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Reorganized Trunk Muscle Activity During Multidirectional Floor Perturbations After Experimental Low Back Pain: A Comparison of Bilateral Versus Unilateral Pain

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Abstract: **Low back pain changes trunk muscle activity after external perturbations but the relationship between pain intensities and distributions and their effect on trunk muscle activity remains unclear. The effects of unilateral and bilateral experimental low back pain on trunk muscle activity were compared during unpredictable multidirectional surface perturbations in 19 healthy participants. Pain intensity and distribution were assessed using a visual analogue scale (VAS) and pain drawings. Root mean square (RMS) of the electromyographic (EMG) signals from 6 trunk muscles bilaterally after each perturbation was extracted and averaged across perturbations. The difference (ΔRMS-EMG) and absolute difference (absolute ΔRMS-EMG) RMS from baseline conditions were extracted for each muscle during pain conditions and averaged bilaterally for back and abdominal muscle groups. Bilateral compared with unilateral pain induced higher VAS scores ($P < .005$) and larger pain areas ($P < .001$). Significant correlation was present between VAS scores and muscle activity during unilateral ($P < .001$) and bilateral pain ($P < .001$). Compared with control injections ΔRMS-EMG increased in the back ($P < .03$) and abdominal ($P < .05$) muscles during bilateral and decreased in the back ($P < .01$) and abdominal ($P < .01$) muscles during unilateral pain. Bilateral pain caused greater absolute ΔRMS-EMG changes in the back ($P < .01$) and abdominal ($P < .01$) muscle groups than unilateral pain.**
Perspective: ***This study provided novel observations of differential trunk muscle activity in response to perturbations dependent on pain intensity and/or pain distribution. Because of complex and variable changes the relevance of clinical examination of muscle activity during postural tasks is challenged.***

Key words: *Lumbar spine, pain induction, motor control, motor strategy, electromyography.*

The lifetime prevalence of low back pain (LBP) is up to 38.9%[40] and the evidence on causality is poor.[31,37] Nonetheless, genetic[50] and psychosocial factors[45,58,61] have been proposed as risk factors in LBP, and movement strategies and muscle activation patterns might be potential factors.[1,37] Muscle function and coordination are usually altered in LBP patients[20,37] and impaired trunk muscle activation and activity gained much attention as an explanatory model for LBP.[30] Although the underlying mechanisms in trunk motor control and pain are sparsely linked,[58] trunk muscle training is widely implemented clinically and in sports[70] with underlying assumptions on trunk muscles as spinal stabilizers during functional tasks.[69,70] However, the nature of possible changes of inconsistent complex muscle pain adaptation is evident.[31,52] Additionally, stabilization exercises have no long-term effect[18] or are not superior to other treatments.[10,13,68]

Experimental pain models therefore have been used extensively to explore the effects of LBP, and aim to mimic pain and yet exclude confounding factors in LBP patients.[3,20] In previous studies lumbar pain was induced unilaterally, but differences in pain characteristics between subacute LBP patients with greater prevalence of unilateral pain and persistent LBP patients predominantly indicating bilateral pain[11] highlight the importance of understanding whether pain-related

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mechanisms during motor tasks differs between unilateral and bilateral pain conditions.

Gait is the primary human locomotion function and on the basis of gait studies in LBP it is evident that complex muscle control is related to specific, and individual, temporal and spatial demands.[37] LBP patients showed inconsistent muscle activity with for example, increased back muscle activity during the swing phase[4] and increased coactivation of erector spinae and rectus abdominis muscles,[76] with increased lumbar and decreased abdominal muscle activity present in patients older than 50 years.[22] van den Hoorn et al[75] additionally found individualized synergistic muscle strategies during treadmill walking and the trunk control synergies were affected by back and leg pain in some subjects.

The nature of the gait task is complex and involves motor planning as well as motor adaptation and the effect of pain on the underlying mechanisms in motor control during gait is challenged. Contrarily, surface perturbation is a highly standardized and still complex motor task because unpredictable surface perturbation is challenging[39] because of nonpredictable, high-velocity changes from the external perturbation.[38,72] Multidirectional floor perturbations resulted in increased cocontraction of the trunk muscles in persistent LBP patients compared with a control group, which indicated a trunk-stiffening strategy.[44] In contrast, Boudreau et al[7] found decreased trunk muscle activity after anterior and posterior perturbations after pain induction in healthy participants. It remains unknown if these observed alterations are related to the differences in the surface perturbation protocol or if the underlying musculoskeletal impairments are important. Although studies showed no changes in proprioception in LBP patients,[56,59] postural repositioning is generally challenged and decreased variability in postural adjustments to perturbations after acute[53] and persistent LBP[41] furthermore might indicate complex trunk muscle timing and activity.[60] Various motor adaptations in functional tasks are generally accepted,[3,29,37] but although experimental unilateral pain affects trunk muscle activity bilaterally[7] and pain-related reorganization of the trunk muscle strategies during LBP is evident between[29] and within[16] muscles, the underlying interactions between muscles are not well understood[34] and the effect of unilateral and bilateral pain on the trunk muscle response is unknown.

The aim of the study was to compare the effects of unilateral and bilateral experimental LBP on trunk muscle activity during unpredictable multidirectional surface perturbations in healthy participants. It was hypothesized that: 1) unilateral LBP will decrease, and 2) bilateral LBP will increase trunk muscle activity during multidirectional unpredictable surface perturbations.

Methods

Participants

Nineteen healthy participants (4 female with a mean age of 26 years [range, 19–39 years]; mean height of 180 cm [range, 160–200 cm], mean body mass index of 23.7 [range, 20.4–29.2]) without lower extremity or back-related pain or dysfunction participated in the study. The study was conducted in accordance with the Declaration of Helsinki and was approved by the local ethics committee (N-20090053) and informed consent was obtained from each participant.

Protocol

The subjects participated in 1 baseline perturbation session and 3 successive experimental perturbation sessions on the same day with a minimum of a 15-minute break in between conditions: 1) bilateral experimental saline-induced LBP, 2) bilateral control condition, and 3) unilateral experimental saline-induced LBP. In each session, the subject was standing on a marked position on a moveable platform during a series of 20 randomized multidirectional surface perturbations delivered after an auditory warning signal. Between sessions the subjects were allowed to sit on a chair.

Experimental LBP

The injection procedure was performed with the subject lying prone. The Th12 segment was located and L2 was down-counted and verified by palpation of L4 at the line between the iliac crest bilaterally where the L2 location was estimated.[15] At the L2 level the most bulky part of the longissimus muscle was palpated (typically 3–5 cm from the midline) and marked as the injection site. Sterile isotonic (1.0 mL, 0.9%) or hypertonic (1.0 mL, 5.8%) saline was injected perpendicular to the skin surface with a 25-gauge × 19 mm needle, after cleaning the injection site with alcohol. Hypertonic and isotonic saline was injected bilaterally (experimental condition 1 and 2, respectively) and in experimental condition 3 one hypertonic saline injection was given in the right side immediately followed by an injection of isotonic saline in the left side. The participants were informed about receiving injections, but were blinded to the type of saline injected. In the bilateral conditions the right injection was performed before the left and the time between injections was 30 to 60 seconds. Immediately after completion of both injections, the participant was assisted to the standing position on the platform for perturbations and started scoring the pain intensity.

During the perturbations the pain intensity was assessed using a 10-cm electronic visual analogue scale (VAS) with an external handheld slider. The VAS was anchored with 'no pain' and 'maximum pain' at 0 cm and 10 cm, respectively. The signal from the VAS was recorded after each injection until the pain vanished (sample frequency of 20 Hz). During the complete period including perturbations the mean VAS score was extracted in the time window from onset to the subsequent perturbation and the maximum VAS and average VAS scores were extracted among the 20 perturbations. The subjects were asked to recover their balance as fast as possible after the perturbation, and only then, they were allowed to update the VAS. After each condition the subjects were asked to indicate the pain distribution on a body chart. The pain area was extracted from the