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Does insertion of intramuscular electromyographic electrodes alter motor behavior

during locomotion?

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testing, fear avoidance

Abstract

Intramuscular electromyography (EMG) is commonly used to quantify activity in the trunk musculature. However, it is unclear if the discomfort or fear of pain associated with insertion of intramuscular EMG electrodes results in altered motor behavior. This study examined whether intramuscular EMG affects locomotor speed and trunk motion, and examined the anticipated and actual pain associated with electrode insertion in healthy individuals and individuals with a history of low back pain (LBP). Before and after insertion of intramuscular electrodes into the lumbar and thoracic paraspinals, participants performed multiple repetitions of a walking turn at self-selected and controlled average speed. Low levels of anticipated and actual pain were reported in both groups. Self-selected locomotor speed was significantly increased following insertion of the electrodes. At the controlled speed, the amplitude of sagittal plane lumbo-pelvic motion decreased significantly post-insertion, but the extent of this change was the same in both groups. Lumbo-pelvic motion in the frontal and axial planes and thoraco-lumbar motion in all planes were not affected by the insertions. This study demonstrates that intramuscular EMG is an appropriate methodology to selectively quantify the activation patterns of the individual muscles in the paraspinal group, both in healthy individuals and individuals with a history of LBP.

Introduction

Intramuscular or fine-wire electromyography (EMG) is commonly used to quantify the activity of the trunk musculature during static or dynamic motor tasks. In particular, intramuscular EMG methodology is often employed in research investigating

alterations in postural control of the trunk in individuals with low back pain (LBP)
[MacDonald et al. 2009; Tsao et al. 2011; Hall et al. 2009]. Intramuscular EMG electrodes enable the measurement of activity in the deep muscles of the trunk that are not accessible to surface EMG electrodes. These include the internal oblique, transversus abdominis and the deep fibers of the lumbar multifidus [Beneck et al. 2013; MacDonald et al. 2009]. In the paraspinal muscle group, the use of intramuscular EMG also minimizes potentially confounding cross-talk from adjacent musculature that may have a

different functional role [Lee et al. 2009].

However, a potential disadvantage of intramuscular EMG is that the pain associated with the insertion of the electrodes may alter motor behavior [MacDonald et
al. 2009]. For example, Young et al., [Young et al. 2004] demonstrated that in children with cerebral palsy, self-selected locomotor speed, cadence, and step length significantly decreased following insertion of intramuscular electrodes into the lower extremities. Similarly, Jacobson et al., [Jacobson & Gabel 1995] reported that after intramuscular electrode insertions into the vastus medialis and biceps femoris, two of their healthy adult
subjects had an antalgic gait pattern during walking and running and two others required a break in testing due to anxiety. Despite the large number of studies utilizing this methodology, to date it has not been established whether inserting intramuscular EMG electrodes into the paraspinal muscles alters trunk control or locomotor kinematics.

It is clear however that in healthy individuals, experimentally induced pain in the 25 paraspinals alters postural control of the trunk during standing and walking [Moseley et al. 2004; Lamoth et al. 2004; Arendt-Nielsen et al., 1995; Moe-Nilssen et al. 1999]. These changes in postural control during experimental pain are on the whole suggestive of a "guarding' or splinting strategy to reduce motion in the painful area [Moe-Nilssen et al. 1999; Lamoth et al. 2004]. Trunk control is also affected by the anticipation of pain in the low back, even in the absence of actual pain itself [Moseley et al. 2004]. However, as 30 studies that utilize intramuscular EMG in the trunk do not routinely quantify the level of pain associated with this methodology, it is unclear whether discomfort following insertion is of sufficient intensity or duration to elicit changes in motion in the trunk during motor activities after the electrode insertions. Individuals with a history of LBP 35 may have a more pronounced response to the insertion of intramuscular electrodes than healthy individuals due to elevated fear avoidance behaviors or lowered pain thresholds [Imamura et al. 2013; Wand et al. 2011]. Therefore, it is also important to determine if the magnitude of any change in motion in response to electrode insertion is the same in healthy individuals and individuals with a history of LBP.

40 Turning during walking is a common locomotor perturbation. Walking turns can be performed in the direction either ipsilateral to or contralateral to the stance limb. In comparison with steady-state locomotion, ipsilateral walking turns are associated with greater postural demand [Taylor et al., 2005] and increased paraspinal muscle activation [Armour Smith & Kulig, unpublished data]. As a result, analysis of walking turns may provide greater insight into changes in locomotor kinematics in response to intramuscular EMG insertion than steady-state locomotion. Therefore, the primary purpose of this study

was to investigate if insertion of intramuscular EMG electrodes into the paraspinal musculature in healthy individuals and individuals with a history of recurrent low back pain resulted in reduced locomotor speed and reduced amplitude of trunk motion during ipsilateral walking turns. We hypothesized that there would be no difference in locomotor kinematics following electrode insertion. The secondary purpose of this study was to

quantify the anticipated and actual amount of pain associated with insertion of intramuscular electrodes into the paraspinal muscles.

Methods

50

55 **Participants**

Twenty-nine young adults between the ages of 22 and 31 years participated in the study (17 women, 12 men). Participants were recruited via word of mouth and study flyers. Control participants (CTRL) were individually matched to participants with recurrent LBP (RLBP) by age (± five years), height in m (± 10 %) weight in kg (± 10 %)
and activity level in metabolic equivalents (METS, ± 15 %; Table 1). Physical activity level was quantified using the Physical Activity Scale [Aadahl & Jorgensen 2003]. One participant with a history of recurrent LBP did not complete the data collection due to a transient episode of vasovagal syncope in response to the intramuscular EMG insertion. Therefore only the remaining fourteen participants with a history of recurrent LBP were
matched to control participants. The Institutional Review Board of the University of Southern California approved the procedures in the study. Participants gave written informed consent after a full explanation of the study procedures and the potential benefits and risks of participating.

Participants were included in the RLBP group if they were between 18 and 40 70 years of age, had a history of more than one year of recurrent episodes of primarily unilateral LBP, reported at least two functionally limiting pain episodes of at least 24 hours' duration in the preceding year [Stanton et al. 2009], and were in symptom remission at the time of the data collection (defined as a score of less than 0.5/10 cm on a visual analogue scale (VAS) for current pain at the start of the data collection). Participants were eligible for inclusion in the control group if they could be individually 75 matched to a participant in the RLBP group as previously described and did not have any history of LBP requiring modification of activity or medical care. Participants in both groups were excluded if they had a history of diabetes mellitus, rheumatic joint disease, any blood-clotting disorder or current anti-coagulant therapy, polyneuropathy, history of 80 low back surgery, history of bilateral leg pain, spinal stenosis or scoliosis, spinal malignancy or infection, lumbar radiculopathy, current or previous musculoskeletal injury or surgery affecting locomotion, or were currently pregnant.

Assessment of symptoms

In the RLBP group, fear avoidance beliefs were quantified using the physical activity sub-scale of the Fear Avoidance Beliefs Questionnaire (FABQ) [George et al. 2010]. All participants completed a baseline VAS for current pain, anchored at 0 with "no pain" and at 10 with "worst possible pain" [Carlsson, 1983]. At baseline, participants also completed a VAS for the amount of pain they anticipated feeling during the electrode insertions and the amount of pain that they anticipated feeling during the locomotor trials 90 following the insertions [Al-Obaidi et al. 2003]. Immediately after the electrode insertions they completed a further VAS for the actual amount of pain they felt during the insertions, and at the end of the data collection they completed a VAS for the actual amount of pain they felt during the locomotor trials that followed the insertions.

Experimental task

95 Each locomotor trial consisted of three laps of a walking circuit. The circuit required both straight locomotion and a series of walking turns (Figure 1). Participants performed the circuit both at a relaxed, self-selected speed (SELF) and at a controlled average speed of 1.5 m/s \pm 5 % (FAST). Average speed was measured from the time taken to complete the standardized length of the circuit and was measured using 100 photo-electric triggers. Participants executed an ipsilateral pivot turn in the same location in each repetition of the circuit. They stepped into an outlined 70 cm by 70 cm area with the foot ipsilateral to the turn direction and turned briskly 90° to the ipsilateral side (Figure 1a). The strategy used to perform the other walking turns in the circuit was not specified. Each participant practiced the circuit until they were consistently able to 105 achieve the correct foot placement for the turn without looking down or breaking stride. At least seven successful trials of the circuit at each speed were collected for each participant, resulting in a total of at least 21 ipsilateral pivot turns in the defined turning area for analysis for each condition (Figure 1b). All participants walked the circuit in the direction contralateral to the side of their EMG instrumentation.

110 Instrumentation

Participants were first instrumented with motion-capture markers. Retro-reflective markers were attached to anatomical landmarks to define body segments and joint axes. Rigid kinematic models of the pelvis and the lumbar and thoracic regions of the spine were defined using individual markers bilaterally on the anterior superior

- 115 iliac spines, iliac crests, greater trochanters and on the L5/S1 disc space (pelvis), a rigid triad of markers affixed over the spinous process of L1 (lumbar spine) and a rigid triad of markers over the spinous process of T3 (thoracic spine) [Popovich & Kulig 2012]. Wireless force-sensitive resistor foot switches were also attached bilaterally to the sole of participants' shoes under the lateral heel and the first metatarsophalangeal
- 120 joint (TeleMyo DTS Telemetry, Noraxon USA Inc, Scottsdale, USA). Kinematic data were collected using an 11-camera digital motion capture system sampling at a frequency of 200 Hz (Qualisys AB, Gothenburg, Sweden). After instrumentation with the motion capture markers and footswitches, participants performed the first set of walking trials at both self-selected (pre-insertion SELF) and controlled speed (pre-insertion FAST).
- We then performed the fine-wire EMG insertions, leaving the motion capture markers in situ. Fine-wire intramuscular electrodes were inserted into the deep fibers of the lumbar multifidus at the level of L4, the longissimus thoracis pars lumborum at the level of L4, and the longissimus thoracis pars thoracis at the level of T10 using a previously described protocol with real-time ultrasound imaging guidance [Beneck et al. 2013] (8 MHz linear transducer, SONOLINE Antares[™], Siemens Medical Solutions Inc, USA; nickel chromium alloy wires, 50 um gauge, polyurethane/nylon coating, tips bent back 5 and 3 mm with 2 mm wire exposed, 25 gauge hypodermic needles). Electrodes were inserted into the predominant side of pain reported by participants with a history of RLBP and the same side for their matched control. The needle was immediately removed
- 135 following the electrode insertion. Correct electrode placement was confirmed observing the contraction induced by light electrical stimulation using ultrasound imaging.

The electrodes were connected to wireless differential preamplifiers (TeleMyo DTS Telemetry, Noraxon USA Inc, Scottsdale, USA: baseline noise < 1 uV RMS, Input impedance > 100 Mohm, CMR > 100 dB, Input range +/- 3.5 mV, Base gain 400). The

EMG and foot switch data were transmitted via a wireless receiver (TeleMyo DTS Telemetry, Noraxon USA Inc, Scottsdale, USA), digitally sampled at 3000 Hz at 16 bit resolution and synchronized with the kinematic data using photoelectric triggers (Qualisys Track Manager v2.6, Qualisys AB, Gothenburg, Sweden). Immediately after the fine-wire EMG insertions, participants walked freely around the laboratory to allow any residual soreness or anxiety to dissipate. They then performed a second set of walking circuit trials at both self-selected and controlled speed (post-insertion SELF and

FAST respectively).

Data processing

Between 15 and 21 turning trials were analyzed for each participant for each 150 condition. Kinematic data were first processed using Visual3DTM software (C-Motion Inc., MD, USA) before being exported to MATLAB[®] (MathWorks, MA, USA) for further analysis. Marker trajectories were low-pass filtered with a 10 Hz recursive fourth order Butterworth filter [Angeloni et al., 1994]. The stride cycle of each ipsilateral pivot turn (Figure 1b) was determined using the voltage signals of the foot switches 155 and confirmed with a visual check of the horizontal velocity of a motion capture marker positioned on the posterior heel. Local coordinate systems for each segment, relative to the global laboratory coordinate system, were determined from a standing calibration trial. Segment and joint kinematics were then calculated across the turn stride cycle using Cardan angles and a rotation order of XYZ

- 160 (flexion/extension; abduction/adduction; axial rotation; Schache et al. 2002). The alignment of the lumbar and thoracic segments was normalized to the static standing trial to account for individual postural alignment [Popovich & Kulig 2012]. Average peak-topeak amplitude of angular lumbo-pelvic and thoraco-lumbar motion in all planes during the walking turns at the controlled speed (FAST) was calculated for each participant.
- 165 Average self-selected walking speed during the walking circuit pre- and post-insertion was calculated for each participant from the time taken to complete each locomotor trial. Average duration of the turn stride cycle at the controlled speed was also determined in order to compare the speed that the turn was executed pre- and post-insertion. The ensemble average for the RLBP and CTRL groups for all variables was then calculated.

170 Statistical analysis

The dependent variables that were compared pre and post-intramuscular EMG electrode insertion were i) average self-selected walking speed (pre- and post-insertion SELF); ii) duration of the turn stride cycle at the controlled speed (pre- and post-insertion FAST); iii) amplitude of peak-to-peak lumbo-pelvic and thoraco-lumbar motion in all planes at controlled walking speed (pre- and post-insertion FAST). The extent of change in each variable from pre- to post-insertion was compared between the RLBP and CTRL groups using paired t-tests (parametric data) and Wilcoxon signed ranks tests (non-parametric data). To correct for multiple comparisons a Bonferroni correction was used resulting in a level of significance set at $\alpha = 0.0063$. Additionally, anticipated and actual pain during the insertions and during the post-insertion walking trials was compared using Wilcoxon signed rank tests.

Inferential confidence intervals were used to determine whether the variables

were equivalent in the pre- and post-insertion conditions, or if there was a significant change in the variables from the pre- to post-insertion [Tryon 2001; Stegner et al. 1996].
This approach was used because equivalence between two conditions is not proven simply by a failure to reject the null hypothesis using a standard hypothesis-testing approach. Testing equivalence using an inferential confidence interval approach avoids the problem of the likelihood of being able to demonstrate equivalence actually becoming smaller as the study variance decreases [Stegner et al., 1996], and also defines *a-priori* an amount of change in the variable that is considered to be meaningful [Tryon 2001]. If the

- difference between the lower limit of the 95% confidence interval for the smaller of the pre- and post-insertion means and the upper limit of the 95% confidence interval for the larger of the two means falls within the pre-defined range of equivalence for that variable, no statistically significant or meaningful change in the variable has occurred
- [Tryon 2001]. Conversely, if there is no overlap between the inferential 95% confidence intervals for the two means, there is a statistically significant difference in the variable from pre- to post-insertion at the p = 0.05 level. For this study, the range of equivalence was defined as ± the minimal detectable change (MDC) for each of the variables. MDC for self-selected walking speed was determined by having five healthy individuals perform two blocks of trials of the locomotor circuit at a self-selected speed, separated by a period of relaxation of approximately 15 minutes. MDC for the kinematic variables was determined by having four healthy individuals perform two blocks of trials of the controlled speed. The two blocks of trials were separated by a period of approximately 15 minutes during which they performed a different sub205 maximal motor task (straight walking). Intra-class correlation coefficients (ICC_{3,1}) and

the standard error of the measure (SEM) were calculated for each variable. MDC was then determined using the equation MDC = $1.96 \times \sqrt{2} \times SEM$ [King 2011]. All statistical analyses were performed using PASW Statistics (Version 18, IBM Corp., Armonk, NY).

Results

Average duration of symptoms in the RLBP group was 5.8 ± 4.2 years. At baseline, average current pain was 0.12 ± 0.24 cm in the participants with a history of RLBP and 0.0 cm in the healthy controls. Median \pm inter-quartile range FABQ physical activity score in the RLBP group was 12.50 ± 6.7 .

Anticipated and actual pain associated with EMG electrode insertion

The anticipated and actual pain VAS scores for EMG insertion and locomotor trials are shown in Table 2. There was no significant difference between groups for any of the VAS scores (Table 2).

Effects of intramuscular EMG electrode insertion

Due to problems with marker occlusion during the pre-insertion trials for one 220 participant in the RLBP group, the data from this participant and the matched control participant were not included in data analysis, leaving a sample size of 26. There were no significant differences between the RLBP and CTRL groups in the extent of change in any of the variables in response to electrode insertion (SELF walking speed group comparison p = .369; FAST stride duration p = .260; FAST lumbo-pelvic motion sagittal 225 plane p = .643, frontal plane p = .854, axial plane p = .276; FAST thoraco-lumbar motion sagittal plane p = .807, frontal plane p = .279, axial plane p = .237). Therefore the inferential confidence intervals were calculated using the pooled data from both groups. The controlled speed was faster than the self-selected speed in all individuals except one CTRL participant. Self-selected walking speed significantly increased after the insertion of intramuscular EMG electrodes (mean \pm standard deviation, pre-insertion SELF 1.15 \pm 0.14 m/s, post-insertion SELF 1.22 \pm 0.12 m/s, Figure 3a). At the controlled speed, there was no difference in the duration of the stride cycle between pre- and postinsertion (pre-insertion FAST 1.03 \pm 0.05 s, post-insertion FAST 1.02 \pm 0.06 s, Figure

3b).

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In the sagittal plane, the peak-to-peak amplitude of lumbo-pelvic motion across the turn stride cycle decreased significantly from pre- to post-insertion (pre-insertion FAST 9.56 ± 2.30°, post-insertion FAST 8.45 ± 2.19°, Figure 4a). This decrease in the amplitude of sagittal lumbo-pelvic motion occurred in the majority of participants in both groups (CTRL n = 10, RLBP n = 10) and was due to reduced peak flexion in 7 participants, reduced peak extension in 7 participants, and a reduction in both peak flexion and extension in 6 participants. There was no change in lumbo-pelvic frontal motion (pre-insertion FAST 5.77 ± 1.58°, post-insertion FAST 5.37 ± 1.32°) or lumbo-pelvic axial motion (pre-insertion FAST 8.24 ± 1.75°, post-insertion FAST 8.85 ± 1.90°, Figure 4a). Thoraco-lumbar motion in all planes was equivalent pre and post-insertion 245 (sagittal plane pre-insertion FAST 5.36 ± 1.04°, post-insertion FAST 4.86 ± 1.26°; frontal plane pre-insertion FAST 9.19 ± 2.40°, post-insertion FAST 9.75 ± 3.33°; axial plane

Discussion

This study is the first to directly investigate if insertion of intramuscular EMG electrodes into the paraspinal muscles results in reductions in walking speed or amplitude

pre-insertion FAST $17.62 \pm 5.03^{\circ}$, post-insertion FAST $18.31 \pm 4.94^{\circ}$, Figure 4b).

of trunk motion. Unexpectedly, self-selected locomotor speed increased following the insertions. There was a significant decrease in the amplitude of lumbo-pelvic motion in the sagittal plane in response to electrode insertions. However, the amplitude of lumbo-pelvic motion in frontal and axial planes, and thoraco-lumbar motion in all planes, was not affected by intramuscular EMG. The levels of anticipated and actual pain associated with electrode insertion were low in both groups.

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Inferential confidence interval testing demonstrated that the amplitude of sagittal plane motion in the lumbar spine when walking at the controlled speed was significantly smaller following insertion of the EMG electrodes. Although the absolute value of the 260 decrease in this motion was small (an average reduction of peak-to-peak amplitude of 1.11°) it exceeded the minimal detectable change calculated for this task under these experimental conditions. There were different sources of reduced peak-to-peak amplitude in individual participants, with some demonstrating reduced peak flexion, others demonstrating reduced peak extension, and some demonstrating a reduction in both peak 265 flexion and extension. As the duration of the turn at the controlled speed was not affected by electrode insertion, it is likely that the reduction in sagittal plane motion was due to subject-specific changes in paraspinal agonist and antagonist activity. The fact that changes in locomotor kinematics were evident in sagittal lumbo-pelvic motion but not in thoraco-lumbar motion may be due to the greater number of insertions performed in the 270 lumbar region.

Importantly, this study demonstrated that although there were small changes in sagittal lumbar motion in response to electrode insertion, these changes were the same in individuals with a history of recurrent LBP as in healthy individuals. In addition, the

individuals in this study with a history of recurrent LBP did not demonstrate elevated

- anticipation of pain or actual pain in comparison with healthy individuals. This is despite the fact that persistent LBP is associated with altered cortical processing of painful and non-painful sensory stimuli [Wand et al. 2011], central and peripheral sensitization, and reduced pain pressure threshold, particularly in the area of symptoms [Imamura et al. 2013]. The lack of group difference in anticipated and actual pain intensity in the present study may be due to the fact that the individuals with a history of LBP were
- asymptomatic at the time of the data collection and had relatively low levels of fear avoidance [Calley et al. 2010].

Self-selected locomotor speed increased following insertion of the intramuscular electrodes. Two previous studies investigating the effect of electrode insertions in the 285 lower limbs on locomotor speed have demonstrated both significantly decreased and unaltered self-selected speed following insertion [Young et al. 2004; Krzak et al. 2013]. However, since both of these studies investigated children with cerebral palsy they are likely not representative of the response in adults without neurological disorders. As LBP is normally associated with reduced rather than increased self-selected locomotor speed 290 [Selles et al. 2001; Lamoth et al. 2006] it is probable that the increase in average selfselected speed evident in this study was a result of an order effect rather than actual or anticipated discomfort in the insertion area. Prior to the second set of self-selected speed trials, the participants had experienced performing the walking circuit at a speed that was faster than their comfortable walking pace. This may have resulted in an after-effect 295 during the post-insertion self-selected speed trials.

Numerous studies have demonstrated changes in motor behavior in response to acute experimental pain [Farina et al. 2004; Graven-Nielsen & Arendt-Nielsen 2008; Hodges et al. 2003; Arendt-Nielsen et al. 1995]. In this present study however, participants reported levels of pain both during the insertion of the EMG electrodes and during the locomotor trials after the insertions that were less than the pain typically induced during experimental pain protocols [Moseley et al. 2004; Ervilha et al. 2005 Farina et al. 2004; Arendt-Nielsen et al. 1995]. The median VAS value during locomotor trials in this study was also less than the pain reported by participants during fast and slow walking following insertion of intramuscular electrodes into the gluteal muscles [Semciw et al. 2013]. The lower levels of pain experienced in this present study during walking may be a result of the slightly lower level of activity in the paraspinal muscles during walking in comparison with the gluteals [Saunders et al. 2005; Callaghan et al. 1999; Perry & Burnfield 2010].

Fear of pain and anxiety about future pain may also result in altered movement strategies and limitation in motor activities [Pincus et al. 2006]. Fear of pain has been demonstrated to be more correlated with guarded movement patterns than actual pain intensity [Vlaeyen & Linton 2000] and anticipation of pain is highly associated with deficits in locomotor speed in individuals with back pain [Al-Obaidi et al. 2003]. However, participants in this study demonstrated low levels of anticipated pain for both the insertion of the electrodes and the locomotor trials following insertion of electrodes. The VAS scores for anticipated pain were lower than those previously demonstrated during induction of experimental low back pain in healthy subjects [Moseley et al. 2004] or during fast walking in individuals with LBP [Al-Obaidi et al. 2003]. Individuals who

were eligible to take part in this study were given a full explanation of the intramuscular

320 EMG procedures prior to volunteering to participate. Therefore, it is likely that individuals with elevated fear of needles or intramuscular EMG methodology, who may have experienced greater anticipated pain, chose not to participate. Future studies utilizing intramuscular EMG may benefit from utilizing similar measures of anticipated and actual pain in order to monitor if their participants may be at 325 greater risk of demonstrated fear-related changes in movement behavior.

It is important to note that this study investigated the effect of intramuscular EMG during a sub-maximal task. During both steady-state locomotion and walking turns the function of the paraspinal musculature is postural rather than propulsive, and paraspinal activity remains below 20 % of the amplitude of maximum voluntary contraction in these 330 muscles [Saunders et al. 2005]. Therefore, it is possible that a larger effect might be evident during a motor activity that requires a greater level of paraspinal activity or a greater range of motion, particularly in the sagittal plane. It should also be noted that in this study, three electrode insertions were performed on each participant. This number of insertions is consistent with previous studies utilizing intramuscular EMG methodology 335 to investigate trunk control [MacDonald et al. 2009; Beneck et al. 2013]. However, more significant effects may also be evident in response to a larger number of electrode insertions. In three participants, one of the electrode insertions was repeated due to the electrode wires being dislodged during removal of the inserting needle (n = 2) and poor electrode placement (n = 1). Electrode re-insertion did not have a systematic effect on actual pain experienced during the insertions and locomotor trials, or on locomotor 340

kinematics in these three individuals.

The findings from this study indicate that other than a small but systematic change in sagittal plane lumbo-pelvic motion, trunk motion is not affected by insertion of intramuscular EMG electrodes Therefore this is an appropriate method for investigating trunk postural control in both individuals with a history of LBP and healthy controls. Future research is needed to clarify if insertion of intramuscular EMG electrodes results in more limited sagittal plane motion in motor tasks that require greater amplitude of motion or higher levels of paraspinal activity than the walking turn in the present study.

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Table 1

Participant demographics (median ± *inter-quartile range)*

	CTRL ^a	RLBP ^a	р
Age (years)	24.5 ± 1.75	26.5 ± 4.75	.068
Height (m)	1.73 ± 0.05	1.73 ± 0.09	.664
Mass (kg)	66.68 ± 14.97	67.70 ± 23.42	.152
PAS score (MET-time)	47.60 ± 5.00	48.20 ± 7.55	.470

 $a_{n} = 14$

Table 2

Visual analogue scores for anticipated and actual pain associated with intramuscular EMG electrode insertion (Median \pm inter-quartile range, n = 28)

	CTRL	RLBP	р
Anticipated pain			
during insertions*	1.35 ± 2.43	1.90 ± 1.63	.730
during locomotor trials*	0.60 ± 0.78	0.50 ± 0.35	.937
Actual pain			
during insertions*	2.45 ± 2.65	1.90 ± 2.38	.753
during locomotor trials*	0.45 ± 0.70	0.50 ± 0.70	.779

Figure 1. a) Schematic of the walking circuit, with the turning area for the ipsilateral pivot turn indicated. Circuit set up for participant instrumented on the left side and therefore turning towards the right. b) Stride cycle of an ipsilateral pivot turn to the right.

Figure 2. Axial ultrasound image (left) and schematic (right) demonstrating insertion of intramuscular EMG electrode into the deep fibers of the lumbar multifidus muscle (SP = spinous process, H = hypodermic needle).

Figure 3. Average a) self-selected locomotor speed, and b) stride duration at controlled speed pre- and post-insertion. Pooled groups, n = 26, error bars = 95% confidence interval, shaded areas = range of equivalence (± minimal detectable change, MDC).

Figure 4. Peak-to-peak amplitude of a) lumbo-pelvic motion, and b) thoraco-lumbar motion at the controlled speed pre- and post-insertion. Pooled groups, n = 26, error bars = 95% confidence interval, shaded areas = range of equivalence (± minimal detectable change, MDC).

Figure 1 a

b







а



b



Lines 111-113: A figure or precise description of the markers on the upper arm, forearm, hand, thigh, shank, and foot are required (with citation(s) is needed). Why describe procedures for kinematics data collection from these markers in the methods when the results from these are not reported (with the exception of the foot marker)?

A full body marker set was utilized as part of a larger study and for completeness this was reflected in the text. However, as only the trunk and pelvis markers were utilized in this study the authors agree that this is confusing and therefore the reference to the other markers comprising the full body marker set has been removed. The heel marker that was only used to double-check the locomotor events is described in the data processing section (line 156).

Lines 177-180: If a correction for multiple t-test comparisons was used, which method was applied? Otherwise why was a correction not applied?

A Bonferroni correction has now been made to the t-test comparisons between groups. This is detailed in the statistical analysis section, line 177. The correction does not alter the results, as they were already all non-significant and demonstrate no difference in the extent of change in any of the variables between groups.

Results

Which values are reported in the results - those for the t-tests or for the Wilcoxon signed ranks tests?

The variables which did not follow a normal distribution were the change in thoraco-lumbar sagittal plane and frontal plane data. Therefore Wilcoxon signed ranks tests were used for those group comparisons. T-tests were used for all of the other group comparisons.

Line 155-156: please include a citation for using a 10 Hz recursive fourth order Butterworth filter for these data.

Optimal cut-off frequency for the filter was determined by examination of the frequency content of the data and was in agreement with the findings from Angeloni et al., (1994). This citation has been added to the text.

Yours sincerely

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