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Spinal stenosis and interspinous spacers: An mri study of recurrent claudication

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SPINAL STENOSIS AND INTERSPINOUS SPACERS: AN MRI STUDY OF RECURRENT CLAUDICATION

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Abstract:

Interspinous spacers represent a potentially less invasive treatment for patients with intermittent neurogenic claudication, secondary to lumbar spinal stenosis. Previous anatomic studies have suggested that these devices may bring about an "indirect decompression" of the neural elements by increasing the dimensions of the spinal canal and foramina. However, the mechanism of failure associated with the development of recurrent claudication has not been fully elucidated. The purpose of this investigation is to quantify the changes in these parameters at the stenotic and adjacent levels by comparing the pre- and follow-up magnetic resonance imaging studies of a series of patients who developed recurrent symptoms following the implantation of interspinous spacers.

A cohort of ten subjects, who had been evaluated with follow-up MRI studies for complaints of recurrent neurogenic claudication following placement of interspinous spacers (X-STOP, Medtronic, Memphis, TN) at one or two levels of the lumbar spine, were prospectively identified. Using a PACS system, various anatomic parameters were measured from the preoperative and follow-up MRI studies including cross-sectional area and anterior/posterior diameter of the central canal, subarticular diameter (bilaterally), foraminal area, height, and width (bilaterally), anterior and posterior disc height, and intervertebral angle. For each patient, these values were recorded at both the operative and adjacent levels. Whenever applicable, the left- and right-sided values were pooled and the preoperative and follow-up values were compared using the Welch two-sample t-test.

The placement of interspinous devices resulted in significant increases in the dimensions of the central canal, lateral recesses, and foramina at the levels of implantation. However, there were no statistically significant changes observed in these parameters at adjacent levels.

The anatomic parameters measured in this study were significantly improved at the levels at which interspinous implants had been placed. Thus, even in patients with recurrent claudication, the interspinous spacer appears to increase the dimensions of the spinal canal and foramina at the stenotic levels with no significant effects on adjacent segments.

Findings from this study suggest that the failure of these devices is not necessarily related to an inability to bring about improvements in spinal anatomy. Further research is necessary to elucidate this mechanism of failure, an understanding of which will be instrumental for refining the appropriate surgical indications for these devices.

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Introduction:

Neurogenic intermittent claudication (NIC), first described in 1954 by Verbiest, is a clinical syndrome caused by stenosis, or narrowing, of the spinal canal.[1,2] The syndrome is characterized by symptoms of radicular pain, paresthesias, or pain originating in the buttocks and radiating down the legs. Often accompanying the characteristic sensory abnormalities is leg weakness that may impair walking. Interestingly, symptoms are generally improved by sitting or leaning forward.[1,2] Most commonly, NIC results from spinal stenosis that is the end result of a cascade of degenerative changes and often presents in the fifth or sixth decade of life. The reported incidence of spinal stenosis among the general population is 8%.[3] Although the exact pathogenesis of NIC is unknown, it is clear that impingement of neural elements is the inciting factor.

Several proposed theories exist to explain the mechanism by which impingement brings about the characteristic symptomatic changes. The ischemic theory ascribes the symptoms to segmental arterial compression preventing sufficient blood flow. Similarly, the stagnant anoxia theory suggests compression resulting in impaired venous return. The mechanical compression theory postulates that it is simply the compression of nerves themselves that produces symptoms.[1,4] Narrowing can occur at several levels: within the spinal canal, while passing beneath the facet joint (subarticular stenosis), or while exiting the canal through the intervertebral foramina.[5] The stenosis most commonly arises as a result of lumbar spondylosis. Age-related degenerative changes cascade from disc narrowing/dehydration and

posterior annulus degeneration, leading to bulging and associated ligamentum flavum thickening and buckling. These changes cause canal narrowing and also contribute to increased stresses that lead to facet arthrosis, subluxation, and osteophyte formation.[5,6] Additional causes of lumbar spinal stenosis can be broken down into congenital, iatrogenic, spondylolitic, and post-traumatic. Congenital stenosis is due to variation in the osteoanatomy of the vertebrae, with shortened pedicles causing the congenital narrowing of the spinal canal, with symptoms of stenosis developing in the third or fourth decade. Achondroplastic dwarves may also develop congenital stenosis due to congenitally small vertebral bodies.[5] Iatrogenic causes include stenosis that may occur at adjacent vertebral levels, postlaminectomy or postfusion. Stenosis may also occur as a result of trauma or from spondylolisthesis. Several diseases may also predispose to spinal stenosis. These include Cushing's syndrome, due to deposition of excess epidural fat, as well as Paget's disease of the spine, resulting in excess bone depositon at effected sites and subsequent compression of neural elements.[5,7]

Diagnosing – Identifying NIC

Regardless of which theory is correct, or the exact location of impingement, symptomatic improvement with flexion is a classic clinical sign present in 88% of affected individuals.[5] Patients may also describe exacerbation of symptoms with active back extensions. Sensory or motor deficits occur in roughly 50% of patients and can be bilateral and polyradicular.[8] Romberg may be positive and patients may have a wide-based gait. Though weakness may be present on exam, it is often mild

and is generally not function limiting.[5] It is important to distinguish spinal stenosis from similarly presenting conditions, including vascular claudication, peripheral neuropathy, hip osteoarthritis and trochanteric bursitis, which can be differentiated easily with a thorough history and physical exam.

Diagnostic Imaging

In patients whose exam is unclear, or in whom further testing is required, imaging may be used. Plain film radiographs may be used for initial evaluation and may point to possible causes, such as foraminal osteophytes causing foraminal stenosis, facet joint hypertrophy, disc-space narrowing, and endplate sclerosis.[8] CT and MRI can confirm stenosis through direct measurement of the cross-sectional area of both the central canal and foramina, as well as further characterization and the degree of disc protrusion, ligamentum flavum hypertrophy, and facet arthrosis and osteophytes. Both CT and MRI have over 70% sensitivity for spinal stenosis, however, the specificity remains somewhat low due to the presence of radiographic evidence for spinal stenosis in over 20% of asymptomatic individuals over the age of 60.[9-12]

Spine Biomechanics

Panjabi was the first to demonstrate that degenerated segments caused a decrease in the intervertebral foraminal area compared to unaffected segments.[13] Mayoux-Benhamou also showed that foraminal dimensions were decreased when the spine moved from a position of flexion, to one of extension.[14] These studies were followed by biomechanical cadaveric studies. Schonstrom quantified the reduction in spinal

canal area at 40mm^2 during extension of the spine.[15] Inufusa carried out a similar study and found a 16% reduction in spinal canal cross-sectional area and a 24% reduction in foraminal cross-sectional area.[16] Schmid and Chung both performed MRI studies on asymptomatic individuals to determine if back extension caused similar narrowing, and found that canal area decreased by 16% and 61 mm^2, respectively.[17,18]

A critical area of 100mm^2 has been proposed for the dural sac, below which symptoms of NIC begin to appear.[19] Other scales for stenosis have been set forth based on central canal sagittal diameter, indicating that 15-18 mm is considered normal, 10–14 mm is considered relative stenosis, and below 10 considered absolute stenosis. A foraminal height of less that 15 mm and a posterior disc height of less than 4 mm have also been indicated to be parameters for stenosis.[20] Lumbar extension causes anterior buckling of the ligamentum flavum, as well as posterior bulging of the posterior annulus. These both contribute to narrowing of the central canal[15-18] as well as the lateral recesses and foramina,[13,14,16,21] which has been documented by several studies.[22,23] For patients in whom greater imaging detail is required, and MRI is contraindicated, CT myelogram may be used. Electromyography is generally not warranted due to its poor diagnostic utility in differentiating possible etiologies.[5]

Disease Progression

The disease course for spinal stenosis is generally stable and non-progressive. Therefore, prophylactic treatments are not warranted, as most symptomatic

patients report no significant changes at one year and their symptoms are thus unlikely to worsen.[24-26] The same reasoning, however, makes expectant management a poor strategy for patients with intolerable symptoms, as they are just as unlikely to improve. Clinical impression, then, can lead to either non-operative management or operative management.

Non-operative Treatment

Non-operative treatment consists largely of physical therapy and pain management. Pain control is generally achieved with acetaminophen, and if poorly controlled, progressing to non-steroidal anti-inflammatory drugs (NSAIDs) and then mild narcotic analgesics (if tolerated). Exercises to strengthen abdominal wall musculature may also help in relieving some degree of lumbar extension and should be encouraged. Abdominal corsets may be used to maintain a posture in slight flexion, however, use should be limited, so as not to result in paraspinal muscle atrophy. In general, exercises done in a position of flexion are more comfortable for patients, such as bicycling, and are thus better tolerated.[5] Epidural corticosteroid injections may also be tried; however, data on efficacy of this approach is limited. While patients often obtain temporary amelioration of symptoms for several weeks to months, functional improvement is often limited.[27,28]

Operative Treatment

For patients who fail conservative management, operative treatments are often indicated. Surgical intervention is aimed at decompressing the spinal canal and

foramina, thereby removing the source of pressure on the nerves. The traditional operative treatment is a laminectomy and facetectomy, with or without arthrodesis. Several studies support surgical intervention being superior to nonoperative management due to statistically significant improvement in leg and back pain.[29] The Spine Patient Outcomes Research Trial (SPORT) was the first large randomized clinical trial to be performed that compared traditional nonsurgical management with decompressive surgery.[57] Patients with a minimum of twelve weeks of symptoms, and imaging confirmed stenosis with no spondylolisthesis, were randomly placed and underwent either surgical decompressive surgery or nonsurgical management. Outcomes measured in this trial were bodily pain and physical function on the SF-36 and modified Oswestry Disability Index at 6 weeks, 3 months, 6 months, one year, and two years. Although complicated by nonadherence, as-treated analysis showed a statistically significant improvement in patients who underwent decompressive surgery when compared to those who received nonsurgical management in all measures of outcome and at all time points.[57] Follow-up reports of the same ongoing trial have continued to show statistically significant improvement in the surgically treated group, currently up to four years.[58] Observational studies also support surgical intervention, with greater pain relief achieved earlier and superior improvement in functional status shown in surgical patients. Although, the difference narrowed over the course of follow-up.[30] Complications for decompressive laminectomies for lumbar spinal stenosis include perioperative mortality (0.32%), dural tears (5.91%), deep infection (1.08%),

superficial infection (2.30%), deep vein thrombosis (2.78%), and overall complication rate (12.64%).[5]

Interspinous Spacers

A less invasive method for decompression of the neural elements is distraction of spinous processes with an interspinous spacer. Within the marked postural dependence of NIC lies the interspinous process spacer's key.[22] The aim of an interspinous spacer (ISS) is to restore foraminal height, expand central canal area, unload the facet joints, and provide stability in extension, while maintaining normal motion and conferring less risk of adjacent segment degeneration.[31]

Symptomatic improvement of NIC during flexion at the stenotic segment results from decreased epidural pressure, decreased nerve compression, and increased area of the spinal canal and intervertebral foramen.[16,32,33] Interspinous spacers were developed to relieve symptoms based on the principle that flexion brings about neural decompression and relieves symptoms of NIC. The first interspinous implant was developed in the 1950s by Knowles, however, its use was abandoned due to flaws in design, material, technique, and indications for use.[34] Newer renditions appeared in the 1990s in Europe and South America to improve clinical outcomes after discectomy.

The first FDA-approved interspinous spacer, the X-STOP, was approved in the United States for use in treatment of NIC in 2005.[34] The spacer is indicated for use in patients over the age of 50 with moderate symptoms of NIC secondary to stenosis confirmed by imaging (XRAY, MRI and/or CT) and who experience relief of

symptoms with flexion. Patients who have undergone 6 months of nonoperative management with failed improvement, and in whom no more than 2 levels are indicated, can be considered for placement of one or two level lumbar decompression with the spacer.[59]

The interspinous spacer is inserted between adjacent spinous processes and forces the adjacent vertebra into a position of flexion. The spacer is placed under general or local anesthetic, with the patient in lateral decubitus positioning.[59] Alternatively, the patient can be positioned prone with the lumbar spine placed in flexion. Utilizing a midline incision, the paraspinal muscles are stripped from the spinous processes, while preserving the supraspinous and interspinous ligaments.[23] The spacer is not fixed to bone, however, movement is restricted caudally and cranially by the spinous processes, and anteriorly and posteriorly by the laminae and supraspinous ligaments, respectively.[1]

Several contraindications to placement of an interspinous spacer exist, including titanium allergy, severe osteoporosis, cauda equina syndrome, severe scoliosis (Cobb angle > 25º), ankylosed segments at affected level, acute fracture of spinous process or pars interarticularis, and systemic infection at the time of surgery. Additionally, high failure rates have been reported when used in patients with stenosis due to spondylolisthesis and it should be avoided in this population as well, in favor of laminectomy and fusion.[35]

By forcing the spinous processes apart at the level of the device, the adjacent vertebrae are put in a position of flexion, preventing extension. The design of the

implant is such that it aims to prevent motion in only one direction (preventing extension) without restricting motion in any other plane. Several studies have shown that this is in fact the case, and extension is restricted without affecting range of motion in flexion, lateral bending, or axial rotation at the decompressed level. Importantly, motion at adjacent levels remains unaffected. Additionally, the range of motion in flexion–extension as well as axial rotation and lateral bending were not significantly affected at adjacent segments.[1,31]

Sobottke showed that foraminal cross-sectional area, foraminal height, foraminal width, intervertebral angle and disc height improved significantly with spacer use. Using the Visual Analog Scale [VAS], postoperative symptom relief was significant. Interestingly, the study found only a weak correlation of radiographic parameters with symptom relief, and actually noted a dissociation at follow-up with radiographic measurement trending towards preoperative values, while VAS scores continued to show symptomatic relief.[20]

Interspinous spacers have been shown in multiple studies to increase both the spinal canal area and the foraminal area in cadaveric studies, as well as in MRI evaluated patients.[23,36,37] A study by Siddiqui evaluated patients preoperatively and postoperatively using MRI and found that cross-sectional areas of both the central canal as well as foramina were significantly increased after placement of an interspinous spacer.[23] Richards conducted another study using MRI that evaluated identical radiographic parameters, but with the spine in 15 degrees of flexion and 15 degrees of extension. Implantation of a spacer increased the cross-sectional area

of the foramina by 25%, the foraminal width by 41%, the subarticular diameter by 50%, and the spinal canal area by 18%.[36]

The concern for degeneration of adjacent levels that exists in cases of lumbar spinal fusion has also been studied repeatedly with interspinous spacer use. Multiple clinical, observational and biomechanical studies of post-arthrodesis spines report radiographic evidence of adjacent level changes as well as adjacent level instability and failure.[3] The increased stress at adjacent levels in the form of increased intradiscal or facet joint pressures that are found with spinal fusions are not found with placement of interspinous spacers.[3,38] In fact, a study by Adams showed that there is actually a decrease in posterior pressure, possibly due to a fulcrum effect of the device. Spacers have been found to off-load stress at the facet joints, with a pressure reduction of 55%.[38,39] Restoration of disc height also led to a decrease in intradiscal pressure of up to 63%, which may even promote disc regeneration.[3,23]

The success rate with interspinous spacer use was originally reported to be 78%, which was corroborated by several other studies.[34,37,40,41] Complication rates were reported to be around 7% (including 6% for device failure).[42] Another study by Chou supported use of interspinous spacers based on modified-network meta-analysis showing that ISS were superior using both disability and pain as outcomes at 12-months follow-up.[43] However, more recent publications have suggested that the success rate may be somewhat lower, and the complication rate higher than originally reported.[44,45]

Zucherman showed that at one-year follow-up 75% of patients continued to have improvement in symptoms and 70% were satisfied with their treatment.[40] Lee conducted a similar study, though with variable follow-up times ranging from 9 to 18 months, and found similar satisfaction rates, but with only 40% with improved symptom severity.[37] Siddiqui found symptom severity improvement in 54% with a 71% satisfaction rate, and notes that maximal clinical improvement occurs by 3 months, then gradually declines. All unsatisfied patients in this study opted for caudal epidural injection of corticosteroids after 12 months over decompressive laminectomy.[23] Kondrashov conducted a longer-term 4-year follow-up study and found similar results to Zucherman's original study. The Oswestry Disability Index was used and they determined that 78% had successful outcomes.[34] A study by Pratt in 2004 showed that both the ODI and ZCQ are well correlated.[23]

Other options for treatment of NIC include conservative nonsurgical management and surgical decompression laminectomies. In order to provide a comparison to other current treatment regimens, several studies have also looked at outcome measurements with decompressive laminectomies. Johnsson found that in surgically treated patients, 60% graded their condition as improved.[46] Amundson found that 31% considered their pain to be completely resolved or only slightly present.[47] Atlas conducted several studies as well, showing that 50-70% considered their symptoms to be improved, with 60-70% satisfied.[30] Instituting a more strict definition for clinical improvement, Gunzburg reported 58% improvement.[48] Comparing decompressive laminectomy and ISS data it is apparent that outcomes between the two procedures are quite similar.

The mean procedure time for placement of an interspinous spacer is reported to be 54 min, which is significantly lower than the mean operative time for a decompressive laminectomy of 114 minutes. Mean blood loss is also less for spacer placement at a mean of 46cc, versus a range of 120-1040cc for lamincectomies.[41]

Cost Effectiveness

Interspinous spacer decompression has been shown in several studies to be superior and more cost effective than nonsurgical conservative management.[40,42] Additionally, they have been shown to be cheaper and possibly more cost effective at 12 months than laminectomies due to lower complication rates.[43,49] The favorable cost-effectiveness compared to conservative management has been attributed to the high treatment failure rate. When compared against laminectomy, the favorable cost-effectiveness stems from the higher incidence of adverse events. The determined cost per quality-adjusted life year for placement of an interspinous spacer was $17,894, which is much lower (47%) than the $37,856 for decompressive laminectomy with fusion.[49]

The most common reason for failure of ISS has been reported to be indication error, followed by technical errors, anatomic variation of patients, and device failure.[50,51] Reported causes of device failure consist of device dislocation, spinous process fracture, and erosion of the spinous process beneath the device.[50,52-54] Although spinous process fractures have been reported to range from 1%-5%, they are difficult to visualize on plain X-rays, and a CT based study found an occult spinous process fracture rate of 22%, and patients reported no traumatic incidents. Patients

with fractures tended towards poorer outcomes and lower satisfaction rates, compared to patients without fracture. However, the majority of patients with fracture denied experiencing any back pain.[52] Some decrease in distraction may also be attributable to bony erosion of spinous process.

Biomechanical studies have shown that lateral failure load is much less than the mean lateral load during insertion, and therefore fracture is unlikely to occur during placement of the device. This is supported by the very low intraoperative fracture rate. Fracture, then, may have more to do with the role of the spinous process changing from one of tension to one of compression load bearing.[55]

With a large amount of published data on the effectiveness of interspinous spacers and understanding its mechanism of function, there is a paucity of studies to better understand patients who fail to improve. In the absence of indication error or device failure, there is a subset of patients who fail to improve clinically after placement of the device. The mechanism of this failure is currently unknown.

It is unclear why these patients, who have symptomatic relief with forward flexion of the spine, fail to respond when the stenotic segments are forced into flexion by the spacer. It is possible that for some reason, the interspinous spacer fails to force the lumbar vertebra into flexion, thereby failing to bring about anatomic decompression, and the neural elements remain compressed as a result. The aim of this study was to carry out a critical analysis of vertebral column dimension on a series of patients in whom the ISS had failed symptomatic improvement. By studying vertebral column dimensions in these patients we hope to gain further

insight into the cause of ISS failure in these patients, and determine whether the spacer has in fact failed to decompress the spine, or whether the expected anatomic decompression has occurred.

Statement of Purpose:

The purpose of this study is to compare preoperative and follow-up MRIs of the lumbar spine in patients with persistent symptoms of NIC, who had been treated with interspinous decompression, to determine an anatomic explanation for persistent claudication despite proper placement of interspinous spacers or device failure.

Hypothesis:

In a subset of patients, although the interspinous spacer is placed correctly, it fails to bring about the anatomic decompression expected and thus canal and foraminal dimensions are not significantly increased, leading to persistent claudication.

Materials and Methods:

The records from all patients who underwent treatment with interspinous devices by our senior author during the period from 2010 through 2013 were reviewed, during which time 57 patients had undergone implantation of an X-STOP interspinous spacer. Ten patients were identified retrospectively who developed recurrent symptoms of neurogenic claudication and who underwent a subsequent MRI. Original indications for surgery were patients with symptoms of neurogenic claudication with radiographic evidence of corresponding spinal stenosis, with symptomatic worsening with standing and improvement with sitting or leaning forward, and who had failed at least six weeks of conservative treatment. All patients had undergone surgical implantation of the X-STOP interspinous implant using the technique described by Zucherman et al.[40] Patients who developed recurrent symptoms of NC after implantation of the interspinous spacer underwent subsequent MRI imaging at follow-up. Recurrent NC was defined as a subjective return of original symptoms of neurogenic claudication after a period of relief within two years after implantation of the interspinous spacer. MRIs were obtained without contrast and were performed at a number of different institutions with varying magnet strength. All patients had implants at either one or two levels, the majority being in the L3-L4 and L4-L5 levels. Patients included in the study showed no signs of gross mechanical implant failure, such as implant displacement or spinous process fracture. A careful history and focused physical exam were used in combination with imaging studies to rule out alternative mechanical, neurologic or

vascular etiologies. IRB approval was obtained to review and record anonymous observations from this series of preoperative and subsequent MRIs.

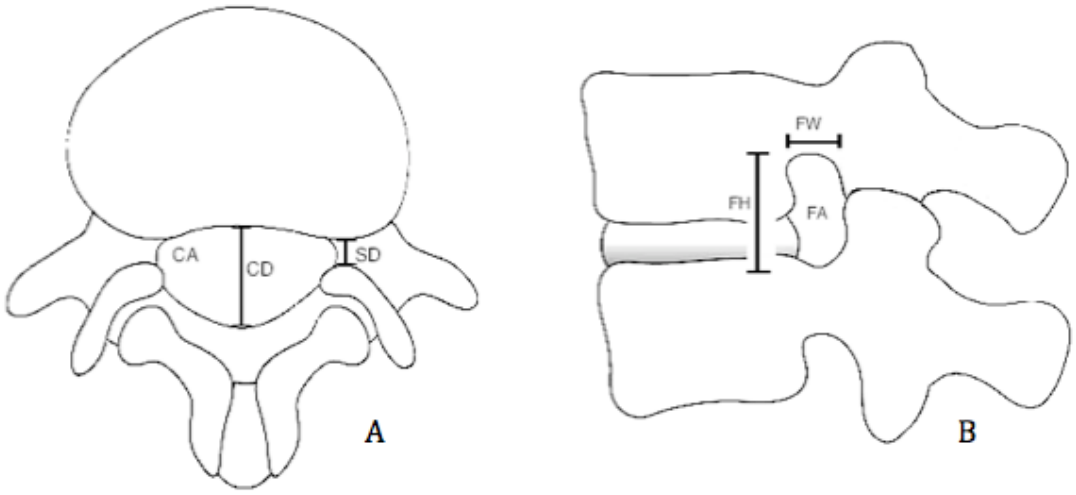


Figure 1. A schematic of the measurements for both the axial (A) and sagittal (B) MRI slices. CA = Canal area; CD = Canal diameter; SD = Subarticular diameter; FA = Foraminal area; FH = Foraminal height; FW = Foraminal width.

Data Collection

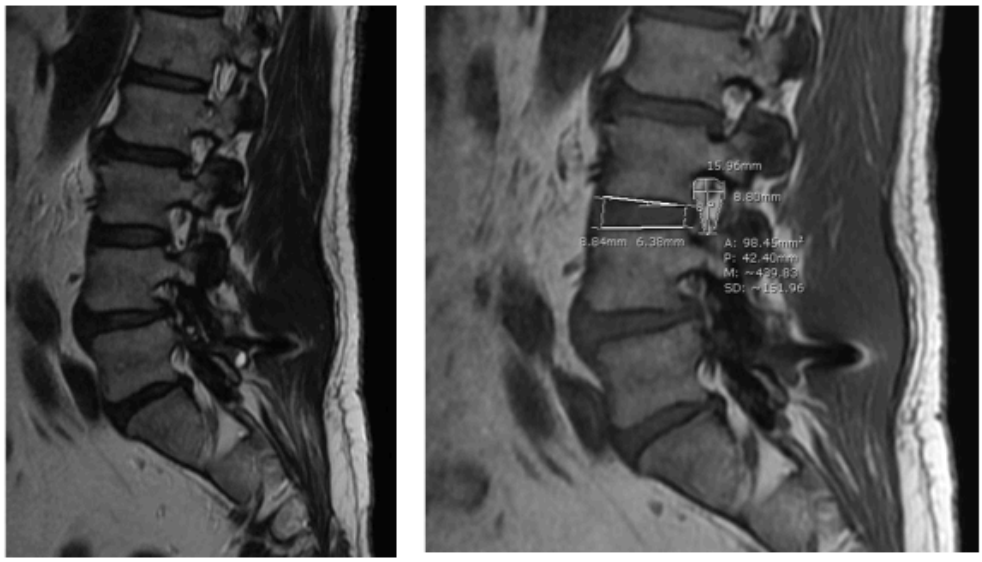
All MRIs were obtained with patients positioned supine. Noncontinguous axial images parallel to the intervertebral discs were used to measure canal area, canal diameter, and subarticular diameter. T2 weighted sagittal images were used to measure foraminal area, foraminal height, foraminal width, anterior disc height, posterior disc height, and intervertebral angle. See Figure 1.

A PACS system was used to measure all variables. Linear measurements were taken using the PACS standardized ruler. Area measurements were taken using a PACS freehand area measurement tool. (See Figure 2) For each patient, all measurements were taken at implanted levels as well as adjacent levels in both preoperative and

subsequent series. All measurements were done in duplicate by two different observers; one fourth-year orthopaedic surgery resident and one fourth-year medical student.

Data Analysis

The left and right side measurements of the subarticular diameter, foraminal area, foraminal width, and foraminal height were pooled. The mean values of these pooled measurements as well as the mean values for canal diameter, canal area, anterior disc height, posterior disc height and intervertebral angle were compared between preoperative and subsequent series for each level and in both operative levels and adjacent levels. For each measurement or pooled measurement, the mean percentage change was calculated, and the means were compared between each pair of levels and those with an ISS versus adjacent segments. The comparisons were done using Welch's t-test, with a significance level of 0.05.



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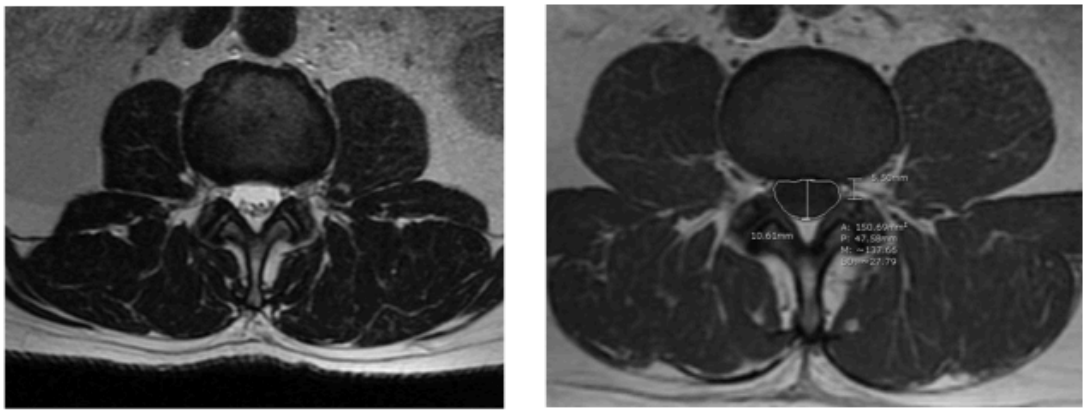

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Figure 2. Example measurements taken from both the sagittal **(A)** and axial **(B)** MRI slices.

Results:

A total of 10 patients were studied with a mean age of 61.4 (age range 47 - 78), and a gender split of seven males and three females. All preop MRIs were done within 2 months before the surgery and the mean time to follow-up MRI was 11.7 months. See Table 1.

Table 1. Characteristics of patient population included in this study, including age at surgery, gender, and the levels that were implanted with an ISS.

Patient Age	Patient Gender	Levels implanted
62	Female	L3-L4, L4-L5
66	Male	L3-L4, L4-L5
64	Male	L3-L4, L4-L5
60	Male	L4-L5
59	Male	L3-L4, L4-L5
47	Female	L3-L4, L4-L5
51	Male	L3-L4, L4-L5
65	Female	L2-L3, L3-L4
62	Male	L3-L4, L4-L5
78	Male	L3-L4, L4-L5

Canal Area:

At levels with implanted spacer devices, the mean preoperative canal area was 85 mm^2, and the mean area at follow-up was 91 mm^2 (Table 2). Although the mean percentage increase of the canal area was 8%, there was a large variability between patients, with a standard deviation for mean increase of 32% (Table 3). This

increase was not statistically significant (p=0.3946). The 8% increase at implanted levels was only slightly higher than that of unimplanted levels, at 3%. This difference was not significant, however (p=0.6142).

Canal Diameter:

The canal diameter had a mean preoperative distance of 8.4 mm, with a diameter of 8.6 mm at follow-up, which was not statistically significant (Table 2). The percentage increase in canal diameter at levels with implanted spacers was 5%, while in unimplanted levels, there was a 5% decrease seen (Table 3). However, this difference did not reach significance (p=0.1064).

Subarticular Diameter:

The subarticular diameter for the implanted L3-L4 and L4-L5 levels showed a much larger increase in distance, with a 58% and 53% increase in diameter at the two levels, respectively. The unimplanted levels at L2-L3 and L5-LS1 actually showed minor decreases in distance of 13% and 5%, respectively. These differences were significant for the implanted levels (p=0.00354) but the change was not significant for the unimplanted levels (0.1042). The mean preoperative diameter was 1.9 mm, compared to 2.5 mm at follow-up, which was statistically significant, with a p-value of 0.0035 (Table 2). The mean percentage increase in subarticular diameter of both of the implanted levels (45%) was significantly greater than at the unimplanted levels (-8%), with a p value of 0.68e-8 (Table 3).

Foraminal Area:

Foraminal area, including both the right and left side, showed an increase in mean area from 60 mm^2 preoperatively, to 68 mm^2 at follow-up. This was a statistically significant increase in areas, with a p-value of 0.025. At the implanted levels of L2-L3 and L4-L5, the mean increases in total foraminal area were 20% and 40%, respectively, compared to very small changes at adjacent, unimplanted levels (3% decrease at L2-L3 and a 1% increase at L5-S1). The percentage increase in foraminal area was significant at implanted levels and not at unimplanted levels, with a p-value of 0.2057 (Table 2). When the mean percentage increase at both implanted levels (23%) was compared with the mean percentage change in area at unimplanted levels (3%), the percentage increase was significant compared to unimplanted levels, with a p-value of 0.001443 (Table 3).

Foraminal Width:

The mean foraminal width increased from 8.0 mm preoperatively to 8.6 mm at follow-up. The percentage change in the foraminal width was relatively unchanged at unimplanted L2-L3 and L5-S1 levels at 5% and <1%. At the implanted levels L3-L4 and L4-L5, only L4-L5 was increased, at 19%, compared to the unimplanted levels. The L3-L4 level was similar to the unimplanted levels at 5%. Mean increase at implanted levels was 10%, and 2% at unimplanted levels (Table 2). The change in implanted levels was significant (p=0.02435) while that at unimplanted levels was not (p=0.958). The difference between implanted and unimplanted levels, however did not reach significance, with a p-value of 0.1083 (Table 3).

Foraminal Height:

The foramen at L3-L4 and L4-L5 implanted levels showed a mean 10% and 18% increase in height, respectively. The mean preoperative foraminal height was 10.2 mm, with a height of 11.3 mm at follow-up. The mean height increased by 3% at the unimplanted L2-L3 level and decreased by 2% at the L5-S1 level. The increase in height did not reach significance at the implanted level (p=0.0605), nor at the unimplanted level (p=0.3767). The mean percentage height changes of implanted levels (14%), however, was significantly higher than that of unimplanted levels (0.3%), with a p-value of 0.01198 (Table 3).

Disc Height and Angle:

The anterior disc height of implanted levels showed a mean decrease from 8.5 mm preoperatively, to 7.5 mm at follow-up. This decrease in height, however, was not significant (Table 2). The implanted levels showed a decrease of 11%, while that of unimplanted levels showed a mean increase in height of 8% (Table 3). Anterior disc height was significantly decreased compared to unimplanted levels (p=0.004128).

The posterior disc height had a mean preoperative distance of 4.5 mm, with a height of 3.7 mm at follow-up. This decrease in height was statistically significant, with a p-value of 0.0223. When changes in implanted height were compared to changes in unimplanted height, however, they were not significant (p=0.1104), with implanted levels showing a mean decrease of 17%, and unimplanted levels showing a mean height increase of 0.3% (Table 3).

Intervertebral angle of implanted levels showed a mean percentage increase in angle of 21%, while angles at unimplanted levels showed relatively little change, at

3%. This difference did not reach statistical significance, however, with a p-value of 0.5087.

Table 2: Mean values for vertebral dimensions measured in the study. Lengths are reported in mm and areas in mm^2.

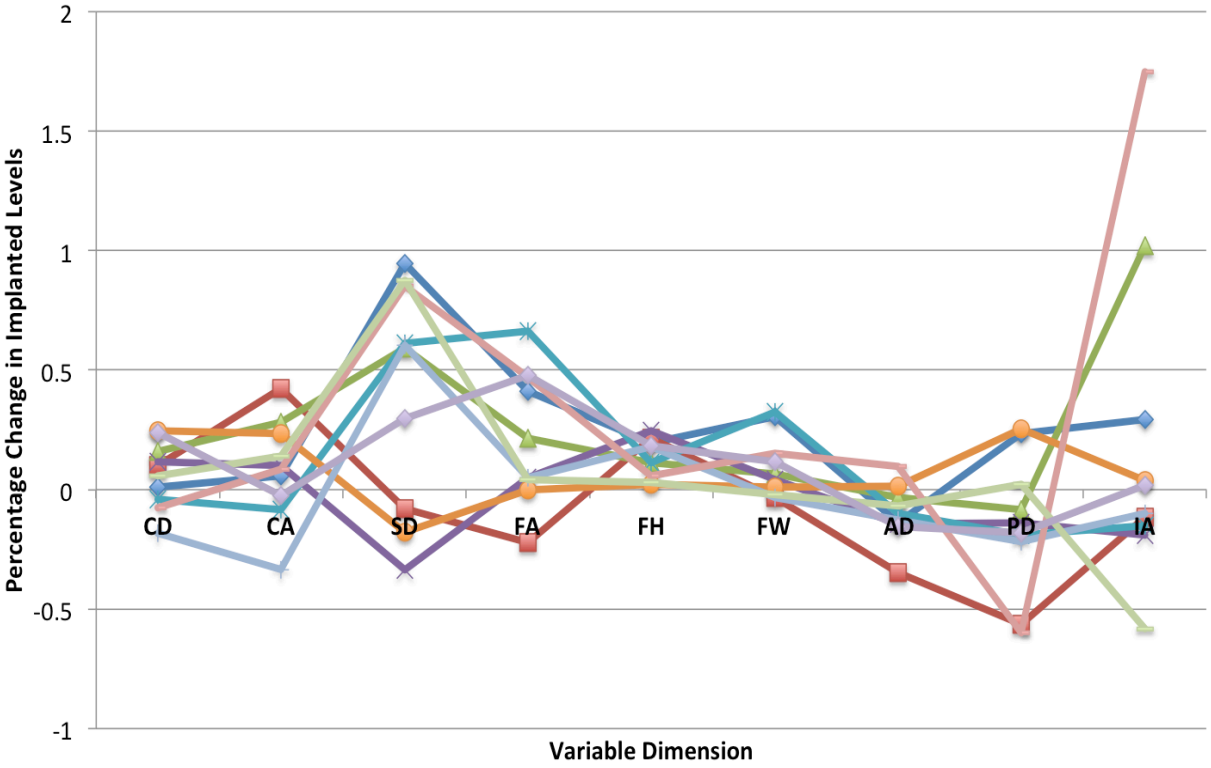
Variable Dimension	Pre-op Mean ± SD	Post-op Mean ± SD	Significance Level (p-value)
Canal Area	84.6 ± 28.1	90.8 ± 34.1	0.3946
Canal Diameter	8.4 ± 2.1	8.6 ± 2.1	0.7751
Subarticular Diameter	1.9 ± 1.0	2.5 ± 1.1	0.0035
Foraminal Area	60.1 ± 23.8	68.1 ± 19.8	0.0251
Foraminal Height	10.2 ± 3.6	11.3 ± 3.3	0.0605
Foraminal Width	8.0 ± 1.6	8.6 ± 1.5	0.0244
Anterior Disc Height	8.5 ± 3.0	7.5 ± 3.1	0.1704
Posterior Disc Height	4.5 ± 1.5	3.7 ± 1.5	0.0223
Intervertebral Angle	7.9 ± 4.7	7.4 ± 4.3	0.5787

Table 3. Percentage change in dimensions of canal and foramina between preoperative to follow-up MRIs in both ISS implanted levels, compared to adjacent non-implanted levels.

Variable Dimension	Mean Increase in implanted levels (%) ± SD	Mean Increase in adjacent non-implanted levels(%) ± SD	P-value
Canal Area	8.0% ± 32.2	3.3% ± 24.3	0.6142
Canal Diameter	5.3% ± 24.0	-5.0% ± 12.6	0.1064
Subarticular Diameter	45.0% ± 68.4	8.2% ± 33.7	0.000068
Foraminal Area	22.7% ± 39.1	3.2% ± 28.8	0.0014
Foraminal Height	14.2% ± 23.4	0.3% ± 26.3	0.0120
Foraminal Width	9.8% ± 24.1	1.7% ± 19.3	0.1083
Anterior Disc Height	-10.6% ± 20.1	8.3% ± 18.5	0.0041
Posterior Disc Height	-16.8% ± 35.4	0.3% ± 29.2	0.1104
Intervertebral Angle	20.7% ± 102.9	3.4% ± 48.0	0.5085

Figure 3 shows the percentage change in implanted levels for each patient. The variable dimensions are listed along the x-axis, and the percentage change at implanted levels is given on the y-axis. Each line corresponds to a different patient in the study. This figure shows that some dimensions were more consistent across patients (CD, FH, FW, and AD), while other dimensions showed a wide variation among patients (CA, SD, FA, PD, and IA). The trajectory of each individual patient could be used to further analyze the cause of implant failure.

Figure 3. Percentage change in dimensions of canal and foramina for implanted levels between preoperative to follow-up MRIs. Each line corresponds to a patient in the study.



To look at inter-rater reliability, R^2 values were determined for each parameter. The average value was 0.91, indicating high reliability of measurements between the two referees. The lowest correlation was 0.71, which still shows very high correspondence between the two referees. R^2 values for each individual parameter measured are presented in Table 4 below.

Table 4: R^2 Values are presented for each of the MRI parameters measured in the study.

Parameter	R^2 Value
Canal Diameter	0.95
Canal Area	0.98
Right Subarticular Diameter	0.91
Left Subarticular Diameter	0.89
Right Foraminal Area	0.98
Left Foraminal Area	0.97
Right Foraminal Height	0.94
Left Foraminal Height	0.91
Right Foraminal Width	0.84
Left Foraminal Width	0.71
Anterior Disc Height	0.95
Posterior Disc Height	0.92
Intervertebral Angle	0.88

Discussion:

Based on the results of this study it does not appear as if the interspinous spacer device failed to improve canal and foraminal dimensions. Despite the development of recurrent symptoms, the implants still appear to be increasing the dimensions of the spinal canal. The biomechanical principle on which the device was designed is that positioning the vertebrae in relative flexion causes a relative increase in space available for the neural elements.

Several studies have been published investigating the changes in the vertebral dimensions from a position of flexion, to a position of extension, in normal vertebral columns. Chung et al. looked at the variation in canal area, canal diameter and subarticular diameter in normal healthy patients in the supine position using MRI in both flexion and extension positioning. They found a mean canal area of 399 in a flexed spine and 331 in an extended spine as well as reductions in both the canal diameter and subarticular diameter.[18]

Fujiwara et al. conducted a similar study using normal cadaveric spines. Helical CT was then used to determine foraminal dimensions throughout flexion-extension, lateral banding, and axial rotation movements. The authors also found a statistically significant increase in foraminal area, height and width with flexion, while the same parameters were significantly decreased during extension.[21]

Inufusa et al. conducted a study on frozen cadaveric lumbar vertabra as well, and determined the changes in vertebral dimensions when moving into a position of flexion as well as into a position of extension under load. Dimensions were

determined using CT imaging of frozen spines and a cryomicrotome, and nerve root compression was also examined. The authors reported a statistically significant increase in parameters during a position of flexion (mean increase of 11% in canal area and 12% increase in foraminal area), and significant decrease in parameters in a position of extension, which held true throughout varying degrees of stenosis (normal, relative stenosis, and absolute stenosis). See Table 5. They also noted that nerve root compression was increased in extended spines and decreased in flexed spines, and the decreased nerve root compression correlated with increased vertebral parameters.[16]

Schmid et al. also published studies on normal spinal anatomy of healthy patients using MRI in seated neutral, flexed, and extended positioning as well as supine extended positioning. The authors also found similar reductions of dimensions during extension and increases in flexion compared to previous studies, but with slightly reduced mean canal areas compared to Chung et al. Interestingly, they also found that the decreases in dimensions were not as prominent in the supine extended position from neutral, as compared to the seated extended position from neutral.[17]

Albietz et al. published a study on the anterior disc height, posterior disc height, and interveterbral angle of healthy patient spines. Mean anatomic dimensions are presented in Table 5. He also noted, however, that there was poor correlation between disc height and angle, with interspinous height.[60]

The biomechanical principles proven in the above studies of normal living and cadaveric spines held true in our study as well. Even in these patients who had failed symptomatic improvement with the interspinous spacer, we found that there were significant increases in several of the dimensions that were measured, although subarticular diameter and foraminal area were the most pronounced.

Previously, published studies investigating the effect of the interspinous spacer device on canal and foraminal dimensions include both cadaveric and in-vivo models. A cadaveric study by Richards et al. demonstrated that in extension, the ISS increased the canal area by 18%, the subarticular diameter by 50%, the canal diameter by 10%, and the foraminal area by 25%. This study design differed from the present study in that patients included in their study had device implantation at a single level only, L3-L4, MRI imaging was taken, and changes in canal and foramen dimensions were examined, in both flexion and extension positioning. While the study results of Richards et al. demonstrated increases in dimensions in all positions, a statistical difference was found only when the specimens were placed in a position of 15 degrees of extension. Comparing our results to the results found in this cadaveric model, we found a similar increase in the majority of the parameters. The patients in our study were imaged in a neutral position and when the percentage change in dimensions between our study and that of Richards et al the canal area, canal diameter, subarticular diameter, foraminal area, foraminal height, and foraminal width are all increased more so than the neutral in Richard's study. (See Table 5) Several of the differences are not significant, however this is true in both our study, as well as in the cadaveric study. The difference was much

more pronounced with higher levels of significance in a position of extension in the cadaveric study, however this was not done in the current study. The correlation between this ex-vivo model of ISS biomechanical function and our study suggests that the ISS succeeded in increasing the anatomic space available.

Several in-vivo studies have also been published, some of which use MRI, CT or dynamic fluoroscopy to evaluate anatomic changes induced by interspinous spacers. Siddiqui et al used MRI studies to determine that there was a postoperative increase in foraminal area by 16% and 17% in extension and flexion, respectively.[23] Interestingly, these findings were only statistically significant on the left side. These percentage increases are comparable to our values. Their study did not use static MRI, but instead measured changes in the foramen and spinal canal through a range of motion.

An additional study, recently published by Wan et al, used three-dimensional image modeling of the vertebral column to record preoperative and postoperative dimensions from patients who had undergone treatment with an interspinous spacer. Similar to previous studies, they found the most significant improvement in anatomic dimensions when the patients were positioned in extension, indicating that the interspinous spacer worked to prevent narrowing of the canal and foramen in this position, but did not reach statistical significance when the vertebral column was held in a neutral or flexed position.

Sobottke conducted a study to measure radiographic decompression as well as pain improvement, using the visual analog scale [VAS] after treatment of lumbar spinal

stenosis with an interspinous spacer. He then compared radiographic improvement with pain scores. He noted that after treatment with interspinous devices, postoperative imaging consistently showed improvement in radiographic parameters for effective decompression, specifically showing significant increases in foraminal cross-sectional area, foraminal height, foraminal width, intervertebral disc angle as well as anterior and posterior disc height. In addition, he found significantly improved pain scores in the same patient population. Interestingly, he noted only a very weak correlation between the magnitude of radiographic improvement and the degree of pain improvement. Furthermore, at follow-up imaging, radiographic parameters tended to revert or trend towards original values, while pain scores continued to remain improved.[20]

A study by Lee et al. evaluated preoperative and postoperative MRIs in a series of ten elderly patients undergoing treatment with an ISS device. Patients were evaluated with preoperative and postoperative MRIs, in a similar manner to our study. Nine out of the ten patients included in their study had undergone single-level placement of the device. Their results demonstrated a 23% increase in area of the canal and a 36% increase in foraminal area compared to 7.3% and 13%, respectively, in our study.[37] The increase in both of these dimensions, but particularly that for canal diameter, are larger than the increases detected in our study. Though this finding may be due to differences in measurement technique, it is possible that patients in our study with recalcitrant symptoms of NIC may have had less successful decompression of their spinal canals relative to their foramen. It is interesting to note that although the canal area in our study was not significantly

increased, the percent increase in area is comparable to what has been reported in many of the other studies and therefore may be limited by our small patient population. However, it is also important to realize that although the percentage increase in canal area was comparable to other studies at nearly 8%, the baseline mean area was quite low at 84 mm^2. In these patients, the mean canal area at follow-up was increased to 91 mm^2, which is still below the suggested 100 mm^2 minimal dural sac area required to prevent stenosis.[19]

It is important to note also, that the results of our study further corroborate previously published studies stating that ISS use does not affect anatomic dimensions of adjacent levels as seen in Table 3.

Our study is limited by the static nature of our preoperative and follow-up MRIs. As other studies have demonstrated, the ISS is most effective at preventing neural compression with the spine in a position of extension. Danielson also showed a significant reduction in spinal canal cross-sectional area when axially loaded.[56] This study does not address dimensions in that position, as all MRIs were done with patients in a supine position. Siddiqui's study compared erect, seated-neutral, and seated extension position and notes that although there were increases in all positions, the increases were only significant in erect and seated neutral positions.[23] This study also did not include a comparison of canal and foraminal dimensions immediately postoperatively. It would potentially be useful to have immediate postoperative radiographic studies, which could be compared to studies obtained following recurrence of symptoms. Such a study could potentially identify settling of the vertebrae, change in implant position, or worsening of stenosis as a

cause of recurrent NIC. The lack of an internal control may mask some of these postoperative changes, and subsequently, some additional information as to the cause of recurrent claudication.

Despite the limitations of this study, however, we found that there were significant improvements in stenosis as measured on preoperative and follow-up MRI in this group of patients with recurrent NIC. We conclude that the failure of the ISS device to resolve symptoms is not due to its inability to increase space available for the neural elements, and that further investigation is required to elucidate the reasons for recurrent symptoms of claudication in patients who have undergone interspinous spacer decompression.

Table 5: Published Mean Values and Percent Changes of Spinal Dimensions

		Canal Area	Canal Diameter	Subarticular Diameter	Foraminal Area	Foraminal Height	Foraminal Width	Anterior Disc Height	Posterior Disc Height	Intervertebral Angle
Chung et al[18]		Flex: 399 Ext: 331	Flex: 25 Ext: 23	Flex: 5.7 Ext: 3.2	-	-	-	-	-	-
Fujiwara et al[21]		-	-	-	Flex: 104 Ext: 83.9	Flex: 17.9 Ext: 18.2	Flex: 4.0 Ext: 2.2	-	-	-
Inufusa et al[16]		Flex: 248 Ext: 208	Flex: 20.2 Ext: 17.7	Flex: 5.8 Ext: 4.7	Flex: 141 Ext: 107	Flex: 20.0 Ext: 20.3	Flex: 5.8 Ext: 3.5	-	-	-
Schmid et al[17]		Flex: 268 Ext: 224	-	-	Flex: 167 Ext: 115	-	-	-	-	-
Albietz et al[60]		-	-	-	-	-	-	11	5.5	10 (L4-L5) 14 (L5-S1)
Lee et al[37]		Pre: 73.6 Post: 90.2 Δ: 22.6%	-	-	Pre: 60.3 Post: 82.3 Δ: 36.5%	-	-	-	Pre: 5.9 Post: 7.7 Δ: 31%	Pre: 11.1 Post: 9.0 Δ: - 19%
Richards et al[36]	flexed	No ISS: 286 ISS: 276 Δ: 3.5%	No ISS: 19.3 ISS: 19.0 Δ: -3.2%	No ISS: 4.5 ISS: 4.1 Δ: -8.9%	No ISS: 149 ISS: 147 Δ: -1.3%	No ISS:23.2 ISS: 22.4 Δ: -3.4%	No ISS: 5.8 ISS: 6.0 Δ: 3.4%	-	-	-
	neutral	No ISS: 251 ISS: 265 Δ: 5.6%	No ISS: 18.3 ISS: 18.9 Δ: 3.3%	No ISS: 3.2 ISS: 3.7 Δ: 16%	No ISS: 128 ISS: 137 Δ: 7.0%	No ISS:21.2 ISS: 22.1 Δ: 4.2%	No ISS: 4.7 ISS: 5.0 Δ: 6.4%	-	-	-
	extended	No ISS: 231 ISS: 273 Δ: 18%	No ISS: 17.8 ISS: 19.5 Δ: 9.6%	No ISS: 2.5 ISS: 3.7 Δ: 48%	No ISS: 106 ISS: 133 Δ: 25%	No ISS:21.3 ISS: 21.2 Δ: -0.5%	No ISS: 3.4 ISS: 4.8 Δ: 41%	-	-	-
Siddiqui et al[23]	flexed	Pre: 97.7 Post: 99.8 Δ: 2.1%	-	-	Pre: 97.8 Post: 114.9 Δ: 17.6%	-	-	-	-	-
	extended	Pre: 84.4 Post: 92.4 Δ: 9.5%	-	-	Pre: 82.3 Post: 95.75 Δ: 16%	-	-	-	-	-
Sobottke et al[20]		-	-	-	Pre: 157 Post: 208 Δ: 35.5%	Pre: 19.3 Post: 22.6 Δ: 17.5%	Pre: 9.2 Post: 11.1 Δ: 28.3%	Pre: 10.7 Post: 9.2 Δ: -12.6%	Pre: 5.9 Post: 8.6 Δ: 52.8%	Pre: 6 Post: 1
Current study		Pre: 84.6 Post: 90.8 Δ: 7.3%	Pre: 8.4 Post: 8.6 Δ: 2.4%	Pre: 1.9 Post: 2.5 Δ: 32%	Pre: 60.1 Post: 68.1 Δ: 13%	Pre: 10.2 Post: 11.3 Δ: 11%	Pre: 8.0 Post: 8.6 Δ: 7.5%	Pre: 8.5 Post: 7.5 Δ: -12%	Pre: 4.5 Post: 3.7 Δ: -18%	Pre: 7.9 Post: 7.4 Δ: 6.3%

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