Exp Brain Res (2004) 156: 64–71
DOI 10.1007/s00221-003-1766-0

RESEARCH ARTICLES

G. Lorimer Moseley · M. K. Nicholas · Paul W. Hodges

Pain differs from non-painful attention-demanding or stressful tasks in its effect on postural control patterns of trunk muscles

Received: 28 May 2003 / Accepted: 16 October 2003 / Published online: 19 December 2003

Abstract Pain changes postural activation of the trunk muscles. The cause of these changes is not known but one possibility relates to the information processing requirements and the stressful nature of pain. This study investigated this possibility by evaluating electromyographic activity (EMG) of the deep and superficial trunk muscles associated with voluntary rapid arm movement. Data were collected from control trials, trials during low back pain (LBP) elicited by injection of hypertonic saline into the back muscles, trials during a non-painful attention-demanding task, and during the same task that was also stressful. Pain did not change the reaction time (RT) of the movement, had variable effects on RT of the superficial trunk muscles, but consistently increased RT of the deepest abdominal muscle. The effect of the attention-demanding task was opposite: increased RT of the movement and the superficial trunk muscles but no effect on RT of the deep trunk muscles. Thus, activation of the deep trunk muscles occurred earlier relative to the movement. When the attention-demanding task was made stressful, the RT of the movement and superficial trunk muscles was unchanged but the RT of the deep trunk muscles was increased. Thus, the temporal relationship between deep trunk muscle activation and arm movement was restored. This means that although postural activation of the deep trunk muscles is not affected when central nervous system resources are limited, it is delayed when the individual is also under stress. However, a non-painful attention-demanding task does not replicate the effect of pain on postural control of the trunk muscles even when the task is stressful.

Keywords Experimental pain · Attention · Stress · Back pain

G. L. Moseley (✉)
Department of Physiotherapy, Royal Brisbane Hospital,
4029 Herston, Queensland, Australia
e-mail: l.moseley@uq.edu.au
Tel.: +61-7-36362590
Fax: +61-7-36362595

G. L. Moseley · P. W. Hodges
Prince of Wales Medical Research Institute,
Sydney, New South Wales, Australia
e-mail: l.moseley@uq.edu.au

G. L. Moseley · P. W. Hodges
Department of Physiotherapy, The University of Queensland,
Brisbane, Queensland, Australia

G. L. Moseley · M. K. Nicholas
Pain Management and Research Centre, University of Sydney
and Royal North Shore Hospital,
Sydney, New South Wales, Australia

Introduction

Changes in postural activation of the trunk muscles during functional tasks have been demonstrated in chronic pain patients (Arendt-Nielsen et al. 1996; Hodges and Richardson 1996) and in asymptomatic subjects given acute experimental pain (Arendt-Nielsen et al. 1996; Zedka et al. 1999; Hodges et al. 2003). Changes in muscle activity during acute experimental pain may reflect changes in spinal motoneuron activity. However, changes associated with low back pain (LBP) have been most often attributed to supraspinal effects (Venna et al. 1994; Lorenz et al. 1997a; Lorenz and Bromm 1997; Derbyshire et al. 1998; Kuukkanen and Malkia 1998; Luoto et al. 1998, 1999).

During single arm movements, postural activation of the trunk muscles is altered when LBP is induced by injection of hypertonic saline into the back muscles (Hodges et al. 2003). The effects on superficial trunk muscles vary between individuals but the timing and amplitude of activation of the deepest abdominal muscle is affected in a consistent manner between individuals. The effect of experimentally induced LBP is similar to that observed in chronic recurrent LBP patients (Hodges and Richardson 1996), and further work in that group demonstrated that the changes are consistent with alterations in the organisation of the postural responses rather than changes in motoneuron excitability (Hodges 2001).

There are several potential explanations for an effect of pain on the planning of postural responses. The current experiment investigated two possibilities. One possibility relates to the information processing requirements of pain. It is widely assumed that pain receives high supraspinal priority because it is directly relevant for survival (for review see Price 2000). Several studies have supported this assumption. For example, recordings of event-related potentials in the cortex (Rosenfeld et al. 1993), brain imaging studies (Derbyshire et al. 1997, 1998), cognitive performance tasks (Crombez et al. 1998a, 1998b, 1999; Eccleston and Crombez 1999) and a combination of these methods (Lorenz et al. 1997b; Lorenz and Bromm 1997) indicate longer latencies to cortical and behavioural responses and more errors in performance with pain than without pain. Although preparatory postural activation of the trunk muscles during voluntary movement is not under attentional control, it needs to be relevant to the movement response. Therefore, postural responses should be indirectly affected by those factors by which the voluntary movement is directly affected. If voluntary movement is disrupted during pain because the two phenomena share common and capacity-limited information processing systems, then the postural responses associated with voluntary movements may also be disrupted.

Another possibility relates to the effect of stress on motor output, which is relevant because pain is inherently stressful. Little is known about the effect of stress, in this sense a perception of threat, on preparatory postural control. However, effects on motor output have been shown (Weinberg and Hunt 1976; Jones and Cale 1997; van Galen and van Huygevoort 2000). For example, Marras et al. (2000) reported an alteration in trunk muscle activity during a lifting task when the task was performed in the presence of psychosocial stressors, and Ekberg et al. (1995) showed that work-related stress altered shoulder muscle activity during keyboard work. In chronic pain patients, disruption of motor control has been linked to subjective measures of distress and anxiety, rather than just to the intensity of pain (Flor and Turk 1989; Flor et al. 1992; Vlaeyen et al. 1999).

Thus, the aim of the current study was to determine whether the effect of pain on control of postural activation of the trunk muscles during rapid arm movements can be replicated by non-painful but attention-demanding or stressful tasks. We hypothesised that neither task has the same effect on the postural activation of the trunk muscles that is observed during pain.

Methods

Subjects

Five male and three female subjects participated, with an age, weight and height of 32±7 years, 63±13 kg and 170±11 cm, respectively. Subjects were excluded if they had been diagnosed with any respiratory or neurological condition, did not score within normal distribution for the Speilberger State-Trait Anxiety Inventory (STAI; Spielberger et al. 1970), or if they had experienced LBP in the previous 2 years. Subjects provided written consent and were free to withdraw from the study at any time. All procedures were approved by the institutional research ethics committee and conformed with The Declaration of Helsinki.

Electromyography

EMG of the deep [deep multifidus (Deep MF), and transversus abdominis (TrA)] and superficial [superficial multifidus (Sup MF), obliquus externus (OE) and obliquus internus (OI)] trunk muscles was recorded with selective bipolar intramuscular electrodes, fabricated from Teflon-coated stainless-steel wire (75 µm), with 1 mm of Teflon removed from the cut ends. The tips were bent back 1 mm and 2 mm from the end to form a hook and the electrodes were threaded into a hypodermic needle (32×50 mm or 70×55 mm).

With the subject supine, electrodes were inserted, under the guidance of real-time ultrasonography (128XP/4; Acuson, Mountain View, CA, USA), into the deep (TrA) and superficial (OE and OI)

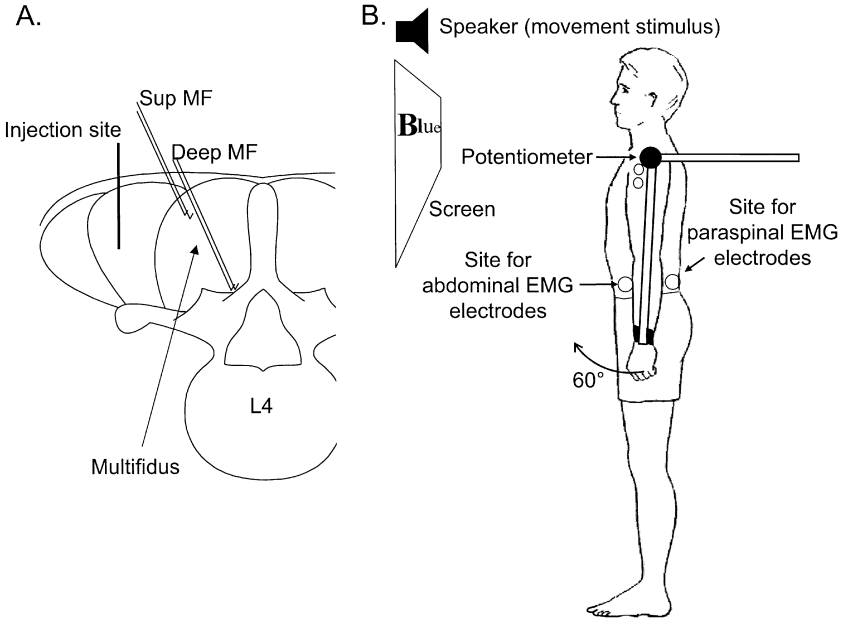

Fig. 1 **A** The site for injection of hypertonic saline relative to the intramuscular electromyograph (EMG) electrodes placed in lumbar multifidus. **B** The experimental set up showing the screen used for the Stroop test and the speaker for the (auditory) movement stimulus. Intramuscular electrodes recorded EMG from the anterolateral abdominal muscles and the deep and superficial components of the multifidus muscle (Deep MF and Sup MF, respectively). Surface electrodes recorded EMG from anterior deltoid. Galvanic skin response and heart rate were used as physiological measures of stress and measured via the right hand

abdominal muscles, through the anterolateral abdominal wall, midway between the iliac spine and the inferior border of the rib cage. In a similar manner but with the subject in sitting, electrodes were inserted into the deep and superficial back muscles (lumbar multifidus) at the level of the L4 vertebral lamina (Fig. 1A) (Moseley et al. 2002).

Pairs of surface EMG electrodes (Ag/AgCl discs, 1 cm diameter, 2 cm inter-electrode distance) were placed longitudinally over the left anterior deltoid muscle. A ground electrode was placed over the right iliac crest. EMG data were differentially amplified with a gain of 10,000–50,000, bandpass filtered between 53 Hz and 1 KHz, and sampled at 2 KHz, using a Power 1401 and Spike 2 software (Cambridge Electronic Design, Cambridge UK). Data were exported for analysis with Matlab 5.1 (Mathworks, Natick, MA, USA).

Stress and pain

The galvanic skin response (GSR), which provides a measure of sympathetic nervous system activity, was recorded using Ag/AgCl discs (1 cm diameter) placed on the palmar and dorsal surface of the right hand. Heart rate (HR) was recorded using a pulse-oximeter. GSR and HR data were sampled at 2 KHz along with the EMG data. After each set of trials in each condition, subjects responded to a single item, "How distressing did you find that trial?", using an 11-point numerical rating scale (NRS) anchored with "not at all distressing" and "extremely distressing". We used an 11-point NRS anchored with "no pain" and "worst possible pain" to record the intensity of pain experienced by the subject. During the pain condition, the subject was asked "How would you rate the pain during that trial?" after completion of the arm movement trials.

Procedure

Subjects performed single arm movements under four conditions: (1) control, (2) attention-demand, (3) stressful attention-demand, and (4) experimentally-induced pain. Attention-demand and stress were conducted in random order, but experimentally induced pain was always conducted last because previous work showed that experimentally induced pain can have lasting effects on trunk muscle activity (Hodges et al. 2003). The experimental set up is shown in Fig. 1B.

Control trials

Subjects stood relaxed. A lightweight bar connected to a potentiometer was fixed to the left wrist. In response to an auditory signal, the subject moved the left arm forwards as quickly as possible (Fig. 1B). Emphasis was placed on the speed of movement, rather than the distance moved.

Attention-demand condition

Subjects performed a modified colour–word Stroop test in which colour names, written in different colours, are presented on a black screen at 0.6-s intervals. Subjects are required to state the colour in which the word is written. However, because the trunk muscles are involved in respiration and speech (Hodges et al. 1997), it would be difficult to delineate EMG associated with postural demands from that associated with speech. Therefore subjects were instructed to respond verbally only when the name of the word was the same as its colour. Fifteen percent of the frames were matched in this way. While performing the Stroop test, subjects continued to respond to the auditory stimulus. Subjects were advised that they were performing well regardless of their actual performance. Because the objective was to perform an attention-demanding task that was not stressful, trials were repeated if a stress response was observed in HR or GSR.

Stressful attention-demand condition

To make the Stroop test stressful, subjects were advised that (1) they were to receive the test on the second easiest setting and that they were expected to be excellent at the task, (2) they were to keep up with the computer and that missed frames and false or slow calls would be recorded and contribute to a "Proficiency Score", and (3) they were not performing well regardless of their actual performance. No false feedback about specific responses was provided. Investigators appeared unhappy and annoyed with each other and with the subject.

Experimentally induced LBP

Hypertonic saline (5%, 1–1.5 ml bolus) was injected into the right erector spinae (ES) ~5 cm lateral to the L4 spinous process at a depth of ~3 cm. Prior to saline injection, approximately 0.5 ml lignocaine (lidocaine 2% with adrenaline) was injected beneath the skin. At 15-s intervals, pain was reported using the NRS for pain. Arm movement trials were commenced when pain reached a NRS ≥5. Previous work has verified that no general change in activity of the back muscles is evident after injection of isotonic saline (0.9%) (Hodges et al. 2003). Thus, an injection of isotonic saline was not included as a control in the current study.

Data analysis

Temporal and spatial parameters of EMG data were analysed. To remove bias, EMG traces were displayed individually and without reference to muscle or trial. 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. The reaction time (RT) of each muscle and the latency between the onset of EMG of the trunk muscles and that of deltoid EMG were analysed. Trials were excluded if the onset of trunk muscle EMG occurred less than 200 ms before or after that of deltoid, because EMG during this period is unlikely to be related to the task (Aruin and Latash 1995). To confirm the onset data in a manner that is not dependent on the ability to detect onset of trunk muscle EMG, the magnitude of the rectified EMG signal was also measured during epochs of 50 ms duration each, for 100 ms before and after the onset of deltoid EMG. The mean baseline EMG was determined from 500–100 ms prior to deltoid EMG onset, and the mean EMG amplitude during each 50-ms epoch, normalised to the control condition, was determined. Normalisation in this manner allows comparison between control and experimental conditions for each epoch but not between epochs within each condition.

Statistical analysis

A one-way analysis of variance (ANOVA) was used to compare stress measures between conditions. For single movement trials, EMG onset latency relative to the onset of deltoid EMG, and mean EMG amplitude for each epoch, were averaged for all trials across the sample. Because altered arm kinematics have an effect on the postural demands associated with movement (Hodges and Richardson 1997), standard *t*-tests compared peak shoulder acceleration between conditions to verify similar performance of the task. As the data were normally distributed, a multiple analysis of covariance (MANCOVA) with shoulder acceleration entered simultaneously as a covariate, was used to compare the EMG onset latency between conditions for each muscle. A separate MANCOVA, with shoulder

acceleration again entered simultaneously as a covariate, was used to compare the mean rectified EMG amplitude for each epoch, for each muscle, between conditions. Post hoc testing of EMG onset latency between conditions, and the epoch data between conditions, was undertaken with Duncan's multiple range test. Significance was set at the 0.05% level of probability.

Results

Stress and pain

Figure 2A–C demonstrates the reported distress and pain, HR and GSR during the control and experimental conditions. No subjects reported a change in back pain during arm movements. All three measures were greater during the stressful task and pain than during the attention-demanding task and control ($P<0.01$ for both). There were no differences in these measures between control and the attention-demanding task. Taken together, these measures indicate that the experimental protocol was effective at selectively targeting attention-demand and stress.

Arm movements

There was no difference in peak shoulder acceleration between conditions ($P>0.53$ for all), which suggests that the task was performed in a similar manner during each condition. Less than 15% of trials for each subject were excluded or rejected because of difficulty in detecting onset. Raw EMG from a representative subject (Fig. 3) and group data (Fig. 4) for the control and experimental conditions show the general effects of attention-demand, stressful attention-demand and pain on the movement task. The mean ±SD latency of the onset of deltoid and trunk muscle activation is presented in Table 1. Attention-demand caused an increase in the RT of the task (as evidenced by increased RT of arm movement and deltoid EMG) ($P<0.01$), but had no effect on activation of the deep trunk muscles. Stressful attention-demand caused a similar increase in the RT of the task but also caused a delay in response of the deep trunk muscles ($P<0.01$) (Fig. 4A). Therefore, relative to the onset of deltoid EMG, the deep trunk muscles were active earlier during attention-demand than during the other conditions ($P<0.01$). During pain, RT of deltoid and the superficial trunk

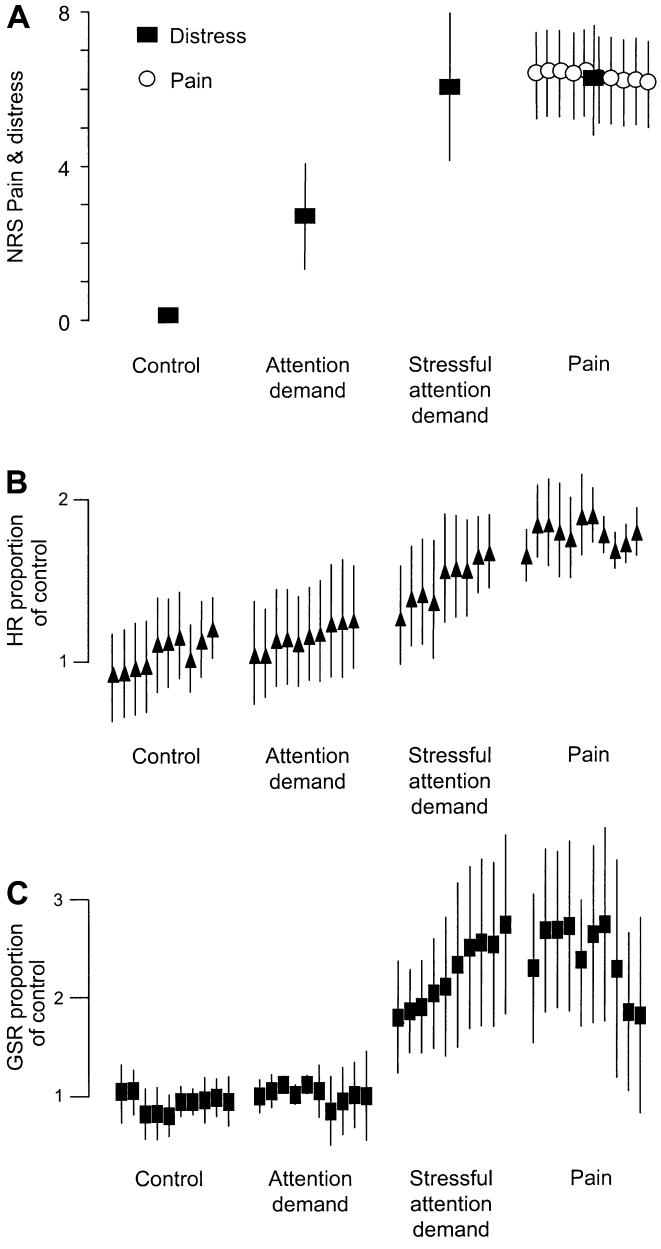

Fig. 2 **A** Means ±SD for reported distress (*circles*) and pain (*boxes*) during the control and experimental conditions assessed on 11-point numerical rating scales (NRS). **B** Means ±SD for heart rate (HR) as a proportion of mean HR during the control trials, for the experimental conditions. **C** Mean ±SD for galvanic skin response

Table 1 Mean ±SD reaction time of deltoid and the trunk muscles (*TrA* transversus abdominis, *Deep MF* deep multifidus, *OI* obliquus internus, *OE* obliquus externus, *Sup MF* superficial multifidus) during single arm movements under control and experimental conditions

Condition	Reaction time (ms)					
	Deltoid	TrA	Deep MF	OI	OE	Sup MF
Control	180±14	154±15	176±14	168±16	220±37	185±11
Attention-demand	280±16*	164±21	195±35	263±23*	325±46*	281±17*
Stress	286±20*	257±46*	289±41*	263±39*	321±40*	285±20*
Pain	187±16	189±19*	185±50	163±48	212±51	182±42

*Significant difference from control ($P<0.01$)

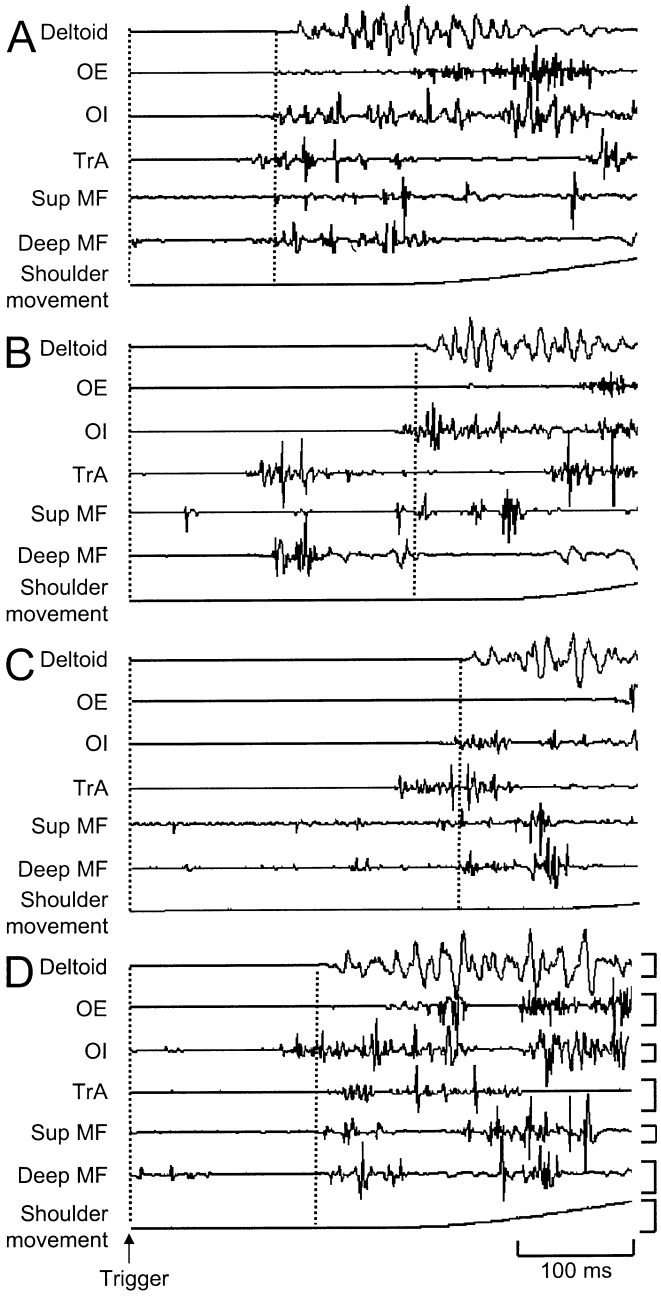

Fig. 3A–D Raw EMG data for deep (Deep MF) and superficial multifidus (Sup MF), transversus abdominis (TrA), obliquus internus (OI), obliquus externus (OE) and deltoid muscles from a single subject during the control (**A**), attention-demand (**B**), stressful attention-demand (**C**) and pain (**D**) conditions. *Vertical dashed line* indicates the onset of EMG in deltoid. *Scale bars* on the right denote 2 mV for the EMG traces, and 60° for the movement traces. Note the delay of deltoid, OE, OI and Sup MF but not TrA and Deep MF during attention-demand, and the delay relative to the attention-demand for TrA and Deep MF during the stressful condition. Finally, note the delay in TrA relative to deltoid during the pain condition

muscles was neither greater than that during control ($P>0.1$) nor less than that during the attention-demand or stressful attention-demand ($P>0.1$); however, for every subject, activation of at least one superficial trunk muscle occurred earlier relative to deltoid. The RT of the deep abdominal muscle TrA was greater (i.e. delayed) during pain than during control and the attention-demand ($P<0.01$) and, relative to the onset of deltoid EMG, the onset of TrA occurred later during pain than during the other conditions ($P<0.01$) (Fig. 4B).

The epoch data corroborate the timing data. Figure 5 shows that during attention-demand there was a two-fold increase in mean EMG amplitude during the first two epochs (0–100 ms prior to deltoid EMG onset) for TrA and Deep MF ($P<0.05$), which suggests the burst of EMG occurred earlier relative to deltoid. During pain, mean EMG amplitude during the first two epochs for TrA was lower than control ($P<0.04$), which suggests the burst of EMG occurred later relative to deltoid. Mean Deep MF EMG amplitude during the 50-ms epochs prior to deltoid onset was higher during pain than during control ($P<0.05$). Similar to the Deep MF epoch data, Sup MF EMG was greater during the epochs either side of deltoid onset, and Sup MF and OE baseline EMG was greater during pain than during control ($P>0.05$).

Discussion

This study shows that a non-painful attention-demanding task does not replicate the changes in planned postural responses of the trunk muscles that are caused by pain, even when the task is also stressful. Notably, however, both the non-painful attention-demand and the stressful attention-demand conditions had differential effects on activation of the deep and superficial trunk muscles. The current results imply that the effect of LBP on postural activation of the trunk muscles during arm movements is not simply a result of compromised central nervous system (CNS) performance caused by the information processing requirements of pain, nor simply a consequence of being under stress.

In this study, experimentally induced LBP consistently impaired postural activation of TrA but the effects on the superficial trunk muscles varied between subjects. Each of the superficial trunk muscles was sometimes augmented, sometimes delayed and sometimes unaffected. These findings support those from a previous study (Hodges et al. 2003) and are similar to findings from chronic recurrent LBP patients (Hodges and Richardson 1996). Although it remains feasible that activation of TrA may be impaired during pain by inhibitory influences at a spinal level, two points suggest otherwise: the variable effect on the superficial trunk muscles between individuals, and similar findings from patients in whom the alteration in postural responses has been shown to be caused by changes in postural planning (Hodges 2001).

A more likely explanation is that the changes reflect a CNS strategy to splint the trunk. If trunk motion is restricted by coactivation of antagonist (superficial) trunk muscles (it is notable that baseline activity of OE and Sup MF was increased during pain), then the CNS may perceive that the demand for activation of TrA is reduced, which in turn leads to decreased recruitment during

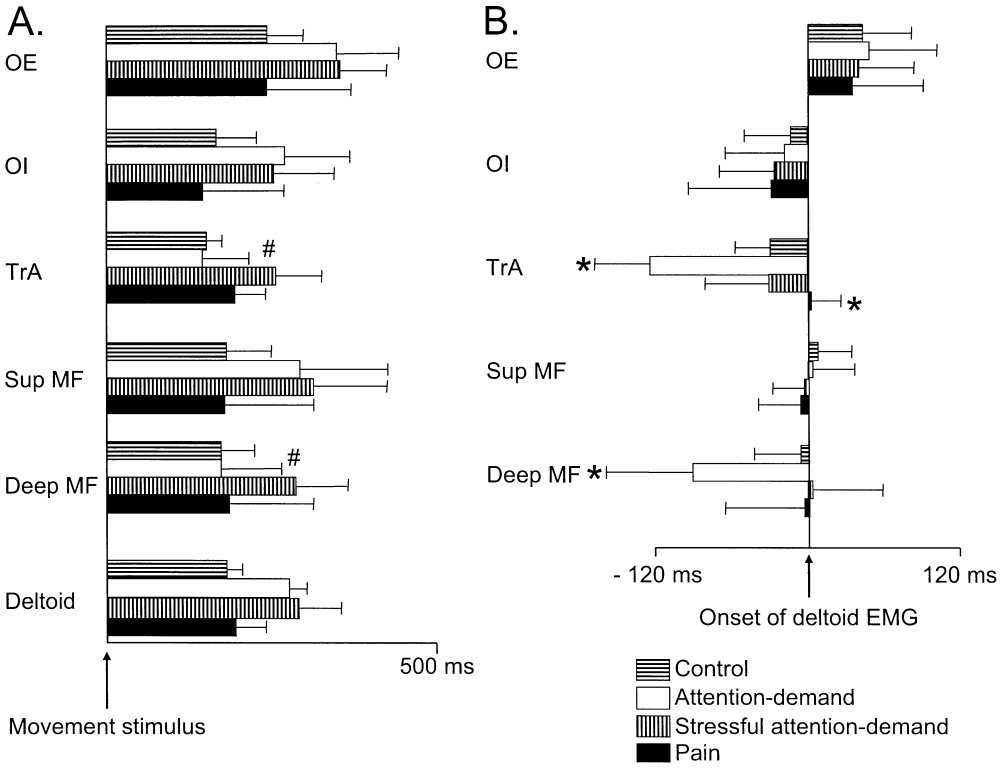

Fig. 4 **A** Group means and SD for the onset of EMG in the deep (Deep MF) and superficial multifidus (Sup MF), transversus abdominis (TrA), obliquus internus (OI), obliquus externus (OE) trunk muscles and deltoid relative to the movement stimulus for control and experimental conditions. **B** Group mean and SD for the onset of EMG relative to deltoid in the trunk muscles for the control and experimental conditions. *Vertical line* indicates the onset of deltoid EMG

movements. Although there is debate as to hyperactivity and hypoactivity of the trunk muscles during pain, for example studies of ES EMG activity during pain have reported increased activity (Arena et al. 1989), decreased activity (Collins et al. 1982; Nouwen and Bush 1984) and no change in activity (Hoyt et al. 1981), it seems generally agreed that changes are task-dependent. The changes are thought to serve to decrease the force, displacement and velocity of movement (the "pain adaptation model", Lund et al. 1991). Because motor control strategies thought to limit movement have also been reported during stress (Flor and Turk 1989; Flor et al. 1992; Ekberg et al. 1995; Marras et al. 2000), we expected to observe this effect during the non-painful but stressful attention-demanding task. However, we found no effect on spatial parameters of EMG activity of any of the trunk muscles tested. The possibility that there was an increase in activity of muscles that were not investigated, for example rectus abdominis and latissimus dorsi, cannot be excluded.

Although activation of deltoid and the superficial trunk muscles during arm movements was delayed by the attention-demanding task, activation of the deep trunk muscles was not. This result is consistent with previous findings that RT of TrA remains constant despite changes in deltoid RT, when preparation for movement is manipulated (Hodges and Richardson 1999). Together these findings are important because they suggest that postural activation of the deep trunk muscles involves a simplistic and automatic postural response that requires limited CNS information processing. In contrast, postural activation of the superficial trunk muscles is dependent on further CNS processing, including processing of the spatial requirements of the task and selection of a particular movement response. Because pain and non-painful attention demand have opposite effects on postural control of the trunk muscles, it is reasonable to conclude that the effect of pain on postural control of the trunk muscles is independent of the information processing requirements of pain.

The addition of stress to the attention-demanding task had an effect on postural activation of only the deep trunk muscles: there was an increased RT of the deep trunk muscles but not of deltoid or the superficial trunk muscles. Importantly, however, stress did not disrupt the normal temporal relationship between postural activation of the trunk muscles and shoulder movement. It is possible that the subjects were simply more distracted during the stressful task than during the non-stressful task and that an increased attentional demand caused the delay in activation of the deep trunk muscles. If so, we would expect a longer delay in activation of the deltoid muscle and superficial trunk muscles, which was not observed. Thus, the results suggest that when an attention-demanding task is also stressful, it is sufficient to alter the automatic and simplistic postural response of the deep trunk muscles, but it imparts an effect that is different from that caused by pain.

The current paradigm aimed to make subjects distressed and the physiological and subjective data suggest that this did occur. Considering that chronic back pain patients are often characterised by distress (Vlaeyen and Linton 2000), and that postural activation of the deep trunk muscles is disrupted in this group even when they are pain-free (Hodges and Richardson 1996), the current findings raise

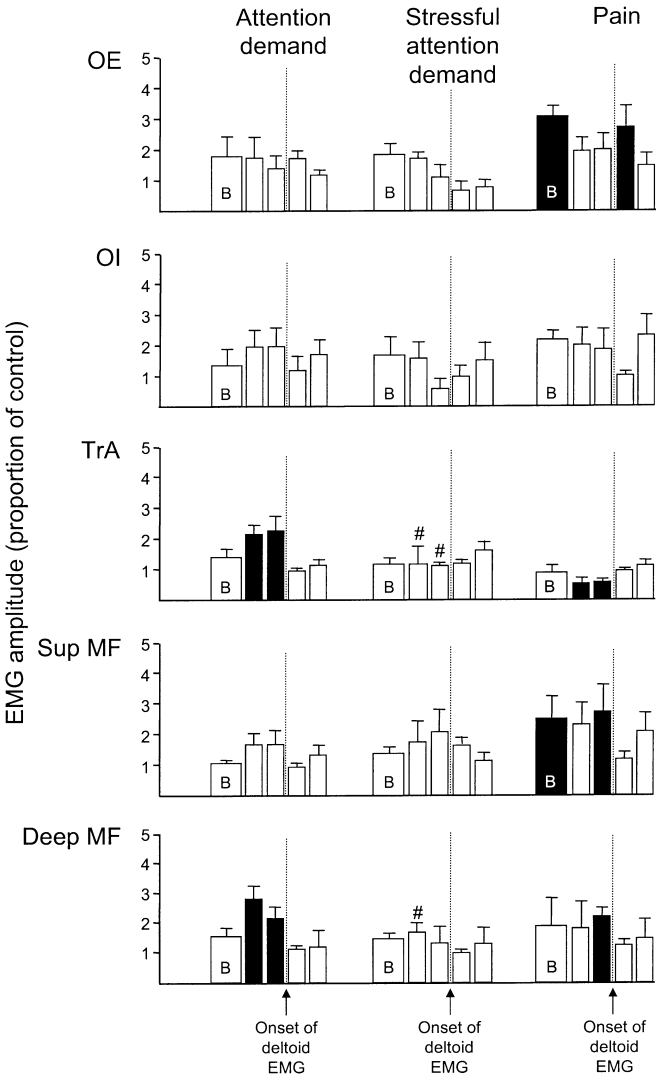

Fig. 5 Group means with SEM for subjects across trials for root mean square EMG at baseline (*B*) and at each 50-ms epoch for 100 ms either side of deltoid onset in each experimental condition, as a proportion of the amplitude during the control condition, for the trunk muscles tested (*OE* obliquus externus, *OI* obliquus internus, *TrA* transversus abdominis, *Sup MF* superficial multifidus, *Deep MF* deep multifidus). *Vertical dashed lines* indicate deltoid onset. *Filled columns* indicate a significant difference to the control condition ($P<0.05$). Note the increased baseline EMG activity for OE, Sup MF and Deep MF during pain. The increase during attention-demand in EMG amplitude for TrA and Deep MF during epochs 1 and 2 corroborate the onset latency data. Note the reduction in EMG amplitude for TrA during epochs 1 and 2 during pain. #Significant difference from the attention-demand condition(note error bars denote SEM not SD)

the possibility that distress may contribute to motor control dysfunction *initiated* by pain. Perhaps context-specific threat [e.g. fear of pain or (re)injury] would impart a greater effect. These proposals are speculative but warrant investigation because according to some models of spinal control, deep trunk muscle dysfunction may lead to stimulation of nociceptors in spinal structures and promote a vicious cycle of pain and motor dysfunction (Panjabi 1992).

In summary, our data show that non-painful attention-demanding task does not replicate the effect of pain on postural activation of the trunk muscles during arm movement, even when the task is also stressful. The results suggest that postural activation of the deep trunk muscles is a simplistic response that requires limited CNS resources. Attention-demand delays the onset of the deltoid (prime mover) and the superficial trunk muscles but does not affect this simplistic response in the deep trunk muscles. When the task becomes stressful, this differential is normalised such that all muscles are equally delayed. The results imply that the effect of LBP on postural activation of the trunk muscles during arm movements is not simply a result of compromised CNS performance caused by the information processing requirements of pain, nor simply a consequence of being under stress.

References

Arena JG, Sherman RA, Bruno GM, Young TR (1989) Electromyographic recordings of 5 types of low back pain subjects and non-pain controls in different positions. Pain 37:57–65

Arendt-Nielsen L, Graven-Nielsen T, Svarrer H, Svensson P (1996) The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. Pain 64:231–240

Aruin AS, Latash ML (1995) Directional specificity of postural muscles in feed forward postural reactions during fast voluntary arm movements. Exp Brain Res 103:323–332

Collins GA, Cohen MJ, Naliboff BD, Schandler SL (1982) Comparative analysis of paraspinal and frontalis EMG, heart rate and skin conductance in chronic low back pain patients and normals to various postures and stress. Scand J Rehabil Med 14:39–46

Crombez G, Eccleston C, Baeyens F, Eelen P (1998a) Attentional disruption is enhanced by the threat of pain. Behav Res Ther 36:195–204

Crombez G, Eccleston C, Baeyens F, Eelen P (1998b) When somatic information threatens, catastrophic thinking enhances attentional interference. Pain 75:187–198

Crombez G, Eccleston C, Baeyens F, van Houdenhove B, van den Broeck A (1999) Attention to chronic pain is dependent upon pain-related fear. J Psychosom Res 47:403–410

Derbyshire SW, Jones AK, Gyulai F, Clark S, Townsend D, Firestone LL (1997) Pain processing during three levels of noxious stimulation produces differential patterns of central activity. Pain 73:431–445

Derbyshire SW, Vogt BA, Jones AK (1998) Pain and Stroop interference tasks activate separate processing modules in anterior cingulate cortex. Exp Brain Res 118:52–60

Eccleston C, Crombez G (1999) Pain demands attention: a cognitive-affective model of the interruptive function of pain. Psychol Bull 125:356–366

Ekberg K, Eklund J, Tuvesson M (1995) Psychological stress and muscle activity during data entry at visual display units. Work Stress 9:475–490

Flor H, Turk DC (1989) Psychophysiology of chronic pain: do chronic pain patients exhibit symptom-specific psychophysiological responses? Psychol Bull 105:215–259

Flor H, Birbaumer N, Schugens MM, Lutzenberger W (1992) Symptom-specific psychophysiological responses in chronic pain patients. Psychophysiol 29:452–460

Hodges PW (2001) Changes in motor planning of feedforward postural responses of the trunk muscles in low back pain. Exp Brain Res 141:261–266

Hodges PW, Richardson CA (1996) Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. Spine 21:2640–2650

Hodges PW, Richardson CA (1997) Relationship between limb movement speed and associated contraction of the trunk muscles. Ergonomics 40:1220–1230

Hodges PW, Richardson CA (1999) Transversus abdominis and the superficial abdominal muscles are controlled independently in a postural task. Neurosci Lett 265:91–94

Hodges PW, Gandevia SC, Richardson CA (1997) Contractions of specific abdominal muscles in postural tasks are affected by respiratory maneuvers. J Appl Physiol 83:753–760

Hodges PW, Moseley GL, Gabrielsson A, Gandevia SC (2003) Experimental muscle pain changes feedforward postural responses of the trunk muscles. Exp Brain Res 151:262–271

Hoyt W, Hunt H, DePauw M, Bard D, Shaffer F, Passias J, Robins D, Runyon D, Semrad S, Symonds J, Watt K (1981) Electromyographic assessment of chronic low back pain syndrome. J Am Osteopath Assoc 80:57–59

Jones G, Cale A (1997) Goal difficulty, anxiety and performance. Ergonomics 40:319–333

Kuukkanen T, Malkia E (1998) Effects of a three-month active rehabilitation program on psychomotor performance of lower limbs in subjects with low back pain: a controlled study with a nine-month follow-up. Percept Mot Skills 87:739–753

Lorenz J, Bromm B (1997) Event-related potential correlates of interference between cognitive performance and tonic experimental pain. Psychophysiology 34:436–445

Lorenz J, Beck H, Bromm B (1997a) Cognitive performance, mood and experimental pain before and during morphine induced analgesia in patients with chronic non-malignant pain. Pain 73:369–375

Lorenz J, Beck H, Bromm B (1997b) Differential changes of laser evoked potentials, late auditory evoked potentials and P300 under morphine in chronic pain patients. Electroencephalogr Clin Neurophysiol 104:514–521

Lund JP, Donga R, Widmer CG, Stohler CS (1991) The pain-adaptation model: a discussion of the relationship between chronic musculoskeletal pain and motor activity. Can J Physiol Pharmacol 69:683–694

Luoto S, Aalto H, Taimela S, Hurri H, Pyykko I, Alaranta H (1998) One-footed and externally disturbed two-footed postural control in patients with chronic low back pain and healthy control subjects. A controlled study with follow-up. Spine 23:2081–2089

Luoto S, Taimela S, Hurri H, Alaranta H (1999) Mechanisms explaining the association between low back trouble and deficits in information processing. A controlled study with follow-up. Spine 24:255–261

Marras WS, Davis KG, Heaney CA, Maronitis AB, Allread WG (2000) The influence of psychosocial stress, gender, and personality on mechanical loading of the lumbar spine. Spine 25:3045–3054

Moseley GL, Hodges PW, Gandevia SC (2002) Deep and superficial fibers of the lumbar multifidus muscle are differentially active during voluntary arm movements. Spine 27: E29–E36

Nouwen A, Bush C (1984) The relationship between paraspinal EMG and chronic low back pain. Pain 20:109–123

Panjabi MM (1992) The stabilizing system of the spine. Part II. Neutral zone and instability hypothesis. J Spinal Disord 5:390–396

Price DD (2000) Psychological mechanisms of pain and analgesia. IASP Press, Seattle

Rosenfeld JP, Johnson MM, Koo J (1993) Ongoing ischemic pain as a workload indexed by P3 amplitude and latency in real- versus feigned-pain conditions. Psychophysiology 30:253–260

Spielberger C, Gorsuch R, RD L (1970) STAI Manual for the state-trait anxiety inventory. Consulting Psychologists Press, Palo Alto CA

van Galen GP, van Huygevoort M (2000) Error, stress and the role of neuromotor noise in space oriented behaviour. Biol Psychol 51:151–171

Venna S, Hurri H, Alaranta H (1994) Correlation between neurological leg deficits and reaction time of upper limbs among low-back pain patients. Scand J Rehabil Med 26:87–90

Vlaeyen JW, Linton SJ (2000) Fear-avoidance and its consequences in chronic musculoskeletal pain: a state of the art. Pain 85:317–332

Vlaeyen JW, Seelen HA, Peters M, de Jong P, Aretz E, Beisiegel E, Weber WE (1999) Fear of movement/(re)injury and muscular reactivity in chronic low back pain patients: an experimental investigation. Pain 82:297–304

Weinberg RS, Hunt VV (1976) The interrelationships between anxiety, motor performance and electromyography. J Mot Behav 8:219–224

Zedka M, Prochazka A, Knight B, Gillard D, Gauthier M (1999) Voluntary and reflex control of human back muscles during induced pain. J Physiol (Lond) 520:591–60