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RESEARCH NOTE

Paul W. Hodges

Changes in motor planning of feedforward postural responses of the trunk muscles in low back pain

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Abstract Changes in trunk muscle recruitment have been identified in people with low-back pain (LBP). These differences may be due to changes in the planning of the motor response or due to delayed transmission of the descending motor command in the nervous system. These two possibilities were investigated by comparison of the effect of task complexity on the feedforward postural response of the trunk muscles associated with rapid arm movement in people with and without LBP. Task complexity was increased by variation of the expectation for a command to either abduct or flex the upper limb. The onsets of electromyographic activity (EMG) of the abdominal and deltoid muscles were measured. In control subjects, while the reaction time of deltoid and the superficial abdominal muscles increased with task complexity, the reaction time of transversus abdominis (TrA) was constant. However, in subjects with LBP, the reaction time of TrA increased along with the other muscles as task complexity was increased. While inhibition of the descending motor command cannot be excluded, it is more likely that the change in recruitment of TrA represents a more complex change in organisation of the postural response.

Keywords Low-back pain · Postural control · Lumbar spine · Motor control · Feedforward

Introduction

Changes in muscle control have been identified in people with clinical pain syndromes (Hodges and Richardson 1996; Wilder et al. 1996) and when pain is induced in an experimental setting by injection of substances, such as hypertonic saline, into limb and trunk muscles (e.g., Svensson et al. 1995; Arendt-Nielsen et al. 1996; Stohler et al. 1996; Zedka et al. 1999b). Although it is accepted that muscle function is altered by pain, the mechanism for these changes is poorly understood.

Pain may affect the motor output at any level of the nervous system, including peripheral, spinal and supraspinal structures. For instance, changes in regional cerebral blood flow in motor and premotor areas [e.g. anterior cingulate cortex, premotor cortex (Derbyshire et al. 1997)] and changes in spinal reflexes (Svensson et al. 2000) have been reported as a result of experimentally-induced pain. In studies of natural movements it is generally not possible to speculate on the mechanisms for altered motor behaviour. However, recent studies have identified a strategy used by the central nervous system to coordinate the postural response of the trunk muscles that could provide insight into this mechanism (Hodges and Richardson 1997; Hodges and Richardson 1999b).

During limb movements, activity of trunk muscles generally occurs in advance of the movement to prepare the spine for the perturbation that results from the associated reactive moments (Bouisset and Zattara 1981; Aruin and Latash 1995; Hodges and Richardson 1997). Transversus abdominis (TrA), the deepest of the abdominal muscles, is the first trunk muscle active regardless of the direction of limb movement (Hodges and Richardson 1997). These responses are considered to be "feedforward" as they occur in advance of the limb movement (Belenkii et al. 1967; Bouisset and Zattara 1981). Thus, feedforward responses are pre-planned by the central nervous system (Massion 1992). However, the response of TrA is delayed (Hodges and Richardson 1996) or absent (Hodges and Richardson 1999a) when people have chronic recurrent low-back pain (LBP). Hypothetically, this change could occur if either "planning" of the motor response is altered, or the transmission of the descending drive to the motoneuron is inhibited at some level(s) of the nervous system. While it is not possible to distinguish between these possibilities from simple analysis of the

P.W. Hodges (✉)
Prince of Wales Medical Research Institute,
Barker Street Randwick, Sydney, NSW 2031, Australia
e-mail: p.hodges@shrs.uq.edu.au
Tel.: +61-7-33652008, Fax: +61-7-33652775

Current address:
P.W. Hodges, Department of Physiotherapy,
The University of Queensland, Brisbane, QLD 4072, Australia

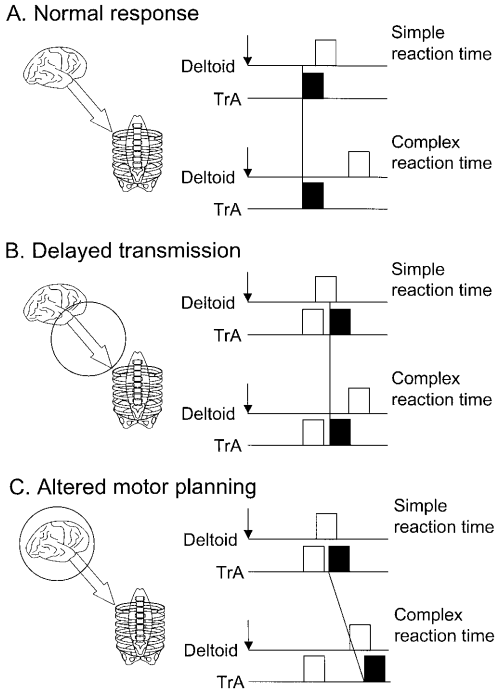

Fig. 1 Hypothetical alternatives for the affect of pain on trunk muscle activity. **A** When the reaction time for arm movement is increased in people without LBP by increased task complexity, the reaction time of the muscle responsible for limb movement is increased but that of TrA remains unchanged. The stimulus to move is indicated by the arrow. **B** If the delay in onset of activity of TrA in chronic LBP is due to delayed transmission of the descending command in the nervous system it may be expected that the onset of TrA would be delayed, but the general strategy would remain unchanged (i.e. the response of TrA would maintain a constant temporal relationship to the movement stimulus between conditions). The *dotted boxes* indicate the response of TrA in a person without LBP (i.e. as for panel A). **C** However, if the response of TrA does not maintain a constant temporal relationship to the movement stimulus in conjunction with an increased reaction time for arm movement this would suggest a change in strategy rather than a simple delay in transmission of the descending command

change in trunk muscle recruitment with LBP, an additional feature of the feedforward response of TrA may permit further interpretation of this mechanism. In people with no history of LBP the onset of electromyographic activity (EMG) of TrA does not vary when the reaction time of limb movement is varied by changes in task complexity (Hodges and Richardson 1999b). This is demonstrated in Fig. 1 A. If the change in TrA recruitment in LBP is due to impaired transmission of the descending drive in the nervous system, this would lead to delayed reaction time of TrA, but the reaction time would remain similar between trials, irrespective of changes in the reaction time of the movement (Fig. 1B). In contrast, a change in motor planning would be indicated if the reaction time of TrA was delayed *and* it varied along with variation in movement reaction time in association with changes in task complexity (Fig. 1C). The aim of the present experiment was to investigate these alternatives.

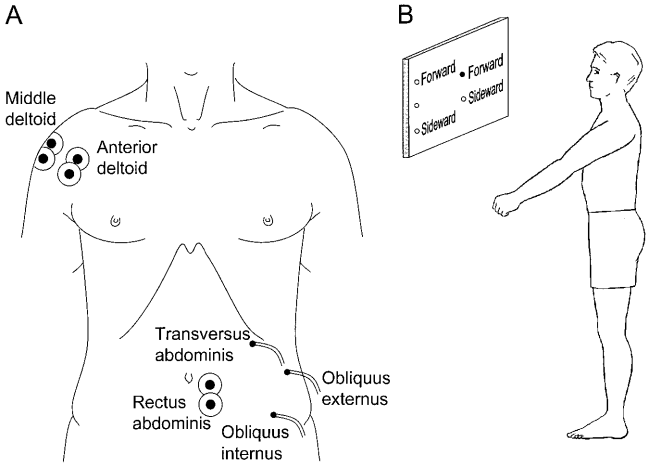

Fig. 2 Methods. **A** Sites for EMG electrode placement. **B** Upper limb movement task (flexion) shown with the set-up for the visual information of expectation and movement to be performed

Subjects and methods

Subjects

Fourteen subjects with a history of chronic recurrent LBP and 14 age- (±3 years) and sex-matched control subjects participated in the study. The subjects for the LBP group were selected on the basis of clinical criteria. They were to have had episodic LBP for at least 18 months that was of sufficient intensity to limit function and for which the subjects sought medical or allied health intervention, and at least one episode of pain each 12 months. Subjects were tested when they were pain-free and not taking any pain-relieving medication. Subjects had pain for 9±8 years, with 10±9 episodes per year of 15±23 days duration. All subjects were medically screened prior to inclusion in the study to exclude any non-musculoskeletal aetiology for their symptoms. In addition, subjects were excluded if they had chronic, unremitting pain, neurological symptoms, pain extending beyond the gluteal fold, abdominal or spinal surgery, recent pregnancy, visual impairment that would preclude the use of a visual stimulus or any neurological or respiratory condition. The control subjects had no history of LBP that had limited function or for which they had sought medical intervention. Subjects were excluded from the study if they had any history of respiratory or neurological conditions. The control and LBP subjects scored 8.9±0.4 and 8.5±0.3, respectively, on a standard activity questionnaire (Baecke et al. 1982). This indicates an average activity level, and the two groups were not different when assessed with Student's *t*-test for independent samples ($P<0.05$). The mean (±SD) age, height and weight of the control and LBP subjects were 29±2 years, 1.72±0.03 m, 66±3 kg and 30±2 years, 1.74 ± 0.02 m, 63 ± 8 kg, respectively. There were no differences in any of these parameters between groups ($P<0.05$). Subjects were naive to the purpose of the study. The study was approved by the institutional Medical Research Ethics Committee.

Electromyographic recordings

EMG recordings were made from the left abdominal muscles and the anterior and middle portions of the right deltoid as the prime movers of shoulder flexion and abduction, respectively. Bipolar fine-wire electrodes were inserted into the TrA, obliquus externus abdominis (OE) and obliquus internus abdominis (OI) under the guidance of ultrasound imaging using a 5 MHz curved array transducer (ATL, USA) (DeTroyer et al. 1990; Hodges and Richardson 1997) (Fig. 2 A). Pairs of Ag/AgCl surface electrodes were placed 32 mm apart over the muscle bulk of the anterior and

middle portions of deltoid and rectus abdominis (RA) following careful skin preparation (Fig. 2A). EMG was bandpass filtered between 20 Hz and 1 kHz and sampled at 2000 Hz (AMLAB, Associative measurements, Australia). The onset of EMG was identified using a computer algorithm as the point at which the EMG amplitude for the subsequent 50 consecutive samples were 3 SD from the mean of the baseline amplitude recorded for 50 ms prior to a warning light (Hodges and Bui 1996). Each computer-derived EMG onset was checked visually. For visual checking each unrectified, raw EMG trace was displayed individually without reference to the muscle, condition or any parameter that gave reference to the onset of movement or deltoid EMG. The onset was identified visually as the point at which the EMG onset deviated from the baseline. As the subjects were instructed to stand in a relaxed posture in the majority of trials this involved an increase from a silent background. Less than 10% of onsets were changed following visual inspection, and less than 5% were rejected due to an inability to be confident of the onset as a result of movement artefact, electrocardiogram or high background activity. The reaction time from the stimulus to EMG onset and the latencies between the onset of deltoid EMG and that of the abdominal muscles were analysed.

Procedure

Standing subjects performed flexion (Fig. 2B) or abduction of the right shoulder as fast as possible through approximately 60° from the resting position with the arm beside the body. Subjects stood without shoes with their feet shoulder width apart on a force plate (Balance performance Monitor, SMS Healthcare, UK) that provided auditory feedback if they failed to maintain equal weight (±4%) on each foot. Movement was performed in response to a visual stimulus that indicated the direction of movement required. Subjects were instructed to respond as quickly as possible when they saw the stimulus to move which was provided by one of two green lights, one labelled "forward" and the other "sideward" (Fig. 2B). The reaction-time for limb movement was varied by manipulation of the degree of expectation of which movement direction would be required. Expectation was varied by illumination of one of three red warning lights 0.5–4 s before the stimulus to move. The warning lights were positioned to the left of the movement light that provided an indication of the likelihood of which movement light would be illuminated (Fig. 2B). Illumination of the uppermost light indicated an 80% probability that the subject would be required to perform flexion, and the bottom light indicated an 80% probability of abduction. The trials in which preparatory information indicated the same direction of movement as the movement stimulus were referred to as the *correct* preparation condition. In 20% of trials the flexion or abduction preparatory light was followed by the opposite stimulus to move. This was the *incorrect* preparation condition. Subjects were instructed that when the flexion or abduction light was illuminated there would be an 80% chance that it would be correct and they were to assume that it would be correct. The middle preparatory light indicated a 50% probability of either movement direction, i.e. the *neutral* preparation condition. The probabilities were similar to those used previously (Brown and Frank 1987; Hodges and Richardson 1999b).

Twenty practice trials were completed followed by 72 trials comprising an equal number of trials in each direction in the proportion of 22:40:10 for the *neutral*, *correct* and *incorrect* conditions. Many trials were performed in order to maintain the appropriate proportions (i.e. 80% probability of correct preparation when direction information was provided) of each condition and to reduce the possibility that the subject could anticipate the preparatory condition. Twenty-four trials (4 trials in each condition for each movement direction) were retained for data analysis. The order of these trials was randomised and separated by two "dummy" trials that were randomly selected from the remaining pool of trials. Identical instructions were given for the practice and experimental trials. This complex trial organisation was implemented for two reasons. First, it was necessary for the *correct* trials to outnumber the *incorrect* trials by 4:1, and it was considered important that the same number of trials be analysed for each condition. In order to avoid fatigue, subjects rested for a minimum of 20 s between each trial and rested in sitting for 2 min between each block of 24 trials.

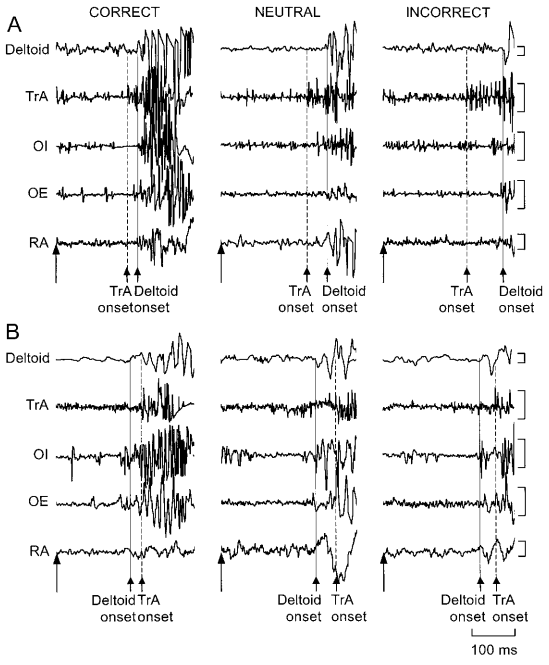

Fig. 3 Representative raw electromyographic (EMG) recordings from a control subject (**A**) and a subject with chronic recurrent LBP (**B**). Responses are shown for all muscles in each of the preparatory conditions for trials of shoulder flexion. The *solid and dashed lines* denote the onsets of deltoid and TrA EMG, respectively. The *large arrow* at the bottom left of each panel indicates the time of the movement stimulus. Note that the reaction time of TrA did not increase along with that of deltoid, RA, OE and OI as the preparation for movement decreased for the control subjects. However when people with LBP performed the same task the reaction time of TrA was increased with the other muscles. EMG calibration: 100 μV

Statistical analysis

For each group the reaction time and latency between the onset of EMG of each muscle were compared between conditions using a repeated measures one-way analysis of variance (ANOVA) and Duncan's multiple range test. The flexion and abduction data were combined for the analysis. In a previous study, only OE displayed a direction specific variation between these two movements (Hodges and Richardson 1997), and comparison of OE between preparatory conditions separately for each direction using identical statistics revealed the same relationship as the combined data. Significance was set at α=0.05.

Results

Control subjects

When subjects rapidly moved an upper limb in response to a visual stimulus, the onset of TrA EMG preceded that of deltoid in all conditions (Figs. 3A, 4A, B). In contrast, the EMG onsets of OI, OE and RA followed that of deltoid (Figs. 3A, 4A, B). When subjects were prepared

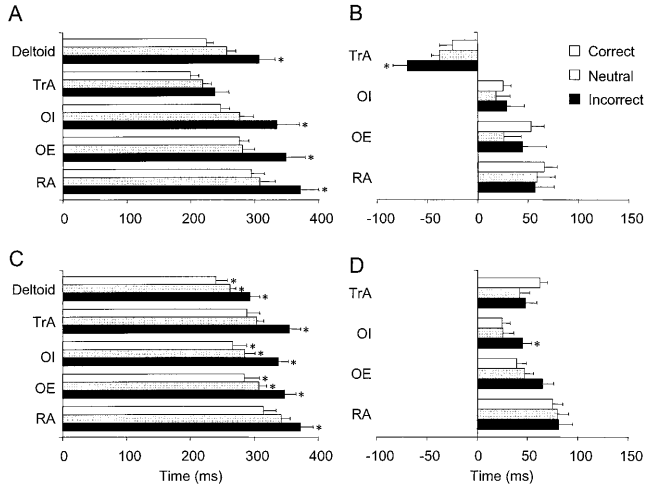

Fig. 4 Mean (SEM) reaction times for the control (**A**) and LBP (**C**) subjects and the latency between the onset of the trunk muscle EMG and that of deltoid for the control (**B**) and LBP subjects (**D**) are shown. Data for flexion and abduction have been combined. In (**A**) and (**C**) the stimulus is at time zero. Note the increased reaction time of deltoid, RA, OE and OI with decreased movement preparation (**A**) and the constant temporal relationship between the onsets of RA, OE and OI EMG and that of deltoid (**B**) for the control subjects. The reaction time of TrA did not change with the other muscles between conditions (**A**) and, thus, the latency between the onset of TrA EMG and that of deltoid increased with decreased preparation (**B**). When the LBP subjects performed the same task, the reaction time of TrA increased along with the other trunk muscles (**C**) and the latency between the onset of TrA EMG and that of deltoid remained constant between conditions (**D**). The '*' indicates a significant difference between conditions for a muscle with $P<0.05$.

in a *neutral* or *incorrect* manner, the reaction time from stimulus to move to the onset of deltoid EMG was increased compared with movements following *correct* preparation (Fig. 3 A). The reaction time of deltoid EMG was different between the *neutral* and *incorrect* preparation conditions.

Consistent with previous data (Hodges and Richardson 1999b), the reaction time of TrA was not influenced by preparation. Figures 3A and 4A show that the reaction time between the movement stimulus and onset of TrA EMG was not significantly influenced by different preparatory conditions, despite changes in deltoid reaction time. Furthermore, the latency between the onset of EMG of TrA and that of deltoid was increased when the subjects were incorrectly prepared (Fig. 4B).

Unlike TrA, the reaction times of RA, OE and OI were influenced by preparation along with deltoid. Figure 4A shows that when subjects were incorrectly prepared, the reaction times of these muscles were delayed compared with the *correct* preparation condition. There was no difference between *correct* and *neutral* conditions. The latencies between the onset of deltoid EMG and that of RA, OE and OI were not different between conditions (Fig. 4B). The influence of movement preparation on the reaction times of all muscles was consistent for both the flexion and abduction data, and all subjects responded in a similar manner.

LBP subjects

Similar to the control group, the reaction time of deltoid increased as the level of movement preparation decreased in the LBP subjects (Figs. 3B, 4C). However, several differences in the response of TrA were identified in the LBP group compared to the control subjects. First, the reaction time of TrA was significantly increased following incorrect preparation (Figs. 3B, 4C). Thus, the response of TrA did not maintain a consistent temporal relationship with the stimulus to move, but instead the latency between the onset of EMG of TrA and that of deltoid remained constant between the preparatory conditions (Figs. 3B, 4C). This change in coupling of the TrA response from the movement stimulus to the initiation of deltoid EMG was consistent for all subjects. Secondly, the onset of EMG of TrA failed to precede that of deltoid with movement in any of the preparatory conditions (Figs. 3B, 4D).

Similar to the control subjects the reaction times of RA, OE and OI increased with decreased preparation. Figures 3B and 4C show that when subjects were incorrectly prepared, the reaction time of each muscle was delayed compared with the correct preparation condition. The reaction times of OE and OI were also different between the correct and neutral preparation conditions (Fig. 4C). The latencies between the onset of deltoid EMG and that of RA and OE did not change with variation in preparation (Fig. 4D). In contrast to the control group the latency between the onset of deltoid and that of OI was shorter with neutral preparation (Fig. 4D). Thus, in general terms the responses of RA, OE and OI were similar to those identified for the control group, with only one minor exception.

Discussion

These results support the hypothesis that the organisation of the trunk muscle response is altered in chronic recurrent LBP. The data show that the change in recruitment of the deep abdominal muscle, TrA, cannot be explained by a delay in the transmission of the descending motor command in the central nervous system alone. The reaction time of TrA EMG was not simply delayed, but varied with the changes in reaction time for limb movement. This is in contrast to the control subjects in whom the reaction time of TrA EMG was constant despite the variation in movement reaction time. While these data cannot exclude the possibility that the transmission of the motor command to the motoneuron may have *also* been delayed, such a delay cannot explain the complexity of the change in motor strategy identified in the trunk muscles of chronic pain patients.

Differential control of the abdominal muscles in people without LBP

Consistent with previous studies, the present data indicate that TrA is controlled independently of the other

abdominal muscles for postural control of the trunk in people without LBP (Hodges and Richardson 1999b). Although the reaction time of several trunk (Hodges and Richardson 1999b; present data) and limb muscles (Brown and Frank 1987) have been shown to maintain a constant temporal relationship to the muscle responsible for limb movement (i.e. their reaction time varies with the limb movement reaction time), the latency between the onset of TrA and deltoid EMG varied significantly between conditions (i.e. the reaction time of TrA remained relatively unchanged). This finding suggests that the recruitment of TrA is not coupled temporally to the command for limb movement, whereas the activity of the other muscles may be organised in either a "hierarchical" or "parallel" manner with the descending command for limb movement (Massion 1992).

Changes in trunk muscle recruitment with LBP

Numerous changes in the recruitment of trunk muscles with LBP have been reported in the literature. In general terms these changes are consistent with either augmentation or impairment of the motor output to trunk muscles. For example, studies have shown reduced force output during maximal voluntary contraction (Alston et al. 1966; Thorstensson and Arvidson 1982), decreased erector spinae activity in static positions (Collins et al. 1982) and delayed recruitment of TrA with arm movements (Hodges and Richardson 1996). In contrast, other studies have reported increased activity of the erector spinae at rest (Jalovaara et al. 1995) and at the end of range of static trunk flexion in standing (Triano and Schultz 1986; Ahern et al. 1988; Arena et al. 1989), delayed relaxation of erector spinae and OE with unexpected removal of a load from the trunk (Radebold et al. 2000)and tonic activity in response to unexpected addition of a load (King et al. 1988). Yet, other studies report both increased and decreased activity during gait and voluntary trunk movements. In general, these studies report reduced erector spinae activity when the muscle is active as an agonist [i.e. voluntary trunk extension from a flexed position (Zedka et al. 1999b), and double-stance phase in gait (Arendt-Nielsen et al. 1996)]. In the same tasks, erector spinae has increased activity when this muscle is normally silent or minimally active [i.e. full-trunk flexion (Zedka et al. 1999b), and swing phase in gait (Arendt-Nielsen et al. 1996)]. While this complex combination of both facilitory and inhibitory influences has been argued to be due to interaction between nociceptive afferents and the motoneuron pool (Lund et al. 1991), it is difficult to establish the mechanism for these changes.

The present data provide evidence that changes in trunk muscle recruitment may result from alteration of the planning of the postural response rather than facilitation or inhibition of the transmission of the descending motor command. The present data represent a change in strategy from "locking" of the response of TrA to the stimulus to move (constant reaction time;Fig. 1A) to "locking" of the TrA response to the reaction time of the motor command for limb movement (i.e. constant latency between onset of deltoid and TrA EMG, and variable reaction time; Fig. 1C). While few studies have investigated changes in motor planning (e.g. Luoto et al. 1999), recent studies report no change in excitability of the motor cortex or alpha motoneuron and no change in corticospinal transmission following stimulation of small diameter afferents by injection of hypertonic saline into the jaw or upper arm (Zedka et al. 1999a; Romaniello et al. 2000) or with ischaemia after prolonged maximal contraction of the elbow flexor muscles (Gandevia et al. 1996; Butler et al. 1999). However, changes were present in recruitment of the painful muscle during a movement task (Zedka et al. 1999b). Thus, it has been argued that the changes in motor output may be the result of changes "upstream" of the motor cortex (Zedka et al. 1999a). The present data are consistent with such a change.

The present data do not provide evidence of the location(s) at which pain may affect motor planning or its mechanism. However, numerous studies have reported increased regional blood flow in areas of the CNS involved in motor planning (Ahern et al. 1988); see Derbyshire et al. [(1997) for a review], and many mechanisms have been postulated for how pain may affect motor planning. For example, factors such as altered attention (Eccleston 1994; Luoto et al. 1999) and fear avoidance (Biederman et al. 1991) have been implicated. Investigation of these possibilities is the subject of further study. Regardless of the mechanism, these data have implications for rehabilitation of patients with LBP. The identification of changes in motor planning in people with LBP suggests that rehabilitation strategies must be directed at retraining the complex strategy for coordination of the trunk muscles.

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