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RESEARCH ARTICLE

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Experimental muscle pain changes feedforward postural responses of the trunk muscles

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Abstract Many studies have identified changes in trunk muscle recruitment in clinical low back pain (LBP). However, due to the heterogeneity of the LBP population these changes have been variable and it has been impossible to identify a cause-effect relationship. Several studies have identified a consistent change in the feedforward postural response of transversus abdominis (TrA), the deepest abdominal muscle, in association with arm movements in chronic LBP. This study aimed to determine whether the feedforward recruitment of the trunk muscles in a postural task could be altered by acute experimentally induced LBP. Electromyographic (EMG) recordings of the abdominal and paraspinal muscles were made during arm movements in a control trial, following the injection of isotonic (non-painful) and hypertonic (painful) saline into the longissimus muscle at L4, and during a 1-h follow-up. Movements included rapid arm flexion in response to a light and repetitive arm flexion-extension. Temporal and spatial EMG parameters were measured. The onset and amplitude of EMG of most muscles was changed in a variable manner during the period of experimentally induced pain. However, across movement trials and subjects the activation of TrA was consistently reduced in amplitude or delayed. Analyses in the time and frequency domain were used to confirm these findings. The results suggest that acute experimentally induced pain may affect feedforward postural activity of the trunk muscles. Although the response was variable, pain produced differential changes in the motor control of the trunk muscles, with consistent impairment of TrA activity.

Keywords Motor control · Experimental pain · Postural control · Trunk muscles · Stability

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Introduction

Deficits in motor control in low back pain (LBP) are reported frequently in the literature. Changes range from delayed reaction time for finger movements (Luoto et al. 1995) to delayed trunk muscle recruitment with predictable (Hodges and Richardson 1996, 1999) and non-predictable challenges (King et al. 1988; Wilder et al. 1996) to spinal stability. On the basis of this data, emphasis has been placed on motor control training in LBP rehabilitation (O'Sullivan et al. 1997; Mannion et al. 1999; Richardson et al. 1999). The rationale for these techniques is to restore control of spinal motion and normal proprioceptive input to the central nervous system (CNS). Despite the success of these techniques in the management of LBP (Hides et al. 1996; O'Sullivan et al. 1997) and neck pain (Jull et al. 2000), fundamental questions remain unanswered. It has not been determined whether the abnormalities in motor control precede or follow the onset of pain and the mechanism of such abnormalities is not known.

Control of spinal motion and stability is dependent on the integrated contribution of passive elements and surrounding muscles. However, of critical importance is the control system, which detects the status of stability, and plans trunk muscle activity, in advance of, or in response to, perturbations to the spine (Panjabi 1992). According to this model, any alteration of the recruitment

strategy for spinal control may lead to microtrauma and subsequent nociceptor stimulation and pain (Panjabi 1992). Moreover, the control system may adapt to increase the muscular control of the spine. Consistent with this hypothesis, studies of patients with LBP have identified delayed recruitment of the abdominal muscles, including transversus abdominis (TrA), in association with limb movements that challenge the stability of the spine (Hodges and Richardson 1996, 1998). If TrA contributes to spinal stability then such changes may reduce the effectiveness of the spinal stability mechanism. In contrast, other studies have reported augmentation of trunk muscle activity such as delayed relaxation of obliquus externus abdominis (OE) and the erector spinae (ES) following the sudden unloading of the trunk (Radebold et al. 2000) in LBP. However, interpretation of these findings is difficult due to the heterogeneous nature of clinical spinal pain and the difficulty of extracting cause and effect. One approach, which may provide insight into postural mechanisms associated with pain, comes from investigations of the effect of experimentally induced pain on the motor control of the trunk muscles.

Numerous studies have evaluated the effect of pain elicited by injection of hypertonic saline into muscles of the limbs and trunk. Hypertonic saline is used because it is considered to simulate clinical pain (Kellgren 1938). Although hypertonic saline injection is unlikely to simulate all aspects of mechanical LBP, it provides a model to investigate the effect of nociceptor stimulation and pain (in the absence of mechanical disruption of spinal structures or inflammatory changes). While numerous studies have investigated the effect of nociceptor stimulation and pain on motoneuron excitability and reflex responses (Matre et al. 1998), studies of postural responses of the trunk muscles are likely to provide data about the effects of pain on more complex features of planning of motor responses. Such postural responses may include those that prepare the spine for the reactive moments from limb movements. It was hypothesized that the changes in motor control of the abdominal muscles that have been identified in people with clinical LBP may be reproduced by nociceptor stimulation and pain induced by intramuscular injection of hypertonic saline.

The aims of this study were to determine: (1) whether trunk muscle recruitment in a postural task could be altered by experimentally induced pain, (2) whether there is a differential effect of pain on individual trunk muscles, (3) whether such changes, if present, are consistent with those identified in clinical pain, and (4) the time course of motor control changes.

Materials and methods

Subjects

Seven healthy volunteers participated in the experiment (five male, two female). The mean (SD) age, height and weight of the subjects were 28.6 (3.6) years, 1.74 (0.05) m and 69 (11) kg, respectively. Subjects were excluded if they had any respiratory or neurological condition, or any history of LBP that had limited function. All procedures were approved by the Institutional Ethics Committee.

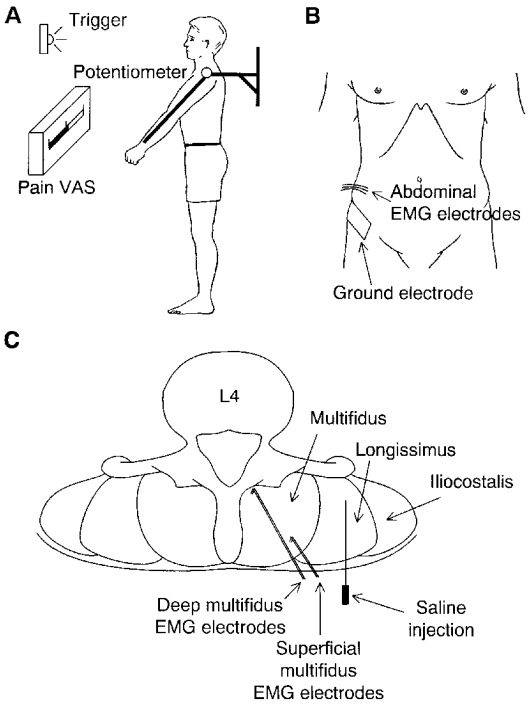

Fig. 1A–C Methods. **A** Experimental setup. Subjects moved the left arm in response to a light. After saline injection, subjects scored the pain on an electronic visual analogue scale (*VAS*), using a knob situated at their right hand. The knob was attached to a potentiometer, which progressively illuminated the VAS. **B** Site for insertion of intramuscular electrodes into the abdominal muscles. The ground electrode was placed over the right superior iliac spine. **C** Placement of intramuscular electrodes into multifidus adjacent to the L4 vertebra, such that the deep multifidus electrode recorded from those fibers adjacent to the lamina and the superficial multifidus electrode recorded from those fibers ~1 cm from the superior border. Saline was injected into the longissimus at a depth of ~35 mm

Electromyography activity

Electromyography (EMG) of the right abdominal and multifidus muscles was recorded with bipolar intramuscular electrodes made from Teflon-coated stainless-steel wire (75 μm). Teflon was removed from the ends (~1 mm), the tips were bent back ~1 and 2 mm to form a hook and the wires were threaded into a hypodermic needle (0.32×0.50 mm/0.32×0.50 mm). EMG recordings were made only from the opposite side of the body for several reasons: to reduce the potential for movement artifact from the moving limb, to allow direct comparison with previous data of patients with clinical LBP, and to minimize the number of invasive recordings. Electrodes were inserted into TrA, OE and obliquus internus abdominis (OI) with ultrasound guidance (7 MHz transducer: Acuson, USA) between the anterior superior iliac spine and the ribcage (Fig. 1A). Electrodes were inserted into multifidus at the L4 level with ultrasound. One electrode was inserted ~40 mm from the midline and directed to the lamina to record from the deep fibers (DM) (Fig. 1B). Another electrode was inserted at the same site to a depth of ~10 mm, medial to the lateral border of multifidus, to record from the superficial fibers (SM) (Fig. 1B). Before

electrode insertion, ~0.5 ml of lignocaine (lidocaine 1%) was injected under the skin.

Pairs of Ag/AgCl surface EMG electrodes (20 mm interelectrode distance) were placed over the left anterior and posterior deltoid muscles and either side of the site for injection of saline into ES. A ground electrode was placed over the iliac crest. EMG data were preamplified and further amplified with a total gain of 1000×, band-pass filtered between 20 and 1000 Hz and sampled at 2 kHz using Spike2 (CED, UK). Data were exported for analysis with Matlab 5.1 (Mathworks, USA).

Arm movement

Angular displacement of the left arm was measured using a potentiometer attached to a bar, which was strapped to the left wrist (Fig. 1C), and the axis aligned with the glenohumeral joint. Movement data were sampled at 1000 Hz.

Pain measurement

In trials with injection of saline, pain intensity was rated by the subject by turning a knob on a potentiometer that was connected to a 100-mm VAS (Visual Analogue Scale) anchored with "no pain" and "worst possible pain." Because pain is subjective, it has been argued that patients' self-reports provide the most valid measure of the experience (Katz and Melzack 1999). Numerous studies have confirmed the reliability and validity of the VAS in acute pain contexts (Bijur et al. 2001; Gallagher et al. 2002), and measurement of VAS has been used extensively in the literature to measure pain intensity in studies of experimental pain (e.g., Svensson et al. 1995; Arendt-Nielsen et al. 1996; Graven-Nielsen et al. 1997). Subjects completed a McGill Pain Questionnaire (MPQ) at the completion of the arm movement trials in order to evaluate the qualities of the pain experienced.

Procedure

Subjects performed a 'set' of single and repetitive arm movements (see description below of arm movement conditions) in four conditions: (1) control trials prior to saline injection, (2) after injection of isotonic saline (0.9%), (3) after injection of hypertonic (5%) saline that achieved a VAS of $\geq$4/10, and (4) at 10-min intervals during a follow-up period of 1–2 h after the hypertonic saline injection. Studies commenced with a 'set' of control single and repetitive movement trials. This was followed by 'sets' of movements after an injection of isotonic saline. This was used to confirm that any change in trunk muscle activity induced by hypertonic saline injection was due to pain rather than the mechanical effects of the injection. While the isotonic saline injection always preceded that of hypertonic saline, subjects were not aware which injection they were to receive. They were told that two would be given, and that one, both, or neither might be painful. Information of the order of injections was withheld from the subjects to control for factors associated with their expectation of the effects of the injection. Both injections were aimed at the lateral portion of the longissimus muscle and were performed after ultrasound visualization of the morphology of the muscle. The site was ~60 mm lateral to L4 (Fig. 1B) and at a depth of ~35 mm. Prior to the saline injections, 0.5 ml of lignocaine (lidocaine 1%) was injected subcutaneously to minimize cutaneous sensations. For isotonic trials a 1.5-ml bolus was injected over ~10 s. Subjects indicated pain on the VAS and arm movements commenced after the pain (if reported) had stabilized (~60 s). Hypertonic saline (5%, 1.5 ml) was injected at the same site ~15 min after the isotonic saline injection. If a subject reported a VAS of <4 after 2 min, further injections were given with 0.5-ml increments. Arm movements commenced once pain was stable for ~30 s. If pain fell below 4/10, additional 0.5-ml injections were given. In the follow-up period, 'sets' of single arm movements were repeated at 10-min intervals for 1 h. No repetitive arm movement data were collected in the follow-up period.

For trials of single arm movement, subjects stood relaxed with their arms beside the body and the left arm was moved rapidly in response to a light (Fig. 1C) that followed a verbal warning by 0.5–2 s. Our preliminary data and previous studies indicate that similar results were obtained for movements of the right and left arm, when contralateral EMG data are recorded (Hodges and Richardson 1997; Hodges et al. 2000). Subjects were instructed to remain relaxed until the stimulus. Investigators observed EMG activity prior to the trial and provided feedback to relax their standing posture if background activity was detected. This was necessary to ensure that background activity was consistent between experimental conditions and to facilitate the identification of EMG onsets. Frequent instruction to relax was only required for two subjects. Subjects were instructed to maintain equal weight bearing during the trial. Each set of single arm movements involved 15 repetitions of arm flexion to ~60° with the emphasis on speed rather than distance moved. Trials were rejected if the onset of deltoid EMG occurred less than 100 ms or greater than 200 ms after the light (<5% of trials) as trials earlier than this have a reaction time that indicates the subject preempted the light and later trials indicate that the subject failed to respond as 'quickly as possible.' Previous studies indicate that postural responses are affected by context of the task (Lee et al. 1987; Benvenuti et al. 1990).

For trials of repetitive arm movement, subjects moved their left arm repetitively beside their body in the sagittal plane between 15° of extension and 15° flexion. Subjects moved their arm "as fast as possible" for 10 s while they held their breath at normal end-expiratory volume. Breathing was ceased during the trial as trunk muscle activity is modulated with respiration (Hodges and Gandevia 2000b). Subjects were given visual feedback of shoulder displacement to ensure the movement amplitude was consistent between conditions. Repetitive movement data were not collected during the follow-up period.

Data analysis

For trials of single arm movement the timing and amplitude of trunk muscle EMG was evaluated. EMG onset was identified from raw data as the point at which EMG increased from baseline. Traces could be enlarged to a resolution of 0.5 ms and were displayed individually, and in random order. There was no reference to muscle, trial, or arm movement to remove the potential for observer bias. Although the onset of EMG could be identified with all recordings, the selective fine-wire electrodes used to record the EMG activity of TrA, OI, OE, DM and SM increased the clarity of onset detection as the onset could be clearly identified from discrete action potentials. Two separate examiners assessed EMG onsets. Both examiners were trained together to ensure consistency of selection of EMG onsets. Preliminary results indicated a high level of agreement. Data from the first assessment were used for analysis but onsets were excluded from the analysis if they differed more than 5 ms from those of the second assessor (<5% trials). Visual detection of EMG onset was selected as it has been shown to be reliable and is less affected by factors such as amplitude of background EMG or the rate of increase in EMG amplitude (Hodges and Bui 1996). EMG onset was expressed relative to that of deltoid. Trials were excluded if the onset of trunk muscle EMG occurred more than 100 ms before, or 200 ms after, that of deltoid (<5% of trials). EMG activity of the trunk muscles outside this window is unlikely to be related to the perturbation resulting from movement of the arm (Aruin and Latash 1995).

To confirm that changes in EMG activity were present in a manner that was independent of the detection of EMG onsets of the trunk muscles, data were analyzed by comparison of the amplitude of trunk muscle EMG during 50-ms epochs around the onset of deltoid EMG. The onset of deltoid EMG was identified from the raw data and the root mean square EMG amplitude for each 50-ms epoch was determined (Epoch 1: −100 to −50 ms; Epoch 2: −50 to 0 ms; Epoch 3:0 to 50 ms; Epoch 4:50 to 100 ms).

Analysis of repetitive arm movements involved measurement of the mean EMG amplitude during movement and identification of temporal and spatial features of trunk muscle EMG from time-locked averages and frequency analysis. Rectified EMG averages were triggered from the onset of forward arm movement (identified as the peak posterior displacement from the arm movement data). Each repetition was time-normalized to 100 samples per cycle to control for variation duration of the arm movement cycle, and all repetitions of shoulder movement for the 10-s trial were averaged. The amplitudes of the peaks and troughs in the averages were measured.

Frequency domain analysis investigated the relationship between the frequency of trunk muscle EMG bursts and that of arm movement. Power spectra were generated by Fast Fourier Transform analysis. For the frequency analysis, data were high-pass filtered (100 Hz) to remove non-stationarity and movement artifact and were rectified and low-pass filtered (30 Hz). We evaluated the relationship between signals (arm movement and trunk EMG), in terms of the similarity in frequency content, by calculation of coherence. Coherence is a measure of the presence of a constant temporal and spatial relationship between the phasic changes in two signals (Bendat and Piersol 1966). Power and coherence were measured at the arm movement frequency. These parameters have been used previously (Zedka and Prochazka 1997; Hodges and Gandevia 2000a).

The peak acceleration for single arm movements and time-locked averages of repetitive movement were calculated by twice differentiating the angular displacement data. These data were used to confirm that the movement characteristics were consistent between trials.

Statistical analysis

For trials of single arm movement the mean latency between the trunk muscle EMG onset and that of deltoid, and the peak arm acceleration were compared between conditions using a repeated measures analysis of variance (ANOVA). Only the initial follow-up period was used for the main analysis. Differences for individual subjects were calculated using a separate repeated measures ANOVA for each muscle with a Bonferroni correction for multiple measures. Each follow-up period was compared separately to identify the EMG onset. The Epoch data were compared between conditions using a separate repeated measures MANOVA with fixed effects for each muscle. Data are also presented for individual subjects.

In repetitive arm movement trials the EMG amplitude of the mean, peak and trough, the amplitude of the power from the frequency spectra and coherence data, and the frequency and peak acceleration of the arm were compared between conditions for each muscle using one-way analyses of variance (ANOVA) with fixed effects and Duncan's multiple range test with the Bonferroni correction.

Results

The mean (SD) intensity of the worst pain reported on the VAS was 6.2 (1.0). The group average of the time course of the experimentally induced pain is presented in Fig. 2. In all subjects the pain initially exceeded 4/10 and remained above this level for 2–16 min. Three subjects required an additional injection of hypertonic saline (1 ml) to complete all of the movement tasks with pain of sufficient intensity. Only one subject experienced pain greater than 1/10 with injection of isotonic saline. The descriptor most frequently selected from the McGill Pain Questionnaire was 'aching' (n=6).

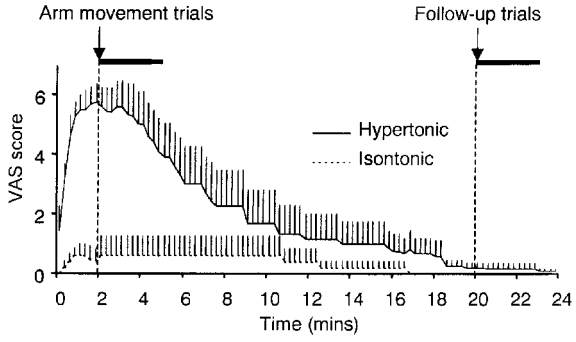

Fig. 2 Pain data. Mean and standard error of the VAS following injection of isotonic (*dashed line*) and hypertonic saline (*solid line*). The approximate time of initiation and duration of the arm movement trials is identified by the *arrow* and *bar,* respectively. Recordings of VAS were made each 15 s

With single repetitions of arm flexion the onset of TrA EMG preceded that of deltoid by 18.9 (2.4) ms. The EMG onsets of all muscles are presented in Table 1. With isotonic saline injection there was no change in EMG onset, except for SM (Table 1). However, after hypertonic saline injection the TrA EMG onset was delayed and remained delayed during the initial follow-up period (p<0.05). However, there were individual differences in time course of the changes in TrA EMG. In five subjects the response of TrA was delayed during the period of pain, in one subject the onset of TrA was not delayed until the initial follow-up period and in one subject there was no change in TrA EMG. The TrA EMG onset of Subject 2 had not returned to control values 1 h after hypertonic saline injection. In all other subjects the response had recovered by the second follow-up period. Of the other muscles, DM EMG onset was earlier after hypertonic saline (p<0.01), OE EMG onset was earlier during the follow-up (p<0.01), and there was no difference between conditions for ES and OI EMG (Table 1). Figure 3 shows data from a subject who is representative of the 6/7 subjects who had reduced TrA EMG.

Table 1 Mean (SE) of the latency between the onsets of trunk muscle EMG and that of deltoid for group. Data presented for the follow-up period represent only the initial follow-up period

	Control	Isotonic saline	Hypertonic saline	Follow-up
DM	47.3 (5.5)	47.7 (5.8)	27.4 (5.5)*	47.9 (5.1)
SM	29.2 (6.0)	54.3 (7.1)*	16.5 (4.3)	25.2 (4.4)
ES	10.7 (3.4)	18.9 (5.8)	14.3 (3.6)	11.6 (3.4)
TrA	−18.9 (2.4)	−13.7 (4.0)	−8.6 (4.1)*	2.6 (4.7)*
OI	−11.2 (4.4)	−15.6 (3.9)	−7.7 (6.4)	1.1 (5.1)
OE	53.6 (9.4)	66.9 (8.1)	74.5 (7.6)	37.4 (6.1)*

*difference from the control condition (p<0.05)

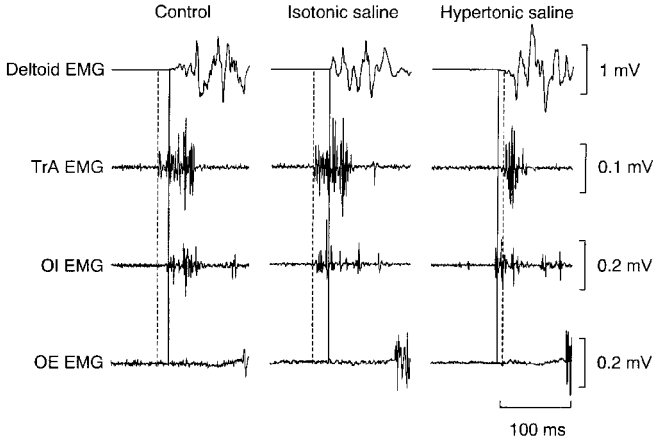

Fig. 3 Changes in abdominal muscle activity with injection of saline. Raw EMG data from a subject, representative of the 6/7 subjects who had a reduction in TrA EMG during single arm flexions. *Solid* and *dashed lines* identify the onsets of deltoid and TrA EMG, respectively. Note the delay in onset and reduced amplitude of TrA EMG following injection of hypertonic saline

The EMG amplitude for each 50-ms epoch before and after the onset of deltoid EMG for a single subject is presented in Fig. 4. In this subject TrA EMG was reduced during Epochs 3 and 4 (i.e., 0–50 ms and 50–100 ms after deltoid EMG onset) during pain. This subject also had an increase in OE EMG during Epoch 3. The group-mean data indicate that there was no change in EMG amplitude following the injection of isotonic saline, with the exception of DM during Epoch 4 (Fig. 5). Following hypertonic saline injection the group data show a reduction in TrA EMG during Epochs 2 ($p<0.02$), 3 ($p<0.0005$) and 4 ($p<0.05$). Furthermore, the reduction of TrA EMG in Epoch 3 remained in the first follow-up trial (i.e., 10 minutes after the injection). OE EMG was reduced during Epochs 3 ($p<0.01$) and 4 ($p<0.05$). While the amplitude of OI in Epoch 3 was also reduced following hypertonic saline injection ($p<0.01$), it was increased during Epoch 4 ($p<0.01$). DM EMG was increased during Epoch 1 ($p<0.005$) and 4 ($p<0.01$) following the injection of hypertonic saline. However, the amplitude of DM EMG during Epoch 4 was also increased following the injection of isotonic saline. The amplitude of SM EMG was reduced during Epoch 4 ($p<0.02$) in the follow-up.

The peak acceleration of the arm was not different between conditions for 5/7 subjects. One subject moved faster after hypertonic saline ($p<0.005$) and another in the control condition ($p<0.01$).

When subjects repetitively moved their arm for 10 s in the control condition, phasic bursts of trunk muscle EMG were present in each movement cycle (Fig. 6). This phasic activity occurred as one or two discrete bursts. TrA and DM EMG were frequently biphasic with EMG bursts during each direction of arm movement. Note the multiple peaks in the triggered averages in Fig. 7A and the large peak in the frequency spectrum at twice the arm movement frequency in Fig. 7B. In contrast, activity of

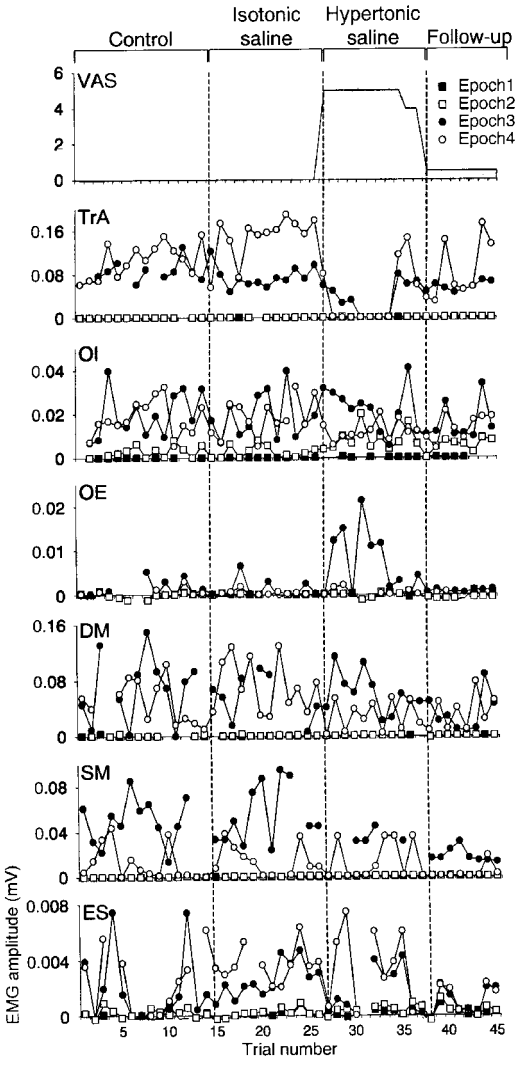

Fig. 4 Epoch data over time. Data are shown for a subject, representative of the 6/7 subjects who had a reduction in TrA activity. Changes in EMG amplitude during each epoch (Epoch 1: −100 to −50 ms, Epoch 2: −50 ms to 0, Epoch 3: 0 to 50 ms, Epoch 4: 50 to 100 ms relative to onset of deltoid EMG) are shown. Control, experimental and follow-up trials are indicated at the top and are separated by *vertical dashed lines*. The number of trials presented in each condition varies due to rejection of some trials (see "Materials and methods" for justification). The number of trials that were rejected was greater for the back muscles due to interference from ECG and greater frequency of movement artifact. Only the initial trial is shown for each 'set' in the follow-up periods. Note the decrease in TrA EMG following hypertonic saline injection and the subsequent recovery. Also note the increase in OE EMG following hypertonic saline injection

OE, OI, ES and SM usually, although not exclusively, occurred as a single burst (Figs. 6, 7A). The frequency of arm movement in the control condition was 3.9 (0.2) Hz.

With injection of saline (both hypertonic and isotonic) there was no difference in the frequency of arm movement ($p=0.44$), peak displacement ($p=0.37$) or peak acceleration of the arm ($p=0.13$). Despite the similar movements, the peak ($p<0.002$), trough ($p<0.002$) and mean (0.02) amplitudes of TrA EMG were reduced during

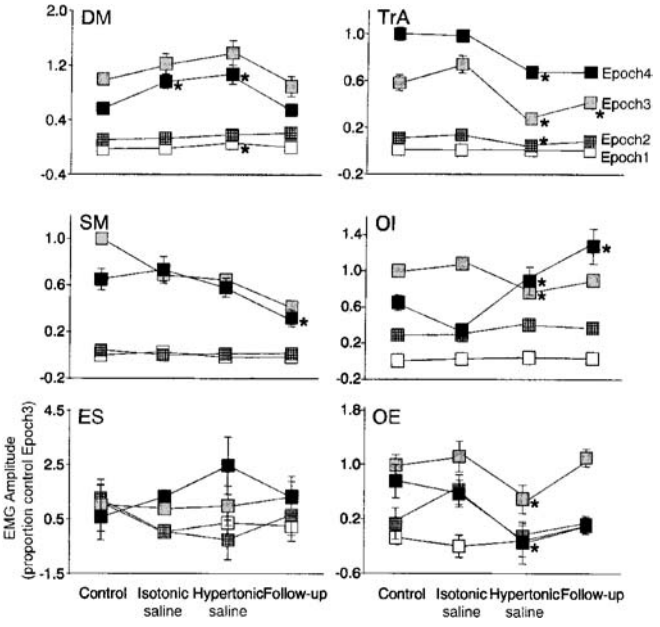

Fig. 5 Epoch data for group. EMG amplitude expressed as a proportion of the Epoch 3 EMG in the control trials is shown for each epoch in all conditions. Each column of data is a single condition. Standard error is shown. Note the decrease in TrA, OE and OI EMG and the increase in DM EMG following injection of hypertonic saline. *Asterisk* indicates differences between conditions (p<0.05)

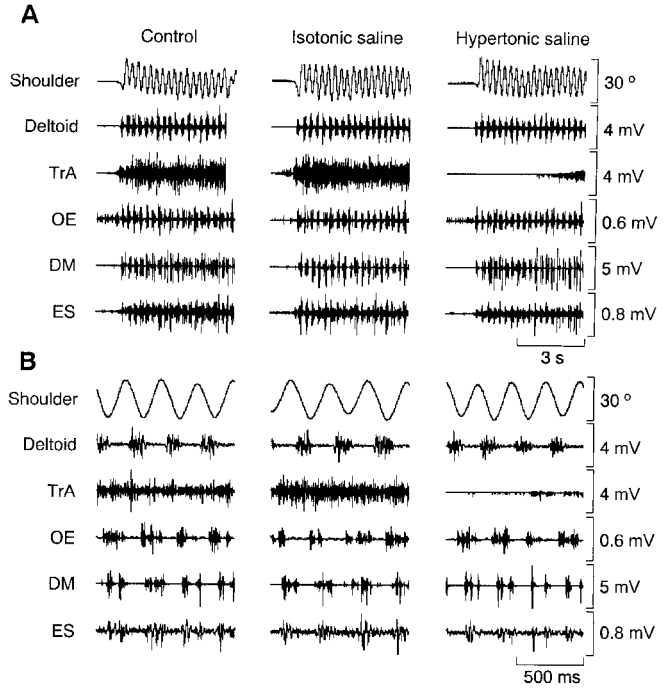

Fig. 6A, B Raw EMG data for repetitive arm movements. Raw EMG data for a subject, representative of the 6/7 subjects who had reduced TrA EMG. **A** and **B** show the same trial on different time scales to show the overall response **(A)** and detail of the burst pattern for individual arm movements **(B)**. Note the phasic bursts in trunk muscle EMG with repetitive movement. The amplitude of TrA EMG is reduced following hypertonic saline injection. The initiation of recovery of TrA EMG can be seen at the end of the hypertonic saline panel

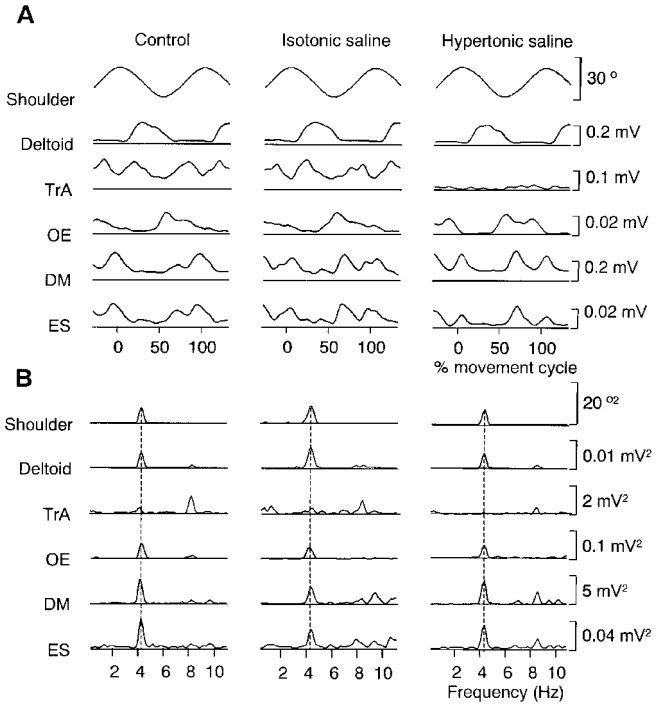

Fig. 7A, B Triggered averages and frequency spectra for a single subject. **A** EMG averages triggered from the onset of arm flexion are shown for the same subject as Fig. 6. Averages are time-normalized to 100 samples for each arm movement cycle. The amplitude of the EMG activity is reduced compared to the raw data as a result of averaging. Note the phasic and biphasic bursts of the trunk muscles with repetitive movement of the arm. Also note the reduction in EMG amplitude of TrA following the injection of hypertonic saline. **B** The frequency spectra calculated for the same subject as Fig. 7. The *dashed line* denotes the frequency of arm movement. Note the peaks in the power spectra at the frequency of arm movement and/or a frequency twice that of the arm movement. This second peak was, in part, due to the modulation of EMG at twice the movement frequency (see TrA EMG in **A**) and because the signals are not perfectly sinusoidal. The amplitude of the TrA peak at the frequency of arm movement was decreased following the injection of hypertonic saline

pain. This is presented for a representative subject in Fig. 7A. The group data, normalized to the control trials, are shown in Fig. 8. There was no difference between the control and isotonic injection trials (peak: p=0.15, trough: p=0.12, mean: p=0.17). Of the other muscles, only the mean ES EMG was reduced after hypertonic saline injection (p<0.001) (Fig. 8).

Changes in amplitude data were supported by similar changes in the frequency data. The power of the TrA EMG frequency spectrum was reduced at the frequency of arm movement (p<0.019) (Figs. 7B, 9) and the coherence between TrA EMG and movement was reduced from 0.9 (0.0) to 0.7 (0.1) after hypertonic saline injection (p<0.009) (Figs. 7B, 9). There was no difference in the power (p=0.1) or coherence (p=0.96) at the movement frequency between control and isotonic injection trials.

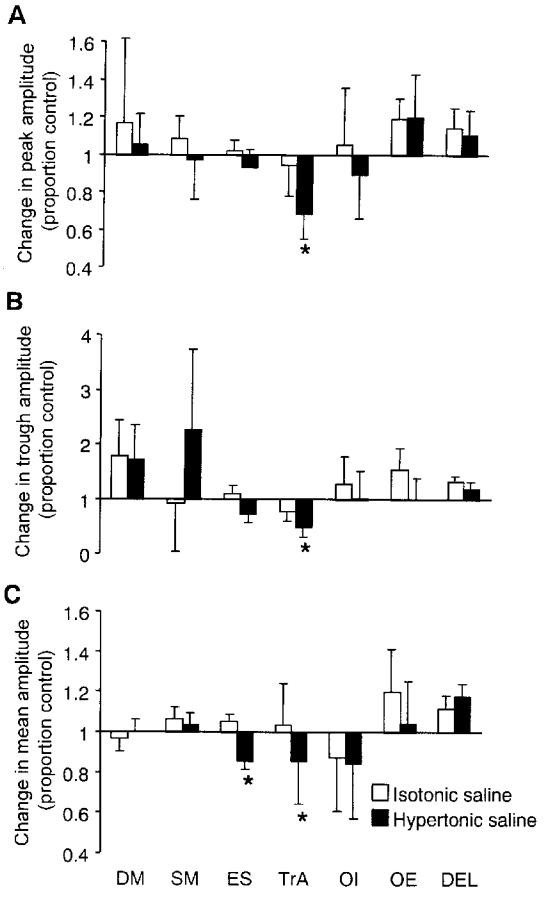

Fig. 8 Mean EMG amplitudes. EMG amplitudes of the peak **(A)** and trough **(B)** of the triggered averages, and the mean EMG across the movement **(C)** are shown. All values are reported as a proportion of the amplitude in the control condition. Note the reduction in TrA EMG following the injection of hypertonic saline. *Asterisk* indicates differences between conditions ($p<0.05$)

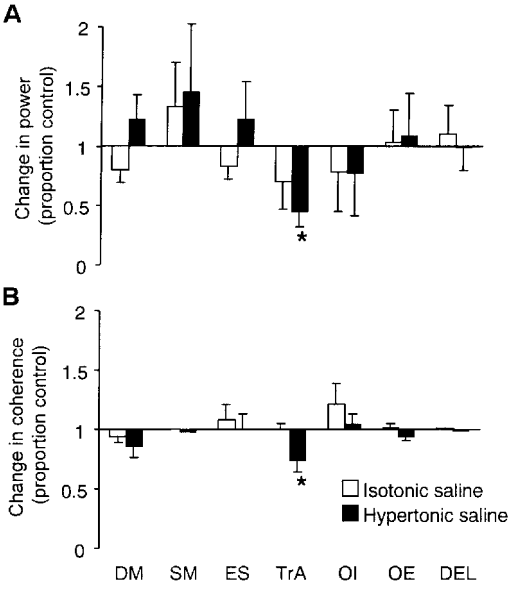

Fig. 9A, B Mean frequency parameters. The amplitude of the power of the frequency spectrum of the trunk muscles **(A)** and the coherence between trunk muscle EMG and arm movement **(B)** at the frequency of arm movement are shown. Note the reduction in amplitude of both parameters for TrA following injection of hypertonic saline. This finding provides additional evidence for a reduction in TrA EMG activity associated with arm movement during experimentally induced pain. *Asterisk* indicates differences between conditions ($p<0.05$)

Discussion

The results of the present study demonstrate that feedforward recruitment of the trunk muscles is altered by acute experimentally induced pain. This was evidenced by changes in the spatial and/or temporal features of the EMG response to voluntary arm movements. While TrA showed consistent changes toward delayed activation or reduced EMG amplitude, changes in other muscles were more variable and included augmentation or delay and/or reduced or increased EMG activity. Because the trunk muscles are normally active in advance of the movement, the effect of pain observed here is not caused by alterations in short or long latency feedback mechanisms.

Although fundamental differences exist between experimentally induced and clinical pain, the findings of the current study suggest that some changes in motor control that occur in patients with LBP may be caused by pain. In this regard, three features of the results are notable. The first feature is the delay observed in TrA EMG across tasks during single movements in ~83% of subjects, and the marked reduction in amplitude of the mean EMG and EMG power at the frequency of limb movement during the repetitive arm movements across the group. Impairment of TrA has been identified during similar limb movement tasks in patients with recurrent LBP (Hodges and Richardson 1996, 1998, 1999). The second feature is that the effect of pain on the other trunk muscles varied between subjects and between movement tasks. Wide variability in trunk muscle EMG in patient groups has been reported in the literature, and there is marked variability between studies (Collins et al. 1982; Nouwen et al. 1987; Arena et al. 1989; Shirado et al. 1995; Wilder et al. 1996; Radebold et al. 2000). Previous studies of experimentally induced pain during gait (Arendt-Nielsen et al. 1996) and voluntary trunk movements (Zedka et al. 1999b) have also reported wide variability. These two features raise the possibility that motor control deficits identified in patients may be consequent to pain, although it remains plausible that they may, at least in some subjects, also lead to LBP (e.g., Janda 1978).

An additional feature that is consistent with patients is the potential for changes in trunk muscle activity to persist after the resolution of pain. Previous studies of trunk muscle activity in association with limb movements have used subjects who have recurrent episodes of LBP but were pain-free at the time of testing (Hodges and Richardson 1996, 1998, 1999). Those findings suggest that the trunk muscle recruitment strategy does not resolve spontaneously on the resolution of symptoms.

This suggestion has also been made by others (e.g., Hides et al. 1996). In the present study the response of TrA to single arm movements was delayed across the group during the follow-up period, even though pain intensity was minimal. In one subject the response of TrA did not recover within 1 h of the saline injection. This may be an important finding because it raises the possibility that minimal exposure to painful stimuli may have long-term sequelae. Further research is required to substantiate this possibility.

In general, the present results are consistent with previous findings of ES activity following injection of hypertonic saline during gait (Arendt-Nielsen et al. 1996) and voluntary trunk movements (Zedka et al. 1999b). Those studies reported increased or decreased activity during different phases of the tasks. This pattern of response has been considered to be consistent with the pain adaptation model of Lund et al. (1991), which postulates that, by increasing antagonist activity and decreasing agonist activity, the maximum force, displacement and velocity of movement is decreased. It is difficult to categorize agonistic and antagonistic trunk muscles during rapid arm movements; however, in terms of minimization of displacement and velocity of the trunk in response to perturbation, the pain adaptation model may predict increased activity of trunk muscles in order to splint the trunk. In the current study, increased and/or augmented activation of one or more of the trunk muscles was evident across tasks and across subjects.

The variability observed between subjects is perhaps not surprising and may reflect the non-homogeneity of clinical LBP. However, this issue is unlikely to have had a substantial impact in the present study because the nociceptive stimulus and experimental context and environment was standardized across the group. It is more likely that the intersubject and intertask variability are due to factors associated with variability in the experience of pain and/or the redundancy in the motor system. That is, cognitive and behavioral factors probably introduced variability in pain and pain report, and because many muscles surround the spine, there is considerable capacity for variation in the motor response to pain.

One feature of the current results that appears inconsistent with data from patient groups is the lack of a consistent effect on multifidus activation. There is an extensive literature describing changes in multifidus in LBP patients. For instance, studies have reported reduced fatigue resistance (Roy et al. 1989), changes in muscle fiber composition (Rantanen et al. 1993), reduced activation during gross movement tasks (Sihvonen et al. 1997), and reduced cross-sectional area (Hides et al. 1994). Thus we expected changes in the recruitment of this muscle during experimentally induced pain, particularly the deep fibers that mirror the activation of TrA during limb movement tasks (Moseley et al. 2002). However, we found no consistent change in feedforward activation of this muscle during experimentally induced pain.

Although the current work demonstrates that feedforward responses can be altered by LBP, the mechanism by which this occurs is not clear. Several factors are critical for planning feedforward strategies, for example, evaluation of the current position, stability and movement status of the spine and prediction, planning and implementation of an appropriate strategy to overcome the impending perturbation. The impairment identified in the present study may be due to a change in any of these components. Consistent with this possibility there is compelling evidence that pain has strong effects at the supraspinal level (Venna et al. 1994; Derbyshire et al. 1997; Lorenz and Bromm 1997; Kuukkanen and Malkia 1998; Luoto et al. 1998, 1999; Hodges 2001). Both short- and long-term changes are thought to occur in activity of the supraspinal structures including the cortex with pain. Many studies have reported changes during experimental pain in activity of regions of the brain involved in movement planning and performance (see Derbyshire et al. 1997 for review). Alternatively, the changes may be explained by changes in motoneuron and motor cortex excitability, or conduction velocity in the CNS. Widespread changes in excitability have been identified at many levels of the motor system during pain. Acute experimental pain has been shown to cause changes in spinal motoneuron activity (Matre et al. 1998; Svensson et al. 1998, 2000). For instance, increased stretch reflex amplitude of the soleus muscle has been reported after intramuscular injection of hypertonic saline (Matre et al. 1998). Others report reduced amplitude of motor potential evoked by transcranial magnetic stimulation over the motor cortex in response to experimental pain (Valeriani et al. 1999). However, these responses may be task or muscle specific as other studies have reported no changes in excitability of the motoneuron or motor cortex (Gandevia et al. 1996; Zedka et al. 1999a) and we have shown that the changes in TrA in clinical LBP are inconsistent with a change in excitability (Hodges 2001). Finally, a mechanical hypothesis for the changes in TrA may also be considered: perhaps the splinting effect of the superficial muscles negates the need for TrA to contribute to spinal control.

Regardless of the mechanism(s) involved, consistent impairment of TrA requires further investigation. On the basis of studies of trunk muscle EMG activity (Cresswell et al. 1992, 1994; Hodges and Richardson 1997) and theoretical studies (Tesh et al. 1987; Snijders et al. 1995; Daggfeldt and Thorstensson 1997), TrA is thought to provide an important contribution to spinal stability via its role in control of IAP, tension in the thoracolumbar fascia and force-closure of the sacroiliac joints. Recent in vivo studies provide evidence for this role in spinal stability and argue that contraction of this muscle may be particularly important for the fine-tuning of intersegmental control of the spine and pelvis (Richardson et al. 2000; Hodges et al. 2001). Thus, although increased activity of the superficial trunk muscles may splint the spine, the fine-tuning of intersegmental control may be impaired.

Conclusions and clinical significance

The present data confirm that feedforward responses of the trunk muscles are affected by pain. While the response of most muscles to pain was variable, TrA responded more consistently to pain and results were similar to that established in people with chronic LBP. It is hypothesized that the abnormal TrA recruitment compromises the precision of spinal stability, although the effect of chronic impairment of TrA activity remains uncertain. The changes in motor control that were produced by experimentally induced pain were more complex than simple inhibition and include changes in motor planning.

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