SPINE Volume 31, Number 6, pp 698–704

Differences in Sitting Postures are Associated With Nonspecific Chronic Low Back Pain Disorders When Patients Are Subclassified

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Study Design. A comparative study.

Objectives. To investigate sitting postures of asymptomatic individuals and nonspecific chronic low back pain (NS-CLBP) patients (pooled and subclassified) and evaluate the importance of subclassification.

Summary of Background. Currently, little evidence exists to support the hypothesis that CLBP patients sit differently from pain-free controls. Although classifying NS-CLBP patients into homogeneous subgroups has been previously emphasized, no attempts have been made to consider such groupings when examining seated posture.

Methods. Three angles (sacral tilt, lower lumbar, and upper lumbar) were measured during "usual" and "slumped" sitting in 33 NS-CLBP patients and 34 asymptomatic subjects using an electromagnetic measurement device. Before testing, NS-CLBP patients were subclassified by two blinded clinicians. Twenty patients were classified with a flexion motor control impairment and 13 with an active extension motor control impairment.

Results. No differences were found between control and NS-CLBP (pooled) patients during usual sitting. In contrast, analyses based on subclassification revealed that patients classified with an active extension pattern sat more lordotic at the symptomatic lower lumbar spine, whereas patients with a flexion pattern sat more kyphotic, when compared with healthy controls ($F = 19.7$; $df_1 = 2$, $df_2 = 63$, $P < 0.001$). Further, NS-CLBP patients had less ability to change their posture when asked to slump from usual sitting ($t = 4.2$, $df = 65$; $P < 0.001$).

Conclusions. Differences in usual sitting posture were only revealed when NS-CLBP patients were subclassified. This highlights the importance of subclassifying NS-CLBP patients.

Key words: sitting, spinal angles, posture, ergonomics, low back pain, subclassification. **Spine 2006;31:698–704**

Low back pain (LBP) is one of the most common and costly musculoskeletal pain syndromes of modern society.[1,2] Despite the large number of pathologic conditions that can give rise to LBP, 85% of cases are without a detected pathoanatomic/radiologic abnormality. This population is classified as having "nonspecific"[3,4] LBP, which often develops into a chronic fluctuating problem with intermittent flares.[5,6]

It has been proposed that the heterogeneous nonspecific chronic low back pain (NS-CLBP) group conceals subgroups of patients.[7–10] The lack of success in defining subgroups of patients has been offered as an explanation for the inability to identify effective treatments for NS-CLBP.[9,11] Consequently, the development of valid classification systems for these patients has been ranked as a top research priority.[8]

For LBP, several classification systems with differing bases have been proposed. A recent review highlights that the multidimensional nature of LBP is not reflected in most classification systems.[12] Further, there is a special need for a mechanism-based classification system acknowledging the bio-psycho-social dimensions of this disorder.[12,13] When the mechanism behind a disorder is known, as long as it is amenable for treatment, treatment of the cause rather than its individual signs and symptoms may be more effective.[14]

A multidimensional mechanism-based classification system has been proposed by O'Sullivan.[15,16] In this classification system, patients with NS-CLBP and clinical signs of motor control impairment can be subgrouped with high reliability by experienced clinicians.[17] It is hypothesized that these patients have a motor control impairment of their lumbar spine that exposes them to repeated stress and strain, thereby providing a basis for ongoing pain. There is growing support in the literature for the presence of motor control impairments in subjects with NS-CLBP, although the nature of the impairment is highly variable[15,18–22] and many mechanisms have been postulated for how pain may alter motor planning.[23,24]

Based on clinical observations, deficiencies in motor control during sitting, which in turn may lead to changes in posture, have been suggested.[7,15,16,21] Consequently, clinicians commonly attempt to improve the sitting posture of LBP patients.

There is conflicting evidence with regards to sitting as a risk factor for LBP, with studies reporting sitting as a major contributing factor[25–27] and other studies finding

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Acknowledgment date: November 4, 2004. First revision date: March 25, 2005. Acceptance date: March 28, 2005.

This study was carried out while the first author (W.D.) was an International Postgraduate Research Scholar in Australia and was supported financially by the Head of School of Physiotherapy Scholarship, Curtin University of Technology, Western Australia.

The manuscript submitted does not contain information about medical device(s)/drug(s).

Federal and Institutional funds were received in support of this work. No benefits in any form have been or will be received from a commercial party related directly or indirectly to the subject of this manuscript.

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no conclusive evidence of increased risk.[28] There is more consensus that sitting commonly exacerbates and perpetuates LBP.[7,16,21,29] It has been reported that LBP patients frequently demonstrate difficulty in adopting a neutral midrange position of the lumbar spine.[30,31] Furthermore, studies have described that during sitting NS-CLBP patients with a flexion pattern disorder position themselves near the end of the available flexion range at the symptomatic region of the spine,[15,16,21,32,33] whereas patients with an active extension pattern disorder hold themselves actively into hyperextension (potentially leading to abnormal tissue strain and pain).[15,16]

Because of the reported link with LBP and the fact that in industrialized countries more of the population acquires a sedentary lifestyle, research examining sitting postures is becoming increasingly relevant. Few quantitative studies have compared sitting posture in LBP patients to asymptomatic controls,[29,34,35] and there is clearly a paucity of studies that quantitatively examine different sitting postures among the LBP population. Therefore, the aim of this study was to compare sitting postures between NS-CLBP patients (pooled and subclassified based on a novel classification system) and asymptomatic controls. The study also aimed to investigate the importance of classifying the NS-CLBP population into homogeneous subgroups.

■ Methods

Participants. Sixty-seven volunteers (33 NS-CLBP patients and 34 controls) were recruited from the Perth metropolitan area. The Human Research Ethics Committee, Curtin University of Technology, approved the study. All subjects provided written informed consent before testing.

Control subjects were excluded from the study if they had a history of LBP or leg pain over the previous 2 years and/or had received previous postural education. NS-CLBP patients were recruited from a private multidisciplinary orthopedic clinic. All patients were assessed by two blinded musculoskeletal physiotherapists (W.D. and P.O.) and subclassified. The assessment was based on a comprehensive subjective and physical examination.[15,16] Patients presenting with clinical signs of motor control impairment were subclassified based on the classification system proposed by O'Sullivan.[15,16] Only patients presenting with a clinical presentation of a flexion pattern (herewith the flexion pattern subgroup) or an active extension pattern (herewith the active extension pattern subgroup) disorder as determined independently by both clinicians were selected for this study. Table 1 presents the strict inclusion/exclusion criteria and a summary of clinical features of these two clinical patterns. The NS-CLBP (pooled) group refers to all LBP patients.

Gender distribution, age, anthropometric data (weight, height, and body mass index), self-reported pain ratings, subjective disability (Revised-Oswestry[36]), and duration of LBP are shown (as mean ± standard deviation) in Table 2.

Experimental Protocol. Each participant sat on a stool with a flat, horizontal surface. The height of the stool was adjusted to ensure that the participants' upper legs were horizontal (line through femoral lateral epicondyle and trochanter major) and the lower legs vertical (line through femoral lateral epicondyle and lateral malleolus). The feet were positioned shoulder width apart with arms hanging relaxed next to the thighs. In both postures, participants viewed a visual target set at eye

Table 1. Inclusion Criteria and Clinical Features and Exclusion Criteria for Flexion Pattern and Active Extension Pattern Classification

Inclusion criteria for nonspecific CLBP with motor control impairment of flexion pattern or active extension pattern
History of chronic (>3 mo) nonspecific (no radiologic diagnosis) LBP with at least moderate disability (Revised Oswestry score >15%)
Pain localized to the lower lumbar spine (L4–L5 or L5–S1) region with minimal radiation
Absence of red flags
Absence of dominant yellow flags
Clear mechanical basis of disorder: specific postural and functional movements that aggravate and ease symptoms; relief of symptoms when reducing the strain to the symptomatic spinal segment in the provocation direction
Associated impairments in the control of the motion segment(s) in the provocative movement direction(s)
Absence of impaired movement of the symptomatic segment in the painful direction of movement or loading (based on clinical joint motion palpation examination)
Clinical diagnosis of a flexion pattern or active extension pattern disorder, both clinicians (independently) agreed upon the diagnosis
Key clinical features flexion pattern
Aggravation of symptoms with movements and postures involving flexion of the lower lumbar spine
Loss of segmental lordosis at symptomatic level, difficulty assuming and/or maintaining neutral lordotic postures with a tendency to drop into flexion
Pain relief with spinal extension
Key clinical features active extension pattern
Aggravation of symptoms with movements and postures involving extension of the lower lumbar spine (commonly reported as a provocative activity is forward bending and sitting, with the key feature here being the tendency to hold the lumbar spine into segmental hyper-extension)
Excess of segmental lordosis at symptomatic level with posture and movements
Difficulty assuming and/or maintaining neutral lordotic postures with a tendency to position themselves into hyperextension
Pain relief with spinal flexion
Exclusion criteria for nonspecific CLBP with motor control impairment of flexion pattern or active extension pattern
Not fulfilling inclusion criteria: low (<15) Revised Oswestry score, signs of neurologic involvement (radicular pain), nonmechanical pain, more generalized pain, evidence of specific diagnosis, *e.g.*, spondylolisthesis, inflammatory disease, no agreement upon the motor control impairment between the two independent clinicians
Presence of red flags
Presence of dominant yellow flags
Previous spine surgery, pregnant at the time of the study or 6 months postpartum, recently undergone a period of motor control rehabilitation

Table 2. Characteristics of Subjects per Group

	No LBP Controls (n = 34)	Flexion Pattern (n = 20)	Active Extension Pattern (n = 13)
Male	18	16	5
Female	16	4	8
Age (yr)	32.0 (12.2)	35.7 (11.2)	39.9 (11.3)
Weight (kg)	68.4 (11.6)	80.1 (10.6)	72.8 (15.7)
Height (m)	1.71 (.09)	1.8 (0.10)	1.70 (0.10)
BMI (kg/m^2)	23.3 (2.9)	24.6 (2.5)	24.2 (2.8)
VAS (average/wk/10)	—	4.1 (1.2)	4.7 (1.1)
VAS (minimum/24/10)	—	1.6 (1.6)	2.7 (1.6)
VAS (maximum/24/10)	—	4.2 (1.9)	5.7 (2.1)
R-OSW (%)	—	36.6 (11.0)	41.2 (14.2)
Duration of LBP (yr)	—	4.9 (5.3)	7.4 (5.3)

BMI = body mass index; VAS = Visual Analogue Scale; R-OSW = Revised Oswestry; LBP = low back pain.
Values are mean (SD). There were no significant differences ($P < 0.05$) between the No LBP and the chronic LBP groups for age, weight, height, and BMI.

level 1.5 m in front of the participants, to standardize the head posture. The two sitting conditions under investigation were usual sitting and slumped sitting. Usual sitting was defined as the sitting posture they would usually adopt during unsupported sitting. Slumped sitting was defined as sitting in an attempt to fully slouch the spine. No other instructions were given to participants.

Data Collection and Analysis. Three-dimensional lumbopelvic data were recorded using 3Space Fastrak (Polhemus Navigation Science Division, Kaiser Aerospace, VT). The Fastrak system is a noninvasive electromagnetic device, which measures the position and orientation of points in space in three dimensions. This apparatus has been shown to be both reliable and valid for measurement of lumbar spine movement with an accuracy of 0.2°.[37] Sensors were placed on the skin over the spinous processes of T12, L3, and S2 using double-sided tape (Norton, Pty Ltd., NSW, Australia) and Fixomull sports tape (Beiersdorf AG, Hamburg, Germany) with the participant in slight spinal flexion to minimize displacement caused by skin movement.

Data were collected at 25 Hz using a customized program in LabVIEW V6.1 (National Instruments). For both usual and slumped sitting postures, three trials of 5 seconds duration were recorded. The three lumbopelvic angles calculated in the sagittal plane are shown in Figure 1 and were defined as follows:

Sacral Tilt. Sacral tilt is the inclination of the sensor at S2 relative to the vertical.[38] A positive angle indicates a posterior sacral tilt.

Lower Lumbar Angle. Lower lumbar angle is the angle between two intersecting lines, one indicating the inclination of the sensor at L3 and the other the inclination of the sensor at S2. A negative lower lumbar angle indicates lumbar lordosis.

Upper Lumbar Angle. Upper lumbar angle is the angle between two intersecting lines, one indicating the inclination of the sensor at T12 and the other the inclination of the sensor at L3. A negative upper lumbar angle indicates lumbar lordosis.

Before processing the raw data, a customized quality control program in conjunction with visual inspection was used to detect and eliminate movement artifact. The abovementioned angles were calculated for the two sitting conditions. The difference between the value of the angle in usual and slumped sitting was also calculated. The intertrial reliability[39] was assessed on all subjects and was excellent. Intraclass correlation coefficient [$ICC_{(3,1)}$] values ranged between 0.85 and 0.99. Standard error of measurement ranged from 5.1° to 0.7°.

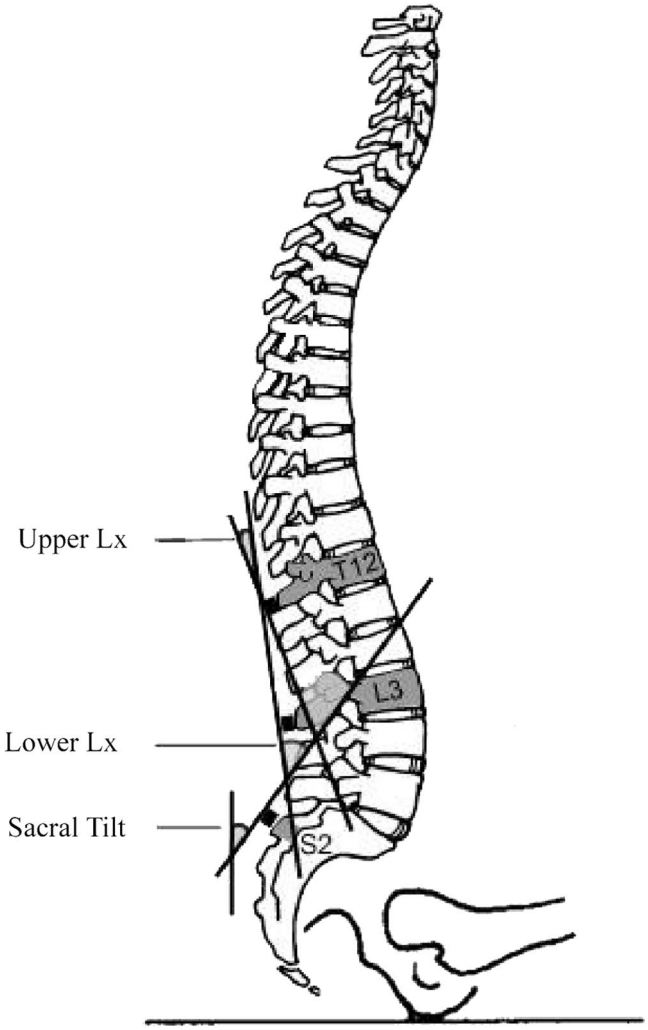

Figure 1. Spinal model used for the calculation of the angles (Lx = lumbar angle).

Statistical Analysis. Postural angles were averaged across the three trials for each subject. Independent *t* tests were used to compare the differences between the No-LBP and NS-CLBP (pooled) groups. Further, a one-way ANOVA with *post hoc* comparisons (Bonferroni) was used to compare the differences between the No-LBP, flexion pattern, and active extension pattern subgroups. SPSS V11.5 (SPSS Chicago, IL) was used to perform all statistical tests and the alpha level was set at 0.05.

■ Results

The lumbosacral angles (mean and standard deviation) are presented in Figure 2 per sitting condition for each group.

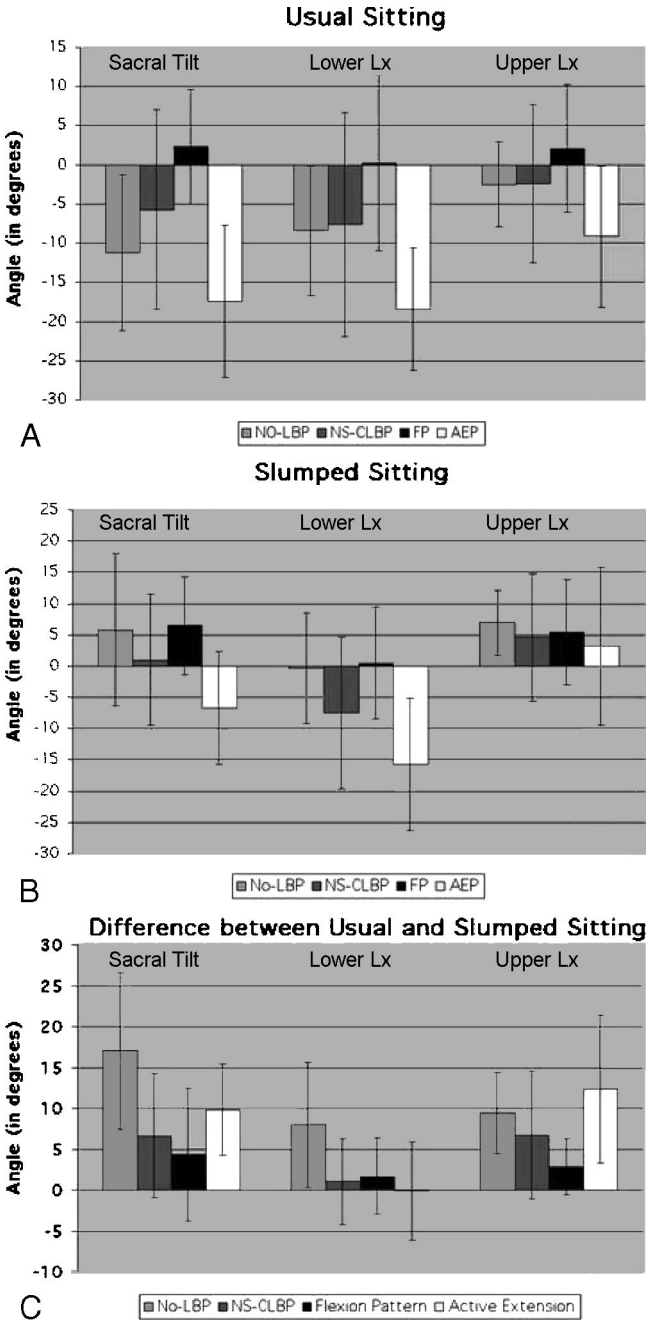

Figure 2. Lumbosacral angles (mean and standard deviation) per sitting condition (Lx = lumbar angle).

No-LBP Versus *Pooled NS-CLBP*

Analyses in usual sitting between No-LBP and NS-CLBP (pooled) groups revealed no significant differences (sacral tilt: $t = -1.95$, $P = 0.06$; lower lumbar angle: $t = -0.31$, $P = 0.75$; upper lumbar angle: $t = -0.02$, $P = 0.99$). A difference was observed between the two groups in the slumped sitting condition for the lower lumbar angle ($t = 2.78$; $P < 0.007$) with NS-CLBP patients sitting with more lordosis. A difference was also observed between the groups for sacral tilt ($t = 4.82$; $P < 0.001$) and lower lumbar angle ($t = 4.29$; $P < 0.001$) based on the change in posture between usual and slumped sitting. The NS-CLBP patients showed less ability to change their lumbopelvic posture when moving from usual to slumped sitting.

No-LBP Versus *Subclassified NS-CLBP*

Analyses between No-LBP subjects and subclassified NS-CLBP patients revealed that the sacral tilt during usual sitting was significantly different ($F_{2,63} = 20.44$; $P < 0.001$) for the active extension pattern subgroup and No-LBP subjects, when compared with the flexion pattern subgroup (who showed a kyphotic posture). There were also significant differences between all three groups for the lower lumbar angle ($F_{2,63} = 19.76$; $P < 0.001$) and for the active extension pattern subgroup compared with the No-LBP subjects and the flexion pattern patients for the upper lumbar angle ($F_{2,63} = 9.86$; $P < 0.001$). Active extension pattern patients sat with more lumbar lordosis than No-LBP subjects, and the flexion pattern patients sat with a more kyphotic lumbar spine.

Analyses of the slumped sitting posture showed that the active extension pattern subgroup presented with more anterior sacral tilt ($F_{2,63} = 9.05$; $P < 0.001$) and a larger lower lumbar angle ($F_{2,63} = 16.31$; $P < 0.001$) compared with the No-LBP subjects and the flexion pattern subgroup. No differences were noted for the upper lumbar angle between the three groups in slumped sitting ($F_{2,63} = 1.03$; $P = 0.36$).

Calculating the change in lumbopelvic posture between usual and slouched sitting revealed less change in sacral tilt ($F_{2,63} = 12.97$; $P < 0.001$) and lower lumbar angle ($F_{2,63} = 9.42$; $P < 0.001$) for the active extension subgroup and flexion pattern subgroup compared with the No-LBP subjects. For the upper lumbar angle, the active extension pattern and No-LBP subgroups showed greater change ($F_{2,63} = 12.52$; $P < 0.001$) between the two sitting postures when compared to the flexion pattern subgroup.

■ Discussion

The rationale for investigating sitting posture within this NS-CLBP population was supported by the fact that all patients in the current study reported aggravation of their LBP with sustained sitting.

As a result of opposing subgroup differences within the pooled NS-CLBP group, only analysis based on subgrouping revealed differences in usual sitting posture between NS-CLBP patients and control subjects. Rose[40] termed this phenomenon the "wash-out effect," wherein the findings in one subgroup of patients is washed out by the opposite findings of patients belonging to another subgroup. A similar pattern is observed in slumped sitting, where the findings from the flexion pattern subgroup are washed out by the hyperlordotic sitting pattern from the active extension pattern subgroup. This clearly highlights the importance of defining specific subgroups and developing a clinically meaningful classification system for the NS-CLBP population.

Flexion Pattern

The identification of a subgroup of NS-CLBP patients that sits with a more kyphotic lumbar spine is supported by field studies reporting an association between flexion related pain and assuming end range flexed postures.[21,32,33] These findings are also consistent with previous investigators who have reported an interaction between LBP and decreased lumbar lordosis. Keegan[35] found the most important factor for the development of LBP with prolonged sitting to be flattening of the lumbar spine, whereas Murphy *et al*[34] showed significant association between flexed postures and self-reported LBP in schoolchildren. Using a lumbar roll that increases lumbar lordosis has been found to decrease LBP.[29]

The loss of lumbar lordosis in sitting, as demonstrated in this study during usual sitting posture in the flexion pattern subgroup, may produce significant mechanical stress.[7] Cadaveric studies have highlighted that the degree of lumbar lordosis effects disc pressure by changing the distribution of load between the disc and zygapophysial joints.[41,42] The lumbar zygapophysial joints have been found to resist none of the compressive load when sitting with a slightly flexed lumbar spine.[41]

Active Extension Pattern

Hyperlordotic sitting and its effect on LBP have not been well investigated. Conflicting evidence emerges from studies examining sagittal plane posture in standing linking discrete postural profiles with LBP. Using a photographic technique, Christie *et al*[43] found that CLBP patients exhibited an increased lumbar lordosis in standing compared with controls. Conversely, Jackson and McManus[44] using standing radiographs found that the total lumbar lordosis was decreased in LBP patients. The conflicting findings in standing may be due to the "wash-out effect" just described.[40]

The findings of this study clearly suggest the presence of a subgroup of NS-CLBP patients (active extension pattern subgroup) with a hyperlordotic sitting strategy. When sitting with an increased lordosis, the forces on the facet joints are supported by the articular surfaces and the capsular ligaments. Using a finite element model, Shirazi-Adl and Drouin[45] reported that the facet joints carry large contact forces in extension, whereas they carry none in small amounts of flexion. Sitting in hyperextension has the potential to induce muscle fatigue and increase loading of the posterior spinal structures *via* compressive forces generated by the extensor muscles.[46] Extension is also associated with a decrease in the space available within the spinal canal and especially the foramens, with potentially a compressive effect on the cauda equina and the nerve roots.

Usual Sitting and the Neutral Zone

The results of the study show that the subgroups tend to sit at opposite ends of the lumbar posture spectrum away from the "neutral zone"[47] with less ability to change their posture at the (symptomatic) lower lumbar spine (Figure 2c) when asked to slump.

The kyphotic sitting posture adopted during usual sitting by the patients with a flexion pattern disorder was within 0.3° of their available voluntary range of motion into flexion in sitting. Patients with an active extension pattern tended to remain in hyperextension, with only 2.7° change in the lower lumbar angle when asked to slump. In contrast, control subjects adopted a more neutral usual posture and moved 8.0° when asked to slump sit. These findings are consistent with previous reports of a loss of neutral repositioning sense in the lumbar spine in CLBP subjects.[31]

These results suggest that flexion pattern and active extension pattern patients may have decreased ability to find an equilibrium around the "neutral zone"[47] and therefore position themselves at the end of a spectrum with minimal change when asked to slump. Tissue strain and, consequently, the risk of tissue irritation and/or damage increases as a function of the rotation away from this elastic equilibrium.[45]

Recommendations for Further Studies and Limitations

Interestingly, during testing NS-CLBP subjects adopted this posture before the onset of LBP as most of the subjects reported pain only after prolonged sitting. This suggests that it is an inherent postural control fault rather than a reflex response to pain. The concept that this represents a motor control impairment that predisposes one to the development of pain is supported by subjects who reported relief from LBP by enhancing lordosis (if they had a flexion pattern classification) or stretching the low back into flexion (if they had an active extension pattern classification). Although these findings lend support to the clinical classification of flexion pattern and active extension pattern as proposed by O'Sullivan,[15,16] clearly further investigations are required. It is acknowledged that in order to further test the hypothesis of a motor control impairment as an underlying mechanism for LBP, a motor learning intervention directed at reducing the flexion (for the flexion pattern subgroup) and extension (for the active extension pattern subgroup) strain at the low lumbar spine during sitting should be trialed. EMG analysis of the low back musculature is also needed to determine the activation patterns used during sitting.

Several potential limitations need to be considered when the results of the current study are interpreted. First, this study reported on very short periods of unsupported static sitting. Studies monitoring lumbopelvic sitting for a longer duration are required. Previous studies have noted that sitting posture depends largely on the task performed.[25] Therefore, the effect of occupational factors should be considered. Further, measurements using external markers tend to overestimate the true angle.[37] However, the measuring system was consistent for both the nonpain and pain groups.

■ Conclusion

If the NS-CLBP patients were pooled into one group, no differences were detected in lumbopelvic posture during usual sitting when compared with controls. In contrast, the authors found differences in lumbopelvic posture with respect to No-LBP *versus* two subgroups with motor control impairment (classified as Flexion Pattern and Active Extension Pattern based on O'Sullivan's classification system).[15,16] This study highlights the heterogeneity of the NS-CLBP population and the importance of subclassification in both clinical and research settings.

Acknowledgments

The authors thank Paul Davey (research assistant) and Dr. Ritu Gupta (statistician) of Curtin University of Technology for their kind assistance throughout this study.

■ Key Points

- The difference in sitting postures (as measured by sagittal plane lumbopelvic angles) was studied on pain-free individuals and nonspecific chronic LBP patients.
- No differences were found during usual sitting when the patients were pooled. Analysis based on subgrouping the patients, using a novel classification system, revealed significant differences in sitting posture between the subgroups of chronic LBP patients and control subjects.
- The current findings highlight the importance of evaluating sitting posture in patients with chronic LBP and the importance of subclassifying chronic LBP patients.

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