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Experimentally induced neck pain causes a decrease in thoracic but not lumbar spine stability

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ABSTRACT

Maintenance of spine stability is considered to be a critical component of spine health. Ross et al. (2015) used a topical capsaicin/heat pain sensitization model to experimentally induce lower back pain, and demonstrated that the experimental pain experience caused a decrease in the muscular contribution to lumbar spine rotational stiffness (related to mechanical stability) as well as lower back local dynamic stability (LDS). It has yet to be established if pain elsewhere in the body, specifically in other regions of the spine, can similarly affect the stability of the lower back. The purpose of this investigation was therefore to quantify thoracic and lumbar spine LDS as well as the muscular contribution to lumbar spine rotational stiffness after an experimental neck pain protocol. Results demonstrated that LDS of the thoracic spine decreased in response to the capsaicin/heat induced neck pain. Limited adaptation was required at the lumbar spine as demonstrated by the lack of statistically significant changes in lower back LDS or rotational stiffness.

1. Introduction

Lower back pain (LBP) is most commonly diagnosed as non-specific (Koes et al., 2006) and the etiology and consequences of LBP are far from fully understood. Since the isolated ligamentous lumbar spine buckles with a compressive load of less than 90 N (Crisco et al., 1992), the maintenance of spine stability has been conceptualized to be a critical component of spine health. It is believed that disruption in the function of the passive, active or control (neurological) systems of the spine can lead to instability which can cause, or be a consequence of, injury or pain (Panjabi, 1992). A transient loss in a vertebral segment's stability can conceivably cause unexpected and uncontrolled displacements that could irritate nociceptors or free nerve endings of surrounding tissues or cause buckling (Cholewicki and McGill, 1996). In response, the active and control systems work to counteract these displacements, but at the risk of overloading or injuring muscles and, over time, causing cumulative overload of the spine itself. Evidence strongly suggests that the possibility of spine instability causing injury and pain has biological plausibility (Preuss and Fung, 2005).

LBP patients are often studied in an attempt to uncover differences compared to healthy controls. The caveat to studying LBP patients is that LBP is heterogeneous such that the cause of pain can vary and cannot be reliably diagnosed. It is reasonable that different pathologies would be associated with spine (in)stability in different ways. Therefore studying a homogenous sample population offers advantages for uncovering specific mechanistic causes or consequences of pain and injury. This can be facilitated by inducing pain in healthy participants through a common stimulus using experimental models (e.g. Arendt-Nielsen et al., 1996; Hodges et al., 2003; Adamczyk et al., 2018). Recent research has used a topical capsaicin/heat pain sensitization model to experimentally induce lower back pain, and demonstrated that the experimental pain experience caused a decrease in the muscular contribution to lumbar rotational stiffness (related to mechanical stability) as well as lower back local dynamic stability (LDS) (Ross et al., 2015). It has yet to be established if pain elsewhere in the body, specifically in other regions of the spine, can similarly affect the stability of the lower back.

Joints of the body are interlinked through anatomical structure and hence function. They work in a coordinated fashion to perform movement. The function of each segment/joint can directly influence the demand and hence the function of the segments/joints adjacent to it. For example, surgical fusions that provide strong mechanical restriction to individual vertebral segments are

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associated with degenerative changes at adjacent vertebral segments (Mannion et al., 2014). Even in the case where minimal mechanical restriction is applied to the lower back, for example using a liquid bandage to cue a change in motion at a single vertebral segment, motion is altered at adjacent segments (Beaudette et al., 2018). Further, restriction of the whole lower back using an orthopedic brace, (Wu et al., 2014) or using tactile feedback to cue an individual to restrict lumbar motion (Pinto et al., 2018), can influence the motion at adjacent joints such as the hips and knees. Restricting joints that are more distal, such as the ankle, can influence lumbar spine mechanics, as it can influence the entire kinetic chain (Beach et al., 2014). Thus the mechanics of the lower back can influence the segments/joints adjacent to it, and vice versa. This served as the motivation to investigate whether pain in one region of the spine (neck) could influence the stability of more caudal spine regions.

Neck pain is the fourth leading cause of disability in the United States of America, ranked behind LBP which is the first (Murray et al., 2013). The incidence of neck pain is strongly associated with LBP and those with pain in one of these regions (neck or low back) are fifteen times more likely to report pain in the other region (Fernández-de-las-Peñas et al., 2011). Since the causes of LBP are yet to be fully understood, understanding how the pain or injury in other regions of the spine, such as the neck, can influence the risk of injury at the lower back is of utmost importance.

The purpose of this investigation was therefore to quantify thoracic and lumbar spine LDS as well as the muscular contribution to lumbar spine rotational stiffness after an experimental neck pain protocol. It was hypothesized that experimentally induced neck pain would cause LDS and stiffness to be reduced compared to baseline, as in Ross et al. (2015). Further, it was hypothesized that the decrease in LDS would be larger in the thoracic compared to the lumbar spine, due to the thoracic region's proximity to the site of pain.

2. Materials and methods

2.1. Participants

Twenty one healthy males (mean ± standard deviation: age 26 ± 4.7 years; mass 83.6 ± 25.1 kg; height 178.7 ± 5.1 cm) were recruited. Exclusion criteria were any lower back or neck pain or injury within the last year, or allergies to adhesives or gels. Upon arrival, participants completed a general health questionnaire asking them about previous and current pain and injury. All procedures were approved by the University Research Ethics Board.

2.2. Equipment

Skin overlaying the abdominal and back muscles of interest was cleaned with alcohol and shaved if necessary. Surface EMG electrodes (Ambu Blue Sensor, Medicotest Inc., Olstykke, Denmark) were applied bilaterally to the skin overlaying the latissimus dorsi (LD), thoracic erector spinae (TES), lumbar erector spinae (LES), external oblique (EO), internal oblique (IO) and rectus abdominis (RA) (Fig. 1). Ground electrodes were placed bilaterally over the acromion processes. Three maximal voluntary contractions (MVCs) for each of these muscle groups were performed isometrically against the resistance, similar to previously published methods (Vera-Garcia et al., 2010).

Electromagnetic sensors (Polhemus Liberty, Polhemus, Colchester, VT, USA) were affixed with double sided tape to the skin overlaying the first sacral vertebra (S1), twelfth thoracic vertebra (T12), third thoracic vertebra (T3), and the occiput using a headband (Fig. 1). Participants then performed a quiet standing trial, maximum spine flexion/extension range of motion (ROM) trial, and a static 5 s hold with the hip flexed and trunk angle at approximately 45° holding a 10 kg mass. The maximum flexion/extension ROM and 45° trunk flexion trials were used to account for variability amongst individuals when using the rotational stiffness model (described later).

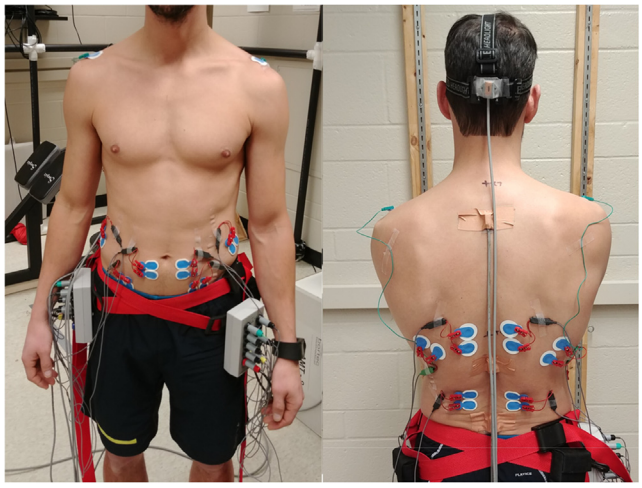

Fig. 1. Equipment set up showing placement of EMG electrodes (blue stickers) and electromagnetic sensors. (For interpretation of the references to color in this figure legend, the reader is referred to the web version of this article.)

EMG signals were differentially amplified (AMT-16, Bortec Biomedical, Calgary AB, bandwidth 10–1000 Hz; CMRR = 115 dB at 90 Hz; input impedance = 10 GΩ) and recorded at 2048 Hz simultaneously to the direction cosines of the electromagnetic sensors recorded at 32 Hz.

2.3. Protocol

A total of eight trials (4 baseline (B), 4 pain (P); Fig. 2), each consisting of 38 repetitions of continuous spine flexion/extension, were performed at a pace of 0.25 Hz (4 s per flexion/extension cycle). Data collection started after the first three cycles (35 flexion/extensions were recorded per trial). Participants were required to repeatedly touch a target at chest height and a target at knee height with arms extended (Ross et al., 2015). To ensure each movement predominantly occurred through the spine, hip motion was constrained by strapping the posterior of the pelvis to an adjustable-height table. Before each trial, participants completed a 10 cm visual analog scale (VAS) indicating how much pain they were experiencing in their neck, from no pain to the worst pain imaginable. Participants rested for at least five minutes between each trial except during the pain administration protocol (between B4 and P1; Fig. 2) where they rested on a massage bench for a minimum of 15 min. Careful consideration was taken to monitor and question each participant about his perceived state of fatigue before and after each trial. Feet position, the adjustable table height (hip constraint) and target heights were kept consistent between each trial for each participant.

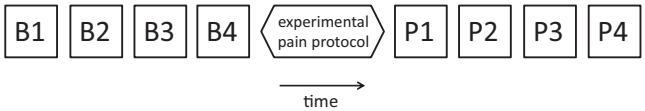

Fig. 2. Pictorial representation of protocol. B1, B2, B3 and B4 refer to the first four baseline trials. P1, P2, P3 and P4 refer to the four trials after the pain protocol. VAS pain reports were acquired before B1, B2, B3, B4, P1, P2, P3 and P4.

After the first four flexion/extension trials (baseline trials: B1-B4, Fig. 2), the participant lay prone on a massage table and the neck region between the participant's hairline and C7 was pre-heated with moist towels heated with 40 °C water for 5 min. Towels were replaced every 30 s. Next, topical capsaicin (0.075%; Zostrix, Medicis, Toronto, ON, Canada) was applied to the neck region between the participant's hairline and C7 (superior and inferior borders) and the sternocleidomastoid muscles (lateral borders). The cream was rubbed in and allowed to soak for five minutes. After this period a moist towel, heated with 40 °C water, and a hot gel pack were applied for five minutes. Participants then performed another four flexion/extension trials (pain trials: P1-P4; Fig. 2).

2.4. Data analysis

Direction cosines were used to calculate relative 3D Cardan angles between sensors using a flexion-extension, lateral bend and axial twist sequence for the lumbar spine (T12 to S1), thoracic spine (T3 to T12) and neck (back of the skull to T3). However, the sensor attached to the back of the skull routinely exceeded the validated range of the sensor (i.e. traveled too far from the electromagnetic source) and therefore these data were not considered reliable and the neck data will not be reported. Data were low-pass filtered using a 10 Hz dual-pass effective fourth order Butterworth filter and standing bias was removed by subtracting angles calculated from the quiet standing trial.

Local dynamic stability (LDS) for both the thoracic and lumbar regions of the spine was estimated using the maximum finite-cycle Lyapunov exponent (λ_{max}). To do so, the first five cycles of the calculated 3D angles were removed and the 30 remaining cycles were time normalized to 3840 frames thereby maintaining any temporal cycle-to-cycle variability. Angles were shifted to positive values and the sum of squares was calculated to account for kinematic fluctuations in all three dimensions (Beaudette et al., 2016). A state space with six dimensions (selected by a global false nearest neighbours analysis (Kennel et al., 1992)) was reconstructed using the following method of delays:

$$Y(t) = [N(t), N(t+Td), N(t+2Td), \cdots, N(t+(n-1)Td)] \quad (1)$$

where $Y(t)$ is the n-dimensional state space, $N(t)$ is the sum of squares data, T_d is the constant time delay set to 13% (determined through time series autocorrelation analysis) (Rosenstein et al., 1994), and n is the number of reconstructed dimensions. Maximum finite-cycle Lyapunov exponents were then calculated by analyzing the exponential rate of divergence of neighboring trajectories in the reconstructed state space, using the equation from Rosenstein et al. (1993):

$$y(i) = \frac{1}{\Delta t}\{\ln dj(i)\} \quad (2)$$

where $\{\ln d_j(i)\}$ represents the average logarithmic divergence ($d_j(i)$), for all pairs of nearest neighbours (j), throughout a certain number of time delays (Δt). λ_{max} was estimated using the slope of the linear line of best fit of the average logarithmic rate of divergence, for 0–0.5 movement cycles. Note that a lower λ_{max} indicates a higher LDS (more stable movement pattern).

EMG data were processed by first removing any bias from the signals and then low-pass filtered using a 500 Hz dual-pass effective fourth order Butterworth filter. EMG data were then full-wave rectified and low-pass filtered, using a 2.5 Hz second order Butterworth filter, to create a linear envelope. This was then normalized to the participant's respective linear enveloped maximum MVC. The normalized linear enveloped EMG was down-sampled to 32 Hz to match the kinematic data.

Both lumbar spine angles and normalized linear enveloped EMG (12 muscle sites as described earlier) were used to determine the muscular contributions to lumbar spine rotational stiffness, using an anatomically detailed EMG-driven model representing 58 muscle lines of action crossing the L4/L5 joint (Brown and McGill, 2010). Model kinematics consist of 18 degrees of freedom (three rotational degrees of freedom at each joint from L4/L5 superiorly to T12/L1). Activations of muscles which are too deep to record from using surface EMG were estimated as follows: multifidus from lumbar erector spinae, quadratus lumborum from lumbar erector spinae, psoas major from internal oblique (McGill et al., 1996; Brown & Potvin, 2007). Muscle force was modeled as:

$$F_m = NEMG_m \times PCSA_m \times \sigma_m \times l_m \times \nu_m \times G \quad (3)$$

where F_m is the force produced by muscle (m) about its line of action (in Newtons), $NEMG_m$ is the normalized EMG signal for each muscle (% MVC), $PCSA_m$ is the physiological cross-sectional area of each muscle (cm^2), σ_m is the stress generated by each muscle (set at 35 N/cm^2), l_m is the length coefficient of each muscle (unitless), ν_m is the velocity coefficient of each muscle (unitless), and G is the participant-specific calibration gain (unitless). Muscle force-length and force-velocity coefficients were adapted from McGill and Norman (1986). To accommodate for inter-participant variance (e.g. differences in physiological cross-sectional areas, stress generated by the muscles, moment arms) as well as any muscles not modeled by the current analysis, a participant-specific gain factor (G) was calculated by matching the L4/L5 moment calculated by inverse dynamics using 3DSSPP (Centre for Ergonomics, University of Michigan, Ann Arbor, MI) during the static 45° loaded trunk flexion trial and the moment estimated by the EMG-driven model. The mean ± standard deviation of the gain factors calculated for the 20 participants in the current study were 2.9 ± 0.8.

The muscular contributions to lumbar spine rotational stiffness were calculated about three orthogonal anatomical axes (flexion/extension, lateral bend, axial twist) at the L4/5 joint, as per Potvin and Brown (2005):

$$S_z = \sum_{m=1}^{58} F_m \left[\frac{A_X B_X + A_Y B_Y - r_z^2}{l} + \frac{q r_z^2}{L}\right]_m \quad (4)$$

where S_z is the rotational stiffness about the flexion/extension (medio-lateral) axis of the L4/L5 joint, F_m is muscle force (N), l is the 3D length of the muscle vector that crosses L4/L5 (between node points on either side of the L4/L5 joint for muscles that have curvature), L is the full 3D length of the muscle, r_z is the moment arm of the muscle force vector about the flexion/extension axis, A_X *and* A_Y (x and y axes are the antero-posterior and vertical axes, respectively) are the origin coordinates with respect to the L4/L5 joint at (0,0,0) m, B_X *and* B_Y are the initial deflection or insertion (without deflection points) coordinates with respect to L4/L5, and q is the stiffness gain relating muscle force and length to stiffness (value of 10 used (Cholewicki and McGill, 1995; Crisco and Panjabi, 1991)). Rotational stiffness was also calculated about lateral bend and axial twist axes by appropriate substituting of coordinates. Rotational stiffness values were separated into the same 30 individual cycles as used for the Lyapunov analysis. Maximum, mean, and minimum values were extracted for each cycle and then averaged across cycles within each trial for statistical analysis (Ross et al., 2015).

2.5. Statistics

To analyze the effect of the experimental pain protocol, one-way repeated measures ANOVAs were conducted on the average of the last three baseline trials (average of B2, B3 and B4), to account for any potential variability during baseline performance,

compared to each of the four trials after the experimental pain protocol (P1, P2, P3 and P4). The first baseline trial (B1) was not included in this average, as it was considered a learning or practice trial. The independent variable was condition (Baseline, P1, P2, P3 and P4) and the dependent variables were λ_{max} or maximum, mean or minimum rotational stiffness about each axis (flexion/extension, lateral bend, axial twist) or VAS score ($\alpha = 0.05$). Tukey HSD post-hoc tests were conducted where a statistically significant main effect was found. Finally, Pearson's correlations were calculated between VAS scores taken immediately prior to P1 and the P1 values of each LDS and rotational stiffness variables. All statistical tests were conducted in SAS 9.4 (SAS Institute, Cary NC, USA).

3. Results

One participant was excluded from the analysis as they verbally described a feeling of fatigue and visually struggled to perform the flexion extension movements toward the end of the protocol. No other participant reported a feeling of fatigue or showed any visual signs of fatigue.

There was a significant main effect of trial on neck VAS scores ($p < 0.0001$). Specifically, VAS scores were significantly higher during P1 and P2 compared to all the other trials, and P3 was significantly higher than P4 and baseline (Fig. 3). There were no significant correlations between VAS and any of the LDS or rotational stiffness variables (all were $> p = 0.46$).

There was a significant main effect of trial on both thoracic ($p = 0.0078$) and lumbar ($p = 0.0278$) λ_{max}. Specifically, thoracic λ_{max} was higher (indicating reduced LDS) in the first post pain trial (P1) compared to baseline and the third post pain trial (P3; 15 min post pain protocol) (Fig. 4). Lumbar λ_{max} was lower in the third post pain trial (P3) than in the first post pain trial (P1) (Fig. 5), but there were no significant differences compared to baseline.

There were no significant differences amongst the baseline or any of the post pain trials for any of the rotational stiffness variables (Figs. 6–8). Mean flexion/extension stiffness was lower during baseline than the pain trials and neared statistical significance ($p = 0.0730$) (Fig. 6). Similarly, maximum axial twist stiffness was lower during P1 than baseline or the other pain trials and neared statistical significance ($p = 0.0580$) (Fig. 8).

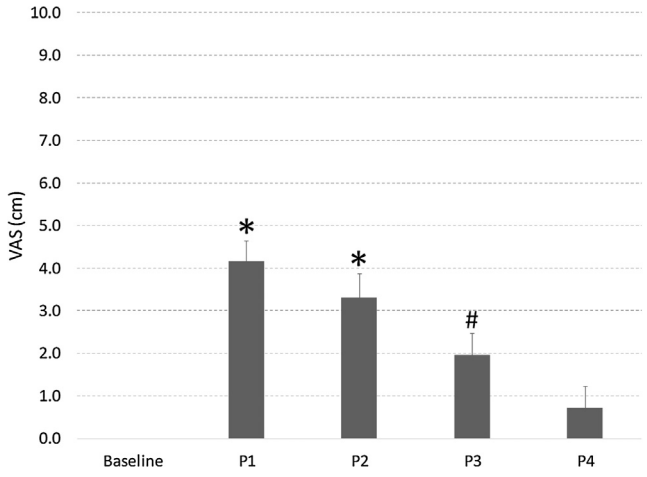

Fig. 3. Mean (+SEM) VAS for neck pain, from no pain (0 cm) to worst pain imaginable (10 cm), taken before each flexion/extension trial for Baseline (average of B2-B4), and before the four trials after the experimental pain protocol (P1, P2, P3, P4). P1 and P2 were significantly higher than all other trials denoted by "*" and P3 was significantly higher than Baseline and P4 denoted by "#". Note that Baseline values were zero since none of the participants had any neck pain prior to the experimental pain protocol.

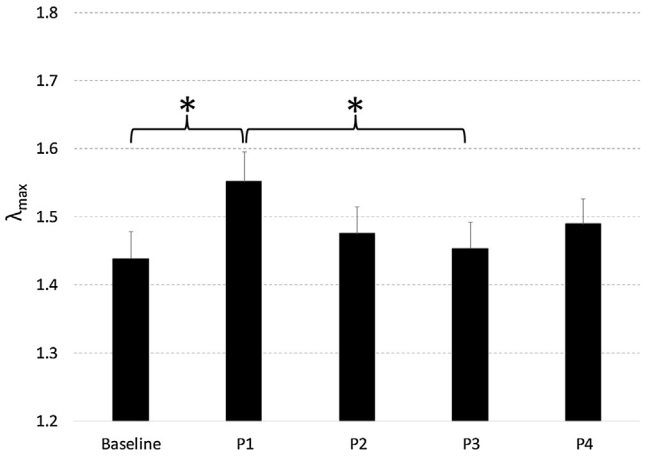

Fig. 4. Mean (+SEM) thoracic LDS for the average (B2-B4) baseline trial and four post pain protocol trials. P1 was significantly higher than baseline and significantly higher than P3.

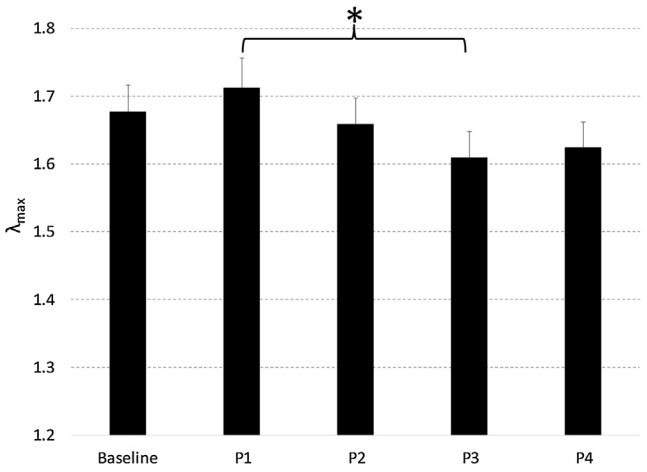

Fig. 5. Mean (+SEM) lumbar LDS for the average (B2-B4) baseline trial and each of the four post pain protocol trials (P1, P2, P3 and P4). P1 was significantly higher than P3.

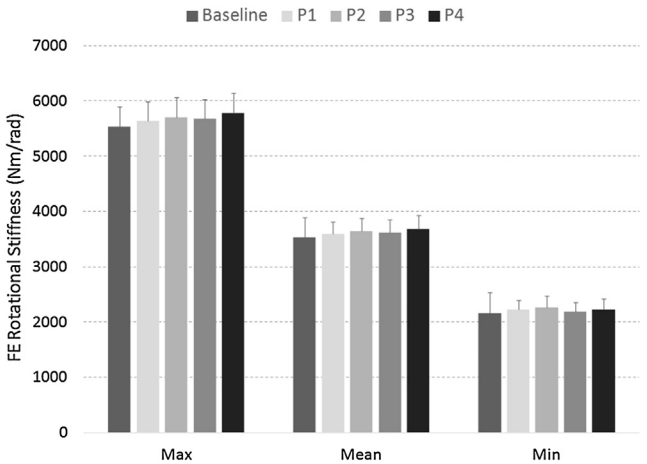

Fig. 6. Participant mean (+SEM) flexion/extension (FE) maximum, mean and minimum rotational stiffness for the average baseline (B2-B4) and the four post pain protocol trials (P1, P2, P3 and P4). No statistically significant differences were found.

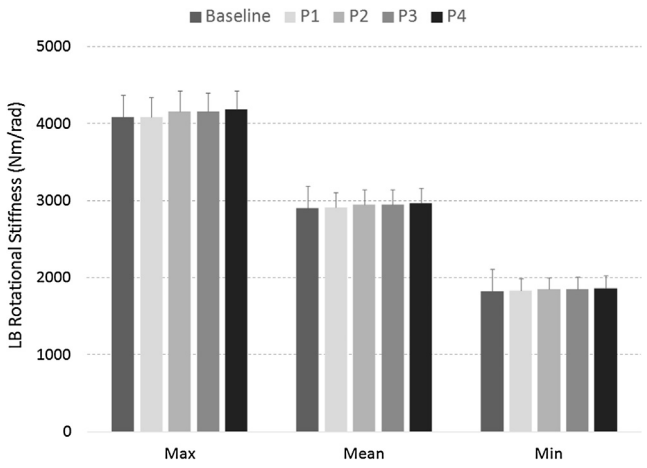

Fig. 7. Participant mean (+SEM) lateral bend (LB) maximum, mean and minimum rotational stiffness for the average baseline (B2-B4) and the four post pain protocol trials (P1, P2, P3 and P4). No statistically significant differences were found.

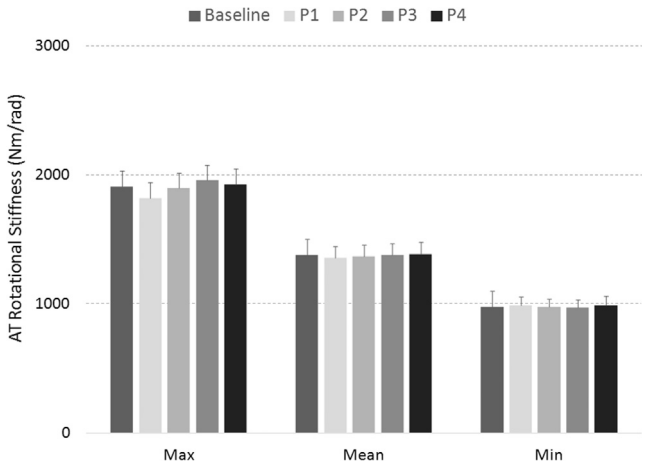

Fig. 8. Participant mean (+SEM) axial twist (AT) maximum, mean and minimum rotational stiffness for the average baseline (B2-B4) and the four post pain protocol trials (P1, P2, P3 and P4). No statistically significant differences were found.

4. Discussion

The primary purpose of this investigation was to quantify LDS and the muscular contribution to lumbar spine rotational stiffness after an experimental neck pain protocol. The experimental neck pain protocol was hypothesized to reduce LDS and rotational stiffness values compared to baseline, as in Ross et al. (2015). The current investigation found that thoracic LDS was reduced immediately after (P1) the experimental pain protocol and elevated in the third post pain trial (P3). Lumbar LDS was only significantly elevated during P3 compared to the first post pain trial (P1). There were no significant differences amongst the baseline or any of the post pain trials for any of the rotational stiffness variables. Hence our hypothesis was confirmed for the thoracic spine regarding LDS but not the lumbar spine regarding LDS or rotational stiffness.

Immediately after the neck pain protocol the thoracic LDS was significantly reduced (higher λ_{max}) compared to baseline and then elevated back toward baseline as the participant felt less pain in the subsequent trials. This indicates that the thoracic region of the spine immediately responded to the neck pain by increasing the expansion of its kinematic variance (becoming less kinematically stable). This may suggest that the thoracic region was not immediately able to respond appropriately to the neck pain, thus resulting in more unstable movement behaviour. However, it's important to note that all of our participants were still able to continue the experimental task (repetitive flexion/extension trials) and none of them reported any upper back discomfort. An alternate way to interpret these changes is that the upper back may have had to increase the expansion of its kinematic variance to adjust to any kinematic alterations in the neck region, due to pain, in order to maintain its intended trajectory while keeping the whole spine within an acceptable margin of kinematic behaviour. Importantly, the lumbar region of the spine did not demonstrate any changes to either LDS or rotational stiffness in response to the neck pain. Therefore it appears that any negative impact of the experimental neck pain was adequately compensated for by the upper back and therefore did not require or compel adaptations at the low back.

Interestingly, in the current study as reported pain (VAS) decreased over time after the pain protocol, both thoracic and lumbar LDS were significantly elevated (lower λ_{max}) during the P3 time point compared to P1. This provides some evidence for an association between pain experience and biomechanical behaviour. For the lumbar region, this may indicate that some effect of neck pain did manifest as a stabilizing influence down to the lower back, albeit at a later time point that coincided with the thoracic region's nearest return to baseline. The lack of difference in thoracic and lumbar LDS at the P4 time point compared to P1 suggests some variability in the return in LDS toward baseline after the pain experience.

Overall, relative to baseline, no statistically significant changes were observed in the lumbar spine LDS or stiffness in response to neck pain via capsaicin/heat application. This suggests that neck pain did not negatively influence the mechanics of the lower back. However, as suggested earlier these results may be influenced on the type or intensity of pain. Mean VAS scores prior to the P1 trial were 4.2 (out of 10), which represents a clinically significant increase from baseline (Kelly, 1998). However, it is of course plausible that a more intense pain response would have caused an even greater response at the upper back and a potentially significant response at the lower back. Further, when participants in the current study were asked how they felt the experimental pain protocol compared to their worst pain experience, they noted that they were not as threatened or fearful of the experimental pain since they knew its cause and that it would pass. This perception may have limited the extent of the biomechanical effect of this experimental pain experience.

A limitation of the study are the lack of data from the sensor placed on the head as this prevented us from expanding our hypothesis to determine whether the largest reductions in LDS in response to pain would occur at the site of pain in the neck region. These data would have provided an understanding of the impact of the experimental neck pain protocol on neck control and stability, and how these are interconnected with the more caudal regions of the spine studied here (thoracic and lumbar). We anecdotally observed that some participants appeared to move their neck less after experiencing the pain protocol. When asked "what does the pain protocol feel like?", one participant noted that he felt he was moving his neck less compared to the baseline trials. Individuals with neck pain can have reduced range of motion (Stenneberg et al., 2017) that is usually associated with further negative outcomes (Snodgrass et al., 2014). Those with chronic neck pain have also been found to move their neck with lower velocities and acceleration (Tsang et al., 2013) and demonstrate increased muscle co-activation (Tsang et al., 2014) compared to healthy individuals. This suggests that at least a chronic form of neck pain has a direct association with altered neuromuscular control of the neck region, but continued work will need to probe the relationships between

acute neck pain and corresponding neck mechanics and how this directly influences mechanics and control of the more caudal (thoracic and lumbar) regions of the spine. Additionally the use of the pelvis/hip restraint used here likely affects natural spine motion, but has been used in previous work in attempt to isolate movement to the spine and has demonstrated utility in differentiating between movement strategies (e.g. Granata and England, 2006; Graham et al., 2014; Ross et al., 2015). Next, despite none of the 20 analyzed participants indicating that they perceived any fatigue during the testing nor displaying any visual signs of fatigue, we cannot rule out the possibility that some did experience a low level of fatigue that could have influenced muscle recruitment patterns. Finally, participants were fully aware that the pain stimulus was only temporary, and thus the results of the study can only provide direct insight into their response to this short-term experience.

In summary, LDS of the upper back decreased in response to the capsaicin/heat induced neck pain. Limited adaptation was required at the lumbar region as demonstrated by the lack of statistically significant changes in lumbar spine LDS or rotational stiffness.

Acknowledgements

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Conflict of interest

The authors have no conflicts of interest associated with the work presented in this manuscript.

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