

Effects of prolonged sitting on the passive flexion stiffness of the in vivo lumbar spine

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Abstract

BACKGROUND CONTEXT: Prolonged sitting may alter the passive stiffness of the lumbar spine. Consequently, performing full lumbar flexion movements after extended periods of sitting may increase the risk of low back injury.

PURPOSE: The purpose was to quantify time-varying changes in the passive flexion stiffness of the lumbar spine with exposure to prolonged sitting and to link these changes to lumbar postures and trunk extensor muscle activation while sitting. A secondary objective was to determine whether men and women responded differently to prolonged sitting.

STUDY DESIGN: Passive lumbar flexion moment-angle curves were generated before, during and after 2 hours of sitting. Lumbar flexion/extension postures and extensor muscle activation levels were measured while sitting.

SAMPLE: Twelve (6 men, 6 women) university students with no recent low back pain were studied.

OUTCOME MEASURES: Quantified changes in the shapes of the passive flexion moment-angle curves (slopes, breakpoints and maximum lumbar flexion angles) were the outcome measures. While sitting, average lumbar flexion/extension angles, the distribution of lumbar flexion/extension postures, average electromyogram (EMG) amplitude, the number and average length of EMG gaps, and trunk extensor muscle rest levels were measured.

METHODS: Participants performed deskwork for 2 hours while sitting on the seat pan of an office chair. Moment-angle relationships for the lumbar spine were derived by pulling participants through their full voluntary range of lumbar flexion on a customized frictionless table.

RESULTS: Lumbar spine stiffness increased in men after only 1 hour of sitting, whereas the responses of women were variable over the 2-hour trial. Men appeared to compensate for this increase in stiffness by assuming less lumbar flexion in the second hour of sitting.

CONCLUSIONS: Changes in the passive flexion stiffness of the lumbar spine may increase the risk of low back injury after prolonged sitting and may contribute to low back pain in sitting. © 2005 Elsevier Inc. All rights reserved.

Keywords:

Lumbar spine; Low back; Sitting posture; Flexion; Muscle activation; Passive stiffness; Injury prevention; Office ergonomics; Gender

Introduction

As a strategy to prevent or reduce low back pain associated with prolonged sitting, many advocate that extended periods of sitting be interrupted with other non-sedentary activities. In the workplace, for example, individuals who perform extended periods of seated deskwork may be encouraged or required to periodically engage in other occupational tasks to promote changes in posture. However, the time-varying changes in the stiffness of the lumbar spine while sitting are largely unknown, and consequently

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performing certain movements after prolonged sitting may increase the risk of low back injury.

When performing deskwork, individuals adopt flexed lumbar spine postures [1], which may result in an increase in the relative contribution of the passive tissues to the maintenance of an upright torso during sitting [2]. If flexed lumbar postures are sustained, the passive flexion stiffness (PFS) of the lumbar spine can decrease over time because of viscoelastic creep [3] or stress-relaxation [4] in the posterior lumbar tissues. Increased intervertebral joint laxity observed with sustained flexion loading [5] was also attributed to fluid loss in the intervertebral discs [6]. Decreased PFS can increase the risk of a hyperflexion injury in situations whereby prolonged sitting is followed by tasks that involve full lumbar flexion.

Alternatively, evidence suggests that PFS can increase in response to prolonged sitting. Previous studies reported increased height of the spine [7,8] and decreased range of lumbar motion [1] after exposure to prolonged sitting. Although the mechanisms responsible for these observations are not completely understood, it is possible that intervertebral discs recovered height during sitting because of time-varying postural adjustments [9] and/or because of decreased magnitude of spinal loading in sitting relative to that endured in preceding activities [10]. Regardless of the mechanism, increased intervertebral disc height is believed to increase PFS by reducing slack in the flexion-resisting ligaments and posterior fibers of the annulus [5]. This reduction in intervertebral joint laxity could also subject ligaments and intervertebral discs to injurious stresses if lumbar flexion movements are performed after prolonged sitting. These two mechanisms for altered spine mechanics (ie, increased or decreased PFS) would influence the development of injury prevention strategies (eg, job-rotation schemes), depending on the specific time-varying changes in PFS with exposure to prolonged sitting.

The primary purpose of this investigation was to quantify time-varying changes in PFS with exposure to prolonged (2 hours) sitting and link any observed changes in PFS with lumbar postures and activation patterns of the trunk extensor musculature measured during sitting. A secondary objective was to determine whether men and women exhibit different responses to this exposure.

Methods

Participants

Twelve volunteers (6 men and 6 women) were recruited from a university student population (Table 1). All individuals reported no low back pain at the time of collection and

had not experienced any bouts of disabling low back pain for a minimum of 1 year before testing. Informed consent was obtained from all participants for the protocol, which had been reviewed and approved by the university's office of research.

Instrumentation

Four pairs of disposable surface electromyogram (EMG) recording electrodes (Ag-AgCl; Medi-Trace; Kendall-LTP, Chicopee, MA) were adhered to the skin bilaterally over the muscle bellies of the lumbar (L3 spinal level) and thoracic (T9 spinal level) erector spinae (ES) muscle groups [11]. A reference electrode was applied over the acromion process of the left scapula. Raw EMG signals were bandpass filtered (10–1000 Hz) and differentially amplified (common-mode rejection ratio: 115 dB at 60 Hz; input impedance: 10 G Ω) (model AMT-8; Bortec, Calgary, AB, Canada) to produce a ± 2.5 V signal. The amplified EMG signals were then A/D converted at 2048 samples/second using a 12-bit ± 2.5 V A/D conversion system.

A force transducer (model LC101-500; Omegadyne Inc, Sunbury, OH) was used during passive flexion trials to measure cable tension. Force transducer outputs were amplified (model S7DC; RDP Electrosense Inc, Pottstown, PA) to produce a ± 1 V signal and were A/D converted at 2048 samples/second using a 12-bit ± 1 V A/D conversion system.

A 3-SPACE Isotrak II system (Polhemus Inc, Colchester, VT) was used to measure lumbar flexion/extension angles. The source, emitting pulsed electromagnetic waves, was mounted over the sacrum with a specialized nylon strap, and the signal-sensing module was taped to the skin overlying the L1 spinous process. As mounted, this system was shown to provide both accurate and reliable measurements of lumbar and flexion motion in vivo [12]. Custom software was used to compute time-varying lumbar flexion and extension angles, based on the position of the source with respect to the sensor and to A/D conversion of the outputs at 30 samples/second. This software also controlled the EMG collection system so that EMG, 3-SPACE and force data were all synchronized.

Normalization procedures

An isometric maximum voluntary contraction (MVC) of the monitored muscle groups was executed according to the procedures described by McGill [11], and baseline EMG values were collected while participants rested quietly in the prone position. Digitized EMG signals were full-wave rectified before being passed through a digital Butterworth low-pass filter (2.5 Hz cut-off frequency) [13] to produce linear-enveloped EMG. All EMG signals were normalized to the maximum values obtained in the MVC task.

After the EMG MVC trial, two maximum voluntary lumbar spine flexion trials were performed. From a normal relaxed upright standing posture (regarded as the zero position), participants were instructed to maximally flex the

Table 1
Average (1 standard deviation) of participant characteristics

Participants	n	Age, y (SD)	Height, m (SD)	Mass, kg (SD)
Men	6	24.5 (1.9)	1.77 (0.06)	76.8 (15.0)
Women	6	23.3 (1.8)	1.62 (0.06)	58.6 (7.0)

lumbar spine by bending forward to touch their hands to the ground (if possible) while maintaining both knees in full extension. Maximum measured lumbar flexion angles were used to normalize lumbar flexion/extension angles during sitting. Sitting postures were normalized in this manner to permit direct quantitative comparisons with those documented in previous studies [1–3,14]. In addition, this normalization procedure provided a biologically relevant measure of lumbar range of motion that could be compared across individuals.

Collection protocol

The effect of prolonged sitting on PFS was investigated by implementing three passive flexion testing sessions: an initial session, one after the first hour of sitting, and one after the second hour of sitting. Tests were performed by using a custom jig [15] that was a modified version of the apparatus designed and introduced by McGill et al. [16] and used more recently by Green et al. [17]. The jig consisted of three main elements: a level horizontal Plexiglass surface (dimensions: 1.22 m×1.83 m×2.54 cm) on which nylon ball bearings (diameter: 1.2 cm; Salem Specialty Ball Inc, Canton, CT) were evenly dispersed, a “floating” upper-body

cradle (lined with 0.64-cm thick Plexiglas) overlaying the horizontal Plexiglas-ball bearing surface and a vertically adjustable lower-body restraining platform (Fig. 1). Functionally, this arrangement provided a frictionless surface over which the cradle could glide. Since it was known a priori that the electromagnetic 3-SPACE system was to be used in the measurement of lumbar flexion/extension angles, the jig was constructed from non-magnetic materials.

With participants lying on their right side, nylon straps were used to affix their upper and lower body to the floating cradle and restraining platform, respectively (Fig. 1). Participants were instructed to fold their arms around the vertical column attached to the floating cradle to minimize axial rotation of the lumbar spine. This position also functioned to limit any relative movement between the upper body and the floating cradle. Lateral curvature of the spine was avoided by making adjustments to the height of both the lower body and head. Repeatability of positioning was established by aligning the anterior superior iliac spine of the pelvis and a mark applied to the left arm with vertical columns attached to the restraining platform and floating cradle, respectively. To further enhance repeatability, an adjustable brace connecting the floating cradle to the restraining platform was used

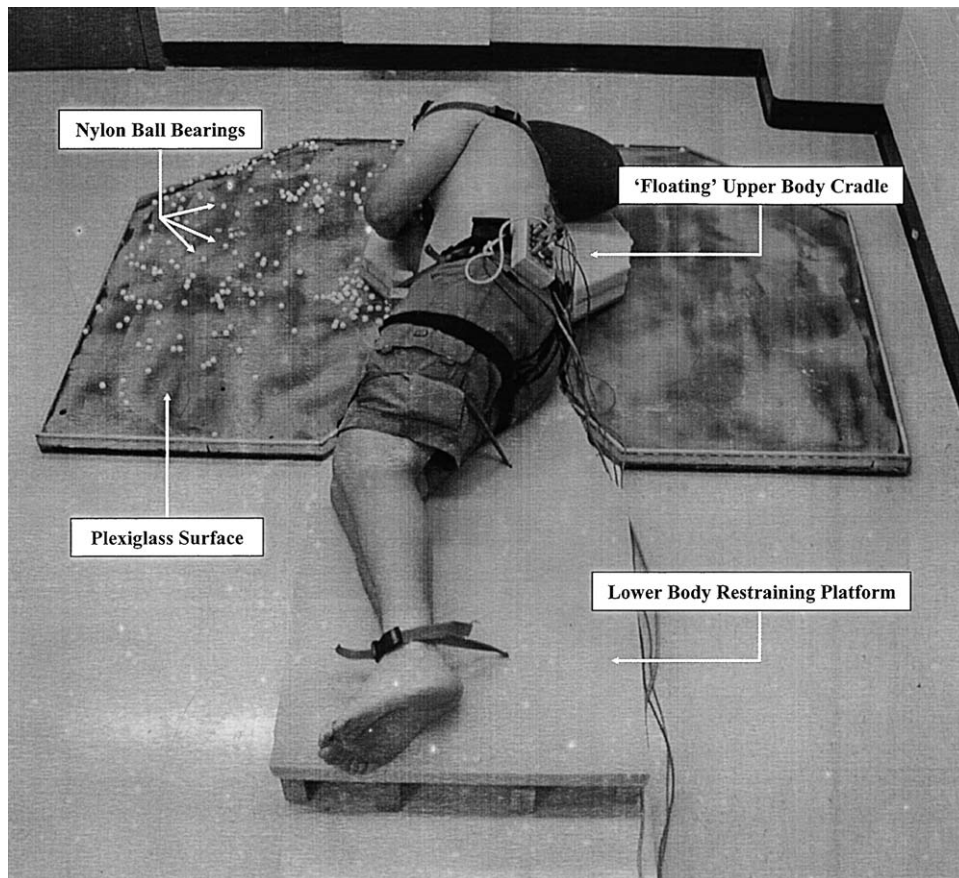


Fig. 1. The frictionless jig with a participant's upper and lower body independently secured to the floating cradle and restraining platform in the initial testing position.

during positioning. Repeatable positioning provided an absolute reference point, such that any observed changes in stiffness were not because of differences in alignment between testing sessions.

Force was applied by the experimenter to the top of the floating cradle, inducing a flexion moment about the lumbar spine. Each passive flexion trial was initiated by an auditory cue and was terminated when the participants reached their voluntary end range of flexion. Passive flexion trials in which ES activation exceeded 5% MVC were recollected, as the purpose was to isolate changes in PFS. The levels of ES activation were monitored on line, using an oscilloscope. Three acceptable flexion trials, constituting one testing session, were collected to enhance the stability of the measurements. A preliminary analysis was performed to verify that passive flexion trials within individual testing sessions were repeatable.

During the sitting sessions, participants were stationed at a desk and were required to engage in non-computer-based tasks (ie, reading and writing). The chair, used by all participants, was the seat pan of an office chair with the back support removed. This chair design was necessary because of the instrumentation used and was not believed to be an important limitation according to the observation that individuals tend to lean forward when reading or writing at a desk [18]. Adjustments to the seat height were made to ensure 90-degree sagittal plane knee angles at the start of each sitting session. ES activation data and lumbar flexion/extension angles were collected for the full hour in each sitting session and were stored in 30-minute increments.

Data analysis

Extensor reaction “moments” were calculated by multiplying the magnitude of the applied force by the perpendicular distance measured between its point of application and the location of the L4–L5 intervertebral joint. Lumbar flexion angles were normalized with respect to the maximum angle achieved in the first trial of the first testing session (Trial 1a) and were plotted against moments. Lumbar flexion angles were normalized in this manner to remove interindividual differences in range of motion and to establish pre-exposure, or baseline, values to which changes could be compared.

Moment-angle curves were then fit with sixth-order polynomials [19] (average $r^2 \pm 1$ SD = 0.98 ± 0.01) to obtain equations that could be numerically differentiated using a 5-point differentiation formula. Differentiation of the moment-angle curves permitted the identification of three zones (low, transition and high stiffness zones) by locating the points at which the greatest percentage of change in the slopes were evident (low and high breakpoints). The slopes of linear trend-lines that were independently fit to the original moment-angle data in each of the low, transition and high stiffness zones were used as a measure of PFS. Changes in low and high moment-angle curve breakpoints were also documented, as

was the range of lumbar flexion between these breakpoints (transition range) (Fig. 2). A left- or right-shifting of the breakpoints was considered to represent increased or decreased PFS, respectively. The final measure used to represent PFS was the maximum voluntary lumbar flexion angle to which participants could be pulled on the frictionless jig. Increased or decreased range of lumbar motion was considered to represent decreased or increased PFS, respectively.

Lumbar postures adopted while sitting were first characterized by calculating average lumbar flexion/extension angles. Normalized lumbar angles were then binned in 1% maxflex increments to generate amplitude probability distribution functions and were used to determine whether participants assumed a static or dynamic sitting strategy [1,14] while performing deskwork. A static sitting strategy was defined as maintaining a sitting posture that was within a 15% range of lumbar flexion for at least 85% of the collection time [14].

Several processing methods were used to quantify ES activation while sitting. Average EMG levels were calculated for each sitting session. Normalized EMG levels were then binned in 1% MVC increments to generate amplitude probability distribution functions and were used to document the amount of time that the ES were at rest while sitting (probability of 0% MVC occurring). EMG gaps analyses were subsequently performed, wherein the number of EMG gaps and the average EMG gaps length while sitting was determined. EMG gaps were defined as periods when ES activation was below 0.5% MVC for longer than 0.2 seconds [20].

Statistical analysis

Two-way repeated measures analyses of variance (general linear model) with one within (session) and one between (sex) factor (repeated measure=session) were used to compare dependent variables across the passive flexion tests and sitting sessions. Tukey post hoc analyses were used when significant differences were found. One-way repeated measure analyses of variance were used when significant sex \times session interactions were observed in the initial two-way analyses. In all statistical tests, a p-value less than .05 was considered to be statistically significant.

Results

Low, transition and high slopes

Slopes were highly variable across participants and testing sessions in all three (low, transition and high) stiffness zones. Despite this variability, transition slopes were significantly different across testing sessions ($p=.0053$). Specifically, transition slopes obtained before the first hour of sitting were lower than those measured after the first and second hours of sitting (Table 2). Systematic changes in low and high slopes were not observed, and, as a result, values were not significantly different across testing sessions ($p=.5083$

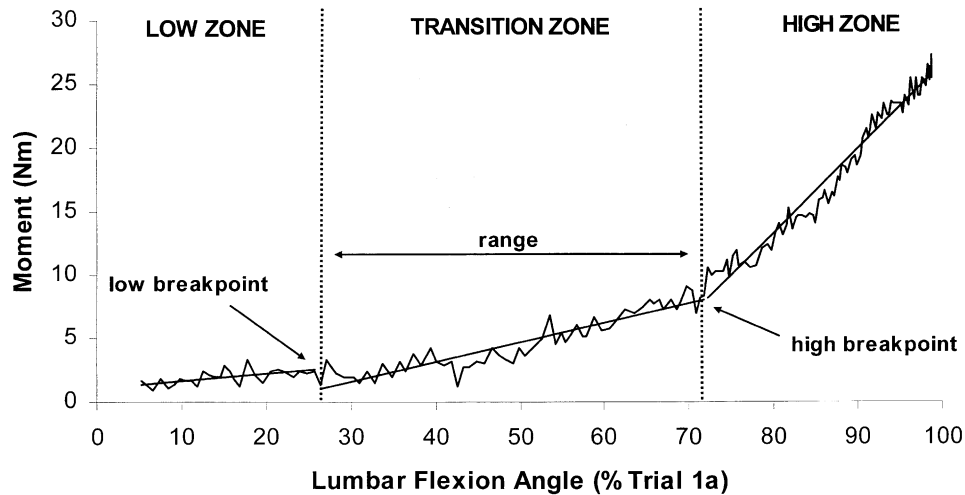


Fig. 2. Typical moment-angle curve. Dashed vertical lines intersect the moment-angle curve at the low and high breakpoints and form the boundaries of the low, transition, and high stiffness zones. The slopes of linear trend-lines, fit to data in each stiffness zone, were used as one measure of passive flexion stiffness (PFS).

and $p = .3509$, respectively). Low and high slopes calculated for men were greater than those for women, although differences in high slopes were not statistically significant (Table 3).

Moment-angle curve breakpoints

The responses of moment-angle curve breakpoint measures differed according to sex, as indicated by a significant sex \times session interaction for low breakpoint measures ($p = .0225$). In men, both the low and high breakpoints were significantly different across testing sessions ($p = .0123$ and $p = .0190$, respectively). Specifically, low and high breakpoints for men occurred at a greater percentage of Trial 1a lumbar flexion in the first testing session than they did in the second and third testing sessions; breakpoints were not different between the second and third testing sessions (Fig. 3, top). Women did not exhibit the same responses, as low and high breakpoints were not significantly different across testing sessions ($p = .8040$ and $p = .7291$, respectively) (Fig. 3, bottom).

The transition range across testing sessions was not significantly different for men ($p = .9472$) or women ($p = .6909$). When coupled with the finding that low and high breakpoints

occurred at lower percentages of lumbar flexion after 1 hour of sitting, unchanged range values indicated that the moment-angle curves generated for men in the second and third testing sessions were left-shifted with respect to those obtained in the first testing session. An illustration of this response is provided in Fig. 4.

Maximum lumbar flexion angles

Differences between men and women were also observed with regard to the maximum lumbar flexion angles to which the experimenter could pull individuals after prolonged sitting ($p = .0237$). In men, significant differences in maximum angles were measured across testing sessions ($p = .0072$). Specifically, maximum angles were greater in the first testing session than those measured in the second and third sessions for male subjects; no differences between angles obtained in the second and third sessions were found (Fig. 5, top). In women, no significant differences were observed in maximum lumbar flexion angles across testing sessions ($p = .3045$) (Fig. 5, bottom).

Lumbar spine postures and muscle activation during sitting

A significant sex \times session interaction ($p = .0199$) for average lumbar flexion/extension angles assumed while

Table 2
Comparisons of the low, transition and high slopes (Newton-meters/% Trial 1a) across testing sessions

Session	Zone		
	Low	Transition	High
1	0.0 (0.1)	0.1 (0.1)	0.7 (0.3)
2	0.1 (0.1)	0.2 (0.1)	1.0 (0.7)
3	0.0 (0.1)	0.2 (0.1)	1.0 (0.9)
p-value	.5083	.0053*	.3074

Values are expressed as average (1 standard deviation) collapsed across all 12 participants.

* Significantly different.

Table 3
Male–female comparisons of the low, transition and high slopes (Newton-meters/% Trial 1a)

Participants	Zone		
	Low	Transition	High
Men	0.1 (0.1)	0.2 (0.1)	1.1 (0.9)
Women	0.0 (0.0)	0.2 (0.1)	0.7 (0.3)
p-value	0.0146*	0.4217	0.3509

Values are expressed as average (1 standard deviation) collapsed across all testing sessions.

* Significantly different.

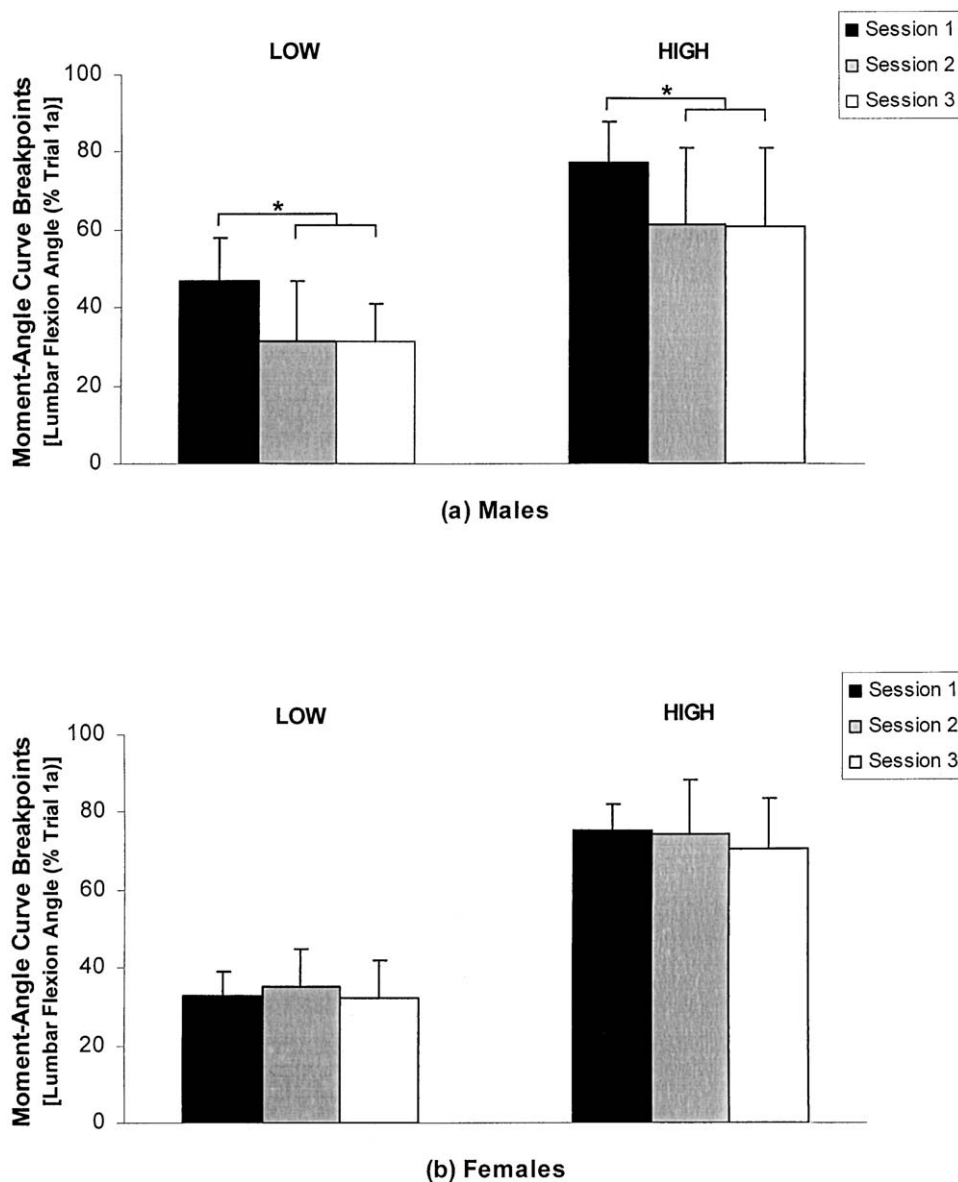


Fig. 3. Locations of the low and high moment-angle curve breakpoints for men and women. The averages of (top) 6 men and (bottom) 6 women are plotted. Error bars represent 1 standard deviation; *significant differences.

sitting also indicated that men and women responded differently to prolonged sitting. The lumbar spines of men were observed to be less flexed in the second hour of sitting, although the differences were not statistically significant ($p=.0927$) (Fig. 6, top). Lumbar flexion angles for women tended to increase over the 2-hour sitting trial; however, these differences were also not statistically significant ($p=.2386$) (Fig. 6, bottom). According to the criterion proposed by Salewytch and Callaghan [14], 7 of 12 participants (4 men, 3 women) exhibited a static sitting strategy. However, no associations could be drawn between a particular sitting strategy and systematic stiffness changes.

No differences were observed in any of the ES activation variables when compared across sitting sessions (minimum $p=.1718$). Sex-related differences in the number of EMG gaps ($p=.0131$) and rest levels ($p=.0218$) were observed

only in the left thoracic ES group. However, these differences were likely because of dissimilarities in the way the individuals performed the deskwork [21] and were not attributed to biological differences in the way that men and women activated their musculature while sitting.

Discussion

The results of this study indicated that PFS is significantly altered in men with exposure to prolonged sitting. The lumbar spines of men became stiffer after 1 hour of sitting, whereas women demonstrated variable responses to the seated exposure. Transition slopes were observed to increase in both men and women after 1 hour of sitting, possibly indicating that women also experienced some lumbar

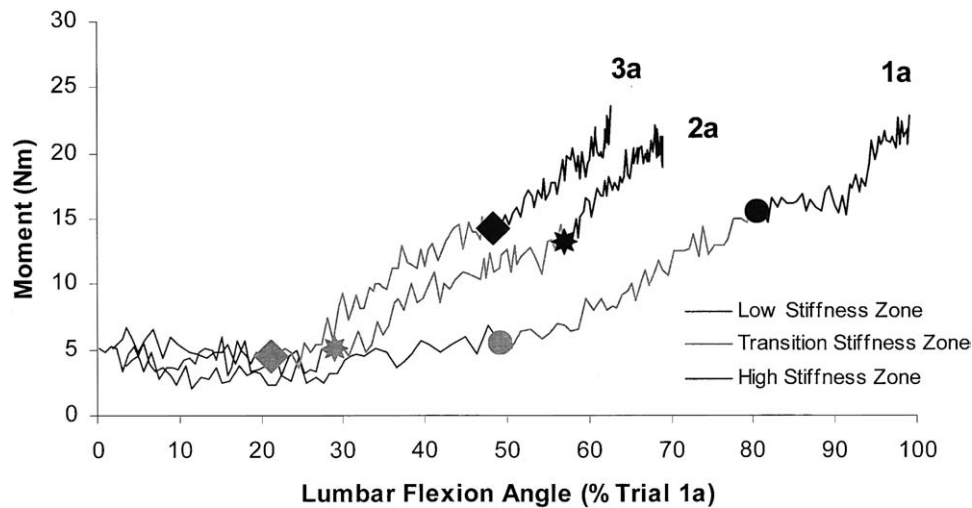


Fig. 4. Illustration of the male passive flexion stiffness (PFS) response to prolonged sitting. Moment-angle curves generated after the first and second hours of sitting were left-shifted with respect to curves generated in the first testing session, indicated here by the location of the low and high breakpoints in trials 1a (●,●), 2a (*,*), and 3a (◆,◆) for 1 male participant.

spine stiffening effects. These findings suggest that the tissue level changes occur with relatively short duration exposures.

The moment-angle curves generated for men after the first and second hours of sitting were left-shifted with respect to those generated before sitting (Fig. 4). Furthermore, after the first and second hours of sitting, men were unable to be pulled through the same range of lumbar flexion angles that were attained before sitting (Fig. 5, top). Decreased range of lumbar flexion in men after prolonged sitting was also observed by Callaghan and McGill [1]. These findings suggest that structures providing passive resistance to lumbar flexion (eg, posterior ligaments and intervertebral discs) may be recruited at lower lumbar flexion angles and, therefore, may be subjected to higher stresses at a given lumbar angle after prolonged sitting. With regard to injury prevention, this finding implies that individuals should avoid tasks that induce full lumbar flexion (eg, stooped lifting) after extended periods of sitting. Similar suggestions were offered by Parkinson et al. [15] and Green et al. [17], who detected time-varying changes in low back stiffness in response to different exposures.

Increased stiffness in the moderate ranges of lumbar flexion in both men and women, indicated by increased transition slopes, could be due to time-varying changes in the passive elastic properties of muscles. Using equations provided by Adams and Dolan [22], McGill et al. [16] concluded that muscles were the primary flexion-resisting tissues in the moderate ranges of lumbar flexion. However, it is not currently possible to validate such an analysis in vivo due to the anatomical and functional complexity of the tissues comprising the lumbar torso. Furthermore, no specific measures of ES activation in this study were able to support or refute the suggestion that changes in the passive properties of muscles contributed to the observed changes in transition slopes.

Men appeared to compensate for these stiffness changes by sitting in decreased lumbar flexion in the second hour of sitting (Fig. 6, top). However, in an occupational setting these postural adjustments might not be possible because of constraints imposed by task requirements or workstation design. This could partially explain why prolonged sitting is often associated with low back pain [23–25], as constrained flexion postures coupled with increased PFS can stimulate pain receptors in the posterior spinal ligaments [26–28].

Measurements of lumbar flexion/extension angles and ES activation made during sitting sessions did not offer any insight into the potential mechanisms responsible for the sex-specific changes in PFS that were observed. Although the 3-SPACE system was capable of distinguishing between individuals who adopted static or dynamic sitting strategies [1,14], the criteria used to make this distinction [14] might not be sensitive enough to detect potential sex-related differences in time-varying lumbar flexion and extension postures. Additionally, measurements made with the 3-SPACE system, as used in this study, were not able to uncover whether sex-related differences existed in relative rotations between individual motion segments comprising the lumbar spine (ie, time-varying differences in lumbar lordosis or kyphosis). Moreover, knowledge pertaining to the timing or magnitude of intrinsic spinal muscle activation (presumed to control small changes in lumbar lordosis or kyphosis) could not be acquired with the use of surface EMG electrodes applied at the levels of L3 and T9. According to the notion that the fluid content of intervertebral discs is modulated by movement [9] and posture [6], undetected changes in lumbar flexion/extension angles might have led to increased fluid absorption in the lumbar intervertebral discs of the men studied. This may have increased intervertebral disc height, resulting in increased lumbar spine stiffness (due to reduced intervertebral joint laxity associated with disc swelling). Exposure to prolonged sitting was shown to be associated with

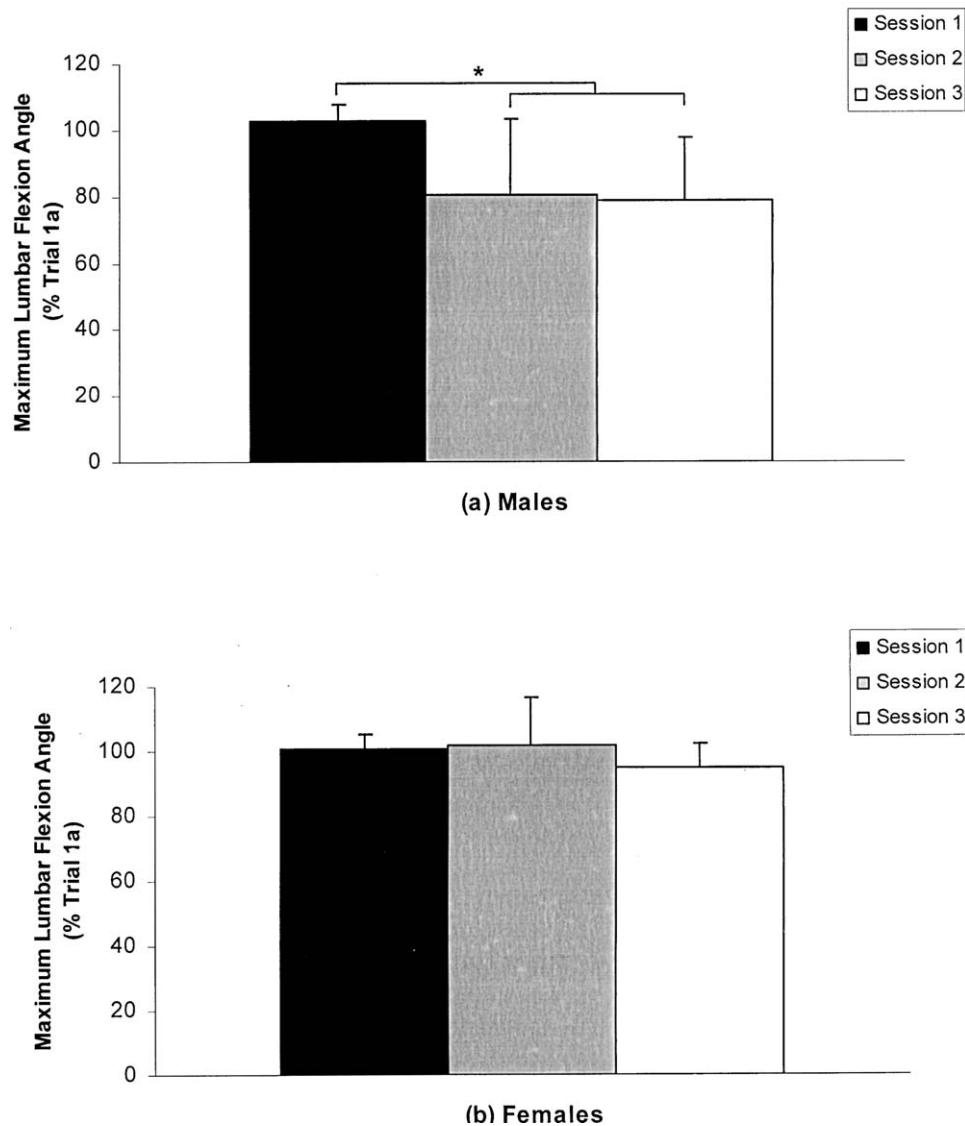


Fig. 5. Maximum lumbar flexion angles to which men and women could be pulled during passive flexion tests. The averages of (top) 6 men and (bottom) 6 women are plotted. Error bars represent 1 standard deviation; *significant differences.

increases in spine height [7,8], possibly because of swelling of the intervertebral discs. Although the responses of men and women were not different in these studies when sitting postures were matched (where possible) to those observed in this investigation, time-dependent measurements of spinal shrinkage in sitting were recently reported to vary according to sex [29].

Cadaveric lumbar motion segments, loaded in approximately 70% of maximum flexion for 1 hour, exhibited decreased resistance to bending after loading [4]. In vivo, passive tissues of the low back were discovered to creep in men and women who were seated in maximal lumbar flexion for 20 minutes [3]. On the basis of these findings and on the observation that some individuals in this study assumed sitting postures that positioned their lumbar spine in approximately 90% maxflex (Fig. 6), it would be reasonable to presume that lumbar spines would show evidence of

decreased stiffness after prolonged sitting. Although this trend was evident in the slopes calculated for some individuals, consistent associations between participants who exhibited the greatest lumbar flexion angles while sitting and participants demonstrating decreasing stiffness could not be drawn. Factors likely contributing to the discrepancies between the findings of this study and the work of others include the inability of in vitro work to replicate physiologic conditions and processes that exist in vivo, the removal of potential lumbar flexion-resisting tissues and organs (eg, musculature, lumbodorsal fascia, skin and viscera) in in vitro investigations and, possibly, the noncomparability of results derived from static and quasistatic loading scenarios (both in vitro and in vivo) to the unconstrained lumbar postures measured here. Furthermore, it is not clear whether, or to what extent, prolonged posterior displacement of the nucleus

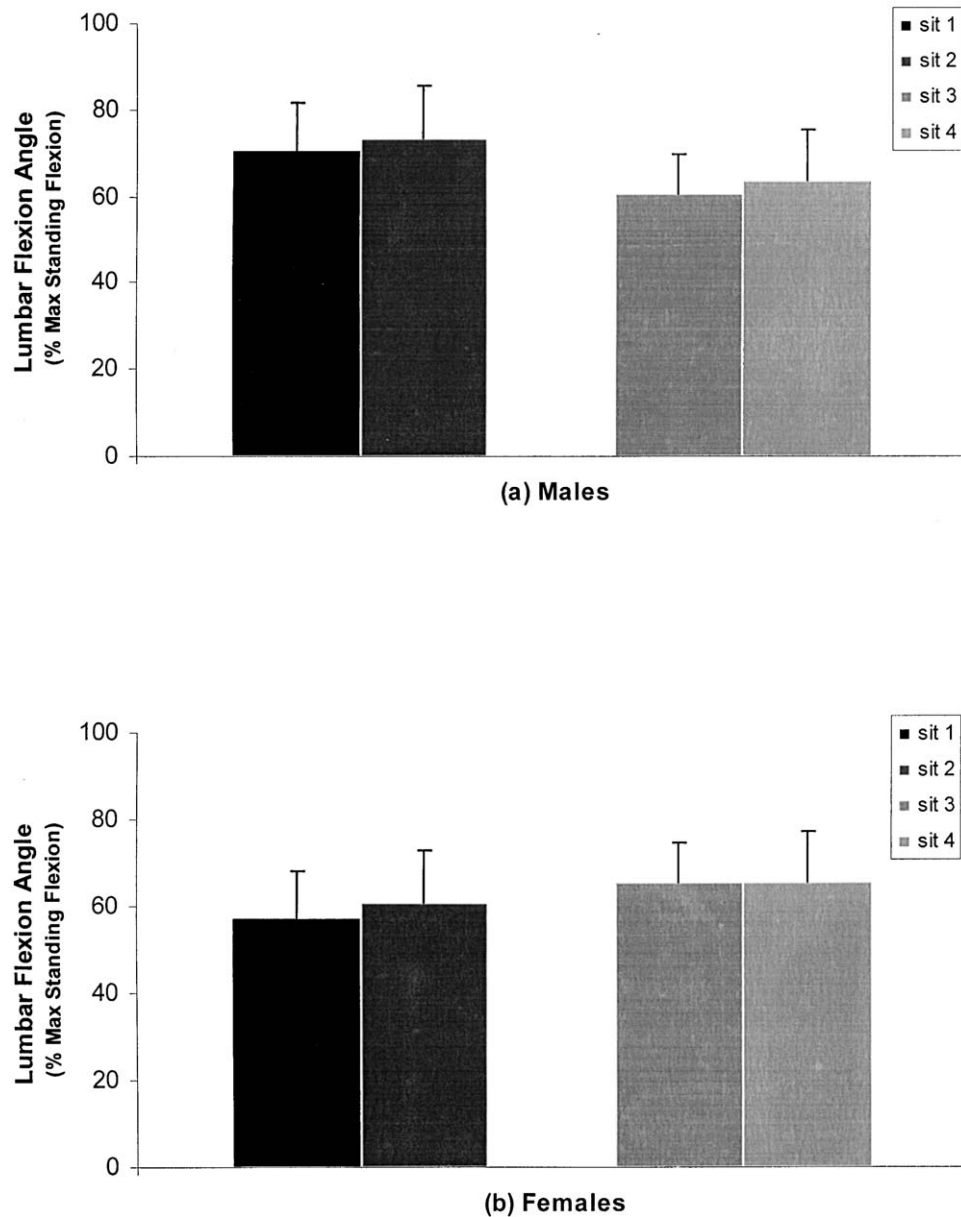


Fig. 6. Average lumbar flexion angles for men and women during sitting. Sit 1 and 2 represent the average lumbar flexion angles calculated for the first and last 30 minutes (respectively) of the first hour of sitting. Sit 3 and 4 represent the average lumbar flexion angles calculated for the first and last 30 minutes (respectively) of the second hour of sitting. The averages of (top) 6 men and (bottom) 6 women are plotted; error bars represent 1 standard deviation.

pulposus during sitting [30] altered the mechanics of the intervertebral joints during passive flexion tests.

Time-varying changes in the contour of the moment-angle relationships, denoted by changes in the slopes, were highly variable across individuals and testing sessions. High intraindividual and interindividual variability in similar measures of low back stiffness has been documented elsewhere [15–17] and was proportional to the variability reported here. There are several potential factors, both methodologic and biological, that could contribute to the variability. First, abdominal muscle activation was not measured in this study because of the difficulty in maintaining adequate electrode–

skin contact during stiffness testing sessions. However, participants were instructed not to contract the abdominals because this would decrease force transducer outputs, and any trials in which the experimenter observed that participants initiated or contributed to flexion were recollected. Second, although participants were encouraged to arrive at the laboratory at approximately the same time on testing day, the amount of time that had elapsed between their arrival and the time at which they arose from bed, and the activities performed before their arrival were not strictly controlled. Hence, responses in lumbar spine stiffness could have varied according to on interindividual differences in the

heights of the intervertebral discs on their arrival [5,10]. Third, interindividual differences in the magnitudes and distribution of tissue loads during sitting, indicated by the observed variability in lumbar sitting postures and ES activation patterns [1], could have altered individual stiffness responses.

Conclusions

Time-varying changes in the passive stiffness of the lumbar spine were demonstrated with exposure to prolonged sitting. The findings of this study suggest that individuals who sit for extended periods can be at increased risk of injury if full flexion movements are attempted after sitting. These changes were evident after 1 hour of sitting, which could be of particular concern for those who design work-rest schedules and job-rotation schemes.

References

- [1] Callaghan JP, McGill SM. Low back joint loading and kinematics during standing and unsupported sitting. *Ergonomics* 2001;44:280–94.
- [2] Callaghan JP, Dunk NM. Examination of the flexion relaxation phenomenon in erector spinae muscles during short duration slumped sitting. *Clin Biomech* 2002;17:353–60.
- [3] McGill SM, Brown S. Creep response of the lumbar spine to prolonged full flexion. *Clin Biomech* 1992;7:43–6.
- [4] Adams MA, Dolan P. Time-dependent changes in the lumbar spine's resistance to bending. *Clin Biomech* 1996;11:194–200.
- [5] Adams MA, Dolan P, Hutton WC. Diurnal variations in the stresses of the lumbar spine. *Spine* 1987;12:130–7.
- [6] Adams MA, Hutton WC. The effect of posture on the fluid content of lumbar intervertebral discs. *Spine* 1983;8:665–71.
- [7] Althoff I, Brinckmann P, Frobin W, Sandover J, Burton K. An improved method of stature measurement for quantitative determination of spinal loading. *Spine* 1992;17:682–93.
- [8] Leivseth G, Drerup B. Spinal shrinkage during work in a sitting posture compared to work in a standing posture. *Clin Biomech* 1997;12:409–18.
- [9] van Deursen DL, Snijders CJ, van Dieen JH, Kingma I, van Deursen LLJM. Passive vertebral rotation causes instantaneous desiccation of the nucleus pulposus. *J Biomech* 2001;34:405–8.
- [10] Kraemer J, Kolditz D, Gowin R. Water and electrolyte content of human intervertebral discs under variable load. *Spine* 1985;10:69–71.
- [11] McGill SM. Electromyographic activity of the abdominal and low back musculature during the generation of isometric and dynamic axial trunk torque: implications for lumbar mechanics. *J Orthop Res* 1991;9:91–103.
- [12] Pearcy MJ, Hindle RJ. New method for the non-invasive three-dimensional measurement of human back movement. *Clin Biomech* 1989;4:73–9.
- [13] Brereton LC, McGill SM. Frequency response of spine extensors during rapid isometric contractions: Effects of muscle length and tension. *J Electromyogr Kinesiol* 1998;8:227–32.
- [14] Salewytch AJ, Callaghan JP. Can quantified lumbar spine postures and trunk muscle activation levels predict discomfort during prolonged sitting? Proceedings of the 31st Annual Conference of the Human Factors Association of Canada. University of Ottawa, Ottawa, Ontario, Canada, 1999:316–21.
- [15] Parkinson RJ, Beach TAC, Callaghan JP. The time-varying response of the in vivo lumbar spine to dynamic repetitive flexion. *Clin Biomech* 2004;19:330–6.
- [16] McGill SM, Seguin J, Bennett G. Passive stiffness of the lumbar torso in flexion, extension, lateral bending, and axial rotation. *Spine* 1994;19:696–704.
- [17] Green JP, Grenier SG, McGill SM. Low-back stiffness is altered with warm-up and bench rest: implications for athletes. *Med Sci Sports Exerc* 2002;34:1076–81.
- [18] Vergara M, Page A. System to measure the use of the backrest in sitting-posture office tasks. *Appl Ergon* 2000;31:247–54.
- [19] Dickey JP, Gillespie KA. Representation of passive spinal element contributions to in vitro flexion-extension using a polynomial model: illustration using the porcine lumbar spine. *J Biomech* 2003;36:883–8.
- [20] Veiersted KB, Westgaard RH, Andersen P. Pattern of muscle activity during stereotyped work and its relation to muscle pain. *Int Arch Occup Environ Health* 1990;62:31–41.
- [21] van Dieen JH, de Looze MP, Hermans V. Effects of dynamic office chairs on trunk kinematics, trunk extensor EMG and spinal shrinkage. *Ergonomics* 2001;44:739–50.
- [22] Adams MA, Dolan P. A technique for quantifying the bending moment acting on the lumbar spine in vivo. *J Biomech* 1991;24:117–26.
- [23] Frymoyer JW, Pope MH, Costanza MC, Rosen JC, Goggin JE, Wilder DG. Epidemiologic studies of low back pain. *Spine* 1980;5:419–423.
- [24] Andersson GBJ. Epidemiologic aspects of low-back pain in industry. *Spine* 1981;6:53–60.
- [25] Videman T, Nurminen M, Troup JDG. Lumbar spinal pathology in cadaveric material in relation to history of low back pain, occupation and physical loading. *Spine* 1990;15:728–40.
- [26] Bogduk N. The innervation of the lumbar spine. *Spine* 1983;8:286–93.
- [27] Cavanaugh JM. Neural mechanisms of lumbar pain. *Spine* 1995;20:1804–9.
- [28] Cavanaugh JM, Kallakuri S, Zatkay AC. Innervation of the rabbit lumbar intervertebral disc and posterior longitudinal ligament. *Spine* 1995;19:2080–5.
- [29] Kanlayanaphotporn R, Trott P, Williams M, Fulton I. Effects of chronic low back pain, age and gender on vertical spinal creep. *Ergonomics* 2003;46:561–73.
- [30] Fennel AJ, Jones AP, Hukins DWL. Migration of the nucleus pulposus within the intervertebral disc during flexion/extension of the spine. *Spine* 1996;21:2753–7.