SPONDYLOLYSIS AND SPONDYLOLISTHESIS: A NARRATIVE REVIEW OF ETIOLOGY, DIAGNOSIS, AND CONSERVATIVE MANAGEMENT

DANIEL W. HAUN, DC[a], NORMAN W. KETTNER, DC[b]

[a]*Resident, Department of Radiology, Logan College of Chiropractic, Chesterfield, MO.* [b]*Chairman, Department of Radiology, Logan College of Chiropractic.*
Submit requests for reprints to: Dr. Norman Kettner, Logan College of Chiropractic, Department of Radiology, PO Box 1065, 1851 Schoettler Road, Chesterfield, Missouri 63006–1065. Email: norman.kettner@logan.edu, Phone: (636) 227-2100 (x1830), Fax: (636) 207-2429
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ABSTRACT

Objective: To review current literature regarding the etiology, diagnosis, and conservative treatment of spondylolysis and spondylolytic spondylolisthesis.

Methods: The PubMed database was searched for articles on spondylolysis and/or spondylolisthesis and their incidence, diagnosis, imaging, treatment, and prognosis. The bibliographies of articles determined to be relevant were also reviewed.

Results: A PubMed search of spondylolysis or spondylolisthesis yielded over 800 citations. Sixty-eight articles were selected based on an opinion of perceived relevance to the subjects of spondylolysis and spondylolisthesis.

Conclusions: Spondylolysis affects approximately 6% of the population. The lesion likely represents a stress fracture and the typical age of onset is early childhood and adolescence. Most individuals are asymptomatic. Adolescents with low back pain may have an impending or new pars defect. A high index of suspicion for a new pars defect should prompt utilization of physiologic imaging to determine the likelihood of pars union in young patients. Restrictive bracing may lead to healing of the fracture and cessation of pain. Spondylolisthesis is a common complication of spondylolysis. Spondylolisthesis progression is typically small and most likely in young individuals. Significant progression in adults is rare. The finding of spondylolysis and spondylolisthesis in an adult patient is usually incidental and not likely to be a direct source of pain unless there is concurrent instability. (J Chiropr Med 2005;4:206–217)

Key Indexing Terms: Spondylolysis; Spondylolisthesis; Radiography; Chiropractic

INTRODUCTION

Most primary care clinicians would agree that low back pain is a common presenting complaint and that spondylolysis is a fairly common radiographic finding in their low back pain patients. But, despite the frequency of the finding, there still seems to be confusion regarding this entity and its significance. This article addresses the populations at risk for spondylolysis, when and how the lysis occurs, the clinical significance of spondylolysis, and the optimal diagnostic tests to diagnose and determine clinical relevance. Treatment issues are also discussed.

METHODS

The PubMed database was searched for articles on spondylolysis and/or spondylolisthesis and their incidence, diagnosis, imaging, treatment, and prognosis. The bibliographies of articles determined to be relevant were also reviewed.

RESULTS

The search yielded over 800 citations. Sixty-eight articles were selected by the authors based on an opinion of perceived relevance to the subjects of spondylolysis and spondylolisthesis.

Definition

Spondylolysis is defined as an osseous defect in the pars interarticularis of a vertebral arch. When this defect is bilateral, the anterior and posterior portion of the neural arch separate. The inferior articular processes, lamina, and spinous process no longer remain connected to the superior articular processes, pedicles, and vertebral body (Fig 1). Because

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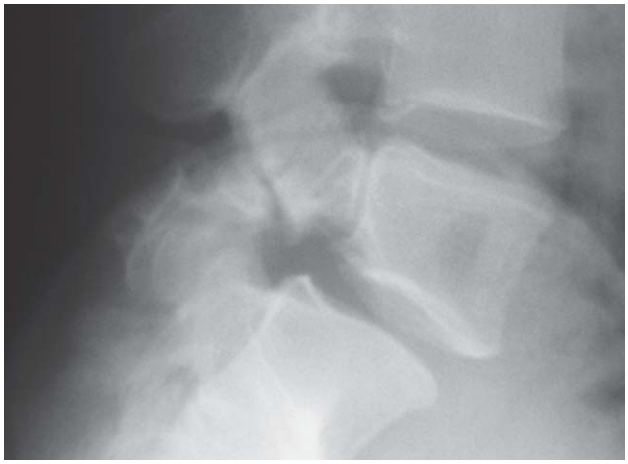

Figure 1. A spot lateral radiograph of L5/S1 demonstrates clear evidence of spondylolysis. The vertebral body, pedicles, and superior articular facets are separated from the inferior articular facets and spinous process.

of the discontinuity within the neural arch, a common complication is spondylolisthesis. In the strictest sense, spondylolisthesis means that there is movement or translation of the vertebral body in relation to an adjacent vertebra. This could mean forward, backward, or sideways. But, an anterolisthesis, or forward movement, is generally implied by this term.

Spondylolisthesis has a number of etiologies. Classification of spondylolisthesis has traditionally used the system proposed by Wiltse et al.[1] A different classification system has recently been proposed by Marchetti and Bartolozzi.[2] This new system differentiates developmental spondylolisthesis with lysis and acquired spondylolytic spondylolisthesis. This was done to remedy confusion over natural history and treatment, and that these fell under the same category in the prior classification system.

Another classification system has been proposed by Herman and Pizzutillo.[3] This classification system applies only to children and adolescents. It uses elements of both the Wiltse and Marchetti-Bartolozzi classifications. It separates out subjects that have an incidental finding of spondylolysis from those with a history of back pain, sports participation, and spondylolysis. The authors suggest that a child or adolescent without back pain and with spondylolysis was predisposed genetically to pars failure, citing the fact that a history of spondylolysis has been shown in 26% of first degree relatives. This group does not need athletic participation to cause stress injury, only normal daily stresses. Four percent of children and 8% of adults may be classified into this category.

Etiology and Pathogenesis

Analysis of the general adult population shows spondylolysis is present in approximately 3–6% of individuals.[4–6] This lytic defect is almost always at the L5 level.[5–7] Caucasians are affected about 3 times more frequently than African Americans.[6] Males are twice as likely as females to be affected.[4,7,8] Radiographic studies of newborns have shown the incidence to be 0%.[4] Therefore, this is clearly not a congenital defect. In a study of 500 first grade children, the prevalence of spondylolysis was 4.4%. When the subjects were followed to adulthood, the incidence rose to 6%.[4,5] Therefore, two-thirds of adults with spondylolysis developed it prior to reaching school age. The remaining third acquired the defect at some point during childhood or adolescence.

There seems to be some degree of genetic predisposition to the development of spondylolysis. In the same study of school age children previously mentioned, radiographs were taken of the family members of children with spondylolysis. There was an increased prevalence of the finding within these families. Fathers were affected 32% of the time, mothers 17%, and male siblings 34%.[5] In another study of first degree relatives of individuals with spondylolysis, it was found that defects were also present in 16% of parents, 14% of siblings, and 16% of children.[8] Studies of Eskimo populations, which could be considered as one large genetic pool due to their isolation, showed spondylolysis prevalence between 17–54%.[9–11] Lifestyle also plays a part. Eskimos living in an urban setting show a lower incidence of lysis than those in a rural (hunter/gatherer) setting.[9] Furthermore, a genetically determined bone density could explain why spondylolysis is less frequent in the African American population. Bone density is higher in African Americans than Caucasians, and a higher cortical bone density within the pars interarticularis has been shown to offer a degree of protection against spondylolysis.[12–14] Whitesides et al[15] studied specific radiographic parameters measured in archeological skeletal remains in two genetically and geographically distinct groups. The tilt of the sacral base relative to a line along the posterior sacrum (sacral table angle) was found to vary between genetically distinct groups. The lower the sacral table angle, the

higher the rate of spondylolysis. Thus, genetic differences in the morphology of the sacrum may play a role in spondylolysis.[15]

Aside from genetic predisposition, certain other groups of people have a higher incidence of spondylolysis than the population at large, presumably due to higher levels of physical stress. The incidence of spondylolysis has been documented to be significantly higher in athletes involved in sports such as diving (63%), weight lifting (36%), wrestling (33%), and gymnastics (33%).[6] The numbers do not include asymptomatic patients with lysis, since the study population presented with back pain prior to radiography. Similar associations between increased spondylolysis incidence and football have been found.[16]

The most accepted theory is that spondylolysis is a stress or fatigue fracture.[1,17,18] A study of the biomechanical loading in the lumbar spine showed that the point of highest stress within a vertebral segment is found within the pars interarticularis.[18] This correlates well with the concept of spondylolysis as a fatigue fracture. While an acute fracture of the pars is possible and documented, most individuals with spondylolysis are unable to identify any particular traumatic incident that could have been capable of causing this fracture. Also, in consideration of the higher incidence of spondylolysis within certain athletic endeavors, a stress fracture etiology seems most likely. These individuals are repeatedly loading the spine in a manner greater than the average person, but without a specific significant trauma. The proposed movements that cause a stress (fatigue) fracture are typically repeated hyperextension of the lumbar spine. Repeated alternating flexion and extension have also been proposed as a plausable mechanism.[19]

The rarity and unique characteristics of an acute traumatic spondylolysis rather than one resulting from a repetitive stress etiology is evidenced in a study by Hilibrand et al.[20] In reviewing ten years of records from the University of Michigan Hospitals, they were able to find only 7 cases of acute traumatic lumbar spondylolisthesis. Of those, only 5 were due to a fracture in the pars. Those 5 patients all presented with significant injury and frequently had multiple fractures in other locations. Several of these patients had or developed neurological deficits as a result of their injury, and these patients had a greater tendency for progression of their spondylolisthesis. The increased tendency for progression of the slip is likely due to disruption of the supporting ligamentous structures in addition to fracture, a feature not found with stress etiologies.

CLINICAL PRESENTATION

The majority of individuals with spondylolysis (at least 2/3) developed it as a young child. Those children are usually asymptomatic. The remaining one-third that develop a pars defect during adolescence or early adulthood have symptoms during and around the time of its development.[5] In a study of inheritance and spondylolysis, only 4% of relatives of people with spondylolysis admitted to having any symptoms, but 19% were found to have a defect.[8] Thus, the majority of affected individuals are asymptomatic, displaying clinically inactive spondylolysis. It is not clear, even with these individuals, if the pain is actually due to the spondylolysis. The individuals most likely to have a symptomatic spondylolysis are adolescents that are developing a defect. As will be discussed in detail later, patients with unstable spondylolysis may be more prone to low back pain.

Diagnosis

Plain film radiography is the most commonly used method of detection and documentation of a spondylolysis. Radiographically, a spondylolysis will be seen as a linear radiolucency in the region of the pars. Typically, lumbar oblique views are utilized to visualize a pars defect. A study of the orientation of pars defects showed that the majority of defects do not lie perpendicular to the pars.[21] The majority of pars defects were found to lie within 0–30 degrees of the coronal plane, thereby making a lateral projection (particularly a coned lateral spot view) the most likely view to visualize the defect. Other authors have reached similar conclusions. Amato et al[22] found the most useful projection to be a spot lateral, showing 84% of the defects. Most lesions were not located in a plane that will be visualized by a 45-degree oblique radiographic position. Only 32% of spondylolytic defects were within 15 degrees of the 45-degree plane, thereby decreasing the likelihood of being seen in this projection. Only 19% of defects were seen on the oblique projection alone.[21] It must be noted that many defects are seen on both lateral and oblique views. This may be explained by two hypotheses. First, some pars defects are curved, and thus lie in multiple anatomical

planes. Second, a wide defect will be more easily visualized on both lateral and oblique views.[21]

Regardless of the orientation of the defect or the radiographic views, the greater the degree of anterolisthesis, the easier it is to see the lesion. A wide fracture line is easier to see than a thin, nondisplaced one. The lateral projection is optimal for documenting a lytic defect in the pars, but a Mach line can mimic a real defect. On the lateral projection, the S1 facets can, at times, project over the region of the pars. Due to the cortical density from the facet, a lucency can appear in the pars that is a summation artifact simulating fracture.

The lateral radiograph is also most useful for the documentation of spondylolisthesis. The degree of anterolisthesis is measured by varied methods. One method is to measure the distance from the posterior cortex of the S1 body to posterior cortex of the L5 vertebral body. This will result in a measurement of the amount of anterolisthesis. Simply reporting the amount of anterolisthesis as a direct measure is discouraged. Comparison with previous or subsequent radiographs cannot accurately be made with this type of mensuration because variations in focal-film or object-film distance will cause magnification errors. There are two commonly accepted mensuration methods. The first is to report anterolisthesis as a Grade I, II, III, IV, or V (known as the Meyerding classification). Grade I is an anterolisthesis from 0–25%. Grade II is 26–50%, and so on. The second is to simply report the exact percentage of slip (14%, 23%, 47%, etc.). This is more precise and more valuable for comparison over time if only radiology reports (not films) are available. Two sets of radiographs taken a few years apart may both report a Grade I slip and it would appear there was no progression over time. But, the first set could have shown a 6% anterolisthesis while the second 24%. Clearly this is a progression, but it would not be obvious by reporting the grade only.

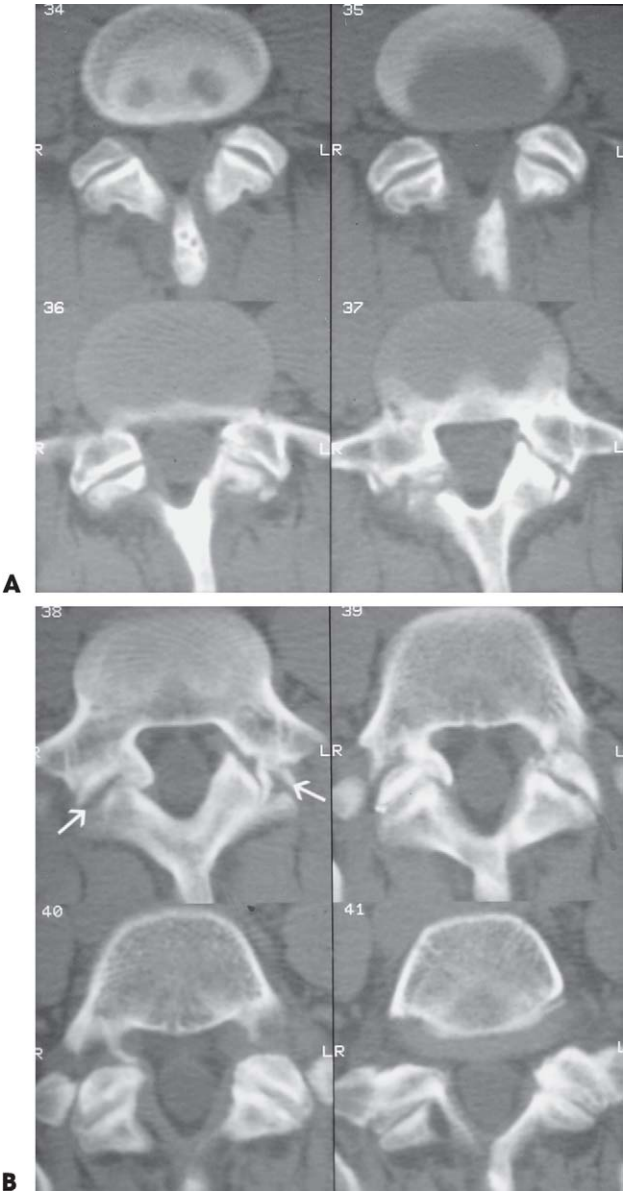

Figure 2 A and B. Axial CT bone window of L5. A and B represent progressively caudal slices. Note in all slices there is a lucency somewhat resembling an apophyseal joint. These lucencies represent bilateral spondylolytic defects (arrows).

Computed tomography (CT) examination may also be employed to better visualize a pars defect. If what appears to be an apophyseal joint is seen at the level of the pedicles, then that may represent a pars defect. (Figure 2) Sagittal reformatting can be helpful. Also, when CT scanning is done to document spondylolysis, a reverse gantry angle (gantry angle perpendicular to the disc plane rather than parallel to it) can help orient the slice into a plane more parallel with the neural arch, thereby making pars defects more visible. In general, CT scanning is not necessary for most cases.

Magnetic resonance imaging (MRI) has also been utilized in the imaging of spondylolysis and spondylolisthesis (Fig 3). Stress injury to the pars can be seen as increased signal intensity on T2 weighted images. This represents bone marrow edema secondary to pars microfractures. Acute fracture also

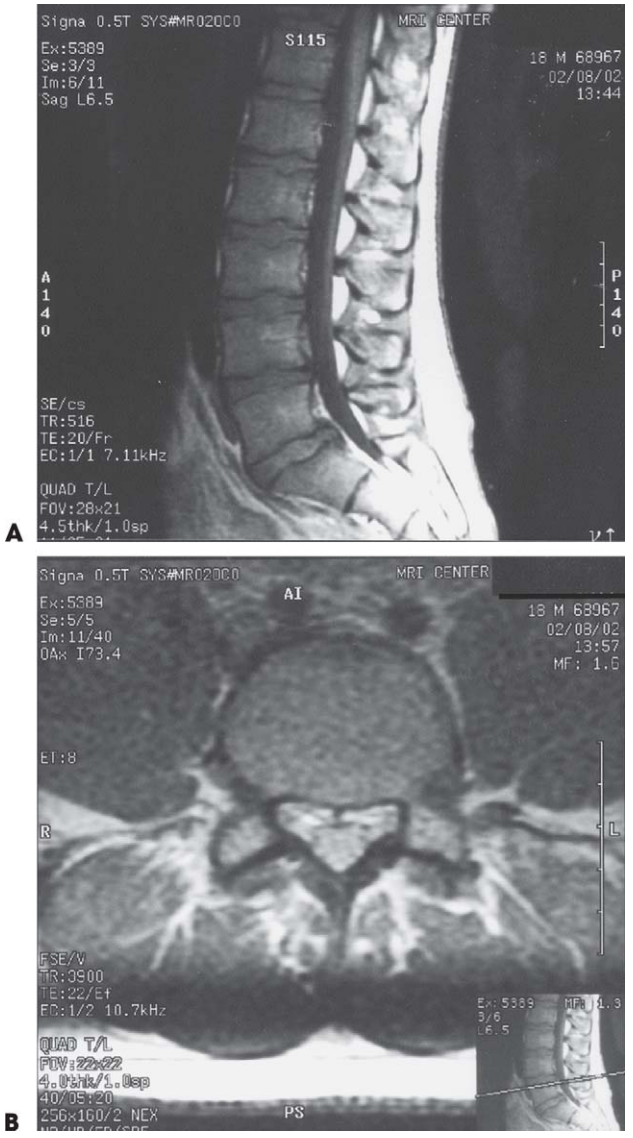

Figure 3 A and B. Sagittal T1-weighted MRI of lumbar spine (A) shows spondylolisthesis of L5/S1 and a widened AP diameter of the vertebral canal. Axial proton density-weighted MRI of lumbar spine (B) reveals bilateral linear low signal intensity through the pars interarticularis bilaterally, indicative of chronic pars defects.

may demonstrate bone marrow edema, along with disruption of the cortex. In old pars defects, low signal on T1 and T2 weighted images represent sclerosis. A widened canal diameter on sagittal images has been shown to be a reliable sign of spondylolysis without spondylolisthesis.[23] In a study of 29 patients with a pars defect, 90% were shown by MRI to have a fibrocartilaginous mass at the defect site. Mass effect on the thecal sac that required surgery was present in 21% of the spondylolysis patients.[24]

As previously mentioned, most cases of spondylolysis are not symptomatic. How can spondylolysis be correlated to low back pain? The key is to obtain some form of physiologic imaging rather than just anatomic imaging. Physiologic imaging modalities such as radionuclide scintigraphy (bone scan) and SPECT (Single Photon Emission Computed Tomography) can aid in distinguishing symptomatic (active) from asymptomatic (inactive) spondylolysis.[25–27] An active pars lesion could be either an impending stress fracture or a newly completed one. Impending stress fractures are negative on plain film radiography or CT but show high radionuclide uptake (hot spots) on bone scintigraphy. A new stress fracture would be seen on radiography and would be hot on bone scintigraphy. In both cases, there is locally increased osteoblastic activity and therefore the potential for osseous healing. With an inactive lesion, a pars defect may be evident on radiography, but the scintigraphic exam would be normal. Osteoblastic activity in this setting has ceased and resulted in a chronic non-union. Healing of the pars defect at this point is unlikely. This concept of active and inactive lesions is novel for many clinicians, but is quite valuable when considering treatment options. Since radionuclide skeletal scintigraphy has the capacity to diagnose pending spondylolysis, it should be considered very early in the workup of adolescents with low back pain. Planar (conventional) bone scan is acceptable, although SPECT (Fig 4), with multiplanar acquisition technology, is even more sensitive.[28,29] Some authors recommend avoiding radiography entirely and proceeding directly to SPECT imaging.[30]

Active lesions are most likely found in an adolescent or young adult who is engaged in a high level of physical activity and has an impending or newly acquired pars defect. In a study of young military recruits with a known spondylolysis, 43% of those individuals with a relatively recent onset of back pain were positive on bone scintigraphy. In comparison, those spondylolytic patients with a long history of back pain were positive on bone scan only 13% of the time.[31] Presumably, those with an acute onset of back pain have a newly acquired defect which is undergoing increased osteoblastic activity in an attempt at repair. The chronic low back pain patients, on the other hand, have likely developed a non-union and osteoblastic attempt at repair of the pars defect has diminished or ceased.

Increased radionuclide uptake can be seen in subjects with chronic low back pain. Lusins et al[32]

Figure 4. Coronal SPECT scan of the lumbar spine. Note the solitary area of high radionuclide uptake. This corresponds to a unilateral L4 spondylolysis (an unusual level). On standard radiography and conventional planar scintigraphy, this finding was occult.

studied subjects with confirmed spondylolysis, but instead of looking at the duration of symptoms, the study groups were categorized by the degree of spondylolisthesis and imaged with SPECT. In subjects with spondylolysis but without spondylolisthesis, 25% had positive scans and uptake was localized to the pars. Those subjects with positive findings had a mean age of 15.3 years while those with negative a mean age of 35.9 years. Contrast those findings with subjects displaying a prominent degree of spondylolisthesis. In this group, 75% had positive scans and increased radionuclide uptake was in a more anterior (vertebral body) location. The mean age of those with positive scans was 39.8 years. This anterior location of increased radionuclide uptake was likely the result of degenerative disc disease. These subjects were significantly older and within an age where degenerative change is expected. So, the older subjects with spondylolysis had pain, but physiologic imaging showed higher bone metabolic activity due to degenerative changes at the discovertebral joint, not due to a pars fracture.

This raises a question that is not well documented in the literature. Does a spondylolysis with or without a spondylolisthesis predispose to earlier or more prominent degenerative change in adjacent intervertebral discs? If this is the case, this may be a generator of back pain. Cohen et al[33] performed provocative discography on fourteen adult subjects (mean age 37 yr; range 24–54 yr) with a previous diagnosis spondylolytic spondylolisthesis. Provocative discography was performed from L2–S1 on all subjects. Seven of 14 subjects had concordant pain at a level adjacent to the spondylolisthesis. Twelve of 14 subjects had pain at the level of the spondylolisthesis.[33] Other authors have alluded to the fact that the pain in adult spondylolytic spondylolisthesis was due to disc degeneration and not the pars defect itself.[34,35] What conclusions can be reached from this information? It is probably safe to assume that adults with low back pain have the pain for some reason other than their spondylolysis, except in the setting of concurrent instability. Back pain in childhood or adolescence should raise the suspicion of a newly developed or impending spondylolysis, and an appropriate attempt at diagnostic confirmation should follow.

However, the concept of a pars defect as a pain generator cannot be entirely ruled out, even in older individuals. There have been small studies in the last few years that support the view that the pars defect itself may be painful in some spondylolytic patients. Neural elements, specifically free nerve endings capable of nociception, have been histologically documented within the scar and connective tissue that bridges the gap of a pars defect.[36] But, at this time, there is insufficient documentation to reach any definitive conclusions regarding the likelihood that these neural elements are truly a source of pain.

NATURAL HISTORY AND PROGNOSIS

Approximately 25% of radiographically detected spondylolysis is accompanied by spondylolisthesis.[5] The question of whether spondylolisthesis is progressive in nature has been studied extensively and the conclusions tend to be very similar. Despite varying follow-up periods among the different studies, there is a very low degree of progressive slip.[37–39] Spondylolisthesis progression in adults is less studied than progression in children, although the evidence seems to point to a minimal risk of progression in most individuals. Ohmori et al[39] studied 22 adult patients with spondylolysis. When comparing the initial presentation with follow-up approximately 12 years later, they found that of 18 patients without a listhesis initially, 13 still had no

slip, and 5 had progressed to an average of 16.6% slip. There was only an average of 6% increase in slip among those patients who had anterolisthesis initially. Saraste[40] studied 210 patients over a time period of 20 to 44 years. Patients initially ranged from 9 to 40 years of age (mean = 24), and at follow-up were between 30–84 years of age (mean = 53). The mean vertebral slip at diagnosis was 7.0 mm. The average progression over the observation period was only 4.0 mm.

Many predisposing risk factors have been proposed for progressive anterolisthesis. Two commonly seen radiographic parameters originally thought to increase the risk of slip are a trapezoidal shape of the L5 vertebral body and rounding of the sacral base. These morphological changes are frequently seen in association with spondylolysis and spondylolisthesis, but they have no predictive value.[5,37,38,41] These changes seem to be the result of spondylolysis and spondylolisthesis rather its cause.[5,37,42,43] Recent studies elucidate that epiphyseal injury in the juvenile spine is a determining factor for progression and a cause for abnormal morphology.[42–45] Sairyo et al[46] proposed, using a rat model of lumbar spine slippage, that vertebral forward slippage in the immature spine due to separation of the epiphysis and is unrelated to disc degeneration. Inoue and colleagues[47] have concluded that slips with deformities of the sacral table most likely had developed in adolescence, while slips without deformity likely developed during adulthood. Thus, they state that low-grade spondylolytic spondylolisthesis can be categorized into "adolescent and adult vertebral slips".[47] Also, numerous studies have confirmed spina bifida occulta as a high coincident anomaly with spondylolysis,[5,6,8] but it has not proven to be a predictor of anterolisthesis progression.[37,38]

The degree of anterolisthesis present at a spondylolytic segment may actually be of minimal clinical significance. It has been shown that the degree of low back pain experienced by spondylolytic patients has poor correlation with the amount of static anterolisthesis, but good correlation with the degree of instability.[48,49] Prominent instability in a spondylolytic level with minimal anterolisthesis is more problematic than stable segments with prominent anterolisthesis. Traditionally, instability has been documented by performing lateral lumbar views done in flexion and extension. Recently, traction and compression views were also found to demonstrate instability.[48–50] In traction, the patient is asked to hang from a bar overhead such that the weight of the lower body causes a distractive force that can minimize the degree of anterolisthesis. In compression, the patient wears a weighted backpack in order to apply additional axial compression to the olisthetic level. This can cause increased anterior translation (Fig 5). The documentation of instability should never be attempted during instances of acute muscle spasm. The spasm has a stabilizing effect that may mask any signs of inherent instability.[49] Translational movement between traction and compression of more than 3.6% is indicative of instability.[50]

A recent study has claimed that flexion/extension radiography documented more instability than traction/compression.[51] The validity of this claim, at least with regard to lytic spondylolisthesis, is questionable. The study only included 5 individuals with documented spondylolytic instability. In these 5 patients, only 1 showed instability on traction/compression. Such a small patient sample is not sufficient to draw any definitive conclusions relative to methodological advantages.

The mensurations used to determine the degree of anterolisthesis and instability are subject to measurement error. Simply measuring and comparing the distance that a vertebral segment has moved is not reliable for comparison purposes. Any change in focal-film distance or object film distance (ie, the patient experienced an increase in girth and the spine is now farther from the film) will cause magnification errors. For that reason, measuring a percentage of translation as described earlier is preferred because there is at least an internal reference; L5 and S1 will be projected bigger or smaller together. Even this is subject to error depending on exactly how the lines are drawn and measured. Choosing an anterior and posterior margin for the superior endplate of S1, for example, can at times be nothing more than a best guess due to poor contrast or anatomic variations. Small changes on sequential films may be nothing more than measurement error. Interval changes in spondylolisthesis as high as 20% have been considered to be within measurement error by some.[52,53]

CONSERVATIVE MANAGEMENT

Most patients with spondylolysis and/or spondylolisthesis respond to conservative treatment.[54] Man-

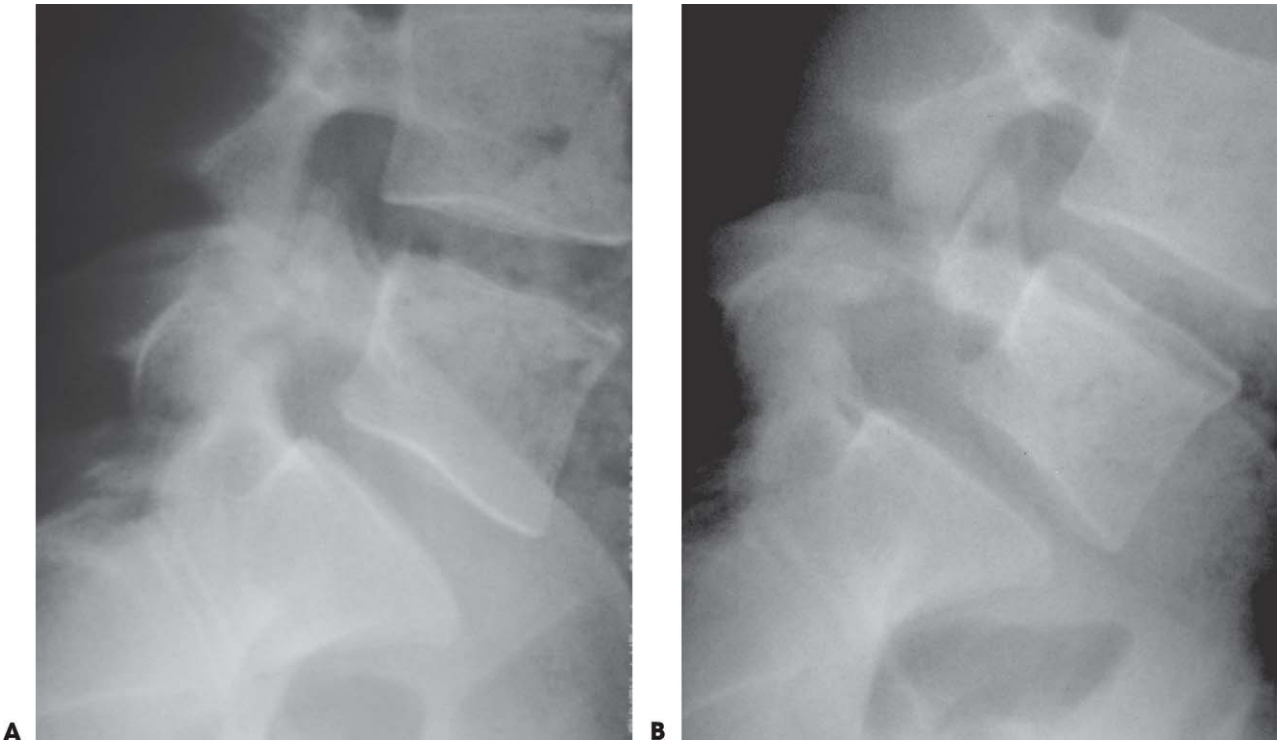

Figure 5 A and B. Traction (A) and compression (B) radiographic views of an L5 spondylolysis demonstrate significant instability. L5 translates anteriorly approximately 25% with a compressive load.

agement will differ depending on the clinical presentation of the patient. We propose that patients may classified into one of the following categories (Fig 6): 1) known spondylolysis without low back pain regardless of age; 2) the young patient with low back pain with suspected or known spondylolysis; or 3) the adult patient with low back pain with suspected or known spondylolysis.

In an asymptomatic spondylolytic patient, treatment of any sort (for the spondylolysis) will not be necessary. Most patients with spondylolysis develop it in childhood and therefore rarely have associated complaints. Asymptomatic lesions are not likely to be undergoing high levels of osteoblastic activity at the defect site, so the ability to achieve osseous healing is unlikely. The spondylolysis is probably in chronic non-union. Spondylolysis and associated spondylolisthesis in this group should never be used as a reason to restrict activity or cause denial of employment.[55]

A clinician has the potential to do much for a young person (child or adolescent) with a spondylolysis or pending spondylolysis and a relatively recent onset of back pain. This is the patient population in which pain is most likely to be due to the pars defect itself. This patient group would likely have an area of increased uptake in the region of the pars on bone scintigraphy or SPECT. This would indicate an active spondylolysis. One popular treatment method is to place the patient into a restrictive brace. Braces that maintain lordosis as well as those that are anti-lordotic have both been used with good results.[56,57] The goal of bracing is to prevent motion at the stress fracture and allow for osseous healing at the defect. Healing occurs in some, but not all cases. Fujii et al[58] found that the stage of the defect and the vertebral level involved were the predominant prognostic indicators for union. Early stage defects were much more prone to union than progressive or late stage defects. Also, defects at L4 achieved union more commonly than at L5. Even without bony fusion, the patient outcome is typically very good. Iwamoto et al[59] stated that although bony union is preferred, stable, pain-free fibrous union that enables full activity is an acceptable outcome of conservative treatment. Some clinicians have restricted their patient's activity while braced,[57] while others have allowed full athletic participation while the brace is in posi-

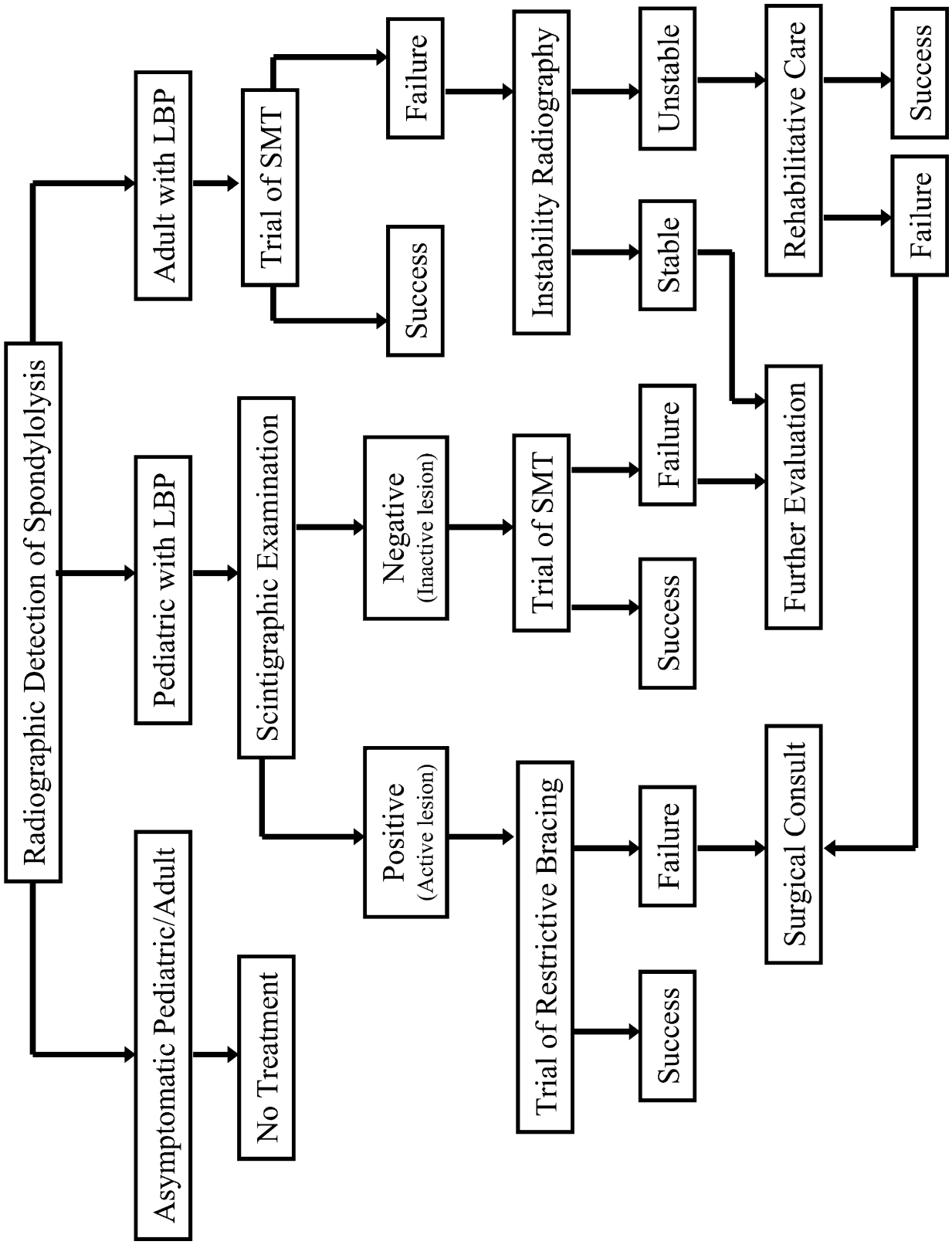

Figure 6. Flowchart for management of spondylolysis. LBP = low back pain, SMT = spinal manipulation therapy.

tion.[56] In addition to bracing, stretching and strengthening exercises are advised.[56,57]

The last group to consider is the adult spondylolytic with low back pain. As previously discussed, acute onset of pars fractures in adults is extremely rare. Therefore any pars lesions in these patients can be considered non-union and probably not the source of low back pain unless there is associated segmental instability. A course of high-velocity low-amplitude (HVLA) manipulative therapy to areas of fixation in the lumbar spine has been shown to be as effective at reducing chronic pain in spondylolytic patients as in non-spondylolytic patients.[60] This study evaluated manipulation to the joints of the lumbar spine in side-posture position and avoided direct manipulation of the spondylolytic segment. A specific exercise regimen for strengthening the deep abdominal muscles and lumbar multifidus in order to provide dynamic stability to the spine has been proven to give significant pain relief.[61]

In the setting of unstable spondylolisthesis and chronic low back pain, a course of rehabilitation may be in order. Lindgren[62] prescribed a course of therapy including muscle stretching, coordination activities, and muscle strengthening. Comparison of pre- and post-therapy symptoms, electromyographic (EMG) findings, and radiographic instability were made. There was no change in the degree of instability seen at radiography. Interestingly, clinical symptomatology and EMG findings improved significantly.[62]

As stated previously, manipulation of the lumbar spine may have some benefit in adults with inactive spondylolytic spondylolisthesis. However, attempts at manipulating a spondylolisthetic segment "back into place" are not supported in the literature. Infact, the spondylolysis "gap" is not open, but is filled with fibrous scar tissue that would prohibit reduction of the vertebral body "back" into a more posterior location.[24] When examining the histological specimens for evidence of nerve endings, Schneiderman[36] reported the presence of extensive scar tissue formation throughout all the specimens. The specimens showed evidence of fibrofatty tissue to dense collagenous scar, consistent with what the authors termed a pars pseudoarthrosis.[36] The surgical literature specifies removing fibrous tissue at the pars defect before performing repair of the defect.[63,64] Also, excessive vertebral body translation would indicate the presence of instability and thereby contraindicate high velocity, low amplitude spinal manipulation.

If persistent back pain and/or radicular pain are resistant to conservative treatment or the slippage of the vertebra is increasing, surgical treatment is usually indicated.[63] The methods of stabilization are varied and multiple.[64–68] Evaluation of which surgical methods yield the best results is beyond the scope of this paper.

CONCLUSIONS

Spondylolysis, a break in the pars interarticularis, is a common radiographic finding. Evidence shows that this condition typically develops in childhood or adolescence, not adulthood. In the adult population, back pain in concurrence with spondylolysis is most likely due to other sources, and not the pars defect. An unstable spondylolysis with spondylolisthesis is less frequent, but may be symptomatic. Progression of the spondylolisthesis most commonly occurs in adolescence, and is likely the result of epiphyseal injury. In general, severe progression of spondylolisthesis is not common. Radiography is the most common imaging modality used to detect spondylolysis and spondylolisthesis. Computed tomography, especially when a reverse gantry angle is used along with coronal or sagittal reformatting, is more sensitive than plain radiography. Bone scintigraphy (bone scan) and SPECT are physiologic imaging modalities that should be employed when evaluating a young patient with spondylolysis. Most patients with spondylolisthesis and/or spondylolysis respond to conservative treatment.

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