Postural recovery following voluntary arm movement is impaired in people with chronic low back pain

Nicola W Mok[1,2], PhD; Sandra G Brauer[1], PhD; Paul W Hodges[1], DSc, MedDr (Neurosci), PhD

1. NHMRC Centre of Clinical Research Excellence in Spinal Pain, Injury and Health and Division of Physiotherapy, The University of Queensland, Brisbane, Australia.

2. Department of Rehabilitation Sciences, The Hong Kong Polytechnic University, Hong Kong SAR.

Address for correspondence:

Dr Paul Hodges

NHMRC Centre of Clinical Research Excellence in Spinal Pain, Injury and Health and School of Health and Rehabilitation Sciences

The University of Queensland,

Brisbane, Qld, 4072 Australia

E-mail: p.hodges@uq.edu.au

Tel: +61 7 3365 4567

Fax: +61 7 3365 4567

Acknowledgments

Abstract

Study Design. Recovery of postural equilibrium following bilateral voluntary arm movement was evaluated using a case-control study, with 13 subjects with chronic LBP and 13 age- and gender-matched control subjects.

Objectives. To evaluate control of the Centre-of-Pressure (COP), as a marker of the quality of control of postural equilibrium associated with voluntary arm movements, in people with and without LBP.

Summary of background data. When healthy individuals perform rapid voluntary arm movements, small spinal movements (preparatory movement) opposite to the direction of the reactive moments precede voluntary arm movements. Evaluation of trunk movement in people with LBP suggests that this strategy is used infrequently in this population and is associated with an increased spinal displacement following arm flexion. As the preparatory spinal movement was also thought to be an anticipatory mechanism limiting postural perturbation caused by arm movements, we hypothesized that LBP subjects would have compromised control of postural equilibrium following arm flexion.

Methods. Subjects performed bilateral voluntary rapid arm flexion while standing on support surface of different dimensions with eyes opened or closed.

Results. Results indicated that people with LBP consistently took longer to recover postural equilibrium and made more postural adjustments in different stance conditions. However, there was no increase in the excursion of the COP during the recovery period in the LBP group.

Conclusion. These data suggest that while COP is tightly controlled during postural recovery, the fine-tuning of the control of postural equilibrium is

compromised in people with LBP. Postural control dysfunctions should be considered in the management of chronic low back pain.

Key Points

- Control of postural equilibrium associated with bilateral voluntary arm flexion was evaluated in 13 study participants with chronic LBP and 13 matched control subjects.

- Subjects with LBP consistently took longer to recover postural equilibrium and made more postural adjustments in different support and visual conditions.

- The inferior quality of postural control may be associated with altered spinal movement control in people with chronic LBP.

Introduction

People with low back pain (LBP) commonly have impaired postural control[1,2]. In particular, LBP subjects have difficulty controlling lumbopelvic movements associated with postural adjustments[3,4]. For instance, it is known that lumbopelvic movement is critical to maintain upright stance when standing on a short base[5] and subjects with LBP frequently lose balance when standing quietly on a short base with no visual input[3]. In trials when they were able to stand on a short base, the LBP group had reduced antero-posterior (AP) shear force which suggests reduced use of AP lumbopelvic movement during quiet stance.

An important element of postural control is appropriate anticipatory control in association with voluntary movement. When healthy individuals perform rapid bilateral arm flexions, the lumbar spine flexes as a result of reactive forces, altered body configuration, and anterior shift of the body's Centre-of-Mass (CoM). This flexion movement has been termed *resultant motion* of the lumbar spine as it is the result of the reactive forces from arm movement[6]. In healthy individuals, this resultant motion is preceded by a small *preparatory* trunk extension, which is in the opposite direction to the resultant motion[4,6]. Although preparatory motion appears to provide an ideal mechanism to limit the perturbation to the trunk, this strategy is used relatively infrequently by people with LBP. Notably, trials with no preparatory spinal movement are associated with increased spinal displacement following arm movement in people with LBP[4]. This compromise in the quality of spinal control may contribute to balance impairment in this population.

Reduced spinal motion prior to the onset of arm movement was thought to be a biomechanical consequence of altered muscle control in people with LBP[4,7]. Increased co-activation of the trunk muscles has been reported in people with LBP

during various motor tasks[8,9], and in association with arm movements when LBP is induced experimentally[10]. Co-activation has been argued to be an adaptation to limit spinal movement and therefore increase spinal stability. This may be a strategy used by the nervous system to protect the spine from further pain and injury or to compensate for impaired proprioception[8]. However, this strategy to reduce spinal movement in people with LBP may adversely affect postural control as multisegmental movement is a critical component of balance.

It has been acknowledged by many authors that movement of the spine is essential to overcome balance disturbance as ankle moment alone is insufficient to maintain equilibrium[11]. With fast support surface translations, movement of the hip/trunk is necessary to adjust the CoM over the new base of support[5]. Even in quiet stance, movement of the spine is ongoing to overcome the challenge to balance imposed by the rhythmic cardiac and respiratory events[12]. Furthermore during arm movements, it is argued that trunk movements contribute not only to the control of perturbation of the trunk but also to assist in the maintenance of the position of the CoM[6,13]. We hypothesized that chronic LBP may be associated with impaired control of Centre-of-Pressure (COP) displacement and whole body equilibrium. The aim of the present study was to test this hypothesis by investigation of the quality of postural recovery following arm movement in people with and without LBP. In the present study, the quality of postural recovery is examined by two characteristics; the time taken to regain postural recovery and the number of postural adjustments during the period of postural recovery following rapid bilateral upper limb movement.

Method

Subjects

Twenty-six subjects participated in the study; 13 in the LBP group with a mean (SD) age of 28.8 (5.3) years and 13 age- (±3 years) and gender-matched control subjects, aged 27.7 (4.2) years. All subjects were on full work duty at the time of testing. Inclusion criteria for the LBP group were: a history of episodic LBP of >18 months duration, at least 1 episode of LBP in the preceding 6 months or pain that was semi-continuous with periods of greater and lesser pain, LBP of musculoskeletal origin, and of a severity that required treatment or sick-leave from work. Subjects were included in the control group if they had no history of significant LBP (defined as an episode that required treatment or sick leave). Subjects with any known sensory or neurological disorders, previous surgery to the spine, unresolved lower limb musculoskeletal pathology, or any condition or medication that could affect balance were excluded from either group. All procedures were approved by the Institutional Medical Research Ethics Committee and were conducted in accordance with the Declaration of Helsinki. All subjects participated in an earlier experiment[4].

Demographic data

Prior to testing, participants completed a Habitual Physical Activity Questionnaire (HPA)[14] and anthropometric measures (height, weight and foot length) were recorded. Subjects in the LBP group completed an additional questionnaire related to their LBP history. Severity of LBP was measured using a 10 cm Visual Analog Scale (VAS) and the Roland Morris Disability Questionnaire (RMS)[15]. The characteristics of subjects are listed in Table 1. T-tests for independent samples showed no difference between groups.

Limb kinematics

Motion of the upper limb was measured with an electromagnetic motion analysis system (Accension, USA). Angular displacement of the arm was recorded

with a single sensor attached to the skin over the mid shaft of shoulder. Data were collected at 100 Hz using MotionMonitor software (Innsport, USA).

Force Plate Measurements

A force platform (9286A, Kistler, USA) was used to detect the ground reaction forces. Data were acquired simultaneously with the limb kinematics using MotionMonitor software (Innsport, USA).

Procedure

In response to an auditory signal, subjects rapidly flexed the arms bilaterally at shoulders to ~60° as fast as possible while standing on either a flat surface (FS) or a short base (SB, anteroposterior dimension - 12 cm). The subject performed 5 individual trials on each support surface. The SB condition was included with the aim to force the use of trunk and hip movement, as ankle torque alone is insufficient to control balance in this condition[5]. Subjects stood relaxed with bare-feet so that the mid-points of the heels were separated by a distance equals to half the foot length and the feet externally rotated up to 15°, and were encouraged to maintain equal weight bearing during the maneuver. In the SB condition, subjects stood on the block across the middle of the soles of the feet. The sequence of the FS and SB was randomized. A 30 s rest period was available between repetitions. An auditory warning preceded the trigger by a random period of 0.5 – 2 s. Three practice trials were provided before data collection. Data were collected at 100 Hz for 2.5 s, from 0.5 s before to 2 s after the auditory trigger for each trial.

Data analysis

Shoulder movement – the time of shoulder movement onset and peak, duration of movement and peak range of movement were identified using the movement trace recorded by the motion analysis system. Peak angular acceleration of shoulder

flexion in the sagittal plane was calculated by twice differentiating the angular displacement data using Matlab 6.0 software (The Mathworks, USA).

COP excursion – ground reaction forces recorded from the force plate were used to calculate the COP range of excursion (max AP position – min AP position). The velocity of the COP (COPV) was calculated from the instantaneous position of COP during the trial using Matlab 6.0 software (The Mathworks, USA). Only the variables in anteroposterior dimension (i.e. COPap and COPVap) were analysed as postural perturbation induced by the arm movement occurred primarily in the sagittal plane.

Time to postural stabilization – the time taken for the COPap velocity to return to a pre-perturbation level was calculated[16]. This was calculated as the time for the rectified COPVap trace to return to a level consistent with the baseline (mean COPVap from 100 ms to 400 ms before onset of shoulder movement plus 2 standard deviations), and remain below this velocity for 30 ms following shoulder movement (Figure 1a).

Number of adjustments – the number of adjustments was recorded as the number of times the COPVap crossed zero (which represents major direction change of the COPap trajectory) in the period from shoulder movement onset until the time to stabilization using the plot of un-rectified COPVap against time (Figure 1b).

Statistical analysis

Force platform measures (baseline COPVap, time to postural recovery, number of postural adjustments made during postural recovery and COPap excursion,) were compared between groups (LBP vs. control), base (FS vs. SB), and visual (eyes open vs. closed) conditions using a linear mixed model and significance was tested using F statistic of the analysis of variance table (ANOVA). Between groups

differences in the ordinal data of the number of postural adjustments were analysed using Wilcoxon's rank sum test. Between-group characteristics including shoulder movement were compared using independent t-tests. SPSS v11.0 was used for all analyses and a p-value of 0.05 was set for significance.

Results

Shoulder movement

When subjects moved the arms forward rapidly, there was no difference in the range ($F_{1,23} = 0.12$, p = 0.74) and peak acceleration ($F_{1,23} = 0.96$, p = 0.34) of shoulder movement between groups (Table 2).

Baseline COPVap

There was no difference in baseline COPVap between the two groups ($F_{1,23} = 0.67$, P = 0.42) (Table 2). However, there was a significant main effect for base (P < 0.01), visual condition (P < 0.02) and an interaction between base and visual condition (P = 0.01). Antero-posterior COP velocity trajectory was increased with reduced base length and when visual input was removed and this was identical for both groups.

Time to stabilization

Comparison of the mean time to stabilization of COPVap following arm movement between the control and the LBP groups indicated that there was a significant main effect for group ($F_{1,23} = 29.8$, P < 0.001). Following voluntary bilateral arm flexion, the average time for COPVap return to pre-movement level was significantly longer in the LBP group (679 ± 45 ms) compared with the controls (513 ± 26 ms). As shown in Figure 2, there was no significant interaction between group

and visual or base condition (all: $P > 0.47$). This indicates that the LBP group consistently took longer for postural recovery after voluntary arm movement.

Number of postural adjustments

The number of postural adjustments during the period between onset of shoulder movement and postural recovery was significantly greater ($F_{1,23} = 13.3$, $P = 0.01$) in the LBP group (5.4 ± 0.7) compared with the controls (3.8 ± 0.2) (Figure 3). Again, no interaction was found between group and either visual or base condition (all: $P = 0.06$). This indicates that people in the LBP group consistently make a greater number of postural adjustments in the period of postural recovery.

Excursion of COP

There was a significant main effect for base ($P = 0.01$), visual condition ($P = 0.02$) and an interaction between base and visual condition ($P = 0.02$). Antero-posterior excursion of COP trajectory was increased with reduced base length and absence of visual input for both groups. However, there was no difference in the range of COPap during the period of postural recovery between the two groups ($F_{1,23} = 0.53$, $P = 0.47$) (Figure 4, Table 2).

Discussion

This is the first study that reveals people with LBP have impaired ability to recover postural stability after internal perturbations induced by arm movement. When compared with their age- and gender-matched pain-free controls, LBP participants took a longer time to regain postural stability and required a greater number of postural adjustments during recovery. It is unlikely that the impaired postural recovery in the LBP group is due to deconditioning as all subjects were

performing full job duties, there was no between-group difference in physical activity level, and the reported pain level in the LBP group was low at the time of testing.

Inferior quality of postural recovery in people with LBP

In the present study, balance performance was examined using COP velocity, and the quality of postural recovery was quantified using two parameters; the time taken for the COPVap to return to the pre-perturbation level and the number of postural adjustments during the period of postural recovery. COPV reflects the speed of movement of the COP, and its timing to resume steadiness has been used as a measure of quality of postural recovery[16,17]. It has been that suggested that increased time taken for postural stabilization is associated with poor postural control[16,18]. For instance, time to regain postural equilibrium is increased in: elderly individuals compared with a younger population[18]; and elderly with known reduced balance performance (versus elderly with better balance performance)[16]. The number of postural adjustments represents major direction changes of the COP around its neutral position which reflects the fine tuning of postural control during the recovery period, and was used to quantify the quality of postural recovery after external postural perturbation[17]. Previous work has shown that this measure of quality of postural recovery is sensitive to changes in postural ability as a result of changed mechanical demands in healthy individuals[17]. It has been shown that postural recovery does not correlate with postural steadiness during quiet stance and should be considered independently when assessing balance performance[18,19]. The present study extends existing findings of balance deficits in LBP (static balance[1-3] and following unexpected external perturbations[2,7,20]. As the perturbation was performed by the individual and therefore predictable, the postural recovery is initiated in a preplanned

manner before the movement is started. The reduced quality of recovery implies that preplanned strategies and later refinement of these strategies (once feedback becomes available) is not ideal in people with chronic LBP.

There are reports of several neuromuscular impairments in people with chronic LBP which might contribute to this finding. First, spinal proprioception is compromised in this population. As proprioception constitutes one of the sensory inputs for regulation of postural stability, defective input may affect the accuracy of postural control. Previous studies reported that LBP subjects are less sensitive to detect rotary motion in both detections[21], make errors with reproduction of a previously presented lumbopelvic angle[22], and consistently tend to undershoot target angles during active repositioning of sacral tilt[23]. If the quality of proprioceptive feedback from the lumbar spine is poor, control of the CoM position might become ineffective when the lumbopelvic movement is involved in postural control (i.e. hip strategy). A hip strategy is more complex and requires calculation of CoM position from interpretation of angle changes at the angle, hip and spine[24]. If spine position is uncertain, this task could be near-impossible. This may explain the reduced tendency for people with LBP to use the hip strategy for balance control.

As ankle torque is generally sufficient to maintain balance in association with minor disturbances, it could be argued that this strategy may be sufficient to control equilibrium in a simple task such as voluntary arm movement when standing on a flat surface. However, lumbopelvic movement has been shown to be an essential component during this task[6]. Notably, lumbopelvic motion is less frequently used by people with LBP: people with LBP used preparatory lumbar movement less frequently and this was associated with increase spinal displacement following voluntary arm movement[4]. Furthermore, the reduced tendency to use shear forces at

the ground (suggesting reduced hip strategy) and the corresponding dominance of ankle torque for postural control in people with LBP accounted for the increased frequency of balance loss when standing on a short base[3]. Thus, in order to maintain balance and spinal control precise control of spinal movement is needed. The tendency of people with LBP to undershoot a previously set position[23] and the reduced sensitivity for spinal motion might compromise the effectiveness of resumption of the pre-perturbation position.

Second, people with LBP have been found to have altered muscle control. Superficial trunk muscle activity is increased in people with LBP during various voluntary tasks[8-10]. Trunk muscle co-contraction has been shown in *in vivo* and modeling studies to increase spinal stiffness[25,26], and has been argued to be an attempt to compensate for insufficiency in the osseoligamentous system or to prevent further pain and/or injury[8]. However, the resultant increase in trunk stiffness would reduce spinal movement, which has been reported in people with chronic or experimental LBP[27,28]. Similar increases in co-contraction have been observed in less-skilled performers and older adults[29] as an attempt to reduce variability of movement[18]. Regardless of the mechanism, the potential advantages of this strategy (prevention of pain and (re)injury, decreased variability) may be offset by the loss of flexibility to use spinal movement for postural control in people with LBP. It is well accepted that control of postural equilibrium is dependent on movement of the hips and spine, particularly when stability is challenged dynamically. Eng et al.[11] and Friedli et al.[13] argued that spinal movement is a crucial element in the control of postural stability if the multi-segmental nature of human body is taken into consideration. Although adjustment of anteroposterior instability would still be possible via movement of the lower limbs, especially hip and ankle[5], previous data from our laboratory show no

evidence of increased compensation by hip during similar tasks[4] and respiration[30] in people with LBP. However, healthy individuals appear to have the capacity to adapt to use other joints when conditions are changed[31]. This suggests that people with LBP have an impaired ability to adapt with changed postural set.

No change in COP excursion

The results showed no difference in the COPap excursion between LBP and the control subjects during postural recovery following voluntary arm movement. This suggests postural steadiness is tightly constrained during the recovery period after voluntary arm movement in people with LBP in order to prevent falling.

Methodological considerations

Several methodological limitations require consideration. First, the case-control methodology is not able to establish a temporal sequence of postural dysfunction and development of chronic LBP. Second, although the sample size was small the sample was sufficient to identify group differences, additional studies are required to establish the generalisability of the results. Third, the time taken for the COPV to return to a pre-perturbation level as a measure of the quality of postural recovery may be influenced by a difference in the baseline variability of COPV between groups. However, no between group difference was found in this study and this can be excluded as an explanation for our results. Fourth, participants in this study were involved in an earlier experiment with similar protocol. As such the observed recovery strategy may vary slightly from a naive cohort, but we do not anticipate that this would compromise the main conclusions of the study as this would be similar for both groups.

Conclusion

During voluntary arm movements postural instability is self-imposed and predictable to the nervous system. Thus, the current data imply compromised organization of preplanned mechanisms of postural control. The inability to achieve timely postural recovery associated with the minor internal perturbation in this task may place the individuals with LBP at risk of overbalancing or falling during situations where rapid recovery is critical. Clinical assessment and treatment of balance are not commonly addressed in the management of LBP. Clinicians should consider the postural stability of patients with LBP during the course of rehabilitation.

References

1.   Byl NN, Sinnott PL. Variations in balance and body sway in middle-aged adults. Subjects with healthy backs compared with subjects with low-back dysfunction. Spine 1991; 16(3): 325-30.

2.   Dvir Z, Daniel-Atrakci R, Mirovski Y. The effect of frontal loading on static and dynamic balance reactions in patients with chronic low back dysfunction. Basic Appl Myol 1997; 7(2): 91-96.

3.   Mok NW, Brauer SG, Hodges PW. Hip strategy for balance control in quiet standing is reduced in people with low back pain. Spine 2004; 29(6): E107-12.

4.   Mok NW, Brauer SG, Hodges PW. Failure to use movement in postural strategies leads to increased spinal displacement in low back pain. Spine 2007; 32(19): E537-43.

5.   Horak FB, Nashner LM. Central programming of postural movements: adaptation to altered support-surface configurations. J Neurophysiol 1986; 55(6): 1369-81.

6.   Hodges P, Cresswell A, Thorstensson A. Preparatory trunk motion accompanies rapid upper limb movement. Exp Brain Res 1999; 124(1): 69-79.

7.   Mok NW, Brauer SG, Hodges PW. Changes in lumbar movement in people with LBP are related to compromised balance. Spine 2011; 16(1): E45-52.

8.   Van Dieen JH, Cholewicki J, Radebold A. Trunk muscle recruitment patterns in patients with low back pain enhance the stability of the lumbar spine. Spine 2003; 28(8): 834-41.

9.   Arendt-Nielsen L, Graven-Nielsen T, Svarrer H, & Svensson P. The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. Pain 1995; 64(2): 231-40.

10.   Hodges PW, Moseley GL, Gabrielsson A, & Gandevia SC. Experimental muscle pain changes feedforward postural responses of the trunk muscles. Exp Brain Res 2003; 151(2): 262-71.

11.   Eng JJ, Winter DA, MacKinnon CD, & Patla AE. Interaction of the reactive moments and centre of mass displacement for postural control during volumtary arm movements. Neurosci Res Commun 1992; 11(2): 73-80.

12.   Hodges PW, Gurfinkel VS, Brumagne S, Smith TC & Cordo PC. Coexistence of stability and mobility in postural control: evidence from postural compensation for respiration. Exp Brain Res 2002; 144(3): 293-302.

13.   Friedli WG, Cohen L, Hallett M, Stanhope S & Simon SR. Postural adjustments associated with rapid voluntary arm movements. II. Biomechanical analysis. J Neurol Neurosurg Psychiatry 1988; 51(2): 232-43.

14.   Baecke JA, Burema J, Frijters JE. A short questionnaire for the measurement of habitual physical activity in epidemiological studies. The American Journal of Clinical Nutrition 1982; 36(5): 936-42.

15.   Roland M, Morris R. A study of the natural history of low-back pain. Part II: development of guidelines for trials of treatment in primary care. Spine 1983; 8(2): 145-50.

16.   Brauer SG, Woollacott M, Shumway-Cook A. The interacting effects of cognitive demand and recovery of postural stability in balance-impaired elderly persons. J Gerontol A Biol Sci Med Sci 2001; 56(8): M489-96.

17.   Huang QM, Hodges PW, Thorstensson A. Postural control of the trunk in response to lateral support surface translations during trunk movement and loading. Exp Brain Res 2001; 141(4): 552-9.

18.   Stelmach GE, Zelaznik HN, Lowe D. The influence of aging and attentional demands on recovery from postural instability. Aging (Milano) 1990; 2(2): 155-61.

19.   Mackey DC, Robinovitch SN. Postural steadiness during quiet stance does not associate with ability to recover balance in older women. Clin Biomech (Bristol, Avon) 2005; 20(8): 776-83.

20.   della Volpe R, Popa T, Ginanneschi F, Spidalieri R, Mazzocchio R & Rossi A. Changes in coordination of postural control during dynamic stance in chronic low back pain patients. Gait Posture 2006; 24(3): 349-55.

21.   Taimela S, Kankaanpaa M, Luoto S. The effect of lumbar fatigue on the ability to sense a change in lumbar position. A controlled study. Spine 1999; 24(13): 1322-7.

22.   Gill KP, Callaghan MJ. The measurement of lumbar proprioception in individuals with and without low back pain. Spine 1998; 23(3): 371-7.

23.   Brumagne S, Cordo P, Lysens R, Verschueren S & Swinnen S. The role of paraspinal muscle spindles in lumbosacral position sense in individuals with and without low back pain. Spine 2000; 25(8): 989-94.

24.   Nashner LM, Shupert CL, Horak FB. Head-trunk movement coordination in the standing posture. Prog Brain Res 1988; 76: 243-51.

25.   Cholewicki J, Juluru K, McGill SM. Intra-abdominal pressure mechanism for stabilizing the lumbar spine. J Biomech 1999; 32(1): 13-7.

26.   Gardner-Morse MG, Stokes IA. Trunk stiffness increases with steady-state effort. J Biomech 2001; 34(4): 457-63.

27.   Kaigle AM, Wessberg P, Hansson TH. Muscular and kinematic behavior of the lumbar spine during flexion-extension. J Spinal Disord 1998; 11(2): 163-74.

28.   Moe-Nilssen R, Ljunggren AE, Torebjork E. Dynamic adjustments of walking behavior dependent on noxious input in experimental low back pain. Pain 1999; 83(3): 477-85.

29.   Manchester D, Woollacott M, Zederbauer-Hylton N, & Marin O. Visual, vestibular and somatosensory contributions to balance control in the older adult. J Gerontol 1989; 44(4): M118-27.

30.   Grimstone SK, Hodges PW. Impaired postural compensation for respiration in people with recurrent low back pain. Exp Brain Res 2003; 151(2): 218-24.

31.   Smith M, Coppieters MW, Hodges PW. Effect of experimentally induced low back pain on postural sway with breathing. Exp Brain Res 2005; 166(1): 109-17.

Tables and Captions

Table 1. Characteristics of back pain and matched control subjects.

	Low Back Pain Subjects (n=13) [mean (SD)]	Matched Controls (n=13) [mean (SD)]	P
Age (yr)	28.8 (5.3)	27.7 (4.2)	0.57
Height (m)	1.74 (0.12)	1.75 (0.06)	0.82
Weight (kg)	75.1 (14.7)	70.4 (12.0)	0.34
Body Mass Index	24.7 (3.5)	22.9 (3.2)	0.20
Habitual Physical Activity			
Work (scale 1-5)	2.5 (0.5)	2.4 (0.3)	0.60
Sports (scale 1-5)	2.7 (1.1)	2.8 (0.6)	0.65
Leisure (scale 1-5)	2.8 (0.7)	3.0 (0.3)	0.18
Duration of Back Pain (yr)	5.0 (5.3)	NA	
VAS score (scale 0-10)	1.9 (1.8)	NA	
Roland Morris Scale (scale 0-24)	2.5 (1.6)	NA	

P values indicate results of independent t test.
NA indicates not applicable.

Table 2. Group data (mean ± SD) of range and acceleration of shoulder flexion, and baseline Centre-of-Pressure Velocity in antero-posterior direction in different stance conditions.

	FS/EO	SB/EO	FS/EC	SB/EC
Shoulder flexion range (º)				
Control	60.0 (16.9)	61.2 (13.7)	62.0 (17.8)	60.7 (13.5)
LBP	62.9 (18.3)	55.3 (13.8)	63.2 (15.0)	59.0 (12.7)
Shoulder acceleration (º/sec²)				
Control	555.0 (125.4)	551.7 (225.9)	488.8 (162.0)	543.9 (208.5)
LBP	501.8 (275.7)	551.4 (270.9)	694.0 (364.8)	668.8 (360.6)
COP Velocity in Antero-posterior direction (mm/sec²)				
Control	1.83 (0.71)	2.30 (1.39)	1.93 (0.61)	3.88 (2.17)
LBP	2.27 (1.98)	3.23 (2.86)	2.78 (2.36)	4.19 (3.12)

FS = flat surface; SB = short base; EO = eyes open; EC = eyes closed. Values are mean (SD).