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.

P. d'Hemecourt (✉) · M. O'Brien
Boston Children's Hospital, Division of Sports Medicine, Boston, Massachusetts, USA
e-mail: Pierre.D'Hemecourt@childrens.harvard.edu

M. O'Brien
e-mail: Michael.obrien@childrens.harvard.edu

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].

L. Micheli et al. (eds.), *Spinal Injuries and Conditions in Young Athletes,* Contemporary Pediatric and Adolescent Sports Medicine, DOI 10.1007/978-1-4614-4753-5_6,

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.

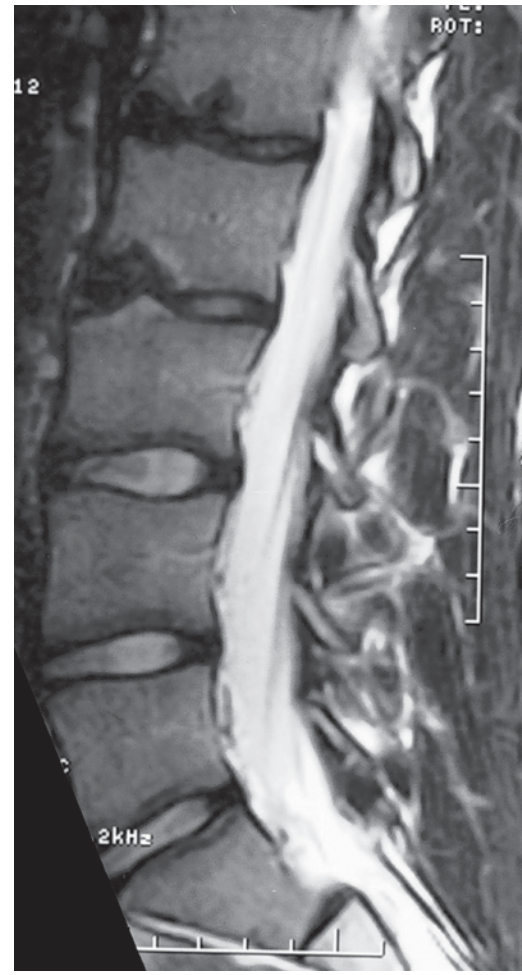

Fig. 6.1 Lumbar Scheuermann's

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-

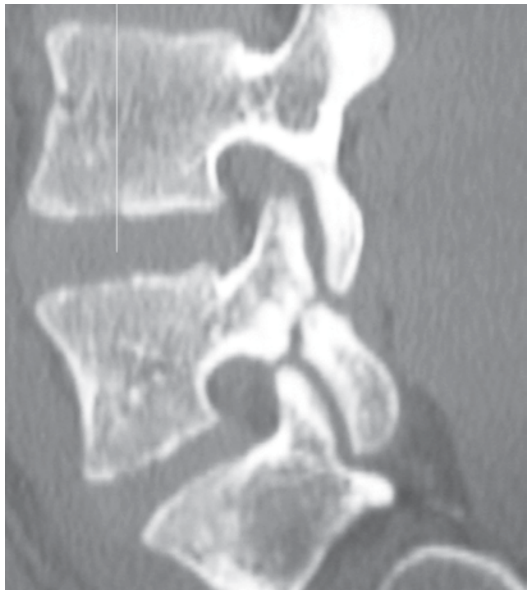

Fig. 6.2 Spondylolysis

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.

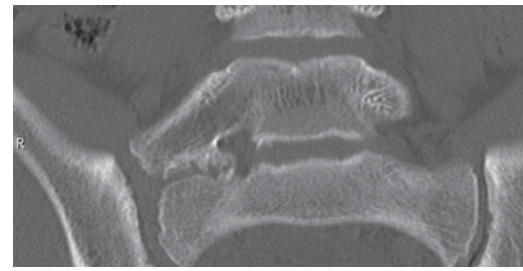

Fig. 6.3 Transitional pseudarthrosis

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.

References

1. Mirovsky Y, Jakim I, Halperin N, et al. Non-specific back pain in children and adolescents: a prospective study until maturity. J Pediatr Orthop. 2002;11:275–8.
2. King HA. Back pain in children. Pediatr Clin North Am. 1984;31(5):1083–95.
3. d'Hemecourt PA, Gerbino PG II, Micheli LJ. Back injuries in the young athlete. Clin Sports Med. 2000;19(4):663–79.
4. Hutchinson MR. Low back pain in elite rhythmic gymnasts. Med Sci Sports Exerc. 1999;31(11):1686–88.
5. National Council of Youth Sports. Report on Trends and Participation in Organized Youth Sports. http://www.ncys.org/publications/2008-sports-participation-study.php.
6. Wojts EEM, Ashton-Miller JA, Huston LJ, et al. The association between athletic training time and the sagittal curvature of the immature spine. Am J Sports Med. 2000;28(4):490–8.
7. Krabbe S, Christiansen C. Effects of Puberty on rates of bone growth and mineralisation. with observations in male delayed puberty. Arch Dis Child. 1979;54:950–3.
8. Bailey DA, Wedge JH, McCulloch RG, Martin AD, Bernhardson SC. Epidemiology of fractures of the distal end of the radius in children as associated with growth. J Bone Joint Surg. 1989;71A:1225–31.
9. Nadler SF, et al. Hip muscle imbalance and low back pain in athletes: influence of core strengthening. Med Sci Sports Exerc. 2002;34(1):9–16.
10. Nadler SF, et al. Relationship between hip muscle imbalance and occurrence of low back pain in collegiate athletes: a prospective study. Am J Phys Med Rehabil. 2001;80(8):572–7.
11. Kujala UM, et al. Lumbar mobility and low back pain during adolescence. A longitudinal three-year follow-up study in athletes and controls. Am J Sports Med. 1997;25(3):363–8.
12. Sjolie A, Ljunggren A. The significance of high lumbar mobility and low lumbar strength for current and future low back pain in adolescents. Spine. 2001;26(23):2629–36.

13. D'Hemecourt PA, Gerbino PG II, Micheli LJ. Back injuries in the young athlete. Clin Sports Med. 2000;19(4):663–79.
14. Loud KJ, Micheli LJ. Common athletic injuries in adolescent girls. Curr Opin Pediatr. 2001;13(4):317–22.
15. McTimoney CA, Micheli LJ. Managing back pain in young athletes. J Musc Med. 2004;21(2):63–9.
16. Omey ML, Michelu LJ, Gerbino PG II. Idiopathic scoliosis and spondylolysis in the female athlete: tips for treatment. Clin Orthop Relat Res. 2000;372:74–84.
17. Rhee JM, Schaufele M, Abdu WA. Radiculopathy and the herniated lumbar disc. Controversies regarding pathophysiology and management. Bone Joint Surg Am. 2006;88(9):2070–80.
18. Buckwalter JA. Aging and degeneration of the human intervertebral disc. Spine. 1995;20(11):1307–14.
19. Boos N, Weissbach S, Rohrbach H, Weiler C, Spratt KF, Nerlich AG. Classification of age-related changes in lumbar intervertebral discs: 2002 Volvo Award in basic science. Spine. 2002;27(23):2631–44.
20. Bogduk N. The innervation of the lumbar spine. Spine. 1983;8(3):286–93.
21. Crock HV. Internal disc disruption. A challenge to disc prolapse fifty years on. Spine. 1986;11(6):650–3.
22. Coppes MH, Marani E, Thomeer RT, Groen GJ. Innervation of "painful" lumbar discs. Spine. 1997;22(20):2342–9.
23. Brown MF, Hukkanen MV, McCarthy ID, et al. Sensory and sympathetic innervation of the vertebral endplate in patients with degenerative disc disease. J Bone Joint Surg Br. 1997;79(1):147–53.
24. Saal JS, Franson RC, Dobrow R, Saal JA, White AH, Goldthwaite N. High levels of inflammatory phospholipase A2 activity in lumbar disc herniations. Spine. 1990;15(7):674–8.
25. Weiler C, Nerlich AG, Bachmeier BE, Boos N. Expression and distribution of tumor necrosis factor alpha in human lumbar intervertebral discs: a study in surgical specimen and autopsy controls. Spine. 2005;30(1):44–53; discussion 54.
26. Kirkaldy-Willis WH, Wedge JH, Yong-Hing K, Reilly J. Pathology and pathogenesis of lumbar spondylosis and stenosis. Spine. 1978;3(4):319–28.
27. Panjabi MM. The stabilizing system of the spine. Part I. Function, dysfunction, adaptation, and enhancement. J Spinal Disord. 1992;5(4):383–9; discussion 397.
28. Mautner KR, Huggins MJ. The young adult spine in sports. Clin Sports Med. 2012;31(3):453–72.
29. McCarron RF, Wimpee MW, Hudkins PG, Laros GS. The inflammatory effect of nucleus pulposus. A possible element in the pathogenesis of low-back pain. Spine. 1987;12(8):760–4.
30. Baker RJ, Patel D. Lower back pain in the athlete: common conditions and treatment. Prim care. 2005;32(1):201–29.
31. Webb JH, Svien HJ, Kennedy RL. Protruded lumbar intervertebral disks in children. J Am Med Assoc. 1954;154(14):1153–4.
32. Micheli LJ, Wood R. Back pain in young athletes. Significant differences from adults in causes and patterns. Arch Pediatr Adolesc Med. 1995;199:15–8.
33. McCormack RG, McLean N, Dasilva J, Fisher CG, Dvorak MF. Thoraco-lumbar flexion-distraction injury in a competitive gymnast: a case report. Clin J of Sport Med. 2006;16(4):369–71.
34. Hosea T, Hannafin J, Bran J, O'Hara D, Seuffert P. Aetiology of low back pain in young athletes: role of sport type. BJSM. 2011;45(4):352.
35. Barr KP. Review of upper and lower extremity musculoskeletal pain problems. Phys Med Rehabil clin N Am. 2007;18(4):747–60, vi–vii.
36. Gaunt A, Herring S, O'connor F. Caring for patients who have acute and subactute low back pain. CME bulletin. 2008;7(2):1–8.
37. Lawrence JP, Greene HS, Grauer JN. Back pain in athletes. J Am Acad Orthop Surg. 2006;14(13):726–35.
38. Chou R, Qaseem A, Snow V, et al. Diagnosis and treatment of low back pain: a joint clinical practice guideline from the American College of Physicians and the American Pain Society. Ann Intern Med. 2007;147(7):478–91.
39. Chou R, Fu R, Carrino JA, Deyo RA. Imaging strategies for low-back pain: systematic review and meta-analysis. Lancet. 2009;373(9662):463–72.
40. Espeland A, Baerheim A, Albrektsen G, Korsbrekke K, Larsen JL. Patients' views on importance and usefulness of plain radiography for low back pain. Spine. 2001;26(12):1356–63.
41. Kendrick D, Fielding K, Bentley E, Kerslake R, Miller P, Pringle M. Radiography of the lumbar spine in primary care patients with low back pain: randomised controlled trial. BMJ. 2001;322(7283):400–5.
42. Jarvik JG. Imaging of adults with low back pain in the primary care setting. Neuroimaging Clin N Am. 2003;13(2):293–305.
43. Roudsari B, Jarvik JG. Lumbar spine MRI for low back pain: indications and yield. Am J Roentgenol. 2010;195(3):550–9.
44. Boden SD, McCowin PR, Davis DO, Dina TS, Mark AS, Wiesel S. Abnormal magnetic-resonance scans of the cervical spine 2 asymptomatic subjects. A prospective investigation. J Bone Joint Surg Am. 1990;72(8):1178–84.
45. Jensen MC, Brant-Zawadzki MN, Obuchowski N, Modic MT, Malkasian D, Ross JS. Magnetic resonance imaging of the lumbar spine in people without back pain. N Engl J Med. 1994;331(2):69–73.
46. Weishaupt D, Zanetti M, Hodler J, Boos N. MR imaging of the lumbar spine. prevalence of intervertebral disk extrusion and sequestration, nerve root compression, end plate abnormalities, and osteoarthritis of the facet joints in asymptomatic volunteers. Radiology. 1998;209(3):661–6.
47. Modic MT, Obuchowski NA, Ross JS, et al. Acute low back pain and radiculopathy: MR imaging findings and their prognostic role and effect on outcome. Radiology. 2005;237(2):597–604.

48. Dang L, Liu Z. A review of current treatment for lumbar disc herniation in children and adolescents. Eur Spine J. 2010;19(2):205–14.
49. Goel VK, Kong W, Han JS, Weinstein JN, Gilbertson LG. A combined finite element and optimization investigation of lumbar spine mechanics with and without muscles. Spine. 1993;18(11):1531–41.
50. Wilke HJ, Wolf S, Claes LE, Arand M, Wiesend A. Stability increase of the lumbar spine with different muscle groups. A biomechanical in vitro study. Spine. 1995;20(2):192–8.
51. Hides JA, Jull GA, Richardson CA. Long-term effects of specific stabilizing exercises for first-episode low back pain. Spine. 2001;26(11):E243–E8.
52. Hides JA, Richardson CA, Jull GA. Multifidus muscle recovery is not automatic after resolution of acute, first-episode low back pain. Spine. 1996;21(23):2763–69.
53. Ferreira PH, Ferreira ML, Maher CG, Herbert RD, Refshauge K. Specific stabilisation exercise for spinal and pelvic pain: a systematic review. Aust J physiother. 2006;52(2):79–88.
54. Manchikanti L, Cash KA, McManus CD, Pampati V, Smith HS. Preliminary results of a randomized, equivalence trial of fluoroscopic caudal epidural injections in managing chronic low back pain: Part 1–Discogenic pain without disc herniation or radiculitis. Pain Physician. 2008;11(6):785–800.
55. Buttermann GR. The effect of spinal steroid injections for degenerative disc disease. Spine. 2004;4(5): 495–505.
56. Cahill KS, Dunn I, Gunnarsson T, Proctor MR. Lumbar microdiscectomy in pediatric patients: a large single-institution series. J Neurosurg Spine. 2010;12(2):165–70.
57. Iwamoto J, Sato Y, Takeda T, Matsumoto H. Return to sports activity by athletes after treatment of spondylolysis. World J Orthop. 2010;1(1):26–30.
58. Iwamoto J, Takeda T, Sato Y, Wakano K. Short-term outcome of conservative treatment in athletes with symptomatic lumbar disc herniation. Am J Phys Med Rehabil/ Association of Academic Physiatrists. 2006;85(8):667–74; quiz 675–7.
59. Young WK, d'Hemecourt PA. Back pain in adolescent athletes. Phys Sportsmed. 2011;39(4):80–9.
60. Sward L, Hellstrom M, Jacobsson B, Karlsson L. Vertebral ring apophysis 2 in athletes. Is the etiology different in the thoracic and lumbar spine? Am J Sports Med. 1993;21(6):841–5.
61. Blumenthal SL, Roach J, Herring JA. Lumbar Scheuermann's. A clinical series and classification. Spine. 1987;12(9):929–32.
62. Bick EM, Copel JW. The ring apophysis of the human vertebra; contribution to human osteogeny. II. J Bone Joint Surg Am. 1951;33-A(3):783–7.
63. Bick EM, Copel JW. Longitudinal growth of the human vertebra; a contribution to human osteogeny. J Bone Joint Surg Am. 1950;32(A:4):803–14.
64. Salter RB, Harris WR. Injuries Involving the Epiphyseal Plate. J Bone Joint Surg. 1963;45(3):587–622.
65. Begg AC. Nuclear herniations of the intervertebral disc; their radiological manifestations and significance. J Bone Joint Surg Am Br. 1954;36-B(2):180–93.
66. Greene TL, Hensinger RN, Hunter LY. Back pain and vertebral changes simulating Scheuermann's disease. J Ped Orthop. 1985;5(1):1–7.
67. Kozlowski K. Anterior intervertebral disc 2 in children. Report of four cases. Pediatr Radiol. 1977;6(1):32–5.
68. McCall IW, Park WM, O'Brien JP, Seal V. Acute traumatic intraosseous disc herniation. Spine. 1985;10(2):134–7.
69. Schmorl G, Junghanns, H. The Human Spine in Health and Disease. 2nd ed. New York; 1971.
70. White A, Panjabi, MM. Clinical Biomechanics of the Spine. Philadephia: JB Lippincott Company; 1978. 61–90.
71. Gurd DP. Back pain in the young athlete. Sports Med Arthroso. 2011;19(1):7–16.
72. Chang CH, Lee ZL, Chen WJ, Tan CF, Chen LH. Clinical significance of ring apophysis fracture in adolescent lumbar disc herniation. Spine. 15 2008;33(16):1750–4.
73. Blumenthal SL, Roach J, Herring JA. Lumbar Scheuermann's. A clinical series and classification. Spine (Phila Pa 1976). 1987;12(9):929–32.
74. Goldstein JD, Berger PE, Windler GE, Jackson DW. Spine injuries in 2 and swimmers. An epidemiologic investigation. Am J Sports Med. 1991;19(5):463–8.
75. Labelle H, Roussouly P, Berthonnaud E, Dimnet J, O'Brien M. The importance of spino-pelvic balance in L5-S1 developmental spondylolisthesis: A review of pertinent radiologic measurements. Spine 2005;30 (6, suppl): S. 27–34.
76. Morita T, Ikata T, Katoh S, Miyake R. Lumbar spondylolysis in children and adolescents. J Bone Joint Surg Br. 1995;77(4):620–5.
77. Beutler WJ, Fredrickson BE, Murtland A, Sweeney CA, Grant WD, Baker D. The natural history of spondylolysis and spondylolistheis: 45-year follow-up evaluation. Spine. 2003;28(10):1027–35.
78. Klein G, Mehlman CT, McCarty M. Nonoperative treatment of spondylolysis and grade I spondylolisthesis in children and young adults: a meta-analysis of observational studies. J Pediatr Orthop. 2009;29(2):146–56.
79. McPhee IB, O'Brien JP, McCall IW, Park WM. Progression of lumbosacral spondylolisthesis. Australas Radiol. 1981;25:91–5.
80. Bellah RD, Summerville DA, Treves ST, Micheli LJ. Low-back pain in adolescent athletes. Detection of stress injury to the pars interarticularis with SPECT. Radiology. 1991;180(2):509–12.
81. Campbell RS, Grainger AJ, Hide IG, Papastefanou S, Greenough CG. Juvenile spondylolysis: a comparative analysis of CT, SPECT and MRI. Skeletal Radiol. 2005;34(2):63–73.

82. Ganiyusufoglu AK, Onat L, Karatoprak O, Enercan M, Hamzaoglu A. Diagnostic accuracy of magnetic resonance imaging versus computed tomography in stress fractures of the lumbar spine. Clin Radiol. 2010;65(11):902–7.
83. Morita T, Ikata T, Katoh S, Miyake R. Lumbar spondylolysis in children and adolescents. J Bone Joint Surg Br. 1995;77(4):620–5.
84. Cohen E, Stuecker RD. Magnetic resonance imaging in diagnosis and follow-up of impending spondylolysis in children and adolescents. Early treatment may prevent pars defects. J Pediatr Orthop B. 2005;14(2):63–7.
85. d'Hemecourt PA, Zurakowski D, Kriemler S, Micheli LJ. Spondylolysis. returning the athlete to sports participation with brace treatment. Orthopedics. 2002;25(6): 653–7.
86. Sairyo K, Sakai T, Yasui N, Dezawa A. Conservative treatment for pediatric lumbar spondylolysis to achieve bone healing using a hardbrace: what type and how long? J Neurosurg Spine. 2012 Jun;16(6):610–4.
87. Sairyo K, Sakai T, Yasui N. Conservative treatment of lumbar spondylolysis in childhood and adolescence: the radiological signs which predict healing. J Bone Joint Surg Br. 2009 Feb;91(2):206–9.
88. Fellander-Tsai, L. and Micheli, L. Treatment of Spondylolysis with External Electrical Stimulator and Bracing in Adolescent Athletes. Clin J. Sport Med. 1998;8:232–42.
89. Mannor DA, Lindenfeld TN. Spinal process apophysitis mimics spondylolysis. Case reports. Am J Sports Med. Mar-Apr. 2000;28(2):257–60.
90. Castellvi AE, Goldstein LA, Chan DP. Lumbosacral transitional vertebrae and their relationship with lumbar extradural defects. Spine (Phila Pa 1976). 1984;9(5):493–5.
91. Connolly LP, d'Hemecourt PA, Connolly SA, Drubach LA, Micheli LJ, Treves ST. Skeletal scintigraphy of young patients with low-back pain and a lumbosacral transitional vertebra. J Nucl Med. 2003;44(6):909–14.
92. Vergauwen S, Parizel PM, van Breusegem L, et al. Distribution and incidence of degenerative spine changes in patients with a lumbo-sacral transitional vertebra. Eur Spine J. 1997;6(3):168–72.
93. Quinlan JF, Duke D, Eustace S. Bertolotti's syndrome. A cause of back pain in young people. J Bone Joint Surg Br. 2006;88(9):1183–6.
94. Greenman PE. Clinical aspects of sacroiliac function in human walking. In: Vleeming A, ed. First interdisciplinary world congress on low back pain and its relation to the sacroiliac joint. 1992: San Diego: Churchill-Livingstone; 1992;353–9.
95. Vleeming A, Stoeckar R. The role of pelvic girdle in coupling the spine and legs: A clinical anatomical perspective on pelvic stability. In: Vleeming A, Mooney V, Stoeckart R, eds. Movement, stability and lumbopelvic pain: integration of research and therapy. New York: Churchill Livingstone; 2007:IX, 658.