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Posterior lumbar interbody fusion in the treatment of symptomatic spinal stenosis

Domagoj Coric, M.D., and Charles L. Branch, Jr., M.D.

Department of Neurosurgery, Bowman Gray School of Medicine of Wake Forest University, Winston-Salem, North Carolina

Lumbar spinal stenosis is often the result of advanced degeneration of motion segments of the lumbar spine. Loss of disc height, facet displacement and hypertrophy, spondylosis, and spondylolisthesis, as well as buckling of the ligamentum flavum and annulus fibrosus, all contribute to impingement on the spinal canal and intervertebral foramen in lumbar stenosis. There is a subgroup of patients with spinal stenosis in whom the spine is unstable preoperatively or becomes destabilized following decompression who would benefit from an initial fusion procedure. Posterior lumbar interbody fusion (PLIF) addresses several aspects of the multifactorial pathophysiology responsible for spinal stenosis and may arrest the degenerative changes at the fused level. Fusion, in particular PLIF, should be considered in complex cases of lumbar spinal stenosis, most notably in patients with postlaminectomy stenosis or stenosis associated with spondylolisthesis.

Key Words * posterior lumbar interbody fusion * lumbar spinal stenosis * spondylolisthesis * fusion

Spinal stenosis and herniated nucleus pulposus are among the most common degenerative conditions of the lumbar spine. Spinal stenosis is the most common diagnosis associated with lumbar spine surgery in persons older than 65 years of age.[2,17] Unlike radiculopathy secondary to herniated nucleus pulposus, lumbar spinal stenosis is not a distinct diagnostic entity. Both the pathological anatomy leading to spinal stenosis and the clinical symptoms that are manifested by the disease process are heterogeneous. Lumbar stenosis encompasses diverse abnormalities that culminate in narrowing of the central spinal canal, lateral canals, recesses, or intervertebral foramen.[3,31] The disease may be local, segmental, or generalized and may consist of decreased disc space height and protrusion of annulus fibrosus, hypertrophic osteoarthritic changes in the inferior or superior facets, osteophyte formation, ligamentum flavum buckling or hypertrophy, or various combinations of these factors leading to entrapment or compression of neural structures.[2,3,25,43,63] Lumbar stenosis has been classified anatomically as central or lateral (or mixed) and developmentally as congenital or acquired. Central stenosis involves narrowing predominantly of the spinal canal.[3,19,41,43,63] Lateral stenosis has been subdivided as entrance zone stenosis, mid-zone stenosis, and exit zone stenosis.[43] Lateral stenosis has also been referred to as lateral recess, foraminal canal, subarticular, subpedicular, intervertebral foramen, and

lateral gutter stenosis.[43,59] Congenital stenosis may be idiopathic or due to achondroplasia.[1] Acquired stenosis is subdivided into degenerative, spondylolisthetic, spondylotic, postlaminectomy, postfusion, posttraumatic, and metabolic stenosis.[3,11] An eclectic group of clinical symptoms have commonly been attributed to stenosis including: postural pain; aching and cramping of the lower extremities; neurogenic claudication; back pain; unilateral or bilateral radiculopathy; and morning stiffness. Lower-extremity numbness and paresthesias, as well as neurological deficit including reflex loss, motor weakness, sphincter disturbance, and muscle atrophy may also be part of the clinical presentation.[3,11,19,23,26,31,41,63]

ANATOMICAL CHARACTERISTICS

The vertebral bodies and discs account for the majority of the axial load bearing capability of the spine. The disc absorbs load and stress, provides support, and resists movement. The superior facet and associated ligamentum flavum form the roof of the lateral recess, where nerve roots exit the canal and enter the foramen. The lumbar facet complex is biplanar with the medial portion oriented in the coronal plane and the lateral portion in the sagittal plane. The medial portion (coronal) limits forward translation and the lateral portion (sagittal) resists lateral rotation.[30,62,72] Therefore, the total facet load consists of a component responsible for sharing axial load bearing with the disc as well as components for resisting anterior and lateral shear.[54] Ligaments play a primary role in resisting flexion rotation and posterior shear.[62] Biomechanical studies on cadavers demonstrate that the posterior ligaments, notably the supraspinous and interspinous ligaments, exert a significant effect on tensile stiffness.[4]

The neural foramen is bordered by the superior and inferior articular processes and pars interarticularis of the superior vertebra dorsally, sequential pedicles superiorly and inferiorly, and the intervertebral disc and the posterolateral surface of the vertebral body ventrally. The nerve root exists caudal to the pedicle, and the dorsal root ganglion lies in the superior and lateral portion of the foramen.[25,41,59]

PATHOLOGICAL CHARACTERISTICS

Degenerative lumbar spinal stenosis is the end result of a cascade of progressive changes that inevitably result in a reduction of spinal canal and/or neuroforaminal volume.[38,41,59,74] A motion segment or functional spine unit consists of two adjacent vertebral bodies, the intervertebral disc, associated ligaments, and facet joints.[55] The ligaments, facets, and disc stabilize the motion segment. Certain configurations, or damage to any of these stabilizing structures, place additional stress on the remaining structures.[41,62,75] Increased sagittal orientation of the facets has been correlated with degenerative spondylolisthesis and lateral stenosis.[24,58,62] Removal of a part of the medial facet during standard hemifacetectomy reduces the resistance to forward (sagittal) translation leading to postdecompression spondylolisthesis.[24,30,47] The ability of the normal intervertebral disc to resist substantial axial stresses decreases with the aging process. Over time, there is a decrease in intradiscal water concentration and a concomitant increase in fibrous tissue concentration. The disc becomes desiccated, and ultimately there is loss of the ability of the nucleus pulposus to evenly redistribute biomechanical loads.[33] This condition ultimately leads to internal disc disruption, disc resorption, annular buckling, and formation of bridging osteophytes.[6,33] As the load-bearing capacity of the disc degenerates there is loss of disc height, which leads to facet displacement and may, ultimately, result in segmental instability. The decreased disc space height may result in nerve root impingement by caudal migration of the superior pedicle. The shape of the intervertebral foramen is also altered by disc degeneration. Anatomical studies on cadavers demonstrate that disc collapse significantly reduces the diameter of the

foramen.[14,25,59] Increased stresses lead to reactive thickening of the lamina and hypertrophy of the ligamentum flavum, further reducing canal and foramen diameter.[33,75] Increased load and instability lead to pathological changes in the facet joints including: degeneration of the synovium and articular cartilage; fracture of the articular processes; laxity of the joint capsule; and osteophyte formation.[41] Excessive facet hypertrophy and osteophyte formation impinge on the spinal canal and lateral recess. Primary enlargement of the inferior facet and lamina narrows the central canal. Enlargement of the superior facet causes encroachment of the lateral recess and intervertebral foramen.[25,33,43,63]

It is evident that decompressive lumbar surgery plays a significant role in destabilizing the spine in a subgroup of patients with stenosis.[10,28,32,50] Surgical removal of the posterior elements during decompressive laminectomy or hemilaminectomy places additional load on the disc and may accelerate degeneration.[4,30,47,51] Postoperative progressive slippage as a result of segmental instability often leads to recurrent symptomatic spinal stenosis.[26] Brodsky[10] evaluated a series of 552 patients with lumbar stenosis and determined that nearly half (41%) had undergone previous laminectomy or fusion procedures. Cauchoix and associates[12] reported that additional spondylolisthesis and spinal instability are complications of decompression for stenosis caused by degenerative spondylolisthesis, and they recommended fusion for that condition. Tile, et al.,[67] reported that all of their patients with spondylolisthesis developed progressive slippage postoperatively. Shenkin and Hash[64] followed 59 patients after multiple-level bilateral laminectomies and facetectomies and reported that 15% of patients in whom three or more levels were removed developed progressive spondylolisthesis. Robertson, et al.,[58] reported that 58% of 33 patients treated by means of decompressive laminectomy showed postoperative spondylolisthesis of greater than 5% within 1 year of surgery. In 1990, Iida and associates[32] evaluated 46 patients who had undergone decompressive laminectomy for spinal stenosis and on follow-up examination confirmed instability at the operated level, adjacent level, or both in 83% of that group. Herkowitz and Kurz[26] reported spondylolisthetic progression in 96% of patients treated by decompression alone. Recently, Fox and colleagues[22] retrospectively studied 124 patients with degenerative lumbar stenosis treated with and without fusion; they reported progressive postoperative spondylolisthesis in 31% of patients with normal preoperative alignment and in 73% of patients with preoperative spondylolisthesis treated without fusion.

TREATMENT OPTIONS

There have been no randomized trials comparing surgical with nonsurgical therapy in patients with spinal stenosis, but nonsurgical treatment is notoriously ineffective in relieving the severe symptoms of lumbar stenosis.[5,35,73] A conventional operative treatment for central spinal stenosis is wide decompression of the affected neural structures. Traditionally, this has entailed a total laminectomy at the involved segment(s) with varying degrees of facetectomy and/or foraminotomy.[2,3,19,23,27,29,40,45,51,53,54,63,68-70] A more limited interlaminar decompression with medial facetectomy has been advocated for lateral stenosis without a significant central component.[43,45,57,64] Decompressive surgery alone does not address the underlying degenerative process responsible for creating the stenosis and, in some circumstances, may serve to accelerate it.[9,10,26,28-30,32,36,42,51,57,65] The ideal surgical intervention would remove only the bone and ligament needed to free compressed neural elements while preserving spinal stability without contributing to the degenerative process. When this is impossible or impractical, the corrective surgical procedure should decompress the neural elements while reducing the impact of the existing degenerative changes. Improving body mechanics and muscular conditioning may slow the degenerative process, but ultimately elimination of the motion segment may be necessary. At this point, segmental arthrodesis

should be considered.

POSTERIOR LUMBAR INTERBODY FUSION

Posterior lumbar interbody fusion (PLIF) allows for decompression of the neural elements while preserving certain components of the posterior supporting structures, including the spinous processes and interspinous ligaments, and accomplishing fusion of the motion segment. The neural elements are decompressed through bilateral laminotomies and medial facetectomies. The nerve roots are visualized and freed of any adhesions in preparation for the total discectomy. The near-total discectomy facilitates the placement of substantial interbody graft and minimizes the risk of recurrent stenosis by bulging annulus or recurrent disc herniation. The interbody graft restores disc space height (Fig. 1) and the normal anatomical configuration of the neural elements and the motion segment.[7,8,13,16,44,46]

Fig. 1. Plain lateral lumbar radiograph obtained 9 months postoperatively, demonstrating solid osseous fusion of an allograft PLIF at L4-5. Note the maintenance of disc space height and foraminal area in relation to the normal adjacent levels.

In fact, PLIF restores all the anatomical properties of the functional spinal unit with the exception of motion. Cadaveric studies demonstrate a significant increase in foraminal area with distraction of the disc space.[59] The restoration of the disc space height ameliorates impingement on the nerve root by the pedicle and reconstitutes the neural foramen. Correction of forward slippage in degenerative spondylolisthesis is possible with PLIF (Fig. 2). Successful interbody arthrodesis stabilizes the motion segment, eliminating pathological motion, and arresting further spondylolisthesis.[20,31,46,60] It is well recognized that lateral intertransverse process fusion does not reliably fuse the segment or prevent progression of spondylolisthesis.[9,29,37,42,73] Furthermore, there have been reports of persistent back pain despite intact posterolateral fusion.[71] Posterior lumbar interbody fusion minimizes the risk of recalcitrant pain in such cases of internal disc disruption or discogenic pain.

Fig. 2. Plain lateral lumbar radiograph demonstrating a postlaminectomy spondylolisthesis at L4-5 in a 65-year-old woman (left). Postoperative radiographs at 3- (middle) and 9-month follow-up (right) examinations show reduction of spondylolisthesis with transpedicular fixation and solid interbody fusion.

Because the graft is placed under compression, enhancing primary bone healing, PLIF is a biomechanically superior fusion technique.[6,13,44] Summation of loading is greatest at the lowest lumbar segments where 80% of weight bearing stress occurs across the interbody space.[8,13,60] Theoretically, because PLIF provides a load-bearing graft, the procedure should result in superior fusion rates that can be further augmented by posterior fixation through the same exposure. Posterior lumbar interbody fusion provides the graft with a wide area of bone surface contact and a copious blood supply, as well as placing the anterior and posterior ligaments in tension.[6,13,46] The documented risk of recurrent stenosis caused by bone overgrowth of a posterior fusion mass is avoided with the interbody graft.[56]

Decompressive laminectomy deals with one symptomatic aspect of the complex disease process leading to lumbar stenosis. This procedure is designed simply to relieve neural compression without regard to the underlying pathophysiological mechanisms responsible for creating the condition and, in the process, may serve to exacerbate the underlying disease. However, PLIF allows for neural decompression while addressing a number of the multifactorial causes of stenosis including pathological changes in the disc space and facet complexes. Furthermore, successful interbody fusion directly treats the common symptom of back pain associated with segmental instability originating from the disc and facets as well as providing prophylaxis against future instability and spondylolisthesis. It is apparent that PLIF, unlike decompressive laminectomy, does not contribute to the degenerative cascade in lumbar stenosis and may serve to arrest this process; this supposition has been supported by limited reports of clinical experience. Lin, et al.,[46] reported a 74% satisfactory clinical result and a 93% fusion rate in 71 patients with spinal stenosis treated with PLIF. Hutter[31] reviewed 142 patients with spinal stenosis treated with PLIF and obtained good or excellent results in 78% as well as a 91% fusion rate.

Technical innovations, including specialized instruments and graft materials, continue to decrease the operative morbidity rates and time associated with PLIF. The use of allograft bone avoids the significant

morbidity and time associated with autograft harvest. Newer interbody devices, including the recently Federal Drug Administration-approved threaded titanium cage constructs, offer the advantages of relative ease of application and increased rigidity (Fig. 3). Long-term follow-up review is necessary to confirm the early promise of these new interbody devices, especially regarding pseudarthrosis rates and the potential of stress shielding of the graft inside the stiff cage construct.

Fig. 3. Plain lateral lumbar radiograph demonstrating interbody grafts at L3-4 and L4-5 with titanium threaded cages. The patient is a 58-year-old man who presented with lumbar stenosis at L4-5 secondary to a large, recurrent disc herniation and ligamentous hypertrophy. The patient had undergone three previous lumbar operations.

DISCUSSION

The current surgical treatment of lumbar spinal stenosis is not entirely satisfactory. Several recent, well-designed studies underscore the need for more effective treatments and outcome measures.[18,39,40,51,68-70] Katz and associates[39] prospectively studied 194 patients treated with decompressive laminectomy for degenerative lumbar stenosis and reported that 22% of patients were somewhat or very dissatisfied with the results of surgery 6 months postoperatively. McCullen, et al.,[50] reviewing 118 patients who underwent lumbar decompression for acquired spinal stenosis, reported 50% good, 27% fair, and 23% poor outcomes with an overall reoperation rate of 9%. Furthermore, preoperative spondylolisthesis as well as progressive postoperative slippage clearly predisposed to poor outcome. Tuite and coworkers[68,69] reviewed 119 patients treated by decompressive lumbar laminectomy and found 66% good outcomes and 34% bad outcomes with a 15% reoperation rate. They

also reported that surgical levels undergoing laminectomy were significantly more likely to develop progressive spinal deformity, including progressive spondylolisthesis, disc space angulation, and decreased disc space height. Herno and associates[27] reported 7- and 13-year follow-up intervals in 108 patients with lumbar canal stenosis treated with decompressive laminectomy and found good-to-excellent outcomes in 67% and 69%, respectively. Caputy and Luessenhop[11] reported a 16% incidence of recurrent stenosis in 100 patients with degenerative stenosis treated by decompressive surgery. Silvers and associates[66] reviewed the long-term outcomes of 258 consecutive patients treated by means of decompressive laminectomies and reported only 64% pain relief and 56% return to normal activity rates. In 1992, Turner and associates[70] attempted a metaanalysis of the literature evaluating the effects of surgery for lumbar stenosis and reported an average of 64% good-to-excellent outcomes. In 1991, Katz and associates[40] reviewed 88 consecutive patients treated with decompressive laminectomy and concluded that the long-term outcome for lumbar stenosis was less favorable than had previously been reported in the literature. They reported an 18% reoperation rate with 43% of all patients meeting the criteria for poor outcome. Hopp and Tsou[30] reviewed 344 patients surgically treated for spinal stenosis and reported a 17% reoperation rate due to postdecompression lumbar instability. Recently, Fox and colleagues[22] retrospectively evaluated 124 patients with degenerative lumbar stenosis treated with and without concomitant arthrodesis and reported better outcomes in patients treated with fusion.

Feffer, et al.,[21] compared two groups of patients with stenosis secondary to degenerative spondylolisthesis and found that patients undergoing decompression and fusion had more favorable outcomes than those treated with decompression alone. Herkowitz and Kurz[26] prospectively studied 50 patients with spinal stenosis associated with degenerative lumbar spondylolisthesis. They compared decompressive laminectomy alone versus decompression with intertransverse process fusion and found significantly better relief of back and lower-extremity pain in the latter group (44% vs. 96%, respectively), despite a pseudarthrosis rate of 36% in the fusion group. Furthermore, patients treated with decompression alone reported significantly more residual pain. Increased spondylolisthesis was noted postoperatively in 28% of patients treated with arthrodesis and in 96% of patients treated with decompression alone. Mardjetko and associates[48] completed a metaanalysis of the literature from 1970 to 1993 addressing the treatment of stenosis due to degenerative lumbar spondylolisthesis. These authors reported significantly poorer results in patients treated with decompression alone (70% success rate) compared with patients who underwent fusion with and without instrumentation (86% and 90% success rates, respectively).

Several factors appear to predispose patients with lumbar stenosis to poor outcomes. Numerous studies have implicated degenerative spondylolisthesis as a poor prognostic indicator.[9,26,30,36,42,50,61,65,68,69,73] Several authors have reported less favorable outcomes in women,[9,19,27,34,50,65,68,69] younger patients,[32,64,67,73] and patients undergoing repeated lumbar surgery.[15,28,52,61,72] Patient characteristics that warrant the consideration of arthrodesis (PLIF) in addition to decompression are listed in Table 1.

TABLE 1 PATIENT CHARACTERISTICS FAVORING PLIF
spondylolisthesis (>3 mm slippage) spinal instability (>2 mm motion on dynamic radiographs) postdecompression stenosis postfusion adjacent-level stenosis younger age (<60 yrs) no complicating medical conditions (osteoporosis, diabetes mellitus)

In an effort to reduce poor outcomes and recurrent stenosis, some surgeons have attempted to identify a subgroup of patients with lumbar stenosis who would benefit from spinal arthrodesis.[9,18,21,26,30,49,50,52] Spinal fusion, PLIF in particular, is technically more demanding to perform than decompressive surgery. Potential long-term consequences of segmental fusion, notably accelerated adjacent-level degeneration, must be further investigated and characterized. The higher operative morbidity rate and initial cost associated with arthrodesis warrant careful patient screening and appropriate selection of a high-risk population that would ultimately benefit from this procedure. Future prospective studies using standardized outcome measures with careful patient selection and long-term follow-up review are necessary to evaluate specific preoperative characteristics of patients with lumbar stenosis who would benefit from arthrodesis compared with decompression alone.

CONCLUSIONS

The pathogenesis of lumbar spinal stenosis is multifactorial. Intuitively, no single procedure can be successfully applied to treat such a diverse diagnostic entity. Using conventional decompressive surgery alone, there are relatively high failure (30-40%) and reoperation (10-20%) rates. The judicious application of PLIF, using strict patient selection criteria, has the potential to raise the overall success rate of operative intervention in the treatment of lumbar stenosis to a more acceptable level. There is a subgroup of high-risk patients in whom the spine is unstable preoperatively or becomes destabilized following decompression who would benefit from an initial fusion procedure. Therapeutic success would be significantly improved if this subgroup could be identified on the basis of preoperative characteristics. Treatment planning in each individual case should encompass such factors as the predominant site of neural compression (canal, lateral recess, or foramen) causing clinical symptoms; the predominant pathological process (bone encroachment, ligamentous hypertrophy, spondylolisthesis, buckling annulus/herniated disc) leading to neural compression; and high-risk factors (age, sex, underlying medical condition, previous surgery, spinal stability, and preoperative slippage). Posterior lumbar interbody fusion should be considered in complex cases of lumbar stenosis, most notably in younger, female patients with postlaminectomy stenosis or stenosis associated with spondylolisthesis.

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Address reprint requests to: Domagoj Coric, M.D., Department of Neurosurgery, Bowman Gray School of Medicine, Wake Forest University, Medical Center Boulevard, Winston-Salem, North Carolina 27157-1029.