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The Treatment Mechanism of an Interspinous Process Implant for Lumbar Neurogenic Intermittent Claudication

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Study Design. The spinal canal and neural foramina dimensions of cadaver lumbar spines were quantified during flexion and extension using magnetic resonance imaging before and after placement of an interspinous process implant.

Objective. To quantify the effect of the implant on the dimensions of the spinal canal and neural foramina during flexion and extension.

Summary of the Background Data. Lumbar neurogenic intermittent claudication symptoms are typically exacerbated during extension and relieved during flexion. It is understood that the dimensions of the spinal canal and neural foramen increase in flexion and decrease in extension. The authors hypothesized that an interspinous process implant would significantly prevent narrowing of the canal and foramina in extension and have no significant effect in flexion.

Methods. Eight L2–L5 specimens were positioned to 15° of flexion and 15° of extension using a positioning frame. Each specimen was magnetic resonance imaged with and without an interspinous implant (X STOP) placed between the L3–L4 spinous processes. Canal and foramina dimensions were compared between the intact and implanted specimens using a repeated measures analysis of variance with a level of significance of 0.05.

Results. In extension, the implant significantly increased the canal area by 18% (231–273 mm^2), the sub articular diameter by 50% (2.5–3.7 mm), the canal diameter by 10% (17.8–19.5 mm), the foraminal area by 25% (106–133 mm^2), and the foraminal width by 41% (3.4–4.8 mm).

Conclusions. The results of this study show that the X STOP interspinous process implant prevents narrowing of the spinal canal and foramina in extension.

Key words: lumbar spinal stenosis, neurogenic intermittent claudication, spinal canal, neural foramen, biomechanics. **Spine 2005;30:744–749**

Neurogenic intermittent claudication (NIC) secondary to lumbar spinal stenosis (LSS) has been described and proved to be a posture-dependent condition in which symptoms such as lower limb tingling, pain, and numbness are typically exacerbated in extension and relieved in flexion. Stenotic symptoms caused by thickened ligamentum flava were described by Towne and Reichert in 1931[1] and Spurling *et al* in 1937.[2] However, the posture-dependency of the condition was not described until 20 years later by Verbiest.[3] The 2 decades following Verbiest's initial findings include numerous publications focused on NIC.[4–20]

The posture-dependent nature of NIC is well understood, and the mechanisms have been described in a number of biomechanical and clinical studies.[21–26] The current treatments for patients with NIC include both nonoperative therapy and surgery. Studies suggest that conservative care may be more appropriate for patients with mild symptoms, while surgery may be more suitable for patients with severe symptoms and physical limitations.[27,28] There is little consensus on the appropriate treatment for patients with moderate symptoms, although the literature seems to suggest that surgery may be more effective than nonoperative therapy.[27,28]

Decompressive surgery typically involves excision of the ligamentum flavum and partial removal of the laminae. Medial facetectomies and foraminotomies are often performed as well, depending on the source of the stenosis, and fusion with or without instrumentation may be necessary for concomitant segmental instability. The goal of decompressive surgery is to remove the source of neurologic compression, thus, hopefully, relieving the NIC.

Based on the findings of Verbiest and others described above who showed that patients with NIC often have symptom relief in flexion and worsening in extension, an alternative treatment for NIC has been developed. An interspinous process implant (X STOP, St. Francis Medical Technologies, Alameda, CA; Figure 1) is placed be-

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The device(s)/drug(s) that is/are the subject of this manuscript is/are being evaluated as part of an ongoing FDA-approved investigational protocol (IDE) or corresponding national protocol for intermittent claudication secondary to lumbar spinal stenosis.

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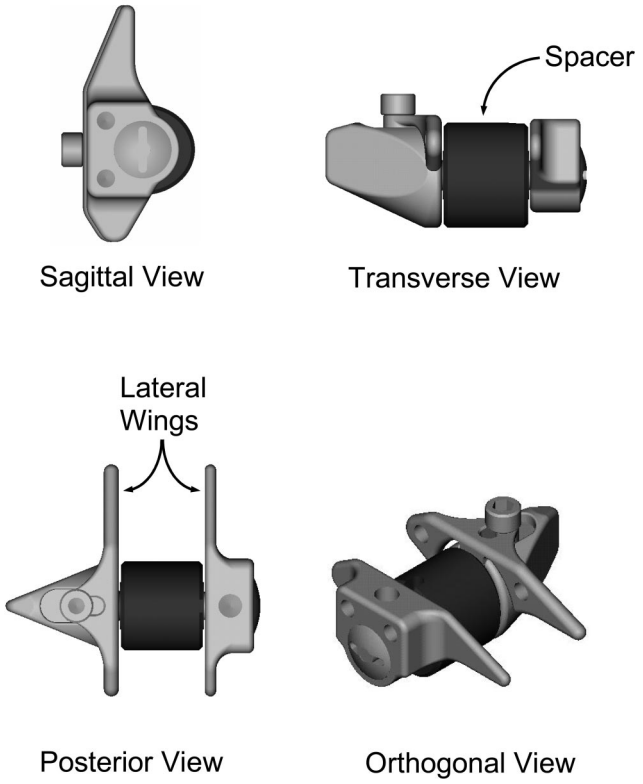

Figure 1. A schematic of the X STOP implant (St. Francis Medical Technologies, Alameda, CA). The implant is inserted laterally between the spinous processes of the affected level without the adjustable lateral wing (shown on the left of the posterior view). The implant is inserted until the spacer is between the spinous processes and then the lateral wing is secured. The implantation procedure allows the supraspinous ligament to remain intact, which prevents the implant from migrating posteriorly. Implant migration is prevented laterally and anteriorly by the lateral wings.

tween the spinous processes to prevent extension of the stenotic level(s), yet allow flexion, axial rotation, and lateral bending.[29] The implant is intended to prevent impingement of the neural structures at the stenotic level(s) that typically occurs in patients with NIC during standing and walking. Other interspinous implants have been introduced in the past, but they were typically indicated for conditions other than NIC, such as herniated nucleus pulposis, instability, and degenerative disc disease, and were often associated with fusion.[30–32] Other interspinous devices currently available, which restrict extension and flexion, are indicated for degenerative disc disease, adjacent level syndrome, LSS, and herniated disc.[33–35]

The specific aim of the current study was to quantify the effect of the interspinous process implant on the dimensions of the lumbar spinal canal and neural foramens in the flexed, neutral, and extended positions. The authors hypothesized that the implant would prevent a decrease in the dimensions during extension, and maintain the dimensions in the neutral and flexed positions. In addition, the authors hypothesized that the implant would have no significant effect on the dimensions of adjacent spinal canal or neural foramens in any position.

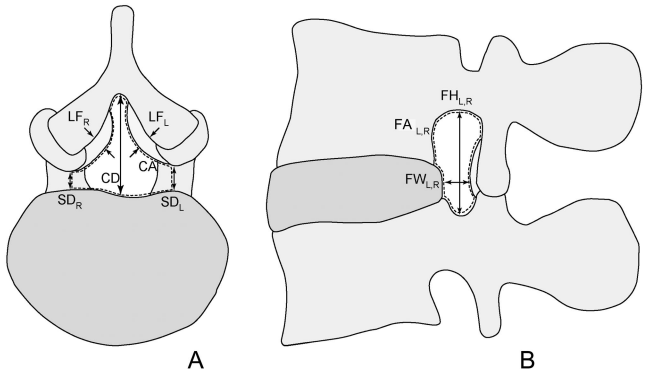

Figure 2. A schematic of the measurements made from the axial magnetic resonance imaging slices (**A**) and magnetic resonance imaging slices in the pedicular plane (**B**). CA = area of the canal; CD = midsagittal canal diameter; FA_L = left foraminal area; FA_R = right foraminal area; FH_L = left foraminal height; FH_R = right foraminal height; FW_L = left foraminal width; FW_R = right foraminal width; LF_L = left ligamentum flavum thickness; LF_R = right ligamentum flavum thickness; SD_L = left subarticular diameter; SD_R = right subarticular diameter.

■ Materials and Methods

Eight human lumbar cadaver specimens (L2–L5) were cleaned of all muscle and adipose tissue. The cranial half of the L2 vertebra and the caudal half of the L5 vertebra were secured in polymethyl methacrylate (PMMA) and placed in a custom acrylic frame. The frame was designed to maintain each specimen in the neutral position, 15° of flexion, or 15° of extension during testing. The neutral position was defined as the natural, unloaded position of each cleaned specimen. In this neutral position, the 2 PMMA blocks were parallel to each other, and the angle was defined as 0°. Flexion and extension were achieved by rotating each of the L2 and L5 PMMA blocks in 7.5° of flexion or extension relative to the neutral position to achieve the desired position.

Once in the positioning frame, the specimens were submerged in a saline bath and placed in a 1.5 Tesla magnetic resonance imaging (MRI) scanner (Signa, GE Medical Systems, Milwaukee, WI). Each specimen was scanned in 3 positions (15° flexion, neutral and 15° extension), with and without the interspinous implant placed at the L3/4 interspinous space. Four millimeter noncontiguous axial slices parallel to each intervertebral disc were used to measure the: canal area; subarticular diameter; canal diameter; and ligamentum flavum thickness at the L2/3, L3/4, and L4/5 levels (Figure 2). Three millimeter noncontiguous slices parallel to the left and right pedicular planes were used to measure the: foramen area; foramen height; and foramen width at the L2/3, L3/4, and L4/5 levels (Figure 2). The slice thickness used for all images was chosen based in the signal-to-noise ratio; attempts to acquire thinner slices in each case resulted in images that were inadequate for analysis. Custom software (Q-Brain, MRSC, University of California, San Francisco, CA) developed to analyze MRI was used to digitally measure linear dimensions and areas. Two observers, an orthopedic resident (J.C.R.) and a biomechanical engineer (S.A.Y.), made each measurement.

The left and right side measurements of the subarticular diameter, ligamentum flavum thickness, foraminal area, foraminal width, and foraminal height were pooled, and the mean values of these pooled measurements were compared be-

Table 1. Canal and Foraminal Dimensions at the Instrumented and Adjacent Levels

	L2/3 - Cranial Adjacent Level					
	Flexion		Neutral		Extension	
Dimension	INT	X STOP	INT	X STOP	INT	X STOP
CA (mm^2)	304 ± 46[a]	307 ± 37[b]	278 ± 32	271 ± 22	262 ± 30[a,<0.0001]	261 ± 30[b,<0.0001]
CD (mm)	20.8 ± 1.2[c]	21.5 ± 2.1[d]	19.9 ± 1.6	20.1 ± 1.6	18.8 ± 2.7[c,<0.0030]	19.2 ± 2.7[d,<0.0008]
SD (mm)	4.6 ± 1.2[e]	4.6 ± 1.2[f]	4.0 ± 1.3	3.5 ± 1.4	3.1 ± 1.0[e,<0.0001]	2.9 ± 1.2[f,<0.0001]
LF (mm)	2.7 ± 0.6	2.9 ± 0.8	3.1 ± 0.9	2.8 ± 0.7	3.0 ± 0.6	2.9 ± 0.7
FA (mm^2)	167 ± 34[g]	164 ± 42[h]	142 ± 34	137 ± 31	114 ± 37[g,<0.0001]	115 ± 35[h,<0.0001]
FW (mm)	6.4 ± 2.0[i]	6.6 ± 2.0[j]	5.4 ± 2.2	4.7 ± 2.0	4.2 ± 2.1[i,<0.0001]	4.0 ± 2.4[j,<0.0001]
FH (mm)	22.2 ± 2.3[k]	22.4 ± 1.9[l]	21.5 ± 2.0	20.9 ± 2.2	20.4 ± 2.2[k,<0.001]	20.1 ± 2.4[l,<0.0001]
Dimension						
	L3/4–Implanted Level					
	Flexion		Neutral		Extension	
	INT	X STOP	INT	X STOP	INT	X STOP
CA (mm^2)	286 ± 68	276 ± 63	251 ± 60[a]	265 ± 65[a,<0.0499]	231 ± 56[b]	273 ± 68[b,<0.0001]
CD (mm)	19.3 ± 1.7	19.0 ± 1.9	18.3 ± 1.9	18.9 ± 2.1	17.8 ± 1.5[c]	19.5 ± 2.0[c,<0.0038]
SD (mm)	4.5 ± 1.1	4.1 ± 0.9	3.2 ± 1.0[d]	3.7 ± 1.0[b,<0.0294]	2.5 ± 1.1[e]	3.7 ± 1.2[e,<0.0001]
LF (mm)	3.0 ± 0.5	2.9 ± 0.5	2.7 ± 0.4	2.7 ± 0.3	2.9 ± 0.4	2.9 ± 0.5
FA (mm^2)	149 ± 37	147 ± 42	128 ± 31	137 ± 35	106 ± 32[f]	133 ± 30[f,<0.0001]
FW (mm)	5.8 ± 1.8	6.0 ± 1.9	4.7 ± 1.5	5.0 ± 1.7	3.4 ± 1.6[g]	4.8 ± 1.9[g,<0.0001]
FH (mm)	23.2 ± 1.9	22.4 ± 1.6	21.2 ± 2.1	22.1 ± 2.0	21.3 ± 1.8	21.2 ± 2.2
Dimension						
	L4/5 - Caudal Adjacent Level					
	Flexion		Neutral		Extension	
	INT	X STOP	INT	X STOP	INT	X STOP
CA (mm^2)	303 ± 64[a]	309 ± 69[b]	269 ± 72	277 ± 60	231 ± 67[a,<0.0001]	244 ± 61[b,<0.0001]
CD (mm)	18.7 ± 3.0[c]	18.5 ± 2.4[d]	17.3 ± 2.7	18.5 ± 2.7	16.7 ± 1.8[c,<0.0073]	16.7 ± 2.3[d,<0.0139]
SD (mm)	4.3 ± 1.7[e]	4.3 ± 1.6[f]	3.7 ± 1.5	3.5 ± 1.5	2.6 ± 1.4[e,<0.0001]	2.4 ± 1.5[f,<0.0001]
LF (mm)	2.9 ± 0.8	2.8 ± 0.8	2.6 ± 0.6	2.9 ± 0.7	3.4 ± 1.0	3.3 ± 0.9
FA (mm^2)	155 ± 39[g]	155 ± 39[h]	129 ± 36	119 ± 28	102 ± 27[g,<0.0001]	105 ± 29[h,<0.0001]
FW (mm)	5.2 ± 1.6[i]	5.8 ± 1.8[j]	4.1 ± 1.7	3.5 ± 1.9	2.2 ± 1.4[i,<0.0001]	2.5 ± 1.5[j,<0.0001]
FH (mm)	24.2 ± 2.3[k]	24.8 ± 1.9[l]	23.1 ± 2.2	22.3 ± 2.4	20.7 ± 1.9[k,<0.0001]	21.3 ± 3.3[l,<0.0001]

For a given level, significant differences are depicted by common superscripts. The corresponding *P* values are next to the right-most superscripts.
CA = canal area; CD = canal diameter; FA = foramen area; FH = foramen height; FW = foramen width; INT = interspinous implant; LF = ligamentum flavum thickness; SD = subarticular diameter.

tween intact and implanted specimens. Measurements were compared between intact and implanted specimens for a given position, and between positions for a given treatment using a repeated measures analysis of variance, with a level of significance of 0.05.

■ Results

Canal Area

At the adjacent levels, L2–L3 and L4–L5, there was no significant difference in mean canal area between the intact and implanted specimens for a given position (Table 1). However, mean canal area at the adjacent levels did significantly decrease from flexion to extension for both the intact and implanted specimens. On the other hand, at the L3–L4 level in the neutral and extended positions, the mean canal area of the implanted specimens was significantly greater than that of the intact specimens; there was no significant difference in flexion. In the extended position, the mean canal area of the implanted specimens was 18% greater (273 *vs.* 231 mm^2, $P < 0.0001$) than that of the intact specimens. In the neutral position, the mean canal area of the implanted specimens was 6% greater (265 *vs.* 251 mm^2, $P < 0.0499$).

Canal Diameter

At the adjacent levels, L2–L3 and L4–L5, there was no significant difference in mean canal diameter between the intact and implanted specimens for a given position (Table 1). However, mean canal diameter at the adjacent levels did significantly decrease from flexion to extension for both the intact and implanted specimens. On the other hand, at the L3–L4 level in the extended position, the mean canal diameter of the implanted specimens was significantly greater than that of the intact specimens; the diameter of the implanted specimens was 10% greater (19.5 *vs.* 17.8 mm, $P < 0.0038$) than that of the intact

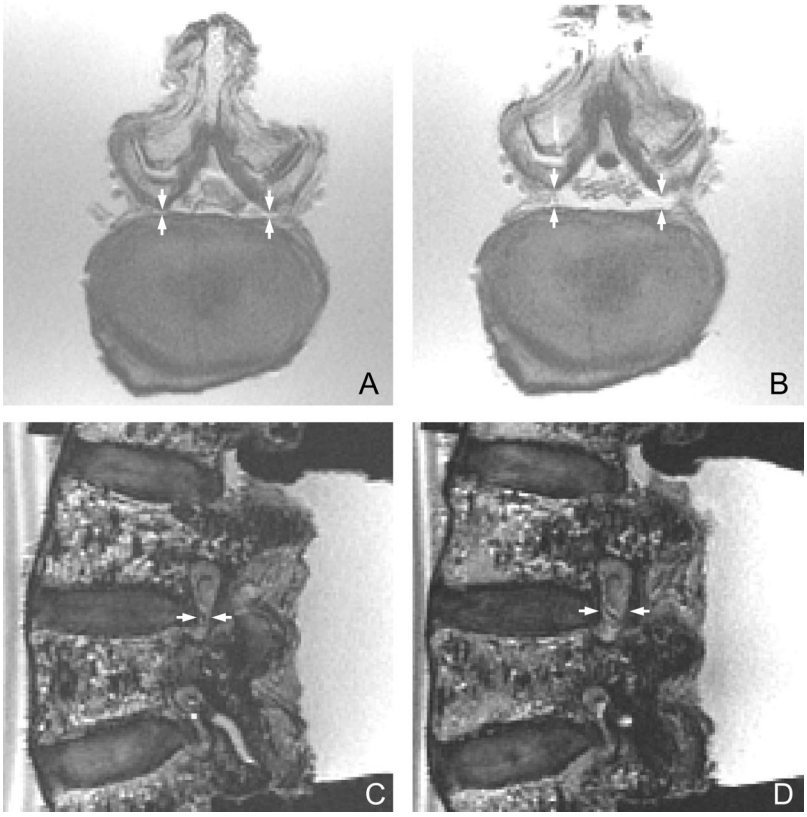

Figure 3. ***A*** and **B** , Axial and (**C** and **D**) pedicular plane magnetic resonance imaging of a specimen in the extended position with and without the implant. The axial slices were taken through the middle of the L3/4 intervertebral disc. **A** is an intact specimen in the extended position. Notice the narrow subarticular diameter between the anterior facet and posterior anulus (arrows). **B** is of the same specimen with an X STOP placed between the L3 and L4 spinous processes. Notice the subarticular diameter in the implanted specimen (arrows). **C** is of the intact specimen in the extended position, and **D** is of the same specimen in the extended position with the implant placed at L3/4. The foraminal area and width are noticeably greater in the implanted specimen (arrows).

specimens. There was no significant difference in the neutral or flexed positions.

Subarticular Diameter

At the adjacent levels, L2–L3 and L4–L5, there was no significant difference in mean subarticular diameter between the intact and implanted specimens for a given position (Table 1). However, mean subarticular diameter at the adjacent levels did significantly decrease from flexion to extension for both the intact and implanted specimens. At the L3–L4 level in the neutral and extended positions, the mean subarticular diameter of the implanted specimens was significantly greater than that of the intact specimens (Figure 3). There was no significant difference in flexion. In the extended position, the mean subarticular diameter of the implanted specimens was 48% greater (3.7 *vs.* 2.5 mm, $P < 0.0001$) than that of the intact specimens. In the neutral position, the mean canal area of the implanted specimens was 16% greater (3.7 *vs.* 3.2 mm, $P < 0.0294$).

Ligamentum Flavum

There was no significant difference in the measured ligamentum flavum thickness at the treated or adjacent levels in any position, with or without the implant in place (Table 1).

Foraminal Area

At the adjacent levels, L2–L3 and L4–L5, there was no significant difference in mean foraminal area between the intact and implanted specimens for a given position (Table 1). However, mean foraminal area at the adjacent levels did significantly decrease from flexion to extension for both the intact and implanted specimens. At the L3–L4 level in the extended position, the mean foraminal area of the implanted specimens was significantly greater than that of the intact specimens; there was no significant difference in the neutral or flexed positions. In the extended position, the mean foraminal area of the implanted specimens was 25% greater (133 *vs.* 106 mm^2, $P < 0.0001$) than that of the intact specimens.

Foraminal Width

At the adjacent levels, L2–L3 and L4–L5, there was no significant difference in mean foraminal width between the intact and implanted specimens for a given position (Table 1). However, mean foraminal width at the adjacent levels did significantly decrease from flexion to extension for both the intact and implanted specimens. On the other hand, at the L3–L4 level in the extended position, the mean foraminal width of the implanted specimens was significantly greater than that of the intact specimens; there was no significant difference in the neutral or flexed positions (Figure 3). In the extended position, the mean foraminal width of the implanted specimens was 41% greater (4.8 *vs.* 3.4 mm, $P < 0.0001$) than that of the intact specimens.

Foramen Height

At the adjacent levels, L2–L3 and L4–L5, there was no significant difference in mean foraminal height between the intact and implanted specimens for a given position (Table 1). However, mean foraminal height at the adja-

Table 2. Published Mean Values of Canal and Foramen Dimensions

Dimension	Position	Chung *et al*[22]	Fujiwara *et al*[21]	Inufusa *et al*[23]	Schmid *et al*[24]	Current Intact	Current X STOP
CA (mm^2)	Flex	399	-	248	268	286	276
	Ext	331	-	208	224	231	273
CD (mm)	Flex	25.0	-	20.2	-	19.3	19.0
	Ext	23.0	-	17.7	-	17.8	19.5
SD (mm)	Flex	5.7	-	5.8	-	4.5	4.1
	Ext	3.2	-	4.7	-	2.5	3.7
LF (mm)	Flex	1.8	-	3.5	1.8	3.0	2.9
	Ext	2.5	-	2.9	4.3	2.9	2.9
FA (mm^2)	Flex	-	104	141	167	149	147
	Ext	-	83.9	107	115	106	133
FH (mm)	Flex	-	17.9	20.0	-	23.2	22.4
	Ext	-	18.2	20.3	-	21.3	21.2
FW (mm)	Flex	-	4.0	5.8	-	5.8	6.0
	Ext	-	2.2	3.5	-	3.4	4.8

CA = canal area; CD = canal diameter; Ext = extended; FA = foramen area; FH = foramen height; Flex = flexed; FW = foramen width; LF = ligamentum flavum thickness; SD = subarticular diameter.

cent levels did significantly decrease from flexion to extension for both the intact and implanted specimens. At the L3–L4 level, there was no significant difference between the mean foramen height of the intact and implanted specimens in any position (Table 1).

■ Discussion

The results of the current *in vitro* study show that the X STOP interspinous process implant prevents narrowing of the spinal canal and foramina at the treated level during extension but does not significantly affect the dimensions of the canal and foramens at the adjacent levels. The design rationale of the X STOP is based on biomechanical and clinical findings that show the spinal canal and neural foramina become narrow in extension and expand in flexion.[21–26]

The mechanism of this dynamic process is based on both the deformation of soft tissues and the relative position of osseous structures. During extension, the ligamentum flavum buckles anteriorly into the spinal canal and lateral recess, and the posterior anulus fibrosis bulges posteriorly into the spinal canal and lateral recess.[21,22,24] Furthermore, the ligamenta flava and facet capsules are thought to be displaced forward by the superior articular process of the caudal vertebra.[36] All of these mechanisms are reversed in flexion. The current study corroborates these findings in the intact specimens; the canal area, canal diameter, subarticular diameter, foraminal area, foraminal width, and foraminal height were all significantly greater in flexion than extension at all 3 levels. However, the effect of ligamentum flavum thickening in extension and thinning in flexion was not measured in the current study.

The results of the current study are also comparable to previously published values (Table 2). Inufusa *et al*[23] studied the anatomic features of 25 motion segments frozen and microtomed in the flexed, neutral, or extended positions. They showed that the canal and foraminal dimensions significantly increased in flexion and decreased in extension, as shown in the current study. In addition, despite the different measuring techniques used in the current study and by Inufusa *et al*,[23] all dimensions are very similar (Table 2). Chung[22] and Schmid[24] *et al* used flexion and extension MRI of healthy volunteers to measure the changes in flexion and extension, and Fujiwara *et al* [21]used computerized tomography of cadaver specimens to make their measurements. All 3 studies reported similar findings to those of the current study.

The current study is the first to report changes in the lumbar spinal canal and foramen following the placement of an interspinous process implant. Although the concept of placing an implant in the interspinous space is not unique to spinal surgery, doing so exclusively for NIC secondary to LSS is unique. In 1958, Knowles[31] reported on a vertebral support placed between the spinous processes that was indicated for contained disc herniations. Years later, Minns and Walsh[32] reported on a silicone interspinous spacer that was indicated for "sagittal plane instability." The history of the Minns device is unknown, and it is unclear whether the implant advanced much further than the laboratory setting. In addition to the X STOP, a number of interspinous implants are commercially available in Europe. Kaech *et al*[34] have reported on the interspinous 'U' that is indicated for protection against adjacent level disc disease and restabilization of a lumbar laminectomy. Similarly, Senegas[35] has reported on the Wallis interspinous implant, which is indicated for discectomy following a herniated disc and adjacent level disc disease, and Caserta *et al*[33] has reported on the DIAM implant, which is indicated for a number of conditions, including degenerative disc disease, herniated nucleus pulposis, and lumbar instability. The X STOP is indicated for lumbar NIC secondary to LSS,[37] and the current study shows that the X STOP prevents narrowing during extension, which is the underlying mechanism believed to be responsible for the symptoms of the condition.

There were a few important limitations of the experimental model that must be addressed. First, because the study incorporated a displacement or angle-controlled

model, loads consistent with those experienced *in vivo* were not applied to the specimens. Despite this limitation, the measurements were consistent with those previously measured using both displacement-controlled experimental models and *in vivo* models (Table 2). Second, the cadaver specimens were not screened for the presence of NIC before the study and may or may not have reflected the absolute changes that occur in such a patient population. One of the goals of the current study was to quantify the relative changes that occur in the lumbar spine during flexion and extension, and not necessarily those that occur in a specific patient population. Third, thinner image slices would have increased the accuracy of the measurements due to less volume averaging. However, the slice thickness in the current study was dictated by the signal-to-noise ratio of the images, and attempts to acquire thinner slices resulted in poor quality images.

The findings in the current report confirm that the X STOP achieves its intended anatomic objective. These *in vitro* imaging results show that the implant prevents narrowing of the canal and foraminal during extension. The current study provides imaging data consistent with the proposed mechanism of action of the X STOP, as well as visual evidence that may illustrate the basis for the described clinical success[37] of the implant in the treatment of NIC.

■ Key Points

- The spinal canal and neural foramina dimensions of cadaver lumbar spines were quantified during flexion and extension using MRI before and after the placement of an interspinous process implant.
- The interspinous process implant significantly prevents narrowing of the lumbar spinal canal and neural foramens in extension.

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