BRIEF COMMUNICATIONS

Reduced Variability of Postural Strategy Prevents Normalization of Motor Changes Induced by Back Pain: A Risk Factor for Chronic Trouble?

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Variability is fundamental to biological systems and is important in posturomotor learning and control. Pain induces a protective postural strategy, although variability is normally preserved. If variability is lost, does the normal postural strategy return when pain stops? Sixteen subjects performed arm movements during control trials, when the movement evoked back pain and then when it did not. Variability in the postural strategy of the abdominal muscles and pain-related cognitions were evaluated. Only those subjects for whom pain induced a reduction in variability of the postural strategy failed to return to a normal strategy when pain stopped. They were also characterized by their pain-related cognitions. Ongoing perception of threat to the back may exert tighter evaluative control over variability of the postural strategy.

Keywords: motor control, posture, motor learning, pain, cognition

The present study involved 16 healthy, consenting subjects (7 male, 9 female) who had participated in a previous study in which delivery of a painful shock when subjects moved their arm altered the postural strategy of the abdominal muscles, which usually returned to normal when the pain stopped (Moseley & Hodges, 2005). Sitting subjects moved their straight right arm as quickly as possible from neutral position (by their side) to about 60° flexion in response to one light or 60° extension in response to another. Light stimuli were delivered randomly throughout the paradigm. Muscle activity (measured with an electromyogram; EMG) of the shoulder and abdominal muscles was recorded with surface electrodes placed over right anterior and posterior deltoid muscles and shoulder and abdominal muscles was recorded with surface electrodes placed over right anterior and posterior deltoid muscles and right obliquus externus. EMG data were amplified (×2000), band-pass filtered (53 Hz – 1 kHz), sampled (2 kHz), and then analyzed. The onset of the postural activity in the abdominals, relative to the onset of activity in the shoulder muscle, was analyzed with a valid and reliable method that is described in detail elsewhere (Hodges & Bui, 1996). An important point to note is that postural activation of the abdominal muscles in this task can be considered a feed-forward response because it occurs too early to be related to afferent input evoked by arm movement. Such preparatory postural responses are typically seen to limit the impact of limb movements on other body parts (Aruin & Latash, 1995). Because the timing of a postural response is affected by the muscle’s background EMG, we monitored background EMG in real time and cued subjects to relax if we noticed a change in background level.

Twenty movements in each direction (order randomized) were performed as control trials. A cutaneous stimulation to the back (60 Hz, 100-ms train, 1-ms pulse duration, via surface electrodes placed over right posterior superior iliac spine, which does not evoke muscle activity in the abdominal muscles tested) that was rated by the subject as moderately painful was determined. This equates to about 60 mm on a 100-mm visual analogue scale for pain intensity (Moseley, 2001). Subjects were told that they might receive the painful stimulus during the ensuing arm movement...
trials. Seventy pain trials (in which a stimulus was delivered for forward arm movements, but not for backward arm movements, 100 ms after the onset of EMG of the anterior shoulder) and then 70 no-pain trials were undertaken. Subjects for whom the timing of abdominal muscle activation in the last 10 no-pain trials was no different from that in the last 10 pain trials were defined as nonresolvers, and the remaining subjects were defined as resolvers. Standard deviation (SD) of the relative onset of the abdominal muscles estimated variability of the postural strategy of the abdominal muscles, during five batches of trials: (a) the last 10 control trials; (b) the first 10 and (c) last 10 pain trials; and (d) the first 10 and (e) last 10 no-pain trials. Although coefficient of variation is preferable for estimating variability in the magnitude of a signal because it considers the mean of the set of values, SD is preferable for estimating variability in relative time points, such as the onset of one muscle relative to the onset of another, because there is no absolute zero time point. Moreover, SD has been widely used to estimate variability in biological systems (e.g., Christ, Seyffart, & Wehling, 1999). We also evaluated pain-related beliefs and attitudes, which have been shown to change motor output (Moseley, 2004), using the Back Beliefs Questionnaire (Symonds, Burton, Tillotson, & Main, 1996), the Survey of Pain Attitudes (Jensen, Karoly, & Huger, 1987), and the Pain Catastrophizing Scale (Sullivan, Bishop, & Pivik, 1995), which are reliable.

Variability of the postural strategy of the abdominal muscles within each batch of trials was compared between resolvers (n = 13) and nonresolvers (n = 3), using a Kruskal–Wallis nonparametric test. Figure 1 shows that variability was similar during control and the first 10 pain trials (χ² < 2.00, p > .16, for both). However, variability was less for nonresolvers than for resolvers during the last 10 pain trials, the first 10 no-pain trials, and the last 10 no-pain trials (χ² > 6.58, p < .01 for all). Two repeated measures analyses of variance (ANOVA) that compared variability between batches for nonresolvers and resolvers, respectively, were significant, F(1, 12) = 342.5, p < .001, for resolvers and F(1, 2) = 469.8, p = .002, for nonresolvers. For resolvers, variability was greater during the first 10 pain trials and the first 10 no-pain trials than for the other batches (p < .001 for all). Nonresolvers showed a different pattern: Variability was greater during the first 10 pain trials than all other batches (p < .01 for all) and was greater during control trials than the last 10 pain trials and the first 10 and last 10 no-pain trials (p < .04 for all). All subjects reported that pain stopped immediately after the stimulus. Thus, a transient pain stimulus seemed to induce a persistent reduction in variability of postural strategy of the abdominal muscles in the three subjects, for whom the strategy did not return to normal when pain stopped. There was no effect of condition on the magnitude of baseline EMG for 500 ms prior to the postural response for either resolvers or nonresolvers (p > .63 for both), and there was no difference between groups in the intensity of the cutaneous stimulus that was delivered (χ² < 1.09, p > .46). The questionnaire scores did not relate (partial correlations < 0.24, p > .25), which confirms assertions in the literature that they estimate different domains (Turk & Melzack, 2001). Figure 2 shows that the summed questionnaire score was lower for resolvers than for nonresolvers, Wilcoxon’s W = 91.0, P(corrected) = 0.004, which suggests that nonresolvers found back trouble more threatening than resolvers did.

These data support the hypothesis that when pain induces a decrease in the variability of the postural strategy, then normal resolution of the postural strategy does not occur. A secondary finding is that beliefs about back pain delineated those in whom variability was lost from those in whom it was not. A large amount of research evaluates variability of the motor constituents of a task (Latah, Scholz, & Schoner, 2002), but this is a new finding. It is consistent with the idea that the robustness of motor tasks provides the freedom to generate exploratory variation in the task (McCollum & Leen, 1989), persistent stimulation of perception–action systems, and motor learning and coordination (Riccio & Stoffregen, 1991). We propose that, if exploratory variation is lost, then the perception–action system cannot specify functionally relevant alterations in motor output, which prevents adaptation to new perceived demands.

In the present context, new perceived demands may relate to avoidance of pain or injury; it would seem sensible to limit excursion of the painful part, and the postural strategy observed here would probably serve that objective. This possibility cannot be verified because we did not measure pain for each trial or the speed or magnitude of trunk muscle imparted by the arm movement. However, there are several reasons why we think it is likely. First, this type of change in postural strategy is induced by experimental back pain but not by experimental elbow pain (Moseley, Nicholas, & Hodges, 2004a) or by nonpainful stressful stimuli (Moseley, Nicholas, & Hodges, 2004b). Second, the postural strategy induced by experimental back pain in asymptomatic patients...
mimics that observed in back pain patients (Hodges, Moseley, Gabrielsson, & Gandevia, 2003). Third, biomechanical models suggest that this type of change in postural strategy reduces torques about the back, albeit with a concomitant increase in compressive load (Cholewicki, Panjabi, & Khachatryan, 1997), which may increase the risk of long-term trouble (Moseley et al., 2004a).

Why might some people not reestablish normal variability even when the pain has stopped? Relevant here is the secondary finding that nonresolvers were characterized by their beliefs about back trouble. These are the first data that link beliefs to motor variability and learning and suggest that the link may be mediated by evaluative processes, which contribute to motor variability. Evaluative processes are those that relate to the functional consequences of a motor task (e.g., the likelihood of falling) or to the meaning of a motor task (Riccio & Stoffregen, 1991). That is poignant because when a movement becomes painful, the meaning of that movement changes. Thus, perhaps when people who perceive their back to be vulnerable experience back pain, they continue to exert tighter evaluative control over variability, even when the back pain has resolved. That asymptomatic controls demonstrate a protective postural strategy when they expect their back to hurt (Moseley et al., 2004a) and that recurrent back pain patients demonstrate it even when they are pain free (Hodges & Richardson, 1996) seem consistent with that possibility.

These results should be considered in light of methodological limitations. The study was probably underpowered to detect other differences between resolvers and nonresolvers, should they exist, and did not evaluate spatial parameters of the EMG data. However, although the timing of EMG responses is affected by changes in baseline EMG (Hodges & Bui, 1996), we chose a task in which the abdominal muscles tested can remain relaxed, and we monitored background EMG in real time and cued the subject to correct any changes that occurred.

In summary, the findings show that when pain induces a loss of normal variability in the postural strategy, then normal strategy does not return, which is important because nonresolution of the strategy probably increases the likelihood of further back trouble. Loss of variability may be mediated by evaluative processes associated with the meaning of back pain.

References


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