Management of Lumbar Spinal Stenosis through the Use of Translatoric Manipulation and Lumbar Flexion Exercises: A Case Series

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Abstract: Lumbar spinal stenosis is a narrowing of the spinal canal or intervertebral foramen that can produce low back pain and leg pain and weakness. Surgical intervention is commonly performed to relieve these symptoms. Symptom reduction and longitudinal management of functional deficits with conservative care is less well documented. The purpose of this case series was to describe the outcomes of a conservative physical therapy program consisting of low- and high-velocity translatoric manipulations of T1-T9 and L1-L3, and two lumbar flexion exercises on 6 subjects diagnosed with lumbar spinal stenosis and neurogenic claudication. A treadmill test was repeated on a weekly basis and at discharge for each patient. All six subjects demonstrated improvements in treadmill walking time prior to the onset of neurogenic claudication (range: 1 min 34 sec to 26 min); in Oswestry Low Back Pain Disability Index scores (range: 7.5% to 64.7%); and in McGill Pain Questionnaire scores (range: 25% to 57%). Five subjects were measured using the Schober technique, and all showed improvement in thoracolumbar flexion mobility. Combined use of translatoric manipulation and spinal flexion exercises may have resulted in improved spinal flexibility, ambulatory abilities, and pain and functional status in six subjects with lumbar spinal stenosis.

Key Words: Lumbar Spinal Stenosis, Translatoric Manipulation, Flexion Exercises, Case Series

Approximately 90% of the people in the US will experience significant low back pain (LBP) at some point in their life[1]. Lumbar spinal stenosis (LSS) is identified as one of the primary causes of back pain in the elderly[1] and is the most common diagnosis requiring lumbar spine surgery for patients over 65[2]. It is estimated that 3-4% of patients who seek assistance from general practitioners for LBP are eventually diagnosed with LSS. Data from the national hospital discharge survey showed an eightfold increase in the number of surgeries for LSS between 1979 and 1992. Annually, over 30,000 surgical procedures are performed in the US for this condition with an associated inpatient expense of approximately one billion dollars[3-5].

LSS is defined as a narrowing of the central spinal canal and/or the intervertebral foramen most often caused by degenerative changes[6,7]. Anatomically, LSS is classified as central or lateral stenosis. Central stenosis can be caused by posterior disc bulging, thickening of the ligaments inside the central spinal canal, osteophytic outgrowth of the facet joints, and/or degenerative spondylolisthesis and retrolisthesis[6]. Lateral stenosis occurs where the nerve exits the spinal canal and can be caused by (postero)lateral disc bulging, asymmetrical loss of disc height, and osteophytic overgrowth of the pedicles and superior lumbar facets[6].

As a result of LSS, the space available for the neural structures within the central spinal canal or interver-

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tebral foramen becomes compromised, and neurogenic claudication may result. Neurogenic claudication and, in the case of lateral stenosis, radicular irritation are the result of compressive forces caused by both the above-mentioned morphological changes and physiological posture-related increases in epidural pressure. Compressive ischaemia results in disruption of the normal neural function and can present as leg weakness and fatigue, sensory disturbances, or a feeling of heaviness in the legs[8]. Some studies[1,7] have reported that the symptoms associated with LSS correlate with the extent and progression of structural abnormalities. In contrast, many researchers have found significant radiographic changes in asymptomatic individuals[8,9]. In addition, no consistent relationship has been identified between imaging findings and treatment outcomes for patients with LSS[1,3,10-15].

Clinical studies have suggested that symptoms associated with LSS are posture-dependent[1,16]. Lumbar extension narrows the central spinal canal and intervertebral foramen, and it results in venous congestion in the cauda equina and nerve roots, respectively. This may lead to inadequate arterial blood supply to these neural structures in response to exercise such as walking[10]. Lumbar flexion may reduce anatomical encroachment by changing the bony positional relationships and by opening up the central canal and intervertebral foramen, thus relieving circulatory congestion[10,16]. Takahashi et al[16] studied the effects of posture on epidural pressure in 10 patients aged 51-72 with leg pain, weakness, sensory changes indicative of L4-L5 involvement, and symptoms of neurogenic claudication. Lumbar extension led to significantly higher ($P<0.001$) epidural pressures (116.5±38.4 mmHg) than did standing (66.9±27.5 mmHg) and 30º of lumbar flexion (27.3±19.7 mmHg). Clinically significant was the pain relief reported with flexed postures.

The purpose of this case series was to report on a conservative intervention consisting of high- and low-velocity translatoric manipulation to the thoracic and upper lumbar spine and two lumbar lordosis-flattening (flexion) exercises, and its outcome with regard to spinal flexibility, ambulatory abilities, and overall spinal pain and functional status in 6 patients with a clinical diagnosis of lumbar spinal stenosis.

Current Treatment Options

Current treatment options for LSS include both surgical and conservative management. We searched the *Medline* and *CINAHL* electronic databases for articles published from 1995 to 2005 and did a hand search of the bibliographies of the articles collected. Key words used were *lumbar spinal stenosis*, *lumbar stenosis*, *physical therapy*, *manual therapy*, *therapeutic exercise*, and *lumbar spine surgery*. The search was limited to articles written in English.

Zdeblick[17] concluded in a literature review that surgery was the only definitive treatment for LSS because no other treatment could reverse or change the anatomic condition. Yone et al[18] reported that the gold standard for surgical therapy was laminectomy to relieve pressure and symptoms of neurogenic claudication. The goals of surgery for LSS include removal of pressure on neural elements, maintenance of stability of the spinal segment, avoidance of iatrogenic surgical injury, and the prevention of future problems[19]. While some physicians believe LSS to be a progressive disorder with surgery as the only treatment option, the literature does not necessarily support this view because not all patients deteriorate over time[6,11,15,20].

Several authors[3,6,13,17,20] have reported on the initial conservative management options when a patient first presents with signs and symptoms consistent with LSS. Fritz et al[6] noted that LSS may not be a progressive disorder and that conservative management may be a valid alternative. In a study with a 4-year follow-up of 32 patients with LSS, Johnsson et al[20] found that 90% of conservatively managed patients either noted improved or unchanging pain levels and that 42% improved in walking capacity. In a cohort study of 100 patients with symptomatic LSS, Amundsen et al[13] compared surgical with conservative care management and found that almost half of the patients randomly assigned to conservative care maintained improvement at the 10-year follow-up.

Conservative treatment options have historically included a combination of non-steroidal anti-inflammatory medication, general conditioning exercises emphasizing lumbar flexion, abdominal strengthening exercises, pelvic traction, lumbar corsetry, aquatic exercises, and partially unloaded treadmill ambulation[3,6]. The success rate for similar forms of conservative care was demonstrated in a cohort study of non-operatively treated patients with LSS by Simotas et al[21], where after three years one-quarter of the subjects followed were significantly better and half noted overall improvement in pain level. This study[21] supports the premise that patients undergoing non-surgical management of LSS do not face inevitable neurological and functional decline. However, the number of interventions applied to the subjects made it difficult to establish the efficacy of any one treatment or even a select combination of treatments. In an 11-subject case series, Kirkaldy-Willis[22] reported on the use of spinal manipulation as a stand-alone intervention for patients with LSS. These patients had been unresponsive to other forms of conservative care. Six subjects reported improvement in symptoms with spinal manipulation.

Methods

Subject Selection

The subjects for this case series were a sample of convenience recruited from patients visiting an outpa-

tient orthopedic physical therapy clinic in southeastern Michigan for various orthopedic diagnoses. Subjects with decreased cardiovascular capacity, lumbar spinal fusion surgery, vascular claudication, vestibular problems, or other disorders that would prevent them from safely ambulating on a treadmill were excluded. Inclusion criteria consisted of 1) a positive history for LSS, 2) positive radiographic findings indicating LSS, 3) a bicycle test negative for vascular claudication, 4) the absence of vascular changes affecting the legs, 5) a decreased lumbar lordosis, 6) decreased lumbar flexion ROM, 7) provocation of symptoms upon lumbar backward bending, 8) L5 motor weakness, and 9) a treadmill test indicative of LSS.

Clinical Tests and Measures

In a study of 66 patients with LSS diagnosed with myelography and confirmed during surgery, Hall et al[23] reported that 94% of these patients described a classic history of LBP with an insidious onset, followed by a gradual onset of radiating pain into the buttock and legs. These symptoms were initiated by walking and relieved by sitting or bending forward. All subjects in this case series presented with histories of insidious development of LBP and eventual leg pain aggravated by walking and relieved by bending forward.

Diagnostic imaging tests have been reported as useful in the clinical diagnosis of LSS. Katz et al[24] found positive CT and MRI findings for LSS in 88% of 43 patients where an expert clinician believed at a ≥80% confidence level that the symptoms these patients were experiencing were the result of LSS. All 6 patients in this case series presented with MRI findings indicative of degenerative LSS at the L4-L5 and L5-S1 spinal levels.

In a single subject case report on a 68-year old patient suspected of having LSS, Dyck and Doyle[25] used lateral myelography views to confirm blockage of cerebral spinal fluid (CSF). While the subject was pedaling with the lumbar spine in an extended position, a partial high-grade blockage was seen; pedaling with the lumbar spine in flexion restored free flow of the CSF. Tenhula et al[26] also noted that this bicycle test may have value in the differential diagnosis of neurogenic claudication and that it may be used as an objective test of postoperative outcome. In the patients described in this case series, the bicycle test was used to exclude vascular claudication: Each patient cycled for 10 minutes with the lumbar spine flexed without provocation of leg pain.

Without providing data on reliability and validity of these observational tests, Thomas[27] noted that visual inspection for alopecia affecting the legs, nail dystrophy, and changes in foot color might assist in differentiating vascular from neurogenic leg complaints. No subject in the current case series presented with these findings.

Again without data on reliability and validity, Thomas[27] also commented on the tendency to find flattening of the lumbar lordosis in patients with LSS. Fedorak et al[28] demonstrated that the intrarater reliability for visual assessment of lumbar lordosis using a 3-point scale (decreased, normal, increased) was only fair (κ= 0.50; 95% confidence interval, 0.02-0.98). Four of six subjects in this case series presented with a kyphotic lumbar region.

Schober[29] initially described a technique to assess thoracolumbar flexion by measuring with a tape measure skin distraction (in cm) between T1 and S1 from a neutral to a flexed position. The modified Schober described by Macrae and Wright[30] has achieved more common clinical usage. This version of the test quantifies the change in position between two marks, one at the S1 level and the other at a point 15 cm above S1. Williams et al[31] reported on the interrater reliability of the modified Schober test: Pearson product-moment correlation coefficients for test-retest reliability varied from 0.78-0.89 for lumbar flexion and 0.69-0.91 for lumbar extension. Without reference to a specific study, patient population, or age range, Greene and Heckman[32] reported 10 (cm) of thoracolumbar (T1-S1) flexion as a normative value. Five of the six patients in this case series were measured using the Schober test and all five had <10 (cm) of thoracolumbar (T1-S1) flexion.

In a study of 43 patients with suspected degenerative LSS, Katz et al[24] noted a correlation between the reproduction of leg symptoms experienced during ambulation and the development of similar symptoms in the legs following 30 seconds of sustained lumbar extension performed in standing. Of these 43 patients, 38 had imaging reports confirming either central or lateral stenosis. Without indicating the duration of lumbar extension or the position in which it was performed, Lyle et al[33] found back, buttock, and leg pain to be moderately correlated with lumbar extension in patients with LSS (Spearman's ρ=0.31, P=0.007). All six patients in this case series experienced increased low back, buttock, and leg pain when their maximum available range of active lumbar extension was held for 30 seconds while standing.

Hall et al[23] noted that 43% of 68 patients with LSS that had been confirmed on myelography presented with L5 myotomal weakness. All six subjects in this series demonstrated positive findings for mild weakness in the L5 myotome.

Without commenting on how a diagnosis of LSS was confirmed, Fritz et al[34] noted in 45 patients that an earlier onset of pain with level walking, increased total walking time on an inclined treadmill causing a more flexed posture, and a prolonged recovery period following the cessation of walking were significantly associated with LSS. Yukawa et al[35] had 62 patients undergoing surgery for symptoms related to LSS perform a pre- and post-surgery treadmill test: 58 (94%) had a positive pre-surgery treadmill test and only six patients had a positive post-surgery test lending validity

to this test for the diagnosis of symptomatic LSS. Each patient in the present series was asked to walk without support and in a fully upright position on a treadmill (Cybex Trotter 710t) at his or her preferred walking pace. Prompting and physical cueing was used to have the patient maintain their maximum lumbar lordosis during ambulation. Patients were instructed to report the time to first symptoms in the legs and when the pain had reached a maximum tolerable point. At this point, each patient was instructed to grasp the front handrail, fully flex the spine, and continue to walk at the same pace. Each patient was then asked whether leg pain had increased, decreased, or stayed the same with all six patients reporting complete resolution of leg pain.

In addition to the tests and measures used to determine eligibility for inclusion in this case series, passive thoracic and lumbar segmental flexion testing and passive translatoric (A-P) joint play testing were used to assess passive segmental thoracolumbar (T1-S1) motion. These two passive segmental movement tests were performed with the patients in side lying and their hips positioned at approximately 60 degrees of flexion. Each segment from T1 through S1 was palpated at the interspinous space while the examiner produced either passive flexion or anterior-to-posterior (AP) translation. Movement occurring between the spinous processes was palpated and was used as a gauge of the underlying segmental motion. Lumbar joint play or AP translation was performed with the patient positioned as described above. A "push-pull" movement parallel to the long axis of the patient's femurs was generated by the examiner's hand and pelvis while the amount of segmental translation, including end feel, was graded[36]. Segmental motion was graded on a 3-point scale as hypomobile, hypermobile, or normal. Inscoe et al[37] reported on a similar passive lumbar segmental flexion examination technique performed in sidelying also using a 3-point mobility scale. Mean intrarater agreement was 66.67% and 75%; mean interrater agreement was 48.61%. A review of reliability of spinal segmental motion tests[38] reported no research on techniques similar to the translatoric joint play testing used in the examination of these patients. Segmental motion was graded as hypomobile at T1-T9 and L1-L3, and hypermobile at L4- L5 and L5-S1 in all six patients included in this case series[36].

Outcome Measures

The outcome measures used in this case series were the McGill Pain Questionnaire (MPQ), the original version of the Oswestry Disability Index (ODI), the Schober technique for measuring thoracolumbar flexion, and a treadmill test.

In a prospective observational cohort study of 57 patients with osteoarthritis, the MPQ was administered two times, five days apart, to evaluate test-retest reliability[39]. For the MPQ total, sensory, affective, and average pain score ICC-values of 0.96, 0.95, 0.88, 0.89, respectively, were reported.

In a study of 32 patients with LSS, Pratt, Fairbank, and Virr[40] reported an ICC-value of 0.89 for the test-retest reliability of the ODI. Several studies have reported on the minimal detectable change (MDC) for this measure. Fairbank et al[41] reported an improvement of 16 points as the MDC in the original version of the ODI. In a prospective multi-site study of 106 patients with LBP, Davidson and Keating[42] administered five different disability questionnaires and evaluated them for both reliability and responsiveness. Repeated measures at a six-week interval were taken for the modified version of the ODI. The MDC was determined in two subgroups of patients. The MDC value was 15 points in a group of 47 patients with LBP, who reported that their overall disability status was unchanged. The MDC value was 10.5 for patients with LBP, who self-rated as "about the same." The minimally clinically important difference (MCID) can be defined as the amount of change that best distinguishes between patients who have improved and those who remain stable. Fritz and Irrgang[43] reported that the MCID value was approximately 6 points for the modified ODI in a group of 67 patients with acute, work-related LBP. For this case series, we used the original version of the ODI.

Reliability of the modified Schober test is discussed above. In a study of 31 patients comparing the Schober test to plain film radiography, Tousignant et al[44] reported that 1 (cm) improvement in spinal motion on the Schober test should be considered the MDC.

The use of a treadmill to measure baseline functional status in terms of ambulatory abilities in patients with LSS is a functionally relevant outcome measurement for patients with this condition[45,46]. In a study of patients with severe LSS, Deen et al[47] measured time to first symptoms and total ambulation time. No intervention was applied between tests. The concordance correlation coefficients, a method of measuring correlation when data are measured on a continuous scale, were 0.90 and 0.89 for time to first symptoms, and total ambulation time at 1.2 mph saw 0.98 and 0.96 for these same parameters, respectively, at the patient's preferred walking speed, both indicating excellent test-retest reliability.

Interventions

Four distinct interventions were used in the therapeutic management of all six subjects in this case series:

- T1-T9 low- and high-velocity bilateral thoracic facet joint traction manipulation (Figure 1)[48,49]
- L1-L3 low- and high-velocity translatoric lumbar side-bending manipulation (Figure 2)[48,49]
- A posterior lumbopelvic tilting performed in a side lying position
- A seated resisted lumbar flexion home exercise (Figures 3 and 4)

Each low-velocity translatoric manipulation was performed at a grade III level and held for 7-10 seconds at each of the segments listed. A low-velocity grade III movement is applied after the slack has been taken up and tissues crossing a joint or segment become taut. A grade III stretching force must be applied for a sufficient period of time in order to stretch the tissues[36]. Low-velocity techniques were repeated up to 3 times at a segment. High-velocity translatoric manipulation was performed by fully taking up soft tissue slack in the desired direction of translation (grade III movement) followed by a short quick straight-line or translational motion (impulse) in the same direction and just through the tissue slack at the segment in question[48,49]. When high-velocity translatoric techniques were performed, only one repetition was applied to the segment.

Figure 1 shows a bilateral facet traction manipulation. The therapist stands facing the patient's head and places the base of a firm wedge on the transverse processes of the caudal vertebra in the treatment segment. The wedge is pressed in the direction of the transverse processes of the caudal vertebra. The slack and translatoric impulses are directed ventrally, ventrocaudally, or ventrocranially, depending on where in the thoracic spine the facet distraction is performed[49].

Figure 2 shows a translatoric side-bending manipulation technique that can be performed from L1 through L4. The patient is positioned in left side lying with the lumbar and thoracic spine in flexion, right side-bending, and left rotation. The therapist stands facing the patient and presses in the direction of the right articular surfaces of the cranial and caudal vertebra and the spinous process of the cranial vertebra in the treatment segment with the ulnar border of the right hand. The therapist's right thigh is placed anteriorly against the abdomen to support the lumbar flexion. The therapist's right wrist is slightly extended and radially deviated. The left hand is used to reinforce the position of the wrist. Slack in the lumbar spine is taken up medially by leaning the body weight onto the right hand. To provide a translatoric impulse, the therapist lifts his head, neck, and upper thorax slightly while maintaining the same end slack tension within the segment. The impulse is directed medially through the dropping of the chest and head toward the patient[49].

The posterior lumbopelvic tilting exercise was performed in a side lying position during each clinical visit. The patients were instructed to hold the posterior pelvic tilt for 6 seconds and to repeat the movement 10 times. This exercise was not prescribed as part of the subject's long-term home exercise program. The second therapeutic exercise was a seated resisted lumbar flexion procedure. An 8-foot piece of blue Theraband™ was knotted in the middle and secured by closing a door on it. This left two strands of Theraband™ hanging from the top of the door to the top of the subject's shoulders. The subject straddled a chair of standard size and shape (42cm high and 38cm deep), pulled the Theraband™ down to the pectoral line, and then sat down in the chair thus creating tension in the band. The patients were then instructed to gently drop the head and chest, and to round (flex) the lumbar region. This movement was performed against the resistance provided by the band (Figure 3). The therapist visually confirmed that the subject flexed the entire lumbar spine. The tension on the Theraband assisted the subjects in returning to an upright but not fully extended position (Figure 4). This portion of the exercise movement provided active

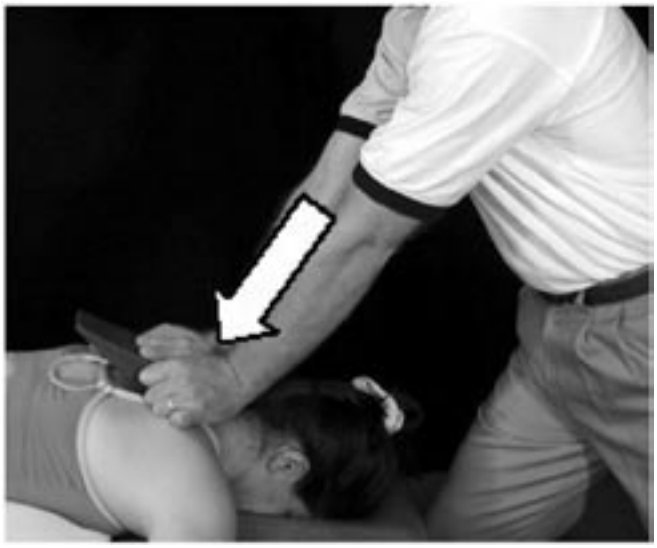

Fig. 1: Bilateral facet joint traction manipulation.

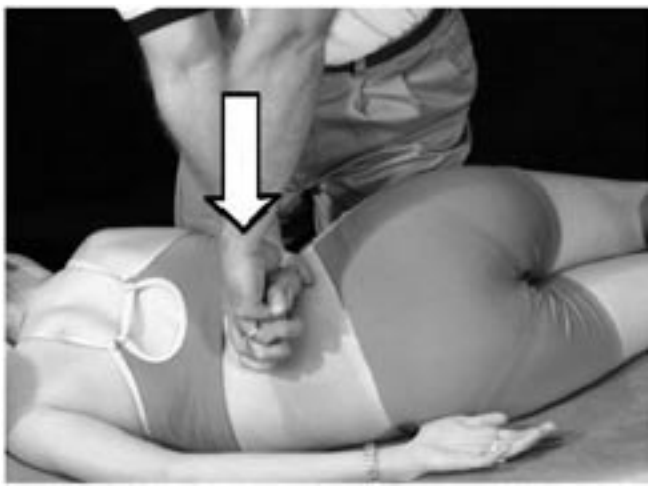

Fig. 2: Translatoric lumbar side-bending manipulation with the entire spine locked in flexion, side-bending, and rotation to the opposite side.

Fig. 3: End position for resisted lumbar flexion home exercise.

assisted training to the lumbar extensor muscles. The patients were instructed to perform 30 repetitions of this exercise 3 times per day. Patient compliance was verbally monitored during each clinical visit and throughout the entire course of intervention with a patient report of 100% compliance.

Results

Table 1 contains individual subject age, diagnosis, functional deficits, and pain distribution. Results of the various outcome measures can be seen in Table 2. Limited long-term outcome data were available for two subjects: Subjects 3 and 4 performed treadmill tests at 4 and 5 months after discontinuing all forms of intervention except 90 daily repetitions of the resisted lumbar flexion home exercise. Subject 3 ambulated 12 minutes at discharge and after four months was able to ambulate for 14 minutes. At discharge subject 4 ambulated 30 minutes and after five months was able to ambulate 32 minutes before reaching the maximum tolerable point related to leg pain.

Discussion

The interventions we chose for the management of

Fig. 4: The Theraband™ assists the patient back to the upright position.

these patients with a clinical diagnosis of LSS was based mainly on basic science research. Studies have shown that the dimensions of both the central spinal canal and the intervertebral foramen are determined by both the amount of axial loading and by positioning. Lumbar flexion increases the sagittal plane dimensions of the central spinal canal; contralateral side-bending and flexion increase the cross-sectional area of the lumbar intervertebral foramina[50-53]. As discussed above, lumbar flexion reduces epidural pressure values when compared to values in standing and extension[16]. The patients in this case series appeared to benefit from the performance of repetitive lumbar flexion exercises. However, one must question whether the dimensional and pressure changes provided by the three times daily performance of flexion exercises translates into prolonged physiological changes with resultant improvements in ambulatory abilities, pain, and functional status in patients with a clinical diagnosis of LSS.

The manipulation techniques shown in Figures 1

Table 1: Subject Characteristics

Subject	1	2	3	4	5	6
Age	82	71	52	64	72	81
Medical diagnosis	Severe lumbar arthritis	Hamstring pain	LSS	LSS	Lumbar degenerative disc disease	LSS
Pain distribution	Central LBP Bilateral buttock pain	Central LBP Right buttock, posterior thigh pain	Central LBP Left buttock, posterior thigh/ calf pain	Central LBP Right buttock, posterior thigh pain	Central LBP Right buttock, posterior thigh/calf pain	Central LBP Bilateral buttock pain and leg pain
Functional deficits	Standing & ambulatory difficulties secondary to leg pain	Ambulatory difficulties secondary to leg pain	Standing & ambulatory difficulties secondary to leg pain	Standing & ambulatory difficulties secondary to leg pain	Ambulatory difficulties secondary to leg pain	Standing & Ambulatory difficulties secondary to leg pain

LSS-lumbar spinal stenosis; LBP-low back pain

Table 2: Outcome Measures

Subject	1	2	3	4	5	6
Schober test initial (cm)	1.0	1.5	3.0	Not measured	1.5	3.0
Discharge (cm)	3.0	5.0	5.5		6.0	6.0
MPQ initial	10	44	6	35	36	40
Discharge	6	29	4	15	27	12
ODI initial (%)	53	18	24	31	51	58
Discharge (%)	49	12	12	21	18	36
Treadmill time* initial (min/sec)	0'30	0'40	3'45	4'00	1'21	3'00
Discharge (min/sec)	2'40	15'00	12'00	30'00	6'30	5'30
Number of clinic visits	9	9	8	13	17	9

*Maximum tolerable point MPQ-McGill Pain Questionnaire; ODI-Oswestry Disability Index.

and 2 were applied to improve thoracolumbar flexibility and, theoretically, to reduce movement stress on the L4-L5 and L5-S1 segments during the performance of the resisted lumbar flexion exercise and during daily activities requiring these movements. The L4-L5 and L5-S1 segments were evaluated as hypermobile with excessive AP gliding, as per the side lying lumbar joint play test, and therefore as not requiring translatoric manipulation.

One of the limitations of a case series or case report is that this format does not allow a cause-effect relationship to be established, so it is not clear whether the interventions described here truly caused the observed positive effects[52]. However, all 5 patients measured at the initial evaluation and at discharge with the Schober technique exceeded the MDC for this measure, indicating that a true increase in thoracolumbar flexion mobility had in fact occurred. Similarly, all subjects showed improvement in pain levels as measured by the MPQ and in perceived disability as measured by the ODI. Three subjects exceeded what might be considered a relevant MCID on the ODI-measure. We were unable to locate specific data on responsiveness (i.e., MDC, MCID) for the treadmill ambulation test in subjects with LSS. However, after the treatment sessions, all subjects in this series reported improvements in their ability to ambulate with longer periods of time prior to the onset of leg pain (Table 2). We believe that for the subjects

in this series, this change in ambulatory abilities was clinically relevant. Furthermore, all subjects reported an improved ability to perform ADL that required standing and ambulation. Subjects 2, 3, and 4 reported resuming their fitness walking programs and subject 4 was able to resume "walking" nine holes of golf. The limited long-term outcomes on two of the subjects reported here would seem to indicate possible long-term benefit from the conservative intervention described in this article, if not by further improvement, then at least by maintenance of the post-intervention improved functional status.

This case series has several other limitations. Although we feel that the patient history discussed and the physical examination procedures applied are useful in making a clinical diagnosis of LSS in older patients with chronic lumbar and leg pain, we realize that data on reliability and validity of the isolated tests and measures used to establish this clinical diagnosis and to determine the interventions chosen are very limited. And when these values are available they may not always be supportive of the diagnostic utility of the tests. Similarly, reliability and validity of the multi-test regimen we used to establish this clinical diagnosis has not been determined. Further, while the clinician who applied the various tests, measures, and interventions had over 20 years of clinical experience, intrarater reliability of the tests performed by this rater was not assessed. Therefore, it is not clear whether other subjects matching these diagnostic criteria would also respond in a similarly positive manner to the management program described here. Despite these limitations, we believe that the results of this case series lend support to the work of other authors who have suggested that conservative intervention may provide some patients with an alternative to surgical interventions in relieving the pain and functional loss associated with LSS[3,6,11,19-21].

Conclusion

We applied a multi-test regimen to establish a clinical diagnosis of LSS, to rule out other potential causes of leg pain, and to determine the segmental level at which to apply specific manipulative interventions. Six subjects with a clinical diagnosis of LSS received translatoric manipulation to the thoracic and upper lumbar segments and performed two lumbar spinal flexion exercises. All six subjects showed improvement in thoracolumbar flexion mobility, ambulatory abilities, pain status, and perceived disability. This case series adds to a growing body of evidence supporting the potential value of conservative management of LSS in the form of exercise and manual therapy. In addition, it would seem to indicate the need for future research into the diagnostic value of the multi-test regimen used here for clinical diagnosis and conservative interventions consisting of orthopaedic manual physical therapy and exercise in the management of patients with complaints resulting from LSS.

Acknowledgements

The authors would like to thank Jeffrey Dehn, MPT; Bryan Kuhlman, MPT; Susan Phillips, MPT; and Ross Walker, MPT, for their contributions to this paper. ■

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