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The Effects of an Interspinous Implant on the Kinematics of the Instrumented and Adjacent Levels in the Lumbar Spine

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Study Design. Measurement of the kinematics of the lumbar spine after insertion of an interspinous spacer *in vitro*.

Objectives. To understand the kinematics of the instrumented and adjacent levels due to the insertion of this interspinous implant.

Summary of Background Data. An interspinous spacer (X Stop, SFMT, Concord, California) has been developed to treat neurogenic intermittent claudication by placing the stenotic segment in slight flexion and preventing extension. This restriction of motion by the interspinous implant may affect the kinematics of levels adjacent to the instrumented level.

Methods. Seven lumbar spines (L2–L5) were tested in flexion–extension, lateral bending, and axial rotation. Images were taken during each test to determine the kinematics of each motion segment. The interspinous implant was placed at the L3–L4 level, and the test protocol was repeated.

Results. The flexion–extension range of motion was significantly reduced at the instrumented level. Axial rotation and lateral bending ranges of motion were not affected at the instrumented level. The range of motion in flexion–extension, axial rotation, and lateral bending at the adjacent segments was not significantly affected by the implant.

Conclusions. The implant does not significantly alter the kinematics of the motion segments adjacent to the instrumented level. [Key words: spinal stenosis, neurogenic intermittent claudication, interspinous implant, kinematics] **Spine 2003;28:2192–2197**

Neurogenic intermittent claudication is the most common and characteristic syndrome of lumbar spinal stenosis. First described by Verbiest, the symptoms of neurogenic intermittent claudication present on standing and walking and include bilateral radicular pain, sensation disturbance, and loss of strength in the legs.[1] These symptoms are relieved on resting or flexing the lumbar spine.[2,3]

Arbit and Pannullo[4] presented the three prevailing theories explaining intermittent neurogenic claudication: the ischemic theory, the mechanical compression theory, and the theory of stagnant anoxia. The ischemic theory postulates that segmental compression prevents sufficient blood flow during increased metabolic activity leading to ischemia.[5,6] The mechanical compression theory is based on the clinical observation that symptoms are commensurate with posture and not activity.[2,3,7] The last theory, the theory of stagnant anoxia, postulates that bone and soft tissues compress the neural elements, interfering with venous return.[8] Although the mechanism behind intermittent neurogenic claudication is unclear, it is clear that the symptoms are exacerbated during extension and relieved by flexing the stenotic segment, which decreases epidural pressure,[9] increases the cross sectional area of the spinal canal,[7,10–12] increases the area of intervertebral foramen,[10,13] and decreases nerve root compression.[7,10] Based on the concept that flexion decreases the symptoms, a novel interspinous spacer has been developed to relieve the effects of intermittent neurogenic claudication.

The interspinous spacer (X Stop, SFMT, Concord, CA; http://www.sfmt.com) was developed to treat intermittent neurogenic claudication in patients who obtain relief in sitting and flexion. The spacer consists of a titanium oval spacer with two lateral wings to prevent lateral migration (Figure 1). Surgical implantation is performed under local anesthesia with the patient slightly flexed in the lateral decubitus position. The procedure requires that the paraspinal muscles be stripped from the spinous processes while all midline structures are left intact. The spacer is inserted between the spinous processes of the affected level and places the motion segment in slight flexion, while at the same time preventing extension. Although the implant is not rigidly attached to the bony anatomy, it is restricted from migrating posteriorly by the supraspinous ligament, anteriorly by the laminae, cranially and caudally by the spinous processes, and laterally by the two wings or stops on the implant. A novel aspect to the spacer is that it only restricts motion in one direction, thus allowing axial rotation and lateral bending of the instrumented segment.

In this study, we investigated the use of the interspinous spacer on the kinematics of the lumbar spine. Pre-

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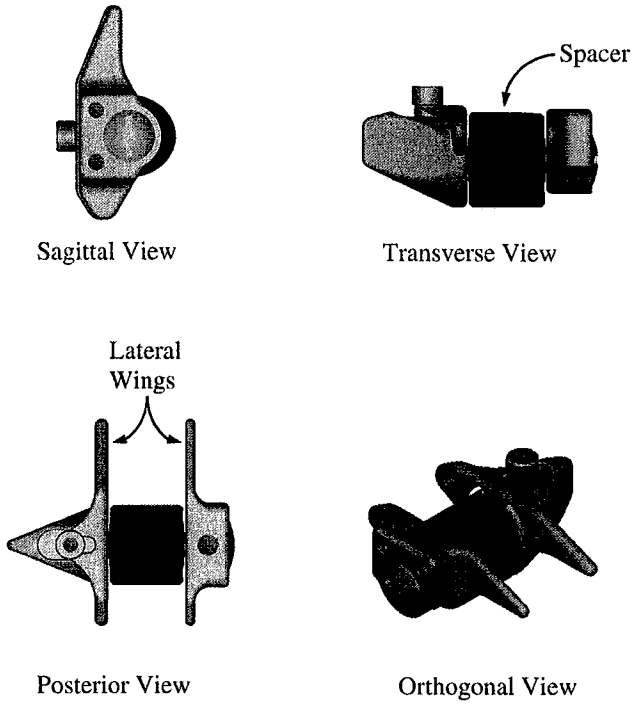

Figure 1. A drawing of the interspinous spacer in three views plus an orthogonal view. The spacer is placed between the spinous processes and the wings rest lateral to the spinous processes.

vious studies have reported that motion restricting surgical techniques, such as fusion, restrict the kinematics of a motion segment and can produce abnormal motion of the adjacent levels leading to instability.[14–16] A biomechanical study of short lumbosacral fusions by Lee and Langrana showed a redistribution of mobility with the proximal level accommodating the extra deformation.[17] Another study by Chow *et al* investigated the effects of anterior lumbar interbody fusion and found that the segment mobility of the adjacent segments was increased in both flexion and extension.[18] Shono *et al* investigated the effects of posterior spinal instrumentation and found higher segmental displacement values at the cephalad motion segment in flexion–extension, lateral bending, and axial rotation.[19] A possible consequence of implanting an interspinous spacer is that by restricting extension at one level, the kinematics of the adjacent levels may be altered, leading to degeneration and instability. We hypothesized that the implant will reduce the range of motion (ROM) of the treated level in flexion–extension while not affecting the treated level in axial rotation or lateral bending. We also hypothesized that the adjacent levels would not be affected.

■ Materials and Methods

Seven human lumbar (L2–L5) cadaver specimens were obtained from human donors aged 17 to 55. The specimens were visually inspected for gross abnormalities and for evidence of metastatic and metabolic diseases. Each specimen was separated into segments consisting of four vertebrae (L2–L5) and the three associated intervertebral discs. All muscle and adipose tissue was removed, leaving the ligamentous structures intact. The cranial portion of L2 and caudal portion of L5 vertebrae were secured in polymethylmethacrylate (PMMA).

Each specimen was placed in a spinal loading frame capable of applying independent bending moments and axial loads (MTS 858, MTS Systems, Eden Prairie, MN). Labeled steel pins 10 cm in length were placed in each vertebra and on the upper and lower actuators to indicate the angular position (Figure 2). Two CCD cameras were used to record the position of the pins during the testing. The first camera (Model XC-77; Sony, Tokyo, Japan) was placed perpendicular to the flexion–extension/lateral bending plane and recorded images only during those tests. The second camera (Model DC-37; Sony) was placed perpendicular to the plane of axial rotation and recorded images only during axial rotation testing. Three images

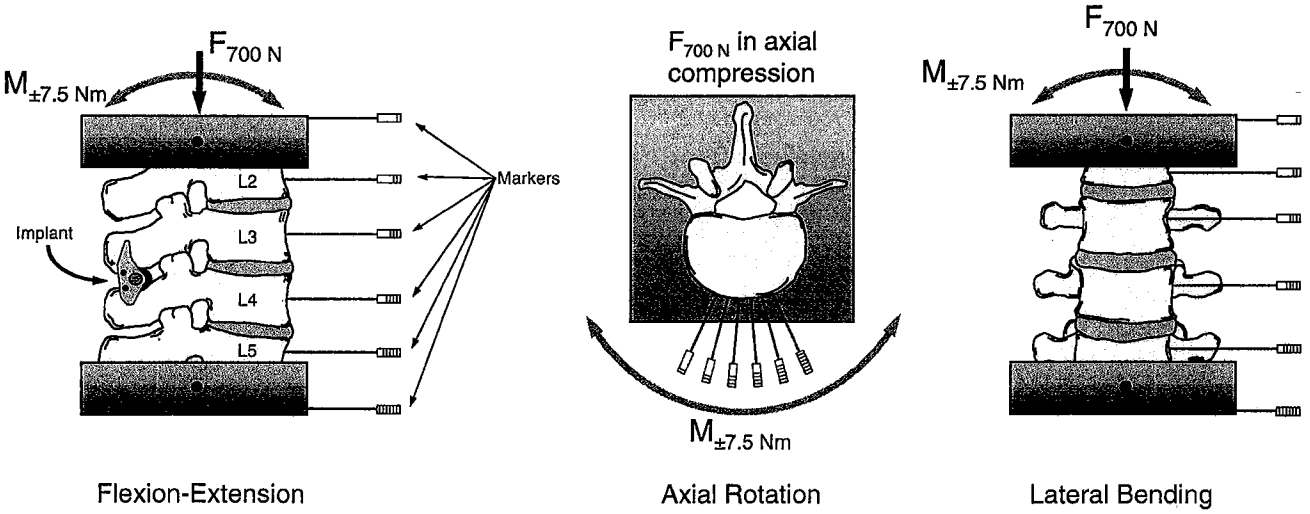

Figure 2. A schematic of the pin placement for flexion–extension, axial rotation, and lateral bending. A 700 N axial load was applied in each testing scenario along with a ±7.5 Nm in the respective direction.

were taken during each test cycle. During flexion–extension, images were recorded with the specimens in the flexed, neutral, and extended positions. During lateral bending, images were recorded during right bending, neutral, and left bending. Finally, during axial rotation, images were recorded during right rotation, neutral, and left rotation.

Specimens were initially tested intact by applying a ±7.5 Nm bending moment with a superimposed 700 N compressive load in flexion and extension, left and right axial rotation, and left and right lateral bending (Figure 2). After testing flexion–extension and axial rotation, the specimen was removed from the loading frame, rotated 90°, and returned to the loading frame for lateral bending testing. This step is required because the loading frame constrains lateral bending during flexion–extension and *vice versa*. Angle, force, and torque data were recorded for each motion at 10 Hz by the MTS. Following the intact testing, the specimens were removed from the loading frame and an appropriately sized interspinous spacer (X Stop, SFMT) was placed between the L3–L4 spinous processes in each specimen. The implant was placed by first piercing the anterior margin of the interspinous ligament between the L3 and L4 spinous processes, then dilating the interspinous space to the appropriate size with the specimen slightly flexed using custom sizers of 6 to 12 mm in 2 mm increments. The implant was then placed in the anterior margin of the interspinous space. After the implant was placed, the specimens were returned to the loading frame and the previously described loading regimen was applied to each specimen.

Technique Validation. The ROM of the upper and lower actuator was calculated using the MTS data at the time of video capture for all three movements and denoted MTS ROM. The range of motion between pins 1 and 6 was calculated in same manner aforementioned and denoted Video ROM. The MTS and Video ROM were compared using a paired t test with a level of significance of 0.05 for all movements. A linear regression was also run to determine the slope and R^2 value (StatView 5.0.1, SAS Institute, Cary, NC).

Data Analysis. The angle of each pin was determined using Scion Image (Scion Corp., Frederick, MD) for all three images of each test cycle, and the difference between adjacent levels was recorded as the ROM. The mean ROM at each level for each movement was compared between the intact and implanted specimens using a single-factor analysis of variance (ANOVA) followed by a Fisher PLSD follow-up test with a level of significance of 0.05 (StatView 5.0.1). The mean ROM of the specimen (L2–L5) for each movement was compared between the intact and implanted specimens using a paired t test with a level of significance of 0.05.

The absolute position of the entire L2–L5 specimen during flexion–extension, lateral bending, and axial rotation was determined from the MTS; the video data were useful for measuring ROM but did not allow for absolute position relative to a fixed reference frame to be measured. A repeated measures ANOVA and a Fisher PLSD follow-up test with a level of significance of 0.05 was used to compare the mean L2–L5 angles for the intact and implanted cases for each of the following positions: 1) flexion, neutral, and extension; 2) left, neutral, and right lateral bending; and 3) left, neutral, and right axial rotation.

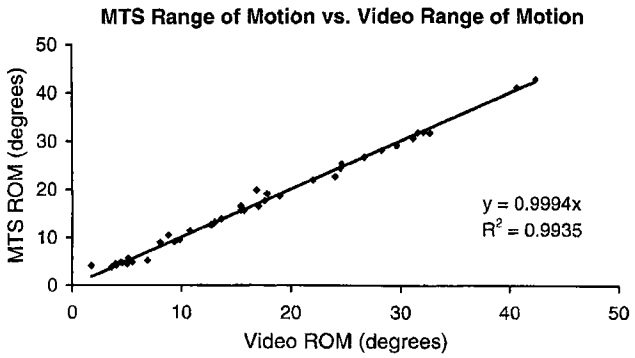

Figure 3. A linear correlation between the angles measured by the MTS transducers and those measured using the video system for all tests in flexion–extension, lateral bending, and axial rotation.

Results

Technique Validation

For technique validation, 42 ROM test points collected with the MTS system and video system were compared with no significant difference observed ($P > 0.49$). The maximum error between the MTS and video system was 2.9° with an average error of 0.094°. The resulting slope of the linear regression was 0.9987 deg/deg with an R^2 correlation coefficient of 0.9980 (Figure 3).

Flexion–Extension

In the motion from the flexed to extended positions, the implant significantly reduced ($P < 0.0001$) the ROM of the L3–L4 motion segment (Table 1; Figure 4). In addition, the motion of the L3–L4 motion segment was significantly reduced from flexion to neutral ($P < 0.0001$; Table 1) but not from neutral to extension ($P > 0.052$; Table 1). At L4–L5, the motion segment was significantly reduced from neutral to extension ($P < 0.035$; Table 1). All other ROM values between vertebrae were not significantly different between the intact and implanted specimens (Table 1). Finally, the overall ROM of the entire L2–L5 specimen from flexion to extension was significantly affected by the implant (intact: 25.8°, implant: 20.8°; $P < 0.0011$).

In the flexed and neutral positions, there were no statistically significant differences between the mean angles of the intact and implanted specimens (Table 2). In the extended position, however, the mean angle of the intact specimens was significantly greater than that of the implanted specimens ($P < 0.029$; Table 2). Also, there was no significant difference between the mean position of the intact specimens in the neutral position (0.6°) and the mean position of the implanted specimens in the extended positions (3.5°) ($P > 0.13$).

Axial Rotation

There were no significant differences in the mean ROM during: 1) right to neutral axial rotation; 2) neutral to left axial rotation; and 3) right to left axial rotation at any level (Table 1). In addition, the mean ROM of the entire L2–L5 specimen was not affected by the implant (intact: 4.7°, implant: 5.1°; $P > 0.29$).

Table 1. Range of Motion for Individual Levels During Flexion-Extension, Axial Rotation, and Lateral Bending

	Flexion–Extension ROM (Degrees)								
	Flexion to Neutral			Neutral to Extension			Flexion to Extension		
Treatment	L2 to L3	L3 to L4	L4 to L5	L2 to L3	L3 to L4	L4 to L5	L2 to L3	L3 to L4	L4 to L5
Intact	5.9 ± 2.0	6.3 ± 2.4*	7.0 ± 2.4	2.3 ± 1.1	1.3 ± 0.4	3.0 ± 1.3†	8.2 ± 2.2	7.6 ± 2.5‡	10.0 ± 2.9
Implant	6.1 ± 1.7	2.5 ± 2.1*	7.3 ± 3.1	2.1 ± 1.1	0.5 ± 0.3	2.2 ± 1.0†	8.2 ± 2.1	3.1 ± 2.3‡	9.5 ± 3.5
	Axial Rotation ROM (Degrees)								
	Right to Neutral Rotation			Neutral to Left Rotation			Right to Left Rotation		
	L2 to L3	L3 to L4	L4 to L5	L2 to L3	L3 to L4	L4 to L5	L2 to L3	L3 to L4	L4 to L5
Intact	0.7 ± 0.4	0.8 ± 0.3	0.7 ± 0.5	0.8 ± 0.5	0.8 ± 0.5	0.9 ± 0.3	1.6 ± 0.8	1.6 ± 0.7	1.6 ± 0.7
Implant	0.8 ± 0.4	0.9 ± 0.6	0.9 ± 0.2	0.9 ± 0.5	0.8 ± 0.9	0.9 ± 0.7	1.7 ± 0.5	1.7 ± 1.3	1.8 ± 0.8
	Lateral Bending ROM (Degrees)								
	Right to Neutral Bending			Neutral to Left Bending			Right to Left Bending		
	L2 to L3	L3 to L4	L4 to L5	L2 to L3	L3 to L4	L4 to L5	L2 to L3	L3 to L4	L4 to L5
Intact	3.9 ± 2.0	4.1 ± 2.4	3.0 ± 1.8	3.5 ± 1.4	4.2 ± 2.6	2.8 ± 2.1	7.4 ± 3.3	8.2 ± 4.7	5.8 ± 3.9
Implant	3.4 ± 2.0	4.2 ± 1.7	2.7 ± 2.1	3.4 ± 1.7	4.6 ± 2.4	2.8 ± 2.0	6.8 ± 3.6	8.8 ± 4.0	5.5 ± 4.1

Depicted values are mean ± SD.
Significant differences ($P < 0.05$ are denoted by common symbols).
ROM = range of motion.

In the left rotated, neutral, and right rotated positions, there were no statistically significant differences between the mean angles of the intact and implanted specimens (Table 2).

Lateral Bending

There were no significant differences in the mean ROM during: 1) right to neutral lateral bending; 2) neutral to left lateral bending; and 3) right to left lateral bending at any level (Table 1). In addition, the mean ROM of the entire L2–L5 specimen was not affected by the implant (intact: 21.4°, implant: 21.1°; $P > 0.65$).

In the left bending, neutral, and right bending positions, there were no statistically significant differences between the mean angles of the intact and implanted specimens (Table 2).

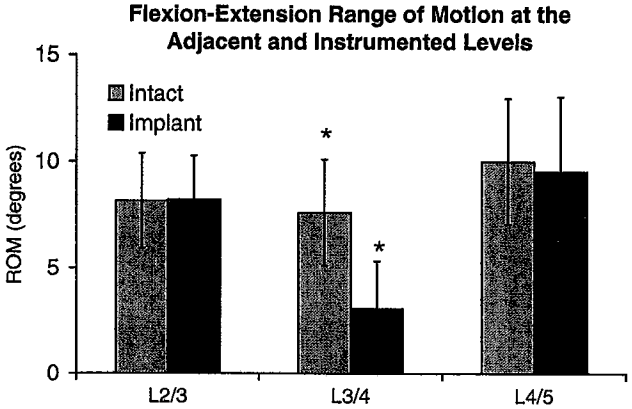

Figure 4. A bar chart of the mean ROM during flexion–extension of the adjacent (L2–L3 and L4–L5) and instrumented (L3–L4) levels with and without the spacer. (* = significant; $P < 0.05$).

■ Discussion

The results of the current study show that the implant significantly affected the instrumented level during flexion–extension. The ROM of the implanted level from flexion to neutral and from flexion to extension de-

Table 2. Mean Specimen (L2–L5) Angle at the Three Positions During Testing (Degrees)

	Flexion–Extension		
Treatment	Flexion	Neutral	Extension
Intact	−17.3 ± 5.9	0.6 ± 2.0	7.7 ± 2.1*
Implant	−16.6 ± 6.0	−1.3 ± 2.4	3.5 ± 2.1*
	Axial Rotation		
	Left Rotation	Neutral	Right Rotation
Intact	4.1 ± 1.3	1.5 ± 0.9	−1.0 ± 1.2
Implant	4.2 ± 1.5	1.5 ± 0.9	−1.3 ± 1.3
	Lateral Bending		
	Left Bending	Neutral	Right Bending
Intact	−10.2 ± 4.8	0.2 ± 1.4	10.7 ± 7.0
Implant	−10.2 ± 4.6	0.5 ± 1.4	10.7 ± 7.0

Depicted values are mean ± standard deviation.
Significant differences ($P < 0.05$) are denoted by common symbols.

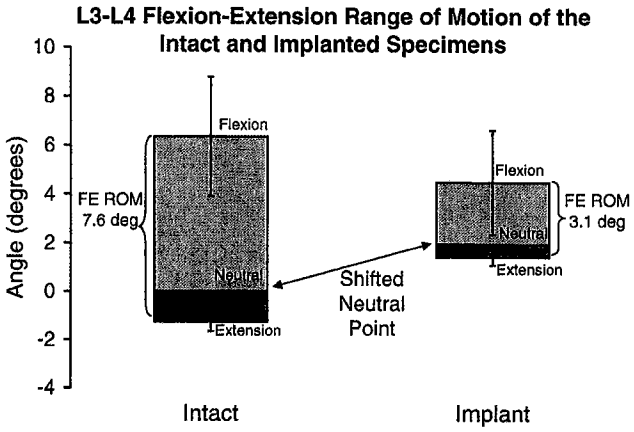

Figure 5. The ROM of the L3–L4 motion segment before and after implantation of the interspinous implant. It is evident that the ROM from the neutral to flexed and neutral to extended positions has decreased, and the neutral point has shifted to a more flexed position.

creased significantly after implantation. The ROM for neutral to extension decreased but was not significant ($P > 0.052$). The intent of the interspinous implant is to place the stenotic segment in slight flexion while preventing extension. Although not statistically significant, the mean neutral position of the L2–L5 specimens changed 1.9° when implanted with the interspinous spacer, which suggests that the mean neutral position of the specimens is in a more flexed position with no loads applied (Table 2). The change in the average neutral angle occurs mainly at the L3–L4 (Table 1; Figure 4). Figure 5 describes the shift in the neutral position and the ROM from neutral to flexion and neutral to extension about the neutral position. It is clear that after shifting the neutral point, the implanted level is not flexed or extended as much as the intact level. Other results from this study support the shift in the neutral position due to the implant: 1) the overall extension angle decreased significantly from the intact to the implanted treatment; and 2) the implanted extension angle was not significantly different from the intact neutral angle. From these results, we conclude that the interspinous spacer places the stenotic segment in about 2° of flexion when unloaded and reduces flexion and extension of that segment.

The results of this study also show that the kinematics of the adjacent levels during flexion–extension, axial rotation, and lateral bending are not significantly affected with one exception. At the caudal L4–L5 level, the ROM from the neutral to extended positions was significantly reduced ($P < 0.035$). Although the ROM at L4–L5 was significantly reduced from the neutral to extended positions, the reduction was only 0.8°. This change, however, is not likely to be clinically significant because the 0.8° reduction in ROM will lead to a reduced stress in contrast to the previously reported increases in ROM leading to degeneration.[17] In addition, the ROM from flexion to extension was not significantly reduced (Table 1; Figure 4). Therefore, we conclude that the kinematics of the motion segments adjacent to the interspinous implant are not significantly affected.

The range of motion values presented in the current study are consistent with those presented in the literature.[20–23] Yamamoto *et al* reported ranges of motion at L2–L3, L3–L4, and L4–L5 during flexion–extension of 10.8°, 11.2°, and 14.5°, respectively.[23] The values presented in the current study are 8.2°, 7.6°, and 10.0° at L2–L3, L3–L4, and L4–L5, respectively, during flexion–extension. During axial rotation, they reported a one-side motion of 2.6°, 2.6°, and 2.2° *versus* the current values of 0.8°, 0.8°, and 0.8°.[23] Lastly, they reported one-side lateral bending values of 7.0°, 5.7°, and 5.7° *versus* 3.7°, 4.1°, and 2.9°.[23] The values from the current study are lower for all test cases likely due to the smaller moments used for the current study (7.5 Nm *vs.* 10 Nm[23]) and the large axial load (700 N) that acts to stabilize the motion segments.[24] Another possible reason for the decrease in the current ROM values is that the current test setup prevents lateral bending during flexion–extension and axial rotation testing and flexion–extension being constrained during lateral bending. Constraining of a single motion decreases the ROM due to the coupled nature of the motions.[25] Although one ROM was constrained during each test in the current study, the ROM values are consistent with those seen in the literature.

In the spinal fusion literature, restriction of segment motion has been shown to result in abnormal or compensatory motion of the adjacent motion segments, leading to instability.[14–16] Previous investigators have reported changes in the kinematics of the adjacent motion segments after fusion.[17–19] Lee and Langrana reported an increase in the motion of the unfused segments above a fusion during combined compression and bending loads.[17] During combined compression-torsion loading, however, Yang *et al* did not find a significant increase in the stress on the adjacent levels.[26] Chow *et al* found increased segmental mobility in the motion segments above an L4–L5 fusion.[18] In the most pertinent study, Shono *et al* investigated the effects of flexion–extension, lateral bending, and axial rotation on the levels adjacent to spinal instrumentation.[19] They found that when using transpedicular screws as fixation for one- and two-level instabilities, motion in the upper intact segment was significantly higher during axial rotation, flexion–extension, and lateral bending. In contrast to these reports where rigid fixation was used, after implantation of the interspinous implant, the adjacent segment motions were not significantly affected during flexion–extension, axial rotation, and lateral bending. In addition, the axial rotation and lateral bending mobility of the specimens were not affected at the instrumented level. Because the instrumented levels were not affected during axial rotation and lateral bending, it is understandable that the adjacent levels would not be affected during the respective motions as well. One reason that significant changes were not observed at the adjacent levels during flexion–

extension may be that the interspinous implant is not a rigid device, such as those used in fusion, and still allows about 3° of flexion during flexion–extension, mostly in flexion (Table 1; Figure 5). Lastly, a remaining possible concern with the implant lies in the neutral position of the instrumented segment. The current data suggests that the instrumented segment is placed in about 2° of flexion in the unloaded position. Whether this change in neutral position leads to adjacent level degeneration remains to be seen through clinical experience, but the current biomechanical data suggests that this is unlikely.

Loss of lumbar lordosis after fusion has been suggested to be a reason for the degeneration of the adjacent segments.[27] Umehara *et al* reported that after L4–L5 fusion in 18 patients, lordosis in the fusion segment decreased 3° after surgery and increased in the proximal 2 segments by 2° each.[27] Long-term clinical results were not reported on these patients, and thus it is unclear if these changes lead to symptomatic adjacent level degeneration. Although the current results describe a 2° decrease in lordosis from L2 to L5, no deleterious effects were noted in the kinematics at the adjacent levels. The one statistically significant effect at L4–L5 from neutral to extension was actually a decrease in motion and is likely not clinically significant.

The current study investigated the kinematics of the lumbar spine implanted with the interspinous process spacer. The results indicate that the implant significantly reduces the amount of bending at the treated level during flexion–extension and does not significantly affect the ROM at the adjacent levels. In addition, the implant does not alter the kinematics during axial rotation and lateral bending at any level. These results were expected because the implant was designed to perform in this manner. The results suggest that, because the implant allows unrestricted axial rotation and lateral bending and motion during flexion–extension, it is unlikely that the implant will have any deleterious effects at adjacent levels.

■ Key Points

- The effect of the interspinous implant on the kinematics of the lumbar spine at the instrumented and adjacent levels was assessed.
- The implant reduced the range of motion during flexion–extension at the instrumented level.
- The range of motion of the adjacent motion segments was not affected by the implant during axial rotation, flexion–extension, or lateral bending.

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