

People With Recurrent Low Back Pain Respond Differently to Trunk Loading Despite Remission From Symptoms

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Study Design. Cross-sectional design.

Objective. To compare lumbar multifidus electromyographic activity (EMG) during predictable and unpredictable trunk loading between people with and without recurrent unilateral low back pain (LBP) during symptom remission.

Summary of Background Data. Unpredictable loading is a common injury mechanism for LBP. Paraspinal muscle responses to trunk loading differ between people with and without a history of LBP, but whether the response differs between specific regions within the paraspinal muscles is unclear. Differences between deep (DM) and superficial fibers (SM) of multifidus have been implicated in other tasks. It is unknown whether DM and SM EMG differ between people in remission from recurrent LBP and healthy people during trunk loading.

Methods. DM and SM EMG was recorded bilaterally at L5 with intramuscular electrodes during predictable and unpredictable trunk loading and compared during 10 milliseconds epochs (250 milliseconds before to 150 milliseconds after loading) between sides, loading conditions, and groups.

Results. DM EMG increased above baseline before and after predictable load onset, but returned to baseline at the time of impact. Both DM EMG bursts were less in the remission group and less on the non-painful side. Peak SM EMG amplitude on the previously painful side was earlier in the remission group than healthy participants. DM and SM EMG were less after unpredictable load onset in the remission group than healthy participants.

Conclusion. Despite symptom remission, DM EMG during predictable loading and DM and SM EMG during unpredictable loading were less in people with recurrent LBP than healthy participants.

Key words: low back pain, recurrence, multifidus, loading, electromyography. *Spine* 2010;35:818–824

Sudden and/or unpredictable trunk loading is a common injury mechanism in low back pain (LBP).^{1–3} Excessive lumbar spine strain in unaccustomed postures,¹ excessive back muscle response⁴ leading to increased spinal loading,⁵ delayed back muscle responses,⁶ and differential changes in deep and superficial back muscle control⁷ are argued to increase the risk of spinal injury from unpredictable trunk loading. Although back muscle responses to trunk loading have been investigated in people with and without LBP, it remains unclear if there are differences between deep and superficial back muscles during LBP or if changes are observed between episodes of recurrent LBP.

Lumbar paraspinal muscle responses to trunk loading differ between people with and without a history of LBP. People with LBP have longer paraspinal muscle reaction times (longer on the painful side than non-painful side),⁶ decreased paraspinal muscle response amplitude⁶ and changes in the latency of the paraspinal muscle response relative to abdominal muscles⁸ during unpredictable trunk loading. Further, unlike the shorter paraspinal muscle reaction time in healthy participants during predictable trunk loading, load predictability has no effect on trunk muscle reaction time in LBP.⁹ Two issues make interpretation of these findings difficult. First, participants were either in pain during testing and the direct effect of pain on muscle responses cannot be excluded, or second, recordings were made with surface electromyography (EMG), which makes it impossible to draw conclusions about activity of specific muscles.^{10,11}

Investigation of the response of specific paraspinal muscles, and their discrete fascicles, to trunk loading is important because paraspinal muscles do not contribute equally to the control of spinal motion nor are they uniformly affected by LBP. Within the paraspinal muscles, the lumbar multifidus contributes up to 2/3 of the stiffness at individual lumbar segments¹² and has a high cross-sectional area and a low fiber length-to-muscle length ratio, making it uniquely designed to generate high forces.¹³ Within the multifidus, the short/deep (DM) and long/superficial (SM) fibers are thought to contribute uniquely to the control of spinal motion by virtue of anatomic and biomechanical differences. For example, SM fibers cross from 2 to 5 lumbar segments, whereas DM fibers cross only 2.^{14–16} SM is capable of generating

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sufficient torque to produce spinal extension, whereas DM is located closer to the predicted instantaneous axis of rotation of the lumbar segments and therefore likely to produce primarily compressive forces.^{17,18} The basis for specific investigation of the paraspinal muscles is further supported by morphological^{19,20} and histochemical^{21,22} evidence that multifidus, rather than adjacent paraspinal muscles, is more affected in LBP.²³ Furthermore, human^{19,21} and porcine²⁰ data suggest morphologic changes are most evident in components of multifidus adjacent to the painful/injured spinal segment and that those changes are present ipsilateral to symptoms. Specific alterations in control have been observed in DM but not SM in people with recurrent unilateral LBP during remission from symptoms compared to healthy people. DM activity, but not SM, is delayed in people with recurrent unilateral LBP and the delay is greater on the previously painful side than the non-painful side during a single rapid arm movement.²⁴ Although EMG investigation of the lumbar multifidus components is justified, and unpredictable loading is a common LBP injury mechanism, it remains unknown if DM and SM EMG activity associated with trunk loading differs between people with a history of recurrent LBP (during symptom remission) and healthy participants.

Investigation of paraspinal muscle control in people with recurrent LBP during symptom remission is important because if changes in control persist after resolution of symptoms, they may contribute to recurrence of LBP.²⁴ On the basis of previous data,²⁴ we hypothesized that (1) DM EMG would be less in people with a history of recurrent unilateral LBP than in healthy participants in response to sudden trunk loading; (2) DM EMG would be less on the previously painful side than the non-painful side; and (3) there would be no difference in SM EMG activity between groups.

Materials and Methods

Participants

A total of 13 people with recurrent unilateral LBP, with multiple episodes of LBP separated by periods of remission (6 male, mean [SD] age: 29 [7] years, height: 171 [9] cm, and weight: 71 [14] kg), and 14 healthy participants [8 male, age: 26 [5] years, height: 174 [10] cm, and weight: 68 [12] kg)

with no history of LBP sufficient to limit function participated in the study.

Participants with LBP history were to have experienced recurrent, intermittent, unilateral symptoms (present on one side >75% of the time) between T12 and the gluteal fold. Each participant was examined by an experienced clinician to ensure an asymmetrical pattern of movement and/or symptom reproduction consistent with a history of unilateral LBP. The physical examination evaluated the active and passive range of motion, neurologic screening, and palpation for tenderness in the lumbar spine. Symptoms were to be present for a minimum of 3 months with a severity sufficient to require medical or allied health intervention and impair the participant's ability to perform activities of daily living. Participants were to be in a period of remission from their recurrent LBP symptoms.

Healthy participants had no LBP in the 2 years before the study and no history of LBP, before that period, which required intervention or limited function. Participants were excluded from either group if that had spinal surgery, major spinal deformities, respiratory or neurologic conditions, or any orthopedic condition that would have limited participant's ability to complete the study.

Written informed consent was obtained. All procedures were approved by the Institutional Research Ethics Committee and conducted in accordance with the Declaration of Helsinki.

Electromyography

Bilateral recordings of DM and SM EMG activity were made at the level of the lamina of L5 using bipolar intramuscular electrodes using a previously established protocol.²⁵ Two Teflon-coated 75 μ m stainless steel wires were inserted into DM and SM, via a hypodermic needle (0.7 \times 50 mm or 0.6 \times 38 mm), with ultrasound guidance (5-MHz linear array transducer, Synergy CFM; Dasonics, Haifa, Israel). Participants were positioned in either supported sitting or side lying for electrode insertion. The L5 vertebral lamina and target muscle were clearly identified. DM electrodes were inserted ~30 mm lateral to the midline and directed anteromedially until the needle tip reached the medial aspect of the L5 lamina. SM electrodes were inserted ~40 mm lateral to the midline and directed anteromedially until the needle tip was visualized in the muscle (Figure 1A). After needle removal, gentle traction of the wires under ultrasound visualization confirmed each electrode's position. Participants reported only mild transient discomfort during insertion and were pain-free during the experiment.

The ground electrode was placed over the right iliac crest. EMG data were amplified 2000 times, band pass filtered be-

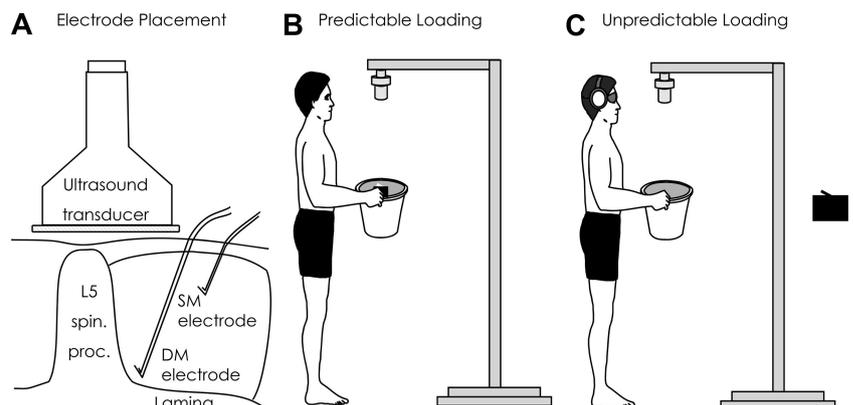


Figure 1. **A**, Intramuscular electrodes were inserted under ultrasound guidance into deep (DM) and superficial multifidus (SM) at the level of the L5 lamina. DM and SM electromyographic activity were recorded during **(B)** predictable and **(C)** unpredictable loading conditions.

tween 30 Hz and 1 kHz, and sampled at 2 kHz using a Power 1401 and Signal software (Cambridge Electronic Design, Cambridge, UK). Data were exported for analysis with Matlab 7.0.1 (Mathworks, Natick, MA).

Procedure

In relaxed standing, participants held a plastic bucket with a metal bottom in both hands with their elbows flexed to 90° and their upper arms placed against their sides. A mass (~1 kg) was released from their eye height into the bucket. Participants were instructed to catch the mass in the bucket and “maintain their arm position.” This instruction aimed to maximize force transfer to the trunk. Contact of the mass with the bucket’s metal bottom initiated EMG recording. Ten trials were recorded during (1) predictable and (2) unpredictable loading. During predictable loading, participants used a hand-held trigger to release the mass (Figure 1B). During unpredictable loading, the investigator released the mass at random intervals with no prior warning and participants wore a blindfold and headphones that provided white noise (Figure 1C). Data were collected from 1 second before load and 0.5 seconds after load.

Data Analysis

Root mean square (RMS) EMG amplitude was calculated in 10-millisecond epochs between 250 milliseconds before, and 150 milliseconds after the load onset during. EMG data were not normalized to a maximum voluntary contraction (MVC) because it has been suggested that LBP patients do not perform MVC’s to their true maximum²⁶ and this is likely to increase the variability in the data more than the potential error associated with analysis of non-normalized data.

Statistical Analysis

To test the hypotheses that DM RMS EMG would be less in people with a history of recurrent unilateral LBP than healthy participants and that DM RMS EMG would be less on the previously painful side than the non-painful side in the recurrent group during sudden trunk loading, a repeated measures analysis of variance (ANOVA) was performed with 2 within subject factors: Condition (predictable and unpredictable) and Epoch (epochs, 1–40), and one between subject factor: Group (3 levels: previously painful side in the recurrent group, non-painful side in the recurrent group, and healthy participants). Data from both sides in healthy participants were pooled because an initial ANOVA indicated that there was no difference between sides (main effect: Side, $P = 0.361$) for DM RMS EMG in either loading condition (interaction: Condition \times Side, $P = 0.675$).

To test the hypothesis that SM RMS EMG would be no different between groups, a repeated measures ANOVA was performed with 2 within subject factors: Condition (predictable and unpredictable), and Epoch (epochs 1–40) and one between subject factor: Group (4 levels: previously painful side in the recurrent group, non-painful side in the recurrent group, and right and left sides in the healthy participants). In healthy participants, right SM RMS EMG amplitude was greater than that on the left (interaction: Epoch \times Side, $P < 0.001$; *post hoc*, $P < 0.023$, epochs 32–35) and data were not pooled for analysis.

Post hoc testing was undertaken with the Duncan multiple-range test. Significance was set at 0.05.

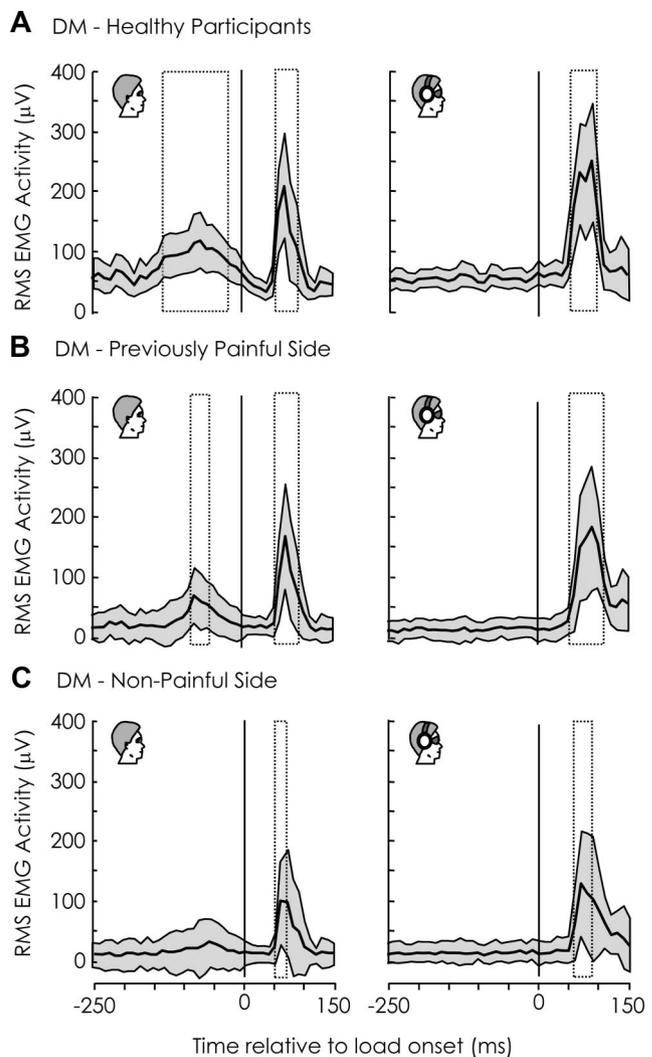


Figure 2. Root mean square (RMS) electromyographic (EMG) amplitude of deep multifidus (DM) in (A) healthy participants, and on the (B) previously painful side and (C) the non-painful side in the recurrent group during predictable (left column) and unpredictable loading (right column). Dashed boxes indicate epochs when DM RMS EMG increased above baseline. Solid vertical lines indicate load onset. Shaded areas denote 95% confidence intervals.

Results

DM EMG in Healthy Participants

During predictable loading, DM EMG increased above baseline between 140 and 30 milliseconds before load onset (preparatory period) (interaction: Condition \times Epoch \times Group, $P = 0.042$; *post hoc*, $P < 0.043$, epochs 13–23) and between 50 and 90 milliseconds after load onset (response period) (*post hoc*, $P < 0.002$, epochs 32–35; Figures 2A, 3A). Preparatory DM EMG peaked ~100 milliseconds before load onset and returned to baseline before load onset (Figures 2A, 3A). Unlike predictable loading, DM EMG during unpredictable loading increased above baseline between 50 and 100 milliseconds after load onset (*post hoc*, $P < 0.001$, epochs 32–36; Figures 2A, 3A). DM EMG response amplitude (between 70 and 100 milliseconds after load onset (epochs 34–36) and duration were greater in unpredictable

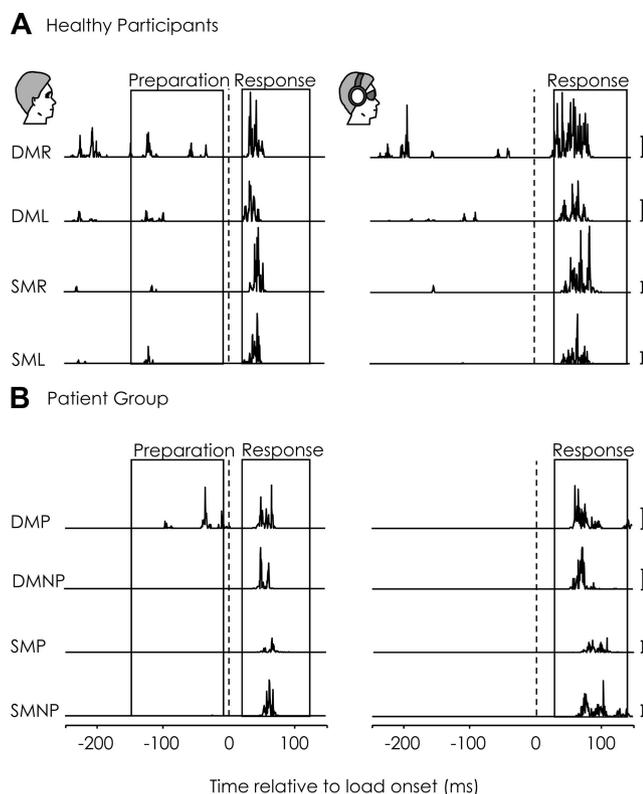


Figure 3. Rectified and high pass filtered (50 Hz) deep (DM) and superficial multifidus (SM) electromyographic (EMG) activity for representative (A) healthy and (B) recurrent group participants (previous right-sided LBP) during predictable (left column) and unpredictable loading (right column). The dashed vertical line represents load onset. Boxes indicate the preparatory and response periods of DM and SM EMG. EMG calibration: SM EMG, 150 μ V; DM EMG, 500 μ V (P indicates previously painful side; NP, non-painful side; L, left, R, right).

than predictable loading (*post hoc*, $P < 0.001$) (Figures 2A, 3A).

Differences in DM EMG Between Groups

During predictable loading, both preparatory and response DM EMG were less on the previously painful side (*post hoc*, $P = 0.042$, epoch 13; *post hoc*, $P = 0.046$, epoch 32, Figures 1B, 2B, 3A) and the non-painful side (*post hoc*, $P < 0.033$, epochs 12–23; *post hoc*, $P < 0.040$, epochs 32–34) than healthy participants (Figures 2A, 3A, 4A). During unpredictable loading, DM EMG response amplitude was less on the previously painful side (*post hoc*, $P < 0.046$, epochs 32, 33, and 35; Figures 2B, 4B) and non-painful side (*post hoc*, $P < 0.012$, epochs 32–36; Figures 2C, 4B) than healthy participants (Figures 2A, 4B).

Differences in DM EMG Between Painful and Non-painful Sides

Although DM EMG was less bilaterally in the recurrent group than healthy participants, and DM EMG response amplitude and duration were greater in unpredictable than predictable loading on both sides in the recurrent group (previously painful side: *post hoc*, $P < 0.029$, epochs 34–38; non-painful side: *post hoc*, $P < 0.037$, ep-

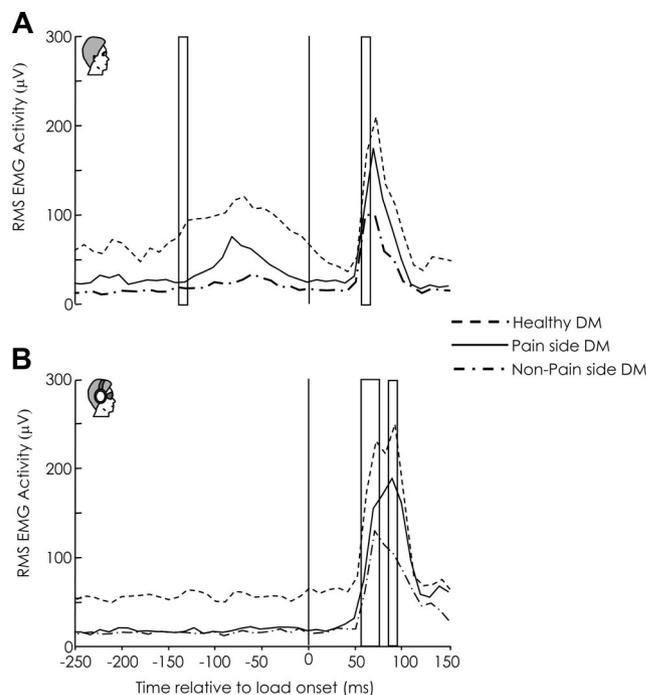


Figure 4. Group data for root mean square (RMS) electromyographic (EMG) amplitude of the deep multifidus (DM) on the previously painful side (—) and non-painful side (- · -) in the recurrent group and healthy participants (---) during (A) predictable and (B) unpredictable loading. Dashed vertical line represents load onset. Boxes indicate epochs when DM RMS EMG is less on the previously painful side in the recurrent group than in healthy participants.

ochs 32, 34–37; Figures 2B, 2C, 4B), there were differences between the previously painful and non-painful sides. DM EMG amplitude increased above baseline in preparation for predictable loading on the previously painful side (*post hoc*, $P < 0.033$, epochs 18–20; Figure 2B), but there was only a tendency for increased activity on the non-painful side (*post hoc*, $P < 0.95$; Figure 2C). Furthermore, DM EMG response amplitude was greater on the previously painful side than the non-painful side during predictable (*post hoc*, $P = 0.026$, epoch 33; Figures 3A, B) and unpredictable (*post hoc*, $P < 0.025$, epochs 35 and 36; Figures 3B, 4B) loading.

Differences in SM EMG Between Groups

Unlike DM EMG in healthy participants and on the previously painful side in the recurrent group, SM EMG amplitude did not increase before load onset on either side in either group (Condition \times Epoch, $P < 0.001$; *post hoc*, $P > 0.05$; Figure 5A). However, peak SM EMG on the previously painful side after predictable loading was earlier than, but of similar amplitude to, SM EMG on both sides in healthy participants (Epoch \times Group, $P < 0.001$; *post hoc*, $P = 0.009$, epoch 32; Figure 5A) and the non-painful side in the recurrent group (*post hoc*, $P < 0.002$, epochs 32 and 33; Figure 5A). However, in unpredictable loading, peak SM EMG amplitude in healthy participants was greater than either side in the recurrent group (previously painful side: *post hoc*, $P < 0.024$, ep-

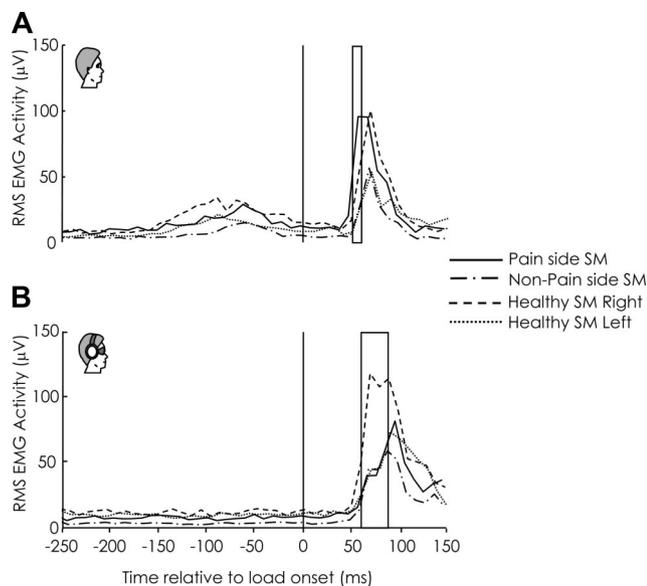


Figure 5. Group data for superficial multifidus (SM) root mean square (RMS) electromyographic (EMG) amplitude on the previously painful side (—) and non-painful side (---) in the recurrent group and on the right (---) and left side (·····) in healthy participants during (A) predictable and (B) unpredictable loading. Solid vertical lines represent load onset. In (A) the boxed area indicates epochs when SM EMG on the previously painful side reaches its peak amplitude before that in healthy participants. In (B) the boxed area indicates the epochs when right SM EMG in healthy participants is greater than on either side in the recurrent group.

ochs 33–35; non-painful side: *post hoc*, $P < 0.047$, epochs 32–36, 38; Figure 5B). In the recurrent group, SM EMG amplitude was greater on the previously painful side (*post hoc*, $P = 0.029$, epoch 36) than the non-painful side in unpredictable loading (Figure 5B).

Discussion

We investigated the control of DM and SM in people with recurrent unilateral LBP during remission from symptoms, and a group of healthy participants. The results support our first hypothesis (DM EMG would be less in people with a history of recurrent unilateral LBP than in healthy participants in response to sudden trunk loading) because DM EMG in the recurrent group was less in both predictable and unpredictable loading than in healthy participants. However, the results did not support our second or third hypotheses (DM EMG would be less on the previously painful side than the non-painful side and there would be no difference in SM EMG activity between groups) because the preparatory DM EMG on the previously painful side was greater than that on the non-painful side in predictable loading, and SM EMG differed between groups with earlier peak activity (predictable loading) and less activity (unpredictable loading) on the previously painful side than healthy participants.

DM EMG increased before predictable loading but returned to baseline at load onset. Although activity before loading is consistent with an attempt to prepare the

spine for the ensuing perturbation, its return to baseline at the time of impact is more difficult to explain. Despite the absence of DM EMG at load onset, force from contraction would be maintained because of the delay between electrical and mechanical events due to electromechanical delay²⁷ and the maintenance of twitch force following cessation of action potentials.²⁸ Perhaps these multiple bursts of DM activity are advantageous because they could reduce the amplitude of trunk oscillation related to loading (e.g., increase damping) in contrast to the stiffness generated by a single sustained burst of activity.²⁹ Consistent with previous work,⁷ SM activation was not initiated before loading and was only active in response to loading. During predictable loading, DM activity may be sufficient to prepare the lumbar spine, but insufficient to counteract the flexion moment applied to the trunk following load onset. In summary, when loading is predictable, and preparation is possible, the nervous system uses a pattern of discrete activation of DM and SM to control the spine.

DM EMG before predictable loading was less on both sides in the recurrent group than in healthy participants. However, peak SM EMG amplitude after predictable loading was earlier on the previously painful side. Because DM contributes to the control of the spine,³⁰ reduced DM EMG before predictable loading could leave the spine less prepared for the subsequent perturbation. An earlier SM EMG burst after predictable loading may be an attempt to compensate for reduced DM EMG before loading, and arguably, the failure to adequately prepare the spine for loading. Alternatively, it could be argued that less DM EMG is required before predictable loading, because the earlier peak SM EMG amplitude after predictable loading may be sufficient to reduce trunk perturbation. Regardless of the argument, the pattern of DM and SM activation on the previously painful side in the recurrent group demonstrates a shift in coordination between fascicles. Although the consequences of this shift in coordination are unclear, it could be speculated that it is not ideal because those people in the recurrent group, who demonstrated this pattern of activation, reported recurrent LBP episodes. Whether the observed changes on the previously painful and non-painful sides in DM and SM EMG are an attempt to increase control of sagittal plane motion (by increasing the use of SM which has a larger extensor moment than DM), or decrease compression on the spine (by decreased use of DM), or some other factor (such as reflex inhibition of DM) remains unclear.

There are clear differences in the mechanisms for control of DM and SM EMG between predictable and unpredictable loading. The primary difference is that predictable loading is self-initiated. Self-initiation of trunk loading allows preparation to begin prior to load release. This combined with the available visual and auditory feedback, optimizes preparation for load onset and, presumably, implementation of the best available control strategy. In unpredictable loading, the responses are

likely to be initiated at a spinal level by mechanisms including the monosynaptic stretch reflex, and the higher center influences on those reflexes. Whether the differences in responses between groups are the result of a decision by the nervous system to prepare the spine differently in the recurrent group irrespective of sensory input, the inability to accurately interpret lumbar spine feedback, the reduced availability of lumbar spine feedback, or modulation of spinal level reflexes, remains to be determined.

Several methodological issues require consideration. Non-normalized EMG permits EMG evaluation within the same muscle (DM for example) between groups (healthy participants and recurrent group) and conditions (predictable and unpredictable loading). However, non-normalized EMG is potentially more variable than EMG normalized to a MVC because it does not control for the effects of electrode placement and recording volume on EMG amplitude. A systematic difference in these recording parameters between groups is unlikely because the electrodes were inserted under ultrasound guidance using easily identifiable bony landmarks and the inter-electrode distance was fixed. Our decision to use non-normalized EMG data were based on the probability that participants in the recurrent group would either not perform a true MVC²⁶ or perhaps use a different strategy than healthy participants during a submaximal effort. Either of these options have the potential to increase data variability more than analysis of the EMG without normalization. Although the sample size used in this study was small, it was sufficient to identify significant differences. However, the small sample size limits the generalizability of the results. It is also unclear to what extent the asymmetrical movement pattern and asymmetry in previous symptoms contribute to the observed differences in multifidus activity between the recurrent group and healthy participants. We are also unable to confirm if there were any differences in trunk movement, or posture/spinal curvature between conditions or groups. However, our findings were consistent across groups and these limitations are unlikely to diminish the main findings of the study.

This study has several clinical implications. Our findings show that when people with recurrent unilateral LBP are unable to prepare for trunk loading, both DM and SM EMG activity is less than normal. The diminished activity of both components of the lumbar multifidus during unpredictable loading could affect the control of lumbar motion and potentially contribute to the increased incidence of LBP with this type of loading. These findings suggest that the level of preparedness for lifting may be an important consideration in developing safe lifting guidelines in addition to how much weight is lifted, the amount of twisting involved, and the distance of the weight lifted from the body.³¹ These findings support team-lifting guidelines that emphasize communication between lifters relating to load adjustment,³² as this

would decrease the incidence of unpredictable trunk loading.

In summary, differences in lumbar multifidus EMG are present in people with a history of recurrent LBP despite symptom remission. These differences are not uniform and vary with the predictability of loading. When the nervous system can predict loading, DM EMG activity is less and, when loading is unpredictable, the activity of both DM and SM are less than healthy people. Such reduced activity may compromise spinal control and contribute to recurrence.

■ Key Points

- We studied responses of discrete fascicles of the multifidus muscle in response to trunk loading in people with and without a history, but no current symptoms, of LBP.
- Despite symptom remission, electromyographic activity of deep multifidus was less in participants with a history of recurrent LBP during predictable and unpredictable trunk loading.
- During predictable trunk loading, peak activity of superficial multifidus on the previously painful side was earlier in participants with a history of recurrent LBP.
- Reduced activity of the lumbar multifidus in people with a history of recurrent unilateral LBP during trunk loading may result in less than optimal spinal control and could contribute to recurrence.

References

1. Magora A. Investigation of the relation between low back pain and occupation. IV. Physical requirements: bending, rotation, reaching and sudden maximal effort. *Scand J Rehabil Med* 1973;5:186–90.
2. Molumphy M, Unger B, Jensen GM, et al. Incidence of work-related low back pain in physical therapists. *Phys Ther* 1985;65:482–6.
3. Manning DP, Mitchell RG, Blanchfield LP. Body movements and events contributing to accidental and nonaccidental back injuries. *Spine* 1984;9:734–9.
4. Marras WS, Rangarajulu SL, Lavender SA. Trunk loading and expectation. *Ergonomics* 1987;30:551–62.
5. Lavender SA, Mirka GA, Schoenmarklin RW, et al. The effects of preview and task symmetry on trunk muscle response to sudden loading. *Hum Factors* 1989;31:101–15.
6. Magnusson ML, Aleksiev A, Wilder DG, et al. European Spine Society—the AcroMed prize for spinal research 1995. Unexpected load and asymmetric posture as etiologic factors in low back pain. *Eur Spine J* 1996;5:23–35.
7. Moseley G, Hodges P, Gandevia S. External perturbation of the trunk in standing humans differentially activates components of the medial back muscles. *J Physiol* 2003;547(pt 2):581–7.
8. McGill S, Grenier S, Bluhm M, et al. Previous history of LBP with work loss is related to lingering deficits in biomechanical, physiological, personal, psychosocial and motor control characteristics. *Ergonomics* 2003;46:731–46.
9. Leinonen V, Kankaanpää M, Luukkainen M, et al. Disc herniation-related back pain impairs feed-forward control of paraspinal muscles. *Spine* 2001;26:E367–72.
10. Stokes I, Henry S, Single R. Surface EMG electrodes do not accurately record from lumbar multifidus muscles. *Clin Biomech* 2003;18:9–13.
11. Wolf SL, Wolf LB, Segal RL. The relationship of extraneous movements to lumbar paraspinal muscle activity: implications for EMG biofeedback training applications to low back pain patients. *Biofeedback Self Regul* 1989;14:63–74.

12. Wilke HJ, Wolf S, Claes LE, et al. Stability increase of the lumbar spine with different muscle groups. A biomechanical in vitro study. *Spine* 1995;20:192–8.
13. Ward SR, Kim CW, Eng CM, et al. Architectural analysis and intraoperative measurements demonstrate the unique design of the multifidus muscle for lumbar spine stability. *J Bone Joint Surg Am* 2009;91:176–85.
14. Jemmett RS, Macdonald DA, Agur AM. Anatomical relationships between selected segmental muscles of the lumbar spine in the context of multi-planar segmental motion: a preliminary investigation. *Man Ther* 2004;9:203–10.
15. Lewin T, Moffett B, Vidik A. The morphology of the lumbar synovial joints. *Acta Morphol Neerl Scand* 1962;4:299–319.
16. Macintosh JE, Valencia FP, Bogduk N, et al. The morphology of the human lumbar multifidus. *Clin Biomech* 1986;1:196–204.
17. Bogduk N, Macintosh JE, Pearcy MJ. A universal model of the lumbar back muscles in the upright position. *Spine* 1992;17:897–913.
18. Macintosh JE, Bogduk N. The biomechanics of the lumbar multifidus. *Clin Biomech* 1986;1:205–13.
19. Hides JA, Stokes MJ, Saide M, et al. Evidence of lumbar multifidus muscle wasting ipsilateral to symptoms in patients with acute/subacute low back pain. *Spine* 1994;19:165–72.
20. Hodges P, Holm AK, Hansson T, et al. Rapid atrophy of the lumbar multifidus follows experimental disc or nerve root injury. *Spine* 2006;31:2926–33.
21. Zoidl G, Grifka J, Boluki D, et al. Molecular evidence for local denervation of paraspinal muscles in failed-back surgery/postdisectomy syndrome. *Clin Neuropathol* 2003;22:71–7.
22. Sihvonen T, Herno A, Paljarvi L, et al. Local denervation atrophy of paraspinal muscles in postoperative failed back syndrome. *Spine* 1993;18:575–81.
23. MacDonald DA, Moseley GL, Hodges PW. The lumbar multifidus: does the evidence support clinical beliefs? *Man Ther* 2006;11:254–63.
24. MacDonald D, Moseley GL, Hodges PW. Why do some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain. *Pain* 2009;142:183–8.
25. Moseley G, Hodges P, Gandevia S. Deep and superficial fibers of the lumbar multifidus muscle are differentially active during voluntary arm movements. *Spine* 2002;27:E29–36.
26. Lariviere C, Arseneault AB, Gravel D, et al. Surface electromyography assessment of back muscle intrinsic properties. *J Electromyogr Kinesiol* 2003;13:305–18.
27. van Dieen JH, Thissen CE, van de Ven AJ, et al. The electro-mechanical delay of the erector spinae muscle: influence of rate of force development, fatigue and electrode location. *Eur J Appl Physiol Occup Physiol* 1991;63:216–22.
28. Burke RE, Levine DN, Tsairis P, et al. Physiological types and histochemical profiles in motor units of the cat gastrocnemius. *J Physiol* 1973;234:723–48.
29. Hodges P, van den Hoorn W, Dawson A, et al. Changes in the mechanical properties of the trunk in low back pain may be associated with recurrence. *J Biomech* 2009;42:61–6.
30. Kaigle AM, Holm SH, Hansson TH. Experimental instability in the lumbar spine. *Spine* 1995;20:421–30.
31. Kuijjer W, Dijkstra PU, Brouwer S, et al. Safe lifting in patients with chronic low back pain: comparing FCE lifting task and Niosh lifting guideline. *J Occup Rehabil* 2006;16:579–89.
32. Cal/OSHA Consultation Service, Research and Education Unit, Division of Occupational Safety and Health, CA Department of Industrial Relations. *Ergonomic Guidelines for Manual Material Handling*. San Francisco, CA: Centre for Disease Control and Prevention, National Institute for Occupational Safety and Health, California Department of Industrial Relations; 2007.