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Temporal patterns of the trunk muscles remain altered in a low back injured population despite subjective reports of recovery

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Title:

Temporal patterns of the trunk muscles remain altered in a low back injured population despite subjective reports of recovery

Abstract

Objective: To compare temporal activation patterns from twenty-four abdominal and lumbar muscles between healthy subjects (ASYM) and those who reported recovery from recent low back injury (LBI).

Design: Cross-sectional comparative study

Setting: University Neuromuscular Function Laboratory

Participants: 81 healthy adult volunteers: 30 LBI, 51 ASYM

Interventions: Trunk muscle EMG activity was collected during two difficulty levels of a supine trunk stability test aimed at challenging lumbo-pelvic control.

Main Outcome Measures: Principal component (PC) analysis was applied to determine differences in temporal and/or amplitude EMG patterns between groups. Mixed model ANOVAs were performed on PC scores that explained more than 89% of the variance (α=0.05).

Results: Four PCs explained 89% and 96% of the variance for the abdominal and back muscles, respectively, with both muscle groups having similar shapes in the first 3 PCs. Significant interactions or group main effects were found for all PC scores except PC4 for the back extensors. Overall activation amplitudes for both the abdominal and back muscles (PC1 scores) were significantly ($p<0.05$) higher for the LBI group, with both abdominal and back muscles of the LBI group demonstrating increased response to the leg loading phase (PC2 scores) compared to the ASYM. Differences were also found between groups in their preparatory activity (PC3 scores) with LBI group having higher early relative amplitude of abdominal and back extensor activity.

Conclusions: Despite perceived readiness to return to work and low pain scores, muscle activation patterns remained altered in this LBI group, including reduced synergistic co-activation, increased overall amplitudes as well as greater relative amplitude differences during specific phases of the movement. EMG measures provide objective information to help guide therapy and may assist with determining level of healing and return-to-work readiness following a low back injury.

Key Words. Exercise movement techniques; principal component analyses; electromyography; abdominal muscles, back pain

List of Abbreviations:

LBP: low back pain

LBI: low back injury

TST: trunk stability test

EMG: electromyography

PCA: principal component analysis

ASYM: asymptomatic

MVC: maximum voluntary contraction

PC: principal component

FOB: flock of birds motion capture system

ANOVA: analysis of variance

Muscle abbreviations (R or L in front of the abbreviation indicate right or left side)

- URA: upper rectus abdominis
- LRA: lower rectus abdominis
- IO: internal oblique
- EO: external oblique
- L48: quadratus lumborum electrode site; 4th lumbar vertebrae level, 8 cm lateral
- L52: multifidus electrode site: 5th lumbar vertebrae, 2 cm lateral
- ES: erector spinae

L13, L16: erector spinae electrode sites; first lumbar vertebrae level, 3 and 6 cm lateral, respectively

L33, L36: erector spinae electrode sites: 3rd lumbar vertebrae level, 3 and 6 cm lateral, respectively

The trunk musculature is required to have sufficient strength and produce forces in a coordinated manner to maintain spinal stability in response to external forces associated with different tasks.[1-4] There is consistent evidence of decreased trunk muscle strength and altered muscle activation patterns in chronic low back pain (LBP) populations,[5-11] with the latter supported by reports of muscle fibre atrophy in specific trunk muscles for low back pain populations[5, 12, 13]. The former can be attributed to altered proprioception,[14, 15] decreased reflex responses[16] as well as an alterations in motor control for chronic low back pain populations.[10, 17-19] Less clear is what happens early in the injury–pain process before a chronic condition develops. Of the few studies that have examined those who would not be classified as chronic, trunk muscle strength deficits were found for those who were in remission following a LBI.[20] In addition, lumbar multifidus activation onset times remain altered while performing different tasks despite remission from symptoms.[21-23] A recent study showed that the relative relationship among a comprehensive set of abdominal and back muscle activation amplitudes were different during a standard lift and replace task[24] as were the temporal patterns during a dynamic transfer task[25] in individuals deemed recovered (minimal pain and dysfunction) from a recent low back injury (LBI)(< 12 weeks). At follow-up, the amplitude and temporal patterns varied more in the group that re-injured than the group that did not re-injure, compared to non-LBI controls.[25] Collectively, these studies support the need for interventions early after an injury or pain episode, and given the high rates of recurrence[26] do not support that these episodes should be left to resolve themselves.

Rehabilitation following a LBI frequently incorporates lumbar and pelvic stabilization exercises, which aim to challenge muscle strength through increasing demands or temporal responses to dynamic perturbations, but there is a need to optimize the balance between lumbo-pelvic stability and muscle-induced compressive forces.[27-31] An example, the trunk stability test (TST) challenges the neuromuscular system, primarily the abdominal muscles, to maintain a neutral lumbo-pelvic position in supine, while leg flexion/extension manoeuvres alter the applied external moment.[32-34] The TST protocol is used both to assess function and as an exercise progression, with different levels of increasing difficulty.[33, 34] Abdominal muscle amplitude differences[19] between healthy and chronic LBP (>3 months) populations as well as more asynchronous co-activation patterns among abdominal and back extensor muscles[10] were reported for the TST. Determining whether electromyographic (EMG) pattern alterations are present in pain-free individuals who have recently recovered from a LBI could provide an objective assessment of recovery following a LBI and information to assist in clinical management decisions related to recovery and exercise progression.

Given the high rates of repeat injury and pain episodes reported in the literature, the purpose of the study was to compare the neuromuscular recruitment patterns from a comprehensive set of trunk muscles between individuals deemed recovered from a LBI and controls with no LBI during the TST exercise protocol, which provides a dynamic challenge to the lower spine and pelvis. Based on the findings from the chronic LBP literature and the few studies on remission and recovery we hypothesized that the LBI group would have i) higher abdominal activation, ii) higher relative agonist-antagonist co-activation and iii) more temporal asynchronies among muscles, with both temporal and amplitude differences modulated by changing task demand. Principal component analysis (PCA) is a multivariate statistical technique that has been applied to reduce the dimensionality of large data sets by examining the main patterns found in a data set and reducing them into a smaller number of variables that allow statistical testing of differences in these key patterns[35]. This technique has been applied to EMG waveforms and it characterizes both amplitude and temporal characteristics,[10, 36-38] providing an objective method for examining differences not only in relative amplitudes among a large number of muscles reflecting different demands, but also the temporal synergies or coordination among muscles, a characteristic deemed important to spinal stability during dynamic tasks.[1, 2, 4] The goal is to shed light on our understanding of the alterations that exist in a LBI group, after symptoms have diminished.

Methods

Participants were recruited via advertisements and electronic notices posted at Dalhousie University and 3 from local physiotherapy practices. The LBI group included individuals with a bout of LBP resulting from a specific event, requiring modification of daily activities, within 12 weeks prior to their test session. At time of testing, each participant self-reported minimal residual pain and perceived they were capable of resuming regular activities. Asymptomatic participants (ASYM) reported no recent history of LBP (within one year) and no LBI resulting in time lost from normal activities or requiring medical attention. All participants reported no cardiovascular, neurological or other orthopaedic conditions based on a health screening questionnaire, postural and neurological assessments. Thirty LBI (16 women) and fifty-one ASYM (27 women) participated in this study (Table 1) after signing an Institution's Research Ethics Board approved informed consent. Participants were instructed in the test procedure at an introductory session, occurring within 2 weeks prior to testing. All reported no previous familiarization with the TST. Demographic data, standing pelvic angle and abdominal function[39] were recorded. Written exercise instructions were provided for participants to practice 10 times each on 3 separate days prior to testing.

Test procedure

Each participant completed 2 levels of the TST leg loading task in supine lying: level one required the leg to be lightly supported on the bed during the leg extension phase, whereas the limb was held aloft in level 2. The TST is more fully described in the Figure 1 caption. There were 3 trials at each level; the order of level was randomly assigned, with one minute rest between trials. Exercise performance was judged correct if leg-lifting, leg-extension and leg-lowering phases occurred with minimal observable motion of the lumbar spine and pelvis to an 8-second count.[33]

Normalization trials

Nine previously described maximum voluntary muscle isometric contractions (MVCs)[40] were elicited following the test trials for EMG amplitude normalizations. MVCs were randomized, with two trials of each 3-s MVC performed in succession, and a 2-min rest between trials.

Event markers

Conductive metal strips were attached to the right heel, anterio-distal thigh, wooden contact frame and bed. When the thigh or foot were in contact with the frame or bed, a circuit was completed, providing event markers which temporally divided the TST into 3 phases: 1) right leg lifting from crook lying position to 90° hip and knee flexion ; 2) right hip and knee extension, then flexing back to 90°; and 3) right leg lowering to the start position (Figure 1). These event data were synchronized with the pelvis position and EMG data.

Surface EMG data collection and processing

Surface electrodes (Meditrace Ag/AgCl, 10mm)[a] were placed in a bipolar configuration (30 mm interelectrode distance) over 12 muscle sites bilaterally following standard skin preparation (skin/amplifier impedance ratio: < 0.1%) and electrode placement.[40, 41] Abdominal muscle sites included upper and lower rectus abdominis (URA, LRA), internal oblique (IO) and 3 external oblique sites (EO1-3) representing anterior, lateral and posterior fibres, respectively. Posterior sites included quadratus lumborum (L48), multifidus (L52), erector spinae (ES) at L1 and L3 levels, both 3cm and 6cm from the midline, representing longissimus and iliocostalis sites, respectively (L13, L16, L33, L36). Electrode placement was validated using a series of manual muscle tests.[32, 39] EMG signals were pre-amplified (200X) and further amplified using three AMT-8 EMG systems[b] (band pass 10–1000 Hz; CMRR=115db, input impedance 10GΩ). Raw EMG signals and step voltage event markers were digitized (2000 samples/s) using a National Instruments analog-to-digital conversion board (16-bit resolution) and Labview[c] software (version 7). EMG data were full wave rectified and low pass filtered (6 Hz) using a second order Butterworth recursive filter yielding a linear envelope profile. Data from right foot-off to right foot-on were time normalized to 100% using a linear interpolation algorithm, then amplitude normalized to the appropriate MVC.

EMG ensemble average waveforms for each participant (81), muscle (12) and condition (2) were entered into a PCA model (1944x101) for back extensors and abdominals separately.[36] Briefly, a covariance matrix was calculated for the abdominals and back extensors separately and then an Eigen vector decomposition was performed on the data, for which the Eigen vectors are a set of principal components (PCs) that capture the key features from EMG waveform data.[36] For each EMG waveform, a PC score was calculated, providing a weighting factor for the contribution of the PC to the measured EMG waveform. Waveforms with similar amplitude and shape have similar PC scores. Statistical testing of PC scores provides a quantitative comparison of EMG waveform patterns. PCs explaining approximately 90% of variance were included in the statistical analysis.[10, 36]

Motion Capture

An electromagnetic Flock of Birds Motion Capture system[d] (FOB) recorded 3D angular motion of the pelvis throughout the TST via a sensor placed superior to the left anterior superior iliac crest. Maximum pelvic displacement over the entire exercise was calculated in the global coordinate system which approximates anatomical references (X=frontal, Y=transverse, Z=sagittal plane). These measures were used to verify visual observation of no motion during testing.

Statistical Analysis

Independent Student t-tests were performed on demographic variables (Table 1). Mixed model (group, exercise level) analysis of variance models (ANOVA) were performed on time to complete the exercise and pelvis angular displacements. Three-factor (group/exercise level/muscle) mixed model ANOVAs tested for main effects and interactions for abdominals and back extensor muscles separately. Bonferroni post-hoc tests were performed when appropriate. All analyses were performed using Minitab™ (version 16)[e], $\alpha = 0.05$.

Results

The LBI group was tested 6.5±3 weeks after their injury. At the time of testing, mean pain VAS score was 16.4±19 out of 100 and Roland Morris disability score was 4.5±5 out of 24. Sixteen LBI participants reported that their original pain had been focused on the right side, 5 on the left side and 9 reported central symptoms or were uncertain. There were no significant differences in time taken or pelvis motion between exercise levels. LBI group was significantly older, took slightly longer, and demonstrated less pelvic motion in all three planes (Table 1).

Four PC patterns captured 96% and 89% of variance in the back and abdominal muscle waveforms, respectively (Figure 2). PC shapes were similar between back and abdominal muscles, except PC4 (Figure 2). PC1 captured overall muscle activation amplitude and shape. Higher PC1 scores would be indicative of higher overall muscle activation.[36] PC1 included an initial burst of activity upon left leg lift (late Phase 1) with another peak during left leg lowering (early Phase 3). The abdominals demonstrated a slightly higher increase in amplitude mid-task than the back extensors. PC2 captured the higher relative activity during Phase 2, as the right leg extended and flexed again, when compared to Phases 1 and 3. High PC2 scores indicate greater muscle response to right leg extension/flexion (Phase 2). This, and subsequent PCs, would be additive to PC1. PC3 captured higher activation amplitudes early in Phase 1, as the participants were asked to “pull their abdomen up and in”, and to a lesser extent late in Phase 3 as the right leg lowered. PC4 captured an increased burst in abdominal activity slightly before left leg raise in Phase 1 and a distinctive drop during right leg lowering (Phase 3). Back extensor PC4 demonstrated high initial activity during the abdominal hollowing, followed by a gradual continual decrease over the entire exercise (Figure 2g,o). For completeness, Tables 2 and 3 include the mean and standard deviations for the associated interactions and main effects. The significant ($p<0.05$) interactions and main effects are indicated in Table 4.

Abdominals:

There was a significant group*level*muscle interaction for PC1, PC2 and PC4 scores whereas PC3 had significant two-way interactions (Table 4). LBI group PC1 scores were significantly higher than ASYM for every muscle, both levels (Figure 3a). LEO2 in the LBI group (both levels) was significantly higher than all other muscles within level (excepting LEO1/Level 2), whereas the highest score in the ASYM group was LEO1 (Figure 3a). Other significant PC1 findings are illustrated in Figure 3a.

Post hoc results showed significant L>R asymmetry in the LBI group PC2 scores at IO (both levels) and EO3 (Level 2) (Appendix 1, Figure 3b). Level 1 LBI scores were significantly lower than the ASYM at LEO2 and RIO, but higher at LEO1, LEO3 and REO1 in Level 2 (Figure 3b).

PC3 post-hoc results for the group*muscle interaction are indicated in Figure 3c, showing significantly higher LBI group scores at 8 muscle sites, but not the 4 left oblique sites. In both groups, IOs were significantly higher than all other sites. All ASYM EO scores were significantly higher than the RAs (excepting REO3and RURA), whereas in the LBI group, only REO1 was significantly higher than all RAs.

Post-hoc analysis of the group*level interaction indicated higher PC3 scores in the LBI group at both levels. Mean (SD) PC3 scores were -1.1(28), 6.4(31), -3.3(20), 0.2(19) for the LBI group, levels 1 and 2, and the ASYM group, levels 1 and 2, respectively. Both groups demonstrated significant between-level increases.

Post hoc results reveal significantly higher PC4 scores in ASYM group (Level 1) at 4 RA sites only (Figure 3d). In Level 2, all sites were significantly higher in ASYM group, except LEO2, LEO3 and LIO. The LBI group had no significant changes in individual muscle PC4 scores with increasing level, whereas the ASYM group had significant increases at 7 sites (Figure 3d). The ASYM group had 5 significant L/R differences in Level 1 and one in Level 2, while the LBI group had 3 differences in Level 1, and 5 in Level 2. Thus, the LBI group became more asymmetrical with increased demand. PC4 scores were consistently higher on the left.

Back Extensors:

PC1 explained 91.5% of variance in the back extensors, indicating minimal variation in activation patterns among muscles. Group*level post-hoc analysis showed mean LBI PC1 scores were significantly higher than ASYM at both levels, with mean (SD) scores being 13.8(48), 16.3(49), -8.8(32) and -8.7(31) for the LBI group (levels 1,2) and ASYM group (levels 1,2), respectively. Only the LBI group showed a significant between-level increase.

Group*muscle post-hoc analysis revealed significantly higher PC2 scores in the LBI group at L1 sites (L13, L16) bilaterally, LL36 and LL48, but significantly lower at RL52 (Figure 4a). Left-sided PC2 scores were consistently higher than right, excepting ASYM/L52.

A group*muscle interaction showed significantly higher LBI PC3scores at all 8 sites caudal to L1. (Figure 4b). Both groups demonstrated significant L/R differences at 3 sites.

There was a PC4 muscle main effect for the back extensors only. The mean of all sites was near zero (Table 3).

In summary, both amplitude and temporal waveform differences in the abdominals and back extensors were found between ASYM and LBI groups. Between-group differences were not uniform across all muscles but were specific to muscle site and exercise level.

Discussion

Differences in muscle activation features between groups and between levels were not systematic, illustrating differential increases in muscle activity and altered synergies between muscles. Between-level activation differences could not be explained solely by altered task performance since timing and lumbo-pelvic motions were similar between levels within groups. Significant between-group differences in task performance can be partly explained by low variability associated with this highly constrained task (i.e. between-group difference for timing was only 0.2 sec). While motion differences were small (<2.3°), the LBI group had lower motion in all three planes. This is in keeping with the increased muscle activation amplitudes in both the agonists (Figure 3a) and antagonists of the LBI group, which would effectively increase the stiffness of the trunk and reduce motion.[42]

For both the back and abdominal muscles, the PC1 shape was the dominant pattern, as indicated by the high variance explained (Figure 2a,i). This indicates a high degree of agonist/antagonist temporal co-activation. Minor differences include a less prominent increase/decrease in back extensor activation during leg extension (Phase 2) as expected, since the TST challenges abdominal moreso than back muscles.[33] All LBI muscles were activated to higher overall percentages of maximum (PC1 scores) than the ASYM group (Figures 3,5,6) consistent with findings for a lifting task.[25] PC1 scores have been shown to be highly correlated with root mean squared amplitude,[36] reflecting overall muscular demand. Both groups increased abdominal activation with Level, but only the LBI group also increased back extensor amplitudes, as described in the results section. Higher activation could in part be explained by strength deficits as shown in chronic low back pain.[5] Working at higher amplitudes for both abdominals and back extensors, along with increased relative antagonist activity with demand, could increase risk of re-injury through muscle fatigue or increased lumbar compressive forces, associated with higher co-activation. [30, 43, 44] Conversely, increased co-activation has been shown to increase active stiffness during dynamic tasks, thus increasing spine stability.[45, 46,3] Since our LBI group was slightly older with potential age-related differences in passive structures,[47] the LBI response is consistent with Panjabi's three subsystem model[48] where increased active stiffness can compensate for reduced passive stiffness.

Relative activation increases associated with leg extension (positive PC2 scores) were specific to Level 2, where unsupported leg extension increased overall demand (Figures 2c,d,k,l, 4,5). While between-group differences were isolated to specific muscle sites (Figure 3b,4a, 5a,h), all LBI abdominal Level 1 scores were more negative and Level 2 scores more positive than the ASYM, indicating a greater relative increase in muscle response during Phase 2 in the LBI group. This is similar to previous findings between groups with stable versus unstable pelvis control: the unstable group elicited greater between-level differences than the stable group when performing the TST.[37] In this study, the LBI group manifested less pelvis motion (i.e. more stability) than the ASYM (Table 1), suggesting a compensatory increased trunk muscle co-activation, thus the overall mechanism may differ from the aforementioned paper. Increased response to leg-loading (PC2) was uniform amongst ASYM abdominal sites, whereas the LBI group demonstrated L>R asymmetries at IO (both levels) and EO3 (Level 2) (Figures 3b, 5c,d,g,h), indicating greater relative response in these contralateral muscles. Higher contralateral back extensor site responses in both groups (Figure 4a) can counterbalance the three dimensional force vectors acting on the spine and pelvis during leg loading (i.e. spine right axial rotation, side flexion and anterior pelvic tilt) (Figure 6c,d)[49], but higher LBI scores at 4 of 6 left back extensor sites, including the more lateral sites (L16, L36, L48), indicate a greater relative response was required. These higher contralateral responses may counterbalance the moments, but the higher responses might also reflect a strength asymmetry, but neither abdominal or back extensor strength was measured.

High initial IO activation (PC3) demonstrated by both groups associated with "abdominal hollowing" prior to Phase 1 (Figures 1a, 2e, 3c, 5c,d)[50] supports a feed-forward strategy engaging IO, whereas all other abdominal PC3 scores were near zero or negative for this pattern. The increased early activation (positive PC3 scores) in most back extensor sites in the LBI group and only RL16 in ASYM (Figure 4b) supports a conscious strategy to engage the back extensors prior to leg loading in the LBI group. This is in keeping with previous literature describing earlier onset times in the back extensors during a lifting task in a low back pain population.[18] Combined with the high PC2 scores (increased activation during the leg extension phase), and overall higher muscle amplitudes (PC1 scores) in the LBI group, the result would be longer periods of higher compressive forces in the lumbar spine, potentially putting the LBI group at risk for re-injury.[51] This decreased variability in motor recruitment levels suggests the LBI group has tended to switch from a closed to an open loop motor control system, as described by Magill.[52] Whereas the closed loop system is responsive to feedback, the open loop system does not allow for feedback, thus the system will be less responsive to the changing external moment. Similarly, this change in motor control strategy may partly explain the reduced lumbo-pelvic motion displayed by the LBI group (Table 1).

Explaining less than 4% of waveform variance, PC4 is the only principal pattern that was notably different between the two muscle groups. PC4 abdominal scores were consistently higher in the ASYM group (Figure 3d, Table 2) moreso with increased demand (Level 2). Negative PC4 scores, as seen with most LBI abdominal sites, captured a lower relative first peak when the left leg is lifted relative to activity during leg lowering (Phase 3) (Figure 2h), or a more sustained level of activity over the task. This is contrary to previous research in a chronic LBP group, which showed a more rapid drop in activity during the leg lowering phase compared to controls.[10] The greater number of L/R asymmetries in the LBI group (Level 2) in this study (Figures 3d, 5e,f) is consistent with the more asynchronous activation pattern described in a chronic LBP group[10] as well as LBP groups who were in remission at the time of testing.[17, 24]

The LBI group exhibited greater variability of activation patterns, as evidenced by larger standard deviations when compared to the ASYM (Table 2). This is similar to the increased temporal variability described in a chronic LBP group[10] and more recently for a recovered LBI group performing a functional task.[24] This was particularly evident for the oblique muscle sites and was not confined to amplitude differences; abdominal temporal patterns also had higher variability. For the back extensors, however, variability was noted primarily for PC1 (amplitude) which could indicate greater strength differential within the LBI group or heterogeneity of clinical classification. These should be considered in future studies.

Differences in overall amplitudes and temporal patterns were not all systematic (Tables 2, 3, Figures 3-6); between-group differences varied with muscle site, as did individual muscle responses to exercise level and the applied external moment. Hence, these alterations cannot all be attributed to strength deficits[5] or differences in the ability of the LBI participants to recruit maximal activity during the normalization exercises.[53] as suggested in the literature for those with pain. Furthermore, this LBI group reported minimal pain scores or dysfunction and were deemed recovered at the time of testing.

Study Limitations: Precautions were taken to minimize cross-talk and ensure valid electromyographic recordings by standardizing electrode placement, validation exercises and electrocardiographic artifact removal. [54] Participants in the study self-reported their perception of recovery following a low back injury. All reported minimal to low level of pain and were within 12 weeks of their low back injury, it is difficult to definitely describe their state of feeling as "recovered" or sub-acute. While all participants were instructed to lightly slide across the exercise table, the force on the table was not measured. Thus greater between-level relative difference demonstrated by the LBI group for PC2 abdominal sites could be related to more mass being supported during the leg slide task during Level 1, which would effectively lower the demand and result in a greater relative increase for Level 2. The underlying implication for clinical practice, however, is that this progression should be undertaken with caution; the mass being supported during Level 1 should be considered before progressing to Level 2 when using this exercise protocol.

Conclusions

This study illustrates that subjective reports or observed task performance may not capture trunk neuromuscular alterations following a LBI; objective muscle activation patterns provide additional information that may help assess recovery and guide clinical decision-making. Despite the perception of recovery, this LBI group demonstrated altered trunk muscle recruitment patterns compared to a non-LBI group while performing a highly constrained TST. Muscle activation levels were generally higher in the LBI group, with a larger relative increase during the leg extension phase for all muscles and less variation in response to the leg-lifting and lowering demands of the task. Higher amplitude of agonist/antagonist co-activity in the LBI group supported our hypotheses. While there were between-muscle temporal differences that varied with group, the results provide weak support for greater temporal asymmetries in the LBI group. In contrast, temporal synchrony between abdominals and back extensors related to leg loading and initial bracing (Level 2) were more evident in the LBI group. Greater variability in amplitudes (PC1) for both muscle groups and temporal patterns for the abdominal muscles suggests that the LBI group utilized a wider range of patterns to perform this highly constrained task.

While clinicians may not have routine access to EMG data, these findings show that motor recruitment patterns and relative demands on the abdominal and back musculature are different in those early after a LBI episode despite the perception of recovery. While amplitudes are higher for both muscle groups, isolated strength training only may not address the alterations in those recovered from a LBI. The differences in overall magnitude would reflect a general strength deficit but the difference between groups for both the abdominals and the back extensors were not uniform between the two groups or between levels for features other than overall amplitude. Thus there is a differential motor response. Therapeutic exercises aimed at encouraging motor responsiveness to changing task demands, while diminishing the amount of agonist/antagonist co-activation should be encouraged. However, caution on how these exercises are progressed needs to be employed based on the different responses for the two groups associated with the increase in level. Furthermore, the higher variability in some of the measures for the LBI group support that subgroups might exist that have differing abdominal muscle responses.

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Suppliers' List

[a.] Kendall-LTP, 15 Hampshire St., Mansfield, MA 02048, USA

[b.] Bortec Electronics Inc., 7171 Sierra Morena Blvd, Calgary, Alta, T3H 3G6 Canada

[c.] National Instruments Corporation, 11500 Mopac Expwy, Austin TX 78759, USA

[d.] Ascension Technology Corporation, 107 Catamount Dr., Milton, VT 05468, USA

[e.] Minitab Inc, 301 Enterprise Dr, State College, PA 16801, USA

Figure Legends

Figure 1: Exercise protocol utilized: starting position corresponds with participants being asked to “pull your abdomen up and in toward your chest as if to tuck your stomach under your ribcage”; **a**) right leg lift; **b**) right leg in a position of 90° hip flexion, in contact with the wooden frame; **c**) left leg also lifted to contact frame, then right leg begins to lower; **d**) Level 1, in which the right leg extends with the heel sliding down the bed, until the leg is fully extended, then returned to the flexed position (**e**); **f**) Level 2 is similar to Level 1, except the heel does not touch the table until the leg is fully extended. Following either of these levels, the left (**g**) then right leg are lowered to the starting position (**h**). All exercises took place over an 8 second count, during which participants were encouraged to minimize pelvic motion.

i) Conductive metal strips attached to the right foot, thigh, wooden contact frame and bed completed a circuit upon contact, thus providing a voltage change. The exercise was consequently divided into 3 phases: phase 1= lifting phase, phase 2= leg extension phase (including hip/ knee extension and flexion again), and phase 3= leg-lowering phase. Abbreviations: R=right, F=foot, T=thigh.

Figure 2: For principal patterns 1-4, charts **a, c, e, g, i, k, m**, and **o** are the respective principal patterns, with scaled variance explained indicated by the gray shade. Total variance explained by each pattern is shown in the top left corner of each chart. PC score values appear on the right y-axis, and percentage of explained variance (% Var.) on the left. Abdominal outcomes are in the left column, back extensors on the right. Charts **b, d, f, h, j, l, n, p** show the mean normalized

activation amplitude patterns as a percentage of MVC for the 5 highest (solid lines) and 5 lowest (dashed lines) PC scores, to assist with interpretation. The arrows on the high-low scores for PC2-4 highlights the relative differences associated with the high scores.

Figure 3: Interaction plots for principal components 1-4 of abdominal muscles. Black lines = ASYM group, gray = LBI group; solid/dashed lines = Level1/Level 2 of difficulty. || = significant R/L within muscle pair differences for all interactions. Solid shading indicates significant between-group differences at both levels; diamond shading indicates a significant between group difference in Level 2; diagonal shading indicates a significant between group difference in Level 1. Along the x-axis, # = a significant between level difference in both groups; $ = a significant between level difference in the ASYM group only. **a**) PC1 Group*muscle*level interaction (abdominals) indicating the higher amplitudes of all muscle sites in the LBI group within level; * = EO1,2 scores are significantly higher than the RAs, EO3s and IOs; † = LEO2 is significantly different than LEO1, LEO3, both IOs and all RAs; ‡ = LEO2 is significantly different than LEO3, the IOs and RAs; § = RURA is significantly higher than LLRA, LURA; ¶ = RLRA is significantly higher than RURA, LURA; **b**) PC2 Group*muscle*level interaction (abdominals) indicating the large between-level differences within group; * = RIO is significantly different from all other muscle sites; † = RIO is significantly different from REO1-3, LEO3, LIO, all RAs; ‡ = RIO is significantly different from LEO2, EO3 (R/L), all RAs; § = RIO is significantly different from all RAs; **c**) PC3 Group*muscle interaction (abdominals) demonstrating the significantly higher amplitudes of the LBI group at 8 muscle sites, within groups; * = RIO is significantly higher than all other sites; † = REO1 is significantly higher than all RAs, LEO2, EO3 (R/L); ‡ = LEO1 (LBI) is significantly higher than LLRA, URA(R/L), LEO2, REO3; § = REO2 is significantly higher than LLRA; ¶ = associated ASYM site is

significantly higher than all RAs; ** = REO3 is significantly higher than both LRAs, LURA; **d**) PC4 Group*muscle*level interaction (abdominals) highlighting the consistently higher scores in the ASYM group.

Figure 4: Interaction plots for principal components 2-3 of the back extensor muscles. Black lines = ASYM group, gray = LBI group. || = significant R/L within muscle pair differences for all interactions. Shading indicates significant between-group differences. **a**) PC2 Group*muscle interaction (back extensors) highlighting the R/L sidedness of this pattern as well significant between-group differences at 7 sites; **b)** PC3 Group*muscle interaction (back extensors) showing the significant between-group differences, more so over the caudal sites. $\alpha = 0.05$ for all interactions.

Figure 5: Examples of ensemble average waveforms for specific abdominal muscles. Black lines = ASYM group; grey lines = LBI group; solid/dashed lines = Level1/Level 2 of difficulty; right = R, left = L; **a,b**) R,L LRA illustrating the large bilateral PC2 effect of increased activation mid-trial with Level 2, more pronounced on the R LBI group. **c,d**) R,L IO depicting the higher initial activation (PC3), with a loss of L-sided activity as the trial progresses, indicative of a high PC4 score. **e,f**) R,L EO2 illustrating a moderate effect of PC2 bilaterally in Level 2, as well as the decrease of left sided activation in the latter part of the trial (+PC4), which is not present on the right (-PC4). **g,h**) R,L EO3 shows the R/L differential between mid-trial activations, indicative of higher PC2 scores on the L. The R sided patterns tend to maintain amplitude during the second peak and latter trial, indicative of the more negative PC4 scores on the R.

Figure 6: Examples of ensemble average waveforms for specific back extensor muscles. Black lines = ASYM group; grey lines = LBI group; solid/dashed lines = Level1/Level 2 of difficulty; right = R, left = L **a,b**) R,L L33 motor sites, illustrating the lower LBI amplitude effect (PC1) on the left side, and a subtle PC2 effect L>R, resulting in a flattening mid-trial. **c,d**) R,L L48 sites depicting the positive PC2 scores on the L compared to the negative R sided scores, more so in the LBI group. **e,f**) R,L L52 sites illustrating the higher amplitude differences on the R (PC1), minimal R/L difference in the ASYM group, but the negative PC2 on the R results in relatively more of mid-trail hollow in the LBI group when compared to either the ASYM group or the L side.

Appendix 1: Post-hoc results of the significant ($p < 0.05$) abdominal **PC2** group*muscle*level interaction. Groupings which share a letter are not significantly different from each other.

Group	Muscle	Level	Grouping
LBI	RLRA	2	A
LBI	LLRA	2	A B C
ASYM	RLRA	2	A B
LBI	LURA	2	A B C D
LBI	LE03	2	A B C D
LBI	RURA	2	A B C D
ASYM	LLRA	2	A B C D E
ASYM	LURA	2	B C D E F
LBI	REO1	2	B C D E F G
LBI	REO2	2	B C D E F G
LBI	LEO2	2	C D E F G
ASYM	RURA	2	D E F G
LBI	LEO1	2	E F G H I
LBI	LIO	2	E F G H I J K
ASYM	LEO3	2	F G H
LBI	REO3	2	G H I J K
ASYM	LEO2	2	G H I J K
ASYM	REO2	2	G H I J K
ASYM	REO3	2	G H I J K
ASYM	REO1	2	H I J K
ASYM	RIO	2	H I J K L
ASYM	LIO	2	I K L
ASYM	LEO1	2	J K L
ASYM	LURA	1	L M
LBI	RIO	2	L M N
ASYM	LLRA	1	M N O
ASYM	RLRA	1	M N O
ASYM	RURA	1	M N O
LBI	LURA	1	M N O P
LBI	LLRA	1	M N O P Q
ASYM	LEO3	1	M N O P Q
LBI	LEO3	1	M N O P Q R
LBI	RLRA	1	M N O P Q R
LBI	RURA	1	N O P Q R S
ASYM	REO3	1	O P Q R
ASYM	LEO2	1	O P Q R
ASYM	REO2	1	P Q R S
LBI	LIO	1	O P Q R S T
ASYM	LIO	1	P Q R S T
ASYM	REO1	1	Q R S T
LBI	REO3	1	P Q R S T U
ASYM	LEO1	1	R S T U
ASYM	RIO	1	S T U
LBI	REO2	1	R S T U
LBI	REO1	1	R S T U
LBI	LEO2	1	T U V
LBI	LEO1	1	U V
LBI	RIO	1	V

Table 1: Descriptive statistics for participants, as well as pelvis motion in 3 planes: Sagittal (flexion/extension), transverse (axial rotation) and frontal (side bending).

	ASYM	LBI
count	51	30
% female	53	53
Age (yrs)	31.5(8)	40.7(12)
Height (cm)	171.1(9)	169.9(9)
Weight (kg)	71.5(15)	77.6(20)
BMI (kg/m^2)	24.3(4)	26.6(6)
Pelvic tilt angle (°)	9.9(3)	9.3(6)
Abdominal function test	1.7(1)	1.6(1)
Test time (s)	7.5(0)	7.7(1)
Sagittal (°)	7.0(4)	4.7(3)
Transverse (°)	4.8(2)	4.1(2)
Frontal (°)	6.3(4)	4.0(2)

Note: Values are mean (SD), and include data from both TST levels. Shading indicates a significant difference between the ASYM and LBI groups ($p < 0.05$).
Test time = time taken to complete the task as indicated by a step voltage meter.

Table 2: PC scores (Mean (SD)) for the abdominal muscles, by group, level and muscle for PC1, PC2 and PC4 and for group by muscle and level by muscle interactions for PC3.

PC	RLRA	LLRA	RURA	LURA	REO1	LEO1	REO2	LEO2	REO3	LEO3	RIO	LIO
1 LBI L1	-27.1(78)	-36.5(86)	-20.9(98)	-39.2(78)	49.7(189)	60.0(204)	57.7(151)	80.9(221)	14.2(146)	14.3(164)	4.9(114)	-27.5(84)
1 ASYM L1	-49.3(92)	-57.6(92)	-67.0(78)	-71.3(69)	-11.3(133)	-3.0(150)	-13.7(94)	-15.0(77)	-23.8(81)	-32.0(94)	-41.6(83)	-59.4(75)
1 LBI L2	35.7(104)	18.0(110)	38.0(126)	10.1(98)	119.4(214)	129.9(229)	114.9(169)	148(254)	53.5(164)	64.6(173)	46.8(128)	20.7(106)
1 ASYM L2	-9.1(113)	-23.5(109)	-37.0(96)	-41.0(85)	22.3(147)	28.0(152)	14.3(107)	11.7(86)	-1.3(87)	-6.4(101)	-15.8(97)	-36.6(86)
2 LBI L1	-15.9(24)	-12.7(18)	-16.8(18)	-9.3(15)	-29.8(33)	-37.0(39)	-29.6(28)	-35.2(45)	-23.5(26)	-15.9(20)	-45.1(55)	-20.7(31)
2 ASYM L1	-8.6(21)	-7.6(17)	-8.8(16)	-3.1(15)	-23.0(33)	-28.7(33)	-20.5(22)	-18.0(22)	-17.6(21)	-14.2(20)	-29.4(27)	-22.4(25)
2 LBI L2	40.7(37)	36.9(29)	33.7(33)	34.4(32)	24.9(23)	18.6(29)	23.1(31)	23.0(31)	13.7(28)	33.8(32)	-4.2(55)	18.3(35)
2 ASYM L2	36.2(26)	29.4(22)	22.9(22)	27.1(18)	9.1(32)	5.2(31)	12.7(19)	13.0(20)	12.2(22)	17.3(22)	7.4(26)	6.0(27)
3 LBI	0.01(23)	-2.9(20)	-1.7(20)	-1.4(17)	3.7(27)	2.8(27)	1.2(29)	-0.8(31)	-1.2(17)	-0.5(20)	21.4(53)	11.2(42)
3 ASYM	-8.8(18)	-9.9(18)	-7.0(14)	-7.7(13)	-1.3(25)	3.5(24)	-4.0(15)	-4.7(13)	-4.8(13)	-3.2(12)	15.5(22)	12.9(25)
3 L1	**-8.6(20)**	**-10.1(19)**	**-7.1(16)**	**-7.4(15)**	**-2.0(26)***	**1.4(26)**	**-5.1(19)**	**-5.0(18)**	**-6.3(15)**	**-5.1(16)**	**15.9(34)***	**9.9(32)**
3 L2	**-2.5(20)**	**-4.5(18)**	**-3.0(16)**	**-3.4(14)**	**3.1(26)**	**5.1(24)**	**0.9(22)**	**-0.2(24)**	**-0.6(14)**	**0.7(15)**	**19.5(39)***	**14.6(33)**
4 LBI L1	-12.8(12)	-6.6(11)	-16.4(18)	-8.9(12)	0.7(23)	5.3(18)	-10.5(19)	0.9(13)	-15.0(15)	-3.1(11)	-8.4(24)	5.2(17)
4 ASYM L1	-5.4(15)	1.2(18)	-5.2(13)	-1.0(15)	1.8(27)	8.7(34)	-7.6(18)	1.7(20)	-10.8(19)	-3.8(24)	-3.2(16)	11.9(21)
4 LBI L2	-7.8(19)	-0.2(15)	-12.5(28)	-3.2(20)	-5.6(30)	3.8(33)	-8.4(24)	6.4(28)	-14.2(20)	-0.8(16)	-6.6(20)	10.6(19)
4 ASYM L2	4.5(21)	9.2(19)	0.1(17)	3.9(17)	10.1(23)	15.1(26)	1.5(17)	7.4(20)	-3.0(17)	4.0(17)	4.7(15)	16.7(18)

Note: L1, L2 = levels 1 and 2 of the trunk stability test, respectively, for PC3 these are both groups combined.

Significance value for all comparisons was set at $p<0.05$, with Bonferroni adjustments.

Bolding = significant between-level difference within groups and muscle.

* = significant PC3 Right/Left muscle pair differences within level.

Table 3: PC scores (Mean (SD)) for the back extensors muscles, showing group by muscle and level by muscle interactions for PC2 and PC3 and a muscle main effect for PC1 and PC4.

PC	RL13	LL13	RL16	LL16	RL33	LL33	RL36	LL36	RL48	LL48	RL52	LL52
1 Combined	-18.4(21)*	-19.3(21)	-2.8(29)*	-7.1(30)	-5.8(33)*	-6.3(36)	-2.4(38)*	-4.0(40)	17.4(46)*	12.0(42)	22.0(60)*	14.8(48)
2 LBI	-0.5(4)	2.1(4)	-2.9(6)	6.5(7)	-0.7(3)	1.9(4)	-2.7(5)	4.4(6)	-4.6(7)	6.4(8)	-2.7(9)	-0.7(6)
2 ASYM	-1.8(4)	0.3(3)	-4.7(6)	2.9(6)	-0.3(4)	1.2(5)	-2.2(5)	2.9(5)	-4.5(10)	3.8(6)	-0.6(5)	-0.8(5)
2 L1	**-2.3(3)***	**0.1(3)**	**-5.7(6)***	**3.0(6)**	**-1.7(3)***	**0.1(3)**	**-4.0(5)***	**2.0(4)**	**-7.0(10)***	**2.8(5)**	**-3.1(7)**	**2.5(5)**
2 L2	**-0.2(4)***	**1.8(4)**	**-2.4(6)***	**5.4(7)**	**0.8(4)***	**2.8(5)**	**-0.8(5)***	**4.9(6)**	**-2.1(7)***	**6.8(7)**	**0.4(5)**	**1.1(4)**
3 LBI	0.2(3)	-0.2(3)	2.0(4)	0.3(5)	0.9(3)	0.8(3)	0.6(4)	0.8(3)	1.3(5)	2.3(5)	1.9(6)	0.7(4)
3 ASYM	0.6(4)	0.3(4)	1.5(5)	0.0(4)	-0.9(4)	-1.0(3)	-2.5(9)	-0.9(7)	-2.2(5)	0.1(3)	-0.9(5)	-0.8(4)
3 L1	0.2(4)	-0.2(4)	1.7(4)*	**-0.4(5)**	-0.6(4)	**-1.0(4)**	-1.6(6)	**-0.8(5)**	**-1.6(5)***	**0.1(4)**	**-0.5(6)**	**-0.9(5)**
3 L2	0.6(3)	0.4(3)	1.7(4)*	**0.7(4)**	0.2(4)	**0.3(3)**	-1.2(9)*	**0.2(7)**	**-0.2(5)***	**1.8(4)**	**0.7(5)**	**0.4(4)**
4 Combined	0.6(3)	0.4(3)	2.8(6)*	-0.3(5)	-0.8(3)	0.7(8)	0.3(3)	0.3(3)	-0.9(4)*	0.5(3)	-1.0(5)	-1.4(4)

Note: L1, L2 = levels 1 and 2 of the trunk stability test, respectively. For PC2 and 3, both groups are combined.

Combined= muscle main effect, thus PC Scores were combined across groups and levels.

Significance value for all comparisons was set at $p<0.05$, with Bonferroni adjustments.

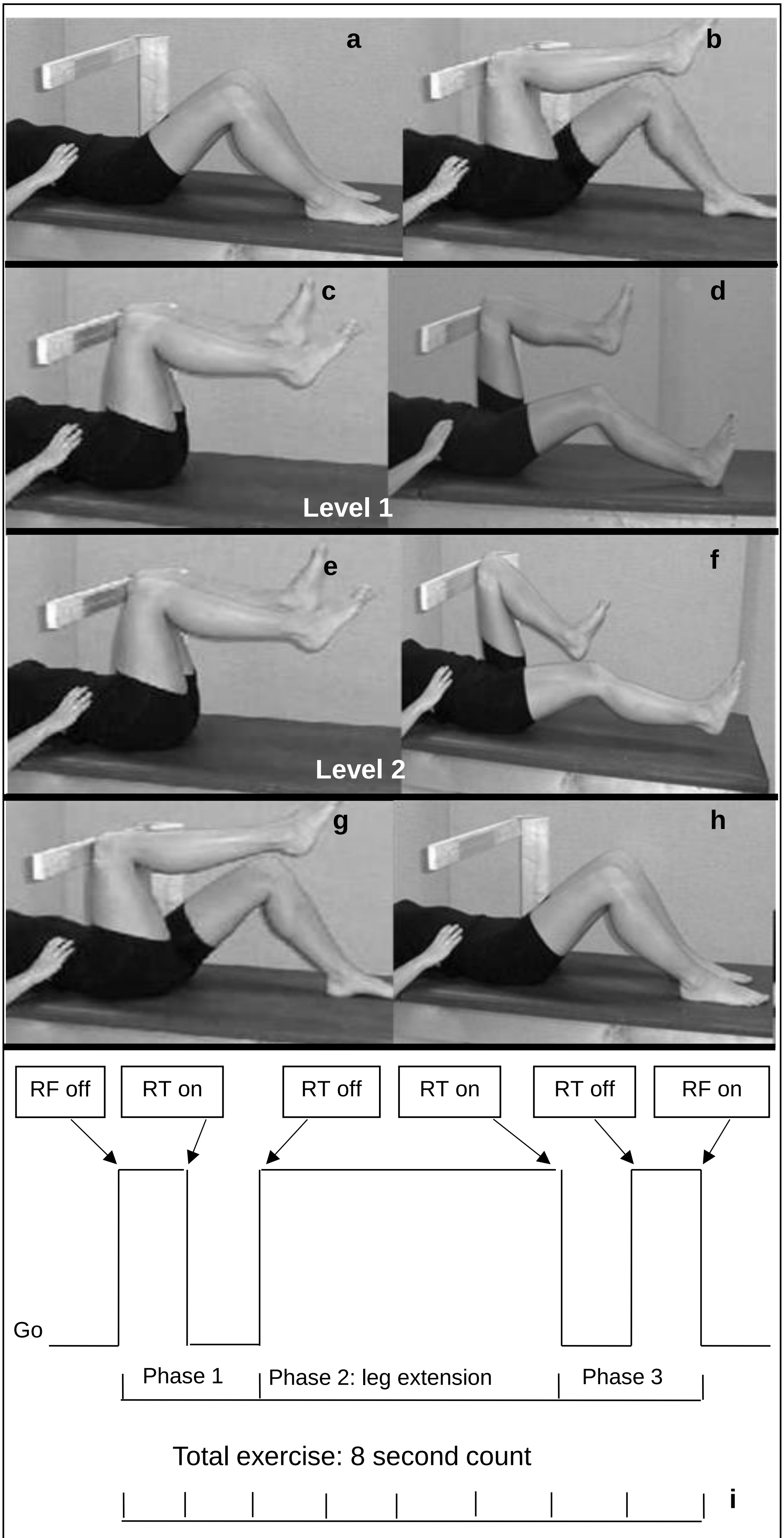
Bolding = significant between-level difference within groups and muscle.

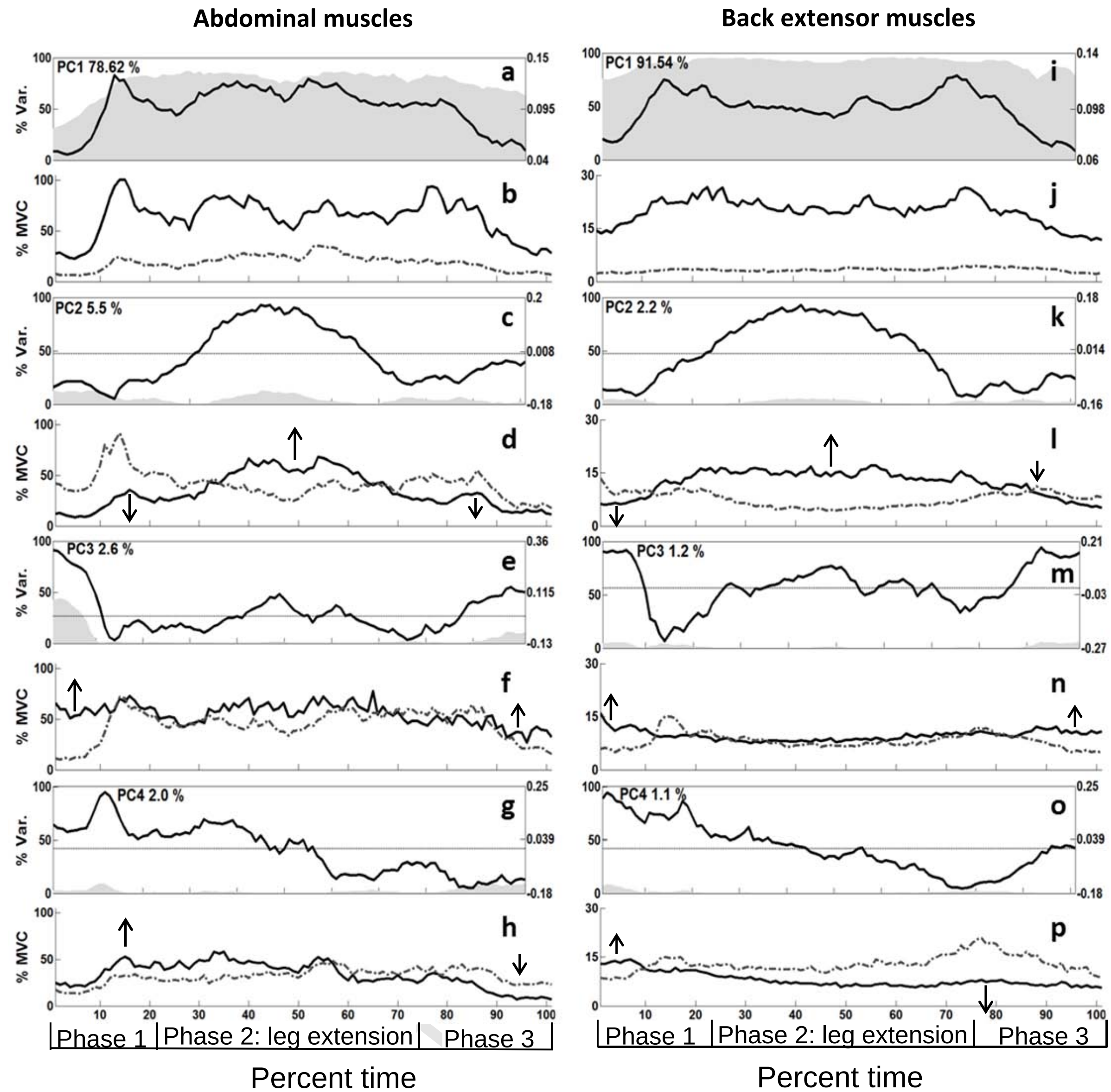
* = significant Right/Left muscle pair differences.

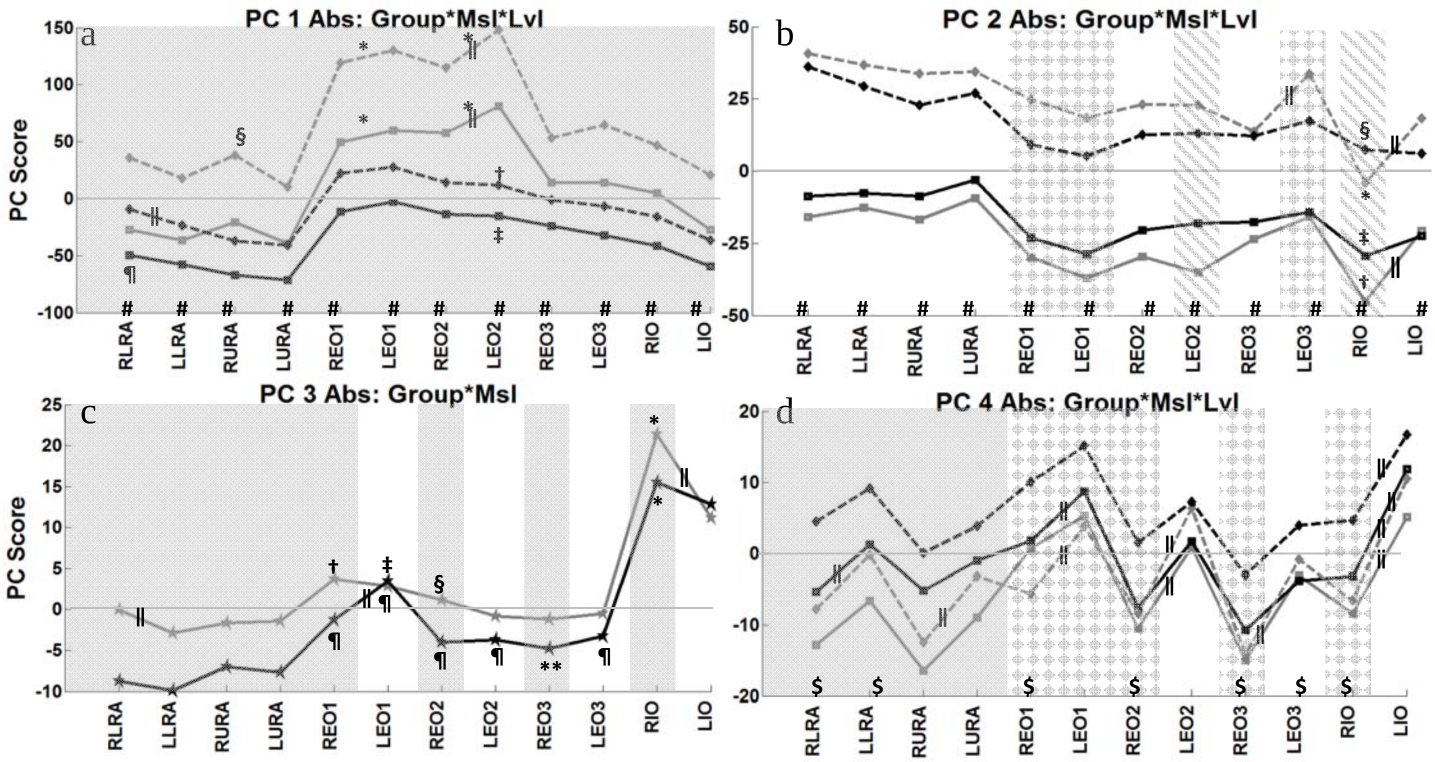
Table 4: Main effects and interactions for the three-factor ANOVAs (2 groups, 2 levels, 12 muscle sites for each of the 4 principal component (PC) scores 1-4 for abdominals and back extensors separately.

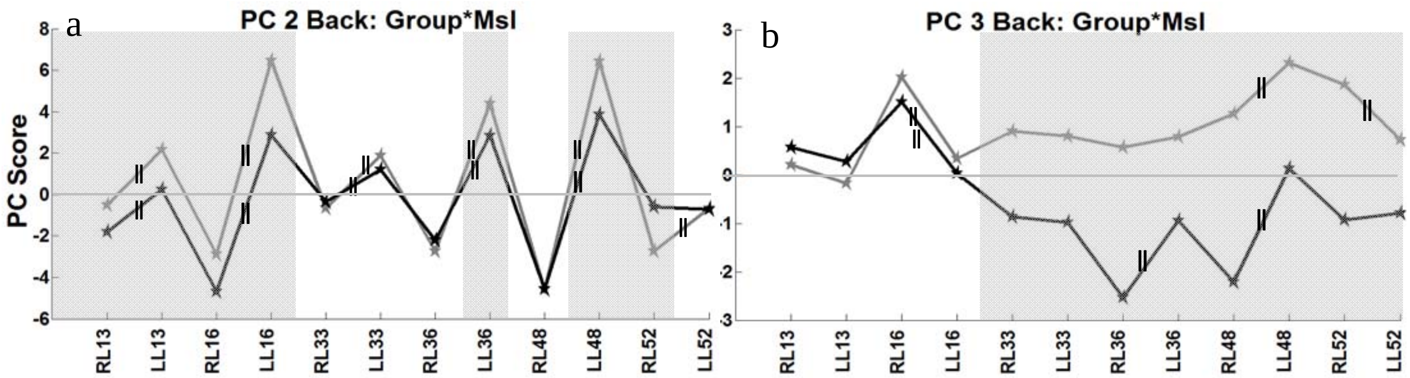
	Abdominals				Back Extensors			
	PC1	**PC2**	**PC3**	**PC4**	**PC1**	**PC2**	**PC3**	**PC4**
Group	< 0.001	=0.913	=0.379	=0.028	=0.001	=0.207	=0.027	=0.865
Level	< 0.001	< 0.001	< 0.001	=0.002	=0.018	< 0.001	< 0.001	=0.299
Muscle	< 0.001	< 0.001	< 0.001	< 0.001	**< 0.001**	< 0.001	< 0.001	**< 0.001**
Group*level	< 0.001	= 0.014	**= 0.053**	= 0.137	**= 0.018**	= 0.232	= 0.196	= 0.164
Group*muscle	= 0.094	= 0.033	**= 0.010**	= 0.375	= 0.571	**= 0.003**	**= 0.003**	= 0.854
Level*muscle	< 0.001	< 0.001	**= 0.001**	= 0.055	= 0.184	**< 0.001**	**< 0.001**	= 0.510
Group*level*muscle	**= 0.030**	**= 0.019**	= 0.426	**= 0.001**	= 0.540	= 0.226	= 0.098	= 0.575

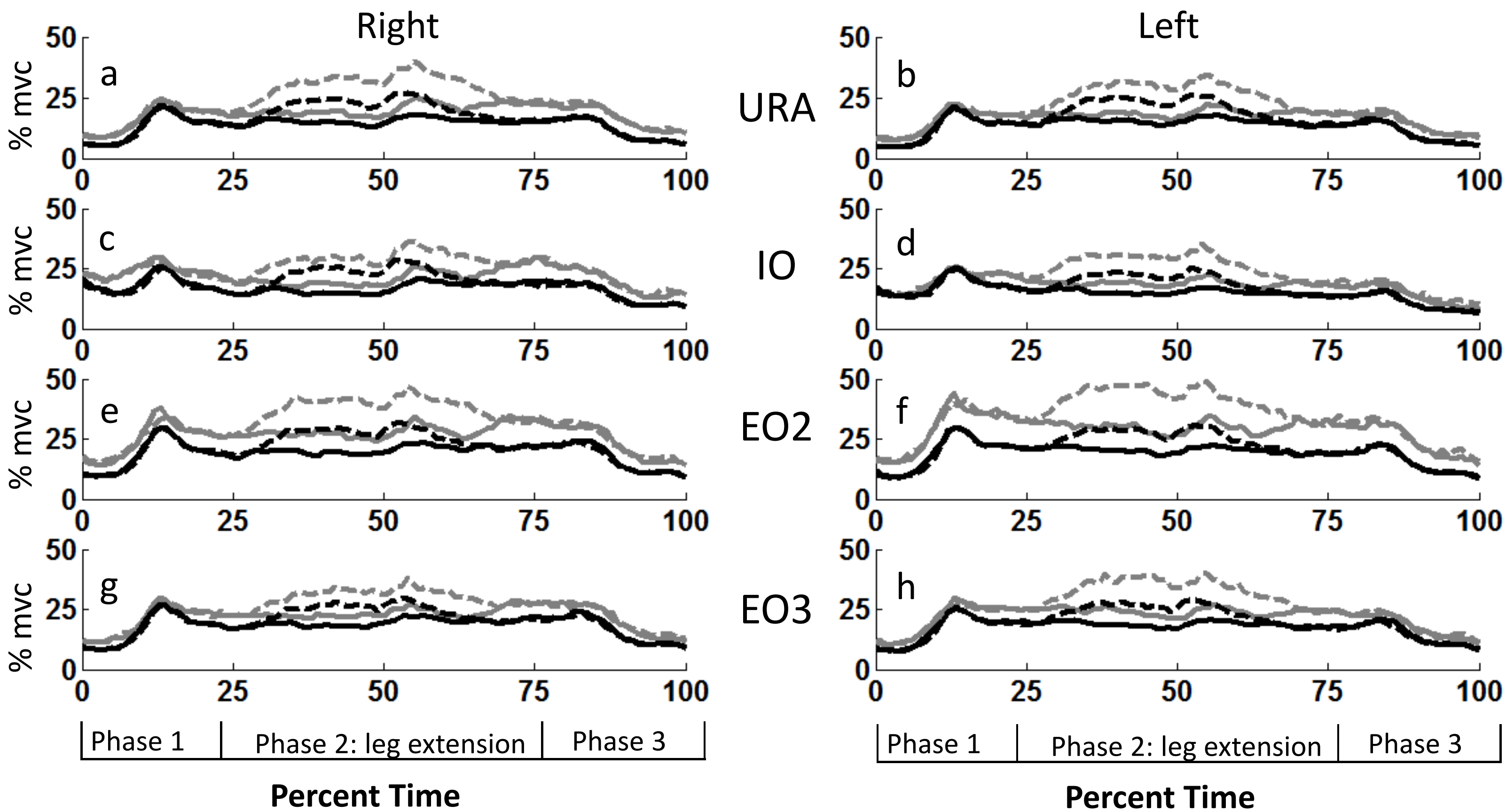
Shading indicates the significant (p < 0.05) main effects or interactions that were subjected to post hoc analyses.

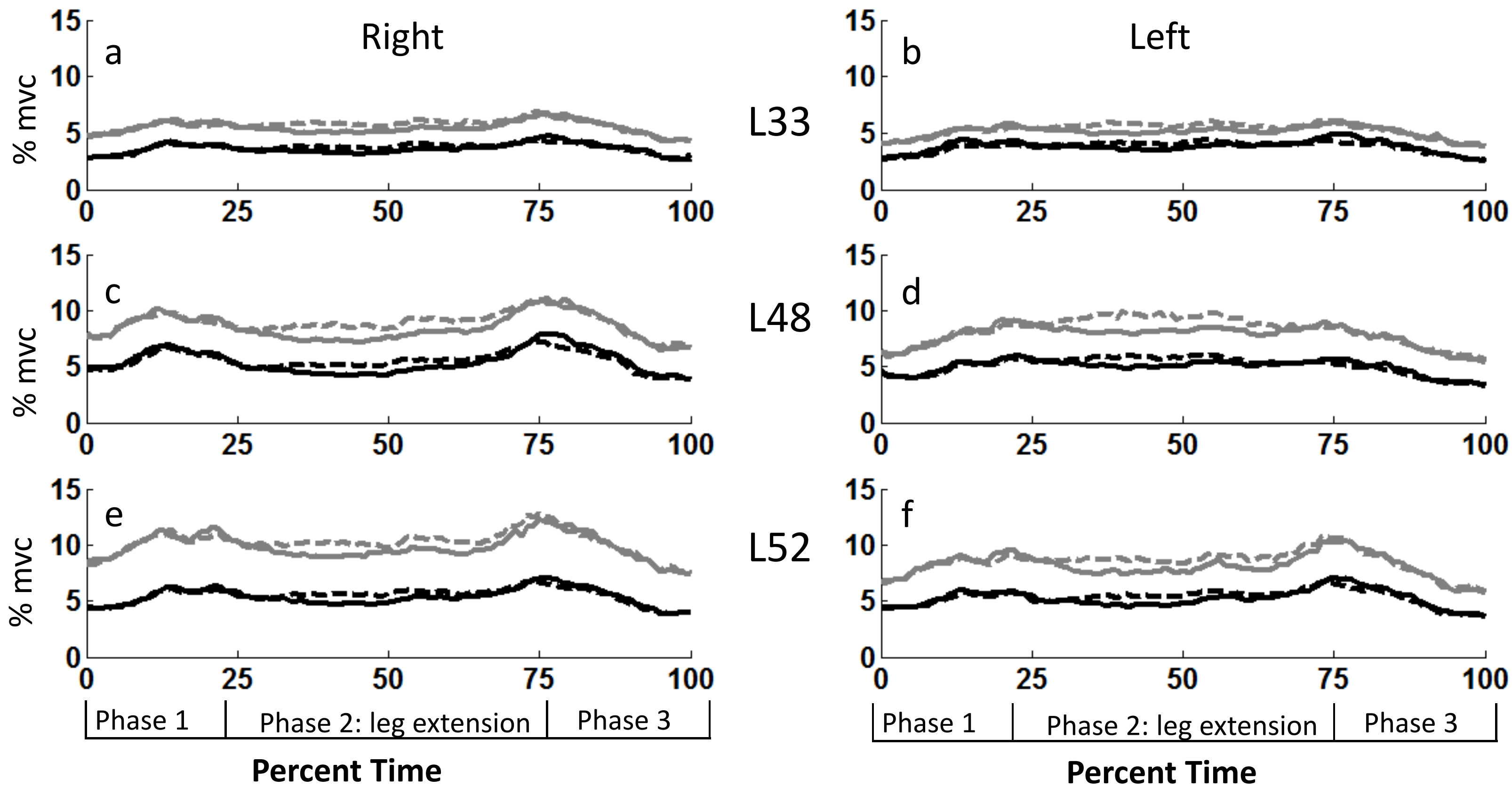

a
b
c
d
Level 1
e
f
Level 2
g
h
RF off
RT on
RT off
RT on
RT off
RF on
Go
Phase 1
Phase 2: leg extension
Phase 3
Total exercise: 8 second count
i


Abdominal muscles
Back extensor muscles
PC1 78.62 %
PC1 91.54 %
PC2 5.5 %
PC2 2.2 %
PC3 2.6 %
PC3 1.2 %
PC4 2.0 %
PC4 1.1 %
% Var.
% MVC
a
b
c
d
e
f
g
h
i
j
k
l
m
n
o
p
Phase 1
Phase 2: leg extension
Phase 3
Percent time


PC 1 Abs: Group*Msl*Lvl
a
PC Score
RLRA
LLRA
RURA
LURA
REO1
LEO1
REO2
LEO2
REO3
LEO3
RIO
LIO
PC 2 Abs: Group*Msl*Lvl
b
PC 3 Abs: Group*Msl
c
PC 4 Abs: Group*Msl*Lvl
d


a
PC 2 Back: Group*Msl
PC Score
8
6
4
2
0
-2
-4
-6
RL13
LL13
RL16
LL16
RL33
LL33
RL36
LL36
RL48
LL48
RL52
LL52
b
PC 3 Back: Group*Msl
3
2
1
0
-1
-2
-3
RL13
LL13
RL16
LL16
RL33
LL33
RL36
LL36
RL48
LL48
RL52
LL52


Right
Left
URA
IO
EO2
EO3
a
b
c
d
e
f
g
h
% mvc
50
25
0
0
25
50
75
100
Phase 1
Phase 2: leg extension
Phase 3
Percent Time



Right
Left
a
b
c
d
e
f
L33
L48
L52
% mvc
15
10
5
0
0
25
50
75
100
Phase 1
Phase 2: leg extension
Phase 3
Percent Time