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A History of Low Back Pain Associates with Altered Electromyographic Activation Patterns in Response to Perturbations of Standing Balance

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A history of low back pain associates with altered electromyographic activation patterns in response to perturbations of standing balance

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Running Head: postural responses with low back pain

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ABSTRACT

People with a history of LBP exhibit altered responses to postural perturbations and the central neural control underlying these changes in postural responses remains unclear. To characterize more thoroughly the change in muscle activation patterns of people with LBP in response to a perturbation of standing balance, and to gain insight into the influence of early- versus late-phase postural responses (differentiated by estimates of voluntary reaction times), this study evaluated the inter-muscular patterns of electromyographic (EMG) activations from 24 people with and 21 people without a history of chronic, recurrent LBP in response to 12 directions of support surface translations. Two-factor general linear models examined differences between the 2 subject groups and 12 recorded muscles of the trunk and lower leg in the percent of trials with bursts of EMG activation as well as the amplitudes of integrated EMG activation for each perturbation direction. The subjects with LBP exhibited (1) higher baseline EMG amplitudes of the erector spinae muscles prior to perturbation onset, (2) fewer early-phase activations at the internal oblique and gastrocnemius muscles, (3) fewer late-phase activations at the erector spinae, internal and external oblique, rectus abdominae, as well as the tibialis anterior muscles, and (4) higher EMG amplitudes of the gastrocnemius muscle following the perturbation. The results indicate that a history of LBP associates with higher baseline muscle activation and that EMG responses are modulated from this activated state rather than exhibiting acute burst activity from a quiescent state, perhaps to circumvent trunk displacements.

Keywords: Low Back Pain; Posture; EMG; Postural Responses; Muscle Synergies

INTRODUCTION

Low back pain (LBP) represents a common, disabling, and costly condition. As many as 85% of people experience LBP (Andersson 1999), rendering LBP a world-wide leading cause of limited activity and disability (Cassidy et al. 1998; Kelsey et al. 1979; Picavet and Schouten 2003; Walker et al. 2004). In addition, yearly expenses due to LBP are estimated to total 100 billion dollars in the United States (Katz 2006). High rates of recurrent or chronic symptoms suggest inadequate treatment or preventative strategies (Andersson 1999; Hestbaek et al. 2003), thereby necessitating a better understanding of LBP in order to facilitate more efficacious treatment strategies and improved patient outcomes.

Postural responses to sudden perturbations associate with the occurrence of LBP, and impaired responses are evident with chronic or recurrent LBP. Slips or sudden changes in load represent one mechanism by which people incur episodes of LBP (Manning et al. 1984). Further, several studies have evaluated the differences between people with and without LBP by examining surface reaction forces and muscle activation patterns associated with unstable stance or sitting as well as their responses to discrete perturbations elicited by weight unloading or sudden movements of the support surface. People with LBP exhibit smaller shear forces during unstable standing conditions (Claeys et al. 2010; Mok et al. 2004). Flexion or extension of the hip and trunk produces horizontal shear forces at the support surface, whereas ankle plantar- or dorsi-flexion generates a greater amount of vertical forces at the support surface (Horak and Nashner 1986). Thus, the smaller shear forces of people with LBP suggest less use of the hip joint for making postural adjustments in order to maintain standing balance. In addition, people with LBP exhibit smaller and slowed center-of-pressure displacements with larger center-of-mass displacements in response to translations of the support surface (Henry et al. 2006). Hip

flexion and extension more rapidly produce large corrective pressure displacements at the support surface in order to more quickly reverse the perturbation-induced fall of the center of mass (Kuo and Zajac 1993). Thus, the slower and smaller center-of-pressure displacements with the larger continued fall of the center of mass suggest people with LBP exhibit postural responses dominated by movements around the ankle rather than the hip. This decreased reliance on the hip joint may be a strategy employed to minimize forces and movement about the trunk. These evaluations of ground reaction forces and their inferences regarding altered postural strategies, however, require confirmation through an evaluation of the underlying muscle response patterns.

Studies evaluating the electromyographic (EMG) responses of people with LBP to postural perturbations have typically limited the perturbation and recordings to the body's trunk (Cholewicki et al. 2005; MacDonald et al. 2010; Radebold et al. 2000; Reeves et al. 2005; Stokes et al. 2006). These studies revealed delayed trunk muscle responses (Cholewicki et al. 2005; Radebold et al. 2000; Reeves et al. 2005), decreased amplitudes of muscle activation (MacDonald et al. 2010; Radebold et al. 2000), as well as evidence of co-contraction (Radebold et al. 2000) or higher baseline muscle activation (Stokes et al. 2006) associated with, or predictive of, LBP. We are aware of only one study to examine the EMG responses of people with and without LBP to postural perturbations in free stance, which demonstrated a decreased incidence of abdominal muscle activation to toes-up rotations of the support surface for people with LBP (Newcomer et al. 2002).

Some of the aforementioned EMG and kinetic studies of postural control in people with LBP suggested that this population might have impaired proprioception and kinesthetic awareness of the trunk (Claeys et al. 2010; Henry et al. 2006; Mok et al. 2004; Radebold et al.

93 2000; Reeves et al. 2005). Thus, the implication is that the reduced hip joint displacements as
94 well as the delayed and decreased EMG responses represent feedback-related changes in motor
95 output because of altered sensory input at the site of the LBP. Although decreased proprioceptive
96 sensitivity likely contributes to the altered postural responses of people with LBP (and is not the
97 subject of this study), some of these studies also acknowledged the possibility that altered
98 postural responses may represent a centrally generated change in muscle synergies reflective of
99 an altered central set (Cholewicki et al. 2005; Henry et al. 2006; MacDonald et al. 2010;
100 Radebold et al. 2000).

101 To gain insight into whether LBP-related changes in postural responses arise due to
102 altered muscle response patterns that are intrinsically organized by the central nervous system,
103 this study evaluated the inter-muscular EMG response patterns of the abdominal, back, and
104 lower-leg musculature to multi-directional support surface translations of freestanding subjects
105 with and without a history of LBP. Therefore, although local impairments at the site of LBP
106 might contribute to the need for altered global response strategies, examining responses of dorsal
107 and ventral trunk muscles as well as muscles distal to the trunk during a freestanding posture will
108 clarify whether a centrally coordinated change in global muscle response patterns (in contrast to
109 a local change in muscle activation at only the low back) contributes to the altered postural
110 responses of people with LBP.

111 In order to gain more detailed information regarding the underlying mechanisms of the
112 subjects' response patterns, this study also evaluated the subjects' early-phase and late-phase
113 EMG response patterns based on the potential influence of voluntary responses (Chan et al.
114 1979; Jacobs and Horak 2007). We base our interpretations on a model of neural control (Jacobs
115 and Horak 2007) in which cortico-striatal circuits first generate preparatory muscle states and

prime potential muscle activation patterns related to a postural response strategy in order to meet the biomechanical, intentional, and environmental constraints that exist prior to a postural perturbation (Horak et al. 1997). Examples of such strategies include feet-in-place hip, knee, or ankle displacements as well as stepping or reaching responses. The muscle synergies that define these strategies are thought to be located within the brainstem. When experiencing a postural perturbation, this primed strategy within the brainstem is then automatically triggered by sensory input related to the perturbation. The execution of this centrally organized strategy can be modified again by executive motor centers higher along the neural axis only in its late phases provided that conduction times allow for such influence. Evaluating preparatory muscle activation states as well as both the early and late phases of the muscle response pattern across proximal and distal segments of the body, therefore, will provide insight into the central neural mechanisms by which people with LBP alter their postural responses.

Guided by the hypothesis that people with LBP would exhibit centrally driven alterations in muscle activation patterns to minimize hip and trunk activity during postural responses, we predicted lower incidence and smaller amplitudes of trunk muscle activations, with higher activation incidence and larger amplitudes at the ankle musculature, during both the early- and late-phase responses. Identifying central neural organization as a contributing mechanism to altered postural responses with LBP would influence treatment strategies to include interventions on motor retraining and strategy selection in addition to interventions that address underlying biomechanical or proprioceptive impairments, all of which may contribute to recurrence of LBP.

METHODS

Subjects

Twenty-four subjects with chronic or recurrent LBP (as defined by Von Korff 1994) and 21 subjects without chronic or recurrent LBP participated in the study following recruitment from the local community through posted advertisements. The subject groups' sex distribution, as well as their average age, height, and weight were not statistically different (Table 1). Subjects with LBP were excluded (by clinical exam or interview) if they reported vertebral fracture, tumor or infection, spinal stenosis, previous spinal surgery, systemic infection, balance or cardiovascular disorders, current pregnancy, history of any surgery in the three months prior to testing, uncorrected vision problems, scoliosis or kyphosis, injury to the lower extremity, or radiating pain below the knee that would be consistent with a disc herniation. Subjects were also excluded if they were receiving disability compensation for their LBP, or if they were in litigation because of the LBP problem. Subjects were not tested during an acute flare-up of their LBP (McGorry et al. 2000) and consequently reported mild levels of pain on the Numeric Pain Rating Scale (Childs et al. 2005) as well as mild levels of disability on the Roland Morris Disability Questionnaire (Roland and Morris 1983) on the day of testing (Table 1). Based on visual analysis of pain body charts, only two subjects identified the location of their LBP as unilateral, left-sided pain, whereas the other 22 subjects identified the location as bilateral. Subjects without LBP were excluded if they had a neurological, psychiatric, cardiovascular or musculoskeletal disorder, uncorrected vision problems, severe musculoskeletal injuries, or history of back pain that required medical attention or resulted in missed work. All subjects were currently employed or active as a full-time student or homemaker.

The subjects represent an overlapping sample of those included in a previous report, which demonstrated that people with LBP exhibit smaller and slowed center-of-pressure displacements with larger center-of-mass displacements in response to translations of the support

surface (Henry et al. 2006). The subject sample was selected on the basis of available EMG data. All subjects provided written informed consent to participate in the protocol, which was approved by the local institutional review board.

Procedures

Subjects were instructed to stand looking forward on a moveable platform at their self-selected stance width (Table 1) and with their arms hanging comfortably at their sides. The subjects were then instructed to maintain their standing balance in response to the platform movements but were not given any instructions about how to respond. Subjects were given three practice trials in each of two perturbation directions (leftward or forward translations) in order to familiarize them with the task. Following these practice trials, three trials in each of 12 directions of linear surface translations (separated by 30-degree increments; Fig. 1) were presented in random order and at unpredictable intervals. The platform translations consisted of 9-cm, ramp-and-hold waveforms with duration of 400 ms, peak velocity of 43 cm/s, and peak acceleration of 127 cm/s^2.

In order to record the muscle activation patterns associated with the subjects' postural responses, bipolar surface EMG was recorded by 1-cm silver, silver-chloride disc electrodes (Norotrodes with fixed 2 cm inter-electrode distance; Myotronics, Kent, WA, USA) placed over the (1) bilateral erector spinae muscles 2.5 cm lateral of the 1^{st} (EST) and the 3^{rd} lumbar (ESP) spinal segments and oriented rostral-caudally, (2) bilateral external oblique (EO) muscles at the lateral midline, 50% of the distance between the iliac crest and lower ribs, and oriented at a 45-degree angle rostral-dorsal to caudal-ventral, (3) bilateral internal oblique (IO) muscles 2.5 cm medial and 2.5 cm rostral to the anterior-superior iliac spine and oriented at a 45-degree angle rostral-medial to caudal-lateral, (4) bilateral rectus abdominae (RA) muscles 2.5 cm lateral to the

umbilicus and oriented rostral-caudally, (5) left tibialis anterior (TA) muscle over the most prominent bulge of the contracted muscle belly located approximately 2.5 cm lateral to the tibia and 33% distal of the length between the tibial condyle and malleolus, oriented rostral-caudally and (4) left gastrocnemius medialis (GM) muscle over the most prominent bulge of the contracted muscle belly, oriented rostral-caudally. The EMG responses of the TA and GM muscles were not recorded bilaterally due to a limited number of available recording channels. Electrode placement was standardized based on anatomical landmarks (e.g., distance from the umbilicus, iliac spines, or spinal segments). Skin impedance was maintained under 10 kΩ. The EMG signals were sampled at 1000 Hz, pre-amplified by 1000 at the skin's surface and then amplified further for a total amplification of 2000-10000.

Data Processing

Using Matlab software (Matlab, Natick, MA, USA), the EMG signals were band-pass filtered at 35-200 Hz, baseline corrected by subtracting the mean of the signal, and full-wave rectified. The high-pass limit was set to minimize cardiac artifact (Drake and Callaghan 2006). The integrated protocol method was then used with an option for manual override to identify EMG activation onset; this method evaluates the point of maximum difference between the integrated signal and an amplitude-normalized integral of the linear envelope, and is less susceptible to changes in baseline amplitude or to false onset detection compared to traditional threshold techniques (Allison 2003). Onset times were then categorized within an early- or a late-activation epoch in order to provide insight about whether changes in muscle coordination patterns occurred when responses are automated versus when responses are potentially under additional voluntary influence (Chan et al. 1979; Jacobs and Horak 2007). The early-phase response was defined from 50-150 ms after perturbation onset for the TA and GM muscles, and

from 50-120 ms after perturbation onset for all other muscles recorded from the trunk. Late-phase responses were defined from after the early-phase epoch to 325 ms after perturbation onset, thereby constraining the analysis within the 400-ms duration of the platform movement (Fig. 1). These epochs were chosen in order to examine the functional synergy of the postural response that contributes to balance recovery after any potential segmental spinal reflexes, which would occur through the first 50 ms after perturbation onset. In addition to isolating these epochs from segmental spinal reflexes, we chose to end the early-phase epoch at 120 ms for the trunk and 150 ms for the leg muscles in order to separate early and late epochs based on the time estimated for voluntary response latencies (Chan et al. 1979; Jacobs and Horak 2007). Each subject's percent of trials with an identifiable onset of muscle burst activity was then computed within the early- and late-phase epochs in order to derive each muscle's incidence of activation in response to the 12 directions of surface translations.

The amplitudes of EMG activation were generated by integrating the rectified EMG signals across five 75-ms epochs, commencing with a baseline activation epoch that began -75 ms from perturbation onset, followed by 4 sequential activation epochs spanning from 25-325 ms after perturbation onset. These five epochs, rather than the early- and late-phase epochs used to identify the incidence of EMG burst onset, were chosen because ongoing muscle activation after an onset could span multiple 75-ms epochs, thereby limiting inferences about whether integrated EMG amplitudes reflect muscle activation with potential voluntary influence. In order to facilitate subject group comparisons, each muscle's integrated EMG amplitudes were normalized to that muscle's maximum amplitude identified from any direction of perturbation and from any of the five epochs. This normalization procedure was necessary due to potential differences in sub-cutaneous fat between groups. A reference contraction generated by this automated postural

task appeared the most plausible choice for normalization rather than the typical maximum voluntary contraction because people with LBP may not be willing to generate a voluntary contraction to their maximum capability (Larivière et al. 2003).

Statistical Analysis

Two-factor generalized linear models evaluated differences between the subject groups (LBP versus no LBP) and among the 12 recorded muscles, with a covariate to correct for the effects of age. These models were applied to each direction of surface translation and each epoch of muscle activation. Muscle was chosen as the second factor in the model (as opposed to perturbation direction or epoch) in order to address our hypothesis that people with LBP exhibit global changes in muscle coordination patterns. When significant group-by-muscle interactions were evident (determined as a p-value < 0.05), post-hoc comparisons between groups for each muscle identified the contributors to the interaction. Bonferroni corrections were applied to these post-hoc comparisons in order to account for the 12 comparisons made on each muscle, rendering the level of significance at a p-value of 0.004. As reported in Table 1, measures of subject characteristics (age, height, weight, heel-to-heel stance widths, pain ratings, and disability scores) were compared using independent samples t-tests or Mann-Whitney tests depending on whether the data satisfied assumptions of normality (determined by Shapiro-Wilks tests), whereas the proportion of males and females was compared using a Fisher's exact test.

RESULTS

Subjects with LBP exhibited a significantly lower incidence of early-phase EMG activation bursts at the bilateral IO muscles and the left GM muscle primarily when responding to surface translations with a backward component (Table 2; Fig. 2). A significant group-by-muscle interaction was also evident in the right-backward direction, but post-hoc comparisons

between groups were not significant. To understand the more subtle contributors to this interaction effect, we identified significant differences in burst incidence between muscles that were evident for one group but not the other (Table 2). In response to right-backward translations, the interactions of the left TA with the left GM as well as the right EO and IO muscles were different for the groups with and without LBP. The burst incidence was not significantly different between the left TA and GM muscles for the group with LBP but was significantly different for the group without LBP. In addition, whereas the burst incidence was not significantly different among the left TA and right EO and IO muscles for the group without LBP, the burst incidence was higher for the left TA than the right EO and IO muscles for the group with LBP.

The group with LBP also exhibited a significantly lower incidence of EMG activation bursts during the late-phase epoch at every recorded muscle except the left GM and right RA muscles (Table 3). In contrast to the early-phase epoch, the directions of surface translation eliciting these significant group-by-muscle interactions were not as systematically constrained to a specific quadrant or hemisphere (Fig. 3).

Before correcting for age and for multiple comparisons, the subjects with LBP exhibited significantly higher amplitudes of normalized integrated EMG at the left ESP, RA, and GM muscles during the baseline epoch just prior to perturbation onset (Fig. 4). After correcting for age and multiple comparisons, the groups statistically differed only at the left ESP (Tables 4 and 5). Although the higher mean baseline amplitudes were consistent across directions of perturbation (as expected for translations of unpredictable direction and timing), the higher amplitudes reached statistical significance ($p < 0.004$) for the left ESP across 6 directions of impending translation. Following perturbation onset, integrated EMG amplitudes were not

significantly different across most muscles, epochs, and directions of surface translation, except the subjects with LBP exhibited significantly higher amplitudes at the left GM to rightward and right-backward translations 175-250 and 250-325 ms after perturbation onset, respectively (Tables 4 and 5).

Age did not represent a significant factor and including age as a covariate did not affect the group-by-muscle interaction statistics on most measures, except the baseline integrated EMG amplitudes. Age significantly affected baseline EMG amplitudes prior to four directions of impending surface translations [$F_{1,44}$ = 4.42, 4.50, 4.81, 5.88; P = 0.041, 0.040, 0.034, 0.020].

DISCUSSION

The results are consistent with the hypothesis that chronic, recurrent LBP associates with an intrinsic central change in the multi-segmental muscle coordination patterns of postural responses, during both the early and late response phases. Specifically, people with LBP exhibited higher normalized baseline EMG amplitudes at the abdomen and back as well as at the ankle, a lower incidence of EMG burst onsets at the distal leg and the trunk muscles, and higher normalized EMG amplitudes at the ankle musculature at least 175 ms after perturbation onset. These results, therefore, suggest that the subjects with LBP attempted to modulate their EMG responses to a balance disturbance from an activated baseline state rather than exhibiting acute burst activity from a quiescent state, perhaps stiffening the body to circumvent a multi-segmental response.

This postural response pattern is consistent with our laboratory's previous report on this overlapping subject sample, in which the subjects with LBP exhibited delayed and smaller displacements of the center of pressure with larger center-of-mass displacements (Henry et al. 2006). Delayed center-of-pressure displacements and larger center-of-mass displacements

299 suggest a loss of rapid hip and trunk flexion or extension because these hip movements are more
300 effective in rapidly moving the center of mass than ankle dorsi- or plantar-flexion (Kuo and
301 Zajac 1993). The results are also commensurate with interpretations that people with LBP exhibit
302 an inhibited hip strategy when maintaining balance in unstable standing conditions (Claeys et al.
303 2010; Mok et al. 2004), as well as with previously reported higher levels of antagonistic co-
304 contraction (Radebold et al. 2000) and of baseline EMG activity prior to perturbations (Stokes et
305 al. 2006). This decreased reliance on the hip strategy may therefore minimize forces and
306 movement about the trunk for people with a history of chronic or recurrent LBP.
307 A more detailed evaluation of each muscle's principle role to overcome the perturbation-
308 induced loss of balance suggested that the group with LBP exhibited fewer EMG bursts that
309 would contribute to trunk/hip flexion (i.e., the IO) and ankle plantar-flexion (i.e., the GM) during
310 the early epoch of responses to forward sway induced by backward perturbations. This result was
311 not precisely as predicted because we anticipated a lower incidence of burst onsets only at the
312 trunk, with higher burst incidence at the ankle. The lower incidence of burst onsets may be
313 explained, however, by the higher baseline activation evident in the subjects with LBP. The
314 coordination pattern of the early postural response is currently hypothesized to arise from the
315 triggering of a primed muscle synergy from within the central nervous system, which generates
316 coordinated muscle activations across the entire body in order to recover postural equilibrium
317 based on initial biomechanical configurations, environmental characteristics, and intentional
318 goals (Jacobs and Horak 2007). Given this study's identified differences in the incidence of burst
319 onsets at sites both proximal and distal from the location of the LBP during the early, automated
320 response phase, these results suggest that LBP associates with altered centrally-organized
321 response patterns or synergies.

Similar to the early-phase EMG burst activity, the lower incidence of EMG onset bursts during the late-phase response occurred in muscles that contributed to overcoming the initial induced body sway. In contrast to the early-phase EMG burst activity, the lower incidence of EMG onset bursts during the late-phase response was also often evident in response to directions of surface translation in which the muscles' activations would not contribute to recovering from the initial induced sway. This result suggests that a lack of muscle activation bursts sometimes represented a diminished contribution of the muscles to counteracting the perturbation-induced sway, but at other times may have represented fewer secondary antagonist muscle responses of an oscillating recovery (i.e., a response of an under-damped mechanical system). The lack of these secondary responses may result from increased stiffness or damping incurred by the higher baseline activation exhibited by the subjects with LBP as well as by a potential intention not to displace the trunk. Such a stiffened, inverted-pendulum response (as opposed to a multi-segmental response), however, may decrease overall stability in response to perturbations of this speed and amplitude (Henry et al. 2006; Ishida et al. 2008), and explain the need for higher GM activation amplitudes demonstrated in this study.

A possible limitation to the study relates to the potential that the lower incidence of burst onsets for the group with LBP might have resulted from an inability to detect an onset due to higher baseline amplitudes (Lee et al. 2007). We are confident that our methods minimized this potential error through the integrated protocol method (Allison 2003) and use of visual inspection. In addition, higher baseline amplitudes were evident for the group with LBP in only the left ES, RA, and GM muscles, whereas significant differences in burst incidence were evident across nearly all muscles recorded. Thus, it is unlikely that the lower incidence of burst onsets with LBP can be explained by an insensitivity to identify burst onsets.

In summary, the centrally organized change in muscle coordination patterns of people with LBP did not simply represent a diminished hip strategy with an enhanced ankle strategy; rather it appears that those with LBP exhibited a lower incidence of acute burst activity across both the ankle and trunk muscles through a higher baseline activation state that may have contributed to increased stiffness in the system. Although at the expense of maintaining stable stance (Henry et al. 2006), such a strategy not only corresponded with a lower incidence of muscle burst activity associated with counteracting the initial induced body sway, but also corresponded with a lower incidence of burst activity at the trunk during secondary (antagonistic) responses. It remains unclear whether these LBP-associated changes in response strategies are beneficial or harmful to the chronicity or recurrence of LBP. Thus, these results suggest the need for future interventional studies on reactive postural control to address these LBP-associated changes in central motor programming in order to determine their benefit on LBP and postural stability.

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DISCLOSURES

The authors have no conflicts of interest, financial or otherwise, to disclose.

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FIGURE LEGENDS

Fig. 1. Schematic of the directions of surface translation with their induced body sway as well as representative EMG. The graphs of representative EMG illustrate the directional dependence and time characteristics of EMG burst activity for antagonistic muscles of the trunk and ankle: from top to bottom, the erector spinae at the 3rd lumbar segment (ESP), rectus abdominus (RA), gastrocnemius medialis (GM), and the tibialis anterior (TA). Black traces represent responses to forward translations that induce backward body sway; gray traces represent responses to backward translations that induce forward sway. The dashed gray boxes identify the early-phase epochs of the ankle- and trunk-muscle responses (50-150 ms and 50-120 ms, respectively); the solid gray boxes identify the late-phase epochs.

Fig. 2. Incidence of early-phase EMG burst onsets. The body schematics illustrate the locations of each muscle by black-outlined ellipses; those filled with gray exhibit significant post-hoc differences between groups following a significant group-by-muscle interaction. Approximate to these muscle locations are spoke wheels that represent each direction of support surface translation. Thick, black lines in the spoke wheel identify the directions of translation with significant group-by-muscle interactions.

Fig. 3. Incidence of late-phase EMG burst onsets. Layout, line types, and fill colors are formatted as defined for figure 2.

Fig. 4. Group mean baseline integrated EMG amplitudes. The vertical axis presents the integrated EMG amplitudes of the baseline epoch (75 ms prior to translation onset) as a percentage of each muscle's maximum activation amplitude exhibited within any of the five 75-ms epochs. The horizontal axis at the bottom of the chart that extends from left to right lists each muscle with a prefix of "l" or "r", representing the left or right side of the body, respectively.

Muscles with significant post-hoc differences between groups following a significant group-by-muscle interaction are highlighted by an asterisk when evident before correction for age or multiple comparisons and by a black box when evident after these corrections. The horizontal axis on the right side of the chart lists the directions of surface translation, ordered counterclockwise from bottom to top and starting from rightward translations (the diagonal directions of translation are not labeled due to space constraints). The black traces represent the grand averages for the group without LBP and the gray traces represent the grand averages for the group with LBP.

Table 1. *Group Characteristics*

	Participant Group		Statistic (P-Value)
	With LBP	Without LBP	
Number (Female, Male)	24 (11, 13)	21 (13, 8)	Fisher's $Chi^2 = 1.16$ (P = 0.37)
Mean (95% CI) Age	40 (35-55) yr	33 (29-38) yr	Mann-Whitney Z = 1.74 (P = 0.08)
Mean (95% CI) Height	1.73 (1.68-1.78) m	1.70 (1.67-1.74) m	T-Test = 0.98 (P = 0.33)
Mean (95% CI) Weight	75 (69-81) kg	68 (62-73) kg	T-Test = 1.65 (P = 0.11)
Mean (95% CI) Heel-to-Heel Stance Width	20.6 (18.5-22.7) cm	21.5 (18.6-24.3) cm	T-Test = 0.48 (P = 0.63)
Median (range) Numeric Pain Rating	2 (0-4)	0 (0-1)	Mann-Whitney Z = 3.87 (P < 0.0005)
Median (range) Roland Morris Disability Score	2 (0-9)	0 (0-1)	Mann-Whitney Z = 4.47 (P < 0.00001)

Table 2. *Group-by-Muscle Interaction Statistics on the Incidence of Muscle Burst Onsets During the Early Response Epoch*

Direction of Perturbation	Group-by-Muscle Statistic	Significant (P < 0.004) Post-Hoc Statistics	Percent of Trials With Muscle Onset by Group		
			Mean LBP	Mean No LBP	99.6% CI for the Difference in Means
Right	$F_{11,506}$=0.93 P=0.51				
Right-Forward	$F_{11,506}$=2.13 P=0.017	lGM: T_{506}=4.17;P<0.0001	lGM: 4	lGM: 36	lGM: 10-55
Forward-Right	$F_{11,506}$=1.02 P=0.43				
Forward	$F_{11,484}$=0.60 P=0.83				
Forward-Left	$F_{11,506}$=1.13 P=0.34				
Left-Forward	$F_{11,495}$=0.87 P=0.57				
Left	$F_{11,495}$=1.02 P=0.42				
Left-Backward	$F_{11,506}$=2.13 P=0.017	lIO: T_{506}=3.73;P=0.0002 rIO: T_{506}=3.66;P=0.0003	lIO: 10 rIO: 3	lIO: 35 rIO: 28	lIO: 6-46 rIO: 5-45
Backward-Left	$F_{11,506}$=2.85 P=0.0013	lGM: T_{506}=5.36;P<0.0001 rIO: T_{506}=3.58;P=0.0004	lGM: 19 rIO: 4	lGM: 59 rIO: 31	lGM: 18-61 rIO: 5-48
Backward	$F_{11,407}$=1.85 P=0.044	lGM: T_{407}=3.20;P=0.0015	lGM: 30	lGM: 56	lGM: 2-49
Backward-Right	$F_{11,495}$=1.89 P=0.039	lGM: T_{495}=3.17;P=0.0016	lGM: 38	lGM: 62	lGM: 2-45
Right-Backward	$F_{11,495}$=2.08 P=0.020	No Direct Comparisons; Significant differences among lTA with lGM, rEO, rIO differ between groups	lTA: 26 lGM: 43 rEO: 6 rIO: 5	lTA: 20 lGM: 61 rEO: 17 rIO: 21	$LBP_{lGM-lTA}$: -1-36 $noLBP_{lGM-lTA}$: 22-61 $LBP_{rEO-lTA}$: 1-38 $noLBP_{rEO-lTA}$: -18-22 $LBP_{rIO-lTA}$: 2-39 $noLBP_{rIO-lTA}$:-21-19

526 **Table 3.** *Group-by-Muscle Interaction Statistics on the Incidence of Muscle Burst Onsets During*

527 *the Late Response Epoch*

Direction of Perturbation	Group-by-Muscle Statistic	Significant (P < 0.004) Post-Hoc Comparisons	Percent of Trials With Muscle Onset By Group		
			Mean LBP	Mean No LBP	99.6% CI for the Difference in Means
Right	$F_{11,506}$=2.10 P=0.019	rEST: T_{506}=3.85;P=0.0001	rEST: 14	rEST: 49	rEST: 9-61
Right-Forward	$F_{11,506}$=1.97 P=0.030	lESP: T_{506}=3.06;P=0.0023 rESP: T_{506}=3.23;P=0.0013 lRA: T_{506}=3.38;P=0.0008	lESP: 19 rESP: 9 lRA: 15	lESP: 49 rESP: 41 lRA: 48	lESP: 2-58 rESP: 3-60 lRA: 5-61
Forward-Right	$F_{11,506}$=2.26 P=0.011	lIO: T_{506}=3.01;P=0.0027 rIO: T_{506}=3.90;P=0.0001	lIO: 19 rIO: 15	lIO: 50 rIO: 55	lIO: 0.1-61 rIO: 10-70
Forward	$F_{11,484}$=1.45 P=0.15				
Forward-Left	$F_{11,506}$=2.11 P=0.019	lESP: T_{506}=3.24;P=0.0013 lIO: T_{506}=4.05;P<0.0001 lRA: T_{506}=3.35;P=0.0009	lESP: 25 lIO: 16 lRA: 30	lESP: 57 lIO: 57 lRA: 64	lESP: 4-62 lIO: 12-70 lRA: 5-63
Left-Forward	$F_{11,495}$=2.58 P=0.0034	lESP: T_{495}=3.32;P=0.0010 rIO: T_{495}=3.28;P=0.0011	lESP: 10 rIO: 13	lESP: 43 rIO: 45	lESP: 4-61 rIO: 4-61
Left	$F_{11,495}$=1.93 P=0.034	lEO: T_{495}=5.54;P<0.0001 lESP: T_{495}=3.17;P=0.0016 lEST: T_{495}=3.10;P=0.0021 lIO: T_{495}=3.21;P=0.0014 rIO: T_{495}=4.10;P<0.0001	lEO: 34 lESP: 15 lEST: 21 lIO: 6 rIO: 8	lEO: 83 lESP: 42 lEST: 48 lIO: 34 rIO: 44	lEO: 23-74 lESP: 3-53 lEST: 2-52 lIO: 3-53 rIO:11-61
Left-Backward	$F_{11,506}$=1.43 P=0.16				
Backward-Left	$F_{11,506}$=2.24 P=0.012	lEO: T_{506}=4.14;P<0.0001 lEST: T_{506}=3.89;P=0.0001 rEST: T_{506}=3.03;P=0.0026 rIO: T_{506}=3.26;P=0.0012	lEO: 7 lEST: 40 rEST: 31 rIO: 9	lEO: 44 lEST: 75 rEST: 58 rIO: 38	lEO: 11-64 lEST: 9-61 rEST: 1-54 rIO: 3-56
Backward	$F_{11,407}$=1.56 P=0.11				
Backward-Right	$F_{11,495}$=2.05 P=0.023	lTA: T_{495}=3.27;P=0.0011 rEO: T_{495}=4.01;P<0.0001 lESP: T_{495}=3.14;P=0.0018 lEST: T_{495}=3.07;P=0.0022 lIO: T_{495}=4.19;P<0.0001 rIO: T_{495}=3.04;P=0.0025	lTA: 12 rEO: 9 lESP: 36 lEST: 25 lIO: 9 rIO: 12	lTA: 44 rEO: 48 lESP: 66 lEST: 55 lIO: 46 rIO: 41	lTA: 4-60 rEO: 11-68 lESP: 2-59 lEST: 2-58 lIO: 13-69 rIO: 1-58

Right-Backward	$F_{11,495}$=3.91 P<0.0001	lTA: T_{495}=2.97;P=0.0031 rEO: T_{495}=3.76;P=0.0002 lESP: T_{495}=4.94;P<0.0001 rESP: T_{495}=3.71;P=0.0002 lEST: T_{495}=3.12;P=0.0019 rEST: T_{495}=4.23;P<0.0001	lTA: 20 rEO: 19 lESP: 13 rESP: 26 lEST: 25 rEST: 23	lTA: 48 rEO: 55 lESP: 60 rESP: 61 lEST: 54 rEST: 63	lTA: 1-55 rEO: 8-62 lESP: 19-73 rESP: 8-62 lEST: 2-56 rEST: 13-66

528 **Table 4.** *Group-by-Muscle Interaction Statistics on the Amplitudes of Integrated EMG Activity*

Direction of Perturbation	Group-by-Muscle Interaction Statistics For Each Recording Epoch				
	Baseline (-75 to 0 ms)	Epoch 1 (25-100 ms)	Epoch 2 (100-175 ms)	Epoch 3 (175-250 ms)	Epoch 4 (250-325 ms)
Right	$F_{11,495}$=1.80 P=0.052	$F_{11,477}$=0.73 P=0.71	$F_{11,477}$=0.84 P=0.60	$F_{11,477}$=1.98 P=0.029	$F_{11,477}$=1.29 P=0.22
Right-Forward	$F_{11,495}$=1.97 P=0.030	$F_{11,476}$=0.56 P=0.86	$F_{11,476}$=0.33 P=0.98	$F_{11,476}$=1.00 P=0.44	$F_{11,476}$=1.23 P=0.27
Forward-Right	$F_{11,495}$=1.97 P=0.030	$F_{11,479}$=0.56 P=0.86	$F_{11,479}$=0.45 P=0.93	$F_{11,479}$=0.34 P=0.98	$F_{11,479}$=0.31 P=0.98
Forward	$F_{11,473}$=1.59 P=0.098	$F_{11,462}$=0.64 P=0.79	$F_{11,462}$=0.66 P=0.78	$F_{11,462}$=0.64 P=0.79	$F_{11,462}$=0.71 P=0.73
Forward-Left	$F_{11,495}$=2.00 P=0.027	$F_{11,479}$=0.79 P=0.65	$F_{11,479}$=0.53 P=0.88	$F_{11,479}$=0.64 P=0.79	$F_{11,479}$=0.49 P=0.91
Left-Forward	$F_{11,484}$=2.05 P=0.023	$F_{11,466}$=0.96 P=0.48	$F_{11,466}$=0.47 P=0.92	$F_{11,466}$=0.30 P=0.99	$F_{11,466}$=0.57 P=0.85
Left	$F_{11,484}$=1.88 P=0.040	$F_{11,466}$=0.64 P=0.80	$F_{11,466}$=1.01 P=0.43	$F_{11,466}$=0.95 P=0.49	$F_{11,466}$=0.54 P=0.88
Left-Backward	$F_{11,495}$=1.56 P=0.11	$F_{11,477}$=0.64 P=0.80	$F_{11,477}$=0.99 P=0.45	$F_{11,477}$=0.62 P=0.81	$F_{11,477}$=0.50 P=0.90
Backward-Left	$F_{11,495}$=1.49 P=0.13	$F_{11,476}$=0.76 P=0.68	$F_{11,476}$=0.95 P=0.50	$F_{11,476}$=0.32 P=0.98	$F_{11,476}$=1.19 P=0.29
Backward	$F_{11,407}$=1.13 P=0.34	$F_{11,401}$=0.59 P=0.83	$F_{11,401}$=0.63 P=0.81	$F_{11,401}$=1.59 P=0.098	$F_{11,401}$=0.30 P=0.99
Backward-Right	$F_{11,484}$=1.35 P=0.19	$F_{11,472}$=0.81 P=0.63	$F_{11,472}$=1.05 P=0.40	$F_{11,472}$=0.37 P=0.97	$F_{11,472}$=0.83 P=0.61
Right-Backward	$F_{11,484}$=2.04 P=0.024	$F_{11,465}$=0.73 P=0.71	$F_{11,465}$=1.50 P=0.13	$F_{11,465}$=0.99 P=0.46	$F_{11,465}$=1.99 P=0.028

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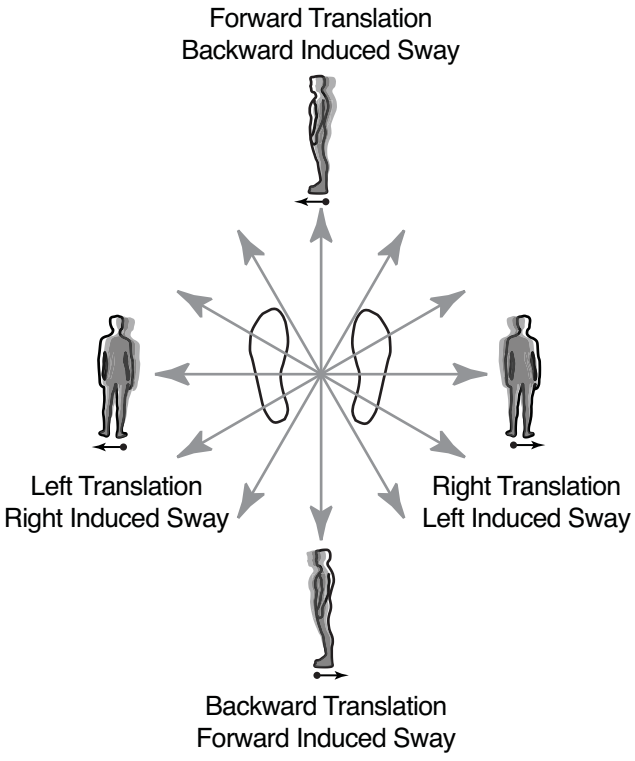
Table 5. *Significant Post-Hoc Comparisons on the Amplitudes of Integrated EMG Activity*

Direction of Perturbation	Significant ($P < 0.004$) Post-Hoc Comparisons	Normalized Integrated EMG Amplitudes By Group		
		Mean LBP	Mean No LBP	99.6% CI for the Difference in Means
Right	lGM_epoch3: $T_{477}=3.80;P=0.0002$	lGM_{epoch3}: 49	lGM_{epoch3}: 28	lGM_{epoch3}: 5-36
Right-Forward	lESP_baseline: $T_{495}=3.82;P=0.0002$	$lESP_{base}$: 81	$lESP_{base}$: 15	$lESP_{base}$: 16-115
Forward-Right	lESP_baseline: $T_{495}=3.65;P=0.0003$	$lESP_{base}$: 75	$lESP_{base}$: 16	$lESP_{base}$: 12-106
Forward				
Forward-Left	lESP_baseline: $T_{495}=3.53;P=0.0005$	$lESP_{base}$: 79	$lESP_{base}$: 17	$lESP_{base}$: 11-106
Left-Forward	lESP_baseline: $T_{484}=3.67;P=0.0003$	$lESP_{base}$: 79	$lESP_{base}$: 17	$lESP_{base}$: 13-111
Left	lESP_baseline: $T_{484}=3.56;P=0.0004$	$lESP_{base}$: 80	$lESP_{base}$: 18	$lESP_{base}$: 12-112
Left-Backward				
Backward-Left				
Backward				
Backward-Right				
Right-Backward	lESP_baseline: $T_{484}=3.78;P=0.0002$ lGM_epoch4: $T_{477}=3.69;P=0.0002$	$lESP_{base}$: 80 lGM_{epoch4}: 48	$lESP_{base}$: 16 lGM_{epoch4}: 33	$lESP_{base}$: 15-113 lGM_{epoch4}: 3-27

Fig. 1


Forward Translation
Backward Induced Sway
Left Translation
Right Induced Sway
Right Translation
Left Induced Sway
Backward Translation
Forward Induced Sway

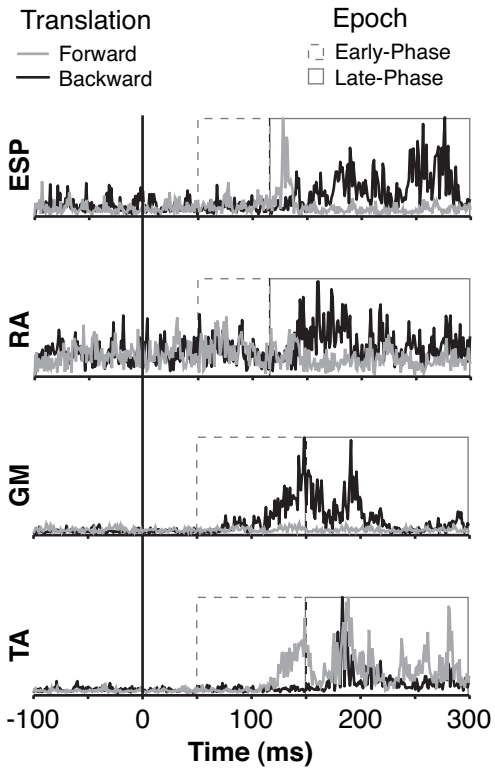

Translation
Forward
Backward
Epoch
Early-Phase
Late-Phase
ESP
RA
GM
TA
-100
0
100
200
300
Time (ms)

Fig. 2

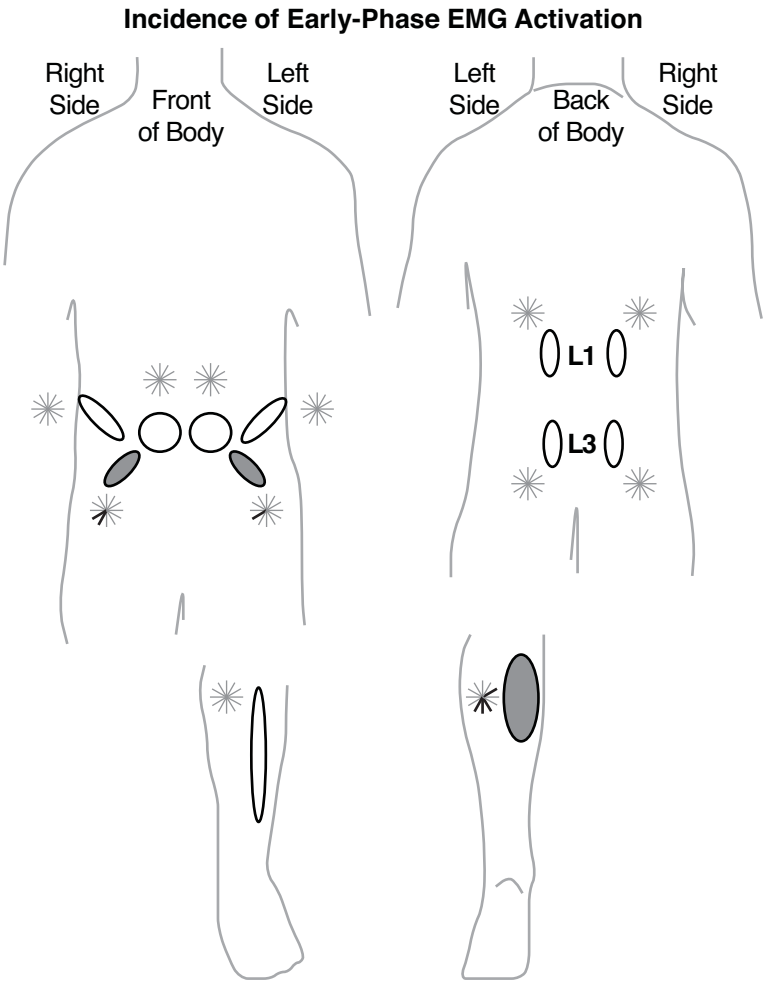

Incidence of Early-Phase EMG Activation
Right Side
Front of Body
Left Side
Left Side
Back of Body
Right Side
L1
L3

Fig. 3

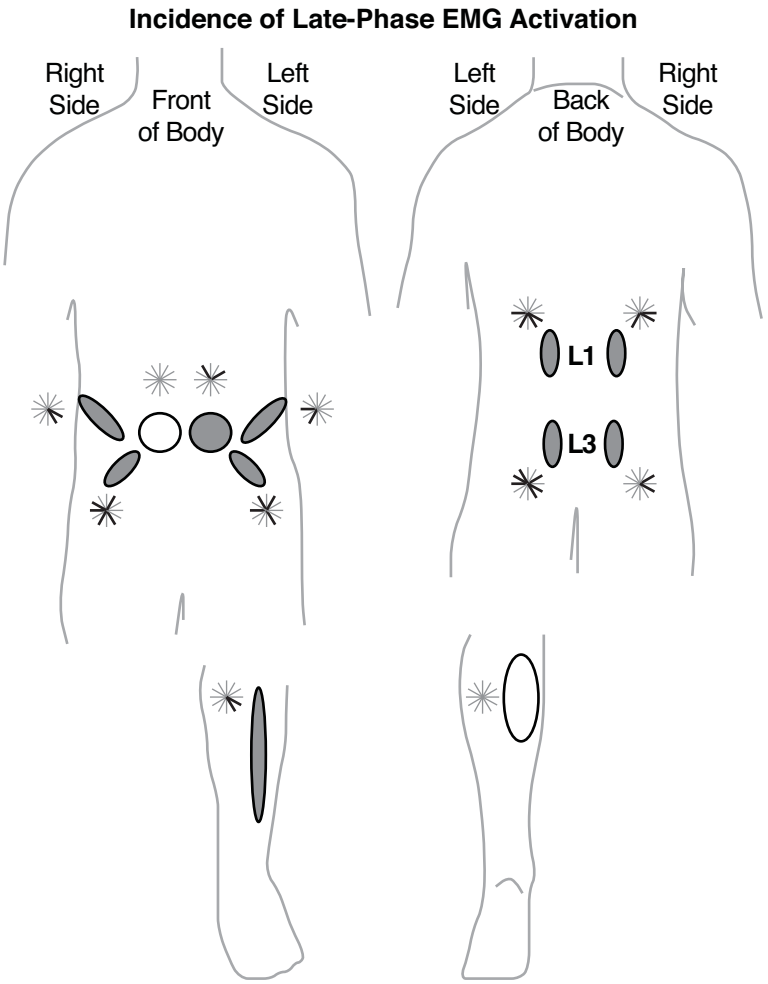
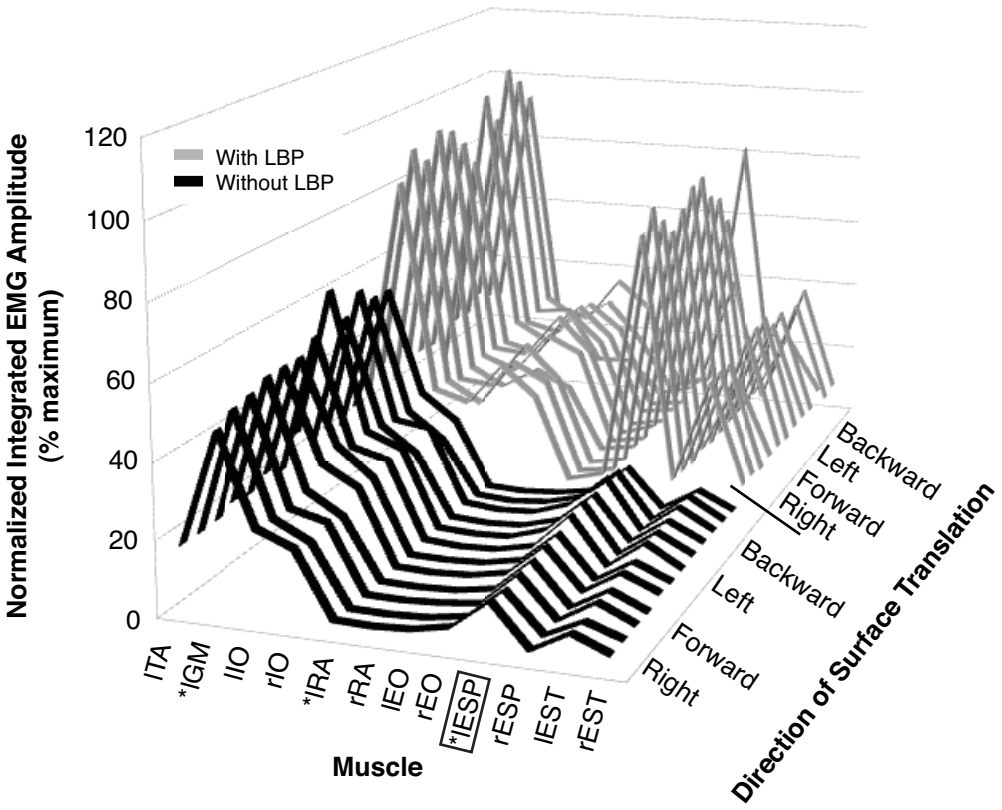

Incidence of Late-Phase EMG Activation
Right Side
Front of Body
Left Side
Left Side
Back of Body
Right Side
L1
L3

Fig. 4


Normalized Integrated EMG Amplitude (% maximum)
120
100
80
60
40
20
0
With LBP
Without LBP
lTA
*lGM
lIO
rIO
*lRA
rRA
lEO
rEO
*lESP
rESP
lEST
rEST
Muscle
Backward
Left
Forward
Right
Backward
Left
Forward
Right
Direction of Surface Translation