The Effects of an Interspinous Implant on Intervertebral Disc Pressures

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Study Design. Measurement of intradiscal pressure was performed after placement of an interspinous implant in a cadaver model.

Objective. To understand the likelihood of accelerated adjacent-level disc degeneration as a result of the implant.

Summary of Background Data. An interspinous implant has been developed to treat lumbar neurogenic claudication secondary to spinal stenosis that places the stenotic segment in slight flexion and prevents extension. Previous biomechanical studies demonstrated that fusing one level may significantly increase the intradiscal pressures at adjacent levels. Moreover, clinical studies have reported an increased incidence of adjacent-level degeneration after lumbar spinal fusion.

Methods. Eight cadaver lumbar specimens (L2–L5) were loaded in flexion, neutral, and extension. A pressure transducer measured intradiscal pressure and annular stresses during each of the three positions at each of the three disc levels. An appropriately sized implant was placed at L3–L4, and the pressure measurements were repeated.

Results. The pressures at the adjacent discs were not significantly affected by the interspinous implant insertion. There was a significant decrease in intradiscal pressure at the L3–L4 disc in the posterior annulus and nucleus in the neutral and extended positions.

Conclusions. The implant does not significantly change the intradiscal pressures at the adjacent levels, yet it significantly unloads the intervertebral disc at the instrumented level in the neutral and extended positions. On the basis of the current findings, it does not appear that the implant causes accelerated disc degeneration at the adjacent levels. [Key words: disc pressure, interspinous implant, neurogenic intermittent claudication, spinal stenosis] **Spine 2003;28:26–32**

Lumbar neurogenic claudication secondary to spinal stenosis is a disabling condition that causes lower back pain and lower extremity pain resulting from narrowing of the spinal canal.[9,21,40,46,53–55] Degenerative lumbar spinal stenosis is the most common type, with a reported incidence of 1.7% to 8% of the general population.[23] Most of these patients initially present in the fifth or sixth decade of life with lower back or lower extremity pain. The pathogenesis of degenerative lumbar stenosis begins with degeneration of the posterolateral annulus, advancing to disc herniation and resorption, then to instability with loss of disc height, and finally to stenosis from hypertrophy of the facet joints.[5,27] Loss of disc height may also cause thickening or "buckling" of the ligamentum flavum at the affected level, contributing to narrowing of the spinal canal.[11,18,41]

Verbiest[53] first described neurogenic intermittent claudication and attributed the pathology to narrowing of the spinal canal and posture. Neurogenic claudication is characterized by one or more of the following symptoms: pain, paresthesias, or decreased sensation and motor power in the legs during walking or standing with relief of symptoms during resting and sitting.[40,53] While the patient is walking, he or she gradually bends forward to relieve symptoms, ultimately needing to stop for complete relief.[40] These symptoms are observed mostly because narrowing of the neural foramen results in impingement of the exiting nerve root.[17,53] Other factors may include radicular ischemia and soft tissue impingement.[40] Lumbar extension, as seen with standing or walking, exacerbates symptoms by decreasing the foraminal width, height, and area at the exiting nerve root, whereas flexion, as seen with sitting, causes improvement of symptoms by increasing the cross-sectional area of the foramen.[17,24,38,45,49,50,56,59,62]

Currently, patients with symptomatic lumbar stenosis have treatment options that range from conservative (nonsteroidal antiinflammatory drugs [NSAIDs], physical therapy, epidural steroid injection, and bracing) to surgical (decompressive laminectomy with or without fusion and instrumentation).[23] Several studies have shown that operative treatment gives better results for relief of symptoms, but there are associated risks, especially in older patients.[22,25,42] It has been estimated that by 2025, the population older than 65 years will have almost doubled.[42] Less morbid surgical options will need to be developed for those considered nonsurgical patients who have failed conservative treatment.
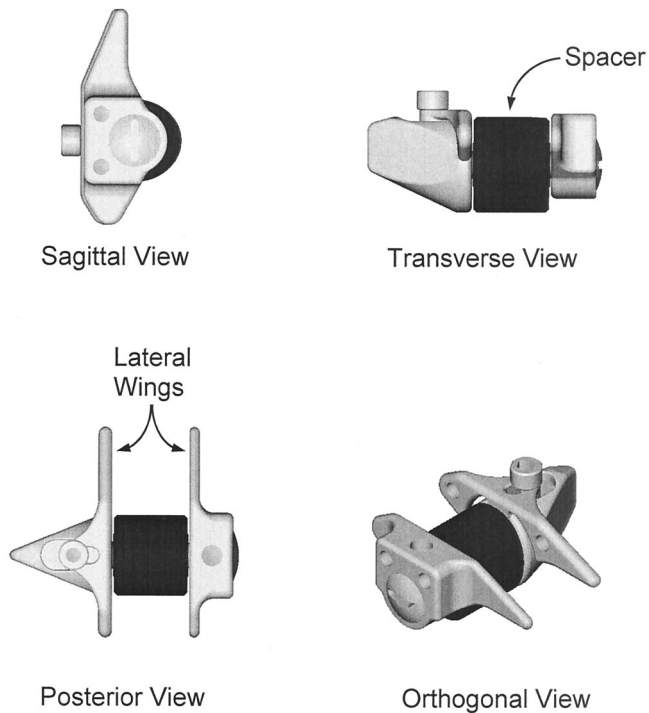
Figure 1. A schematic of the X-Stop implant in three planar views and an orthogonal view. The spacer rests between the spinous processes, and the lateral wings rest lateral to the spinous processes.

A newly developed spinal implant has been designed to treat symptomatic lumbar stenosis, especially in patients with neurogenic claudication who obtain nearly complete relief in sitting or flexing. The device is composed of an oval spacer made of titanium placed between two adjacent lumbar spinous processes (Figure 1). There are two lateral wings on each end of the implant that keep it in place and prevent the implant from migrating laterally. Surgical implantation is performed with the patient under local anesthesia in the lateral decubitus position. The procedure requires muscular stripping of only the paraspinal muscles attached to the spinous processes and not the laminae, and does not require removal or transection of any midline structures. The implant is placed in the interspinous space with the patient in a slightly flexed position. The intent is to position the stenotic segment in slight flexion, and by preventing extension, to relieve the symptoms of lumbar spinal stenosis.

The intent of the current study was to understand changes in intradiscal pressure that occur after placement of the implant. A study by Adams et al[3] measured pressures in the intervertebral disc after applying physiologic loads. They observed that the pressures in the posterior annulus increased with extension and decreased with flexion in discs that were not degenerative. When the discs were subjected to degenerative treatment, there was a paradoxical decrease in posterior annular pressure during extension in most specimens. This was explained by possible stress shielding of the posterior annulus by the zygapophysial joints. Another study by

Cunningham et al[12] reported the changes in intervertebral disc pressures after destabilization of the lumbar spine followed by stabilization with instrumentation. Stabilization with posterior instrumentation resulted in a significant increase in pressure at the adjacent levels. Conversely, others have shown little change in disc pressure with lumbar instrumentation.[10,43,44] There is strong evidence based on clinical and biomechanical findings that increased disc pressure leads to disc degeneration.[12,16,20,30,48,57] A possible concern with the current implant is how the implant affects the disc pressures at the adjacent levels of insertion and at the level of insertion. The authors hypothesized that placement of an interspinous implant would cause a decrease in the intervertebral disc pressure at the level of instrumentation without significantly affecting the disc pressures at the adjacent levels.

■ **Materials and Methods**

Eight cadaver lumbar spines were obtained from donors ages 56 to 80 years and stored at −22 C. The specimens were thawed and separated into motion segments consisting of four vertebrae (L2–L5) and three corresponding vertebral discs. Each specimen was debrided of muscle and adipose tissue with the ligamentous structures left intact. Polymethylmethacrylate (PMMA) was used to secure L2 and L5 endplates. The specimens were then placed in a freezer and stored at −22 C until they were needed for testing.

At the time of the experiment, the specimens were thawed to room temp (22 C) and loaded onto a computer-controlled hydraulic materials testing machine capable of applying independent axial loads and bending moments (MTS 858, Eden Prairie, MN).

The specimens were wrapped in a polyethylene sheet to keep them hydrated during the experiment.[2] Before testing, with the spines placed in the neutral position, a compressive force of 300 N was applied to each specimen for 15 minutes. This technique was performed to precondition the specimens and reduce any postmortem superhydration effects of the intervertebral discs.[2–4] This was done once for each specimen.

A pressure transducer with a diameter of 1.3 mm (Gaeltec, Hackensack, NJ) was placed into the appropriate disc level with the tip just through the posterior annulus to allow for stress profilometry of the respective disc. A linear variable displacement transducer (LVDT; Sensotec, Columbus, OH) was used to measure the position of the pressure transducer as it was drawn through the disc. Both of the transducers were located on the same apparatus, allowing for simultaneous measurements of pressure and displacement (Figure 2). Initially, each motion segment was placed in the loading frame in the neutral position and subjected to an axial force of 700 N for 30 seconds, at which time the pressure transducer was pulled along the midsagittal plane of the disc being measured. A 700-N force was chosen because it is approximately the amount of force observed in the lumbar spine during sitting and has been used in similar *in vitro* disc pressure studies.[2,35] Both superior and lateral components of the compressive stress were measured by rotating the transducer needle 90° during successive tests.[4] Stress profilometry was performed for each disc (L2–L5) with the specimens in neutral, flexed, and extended positions. Flexion and extension were achieved by applying a
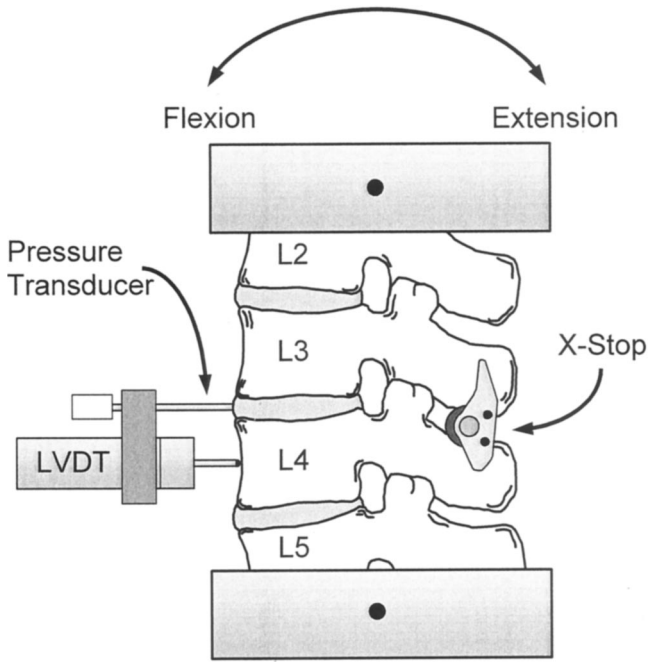
Figure 2. A schematic of the testing configuration. Spines were flexed, extended, and held in the neutral position while a pressure transducer was pulled through the disc from posterior to anterior and the linear variable displacement transducer (LVDT) simultaneously recorded the displacement of the pressure transducer.

7.5-Nm bending moment in the respective direction with a superimposed 700-N compressive load.

Next, an implant (X-Stop; St Francis Medical Technologies, Concord, CA) was placed between the L3 and L4 spinous processes. The size of the X-Stop used was dictated by the size of each individual specimen. The device was placed between the spinous processes of L3 and L4 by creating a rent through the interspinous ligament with a sharp scalpel and then dilating it to the appropriate size. After placement of the implant, the specimen was placed once again in the loading frame. The aforementioned sequence was repeated with the specimens loaded in the neutral, flexed, and extended positions. A 700-N compressive load was used in each position, and a 7.5-Nm bending moment was used to create flexion or extension. Again, a transducer was used to measure the intradiscal pressure during loading, and a displacement transducer was used to measure the travel of the pressure transducer through the disc.

A total of 12 measurements were recorded for each disc level (6 without and 6 with the X-stop). The mean pressures were compared between the intact and implanted specimens for a given level (L2–L3, L3–L4, L4–L5), specimen position (flexion, neutral, extension), transducer direction (superior, lateral), and disc region (posterior, nucleus, anterior). A total of 54 comparisons were made using individual paired t tests, each with 0.05 regarded as the level of significance.

■ Results

As expected, the most notable differences in mean disc pressure were identified at L3–L4 (Figure 3). In extension, the mean pressure in the posterior annulus was significantly reduced with the use of the implant (Figure 4, Table 1): the mean superior pressure by 63% and the mean lateral pressure by 46%. Likewise, the mean pres-
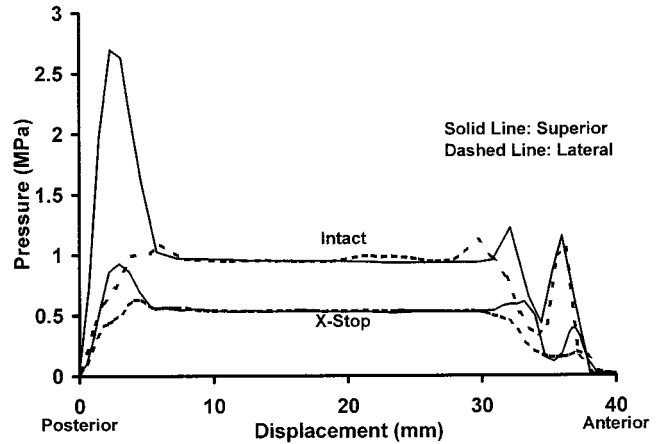

Figure 3. A representative plot of data collected at L3–L4 in extension with and without the X-Stop implant. The plot shows that the implant reduces the pressure in the posterior annulus and nucleus. Also, for a given specimen configuration (intact or X-Stop), the pressure in the nucleus does not change relative to the direction of the pressure transducer, whereas it does change in the anterior and posterior annulus. This indicates that the loading environment in the nucleus is that of hydrostatic compression, whereas the annular walls are loaded in a mixed mode.

sures in the region of the nucleus were significantly reduced after implantation (Figure 4, Table 1): the mean superior pressure by 41% and the mean lateral pressure by 40%. In the neutral position, the mean pressures in the posterior annulus and nucleus were also significantly reduced by the implant (Figure 5, Table 1): the mean superior pressure in the posterior annulus by 38% and the mean superior and lateral pressures in the nucleus by 20% and 17%, respectively. Two other comparisons were significantly different: the mean lateral pressure in the anterior annulus during extension (45%) and the

Figure 4. A bar chart of the mean pressures and standard deviations collected in the posterior annulus, nucleus, and anterior annulus of L3–L4 in extension. Pressures collected in the superior and lateral directions of specimens with and without the implant are presented. The mean superior and lateral pressures in the posterior annulus and nucleus were significantly reduced after implant placement, and the mean lateral pressure in the anterior annulus was significantly reduced.

Table 1. Mean Disc Pressures at the L3–L4 Level for the Intact and X-Stop Implanted Specimens

Position	Transducer Direction	Posterior Annulus		Nucleus		Anterior Annulus	
		Intact	X-Stop	Intact	X-Stop	Intact	X-Stop
Extension	Superior	1.88 ± 0.70[a]	0.70 ± 0.23[a]	0.87 ± 0.21[d]	0.51 ± 0.10[d]	0.94 ± 0.34	0.90 ± 0.31
Neutral	Superior	1.54 ± 0.62[b]	0.95 ± 0.32[b]	0.79 ± 0.17[e]	0.63 ± 0.09[e]	1.33 ± 0.69	1.12 ± 0.41
Flexion	Superior	1.04 ± 0.38	0.99 ± 0.25	0.84 ± 0.18[f]	0.81 ± 0.17[f]	1.91 ± 0.63	1.70 ± 0.55
Extension	Lateral	1.15 ± 0.17[c]	0.62 ± 0.11[c]	0.86 ± 0.21[g]	0.52 ± 0.09[g]	0.93 ± 0.48[i]	0.51 ± 0.22[i]
Neutral	Lateral	0.86 ± 0.18	0.75 ± 0.16	0.77 ± 0.15[h]	0.64 ± 0.09[h]	0.68 ± 0.32	0.56 ± 0.21
Flexion	Lateral	1.01 ± 0.34	0.90 ± 0.20	0.83 ± 0.18	0.82 ± 0.16	0.86 ± 0.29	0.86 ± 0.22

Means with common superscripts are significantly different at a *P* value of 0.05. Values are represented as mean ± standard deviation.

mean superior pressure in the nucleus during flexion (4%).

There were no significant differences between the mean pressures of the intact and implanted specimens at L2–L3 (Table 2). The only significant differences between the intact and implanted specimens at L4–L5 were between the lateral nucleus pressures in the neutral (7%) and flexed positions (9%) and the lateral anterior annulus pressures in the extended position (12%) (Table 3).

■ Discussion

The results of the current study suggest that the interspinous implant will not cause pressure-induced accelerated disc degeneration at levels adjacent to the implant. The most dramatic differences were observed at the level of insertion. The implant significantly decreased the intradiscal disc pressure in the posterior annulus region of the nucleus. In the extended and neutral positions, the implant appears to redirect a large portion of the load away from the intervertebral disc and to transfer that load to the spinous processes. During flexion, no appreciable change in intradiscal pressure was observed at the instrumented level, which suggests that the implant does not
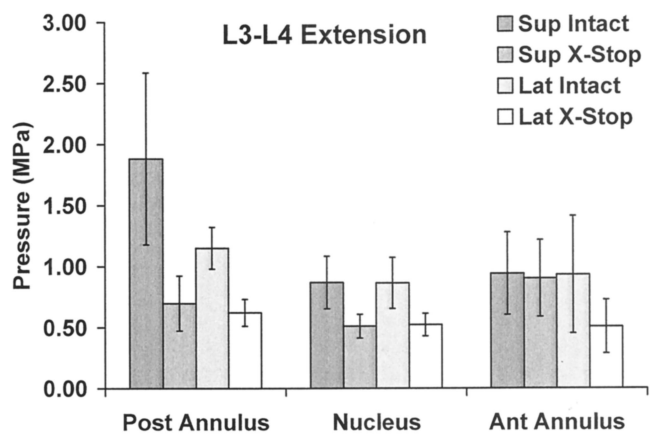

Figure 5. A bar chart of the mean pressures and standard deviations collected in the posterior annulus, nucleus, and anterior annulus of L3–L4 in the neutral position. Pressures collected in the superior and lateral directions of specimens with and without the implant are presented. The mean superior pressures in the posterior annulus and nucleus were significantly reduced after implant placement, and the mean lateral pressure in the nucleus was significantly reduced.

alter the mechanics during flexion. In an experiment, Adams et al[3] noted a paradoxical decrease in posterior annular pressure during hyperextension at the tested level. They attributed this observation to the facet joints acting as a fulcrum and redirecting most of the force from the respective disc.[3] This finding is similar to the current finding, but in the case of the implant, instead of the facet joints redirecting the force, the implant acts to transfer the load from the disc to the spinous processes.

The primary focus of the current study was to understand the intradiscal pressure mechanics at the levels adjacent to the implant and how changes in pressure at these levels may lead to pressure-induced disc degeneration. The concern about degeneration at the adjacent levels was brought about by clinical[16,30,31,39,48,63] and biomechanical[10,12,20,26,36,44,51,57] observations in lumbar spine fusion. In the case of spinal fusion, the motion segment is entirely immobilized, and the adjacent levels are forced to flex and extend appreciably more to compensate for the lack of mobility at the fused level. A number of studies have reported radiographic findings of adjacent-level changes. Aota et al[8] reported on 55 patients who had a stable adjacent level before lumbar fusion. After a mean 25-month follow-up period, an adjacent-level instability developed in 14 (25.5%) of the patients. Similarly, Etebar and Cahill[15] reviewed the records of 125 patients and reported that adjacent-level failure developed in 18 of these patients after a mean of 45 months.

However, many of these radiographic findings do not correlate with clinical symptoms. For example, Guigui et al[19] reported up to a 49% incidence of radiographic changes in 102 patients after an average follow-up period of 8.9 years. However, "no significant correlation was found between the radiographic findings and the final functional results, and only eight patients required a new surgery."[19] Similar findings have been reported by Kumar et al,[28,29] Lehmann et al,[31] Miyakoshi et al,[34] Van Horn and Bohnen,[52] and Whitecloud et al.[58] On the other hand, there are reports of symptomatic adjacent levels that correlate very well with the radiographic findings.[30,48] Lee[30] reported on 18 patients with symptomatic levels adjacent to a fusion in which 11 of the 18 patients had symptoms within 5 years. Similarly, Schlegel et al[48] reported on 58 patients treated for adjacent-
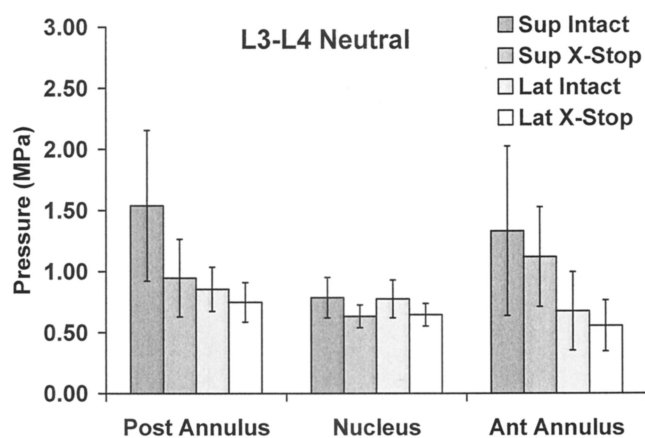
Table 2. Mean Disc Pressures at the L2–L3 Level for the Intact and X-Stop Implanted Specimens

Position	Transducer Direction	Posterior Annulus		Nucleus		Anterior Annulus	
		Intact	X-Stop	Intact	X-Stop	Intact	X-Stop
Extension	Superior	1.92 ± 1.55	1.47 ± 0.85	0.78 ± 0.31	0.77 ± 0.30	1.00 ± 0.61	1.10 ± 0.63
Neutral	Superior	1.18 ± 0.49	1.70 ± 1.05	0.74 ± 0.15	0.76 ± 0.18	1.43 ± 0.53	1.70 ± 0.80
Flexion	Superior	1.00 ± 0.18	1.04 ± 0.37	0.90 ± 0.23	0.89 ± 0.21	2.19 ± 1.20	2.56 ± 1.16
Extension	Lateral	1.12 ± 0.36	1.08 ± 0.28	0.78 ± 0.33	0.76 ± 0.30	0.63 ± 0.39	0.65 ± 0.36
Neutral	Lateral	0.91 ± 0.22	1.02 ± 0.31	0.72 ± 0.15	0.75 ± 0.16	0.81 ± 0.49	0.69 ± 0.19
Flexion	Lateral	1.05 ± 0.32	1.11 ± 0.45	0.89 ± 0.24	0.87 ± 0.21	1.08 ± 0.52	1.11 ± 0.46

There are no significant differences between the means of the intact and x-stop pressures at the L2–L3 level for any specimen position, anatomic location, or transducer position. Values are represented as mean ± standard deviation.

level disc disease who were asymptomatic for an average of 13.1 years. Among these patients, 36 were treated with adjacent-level fusions. Although the radiographic findings of adjacent-level degeneration are not disputed, their correlation to clinical symptoms is a matter of debate. For a better understanding of the mechanisms driving this degeneration, a number of investigators have taken a biomechanical approach.

Disc pressure studies are routinely performed to understand the changes occurring as a result of instrumentation. Chow et al[10] showed that after an L4–L5 fusion, the L3–L4 intradiscal pressure increased only slightly, from 0.30 to 0.31 MPa in flexion and from 0.39 to 0.41 MPa in extension. Similarly, Rohlmann et al[44] showed that an external fixator increased the pressures above and below the fused segments only slightly. On the other hand, Cunningham et al[12] showed that the adjacent-level disc pressures increase substantially during flexion and extension, whereas the pressures at the instrumented levels decrease.

A likely reason for the differences in these biomechanical studies lies in the testing mode. Some studies were performed under load control, whereas others used displacement control. There is no conclusive evidence that one testing mode is preferential over another, but convincing arguments can be made for both modes. In an attempt to identify the differences between the two and a preference of one over the other, Dekutoski et al[13] investigated an animal model followed up with biomechanical testing. On the basis of their findings, they concluded that displacement control appears to simulate the *in vivo* changes caused by immobilization. On the other hand,

Rohlmann et al[44] conducted a number of clinical and biomechanical pressure studies, concluding from their results that "during most daily activities, patients tend to accept the limited motion," and that "load control is probably therefore the adequate loading condition."

The current study was performed under load control based on the assumption that patients tend to apply a constant load instead of attempting to achieve a constant displacement. As a result of this loading modality, the magnitude of the pressures measured in the nucleus, and in anterior and posterior annulus are similar to those reported by others.[1,4,14,32,57,61] In addition, the pressures are similar to those measured in patients by Andersson et al,[6,7] Ortengren et al,[37] Sato et al,[47] Rohlmann et al,[44] and Wilke et al.[60] The pressure within the nucleus was fairly constant despite movements in flexion and extension. In addition, the pressure within the nucleus did not vary with orientation of the pressure transducer, which suggests that the pressure measured in these specimens was hydrostatic pressure. Most of the pressure changes were observed in the anterior and posterior annulus. The superior stress profiles were also more sensitive to compressive loading of the intervertebral disc than the lateral stress profiles, similar to those noted in a previous study.[32]

To the authors' knowledge, there is only one study in the reported literature that addresses change in intradiscal pressure after insertion of an interspinous implant. Minns and Walsh[33] discovered that insertion of a silicone implant between two adjacent spinous processes resulted in a decrease in disc pressure. Their experiment did not include flexion–extension of the motion seg-

Table 3. Mean Disc Pressures at the L4–L5 Level for the Intact and X-Stop Implanted Specimens

Position	Transducer Direction	Posterior Annulus		Nucleus		Anterior Annulus	
		Intact	X-Stop	Intact	X-Stop	Intact	X-Stop
Extension	Superior	2.02 ± 0.72	1.65 ± 0.94	0.81 ± 0.42	0.61 ± 0.17	0.92 ± 0.30	1.07 ± 0.57
Neutral	Superior	1.40 ± 0.77	1.21 ± 0.49	0.65 ± 0.07	0.63 ± 0.07	1.35 ± 0.65	1.10 ± 0.69
Flexion	Superior	0.82 ± 0.43	0.76 ± 0.39	0.94 ± 0.37	0.90 ± 0.31	1.77 ± 0.66	1.66 ± 0.88
Extension	Lateral	1.28 ± 0.63	0.99 ± 0.24	0.80 ± 0.34	0.63 ± 0.14	0.66 ± 0.13[c]	0.59 ± 0.13[c]
Neutral	Lateral	0.75 ± 0.31	0.93 ± 0.41	0.69 ± 0.06[a]	0.64 ± 0.07[a]	0.73 ± 0.19	0.70 ± 0.19
Flexion	Lateral	0.82 ± 0.29	0.88 ± 0.38	0.87 ± 0.39[b]	0.80 ± 0.37[b]	1.17 ± 0.33	1.07 ± 0.20

Means with common superscripts are significantly different at P value of 0.05. Values are represented as mean ± standard deviation.

ments, and they did not measure disc pressures at the adjacent levels. However, the data from the current study follow the same patterns observed by Minns and Walsh,[33] and also give some insight into the pressure changes in the anterior and posterior annulus as well as adjacent levels.

It appears that the implant would not induce any degenerative changes at the adjacent levels, and that it may have some benefit for patients with pressure-related discogenic back pain, although the latter point is pure speculation. The safety and efficacy of its intended use, lumbar neurogenic claudication secondary to spinal stenosis, is currently being evaluated in a prospective randomized clinical trial.

■ Key Points

- An interspinous spacer is presented as an alternative treatment for neurogenic claudication secondary to lumbar spinal stenosis.
- The effect of the implant on disc pressure at the instrumented level and adjacent levels was evaluated.
- The implant did not affect the disc pressure at the adjacent levels and reduced the disc pressure at the instrumented level.

Acknowledgments

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