Lumbar Posture—Should It, and Can It, Be Modified? A Study of Passive Tissue Stiffness and Lumbar Position During Activities of Daily Living

Background and Purpose. Physical therapists commonly attempt to reduce and prevent low back pain by “improving” individuals’ lumbar posture. To investigate the physical therapy clinical practice of attempting to “improve” lumbar posture, measures of passive tissue stiffness and angular deformation during activities of daily living were used. Participants. The lumbar spine posture of 150 university students was measured as the inclinometer angle difference between L1 and S1. Eighteen female participants (6 with hypolordosis, 6 with hyperlordosis, and 6 controls without lumbar spine impairment) were recruited from this lumbar posture database. Hypolordosis and hyperlordosis were clinically classified by physical therapists. Methods. Lumbar passive tissue stiffness was measured during sitting, standing, and walking before and after a 12-week exercise program, and estimates of lumbar passive tissue strain were calculated from those measurements. Results. The neutral zone (NZ), a range of lumbar positions of low passive tissue stiffness, was identified. Prior to training, the subjects with hypolordosis had more passive tissue strain during sitting than the subjects with hyperlordosis, and the subjects with hyperlordosis stood in extension relative to their NZs while the control subjects and subjects with hypolordosis stood within their NZs. Before and after training, subjects in all 3 groups walked with lumbar spine positions within their NZs. After training, the lumbar posture of the subjects with hypolordosis and the subjects with hyperlordosis changed toward a “mean” (mid-range) lumbar posture. After the exercise program, subjects in all 3 groups stood and walked with their lumbar spines in positions within their NZs, and they sat with their lumbar spines flexed relative to their NZs. Discussion and Conclusion. Knowing that tissue failure can be related to passive tissue strain, the results of this study support the clinical practice of attempting to change individuals’ posture-related lumbar spine positions during activities of daily living. Lumbar passive tissue strain, as estimated from the NZ and angular deformation during activities of daily living, can be decreased, but can also be increased, by an exercise program. [Scannell JP, McGill SM. Lumbar posture—should it, and can it, be modified? A study of passive tissue stiffness and lumbar position during activities of daily living. Phys Ther. 2003;83:907–917.]

Key Words: Lumbar elastic equilibrium, Passive tissue strain, Posture.

Joan P Scannell, Stuart M McGill
Extreme lumbar postures, also called “hypolordosis” and “hyperlordosis,” are thought by some physical therapists to be indicative of altered muscle activity and stress patterns such that tolerance of particular activities of daily living (ADL) of an individual with hypolordosis or hyperlordosis is reduced.1,2 There is, however, little data to support this contention, and some research has questioned the relationship. In addition, there are no widely accepted operational definitions for hypolordosis and hyperlordosis. One approach is to attempt to change these extreme lumbar postures toward a mid-range lumbar posture in order to reduce what might be excessive tissue stress. Despite the widespread use of these efforts, little data are available to justify this approach. Several critical issues emerge: Is hyperlordosis or hypolordosis the consequence of individual anatomy such that, for an individual, it is a posture of least elastic strain (ie, elastic equilibrium)? Is the chosen standing posture indicative of tissue strains in other ADL tasks such as sitting? Can a physical therapy intervention change posture, positions assumed, or elastic equilibrium? Insight into these issues would provide evidence for such practice. The hypotheses addressed in this investigation were: (1) Do individuals with hypolordotic lumbar curvature and those with hyperlordotic lumbar curvature function in different regions of the torque-angular deformation relationship of the lumbar passive tissue (which is the angular manifestation of tissue stress or strain) during ADL tasks? and (2) Can a 12-week training program designed to alter lordosis actually do so in people without impairment or known pathology, and if so, are the strain levels on lumbar tissues reduced during ADL tasks?

The lumbar spine posture of least elastic strain, known as “elastic equilibrium,” is a position where passive tissues on either side of a joint balance to zero moment—the angle of minimal joint load.3 Some authors4 have argued that elastic strain can be an etiology of low back pain (LBP) where LBP is indicative of the load (stress) that is applied repeatedly or for a sustained period of time to a tissue, resulting in cumulative strain that exceeds the strain tolerance of the tissue, consequently resulting in pain and eventually in tissue failure.5 Identification of and subsequent training to move the lumbar spine toward a position of elastic equilibrium has merit as it would reduce passive tissue strain and perhaps LBP.

Several studies that have quantified the effects of various degrees of lordotic postures demonstrate the related controversies. One benefit of lumbar lordosis was suggested in a postmortem study by Farfan et al,6 which noted an association between decreased lordosis and increased degeneration of the L5-S1 disk. Since then, numerous researchers have associated decreased lordosis with increased intradiskal pressure (IDP)7 and increased LBP,8,9 but these were not longitudinal studies, which would better reflect cause and effect. In lumbar extension (increased lordosis), the forces on the facet joints are supported by both the articular surfaces and the capsular ligaments. Shirazi-Adl and Drouin,10 using a finite element model, reported that the facet joints carry large forces in extension, whereas in small degrees of flexion they carry none. Under a 10-Nm extensor moment, the L4-5 facet articular processes carried a contact force of approximately 90 N as compared with zero contact force at the L4-5 level under a flexor moment of 10 Nm. Addition of compression tends to increase these contact forces in extension, but it has no effect on them in small degrees of flexion. With hypolordosis (lumbar flexion), there is less compression of the facet joints together with an increase in the space available within the spinal canal and especially of the foramina of exit, which relieves the compressive effect on the nerve roots and the cauda equina. There is, however, no widely accepted method for characterizing whether lordosis is hypolordosis or hyperlordosis, and at present judgments are based on clinical opinions. Flexion stresses are thought to play a role in lumbar disk failure, most commonly in the posterior or posterolateral aspect of the annulus. Increased IDP11,12 and increased posterior annular tension on flexion have been shown. Gordon et al13 produced disk ruptures by combining rotation (1°–3°), flexion (7°), and compres-

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sion (1,334 N) within physiological ranges. Ten of these disks failed through annular protrusions, and 4 disks failed through nuclear extrusion through annular tears. These findings suggest that people who undergo increased total or segment flexion in their ADL are more at risk of disk protrusions when combined with a given level of compression and rotation. More recently, Callaghan and McGill\textsuperscript{12} were able to consistently prolapse porcine cervical disks (posterior or posterolateral) by repeated full flexion under low levels of compression.

Passive tissue stresses are at an average minimum level when the lumbar spine is in a position, or zone, of elastic equilibrium. Tissue strain and the risk of irritation or damage increase as a function of the rotation away from elastic equilibrium. Research\textsuperscript{8–12} supports the proposition that individuals with hypolordotic or hyperlordotic lumbar spine posture have more tissue strain and a smaller prefailure tissue safety margin when performing various ADL tasks such as sitting, standing, and walking. The clinical classification of hyperlordotic or hypolordotic postures and the “ideal” posture clinicians aim to achieve, however, are based on the observations and judgment of the clinician. Furthermore, can physical therapists actually change standing and sitting postures, and, if so, does this reduce the elastic strain?

Three cascading experimental approaches were used to address our hypotheses:

1. Identification of individuals with hyperlordotic and hypolordotic spines from a large population. We used this approach to obtain the required experimental cohort for our study because hyperlordotic and hypolordotic postures are those that many clinicians attempt to change.

2. Measurement of lumbar passive tissue stiffness, from which elastic equilibrium was identified and subsequently from which estimates of passive tissue strain in ADL (sitting, standing, and walking) were calculated. Passive tissue strain has been shown to increase as a function of angular deformation away from lumbar positions of low passive tissue stiffness.\textsuperscript{5} Positions other than elastic equilibrium are indicative of passive tissue strain—a finding, based on individual passive tissue strain, that would support clinical attempts to reduce passive tissue strain.\textsuperscript{3–5}

3. An interventional exercise program, based on a survey of 30 clinicians, aimed at changing extreme lumbar postures to reduce the strains thought clinically to be imposed by natural hyperlordotic and hypolordotic postures. This program was developed prior to testing passive tissue stiffness and was not based on the results of the second experiment.

Method

All 3 experimental approaches were approved by the University of Waterloo Office of Research Ethics, ensuring that the rights of the subjects were protected.

Finding the Experimental Cohort

Our first objective was to obtain a cohort of subjects that had among them individuals with hyperlordotic and hypolordotic spines. After giving their informed consent, 150 undergraduate university students (102 female and 48 male students with a mean age of 19.9 years [SD=1.2, range=18–24]) were screened to identify those with a hyperlordotic or hypolordotic lumbar posture. The initial test involved obtaining inclinometer readings from the L1 and S1 spinous processes of each participant in a relaxed standing position. The posture of the lumbar spine was calculated according to Adams et al\textsuperscript{13} (angle at L1 \(\pm\) angle at S1), a method previously tested for reliability (SD=2.5°) and validity (\(r=0.91\)). Lumbar lordosis measurements recorded using an inclinometer were compared with radiographic measurements of lumbar lordosis. The purpose of our study was to address the clinical management of people with extremes of lumbar postures. A key consideration, therefore, was to define a classification of lumbar posture. Definitions of hypolordosis and of hyperlordosis do not exist in the literature. Our intention was to characterize the extent of lordosis using the 1st through 10th and 90th through 100th population percentiles. Because this approach unnecessarily limited the size of the subject pool, 2 physical therapists, both PhD candidates with over 5 years of clinical experience, made the clinical classifications of hyperlordosis (inclinometer readings of less than \(-25^\circ\)), “mean” (mid-range) lumbar posture (inclinometer readings of \(-17^\circ\) to \(-19^\circ\)), and hypolordosis (inclinometer readings of greater than \(-8^\circ\)). These angles were used to distinguish between the groups. Eighteen female subjects were recruited from this initial group of 150 students based on the posture of their lumbar spine (6 subjects with hypolordotic postures [mean age=19.9 years, SD=1.38, range=18–22], 6 subjects with hyperlordotic postures [mean age=19.6 years, SD=1.63, range=19–23], and 6 controls without lumbar spine impairment [mean age=20.1 years, SD=0.75, range=19–21]).

Identification of Lumbar Elastic Equilibrium

Lumbar angular moment (torque) and angular displacement in the sagittal plane were measured and plotted to obtain the stiffness (slope [\(q\)]) of the lumbar torso\textsuperscript{14} and then to obtain an estimate of the position of elastic equilibrium in the sagittal plane (Fig. 1). To obtain the measurements, the subjects lay on their side with restraining straps fixing their lower extremities and pelvis to a support while their upper torso (top of head to approximately T12) was supported in a cradle that was
floating on a frictionless jig. Movement of the head, neck, and thorax was prevented by the restraints of the cradle. In order to measure elastic equilibrium, muscle activity had to be eliminated. Therefore, 1-cm silver-silver chloride surface electromyographic (EMG) electrodes were applied to the skin over the spine extensors (at the L3 level) and the abdominal external oblique muscles (lateral to the umbilicus) to monitor the activity level of these muscles.

A research assistant monitored the online myoelectric signal and advised the participants if activity was observed. This system enabled the participants to learn to relax their musculature in a matter of minutes. Those trials where more than 0.3 second of 2% maximum voluntary contraction myoelectric activity was recorded were dropped, and only one attempt was made to repeat the trial. Floating in the frictionless cradle, with no muscle activity, created a lumbar curvature in elastic equilibrium. Bending torques were applied (at an average velocity of $4^\circ$/s) to the torso-cradle with a cable whose line of action formed a normal tangent with the top of the cradle, which was aligned tangential with the spine flexion arc. Cable tension was measured with a load cell, and angular displacement was measured with a 3-SPACE Isotrak device.

Data from 3 trials of both flexion and extension were collected for each participant. Each trial lasted 10 seconds. Bending torques were applied in each trial, with the peak torque occurring about 7 to 8 seconds (no differences in loading rate were found). The angular kinematics of the lumbar spine were measured with a 3-SPACE Isotrak system,* where a source producing a high-frequency magnetic field was secured to the participant’s pelvis, over the sacrum, with straps around the torso and between the legs. A sensor module was placed over the T12 spinous process and secured with straps around the participant’s rib cage to isolate lumbar motion.

This system measured the 3-directional cosines about the orthogonal axis of flexion-extension to the accuracy of ±0.3 degree. In order to obtain absolute and relative kinematic measurements during the pretraining and posttraining tests, the device was boresighted, where a particular relationship between the source and the sensor was chosen and considered the position of “0” degrees. When the spine was flexed relative to this boresight position, the Isotrak device recorded the lumbar position as a negative angle. When the spine was extended relative to the boresight position, the Isotrak device recorded the lumbar position as a positive angle. The same boresight position was used for each test, so that the subjects’ lumbar position in ADL on different days could be compared. We attempted to standardize this source-sensor relationship by using a template mold fixed to a horizontal surface, and we recorded this relationship prior to putting the Isotrak device on the subject. The myoelectric channels and load-cell force signals were A-D converted at 100 Hz and stored in computer memory. The Isotrak device contained its own A-D converter, which sampled the signals at 60 Hz while storing the measurements of the angles in binary form on a second computer. The 10-second data collection window was synchronized in time between the 2 computers with a common trigger to the A-D converter on the EMG and load cell collection computer.

Lumbar Spine Position in ADL (Walking, Standing, and Sitting)
Walking, sitting, and standing were considered representative of basic ADL tasks to study.

Walking. The subjects repeatedly walked, self-paced, along a 4.5-m walkway, turning at either end of the walkway, for a total of 60 seconds. Although turning may affect the lumbar positions assumed during the trial, all 3 tests (pretraining, mid-training, and posttraining) were repeated with the same test protocol. In an attempt to record each subject’s true gait pattern, a cognitive task was included to distract the subject from the physical

* Polhemus Navigation Systems, 1 Hercules Dr, Colchester, VT 05446.
task (ie, the subject counted backward from 100 as she walked). The lumbar position data were recorded with the Isotrak device for the full 60-second trial.

Standing. Each subject then stood for 11 minutes. The subject was allowed to shift weight from one side to the other but not to take a step. The subject began watching a movie of her choice (chosen from a selection of 8–10 movies) during the trial. Ten-second trials of lumbar position data were collected with the Isotrak device every 2 minutes during the standing trial.

Sitting. Following the standing and walking trials, each subject sat in a wooden chair for 1 hour and continued to watch the same movie. The chair was a wooden dining room chair with cutouts made in it to accommodate the Isotrak device. The cutout spaces did not contact the participants. The subjects were instructed to sit in any position they preferred and were told that they could move around in the chair as desired, but they could not stand during the trial. Ten-second collections of the lumbar position data were made every 2 minutes with the Isotrak device.

The 12-Week Training Program: Can Lumbar Posture Be Changed? After the initial test, the subjects with hypolordotic and hyperlordotic postures started a 12-week exercise program. The training program (Appendix) used in this study was based on a survey of 30 physical therapists. The 30 therapists surveyed were completing a postgraduate manual therapy lumbar spine assessment and treatment course (run nationally by the orthopedic division of the Canadian Physiotherapy Association). The clinicians were asked (yes/no) if, in their opinion, training the proposed muscles would change the lumbar posture of the subjects with hypolordosis and hyperlordosis. The clinicians also were surveyed regarding the exercise for each of the muscles in question and the progression from one exercise to the next. Over 70% of support for the exercise and progression of the exercise was considered a consensus.

The goals of the training program for the subjects with hyperlordotic postures were: (1) to increase the muscle activity of the abdominal and gluteal muscles (thereby reducing the relative contribution of the erector spinae muscles) and (2) to increase the length of the hip flexor muscles. The goal of the training program for the subjects with hypolordotic postures was to increase the muscle activity of the erector spinae muscles (thereby reducing the relative contribution of the abdominal and gluteal muscles). The training program is in keeping with current clinical practice, as confirmed by results of a survey, and may not be the most effective program to achieve the desired goals. It has not been investigated scientifically prior to this study. The force contribution of a muscle can be increased by increasing the level of activity or the cross-sectional area of the muscle. Changes in lumbar positions can occur by either method. The effects of the exercise program used in this study had not been investigated prior to the study, and changes in force or activity were recognized as possible means of any effects resulting from the program. Abdominal muscle training also was included in the training program of the subjects with hypolordotic postures so that any reduction in the passive tissue contribution to stability would be compensated for by the increase in abdominal muscle activity.

Over the first 6 weeks of the exercise program, the participants were assessed once a week to determine their ability to progress to the next level of the program. The progression of the exercises was assessed according to information provided in the survey. The exercises were performed independently by the participants on a daily basis. Each participant completed a daily log sheet of the exercises she performed. After the first 6 weeks of the training, all 3 groups of participants (subjects with hypolordotic postures, subjects with hyperlordotic postures, and controls) repeated the testing procedure described (ie, mid-training test). The subjects with hypolordotic and hyperlordotic postures were then given a number of exercises to continue independently over the 6 weeks that followed without further review, during which time they continued to complete the daily log sheets to confirm adherence. After a total of 12 weeks, the participants again repeated the testing procedure described (ie, posttraining test).

Data Processing

Identification of elastic equilibrium. The torque-angular deformation curves for each participant were graphed according to the following equations:

\[
\text{Torque (N}\cdot\text{m)} = \text{force}
\]

\[
\text{Moment arm} = [\text{load cell output} \times \text{calibration factor}] \times \text{measured moment arm}
\]

\[
\text{Angular displacement in the sagittal plane (in degrees)} = \frac{\text{Isotrak device output in the sagittal plane}}{\text{Isotrak device output in the sagittal plane}}
\]

Elastic equilibrium is the position the spine would assume in the absence of muscle activity, as determined by the position of least passive tissue stiffness. We expected that the starting position of the lumbar spine would be the same in the stiffness tests of flexion and extension. This was not the case, however, and we came to believe that elastic
equilibrium is a zone ("neutral zone\[NZ\]) rather than a specific lumbar position (Fig. 2).

Within the NZ, the stiffness of the tissues is low, and a small change in torque gives a moderate change in position. Given that there is no standard definition of NZ, on the advice of Professor Manohar Panjabi (personal communication), the originator of the concept of NZ, the definition of "NZ" in this study was chosen to meet reasonable physiological stiffness levels and to delineate among the variable responses observed in our participants. We defined neutral zone as the slope of the section \(0.1 \text{ Nm} / \degree\) and a change in torque over the section \(<7 \text{ Nm}\) (Fig. 3).

Given the typical 3 sections ("toe" phase [removal of crimp], linear phase, and failure) within a biological tissue stress-strain curve, the torque-angular deformation curves were divided into these sections (1="toe" phase, 2=linear phase, 3= failure), with each section having distinctly different slopes (ie, stiffness) (\(q, \text{ Nm} / \degree\)). The limiting angle of each section—angles (a) and (b), respectively—were recorded. The average slope of each section across the 3 trials was calculated. Angle (b) of the highest section (ie, 1, 2, or 3) of a stress-strain curve that met with the NZ criteria was considered the limit of the NZ in that direction of movement. In cases where the slope of the first section was \(>0.1 \text{ Nm} / \degree\), angle (a) was considered the limit of the NZ. The limit of the NZ was identified in both directions of movement in the sagittal plane to give a sagittal-plane NZ for each subject in each test.

**Lumbar position.** The amplitude probability distribution function (APDF) of the raw Isotrak device data collected during each ADL was formed.\(^{15}\) An APDF is the cumulative sum of a variable—in this case, lumbar spine position—over time. For example, the APDF function allows us to report that the subject sat in lumbar positions between x-y degrees for 50% of the trial. Typical of those who use APDFs,\(^{15,16}\) the 50% and 90% levels of each subject’s APDF of each ADL were identified and compared with the NZ of that subject in order to establish her passive tissue strain.

**Data Analysis**

The first hypothesis addressed in our investigation was that individuals with hypolordotic lumbar curvature and those with hyperlordotic lumbar curvature function in different regions of the lumbar passive tissue torque-angular deformation curve when performing ADL tasks. The calculated lumbar passive tissue strain for each of the 3 groups (subjects with hypolordotic lumbar curvature, subjects with hyperlordotic lumbar curvature, and controls) in each of the 3 ADL tasks (sitting, standing, and walking) was compared using a one-way analysis of variance (ANOVA). Also using a one-way ANOVA, further analysis was performed to identify the source of these differences, that is, whether the differences were due to differences in the position of the NZ (location [comparing NZ limiting angles] and size [absolute degrees between the limiting angles] of the NZ) or to...
differences in the lumbar position assumed in a given ADL task. The 50% and 90% levels of the APDF of lumbar position were analyzed. The 50% level is reported in the “Results” section. Any statistically significant results at the 50% level also occurred at the 90% level.

The second question we addressed was whether a 12-week training program designed to alter lordosis actually does so and whether the strain levels on lumbar tissues during ADL tasks are reduced. This question was addressed using a repeated-measures ANOVA (P<.05) to compare changes in: (1) the inclinometer standing lumbar posture readings, (2) the calculated passive tissue strain, (3) the location and size of the NZ, and (4) the lumbar positions assumed in each ADL task among the 3 groups across the 3 tests (pretraining, mid-training, and postraining). The Pearson test was used to test for correlation.

Results
Cohorts operate in different regions of the torque-angular deformation curve of the lumbar passive tissue.

Inclinometer Screening Results
The lumbar lordosis of the 150 university students had a mean of −15.88 degrees (SD=7.67). Mean lumbar lordosis (and standard deviation) was not different between male and female students.

The Neutral Zone
In the pretraining test, there were no group differences in the size and location of the NZ (q=0.1 N·m/°). Increasing the NZ stiffness criteria (eg, to q=0.1 N·m/°) changes the size and location of the NZ, but there were still no group differences in size and location of the NZ. Furthermore, the average stiffness (q) of the first section of the torque-angular deformation graphs for all participants was found to be q=0.13 N·m/° and thus gives physiological support to the chosen NZ stiffness criterion of q=0.1 N·m/°. There were no changes in the size and location of the NZ of each group recorded during the mid-training and postraining tests. Factors that may have influenced the stiffness of the spine or the passive tissue strain results were the participants’ age (potential age-related changes in lumbar spine mechanics), the participants’ height (standard testing setup, possible lumbar spine positional demands imposed), the time of testing (known changes in viscoelastic properties of the tissues over the course of the day may change the stiffness properties), or the movie watched (possible changes in muscle activity). No correlation was found between any of these factors and (1) the position of the participants’ NZ and (2) the lumbar positions assumed during the sitting, standing, and walking trials.

Pretraining Tissue Strain in ADL
Lumbar positions and calculated tissue strain differed between the subjects with hypolordotic lumbar curvature and the subjects with hyperlordotic lumbar curvature (P=.009) during the sitting trial. The subjects with hypolordotic lumbar curvature also sat with their lumbar spine more flexed relative to their NZ than both the controls and the subjects with hyperlordotic lumbar curvature, but the subjects in all 3 groups sat in elastic flexion (lumbar spine flexed relative to their NZ) (Fig. 4). There were no group differences in the location of the NZ; therefore, the differences in the passive tissue strain during ADL tasks probably stem from differences in the lumbar position assumed during ADL tasks. There were differences between the subjects with hyperlordotic lumbar curvature and the subjects with hypolordotic lumbar curvature in lumbar position (50% level of probability function) during sitting (P=.028), standing (P=.004), and walking (P=.004). In the standing trial, the subjects with hyperlordosis stood in elastic extension, whereas the controls and the subjects with hypolordosis stood within their lumbar NZ. There were no group differences in lumbar passive tissue strain. A correlation existed between the inclinometer reading from the pretraining test and the sitting (P=.003, r = −.66), standing (P=.001, r = −.715), and walking (P=.001, r = −.702) positions assumed by the participants in the pretraining test. The greater the degree of lordosis on initial testing, the more extended the spine was in sitting, standing, and walking.

Training Effects on Cohort-Calculated Passive Tissue Strain
Subjects’ adherence to the exercise program, according to their daily log sheets, was not different among the groups. The numbers of subjects who completed the
subjects with hypolordosis became more lordotic changed across the 3 tests. The lumbar curvature of the group with hypolordotic lumbar curvature continued to stand within their NZ (Fig. 6). Relative to the pretraining test, all 3 groups sat in more lumbar flexion during the mid-training test ($P=.005$) (lumbar flexion increased by 4° in subjects with hypolordosis, by 5° in subjects with hyperlordosis, and by 5° in control subjects) and the postraining test ($P>.5$) (flexion increased by only 1° more in all 3 groups relative to the mid-training test results). The changes in the sitting position between the pretraining and mid-training tests were seen in all 3 groups and therefore cannot be considered a treatment effect. Unlike during the pretraining test, differences in the calculated passive tissue strain levels in sitting among groups were not found during the mid-training and posttraining tests. All 3 groups continued to walk within their NZ during the mid-training and postraining tests, and the subjects with hyperlordotic lumbar curvature continued to walk closer to elastic extension (subjects with hyperlordosis were 3° inside the NZ) than did the other 2 groups (subjects with hypolordosis were 22° from elastic extension, and control subjects were 25° from elastic extension).

### Discussion

#### Results as Related to the Hypotheses

The results of this study showed that the estimates of calculated lumbar tissue strain for the subjects with hypolordotic and hyperlordotic lumbar postures were different during various ADL tasks. This finding supports the first hypothesis of our study. According to our results, while sitting for a given period of time, individuals with hypolordotic lumbar postures will sit farther from their NZs (increasing the calculated posterior tissue tension [strain]) than those with hyperlordotic lumbar postures. Group differences in the lumbar spine position in a standing position were not found.

Attempts by the subjects to change their lumbar positions assumed during some ADL tasks, in our opinion, seem to justify the training program. We investigated whether the training program would reduce the calculated lumbar passive tissue strain during ADL tasks, and our hypothesis was not consistently supported. The subjects with hyperlordotic lumbar postures stood within their NZ during the postraining test, and therefore the calculated strains were reduced. However, they sat farther from their NZ during the postraining test than during the pretraining test, which increased their calculated passive tissue strain. Both the controls and the subjects with hypolordotic lumbar postures also sat farther from their NZ during the mid-training and postraining tests. This may have occurred because the pretraining test was performed within 1 month of the participants returning to the university after summer vacation, whereas the postraining test was performed at least 3 months into the academic year. This finding
suggests to us the possibility of a functionally driven change in passive tissue stiffness. The training program reduced the calculated passive tissue strain of the subjects with hyperlordotic lumbar postures in a standing position, but it did not consistently reduce the calculated passive tissue strain of all individuals during all 3 ADL tasks that were tested. Based on our results, we believe clinicians should consider, from a tissue failure point of view, whether the lumbar spines of certain individuals are at risk with certain tasks. For example, should a clinician be more concerned about a person with hypolordosis whose job requires him or her to sit for hours at a time or a person with hyperlordosis performing the same task? A wider reaching question is: Should people meet specific dominant spine kinematic patterns before they are selected for a given activity? The results indicate that a person with hypolordosis could be at greater risk for strain-related tissue failure when sitting than a person with hyperlordosis.

As this is the first study documenting whether lordosis should be and is trainable, no literature exists for comparison. However, the implications of altered tissue loading can be viewed in the context of the existing literature. The current understanding of the mechanism of tissue strain failure gives insight into the particular dysfunction associated with each group of this study. Positions with the lumbar spine either flexed or extended relative to the NZ imply that some passive tissue is beyond the “toe” region of its torque-angular deformation curve and, theoretically, that the prefailure tissue safety margin is reduced. Although subjects in all 3 groups sat with their lumbar spine flexed relative to their NZ, the subjects with hyperlordosis actually sat closer to their NZ than did the subjects in the other 2 groups. People with hypolordosis appear to have greater posterior tissue strain when seated than do people with hyperlordosis. Future work should be directed at whether flexion-associated syndromes (eg, disk herniation) are linked with this population. Perhaps of more interest is facet-joint loading and capsular strain in people with hyperlordosis. Excessive loading of the facet joints can occur in extended postures and full flexion. Concern regarding the increased incidence of tissue failure due to posterior tissue loading in people with hyperlordosis seems to us to be justified based on our data because they stand in more extension and outside their NZ. The data reported here suggest that the safety margin of the lumbar spine tissues of individuals with hypolordosis and hyperlordosis are different and probably should be considered when designing prevention and rehabilitation protocols.

Several limitations modulate the interpretation of our work. In the absence of a standard definition of NZ, the definition used in this study was based on what we consider reasonable physiological stiffness levels. In addition, there are no generally agreed-on definitions of what constitutes a hypolordotic or hyperlordotic posture. The passive tissue torque-angular deformation data in this study is of the intact lumbar torso and not just lumbar spine passive tissues. We considered this to be reasonable because the contributions of viscera, skin, and fat are thought to be relatively small. Unlike most people seen in clinics, the participants recruited in this study did not have a history of LBP. The aim of our study was to investigate the need and ability to alter people’s...
Conclusions

Posture-related differences in lumbar positions assumed, and resulting passive tissue strain as calculated, during ADL exist and perhaps justify attempts to change these lumbar positions. Furthermore, the results of this study suggest that changes in lumbar positions assumed, which increase and decrease passive tissue strain, are possible with training. Walking and standing seem to be safe activities for the lumbar passive tissues because the lumbar spine position during these activities is within the NZ, except in the case of the untrained subjects with hyperlordosis. The data contained in 3 cascading studies suggest potential benefit for physical therapy practice with quantifying and altering passive lumbar strain.

References

## Appendix.
### Training Program Used in the Study

<table>
<thead>
<tr>
<th>Muscle Group</th>
<th>Subjects</th>
<th>Starting Position</th>
<th>Loading Mechanism</th>
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</thead>
<tbody>
<tr>
<td>Abdominals</td>
<td>Subjects with hypolordosis</td>
<td>Lying supine with knees bent, feet on the floor</td>
<td>Lift one foot off the floor</td>
</tr>
<tr>
<td></td>
<td>Subjects with hyperlordosis</td>
<td>Sitting with feet on the floor, neutral spine position</td>
<td>Lift one foot off the floor</td>
</tr>
<tr>
<td></td>
<td>Subjects with hypolordosis</td>
<td>Standing in natural lumbar posture</td>
<td>Sway your body from side to side</td>
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<tr>
<td></td>
<td>Subjects with hyperlordosis</td>
<td>Standing in natural lumbar posture</td>
<td>Lift one foot off the floor</td>
</tr>
<tr>
<td></td>
<td>Subjects with hypolordosis</td>
<td>Lying on your side, support your body on your flexed elbow and your feet</td>
<td>Hold neutral lumbar spine posture in this position</td>
</tr>
<tr>
<td></td>
<td>Subjects with hyperlordosis</td>
<td>Lying on your side, support your body on your hand (elbow extended) and your feet</td>
<td>Hold neutral lumbar spine posture in this position</td>
</tr>
<tr>
<td>Gluteus maximus</td>
<td>Subjects with hyperlordosis</td>
<td>Lying prone with your lumbar spine in neutral posture</td>
<td>Raise one leg (knee flexed to 90°) off the floor</td>
</tr>
<tr>
<td></td>
<td>Subjects with hyperlordosis</td>
<td>Standing with abdominal and gluteal muscles activated</td>
<td>Sway your body from side to side</td>
</tr>
<tr>
<td>Gluteus medius</td>
<td>Subjects with hyperlordosis</td>
<td>Lying on your side with your lumbar spine in neutral posture</td>
<td>Lift your upper leg (ankle, hip, and shoulders in line)</td>
</tr>
<tr>
<td></td>
<td>Subjects with hyperlordosis</td>
<td>Lying on your side with your lumbar spine in neutral posture (knees bent, feet together)</td>
<td>Lift your upper knee, keeping your feet together</td>
</tr>
<tr>
<td>Piriformis</td>
<td>Subjects with hyperlordosis</td>
<td>Standing with one knee flexed and touching a wall</td>
<td>Press your knee into the wall while flexing your supporting knee and keeping your abdominal and gluteal muscles activated</td>
</tr>
<tr>
<td>Synergistic patterning</td>
<td>Subjects with hypolordosis</td>
<td>Four-point kneeling with your lumbar spine in neutral posture</td>
<td>Lift one leg off the floor, straightening it out behind you</td>
</tr>
<tr>
<td></td>
<td>Subjects with hyperlordosis</td>
<td>Standing in lumbar extension</td>
<td>Hold position of lumbar extension</td>
</tr>
<tr>
<td>Extensor stance</td>
<td>Subjects with hypolordosis</td>
<td>Abdominal muscles activated</td>
<td></td>
</tr>
<tr>
<td>Walking</td>
<td>Subjects with hyperlordosis</td>
<td>Abdominal and gluteal muscles activated</td>
<td></td>
</tr>
<tr>
<td>Walking</td>
<td>Subjects with hypolordosis</td>
<td>Four-point kneeling</td>
<td>Keeping your arms in the starting position, sit onto your heels</td>
</tr>
<tr>
<td>Erector spinae</td>
<td>Subjects with hyperlordosis</td>
<td>Standing</td>
<td>Raise your heel to your buttock</td>
</tr>
<tr>
<td>Hip flexors and rectus femoris</td>
<td>Subjects with hyperlordosis</td>
<td>Standing</td>
<td></td>
</tr>
</tbody>
</table>