Motor adaptations to trunk perturbation: effects of experimental back pain and spinal tissue creep

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Running head: Motor adaptations to trunk perturbation

Abstract

Introduction: In complex anatomical systems, such as the trunk, motor control theories suggest that many motor solutions can be implemented to achieve a similar goal. While reflex mechanisms act as a stabilizer of the spine, how the central nervous system uses the trunk redundancy to adapt the neuromuscular responses under the influence of external perturbations, such as experimental pain or spinal tissue creep is still unclear. The aim of this study was to identify and characterize trunk neuromuscular adaptations in response to unexpected trunk perturbations under the influence of spinal tissue creep and experimental back pain.

Methods: Healthy participants experienced a repetition of sudden external trunk perturbations in two protocols: [1] 15 perturbations before and after a spinal tissue creep protocol, [2] 15 perturbations with and without experimental back pain. Trunk neuromuscular adaptations were measured using high-density electromyography to record erector spinae muscle activity recruitment patterns and using a motion analysis system.

Results: Muscle activity reflex attenuation was found across unexpected trunk perturbation trials under the influence of creep and pain. A similar area of muscle activity distribution was observed with or without back pain, as well as before and after creep. No change of trunk kinematics was observed.

Conclusion: While under normal circumstances muscle activity adaptation occurs throughout the same perturbations, a reset of the adaptation process is present when experiencing a new perturbation such as experimental pain or creep. However,

participants are still able to attenuate reflex responses under these conditions using variable recruitment pattern of back muscles.

New & Noteworthy

The current study characterizes, for the first time, trunk motor adaptations using high-density surface electromyography when the spinal system is challenged by a series of unexpected perturbations. We propose that the central nervous system is able to adapt neuromuscular responses using a variable recruitment pattern of back muscles to maximize the motor performance, even under the influence of pain or when the passive structures of the spine are altered.

Introduction

Trunk muscles play an important role in postural stability. In everyday life, several neuromuscular adaptations, such as an increase of reflex muscle activity and/or postural adjustments, are used when the trunk system is challenged by an external perturbation. The complexity of muscles surrounding the trunk makes it a relevant system to better understand how motor variability could be used to face environmental perturbations. However, few studies have investigated the ability of the trunk system to adapt when it is challenged by repetitions of external unexpected perturbations (Abboud et al. 2016c; Skotte et al. 2004). These studies suggest that the CNS is able to adapt the neuromuscular outcomes based on previous experience of a given external perturbation even when it is unexpected. These neuromuscular adaptations, referring to an attenuation of trunk reflex activity amplitude and/or postural oscillations (Abboud et al. 2016c; Skotte et al. 2004) have been also reported in other muscles, such as the neck or lower limbs (Blouin et al.

2003; Nashner 1976; Siegmund et al. 2003). These observations concur with the ability of the CNS to use trunk muscle system redundancy to adapt neuromuscular responses. Based on these findings, it seems reasonable to suggest that these responses occur when trunk muscle control works adequately. This raises the question of the capacity of the CNS to adapt trunk neuromuscular responses to generate proper motor control in the presence of an external condition that has the potential to alter trunk muscle control, such as experimental back pain or spinal tissue creep.

In a recent systematic review, our group explored and synthetized the effects of spinal tissue creep on trunk muscles neuromuscular responses while postural stability was challenged by unexpected trunk perturbations (Abboud et al. 2016a). Most studies reviewed were of good quality but the high heterogeneity and small sample sizes rendered the evidence inconclusive. One of the reason for such discrepancies among the studies could be the lack of information regarding muscle activity recruitment strategies used to face external trunk perturbations. All of the above mentioned studies have been limited by the use of bipolar electromyography (EMG). One could argue that using high-density EMG (HD-EMG), because of its larger size and high number of recording electrodes, could provide a unique perspective on muscle activity reflex responses, such as the topographical distribution of muscle activity (Zwarts and Stegeman 2003). It was recently observed that the level of motor variability, assessed by muscle activity recruitment pattern using HD-EMG, could be influenced by the presence of spinal tissue creep (Abboud et al. 2016b). Under the influence of creep deformation, spinal passive structures are altered, and a reorganization of muscle activity occurs in order to compensate for such changes (Abboud et al. 2016b; Solomonow 2012).

As for the effects of experimental back pain on low back neuromuscular responses, no change in muscle reflex amplitude was observed, while contradictory results were found for the erector spinae reflex latency following unexpected trunk perturbations (Boudreau et al. 2011; Gregory et al. 2008; Miller et al. 2013). It has also been proposed that the level of motor variability can also be influenced by the presence of pain. For instance, a higher motor variability was observed in the upper limb or in the trunk in the presence of acute pain, whereas it was lower under chronic pain conditions (Madeleine 2010; van Dieen et al. 2017). Moreover, it has been proposed that neuromuscular responses to pain are not stereotypical, and that the pattern of muscle activity recruitment varies between individuals submitted to experimental back pain (Hodges et al. 2013). Overall, these observations reflect the trunk muscle system redundancy, which enables the central nervous system (CNS) to choose from several distinct combinations of muscle activations.

Therefore, the first objective of the present study was to identify and characterize the neuromuscular responses in healthy participants when they are submitted to unexpected trunk perturbations in two different experimental conditions (spinal tissue creep and experimental back pain). The second objective of this study was to determine whether the trunk neuromuscular responses to an unexpected perturbation can be modulated by a previous and similar trunk perturbation (trial-to-trial adaptation) under the influence of spinal tissue creep and experimental back pain. Based on the trunk muscle system high redundancy potential and the prediction of Madeleine's motor variability model (2010), we hypothesized that participants would be able to adapt their neuromuscular responses

across trunk perturbations trials even in presence of creep deformation or experimental back pain.

Methods

Participants

Two groups of participants were included in this study: an experimental group and a control group. In the experimental group, twenty healthy participants (4 women and 16 men) were recruited from the university community. Participant mean (*M*) age, height, weight and BMI were respectively 28.2 (standard deviation [*SD*] = 5.4) years, *M* = 1.75 (*SD* = 0.07) m, *M* = 77.3 (*SD* = 13.8) kg and *M* = 25.1 (*SD* = 3.6) kg/m^2. For the control group, fourteen healthy participants (5 women and 9 men) were recruited from the university community. Participant *M* age, height, weight and BMI were respectively 27.1 (*SD* = 6.9) years, *M* = 1.75 (*SD* = 0.09) m, *M* = 71.5 (*SD* = 11.4) kg and *M* = 23.3 (*SD* = 2.3) kg/m^2.

For both groups, the exclusion criteria were: history of acute/chronic thoracic or low back pain in the past 6 months, ankylosing spondylitis, inflammatory arthritis, trunk neuromuscular disease, scoliosis (≥ 15 °), and previous spinal surgery. The project received approval from the university's ethics committee for research with humans (Comité d'éthique de la recherche avec des êtres humains). Before their participation in this study, all participants gave their written informed consent.

Protocol

Healthy participants from the experimental group participated in two different conditions on separate days. In one condition, participants were asked to sit with a flexion of their trunk to induce spinal tissue creep in the lumbar region (creep condition). In the other condition, participants were submitted to an experimental low back pain protocol (experimental pain condition). In order to minimize possible order effects, half of the participants were first submitted to the creep protocol, whereas the other half started with the experimental pain protocol. In both experimental conditions, participants responded to two series of 15 unexpected trunk perturbations. During the experimental pain condition, one of the two series was performed without the influence of experimental back pain and the other one with the influence of experimental back pain. Half of the participants started without the presence of experimental pain, and the other half started with the presence of experimental pain during the trunk perturbations trials. The two series of perturbation trials were separated by a 5-minute rest period. As for the creep condition, all participants started the first series of 15 trunk perturbations before the creep deformation protocol and the second series was performed immediately after. The two experimental conditions were carried out several days apart (minimum of 7 days) to allow full recovery from creep deformations. During these recovery days, participants were asked to avoid any unusual activity, such as a new physical activity. Finally, healthy participants from the control group participated in one protocol during which they were submitted to the same two series of 15 unexpected trunk perturbation with a 5-minutes rest between the two series (rest condition). Figure 1 represents the experiment timeline.

At the beginning of each condition, kinematic sensors and EMG electrodes were placed on the participants. Once the instrumentation in place, participants were asked to perform

maximal voluntary contractions (MVC) in trunk flexion and trunk extension direction. Two or three trials were performed in each direction. Participants were asked to perform a third trial only when their second MVC trials was 5% higher than the first one. For trunk flexion MVC trials, participants had to pull anteriorly on a cable attached at the T8 level to a load cell (Model LSB350; Futek Advanced Sensor Technology Inc., Irvine, CA, USA). For trunk extension, participants had to pull posteriorly on a cable. Verbal cues were provided by assessors to motivate the participants during MVC performances. The MVC trials, which consisted of ramp contractions, were performed in a semi-seated position in a custom-made chair (see Figure 1 and "Trunk perturbation protocol"). Ramp contractions consisted of progressively increased extensor muscles force for approximately 3 seconds in order to reach the maximal strength of these muscles.

[Insert Figure 1 around here]

Creep condition

Before the beginning of the creep protocol and immediately after, the range of motion (ROM) of trunk flexions was measured by the same assessor. To induce spinal tissue creep, participants were asked to sit on a bench for 30 minutes. In this position, they were asked to bend forward to achieve a trunk flexion of approximately 75% of their maximal range of trunk flexion. In this 30-minute interval, the participant trunk was supported by a table to minimize trunk muscle activity. If needed, a cushion was added on the table for the participant's comfort. Moreover, a 90-degree knees flexion was required to limit the occurrence of hamstring muscles stretching. The trunk ROM was measured by placing a

digital dual inclinometer (Dualer IQ Pro™ Digital Inclinometer, JTECH Medical; USA) on the L1 and L5 vertebrae. Trunk ROM was assessed in a straight upright position during which participants were asked to tilt the trunk forward as much as possible, without bending the knees. Three attempts were performed, before and after the creep deformation protocol. The trial with the highest trunk ROM was considered for the analysis.

Experimental Pain Condition

To induce experimental pain, thermal cutaneous stimulations were administered using a 9-cm2 contact thermode (Model TSA-2001; MEDOC Advanced Medical Systems, Ramat Yishai, Israel) placed on the skin over the L3 spinous process, between the two arrays EMG. The thermode was placed at the beginning of the experiment, once the EMG was installed, and was kept in position during the experiment with a custom-made adjustable belt, while the belt was installed over the EMG. After each thermal stimulation, participant had to rate their perceived pain on a validated numerical rating scale (NRS) including verbal and numerical guide: no pain (0/100), light pain (21/100), moderate pain (46/100), strong pain (75/100) and extreme pain (97/100) (Rainville et al. 1992). The level of noxious heat was individually adjusted to induce moderate pain. To achieve a moderate pain, an ascending protocol was used: 15 seconds of noxious stimulation followed by a 15-second rest period without noxious stimulation. The ascending protocol started with the same baseline temperature for all participants set to 42 °C, and increased with steps of 0.5 °C until the participant perceived pain corresponding to a moderate level. The highest temperature used was 50 °C to avoid any

tissue damage. If a participant did not feel moderate pain at 50 °C, they were excluded from the study. The temperature triggering moderate pain was used during the unexpected trunk perturbations. The noxious stimulation started 8 seconds before the random onset of the perturbation and stopped one second after the perturbation onset.

Trunk Perturbation Protocol

To induce unexpected trunk perturbation, a custom-made apparatus was used to generate a posterior to anterior perturbation of the trunk. Participants were in a semi-seated position with ~75 degrees of flexion of the knee, ~110 degrees of flexion of the hip and their trunk in a natural straight position (Figure 2). A harness was installed over their upper body and attached at the T8 level by a cable using a pulley system. The trigger was connected to a small motor by a cable. Once the motor started, it was able to pull the trigger and consequently initiate the trunk perturbation by releasing the tension in the cable, which forced the trunk to control anterior movement. A signal from the motor was sent to a computer to determine the exact moment of the perturbation onset. The trigger was also connected to a load cell (Model LSB350; Futek Advanced Sensor Technology Inc., Irvine, CA, USA) to measure the force exerted by participants in trunk flexion. The magnitude of the trunk perturbation corresponded to 20% of the MVC in trunk flexion. Participants were instructed to maintain this pulling force, and once the perturbation was triggered, to return to their original position. Using a computer screen, visual feedback was provided to the participants to help them reach the target force of 20% of trunk flexion MVC. To avoid any anticipation of the trunk perturbation, the onset of the perturbation varied between 1, 3 or 5 seconds, according to a random sequence generated

by Matlab (Mathworks, Natick, MA, USA). Moreover, participants wore headphones to mute the sound of the perturbation trigger.

[Insert Figure 2 around here]

Data acquisition

Myoelectric activity was recorded from the flexor and extensor trunk muscles. Before the application of any EMG electrode, each location site was prepared through the reduction of skin impedance by shaving body hair, gently exfoliating the skin with fine-grade sandpaper (Red DotTrace Prep, 3 M; St. Paul, MN, USA) and wiping the skin with alcohol swabs. With regards to the extensor trunk muscles, surface EMG of the right and left erector spinae muscles was recorded using high-density EMG (HD-EMG) (model ELSCH064; LISiN-OT Bioelettronica; Torino, Italy). The HD-EMG consisted of two grids composed of 64 electrodes and organized in an 8x8 matrix (10 mm inter-electrode distance). The center of each grid was located at L3 level and the medial edge of the array was at ~2cm from the L3 spinous process (to avoid any contact with the thermode). One bracelet ground electrode was placed on the right wrist. Signals from the bipolar HD-EMG were amplified (64-channel sEMG amplifier, SEA 64, LISiN-OT Bioelettronica; Torino, Italy; –3 dB bandwidths 10–500 Hz) by a factor of 5,000 during the protocol. The signal was sampled at 2048 Hz and converted to digital form by a 12-bit A/D converter. As for the flexor trunk muscles, rectus abdominis and external obliquus abdominis muscle activities were recorded bilaterally, using a differential Ag surface EMG sensor with a common mode rejection ratio of 92 dB at 60 Hz, a noise level

of 1.2 μV, a gain of 10 V/V ± 1%, and a bandwidth of 20–450 ± 10% (Model DE2.1, Delsys Inc., Boston, MA, USA) amplified by a factor 10,000. The signal was sampled at 2048 Hz with a 12-bit A/D converter (PCI 6024E, National Instruments, Austin, TX, USA). Each bipolar signal was filtered using a band-pass filter in the frequency bandwidth-30-450 Hz (2^{nd} order Butterworth filter). Moreover, notch filters were also applied to the EMG signals to eliminate the 60 Hz and 100 Hz power line interferences and their harmonics. The same investigator assessed the placement of each electrode for all participants to avoid inter-rater variability. Rectus abdominis electrodes were positioned parallel to the muscle fibers, so that they were located approximately 2 cm lateral and across from the umbilicus over the muscle belly (Criswell and Cram 2011). As for the external obliquus, the electrodes were placed lateral to the rectus abdominis and directly above the anterior superior iliac spine (halfway between the crest and the ribs parallel to the muscle fibers) (Criswell and Cram 2011). The myoelectric signals from both EMG acquisition systems were collected using the OT Bioelettronica custom software. Muscle activity from all the trunk muscles (extensor and flexor) was normalized with respect to the trunk extension and flexion MVC values.

Kinematics of the trunk during perturbation trials were collected using a 3-D motion analysis system (Optotrak Certus, Northern Digital, Waterloo, ON, Canada). Kinematic sensors (light-emitting diodes) were placed, by the same assessor for each participant, on the left side of participants' trunk over two anatomical landmarks: (1) L1, (2) T11. These markers were positioned a few centimeters on the left side of the trunk to avoid creating interference with EMG signals. Data from kinematic sensors were sampled at 100 Hz and low-pass filtered with a dual-pass, fourth-order Butterworth filter using a cut-off

frequency of 5 Hz. Finally, EMG data and kinematic data were synchronized through a signal triggered by OT Bioelettronica software and Matlab (MathWorks).

Data Analysis

From HD-EMG signals, four variables were computed: the baseline activity, the reflex latency, the EMG reflex and the area of spatial distribution of muscle activity. Left and right sides of the erector spinae muscles were analyzed separately. From trunk flexors EMG signals, reflex activity was also computed. To avoid inclusion of any voluntary responses, reflex response latencies superior to 300 ms from the perturbation onset were excluded from the analysis.

Baseline Activity

Baseline activity of the erector spinae muscles corresponded to the mean EMG amplitude of the root mean square (RMS) using a 500-ms window prior to the onset of the trunk perturbation. The mean of all electrodes for each HD-EMG (left and right) was calculated.

Reflex Latency

Reflex latency was defined as the time delay from the perturbation onset to the reflex onset.

To determine the reflex onset, HD-EMG signals were Butterworth filtered (sixth order, 50 Hz cut-off frequency) and assessed using a sliding window of 25 ms (Lariviere et al. 2010). Muscle activity onset was then computed using an automated method: the SD method (Hodges and Bui 1996). More precisely, the EMG onset was detected when the EMG signals exceeded three SD above the mean baseline activity amplitude.

301 Erector Spinae EMG Reflex Amplitude

302 From each HD-EMG, the EMG reflex amplitude corresponded to the mean RMS value
303 from a window of 100 ms, divided equally (50 ms) on either side of the reflex peak. The
304 reflex peak corresponded to the highest RMS value following perturbation onset. The
305 reflex peak had to be present in a 300 ms window following the perturbation onset to be
306 considered in the analysis.

307 Spatial Distribution Area of Reflex Activity

308 The spatial distribution area of reflex activity was computed using the muscle activity
309 range of displacement (centroid) from the HD-EMG across the perturbation trials; a
310 method described in a previous publication (Abboud et al. 2016c). The spatial
311 distribution area of reflex activity represents the level of motor variability expressed as
312 muscle activity recruitment pattern.

313 Abdominal EMG Reflex Amplitude

314 Across perturbation trials, the reflex activity in the abdominal muscles rarely occurred. In
315 the majority of participants, the EMG reflex amplitude did not exceed three SD above the
316 mean baseline activity amplitude following the second or third perturbations trials.
317 Therefore, mean RMS values of the rectus abdominis and obliquus externus were
318 computed based on the same 100-ms window used for erector spinae data analyses.

319 Trunk Kinematic

320 Trunk kinematics were analyzed using the data from both kinematic sensors to create a
321 vector. Trunk motion was obtained by calculating the trunk flexion angle between the
322 T11-L1 vector and a horizontal vector relative to the ground. From the trunk motion,
323 three variables were extracted. [1] The trunk angle values corresponded to the ROM

between the starting position before the trunk perturbation, and the maximal trunk flexion following perturbation onset. From the trunk angle, [2] peak velocity and [3] time to peak velocity were computed.

Statistical Analysis

For each dependent variable, the normality of distribution was evaluated using the Kolmogorov—Smirnov test, and by visual inspection. Student *t*-tests for dependent samples were used to compare the area of reflex activity spatial distribution before and after the creep protocol, as well as, with and without experimental back pain. Student *t*-tests for dependent samples were also used to identify whether a difference occurred between the left and right side of the abdominal EMG. A mixed model two-way repeated measure ANOVA was conducted to assess [1] the trial-to-trial adaptation effect across perturbations, [2] the condition effect (creep effect or experimental pain effect or rest effect) and [3] the interaction effect (condition x adaptation) for each dependent variable (baseline activity, reflex latency, EMG reflex amplitude for erector spinae and abdominal muscles, and trunk kinematic variables). For the baseline activity, reflex latency, EMG reflex and kinematic variables, the means of the first and last five perturbation trials of the first and the second series of the 15 trunk perturbation trials were considered for the two-way repeated measure ANOVA. When necessary, the Tukey post hoc test was performed for pairwise comparisons. For all statistical analyses, a $p < 0.05$ was considered to be significant.

Results

During the experimental pain condition, the magnitude of the perturbation ranged from 40.0 to 79.4N, with an average of 57.3N. During the creep condition, the magnitude of the perturbation ranged from 39.7 to 78.6N, with an average of 56.2N. During the control condition, the magnitude of the perturbation ranged from 33.1 to 82.5N, with an average of 53.6N. These magnitudes are similar to the ones used in previous studies that used similar trunk perturbation protocols (Abboud et al. 2016c; Radebold et al. 2000).

In all conditions (creep, experimental pain, and control), 4% of all perturbation trials from HD-EMG recordings were excluded from the analyses due to the absence of a reflex response. These excluded trials corresponded to 4% in the creep condition, 5% in the experimental pain condition, and 3% in the control condition. Moreover, for one participant, the left electrode on the rectus abdominis muscle was removed from the analysis during the creep protocol due to a technical issue.

Regarding abdominal EMG, mean values of left and right rectus abdominis, as well as mean values of left and right external obliquus, were used for the analyses, since no statistical difference was identified between both sides (all *p*s > 0.05).

Rest Condition

Between the first series of 15 trunk perturbations and the second series, erector spinae, rectus abdominis and externus obliquus activity in the control condition showed a significant reduction of EMG reflex amplitude (all *ps* < 0.05, except for the right side of the erector spinae, $p = 0.12$) (Table 1). A clear and significant adaptation effect across the perturbations trials before and after the rest period was observed in the erector spinae EMG reflex amplitude (only on the right side, $p = 0.03$, Table 1 and Figure 3), and in the

obliquus externus EMG reflex amplitude (p = 0.03). Most of the other EMG and kinematic variables did not significantly change before and after the rest period, and did not adapt over perturbation trials (Table 1).

[Insert Table 1 around here]

Creep condition

Data from five participants were excluded from the ROM analyses due to technical measurements errors during the ROM assessment. Results showed that participants' ROM in full trunk flexion increased from 37.7° (SD = 11.6) before the creep protocol to 39.9° (SD = 8.7) afterwards (dependent t-tests, p = 0.10). The mean increase corresponded to 4% after the creep deformation protocol.

Following the spinal tissue creep protocol, erector spinae baseline activity tended to be higher (significant only on one side of the erector spinae, p = 0.04) than before creep (Table 2). Moreover, baseline activity and erector spinae EMG reflex amplitude significantly decreased (adaptation effect) across trunk perturbation trials regardless of the presence or not of creep (Table 2 and Figure 3). All the other EMG and kinematic variables did not significantly change before and after the creep condition, and did not adapt over perturbation trials (Table 2). Moreover, dependent t-tests revealed no significant difference between pre- and post-creep conditions for the area of reflex activity spatial distribution of the right erector spinae muscles (pre-creep: M = 1.14, SD = 0.32; post-creep: M = 1.05, SD = 0.31; p = 0.25) nor left sides (pre-creep: M = 1.22, SD = 0.40; post-creep: M = 1.14, SD = 0.32; p = 0.37).

[Insert Table 2 around here]

Experimental pain condition

The mean temperature needed to induce experimental low back pain was 48 °C (SD = 0.9). During the experimental pain condition, participants scored their perceived low back pain with an average of 43/100 (SD = 7.5).

Erector spinae EMG reflex amplitude significantly decreased across trunk perturbation trials, regardless of the reported level of pain (the adaptation effect was only significant on one side, p = 0.04, Table 3). Most of the other dependent variables did not significantly change during the experimental pain condition (Table 3). Dependent *t*-tests revealed no significant difference in the pain condition versus the condition without pain for the spatial distribution of erector spinae reflex activity on both the right (without pain: M = 1.09, SD = 0.34; with pain: M = 1.18, SD = 0.51; p = 0.34) and left sides (without pain: M = 1.14, SD = 0.30; with pain: M = 1.14, SD = 0.35 ; p = 0.99).

[Insert Table 3 and Figure 3 around here]

Discussion

The present study investigated how superficial lumbar muscles adapt following a spinal tissue creep deformation and during experimental pain, while the neuromuscular system is challenged by a series of unexpected trunk perturbations. Despite an increase of pain perception during the pain condition, and an increase trunk ROM during the creep

condition, the study showed that most neuromuscular outcomes were similar in all conditions. Moreover, this study is the first one to show that neither experimental back pain nor creep deformation altered participants' ability to adapt across unexpected trunk perturbation trials.

Creep Effect

A 30-minute static trunk flexion was used to induce creep deformation in the lumbar passive structures. Previous studies have found that a static full trunk flexion sustained for a period of 5 to 20 minutes was enough to induce creep deformation (McGill and Brown 1992; Shin et al. 2009). Moreover, it has been shown that creep deformation can be induced using a static flexion between 70 and 75% of full-trunk flexion lasting 30 to 60 minutes (Abboud et al. 2016b; Sanchez-Zuriaga et al. 2010).

In the present study, an increase of 4% of trunk ROM in flexion was found following the creep deformation, which is similar to a previous study using a similar protocol (Sanchez-Zuriaga et al. 2010). By contrast, results from the current study showed that trunk kinematics following an unexpected perturbation did not change under the influence of creep deformation. Results from a recent study showed that trunk posture (neutral versus flexed) influences the level of muscle activation in the lumbar region when postural stability is challenged. When the trunk is flexed, a higher contribution of the passive system is observed (Maaswinkel et al. 2015). Sufficient stretching of passive structures within the elastic zone will trigger mechanoreceptor responses generating proprioceptive information, potentially improving sensory-motor control via appropriate and coordinated motor responses (Holm et al. 2002; Panjabi 1992a). It has been often proposed that

increasing length and tension in the passive structures is associated with an increase in muscular activation to maintain joint stability (ligamento-muscular reflex (Solomonow 2009; 2006)).

On the other hand, when tissue creep is present, a reduction of the force transmission capabilities of the musculotendinous units could occur (Solomonow et al. 1999). Therefore, it can be hypothesized that an alternative muscle activation strategy was implemented to minimize trunk displacement when it was challenged by an unexpected perturbation (see "Motor Adaptations" section).

In the current study, a slight increase of back muscle activity before the trunk perturbation onset was observed following the creep deformation, which supports the hypothesis that changes in muscle activity act as compensation mechanisms for spinal instability resulting from passive structure laxity (Solomonow et al. 1998). Nevertheless, once the perturbation is triggered, the back muscle activity does not change between pre- and post-creep conditions as observed in this study. Previous studies failed to identify changes in muscle reflex activity when trunk muscles are pre-activated before an unexpected postural perturbation (Stokes et al. 2000). Other studies suggested that increased trunk stiffness due to increased baseline activity leads to a reduction of muscle reflex activation (Granata and Rogers 2007; Shahvarpour et al. 2015). Moreover, the co-contraction of the trunk muscles (rectus abdominis, externus obliquus and erector spinae muscles) did not increase after the creep protocol. The absence of change in muscle activity reflex following the creep protocol concurred with the fact that the passive components contribution to stabilize the spine is negligible, especially in the neutral zone (Panjabi 1992b; Solomonow 2006). Therefore, it can be suggested that alteration of

461 passive components following a creep deformation, does not trigger changes in back
462 muscle activity needed to prevent spinal instability.
463
464 **Experimental Pain Effect**
465 Thermal stimulation has been previously used to activate selective nociceptive fibers
466 (Bosshard et al. 2015; Yeomans et al. 1996). Thermal cutaneous pain has also been
467 previously used to evoke acute LBP in healthy participants and produce neuromuscular
468 responses similar to the adaptations typically reported in patients with chronic LBP
469 (Dubois et al. 2011). In Dubois' study, the authors have observed a nociceptive
470 stimulation yielding painful evaluations by both LBP patients and healthy participants, as
471 well as typical increases in erector spinae muscle activity, often observed in patients with
472 various levels of clinical pain.
473 The present study showed that trunk neuromuscular responses were similar with or
474 without the presence of experimental back pain when the trunk was challenged by
475 unexpected perturbation. It has been suggested that despite neuromuscular changes
476 usually observed under the influence of experimental pain, the overall motor performance
477 remains unchanged (Bank et al. 2013). For instance, in a recent study, it has been shown
478 that pain did not interfere with global performance (movement errors) while participants
479 walked on a treadmill at a control speed while facing a perturbation at the ankle
480 (Bouffard et al. 2016). This could explain the absence of trunk kinematic alteration
481 following an unexpected perturbation under the influence of pain. This behavior concurs
482 with the minimal intervention principle, which states that the irrelevant aspects of a motor
483 task should be left uncorrected in order to improve the resulting performance (Todorov

484 and Jordan 2002). Therefore, it can be hypothesized that trunk movements triggered by
485 the perturbations were too small to challenge spinal stability, and that consequently no
486 trunk kinematic adjustment was needed to optimize the neuromuscular system. As
487 mentioned earlier, the magnitude of the perturbation was similar to the one used in other
488 similar trunk perturbation protocols (Abboud et al. 2016c; Radebold et al. 2000).
489 The presence of experimental back pain did not modify the erector spinae baseline
490 activity. This observation is consistent with previous research (Boudreau et al. 2011;
491 Gregory et al. 2008; Miller et al. 2013). Moreover, in a recent review, it has been
492 proposed that pain has a negligible effect on the muscle experiencing it when it is at rest
493 (Bank et al. 2013). While lumbar muscle reflex latency is longer in patients with chronic
494 low back pain (Abboud et al. 2016a), the current study showed no difference with
495 experimental back pain. Moreover, no change was found in EMG reflex responses. These
496 observations may be surprising, since under the influence of experimental/acute pain, as
497 proposed by the pain adaptation model (Lund et al. 1991), inhibition of agonist muscles is
498 commonly described (Bank et al. 2013). However, inhibition of agonist muscles is not
499 systematically observed in pain conditions. Hodges et al. described no consistent pattern
500 of trunk muscle activity (flexor and extensor) adaptation under the influence of
501 experimental back pain (Hodges et al. 2013). Moreover, the absence of EMG reflex
502 change observed in the current study is in line with another study's findings, which used
503 similar perturbation protocols in participants with acute low back pain (Gregory et al.
504 2008). These authors also observed an increase of trunk muscle co-contractions. It has
505 been recently suggested that an increase in trunk stiffness was also present with
506 experimental pain, and was correlated to a slight increase of trunk muscles co-contraction

(Wong et al. 2016). Since spinal stiffness has been associated with spinal stability (Graham and Brown 2012), it could be suggested that the redistribution of muscle activity is enough to maintain spinal stability under the influence of experimental back pain. Moreover, since no increase in abdominal muscle activity was observed in the current study while participants were submitted to experimental back pain, it could be hypothesized that the redistribution of muscle activity occurred within the erector spinae muscle.

Motor Adaptations

To our knowledge, the current study is the first one investigating how erector spinae muscles adapt across a series of perturbation trials before and after a creep deformation protocol, as well as with and without experimental low back pain. Our results showed that trunk kinematic remained constant across perturbation trials. Since participants did not change their maximal velocity following an unexpected trunk perturbation, it can be suggested that no modification of the time to peak velocity was necessary to maintain stability. This strategy was different from our previous observations, where participant took less time to stop their trunk while their maximal velocity reduced across perturbation trials (although not significant $p = 0.07$) (Abboud et al. 2016c). These differences reflect the important motor redundancy provided by the trunk system to achieve a similar goal and serve as a note of caution regarding results generalizability when studying adaptation process in highly redundant motor systems.

As expected, without the influence of creep deformation or experimental back pain, a clear attenuation of back muscle activity was found through the repetition of the same

530 unexpected trunk perturbation. This suggests that the CNS is able to modulate the trunk
531 neuromuscular responses based on a previous postural perturbation experience. However,
532 when two experiences of the same unexpected trunk perturbation are partitioned by a new
533 external condition, such as experimental pain or creep deformation, this ability is altered.
534 It can be suggested that the learning process used by the CNS is partly reset under such
535 circumstances. On the other hand, when a second set of the same unexpected trunk
536 perturbation is conducted following a rest period, the CNS is still able to use the previous
537 perturbation experience to adapt the motor output (Figure 3). Indeed, participants
538 continued to reduce their EMG reflex amplitude in the second series of perturbation trials
539 in the control group. This observation confirms that the absence of erector spinae EMG
540 reflex attenuation across perturbation trials under the influence of muscle fatigue is a
541 consequence of muscle fatigue, and not a leaning effect (Abboud et al. 2016c).
542 Interestingly, despite a higher EMG reflex value between the last perturbation trials of the
543 control condition (without pain or before creep) versus the first perturbation trials of the
544 experimental condition (pain or creep), adaptations of EMG reflexes were also observed
545 under these two experimental conditions, suggesting that participants are partially able to
546 use across-trial redundancy to adapt their neuromuscular responses. In a recent study, it
547 has been shown that the attenuation of back muscle activity across perturbation trials was
548 limited by the influence of muscle fatigue (Abboud et al. 2016c). Under the influence of
549 muscle fatigue, the spinal active muscle system is altered (e.g. motor unit frequency
550 discharge) (Gandevia 2001; Taylor et al. 2016) and a migration of lumbar muscle activity
551 occurs during a fatigue task (Abboud et al. 2014; Tucker et al. 2009). This suggests that
552 back muscle fatigue reduces the number of available motor solutions to execute a given

553 motor task, which could limit the ability of the CNS to use alternative motor strategies. In
554 the current study, the spatial distribution of muscle activity was similar before and after
555 the creep deformation, as well as with and without experimental back pain. Adaptation in
556 muscle activity distribution within different regions of the erector spinae could be
557 associated with changes in motor units control in this muscle. Unlike muscle fatigue,
558 creep deformation does not modify motor units number availability, which may increase
559 the motor solutions number to achieve a desired goal. Nevertheless, when creep
560 deformation and back muscle fatigue are combined, muscle activity spatial distribution is
561 lower than the one observed during back muscle fatigue only (Abboud et al. 2016b). It
562 can be hypothesized that when passive components of the spine are the only altered
563 stabilization structures, trunk muscles are able to compensate for the loss, while when
564 both of these components are altered, trunk motor redundancy is reduced, limiting the
565 number of motor solutions available to stabilize the spine when it is challenged. As for
566 experimental pain, a similar level of variability, with or without pain, concurs with the
567 model proposed by Madeleine, suggesting that acute pain leads to an increase of motor
568 variability (Madeleine 2010). While these adaptations may have short-term benefit, in the
569 long-term, these changes in neuromuscular control may have negative consequences on
570 sensory-motor control (van Dieen et al. 2017). Based on the findings of the present study,
571 it seems reasonable to suggest that using the trunk system's redundancy, the CNS is able
572 to adapt neuromuscular responses to generate proper spinal stability based on a previous
573 experience, even with the presence of experimental back pain or after a creep
574 deformation.
575

Limitations

Potential limitations include the use of that thermal cutaneous pain that may not exactly reflect clinical musculoskeletal pain. Experimental muscle pain can also be generated using intramuscular injections of hypertonic saline into muscles. However, there is some evidence suggesting that hypertonic saline can excite other motor axons (Kumazawa and Mizumura 1977; Weerakkody et al. 2003), which may alter the sensorimotor control independently from pain effects. Moreover, EMG activity from the injection site may be altered to avoid pain provocation from contraction. This effect is important because the source of the non-specific low back pain, which represents the majority of low back pain, does not necessarily originate from muscle pain. Another limitation of the current study is the absence of direct spinal tissue creep measurements, and the fact that the protocol used to induce spinal tissue creep has not yet been proven to generate passive tissue creep. During the creep protocol, participants were asked to bend forward to a posture of approximately 75% of their maximum ROM, while bracing their upper body on a table. This position might not have caused sufficient tension on posterior passive tissues of the spine. Nevertheless, previous studies showed alteration of trunk responses using similar creep protocol (Abboud et al. 2016b; Sanchez-Zuriaga et al. 2010). Furthermore, one of the assessors was present during the entire creep protocol in order to verify that participants stayed in the same position. Indirect evidence, such as dose-response relationship, also support the use of prolonged trunk flexion to create changes in passive supporting spine structures. It has been shown that creep deformation in the lumbar region increased with increasing exposure duration (Bazrgari et al. 2011; Muslim et al. 2013). In these studies, the authors used prolonged trunk flexion from 1 to 10 minutes to

induce creep deformation and observed a significant increase of trunk flexion range of motion following the longer creep deformation protocol. Another limitation is the potential occurrence of sequence effects. A sequence effect was present across trunk perturbation trials in each conditions (creep, pain). However, in order to minimize possible order effects of conditions, half of the participants started with the creep protocol, and the other half started with the experimental pain protocol. Finally, a methodological consideration of the current study was the time window chosen to determine whether the trunk responses to a perturbation were reflex or voluntary activation. Based on a recent systematic review, it was pointed out that the authors disagreed on what should be considered as reflex responses (shorter than 120 ms to 300 ms) or voluntary movements (Abboud et al. 2016a). Future studies should focus on determining standard latency values for trunk muscles.

Conclusion

The results of the current study suggest that the short-term effect on trunk neuromuscular control of creep deformation as well as experimental back pain was negligible. Moreover, similar neuromuscular adaptations between experimental back pain, creep deformation and control conditions were present across the repetition of the same unexpected perturbation of the trunk. Finally, this study showed that the CNS chose to adopt a variable recruitment pattern of back muscle activation to face trunk perturbations under the influence of creep deformation or experimental back pain. It could be suggested that this strategy helped participants adapt across perturbation trials while maximizing the motor performance.

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Disclosures

The authors declare that the research was conducted in the absence of any commercial or financial relationships that could be construed as a potential conflict of interest.

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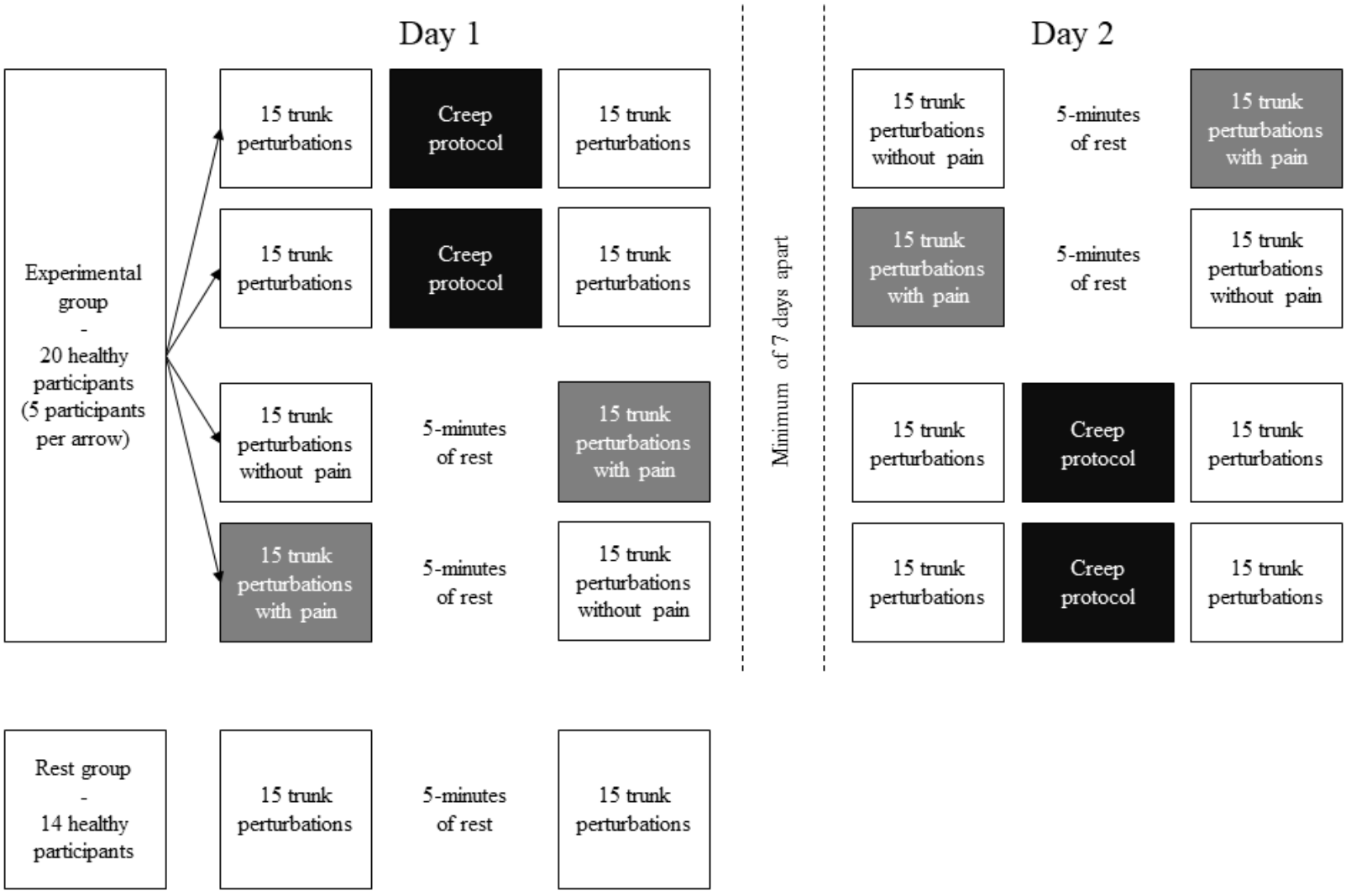
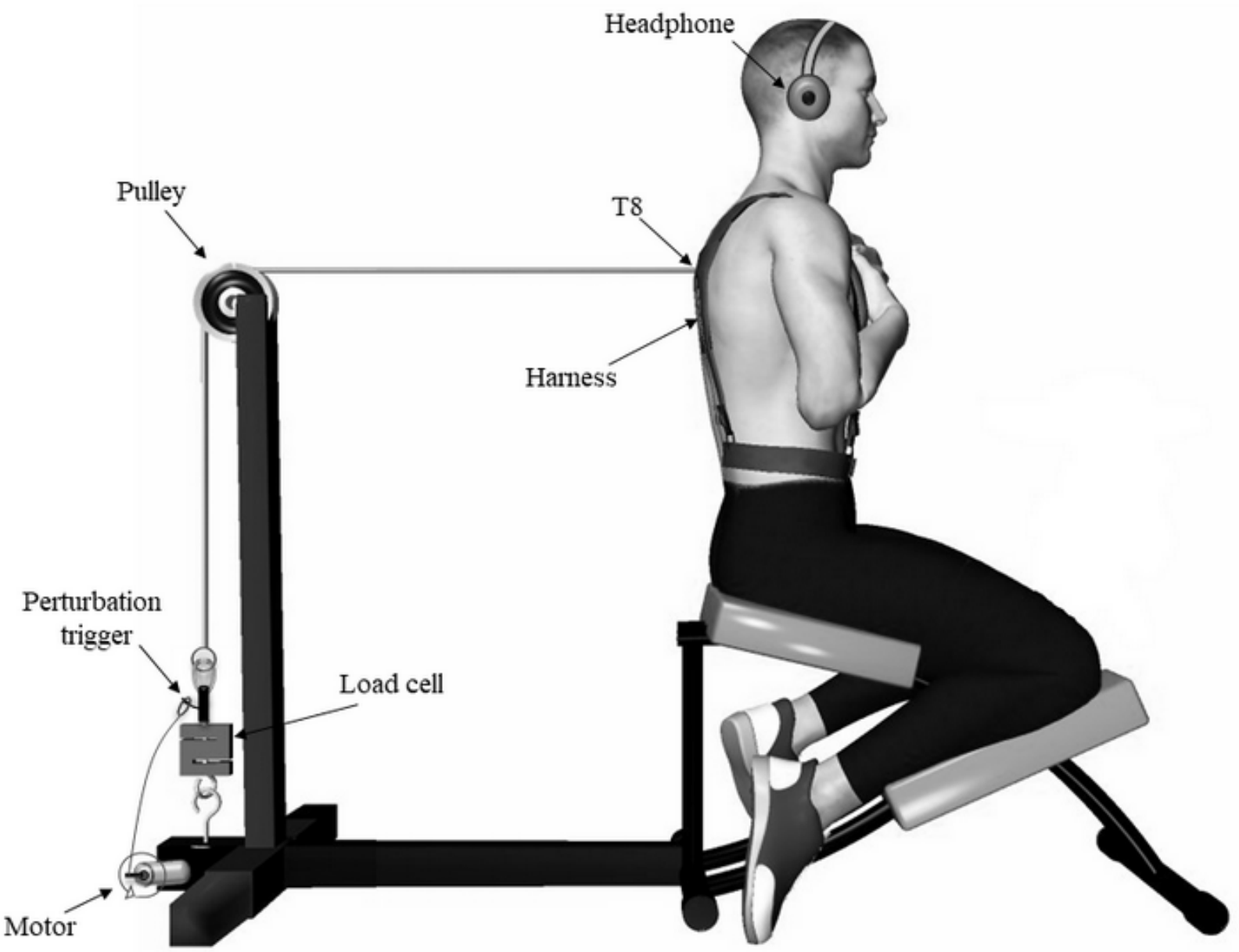
Figure legends

Figure 1. Timeline of the experimental protocol

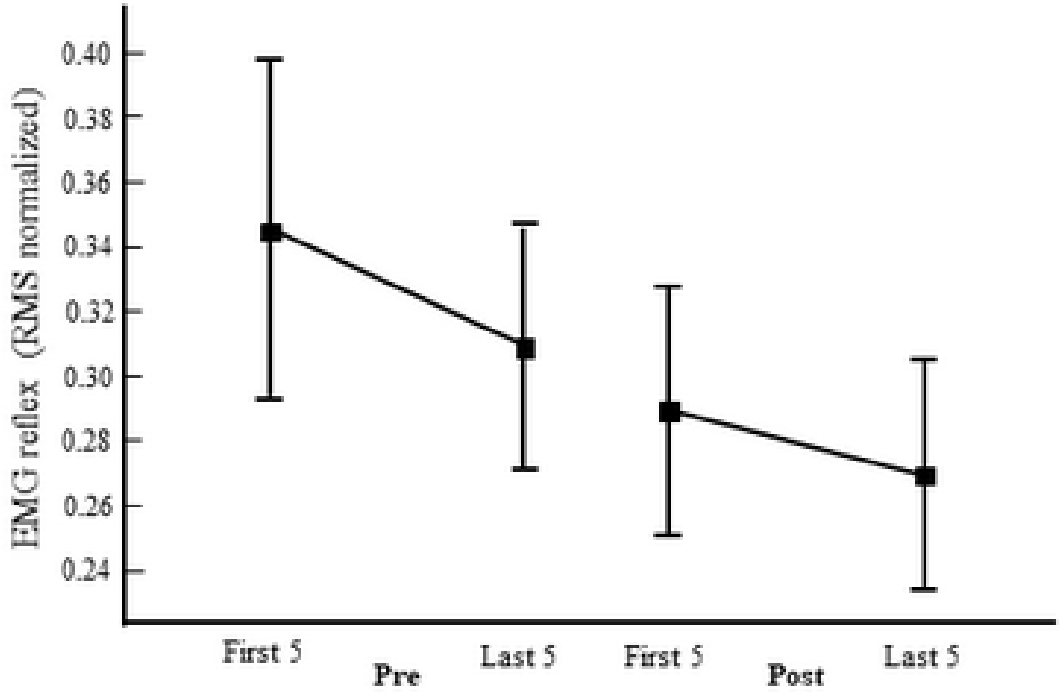
Figure 2. Illustration of the perturbation protocol

Figure 3. Mean erector spinae EMG reflex amplitude results on the left side for all conditions

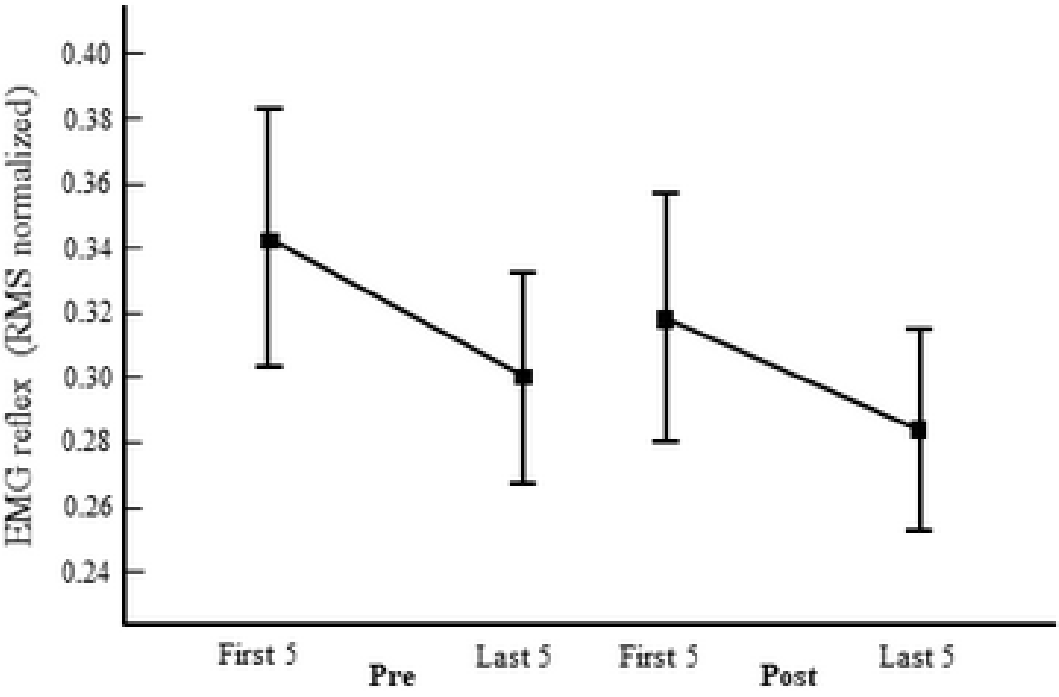

Day 1
Day 2
Experimental group - 20 healthy participants (5 participants per arrow)
15 trunk perturbations
Creep protocol
15 trunk perturbations
15 trunk perturbations
Creep protocol
15 trunk perturbations
15 trunk perturbations without pain
5-minutes of rest
15 trunk perturbations with pain
15 trunk perturbations with pain
5-minutes of rest
15 trunk perturbations without pain
Minimum of 7 days apart
15 trunk perturbations without pain
5-minutes of rest
15 trunk perturbations with pain
15 trunk perturbations with pain
5-minutes of rest
15 trunk perturbations without pain
15 trunk perturbations
Creep protocol
15 trunk perturbations
15 trunk perturbations
Creep protocol
15 trunk perturbations
Rest group - 14 healthy participants
15 trunk perturbations
5-minutes of rest
15 trunk perturbations


Headphone
Pulley
T8
Harness
Perturbation trigger
Load cell
Motor

Control



Creep



Pain

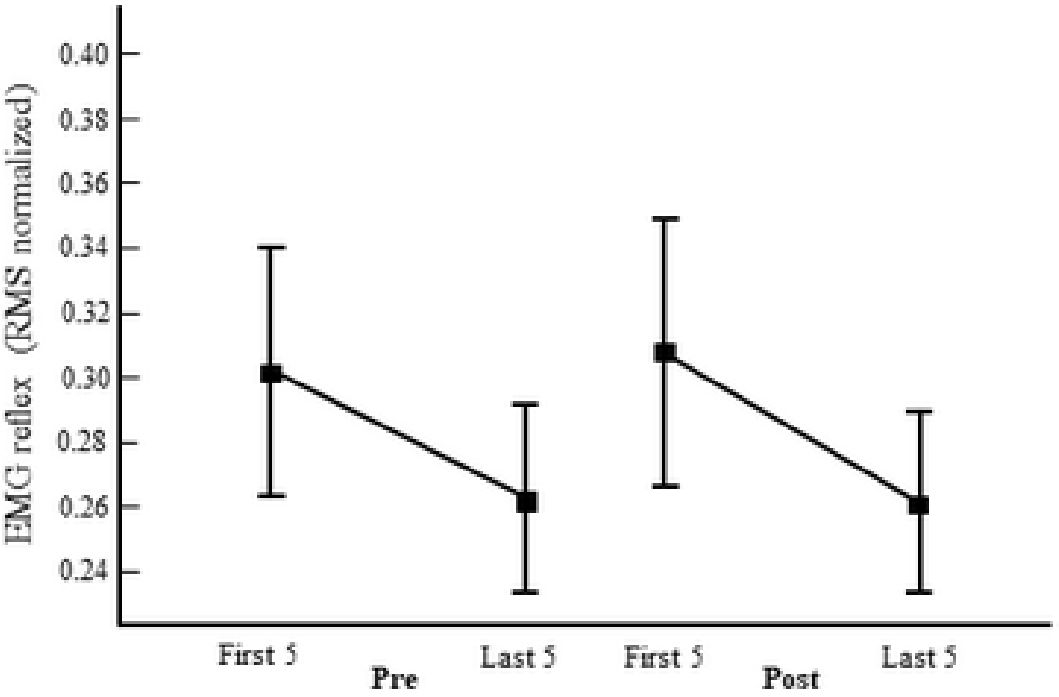


Table 1. Mean and (SD) values of all dependent variables for the control group (L: left side of the erector spinae; R: right side of the erector spinae; RA: rectus abdominis; OE: obliquus externus).

			First 5 trials mean	Last 5 trials mean	*p**	
					Rest	Adaptation
Flexion angle (°)		Pre-rest	4.8 (2.8)	4.7 (2.6)	$p = 0.23$	$p = 0.98$
		Post- rest	5.0 (3.2)	5.1 (2.7)		
Peak velocity (°/s)		Pre-rest	19 (7)	19 (7)	$p = 0.45$	$p = 0.67$
		Post- rest	19 (7)	20 (7)		
Time to peak velocity (ms)		Pre-rest	198 (67)	209 (75)	$p = 0.41$	$p = 0.10$
		Post- rest	212 (80)	216 (74)		
Baseline activity (% MVC)	L	Pre-rest	9 (4)	8 (3)	$p = 0.80$	**$p = 0.04$**
		Post- rest	9 (4)	8 (3)		
	R	Pre-rest	10 (5)	9 (5)	$p = 0.26$	**$p = 0.03$**
		Post- rest	10 (5)	10 (5)		
Reflex latency (ms)	L	Pre-rest	129 (33)	128 (24)	$p = 0.10$	$p = 0.79$
		Post- rest	131 (30)	135 (28)		
	R	Pre-rest	127 (35)	124 (20)	$p = 0.99$	$p = 0.80$
		Post- rest	125 (24)	127 (25)		
Erector spinae EMG reflex amplitude (% MVC)	L	Pre-rest	35 (20)	31 (14)	**$p = 0.03$**	$p = 0.06$
		Post- rest	29 (14)	17 (13)		
	R	Pre-rest	35 (14)	33 (15)	$p = 0.12$	**$p = 0.03$**
		Post- rest	32 (13)	30 (12)		
Abdominal EMG reflex amplitude (% MVC)	RA	Pre-rest	10 (10)	8 (7)	**$p = 0.03$**	$p = 0.07$
		Post- rest	8 (8)	8 (7)		
	OE	Pre-rest	10 (6)	9 (4)	**$p = 0.02$**	**$p = 0.03$**
		Post- rest	9 (5)	8 (4)		

*p** based on the repeated measure ANOVA

Table 2. Mean and (SD) values of all dependent variables before and after the creep protocol (L: left side of the erector spinae; R: right side of the erector spinae; RA: rectus abdominis; OE: obliquus externus).

			First 5 trials mean	Last 5 trials mean	*p**	
					Creep	Adaptation
Flexion angle (°)		Pre-creep	5.8 (3.7)	5.3 (3.4)	$p = 0.10$	$p = 0.60$
		Post-creep	6.1 (3.8)	6.2 (4.3)		
Peak velocity (°/s)		Pre-creep	20 (11)	18 (7)	$p = 0.10$	$p = 0.38$
		Post-creep	20 (8)	19 (7)		
Time to peak velocity (ms)		Pre-creep	196 (58)	196 (60)	$p = 0.16$	$p = 0.59$
		Post-creep	208 (61)	198 (52)		
Baseline activity (% MVC)	L	Pre-creep	10 (7)	9 (7)	$\mathbf{p = 0.04}$	$\mathbf{p = 0.003}$
		Post-creep	11 (7)	10 (7)		
	R	Pre-creep	10 (8)	9 (8)	$p = 0.47$	$\mathbf{p = 0.01}$
		Post-creep	10 (7)	10 (6)		
Reflex latency (ms)	L	Pre-creep	114 (14)	115 (23)	$p = 0.49$	$p = 0.65$
		Post-creep	115 (15)	117 (14)		
	R	Pre-creep	116 (17)	114 (15)	$p = 0.82$	$p = 0.70$
		Post-creep	115 (16)	116 (15)		
Erector spinae EMG reflex amplitude (% MVC)	L	Pre-creep	34 (18)	30 (15)	$p = 0.10$	$\mathbf{p = 0.008}$
		Post-creep	32 (17)	28 (14)		
	R	Pre-creep	36 (17)	31 (16)	$p = 0.09$	$\mathbf{p \leq 0.001}$
		Post-creep	33 (15)	29 (12)		
Abdominal EMG reflex amplitude (% MVC)	RA	Pre-creep	14 (13)	13 (13)	$p = 0.55$	$p = 0.12$
		Post-creep	14 (14)	12 (12)		
	OE	Pre-creep	15 (15)	15 (18)	$p = 0.22$	$p = 0.07$
		Post-creep	15 (13)	11 (9)		

*p** based on the repeated measure ANOVA

Table 3. Mean and (SD) values of all dependent variables before and during experimental pain (L: left side of the erector spinae; R: right side of the erector spinae; RA: rectus abdominis; OE: obliquus externus).

			First 5 trials mean	Last 5 trials mean	*p**	
					Pain	Adaptation
Flexion angle (°)		Without pain	5.7 (3.9)	5.2 (2.9)	$p = 0.81$	$p = 0.54$
		With pain	5.5 (2.7)	5.6 (2.9)		
Peak velocity (°/s)		Without pain	20 (10)	18 (8)	$p = 0.60$	$p = 0.47$
		With pain	18 (7)	19 (8)		
Time to peak velocity (ms)		Without pain	206 (67)	208 (64)	$p = 0.47$	$p = 0.89$
		With pain	202 (77)	199 (65)		
Baseline activity (% MVC)	L	Without pain	8 (5)	8 (4)	$p = 0.87$	$p = 0.35$
		With pain	8 (4)	8 (4)		
	R	Without pain	9 (6)	9 (6)	$p = 0.85$	$p = 0.16$
		With pain	9 (6)	9 (6)		
Reflex latency (ms)	L	Without pain	119 (22)	121 (24)	$p = 0.07$	$p = 0.10$
		With pain	121 (25)	128 (28)		
	R	Without pain	122 (26)	128 (32)	$p = 0.53$	$p = 0.20$
		With pain	123 (31)	123 (25)		
Erector spinae EMG reflex amplitude (% MVC)	L	Without pain	30 (17)	26 (13)	$p = 0.85$	$\mathbf{p = 0.04}$
		With pain	31 (18)	26 (13)		
	R	Without pain	31 (16)	30 (15)	$p = 0.83$	$p = 0.08$
		With pain	34 (21)	28 (14)		
Abdominal EMG reflex amplitude (% MVC)	RA	Without pain	15 (16)	14 (14)	$p = 0.90$	$p = 0.07$
		With pain	16 (15)	14 (14)		
	OE	Without pain	13 (10)	11 (8)	$p = 0.59$	$\mathbf{p = 0.02}$
		With pain	13 (10)	11 (10)		

*p** based on the repeated measure ANOVA