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Gait kinematics and spinal loading in patients with lumbar spinal stenosis and healthy older adults

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Thesis

GAIT KINEMATICS AND SPINAL LOADING IN PATIENTS WITH LUMBAR SPINAL STENOSIS AND HEALTHY OLDER ADULTS

by

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GAIT KINEMATICS AND SPINAL LOADING

IN PATIENTS WITH LUMBAR SPINAL STENOSIS

AND HEALTHY OLDER ADULTS

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ABSTRACT

Background: Lumbar spinal stenosis (LSS) in older adults is a leading cause of pain and limitations to mobility. Compression of the spinal nerves can result in neurological symptoms that can decrease walking capacity and overall quality of life. It is clinically believed that patients with LSS alter their gait pattern to be able to increase their walking capacity but biomechanical assessment of spinal and pelvic motion during walking compared to healthy older adults is lacking. The purpose of this study was to gain further insight into how patients with LSS move and how their posture affects spinal loading compared to healthy older adults.

Methods: Whole body motion data was collected on 9 patients with LSS and 10 healthy older adults. Both cohorts completed a 3D opto-electronic motion analysis during standing and walking trials and patients with LSS were measured during both asymptomatic and symptomatic states. Pelvis, knee, and spine kinematics and spinal loading were obtained via subject-specific musculoskeletal models.

Results: In the LSS group, both asymptomatic and symptomatic trials, the average pelvic tilt was more posteriorly rotated than the healthy adults during standing and walking. Lumbar spine angles in the LSS group adopted a more flexed posture compared to the healthy group's normal lordotic angle. This coincided with higher C7/S2 angles and

distances compared to the healthy group. Lumbar spine loading doubled in both LSS groups compared to the healthy group's standing trials, though little difference was seen during walking. Knee flexion angle increased greatly during both standing and walking.
Conclusions: My results indicate that LSS patients both stand and walk with greater posterior pelvic tilt and lumbar flexion which greater knee flexion to counterbalance compared to healthy counterparts. While the provocation of symptoms did not affect their kinematics, both asymptomatic and symptomatic states showed significant modification from older healthy adults. The clear differences in gait and posture can aid in therapeutic interventions but additional work is needed to better understand the biomechanical differences between these two groups.

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1. INTRODUCTION

Lumbar spinal stenosis (LSS) is one of the more common degenerative spinal conditions that affects 10%–14% of adults over age 50. As the U.S. population's median age grows, LSS prevalence will only increase (Katz and Harris, 2008). This can lead to a rise in health and economic concerns which makes fully understanding the condition paramount. A "cost-of-illness" study run in the UK in 1998 placed economic effects of low back pain (due to LSS and other factors) at £1.63 billion with approximately 35% being paid to the private sector by patients and their families (Boos, 2009). With inflation and a steady rise in the number of patients, that cost has only increased. The need for more effective therapeutic intervention based on *in vivo* measurements is paramount.

LSS is caused by compression of the nerve roots coinciding with a narrowing of the spinal canal and the intervertebral foramen. The main symptom attributed to LSS is neurogenic claudication. This presents as pain and discomfort starting at the lumbar spine and travels down one or both legs. Often the pain correlates with a loss of sensation in the limb, fatigue, weakness, and difficulty maintaining a balanced posture (Suri et al., 2010; Katz and Harris, 2008). Symptoms often begin from walking or prolonged standing, particularly when the lumbar spine is in extended (lordotic or upright) postures. It has been suggested that patients may adopt a more flexed or hunched over posture to increase the space in the spinal canal and foramina and relieve pressure on spinal nerves (Suri et al., 2010, Conrad et al., 2013). Surgical intervention is available for patients with severe pain or functional limitations. A lumbar laminectomy, the removal of the lamina and insertion of a spacer, often paired with spinal fusion across surrounding vertebrae, is the most common surgery performed. Physical therapy is also very common as a non-invasive treatment pre-operatively, and post-operatively after the decompression surgery. However, prescribing therapeutic exercises based solely on clinical observations could be less effective than utilizing quantitative assessments of the LSS patient's movements to increase walking capacity and reduce pain.

Spine loading, or the force placed on vertebrae by body mass, physical activity, or muscular tension, may aggravate claudication and decrease walking capacity by reducing the size of the spinal canal and increasing epidural pressure. A 2007 study showed that loading and unloading the spine through a weight vest or vertical traction harness in LSS patients while walking on a treadmill resulted in shorter and longer time for appearance of symptoms and total walking time, respectively (Oğuz et al, 2007). This shows the compressive loads factoring into the onset of pain. Mousavi et al., 2021 showed no increase in spinal flexion when claudication symptoms were provoked, though there was an increase in spinal loading. Only four studies investigated spine kinematics (postural angles) in patients with LSS during walking (Wang et al., 2021). Three of those studies reported hip and knee flexion moments and paravertebral muscle activities (Kuwahara et al., 2016; Goto et al., 2017; Igawa et al., 2018). However, none of these published studies have directly compared the gait measurements of LSS patients to healthy controls. Addressing this clear lack of information regarding how spine biomechanics during walking in LSS patients differ from normal could clarify targets for therapeutic exercises. Thus, this study aims to provide insights into how patients with LSS move and how their posture affects their spinal loading compared to healthy older adults.

2. METHODS

2.1 Subjects:

This study compared a healthy older adult cohort (Banks et al., 2023) to a cohort of older adults with LSS who were scheduled for spine decompression surgery. Nine participants (aged 44–82 years) with symptomatic LSS confirmed by imaging and clinical examination at Beth Israel Deaconess Medical Center, who were scheduled for surgery (laminectomy with or without fusion) for lumbar spinal stenosis were recruited. The LSS cohort consisted of 5 women and 4 men (aged 44-82 years) and were the mean±SD of age: 65±11.3 years, height: 165±10.1 cm, body mass: 79±26.7 kg, and BMI: 29.1±3.3 kg/m2. The healthy adult participants consisted of 6 women and 4 men (aged 53-78 years) and were the mean±SD of age: 66±9.2 years, height: 167±10.7 cm, body mass: 71±20.0 kg, and BMI: 27±4.2 kg/m^2. The studies were approved by the Institutional Review Board of Beth Israel Deaconess Medical Center, and all participants provided written informed consent prior to participation. Participants were excluded if they had pre-existing conditions (unrelated to LSS) that altered walking or spine function, such as history of traumatic spinal injury or surgery, vascular insufficiency, Parkinson’s disease, stroke, or cognitive impairment.

2.2 Experimental Procedure:

Whole body 3D motion capture data was collected using passive reflective markers and 10 optical-active infrared cameras. Clusters (four markers each) were attached to participants at T1, L1, and S2 levels of the spine, with additional single markers at every thoracic and lumbar spinal levels from C7 to L5, as well as the head, sternum and left/right clavicle, anterior/posterior superior iliac spines, shoulders, upper and lower arms and legs, and feet. Four of the nine LSS patients were tested with a different spinal marker set using only clusters placed at T1, T5, T9, L1, L3, L5, and S2. The marker position was recorded by a motion analysis system (Vicon Motion Systems, Centennial, CO) which operates at sub-millimeter precision (Merrieux et al., 2017). Force plates embedded in the floor collected ground reaction forces (AMTI, Watertown, MA).

2.3 Walking Protocol:

Both cohorts underwent a series of neutral standing and walking trials. LSS patients were tested in the lab between 2–10 days before their surgery. The healthy subjects began with a neutral standing pose and then walked a straight 5m path over the embedded force plates 3-4 times at a self-selected pace. Subjects were instructed to land with one foot completely on the force plate without making conscious adjustments to their gait.

After completing neutral standing poses, LSS participants completed the same 5m path walking task in both asymptomatic and symptomatic conditions. The following tasks were conducted in a consecutive order: 1.) static upright standing posture (asymptomatic), 2.) walking 5m at a self-selected pace without neurogenic claudication symptoms present (asymptomatic) (three trials), 3.) walking after onset of neurogenic claudication symptoms (symptomatic) (three trials), and 4.) static upright standing posture (symptomatic). Almost all LSS patients experienced low back and/or leg pain during the neutral standing position, but they were able to differentiate this pain as not neurogenic claudication symptoms. To produce the symptomatic state, patients performed a standard walking capacity test, walking over ground or on a motorized treadmill at a self-selected pace until reporting the onset of neurogenic claudication symptoms, up to a maximum of 30 min (Mousavi et al., 2021). Pain severity was collected by using the Brief Pain Inventory. Self-reported pain values for LSS patients were collected before and after asymptomatic walking trials, after inducing claudication symptoms, and after symptomatic walking trials (Mousavi et al., 2021).

Healthy subjects performed the standing neutral posture followed by 3–4 walking trials (5m) at a self-selected pace. The healthy group did complete several lifting and simulated activities of daily living trials prior to the walking trials however no great fatigue was documented prior to the walking trials.

2.4 Musculoskeletal Modeling:

Full-body subject-specific musculoskeletal models were made for both cohorts including a fully-articulated thoracolumbar spine model (Bruno et al., 2015, Burkhart et al., 2020). Incorporated into model creation is the subject’s age, sex, height, weight, motion capture data measurements, and standing spine radiographs (LSS patients only). Base models were initially adjusted according to anthropometric data and marker data in a standing neutral posture using the OpenSim scale tool (Delp et al., 2007). For LSS patients, the base model lumbar spine curvature was adjusted from their pretreatment standing radiograph (available from online medical records). Thoracic curvature was estimated based on regression equations using the patient's thoracic angle calculated from spine markers, age, and BMI (Grindle et al., 2020). Intervertebral angles in each model were adjusted accordingly to produce a subject-specific model. Each healthy subject's spine thoracolumbar curvature was calculated from trunk CT data taken during their study visit and 3D motion capture data.

Kinematic constraints were used to limit the spinal degrees of freedom when evaluating underlying spinal motion from motion capture data (Actis et al., 2018; Ignasiak et al., 2018; Beaucage-Gauvreau et al., 2019; Alemi et al., 2021). Constraints were selected allowing six independent spinal degrees of freedom (two each in flexion-extension, lateral bending, and axial rotation), which produce realistic, repeatable spine motions from motion analysis data with relatively low marker position error (Alemi et al., 2021).

2.5 Data Processing & Analysis:

The healthy and LSS groups both performed a 5 second standing trial which was used to compare the walking trials to a neutral standing position. All walking analysis was performed using the same methods as the standing analysis. Walking trials were trimmed to one stride beginning with heel strike of the right foot in stance phase, moving through the swing phase and ending at the initial impact of the second right heel strike. For most healthy subjects, the trial analyzed was their third gait cycle while for LSS patients, it tended to be their fourth or fifth due to smaller stride length and/or taking more strides to reach the center of the motion capture volume.

Analysis was performed on aspects of the pelvis, hip, spine, and right knee. Pelvic tilt, both peak and average, were measured during walking and the standing neutral pose. Pelvic tilt during stride relative to the neutral standing pose was also calculated. Spine angle analysis was composed of lumbar and thoracic average angles as well as individual level comparisons. Spinal compression was computed via the static optimization analysis to evaluate muscle forces, following by joint reaction analysis to determine vertebral compressive loads. C7/S2 distance and angle was measured from the single C7 marker to the vertical line of the S2 cluster centroid. Pelvic tilt angle was measured from the OpenSim output of the 6 pelvis markers (L/R ASIS, L/R PSIS, L/R Iliac Crest) and is expressed as positive being posterior rotation and negative being anterior rotation in degrees. Right knee angle was measured from the greater trochanter marker to the lateral condyle of the femur to the lateral malleolus of the fibula. Knee angle from the standing trial was averaged across the whole trial, while walking trials were trimmed to a single stance phase and averaged. Spatiotemporal metrics such as stride length (m), velocity (m/s), and cadence (steps/min) were measured over 2-3 strides for both groups. Averages were taken of the 3 pre-claudication and 3 post-claudication trials as well as an average of the 3 healthy trials.

Statistical analysis was conducted in JMP Pro 16 (SAS Institute Inc, Cary, NC). Independent variables are group and subject as a random effect. Within group, there are 3 types: Healthy Control, patients without claudication (LSS-NP), and patients with claudication (LSS-P).

Dependent variables are the following outcomes from standing and walking analysis: pelvic tilt (average, peak, range), lumbar and thoracic spinal flexion angles, average right knee flexion angle, spinal compression force both averaged across the lumbar and thoracic spine and at each individual level. Additionally, average C7/S2 distances and angles were examined as postural measurements that might be more easily utilized clinically.

Due to small sample size, non-parametric tests were used. Kruskal-Wallis H Test was used to compare the 3 groups. The subject variable was used as a random effect for both standing and walking. Post-hoc Tukey HSD Tests were run to understand differences between groups. Statistical significance was set at a *P*-value of less than 0.05.

3. Results:

3.1 Participants Characteristics

A comparison of age, body mass, height, and BMI showed no differences between the groups. The healthy group contained one more female than the LSS group.

3.2 Spatiotemporal Metrics

A 0.3m decrease in stride length was measured in both LSS groups compared to the healthy subjects (**Table 1**) ($p<0.05$). Velocity also decreased by approximately 0.25 m/s in both LSS groups compared to the healthy subjects ($p<0.05$). Cadence showed very little change between groups, however the healthy group tended to walk with a higher cadence.

Table 1 | Mean (SD) of spatiotemporal metrics during walking in healthy subjects and patients with LSS. (*) indicates significant difference from healthy ($p<0.05$)

	Healthy	LSS-NP	LSS-P
Stride Length (m)	1.241 (0.17)	0.937 (0.22) *	0.941 (0.24) *
Velocity (m/s)	1.086 (0.19)	0.841 (0.25) *	0.848 (0.26) *
Cadence (step/min)	107.106 (9.20)	104.871 (13.15)	105.608 (13.12)

3.3 Standing Analysis:

Mean (SD) of the pelvic tilt average angles across healthy, LSS-NP and LSS-P participants were 0.36º (0.35), 5.89º (4.34), and 4.85º (4.36), respectively, in standing (**Table 2**). Positive values in pelvic tilt relate to posterior rotation and negative values coincide with anterior pelvic rotation. Both LSS groups had significantly more posterior pelvic tilt than the healthy group ($p=0.001$). Peak pelvic tilt across healthy, LSS-NP and LSS-P were 0.44º (0.71), 6.71º (4.58), 7.22º (3.78), respectively with both LSS groups showing a higher peak from the healthy group ($p=0.002$). The pelvic tilt range was not different among groups.

Analysis of the average lumbar spinal angles for both LSS groups shows greater lumbar lordosis compared to the healthy group. L1/S1 average lordosis angles increased by 4.61º in LSS-NP and 3.58º in LSS-P. The LSS-NP had a significantly higher lumbar angle than the healthy and LSS-P groups ($p<0.05$). The overall range of lumbar angles were significantly higher for both LSS groups by over 1.5º ($p<0.05$). Our results clearly show an increase in lumbar angles among both SLSS groups but with a slight decrease after the claudication symptoms began. No thoracic angle increase was detected.

The C7/S2 measure showed a significant increase in both distance and angle in patients. Compared to healthy C7/S2 distances, LSS groups increased by over 45 mm for the LSS-NP group and decreased by 5mm to 40mm in the LSS-P group. C7/S2 angles also increased from 4.5º in the healthy group to 10.0º in LSS-NP and 8.7º in LSS-P.

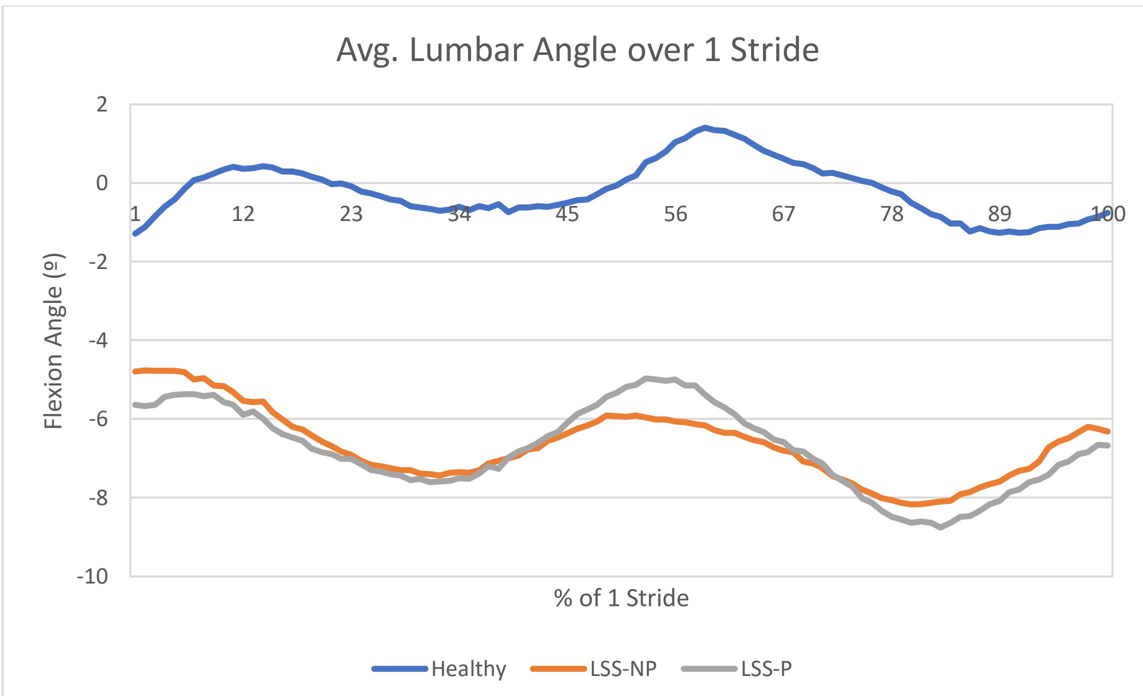
Lumbar loading (L1/S1) displayed high increases in both LSS groups compared to healthy with over 400N increases. This was significantly different than the healthy group ($p<0.05$). Loading in the asymptomatic group was 20N lower than the symptomatic group. Thoracic loading showed small increases in both LSS groups but nothing statistically significant. There was an average increase between the LSS groups and the healthy group of 138N. The overall thoracic loading was not statistically significant ($p=0.084$) but did show increases in both LSS groups.

Right knee flexion angle was measured during the standing neutral trial. The healthy subjects had a mean (SD) angle of -3.03º (4.58). The LSS-NP group had a greater knee flexion angle of -14.42º (11.93) ($p<0.05$) while the LSS-P group had a flexion angle of -11.11º (10.90).

3.4 Walking Analysis:

The mean pelvic tilt angles during walking showed a large difference across the three groups. Healthy average angle was -2.066º (3.031) while LSS-NP was 4.786º (4.907) and LSS-P measured 4.709º (5.158) (**Table 3**). Positive values in pelvic tilt relate to posterior rotation and negative values coincide with anterior pelvic rotation. The pelvic tilt relative to the standing neutral pose pelvic tilt for healthy, LSS-NP, and LSS-P was -2.429º (3.117), -1.1º (3.065), and -0.143º (3.309) respectively. This showed the healthy group being drastically different from both LSS groups ($p=0.0043$). The range of walking pelvic tilt was 3.63º (1.555), 4.445º (1.963), and 4.509º (1.989) for the three groups. While there was no significant interaction between groups, the range relative to the neutral standing position did show a large increase between the healthy and the LSS groups ($p<0.0001$). There was a 1º increase for LSS-NP and a 2º increase for LSS-P in anterior pelvic tilt compared the healthy group. The peak pelvic tilt showed over a 10º shift to the posterior in both LSS groups ($p=0.001$).

The L1/S1 average angle was far higher in both LSS groups compared to the healthy group ($p<0.05$). For lumbar angle, positive values denote extension and negative values denote flexion. The healthy group had an average angle of -0.596º (0.477) over 1 stride while the LSS groups had an average angle of -7.125º (5.727) for the no pain group and -7.425º (6.095) for the pain group (**Figure 1**). The angle relative to the neutral standing pose also shows increases in lumbar lordosis in the LSS groups with an increase of over 2º compared to the healthy group. The peak L1/S1 angle was seen in the healthy group (-11.348º) with the LSS groups containing peaks of around 9º. There was little to no difference in average thoracic angle during walking between groups as well as no differences between standing and walking trials.



The C7/S2 average angle in the healthy group was 9.038º (4.887). There was a 4º increase in both LSS groups with averages of 13.471º (4.696) in the no pain group and 13.379º (5.602) in the pain group.

Lumbar loading during walking increased by 340N for the healthy group compared to standing. This was not statistically significant difference but a large increase nonetheless. There was a 200N increased in thoracic loading for the healthy group and little to no increase in loading during walking for both LSS groups. For both LSS groups, lumbar loads were higher than the healthy group but only by approximately 120N. In addition, the difference between standing and walking lumbar loading was only a 25-30N increase during walking.

Knee angles were measured on the right knee during the stance phase of one stride of the walking trials. The healthy subjects had a mean (SD) angle of -14.84º (5.65) for the right knee. The LSS-NP group had a greater average angle of -22.52º (11.06) for the right knee, while the LSS-P group had a similar angle of -22.64º (10.74) ($p<0.05$).

Table 2 | Descriptive statistics of postural angles and compressive loading during **standing** in healthy subjects and patients with LSS. (*) indicates significant difference from healthy ($p<0.05$).

Standing		Healthy		LSS-NP		LSS-P	
		Mean	SD	Mean	SD	Mean	SD
Pelvic Tilt (º)	Avg	0.357	0.353	5.886*	4.342	4.852*	4.356
	Range	0.509	0.189	2.285	2.772	1.250	0.719
	Peak	0.439	0.708	6.712*	4.576	7.216*	3.783
L1/S1 (º)	Avg	0.018	0.083	-4.587*	4.697	-3.557*	4.936
	Range	0.027	0.010	2.729*	3.758	1.535*	1.284
	Peak	-9.549	5.665	-5.928	5.971	-4.321	5.290
Load (N)	T1/T12	313.004	84.961	451.072	178.413	468.936	194.595
	L1/S1	407.312	114.665	831.945	464.467	851.85*	454.654
C7/S2 (mm, º)	Distance	37.170	24.773	82.868	36.921	74.079	44.694
	Angle	4.567	3.109	10.038	4.552	8.796	5.261
Knee (º)	Right	-3.031	4.575	-14.420*	11.934	-11.111	10.901

Table 3 | Descriptive statistics of postural angles and compressive loading during **walking** in healthy subjects and patients with LSS. (*) indicates significant difference from healthy (p<0.05).

Walking		Healthy		LSS-NP		LSS-P	
		Mean	SD	Mean	SD	Mean	SD
Pelvic Tilt (°)	Avg	-2.066	3.031	4.786*	4.907	4.709*	5.158
	Avg-ref	-2.429	3.117	-1.100*	3.065	-0.143*	3.309
	Range	3.630	1.555	4.445	1.963	4.509	1.989
	Peak	-3.799	4.690	7.601*	5.993	6.072*	8.550
L1/S1 (°)	Avg	-0.596	0.477	-7.125*	5.727	-7.425*	6.095
	Avg-ref	0.615	0.481	2.538	2.327	3.868*	2.307
	Range	0.252	0.082	3.920	2.977	4.011	2.88
	Peak	-11.348	5.387	-8.895	6.972	-9.204	7.224
Load (N)	T1/T12	545.067	215.423	467.236	125.459	467.123	139.109
	L1/S1	746.428	217.528	865.141	241.716	878.567	316.193
C7/S2 (mm, °)	Distance	73.944	38.238	115.255	38.817	113.703	45.481
	Angle	9.038	4.887	13.471	4.696	13.379	5.602
Knee (°)	Right	-14.848	5.652	-22.518*	11.061	-22.635*	10.735

4. Discussion:

The main result of my study shows how patients with LSS have postural changes (when compared to healthy subjects) that increase lumbar loading. The combination of posterior pelvic tilt and lumbar flexion create a spine that is angled further forward compared to healthy controls.

Our previous study examined gait and postural factors in patients after the onset of claudication symptoms (Mousavi et al., 2021), but did not compare patients with healthy controls. In my thesis, I have added an older healthy group to gain insight into how patients with LSS may be different. While the lack of within group differences for the LSS group shows that inducing claudication symptoms does not greatly change their gait kinematics, there is a notable difference in a between patients and healthy subjects.

Focusing on the standing results, the greater posterior pelvic tilt and lumbar flexion may allow the LSS patients to widen the lumbar foramina. This posture is significantly different than the healthy group ($p<0.05$). My results are in line with Goto et al. (2017) who reported increased pelvic sagittal plane angles, but no change in thoracic spine flexion angle in patients while standing. The average thoracic angle was not different between healthy and LSS, showing no hyper kyphosis in the LSS group. Clinical observations show bending forward during standing can relieve pain in patients with LSS, and my results are in line with these observations (Katz and Harris, 2008). The C7/S2 angle was larger by 6º ($p=0.01$) in the LSS group meaning the center of mass was located further forward from the pelvis thus causing an increase in lumbar loading (400+N). In addition to the increased C7/S2 angle, other non-sagittal motion/angles (not analyzed here) could be adding to the lumbar loading. Measuring spinal compression values at all thoracolumbar levels is unique to my work. While the L1/S1 compressive loads for LSS patients were significantly larger ($p<0.05$) compared to healthy group, there was no significant difference between LSS groups.

Peak lumbar angle showed a higher value in the healthy group but that was only because one healthy subject had a larger lordosis angle. Thoracic angles were also measured to check if the upper spine displayed increased kyphosis to possibly account for increased loading.

The changes in knee angle are very pronounced during standing for the LSS patients. If patients are trying to change their pelvis position to allow for more lumbar flexion, then one postural strategy is to increase knee flexion. From a balance standpoint, if the upper body is flexed forward, a coincident movement of the center of mass backwards to achieve balance is needed (Ramari et al., 2018).

While most clinicians will use standing measurements for ease and promptness, a full dynamic walking analysis can allow for greater understanding of the condition. Bending forward during gait has been suggested to relieve pain and symptoms in patients with LSS, but my results did not show a drastic change in lumbar or pelvic angle pre- and post-claudication symptoms (Igawa et al., 2018). During the walking trials, LSS patients walked with posterior pelvic tilt, lumbar flexion, and greater knee flexion. The expectation of the pelvis moving towards a more anteriorly rotated position is seen with a small decrease in the angle compared to standing. There is a 1º rotation towards the anterior (compared to standing) in the N-P group, while the LSS-P had no change in pelvic tilt compared to standing. However, not all subjects walked with their pelvis rotated in the same orientation. The LSS group had 9 of 10 patients walking with posterior rotation, yet after claudication symptoms were provoked, 1 patient changed from a posterior to an anterior pelvic tilt. The LSS group overall walked with a 6º posteriorly rotated (on average) compared to the healthy group. This result can be explained by the LSS patients maintaining a posterior tilt to maintain as wide a spinal foramen opening as possible as symptoms begin. Eight healthy subjects displayed anterior rotation of the pelvis while 2 walked with slight posterior rotation.

The LSS group had close to the same amount of lumbar compression whether they were walking or standing i.e., there was only around a 20N increase. Although the lumbar flexion is greater during walking, the loading only slightly increased compared to standing. Other factors that affect the loading (i.e., out of plane movements) could come into play that we did not analyze. Walking loading in patients was more than in healthy by 120N and this corresponds to differences in the spine posture. There is an association between the lumbar loading and the spinal angle.

Igawa et al., 2018 showed that LSS patients use one of two different strategies during walking. Either a trunk-flexed posture to increase step length and hip extension angle, or with an upright trunk posture to decrease step length and hip extension angle. My stride metrics data shows a smaller stride length compared to healthy subjects, but with a trunk flexed posture. The decrease in stride length and velocity for both LSS groups may be caused by the adoption of this trunk-flexed posture. This compensatory strategy, perhaps temporarily effective in reducing symptoms, may cause the patient to be posturally unstable and energy inefficient, as it demands compensatory motions and higher muscular activity to maintain dynamic balance (Saha et al., 2007; Saha et al., 2008). This may soon lead to general or local muscular fatigue, forcing patients to stop or limit their walking.

Factoring in the knee flexion angles with the spatiotemporal metrics, there is a compelling link between the reduction in stride length and velocity and increase in knee flexion angle during the stance phase. Increased knee flexion is a known compensatory strategy because knee flexion can lead to the pelvis posterior rotating and the lumbar spine flexes or straightens. And conversely, it has been reported that changes in the sagittal profile of the spine affect the hips and knees (Fogel et al., 2003).

Future analyses are needed to explore these possibilities to identify the mechanisms by which lower extremity kinematics and non-sagittal movements affect gait in these patients.

One of the strengths of my study is that in 3 strides with a small sample size, I was able to find significant differences between groups. The protocol used did not allow me to fully fatigue the subjects as they would out in the real world. If a subject was walking in a real-world situation, such as city blocks between work and home and was analyzed by wearable technology such as IMUs, perhaps even greater differences in gait could be uncovered. In other studies (Yokogawa et al., 2015, Loske et al., 2018), subjects would be pushed to greater levels of claudication pain and then tested. We did not want to hurt the patient as they were most often less than a week until their laminectomy surgery. In addition, many subjects were tested during the COVID-19 pandemic which led to challenges in recruitment. All these factors led me to collect the subsequent data and still find significant differences between healthy and LSS as well as pre -and post-claudication within LSS groups.

A future study proposing using wearable sensors to analyze patients outside the lab when they are performing their normal activities of daily living would assist in fully understanding their gait. Additionally, testing patients with preselected paces and analyzing how spine posture and loading changes when they walk faster or on an incline or decline can shed light on the motion of the pelvis and spine.

Currently, physical therapy is a significant aspect in the treatment of LSS, though with low effectiveness and limited evidence (Ammendolia et al., 2013; Schneider et al., 2019). Many current therapeutic exercises rarely target underlying biomechanical factors behind symptom provocation or mobility limitation and seek to temporarily reduce pain. Seated, standing, and supine lumbar flexion stretches do allow the patient a temporary reprieve from neurogenic claudication symptoms by aligning the foramen. However, this is simply an exaggeration of the posture they are already adapting throughout their day. If a new client presents with increased posterior pelvic rotation, lumbar flexion, and decreased stride length and velocity, exercises focusing on strengthening spinal muscles such as the quadratus lumborum, erector spinae, and multifidus. A stable spine may reduce loading and reduce excessive intervertebral motion. In addition to increasing stability in the lumbar spine, working on decreasing the posterior pelvic tilt through strengthening the quadriceps and iliopsoas muscles and bring the patients gait closer to that of a healthy adult. There are some combinations in postural changes and loading that this and future data can be used in tandem with physical therapy to prescribe stabilization exercises to decrease pain and improve quality of life.

5. Limitations:

A small samples size is a limitation of this study and will limit the generalizability of the findings. Working with musculoskeletal models comes with a series of limitations. All spine loads are estimates based off static optimization results from the musculoskeletal model. Healthy subjects performed a series of lifting and static hold tasks prior to performing the walking tasks. This may have caused the healthy subjects to be fatigued prior to walking which may have affected gait kinematics. However, between-group effects were nonetheless found, suggesting any such effects were not of importance.

All kinematic measurements were focused purely in the sagittal plane and do not take into account non-sagittal movements such as medial/lateral sway. To further understand the condition, analysis of the frontal plane movements can be added to that of the sagittal plane. Adding the resultant forces and moments could be helpful as well.

6. Conclusion:

When compared to healthy subjects, patients with LSS had higher posterior pelvic tilt, lumbar flexion, lumbar compression, and C7/S2 angles & distances. This supports the hypothesis that patients adopt a flexed spine posture to increase spinal foramen diameter. My biomechanical analysis showed that spine loading was larger both during walking and standing compared to healthy subjects, thus may contributed to the onset of pain and nerve-related symptoms or it was compensatory to altered gait pattern after provocation of claudication symptoms. My analysis has showed the deepening divide in gait kinematics between older healthy adult populations and those suffering from LSS. It illuminates the differences between posture, spine loading, and gait with a relatively small sample size and with a limited number of strides. However, more studies looking at non-sagittal plane motions and longer duration walking trials are needed to better understand this issue (Suda et al., 2002; Comer et al., 2010). With continued developments in markerless motion capture and the creation of subject-specific kinematics and musculoskeletal models, I see their use being much more available to the public (Camomilla et al., 2020). This will allow far more subjects to be tested with resultant data being much easier to process and analyze. Hopefully one day, lumbar spinal stenosis will not be such a burden on the adult population.

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