Acquired lumbar stenosis: topic review and a case series

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Stenosis of the central and lateral lumbar vertebral canal can be congenital or acquired; the latter is most often caused by a degenerative process. The associated neurogenic claudication and/or radiculopathic symptom complexes are thought to result from compression of the cauda equina and lumbosacral nerve roots by hypertrophy of or encroachment by any combination of the following: canal walls, ligamenta flava, intervertebral discs, posterior longitudinal ligament, or epidural fat.

The authors' technique for the treatment of lumbar stenosis involves extensive unilateral decompression with undercutting of the spinous process and obviates the need for instrumentation by using a contralateral autologous bone fusion. The results in a series of 29 patients in whom the procedure was performed suggest that this decompression method safely and successfully treats not only the radicular symptoms caused by lateral stenosis but also the neurogenic claudication symptoms associated with central stenosis. In addition, the procedure can preserve spinal stability without instrumentation by using contralateral autologous bone fusion along the laminae and spinous processes.

Key Words * lumbar stenosis * neurogenic claudication * lateral recess stenosis * low-back pain

PATHOLOGICAL CONSIDERATIONS

The spinal canal is bordered anteriorly by the vertebral body, intervertebral disc, and posterior longitudinal ligament; posteriorly by the laminae, pars interarticularis, ligamenta flava, and facet joints; and laterally by the pedicles (Fig. 1).[13] Hypertrophy of any of these structures can narrow the central canal (Fig. 2). The central canal is narrowed anteriorly by central intervertebral disc bulging and vertebral body osteophytes; posteriorly by enlarged laminae, hypertrophied inferior facets, which are often pronounced, and ligamenta flava, which are hypertrophied at the caudal edge of the laminae.[1,3,19,22,23,27,33,35,40] At times, marked superior facet hypertrophy can also narrow the central canal.
Fig. 1. Artist's rendering of a normal axial view at a single lower lumbar vertebral level. Each of the bony and ligamentous elements captioned can be involved in spinal stenosis. Note the oblique orientation of the facet joints, placing this level in the lower lumbar region.
The disease process of lateral stenosis most commonly involves the superior facet, ligamentum flavum, intervertebral disc, and pedicle. The lateral recess may be narrowed posteriorly by hypertrophy of the superior facet or ligamentum flavum, anteriorly by a laterally bulging disc, or superiorly by a descending pedicle.[1-3,8,9,14,19,26,34,35,40,41] Significant inferior facet hypertrophy may also narrow the lateral recess.

Farfan and Sullivan[11,12] described the three-joint complex as a functional spinal unit composed of the intervertebral disc and facets. Kirkaldy-Willis and colleagues[19,40] popularized this concept of spinal anatomy and hypothesized that acquired spinal stenosis begins with the degeneration of one of these joints, usually the intervertebral disc. Being a complex, degeneration of one joint ultimately affects the other two, leading to degeneration of each joint in the trio.[19,40] The ensuing degenerative changes may include bulging of the disc and hypertrophy of the ligamentum flavum and the superior and inferior facets.[6,7,11-13,18,19,37,40] Generally, superior facet hypertrophy narrows the intervertebral foramen and lateral recess and inferior facet hypertrophy projects medially into the central canal.[2-4,8,9,11-13,19,27,33,36,40] The manner in which these degenerative changes occur leads to two overlapping but distinguishable pathological and clinical entities: central and lateral stenosis.

**CLINICAL PRESENTATION**

Lumbar stenosis commonly produces symptoms of either radiculopathy or neurogenic claudication. Radicular symptoms are most likely caused by lateral stenosis, whereas neurogenic claudication is probably the result of narrowing of the central region of the spinal canal. Seventy-eight percent of our patients presenting with radiculopathy experienced pain that was worse in or limited to one leg. Similarly, 75% of our patients with neurogenic claudication report that their pain is significantly worse on one side, although symptoms typically occur in both legs. This asymmetry permits us to choose the most appropriate side to decompress fully.

**NATURAL HISTORY**

There are few reports of conservative treatment of symptomatic spinal stenosis. In a recent study of 32 patients with myelogram-proven spinal stenosis, neurogenic claudication was found in 75%, radicular symptoms in 12.5%, and mixed symptoms in another 12.5%. After a mean follow-up time of 49 months, pain was unchanged in 70%, worse in 15%, and improved in another 15% of patients. Although no patient experienced neurological deterioration, the fact that 85% of the pain these patients experienced worsened or continued unchanged during long-term follow-up review makes surgery a reasonable option.[17]

**CURRENT TREATMENT**

To correct spinal stenosis, most spine surgeons perform bilateral laminectomy with foraminotomy, with or without spinal fusion. More extensive decompression is often indicated but it is believed that removal of as little as 30% of the facet joint can produce lumbar instability.[28] To counteract this destabilization, many surgeons now use spinal instrumentation for internal fixation.[21,24,29] Bone fractures, instrument failure, neurological compromise, increased blood loss, erosion into great vessels, increased operative time, extended hospitalization, and infection of the instrument are among the complications and drawbacks reported to be associated with this method of fixation.[10,28,31] In addition, instrumentation
makes it extremely difficult to obtain adequate postoperative radiographic studies because the resulting artifact generated during magnetic resonance imaging frequently obscures any lumbar abnormalities.

Other surgeons use autologous bone grafts, with or without instrumentation, to provide added stability to extensive decompressions. In the past, autologous bone fusion had routinely required autograft harvest from a second site, usually the iliac crest; this method of fixation has its own set of potentially serious complications, which include infection, increased blood loss, increased operative time, graft-site fatigue fracture, iliac hernia, hematoma formation, pelvic instability, and painful scarring.[28,30] Additionally, it has been our experience that patients who undergo transverse process fusion experience much more postoperative pain, probably secondary to a necessarily more extensive dissection of the paraspinous musculature. Therefore, the development of a spinal fusion technique that produces a high rate of fusion without the use of instrumentation or separate autograft harvest would be ideal for concomitant use with decompressive surgery.

**THE IPSI-CONTRA TECHNIQUE**

This technique involves a midline approach that permits total unilateral decompression of affected nerve roots and bilateral decompression of a stenotic central canal by combining extensive ipsilateral decompression at the facets and pedicles with ipsilateral hemilaminectomy through which removal of the ligamentum flavum and undercutting of the spinous process and the ventral surface of the contralateral lamina is performed (Fig. 3). The placement of a large, multilevel, autologous bone fusion graft eliminates the need for internal fixation.

The first goal of the surgery is to enlarge the size of the canal, which is achieved with an upbiting punch drill to remove bone extending to the contralateral side underneath the spinous process. The dura is carefully dissected away. The contralateral ligamentum flavum is also removed with the aid of a blunt nerve hook. This ligamentum is engaged and pulled toward the operating surgeon. The obliquely directed ligamentum is easily removed, and it is also necessary to remove the more horizontal ligamentum flavum that inserts on the superior facet of the opposite side. Using this maneuver it is usually possible to...
decompress the contralateral side enough so that the nerve roots are visualized. If the contralateral decompression still seems inadequate, the surgeon drills through the facet capsule into the facet and spinal canal. Direct decompression of the nerve is performed, and small pieces of fat are then placed into the facet.

The surgeon must then decompress the ipsilateral lateral recess; this is best accomplished while standing on the opposite side of the operating table. The nerve roots are followed out to the lateral recess, and bone is removed as necessary to decompress the nerve root. This often involves removing the inferior facet, thereby exposing the surface of the superior facet. As much of the superior facet as necessary is removed and the nerve is followed out around the pedicle. Often it appears that the nerve root is tethered to the surface of the pedicle. When this is the case, the pedicle is hollowed out with a drill and a curette is used to break down the remaining shell (Fig. 4A-F). The decompressed nerve root often subsequently moves into the space previously occupied by the pedicle (Fig. 4G and H).
Fig. 4. An intraoperative photograph (A) and an artist's rendering (B) of the coring procedure; the patient's head is turned to the right. An intraoperative photograph (C), a drawing in axial view (D), a cadaveric dissection (E), and its illustration (F) of the cracking procedure of the cored pedicle. A cadaveric dissection (G) and its drawing (H) showing how the decompressed pedicle moves into the space previously occupied by the pedicle.

The contralateral bone is then decorticated, including the facet, lamina, and spinous process (Fig. 5). All of the bone that has been removed during the procedure is then added together and firmly packed into the contralateral space (Fig. 6). The muscle is approximated in the midline.

Fig. 5. Artist's rendering showing the decortication of the contralateral spine in preparation for autologous bone graft.
Surgical Considerations

Our method of treating lumbar stenosis tailors the surgical approach not only to the radiographic findings but also to the specific signs and symptoms exhibited by the patient.

Patients who primarily exhibit unilateral radiculopathy and in whom radiographic studies demonstrate evidence of spinal stenosis are considered to suffer from lateral stenosis. This lateral stenosis is usually related to the superior facet but may often be due to inferior facet and ligamentum flavum hypertrophy, scoliosis, or pedicle impingement. An extensive unilateral decompression is performed in these individuals so that the affected nerve root is completely decompressed. This commonly involves a unilateral hemilaminectomy, foraminotomy, pediculectomy, facetectomy, and, occasionally, discectomy. The side without symptoms is not attacked surgically, except perhaps to remove the contralateral ligamentum flavum via the undercut spinous processes and laminae.

Patients who present with unilateral neurogenic intermittent claudication and in whom radiological confirmation of central stenosis can be made undergo an extensive unilateral decompression with hemilaminectomies. Enlargement of the contralateral spinal canal is achieved by extensive undercutting of the spinous processes and total removal of the contralateral ligamentum flavum. We believe that this approach consistently enlarges both sides of the canal while maintaining the anatomical integrity of the spinous processes and contralateral laminae and facets.

In patients who exhibit bilateral neurogenic claudication, the side on which the decompression is performed is determined by comparing the severity of pain in each leg. The more symptomatic side is chosen for major decompression. In patients with claudication or radiculopathy who experience
intolerable bilateral pain and in whom imaging studies demonstrate severe bilateral lateral stenosis, the side with the worst pain is decompressed extensively. In addition, the side with less pain is treated by removing the ligamentum flavum and performing either limited foraminotomies or a transfacetal decompression to free the exiting nerve root (Fig. 7). Again, this leaves the spinous processes, the majority of the laminae, and the facets mechanically intact.

![Contralateral Transfacetal Approach](image)

Fig. 7. Illustration showing an axial view of the contralateral transfacetal approach. This approach may be used in patients with bilateral symptoms and permits a more adequate removal of the ligamentum flavum.

**RESULTS**

Our initial results using this procedure have been published previously.[5] The ipsi-contra procedure was performed in 29 patients over a 19-month period ending in December 1991. The mean patient age was 64 years; only two individuals were younger than 50 years of age. All individuals had been unresponsive to conservative treatment and presented with low-back pain in addition to signs and symptoms consistent with pseudoclaudication or radiculopathy. Nine patients had undergone previous lumbar decompressive surgery. The minimum and mean postoperative follow-up times were 24 and 30 months, respectively.
Of the patients with neurogenic claudication, 69% reported complete pain relief at follow-up review. Of those patients with radicular symptoms, 41% had complete relief and another 23% had mild residual pain, which was rated 3 or less on a pain functionality scale of 0 to 10. For the entire cohort, this surgery decreased pain from 9.2 to 3.3 (paired Student's t-test; dependent t = 8.73, p < 0.0001) on this scale. Patient satisfaction with surgery was 69%. Low-back pain was significantly relieved in 62% of all patients (sign test; 10.0, p < 0.0001). Low-back pain relief correlated negatively with the number of levels decompressed (Chi square test; 3.90, p < 0.05). Total success of bone fusion was difficult to evaluate; however, in follow-up studies radiographic evidence revealed effective fusion and immobility of the affected spine (Figs. 8 and 9).
showing the postoperative result of a left-sided decompression and contralateral fusion.

**DISCUSSION**

In this study, we demonstrated that the unilateral decompression/contralateral fusion technique relieved pain in 14 (64%) of 22 patients who exhibited radicular symptoms. In nine (41%) of these 22 patients, pain was completely relieved. Of the eight patients whose pain functionality score at follow-up review was greater than 3 of 10, only four reported no pain relief from surgery. Therefore, it would seem that extensive unilateral decompression can adequately treat these symptoms and is likely to be more effective than a less extensive lateral neural decompression and less destabilizing than an extensive bilateral decompression. When necessary in patients with severe bilateral radiculopathies, this technique permits the performance of limited partial hemilaminectomies and foraminotomies on the fused side without the need for facetectomy and without causing gross instability.

Unlike the more lateral or localized neural compression suspected with radicular symptoms, intermittent neurogenic claudication is probably the result of a diffuse central narrowing of the spinal canal. Although the exact cause of neurogenic claudication is still the subject of debate, its symptomatic presentation is generally thought to be bilateral in approximately 75% of these patients. Unilateral laminectomy, facetectomy, and pediculotomy, with extensive undercutting of the spinous processes and removal of hypertrophic ligamentum flavum on the contralateral side, enlarge both sides of the spinal canal to treat bilateral intermittent neurogenic claudication symptoms. Of the 16 patients who reported preoperative intermittent neurogenic claudication symptoms, 11 (69%) reported complete relief of these symptoms at the follow-up review and one reported only mild residual pain, rated 3 or less of 10 on the pain functionality scale. This suggests that the unilateral decompression described here creates adequate room for the thecal sac on both sides of the spinal canal. It is interesting to note that of the 10 patients who presented with bilateral claudication symptoms, eight reported complete relief postoperatively.

Regardless of the cause, the unilateral decompression/contralateral fusion technique addressed the disease that resulted in both neurogenic claudication and radicular symptoms. In all patients, the surgery produced a significant decrease in pain: 62% reported little or no pain at follow-up examination and 45% were completely free of pain. Additionally, 20 (69%) of 29 patients were satisfied with their surgery. Of the nine patients with both types of symptoms, seven had complete relief of pain and only one had pain greater than 3 of 10 on the pain functionality scale at follow-up review.

**CONCLUSIONS**

We believe that an extensive lateral decompression is typically needed only on one side to treat radiculopathy, whereas a bilateral decompression of the spinal canal is commonly used to treat claudication. To treat both radiculopathy and claudication, we use: 1) a midline approach for radical unilateral decompression of the nerve roots on the side with radicular symptoms or the more symptomatic neurogenic claudication symptoms; 2) generous undercutting of the spinous processes; and 3) removal of the contralateral ligamentum flavum, which enlarges the spinal canal centrally and bilaterally. In addition, a contralateral, multilevel autologous bone fusion without instrumentation prevents any subtle or gross instability and maintains the surgical decompression.

**Future Treatment**

Although the general treatment of lumbar stenosis has not dramatically changed in the past two decades, several potential improvements are on the horizon: spinal instrumentation and advances in molecular...
approaches to improve bone deposition at the fusion site offer promise.[38,39] Recent advances in molecular biology have made it possible to identify bone growth factors that can be used to promote bone fusion in the lumbar spine.[4] It has been demonstrated in a canine model that when bone morphogenic protein-2 is added to an autologous bone graft, the amount of bone deposition at the fusion site, as well as the rate of solid bone fusion, is greatly enhanced.[15] In addition, type I collagen gel added to the autologous bone graft provides an excellent matrix onto which osteoblasts can migrate, thereby improving bone deposition between the autologous bone chips.[15,32] Collagen may also be an excellent carrier for osteogenic growth factors and may actually bind circulating growth factors that reach the fusion site.[25]

References


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