Are the Changes in Postural Control Associated With Low Back Pain Caused by Pain Interference?

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Background: Voluntary limb movements are associated with involuntary and automatic postural adjustments of the trunk muscles. These postural adjustments occur prior to movement and prevent unwanted perturbation of the trunk. In low back pain, postural adjustments of the trunk muscles are altered such that the deep trunk muscles are consistently delayed and the superficial trunk muscles are sometimes augmented. This alteration of postural adjustments may reflect disruption of normal postural control imparted by reduced central nervous system resources available during pain, so-called "pain interference," or reflect adoption of an alternate postural adjustment strategy.

Methods: We aimed to clarify this by recording electromyographic activity of the upper (obliquus externus) and lower (transversus abdominis/obliquus internus) abdominal muscles during voluntary arm movements that were coupled with painful cutaneous stimulation at the low back. If the effect of pain on postural adjustments is caused by pain interference, it should be greatest at the onset of the stimulus, should habituate with repeated exposure, and be absent immediately when the threat of pain is removed. Sixteen patients performed 30 forward movements of the right arm in response to a visual cue (control). Seventy trials were then conducted in which arm movement was coupled with pain ("pain trials") and then a further 70 trials were conducted without the pain stimulus ("no pain trials").

Results: There was a gradual and increasing delay of transversus abdominis/obliquus internus electromyograph and augmentation of obliquus externus during the pain trials, both of which gradually returned to control values during the no pain trials.

Conclusion: The results suggest that altered postural adjustments of the trunk muscles during pain are not caused by pain interference but are likely to reflect development and adoption of an alternate postural adjustment strategy, which may serve to limit the amplitude and velocity of trunk excursion caused by arm movement.

Key Words: low back pain, electromyograph, trunk muscles, pain interference

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During voluntary limb movement, the central nervous system (CNS) coordinates postural adjustments of the trunk muscles in advance of the movement.[1] Such postural adjustments are involuntary and automatic and limit the postural perturbation that is caused by movement of the limb. Pain causes changes in the timing and coordination of these postural adjustments.[2,3] For example, during arm movements, experimentally induced low back pain (LBP) has variable effects on postural activation of superficial abdominal muscles and a consistent effect on postural activation of the deepest abdominal muscle.[2] This consistent effect has also been observed during fear of LBP induced by impending painful cutaneous shock.[4] In each case, the change in postural adjustments was not caused by changes in the way that the task was performed. The alteration in postural adjustments of the trunk muscles may reflect disturbance of normal postural control or adoption of an alternate postural strategy with which to cope with the new demands introduced by pain or fear of pain.

During pain, or fear of pain, CNS performance is often reduced because pain is the CNS process of highest priority. This disruption of CNS performance during pain or fear of pain is called "pain interference."[5] Pain interference has been demonstrated during experimentally induced pain, appears to be dependent on the threat value of the pain, and is absent immediately when the threat of pain is removed.[6–10] We have previously investigated the possibility that compromised CNS resources lead to disrupted postural adjustments using a dual-task paradigm in which arm movements were performed while also performing an attention demanding task that was not painful.[11] That paradigm did not replicate the effect of pain on postural adjustments. However, that finding does not eliminate the possibility that pain disrupts the control of postural adjustments because pain may place a distinct demand by virtue of its immediate relevance to survival.

An alternative explanation for altered postural adjustments during pain is that the CNS adopts an alternate postural strategy with which to cope with the altered demands associated with pain or threat of pain. We have previously observed impaired postural activation of the deep abdominal muscles and facilitation of at least 1 superficial trunk muscle during pain and fear of impending pain.[2,4] Those changes are consistent with a postural strategy that aims to increase trunk stiffness and limit trunk movement. According to the biomechanical characteristics of the trunk muscles,[12,13] adoption of such a strategy would be consistent with the pain adaptation model, which proposes that pain has contrasting effects on muscles around a painful segment so that the velocity and amplitude of movement is reduced.[14]

We aimed to clarify whether the pain-induced changes in postural adjustments of the trunk muscles during voluntary limb movements are caused by disruption of normal postural control or adoption of an alternative postural control strategy. This issue is important because although the changes in trunk muscle activity that are observed during pain may increase spinal stiffness and prevent lumbar spine buckling,[15,16] such a postural strategy is also associated with increased compressive loading of spinal structures.[17,18] Long-term, a pattern of postural adjustments that relies on superficial trunk muscle activity at the expense of deep trunk muscle activity is thought to lead to stimulation of nociceptors in spinal structures, which in turn promotes a vicious cycle of pain and motor dysfunction.[19]

We investigated postural adjustments of the abdominal muscles by recording electromyographic activity (EMG) during voluntary arm movements. The pattern of change in postural adjustments induced by repeated coupling of arm movements with experimentally induced LBP was evaluated. We hypothesized that if alteration in postural adjustments is caused by pain interference, then the changes induced by pain should follow a given pattern: they should be greatest at the onset of pain or fear,[20] habituate with repeated exposure as the threat value of the pain subsides, and be absent as soon as the threat of pain is removed.[6,21] However, if alteration in postural adjustments is caused by an alternate postural control strategy, then the effect should follow a contrasting pattern that becomes less variable with repeated exposure.

METHODS

Patients

Sixteen patients (seven male) with a mean ± standard deviation (SD) age, weight, and height of 24 ± 5 years, 62 ± 10 kg, and 168 ± 13 cm, respectively, participated in the study. Patients were excluded if they had been diagnosed with any respiratory, neurologic, or psychiatric condition or if they had current pain or a history of chronic recurrent LBP. Written informed consent was obtained. All procedures were approved by the institutional research ethics committee and conformed to The Declaration of Helsinki.

Electromyography

For each trial, EMG of the anterior deltoid and the anterolateral abdominal muscles (transversus abdominis [TrA], obliquus internus [OI], and obliquus externus [OE]) were recorded using pairs of surface EMG electrodes (Ag/AgCl discs, 0.5 cm diameter, 1.5 cm interelectrode distance, Grass, UK) (Fig. 1). The lower pair of electrodes (TrA/OI) were placed over the inferior regions of TrA and OI, ~1.5 cm medial to the anterior superior iliac spine. The electrodes were aligned ~20° inferomedially, which is thought to be the direction of TrA muscle fibers in this region.[22] The upper pair of electrodes (EO) were placed ~2 cm inferomedial to the eighth rib and aligned ~40° inferomedially, which has been shown to be the direction of EO muscle fibers in this region.[22,23] A ground electrode was placed over the right iliac crest. EMG data were differentially amplified with a gain of 2000, band-pass filtered between 53 Hz and 1 KHz, and sampled at 2 KHz using a CED 1401 data acquisition system and Signal 2.09 software (Cambridge Electronic Design, Cambridge, UK).

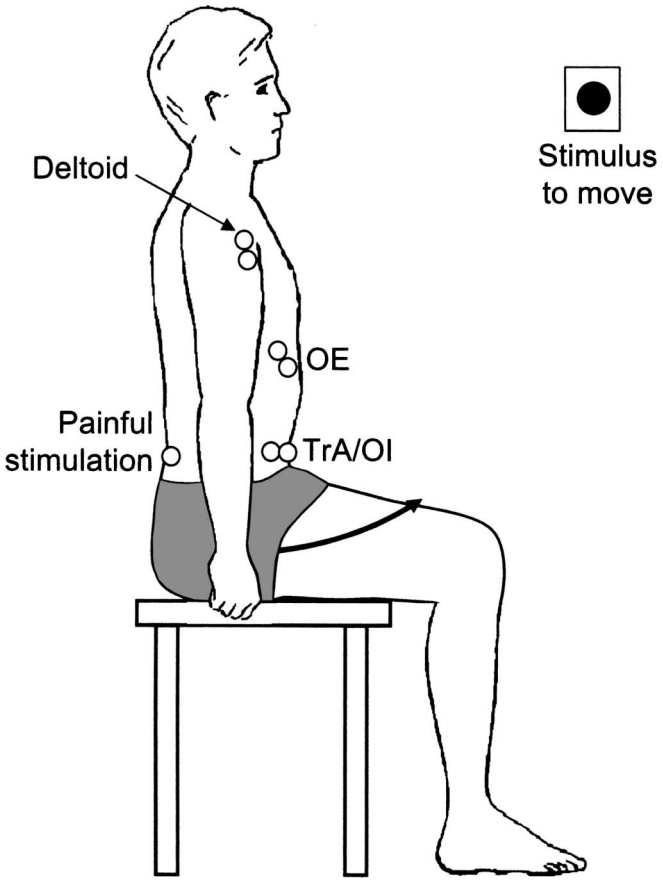

FIGURE 1. Experimental setup. Circles denote surface EMG electrodes for recording EMG from anterior deltoid, obliquus externus (OE), and transversus abdominis/obliquus internus (TrA/OI) and for delivering the painful stimulus (bilaterally posterior superior iliac spines, 60 Hz, 100 msec train, 1 msec pulse duration).

Anxiety

If altered postural adjustments are caused by pain interference, there should be a relationship between change in postural adjustments and anxiety.[21] To investigate this, we measured heart rate (HR) and perceived anxiety. Heart rate data were obtained during each condition from EMG data collected from the electrodes placed over OE. Frames in which 2 heartbeats were captured (~25% of frames) were used to calculate the electrocardiogram (ECG) interval. Perceived anxiety was estimated using an 11-point numerical rating scale (NRS) anchored with "not at all" and "extremely." Immediately after each set of trials, patients were asked "How anxious were you during those trials?"

Pain

Midway through and immediately after the stimulation trials and using an 11-point NRS anchored with "not at all" and "extremely," patients were asked "How painful was the stimulation you received during those trials?"

Determination of the Painful Stimulus

Noxious cutaneous electric stimulation (60 Hz, 100 msec train, 1 msec pulse duration) was used to elicit pain. Surface electrodes (Ag/AgCl discs, 1 cm diameter, 2.5 cm interelectrode distance) were placed over the medial portion of the posterior superior iliac spine bilaterally. The stimulus was then delivered with increasing intensity, and the patient was instructed to indicate when the stimulus became "awful." In previous work, this was equivalent to ~7 cm on a 10 cm visual analog scale.[24] Because motor stimulation would evoke unwanted postural demands, we attempted to minimize motor stimulation by placing the electrodes over bone. If muscle activity was observed in association with the stimulation, the electrodes were relocated and the perceptual threshold and pain stimulus redetermined. Relocation of the electrodes was undertaken in 2 patients.

Investigation of Postural Adjustments of the Abdominal Muscles

Voluntary movements of the right arm in response to a visual stimulus were used to elicit postural adjustments of the abdominal muscles. A light was placed ~1.5 m in front of the sitting subjects, who moved their arm forward as quickly as possible to an elevation of ~40° whenever it was illuminated (Fig. 1). At a random period 0.5 to 2 seconds prior to the visual stimulus, patients received a verbal warning ("ready"). There was a random period (5–15 seconds) between presentations of the stimulus to move. For control trials, 30 movements were recorded, and the final 10 movements were used for analysis ("control"). This strategy aimed to allow the patient to accommodate to the experimental paradigm.

Coupling Arm Movement With Pain—Pain Trials

After control trials, movement of the arm was coupled with painful stimulation of the back. Using Signal 2.09 software (Cambridge Electronic Design, Cambridge, UK), the onset of EMG in anterior deltoid muscle was used to trigger the pain stimulus at a delay of 150 milliseconds. The electromechanical delay between deltoid muscle activation and arm movement was ~50 milliseconds, which meant that the painful stimulus occurred soon after arm movement but well after the onset of postural adjustments of the abdominal muscles. Seventy trials were collected and all trials were analyzed. To detect a pattern of effect consistent with pain interference, such as, maximum effect initially and reduced effect with repeated exposure, the first 10 pain trials and the last 10 pain trials were compared.

Decoupling Arm Movement With Pain—No Pain Trials

Directly proceeding the pain trials, a further 70 trials were conducted in which no painful stimuli were presented. All trials were analyzed.

Data Analysis

Spatial and temporal parameters of the EMG data were analyzed. To minimize bias, EMG traces were displayed individually without reference to experimental condition. For each EMG trace, the onset of EMG was identified visually from the raw data as the point at which EMG increased above the baseline level. This analysis technique is reliable and is preferred to computer-based methods.[25] The reaction time of the EMG response of deltoid and the latency of the contralateral abdominal muscle EMG responses relative to the onset of deltoid EMG (the "onset latency") were used for analysis.

To verify that the arm was moved in a similar manner across experimental conditions, several aspects of the EMG response in deltoid muscle were analyzed. Deltoid mean rectified EMG amplitude was determined for 50 msec epochs from onset of the response to 250 msec after onset. For arm movements during the pain trials, data were not analyzed during the period 150 msec to 250 msec after onset of deltoid EMG due to artifact caused by the pain stimulus. The peak amplitude of the deltoid EMG response and the time to peak deltoid EMG were also compared. Heart rate during each condition was calculated as a proportion of HR during the initial control trials.

Statistical Analysis

Verification of Similar Arm Movement Between Conditions

Repeated measures *t* tests were used to compare group data for: 1) deltoid mean rectified EMG for each 50 msec epoch; 2) deltoid peak rectified EMG; and 3) time from onset to deltoid peak rectified EMG between conditions. T tests were used for this analysis to maximize the likelihood of detecting a difference in deltoid activation and thus performance of the task between conditions.

The Effect of Pain on Anxiety and Postural Adjustments of the Abdominal Muscles

Reported anxiety, HR, onset latency of OI/TrA EMG and OE EMG, the reaction time (RT) of deltoid EMG to the light (ie, RT of the task), and the SD of the response between conditions obtained from the last 10 control trials, the first 10 and last 10 pain trials, and the first 10 and last 10 no pain trials were compared using a series of one-way (variable versus time) analyses of variance (ANOVAs). We expected this selection of trials to be most likely to detect an effect. To further evaluate the pattern of effect on onset latency of the abdominal muscles during pain trials, linear regression analyses between trial number (0–70) and onset latency of TrA/OI EMG and OE EMG were used.

Although multiple measures elevate the probability of a type I error, a Bonferroni correction would elevate the probability of a type II error and reduce significance to $P < 0.006$, which we considered to be too conservative. Because the current work was exploratory in nature, and in light of criticism in the literature of Bonferroni and other corrections,[26] we considered it appropriate to maintain significance at $P < 0.05$.

RESULTS

Anxiety and Pain Measures

Reported anxiety and HR were strongly related ($r = 0.79$, $P < 0.01$). As expected, anxiety and HR were greater during

the first 10 pain trials than during control or the last 10 pain trials ($P < 0.01$) (Fig. 2). The mean ± SD intensity of the pain stimulus was 4.6 ± 1.0 and 4.9 ± 0.9, reported in retrospect midway through and after the pain trials, respectively, which suggests that there was no change in the intensity of the pain stimulus with repeated stimulation.

Spatial and Temporal Parameters of Deltoid Electromyograph

There was no effect of experimental condition on: 1) the RT of deltoid EMG (mean = 241–251 msec, $P = 0.20$); 2) the peak amplitude of deltoid EMG ($P = 0.52$); 3) the time from onset to peak deltoid EMG ($P = 0.78$); and 4) the mean amplitude of deltoid EMG during the 50 msec epochs from onset to 250 msec ($P = 0.6$). Note that during the pain trials, the epoch data for the period 150 to 250 msec after onset were not included in the analysis because of artifact from the pain stimulus (see *Methods*). Taken together, the above results show that activation of deltoid was similar across conditions, which suggests that the arm was moved in a similar manner across conditions.

Electromyograph Activity of the Abdominal Muscles

Figure 3 presents raw EMG data for forward movement of the arm obtained from a single patient during each condition. Note that the onset of TrA/OI EMG relative to deltoid EMG occurred progressively later during the pain trials and then returned to control values during the no pain trials. To show the consistency of the response, Figure 4 presents rectified EMG of TrA/OI obtained from 5 trials from each condition. Figure 5 presents the group latency data. The onset latency of TrA/OI EMG relative to that of deltoid was later during the last 10 pain trials than during control trials, the first 10 pain trials, and during the last 10 no pain trials ($P < 0.01$ for all). A converse response was observed for OE EMG: the onset latency relative to deltoid was earlier during the last 10 pain trials than during the other periods ($P < 0.01$ for all). The standard deviation of the response in OE EMG was greater during the first 10 pain trials and the first 10 no pain trials than during the last 10 trials in each condition or during control trials ($P < 0.05$ for both). During pain trials, trial number was moderately related to onset latency for TrA/OI EMG ($r = 0.47$, $P < 0.01$) and weakly related to onset latency for OE EMG respectively ($r = -0.21$, $P < 0.01$). During the 70 no pain trials, there was a moderate relationship between trial number and onset latency of TrA/OI ($r = -0.38$, $P < 0.01$) but no relationship between trial number and onset latency of OE EMG.

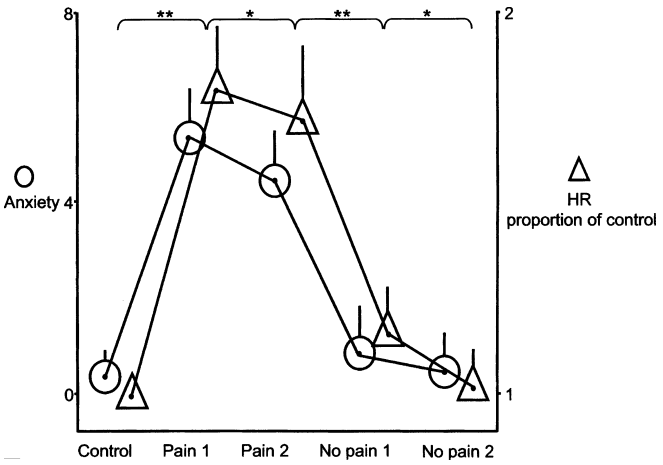

FIGURE 2. Reported anxiety (left y-axis, circles) and heart rate as a proportion of heart rate during control trials (right y-axis, triangles), during the first 10 pain trials (pain 1), the last 10 pain trials (pain 2), the first 10 no pain trials (no pain 1), and the last 10 no pain trials (no pain 2). Difference of both parameters at $P < 0.05$ (*) and $P < 0.01$ (**) is shown.

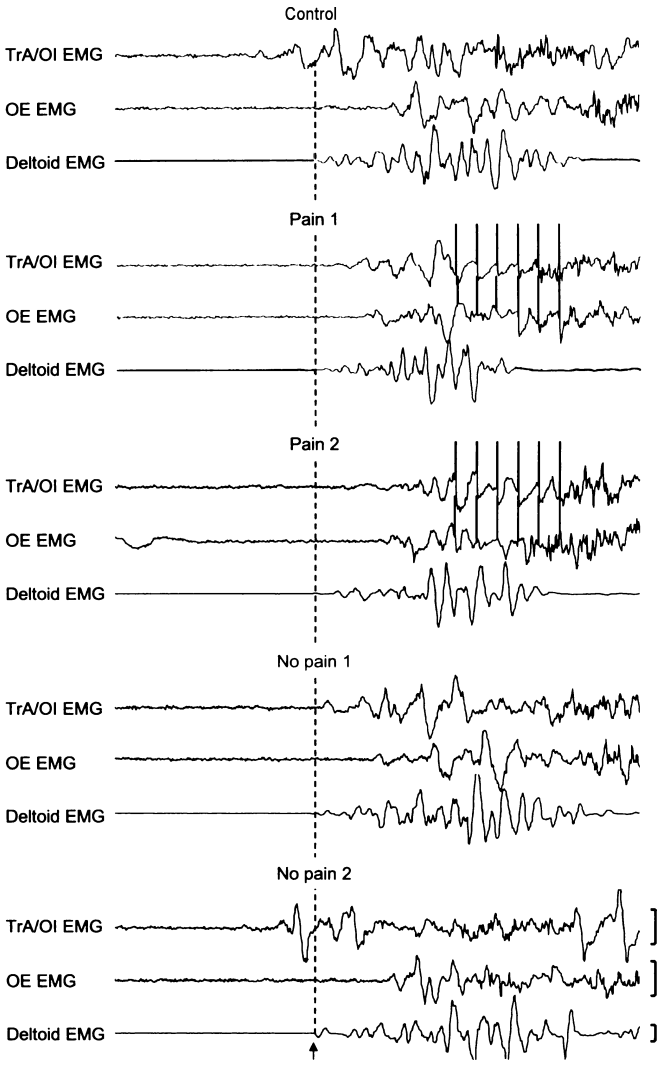

FIGURE 3. Representative raw EMG from TrA/OI and OE during forward movement of the right arm in sitting across conditions. Vertical line denotes onset of anterior deltoid EMG. Note change in onset of TrA/OI EMG and in OE EMG between conditions. Artifact from the painful stimulus can be seen during the pain trials.

DISCUSSION

The results of the current study suggest that when voluntary arm movements are painful, the alterations in

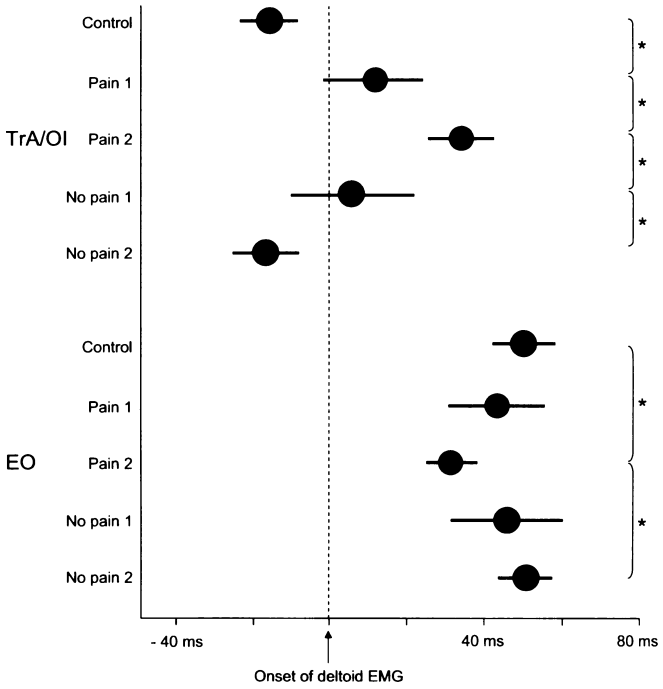

FIGURE 4. Rectified EMG data from TrA/OI for 5 trials in each condition during forward movement of the right arm, obtained from a single sitting patient. Vertical dashed line marks the onset of anterior deltoid EMG. Triangles mark the approximate average onset of TrA/OI EMG for the trials shown. Artifact from the painful stimulus can be seen during pain 1 and pain 2.

postural adjustments of the abdominal muscles do not reflect CNS disruption caused by pain interference but are consistent with adoption of an alternate postural strategy. This interpretation is based on several findings. First, when arm movement was coupled with experimentally induced LBP, changes were small initially but increased with repeated exposure. Repeated performance did not lead to habituation but resulted in a gradual increase in both the delay of postural

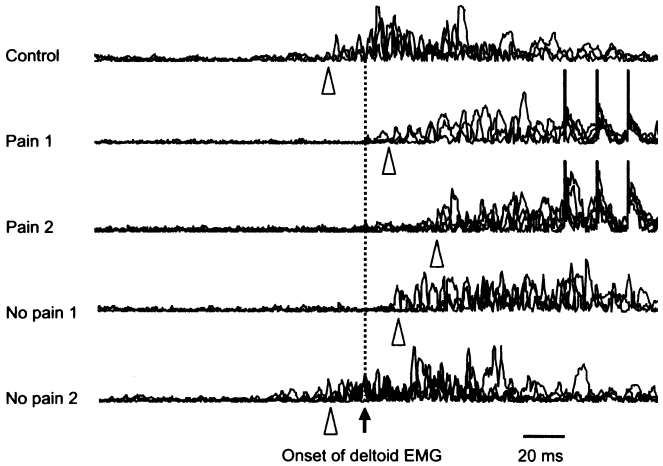

FIGURE 5. Group data for mean (SE) onset of TrA/OI and OE EMG during forward movements of the right arm in sitting. Vertical dashed line marks the onset of deltoid EMG. *$P < 0.01$.

activation of TrA/OI and the augmentation of postural activation of OE. Second, removal of the stimulus did not result in immediate cessation of the effect, but resulted in gradual normalization of the postural response of both TrA/OI and OE. Third, change in anxiety and HR did not reflect change in onset latency of TrA/OI EMG or OE EMG. Finally, the variance of the response was greatest during the first pain trials and first no pain trials, which is consistent with acquisition of a new motor strategy.

The onset latencies of the abdominal muscles observed here are similar to previous results obtained from standing patients[4,27,28] and reflect preparatory (feedforward) postural commands. Importantly, the effect of pain on postural activation of TrA/OI in the present study (~30 milliseconds delay, relative to deltoid) is consistent with previous work in which LBP was induced by intramuscular injection of hypertonic saline (~30 msec delay in TrA EMG),[2] and fear of LBP was induced by unpredictable painful cutaneous shock (~20 msec delay in TrA EMG).[4] Those studies found augmentation of OE in some patients but not others, whereas the present results show that during painful movement, postural activation of OE was augmented across the group. This discrepancy probably reflects differences in the nature of the stimuli used and the timing and context of its delivery in each situation.

The results of the present study suggest that coupling a particular movement with pain leads to development and adoption of an alternate postural strategy. There are 2 aspects of the data that support this possibility. First, the timing of TrA/OI EMG onset was correlated with trial number, and the change in timing of TrA/OI activation was greatest in the last 10 pain trials. Second, the SD of the onset latency of TrA/OI EMG was larger in the first 10 trials in each condition than during the last 10 trials, which suggests reduced variability of the response with repeated performance, a finding consistent with refinement of a motor skill. The short duration over which the SD reduced raises the possibility that altered postural strategies may quickly become robust after acute injury, a possibility that has been raised previously within the context of guarded movements.[29]

The alternate postural strategy observed during pain in the current study is consistent with the pain adaptation model.[14] Because the superficial trunk muscles including OE are well suited to limit trunk motion, increase spinal stiffness, and prevent lumbar spine buckling,[15,16] augmentation of OE activation during painful arm movements may serve to limit the velocity and amplitude of trunk excursion caused by the arm movement. Delay in TrA/OI activation may reflect relevant redundancy of the deep trunk muscles in the presence of increased compression imparted by superficial trunk muscle activity. Although the pain adaptation model is well established for voluntary movement of painful segment,[30,31] the present work offers new evidence of the pain adaptation model applying to involuntary automatic postural adjustments associated with voluntary movements of a nonpainful segment. Although the mechanisms that underlie a pain adaptation effect on involuntary postural adjustments are not clear, our results raise implications for consideration of spinal pain conditions.

First, according to current theories of spinal mechanics, changes in postural adjustments such as those observed here, if

sustained long term, may pose a risk to spinal structures.[32] This suggests that in the presence of back pain, even movements of a nonpainful limb may contribute to secondary problems by virtue of sustained alterations in postural adjustment strategy. Second, the alteration in postural adjustments may be sustained by the threat of pain such that secondary problems may occur even after pain has resolved. This would support preliminary evidence that normal trunk muscle control does not always return when symptoms subside.[33] As such, it may be necessary to actively relearn appropriate postural adjustment strategies, either through practice or targeted motor control training. Finally, the data suggest that belief about impending pain may be sufficient to alter postural adjustments even when the impending pain is not anxiety provoking, which implies that beliefs about pain and injury may be etiologic in secondary and recurrent problems. These issues are currently under investigation.

The present findings should be considered in light of several limitations. One criticism may be that changes in postural activation actually represent part of a movement response away from the pain stimulus. This possibility was minimized by coupling the painful stimulus to activation of the prime mover but offsetting it by 150 msec, which meant that the painful stimulus occurred ~100 msec after the initiation of arm movement. In every patient, onset of TrA/OI EMG always occurred >60 msec prior to the painful stimulus. Second, the clinical relevance of the current work may be limited because cutaneous shock was used to induce pain. This type of pain poorly simulates nonexperimental pain and imparts distinct neurophysiologic effects. However, the experimental question required short-lasting pain of known onset, which meant other more clinically accurate strategies such as intramuscular injection of algesic chemicals were not appropriate. Moreover, this limitation would decrease the power of the design and would have been of greater consequence if no effects were observed.

A final limitation is introduced by the use of surface EMG electrodes to record abdominal muscle activity. Selective recording of EMG from the deep abdominal muscles requires intramuscular electrodes.[28] In the current study, although the TrA/OI electrodes were aligned with TrA muscle fibers in this region,[22] the recorded EMG reflects muscle activity in TrA and OI. This is not a major problem because the impetus for, and significance of, the current work relates to the pattern of changes in postural control. Nevertheless, corroboration of the current findings using selective EMG would clarify the involvement of individual muscles. Evaluation of EMG of other trunk muscles would also be helpful in this regard.

CONCLUSION

The results of the current study suggest that changes in postural adjustments of the abdominal muscles associated with LBP do not reflect compromise of CNS performance caused by pain interference. With repeated exposure, the changes in postural activation of TrA/OI and OE become larger and less variable. The changes probably reflect an alteration in postural strategy, which is consistent with the pain adaptation model and may serve to limit the amplitude and velocity of trunk excursion caused by limb movement. These findings are important because, long term, such postural changes may pose a risk to spinal structures and lead to chronic problems.

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