

Mislocalization of Sensory Information in People With Chronic Low Back Pain

A Preliminary Investigation

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Objectives: The purpose of this study was to establish if people with chronic low back pain (CLBP) demonstrate impairments in the ability to localize sensory information delivered to the back more than pain-free controls and determine whether any sensory abnormalities are related to pain-related variables.

Methods: Vision was occluded and participants were stimulated using light touch or pinprick over a number of body areas in random order. To assess for mislocalizations participants were asked to nominate the location of each stimulus in reference to a marked body chart. To assess referred sensations participants who were asked whether they experienced any sensations elsewhere during stimulation. If referred sensations were reported, testing was repeated with visualization of the stimulated area.

Results: Although a small number of CLBP patients demonstrated referral of sensations, this was not statistically different from what was observed in a pain-free control group ($P = 0.381$). In contrast, mislocalizations were very common in the patient sample and statistically more common than we found in controls ($P = 0.034$). No statistically significant associations were detected between sensory function and the measured pain-related variables (all $P > 0.05$).

Discussion: These data add to a growing body of evidence suggesting that disturbed self-perception is a feature of CLBP. It is plausible that altered self-perception is maladaptive and contributes to the maintenance of the problem and may represent a target of treatment for CLBP.

Key Words: low back pain, referred sensations, sensory perception, cortical reorganization, body image

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A key property of the somatosensory system is the ability to localize the site of sensory input.¹ This is particularly significant for the nociceptive system where accurate localization of the stimulus is likely to be very adaptive.²

Evidence suggests that the ability to localize sensory information is impaired in people with chronic pain problems. Referred sensations, which are somatosensory feelings that are perceived to emanate from a body part other than the one being stimulated,³ have been reported in a range of painful conditions such as phantom limb pain,^{4–9} neuropathic pain after spinal cord injury,¹⁰ brachial plexus injury,^{11,12} and complex regional pain syndrome (CRPS).^{3,13} Referred sensations are thought to represent the clinical correlate of cortical^{4,14,15} and subcortical^{16,17} reorganization, a feature of a number of chronic pain syndromes.¹⁸

In reports of referred sensations, patients commonly perceive sensation both at the stimulated site and the remote referral site. Clinically, we have observed a different type of localization deficit, in which people with chronic back pain experience only 1 site of stimulation but are unable to accurately localize where on the body surface the stimulation occurred, a phenomenon termed atopognosia.¹ This finding is generally displayed in neurological patients after definitive brain injury.^{19–21} However, it is also described in people with CRPS of 1 arm. Those people have not sustained brain damage, yet when a single finger is stimulated, their ability to correctly identify the stimulated finger is lower on the affected hand than on the unaffected hand,²² suggesting that this phenomenon may also be present in patients with pain-induced cortical reorganization.

Chronic low back pain (CLBP) involves extensive changes in central nervous system's structure and function,^{23,24} and there is an increasing interest in identifying clinically accessible markers of cortical dysfunction. There is some evidence, from retrospective chart review, that referred sensations are a feature of CLBP,²⁵ but we are not aware of any empirical investigation of the presence of atopognosia in people with CLBP. This study aimed to determine if people with CLBP demonstrate impairments in the ability to localize sensory information and whether these sensory impairments relate to clinical status. We hypothesized that CLBP patients would be poor in localizing sensory input, and would experience referred sensations more often than pain-free controls. We also hypothesized that the extent of these particular sensory impairments would relate to clinical status.

MATERIALS AND METHODS

Design

This cross-sectional case-control cohort study received institutional ethical approval. Participants provided informed consent and all procedures conformed to the Declaration of Helsinki.

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Participants

A convenience sample of 24 nonspecific CLBP patients was recruited from the Department of Pain Management at The Sir Charles Gairdner Hospital, Perth, WA, and from community physiotherapy practices as part of a randomized cross-over experiment exploring the effect of visual feedback on movement-related back pain.²⁶ The sample size was determined by the power calculation for that cross-over experiment. Participants were eligible if they were aged between 18 and 60 years of age; were proficient in written and spoken English; reported back pain as their main symptom; had experienced nonspecific low back pain for a minimum of 6 months; rated their back pain as at least moderate on a modified version of item 7 of the Short-Form 36,²⁷ and were able to provide consent. Participants were excluded if they presented with nerve root pain or evidence of specific spinal pathology (such as malignancy, infection, fracture, inflammatory disease, etc.); were pregnant or <6 months postpartum; had undergone any lumbar surgery or invasive procedure within the previous 12 months; were currently involved in litigation in relation to their back pain; were judged by their treating clinician to be unsuitable for performance of a repeated movement assessment; and had significant medical or psychological illness or significant visual impairment.

Twenty-four healthy volunteers drawn from staff and students of The University of Notre Dame Australia also participated. Controls were invited to participate if they were currently low back pain-free, reported no back pain at all in the last 6 months, had not experienced any episode of low back pain sufficient to restrict work or leisure within the last 2 years, were proficient in written and spoken English, and were able to provide consent. Controls were excluded if they were pregnant or <6 months postpartum or had any significant extant medical condition.

Participant Profile

Treating medical or physiotherapy staff identified potential participants and checked the study criteria. Potential participants were then seen by a research assistant who clarified inclusion and exclusion criteria, obtained consent, and collected basic demographic data. Volunteers completed a questionnaire that solicited information about the length of the current episode, pain distribution, work status, and current pain medications. In addition, patients completed a set of standardized questionnaires that assessed disability, pain, and psychological functioning. Low back pain-related disability was measured using the Roland Morris Disability Questionnaire.²⁸ Back pain intensity was measured using a Visual Analogue Scale for average pain over the last week, anchored at left with 0 = "no pain" and at right with 100 = "pain as bad as you can imagine." Kinesiophobia was estimated using the Tampa Scale of Kinesiophobia.²⁹ The level of pain-related catastrophization was measured using the Pain Catastrophizing Scale³⁰ and depression and anxiety were assessed with the Hospital Anxiety and Depression Scale.³¹ The control sample provided the same demographic information and completed the Hospital Anxiety and Depression Scale.³¹

Procedure

The protocol was based partly on that used by McCabe et al.³ All testing occurred in a closed room in which ambient noise was kept low and distractions minimized. Before testing, participants were undressed to their

underwear and were shown an A3 size schematic diagram of a posterior view of the body with different anatomic areas marked (Fig. 1). The areas were: lower thigh (popliteal line to mid femur), upper thigh (mid femur to gluteal crease), low lumbar (posterior superior iliac spine to spinous process of L2), upper lumbar (L2-T10), low thoracic (T10-T6), upper thoracic (T6-T2), and shoulder (posterior acromion to mid humerus). The participants were thoroughly orientated to the labels for each body area and the anatomic demarcation between the areas. The verbal descriptions were reinforced by tactile input from the researcher identifying both the boundaries between the areas and the centre of each area where the formal stimulation would occur.

Participants were then positioned comfortably in prone on an examination table. A pillow was placed under the stomach to flatten the lumbar spine and standardize the lumbar position. A large mobile mirror was placed adjacent to the table so that the participants could view a reflection of their back and legs by turning their head to the side. For initial testing, participants were instructed to lay prone with

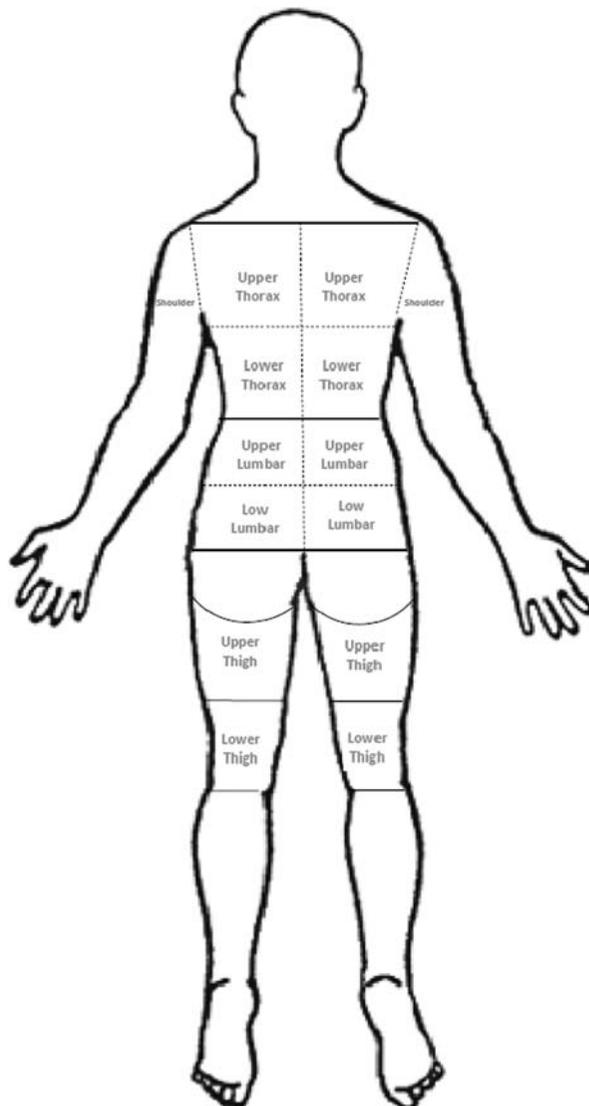


FIGURE 1. Body chart with anatomic area marked.

their face through the hole at the end of the examination table, occluding vision of the back. The schematic diagram of the body was placed on the floor visible to the participant through the face hole. In this position further verbal and tactile reinforcement was given of the boundaries and centre of the marked body areas. For the final stage of preparation, the research assistant lightly marked the centre of each body area to ensure standardization of the stimulation site.

For CLBP patients, the sensory examination was conducted on the side of worst back pain. If patients were unable to differentiate between sides, the side of testing was determined by coin toss. The side of testing for control participants was determined by coin toss. Testing of light touch was undertaken first in all participants using a software-generated sequence that ensured each body area was assessed twice in randomized counterbalanced order. Superficial pain was then assessed in a similar manner using a different random sequence, resulting in 28 stimulations in total. As the participants were recruited from different facilities, the tester was not blinded to the patient's clinical status.

Light touch was assessed by applying 5 slow horizontal strokes with a cotton swab to the centre of each body region marked on the body chart and superficial pain by 5 light depressions with a **Medipin** (Medipin Ltd., Bushey Hertfordshire, UK) in the same area. Five stimuli were chosen, as pilot testing established that participants had difficulty judging both referred sensations and localization based on a single stimulus. To enhance consistency in sensory stimulation the size of the cotton swabs used and the way they were held was standardized and attempts were made to ensure uniformity of applied pressure. For superficial pain testing we attempted to control the depth of depression by use of the **Medipin**, which is a single use neurological testing pin where the point is surrounded by a flattened annulus, thus limiting the depth of depression. Each stimulation was applied at a rate of approximately 1 stimulation/s and a 5-second pause was used between each set of stimulations.

For each series of 5 stimuli, participants were asked to state in which body area they felt the stimuli. If the stated body area was different to the stimulated site this was recorded as a mislocalization. Participants were then asked whether they perceived a stimulus area anywhere else. If participants responded in the affirmative, it was recorded as a referred sensation. When referred sensations were reported, participants were asked to describe the referred sensations and indicate their location. The identical stimulation site was then reassessed, using the same modality, but with visualization of the back and legs, so as to measure the effect of visual feedback on the referred sensation. The effect of vision on mislocalization was not assessed as it was assumed that all mislocalizations would likely be corrected by visual feedback. Patients were informed that the purpose of the study was to investigate the sensitivity of the back in CLBP patients and they were naive to the concept of referred sensations, and blinded to the study hypotheses.

Data Analysis

All analyses were undertaken using PASW for Windows version 18 (SPSS, Chicago IL) or Stata/IC 10.1 for Windows (Statacorp LP, College Station, TX). The demographic and clinical profile of patients and controls were summarized with means and SDs for continuous data and ratios and percentages for categorical data. The 2 outcome variables were counts of (1) mislocalizations; or (2) referred

sensation with sensory stimulation. Data from light touch and superficial pain testing were combined for analysis. A comparison of the distribution of the number of mislocalizations and referred sensations between patient and control groups was made using Fisher exact test. Because of the small number of mislocalizations and referred sensations, the nonparametric Kendall τ -b coefficient was used to test if the number of mislocalizations or referred sensations were associated with pain-related variables (intensity, duration, disability, kinesiophobia, and pain-related catastrophization). Statistical significance was set at $\alpha = 0.05$.

RESULTS

Group Characteristics

Table 1 describes the demographics of all the study participants and the clinical status of the participants with CLBP. Of note, 62.5% of the patient sample reported back pain and referred leg pain.

Differences in Sensory Function

Sixty-seven percent of people with CLBP reported at least 1 mislocalization, whereas only 25% of control participants mislocalized sensory information. This difference was statistically significant (Fisher exact $P = 0.034$). Figure 2 displays the frequency of participants experiencing up to 5 mislocalizations, by patient versus control group (although the maximum possible mislocalizations was 28, a maximum of only 5 was observed).

TABLE 1. Demographics and Clinical Status of Participants [Mean (SD) or n (%)]

| | Controls (n = 24) | CLBP Patients (n = 24) |
|--|----------------------|------------------------------|
| Age (y) | 42.0 (14.7) | 41.8 (15.0) |
| Female sex | 10 (41.7%) | 10 (41.7%) |
| Height (m) | 1.75 (0.11) | 1.73 (0.10) |
| Weight (kg) | 77.5 (14.4) | 80.2 (14.5) |
| Body mass index (kgm ⁻²) | 25.1 (2.8) | 26.9 (5.0) |
| Anxiety (HADS, 0-21) | 4.9 (2.7) | 6.8 (4.4) |
| Depression (HADS, 0-21) | 1.4 (1.8) | 4.8 (3.3) |
| Side tested: right | 10 (41.7%) | 12 (50.0%) |
| CLBP patient clinical status | | |
| Side of pain | | |
| Unilateral | | 8 (33.3%) |
| Bilateral with dominance | | 12 (50.0%) |
| Bilateral and equal | | 4 (16.7%) |
| Length of current episode [y; median (interquartile range)] | | 5.5 (17.5) |
| Taking opioids | | 9 (18.8) |
| Symptom distribution | | |
| Back only | | 9 (37.5%) |
| Above knee | | 6 (25.0%) |
| Below knee | | 9 (37.5%) |
| Off work because of LBP | | 3 (12.5%) |
| Pain intensity (VAS, 0-100) | | 45.0 (19) |
| Disability (RMDQ, 0-24) | | 9.9 (5.6) |
| Kinesiophobia (TSK, 17-68) | | 40.4 (6.5) |
| Catastrophization (PCS, 0-52) | | 18.2 (12.2) |

CLBP indicates chronic low back pain; HADS, Hospital Anxiety and Depression Scale; LBP, low back pain; PCS, Pain Catastrophizing Scale; RMDQ, Roland Morris Disability Questionnaire; TSK, Tampa Scale of Kinesiophobia; VAS, Visual Analogue Scale.

Referred sensations were experienced by 21% of people with CLBP and 12.5% of control participants. This difference was not statistically significant (Fisher exact $P = 0.381$). Figure 3 displays the frequency of participants experiencing up to 9 referred sensations, by patient versus control group (a maximum of 9 referred sensations out of possible 28 was observed). Visual feedback reduced the perception of referred sensations in 71% of referred sensations experienced by the patient group and all referred sensations experienced by the control group.

Relationships Between Sensory Function and Clinical Profile

Table 2 displays the associations between the number of mislocalizations/referred sensations and pain-related variables in the patient group. No statistically significant associations were detected.

DISCUSSION

The results confirmed our first hypothesis that CLBP patients would be poorer than pain-free controls in localizing sensory inputs. Sixty-seven percent of people with CLBP and only 25% of controls made at least 1 error when asked to indicate where on a body chart they had been touched. That is, atopognosia seems to be a feature of CLBP. Our results do not support the second hypothesis that people with CLBP would report more referred sensations than pain-free controls. Twenty-one percent of people with CLBP and 12.5% of controls reported referred sensations at some time during the testing protocol, however, this difference was not statistically significant. Our third hypothesis was also not supported as we found no relationship between sensory function and pain-related variables.

Although quite high rates of referred sensations are reported for phantom limb pain,⁵⁻⁷ previous research has suggested that only around 30% of people with CLBP experience some form of referred sensations,²⁵ a figure very similar to what has been reported in people with CRPS^{3,13} and neuropathic pain related to spinal cord injury.¹⁰ The lower rate reported in our study may represent a difference in severity between the patients in our sample and those in other investigations. In our study, patients were only included if the referring clinician felt they were suitable for

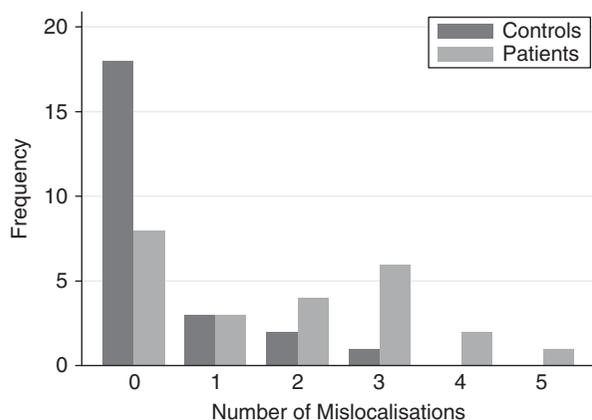


FIGURE 2. Frequency of 0 to 5 mislocalizations in control and patient groups.

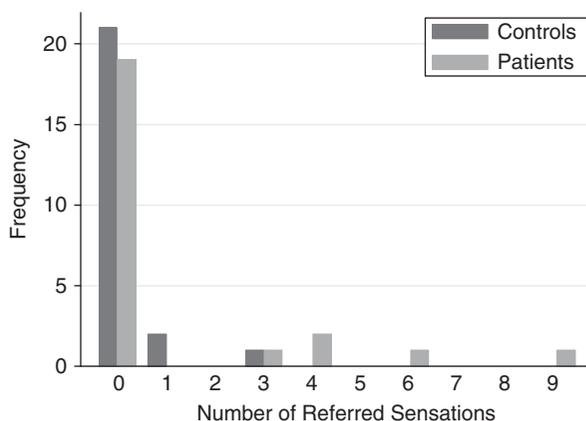


FIGURE 3. Frequency of 0 to 9 referred sensations in control and patient groups.

performance of a repeated movement assessment. This may have led to the exclusion of more disabled and distressed patients. This is particularly relevant as researchers have previously found evidence of somatosensory cortical reorganization in people with CLBP who were distressed but not in those who were not.³² Further investigation of referred sensations in people with CLBP may still be indicated, utilizing larger samples of more severely affected patients.

Although there are anecdotal reports of referred sensations in healthy individuals,³³ none of the experimental studies we identified found evidence of referred sensations in the control groups^{4,10,13} or in pain-free controls.^{5,10} Most studies, including our own, have used modest sample sizes and it may be that the failure of other researchers to identify referred sensations in control groups reflects the difficulty of identifying uncommon phenomena in small populations. Alternatively, it might be related to the body area tested. All stimulation in our study was provided to the posterior surface of the body and primarily to the spine and the thighs. The relatively small representation of these regions in primary sensory cortex, and the fact that these regions are rarely visualized, might make referred sensations from stimulating these segments more likely in healthy controls than they are from regions with larger representations, most notably the hand and the mouth.¹⁵ In addition, the high prevalence rates of low back pain means that identification of a truly healthy control group is likely

TABLE 2. Associations Between Number of Mislocalizations/ Referred Sensations and Pain-related Variables in the Patient Group (n = 24)

| | Mislocalizations | | Referred Sensations | |
|-----------------------------|-------------------|-------|---------------------|-------|
| | Kendall τ -b | P | Kendall τ -b | P |
| Pain intensity | 0.008 | 0.980 | 0.071 | 0.699 |
| Duration of current episode | 0.161 | 0.327 | -0.097 | 0.596 |
| Disability | -0.066 | 0.699 | 0.061 | 0.750 |
| Kinesiophobia | -0.053 | 0.757 | 0.307 | 0.077 |
| Catastrophization | 0.183 | 0.258 | -0.012 | 0.972 |

to be more difficult than with other much less common pain conditions such as CRPS or brachial plexus injury. We did not screen for the absence of back pain beyond 2 years and it is likely that some of the pain-free controls had previously experienced episodes of low back pain and this may have influenced our results. Finally, the protocol we adopted used a series of 5 stimulations that may have influenced the results; different outcomes might be seen with a single stimulation protocol. Clearly, further research of referred sensations on large groups of pain-free controls stimulating a variety of body areas is required.

This is the first report of atopognosia in patients with CLBP, and our data suggest that it is a common phenomenon. Previous studies have found normal tactile detection thresholds, yet deficits in 2-point discrimination^{34,35} and graphesthesia³⁵ over the back in CLBP patients and the findings of the present study add to this growing body of evidence suggesting that deficits in complex sensory function are a feature of the CLBP experience. Investigations of atopognosia in neurological patients suggest a dissociation between tactile detection and tactile localization. Several authors have described neurological cases in which detection of sensory stimulation is minimally affected, yet patients are unable to indicate where they have been touched.^{19–21} This would seem consistent with the data available for CLBP.

Evidence also suggests that primary somatosensory cortex disruption may be implicated in deficits of localization. Large parietal infarcts involving the primary somatosensory cortex have been identified in some neurological patients who demonstrate atopognosia.^{19,20} In addition, Braille readers who use 3 fingers simultaneously for reading display a greater degree of mislocalization of tactile stimulation of the fingers than single-finger Braille readers and non-Braille readers. Importantly, the 3-finger Braille readers demonstrated far greater reorganization in the primary somatosensory cortex than the other 2 groups as well as a significant relationship between cortical reorganization and mislocalisations.³⁶ Schweizer et al³⁷ have noted a worsening of mislocalizations to near threshold tactile stimulation of the hand when healthy patients engage in a training task known to disrupt normal somatosensory cortical maps. Also, Schaefer et al³⁸ used a visual illusion to induce mislocalization of sensory stimulation to the hand in healthy controls. Neuromagnetic source imaging showed that representation of the hand in the primary somatosensory cortex changed during the illusion in comparison to control stimulation that did not induce mislocalization. Finally, temporary disruption of somatosensory cortex function using transcranial magnetic stimulation has been shown to affect both detection and localization of tactile stimulation, although the effect on localization is more profound and long lasting.³⁹ Reorganization^{32,40} and degeneration⁴¹ within the primary somatosensory cortex appear to be a feature of CLBP, and the presence of atopognosia in CLBP patients may be the clinical correlate of these observed central nervous system changes.

The presence of atopognosia may simply be a consequence of ongoing pain and related emergent behaviors. However, it is plausible that atopognosis is maladaptive and contributes, at least in part, to the maintenance of the CLBP experience. Recent models of pain emphasize the importance of threat perception in generation of the pain experience⁴² and the loss of ability to accurately localize where nociceptive information is coming from might contribute to perceived threat. Consistent with this idea visualization of the stimulated body part, which improves

tactile acuity⁴³ and is very likely to improve localization, has been shown to be effective in reducing the intensity of experimental pain.^{44,45}

Some researchers have emphasized the role of cortical mechanisms in maintaining various chronic pain states.^{46,47} Specifically, it has also been argued that movement-related pain may arise as a result of incongruence between predicted and actual sensory feedback by virtue of disrupted body maps and disturbed body schema.^{46,47} A deficit in localization of sensory information from the back is a likely contributor to a mismatch between actual and expected sensory feedback and could contribute to ongoing movement-related pain by this mechanism. In addition, poor sensory function is likely to negatively impact on control of the spine with movement and during static tasks. This may lead to abnormal and noxious loading of spinal tissue and contribute to the maintenance of peripheral nociceptive input as a driver to the chronic pain state.

One final interpretation of the current results relates to the recent discovery of spatially defined deficits in sensory processing in people with back pain.⁴⁸ This work, undertaken in people with unilateral CLBP, revealed that tactile stimuli from the painful side were processed more slowly than identical stimuli from the nonpainful side. Importantly, the same was true when the stimuli were delivered to the hands and the hands were held next to the painful and nonpainful side of the back. This spatially defined deficit has also been observed in people with CRPS of 1 hand⁴⁹ and implicates a deficit in the integration of somatotopically based frame of reference with a space-based frame of reference (see Moseley et al² for review). Such a deficit may also explain problems that people with chronic pain have in performing motor imagery tasks relating to the painful area. The most studied motor imagery tasks involve making left/right judgements of pictured body parts. These tasks require the individual to mentally maneuver their own body part to match the posture of the part shown in the picture. This maneuver requires the transformation of location data between frames of reference, a task that is thought to depend on posterior parietal mechanisms (see Parsons⁵⁰ for review). Performance in this task is disrupted in people with CRPS,^{51,52} chronic back pain,⁵³ and chronic knee pain.⁵⁴ Sensory discrimination training, which aims to improve sensory localization ability and is likely to sharpen the somatotopically based frame of reference, has been shown to be effective in managing phantom limb pain⁵⁵ and CRPS.^{56,57} Our data suggest that these or similar approaches may be worth testing in people with CLBP.

The results presented here need to be interpreted in light of the study limitations. As data collection for patients and controls largely occurred at separate sites, it was not possible to blind the tester to the participants' clinical status, the rigor of this study would have been improved by the use of a blind assessor. It is also possible that attentional problems may underpin the results seen. Attentional functioning is known to be susceptible to pain interference,⁵⁸ and the poorer performance by patients on the localization task may have been influenced by the distracting influence of the pain. We attempted to mitigate this confounder by ensuring that patients were comfortably positioned throughout the testing. Furthermore, the lack of association between pain intensity and mislocalization suggests this might not be an important issue, although assessing present rather than average pain would have enabled better control of this issue. This study was conducted alongside a

randomized experiment analyzing the influence of mirror visual feedback on movement-related pain, and the sample size and patient characteristics were determined based on this experiment. As mentioned previously, this might have influenced the severity of patients accepted in to the study and decreased the representativeness of our sample, most significantly by excluding the more severe and distressed participants. The sample size might also not have been large enough to detect group differences in referred sensations as it appears to be a phenomenon with a fairly low incidence among chronic pain populations.

In summary, mislocalizations are more common in CLBP than in pain-free controls, but referred sensations are not. These data add to a growing body of evidence suggesting that disturbed self-perception is a feature of CLBP.

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