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Lumbar Instability Adjacent to a Previous Fusion

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Disc degeneration that occurs at the level adjacent to a previous fusion is known as adjacent segment degeneration (ASD). Altered biomechanical forces at the adjacent level motion segments may predispose to subsequent degeneration and pain, which may require further surgery consisting of decompression with or without an extension of the fusion. The rapid rise in the rates of lumbar spinal fusion over recent years suggests that ASD may become even more prevalent with time. Adjacent segment degeneration has served as the impetus for the development of motion-preserving alternatives for the treatment of degenerative disc disease, most notably total disc replacement (TDR). This article will highlight the diagnosis, etiology, risk factors, and treatment for lumbar instability adjacent to a previous fusion and discusses the potential role for total disc replacement in the prevention of ASD.

KEYWORDS lumbar instability, adjacent segment disease, adjacent segment degeneration, risk factors, total disc replacement

On January 9, 1911, Russell Hibbs introduced the technique of spinal arthrodesis for the treatment of spinal deformity in a patient with tuberculosis.[1] In that same year, Fred Albee described fusion for the treatment of Pott's Disease.[2] Since that time, spinal arthrodesis has become the treatment of choice for numerous spinal disorders, including spinal trauma, scoliosis, spondylolisthesis, spinal tumors, and degenerative disc disease (DDD).

Despite the use of current techniques and careful patient selection, fusion rates are inevitably higher than clinical success rates. Numerous studies have confirmed inconsistent clinical outcomes following fusion for the treatment of lumbar DDD, which ranges from 65 to 93%, despite the presence of a solid fusion.[3-5]

One of the most common complications following lumbar spinal fusion that may contribute to this discrepancy is the development of disc degeneration above or below a previous fusion, known as adjacent segment degeneration (ASD). Altered biomechanical forces exerted at the spinal motion segments adjacent to fused levels may predispose to subsequent degeneration and pain, which may require further surgery consisting of decompression with or without an extension of the fusion. The alarmingly rapid rise in the rates of lumbar spinal fusion over recent years suggests that ASD may become even more prevalent with time.[6,7] In combination with other potential morbid consequences of lumbar fusion (Table 1), ASD has served as the impetus for the development of motion-preserving alternatives for the treatment of DDD, most notably total disc replacement (TDR). This article will highlight the diagnosis, etiology, risk factors, and treatment for lumbar instability adjacent to a previous fusion and discusses the potential correlation between this instability and adjacent segment degeneration.

Diagnosis of Adjacent Lumbar Instability

As discussed in the first section of this issue, classic segmental lumbar instability has been defined as "a loss of spinal motion segment stiffness, such that force application to that motion segment produces greater displacement than would be seen in a normal structure, resulting in a painful condition, the potential for progressive deformity, and neurologic structures at risk."[8] Instability adjacent to a previous fusion is only one of several possible pathologic processes that have been described at the adjacent level that comprises ASD (Table 2).

Clinically, there exists no specific pattern of pain that correlates with radiologic evidence of instability.[9,10] Symptoms can include but are not limited to recurrent, acute episodes of back pain, radiation of lumbar pain into the lower limbs, and pain on sitting down, which is relieved by standing up.[11,12] It

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Table 1 Potential Consequences of Lumbar Spinal Fusion that May Be Alleviated by Total Disc Replacement

Lack of pain relief
Loss of motion
Pseudarthrosis
Sagittal balance
Adjacent segment disease
Bone-graft donor site morbidity

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has also been suggested that visual inspection and palpation during the physical examination can be useful in detecting lumbar instability.[13] Following lumbar fusion, several authors have noted that a symptom-free period of 12 months following the index procedure is necessary to discern painful pseudarthrosis from other pathology, including ASD.[14-16] Absence of a pain-free interval following lumbar fusion suggests that the fusion did not address the pain-generating lesion.[17]

The classic radiographic criteria for lumbar instability usually include sagittal plane translation of 4.5 mm, or 15% of the sagittal width of the vertebral body, and angulation of 10 to 15° during flexion and extension.[18,19] It is well-known, however, that the diagnostic utility of range-of-motion measurements from static plain films in identifying abnormal motion segments is limited.[20,21] Several authors have shown poor inter- and intraobserver reliability in Cobb angle measurements from which the range of motion is typically calculated.[21-25] Inconsistent patient effort during flexion-extension is another factor that contributes to the highly variable range of motion values typically seen from flexion-extension radiography, further limiting its utility as a reliable study endpoint to evaluate in vivo spinal motion. Therefore, it has been posited that continuous motion analysis using cineradiography or videofluoroscopy is required to more properly evaluate and compare quantitative lumbar motion profiles and facilitate the diagnosis of spinal segmental instability.[26-28] Later in this section we describe an in vivo fluoroscopic technique to evaluate lumbar motion profiles in patients who underwent total disc replacement, circumferential lumbar fusion, and controls.

Etiology of Adjacent Segment Instability

Altered Biomechanics at the Adjacent Level

While the exact etiology of disc degeneration adjacent to a fusion is not known, there exists convincing evidence to suggest that adjacent segment degeneration is at least partly the result of altered biomechanical stresses. Shono and coworkers tested calf lumbosacral spine specimens that were intact or destabilized (multilevel disc dissections), followed by posterior spinal instrumentation using a variety of constructs. Specimens were tested in flexion-extension, rotation, and lateral bending. They showed that the upper uninstrumented adjacent level underwent greater rotational motion compared with intact controls, and that the lower uninstrumented adjacent level underwent greater translational motion.[29] The magnitude of the changes correlated with the number of instrumented levels. Using canine spines, Ha and coworkers reported that after lumbosacral immobilization, facet contact patterns at the adjacent segment were altered and segmental motion increased when the lumbar spine reproduced the same range of motion.[30] Bastian and coworkers also demonstrated hypermobility at the adjacent level, which resolved following removal of the instrumentation.[31] Other human cadaveric studies have demonstrated up to 45% higher intradiscal pressures at discs adjacent to the instrumented fusion level compared with controls.[32,33] Altogether, numerous in vitro experiments using both human and dog cadaveric specimens have revealed increased mobility, intradiscal pressures, facet loads, and tissue stresses at levels above a fusion.[30,31,34-37]

Despite ample in vitro evidence for altered stresses at the level adjacent to a fusion, there are few studies that have shown a causative effect for the subsequent development of ASD. Several in vivo biomechanical animal studies, however, have provided support for this relationship. Cole and colleagues performed two-level posterior spinal fusion on 10 beagles and sacrificed the animals at 6 and 12 months. Postmortem analysis revealed a decrease in proteoglycan (PG) content and aggregating capacity in the nucleus pulposus in the adjacent segment, which was similar to that seen in the fused segment.[37,38] Taylor and coworkers recently showed in vivo that the proximity of lumbosacral discs to a rigid segment (the sacrum) predisposed the disc to decreased PG content, decreased ability for PG to aggregate, and PG catabolism which exceeded the rate of biosynthesis.[39] Phillips and coworkers recently described an in vivo experimental rabbit model of disc degeneration at the adjacent level that developed following posterolateral lumbar fusion. The rabbits were sacrificed at time points up to 9 months. Their results revealed disc degeneration at the adjacent level that is similar to that seen in humans with respect to disc histology, biochemistry, and radiology.[40] Such an animal model may prove to be useful for the study of disc degeneration without the need for invasive annular punctures or pharmacological nuclear alterations.

Table 2 Potential Pathology Seen at the Level Adjacent to Lumbar Arthrodesis

Retrolisthesis, anterolisthesis
Instability
Herniated nucleus pulposus
Stenosis
Hypertrophic facet arthritis
Osteophyte formation
Scoliosis
Vertebral compression fracture

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