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REORGANISED MOTOR CONTROL STRATEGIES OF TRUNK MUSCLES DUE TO ACUTE LOW BACK PAIN

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ABSTRACT

This study assessed how the low back motor control strategies were affected by experimental pain. In twelve volunteers the right m. longissimus was injected by hypertonic and isotonic (control) saline. The pain intensity was assessed on a visual analogue scale (VAS). Subjects were seated on a custom-designed chair including a 3-dimensional force sensor adjusted to the segmental height of T1. Electromyography (EMG) was recorded bilaterally from longissimus, multifidus, rectus abdominis, and external oblique muscles. Isometric trunk extensions were performed before, during, and after the saline injections at 5%, 10%, and 20% of maximum voluntary contraction force. Visual feedback of the extension force was provided whereas the tangential force components were recorded. Compared with isotonic saline, VAS scores were higher following hypertonic saline injections ($P<0.01$). Experimental low back pain reduced the EMG activity bilaterally of the rectus abdominis muscles during contractions at 10 and 20% MVC ($P<0.01$) although force accuracy and tangential force variability was not affected. Increased variability in the tangential force composition was found during pain compared with the non-painful condition ($P<0.05$). The immediate adaptation to pain was sufficient to maintain the quality of the task performance; however the long-term consequence of such adaptation is unknown and may overload other structures.

Key words: experimental muscle pain, EMG, isometric force, three-dimensional force variability

1. Introduction

Most people have had an episode of low back pain (LBP) (Hoy, Brooks, Blyth, & Buchbinder, 2010). The prevalence of LBP have been reported from 6 to 19% (Biering-Sorensen, 1982; Cassidy, Cote, Carroll, & Kristman, 2005; Hestbaek, Leboeuf-Yde, Engberg, et al., 2003) for a first episode and recurrence reach up to 50% over a 5 years period (Hestbaek, Leboeuf-Yde, & Manniche, 2003). Besides the individual suffering this constitutes an enormous socio-economic burden (Andersson, 1999; Filho, Simmonds, Protas, & Jones, 2002; Hoy et al., 2010; Steenstra, Verbeek, Heymans, & Bongers, 2005). Patients suffering from LBP often present a multi-factorial pathogeneses (McCowin, Borenstein, & Wiesel, 1991), with symptoms including referred limb pain (Mellin & Hurri, 1990), often associated with numbness and radiation to the leg (Wolff et al., 2006), as well as muscle weakness (Helewa, Goldsmith, & Smythe, 1993) and biomechanical changes, such as increased spinal stiffness (P. Hodges, van den Hoorn, Dawson, & Cholewicki, 2009), and spinal deformations (Schroeder, Schaar, & Mattes, 2013). Insufficient spine stabilization has been associated with LBP (P. W. Hodges & Richardson, 1996; MacDonald, Moseley, & Hodges, 2009) and may be important for understanding mechanisms involved in LBP.

Experimental pain models, have been widely used to study motor adaptations caused by deep-tissue pain per se, excluding the multiple confounding factors observed in chronic pain patients (Bank, Peper, Marinus, Beek, & van Hilten, 2013). In particular, injection of hypertonic saline in the erector spinae muscle has been shown to mimic the pain sensation perceived in LPB patients and produce motor adaptations comparable to what has been observed in patients (Arendt-Nielsen, Graven-Nielsen, Svarrer, & Svensson, 1996; P. W. Hodges, Moseley, Gabrielsson, & Gandevia, 2003; Smith, Coppieters, & Hodges, 2005; Tsao, Tucker, Coppieters, & Hodges, 2010). In another example, experimental low back pain in healthy subjects delayed the onset of EMG activity of deep abdominal muscles in postural adjustments to fast arm movements (P. W. Hodges

et al., 2003), similar to results found in LBP patients (P. W. Hodges, 2001; P. W. Hodges & Richardson, 1998; Tsao, Galea, & Hodges, 2008; Tsao & Hodges, 2008). Although these adaptations are believed to compromise spinal stability during rapid movements (P. W. Hodges et al., 2003) it is still an open question if they contribute to deficits in trunk stability during sustained submaximal tasks. It is important to note that due to the complex biomechanics of the lumbar spine (Pope, 1989), the anatomical configuration of abdominal and trunk muscles (Harrison, Harrison, & Troyanovich, 1997; van Dieen, 1997), and the high level of muscular redundancy controlling the trunk (Cholewicki & VanVliet, 2002), trunk stability is achieve by a multidirectional control of the lumbar spine. This allows reorganization of the activity from different muscular groups to avoid or reduce the pain sensation while maintaining the task performance, which also causes increase in movement/force variability compared with pain free conditions (Hirata, Arendt-Nielsen, Shiozawa, & Graven-Nielsen, 2012; Salomoni, Ejaz, Laursen, & Graven-Nielsen, 2013; Tucker & Hodges, 2010). When specifically investigating the effects of pain in multidirectional force fluctuations during submaximal tasks, Salomoni *et al.* (2013; 2012a) found increased variability in tangential force components during painful compared with non-painful tasks. This findings support the theory that adaptations to pain might include redistribution of activity within and between muscles (P. W. Hodges & Tucker, 2011) which controls movement in different directions. Therefore, evaluating the effects of pain on multidirectional force fluctuations controlling the trunk can provide deeper understanding on the relationship between insufficient muscular stabilization of the trunk and low back pain (P. W. Hodges & Richardson, 1996; MacDonald et al., 2009).

The aim of the present study was to investigate the effects of pain during different levels of isometric trunk extensions on multidirectional force variability and muscle activation. It was hypothesized that (i) unilateral low back muscle pain will cause a reorganization of muscle

activation during trunk extension, and (ii) the reorganized muscle activity will cause an increase in force variability and alter the tangential forces intensity.

2. Methods

2.1. Subjects

Twelve young volunteers (7 males; age 25 ± 4 yrs.; height 172 ± 11 cm; weight 70 ± 13 kg; mean ± SD) with no known musculoskeletal disorder participated in this study. All participants received detailed written and verbal information and signed an informed consent before inclusion. Four subjects did not complete the entire protocol and were therefore excluded from the data analysis. The study was conducted in accordance with the Declaration of Helsinki and approved by the local Ethics Committee (N-20090036).

2.2. Experimental low-back pain

Acute low-back pain was induced by an intramuscular injection of sterile hypertonic saline (1.0 ml, 5.8%) into the right m. longissimus and injection of isotonic saline (1.0 ml, 0.9%) was used as control. The needle (25G × 38mm) was inserted perpendicular to the surface about 40 mm lateral to spinal process L2 with a depth of 15 to 20 mm. The pain intensity was assessed on a 10-cm electronic visual analogue scale (VAS), where 0 cm indicated “no pain” and 10 cm was anchored to “maximal pain”. Immediately after the injection, the VAS signal was recorded continuously for 10 minutes (sampling frequency of 0.5 Hz) and subjects were asked to update the VAS scores between trials by adjusting an external handheld slider. Additionally, subjects were asked to indicate the pain distribution by filling out a body chart. Five subjects had the hypertonic saline as the first injection and 7 had the isotonic saline as first injection.

2.3. Protocol

A randomized, single-blinded, controlled, crossover design was used to assess the effects of experimental low-back pain on the variability of force. Subjects performed three trunk extension

and three trunk flexion isometric maximal voluntary contractions (MVCs, 5 s) where each was followed by at least 1.5 minutes of rest. The EMG results for the trunk flexion MVC are not shown, and were only used to normalize the subsequent muscle activity recordings. The MVC force in the task-related (anterior-posterior) direction was extracted for calculation of all the following submaximal target force levels. Subjects then performed a total of six series of submaximal isometric trunk extensions with at least 1.5 minutes of rest between series: Before, during, and after the effects of a painful or non-painful injection (baseline, during and post conditions). The sequence of injection type was randomized and balanced across subjects, and the post-injection condition was initiated one minute after the subject reported the last pain sensation. Immediately after the last resting period, one additional MVC trial for trunk extension was performed. The series of contractions included 5%, 10%, and 20% MVC force contractions (45 sec) performed in random order and each followed by at least 40 seconds of rest. During each contraction level, a ramp-and-hold force feedback was provided on a computer screen (41 seconds of hold phase) using a variable visual gain, resulting in a constant visual scale across all target forces. Before starting the recordings, subjects were familiarized with the setup and the protocol by performing 1 maximal and 1 submaximal for each contraction level (5,10 and 20% MVC) as practice trials.

2.4. Force recordings

Trunk extension forces were recorded using a high-sensitivity 3 dimensional force sensor (MC3A, AMTI, USA). During the recordings, the subjects were seated on a custom-designed chair which could be adjusted according to the subject's size. The pelvis was stabilized to avoid movements, while the weight of the subjects was supported mainly by the seat. The subjects did not touch the floor with their feet, and both hips and knees were partially flexed, allowing the shanks to be supported by an extra seat, securing that the feet were always above the floor. The force sensor was

attached to the setup and adjusted in height for each subject at the T1 (2012b). The arms were crossed in front of the chest. The analogue output of the force sensor was low-pass filtered at 500 Hz, amplified (MSA-6, AMTI, USA), sampled at 1 kHz, and stored after 12 bits A/D conversion.

2.5. Surface electromyography (EMG)

EMG signals were recorded bilaterally from *m. longissimus* (LO), *m. multifidus* (MUL), *m. rectus abdominis* (RA), and *m. external oblique* (EO) using pairs of disposable Ag/AgCl surface electrodes (Ambu Neuroline 720, Denmark) in bipolar configuration, placed 2 cm apart and positioned according to the standard recommendations: (i) LO muscles, the electrodes were orientated vertically and placed at 2 finger width lateral from the spinous process of L1. (ii) MUL muscles, the electrodes were placed on and aligned with a line from caudal tip posterior spina iliaca superior to the interspace between L1 and L2 interspace at the level of L5 spinous process. (iii) RA muscles, the electrodes were placed aligned with the umbilicus and oriented parallel with the muscle fibers, and over the muscle belly (identified via palpation). (iv) EO muscles, above the anterior superior iliac spine, halfway between the iliac crest and the ribs at approximately 5 degrees oblique angle in the umbilicus direction. Signals were amplified (Counterpoint MK2, Dantec, Denmark), filtered (10-500 Hz), sampled at 1 kHz, and stored after 12 bits A/D conversion.

2.6. Data analysis

All analyses were performed over the epoch of 20 s (15 s from beginning end of ramp), avoiding excessive fluctuations due to slow force development and anticipation of trial termination (Salomoni & Graven-Nielsen, 2012b).

The average VAS score between all trials was calculated for each injection paradigm. The drawings indicating the pain areas were scanned and each scan was loaded in Matlab [Mathwroks, Version: 8.1.0.604 (R2013a), USA] to extract the pain area in arbitrary units (a.u.).

The EMG signals were digitally band-pass filtered at 20 Hz - 400 Hz using a Butterworth filter of 2nd order. The force signals were filtered with a low-pass 20 Hz, 6th order Butterworth filter (Kamavuako, Farina, Yoshida, & Jensen, 2009). The maximum root-mean-square (RMS) EMG peak activation between the 3 MVCs for both trunk flexion and extension was extracted. The muscle activity for both RA and EO muscles during the submaximal tasks, were normalized by the RMS EMG peak activation for each respective muscle obtained during trunk flexion MVC. For normalizing the MUL and LO muscle activity, the RMS EMG peak activation obtained for the respective muscle during trunk extension MVC was used. After normalization by MVC, the integral (bin length equal to 1 ms) of the filtered EMG signal (iEMG) was calculated.

A bivariate histogram (1000 squares defined by a 100-by-100 equally spaced grid) was fitted to the plot of the two tangential force components (medial-lateral and cephalo-caudal directions) for each contraction (Mista, Christensen, & Graven-Nielsen, 2015) and the centroid of the histogram was calculated. The centroid position represents the preferable (most used) tangential force intensity while performing the task. During and post injection conditions, the centroid position was normalized by subtracting the values obtained during baseline conditions for each contraction level. This normalized variable is then referred to as centroid's position difference (CPD) and the origin in the CPD graphic indicates the forces levels used during baseline. Any deviation from baseline condition values will shift the centroid position away from the origin, indicating that new combinations of tangential forces were used to accomplish the task. Positive values in the x-axis (medial-lateral) indicate forces located to the left when compared with baseline values, while positive values in the y-axis (cephalo-caudal) indicated forces located above (cephalo direction)

baseline values. The absolute value (modulus) of the CPD was also extracted (ACPD) to quantify the absolute deviation from baseline condition. Contraction task accuracy for the task-related force (anterior-posterior direction) during the force matching tasks (provided as biofeedback) was estimated by the mean absolute error between the task force level and the corresponding requested target force

2.7. Statistical analysis

Baseline Conditions

Baseline EMG parameters were analyzed for each muscle by a three-way repeated measures analysis of variance (RM-ANOVA) with *injection* (prior isotonic, prior hypertonic), *side* (left and right), and *force* (5%, 10%, 20% MVC force) as within-subject factors. Baseline force variability, range, accuracy and centroid's position were analyzed by a two-way RM-ANOVA with *injection* (prior isotonic, prior hypertonic) and *force* (5%, 10%, 20% MVC) as within-subject factors.

During and Post-Injection Conditions

The EMG, force variability, range, accuracy, CPD and ACDP values during and post-injection conditions were normalized to the baseline values. The EMG parameters *during* injections and post-injection conditions, were analyzed for each muscle by a four-way RM-ANOVA with *side* (left and right), *injection* (isotonic, hypertonic), *condition* (during, post), and *force* (5%, 10%, 20% MVC force) as within-subject factors. For the force variability, range, accuracy, CPD and ACDP values during and post-injection conditions, a three-way RM-ANOVA was used with, *injection* (isotonic, hypertonic), *condition* (during, post), and *force* (5%, 10%, 20% MVC) as within-subject factors.

For the MVC force after the submaximal contractions, a 2-way RM-ANOVA with *injection* (isotonic, hypertonic), and *condition* (during, post) as main factors was used. In case of significant factors or interactions, the Newman-Keuls (NK) post-hoc test was applied incorporating correction for multiple comparisons. The VAS scores and pain areas elicited by each injection type were analyzed with the non-parametric Wilcoxon test. Statistical significance was considered for P-values lower than 0.05. All results are reported as mean ± standard error of the mean (SEM).

3. Results

3.1. Experimental low back pain

VAS scores elicited by the hypertonic injection was higher than control (isotonic saline) injection (hypertonic: 2.6 ± 0.4 cm, isotonic 0.5 ± 0.2 cm; Wilcoxon, $P < 0.01$). Both saline injections provoked pain unilaterally (right side) around the injection site, however, hypertonic saline injection induced pain in all subjects, while isotonic injection (control) provoked pain in 4 out 12 subjects (Fig. 1). Additionally, 2 subjects (16%) also indicated referral pain (pain occurring outside the injection-pain area) in the gluteous and lower leg area during the hypertonic injection. Subjects drew significantly larger areas during hypertonic saline injection compared with isotonic saline injection (hypertonic: 3180 ± 1911 a.u., isotonic: 460 ± 254 a.u.; Wilcoxon, $P < 0{,}01$).

3.2. *Force variables*

Analysis of force variables during baseline conditions did not reveal any significant main factors (injection and force) or interactions (Table 1).

The analysis of tangential forces is embedded in the centroid's position difference (CPD) parameter illustrated for all subjects immediately after hypertonic and isotonic saline injections at the three contraction levels in Figure 2. Although the CPD for the medial-lateral direction was not significantly affected by pain (Figure 3A), the respective absolute centroid's position difference (ACPD) was higher during hypertonic injection condition than isotonic injection and post injection conditions, regardless the contraction level (Figure 3B; RM-ANOVA: $F(1, 11) = 13.1$, $P < 0.01$, NK: $P < 0.05$).

The MVC force (Newton) after the submaximal contractions (anterior-posterior direction) was not significantly different between conditions [mean (± SEM, N = 12); Baseline 1: 275 ± 24;

Isotonic Injection: 262 ± 32; Post Isotonic Injection: 259 ± 29; Baseline 2: 272 ± 28; Hypertonic Injection 263 ± 27 and Post Hypertonic Injection: 259 ± 30; F (2, 22) = 0.16, $P = 0.84$].

3.3. Muscle activity during submaximal trunk extension

The analysis of baseline conditions indicated that only the factor *force* (5, 10 and 20% MVC) significantly affected muscle activation (iEMG, Table 1). The bilateral *rectus abdominis* muscle showed increased iEMG during both the 20% MVC task compared with the other force levels and 10% MVC compared with 5% MVC (RM-ANOVA: F(2,22) = 34.6, $P < 0.01$; NK: $P < 0.01$). The bilateral *external oblique* muscles showed increased iEMG during the 20% MVC force task compared with the 5% of MVC task (RM-ANOVA; F(2,22) = 13.8, $P < 0.01$; NK: $P < 0.02$).

A significant interaction between *injection*, *condition* and *force* factors showed that pain in the right *m. longissimus* decreased bilaterally the iEMG in the left *m. rectus abdominis* during the trunk extensions at 20% MVC force compared with control (isotonic saline) injection condition (Figure 4; RM-ANOVA: F(2,22) = 4.5, $P = 0.02$; NK: $P = 0.03$).

4. Discussion

The present study is the first to assess how experimental low back pain affects 3-dimensional force steadiness and trunk muscle activation in different high precision isometric force tasks. During submaximal force tasks, pain in the right m. longissimus decreased the activity of left m. rectus abdominis and increased the centroid position difference in the medial-lateral direction suggesting that acute back pain causes an adaptation in the motor control strategies ensuring that the task is still completed without compromising its quality

4.1. Effect of force level on force variability and muscle activation

Motor-output variability is inherent to every muscle contraction, probably due to variability in basic mechanisms involved in force generation such as motor-unit firing rate and recruitment order (Jones, Hamilton, & Wolpert, 2002; Stein, Gossen, & Jones, 2005). Observations of monotonic increases in force variability (less accuracy) with higher force levels during isometric contractions is often described as signal-dependent noise (SDN) in motor control studies (Enoka, Burnett, Graves, Kornatz, & Laidlaw, 1999; Schmidt, Zelaznik, Hawkins, Frank, & Quinn, 1979). However, accuracy, standard deviation (SD) or range of the task-related force component was not significantly different across force levels in this study. Reeves *et al.* (2008) and McCook *et al.* (2009) found an increase in the task-related force variability at higher *vs* lower force levels by using higher target forces (10, 20, 40, 60 and 80% of MVC) or fixing the increment in force (50N) between difference force levels. In the present study all three target forces corresponded to contractions of relatively low intensity (20% MVC is on average 53N) and small force increment between levels (average increase of approximately 13.25N for every 5% of MVC), compared with both Reeves *et al* (2008) and McCook *et al* (2009), which may explain the differences in the results.

In the present study higher force levels (20% MVC) increased bilateral muscle activity in both external oblique and rectus abdominis muscles compared with 5% MVC task. Additionally, bilateral increase in the rectus abdominis muscle was observed during 10% MVC compared with 5% MVC. Given that higher contraction levels of the abdominal muscles during trunk extension would counteract the action of the agonistic muscles (trunk extensors), there is not a straightforward explanation of why the central nervous system adopts such strategy when controlling the trunk. Nevertheless, such phenomenon has been extensively observed in previous studies, and the most recent findings indicates a co-contraction strategy aiming to increase trunk stiffness (Lee, Rogers, & Granata, 2006) and enhance stability during trunk extension (Cholewicki & McGill, 1996; Cholewicki & VanVliet, 2002; McCook et al., 2009; Reeves et al., 2008; van Dieen, Kingma, & van der Bug, 2003). Since the tasks demanded high levels of accuracy, increasing trunk stability would diminish trunk oscillations and improve performance in the task. Interestingly, the trunk extensor muscles (agonistic) evaluated in this study did not show increased muscular activity in higher force levels, contrary to results reported previously (McCook et al., 2009). However, Willigenburg *et al.* (2013) showed recently in both control subjects and LBP patients that during dynamic trunk movements (spiral-tracking task requiring precise trunk movements), there was no correlation between tracking errors and agonistic muscle activation. The absence of increased activity of trunk extensor muscles during progressively increasing the trunk extension force may be explained by several factors: (i) Despite using a similar experimental setup, McCook *et al.* (2009) applied the load through a shoulder harness, while in the present study, the subjects pulled a fixed force transducer attached to the frame; and (ii) Cholewicki *et al.* (2002) showed large redundancy in the motor system (ten major muscle groups were evaluated) while controlling isometric trunk exertions, where none of the muscle groups could be identified as the most important for controlling the trunk stability. Therefore, the small difference in the experimental setup between McCook *et al.*

(2009) and the present study and the high number of degrees of freedom involved in the task might suggest that the extensor trunk muscles that actually had their muscle activity increased were not analyzed in this study, for example deep muscles or muscles located higher at the trunk segment (Cholewicki & McGill, 1996). In addition, the present setup (different spine posture and trunk extension force) allowed a different motor strategy compared with the previous studies, where the extensor torque at the trunk segment might have been achieved via shared muscular contraction among different trunk extensor muscle groups (Cholewicki & VanVliet, 2002). This flexibility in the motor strategy could also have affected the normalization process in this study, where the motor strategy (i.e. muscle activation patterns) used during the MVC does not relate to the one used when the task requires trunk accuracy during low contraction levels. Nevertheless, the present results reinforce the previous findings indicating increased activation in the antagonistic muscles as common strategy when controlling the trunk segment in pain free conditions (van Dieen et al., 2003).

4.2. Effect of pain on force variability and muscle activation

This study used an experimental pain model to test the hypotheses that pain would provoke a reorganized muscle activation pattern and increase in force variability during isometric force-matching tasks in an otherwise healthy system. Similar to observations using experimental knee-related pain (Salomoni et al., 2013), experimental muscle pain per se did not affect the force variability in the tangential directions for which biofeedback was not shown to the subjects. Moreover, in this study, the force range in both tangential directions was not affect by pain, showing that healthy subjects are able to maintain the trunk stability in the frontal plane in presence of pain. Likewise, the variability, range, and accuracy of the task-related force were not altered by pain. Furthermore the MVC force (Newton) after the submaximal contractions (in the anterior-

posterior direction) was not affected by unilateral pain at right m. longissimus, probably indicating that the maximal capacity for producing trunk extension forces was maintained during the experiment. However, pain reduced bilaterally the activation of the rectus abdominis muscle, although the pain drawings indicated that subjects perceived pain only in the ipsilateral side to the painful injection. Such bilateral decrease in muscular activation might have been beneficial for trunk stability by avoiding asymmetrical muscle contraction and therefore, minimizing displacements of the trunk. Indications that such bilateral decreased muscle activity have indeed occurred without affecting the trunk stability is also indicated by the similar accuracy scores before and during pain (Table 1). Decreasing the antagonist muscle activity during painful trunk extension may however be interpreted as contrary to the observed strategy in pain free conditions of this experiment (Table 1), which in turn could impair the trunk stability (Cholewicki & McGill, 1996; Cholewicki & VanVliet, 2002; McCook et al., 2009; Reeves et al., 2008; van Dieen et al., 2003) and decrease the trunk stiffness (Lee et al., 2006), the last not estimated in this study. Overall, the decreased activity of the bilateral rectus abdominis muscles may indicate the robustness (ability to couple with disturbances while performing a movement) of the central nervous system in healthy individuals when utilizing visual feedback to control and correct the trunk force during low isometric trunk extension in presence of pain (Cholewicki & VanVliet, 2002).

Another indication of the motor reorganization while performing the task can be observed in the centroid position difference (CPD). The CPD in figure 2 graphically shows the mean force values in the tangential directions (medial-lateral and cephalo-caudal directions) generated while controlling the task-related force via visual feedback for every subject analyzed. During pain the high level of accuracy in the task-related force was probably preserved by reorganizing the activity of the different muscles in the trunk (including relevant deep trunk muscles not evaluated in this study (P. W. Hodges et al., 2003)), which affected the tangential forces. Similar motor

reorganization during pain has already been suggested in postural control tasks, where experimental pain induced dissimilar muscle contraction strategies among healthy subjects, although balance was maintained while recovering from external perturbations (Hirata et al., 2012). Inline, such phenomenon seems to be observed in Figure 2 by the larger variability and distance of each point (centroid's position) from the histogram origin during the painful condition, whereas during isotonic saline condition (control) the points were more centered on the origin of the histogram. However, the CPD (the difference between the centroid's position between baseline and during injections condition) was not significantly different between injections (painful vs control, Figure 3A and C), indicating that there was not a consistent change among all subjects when controlling the tangential forces during pain. Interestingly, the absolute difference in the CPD (ACPD) was significantly higher during pain compared to control (isotonic saline injection) conditions in the medial-lateral direction (Figure 3B). This indicates that during pain, the mean force generated in the tangential medial-lateral was different from baseline values without altering the force variability (force SD). Interestingly the significant difference was only found in the absolute centroid position difference (ACPD, Figure 3) and not in the difference per se (CPD), reflecting an individualized attempt of the CNS to find the most comfortable or efficient trunk extension movement pattern potentially also reducing the pain. The search pattern for the most optimal strategy is still to be clarified. Although this immediate adaptive strategy to acute pain in healthy subjects seems to be beneficial in maintaining accuracy in the task, using such strategies regularly could result in overloading different structures, which in a long term could lead to other painful states (P. W. Hodges & Tucker, 2011).

5. Conclusions

The present study demonstrated a reorganization of trunk muscle activity during painful trunk extensions. This adaptive strategy was distinct for each subject, although the final motor output did not affected the task-related force variability. Such achievement may be due to the robustness and high redundancy of the trunk motor system in healthy subjects, and in addition, low intensity isometric trunk extensions forces do not provide enough challenge to the sensory-motor system. These findings stress the importance to target and focus on individual treatment procedures in LBP patients to their individual pain adaptation pattern.

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Reference List

Andersson, G. B. (1999). Epidemiological features of chronic low-back pain. [Review]. *Lancet, 354*(9178), 581-585. doi: 10.1016/S0140-6736(99)01312-4

Arendt-Nielsen, L., Graven-Nielsen, T., Svarrer, H., & Svensson, P. (1996). The influence of low back pain on muscle activity and coordination during gait: a clinical and experimental study. [Clinical Trial
Randomized Controlled Trial
Research Support, Non-U.S. Gov't]. *Pain, 64*(2), 231-240.

Bank, P. J., Peper, C. E., Marinus, J., Beek, P. J., & van Hilten, J. J. (2013). Motor consequences of experimentally induced limb pain: a systematic review. [Research Support, Non-U.S. Gov't]. *Eur J Pain, 17*(2), 145-157. doi: 10.1002/j.1532-2149.2012.00186.x

Biering-Sorensen, F. (1982). Low back trouble in a general population of 30-, 40-, 50-, and 60-year-old men and women. Study design, representativeness and basic results. [Research Support, Non-U.S. Gov't]. *Dan Med Bull, 29*(6), 289-299.

Cassidy, J. D., Cote, P., Carroll, L. J., & Kristman, V. (2005). Incidence and course of low back pain episodes in the general population. [Comparative Study
Research Support, Non-U.S. Gov't]. *Spine (Phila Pa 1976), 30*(24), 2817-2823.

Cholewicki, J., & McGill, S. M. (1996). Mechanical stability of the in vivo lumbar spine: implications for injury and chronic low back pain. *Clin Biomech (Bristol, Avon), 11*(1), 1-15.

Cholewicki, J., & VanVliet, J. J. t. (2002). Relative contribution of trunk muscles to the stability of the lumbar spine during isometric exertions. [Research Support, Non-U.S. Gov't]. *Clin Biomech (Bristol, Avon), 17*(2), 99-105.

Enoka, R. M., Burnett, R. A., Graves, A. E., Kornatz, K. W., & Laidlaw, D. H. (1999). Task- and age-dependent variations in steadiness. [Research Support, U.S. Gov't, P.H.S.]. *Prog Brain Res, 123*, 389-395.

Filho, I. T., Simmonds, M. J., Protas, E. J., & Jones, S. (2002). Back pain, physical function, and estimates of aerobic capacity: what are the relationships among methods and measures? [Research Support, Non-U.S. Gov't
Research Support, U.S. Gov't, P.H.S.]. *Am J Phys Med Rehabil, 81*(12), 913-920. doi: 10.1097/01.PHM.0000030729.77020.2A

Harrison, D. E., Harrison, D. D., & Troyanovich, S. J. (1997). The sacroiliac joint: a review of anatomy and biomechanics with clinical implications. [Research Support, Non-U.S. Gov't
Review]. *J Manipulative Physiol Ther, 20*(9), 607-617.

Helewa, A., Goldsmith, C. H., & Smythe, H. A. (1993). Measuring abdominal muscle weakness in patients with low back pain and matched controls: a comparison of 3 devices. [Comparative Study
Research Support, Non-U.S. Gov't]. *J Rheumatol, 20*(9), 1539-1543.

Hestbaek, L., Leboeuf-Yde, C., Engberg, M., Lauritzen, T., Bruun, N. H., & Manniche, C. (2003). The course of low back pain in a general population. Results from a 5-year prospective study. [Research Support, Non-U.S. Gov't]. *J Manipulative Physiol Ther, 26*(4), 213-219.

Hestbaek, L., Leboeuf-Yde, C., & Manniche, C. (2003). Low back pain: what is the long-term course? A review of studies of general patient populations. [Research Support, Non-U.S. Gov't
Review]. *Eur Spine J, 12*(2), 149-165. doi: 10.1007/s00586-002-0508-5

Hirata, R. P., Arendt-Nielsen, L., Shiozawa, S., & Graven-Nielsen, T. (2012). Experimental knee pain impairs postural stability during quiet stance but not after perturbations. *Eur J Appl Physiol, 112*(7), 2511-2521. doi: 10.1007/s00421-011-2226-3

Hodges, P., van den Hoorn, W., Dawson, A., & Cholewicki, J. (2009). Changes in the mechanical properties of the trunk in low back pain may be associated with recurrence. [Research Support, Non-U.S. Gov't]. *J Biomech, 42*(1), 61-66. doi: 10.1016/j.jbiomech.2008.10.001

Hodges, P. W. (2001). Changes in motor planning of feedforward postural responses of the trunk muscles in low back pain. [Research Support, Non-U.S. Gov't]. *Exp Brain Res, 141*(2), 261-266. doi: 10.1007/s002210100873

Hodges, P. W., Moseley, G. L., Gabrielsson, A., & Gandevia, S. C. (2003). Experimental muscle pain changes feedforward postural responses of the trunk muscles. [Clinical Trial

Research Support, Non-U.S. Gov't]. *Exp Brain Res, 151*(2), 262-271. doi: 10.1007/s00221-003-1457-x

Hodges, P. W., & Richardson, C. A. (1996). Inefficient muscular stabilization of the lumbar spine associated with low back pain. A motor control evaluation of transversus abdominis. [Research Support, Non-U.S. Gov't]. *Spine (Phila Pa 1976), 21*(22), 2640-2650.

Hodges, P. W., & Richardson, C. A. (1998). Delayed postural contraction of transversus abdominis in low back pain associated with movement of the lower limb. [Research Support, Non-U.S. Gov't]. *J Spinal Disord, 11*(1), 46-56.

Hodges, P. W., & Tucker, K. (2011). Moving differently in pain: a new theory to explain the adaptation to pain. *Pain, 152*(3 Suppl), S90-98.

Hoy, D., Brooks, P., Blyth, F., & Buchbinder, R. (2010). The Epidemiology of low back pain. [Research Support, Non-U.S. Gov't

Review]. *Best Pract Res Clin Rheumatol, 24*(6), 769-781. doi: 10.1016/j.berh.2010.10.002

Jones, K. E., Hamilton, A. F., & Wolpert, D. M. (2002). Sources of signal-dependent noise during isometric force production. *J Neurophysiol, 88*(3), 1533-1544.

Kamavuako, E. N., Farina, D., Yoshida, K., & Jensen, W. (2009). Relationship between grasping force and features of single-channel intramuscular EMG signals. [Research Support, Non-U.S. Gov't]. *J Neurosci Methods, 185*(1), 143-150. doi: 10.1016/j.jneumeth.2009.09.006

Lee, P. J., Rogers, E. L., & Granata, K. P. (2006). Active trunk stiffness increases with co-contraction. [Research Support, N.I.H., Extramural

Research Support, U.S. Gov't, P.H.S.]. *J Electromyogr Kinesiol, 16*(1), 51-57. doi: 10.1016/j.jelekin.2005.06.006

MacDonald, D., Moseley, G. L., & Hodges, P. W. (2009). Why do some patients keep hurting their back? Evidence of ongoing back muscle dysfunction during remission from recurrent back pain. [Research Support, Non-U.S. Gov't]. *Pain, 142*(3), 183-188. doi: 10.1016/j.pain.2008.12.002

McCook, D. T., Vicenzino, B., & Hodges, P. W. (2009). Activity of deep abdominal muscles increases during submaximal flexion and extension efforts but antagonist co-contraction remains unchanged. [Research Support, Non-U.S. Gov't]. *J Electromyogr Kinesiol, 19*(5), 754-762. doi: 10.1016/j.jelekin.2007.11.002

McCowin, P. R., Borenstein, D., & Wiesel, S. W. (1991). The current approach to the medical diagnosis of low back pain. [Review]. *Orthop Clin North Am, 22*(2), 315-325.

Mellin, G., & Hurri, H. (1990). Referred limb symptoms in chronic low back pain. [Research Support, Non-U.S. Gov't]. *J Spinal Disord, 3*(1), 52-58.

Mista, C. A., Christensen, S. W., & Graven-Nielsen, T. (2015). Modulation of motor variability related to experimental muscle pain during elbow-flexion contractions. *Hum Mov Sci, 39*, 222-235. doi: 10.1016/j.humov.2014.09.006

Pope, M. H. (1989). Biomechanics of the lumbar spine. [Research Support, Non-U.S. Gov't Review]. *Ann Med, 21*(5), 347-351.

Reeves, N. P., Cholewicki, J., Milner, T., & Lee, A. S. (2008). Trunk antagonist co-activation is associated with impaired neuromuscular performance. [Research Support, N.I.H., Extramural]. *Exp Brain Res, 188*(3), 457-463. doi: 10.1007/s00221-008-1378-9

Salomoni, S. E., Ejaz, A., Laursen, A. C., & Graven-Nielsen, T. (2013). Variability of three-dimensional forces increase during experimental knee pain. [Research Support, Non-U.S. Gov't]. *Eur J Appl Physiol, 113*(3), 567-575. doi: 10.1007/s00421-012-2461-2

Salomoni, S. E., & Graven-Nielsen, T. (2012a). Experimental muscle pain increases normalized variability of multidirectional forces during isometric contractions. *Eur J Appl Physiol*.

Salomoni, S. E., & Graven-Nielsen, T. (2012b). Muscle fatigue increases the amplitude of fluctuations of tangential forces during isometric contractions. *Hum Mov Sci*.

Schmidt, R. A., Zelaznik, H., Hawkins, B., Frank, J. S., & Quinn, J. T., Jr. (1979). Motor-output variability: a theory for the accuracy of rapid motor acts. [Research Support, U.S. Gov't, P.H.S.]. *Psychol Rev, 47*(5), 415-451.

Schroeder, J., Schaar, H., & Mattes, K. (2013). Spinal alignment in low back pain patients and age-related side effects: a multivariate cross-sectional analysis of video rasterstereography back shape reconstruction data. *Eur Spine J, 22*(9), 1979-1985. doi: 10.1007/s00586-013-2787-4

Smith, M., Coppieters, M. W., & Hodges, P. W. (2005). Effect of experimentally induced low back pain on postural sway with breathing. *Exp Brain Res, 166*(1), 109-117. doi: 10.1007/s00221-005-2352-4

Steenstra, I. A., Verbeek, J. H., Heymans, M. W., & Bongers, P. M. (2005). Prognostic factors for duration of sick leave in patients sick listed with acute low back pain: a systematic review of the literature. [Research Support, Non-U.S. Gov't Review]. *Occup Environ Med, 62*(12), 851-860. doi: 10.1136/oem.2004.015842

Stein, R. B., Gossen, E. R., & Jones, K. E. (2005). Neuronal variability: noise or part of the signal? [Review]. *Nat Rev Neurosci, 6*(5), 389-397. doi: 10.1038/nrn1668

Tsao, H., Galea, M. P., & Hodges, P. W. (2008). Reorganization of the motor cortex is associated with postural control deficits in recurrent low back pain. [Research Support, Non-U.S. Gov't]. *Brain, 131*(Pt 8), 2161-2171. doi: 10.1093/brain/awn154

Tsao, H., & Hodges, P. W. (2008). Persistence of improvements in postural strategies following motor control training in people with recurrent low back pain. [Research Support, Non-U.S. Gov't]. *J Electromyogr Kinesiol, 18*(4), 559-567. doi: 10.1016/j.jelekin.2006.10.012

Tsao, H., Tucker, K. J., Coppieters, M. W., & Hodges, P. W. (2010). Experimentally induced low back pain from hypertonic saline injections into lumbar interspinous ligament and erector spinae muscle. *Pain, 150*(1), 167-172. doi: 10.1016/j.pain.2010.04.023

Tucker, K. J., & Hodges, P. W. (2010). Changes in motor unit recruitment strategy during pain alters force direction. [Research Support, Non-U.S. Gov't]. *Eur J Pain, 14*(9), 932-938. doi: 10.1016/j.ejpain.2010.03.006

van Dieen, J. H. (1997). Are recruitment patterns of the trunk musculature compatible with a synergy based on the maximization of endurance? [Comparative Study]. *J Biomech, 30*(11-12), 1095-1100.

van Dieen, J. H., Kingma, I., & van der Bug, P. (2003). Evidence for a role of antagonistic cocontraction in controlling trunk stiffness during lifting. [Clinical Trial]. *J Biomech, 36*(12), 1829-1836.

Willigenburg, N. W., Kingma, I., Hoozemans, M. J., & van Dieen, J. H. (2013). Precision control of trunk movement in low back pain patients. *Hum Mov Sci, 32*(1), 228-239. doi: 10.1016/j.humov.2012.12.007

Wolff, A. P., Groen, G. J., Wilder-Smith, O. H., Richardson, J., van Egmond, J., & Crul, B. J. (2006). Do diagnostic segmental nerve root blocks in chronic low back pain patients with radiation to the leg lack distinct sensory effects? A preliminary study. [Randomized Controlled Trial]. *Br J Anaesth, 96*(2), 253-258. doi: 10.1093/bja/aei307

Figures Legend

Figure 1. Superimposed body chart pain drawings (n = 12) after saline injections into the into the right longissimus muscle. The pain distribution after isotonic saline (left) and hypertonic saline (right) injections are illustrated.

Figure 2. Three-dimensional shaded surface based on the mean value for the centroid's position difference (CPD), in bins, for both medial-lateral (ML) and cephalo-caudal (CC) direction during injections of hypertonic and isotonic saline in the right m. longissimus muscle. Positive values in the ML direction indicate that CPD is positioned more to the left compared with the baseline condition. Positive values in the CC direction indicate that CPD is located higher than baseline condition. The centre of the figure (0 x 0) indicates the relative CPD position during baseline for all subjects. Warm colors indicate larger number of subjects showing similar CPD than cool colors. During isotonic injection, the distribution of the CPD is more centered on the origin than during hypertonic injection condition.

Figure 3. Mean (+ SD, N = 12) of centroid's position difference (CPD) and the absolute centroid's position difference (ACPD) in the medial-lateral and cephalo-caudal directions during and post saline injections (hypertonic and isotonic). **(A)** CPD in medial lateral direction. **(B)** ACPD in medial lateral direction. **(C)** CPD in cephalo-caudal direction. **(D)** ACPD in cephalo-caudal direction. During hypertonic saline injections, the ACPD was significantly increased compared with the isotonic saline injection and its respective post injection condition (*, NK: $P < 0.05$)

Figure 4. Mean (+ SD, N = 12) of iEMG (% MVC) values for left (A) and right (B) rectus abdominis muscles for baseline, during (injection) and post-injection conditions during 3

submaximal (5, 10 and 20% MVC) isometric trunk extension. The symbol "*" indicates significant 3-way interaction between the factors *injection*, *condition* and *force* for the iEMG data after normalization by baseline values (NK: P = 0.03).

Table 1: Muscular activity, force variability, accuracy and MVC force during 5, 10 and 20% MVC submaximal tasks (n = 12)

	Baseline 1 - Prior Isotonic Injection			Baseline 2 - Prior Hypertonic Injection		
	5 % MVC	**10 % MVC**	**20 % MVC**	**5 % MVC**	**10 % MVC**	**20 % MVC**
Force Variability, Range and Accuracy (N)						
Medial-lateral force SD	0.39 ± 0.12	0.52 ± 0.33	0.66 ± 0.33	0.52 ± 0.23	0.54 ± 0.25	0.65 ± 0.48
Cephalo-caudal force SD	1.83 ± 0.73	1.93 ± 0.56	2.23 ± 0.85	2.15 ± 1.26	2.56 ± 1.21	2.32 ± 1.20
Anterior-posterior force SD	0.82 ± 0.30	0.83 ± 0.21	0.94 ± 0.24	0.78 ± 0.27	0.83 ± 0.28	0.86 ± 0.36
Medial-lateral force Range	2.18 ± 0.64	2.7 0 ± 1.39	3.10 ± 1.35	2.43 ± 0.93	2.59 ± 0.83	3.23 ± 2.47
Cephalo-caudal force Range	7.48 ± 2.96	8.32 ± 2.76	9.25 ± 3.67	8.97 ± 5.53	10.26 ± 5.34	9.93 ± 5.84
Anterior-posterior force Range	4.99 ± 2.16	5.26 ± 1.33	5.7 ± 1.51	4.99 ± 2.48	4.87 ± 2.18	5.44 ± 2.37
Accuracy - Mean absolute Error	0.62 ± 0.20	0.64 ± 0.17	0.72 ± 0.18	0.59 ± 0.18	0.66 ± 0.22	0.68 ± 0.29
Centroid's Position (mm)						
Cephalo-caudal	53.1 ± 9.4	56.3 ± 9.9	57.7 ± 9.7	55.3 ± 8.7	54.2 ± 8.1	53.0 ± 6.5
Medial-lateral	49.5 ± 4.4	50.8 ± 7.6	49.6 ± 7.6	50.7 ± 8.8	50.8 ± 6.9	49.1 ± 7.5
Muscle Activity (EMG, pooled within left and right side, % MVC)						
Rectus abdominis	20.7 ± 19.4	***24.8 ± 19.4****	***33.9 ± 21.5****	17.1 ± 15.6	***24.7 ± 16.4****	***38.2 ± 25.1****
External oblique	16.9 ± 16.7	18.5 ± 11.4	***25.6 ± 15.9****	14.3 ± 9.0	16.0 ± 10.0	***25.3 ± 16.2****
Longissimus	5.1 ± 2.7	6.7 ± 4.1	6.1 ± 3.1	5.0 ± 2.7	5.7 ± 3.1	8.0 ± 6.6
Multifidus	3.1 ± 1.4	3.5 ± 1.4	3.4 ± 1.4	3.5 ± 2.1	3.3 ± 1.7	5.8 ± 8.0

Data presented as mean (± SD, N = 12) for both baselines conditions (prior hypertonic and isotonic injection). Muscle activity (EMG, % of MVC) and standard deviation (SD) and accuracy (mean absolute error) for the 3 force directions [medial-lateral (ML), cephalo-caudal (CC) and anterior-posterior (AP), in Newton (N)] and centroid's positions for CC and ML direction are presented for each force level: 5%, 10% and 20% of maximal voluntary contraction (MVC). Significant differences in the EMG level between force levels are indicated by: one asterisk "*" when higher than 5% MVC condition; and two asterisks "**" when higher than all other conditions ($P<0.05$).

Figure 1

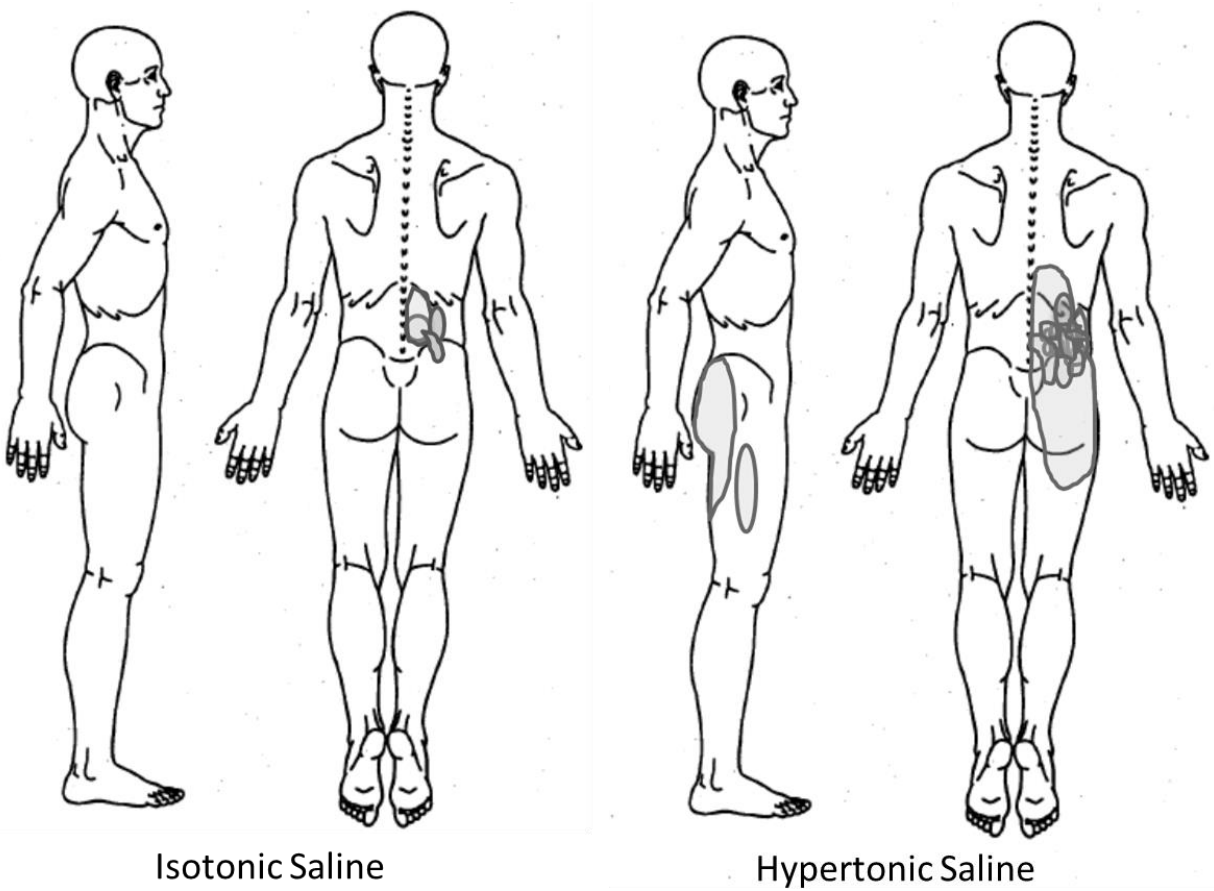


Figure 2

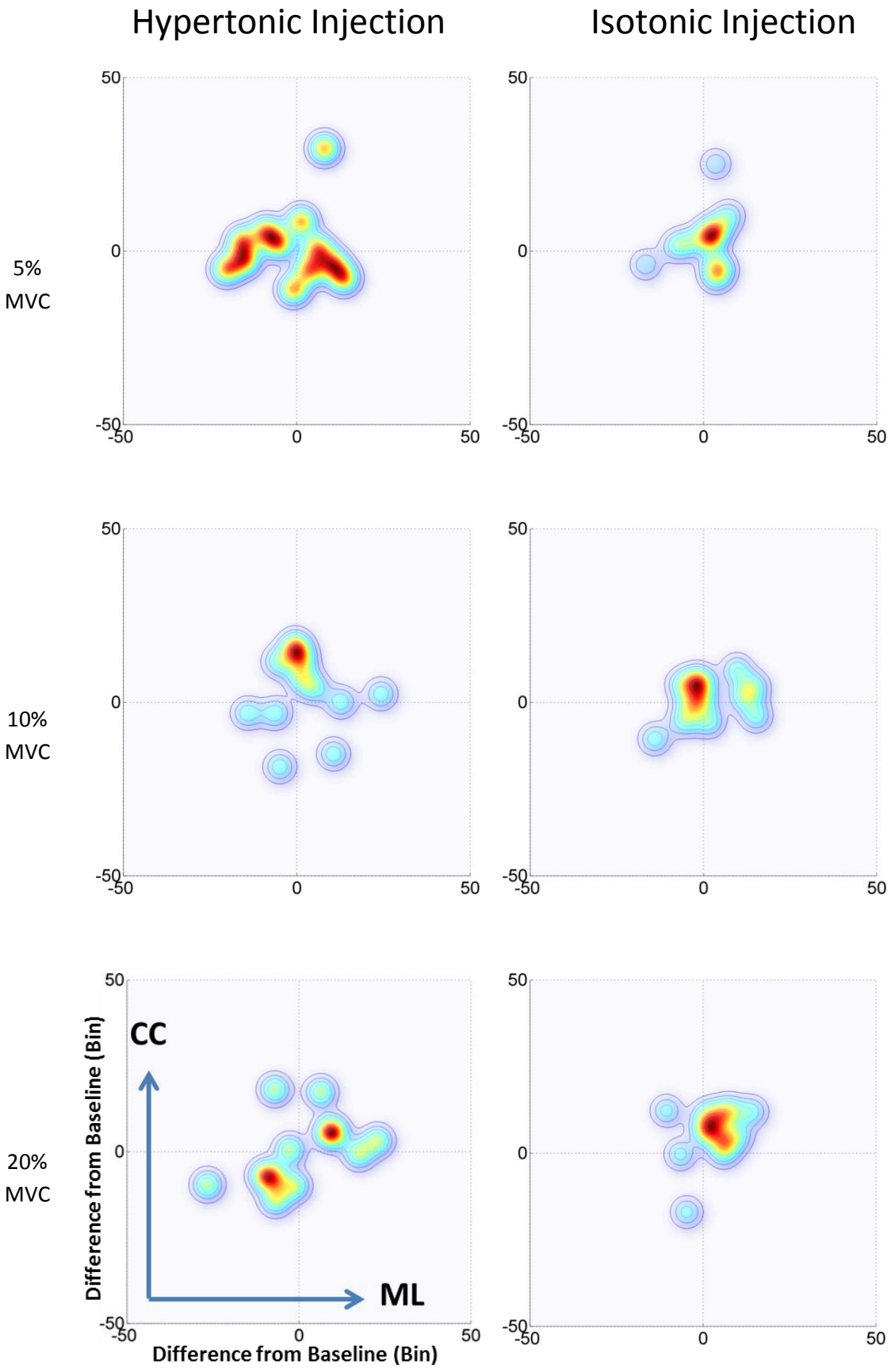

Hypertonic Injection
Isotonic Injection
5% MVC
10% MVC
20% MVC
50
0
-50
CC
ML
Difference from Baseline (Bin)
Difference from Baseline (Bin)

Figure 3

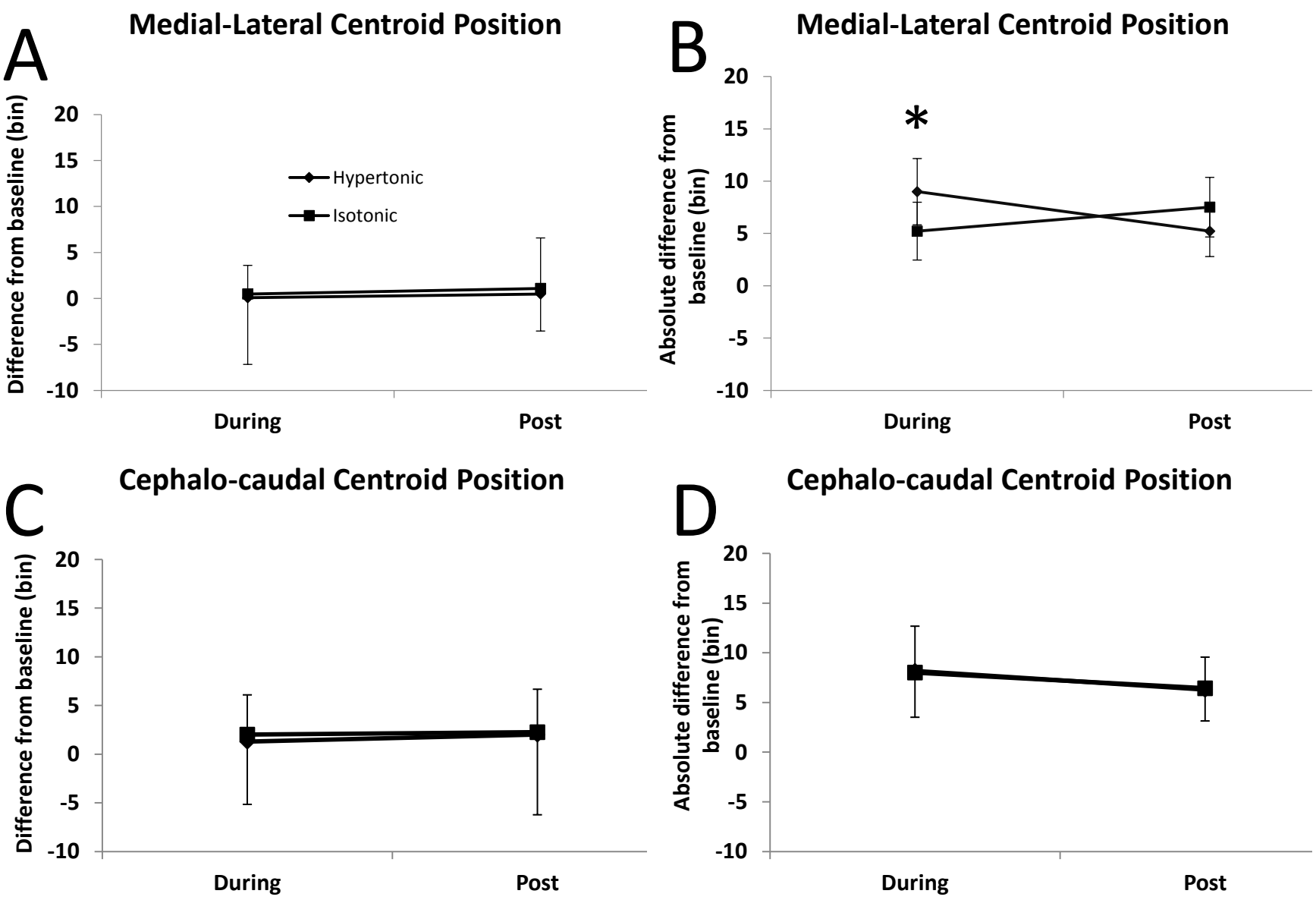
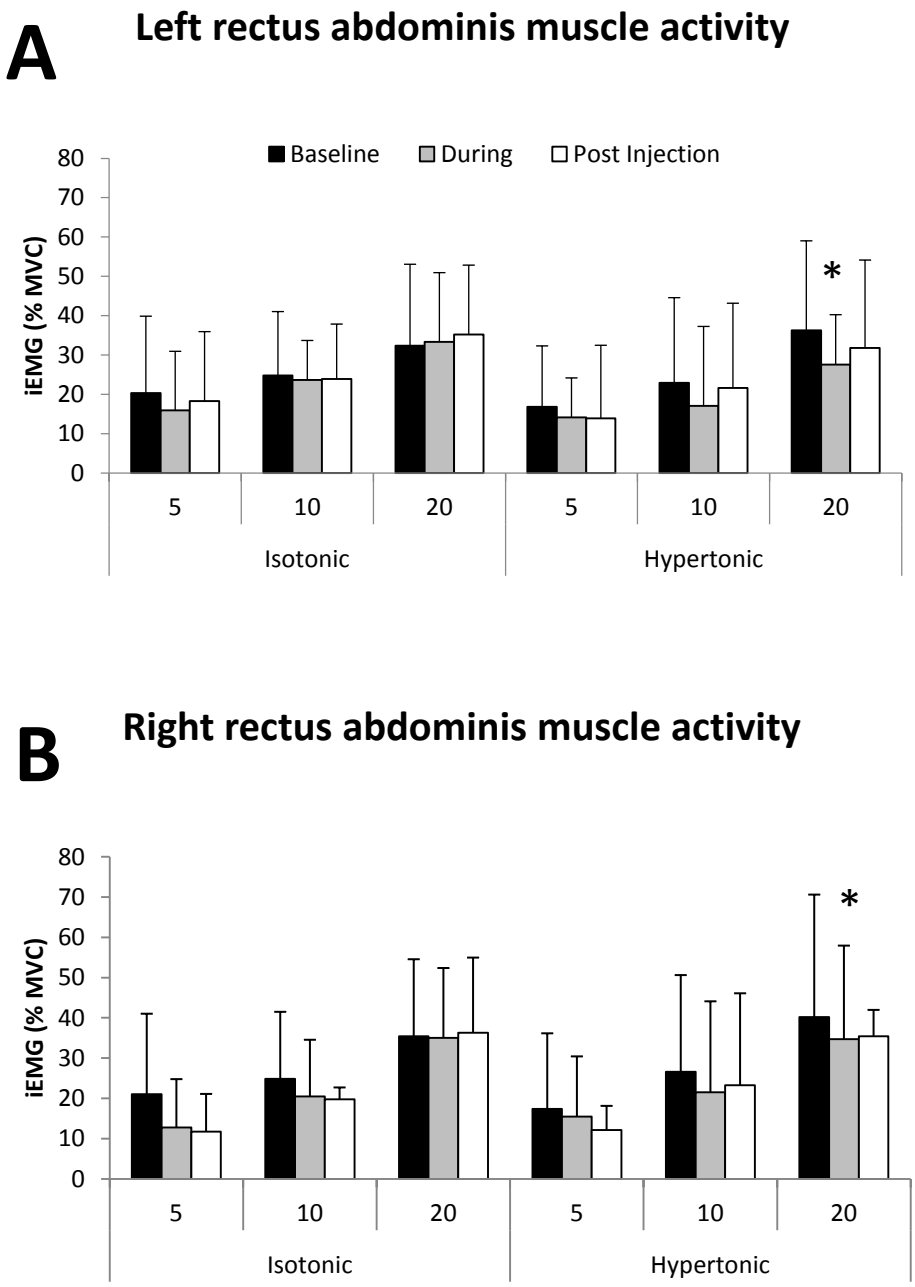

A
Medial-Lateral Centroid Position
Difference from baseline (bin)
20
15
10
5
0
-5
-10
Hypertonic
Isotonic
During
Post
B
Medial-Lateral Centroid Position
Absolute difference from baseline (bin)
*
During
Post
C
Cephalo-caudal Centroid Position
Difference from baseline (bin)
During
Post
D
Cephalo-caudal Centroid Position
Absolute difference from baseline (bin)
During
Post

Figure 4


A
Left rectus abdominis muscle activity
Baseline
During
Post Injection
iEMG (% MVC)
80
70
60
50
40
30
20
10
0
*
5
10
20
5
10
20
Isotonic
Hypertonic
B
Right rectus abdominis muscle activity
iEMG (% MVC)
80
70
60
50
40
30
20
10
0
*
5
10
20
5
10
20
Isotonic
Hypertonic

Highlights

- Experimental pain was applied in the longissimus muscle in healthy subjects while performing isometric trunk extensions.
- Pain decreased the EMG activity bilaterally of the rectus abdominis muscles.
- Performance of the task was not affected by pain.
- Tangential forces generated by the trunk segment were altered by pain.
- The motor adaptations to pain were unique for each individual.