative rehabilitation program

Not enough emphasis can be placed on a proper postoperative rehabilitation program. Failure to do postoperative spinal rehab would be similar to a failure to do postoperative knee strengthening after reconstruction of the knee, or a failure to do postoperative strengthening and range of motion exercises after surgery on a shoulder. The patient wants restoration of function. The surgeon must be able to guide the patient through the restoration of function.

Spinal operations to enhance performance, rather than to relieve disabling pain, are a part of managing the care of athletes, a part that requires a great deal of experience not only in spinal surgery but also in dealing with athletes. There are numerous factors to consider. One must always keep in mind the full longevity of the patient. Young players can take a year off after spinal surgery, and still potentially return to play. Older players are less likely to return to play after a major spinal reconstructive operation. What the player will be like after his/her career—condition of his/her spine at that time—should be of major importance in decision making early in the player's career. A major factor is calculating the risk if the operation is successful. In many sports, after a spinal fusion, for example, or a major resection of a supporting structure in a decompression, the percentage chance of return may be no greater after the operation than without the operation. Therefore, when considering surgery to enable a player to return to sport we must answer: (1) Are we decreasing his/her risk of injury by doing surgery? (2) Are we making him/her a better player? (3) Are we making him/her less likely to be symptomatic later in life?

A surgeon must carefully question his/her advice concerning surgery if he/she does not have a proper alternative to the surgery, namely good, effective nonoperative care. Frankly, if all

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Table 20.1 Treatment algorithm

Bone scan with lumbar SPECT on athletes with greater than 3 weeks back pain
If the bone scan is positive, order CT scan
If the bone scan is positive and the CT shows spondylolysis = active healing lesion
Excellent chance of recovery with the use of trunk stabilization program. Patient must stop the activity that causes the pain. Return to the sport depends on rehabilitation criteria, not just pain relief
Only recommend use of brace if patient has pain preventing activities of daily living, such as school
Potential surgery if trunk stabilization exercise program unsuccessful after at least 6 months
If disc normal is normal on MRI and pars block positive, may be candidate for direct pars repair
If disc is abnormal, discogram is potentially performed and patient may be candidate for fusion. In general, we prefer anterior interbody fusion with percutaneous pedicle screws for athletes without radicular symptoms
If the bone scan is negative, order an MRI
If the bone scan is negative and the MRI is negative
Excellent chance of recovery with the use of trunk stabilization program. Patient must stop the activity that causes the pain
If the pain persists, order a CT scan
If the bone scan is negative, the MRI is negative, and the CT shows spondylolysis = cold, unhealed spondylolysis
Not likely to heal, but not likely to require surgery
If persistently symptomatic, confirmed with pars block, may be candidate for direct pars repair
If the bone scan is negative and the MRI is positive
Disc herniation—usually treatable with trunk stabilization program, stopping the painful activity, anti-inflammatory medications, and spinal injections
If performing discectomy on athlete, get preoperative CT scan to diagnose potential spondylolysis
Inflammatory discitis—usually treatable with trunk stabilization program, stopping the painful activity, anti-inflammatory medications, and spinal injections
May require fusion

one knows is the surgical technique and if one does not have a proper understanding and delivery of a system for nonoperative care, then that person should not advise surgery for the athlete. An appropriate team approach among specialists in nonoperative care and specialists in operative care can be worked out so that the decision for surgery is well founded. The surgeon must understand and participate in that portion of the decision-making process, namely, the surgeon should treat the patient with a potentially effective nonoperative treatment program. Furthermore, a surgeon should not advise surgery to an athlete unless he/she can manage the athlete's postoperative rehabilitation and return to sport.

The surgeon and the therapist or trainer must convince the athlete of the importance of the rehabilitation treatment. The answer to "When can I play?" is "When you can do your level in the rehab program." The athlete, regardless of age, must "buy in" to the rehab program. Adolescents cannot be forced to do the rehab because "mom says so" and "mom cannot fix the problem." It takes hard work and commitment by the athlete. The concept of earning the ability to play effectively and pain-free through hard work at a program that does not entail specifically playing the sport is eventually grasped by all successful athletes regardless of their age.

Trunk Stabilization Program

When prescribing treatment for spine rehabilitation, the clinician must appreciate the important role of the entire cylinder of the trunk and its supporting muscles. The static ligamentous structures of the spine provide considerable resistance to injury, but this resistance in itself would be insufficient to produce proper strength without the additional support provided through the trunk musculature and lumbodorsal fascia. Muscle control of the lumbodorsal fascia allows a much higher resistance to bending and loading stresses. The lumbodorsal fascia and the muscles attaching to it must be considered of equal importance to the

more specialized function of the intervertebral disc and facet joints.

The trunk stabilization program offers an early and graduated return to sports [3]. This program is derived from work by Jeff Saal, MD, Arthur White, MD, and others including Celeste Randolph, Michael Schlink, Ann Robinson, and Clive Brewster. It comprises a combination of activities designed to bring the spine back to a position of balance and power in injured athletes. By training muscles of the trunk to work in coordination, the program produces biomechanically sound spinal function. It uses special isometric strengthening exercises to develop specific trunk muscles that are molded with proprioceptive feedback. Muscle function based on balance and coordination, not strength alone, is the result. Initially, the athlete is taught to maintain a safe, neutral, pain-free, and controlled position. He/she then moves through a series of exercises that combine balance and coordination. Gradually, the athlete, while maintaining good trunk control, is moved in incremental steps through increasingly advanced exercises. In each succeeding exercise, the patient assumes a somewhat more precarious position than he/she had experienced in the one that preceded it.

The emphasis is on endurance. There are hundreds of stabilization exercises available; we concentrate on these eight exercises with longer-timed repetitions: (1) dead bug, (2) partial sit-ups, (3) bridging, (4) prone, (5) quadripedal, (6) wall slides, (7) ball exercises, and (8) aerobics (Table 20.2). Each of these categories consists of five levels, with each level of difficulty amplifying the intensity of performance, increasing the number of repetitions of the exercise performed, varying the body positions, adding resistance when exercising, and so forth. The program starts in a neutral, pain-free position with isometric exercises, then progresses to concentric and eccentric contractions with balance and coordination. Using excellent trunk control, the athlete performs the exercise by the clock, not the number of repetitions. If there is a loss of trunk position and/or pain during the exercise, then the athlete is asked to stop and move on to a different exercise.

Return to sport is based on the Watkins–Randall 1 through 5 rating scale of trunk stabilization exercises. An adolescent athlete should be able to do a full level 3 workout, and a college and professional athlete should be able to do level 4 or level 5 workout before practicing. After achieving a proper stabilization level, the athlete begins a program of sport-specific exercises for their individual sport. For example, a pitcher or quarterback can start shoulder exercises and soft toss at level 2. A football lineman can start light weightlifting at level 3. Steps in returning an athlete to play are as follows:

1. Complete the appropriate level of stabilization program
2. Be in excellent aerobic conditioning compatible with the sport
3. Work with the coaching staff and training staff in a series of sport-specific exercises for the individual sport
4. Return slowly to the sport with playing time or position changes as needed and specific to the sport
5. Maintain the same level of stabilization training after return to the sport for at least 1 year

Diagnosis

The anatomic lesion is critically important in determining the prognosis of surgery. An extruded disc herniation can be very amenable to a one-level microscopic lumbar discectomy. However, an annular tear of the intervertebral disc with mild nerve root irritation probably will not be made better by a decompression surgery, and usually will be made worse because of abnormal motion in the injured disc with potential scarring of the nerve root to the back of the annulus. Another important consideration before performing a discectomy is checking for the presence of a spondylytic defect. This may decrease the stability of the operative level and increase the chance for persistent symptoms.

Degenerative disc disease or inflammatory discitis can be treated with a spinal fusion. Some athletes can return to their sport after a successful spinal fusion and some may not be able to. Part of the danger is in curing the X-ray, and not the patient. Another possibility is curing the

Table 20.2 Watkins–Randall trunk stabilization scale

	Dead bug	Partial sit-ups	Bridging	Prone	Quadriped	Wall slide	Ball	Aerobic
	A	B	C	D	E	F	G	H
1	Supported Arms over 2 min Marching	Forward– Hands on Chest 1 × 10	Slow Reps Double Leg 2 × 10	Gluteal Squeeze Alternating arm or leg lifts 1 × 10 reps	Upper Ext. or Lower Ext. Hold 1 × 10	Less than 90 degree Reps 10 ×	Balance on Ball Leg Press	Walk Land and Water
2	Unsupported Arms over Head/one leg Extended × 3 minutes	3 × 10 Fwd Hands on Chest	Slow Reps Double Leg Weight on Hips 2 × 20	Alternating Arm/Leg Lift 2 × 10 Hold	Arm and Leg 2 × 10 Hold	90 degree Hold 20 seconds 10 ×	Leg press w/arms over head Sit-ups forward No hold Run	10 min. Cycle Water
3	Unsupported Arms 7 min. Over alternate Leg Extended with Weights	3 × 10 Fwd 3 × 10 Right 3 × 10 Left	Single Leg 3 × 20 Hold Double w/weights Double on ball	Ball Flys Swims Superman 2 × 10	Arm and Leg 3 × 20 Hold 5 seconds w/weights	90 degree Hold 30 seconds 10 × Lunges/no weights	Ball sit-ups × 20 Fwd, Right, Left	20–30 min. Swim and Nordic Track
4	Unsupported Arms 10 min Over alternate Leg Extended with Weights	3 × 20 Fwd. 3 × 20 Right 3 × 20 Left Weights on Chest	On Ball Single Leg 4 × 20 Hold dbl on ball w/weights feet on ball double bridge	Ball 10 × 20 Hold Superman w/wts Prayer Pushups Walkouts	Arm and Leg 2 × 20 Hold 10 seconds w/weights Body blade	90 degree Hold 15 seconds w/Weights × 10 Lunges w/weights	Ball, Sit-ups Fwd, Right, Left w/wts 3 × 20, Wand Manual Resistance, Pulleys	45 min. Versiclimor also Step Skip rope
5	Unsupported Bil. LE ext 15 min. total Increased wts Bil. UE w/bil LE extension	3 × 30 Fwd 3 × 30 Right 3 × 30 Left Unsupported Weights overhead and behind	On Ball Single Leg 5 × 20 w/weights holding dbl w/ feet on ball and bil knees flex	Ball, All Exercises w/weights 4 × 20 Body blade	Arm and Leg 3 × 20 Hold 15 seconds w/weights Body blade	90 degree Hold Arms Extended w/weights × 10 Lunges w/weights hold 1 min	Ball Overhead and Lateral Pull through Sports Stick Pulleys Body blade	60 min. also Run

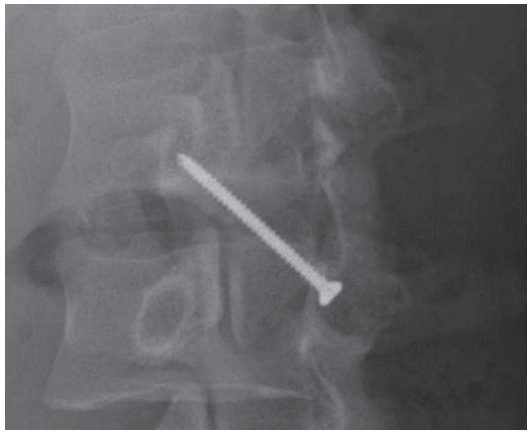

Fig. 20.1 Screw repair of spondylolysis in rugby player

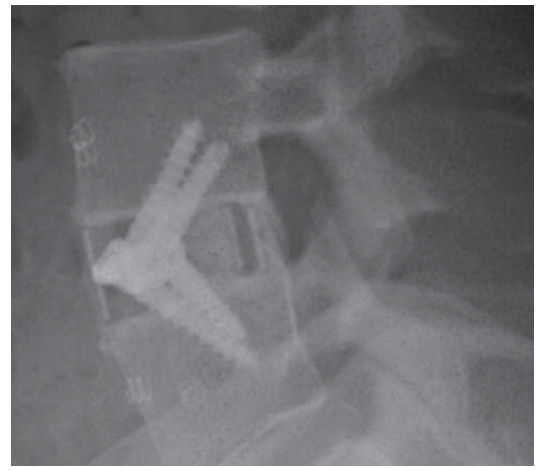

Fig. 20.2 Anterior stand-alone fusion in softball player

patient with a successful operation and leaving the player without a job.

As with all patients, an absolute indication for surgery and lumbar disc disease is progressive cauda equina syndrome or progressive neurological deficit. Strong, relative indications are static significant neurological deficit, unrelenting night pain, and major loss of functional capability. Mild relative indications for surgery fall more under the category of performance enhancement and return to play. There will always be patients who could live the way they are but cannot perform the way they are. This is a relative indication for surgery, but must be a frequent consideration in spine injuries in athletes.

Treatment: Spondylolisthesis/ Spondylolysis

There are very limited indications for the surgical treatment of spondylolysis. In 25 years of treating spondylolysis in adolescent, college, and professional athletes with our trunk stabilization program, we have resorted to surgery only three times. One was in a minor league baseball pitcher with a unilateral defect who had already missed one season and was in danger of missing another. Another was for bilateral spondylolysis in a minor league pitcher. The third was a professional rugby player who had missed one season due to the pain. The incidence of symptomatic acute spondylolysis not responding to stabilization training and preventing return to sport is rare. Studies have shown that at least 90% of children returned to their previous level of activity with conservative management of spondylolytic defects [4, 5]. Experiences with extremely demanding activities such as cricket fast bowlers may be different. Persistent pain and decreased function, despite proper trunk stabilization exercises for at least 6 months, is a reasonable time to consider surgery [6].

Direct repair of the spondylitic defect has been shown to be effective [7–9]. Many different techniques have been described, such as hook screws [7, 8, 10], translaminar screws [7, 8], wiring [7], and pedicle screws with a V-shaped rod [11]. Our operation of choice is an image-guided lag screw across the spondylolytic defect and grafting of the defect with minimal exposure (Fig. 20.1). A prerequisite for a spondylolysis repair is that the disc is normal [12]. This can be determined with **magnetic resonance imaging (**MRI) or discography [9].

Patients with significant disc degeneration may need a one-level fusion. Our preferred fusion is a stand-alone anterior lumbar interbody fusion (ALIF) with plastic graft, titanium screws, and synthetic bone morphogenic protein (Fig. 20.2). Unless the patient has significant radicular symptoms, in which case we may perform a decompression as well. If there is segmental instability,

we often perform ALIF with pedicle screws. We have performed two fusions in professional athletes for spondylolisthesis with a 50% effectiveness rate. Lumbar spinal fusion in professional athletes is not a very successful operation because of the high demands placed on adjacent levels, the amount of time out from the sport for the fusion to heal, and the aggressive demands put on the spine in the early-healed phase. Artificial disc replacement for spondylolisthesis is generally not recommended due to concerns about stability.

After direct repair of a pars defect or a spinal fusion, we limit a patient's activity to only ambulation or recumbent bike for 10 weeks. If the X-rays show solid fixation and the patient has minimal pain, we begin level 1 of the trunk stabilization program (neutral, pain-free, isometric exercises). In general, after 6 months, if the athlete has met the return-to-play criteria and has minimal pain, we will obtain a **computed tomography (**CT) scan to check for bony union. The athlete may return to play if there is evidence of bony union, minimal symptoms, and he/she has completed the five criteria for return to play.

Treatment—Disc Pathology

After a discectomy, athletes start the trunk stabilization rehabilitation program when their pain is minimal, typically 3–6 weeks. The return to sport guidelines have been previously described. If a patient has a complicating factor, such as spondyloysis, we may be less aggressive in our timetable for return to play. If a patient has a flare-up of pain in the postoperative period, we typically treat them with anti-inflammatories, spinal injections, and rest. The amount of pain the player experience determines how aggressive the postoperative rehabilitation proceeds.

Our recent study showed overall return to play after microscopic lumbar disctomy in professional athletes to be 89% [13]. Progressive return to play analyzed if a patient's sport was in-season at 3-month time intervals postop, what percentage of players returned at those times. We found that the chance of a player returning to their sport was 50% at 3 months, 74% at 6 months, and 86% at 12 months, if their sport was in-season at those time intervals. Hsu has demonstrated that if a player is able to return to sport after lumbar discectomy, then their postoperative performance is similar to preoperative [14].

Treatment—Scoliosis

Return to sport after scoliosis surgery relies upon successful fusion as well as appropriate rehabilitation. There are varying opinions within spine surgeons regarding return to play after scoliosis surgery [15]. Trunk stabilization exercises can generally begin at 2–3 months post operation. Return to sport is based on the previously described criteria. Generally, 6 months is the earliest return to noncontact sports and 1 year is typical for contact sports. Obviously, failure of fusion, neurologic deficit, and/or adjacent level degeneration may limit return to sport.

Treatment—Cervical Spine

Return to sports after cervical spine surgery depends on several factors including condition of neural elements, persistence of symptoms, stability and biomechanics of the spine, completion of the rehabilitation program, and risks associated with the specific sport and position. Athletes are generally categorized into one of three groups: extreme risk, moderate risk, or low risk [16]. The risk is for potential neurologic injury and/or chronic pain.

Conditions that would qualify as an extreme risk include:

- Clinical history or physical examination findings of cervical myelopathy
- History of a C1–C2 cervical fusion
- C1–C2 rotatory fixation
- Evidence of a spear tackler spine on radiographic analysis
- A multiple level Klippel–Feil deformity
- An occipital C1 assimilation
- Radiographic evidence (i.e., MRI) of basilar invagination

- MRI evidence of significant Arnold–Chiari malformation
- Radiographic evidence of ankylosing spondylitis or diffuse idiopathic skeletal hyperostosis
- More than two previous episodes of a cervical cord neuropraxia
- Status post a cervical laminectomy
- Status post C1–C2, C2–C3, or C3–C4 fusion in head-contact sport
- Symptomatic disc herniation
- Clinical or radiographic evidence of rheumatoid arthritis
- Three-level lumbar spine fusion

Conditions that would qualify as a moderate risk include:

- Previous history of spinal cord neuropraxia; the patient must have full return-to-baseline strength and cervical range of motion
- Three or more previous stingers or burners of nondiagnostic etiology
- Status post two-level cervical fusion

Conditions that would qualify as a minor risk include:

- Single level Klippel–Feil deformity with no evidence of instability or stenosis
- Spina bifida occulta
- Status post single-level cervical fusion
- Previous history of chronic stingers or burners of known origin
- Status post a single- or multiple-level posterior cervical microforaminotomy

Postoperative Care

Typically, the surgical treatment for herniated, extruded cervical discs is an anterior cervical discectomy and fusion. After such treatment, we often place the player in a mild-risk category, secondary to the biomechanical alterations that must necessarily occur above and below the fused cervical motion segment. It is for this reason that, if given the appropriate clinical indications, we might recommend a microscopic cervical foraminotomy for the treatment of monoradiculopathy secondary to foraminal stenosis in an athlete involved in contact sports. For the athlete with significant intermittent radiculopathy, a positive Spurling's hyperextension test, and foraminal stenosis, a posterolateral foraminotomy is a reasonable approach. The technique of this operation is adopted from Robert Warren Williams [17] and includes a minimal resection of the posterior wall of the foramina and detachment of the ligamentum flavum only until nerve root pulsations are clearly present. A significant facet resection would make return to head-contact sport contraindicated.

Postoperatively, return to play is based on the progression through the trunk stabilization and chest-out posture rehabilitation program. The patient is immobilized in a hard cervical collar for 2 weeks. Most patients are removed from the collar at the 2-week postoperative visit depending on X-rays and symptomatology. In general, foraminotomy patients begin the trunk stabilization program with physical therapist at 4 weeks post operation. Fusion patients typically begin at 6–10 weeks post operation.

The rehabilitation program begins with establishing a stable core of trunk muscles. Isometric trunk exercises and upper body exercises that emphasize chest-out posture strengthen the supporting structures for the cervical spine and the postural muscles necessary for maintaining proper body alignment. Once the patient reaches level 3 of the program in most trunk exercises, then chest-out posture and scapula stabilization exercises are introduced. The patient must establish pain-free neutral trunk strength before beginning rotational exercises. Once a patient has reached the appropriate trunk stability level (3 for high school, 4 for college, and 5 for professional), then sport-specific exercises are preformed.

Aerobic activity begins with ambulation immediately after operation. In the first 6–12 weeks, ambulation on an incline and a recumbent bike are recommended. Once the patient achieves level 3 of trunk stabilization and chest out posture exercises, aerobic exercise can increase to running, elliptical, swimming, etc. Return to play requires the previously described five criteria.

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Adaptive Sport

21

David M. Popoli

Introduction

Disabled sport, also called "adaptive sport" or "Paralympic Sport" when practiced at its most elite level, grew out of a unique treatment paradigm for wounded World War II soldiers. In 1939, during the height of Nazi influence on German politics, Sir Ludwig Guttmann (1899–1980), a Jewish, German-born neurologist and neurosurgeon, applied for and was granted an English visa. He initially took a position at Oxford but later became the Director of the National Spinal Injuries Unit at the Ministry of Pensions Hospital located in Stoke Mandeville, Aylesbury.

Stoke Mandeville was the platform from which Guttmann launched innovative rehabilitation methods for people with spinal cord injury (SCI) or amputation. Former department directors had encouraged rest and antiseptic salves. Guttmann credited his patients for inspiring more unconventional methods; the most controversial of these techniques was the incorporation of sport as a cornerstone of therapy. While on rounds, he happened upon a game of "wheelchair polo"—a group of patients organized into two teams furiously propelling their wheelchairs after a "puck" that they smacked with upside-down walking sticks. Although this particular activity's inherent risk precluded its long-term incorporation into the rehabilitation process, Guttmann promoted sport as "the most natural form of remedial exercise, restoring physical fitness, strength, coordination, speed, endurance, and overcoming fatigue." He also recognized that "The noblest aim of sport is to facilitate and accelerate social reintegration…, [t]o make the spinally injured person as independent as possible and to restore him to his rightful place in social life."

Guttmann's nontraditional approach worked. The mortality rate for the 2,500 patients housed on the Stoke Mandeville rehabilitation unit between 1944 and 1962 was 14.8 % (11.5 % if etiologies unrelated to SCI were eliminated) [1]. The relative risk of mortality for those treated at Stoke Mandeville after an SCI was 3.49, between 1943 and 1952, which compared favorably to a relative risk of 5.59 at other centers [2]. Moreover, Guttmann's program achieved high rates of social reintegration: 1,682 of 2,500 patients were discharged home, and 85 % of those individuals returned to at least part-time work.

Beyond their rehabilitative qualities, the athletics at Stoke Mandeville took on increasingly competitive characteristics. In 1948, the first Stoke Mandeville Games were held and included 16 patients from two hospitals—Stoke Mandeville and the Star and Garter Home. The following year, there were 36 competitors from six hospitals. Guttmann declared the Games would "achieve world fame as the disabled men and women's equivalent of the Olympic Games."

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Even the enthusiastic Guttmann was unlikely to have anticipated the rapid growth of adaptive sport. The first "Paralypmic Games," held in Rome in 1960, saw 138 athletes from 17 countries vying for medals. By the 2008 Beijing games, the numbers swelled to 4,011 athletes representing 146 countries. A total of 28 sports (23 summer, 5 winter) are now represented at the Games, and the field has expanded to include athletes in six major subcategories: amputee, cerebral palsy (CP), intellectual disability, visually impaired, wheelchair, and *les autres* ("the others"—including dwarfism, multiple sclerosis, or congenital deformities). The adaptive sport community has embraced the vision of the Paralympic movement, to "enable athletes to achieve sporting excellence and inspire and excite the world" through the values of "courage, determination, inspiration, and equality" [3].

In addition to the physical demands of any athlete, those involved in adaptive athletics have unique physiology and biomechanics that must be considered. For example, changes to the cardiovascular system alter aerobic capacity, contractures, and postsurgical hardware impact the musculoskeletal system, and wheelchair propulsion itself leads to an increased risk of upper extremity injury. This chapter explores some of the changes the adaptive athlete encounters and examines their effects on athletic performance and injury patterns. It also discusses injury prevention and the health benefits of regular exercise for this unique group of athletes.

Physiology

Cardiovascular

Aerobic

Aerobic power is defined as the maximum oxygen uptake per unit of time ($VO_{2\,max}$), often represented by the Fick equation:

$$VO_{2\,max} = Q\,(a[O_2] - v[O_2])$$

$VO_{2\,max}$ is the product of cardiac output (Q) and arteriovenous oxygen difference ($a[O_2] - v[O_2]$). Cardiac output is the primary factor in this equation and is directly proportional to heart rate and stroke volume.

Those with SCI have reduced $VO_{2\,max}$ levels that correlate with the level of neurological injury. Low thoracic and lumbar paraplegics have no innervation to the lower extremities and therefore lack the muscle pump that aids in venous return. This reduces stroke volume and $VO_{2\,max}$. All SCIs lead to the decoupling of the sympathetic and parasympathetic systems, and injuries above T6 result in the disruption of sympathetic innervation to the splanchnic bed, leading to venous pooling and further reduction in stroke volume. When the high thoracic or cervical spine is lesioned, sympathetic outflow to the heart is interrupted. Catecholamine response is blunted, heart rate declines, and cardiac output falls. Additionally, all individuals with SCI undergo changes to both muscle mass and fiber composition. Significant atrophy occurs below the level of injury, and there is an increase of fast glycolytic type IIB fiber and reduction of slow oxidative fibers [4]. Less oxygen, therefore, is extracted by the skeletal muscle, and the arteriovenous oxygen difference narrows, further decreasing $VO_{2\,max}$.

The overall effect on aerobic power is quite significant: measured $VO_{2\,max}$ can range from 18.8 ml/kg/min in a C7 tetraplegic Paralympic wheelchair racer to 45.8 ml/kg/min in an L4 Paralympic counterpart [5]. By way of comparison, the average elite-level high school cyclist has a $VO_{2\,max}$ of 61.4 ml/kg/min [6]. Reductions in $VO_{2\,max}$ translate to decreased aerobic endurance and lower anaerobic thresholds, and venous pooling in the splanchnic bed can precipitate orthostatic hypotension that causes unpleasant symptoms and saps athletic performance.

Unlike SCI athletes, those with CP have intact innervation to the myocardium, and should theoretically be able to achieve a $VO_{2\,max}$ comparable to the general population. The muscle spasticity frequently seen in CP, however, increases metabolic demands and can lead to premature muscle fatigue before cardiovascular exhaustion. This is reflected in a $VO_{2\,max}$ higher than a tetraplegic athlete but below that of a well-trained paraplegic one.

The measured $VO_{2\,max}$ for athletes with amputations varies broadly with level and laterality. Distal unilateral lower extremity amputees achieve a $VO_{2\,max}$ almost identical to the general

population, but proximal bilateral lower extremity amputees experience significant reductions. The changes in aerobic capacity are related to energy expenditure during ambulation. A unilateral below-the-knee amputation (BKA) only increases metabolic cost by 20–25 %, but a bilateral above-the-knee amputation (AKA) may cost as much as 200 % more than able-bodied controls [7].

Anaerobic

In individuals with SCI, anaerobic capacity is inversely proportional to the neurological level of injury. Those with low lumbar or sacral lesions can achieve anaerobic powers three to four times that of those with a cervical spine lesion.

The anaerobic capacity for CP and amputee athletes has been difficult to study because of small sample sizes, inconsistent controls, and lack of normative data for this population [8]. One small study found anaerobic output in track athletes with CP to be considerably lower than that of comparable wheelchair and amputee competitors [9]. This was attributed to impaired coordination and the possibility of diminished fast-twitch muscle fiber.

Training Considerations

- When designing an aerobic training program for an individual with an SCI, using percentage of maximum heart rate to gauge intensity may not be accurate. SCI blunts heart rate response, and therefore, using perceived exertion (such as the Borg Scale) may yield better results.
- In SCI athletes, the risk of orthostatic hypotension can be reduced by maintaining excellent hydration and avoiding training in high-humidity environments. These athletes may also consider using an abdominal binder to offset venous pooling in the splanchnic system.
- Amputee and CP athletes have no specific metabolic training requirements, but one should consider increased per unit distance energy expenditure during program design.

Training Benefits

- Athletes with SCI who participate in endurance training that meets the American College of Sports Medicine (ACSM) guidelines for intensity, frequency, and duration have been shown to increase $VO_{2\ max}$ between 8 and 28.8 % [8, 10, 11]. Some researchers postulate that the changes stem from improvement in stroke volume, whereas others hypothesize that regular aerobic activity improves $VO_{2\ max}$ by increasing oxygen extraction in skeletal muscle.
- The cardiovascular benefits of intense arm crank ergometry (ACE) can be augmented with the use of functional electrical stimulation (FES) of the lower extremities, and this form of "hybrid" exercise has been shown to increase stroke volume by 33 % [12].
- Regular cardiovascular exercise may improve or eliminate orthostatic intolerance, particularly if performed in an upright or standing position [13, 14].

Neurologic

Most SCIs and all cases of CP represent upper motor neuron lesions. The resulting imbalance of the inhibitory and excitatory inputs to the stretch reflex leads to a velocity-dependent increase in muscle tone—spasticity. Spasticity can not only create significant discomfort in skeletal muscle but can also interfere with function and reduce athletic performance. In extreme cases, spasticity can lead to joint contracture, peripheral neuropathy, or bony fracture [7].

The upper motor neuron changes that occur with SCI and CP also alter the activity of smooth muscle in the genitourinary and gastrointestinal tracts; this manifests as urinary and fecal incontinence or retention. Constipation, bladder distention, and kinked catheters can induce potentially life-threatening autonomic dysreflexia. Furthermore, incontinence compounds the risk of skin breakdown for athletes already at high risk of pressure ulcers due to seating position and/or insensate skin, so close attention to bowel and bladder management programs is essential.

Athletes without a regular voiding program risk a serious physical complication called autonomic dysreflexia. For individuals with neu-

rological injury above T6, the relay between the parasympathetic and sympathetic pathways to the splanchnic system is broken. In response to a painful stimulus distal to the level of the spinal cord lesion (such as bladder/bowel distention or a pressure ulcer), an ascending sympathetic discharge generates peripheral vasoconstriction and a resultant increase in blood pressure. Ordinarily, a corresponding descending parasympathetic response would create a buffer, but the discontinuity created by an SCI prevents signal transmission. Systemic blood pressure can rise to dangerous levels, placing the athlete at risk for cerebral hemorrhage, aphasia, blindness, cardiac arrhythmias, and death [8].

Treatment for autonomic dysreflexia begins with removal of the noxious stimulus. Empty the bladder, evacuate the bowels, and remove tight-fitting garments. Sit the athlete upright to induce orthostatic hypotension. If hypertension persists, then nitropaste applied to the skin or sublingual nifedipine are potential treatment options. Exercise itself can also trigger autonomic dysreflexia, so the adaptive athlete must remain vigilant for early warning signs such as headache, blurred vision, or flushing/sweating above the level of the SCI. Despite these risks, some athletes knowingly induce autonomic dysreflexia (known as "boosting") in order to improve athletic performance. The sympathetic surge increases both heart rate and stroke volume, thereby generating as much as a 10% improvement in $VO_{2\ max}$ [8].

Although it is not life-threatening, neuropathic pain represents a major obstacle for adaptive athletes. Changes in neuronal function can occur at both the central and peripheral nervous system levels. Among other alterations, SCI interrupts the transmission of a descending spinal inhibitory signal that would ordinarily prevent hyperexcitability of the sympathetic nervous system. When the unchecked system is combined with the upregulation of the *N*-methyl-D-aspartate receptor commonly seen after SCI, a feed-forward loop is created that can result in central neuropathic pain. In the peripheral system, neurons demonstrate altered ion channel permeability and produce increased amounts of cytokines. These changes can lead to altered intracellular signaling and paradoxical sprouting of new, hypersensitive nerve endings that are primed to conduct pain signals throughout the body [15].

The dysfunctional signaling pathways of the peripheral and central nervous systems also affect thermoregulation. In the SCI athlete, there are changes in skin blood flow and sweating below the level of the lesion, increasing the probability of hyperthermia during exercise. The severity of risk is higher in those with tetraplegia and with complete lesions [16]. Acclimatization is important for all athletes but in particular for those involved in adaptive sports. Hot, humid environments increase the risk of hyperthermia, so adaptive athletes should arrive at the athletic venue at least 7 days prior to the competition to adjust to conditions.

Training Considerations

- All adaptive athletes should maintain excellent hydration status, as dehydration can worsen spasticity. A simple training tool is the Urine Color Test Card, which gives the athlete a visual frame of reference to determine if he or she is drinking enough water.
- Adaptive athletes should familiarize themselves with any personal spasticity triggers, and if appropriate, carry antispasticity medications. However, caution should be taken when administering these medications around the time of competition, as they can interfere with thermoregulation.
- In SCI athletes, the risk of autonomic dysreflexia can be reduced by regular straight catheterization, which keeps bladder volumes low, and a bowel program to prevent rectal distention. Athletes should be counseled to wear loose-fitting garments to reduce the risk of catheter kinks and shear forces at skin folds that can also trigger autonomic dysreflexia.
- Loose-fitting garments also enhance evaporation of sweat to facilitate cooling. Other measures to improve thermoregulation include avoidance of training in high-humidity environments, ensuring adequate fluid–electrolyte balance, and refraining from competition when ill or injured.

- Neuropathic pain should be monitored closely, as it can be a signal of infection or injury.

Training Benefits

- Regular exercise helps improve bladder and bowel dynamics and may reduce the risk of urinary tract infection in individuals with SCI [17].
- Exercise helps decrease spasticity in individuals with SCI and CP [18].
- In animal models, there is a suggestion that regular exercise may promote new dendrite sprouting and improve nerve function after a SCI [19, 20].

Respiratory

Depending on the level of the lesion, SCIs have a varying impact on the function of respiratory muscles. High cervical lesions (C3, C4, C5) may damage input to the phrenic nerve and eliminate spontaneous respirations altogether. Cervical and high thoracic lesions weaken or paralyze both inspiratory and expiratory muscles and impact lung function; forced expiratory volume in 1 s (FEV_1), forced vital capacity (FVC), and FEV_1/FVC ratio are all significantly lower in individuals with SCI than in able-bodied controls [8]. Although lumbar lesions have a less pronounced effect on respiratory muscle function, the seated position and the forward-crouched posture of wheelchair propulsion result in a biomechanical disadvantage. Athletes with CP experience similar alterations in biomechanics, and respiratory muscle spasticity may compound the problem.

Training Considerations

- The inefficiencies created by poor respiratory mechanics lead to increased use of accessory muscles of respiration and may result in premature fatigue during training.
- Alterations in respiratory mechanics (including poor cough strength) predispose SCI athletes to pneumonia.

Training Benefits

- The respiratory benefits of regular exercise vary broadly depending on the level and completeness of a spinal cord lesion or the degree of spasticity in CP. In one study involving a 6-week trial of arm ergometry, however, Silva et al. reported a 20% increase in respiratory muscle endurance, a 36% increase in FVC, and an 18% decrease in respiratory infections [21].

Musculoskeletal

SCI significantly decreases muscle bulk; the average cross-sectional area of the vastus lateralis drops by nearly 25% 11 weeks after injury [22]. Consequently, there is no skeletal muscle traction at the bone/tendon interfaces of the lower extremities. Coupled with the elimination of weight bearing, bone mineral density subsequently declines at a rate of 2–4% per month in trabecular bone and 2% per month in cortical bone [23]. As a result, even minimal trauma during athletic training can result in osteoporotic-type insufficiency fractures. Moreover, SCI athletes may not report symptoms after sustaining a fracture in an insensate region, so the index of suspicion for fracture must remain high. Amputee athletes may also have altered sensation at the distal aspect of the residual limb, and there have been case reports of fractures attributed to post-amputation osteopenia.

Another potential skeletal complication of SCI is neuropathic spinal arthropathy—so-called "Charcot spine." Denervation of the muscles that ordinarily stabilize the spine leads to increased motion at the facet joints, and the forward-leaning posture of wheelchair propulsion alters loading biomechanics. As a result of these combined factors, the facet joint wears prematurely, which manifests as pain and audible noises with motion. Generally, neuropathic spinal arthropathy is a late complication of SCI, with diagnosis ranging between 6 and 31 years post injury and presenting most commonly in the lumbar region

[24, 25]. The cervical spine is also subject to repetitive loading and altered mechanics, and neck pain, particularly at the cervicothoracic junction, is not uncommon.

Bony injuries are not the only major consideration. Bernardi et al. reviewed 12 months of data regarding "sports-related muscle pain" among elite adaptive athletes and found that the most commonly affected regions were the shoulder (56%), upper limb (33%), and lumbar spine (13%) [26]. Wheelchair athletes place abnormally high stress on the muscles of upper extremity. The rotator cuff, proximal biceps tendon, and glenohumeral joint absorb continuous motion during wheelchair propulsion and are therefore at increased risk of overuse injury. The biceps tendon, in particular, is subject to heavy eccentric loads during the elbow-drive phase of the racing/acceleration stroke [27]. Musculoskeletal demands are highly variable for CP competitors. For those with relatively mild neuromuscular involvement, there is little difference from the able-bodied population. High levels of spasticity, however, can place the athlete at risk for tendon avulsion or joint subluxation/dislocation.

Both SCI and CP athletes face higher rates of scoliosis due to spasticity, decreased core strength, and altered seating position. Depending on its severity, scoliosis can alter loading mechanics through the axial skeleton and contribute to premature thoracic or lumbar facet arthropathy. Surgical rods may prevent the progression of the curvature, but they also make the spine rigid and generate additional wear above and below the fused segment [28]. Although athletes with a lower extremity amputation do not experience higher rates of degenerative disc disease, they too have unique biomechanics. Forces through the "sound" limb often exceed that of the prosthetic one, and some studies have shown increased prevalence of knee osteoarthritis when compared with able-bodied persons [29].

Training Considerations

- SCI athletes and their trainers should pay exquisite attention to any areas of swelling or discoloration denervated limbs, as this may be the only sign of tendon rupture or fracture.
- Pre- and postexercise stretching is an essential part of any musculoskeletal training program for athletes with SCI or CP, as spasticity can decrease muscular performance and place the athlete at risk for injury.
- Training protocols for amputee athletes should avoid excessive pressure over the residual limb and the prosthesis. Like the SCI athlete, amputee athletes can have reduced bone density that increases the risk of insufficiency fracture, and specialized prostheses are highly sophisticated and can be expensive to repair when damaged.
- Strength training that uses motion analysis to ensure maximum biomechanical efficiency may be beneficial for both amputee and CP athlete, by increasing performance and reducing injury risk.
- Specialized equipment for adaptive athletes increases participation, but it can also be associated with increased risk of injury. Athletic wheelchairs generally weigh less and are more aerodynamic, thereby improving speed and agility, but these chairs lack the stability and structural integrity of the average day chair. When they occur, wheelchair accidents carry high risk of injury, because the athlete's mobility impairment precludes him from avoiding impact.

Training Benefits

- Regular exercise, particularly when paired with electrical stimulation of denervated muscle, can help restore muscle mass and prevent changes in muscle fiber composition [30].
- Bone mineral density increases with the combination of exercise and FES [31–33].
- In wheelchair users, regular exercise helps decrease shoulder pain and increase quality of life [34].

Fluid, Electrolytes, and Nutrition

Basal metabolic rate decreases after SCI and is inversely proportional to the level of injury. An elite C7 tetraplegic wheelchair racer has a caloric need of approximately 1,200–1,400 kcal/day [8].

For the amputee athlete, energy expenditure for a given activity is slightly higher than that of the general population. This varies with the level and laterality of amputation but does not have a significant impact on caloric needs. Athletes with CP also demonstrate increased energy expenditure based on severity and laterality of spasticity/tone, but these changes do not affect nutritional demands.

Training Considerations

- There is little scientific literature regarding nutritional supplementation or special diets in the adaptive athlete. One study showed that a 6-day creatine load had no effect on wheelchair racer performance [8]. A recent study by Temesi showed no improvement in arm-cranking performance in wheelchair track athletes after consuming a high-carbohydrate diet [35].
- As discussed in Sect. "Training Considerations" of Sect. "Neurologic", all adaptive athletes should maintain excellent hydration status to avoid complications during training, including orthostatic hypotension and hyperthermia.
- Energy expenditure in adaptive sports athletes is generally less than that for able-bodied counterparts.

Training Benefits

- As in the able-bodied population, regular exercise reduces rates of obesity and improves morbidity.

Psychological

A physical disability influences every facet of an individual's life, including self-esteem, school performance, employment, relationships, and empowerment. Particularly if traumatically acquired, a physical change in one's body can result in feelings of hopelessness, depression, anger, and solitude. In general, however, adaptive athletes face the same psychological obstacles as all individuals engaged in sport, including fear, performance anxiety, and stress management.

Training Considerations

- An important psychological consideration for adaptive athletes is retirement. Like any elite-level athlete, these individuals have committed themselves to rigorous training over many years and often define themselves by their participation in sport. Transitioning to a skill or knowledge-based career can be frustrating and difficult, particularly if the athlete began training at an early age and/or was making a livelihood through competition.
- Current best practice for sports psychology is applicable to adaptive sport and includes mental practice, visualization techniques, and "active rest."

Training Benefits

- Engaging in sport can promote social integration and improve mood. One study of wheelchair athletes found lower anger, confusion, depression, and tension scores than in nonathletic counterparts [36]. The literature is mixed, however, as many studies of adaptive athletes have reported results that do not demonstrate significant differences from the able-bodied population.

Other

For all adaptive athletes, skin integrity is a major issue. Shear forces affect the sacrum and perineum of wheelchair athletes, amputees must ensure proper socket fit of their prostheses to prevent skin breakdown, and CP athletes may have altered gait mechanics that lead to foot problems or increased tone that generates abnormal skin friction. Skin breakdown, when it occurs, often leads to infection, particularly since so many of these athletes have altered skin sensation.

Participation

Sport Options

Once an individual chooses to pursue adaptive athletics, his or her options are essentially

Table 21.1 Current Paralympic sports

	Team	Individual
Summer	Athletics (track and field)	Archery
	Boccia	Athletics (track and field)
	Equestrian	Boccia
	Football 5-a-side	Equestrian
	Football 7-a-side	Judo
	Goalball	Para-canoeing
	Para-cycling	Para-cycling
	Rowing	Para-triathlon
	Sailing	Powerlifting
	Sitting volleyball	Rowing
	Swimming	Shooting
	Table tennis	Swimming
	Wheelchair basketball	Table tennis
	Wheelchair dance	Wheelchair fencing
	Wheelchair rugby	Wheelchair tennis
	Wheelchair tennis	
Winter	Ice sledge hockey	Alpine skiing
	Wheelchair curling	Biathlon
		Cross-country skiing

limitless. Nearly every able-bodied sport has an adaptive equivalent—even extreme sports such as mountain climbing and skydiving. The more "traditional" sports are also numerous. Table 21.1 lists the 28 Paralympic sports. All Paralympic athletes undergo "classification" prior to competition to ensure fairness and equality. This is a standardized process during which athletes are grouped for competition according to the degree of activity limitation resulting from their physical impairment—similar to grouping athletes by weight class or age.

Facilitators of Participation

The primary facilitator of participation is accessibility. At a basic level, the adaptive athlete must be able to travel to and from venues and negotiate the physical space. Regional sport clubs can help organize carpools or may have access to a van or minibus. Traveling with other athletes is not only more cost efficient but it also reinforces the team nature of sports. Once a competitor arrives at his or her venue, it is important to insure that the physical location meets Americans with Disabilities Act (ADA) standards or can be easily modified to meet them. Even highly motivated individuals can be frustrated in their attempts to stay active if they cannot use the fields, restrooms, or locker rooms. Accessibility also extends to equipment. Although some sports can be played with a daily-use wheelchair or prosthetic limb, competitors often need specialized equipment to compete at the elite level. Wheelchairs, for example, are often sport-specific, and the design and function of a lower extremity prosthesis is frequently tailored to the physical demands of the athlete's activity.

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The Spine in Skeletal Dysplasia

22

Lawrence I. Karlin

The Skeletal Dysplasia

The skeletal dysplasias are a group of diseases characterized by an abnormality in bone form or modeling, and are caused by an error intrinsic to bone. [22]. The extent of involvement will vary between the long bones and the spine, and between the various portions of the individual bone. In the long bones, the aberrant modeling may be located in the epiphysis, physis, metaphysis, or diaphysis. In general, skeletal dysplasias will present with short stature. This will be either proportionate, with the trunk and limbs affected similarly, or disproportionate, with either short limb or short trunk forms. The various dysplasias may also be differentiated by the disproportionate involvement of the long bone segments. The most involved portion may be either the proximal, mid, or distal segments, termed respectively rhizomelic, mesomelic, or acromelic.

Systemic abnormalities of collagen or connective tissue also produce clinical deformities that meld with those caused by disturbances in bone or cartilage. These have also been grouped as skeletal dysplasia. Taking this into account, there are over 350 skeletal dysplasias [24]. These vary in their pattern of inheritance and in their clinical presentations. Some are so severe that they are incompatible with life while others have little, if any, effect on the individual's well being. They can be easily diagnosed at birth or go undetected for many years, if not a lifetime (Fig 22.1). Some, achondroplasia, for example, are quite homogeneous with all individuals similarly affected. Others present significant phenotypic variability. There can be differences between subgroups and between individuals within the same subgroup.

The spine is not always involved in the skeletal dysplasias. When it is, there are in general, three types of problems created: spinal instability, deformity, and stenosis (Table 22.1). The instability is usually limited to the cervical spine, while the stenosis may involve the upper cervical or lumbar regions. The deformity is more often increased kyphosis than scoliosis. The more common skeletal dysplasias are discussed and the principles developed can be utilized for others.

These are uncommon disorders that will infrequently be encountered by sports medicine physicians. Certainly many individuals with skeletal dysplasias have handicaps that will prevent even modified sports activities. When the young participant arrives with a diagnosed skeletal dysplasia, it must be established that they have been screened and appropriately treated for the known spinal problems associated with that disease. The activity can then be modified for safety. In general, any type of contact sport is contraindicated in the presence of potential cervical instability. Lumbar extension should be minimized in the presence of lumbar spinal stenosis. On the other hand, some activities or training techniques may

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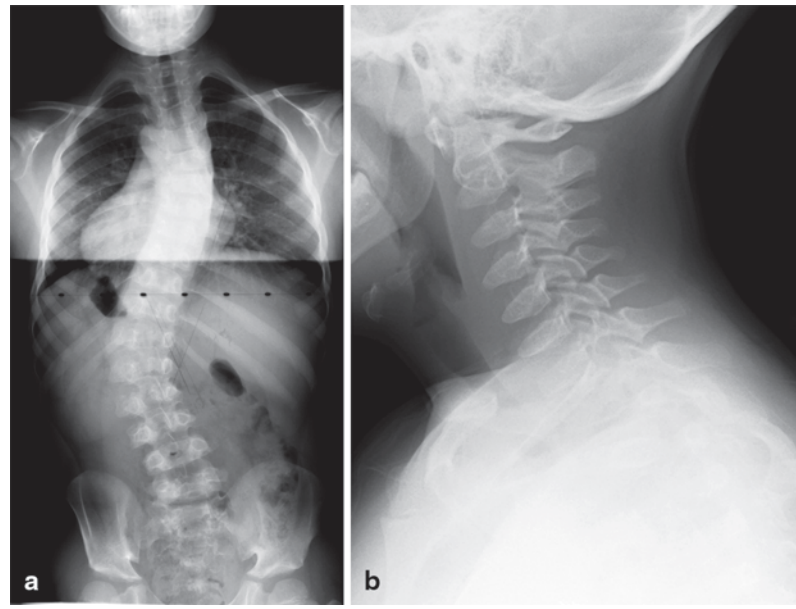

Fig. 22.1 This teenager has spondylometaphyseal dysplasia. There is scoliosis (**a**) and platyspondyly best noted on the cervical radiograph (**b**). His mother, who had scoliosis surgery at a major teaching hospital, has the same condition. She is 4 ft 10 in. tall, but never knew she had this diagnosis until it was identified in her son

Table 22.1 Pathologic spinal conditions associated with selected skeletal dysplasia

	Stenosis			Instability	Deformity		
	Cervical	Foramen magnum	Lumbar	Atlanto/axial odontoid hypplasia	Scoliosis	T/L Kyphosis	Lordosis
Achondroplasia		X	X			X	X
Hypochodroplasia		X	X				X
Pseudochondroplasia				X	X		
Spondyloepiphyseal dysplasia congenital				X	X		X
Spondyloepiphyseal dysplasia tarda				X	X		
Mucopolysaccharidosis Type IV	X			X	X	X	X
Osteogenesis Imperfecta		X			X		

be beneficial, such as core strengthening and hip flexor stretches for hyperlordosis. The sports medicine physician should be vigilant for problems that may occur in children with skeletal dysplasia mild enough to have avoided detection.

Achondroplasia

Achondroplasia is the most common skeletal dysplasia. The reported incidence is 1/30,000 live births [17]. It is inherited as an autosomal recessive with complete penetrance, though in 90% of cases it occurs through spontaneous mutation. There is no mutation variability and the phenotypic presentation is constant [21].

The defect is in enchondral ossification and the result is dwarfism characterized as proximal limb shortness or rhizomelic micromelia. The average height is 4'5" in men and 4'1" in women. The defect involves a mutation at fibroblast growth factor receptor protein-3 (FGFR3) producing a "gain of function" which in this case is an increase in the inhibition of the cells of the

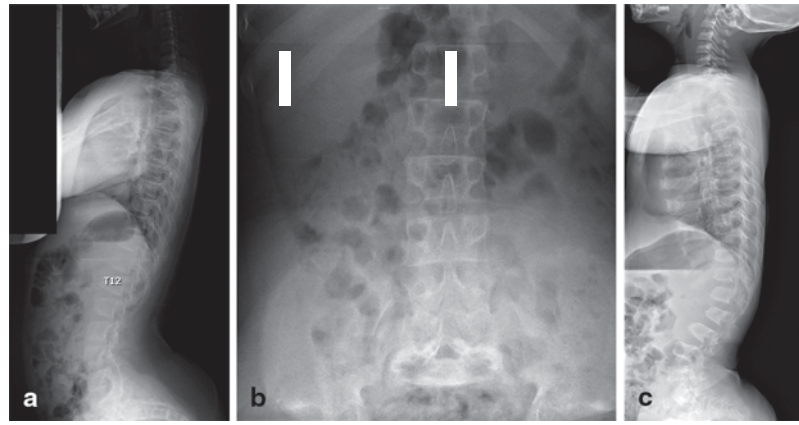

Fig. 22.2 Wedging of the vertebral bodies at the thoracolumbar spine (**a**) and lumbar spinal stenosis occur frequently in children with achondroplasia (**a**, **b**). In some, a kyphotic deformity will occur (**c**)

proliferative zone of the growth plate [7]. The distinguishing clinical characteristics are disproportionally short stature with shortened long bones. The shortening is rhizomelic, or most marked in the proximal segment. There is frontal bossing, macrocephaly, midface hypoplasia, and a trident hand shape. The developmental motor milestones may be delayed.

The primary spinal manifestations are spinal stenosis at the cephalad and caudal portions of the spinal column and a thoracolumbar kyphotic deformity (Fig. 22.2). The foramen magnum stenosis is present at birth. This usually improves over time, but in the infant it may be a cause of hypotonia, sleep apnea, cranial nerve dysfunction, weakness, and developmental delay [23]. Unexpected infant death has been reported [26].

Lumbar spinal stenosis is present in 80–90% of achondroplastic individuals. The neurocentral synchondrosis is responsible for both the elongation of the pedicles and the width of the vertebral bodies. Inhibition of this growth results in biplanar stenosis; the spinal canal is narrower in both the coronal and sagittal plane [14]. Additionally, instead of enlarging from the cephalad to caudal direction as is the norm, the lumbar interpeducular distance, as measured on frontal radiographs, narrows. Early symptoms of spinal stenosis may begin in the teen years and include activity-related cramping and lower extremity fatigue, which may be relieved by maneuvers such as leaning forward or sitting that minimize the lumbar lordosis. As the stenosis worsens either through degenerative changes or through increasing deformity, neurologic symptoms such as numbness, weakness, and bowel and bladder dysfunction may occur. Surgical decompression is eventually required in approximately 25% of patients [3].

Thoracolumbar kyphosis occurs often in these children. Prior to the development of an erect posture that occurs with sitting and standing, all infants have a gentle thoracolumbar kyphosis. In children with achondroplasia, the kyphosis may be exaggerated due to hypotonia, ligamentous laxity, or delayed standing. In some, it persists or increases and takes on a more sharply angulated appearance as the apical vertebral bodies, T12 and L1, become wedged. There is evidence that early treatment with corrective orthotics may reverse this deformity and the recommendation is for brace initiation if the fixed kyphosis is greater than 30°, the anterior vertebral wedging increases, or there is posterior apical displacement [18]. Significant thoracolumbar kyphosis will cause a compensatory increase in the already exaggerated lumbar lordosis and my aggravate symptoms of spinal stenosis.

Injury prevention in this group will include activities that minimize lumbar lordosis. This will include core strengthening and lumbodorsal stretches. Hip flexion contractures contribute to the increased lumbar lordosis and should be treated as well.

Hypochondrodysplasia

This is another FGFR-3 related condition with a similar but less severe clinical presentation than achondroplasia. There is a greater variability in

the causative mutation and accordingly a greater heterogeneity in the clinical characteristics. The individuals have a short stature with rhizomelic limb shortening, increased lumbar lordosis, and narrowed interpedicular distances in the lumbar spine [6].

Pseudoachondroplasia

This autosomal dominant condition is caused by a defect of the oligomeric matrix protein in cartilage. The appearance is similar to achondroplasia with disproportionate short stature and rhizomelic limb shortening. The diagnosis is often delayed due to the mildness deformity and the absence of other distinguishing characteristics. Joint laxity is a common finding [28].

The spinal stenosis so characteristic of achondroplasia is not present, but instability and deformity may be encountered. Here, there is atlantoaxial instability and thoracolumbar kyphosis. Unlike the severe apical wedged vertebra found in achondroplasia, the kyphosis is gradual with mild vertebral wedging over a number of segments. Increased lumbar lordosis is present and may be related to hip flexion contractures. In the absence of spinal stenosis, this deformity is usually not problematic. There may also be thoracic hyperkyphosis [28].

Spondyloepiphyseal Dysplasia

Both a congenital and late or tarda form have been described. The congenital variety is inherited as an autosomal dominant, is caused by defective procollagen type 2 subunits, and presents as severe dwarfism easily recognized at birth [28]. The shortened spine is caused by the severe platyspondyly and the shortened limbs by the epiphyseal and physeal changes. There are angular deformities of the lower extremities. Importantly, atlantoaxial instability, caused by odontoid hypoplasia and ligamentous laxity, is frequent and myelopathy has been reported in 42% of patients.[11, 15]. An increased lumbar lordosis may be associated with hip flexion contractures.

The tarda variety is inherited as X-linked recessive with male only involvement. These individuals have far less obvious characteristics and the diagnosis may not be made until late, often when symptoms of hip pain bring the musculoskeletal system to attention. The final height may be just over 5 ft. There may be increased lumbar lordosis due to hip flexion contractures and back pain due to, perhaps, apophyseal changes in the vertebral bodies.

Mucopolysaccharidosis

The mucopolysaccharidosis comprises a group of disorders linked by their common pathophysiology, an enzymatic defect that leads to an accumulation of metabolites. Over time, these substances produce clinically significant problems not initially appreciated. There are seven distinct types defined by the specific enzymatic defect. The phenotypes vary significantly both between the different groups as well as within each type, and the spectrum of clinical problems ranges from early death to survival well into adulthood. The typical spinal problems are atlantoaxial instability and thoracolumbar kyphosis. Enzyme replacement therapy represents a promising future treatment option [8, 25].

Mucoplysaccharidosis IV (MPS IV), or Morquio syndrome, is the most common form. These children have normal intelligence and longevity. It is an autosomal recessive lysosomal storage disease with intracellular accumulation of glycosaminoglycans. There are three subtypes based on the specific enzymatic deficiency. The clinical presentation is short trunk disproportionate short stature, progressive bony deformities, pectus carinatum, corneal clouding, and aortic valve disease [16].

The primary spine problem is the very common C1/2 instability due to odontoid aplasia or hypoplasia. There is also glycosaminoglycan deposition in the extradural space of the upper cervical canal, and the combination of the stenosis produced by this deposition and instability leads to myelopathy so frequently that prophylactic decompression and fusion have been suggested

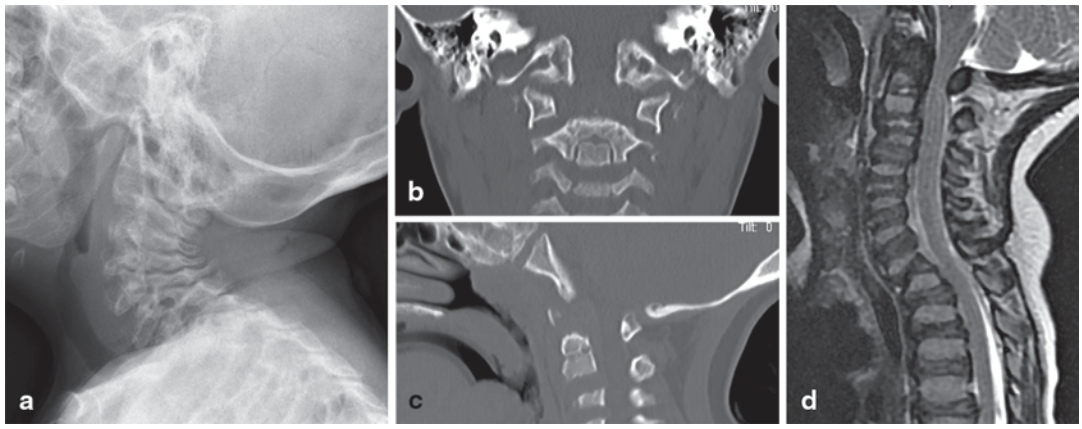

Fig. 22.3 Atlantoaxial instability and stenosis occur frequently in spondyloepiphyseal dysplasia. The pathology may be difficult to visualize on plane radiographs (**a**). Here, the area is compromised by instability caused by odontoid hypoplasia noted best on the CT scan (**b**, **c**), and stenosis secondary to deposits of metabolites in the spinal canal as noted on the MRI (**d**)

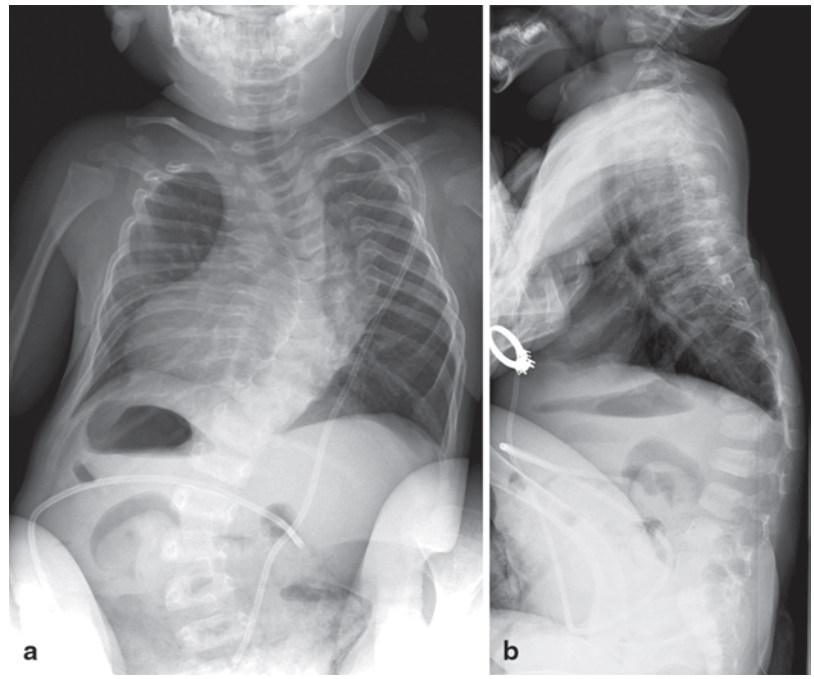

Fig. 22.4 By age 2, this boy with severe osteogenesis imperfecta has already developed a significant scoliosis (**a**). The soft bones also deform into the noted thoracolumbar kyphosis (**b**)

[4, 13] (Fig. 22.3). An increase in the thoracic kyphosis and lumbar lordosis also occurs.

Osteogenesis Imperfecta

This is a heterogeneous group of diseases characterized by susceptibility to fractures and presumed or proven defects in collagen I biosynthesis [27]. The various forms have been classified into seven or eight types based on the mode of inheritance, clinical characteristics, and collagen abnormality [5, 20]. There is a dramatic variability in the clinical consequences. Type II is incompatible with life while type I is associated with an occasional fracture. The more severe forms present with variable combinations of short stature, blue sclerae, abnormal teeth, deafness, frequent

fractures, and ligamentous laxity. Biphosphonate treatment does reduce the fracture risk and improves participation in activities of daily living [9, 12].

While long bone fractures are responsible for the most common morbidities, the spine is not spared. Scoliosis occurs frequently The incidence of scoliosis has been reported to be 26 % in involved children under 5 year of age and 80 % in those over 12 years [21, 22] (Fig. 22.4). Presumably, a combination of ligamentous laxity and weakened bones is responsible for the deformity. The more severe the disease, the greater the incidence of the deformity [1, 2]. The spine is also at increased risk for spondyolisthesis [10].

Deformity at the craniovertebral junction occurs infrequenty but may be catastrophic [19, 29]. The softened bones permit a secondary basilar impression with invagination of the foramen magnum into the posterior fossae. Hydrocephalus, cranial nerve abnormality, long tract signs, and respiratory depression may occur.

Activity must clearly be titrated based on the severity of the condition, and the sports physician should be mindful that some children with the milder forms of oteogenesis imperfecta (O.I.) will be undiagnosed. When a fracture occurs after a seemingly mild traumatic event, the differential diagnosis should include O.I.

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