

Trunk muscle recruitment patterns in specific chronic low back pain populations

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Abstract

Background. It is hypothesized that injury or degeneration of osteoligamentous spinal structures would require compensation by trunk musculature and alterations in motor control to maintain spine stability. While, biomechanical modeling has supported this hypothesis, studies of muscle recruitment patterns in chronic low back pain patients both with and without significant osteoligamentous damage have been limited. This study utilized a non-randomized case-control design to investigate trunk muscle recruitment patterns around the neutral spine position between subgroups of patients with chronic mechanical low back pain and asymptomatic controls.

Methods. Twenty subjects with chronic low back pain attributed to clinical lumbar instability were matched to 20 asymptomatic controls. In addition 12 patients with non-specific chronic low back pain were studied. Surface EMG from five trunk muscles was analyzed to determine activation levels and patterns of recruitment during a standing reach under two different loading conditions.

Findings. The chronic low back pain group with symptoms attributed to clinical instability demonstrated significantly higher activation levels of the external oblique and rectus abdominus muscles and lower abdominal synergist ratios than the control group. No significant differences were found between patient subgroups.

Interpretation. While these data demonstrate altered muscle recruitment patterns in patients with chronic low back pain, the changes are not consistent with Panjabi's theory suggesting that these alterations are driven by passive subsystem damage. However, the higher activation of global abdominal musculature and altered synergist patterns may represent a motor control pattern that has consequences for continued dysfunction and chronic pain.

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1. Introduction

Differences in trunk muscle recruitment or neuromuscular control in patients with mechanical low back pain

have been reported by several investigators (Hodges and Richardson, 1999; O'Sullivan et al., 1997a; Radebold et al., 2000; Van Dieen et al., 2003). It has been hypothesized that these changes in muscle recruitment patterns are an adaptation to underlying spinal instability resulting from osteoligamentous laxity or damage, muscle dysfunction or reduced neuromuscular control (Panjabi, 1992; Paris, 1985). Panjabi (1992) proposed a model for

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a spinal stabilization system which partitioned the responsibility for joint stability and movement into three subsystems: a passive subsystem (connective tissue, bones, and intervertebral discs), an active subsystem of muscles and tendons, and a neural (motor) control subsystem. Panjabi further hypothesized that spinal instability created by dysfunction of the passive support system, resulting in loss of control or excessive motion of a spinal segments neutral zone, would trigger compensation strategies by trunk musculature under the guidance of the neural control systems. The objective of the compensation would be to maintain spinal stability (Panjabi, 1992).

Biomechanical modeling and experimental studies have demonstrated that trunk muscle co-contraction is necessary for spinal stability particularly in neutral upright postures even in the healthy spine (Cholewicki et al., 1997; Granata et al., 2001). Moreover, reduction of a model's passive stiffness component predicts that muscle activation would increase to maintain stability of a spine (Cholewicki et al., 1997). Gardner-Morse and Stokes (2001) lend further support to this hypothesis, by demonstrating that a 10% reduction in segmental stiffness can compromise spine stability. They further suggest that this reduction in segmental stiffness, in conjunction with poor neuromuscular control and reduction in muscle stiffness could result in clinical instability. These modeling predictions are supported by data from animal models (Kaigle et al., 1995; Wilke et al., 1995) and through experiments using healthy individuals, who upon challenges to trunk stability responded by increasing muscle co-contraction (Granata and Orishimo, 2001). This co-contraction is particularly necessary around the neutral spine position and during low load conditions (Cholewicki and McGill, 1996).

While numerous investigators have reported activation pattern differences in patients with non-specific mechanical low back pain, the hypothesis that changes in trunk muscle recruitment patterns are an adaptation to underlying passive subsystem damage that results in an increased neutral zone, segmental hypermobility, and/or clinical spinal instability has not been systematically investigated (Lariviere et al., 2000; Newcomer et al., 2002; Van Dieen et al., 2003). The few investigators who studied patients with radiographic findings associated with clinical lumbar instability (i.e., spondylolisthesis) have found differences in muscle recruitment; however, these findings were demonstrated during the performance of a specific therapeutic exercise or non-functional activity (Lindgren et al., 1993; O'Sullivan et al., 1997a; Sihvonen et al., 1997). These investigators did not study muscle activation patterns of the trunk flexors and extensors simultaneously nor did they consistently address muscle co-contraction or synergist ratios (i.e., *trunk flexors/extensors*, *internal oblique/rectus abdominus*).

On the basis of the assumption that chronic mechanical low back pain (CLBP) patients with significant passive subsystem damage adapt muscle recruitment to compensate for the loss of spinal stability, we have formulated several hypotheses regarding muscle activation levels and patterns. Patients with chronic mechanical low back pain attributed to clinical lumbar instability (CLBP_I) from significant passive subsystem damage would demonstrate increased muscle activation and greater co-contraction of the trunk muscular than asymptomatic controls during a functional reaching task. In addition, work by Bergmark (1989) and Panjabi et al. (1989) suggests that muscle architecture plays a role in effective spine stability. They found through biomechanical modeling that activation of segmentally inserting muscles would be more effective at increasing stability than multi-segmental muscle inserting on the thorax and pelvis. Based upon this work, we also hypothesized that synergist muscle ratios represented by activation of segmental relative to multi-segmental muscles when acting synergistically (i.e., *internal oblique/rectus abdominus*) would be higher in the CLBP_I group as an attempted to increase stability. To further establish if passive subsystem damage associated with findings of clinical lumbar instability was the determinate of muscle pattern changes, a separate subset of patients with non-specific chronic mechanical low back pain (CLBP_N) was compared to the CLBP_I group. Pattern differences between CLBP_I and CLBP_N; would also lend support to the idea that unique impairments exist between these subgroups of the chronic low back pain population. These hypotheses were tested by recording activity of ten trunk muscles during functional reach under two loading conditions.

2. Methods

2.1. Participants

A total of 39 participants with recurrent or chronic low back pain were recruited from an orthopedic surgery practice and completed the testing protocol. Inclusion criteria were current pain episode greater than 3 months, primary complaint of back and not leg pain, and inability to work or perform essential activities of daily living secondary to pain. All of these individuals had failed to resolve their symptoms in a course of conservative care, which included medical management, as well as physical rehabilitation. Potential participants were excluded if they had prior spine surgery, structural deformities or neurological findings indicating radiculopathy. The data from seven subjects were eliminated from this analysis secondary to demonstration of a high degree of psychosocial involvement (three out of five positive findings on Waddell's signs (Waddell, 1987) or inconsistency in performance during the clinical examination or testing).

The data from the remaining 32 CLBP participants were separated into the two groups. Twenty CLBP patients met the criteria for significant passive subsystem damage with moderate to severe degenerative disc disease (DDD) on magnetic resonance imaging (MRI) and positive low pressure discography at one or more corresponding levels. Although not a criteria for admission to this group, three of these individuals had documented spondylolisthesis at a segmental level corresponding to their DDD and positive discography. The underlying assumption was that damage to the major stabilizing structure of the spinal segment (disc) resulted in an increased segmental neutral zone as described by Panjabi et al. (1988). Evidence supporting the relationship between DDD, positive discography and spinal segmental hypermobility is offered in several studies (Eisenstein et al., 1999; Mimura et al., 1994; Tanaka et al., 2001). These medical findings in conjunction with clinical examination results placed these individuals into the clinical lumbar instability (CLBP_I) group. The recommended medical management of these 20 individuals was spinal fusion. The remaining 12 patients were diagnosed with non-specific mechanical low back pain (CLBP_N). They demonstrated DDD consistent with age-related changes and no evidence of spondylolysis or spondylolisthesis on MRI. In addition, these subjects demonstrated either negative discography (10/12) or negative flexion–extension films (2/12) which decreased the suspicion of an increased neutral zone or segmental hypermobility.

Asymptomatic controls ($n = 20$) were matched by age, sex and body mass index to the CLBP_I group. The control subjects reported no history of low back pain that required medical assessment or limited function for more than 3 days. Standard anterior–posterior and lateral flexion–extension views were completed on the control subjects to rule out degenerative changes deemed abnormal for the subject's age or evidence of an asymptomatic segmental hypermobility. All participants were evaluated by the same physician (P.M.), with that physician reading all imaging studies, performing the discography procedures and ruling out other medical diagnoses. Descriptive information for the participants is outlined in Table 1. There were no significant differences between the three groups based upon age or body mass index. Pain (11-point numeric pain rating scale), self-report disability (Roland–Morris disability questionnaire) and clinical measures were not significantly different between the two CLBP subgroups (Table 1). This study was approved by the Institutional Review Board of Drexel University and all subjects signed an informed consent prior to participation.

2.2. Instrumentation

Bipolar, pre-amplified surface electromyography (sEMG) electrodes (CMMR >100 dB, bandwidth 6–

29 kHz, 300–380 gain, inter-electrode distance 35 mm; Motion Control, Inc., Salt Lake City, UT, USA) were applied over five trunk muscles bilaterally: internal oblique (IO) (midway between the anterior superior iliac spine and pubic tubercle above the inguinal ligament), external oblique (EO) (15 cm lateral to umbilicus), rectus abdominus (RA) (3 cm lateral to umbilicus), lumbar erector spinae (ES) (3 cm lateral to midline, centered at the level of the L2 spinous process), lumbar multifidus (LM) (2 cm lateral to midline, centered at the level of the L5 spinous process) and ground over right lateral malleolus. Light skin abrasion and cleansing with alcohol preceded application of electrodes with conduction gel and double-sided foam tape. Electrodes placement was consistent with previous studies (Cholewicki et al., 1997; Ng and Richardson, 1996). Muscle activity was recorded at 1248 Hz. Raw sEMG signals were band pass filtered (Bessel high pass at 10 Hz and a Butterworth low pass at 750 Hz) and differentially amplified with a gain of 1500–3800 to achieve 3–5 V peak to peak activity during the reference contractions.

Kinematic data related to the spine position were collected (40 Hz) using a 3 Space Fastrak (Polhemus Incorporated, Colchester, VT, USA) with a lightweight magnetic receiver directly mounted to the skin over the L1 spinous process with double-sided adhesive tape. The Polhemus transmitter defined the global reference frame. Kinematic data representing trunk position were defined relative to the subject's neutral standing posture. Raw sEMG and kinematic data were simultaneously collected through a custom LabVIEW program (National Instrument, Austin, TX, USA) and digitally stored.

2.3. Testing procedures

Normalization of trunk flexor and extensor muscle activity was completed using submaximal isometric contractions. Each abdominal group was normalized to the highest activation level produced during the isometric hold (5 s) of either a gravity resisted abdominal crunch or crunch with rotation. Extensor muscles were normalized to the highest activation level achieved during submaximal isometric contraction in a modified standing position (20% of subject's lumbar flexion) with hip and pelvic motion restrained. The Kin-Com (Chattecx Corp., Chattanooga, TN, USA) back testing unit was modified for this purpose. The target submaximal force was calculated using 40% of the subject's body weight. This calculation was modified from research by Mayer et al. (1985) and pilot work with similar chronic low back pain subjects.

The functional task, a forward reaching activity, was performed for three continuous trials starting in a position of trunk extension (Fig. 1). The reaching task was completed holding the upper extremities at 90° of

Table 1
Descriptive statistics (mean (SD)) and clinical characteristics of the chronic low back pain and control subjects

	Control (<i>n</i> = 20)	Clinical instability (<i>n</i> = 20)	Non-specific (<i>n</i> = 12)	Significance ^a (<i>P</i> -value)
Sex	4F, 16M	4F, 16M	7F, 5M	–
Age (years)	40.6 (8.9)	42.9 (8.7)	44.3 (5.9)	NS/NS
Body mass index (kg/m ²)	25.2 (2.8)	26.8 (5.4)	29.5 (7.5)	NS/NS
Positive discography (# segments)	NA	2.4 (1.0)	NA	–
Pain location (% back pain only)	NA	60%	75%	NS
Current symptoms onset (years)	NA	2.8 (3.7)	3.8 (3.1)	NS
NPRS ^b pre-test (0–10)	NA	4.7 (2.3)	4.3 (2.3)	NS
NPRS post-test (0–10)	NA	5.4 (2.7)	5.1 (2.7)	NS
Lumbar flexion (cm)	6.4 (2.6)	6.4 (3.8)	5.9 (2.4)	NS
Functional reach (cm)	38.1 (11.7)	32.6 (7.5)	29.7 (9.9)	0.022/NS
Trunk extensor strength (N)	342.2 (148.7)	250.8 (112.3)	202.2 (75.8)	0.002/NS
Trunk flexion (% painful)	NA	29%	10%	NS
Return to standing from flexion (% painful)	NA	48%	40%	NS
Return to standing (% aberrant motion)	9%	48%	30%	0.005/NS
Lumbo-pelvic rhythm (% reversal)	5%	38%	10%	0.013/NS
Extension hinge (%)	23%	57%	70%	0.005/NS
RMQ ^c (0–24)	0.5 (0.21)	11.1 (4.4)	11.5 (4.6)	0.000/NS
SF-36 ^d (physical component score)	55.9 (5.9)	34.7 (7.3)	36.5 (10.8)	0.000/NS
SF-36 (mental component score)	51.2 (9.6)	43.6 (14.0)	45.5 (11.7)	0.035/NS

^a Comparison of combined chronic low back pain to control subjects/comparison of two chronic low back pain groups; NS indicating not significant.

^b Numeric pain rating scale (higher score indicates more painful condition).

^c Roland–Morris disability questionnaire (higher score indicates greater disability).

^d Medical Outcomes Study Short Form (SF-36) Health Status Profile (norm based to general US population mean 50, SD 10; lower score indicates reduced health status).

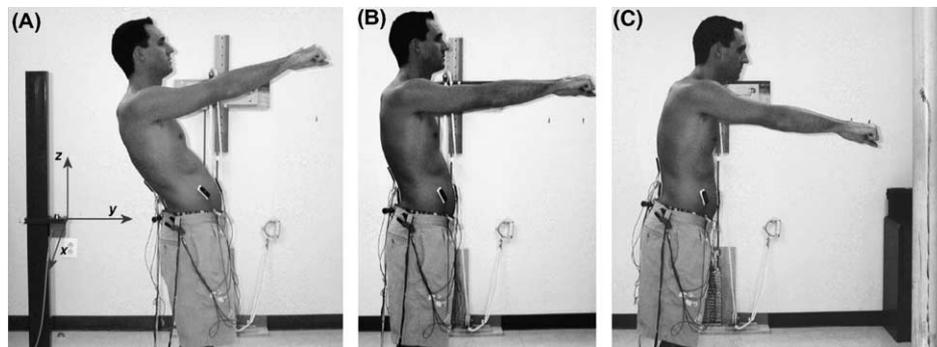


Fig. 1. This series depicts the no load condition of the functional reaching task. Panel A: the start position. Panel B: midrange or neutral position. Panel C: the maximum forward position at target. Subjects moved through a range of approx. 15° of trunk extension to 15° of flexion with respect to the global reference frame of the transmitter. Subjects were given a visual target to mark standardized reaching distance (pole, (C)).

shoulder flexion. Subjects selected a comfortable stance within foot prints shoulder width apart. The hips and pelvis were free to move. The excursion of the reach was standardized at 50% of the participant's forward reaching distance, determined by Functional Reach (Nakamura et al., 1988). Speed of movement was standardized (6 s cadence; approximately 10°/s) to control for its effects on muscle activation levels (Luoto et al., 1996). This movement was relatively slow as it amounted to taking 2 s to reach forward approximately 6–8 in. to a target. Data were collected throughout the reaching motion.

The task was completed under two conditions with a minimum of 1 minute of rest between no load (Fig. 1)

and holding a 5 lb sandbag with both hands. This protocol was developed based upon work by Cholewicki and McGill (1996) indicating that in upright tasks with little muscle demands, such as standing with no load, the spine functions close to the threshold of buckling. The no load condition provided a suitable model for testing the motor control system's ability to provide general trunk stability, while the additional load increased the stability challenge.

2.4. Data management and analysis

To determine sEMG signal amplitude, the raw sEMG data from a baseline resting signal, the reference

contractions and reaching activity was first filtered using an algorithm adapted from Aminian et al. (1988) to reduce heart rate artifact. The signal was further filtered using root-mean-square (RMS) processing with a time constant of 62 ms and then baseline resting levels were subtracted. The RMS sEMG signal for each muscle corresponding to the neutral spine position (0° of trunk flexion) was extracted from the reaching data and computed for each trial. The signal was then averaged over the three trials and between corresponding left and right trunk muscles. Normalized muscle activation was calculated using the submaximal isometric contraction for each muscle group creating a percent activation. Co-contraction (*flexors/extensors*) and abdominal (IO/RA, EO/RA) and extensor (LM/ES) synergist patterns were calculated using the RMS values to create synergist ratios.

The muscle recruitment patterns described in this paper represent trunk muscle activation and patterns of co-contraction at 0° of trunk flexion, during the forward phase of the reaching motion. Comparisons were made between groups and loading conditions. To address the hypotheses related to differences in muscle recruitment pattern between asymptomatic control and the CLBP_I group, a repeated measures ANOVA with between-subject factor of group (asymptomatic and CLBP_I) and within-subject/repeated factor of load (no load, 5 lb load) was used. This analysis was completed on 20 matched pairs of participants. Two planned comparisons using orthogonal contrasts were employed to test the specific hypotheses related to group difference. The first tested the hypothesis regarding differences between the subgroups of CLBP patients and the second tested differences between the asymptomatic control group and the combined CLBP groups. The purpose of the second comparison was to contrast our findings to previously published studies from other laboratories. Activation parameters for each muscle group were evaluated independently with significance level set at $P \leq 0.05$ for each analysis.

3. Results

3.1. Muscle activation levels

Fig. 2 provides group mean muscle activations with standard error for the no load and 5 lb load condition at 0° of trunk flexion. The rectus abdominus ($F_{1,36} = 5.226$, $P = 0.0001$) and external oblique ($F_{1,35} = 18.541$, $P = 0.028$) muscles had significantly higher activation levels in the CLBP_I group compared to matched asymptomatic controls. There was a significant main effect for load in all muscle groups except the external oblique (Table 2). A significant load x group interaction ($F_{1,38} = 6.406$, $P = 0.016$) for the lumbar

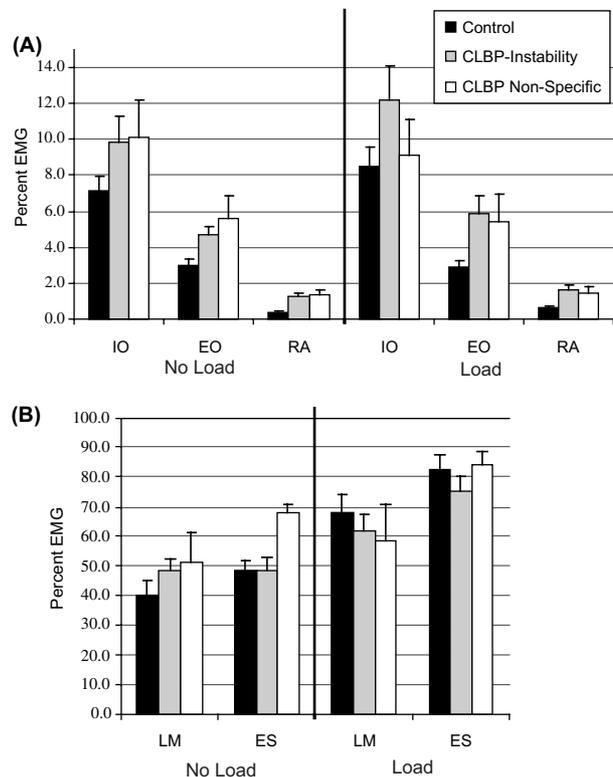


Fig. 2. Group mean and standard error of the normalized muscle activation levels in the no load and 5 lb load conditions. Panel A: abdominal muscle groups: internal oblique (IO), external oblique (EO) and rectus abdominus (RA). Panel B: extensor muscle groups: lumbar multifidus (LM) and erector spinae (ES).

Table 2

Results of the repeated measure ANOVA for muscle activation levels and ratios between the chronic mechanical low back pain instability subgroup ($n = 20$) and matched asymptomatic control group ($n = 20$)

ANOVA	Main effect (group)	Main effect (load)	Interaction
<i>Muscle groups</i>			
Internal oblique (IO)	0.217	0.012	0.633
External oblique (EO)	0.028	0.183	0.522
Rectus abdominus (RA)	0.0001	0.0008	0.172
Lumbar multifidus (LM)	0.962	0.0004	0.016
Erector spinae (ES)	0.748	0.0004	0.486
<i>Synergist ratios</i>			
IO/RA	0.059	0.033	0.609
EO/RA	0.006	0.318	0.964
LM/ES	0.986	0.005	0.782
Flexors/extensors	0.822	0.000	0.181

Data are presented as P -values.

multifidus was found due to a 28% increase in activation in the control group, but only a 14% increase in the CLBP_I group. Planned comparisons between the CLBP_I and CLBP_N groups demonstrated no significant differences (Table 3). The combined CLBP groups demonstrated significantly higher levels of normalized muscle activation than the asymptomatic control group for

Table 3

Results of planned contrast for muscle activation levels or ratios between the two chronic mechanical low back pain subgroups and combined chronic low back pain ($n = 32$) and control ($n = 20$) groups

Planned contrasts condition	CLBP vs. control		CLBP _I ^a vs. CLBP _N ^b	
	No load	Load	No load	Load
<i>Muscle groups</i>				
Internal oblique (IO)	0.321	0.721	0.579	0.520
External oblique (EO)	0.036	0.024	0.345	0.842
Rectus abdominus (RA)	0.0002	0.0003	0.357	0.628
Lumbar multifidus (LM)	0.083	0.325	0.976	0.879
Erector spinae (ES)	0.205	0.979	0.097	0.426
<i>Synergist ratios</i>				
IO/RA	0.025	0.139	0.632	0.266
EO/RA	0.028	0.041	0.860	0.406
LM/ES	0.435	0.997	0.425	0.809
Flexors/extensors	0.405	0.399	0.690	0.441

Data are presented as P -values.

^a Chronic low back pain subgroup defined as clinical instability.

^b Chronic low back pain subgroup defined as non-specific.

the rectus abdominus and external oblique in the both the no load and 5 lb conditions.

3.2. Muscle co-activation patterns

Fig. 3 provides group mean co-activation ratios with standard error for the no load and 5 lb load condition at 0° of trunk flexion. The EO/RA ratio ($F_{1,37} = 8.612$, $P = 0.006$) was significantly lower, with a trend toward a lower IO/RA ratio ($F_{1,34} = 3.813$, $P = 0.059$) for the CLBP_I group compared to the asymptomatic controls. There was a main effect for load with decreases in all ratios except the EO/RA. This includes a significant decrease in the co-contraction ratio (Table 2). Planned comparisons between the CLBP_I and CLBP_N groups demonstrated no significant differences (Table 3). The combined CLBP groups demonstrated a significantly

lower IO/RA ratio (no load) and EO/RA ratio (no load, 5 lb) than the asymptomatic control group (Table 3).

4. Discussion

4.1. Muscle activation levels and patterns

This study compared trunk muscle recruitment pattern between two subgroups of CLBP patients and asymptomatic control using both normalized muscle activation and RMS sEMG patterns of co-activation. The findings demonstrate differences in activation strategies of the CLBP_I subgroup and matched control subjects, but not between the CLBP subgroups themselves. Using both normalized muscle activity and RMS ratios of activation to describe muscle recruitment allowed us to address the limitations associated with EMG normalization and to also look at patterns of synergistic muscle activation (Edgerton et al., 1996; Van Dieen et al., 2003). We choose to normalize our EMG amplitude to standardized submaximal isometric contraction because of reported intolerance to maximal resistance and significant intrasubject variability for maximum voluntary isometric contractions in patients with low back pain (Yang and Winter, 1983). However, these methodological features limit direct comparison of activity levels with studies normalizing to maximal voluntary contractions or submaximal references using different activities or trunk positions. Within these limitations, and others, we believe our data add to the current knowledge of neuromuscular control patterns in specific subgroups of patients with CLBP during a standardized reaching task.

The data from this study supported portions of our hypotheses related to differences between our CLBP_I and matched control group. Muscle activation was generally higher in the CLBP_I group with significantly high-

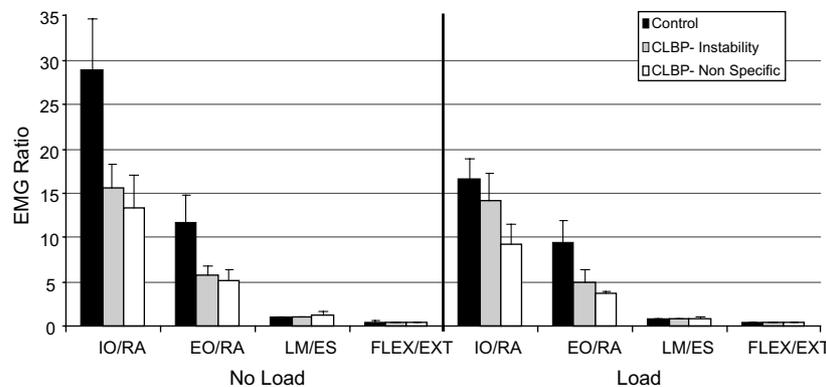


Fig. 3. Group mean and standard error of the synergist ratios in the no load and 5 lb load conditions. Synergist ratios: internal oblique/rectus abdominus (IO/RA), external oblique/rectus abdominus (EO/RA) and lumbar multifidus/erector spinae (LM/ES), and co-contraction ratio defined as abdominals/extensors (FLEX/EXT).

er activation of rectus abdominus and external oblique and a lower EO/RA synergist ratio. While, there appears to be increased muscle activity as either a compensation for or a precipitator of passive subsystem damage, the co-contraction ratio (*flexors/extensors*) was not significantly different between groups. The abdominal synergist ratios although significantly different, did not support our hypothesis that coordinated response from the segmental trunk muscles relative to the multi-segmental muscle would raise those synergist ratios. We found few studies where the investigators specifically assessed trunk muscle recruitment patterns in patients with a diagnosis of clinical lumbar instability. Our findings coincide with data from O'Sullivan et al. (1997a) who reported a significantly lower IO/RA ratio in CLBP patients with spondylolisthesis when performing specific abdominal isometric exercises. In our study, the lower abdominal synergist ratios resulted from greater activation of the rectus abdominus relative to external or internal oblique muscle recruitment. Although we did not find significant differences in our subjects extensor muscle patterns, Lindgren et al. (1993) reported differences in segmental extensor activation in stable vs unstable segments, and Sihvonen et al. (1991) reported a lower ratio of activity for the erector spinae muscles (activation averaged over the entire *flexion/extension* phase of motion) in their subjects with chronic low back pain. The extensor ratio was lowest in their subgroup of CLBP subjects (25/87) diagnosed with segmental hypermobility on flexion–extension radiographs.

Using the results of our planned comparison between the combined CLBP subgroups and asymptomatic control subjects we are able to further discuss our data relative to previously published studies. While our findings are generally supported by Chiou et al. (1998), who also found greater activation around neutral standing of the rectus abdominus and external oblique muscles in their low back pain subjects, additional comparisons are limited by methodological differences and the lack of description of their low back pain subjects. Sihvonen et al. (1991) also reported a decreased ratio of extensor activity in their non-specific CLBP subjects. While, Van Dieen et al. (2003) did not find a significant difference in the IO/RA ratio, between their group of non-specific CLBP subjects and controls; they did report a significantly higher co-contraction ratio (*flexors/extensors*) and ratio of lumbar to thoracic erector spinae activity. However, it should be noted that in addition to their blocking pelvic/hip motion during testing, their CLBP group was different from ours relative to pain intensity (lower), self-reported disability (less) and work status (working) at the time of their data collection.

To date, no optimal pattern of activation has been experimentally determined and results of biomechanical modeling studies do not indicate that one particular muscle group is the best stabilizer of the lumbar spine

(Cholewicki and VanVliet, 2002). Several studies do demonstrate that increased co-contraction (flexors/extensors) and higher synergists ratios (IO/RA, lumbar/thoracic ES) result in enhanced spine stability particularly in upright neutral postures (Cholewicki et al., 1997; Granata and Orishimo, 2001; Van Dieen et al., 2003). In addition, Gardner-Morse and Stokes (1998) demonstrated through modeling that the abdominal muscles may play a more important role than the extensors in providing trunk stability. Therefore, it is plausible that an altered abdominal recruitment pattern may lead to deficiencies in spine stability.

Several authors have suggested that lumbar stability is maintained by segmental muscles and/or a coordinated response of segmental and multi-segmental synergist muscle groups (Cholewicki and VanVliet, 2002; Crisco and Panjabi, 1991). In light of this work, we propose that the trunk muscle recruitment pattern demonstrated by our CLBP subjects reflects a muted response of segmental musculature (in our study the IO and LM) and reliance upon multi-segmental muscle activation. This pattern of recruitment may suggest muscle or motor control impairment and represent an inability to successfully meet the demand for maintaining spinal stability. These findings are consistent with recent research that indicated dysfunction of the segmental abdominal musculature (internal oblique and transverse abdominus) in CLBP patients during perturbation (Hodges and Richardson, 1998). Additionally, atrophy and altered function of the trunk extensors, particularly the lumbar multifidus, has been reported (Hides et al., 1996; Hides et al., 1994). We acknowledged that there is an ongoing debate related to the ability to accurately determine lumbar multifidus muscle function using surface electrodes (Stokes et al., 2003). Thus, our findings, relative to independent activation of the LM may be contaminated by crosstalk with the erector spinae.

The second hypothesis related to proposed differences between the two CLBP subgroups was not upheld by the data. The absence of this difference suggests that clinical lumbar instability, as defined in this study (moderate to severe DDD and positive discography), may not be the determinant of the alterations in muscle activation patterns. These pattern changes could be the result of other factors (altered mechanoreceptor information, muscle atrophy, reflex inhibition or pain) common to the two CLBP subgroups (Brumagne et al., 2000; Hides et al., 1996; Sterling et al., 2001). This is a reasonable alternative hypothesis given that our CLBP subgroups did not demonstrate differences in time since symptom onset, pain intensity or location, lumbar flexion ROM, clinically observed movement patterns or disability level. In addition, our operational definition of clinical lumbar instability, while suggestive of underlying neutral zone changes or segmental hypermobility, does not directly

measure these parameters. Thus it is possible that our CLBP groups were not significantly different in this attribute. Faced with these findings we also ran an additional comparison of CLBP subgroups, this time separating them based upon the number of lumbar segments demonstrating degenerative change (one level vs. greater than one level). The hypothesis being that those individuals with a greater amount of passive subsystem damage would demonstrate a different recruitment pattern. Again we found no differences between the subgroups.

4.2. Effects of load

The response of the musculature to an increased external load resulted in an expected significant increase in activation level for both the CLBP and control groups. The abdominal muscles on average increased activation by 1–3% while the extensors increased by 13–31%. The increase in activity due to increased load is consistent with previously reported findings in CLBP subjects and asymptomatic individuals (Huang et al., 2001; Ross et al., 1993). The greater increase by extensor muscle activity was expected due to the increased trunk flexion moment. The reason for the lack of significantly increased muscle activity of the external oblique or the lumbar multifidus interaction (control groups approximate twofold increase over CLBP_I group) cannot be directly explained and its effect on spinal stability are unclear. These findings may be associated with use of adaptive movement patterns to decrease the external flexion moment, an inability to further increase activation secondary to muscle inhibition, or an altered recruitment pattern in response to tissue injury, pain or avoidance behavior. It may be that the general increase in all trunk muscle activity satisfied the need for any additional stability through compression forces alone (Cholewicki et al., 2000). Future studies would benefit from the calculation of spinal stability or stiffness achieved by specific muscle recruitment patterns.

The synergist and co-contraction (*flexors/extensors*) ratios were affected by the load increase, with the exception of EO/RA. Thus, the additional 5 lb load in the hands significantly changed the recruitment, but did not assist in differentiation of groups with the exception of the lumbar multifidus response discussed previously. The decreased synergist and co-contraction ratios were primarily the result of a greater relative increase in the multi-segmental muscle response (RA, ES) and load sharing.

5. Conclusions

The data from our subjects does not support the theory that passive subsystem damage drives the muscle

recruitment patterns of patients with CLBP. While the altered abdominal recruitment patterns demonstrated by our CLBP patients suggest reliance on multi-segmental abdominal musculature, the 1–2% mean group difference in individual muscle activation, although statically significant, may have limited clinical implications. However, we believe the synergists ratios are a better and more meaningful indicator of trunk motor control, particularly given the issues surrounding normalization of EMG data. The model proposed by Panjabi (1992) would suggest that these pattern changes were driven by the need to provide increased spinal stiffness around the subject's neutral spine position. The CLBP subjects in our study increased trunk muscle activity overall which would serve to enhance trunk stability, however no difference in the co-contraction ratio and their synergist pattern of a lower IO/RA might suggest they were not successful at achieving the goal. Perhaps our CLBP subjects represent those individuals who are unable to adequately compensate for their spinal dysfunction "non-copers" and this has resulted in their chronic symptoms and prolonged functional limitations. This is supported by their history of long standing low back pain and moderate to severe functional limitations as per self-report disability scores. As such, interventions that address trunk muscle recruitment strategies, particularly relative activation levels of the abdominal musculature may be an important component of a therapeutic exercise program for these individuals. At this time clinicians and researchers are theorizing that improved activation of the segmental trunk muscles with a goal of achieving higher segmental to multi-segmental synergist ratios of activation is the most efficient means of attaining needed trunk stability (Van Dieen et al., 2003), reducing pain and improving function (Hides et al., 2001; O'Sullivan et al., 1997b; Rasmussen-Barr et al., 2003).

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