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DIAGNOSTICS

Smudging of the Motor Cortex Is Related to the Severity of Low Back Pain

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Study Design. Cross-sectional design.

Objective. Here we aimed to determine whether motor cortical reorganization in low back pain (LBP) can be identified using noninvasive surface electromyographic (EMG) recordings of back muscles at different lumbar levels, and whether cortical reorganization is related to clinical features of LBP.

Summary of Background Data. Reorganization of motor regions of the brain may contribute to altered motor control, pain, and disability in chronic LBP. However, data have been limited by the need for invasive recordings of back muscle myoelectric activity. The relationship between altered cortical organization and clinical features of LBP remains unclear.

Methods. In 27 individuals with recurrent, nonspecific LBP and 23 pain-free controls, we mapped the motor cortical representation of the paraspinal muscles using transcranial magnetic stimulation in conjunction with noninvasive surface EMG recordings at L3 and L5 levels. Clinical measures of pain severity, location, and duration were made.

Results. The results demonstrate a loss of discrete motor cortical organization of the paraspinal muscles in chronic LBP that can be identified using noninvasive EMG recordings. A loss of discrete cortical organization was clearer when surface electrodes were positioned at L3 rather than L5. A novel finding was that altered motor cortical organization (number of discrete peaks and map volume) was associated with the severity and location of LBP.

Conclusion. These data suggest that surface EMG positioned at L3 is appropriate for the identification of changes in the motor cortex in LBP. Furthermore, our data have implications for treatment strategies that aim to restore cortical organization in LBP.

Key words: chronic low back pain, electromyography, motor control, motor cortex reorganization, pain duration, pain location, pain severity, paraspinal muscles, transcranial magnetic stimulation.

Level of Evidence: 2

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Low back pain (LBP) is associated with poor rates of recovery and high rates of recurrence.[1,2] Although persistence of symptoms is multifactorial, altered control of back muscles has been identified as a predictor of pain onset and recurrence.[3] Despite this, the mechanisms that underpin adaptation of the motor system, and their relationship to pain and disability, remain poorly understood. Reorganization of the primary motor cortex (M1) has been identified in LBP,[4,5] and this may contribute to altered motor control, pain, and disability. However, interpretation of these findings is limited by the use of invasive recordings that restrict the number of individuals tested. The relationship between brain organization, motor control and clinical features of LBP will remain unclear until larger populations can be tested using less invasive methods.

Maps of M1 generated for 2 back muscles (lumbar longissimus and deep multifidus [DM]) using transcranial magnetic stimulation (TMS) demonstrate a change from 2 discrete map peaks in healthy individuals to a single, overlapped peak in LBP.[4] Increased overlap (''smudging'') in the cortical representations of lumbar longissimus and DM may explain the loss of differentiated control of the paraspinal muscles and tendency for back muscles to be recruited *en masse* in this population.[6,7] A key feature of this work was the discrete recording of electromyography (EMG) from individual muscle fascicles with intramuscular fine-wire electrodes. Although fine-wire electrodes enabled resolution of the origin of the EMG signals, their invasive nature restricts the size of the participant group and thus, sample sizes have been insufficient to address the relationship between motor cortical organization and clinical symptoms.

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No studies have investigated whether reorganization of the cortical outputs to the back muscles in LBP is identifiable with noninvasive recordings. Furthermore, motor cortical mapping has been limited to investigation of a single level of the spine, and it is unclear whether changes are present across multiple spinal levels.

This study aimed to determine whether reorganization of M1 in LBP (1) can be identified from noninvasive surface EMG recordings of back muscles; (2) is present in individuals who have LBP at the time of testing; (3) differs between levels of the lumbar spine; and (4) is related to clinical features of mild to moderate LBP.

MATERIALS AND METHODS

Participants

Twenty-seven individuals with nonspecific, recurrent LBP and 23 pain-free individuals participated. Individuals with LBP were included if they experienced episodic pain in their low back, sufficient to limit function, for greater than 3 months. Individuals were excluded if they presented with suspected spinal pathology; major circulatory, neurological, or psychiatric conditions; previous spinal surgery; or recent/current pregnancy. LBP participants indicated the region of worst pain on a body chart. From the body chart, the side of worst pain was identified and participants allocated to either "upper" or "lower" lumbar pain on the basis of whether pain was indicated above or below the line representing the iliac crest. Participants rated their current pain intensity on an 11-point numerical rating scale (NRS). Participants were asked to estimate the duration of LBP as the total time since their first episode, regardless of periods of remission. Participant characteristics are provided in Table 1.

All procedures were approved by the institutional ethics committee and conformed to the Declaration of Helsinki.

Electromyography

Surface EMG was used to record activity of the paraspinal muscles at 2 sites: 3 cm lateral to the spinous process of L3 and 1 cm lateral to the spinous process of L5, on the side of worst pain (silver-silver chloride disposable electrodes; Noraxon USA Inc, Scottsdale, AZ). These sites record EMG from multiple back muscles.[8] As the purpose of this study was to determine whether multiple peaks in the TMS map could be recorded from surface electrodes, it was necessary to record from multiple muscles simultaneously. EMG data were amplified 1000 ×, filtered 20 to 1000 Hz, and sampled at 2000 Hz.

Motor Cortex Mapping

Single-pulse TMS (width 1 ms) was delivered to M1 contralateral to the side of worst pain (figure-of-eight coil; Magstim Co. Ltd. Dyfed, United Kingdom).[4,9] The vertex was determined using the 10/20 International EEG Electrode Placement system, and this point registered using a Brainsight2 neuronavigation system (Rogue Resolutions Ltd, Cardiff, United Kingdom). Starting at the cranial vertex, used as the standard reference for reporting location of TMS of the brain,[4,9,10] 5 magnetic stimuli were delivered at 1-cm intervals on a 6×7 cm grid. Accurate coil placement was determined using neuronavigation. Stimuli were applied at 100% (maximum) stimulator output. During TMS mapping, participants were requested to activate the paraspinal muscles to 20% of the EMG amplitude recorded during a maximum voluntary contraction. This level of activation of the extensor muscles was based on that used in previous studies[4,9] and was required for 2 reasons. First, activation of muscles facilitates the corticomotor pathway and thus increases Motor evoked potential (MEP) amplitude. This is often necessary to evoke a MEP in trunk muscles. Second, control of the intensity of contraction standardizes the facilitation of the pathway between participants. The target EMG amplitude was determined as 20% of the highest root mean square EMG for 1 second during three, 3-second maximal trunk extension efforts performed against manual resistance in sitting. Visual feedback was provided on a computer monitor and the 20% maximum voluntary contraction target achieved by sitting forward with the back straight.[11,12] All procedures adhered to the TMS checklist for methodological quality.[13]

Data Analyses

EMG was full-wave rectified and the 5 MEPs at each scalp site averaged. MEP onset and offset were visually identified from the averaged traces and MEP amplitude calculated as the root mean square EMG amplitude between the onset and offset.[4,5,14–16] Background EMG from 55 to 5 ms prior to stimulation was subtracted.[4,5,15] MEP amplitudes were superimposed over the respective scalp sites to produce a topographical representation of the target paraspinal muscle and normalized to the peak amplitude for each participant. Normalized values less than 25% of the peak response were removed and the remaining values rescaled from 0% to 100%.[4] Three parameters were calculated from motor cortical maps. First, map volume, a measure of the total

TABLE 1. Participant Characteristics (Mean ± Standard Deviation)

	Pain-free Controls (n = 23)	Low Back Pain (n = 27)
Age (yr)	27 ± 5	30 ± 9
Sex (male:female)	12:11	13:14
Pain (NRS)	...	4.6 ± 1.9
Pain duration (yr)	...	5.3 ± 4.0
Side of worst pain (right:left)	...	18:9
Site of worst pain (upper:lower)	...	11:16

Pain: current pain intensity rated on an NRS. Pain duration: total time that participants had experienced recurrent low back pain including periods of aggravation and remission.

NRS indicates numerical rating scale.

excitability of the cortical representation, was calculated as the sum of the mean normalized MEP peak-to-peak amplitude at all active sites. To be considered "active," a scalp site was required to evoke a MEP with a normalized amplitude of 25% or greater peak response. Second, the center of gravity (CoG), defined as the amplitude weighted center of the map,[17,18] was calculated for each muscle using the formula: $CoG = \sum V_i \; x \; X_i / \sum V_i, \; \sum V_i \; x \; Y_i / \sum V_i$, where: V_i = mean MEP amplitude at each site with the coordinates X_i, Y_i. Finally, the number of discrete map peaks was determined using criteria that were derived on the basis of inspection of data from a previous study[4] and initial pilot work. Accordingly, a peak was identified if its amplitude was greater than 60% of the maximum MEP amplitude and was separated from any adjacent areas above the threshold by a reduction in MEP amplitude of at least 20%. Peaks in addition to the primary peak were registered only if they were separated from the primary peak along an anterior-posterior axis.[4]

Statistical Analyses

Map volume, CoG position, and the number of discrete map peaks were compared between groups (LBP/control) using 1-way analyses of variance. *Post hoc* tests were performed using Holm-Sidak tests for multiple comparisons. Possible relationships between clinical features and properties of the cortical map were investigated in 2 ways. First, linear relationships between continuous measures of map volume and measures of pain severity and duration were examined using Pearson coefficients. Second, the relationship between clinical features and ordinal data (*i.e.*, number of discrete map peaks) was compared between individuals on the basis of pain duration (long duration $\geq$60 mo; short duration $<$60 mo; based on group median duration of 60 mo), pain location (upper or lower [above or below the iliac crest]), and pain severity (moderate to severe: LBP $>$ 5 on the NRS; mild: LBP $\leq$ 5 on the NRS—based on recommendation of NRS of 5 as an optimum cutoff to distinguish between subgroups of LBP).[19,20] Significance was set at P value of less than 0.05. Values in text are mean $\pm$ standard deviation.

RESULTS

Altered Motor Cortical Organization Can Be Identified Using Surface EMG

Figure 1 shows normalized TMS maps. The number of discrete peaks was less in the L3 map for the LBP participants (1.3 ± 0.5, $P = 0.009$; Figure 1A) than for the pain-free participants (1.7 ± 0.5; Figure 1C). At the L3 recording site, 70% of pain-free controls and only 33% of individuals with LBP displayed 2 discrete map peaks. Although there was no significant difference in the number of discrete peaks in the L5 map between individuals with (1.4 ± 0.5; Figure 1B) and without LBP (1.7 ± 0.5, $P = 0.15$; Figure 1D), 65% of pain-free controls and only 44% of individuals with LBP displayed 2 discrete peaks. The map generated from averaging data for the group shows 2 separate peaks when EMG is recorded with the electrodes at L3 but only 1 site when EMG was recorded at L5.

The CoG was located more anteriorly for both the L3 and L5 maps in individuals with LBP (L3: LBP 1.4 ± 0.61, pain-free 0.8 ± 0.77, $P = 0.006$; L5: LBP 1.5 ± 0.7, pain-free 1.0 ± 0.8, $P = 0.02$; Figure 2).

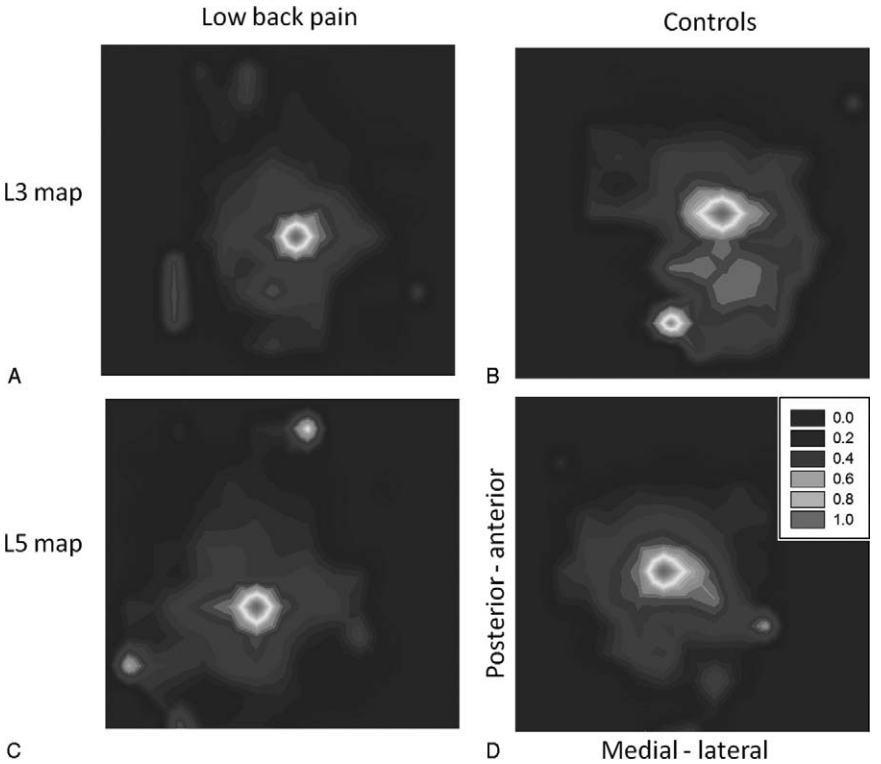

Figure 1. Normalized motor cortex maps aligned to the maximum motor evoked potential (MEP) for each participant obtained for electromyographic electrodes placed at the level of L3 in low back pain (**A**) and pain-free controls (**C**) and at the level of L5 in low back pain (**B**) and pain-free controls (**D**). The colored scale represents the proportion of the maximum motor evoked potential (MEP) amplitude. Note that the L3 motor cortical map in the control participants demonstrates 2 discrete peaks, whereas maps obtained at L5 in healthy controls and at both recording sites in those with low back pain demonstrate only 1 discrete peak.

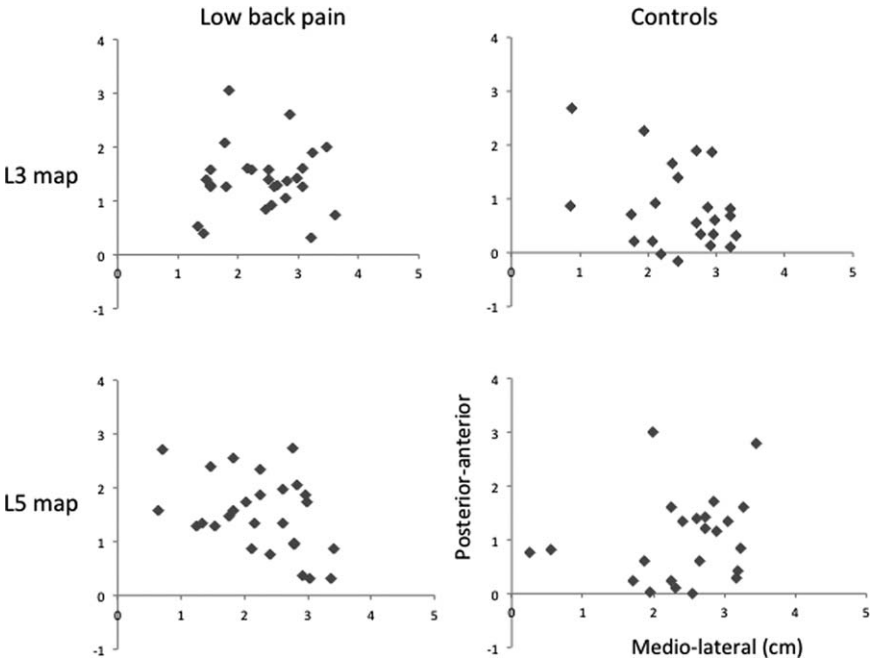

Figure 2. Individual data for the center of gravity (CoG) of the motor cortex maps obtained at the L3 and L5 electromyographic recording sites in those with low back pain and in pain-free controls. The coordinate (0,0) denotes the vertex. Note that the distribution of locations of the CoG is more anterior at both sites in those with low back pain than that in pain-free participants.

Map volume did not differ between LBP and pain-free participants at either the L3 (LBP 9.8 ± 5.1, controls 10.5 ± 5.1, $P = 0.65$) or L5 recording sites (LBP 10.1 ± 5.5, pain-free 10.5 ± 5.0, $P = 0.81$) regardless of the location of pain (L3, $P = 0.87$; L5, $P = 0.93$).

Altered Motor Cortical Organization Is Related to Clinical Features of LBP

All individuals with moderate-severe LBP (>5 on the NRS, n = 8) displayed a single discrete peak in the topographical map of L3 (moderate-severe LBP 1 ± 0; Figure 3A), whereas this was apparent for only 53% of participants with mild LBP (≤ 5 on the NRS; n = 19; 1.5 ± 0.5, $P = 0.016$; Figure 3C) and 30% of pain-free controls (1.7 ± 0.5; Figure 3E). The number of discrete peaks in the L5 map was not related to pain severity (moderate-severe LBP 1.6 ± 0.5 peaks; Figure 3B; mild LBP 1.4 ± 0.5 peaks; Figure 3D; pain-free controls 1.7 ± 0.5 peaks; Figure 3F; $P = 0.24$). The number of discrete peaks did not differ on the basis of pain duration (L3: $P = 0.82$; L5: $P = 0.8$) or pain location (L3: $P = 0.79$; L5: $P = 0.41$).

Although map volume did not differ between LBP and control subjects, our data revealed a relationship between map volume and pain severity that was dependent on the

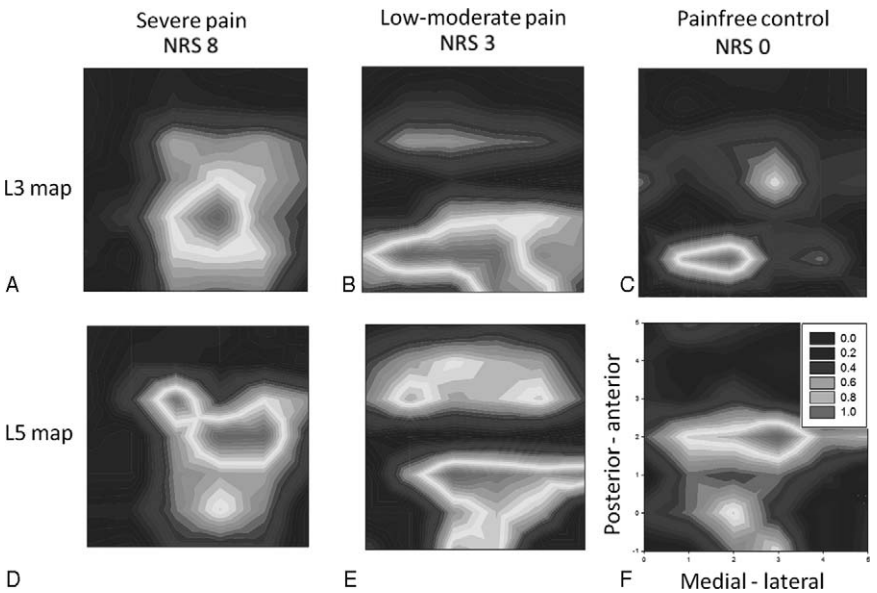

Figure 3. Normalized motor cortex maps obtained at the L3 (top panels) and L5 (bottom panels) electromyographic recording sites from representative individuals with severe low back pain (LBP, **A** and **B**), mild LBP (**C** and **D**), and no history of LBP (**E** and **F**). The vertex is coordinate 0,0. Note the single discrete peak in the L3 map of the individual with severe pain, but the 2 peaks observed in the L5 map, in the individual with mild pain, and the pain-free control participant. NRS indicates Numerical Rating Scale.

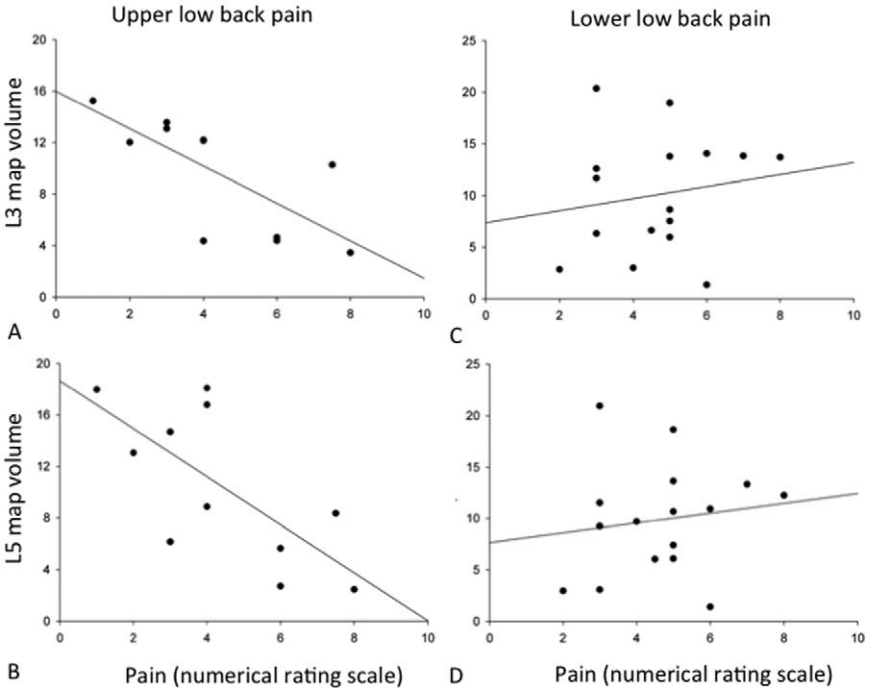

Figure 4. Relationship between L3 map volume (sum of normalized map volume) and pain severity in individuals with upper (**A**) and lower (**C**) low back pain and between L5 map volume and pain severity in individuals with upper (**B**) and lower (**D**) low back pain. Smaller L3 and L5 map volume was associated with greater pain severity in those with upper low back pain. The same pattern was not present for those with lower back pain.

location of pain. In individuals with upper LBP (n = 11), a smaller map volume at both L3 and L5 was related to higher pain severity (L3: $r = 0.73$, $P = 0.01$; Figure 4A; L5: $r = 0.69$, $P = 0.01$; Figure 4B). There was no such relationship for individuals with lower LBP (n = 16; L3: $r = 0.16$, $P = 0.53$; Figure 4C; L5: $r = 0.14$, $P = 0.59$; Figure 4D). Although not significant, there was a tendency for an association between map volume at L3 and pain duration in those with upper LBP (*i.e.*, maps tended to be larger the longer the pain duration; $r = 0.54$, $P = 0.08$). Similar trends were not present when L5 map volume was considered in relation to pain duration (upper pain: $r = 0.27$, $P = 0.41$; lower pain: $r = 0.22$, $P = 0.41$).

DISCUSSION

Our findings show that loss of discrete motor cortical organization of the paraspinal muscles can be identified using noninvasive EMG in individuals with persistent LBP who have symptoms at the time of testing. Cortical reorganization in the LBP group most closely resembles that obtained using fine-wire recordings when surface EMG electrodes are positioned at the level of L3. A new finding is that features of altered motor cortical organization are associated with the severity and location of LBP.

Organization of M1 Can be Measured With Noninvasive EMG

A key finding is that organization of cortical networks with outputs to the paraspinal muscles can be evaluated in humans with, and without, LBP using noninvasive surface EMG. Previous work using invasive fine-wire EMG positioned at L4 revealed several characteristics of the "motor brain" that differed between healthy individuals and those with LBP: (1) a shift from 2 discrete peaks in the topographical representation of the paraspinal muscles in healthy individuals to a single peak in LBP; (2) smaller map volume in LBP than healthy controls; and (3) a CoG that was located more posteriorly in LBP.[4] We show similar changes in the number of discrete peaks between LBP and controls using noninvasive EMG at L3 (70% of controls and 33% of LBP displayed 2 peaks). Some differentiation of discrete map peaks between the healthy and LBP groups at L5 was also present, although less clear (65% controls and 44% of LBP with 2 peaks). Surface electrodes have a broad detection area and include contribution from muscles across multiple spinal levels. However, our finding that differentiation of cortical representations was clearer when surface EMG was recorded at L3 could be considered surprising given that LBP is more common in lower regions, and multifidus wasting is also more common at lower sites.[21] Differences in the relationship between the paraspinal muscles and the surface EMG recordings at the 2 sites, for example, as a result of differences in relative muscle bulk of deep short and long superficial muscles, may influence the sensitivity to detect differences in cortical representations for these muscles at different lumbar regions.

Our data are the first to determine the proportion of LBP and pain-free individuals who display a single map peak in the cortical representation of the paraspinal muscles. A reduction in map volume was also observed using surface EMG at L3 and L5 but only in individuals with more severe pain in the upper lumbar spine. In contrast to previous findings,[4] the CoG was located more anteriorly for both the L3 and L5 maps in LBP than for controls. Extrapolation of previous CoG findings in LBP[4] is limited by the use of noninvasive recordings in our data, in which the cortical representation of both

lumbar longissimus and DM contributes to the total map. This contrasts invasive fine-wire recordings that allow the CoG for lumbar longissimus and DM to be determined separately.[4] Calculation of the CoG for separate paraspinal muscles is one limitation of noninvasive surface EMG recordings but does not limit the use of these recordings to interpret overall organization of the M1 map.

Reorganization Is Related to Clinical Features of LBP

Our data provide evidence of a relationship between organization of M1 in LBP and pain severity, duration, and location. A unique finding is that greater smudging of the cortical representation at L3 (single-map peak) was more consistently present in individuals with higher pain severity, whereas individuals with lower severity exhibit a pattern of cortical organization that is more similar, on average, to that of controls. A loss of discrete organization of the cortical networks that control paraspinal muscles has potential functional consequences. Individuals with focal dystonia, a condition characterized by excessive and inappropriate muscle activity during skilled motor tasks, have a reduced ability to independently contract involved muscles and move fingers independently. This motor dysfunction is associated with greater overlap of the cortical representations of the hand muscles compared with healthy individuals.[10,22] Taken together with the observation of reduced discrete activation of the paraspinal muscles in LBP,[4,6,7] smudging of cortical areas could explain compromised activation of discrete muscles in LBP. Impaired control of individual back muscles in the presence of pain may represent adoption of a new movement strategy to contract the muscles *en masse* to protect a painful body region.[23–25] If correct, this mechanism may explain our finding of a more pronounced reorganization with more severe pain.

Reduced map volume at the L3 and L5 recoding sites was associated with higher pain severity but only in those who reported upper LBP. Smaller map volume suggests reduced excitability of corticomotor projections to paraspinal muscles and/or a smaller cortical territory devoted to control of these muscles. This is consistent with previous reports of cortical reorganization in LBP.[4] It is unclear why the relationship between map volume and pain was identified only for those with upper LBP. One possibility is that, for anatomical reasons, surface electrodes can more adequately detect reduced map volume in upper lumbar regions. For instance, the muscles that are altered when pain is in the upper low back are likely to be those located in the more cranial regions of the lumbar spine (greater relative mass of the long superficial to short deep muscles), and these may be reflected differently in the surface recordings than when more caudal regions (with bias toward greater mass of deep short muscle bulk) are affected.

Apart from the requirement to satisfy general criteria (pain for >3 mo; sufficient pain to cause functional limitation; no spine surgery), no attempt was made to select LBP participants with a specific pathology or from a specific subgroup. This decision was based on an earlier study using intramuscular EMG that identified changes in organization of M1 in people with nonspecific LBP.[4] The present data suggest that on average, there is a difference in M1 organization in people with nonspecific LBP, but there was variation between participants. Future work should consider whether these changes depend on individual features of LBP such as movement behavior or specific pathology.

Clinical Implications

Our data suggest that moderate to severe LBP is more likely to be associated with cortical reorganization, characterized by smudging (1 map peak) and smaller map volume, than mild LBP. Altered map volume may also be influenced by the location of pain. This has implications for treatment strategies that aim to restore normal cortical organization. Although the optimum method to restore cortical organization in LBP is yet to be established, studies in focal hand dystonia suggest motor retraining using isolated movements[22,26] or asynchronous electrical stimulation,[10] restore cortical representations and improve pain. Furthermore, interventions such as voluntary motor training[15] and combined noninvasive brain and peripheral stimulation[9] have been shown to restore cortical representations, and this is associated with improved symptoms in LBP. Our data suggest that those with moderate to severe LBP may derive more benefit from motor skill training than those with milder symptoms. This requires further investigation.

➢ Key Points

- ❑ Motor cortical organization is associated with the severity and location of LBP.
- ❑ Altered motor cortical organization in LBP can be measured noninvasively using surface EMG recordings.
- ❑ A relationship between motor cortical organization and LBP severity and location has implications for treatments that aim to restore cortical organization in LBP.

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