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People with chronic low back pain exhibit decreased variability in the timing of their anticipatory postural adjustments

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Abstract

Variability in the constituents of movement is fundamental to adaptive motor performance. A sustained decrease in the variability of anticipatory postural adjustments (APAs) occurs when performing cued arm raises following acute, experimentally induced low back pain (LBP) [Moseley and Hodges, 2006, *Behavioral Neuroscience*, *120*, 474–476]. This observation implies these changes in variability may also be relevant to people with chronic LBP. To confirm that this reduced variability in the timing of APAs is also evident in people with chronic LBP, we examined the standard deviations of electromyographic onset latencies from the bilateral internal oblique (IO) and erector spinae muscles (relative to deltoid muscle onset) when 10 people with chronic LBP and 10 people without LBP performed 75 trials of rapid arm raises. The participants with LBP exhibited significantly less variability of their IO muscle onset latencies, confirming that the decreased variability of postural coordination that is evident following acutely induced LBP is also evident in people with chronic LBP. Thus, people with chronic LBP may be less capable of adapting their APAs to ensure postural stability during movement.

Keywords

low back pain; anticipatory postural adjustment; posture; variability; EMG

Variability in the kinematic, kinetic, and electromyographic (EMG) components of movement is hypothesized to contribute to motor adaptation in order to preserve performance of the motor endpoint (Davids et al., 2003). Moseley and Hodges (2006) recently reported that, when performing rapid arm raises in response to cues, healthy participants decrease the variability of the ipsilateral external oblique muscle's EMG onset latency relative to that of the deltoid muscle following acute, experimentally induced low back pain (LBP). The authors noted that the activations of the external oblique muscle represent anticipatory postural adjustments (APAs), which are generated in a feed-forward capacity by the central nervous system to stabilize the body against the anticipated perturbing forces generated by limb movements (Massion, 1992). The authors also reported that the reduced variability of the external oblique muscle's onset times depended on the participants' perceived vulnerability to back pain and that the decreased variability persisted several trials after the induced pain was ceased. Based on these observations, the authors then suggested that this decrease in variability represented a change in the central neural control of the postural strategy based on the participants' perceived consequences of performing the movement. Moseley and Hodges (2006) then

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speculated that such a change in central motor control might also contribute to chronic LBP by reducing a person's ability to adapt their APAs to different contexts of movement.

Several questions remain, however, following the report by Moseley and Hodges (2006): (1) can their speculations about chronic LBP, which were based on acutely induced LBP, be validated by demonstrating decreased variability of APA timing from people with chronic LBP, (2) does the decrease in the variability of APA onset latencies generalize to self-initiated voluntary movements, and (3) is this proposed change in the neural control of APA coordination specific to the abdominal muscles ipsilateral to the arm movement? Answers to these questions are necessary to confirm the clinical significance of the study reported by Moseley and Hodges (2006) on cued movements following acute, experimentally induced LBP.

Guided by the hypothesis that people with chronic LBP would exhibit decreased variability in the timing of their APAs when performing self-initiated arm raises, we examined the APA onset latencies of 10 participants with chronic, recurrent LBP and 10 participants without LBP. The participants gave written informed consent to participate in the protocol, which was approved by the Institutional Review Board of the University of Vermont. Participants with and without LBP were differentiated by a history of pain in the lumbar area that impaired at least three activities of daily living for a minimum of 12 months. All participants were active in work, homemaking, or school. Other than LBP, participants did not have a neurological, cardiovascular, or psychiatric disorder, nor did they have a neuromuscular or joint disease, systemic infection, a known problem with alcoholism, tumor or suspected carcinoma, surgery in the previous three months, a history of any back surgery, spinal fracture or dislocation, or structural spinal deformity.

The groups were matched for sex (five females and five males in each group), and were of similar age and body-mass index. The mean (95% confidence interval; CI) age of the group with LBP was 39 (6) years compared to 35 (5) years for the group without LBP [$T = 0.91$, $P = 0.37$]. The mean (95% CI) body-mass index of the group with LBP was 25 (1) kg/m^2 compared to 23 (3) kg/m^2 for the group without LBP [Mann-Whitney $Z = 1.21$, $P = 0.24$]. The participants with LBP were not in an acute symptom flare-up and reported mild levels of pain and disability on the day of testing just prior to performing the protocol: mean (standard deviation) scores on the 0–10 numeric pain rating scale (Childs et al., 2005) equaled 1.78 (0.92) and mean (standard deviation) scores on the modified Oswestry disability questionnaire (Fritz and Irrgang, 2001) equaled 13 (7) % of the maximum possible disability score. Pain was not assessed during or after testing.

Prior to performing the task of rapid arm raises, electrodes were attached for EMG recordings of the participants' APAs. Double-differential surface EMG electrodes were placed 2 cm apart along the length of the contracted muscle belly of the dominant arm's anterior deltoid (DELT; a shoulder muscle activated for raising the arm) as well as bilaterally over the internal oblique muscles (IO; abdominal muscles used as part of the APA to stabilize the trunk when the arm is raised) and erector spinae muscles (ESP; back muscles that are also used as part of the APA to stabilize the trunk when the arm is raised). Electrodes over the IO muscles were placed 2.5 cm medial and 2.5 cm rostral to the anterior-superior iliac spine, and those for the ESP muscles were placed 2.5 cm lateral to the third lumbar segment of the spine. A tri-axial accelerometer (NeuwGhent Technology, Lagrangeville, NY) was also affixed at the dorsal midpoint between the distal end of the ulna and radius of the participants' dominant arm to record the accelerations of the participants' arm raises.

When performing arm raises, the participants sat upright in a stable, adjustable stool with their back unsupported, feet on the floor, and arms at their sides. Joint angles were measured to

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establish 90-degree flexion at the hip and knee as well as zero-degree plantar-/dorsiflexion at the ankle in order to ensure similar postural orientations across the participants. This initial position was intermittently verified during testing by visual inspection and confirmation with a goniometer. The participants were instructed to exhale and relax their abdomen prior to raising their arm in order to (1) prevent APA activity associated with breathing from confounding the APA activity associated with the arm raise, and (2) minimize background EMG activation, which also facilitated the identification of muscle onset times. Background EMG activation was monitored online and, if necessary, the participants were reminded to relax their abdominal or back muscles in order to maintain low levels of background EMG activity.

With the participants' arms initially positioned in approximately zero degrees of both glenohumeral flexion and abduction, they flexed their dominant arm about 90 degrees as fast as they could and then returned the arm to its initial position. The participants initiated their arm raises at a self-initiated pace (without cues or prompting) of approximately once every 10 seconds. Hardware limitations prevented the simultaneous recording of arm accelerations and EMG activations. Therefore, the participants performed 25 trials for recording their arm accelerations, followed by 75 trials for recording EMG onset times, and then another 25 trials for recording arm accelerations. The participants were not made aware of the different reasons for recording each set of trials. The participants rested, at minimum, every 25 trials and were instructed to request a rest when needed in order to prevent fatigue or soreness.

Continuous EMG signals were recorded from a DUO amplifier using Harmonie software (Stellate, Montreal, Canada). Data were sampled at 1000 Hz with 16-bit resolution, amplified by 1000, and bandpass filtered at 10–400 Hz. Input impedance was held under 10 kΩ. Signals from the accelerometer were recorded at 200 Hz with a band-pass filter of 0.07–25 Hz using a direct-current interface box into the DUO amplifier. The data were subsequently reduced and analyzed offline using Matlab software (MathWorks, Inc., Natick, MA). The continuous data were first spliced into 6-s trials, with three seconds before and three seconds after the activation onset of the DELT muscle. Muscle onset times were selected from rectified EMG traces plotted in an interactive graphing program and were defined as the moment when the amplitude of the EMG signal first began to increase 3 standard deviations above the mean baseline activity of the 6-s epoch's first 500 ms. Muscle onset times were selected for each trial, and those of the DELT muscle were subtracted from those of the IO and ESP muscles to generate the APA onset latencies of the IO and ESP muscles. The variability of the participants' APA onset latencies was defined as the standard deviation of each participant's IO and ESP muscle onset latencies. Differences between participants with and without LBP in the standard deviation of the bilateral IO and ESP muscle onset latencies were compared by MANOVA. Pearson's correlation coefficients determined associations among the participants' APA onset variability and their pain and disability scores. The maximum tangential accelerations of the participants' arm raises were determined from the accelerometer signal in the sagittal plane. Differences in average peak arm-raise accelerations between participants with and without LBP were determined by separate two-tailed t-tests for the sets of 25 trials performed before and after the set of 75 trials performed for recording the APA onset latencies.

The results showed that the group with LBP exhibited less variability of their APA onset latencies than the group without LBP [Wilks Lambda $F = 4.80$, $P = 0.011$] (Figure 1). Post-hoc comparisons demonstrated that the decrease in variability was statistically significant for the IO muscles but not for the ESP muscles [$F = 8.66$, $P = 0.009$ for the contralateral IO muscle; $F = 4.65$, $P = 0.045$ for the ipsilateral IO muscle; $F = 1.96$, $P = 0.18$ for the contralateral ESP muscle; $F = 0.51$, $P = 0.48$ for the ipsilateral ESP muscle]. The variability of APA onset times for the participants with LBP was not significantly correlated with their reported levels of pain or disability: Pearson correlation coefficients between APA onset times and numeric pain rating

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scores ranged from 0.17 to 0.45 [P = 0.22–0.66] and those between APA onset times and Oswestry disability scores ranged from 0.03 to 0.15 [P = 0.70–0.94]. Maximum accelerations of the arm raises were similar between the participants with and without LBP: during the first trial-set, the mean (95% CI) peak accelerations of the participants' arm raises equaled 23.5 (5.5) m/s^2 for those with LBP and 20.8 (4.8) m/s^2 for those without LBP [T = 0.73; P = 0.48]; during the last trial-set, 26.3 (5.1) m/s for those with LBP and 29.9 (7.7) m/s^2 for those without LBP [T = 0.76; P = 0.46].

The results, therefore, demonstrate that the variability of APA onset times decreases for people with chronic LBP similar to previous reports on healthy individuals experiencing acute, experimentally induced LBP (Moseley and Hodges, 2006). The results also demonstrate that, with LBP, a decrease in the variability of APA onset latencies generalizes to self-initiated, voluntary movements. The decreased variability was not likely due to biomechanical factors because both groups performed the task with similar initial postural orientations and respiratory states, as well as with similar arm accelerations. Further, the decreased variability of APA onset latencies was not significantly associated with the participants' reported levels of pain or disability on the day of testing, suggesting these variables were not the cause of the altered strategies of postural coordination exhibited by the participants with LBP.

The cause of this decreased variability in postural coordination thus remains unclear. It has been suggested that people with chronic LBP alter their muscle coordination patterns in order to promote spinal stability (van Dieen et al., 2003). In addition, healthy individuals who decrease the variability of their APA onset latencies following acute, experimentally induced LBP report greater vulnerability to LBP (Moseley and Hodges, 2006). Thus, it is feasible that people with chronic LBP decrease the variability of their APAs in an attempt to minimize spinal motion due to anxiety that the movement may induce LBP. These attempts to minimize preparatory spinal motions, however, may actually increase the subsequent spinal motion induced by the movement (Mok et al., 2007). Thus, decreased variability of APA onset latencies may contribute to the persistence of LBP by purportedly impairing the ability of a person with chronic LBP to adapt the timing of the APA according to contextual constraints. This impaired adaptation would then contribute to increased tissue loads and trauma due to poor postural stabilization of the forces induced by movement. Further study is required to confirm the underlying causes of the decreased variability in APA onset latencies exhibited by people with chronic LBP and to determine whether this decrease in APA variability impairs the adaptation of APAs when performing arm raises in changing contexts.

Variability in the motor constituents of a movement task is essential for adaptive motor control. A decrease in variability generates a rigid repertoire of motor strategies with which to accomplish a movement goal under differing contexts (Stergiou et al., 2006). That is, a stable orientation of the trunk or body may be compromised for individuals with chronic LBP exhibiting decreased variability in postural coordination (Brumagne et al., 2008). Thus, rehabilitation of chronic LBP should include movement re-education under varied postural sets in order to restore properly adapted APA patterns to different movement contexts (Stergiou et al., 2006). Psychological interventions may also be required because the diminished variability of postural coordination may associate with beliefs of vulnerability to LBP associated with movement (Moseley and Hodges, 2006).

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REFERENCES

Brumagne S, Janssens L, Knapen S, Claeys K, Suuden-Johanson E. Persons with recurrent low back pain exhibit a rigid postural control strategy. European Spine Journal 2008;17:1177–1184. [PubMed: 18594876]

Childs JD, Piva SR, Fritz JM. Responsiveness of the numeric pain rating scale in patients with low back pain. Spine 2005;30:1331–1334. [PubMed: 15928561]

Davids K, Glazier P, Araujo D, Bartlett R. Movement systems as dynamical systems: the functional role of variability and its implications for sports medicine. Sports Medicine 2003;33:245–260. [PubMed: 12688825]

Fritz JM, Irrgang JJ. A comparison of a modified Oswestry Low Back Pain Disability Questionnaire and the Quebec Back Pain Disability Scale. Physical Therapy 2001;81:776–788. [PubMed: 11175676]

Massion J. Movement, posture and equilibrium: interaction and coordination. Progress in Neurobiology 1992;38:35–56. [PubMed: 1736324]

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:E537–E543. [PubMed: 17762795]

Moseley GL, Hodges PW. Reduced variability of postural strategy prevents normalization of motor changes induced by back pain: a risk factor for chronic trouble? Behavioral Neuroscience 2006;120:474–476. [PubMed: 16719709]

Stergiou N, Harbourne R, Cavanaugh J. Optimal movement variability: a new theoretical perspective for neurologic physical therapy. Journal of Neurologic Physical Therapy 2006;30:120–129. [PubMed: 17029655]

van Dieën JH, Selen LP, Cholewicki J. Trunk muscle activation in low-back pain patients, an analysis of the literature. Journal of Electromyography and Kinesiology 2003;13:333–351. [PubMed: 12832164]

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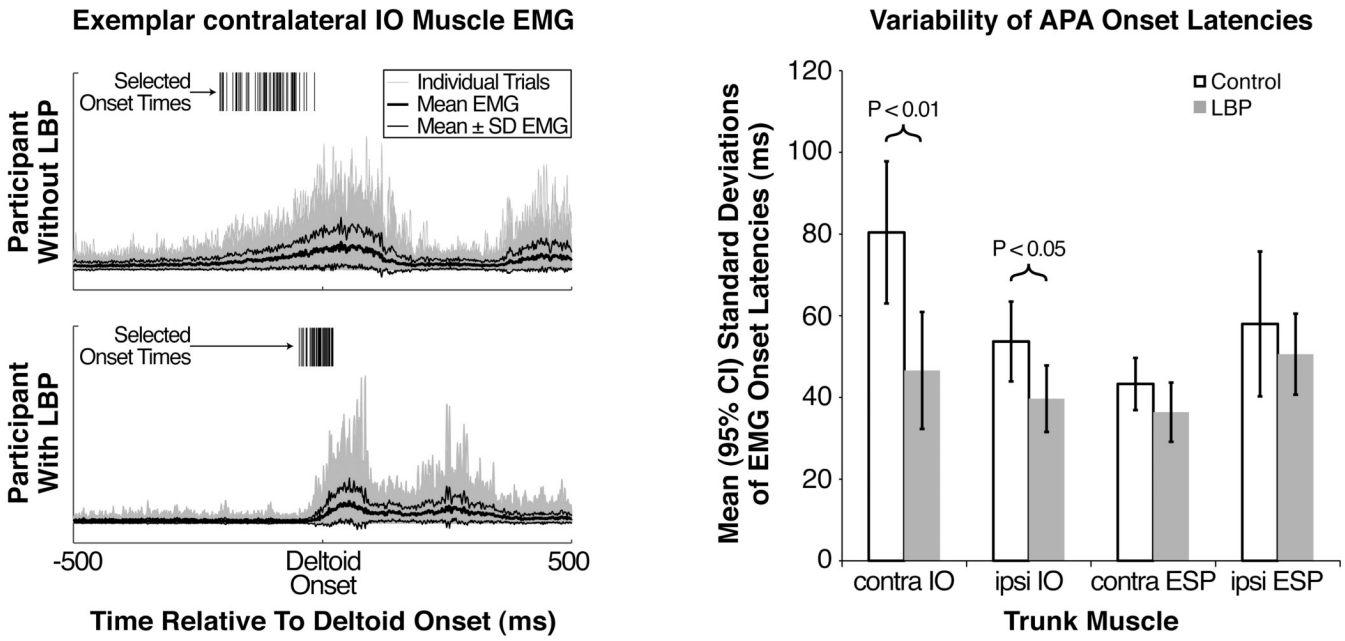

Fig. 1.

Variability of EMG onset latencies for each group. The chart on the left illustrates exemplar rectified EMG traces of the IO muscle contralateral to the arm movement for a participant without LBP (top) and a participant with LBP (bottom). The gray traces represent 75 overlaid trials; the thick black line, the average EMG trace; the thin black lines, one SD above and below the mean EMG trace. The vertical black lines at the top of each chart illustrate the selected onset latencies. The chart on the right illustrates the group means (± 95% CI) of the standard deviations of EMG onset latencies for the IO and ESP muscles contralateral (contra) and ipsilateral (ipsi) to the arm movement for the participants with LBP (gray bars) and those without LBP (open bars).