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Does experimental low back pain change posteroanterior lumbar spinal stiffness and trunk muscle activity? A randomized crossover study

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ABSTRACT

Background: While some patients with low back pain demonstrate increased spinal stiffness that decreases as pain subsides, this observation is inconsistent. Currently, the relation between spinal stiffness and low back pain remains unclear. This study aimed to investigate the effects of experimental low back pain on temporal changes in posteroanterior spinal stiffness and concurrent trunk muscle activity.
Method: In separate sessions five days apart, nine asymptomatic participants received equal volume injections of hypertonic or isotonic saline in random order into the L3–L5 interspinous ligaments. Pain intensity, spinal stiffness (global and terminal stiffness) at the L3 level, and the surface electromyographic activity of six trunk muscles were measured before, immediately after, and 25-minute after injections. These outcome measures under different saline conditions were compared by generalized estimating equations.
Findings: Compared to isotonic saline injections, hypertonic saline injections evoked significantly higher pain intensity (mean difference: 5.7/10), higher global (mean difference: 0.73 N/mm) and terminal stiffness (mean difference: 0.58 N/mm), and increased activity of four trunk muscles during indentation ($P < 0.05$). Both spinal stiffness and trunk muscle activity returned to baseline levels as pain subsided.
Interpretation: While previous clinical research reported inconsistent findings regarding the association between spinal stiffness and low back pain, our study revealed that experimental pain caused temporary increases in spinal stiffness and concurrent trunk muscle co-contraction during indentation, which helps explain the temporal relation between spinal stiffness and low back pain observed in some clinical studies. Our results substantiate the role of spinal stiffness assessments in monitoring back pain progression.

1. Introduction

Low back pain (LBP) is the most common and debilitating musculoskeletal disorder in the world (Vos et al., 2012). Given the heterogeneous cause of LBP (Brennan et al., 2006), clinicians attempt to use various strategies to classify patients into different subgroups to guide treatment selection. Posteroanterior spinal stiffness assessment is used as an objective measure to evaluate the mechanical responses of the spine to external forces (Abbott et al., 2009). During this examination, a manual/mechanical posteroanterior force is applied over the spinal processes of a prone patient while the corresponding spinal stiffness/movement is either perceived by the clinician or measured by a mechanical device (Maitland et al., 2005; Owens et al., 2007). The presence of hypomobile lumbar segment(s) is one of five clinical characteristics in a clinical prediction rule for identifying likely responders to successful spinal manipulation (Flynn et al., 2002). Similarly, immediate reduction in spinal stiffness following spinal manipulation may be related to favorable treatment outcomes (Wong et al., 2015).

Despite recent interests in spinal stiffness assessments, no consensus has been reached regarding the relation between spinal stiffness and LBP. Some clinical studies using manual/mechanical assessments found that people with LBP demonstrated higher lumbar stiffness than asymptomatic controls, and some patients' spinal stiffness decreased significantly during LBP remission (Brodeur and DelRe, 1999; Ferreira et al., 2009; Latimer et al., 1996). Conversely, other studies using similar lumbar stiffness assessments revealed no relation between spinal stiffness and LBP (Owens et al., 2007; Wong et al., 2013).

While these mixed results may be partly attributed to different stiffness measurement methods, it is plausible that some, but not all, patients with LBP display pain-related hyperactivity of trunk muscles that increases spinal stiffness (Hu et al., 2009; Shirley et al., 1999). Shirley and Lee found that some patients with LBP, as compared to their asymptomatic counterparts, displayed higher spinal stiffness and concurrent bilateral erector spinae (ES) activity during mechanical

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spinal stiffness tests (Shirley and Lee, 1993). They also found that a 10% maximum voluntary contraction of ES in asymptomatic individuals yielded an average increase in spinal stiffness of 11.8% (Shirley et al., 1999). Unfortunately, these cross-sectional studies did not examine the interrelation among LBP, spinal stiffness and trunk muscle co-contraction, which may help refine the existing clinical prediction rules (Flynn et al., 2002) or guidelines for LBP interventions.

Because it is difficult to quantify the temporal associations among the aforementioned parameters in a heterogeneous clinical population, these relations are best investigated by an experimental pain model. Numerous studies have used experimental pain models to explore the sensory and motor response of LBP (Hodges et al., 2003; Tsao et al., 2011). These models allow the control of intensity and duration of pain in individuals (Dickx et al., 2010). They also enable the investigations of causal effects of nociception on the changes of physical parameters.

For these reasons, the primary objective of the current study was to elucidate the effect of experimentally-induced pain on the temporal changes of spinal stiffness and simultaneous trunk muscle activity during spinal indentation. The secondary objective of the present study was to examine the correlation among pain intensity, spinal stiffness and trunk muscle activity. We hypothesized that experimental pain would transiently increase spinal stiffness and involuntary activity of some trunk muscles during indentation, which would return to baseline levels during pain remission. We also hypothesized that pain intensity, and percent changes in spinal stiffness and trunk muscle activity were closely related to one another.

2. Methods

2.1. Participants

Asymptomatic volunteers (age 18–60 years) without history of back/pelvic pain in the last 12 months were recruited by posters at the University of Alberta. Exclusion criteria were: major orthopedic, neurological or cardiorespiratory diseases, history of any back or abdominal surgery, malignancy, or potential/confirmed pregnancy. Written informed consent was obtained from participants. This study was approved by the institutional Research Ethics Board and registered with ClinicalTrials.gov (NCT01761838).

2.2. Study design

This study employed a randomized crossover design. Participants attended 2 sessions at the same time of day at least 5 days apart in a rehabilitation clinic. During the first session, participants completed a battery of self-reported questionnaires and underwent a physical examination. Surface electromyography (EMG) electrodes were attached to six trunk muscles (i.e. bilateral obliquus externus (OE), obliquus internus/transversus abdominis (OI/TrA), and ES at the L3–4 levels). Participants were instructed to perform maximum voluntary isometric contraction (MVIC) of each trunk muscle against manual resistance (Table 1) (Kavcic et al., 2004; Hodges et al., 2013; Marshall and Murphy, 2003; Imai et al., 2010). The EMG signals during MVIC were used for subsequent EMG normalization. The participant then underwent standardized assessments at 3 time points: 1) before (baseline), 2) immediately after, and 3) 25-min after saline injection (Fig. 1). The 25-min follow-up duration was chosen because the present pain provocation method induced pain lasting approximately 14 min (Tsao et al., 2010a). The standardized assessments include three instrumented spinal stiffness tests and concurrent trunk muscle EMG measurements (Fig. 1). After the baseline standardized assessments, either 0.3 ml of 0.9% isotonic saline or 5% hypertonic saline was injected into the L3–4 and the L4–5 interspinous ligaments (Tsao et al., 2011). Hypertonic saline injection was used to induce pain, while isotonic saline injection was used to control for the volumetric effect of injection on spinal stiffness. Because saline injections into the interspinous ligament might change the mechanical properties of passive tissues and result in altered spinal stiffness, isotonic saline was chosen to control for potential volumetric effects. The saline concentration injected at the first session was randomly assigned. Participants were reminded not to perform volitional trunk muscle contraction during the spinal stiffness tests.

Table 1
Placement of surface electromyography electrodes and measurement of maximum voluntary isometric contraction of trunk muscles.

Muscle/ground electrode	Placement of electrodes	Maximum voluntary isometric contraction measurement
Obliquus externus	Approximately 15 cm lateral to the umbilicus	It was tested in supine with bent knees and shoulder in 90° flexion and full elbow extension (Hodges et al., 2013). Manual resistance was applied at the bent knees and the arms to resist right or left trunk rotation.
Obliquus internus/transversus abdominis	2 cm medial and inferior to the anterior superior iliac spine (Marshall and Murphy, 2003)	A maximal expiratory maneuver with abdominal hollowing was performed in a sit-up position (Imai et al., 2010).
Erector spinae	3 cm lateral to the L3 spinous process (Kavcic et al., 2004)	Performing prone extension against resistance applied to the upper trunk in a PA direction while the arms were placed beside the body and legs were tied to the plinth by a strap (Imai et al., 2010).
Ground electrode	The left acromion	–

During the second session, the participants underwent the same procedures but with the previously unused saline concentration. A 5-day washout period was chosen to mitigate any carryover effects of previous injections. To reduce participant bias toward the anticipation of saline concentrations, participants were told that they would receive injections of two distinct saline concentrations that might elicit different pain intensities. At the end of the second session, the order of saline injections was revealed to each participant.

2.3. Self-reported measures

On the first session, participants completed the fear of pain questionnaire—III (FOP-III) (McNeil and Rainwater, 1998), which evaluated participants' fear toward various pain provoking conditions (McNeil and Rainwater, 1998). Pain intensity was measured on an 11-point numeric pain rating scale (NPRS) where 0 represents “no pain” and 10 represents “worst pain imaginable” (Williamson and Hoggart, 2005). Pain intensity was measured at baseline, at every 30 s after the injection until the experimental pain completely subsided, and at 25 min after the injection. The location and size of the experimental pain were marked on a body pain diagram (Werneke et al., 1999).

2.4. Spinal stiffness assessment

Spinal stiffness was assessed using a mechanical indentation device whose performance has been detailed elsewhere (Wong et al., 2015; Wong et al., 2013). Briefly, the device comprises of a motorized indentation probe equipped with a load cell transducer and a rotary encoder. Custom written LabVIEW software (National Instruments, Austin, USA) was used to control the loading speed (2.0 mm/s) of the probe and to collect force and displacement signals.

The L3 spinous process of the prone participant was identified by ultrasonography. The indenter was then positioned above the L3 level. The participants held their breath at the end of normal exhalation during indentation at the L3 level (Wong et al., 2015; Wong et al., 2013). All