Changes in lumbar movement in people with LBP are related to compromised balance

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Introduction

Postural control involves coexistence of stability and mobility. The hip and spine articulations are suggested to contribute to maintenance of postural stability even with subtle postural disturbances caused by vital repetitive movements like respiration\textsuperscript{1-3}. In comfortable stance, postural stability is typically maintained by movement of the body in an anterior-posterior direction about at the ankle. When standing on a short base, such as across a beam or when the postural perturbation is large, lumbopelvic movement is increased to assist in maintaining postural stability\textsuperscript{4}. This is referred to as a “hip strategy”\textsuperscript{4}. Both compromised spinal movement and impaired balance control are evident in people with low back pain (LBP)\textsuperscript{5,6}.

Arm movements are preceded by small movements of the spine in the opposite direction to the reactive moments\textsuperscript{7}, which are thought to reduce the effect of the perturbation on balance and spinal control. People with LBP who do not prepare the spine with movement have greater perturbation of the spine following arm movement\textsuperscript{6}. This is associated with delayed postural recovery and use of a greater number of postural adjustments (Mok et al, unpublished observation). In order to determine whether the inter-relationship between spinal motion and balance is a generalisable phenomenon it is necessary to study other postural tasks that involve different mechanisms.

The response to unexpected perturbation provides an alternative method to evaluate this inter-dependence. The response of the trunk muscles to sudden loading either directly to the trunk or via catching a load with the upper limbs has been studied in LBP\textsuperscript{8-12}. Although the response of muscles stretched by the perturbation is delayed in chronic LBP\textsuperscript{8,10}, this is accompanied by delayed offset or increased activation of the shortened muscles on the other side of the trunk\textsuperscript{10,12}. This co-contraction strategy\textsuperscript{10,12,13} suggests an adaptation to increase trunk
stiffness and stability of the spine in LBP, to reduce the possibility of pain and injury. Although co-contraction may have short-term benefit for spine protection, it is likely to compromise the movement required for postural recovery following perturbation. In particular, the time taken and number of postural adjustments needed to regain stability following a perturbation have been shown to change with postural challenge or in those with reduced balance control. We hypothesized that spinal motion will decrease following unexpected trunk perturbation and this would be associated with compromised restoration of postural equilibrium.

This study aimed to investigate the movement response of the lumbar spine and the quality of postural recovery following sudden loading in individuals with and without chronic LBP. A further aim was to investigate the relationship between lumbar movement and the quality of postural recovery.

**Method**

**Subjects**

Eleven healthy subjects and 11 subjects with chronic LBP participated in the study. Subjects were included in the LBP group if they had a history of LBP that required sick leave from their usual occupation or treatment, which lasted for > 18 months with at least 1 episode of LBP in the preceding 6 months or pain that was semi-continuous with periods of greater and lesser pain. Control subjects were included if they had no history of LBP that required treatment or sick leave. Subjects with sensory or neurological disorders, previous spine surgery, unresolved or recurrent lower limb musculoskeletal pathology, condition or medication that could affect balance were excluded from the study. All subjects were on full work duty at the time of testing.
Procedures were approved by the Institutional Medical Research Ethics Committee and were conducted in accordance with the Declaration of Helsinki.

Participants completed a Habitual Physical Activity Questionnaire (HPA)\cite{16} and anthropometric measures were recorded. It has been shown that the HPA has good to excellent test-retest reliability in the chronic LBP population (ICC = 0.90, 0.77 and 0.70 for the work, sports and leisure domain respectively)\cite{17}, and is comparable to that in the healthy population (ICC = 0.88, 0.81 and 0.74 for the work, sports and leisure domain respectively)\cite{16}. T-tests for independent samples showed no difference between groups in the HPA and anthropometric measures (P > 0.05). Subjects in the LBP group completed an additional LBP questionnaire, the Roland Morris Disability Questionnaire (RMS)\cite{18,19}, and a 10 cm Visual Analog Scale (VAS) to indicate the level of pain just prior to testing. Subject demographics are presented in Table 1.

*Trunk kinematics*

An electromagnetic motion analysis system (Accension, USA) measured motion of the lumbar spine and a box into which a weight was dropped (Fig. 1A). Data were collected at 100 Hz and converted to 3D coordinates using MotionMonitor software (Innovative Sports Training, USA). The system has a static accuracy up to 1.8 mm and 0.5° and a resolution of 0.5 mm and 0.1° (Ascension Technology Corporation, USA). The relative reliability (ICC) and absolute reliability (SEM) of the system during quiet stance on a firm surface has been reported to be 0.97 and 0.00456° for the trunk and 0.85 & 0.01512° for the hip during trials with eyes closed\cite{20}.

*Force plate measures*
Ground reaction forces were detected using a force platform (9286A, Kistler, Switzerland). Data were acquired at 100Hz, and simultaneously with the kinematic data. Force plate data were filtered with a 4th order Butterworth filter with a cutoff frequency at 20Hz.

Procedure

Subjects were blind-folded and listened to white noise with headphones to minimise any visual or auditory cues of weight release. Subjects stood relaxed with bare feet with a distance of half the foot length between mid-point of the heels and were encouraged to maintain equal weight bearing while standing on a flat surface (FS) or a short base (SB, a block with anteroposterior dimension of 12 cm across the middle of the feet). The SB condition was used to force the use of trunk and hip movement as ankle torque alone is insufficient to control balance in this context. A sudden anterior load was imposed on the subjects by dropping a 1 kg weight (diameter - 7.5 cm, height – 11cm) from a 30 cm height by release of an electromagnet. Subjects caught the weight in a container (width – 19 cm, length - 19 cm, height - 16 cm) that was held in the hands with their elbow at 90° flexion and upper arms by the side (Fig. 1A). Subjects were instructed to maintain the position of the elbows when the weight hit the container. Five repetitions were performed with rests of ~ 30 s between repetitions. The sequences of the support surfaces were randomized. Three practice trials on each surface were completed before data collection.

Data processing

Movement data – lumbar movement was analyzed as linear and angular displacement in the sagittal plane as loading imposed a sagittal perturbation. Onset of linear displacement was identified from the L1 marker. A lumbopelvic angle was calculated as the angle between lumbar
and pelvic segments (Fig. 1B). Range of lumbar motion was calculated from the maximum and minimum of the angle data (Fig. 1B) using Matlab software (The Mathworks, USA). The onset of lumbar motion was determined using an algorithm that detected the time when at which the angle deviated from the baseline angle (mean of 100 - 400 ms prior to loading) by 3 standard deviations for 30 ms (Fig. 1C). The onset of linear translation of the L1 sensor was identified using the same criteria. Onset of postural perturbation was identified from downward displacement of the marker on the box using similar criteria.

Centre-of-Pressure (COP) excursion – the range of COP excursion (COPE, max– min AP position) was calculated using the ground reaction forces. The velocity of COP (COPV) was calculated by differentiation of COP displacement using Matlab 6.0 software (The Mathworks, USA). Only the variables in anteroposterior direction (i.e. COPEap and COPVap) were analyzed.

Time to postural stabilization – the time taken for the COPV to return to a pre-perturbation level (i.e. time for rectified COPVap to return to 2 SD from baseline [mean COPVap from 100 to 400 ms before onset of loading] for 30 ms, Fig. 2A) was calculated.

Number of postural adjustments during postural recovery – the number of adjustments was recorded as the number of times the COPVap crossed zero (i.e. major direction change of the COP trajectory) from loading to postural stabilization (Fig. 2B).

Statistical analysis

Distribution of the data was tested with one-sample Kolmogorov-Smirnov test, all variables in both groups were normally distributed. A linear mixed model was used to compare movement (mean onset of L1 acceleration, onset and range of lumbar flexion motion) and force plate measures (mean COPE, time to stabilization, number of postural adjustments) between groups (LBP vs control) and base (FS vs SB). Significance was tested using F statistics of the
analysis of variance (ANOVA) table. The Wilcoxon’s rank sum test was used to analyse the between-group differences in ordinal data (number of postural adjustments). When there was a significant interaction, T-tests were used to compare the between group characteristics. Pearson’s r was calculated to test the correlation between the onset of lumbar flexion motion and duration of postural recovery or number of postural adjustments for all trials. SPSS v16.0 was used for all analysis and the significance was set at $p \leq 0.05$.

**Results**

*Lumbar movement*

In response to sudden loading, the spine began to translate anteriorly ~ 160 ms after acceleration of the box due to loading (Table 2). Although the onset of translation was not different between groups ($p = 0.842$), the onset of lumbar flexion was significantly later after load contact ($F_{1,20} = 32.7, p < 0.001$) in the LBP group (187.75 (4.62) ms) compared with the controls (122.11 (2.13) ms) (Table 2). The duration between onset of lumbar translation and the initiation of lumbar flexion was significantly greater in the LBP group compared with the controls ($F_{1,20} = 14.3, p = 0.001$). The latency to the onset of first lumbar motion (translation or flexion) was shorter for controls. There was a significant effect of surface ($F_{1,20} = 7.45, p = 0.013$) and an interaction between surface and group ($F_{1,20} = 8.64, p = 0.008$). Post hoc analysis showed that LBP subjects translated the lumbar spine anteriorly before it flexed whereas control subjects initiated lumbar flexion before anterior translation. When standing on a short base, onset of lumbar flexion was earlier than on a flat surface for the healthy subjects. Furthermore, flexion movement onset was later in the LBP group compared with controls in the SB condition ($p < 0.001$, Fig. 3A). Taken together these data suggest that the spine translates anteriorly for a longer
period without flexing in the LBP group. Although the initiation of lumbar spine flexion was delayed in the LBP group, there was no difference in the total excursion of lumbar movement during the period of postural recovery between groups (P = 0.111, Table 2).

Time to stabilization

Following unexpected loading, the average time for COPVap to return to pre-loading values was significantly longer in the LBP group (761 (194) ms) compared with the control group (460 (123) ms). There was a significant main effect for group for comparison of the mean time to stabilization of COPVap in response to loading between groups (F$_{1,20} = 43.16$, P < 0.001), but no interaction between group and support surface (P = 0.871). LBP subjects took longer to regain postural recovery after unexpected loading regardless of support surface (Fig. 3B).

Number of adjustments

LBP subjects made significantly more adjustments (changes in COPap direction) (6.1 (2.1)) compared with controls (3.9 (1.1)) (F$_{1,20} = 19.702$, P < 0.001). There was no interaction between surface and group conditions (P = 0.367), suggesting that LBP subjects made more postural adjustments during postural recovery (Fig. 3C), regardless of support surface.

Maximum anteroposterior excursion of COP

During the period of postural recovery, there was no difference in the COPEap between groups (P = 0.799, Table 2).

Correlation between postural recovery and onset of lumbar movement

There was a significant positive correlation between the time of onset of lumbar flexion (after loading) and both the time to postural recovery ($r^2 = 0.655$, P < 0.001; Fig. 4A) and number of postural adjustments during recovery ($r^2 = 0.604$, p < 0.001; Fig. 4B) for the SB
condition. This positive correlation suggests that later flexion was associated with longer time to postural recovery and greater number of postural adjustments.

**Discussion**

The results of this study showed no difference in the COP excursion in the anteroposterior direction between the LBP and pain-free subjects. Similar results were shown in individuals with chronic LBP following voluntary arm movement, which suggests that COP excursion is tightly constrained by the CNS during internal\(^{21}\) and external perturbation to prevent falls. However, people with LBP use a different lumbar spine movement strategy following sudden trunk loading to controls, and require a longer recovery period during which more postural adjustments were made to resume postural stability. The results are consistent with previous studies that have shown impaired balance reactions in response to unexpected external perturbation in individuals with chronic LBP\(^{22-26}\). The chronic LBP subjects recruited in this study were all performing full job duties, a comparable level of daily physical activity (as the control subjects) and had a very low level of LBP at the time of testing, and hence the altered postural control was unlikely due to disuse in the chronic LBP subjects.

**Different lumbar movement response following unexpected perturbation**

There was a later onset of the first perturbation to the spine (anterior translation or flexion) after loading in the chronic LBP subjects. The increased time taken for the perturbation to be transmitted to the spine may be explained by a change of stiffness in the upper limb (thus allowing elbow extension for longer periods before transmitting force to spine).
Two aspects of lumbar motion were investigated in this study to reveal the movement strategy in response to unexpected perturbation. Anterior translation measures linear motion of the spine in space and angular displacement measures motion of the lumbar spine relative to the pelvis. The increased period of time between the onset of anterior translation and flexion in the LBP group suggests that when the forces were applied to the trunk in the LBP group the trunk initially translates without flexing, whereas pain-free controls flex the spine early. The delayed flexion after perturbation implies increased stiffness of the trunk thus favouring rotation around the ankle in chronic LBP subjects. The apparent increase in trunk stiffness could be due to either active or passive adaptation in LBP patients.

Earlier work has suggested that people with chronic LBP have limited lumbopelvic movement due to restricted flexibility of the soft tissue. However, there was no difference in the maximum range of lumbar motion during postural recovery between the subjects with and without chronic LBP. Thus other active mechanisms are likely to account for the reduced initial movement of the spine. The most likely explanation for the altered lumbar spine movement pattern in people with chronic LBP is active adaptation. People with chronic LBP have increased activity of superficial trunk muscles during various voluntary tasks, which is likely to increase spinal stiffness. This adaptation of trunk muscle co-contraction has been argued to be an attempt to compensate for the insufficiency in the osseoligamentous system or to protect the spine from further pain and injury, even in the absence of reduced osseoligamentous stability. Delayed onset of lumbar flexion in the initial phase of loading in the LBP group may be a consequence of a trunk muscle co-contraction strategy. An association between the adoption of a trunk stiffening strategy and delayed response of postural control to sudden perturbation in chronic LBP has also been suggested by Henry et al. In that study
people with chronic LBP had reduced COP and increased COM peak displacement in the antero-posterior direction following support surface translation. It was suggested to be a consequence of reduced body movement especially movement around the lumbopelvic region (i.e. “hip strategy”) for maintenance of postural stability\textsuperscript{25}. This hypothesis is further supported by evidence that use of lumbopelvic movement for postural control during quiet standing\textsuperscript{5}, and during voluntary arm movements\textsuperscript{6} is reduced in LBP.

*Stiffening of the lumbar spine is associated with less efficient postural recovery*

Although the total movement of the lumbar spine was not changed in people with LBP, the quality of postural recovery appears to be compromised as revealed by the prolonged recovery time and the greater number of postural adjustments during postural recovery. Notably, the time to onset of lumbar flexion motion after loading was positively correlated with the time to postural recovery and number of postural adjustments. The results concur with the previous studies that have reported the lumbar spine to be important for the control of postural stability\textsuperscript{3,6,7}. It has been shown that movement of the lumbar spine is used for the maintenance of postural stability during quiet standing to compensate for the subtle postural perturbation generated by respiratory movement\textsuperscript{3}, and voluntary arm movement\textsuperscript{7} in healthy subjects. In contrast, people with LBP who use lumbar motion less frequently to prepare for voluntary arm movements\textsuperscript{6} have increased spinal perturbation and less efficient postural recovery\textsuperscript{21}.

*Alternative mechanisms for altered motion of the lumbar spine following perturbation*

Apart from attempts to stiffen and protect the spine from further pain and/or injury, altered lumbar motion could also be a compensation for poor proprioception. Proprioceptive
acuity is critical for recovery of postural equilibrium following perturbation\textsuperscript{39,40}. Proprioceptive deficits at the lumbopelvic region have been reported in chronic LBP\textsuperscript{41-46}. Precise information about the position and motion of the lumbar spine is required by the CNS to calculate the position of the COM, when lumbopelvic movement (e.g. during a “hip strategy”) is utilized as part of the movement strategy for postural control. Inaccuracy in this feedback may force people with chronic LBP to avoid using hip strategy in preferences for rotation around the ankle, which allows simpler calculation of COM position from changes at a single joint. However, this change in strategy has consequences. The hip strategy is more efficient for control of postural equilibrium, especially in situations where the perturbation is fast and large\textsuperscript{4,47}. The result may be that people with chronic LBP are closer to the limit of safety, especially in critical conditions when a rapid postural correction is required. Future research investigating links between spinal proprioception and trunk motion during reactive postural tasks is indicated.

\textit{Methodological considerations}

Whilst this study found differences in postural stability and movement strategies between groups, there are methodological issues that require consideration and direct future research. First, measurement of muscle activity via EMG was not reported and is indicated to determine whether the change in trunk kinematics is associated with changes in the coordination of muscle activity. Second, causality cannot be inferred between the changes in trunk kinematics and the balance parameters. Future work could begin to address this in several ways, such as by modulating trunk control in healthy individuals and determine this effect on the quality of trunk motion and postural stability.
Conclusion

This study has found that the efficiency of postural recovery following unexpected perturbation is compromised in people with chronic LBP compared with healthy counter-parts and that this is associated with poor use of spinal motion as a component of the postural strategy. It is therefore important for clinicians to consider that patients with chronic LBP may demonstrate altered postural stability in addition to other known dysfunction such as altered movement of the lumbar spine. Future research is indicated to further understand the mechanisms underlying alterations in movement and postural stability in this population.
References


