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Pain catastrophizing and trunk muscle activation during walking in patients with chronic low back pain



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ABSTRACT

It has been hypothesized that individuals with low back pain (LBP) will have higher trunk muscle activity during gait, in an attempt to limit spine motion, and that this "guarding strategy" may be influenced by the person's psychological response to pain. This study investigated whether the amplitude of trunk muscle activation differs between persons with chronic LBP and healthy individuals during walking, and whether changes in muscle activation were related to pain catastrophizing. Thirty persons with chronic non-specific LBP, stratified into 2 groups of high (HLBP) and low (LLBP) pain catastrophizing, were contrasted with a control group of 15 healthy individuals during walking on a treadmill at a self-selected speed. Surface electromyographic (EMG) data were recorded from 10 trunk muscles. The effects of Group and gait Sub-phase on EMG activation amplitudes were assessed. The HLBP group exhibited higher activation of certain muscles throughout the gait cycle, and reduced variability of others at specific subphases of gait. A significant correlation was found between activation amplitude and pain catastrophizing in most muscles, when controlling for gait speed and pain intensity. These data indicate that altered trunk muscle activation is present in some patients with LBP during walking, but does not represent a universal increase in activation for all muscles. This altered neuromotor control is, however, more strongly associated with pain catastrophizing than with pain intensity, and appears to represent a non-functional, maladaptive behavior, as it alters the normal, phasic pattern of activation in certain trunk muscles.

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1. Introduction

Individuals with low back pain (LBP) tend to walk slowly [1], which may be a means of reducing spine motion [2]. This should normally lead to lower trunk muscle activation [3], but there is evidence that some individuals with LBP increase trunk muscle activity during gait, possibly as a "guarding strategy" to further limit spine motion [4,5]. Furthermore, it has been suggested that muscle guarding is more closely related to the psychological response to pain than to pain intensity [6].

There are conflicting findings on pain-induced trunk muscle guarding during gait. Arendt-Nielsen et al. [7], for example, reported a decrease in the phasic nature of EMG patterns during

gait in individuals with chronic LBP, which could reflect an attempt to splint the spine. Lamoth et al. [8], on the other hand, found that induced pain has only minimal effects on the global pattern of lumbar erector spinae activity. The influence of psychological factors on trunk muscle activity during gait is also unclear. Two previous studies have found no relationship between fearavoidance beliefs and lumbar EMG [4,9]. Pain catastrophizing, however, has been linked to increase erector spinae activity during gait [10].

The objective of this cross-sectional study is twofold: first, to investigate whether the amplitude of trunk muscle activation differs between individuals with and without chronic LBP, when walking on a treadmill at a self-selected speed; second, to determine if the amplitude of trunk muscle activation is affected by pain catastrophizing in individuals with chronic LBP. We hypothesized that higher trunk muscle activation is used by persons with LBP during walking as a guarding strategy that can be influenced by pain catastrophizing.



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2. Methods

2.1. Participants

Thirty individuals with chronic LBP were divided into two ageand gender-matched groups, based on their scores on the Pain Catastrophizing Scale (PCS) [11]: high (HLBP: PCS > 21/52) and low (LLBP: PCS < 20/52). The cut-off value of 20/52 was chosen based on the median value for the PCS, as reported in the PCS User Manual [12]. Inclusion criteria for both groups were: nonspecific LBP (excluding serious pathology, spinal stenosis and radiculopathy [13]) primarily located between the lower ribs and gluteal folds; pain duration >3 months; pain intensity >2/10 on the numeric pain rating scale (NPRS) [14]; an Oswestry Disability Index (ODI) score >12% [15]. Participants in the HLBP and LLBP groups also completed the Fear-Avoidance Beliefs Questionnaire (FABQ) [16], and were asked to identify the primary location of their LBP (right side, left side, middle). At each step of the experimental procedure, patients were asked to rate their pain using the NPRS. Pain intensity did not change by more than 2 points for any participant during testing.

A third group of 15 age- and sex-matched individuals, with no history of LBP within the past year, was also recruited.

Exclusion criteria for all groups included a confirmed diagnosis of arthritis (spine or lower limbs), fibromyalgia, scoliosis, leg length discrepancy >2 cm, spinal surgery, pregnancy within the past year, neurologic, respiratory or lower limb pathology affecting gait.

Demographic and anthropometric descriptors for the three groups are presented in Table 1. Only self-selected gait speed was different between groups (one-way ANOVA comparison), which was lower for the HLBP group than for healthy controls.

Clinical profiles of the HLBP and LLBP groups are also presented in Table 1. Scores on the clinical questionnaires were all significantly higher in the HLBP group (independent samples *t*test comparison), while no difference was found for pain duration.

Participants were recruited from hospitals and clinics, and from the local community, and provided written, informed consent prior to participation. The research ethics board of CRIR provided approval for the study.

2.2. Electromyography (EMG)

Trunk muscle activity was recorded using a Trigno wireless surface EMG system (Delsys, Boston, MA: CMRR > 80 db at 60 Hz, bandwidth 20–450 Hz, gain 1000) at a sampling rate of 2000 Hz. Trigno sensors consist of four 5×1 mm parallel bar silver (99.9%) electrodes, have a dry contact surface, and are applied after careful skin preparation. Sensor placement and notation is shown in

Table 1

Characteristics of the study p	participants (Mean \pm SD).
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	Control	LLBP	HLBP
Gender	9F,6M	9F,6M	9F,6M
Age (y)	$\textbf{33.1} \pm \textbf{6.8}$	32.5 ± 6.4	34.1 ± 6.8
Height (cm)	171.1 ± 8.0	172.3 ± 8.7	169.3 ± 5.3
Weight (kg)	69.5 ± 6.1	72.7 ± 11.4	70.6 ± 12.1
BMI	23.8 ± 2.1	24.4 ± 2.6	24.5 ± 3.3
Speed (cm/s) ^a	125.4 ± 9.6	119.9 ± 10.3	113.6 ± 15.7
NPRS ^b		3.4 ± 1.1	4.9 ± 1.2
ODI (%) ^b		18.7 ± 6.8	27.6 ± 8.4
FABQ ^b		25.3 ± 10.8	35.9 ± 10.6
PCS ^b		13.4 ± 5.6	32.0 ± 8.3
Pain Duration (M)		19.5 ± 11.9	14.3 ± 7.4
Pain Site		4right,4left,7middle	2right,7left,6middle

^a Significant difference (Control vs. HLBP).

^b Significant difference (LLBP vs. HLBP).

Table 2, with the prefix R or L denoting the right or left side (e.g. RRA = Right Rectus Abdominis).

EMG signals were high-pass filtered at 30 Hz to minimize electrocardiographic artifacts, full-wave rectified, and then low-pass filtered at 2.5 Hz to form a linear-envelope as in previous analysis [17]. Butterworth, 4th order, dual-pass filters were used.

Sub-maximal voluntary isometric contractions (sub-MVIC) were performed by all participants, prior to gait testing, according to the protocol described by Dankaerts et al. [18]. For RA and EO, participants lay supine, with hips flexed to 45° and knees flexed to 90°, then raised both feet 1 cm off the plinth for 3 s. For ESL, ESI and MF, participants lay prone, with knees bent to 90°, then lifted both knees 5 cm off the plinth for 3 s. Three trials were performed for each task, and the mean EMG amplitude for the three trials was used to normalize the EMG amplitudes acquired during the subsequent gait testing (%sub-MVIC).

2.3. Self-selected gait speed

Participants walked, over ground, at their usual comfortable speed, for 60 s, while pushing a wheeled odometer. The average gait speed over 3 trials, for each participant, was used as the self-selected gait speed for subsequent testing.

2.4. Gait testing

Participants walked on a split-belt, instrumented R-Mill treadmill (Forcelink B.V, Culemborg, Netherlands). A warm-up period of four minutes was first provided to allow the participants to accommodate to the treadmill. Following a one-minute rest period, participants were again asked to walk on the treadmill, with data acquired for 60 s, at the previously-determined self-selected gait speed, in order to measure at least 20 gait cycles [19].

Surface reaction force data were acquired under each foot, at a sampling rate of 120 Hz, and synchronized with the EMG data using a trigger pulse (Delsys Trigger Module, Boston). The force data was then low-pass filtered at 10 Hz (Butterworth, 4th order, dual-pass design).

2.5. Critical gait events

EMG and force data were re-sampled to 1000 Hz, using a cubicspline interpolation. Critical gait events (foot-contact and foot-off) were identified from the vertical surface reaction force, using a 20 N threshold. One gait cycle was defined as the time between successive right foot contacts, and was divided into four subphases of gait, based on the identified critical events: right double limb support (RDLS: right foot-contact to left foot-off), left swing (LS: left foot-off to left foot-contact), left double limb support (LDLS: left foot-contact to right foot-off), and right swing (RS: right foot-off to right foot-contact) [20].

EMG data for each sub-phase of gait were re-sampled to 100 data points, using a cubic-spline interpolation. For each participant, the average EMG amplitude for each sub-phase, from the first 20 gait cycles, was retained for analysis.

Data processing was performed using Matlab (Mathworks, Nantick, MA).

2.6. Statistical analysis

The %sub-MVIC-normalized EMG amplitude for each muscle was analyzed separately, using a mixed design ANOVA, to determine the main effects of Group (Control, LLBP, HLBP) and Sub-phase of gait (RS, RDLS, LS, LDLS), and any interaction effects. Significant main effects of Group were analyzed by post-hoc Download English Version:

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