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RESEARCH ARTICLE

Low back skin sensitivity has minimal impact on active lumbar spine proprioception and stability in healthy adults

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Abstract The purpose of the current work was to (1) determine whether low back cutaneous sensitivity could be reduced through the use of a topical lidocaine–prilocaine anesthetic (EMLA®) to mirror reductions reported in chronic lower back pain (CLBP) patients, as well as to (2) identify whether reductions in cutaneous sensitivity resulted in decreased lumbar spine proprioception, neuromuscular control and dynamic stability. Twenty-eight healthy participants were divided equally into matched EMLA and PLACEBO treatment groups. Groups completed cutaneous minimum monofilament and two-point discrimination (TPD) threshold tests, as well as tests of sagittal and axial lumbar spine active repositioning error, seated balance and repeated lifting dynamic stability. These tests were administered both before and after the application of an EMLA or PLACEBO treatment. Results show that low back minimum monofilament and TPD thresholds were significantly increased within the EMLA group. Skin sensitivity remained unchanged in the PLACEBO group. In the EMLA group, decreases in low back cutaneous sensitivity had minimal effect on low back proprioception (active sagittal and axial repositioning) and dynamic stability (seated balance and repeated lifting). These findings demonstrate that treating the skin of the low back with an EMLA anesthetic can effectively decrease the cutaneous sensitivity of low back region. Further, these decreases in peripheral cutaneous sensitivity are similar in magnitude to those reported in CLBP patients. Within this healthy population, decreased cutaneous sensitivity of the low back region has minimal influence on active lumbar spine proprioception, neuromuscular control and dynamic stability.

Keywords Neuromuscular control · Cutaneous sensation · Local dynamic stability · Anesthetic · Sensory feedback · Kinematics

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Introduction

To complete dynamic motor tasks and ensure adequate stability of the lumbar spine, sensitivity of the proprioceptive and kinesthetic systems is necessary. Sensory feedback systems, which facilitate coordinated and stable movement patterns, include inputs from visual, vestibular, cutaneous, ligamentous and muscular subsystems (Proske and Gandevia 2012). In the absence of visual information, one's body representation in space (proprioception) has primarily been attributed to muscular receptors whereas a complimentary, supplementary or substitutory role has been attributed to more peripheral receptors, such as those within the skin (Aimonetti et al. 2012; Kavounoudias et al. 2001; Proske and Gandevia 2012). In particular, previous research has classified these skin receptors into separate groups responding independently to target biologically relevant phenomena (Johansson et al. 1982; Johansson and Vallbo 1979; Morioka et al. 2008). These receptors include rapidly adapting Meissner and Pacinian corpuscles which respond to skin slips/lateral movements and high-frequency

vibrations, respectively, as well as slowly adapting Merkel cells and Ruffini endings which respond to indentation and stretch, respectively (Gardner and Johnson 2013; Johnson 2001; Macefield 2005). In addition to these receptors, the skin of the torso also includes receptors located within hair follicles, which are especially sensitive to hair movement (Gardner and Johnson 2013). In chronic lower back pain (CLBP) patients, previous research has noted a decrease in the cutaneous sensitivity of low back using two-point discrimination tests (Moseley 2008; Catley et al. 2014). Furthermore, it has been suggested that the deficits in tactile acuity are related to the guarded/encumbered lumbopelvic motor movements associated with CLBP (Luomajoki and Moseley 2011). How the feedback from these cutaneous sensory receptors located within the low back contributes to the control of lumbar spine movement and proprioception is still unknown.

Previous works have demonstrated that CLBP patients have diminished tactile sensitivity atop the skin of the low back, paired with encumbered motor strategies of the lumbopelvic region (Luomajoki and Moseley 2011; Moseley 2008; Catley et al. 2014). It is possible that the deficits in tactile acuity stem from reorganization within the somatosensory cortex (Flor et al. 1997; Lloyd et al. 2008), changes in peripheral receptor sensitivity (such as is the case in diabetic neuropathies) or some combination of both factors. Based on these findings, recent research has begun to suggest that sensory retraining paradigms that target tactile sensitivity of a painful region can assist in reducing perceptions of pain as well as improving the motor control of the lumbar spine. These paradigms appear to be especially promising when paired with conventional physiotherapy style training paradigms (Wälti et al. 2015). It is currently unknown, however, whether the tactile acuity deficits observed in CLBP (as well as the effects of tactile sensory retraining) act through peripheral (e.g., cutaneous mechanoreceptor sensitivity) or central (cortical) neural mechanisms.

As a means to modulate cutaneous sensory feedback, previous works have employed the use of an anesthetic to decrease/eliminate cutaneous sensitivity from a particular anatomical area (Björkman et al. 2004; Cordo et al. 2011; Howe et al. 2015; Lowrey et al. 2010). With this idealized removal of skin information, researchers are able to speculate on how the eliminated cutaneous mechanosensitive feedback contributes to the control of posture, dynamic movement or proprioception. Alternatively, researchers can also infer whether individuals are able to cope with the loss of accessible sensory information with an increase in the reliance on information originating from the remaining senses. Based on this, the primary goals of the present study were to (1) discern whether decreases in lumbar tactile acuity (as a result of the EMLA treatment) in healthy participants could mirror those reported in CLBP patients, as well as to (2) identify whether these peripheral tactile insensitivities alone would result in larger movement variability, decreased stability and decreased proprioception of the lumbar spine.

Materials and methods

Participants

Fourteen male and fourteen female participants were divided evenly into age, height and weight-matched treatment and PLACEBO groups (Table 1). Participant exclusion criteria included the presence of pain within the lumbopelvic or lower-limb region as well as any diagnosed sensory or motor deficit disorders. All participants ($n = 28$) completed a health screening questionnaire and signed informed consent prior to data collection. The study was approved by the institutional research ethics board in accordance with the Declaration of Helsinki.

Procedure

All collections were completed at the same time of day, each with a total duration of approximately 4.5 h. Upon arrival, participants were randomly assigned into either an EMLA (EMLA®, Astra Zeneca: 2.5 % lidocaine/2.5 % prilocaine, topical anesthetic) or PLACEBO (inert, unscented moisturizing cream) treatment group. The first half an hour was allotted for instruction and practice of the experimental tasks as well as for the consent process. The remaining 4 h was used for skin sensitivity, proprioceptive and stability testing procedures as well as the administration of the anesthetic or placebo skin treatment (Fig. 1a). The order of proprioceptive and stability tasks were randomized and balanced between participants and were held in a constant order during repeated assessments within each participant. The treatment cream (EMLA or PLACEBO) was applied in a dose of 0.1 g/cm^2 atop the skin of the low back for a period of 2 h. During this time, participants lay prone on

Table 1 Mean (±SD) EMLA and PLACEBO group participant characteristics

Group	Height (cm)	Weight (kg)	Age (years)	Dose area (cm)
EMLA (7 males; 7 females)	174.9 (±10.2)	70.6 (±12.5)	24 (±2.2)	217.1 (±46.9)
PLACEBO (7 males; 7 females)	176.8 (±8.8)	76.8 (±16.5)	24 (±2.9)	218.6 (±30.9)

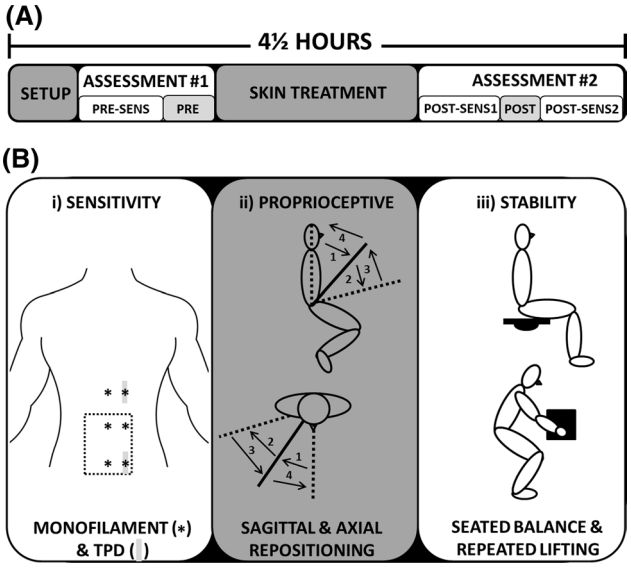

Fig. 1 Schematic depiction of each data collection session showing both the **a** timeline of procedure as well as **b** each individual sensitivity (PRE-SENS, POST-SENS1 and POST-SENS2) or proprioceptive/stability (PRE and POST) assessment. Tests included *i* minimum monofilament tactile sensitivity (MMTS) and two-point discrimination (TPD) threshold testing both within (L1 and L4) and outside (T9) of the experimental test area (*dashed box*), *ii* proprioceptive acuity testing for both flexion/extension and axial twisting movements and *iii* dynamic stability estimation during both seated stability and repetitive sagittal lifting tasks

a chiropractic bench while also completing 2-min walking bouts every 30 min. These short walks were done to mitigate any effect of maintaining a prolonged prone posture (e.g., intervertebral disk swelling or low back discomfort). The boundaries for cream application were defined anatomically based on the superior–inferior (SI) locations of the T12 and S1 spinous processes as well as the medial–lateral (ML) peripheral boundaries of the left and right paraspinal muscle bellies (Fig. 1b, i). This anatomical location was targeted due to reports of decreased cutaneous sensitivity in CLBP patients within this region (Luomajoki and Moseley 2011; Moseley 2008). The average dose area for each participant group is presented in Table 1. During the administration of the EMLA or PLACEBO treatment, all participants, and experimenters taking any measurements, were blinded to the type of skin treatment cream.

Cutaneous sensitivity tests

Cutaneous sensitivity was measured across three separate assessments relative to the application of either the EMLA or PLACEBO treatment cream. These assessments included measures prior to cream application (PRE-SENS), immediately following cream removal (POST-SENS1) as well as immediately following the completion of the second round of lumbar spine proprioceptive and stability tasks (POST-SENS2). The POST-SENS1 and POST-SENS2 assessments were separated in time by ~40–45 min. Control test locations (outside of dose area) were assessed at the T9 level both immediately superior to the vertebral spinous process (T9SP) and superior to the right-side paraspinal muscle belly (T9MB). Experimental test locations (within dose area) were assessed at the L1 and L4 vertebral levels, again both superior to the vertebral spinous process (e.g., L1SP) and muscle belly (e.g., L4MB). Prior to any cutaneous sensitivity tests, each test location was lightly shaved with a disposable razor, with care not to abrade the skin. Two cutaneous sensitivity tests were administered: a Semmes–Weinstein (Semmes et al. 1960; Weinstein 1993) minimum monofilament tactile sensitivity (MMTS) test (e.g., Wand et al. 2010) as well as a two-point discrimination (TPD) threshold test (e.g., Catley et al. 2013; Johnson and Phillips 1981; Luomajoki and Moseley 2011; Moseley 2008; Nolan 1985; Wand et al. 2014). MMTS tests were completed at six anatomical test locations including T9SP, T9MB, L1SP, L1MB, L4SP and L4MB. TPD tests were completed in a superior–inferior orientation at two anatomical test locations, T9MB and L4MB. Both the MMTS and TPD tests utilized a modified stepwise 4-2-1 approach (Dyck et al. 1993) such that minimum sensitivity threshold was defined as a 66 % success rate (e.g., Frost et al. 2015).

Lumbar spine proprioception tests

Proprioceptive acuity was tested across two separate assessments relative to the application of either the EMLA or PLACEBO treatment cream. The first tests were completed prior to cream application, immediately following the cutaneous sensitivity assessments (PRE), while the second tests were completed following cream removal, again immediately following the cutaneous sensitivity tests (POST). Proprioceptive tests included active lumbar repositioning (Fig. 1b, ii) within both the sagittal (e.g., Brumagne et al. 2000) and axial (e.g., Silfies et al. 2007) planes. Sagittal repositioning was performed while seated in a kneeling chair, while axial repositioning was performed while seated upright on a twisting platform. For each task, participants were required to (without the use of vision, at a self-selected pace) either flex or rightward twist, away from their starting neutral position, toward a random target within ~10° of each participant's neutral range of motion (ROM). After holding this target for ~2 s, participants were required to flex/twist to their end ROM and hold this position again for ~2 s. Next, participants were instructed to begin moving back toward the neutral position and, however, were instructed to pause for an additional ~2 s when they believed they were repositioned at the original target location (Fig. 1b, ii). Each of the sagittal

and axial repositioning tests was repeated five times at both PRE and POST assessments. Sagittal repositioning kinematic data were acquired from rigid bodies consisting of three non-collinear kinematic markers affixed at the T12 and S1 vertebral levels. Axial repositioning kinematic data were acquired from a rigid body placed on the twist platform. Both sets of kinematic data were sampled at 100 Hz (Optotrak 3D Investigator, Northern Digital, Waterloo, ON, Canada).

Lumbar spine stability tests

Similar to the proprioceptive acuity tests, each lumbar spine stability test was completed at the same PRE and POST assessments. Stability tests included both a seated balance (e.g., Reeves et al. 2006; van Dieën et al. 2010) test as well as a repeated sagittal lifting (e.g., Beaudette et al. 2014; Graham et al. 2012a, b) tests (Fig. 1b, iii). For the seated balance assessment, participants sat atop a hemispherical wobble board (10 cm radius) placed on a force platform (True Impulse, Northern Digital, Waterloo, ON, Canada); data were sampled at 50 Hz for 60 s. Participants were instructed to remain as stable as possible with their arms crossed against their chest and legs strapped together at the knees and ankles to minimize potential compensatory leg movements. Each seated balance test was repeated five times at both the PRE and POST assessments. For the repeated sagittal lifting test, participants were required to lift a light load (4.5 kg) for 30 consecutive repetitions using similar pacing and setup as outlined in previous works (Beaudette et al. 2014). 3D lumbar spine kinematic data for the repeated lifting test were sampled at 100 Hz from rigid bodies again placed at the T12 and S1 vertebral levels (same as the sagittal repositioning task).

Data processing and analysis

Preprocessing

Raw kinematic data from the repositioning and repeated lifting tasks were filtered (4th order dual-pass Butterworth) with a 6 Hz low-pass cutoff. To quantify 3D lumbar spine angles (for the sagittal plane repositioning task as well as the repeated lifting task), a flexion/extension (FE), lateral bend (LB), axial twist (AT) Cardan rotational sequence was implemented. For the repeated lifting test, these 3D lumbar spine angular data were used in the estimation of lumbar FE, LB and AT ROM as well as FE, LB and AT ROM variability (standard deviation of each repeated lift to lift ROM). To quantify 3D axial rotations during the axial repositioning test, the twist platform rigid body orientation was quantified in 3D global space. For the seated balance test, raw voltages from the force platform were filtered (fourth-order dual-pass Butterworth) with a low-pass cutoff of 10 Hz. These voltages were calibrated into triaxial forces and moments which were subsequently used to calculate anterior–posterior (AP) and ML center of pressure (CoP) trajectories. Using these CoP waveforms, AP and ML displacements and velocities were quantified and assessed using maximum range, root mean square (RMS) and cumulative path length outcome parameters (e.g., van Dieën et al. 2010).

Repositioning error

To assess sagittal or axial angular lumbar spine active repositioning error, three estimates were used: constant error (CE), absolute error (AE) and variable error (VE) (e.g., Brumagne et al. 2000; Rausch Osthoff et al. 2015). CE values assess a participant's tendency toward over-/undershooting the target angle such that positive values represent the magnitude (in degrees) of overshoot. AE values quantify the amount of error, irrespective of direction ($AE = |CE|$), associated with active repositioning and quantifies the proprioceptive accuracy. VE values quantify the variability (standard deviation) in CE values and quantify proprioceptive precision throughout repeated measurements. Previous research has demonstrated good within-session reproducibility ($0.47 < ICC < 0.61$, $0.57° < SEM < 0.73°$) for active repositioning measurements, particularly within the axial plane (e.g., Silfies et al. 2007).

Local dynamic stability (LDS)

To estimate the LDS, the maximum finite-time Lyapunov exponent (LyE) was calculated for each seated balance and repeated lifting time series. Previous research studies have demonstrated good to strong intra-session reliability for LyE estimates of trunk dynamic stability using kinetic seated balance data ($0.75 < ICC < 0.83$; Lee and Granata 2008), as well as kinematic repeated sagittal flexion–extension data ($0.39 < ICC < 0.73$; Graham et al. 2012a, b). Using this method (Rosenstein et al. 1993), positive LyEs are indicative of dissipative (unstable) dynamics and deterministic chaos whereas negative LyEs are representative of convergent (stable) dynamics (Stergiou et al. 2004). These estimates have been used previously in the analysis of trunk movements during walking (e.g., Bruijn et al. 2009; Dingwell and Cusumano 2000), joint movements during repeated lifting (e.g., Graham and Brown 2012) and walking (Beaudette et al. 2015), as well as during standing balance tests (Roerdink et al. 2006). The time series data used to estimate LDS were Euclidean norm lumbar spine angles during the repeated lifting

movements, as well as AP and ML CoP displacement data during the seated balance tasks. To best ensure steady-state repeated lifting dynamics, lifts 1–5 were excluded. Similarly, to mitigate any data collection initiation/termination effects, the first and last 5 s of each seated balance trial were excluded. For repeated lifting, a time delay (Td) of 10 % of a lifting cycle was used, paired with an embedding dimension (d_E) of 6, estimated using a global false nearest neighbors (GFNN) analysis (Kennel et al. 1992). LyE values for the repeated lifting movements were approximated from the average local divergence curve between 0 and 0.5 lifting cycles to mirror previous analyses (Beaudette et al. 2014; Graham et al. 2012a, b; Graham and Brown 2012). For seated balance, a Td of 11 and 10 frames was used for the AP and ML CoP displacement data, respectively, paired with a d_E of eight. These Tds were chosen such that x_i and x_{i+Td} were not strongly correlated ($R^2 < 0.7$) (e.g., Ramdani et al. 2013), and the d_E were again selected based on a GFNN analysis. LyE estimations for the seated balance data were approximated from the average local divergence curve between 0 and 1 s. All of these analyses were performed using custom software developed in MATLAB (The MathWorks, Natick MA, USA).

Statistical analysis

Dependent variables for the statistical analyses included: (1) cutaneous MMTS and TPD thresholds, (2) sagittal and axial CE, AE and VE estimates of repositioning error, (3) seated balance estimates of AP and ML CoP displacement and velocity ranges, RMS, cumulative path lengths and AP and ML LyE estimates and (4) repeated lifting estimates of FE, LB and AT ROM, ROM variability and LyE estimates of LDS. Independent variables included the time point of assessment (e.g., PRE/POST or PRE-SENS/POST-SENS1/POST-SENS2) as well as the location of the cutaneous sensitivity tests (e.g., T9SP, T9MB, etc.). For each cutaneous sensitivity test (MMTS and TPD), two separate (EMLA and PLACEBO) two-way, repeated measures ANOVAs were completed to evaluate the impact of PRE-SENS/POST-SENS1/POST-SENS2 assessment and location on each respective dependent variable. For all other tests (axial and sagittal repositioning, seated balance and repeated lifting), two separate (EMLA and PLACEBO) one-way repeated measures ANOVAs were performed to evaluate the effect of each PRE/POST assessment on each respective dependent variable. For all analyses, post hoc pairwise multiple means comparisons were made using a Tukey adjustment to determine the cause of any significant main effects ($\alpha = 0.05$). All statistical analyses were performed using SAS 9.2 (SAS Institute, Cary NC, USA).

Results

Cutaneous sensitivity tests

Within the EMLA group, there was a significant two-way PRE-SENS/POST-SENS1/POST-SENS2 assessment × location interaction for both the MMTS (F-ratio = 2.11, $p = 0.0243$) and TPD (F-ratio = 9.05, $p = 0.0003$) threshold data. Post hoc tests revealed that the EMLA treatment induced significantly higher MMTS and TPD thresholds, indicating reduced sensitivity, within the test locations, especially at POST-SENS1. Specifically, participants in the EMLA group had larger MMTS thresholds at the L1SP ($p = 0.0434$) and L4SP ($p = 0.0006$) locations during the POST-SENS1 assessment (when compared to PRE-SENS) (Fig. 2a). In addition to these MMTS changes, participants in the EMLA group also had larger TPD thresholds at the L4MB test location during both the POST-SENS1 ($p = 0.0001$) and POST-SENS2 ($p = 0.0082$) assessments (when compared to PRE-SENS) (Fig. 3a). All MMTS and TPD measurements at the control T9 level (T9SP or T9MB) were unaffected by the EMLA treatment. Within the PLACEBO group, MMTS thresholds were constant across all PRE-SENS/POST-SENS1/POST-SENS1 assessments (F-ratio = 0.59, $p = 0.5525$) and across all locations (F-ratio = 0.45, $p = 0.8152$) (Fig. 2b). TPD data from the PLACEBO participant group were observed to differ significantly based on location (F-ratio = 4.22, $p = 0.0433$); however, were unaffected based on the PRE-SENS/POST-SENS1/POST-SENS2 assessment being analyzed (F-ratio = 0.06, $p = 0.9410$). Post hoc tests revealed that TPD thresholds at the L4MB level (28.7 mm) were significantly lower than those at the T9MB level (34.9 mm) in the PLACEBO participant group (Fig. 3b).

Lumbar spine proprioception tests

Within the EMLA group, sagittal repositioning estimates of CE (F-ratio = 0.01, $p = 0.9158$), AE (F-ratio = 1.21, $p = 0.2742$) or VE (F-ratio = 0.46, $p = 0.5051$) were all unaffected by PRE/POST assessment. Similarly, within the PLACEBO group, sagittal repositioning estimates of CE (F-ratio = 1.17, $p = 0.2819$), AE (F-ratio = 0.44, $p = 0.5081$) and VE (F-ratio = 0.05, $p = 0.8187$) were again unaffected by PRE/POST assessment. For the EMLA group, axial repositioning estimates of VE were observed to differ significantly between PRE and POST assessments (F-ratio = 7.04, $p = 0.0134$); however, CE (F-ratio = 0.69, $p = 0.4080$) and AE (F-ratio < 0.01, $p = 0.9918$) estimates were unaffected. Specifically, within the EMLA group axial VE data, post hoc analyses revealed participants had significantly larger VE during POST when compared

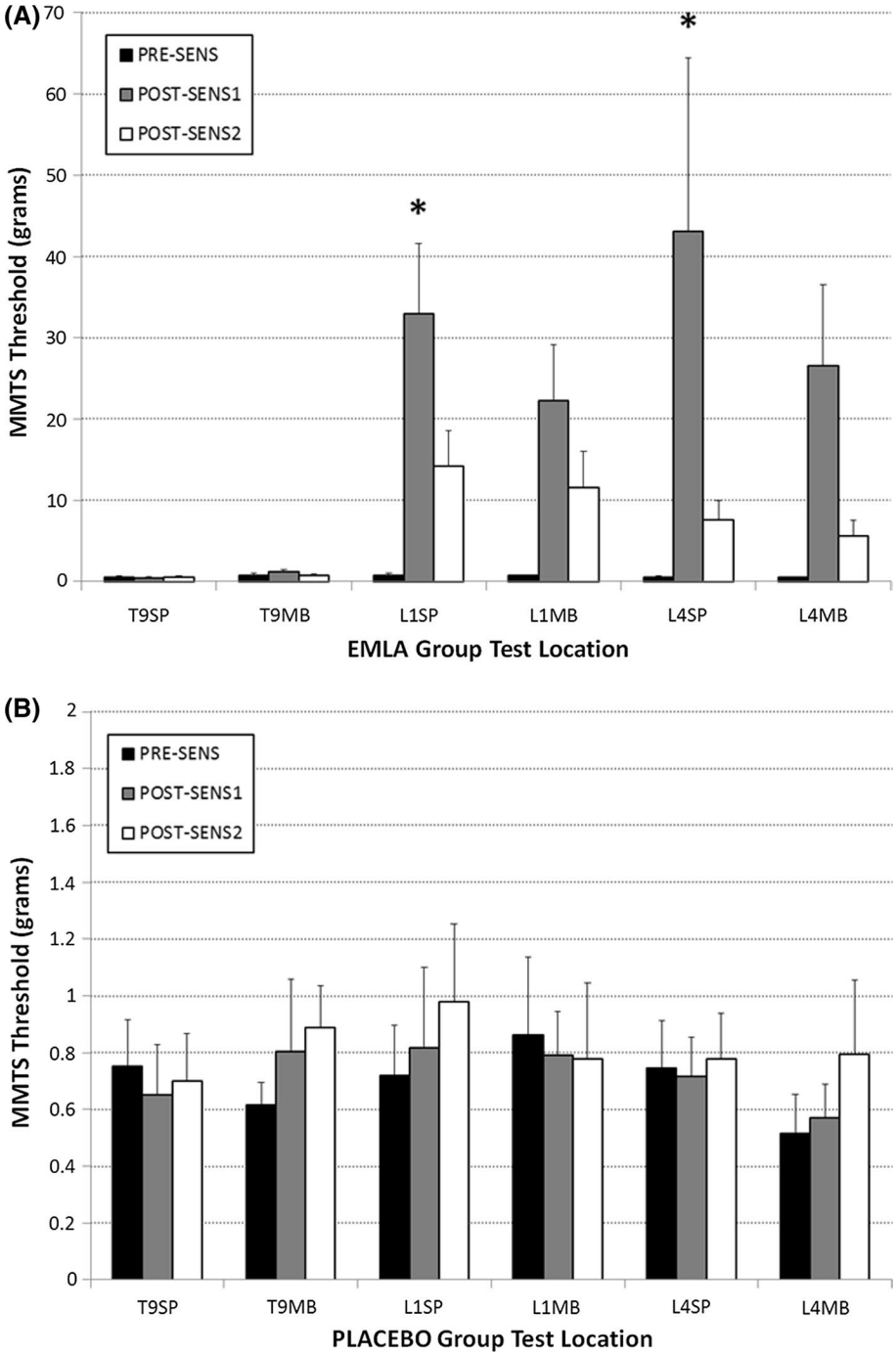

Fig. 2 Minimum monofilament tactile sensitivity (MMTS) threshold (in g) for the **a** EMLA and **b** PLACEBO groups. Note that an increased threshold indicates a decreased sensitivity. Monofilament data are presented from the T9 spinous process (T9SP) and muscle belly (T9MB) control locations as well as the experimental L1 and L4 spinous process (L1SP and L4SP) and muscle belly (L1MB and L4MB) test locations. *Asterisks* show significant post hoc differences ($p < 0.05$) relative to the PRE-SENS assessment at each location. All data are presented as mean ± SEM

to PRE (Table 2). For the PLACEBO group, axial repositioning estimates of CE (F-ratio = 0.19, $p = 0.6645$), AE (F-ratio < 0.01, $p = 0.9748$) and VE (F-ratio = 0.99, $p = 0.3290$) were all unaffected by the PRE/POST assessment. All repositioning data are given in Table 2.

Lumbar spine stability tests

Within the EMLA group, seated balance outcomes of CoP AP velocity range (F-ratio = 7.20, $p = 0.0082$) and AP velocity RMS (F-ratio = 4.66, $p = 0.0327$) were observed to differ significantly across PRE/POST assessments. Further, within the EMLA group, estimates of CoP path length (F-ratio = 3.61, $p = 0.0596$) and ML LyE (F-ratio = 2.99, $p = 0.0861$) tended toward being different across PRE/POST assessments. Specifically, post hoc tests revealed that CoP AP velocity ranges and AP velocity RMS were larger during PRE when compared to POST. Similar trends were observed for the CoP path length and ML LyE estimates. Within the PLACEBO group, seated

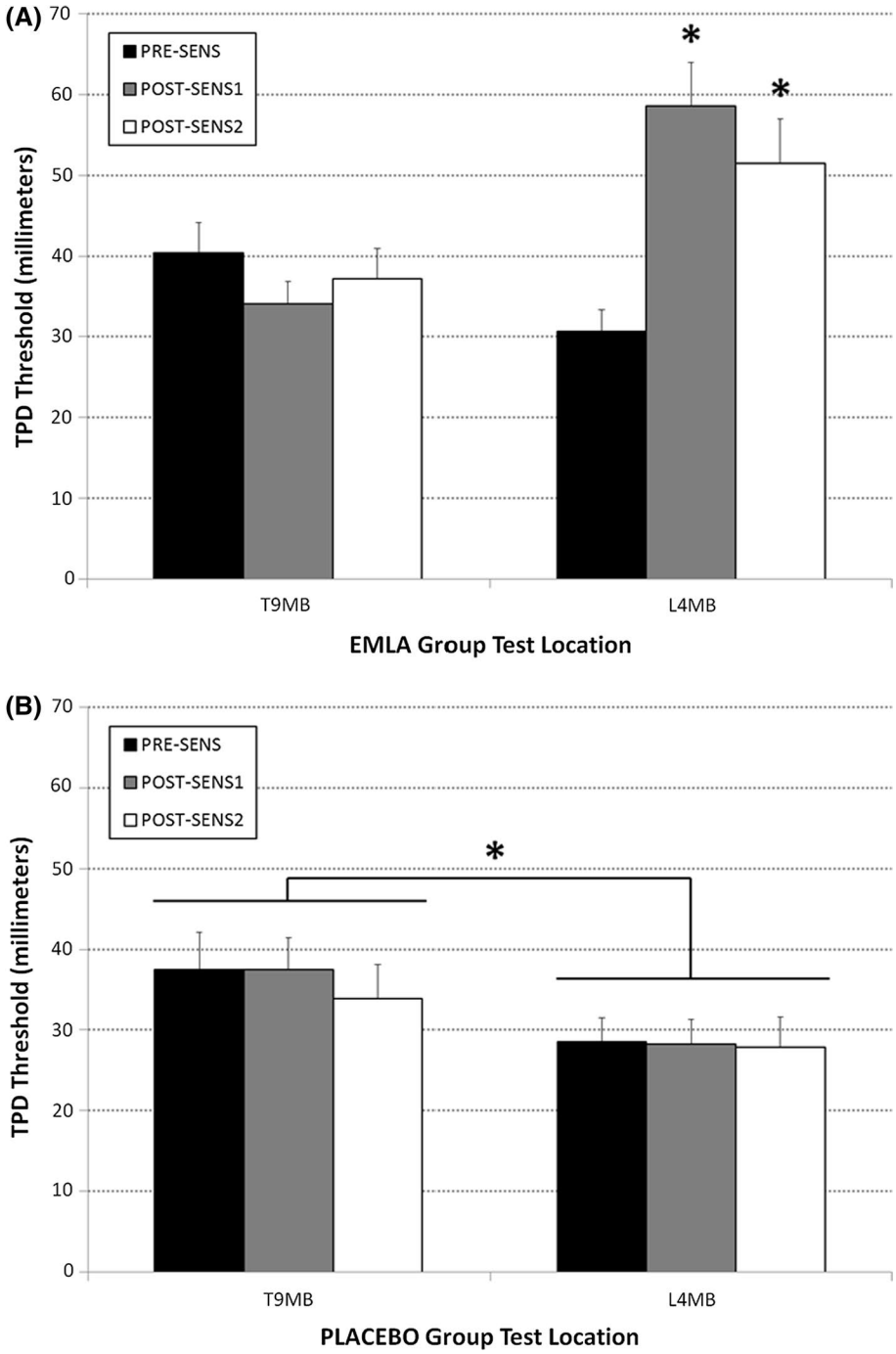

Fig. 3 Minimum two-point discrimination (TPD) threshold (in mm) for the **a** EMLA and **b** PLACEBO groups. Note that an increased threshold indicated a decreased sensitivity. TPD data are presented from the T9 muscle belly (T9MB) control location as well as the experimental L4 muscle belly (L4MB) test location. *Asterisks* show significant post hoc differences ($p < 0.05$) either between locations or relative to the PRE-SENS assessment at each location. All data are presented as mean ± SEM

balance estimates of AP LyE were observed to differ significantly across PRE/POST assessments (F-ratio = 8.34, $p = 0.0045$). Further, estimates of CoP AP velocity range (F-ratio = 2.95, $p = 0.0883$), ML velocity range (F-ratio = 2.98, $p = 0.0868$) and ML velocity RMS (F-ratio = 2.87, $p = 0.0924$) tended toward being different across PRE/POST assessments. Specifically, post hoc tests revealed that the AP LyE estimate was significantly larger during PRE when compared to POST. Further, similar trends were observed for CoP AP velocity range, ML velocity range and ML velocity RMS. All seated balance data are given in Table 3. For both the EMLA and PLACEBO participant groups, all repeated lifting dependent variables (FE, LB and AT range and variability as well as LyE) were unaffected by each PRE/POST assessment. All repeated lifting data are given in Table 4.

Table 2 Mean (±SEM) lumbar spine sagittal and axial active repositioning errors

Assessment	Treatment	Outcome	PRE (°)	POST (°)	*F*-ratio (*p* value)	Effect size (*d*)
Sagittal repositioning	EMLA	CE	3.04 (±2.0)	3.20 (±2.2)	0.01 (0.92)	0.02
		AE	7.08 (±1.0)	8.05 (±1.2)	1.21 (0.27)	0.25
		VE	3.63 (±0.5)	4.20 (±0.7)	0.46 (0.51)	0.31
	PLACEBO	CE	2.70 (±1.7)	4.04 (±1.7)	1.17 (0.28)	0.22
		AE	5.84 (±1.0)	6.41 (±1.0)	0.44 (0.51)	0.17
		VE	3.83 (±0.6)	3.66 (±0.5)	0.05 (0.82)	0.12
Axial repositioning	EMLA	CE	−0.10 (±1.3)	0.65 (±1.2)	0.69 (0.41)	0.16
		AE	4.13 (±0.7)	4.13 (±0.6)	<0.01 (0.99)	<0.01
		VE	2.21 (±0.2)	3.11 (±0.3)	7.04 (0.01)*	1.28
	PLACEBO	CE	0.6 (±0.7)	0.33 (±0.8)	0.19 (0.66)	0.10
		AE	2.77 (±0.4)	2.78 (±0.3)	<0.01 (0.97)	0.01
		VE	2.61 (±0.4)	2.16 (±0.3)	0.99 (0.33)	0.42

Error estimates include constant errors (CE), absolute errors (AE) and variable errors (VE) for each of the EMLA and PLACEBO participant groups

d effect size (Cohen's *d*)

* Significance at the $p < 0.05$ level

Table 3 Mean (±SEM) seated stability outcome measures for the EMLA and PLACEBO participant groups

Treatment	Outcome	PRE	POST	*F*-ratio (*p* value)	Effect size (*d*)
EMLA	AP range (cm)	1.70 (±0.2)	1.56 (±0.2)	1.03 (0.31)	0.26
	ML range (cm)	1.69 (±0.2)	1.61 (±0.2)	0.27 (0.61)	0.14
	AP RMS (cm)	0.26 ($\pm 2.6 \times 10^{-2}$)	0.25 ($\pm 2.9 \times 10^{-2}$)	0.01 (0.93)	0.02
	ML RMS (cm)	0.25 ($\pm 2.1 \times 10^{-2}$)	0.24 ($\pm 3.3 \times 10^{-2}$)	0.11 (0.75)	0.08
	AP vel. range (cm/s)	18.13 (±2.8)	14.09 (±0.7)	7.20 (0.01)*	0.71
	ML vel. range (cm/s)	13.2 (±1.0)	12.40 (±1.0)	0.76 (0.39)	0.27
	AP vel. RMS (cm/s)	1.83 (±0.3)	1.53 ($\pm 7.3 \times 10^{-2}$)	4.66 (0.03)	0.50
	ML vel. RMS (cm/s)	1.32 ($\pm 7.9 \times 10^{-2}$)	1.27 ($\pm 5.7 \times 10^{-2}$)	0.93 (0.34)	0.26
	Cumulative path length (cm)	96.36 (±10.5)	86.37 (±3.3)	3.61 (0.06)†	0.44
	AP LyE	0.58 ($\pm 3.1 \times 10^{-2}$)	0.59 ($\pm 2.7 \times 10^{-2}$)	0.31 (0.58)	0.13
	ML LyE	0.76 ($\pm 1.1 \times 10^{-2}$)	0.73 ($\pm 2.0 \times 10^{-2}$)	2.99 (0.09)†	0.55
PLACEBO	AP range (cm)	1.88 (±0.2)	1.64 (±0.2)	2.22 (0.14)	0.30
	ML range (cm)	1.84 (±0.2)	1.62 (±0.2)	2.23 (0.14)	0.34
	AP RMS (cm)	0.30 ($\pm 3.7 \times 10^{-2}$)	0.28 ($\pm 4.3 \times 10^{-2}$)	0.64 (0.42)	0.16
	ML RMS (cm)	0.28 ($\pm 2.9 \times 10^{-2}$)	0.25 ($\pm 2.9 \times 10^{-2}$)	1.82 (0.18)	0.29
	AP vel. range (cm/s)	15.77 (±1.4)	14.01 (±0.9)	2.95 (0.09)†	0.46
	ML vel. range (cm/s)	13.89 (±1.2)	12.35 (±0.9)	2.98 (0.09)†	0.45
	AP vel. RMS (cm/s)	1.58 ($\pm 9.7 \times 10^{-2}$)	1.53 ($\pm 7.7 \times 10^{-2}$)	0.53 (0.47)	0.15
	ML vel. RMS (cm/s)	1.37 ($\pm 9.6 \times 10^{-2}$)	1.26 ($\pm 6.6 \times 10^{-2}$)	2.87 (0.09)†	0.39
	Cumulative path length (cm)	89.45 (±5.4)	86.24 (±3.9)	0.96 (0.33)	0.20
	AP LyE	0.64 ($\pm 1.9 \times 10^{-2}$)	0.59 ($\pm 3.0 \times 10^{-2}$)	8.34 (<0.01)*	0.66
	ML LyE	0.76 ($\pm 2.3 \times 10^{-2}$)	0.75 ($\pm 2.6 \times 10^{-2}$)	0.43 (0.51)	0.14

Note that a smaller LyE value represents an increase in stability

d effect size (Cohen's *d*)

* Significant PRE/POST assessment differences at the $p < 0.05$ level

† Comparisons tending toward being different across PRE/POST assessments ($p < 0.10$)

Table 4 Mean (±SEM) repeated lifting outcomes for the EMLA and PLACEBO participant groups

Treatment	Outcome	PRE	POST	*F*-ratio (*p* value)	Effect size (*d*)
EMLA	FE ROM (°)	27.65 (±3.3)	26.75 (±3.1)	0.04 (0.85)	0.08
	LB ROM (°)	3.17 (±0.2)	3.41 (±0.3)	0.40 (0.53)	0.27
	AT ROM (°)	2.37 (±0.2)	2.94 (±0.5)	1.13 (0.30)	0.48
	FE ROM variability (°)	3.12 (±0.5)	2.55 (±0.4)	0.71 (0.41)	0.34
	LB ROM variability (°)	1.11 (±0.1)	0.93 (±0.1)	2.71 (0.11)	0.71
	AT ROM variability (°)	0.68 (±0.1)	0.70 (±0.1)	0.06 (0.81)	0.10
	Euclidean norm LyE	2.15 (±0.1)	2.07 (±0.1)	0.25 (0.62)	0.22
PLACEBO	FE ROM (°)	26.29 (±2.2)	26.93 (±2.8)	0.03 (0.86)	0.07
	LB ROM (°)	3.66 (±0.3)	3.73 (±0.3)	0.03 (0.86)	0.07
	AT ROM (°)	2.63 (±0.2)	2.85 (±0.4)	0.23 (0.63)	0.19
	FE ROM variability (°)	2.35 (±0.3)	2.71 (±0.4)	0.60 (0.44)	0.32
	LB ROM variability (°)	1.10 (±0.1)	1.21 (±0.1)	0.35 (0.56)	0.23
	AT ROM variability (°)	0.73 (±0.1)	0.73 (±0.1)	<0.01 (0.96)	0.02
	Euclidean norm LyE	2.04 (±0.1)	1.95 (±0.1)	0.29 (0.60)	0.23

Note that a smaller LyE value represents an increase in stability

d effect size (Cohen's *d*)

Discussion

The primary goals of the present study were to (1) determine whether topical anesthesia (EMLA®) atop the skin of the low back had the capacity to decrease cutaneous mechanosensitivity (mirroring cutaneous insensitivities observed in CLBP patients) and (2) to assess whether this decrease in cutaneous sensitivity of the low back influenced estimates of lumbar spine proprioception, dynamic movement control and LDS in a group of healthy young adults. We were able to confirm a decrease in cutaneous sensitivity in response to an EMLA treatment; however, we were unable to detect any consistent declines in lumbar spine proprioception, dynamic movement control or LDS in response to decreased skin sensitivity.

In accordance with our first hypothesis, the EMLA topical anesthetic decreased cutaneous sensitivity based on our estimates of MMTS and TPD thresholds (Figs. 2a, 3a). Inspecting our POST-SENS1 cutaneous assessments, the current data show that EMLA induced an average increase in MMTS threshold of 6462 %, paired with an average increase in TPD threshold of 200 %, across all test locations (e.g., L1 and L4) relative to the original PRE-SENS assessment. This cutaneous insensitivity persisted until our final POST-SENS2 cutaneous assessment for both the MMTS (2038 % increase) and TPD (175 % increase) threshold measures. These data demonstrate a decrease in mechanoreceptor sensitivity within the skin of the low back, which would suggest a decreased ability of this skin area (mean 217.11 ± 12.5 cm^2) to contribute to any sensory feedback needed to control dynamic movement of the lumbar spine. These reductions are similar to those shown previously using topical anesthesia to decrease the cutaneous sensitivity within the upper and lower limbs (e.g., Björkman et al. 2004; Howe et al. 2015; Lowrey et al. 2010). Further, it is also noteworthy that the decreased TPD sensitivity in our EMLA treatment group is in line with the reported sensory deficit experienced in CLBP patients (e.g., TPD estimates of ~60–70 mm compared to a ~40–50 mm healthy control) (Luomajoki and Moseley 2011; Moseley 2008).

In partial accordance with our second hypothesis, decreased cutaneous sensitivity of the skin of the low back resulted in decreased axial repositioning precision (VE) within our EMLA treatment group (Table 2). This suggests that the cutaneous information from the skin of the low back may be used, to some degree, to provide feedback for the precision of active movements of the lumbar spine within the axial plane. Since the FE ROM is relatively low within these tasks, changes in paraspinal and intrinsic back muscle lengths is likely minimized. As such, being that muscle spindle signaling is especially adept to code for changes in muscle length (Fallon and Macefield 2007), it is possible that the muscular contributions to sensory feedback during the axial repositioning tasks are limited (relative to the sagittal repositioning and repeated lifting tasks). Therefore, it is possible that feedback from the skin of the low back serves a larger role within these axial plane tasks, and further, removal of cutaneous sensory feedback has some effect. However, overall, any potential effects of the decreased cutaneous sensitivity were limited, as no other variables assessed within the current paradigm demonstrated a significant effect. Further, the small effect sizes (Cohen's D) pre- versus post-EMLA application support the notion of a lack of a clinically significant impact. Being that previous research has determined a reliance on

cutaneous sensory feedback to control upright standing using a vibrotactile stimulation paradigm (Lee et al. 2012; Martin et al. 2015), it is possible that our anesthesia treatment was too focal and that other areas of skin (or other sensory systems) were able to compensate for the decreased low back cutaneous sensitivity. It is also possible that under different circumstances (e.g., during standing balance), the contribution of cutaneous sensory feedback may have a more profound effect in the maintenance of lumbar spine proprioception or stability. Further research to assess the relative dependence of muscular and cutaneous sensory feedback, across various static postures and dynamic tasks, is needed to determine whether this is the case.

Contrary to our second hypothesis, it is clear that a decreased sensitivity of the skin of the low back has no significant effect on estimates of active sagittal repositioning error (Table 2), seated balance (Table 3) or repeated lifting (Table 4). Through previous research, active sagittal repositioning tests have been shown to be sensitive to vibrational sensory perturbations in healthy participants (Brumagne et al. 2000; Hidalgo et al. 2013); however, cutaneous sensory perturbations (such as taping techniques designed to illicit skin stretch) have shown mixed results (Hidalgo et al. 2013; Ruggiero et al. 2015). Seated balance tests, similar to those used in the current paradigm, have also been used previously in the analysis of trunk stiffening (Reeves et al. 2006), as well as overall trunk postural control (van Dieën et al. 2010). Furthermore, the repeated lifting test has been shown to be sensitive to levels of muscular mediated stiffening (Beaudette et al. 2014; Graham and Brown 2012), experimental pain (Ross et al. 2015) and movement pace (Granata and England 2006). Therefore, these tests of lumbar spine movement control have been shown previously to be sensitive to various sensory perturbations and task demands. The current results demonstrate that information from the skin of the low back has little effect in providing feedback for dynamic movements of the lumbar spine, especially when the necessary muscular activation is relatively large (e.g., seated balance, repeated lifting), or when the motion is within the sagittal plane (e.g., sagittal repositioning or repeated lifting). As noted above, it is therefore possible that these movements are feedback-controlled predominantly by other remaining sensory sources unaffected by the EMLA topical anesthetic (e.g., muscle spindle sensory feedback). This reliance on muscle-mediated feedback within sagittal plane movements has been noted during previous research using vibrational interventions designed to target muscle spindle sensory channels (e.g., Boucher et al. 2013, 2015; Kiers et al. 2014, 2015; Willigenburg et al. 2012; Willigenburg et al. 2013). Although healthy participants were studied the current research, the data from the present study suggest that the peripheral tactile insensitivities, observed within CLBP patients, alone cannot account for the reported changes in lumbar spine movement control. It is possible, however, that cortical changes (e.g., Flor et al. 1997; Lloyd et al. 2008; Tsao et al. 2011) or changes to other sensory systems may drive the movement control deficits observed in CLBP groups.

In interpreting the results of this study, there are some limitations to consider. First, based on the MMTS threshold data, it is possible that the EMLA topical anesthetic effects are transient (e.g., the desensitization begins to wear off by the POST-SENS2 assessment). However, the current MMTS data demonstrate that even if the desensitization brought on by the EMLA anesthetic was reduced at the POST-SENS2 assessment, its effects are still present (2038 % increase in MMTS threshold at POST-SENS2 relative to PRE-SENS). Second, due to the maximum dose suggested by the EMLA manufacturers ($\leq$400 cm^2), it was only possible to anesthetize a relatively small area of the low back (Table 1). The consistent dose of 0.1 g/cm^2 was chosen to adhere to the maximum dose while also providing a sufficient depth of cream as to completely cover the test area; however, it is possible that a dose over a larger, or different, area may have resulted in greater influence on the low back proprioception, dynamic movement and LDS variables. Third, it is apparent within our seated balance data that both participants groups improved at the task between our PRE and POST time points (Table 3). However, as this was true for both our EMLA and PLACEBO groups, it does not impact our ability to address the influence of decreased cutaneous sensitivity on this task. Finally, all of the analyzed proprioceptive and stability tasks were conducted under active muscular control. These tasks were chosen as: (1) they are functional in nature and (2) best represent movements in which motor control deficiencies have been detected in CLBP populations. It is possible that if passive movements were explored (resulting in reduced muscular sensory feedback), larger effects of cutaneous desensitization would have been observed. However, we were most interested in an ecologically relevant, real-world scenario in which any potential use of cutaneous sensory information would occur in addition to readily available information from other remaining senses.

Conclusions

In conclusion, the current work demonstrates that over-the-counter, topical lidocaine–prilocaine-based anesthetic (1) can effectively decrease the cutaneous sensitivity of low back region and (2) has a minimal effect on low back proprioceptive and movement capability during active tasks. The decreases in low back cutaneous sensitivity observed here mirror those reported in CLBP patients. When linking these findings to the body of research examining patients

with recurrent CLBP, it appears that peripheral cutaneous insensitivities alone cannot explain declines in lumbar spine motor control. It is possible that higher level cortical changes drive any motor declines associated with tactile insensitivity and that benefits from tactile sensory retraining modalities necessitate more than simple changes in peripheral sensitivity alone.

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Compliance with ethical standards

Conflict of interest The authors declare that there are no conflicts of interest.

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