Spondylolysis is a defect in the pars interarticularis of a vertebra. It may be unilateral or bilateral.¹ The earliest age at which these lesions appear has been debated; often they are not apparent until a child is able to stand upright. The incidence of pars defects of the lumbar vertebrae is zero at birth.²⁻⁴ Fredrickson et al² first performed a prospective population-based study to determine the natural history of spondylolysis through childhood and into adulthood. These authors showed a 4.4% incidence at the age of 6 years, and the incidence was approximately 6% in adults. In those patients they were able to follow to skeletal maturity, nearly 90% of spondylolytic lesions were seen at L5. Of these L5 pars defects, more than 70% were bilateral and 17% were unilateral. It is likely that between 3% and 7% of the general population has a spondylolytic lesion.²⁻⁶

Unilateral spondylolysis is much less common than the bilateral type. The incidence of unilateral lumbar spondylolysis is estimated to be less than 2% in the general population, with most seen at L5. Previous studies showed unilateral pars defects in 3% to 33% of the population.⁶⁻⁹

Degeneration is believed to begin in the intervertebral disk, where loss of

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**Abstract**

This study examined the effect of bilateral and unilateral L5 pars defects on the degree of disk degeneration at the L5-S1 level in cadaveric specimens. An observational study was performed of 690 cadaveric specimens selected at random. These specimens represent individuals who died between 1893 and 1938. The study included 558 male and 132 female cadavers. Of the 120 specimens with L5 spondylolysis, 95 cases were bilateral and 25 were unilateral. The remaining 544 specimens were used as the control cohort. Degenerative disk disease was measured by the classification of Eubanks et al. According to this classification, degenerative disk disease was graded from no arthrosis (grade 0) to complete ankylosis (grade IV). Linear regression analysis corrected for age, sex, and race showed that subjects with bilateral spondylolysis at L5 had a statistically significant increase in the amount of disk degeneration (P=.02) compared with those with unilateral lesions. Student’s t tests showed significant differences (P<.001 and P=.002, respectively) in the amount of degeneration seen with both bilateral and unilateral spondylolysis above what would be predicted in the normal control population. A positive correlation was found between the number of pars defects at L5 and the degree of disk degeneration at L5-S1. These results support the idea that individuals with spondylolysis at these levels may be at increased risk for development of low back pain and reduced quality of life. [Orthopedics. 2017; 40(1):e59-e64.]

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*Spondylolysis and End Plate Arthrosis at L5-S1: A Cadaveric Study*

Peter T. McCunniff, MD; HoJun Yoo, BS; Charles Yu, MD; Navkirat S. Bajwa, MD; Jason O. Toy, MD; Uri M. Ahn, MD; Nicholas Ahn, MD

The authors are from the Department of Orthopaedic Surgery (PTM, HY, CY, NSB, JOT, NA), University Hospitals of Cleveland, Cleveland, Ohio; and the New Hampshire NeuroSpine Institute (UMA), Bedford, New Hampshire.

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Correspondence should be addressed to: Peter T. McCunniff, MD, 408 W St Clair Ave #315, Cleveland, OH 44113 (pmccunniff@gmail.com).

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height leads to biomechanical changes that further increase pressure on the joints, leading to arthrosis. Previous research showed that degenerative disk changes are always accompanied by concomitant vertebral rim osteophytosis and that end plate arthrosis may be used as a marker for degenerative disk disease. Although the spine is a 3-joint complex, Eubanks et al noted that degenerative changes of the spine more closely mimicked the changes at the vertebral end plates rather than those of the facet joints, further supporting end plate arthrosis as a superior marker of disk degeneration compared with facet arthrosis. The study hypothesis was that increasing levels of end plate arthrosis would occur at the L5-S1 joint in response to bilateral L5 spondylolysis compared with unilateral spondylolysis and control specimens without spondylolysis. The authors also believed that specimens with unilateral spondylolysis would have a higher level of disk degeneration compared with control specimens without spondylolysis. The authors formed these hypotheses based on the belief that lumbar disk joints were increasingly unstable and that a greater number of pars defects would result in increased degeneration of the disk joint.

**MATERIALS AND METHODS**

An observational study was performed with the 3100 cadaveric specimens from the Hamann-Todd osteologic collection. The authors chose 690 skeletal lumbar spines from the entire collection based on their collective proximity within 2 rows of the collection and their ease of access (no ladder necessary to retrieve the specimens from the shelves). The selection was random because the specimens in the collection are not arranged in any particular order. Specimens were obtained from individuals who died between 1893 and 1938. Specimens were obtained from 558 male and 132 female cadavers (age range, 17-105 years). Of the 120 specimens with L5 spondylolysis, 95 cases were bilateral and 25 were unilateral. The remaining 544 specimens were used as the control cohort (Table 1).

Gross specimens were examined subjectively by 1 examiner for evidence of arthrosis. The specimens were not examined specifically for coexisting spondylolysis, and the examiner was not blinded to the age of the specimens. Degenerative disk disease was measured according to the classification of Eubanks et al, which is a modified version of the Kettler and Wilke classification. According to this classification, degenerative disk disease was graded from no arthrosis (grade 0) to complete ankylosis (grade IV), as seen in Table 2. Linear regression analysis was performed to derive a formula to predict the amount of degenerative disk disease at L5-S1 for the normal control population, taking into account the age, sex, and race of the specimen.

**RESULTS**

Linear regression analysis, correcting for age, sex, and race, showed that subjects with bilateral spondylolysis at L5 had a statistically significant increase in the amount of degenerative disk disease (P=.02) compared with those with unilateral lesions. The number of defects in L5 seemed to have a larger effect, and therefore a greater difference, in the degree of degenerative disk disease as subject age decreased. As seen in Figure 1 and Figure 2, younger patients with bilateral defects had a significantly greater degree of degenerative disk disease compared with the group with unilateral defects and the control group. However, as age increases, the difference in the corresponding degree of degenerative disk disease decreases (Figures 3-4).

Student’s t test showed significant differences (P<.001 and P=.002, respectively) in the amount of degenerative disk disease seen with both bilateral and unilateral spondylolysis above what would be predicted for the normal control population (Table 3).

**DISCUSSION**

Lumbar spondylolisthesis, or degenerative spine disease, is the most common etiology of low back pain, and it can have a substantial effect on quality of life.
Medical costs associated with back pain in the United States doubled to more than $100 billion during the past 7 years, with a 65% increase in national expenditures on back-related issues. The authors are not aware of another study that has examined the relationship between bilateral and unilateral L5 spondylolysis and its correlation with degenerative disk disease at the L5-S1 joint. Based on skeletal observations, the current results support previous reports showing that L5 is the most common vertebra to have a pars defect and that these defects are most commonly bilateral.2,6 The current findings showed a positive correlation between the number of pars defects at L5 (zero, unilateral, or bilateral) and the degree of end plate arthritis and corresponding degenerative disk disease seen at the L5-S1 joint. These differences were statistically significant for bilateral spondylolysis compared with both the unilateral defect group and the control group as well as when disk degeneration resulting from a unilateral defect was compared with control specimens without spondylolysis.

In a biomechanical study of the lumbar spine, Farfan et al18 concluded that, under normal conditions, the L5-S1 intervertebral joint was subjected to the highest forces and therefore was the first joint to be damaged. These changes in transmission of force can predispose the joint to failure of the pars and resultant spondylolysis. They also concluded that torsional forces of the spine were the main cause of damage to the intervertebral disk.19 When this damage is coupled with the effect of unilateral rotation from a single pars defect, this may initiate the cascade of worsening disk degeneration and end plate arthrosis, leading to further changes in transmission of force to maintain stability, with subsequent failure of the contralateral pars. Adams et al20 showed that even minor damage to the vertebral body end plate leads to progressive structural changes in the adjacent intervertebral disk.

This study is not without limitations. Skeletal cadavers were used to examine degenerative disk disease with both bony and soft tissue involvement. Without the availability of soft tissue attachments, it was impossible to comment on an important factor, such as the degree of slip, or spondylolisthesis. One of the current standards for assessing the degree of degenerative disk disease, magnetic resonance imaging, could not be used in this cadaveric study. Over the past 30 years, magnetic resonance imaging has been the most accurate test for showing morphologic abnormalities.21,22 Assuming that changes in the skeletal anatomy of adjacent vertebrae represent later stages of underlying soft tissue derangement, the findings of this study seem to accurately reflect alterations in the soft tissues that

Figure 1: Graph showing end plate arthrosis at the L5-S1 joint, with the classification grade shown on the vertical axis and age shown on the horizontal axis. Solid line, trend line for degree of end plate arthrosis for specimens with unilateral spondylolysis at L5; dashed line, trend line for L5 control subjects.

Figure 2: Graph showing end plate arthrosis at the L5-S1 joint, with the classification grade shown on the vertical axis and age shown on the horizontal axis. Solid line, trend line for degree of end plate arthrosis for specimens with bilateral spondylolysis at L5; dashed line, trend line for L5 control subjects.
make up the intervertebral spaces. Previous studies agree with the current findings that end plate arthrosis in skeletal remains provides an accurate marker of degenerative disk disease. In addition, the examiner was not blinded to the age of the specimens, and this could lead to bias in tending to associate a higher grade of degeneration with older patients. However, the data showed that many of the younger patients with bilateral spondylolysis were given higher grades. The opposite was true for older control subjects who had little degeneration. In addition, because this was a cadaveric study, it was not possible to determine the symptoms that the subjects may have experienced. Also, there is no information available on factors such as smoking status or other systemic illnesses that can contribute to degenerative disk disease. Without that information, it is difficult to determine the clinical significance of the degree of disk degeneration seen in this study.

The literature on causality between degenerative disk disease and low back pain is not clear, with many opinions differed on both sides. In a systematic review, Van Tulder et al.23 reported that lumbar disk degeneration is a risk factor for low back pain in adults. Multiple studies showed an association between degenerative disk disease and low back pain. Salminen et al.27 argued for a causal relationship between early-onset degenerative disk disease and recurrent low back pain throughout the lifetime. Substantial research shows that degenerative changes may be seen in asymptomatic patients and that these findings are not predictive of the development or the duration of low back pain on long-term follow-up.31 Although this study found a statistically significant correlation between the number of pars defects and the extent of disk degeneration, additional factors may affect the degree of disk degeneration seen at this level. Hutton et al.32 concluded that horizontal inclination of L5 was correlated with the likelihood that patients with spondylolysis would have spondylolisthesis and subsequent disk degeneration. They showed that tensile and shear forces acting on the disk were significantly higher when subjects were in a flexed vs partially flexed posture. Toy et al.33 evaluated the relationship between sacro-pelvic geometry and the degree of L5-S1 disk degeneration and concluded that the likelihood of spondylolisthesis and subsequent disk degeneration increased with increasing sacral inclination and pelvic incidence.

The relationship between the normal control population and the unilateral defect group may be the most important finding of this study because unilateral defects often are not followed as closely as bilateral pars defects. However, the current study showed a significant increase in the amount of degenerative disk disease seen in patients with a unilateral defect compared with normal control subjects. Although it is impossible to determine how much this increase would manifest itself in the clinical setting, unilateral lesions are not benign and may progress to bilateral defects or accelerate the degeneration of the spine at L5-S1.34-36

Previous authors concluded that patients who have spondylolysis without progression to spondylolisthesis have normal disk height on x-ray at the time of diagnosis and that disk degeneration and subsequent reduced disk height develop only after the spondylolisthesis process begins. However, disk degeneration increases with age, regardless of the presence of spondylolysis or spondylolisthesis. The finding of normal disk height could be a function of patient age and the concordant degree of disk degeneration.
not the lack of slip. Because these processes are intertwined, it is difficult to determine which factor (age, presence of spondylolysis, degree of spondylolisthesis, or another described variable) is responsible. It is likely that all of these factors function together to produce the final outcome that the study was designed to examine, which is the degree of degenerative disk disease.

**Conclusion**

Without an in vivo study, it is difficult to determine how many factors could affect stability or disk degeneration at this lumbar level. The goal of the current study was to examine the difference in degenerative disk disease between the afore-mentioned lumbar level with bilateral or unilateral pars interarticularis defects beyond what would be expected in patients without spondylolysis. Patients with bilateral spondylolysis at L5 had a statistically significant increase in the amount of degenerative disk disease (P = .02) compared with those with unilateral L5 spondyloytic lesions. The number of defects at L5 had a larger effect on the degree of degenerative disk disease in younger subjects. The study also found significant differences in the amount of degenerative disk disease seen with both bilateral and unilateral spondylolysis above what would be predicted for the normal control population.

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