ORIGINAL ARTICLE

Differences in Feedforward Trunk Muscle Activity in Subgroups of Patients With Mechanical Low Back Pain

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ABSTRACT. Silfies SP, Mehta R, Smith SS, Karduna AR. Differences in feedforward trunk muscle activity in subgroups of patients with mechanical low back pain. Arch Phys Med Rehabil 2009;xx:xxx.

Objective: To investigate alterations in trunk muscle timing patterns in subgroups of patients with mechanical low back pain (MLBP). Our hypothesis was that subjects with MLBP would demonstrate delayed muscle onset and have fewer muscles functioning in a feedforward manner than the control group. We further hypothesized that we would find differences between subgroups of our patients with MLBP, grouped according to diagnosis (segmental instability and noninstability).

Design: Case-control.

Setting: Laboratory.

Participants: Forty-three patients with chronic MLBP (25 instability, 18 noninstability) and 39 asymptomatic controls.

Interventions: Not applicable.

Main Outcome Measures: Surface electromyography was used to measure onset time of 10 trunk muscles during a self-perturbation task. Trunk muscle onset latency relative to the anterior deltoid was calculated and the number of muscles functioning in feedforward determined.

Results: Activation timing patterns ($P<.01$; $\eta=.50$; $1-\beta=.99$) and number of muscles functioning in feedforward ($P=.02$; $\eta=.30$; $1-\beta=.83$) were statistically different between patients with MLBP and controls. The control group activated the external oblique, lumbar multifidus, and erector spinae muscles in a feedforward manner. The heterogeneous MLBP group did not activate the trunk musculature in feedforward, but responded with significantly delayed activations. MLBP subgroups demonstrated significantly different timing patterns. The noninstability MLBP subgroup activated trunk extensors in a feedforward manner, similar to the control group, but significantly earlier than the instability subgroup.

Conclusions: Lack of feedforward activation of selected trunk musculature in patients with MLBP may result in a period of inefficient muscular stabilization. Activation timing was more impaired in the instability than the noninstability MLBP subgroup. Training specifically for recruitment timing may be an important component of the rehabilitation program.

Key Words: Electromyography; Low back pain; Motor skills; Reaction time; Rehabilitation.

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MUSCLE IMPAIRMENT AND motor control dysfunction appear to be strongly associated with chronic and recurrent MLBP.[1-5] While much of the literature has focused on differences in muscle activation level, timing and pattern of recruitment also play an important role in spine stability and movement control. Dynamic trunk stability could be compromised by delayed activation of trunk musculature during challenges to postural control from unexpected perturbation or voluntary movement.

The central nervous system uses several strategies (postural preparation, anticipatory postural adjustments, reactive postural adjustments) to regulate control of posture during movement. *Postural preparation* occurs well before movement in an attempt to increase one's base of support or stiffen a joint or joints prior to a perturbation (ie, holding onto a handrail during stair climbing). Adjustments in posture that occur with or just before initiation of voluntary movement are termed *anticipatory* or *feedforward postural adjustments*. These adjustments occur in anticipation of a known effect of a movement on postural stability and function to minimize the postural disturbance. Reactive or feedback strategies occur after the movement and benefit from input of sensory information to the system that triggers automatic strategies within 100 milliseconds postdisturbance. This strategy is the primary defense against unexpected or external perturbations.[6] Models for testing trunk postural control have been developed for each of

List of Abbreviations

COM	center of mass
Cont	contralateral to side of arm perturbation
DDD	degenerative disk disease
EMG	electromyography
EO	external oblique
ES	erector spinae
ICC	intraclass correlation coefficient
IO	internal oblique
IO/TrA	internal oblique/transversus abdominis
Isp	ipsilateral to side of arm perturbation
LBP	low back pain
LM	lumbar multifidus
MLBP	mechanical low back pain
MRI	magnetic resonance imaging
RA	rectus abdominis
RMQ	Roland-Morris Questionnaire
SF-36	Medical Outcomes Study 36-Item Short-Form Health Survey, v1
TrA	transversus abdominis

these strategies. Postural preparation strategies have been assessed using ramped effort trunk muscle activation followed by transient support surface perturbation.[7] Reactive strategies have used expected or unexpected external loading or unloading of the trunk[8,9] or perturbation of a support surface.[10,11] Anticipatory or feedforward postural control strategies have been assessed using self-perturbation of extremities to test standing trunk postural control. This paradigm can be used to relate the timing of extremity movement or muscle activation to that of the trunk muscle activation.[12-15]

Using a model of self-perturbation of a single upper extremity provides a means to assess trunk muscle timing and activation patterns during an asymmetrical challenge to trunk postural control. Because this perturbation is self-initiated, the central nervous system can predict the changes and thus preprogram its feedforward response. Evidence has been presented that attributes this anticipatory muscle activation to attempted control of COM displacement and trunk orientation.[16,17] In fact, preparatory activity of trunk muscles appears necessary for preservation of postural equilibrium, because electromechanical delay of the reactive strategy of the trunk muscles is greater than 100 milliseconds.[18]

The trunk postural response in healthy subjects using this paradigm indicates that specific trunk muscles—TrA, IO, and superficial LM—act in a feedforward manner by firing prior to or in conjunction with the limb prime mover to dampen the moments created by the perturbation.[13,15] It has been suggested that TrA and IO activation is a general response to a postural challenge, because their feedforward activation is not based on the direction of extremity movement.[19,20] However, interpretation of data from more recent studies suggests this may not be the case.[21,22] Much of the research and clinical focus has been on the role of the TrA, which is proposed to stiffen the spine by creating a musculofascial corset around the lumbar spine or through the creation of intra-abdominal pressure.[23-25] Theoretically, feedforward activation of the TrA contributes to control of spinal segmental motion, which is necessary to prepare the spine for contraction of the larger trunk musculature and for limb movement. Larger and more superficial trunk musculature also responds in a feedforward manner; however, this appears to be related to the direction of extremity perturbation or COM movement.[19,20] For example, unilateral shoulder flexion movements are generally accompanied by a preparatory firing of the trunk extensor musculature. During rapid upper extremity flexion, the COM is moved anteriorly; consequently, the extensors fire prior to limb movement, presumably to dampen the postural disturbance.[16]

Hodges and Richardson[13,26] and Hodges[27] used this self-perturbation paradigm to examine differences in the response of trunk muscles in subjects with and without chronic MLBP. They found that the TrA and IO did not act in a feedforward manner in patients with a history of chronic MLBP. Instead, the LM muscle group activated earliest and in a feedforward manner in the patients with MLBP. These studies suggest that inappropriate muscle recruitment and timing may be a component of or a predisposing factor in chronic or recurrent MLBP.[28,29]

To date, most research reporting impaired feedforward trunk postural control has been completed on small (n=15–20) heterogeneous samples of patients with chronic MLBP, many of whom were demonstrating minimal to no symptoms or disability at the time of the study.[13,27,30] However, the literature indicates that not all patients with chronic or recurrent MLBP share the same underlying cause or level of impairment.[31-34] In addition, it has been suggested that heterogeneity in research samples of patients with MLBP may account for the reported high variability in dependent variables representing muscle

activation data.[35,36] This variability is hypothesized to be the result of concealed patient subgroups.[37,38] Nevertheless, studies comparing trunk muscle timing and activation patterns in subgroups of the MLBP population have not been reported.

The subgroup of patients with MLBP that is most often associated with poor neuromuscular control includes those patients suspected of having segmental hypermobility or spinal instability.[29,39-41] In fact, exercises that target key stabilizing muscles (TrA, LM) of the trunk have become the standard of care for patients with chronic and recurrent MLBP.[24,42,43] These exercises are the same exercises as those prescribed for patients subgrouped into the "stabilization" category of a widely used LBP subclassification system (Treatment-Based Classification System).[40,44,45] In the clinical prediction rule study that identified the stabilization subgroup, over 70% of the patients had previous episodes of LBP.[40] Thus, a connection between lumbar instability and chronic and recurrent LBP seems likely, so this subgroup was chosen for this study.

In addition to a lack of investigation into subgroups, previous studies assessing trunk feedforward control strategies recorded from only 1 side of the trunk or from a limited number of trunk muscles.[27,46] Given the redundancy of the trunk musculature and reported differences in contralateral muscle activations,[21,47] the current literature may provide only a partial picture of the trunk's postural response to self-initiated movement of the extremities. By evaluating bilateral trunk muscles in subgroups of patients with MLBP, we may begin to identify specific dysfunctions in trunk neuromuscular control that could assist with more directed treatment.

The purpose of this study was to describe bilateral trunk muscle activation patterns and to investigate differences in trunk muscle timing between subgroups of patients with chronic MLBP and asymptomatic controls. Based on previous findings of delayed onset of trunk muscles in patients with chronic LBP, we hypothesized that subjects with MLBP would demonstrate an altered pattern of muscle onset and have fewer muscles functioning in a feedforward manner than the asymptomatic control group. We further hypothesized that we would find differences between patients with MLBP attributed to segmental instability and those without clinical signs and symptoms of segmental hypermobility. The subgroup hypothesis was based on clinical experience and research indicating improved treatment outcomes for patients with MLBP who were subclassified.[44]

METHODS

Subjects

Eighty-two subjects completed the testing protocol, 43 patients with chronic MLBP and 39 asymptomatic controls. Subjects with MLBP were recruited from a university orthopedic practice specializing in spine care. All patients with MLBP had current symptom durations in excess of 3 months and LBP pain that significantly limited normal activities. Their primary complaint was LBP with minimal leg pain that failed to resolve adequately with conservative care. Conservative care included a trial of physical therapy (6–8wk) and pharmacologic management. Control subjects were recruited from the university campus and surrounding community. These participants reported no history of LBP that required the attention of a health care practitioner or limited function longer than 3 days. The study was approved by the university's Institutional Review Board, and informed consent was obtained from all participants. All subjects were evaluated by a physical therapist prior to testing to determine their eligibility for participation. Those subjects with a history of spinal or hip surgery, osteoporosis,

Table 1: Descriptive Statistics and Clinical Characteristics of the Chronic Mechanical Low Back Pain and Control Groups

	Control (n=39)	MLBP Instability (n=25)	MLBP Noninstability (n=18)	Control vs MLBP* P	Instability vs Noninstability[†] P
Sex	14 F, 25 M	7 F, 18 M	11 F, 7 M	ND	ND
Age (y)	39.3±9.5	42.5±8.6	41.2±8.4	.17	.63
Body mass index (kg/m²)	25.3±4.6	27.0±5.6	28.7±7.2	.05	.43
Positive diskography (no. segments)	ND	2.2±1.0	ND	ND	ND
Pain location (% back pain only)	ND	64	72	ND	.05
Current symptoms onset (y)	ND	7.1±7.8	5.7±5.6	ND	.05
NPRS[‡] pretest (0–10)	ND	4.2±2.3	3.3±2.5	ND	.88
Lumbar flexion[§] (cm)	6.1±2.2	6.2±3.6	5.5±1.9	.70	.46
Trunk extensor strength (N)	355±138	234±110	238±82	<.01	.90
Painful movement (%)					
Trunk flexion	ND	27	11	ND	.21
Return to standing	ND	52	33	ND	.23
Aberrant trunk motion[‖] (% of subjects)	13	65	50	<.01	.52
Extension hinge[¶] (%)	28	64	61	<.01	.87
RMQ[#] (0-24)	ND	11.6±4.8	8.1±6.3	ND	.05
SF-36** (Physical Score)	56.5±4.8	33.6±8.4	40.8±12.2	<.01	.03
SF-36 (Mental Score)	50.7±8.9	44.1±12.9	47.9±10.7	.04	.32

NOTE. Data represent mean ± SD unless otherwise indicated.
Abbreviations: F, female; M, male; ND, no data.
*P values for comparison of combined chronic MLBP and control groups; significant difference at α<.05.
[†]P values for comparison of chronic MLBP subgroups; significant difference at α<.05.
[‡]Numeric Pain Rating Scale (higher score indicates more painful condition).
[§]Measurement in centimeters, using Modified Schober technique (higher score indicates more motion).
[‖]Observed abnormal pattern of movement during standing trunk range of motion testing.
[¶]Observed hypermobility of a segment during active trunk extension.
[#]Higher score indicates greater disability.
**SF-36 Health Status Profile (norm based to general US population, mean ± SD, 50±10; lower score indicates reduced health status).

inflammatory joint disease, frank neurological loss (ie, lower extremity weakness and sensory loss), pain or paresthesia below the knee, pregnancy, scoliosis, leg length discrepancy, or vestibular dysfunction were excluded from the study.

The patients with chronic MLBP were separated into 2 subgroups, those with a diagnosis of segmental instability (n=25) and those with noninstability (n=18). Their diagnosis was made by an orthopedic spine surgeon. Patients meeting the criteria for the instability subgroup demonstrated at least moderate DDD on MRI with positive low pressure diskography at 1 or more corresponding lumbar levels. This subgroup demonstrated osteoligamentous injury or degeneration consistent with segmental hypermobility or instability.[41,48] The relationship among DDD, concordant pain on diskography, and increased segmental motion or neutral zone is offered by several researchers.[25,49-52] The medical intervention proposed for the subgroup diagnosed with instability was lumbar fusion. The noninstability MLBP subgroup (n=18) demonstrated mild to moderate DDD on MRI. Twelve of the 18 subjects in this subgroup had negative diskographies. The remaining 6 did not undergo diskography based on their medical imaging and symptoms, but did have negative flexion-extension imaging studies. These findings decreased suspicion of segmental hypermobility, or instability, in this subgroup. All of the subjects with MLBP had their medical imaging, diskography procedure, and examination completed and interpreted by the same orthopedic spine surgeon.

To control confounding variables, the controls (n=39) were matched by age (±5y), sex, and body mass index (±4 kg/m²) to the subjects with chronic MLBP. Standard static and dynamic lumbar radiographs were used to rule out significant degenerative changes (those atypical for the subject's age) and asymptomatic segmental hypermobility or instability. These images were interpreted by the same orthopedic surgeon who diagnosed the patients with MLBP.

The subjects' self-perceived health status and function were evaluated using 2 self-report questionnaires. The RMQ, a condition-specific outcome measure designed for the LBP population, and the SF-36, a generic health status questionnaire, both with well documented psychometric properties, were used to assess self-perceived disability.[53-58] An 11-point verbal numeric pain rating scale was obtained pretesting and posttesting along with a pain body diagram and a general history of the symptoms. Table 1 displays the group means and SDs for subject characteristics. To test for significant differences between the control and MLBP group, t tests were used. The clinical findings from the physical therapy examination and self-perceived pain and function are provided to describe our MLBP population better. MLBP subgroup differences were found in location of symptoms, RMQ score, and SF-36 physical component score (see table 1) using chi-square and t test analyses. The interpretation of any differences in physical therapy clinical findings between the subgroups can be found in the Discussion.

Instrumentation

Preamplified bipolar surface electrodes[a] with an interelectrode distance of 35mm (common mode rejection ratio >100 dB; bandwidth=6–29kHz; 300–380 gain) were used to record trunk muscle activity (1248Hz). Data were bandpass-filtered (10–500Hz), and the signal was differentially amplified[b] to achieve a peak of 3 to 5V during a reference contraction. Muscle activity was recorded over 5 trunk muscles bilaterally: IO\TrA, EO, RA, superficial LM, and lumbar ES.[5] The skin was prepped, and electrodes were aligned parallel to muscle fibers and placed in accordance with previous studies demonstrating that these placements maximize signal-to-noise ratio related to levels of cross-talk.[59-61] An electrode was positioned on the dominant upper extremity over the anterior deltoid.[46]
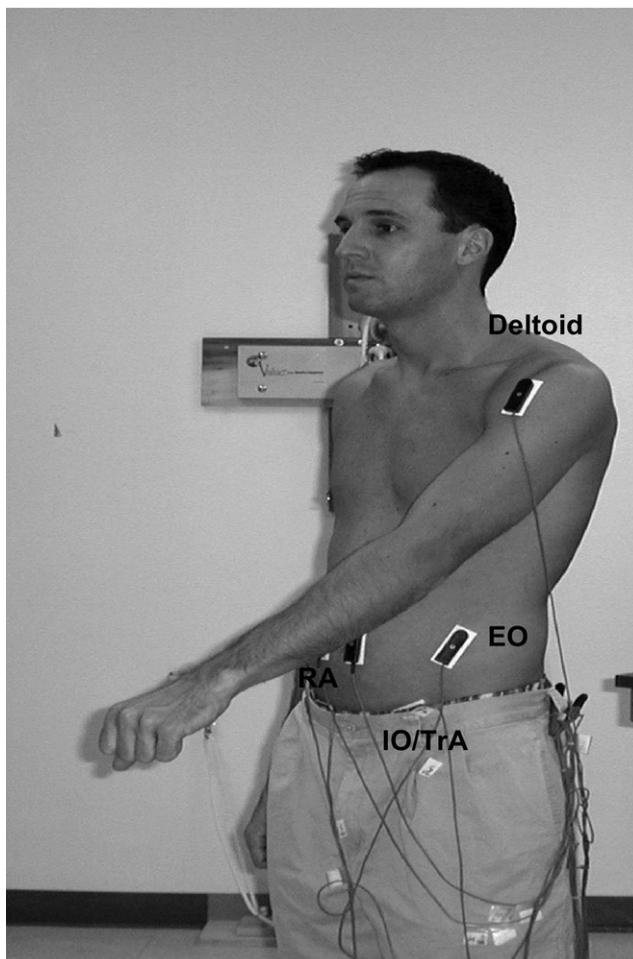
Fig 1. Model of self-perturbation of the upper extremity used to evaluate response of trunk musculature.

Protocol

The upper extremity self-perturbation paradigm was used to test the response of the trunk musculature to perturbation when the trunk was in the neutral position. Subjects stood in a relaxed position with their foot position standardized to shoulder width apart and weight equally distributed. A specific posture or alignment between the lumbar spine and pelvis was not mandated. Subjects were encouraged to relax in this posture for at least 20 seconds prior to each trial in an attempt to lower resting EMG activity. Three repetitions of rapid shoulder flexion with 30 seconds between trials were performed in response to an auditory stimulus (fig 1). An auditory warning stimulus was provided from 1 to 3 seconds prior to the auditory signal to raise the arm rapidly. Subjects performed 2 practice trials to allow for adjustment in direction and speed of arm movement. Because of reported differences in onset time and pattern between slow and fast movements, subjects were asked to move as quickly as possible through at least 60° of shoulder flexion.[13] Trials not meeting these performance criteria were rejected and the test repeated so that each subject had 3 appropriate trials for analysis.

Data Processing

Postprocessing consisted of removal of heart rate artifact,[62] full-wave rectification, and low-pass filtering (10Hz; 2nd-order

Butterworth). Using a custom LabView[c] program, muscle onset was determined as the point where EMG signal amplitude exceeded 2 times the baseline EMG for a minimum of 50 consecutive data points (42ms). Computer algorithm identification of each muscle's onset time was visually inspected by a single rater, blinded to group and muscle. Onset time was manually corrected if residual heart rate or baseline noise interfered with the accuracy of the computer's onset identification. Determination of changes was made using 2-millisecond resolution and recorded as the next data point meeting the established onset criteria. Rater reliability for detecting muscle onset time with blind, visual override was established using 10 randomly selected subjects and trials from the described study and by analyzing the trials 5 days apart. Intrarater reliability of muscle onset ranged from .66 to .99 for individual muscles using an $ICC_{3,1}$.

Muscle onset latency was defined as the time difference between the onset of contraction of individual trunk muscles and the anterior deltoid. Individual subjects' trunk muscle latencies for each trial were then categorized as either feedforward or feedback. Responses were categorized as feedforward if the trunk muscle onset was prior to or within 50 milliseconds of the deltoid onset. This criterion was established and used by several authors.[26,47,63] Onset latency for the 10 trunk muscles in each subject and the number of muscles functioning in feedforward were then averaged across the 3 trials. The subjects' mean values were used for subsequent data analysis.

Within-session reliability of dependent variables (average muscle latencies, number of feedforward muscles) was estimated with repeated testing in a subset of study subjects (n=18) using the same protocol. The subset included both control subjects and subjects with chronic MLBP. $ICCs_{3,3}$ reporting the results for average measures, kappa statistics, and standard error of measurement values can be found in table 2.

Data Analysis

Deltoid reaction time (time from auditory stimulus to anterior deltoid onset) was compared using an analysis of variance. Alpha level was set at 0.05. Because our subjects used the dominant arm to perform the self-perturbation, we categorized each trunk muscle as Isp or Cont relative to the upper extremity perturbation. Muscle latencies between the Isp and Cont RA, LM, and ES were not significantly different across subjects; therefore, data were collapsed for these muscle groups.

To describe each group's pattern of trunk muscle activation, we used repeated-measures ANOVAs to determine whether the

Table 2: Within-Session Reliability of Trunk Muscle Onset Latency, Deltoid Reaction Time, and Feedforward Classification

Muscle*	$ICC_{3,3}$	SEM (ms)	Variance†	CV (%)	κ‡
Isp IO/TrA	.52	93	3276	80	.53
Cont IO/TrA	.40	119	2818	92	.06
Isp EO	.78	36	99	114	.72
Cont EO	.81	46	740	92	.37
RA	.56	73	1733	105	.22
LM	.28	69	69	94	.26
ES	.40	40	155	96	.46
Deltoid	.83	27	167	96	ND

Abbreviation: CV, coefficient of variation; ND, no data.
*Reliability was calculated from a subset of study subjects (n=18) who repeated the protocol within the measurement session. Data represent both control subjects and subjects with chronic MLBP.
†Variance of mean values.
‡Reliability of feedforward classification.

activation latencies were different between the trunk muscles within each subject group. For post hoc muscle comparisons, a familywise error rate was set at alpha equals .15 (per comparison, α=.007).[64-66] This alpha level was used to maintain study power.

To assess group differences, the dependent variables of muscle onset latency and number of muscle functioning in feedforward were analyzed separately with multivariate analysis of variance and planned comparisons. Planned comparisons were used to test the specific hypotheses related to differences between the groups. The first planned comparison tested our hypothesis of differences between the asymptomatic control group and the heterogeneous MLBP group. We used these data to compare our results to those previously reported in the literature for nonspecific chronic LBP groups. The second planned comparison tested our hypothesis of differences between the subgroups of patients with chronic MLBP (instability, noninstability). Familywise error was again set at alpha equals .15 for the planned comparisons resulting in a per comparison error for latency at alpha equals .011 and for number of muscles at alpha equals .025. All statistical analyses were completed using SPSS v15.[d] To maintain study power, we elected to use a familywise error rate of .15 (corrected for the number of analyses) in our post hoc analyses. This may be viewed as an overly cautious approach. Therefore, we provided 95% confidence intervals in our planned comparison data tables to aid in interpretation of these data.

RESULTS

Deltoid Reaction Time

Deltoid reaction times were not different among the 3 groups (F=$2.29_{2,79}$; P=.11; η=.23; 1-β=.45). Mean $\pm$ SD deltoid reaction times were 212$\pm$62, 247$\pm$66, and 225$\pm$67 milliseconds for the control, instability MLBP, and noninstability MLBP groups, respectively.

Patterns of Trunk Muscle Activation

Within the control group (n=39), the mean onset latency of the trunk muscles differed significantly (F=$27.1_{2.5,97}$; P<.01; η=.65; 1-β=1.0). The control group demonstrated a pattern of feedforward activation of the Cont EO, LM, and ES. This represented 64%, 77%, and 56% of the control subjects, respectively. These muscles activated significantly earlier than the other trunk muscles (fig 2A, feedforward identified by the striped boxes; table 3). Onset latencies for the 3 feedforward muscles (Cont EO, LM, ES) were not statistically different.

Within the instability subgroup (n=25), there were no significant differences between group mean muscle latencies (F=$1.75_{4.6,110}$; P=.14; η=.26; 1-β=.56; latency ranged 105–266ms). Their mean trunk muscle onset latencies were predominantly feedback (see fig 2B). The earliest mean activation was in the Cont EO at 105$\pm$191 milliseconds after deltoid onset. In addition, this group demonstrated the most variable response pattern.

Mean onset latency in the noninstability subgroup (n=18) was significantly different between muscles (F=$10.1_{4.4,75}$; P<.01; η=.61; 1-β=1.0). Their mean muscle latency pattern was similar to that of the controls, with feedforward and significantly earlier activation of the LM and ES than other muscles (see fig 2C, feedforward identified by the striped boxes). The LM and ES activated significantly earlier than the Isp IO/TrA and RA. The Isp EO activated significantly earlier than the RA (table 4). Again, those muscles acting in feedforward (LM, ES) did not differ significantly in onset latency. The
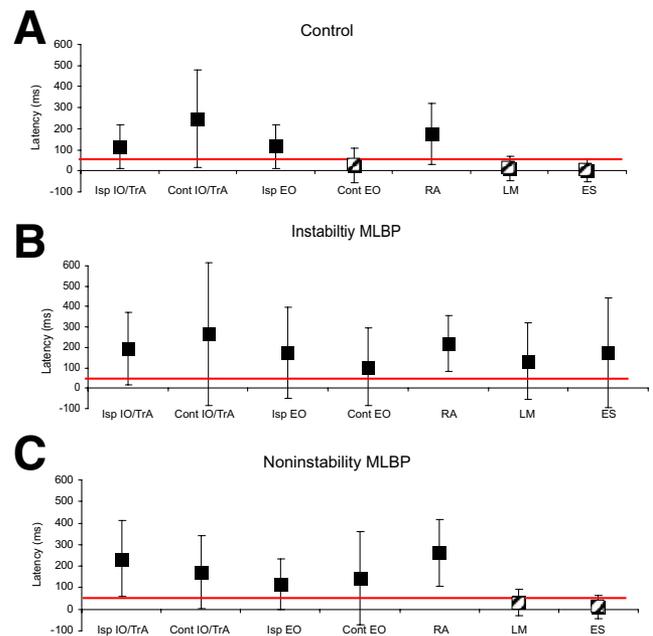

Fig 2. Mean $\pm$ SD onset of each of trunk muscle relative to the anterior deltoid onset (0ms) for (A) control, (B) MLBP instability, and (C) MLBP noninstability groups. The horizontal line represents the end (50ms) of the feedforward period (−100ms to 50ms relative to anterior deltoid onset). Striped boxes represent muscles groups acting in a feedforward manner. Note the differences between the patterns and onset latency variability between groups.

feedforward activation of the extensor muscles in this subgroup is consistent with previous reports.[13,27]

Group Differences

Onset latency of trunk muscles differed significantly between the control and MLBP subgroups (F=$3.61_{14,148}$; P<.01; η=.50; 1-β=.99) (fig 3). The post hoc planned comparison between the control (n=39) and heterogeneous MLBP group (n=43) demonstrated significantly earlier activation of the Isp IO/TrA (P=.003), Cont EO (P=.006), LM (P=.008), and ES (P=.011) in the control group.

In post hoc comparisons between the 2 MLBP subgroups, the instability subgroup demonstrated a significantly later onset of the LM (P=.005) and ES (P=.001) than the noninstability subgroup. There were no significant differences in abdominal muscle activation patterns between the MLBP subgroups. See table 5 for group mean $\pm$ SD and individual planned comparison results with 95% confidence intervals.

The number of muscles functioning in a feedforward manner also differed significantly between the control and MLBP subgroups (F=$2.51_{6,156}$; P=.02; η=.30; 1-β=.83). Post hoc planned comparisons revealed the total number of feedforward muscles was significantly greater for the control (4.3$\pm$2.2) than the heterogeneous MLBP group (3.2$\pm$2.3; P=.025) (table 6). In comparisons between the 2 MLBP subgroups, only the number of feedforward extensors was significantly less (P=.017) in the instability subgroup (1.3$\pm$1.2; noninstability, 2.1$\pm$0.8).

DISCUSSION

Pattern of Trunk Muscle Activation

Asymptomatic control. The group mean trunk muscle response pattern demonstrated by our control subjects confirmed

Table 3: Results of Control Group Post Hoc Pairwise Comparisons for Muscle Onset Latency Pattern

		Mean Difference*	$P^{\dagger}$	95% CI for Mean Difference[‡]	
				Upper Bound	Lower Bound
Isp IO/TrA	Cont IO/TrA	−133 (36)	0.014	−250	−16
	Isp EO	−1 (16)	1.000	−53	52
	Cont EO	88 (17)	0.000	33	143
	RA	−59 (23)	0.345	−134	17
	LM	103 (17)	0.000	48	158
	ES	112 (18)	0.000	54	170
Cont IO/TrA	Isp EO	132 (33)	0.005	26	239
	Cont EO	221 (40)	0.000	92	351
	RA	75 (41)	1.000	−60	209
	LM	236 (34)	0.000	124	348
	ES	245 (35)	0.000	131	359
Isp EO	Cont EO	89 (18)	0.000	31	147
	RA	−58 (17)	0.041	−114	−1
	LM	103 (17)	0.000	48	158
	ES	113 (16)	0.000	62	163
Cont EO	RA	−147 (23)	0.000	−222	−72
	LM	14 (16)	1.000	−39	67
	ES	24 (15)	1.000	−26	73
RA	LM	161 (23)	0.000	85	237
	ES	170 (22)	0.000	100	241
LM	ES	9 (8)	1.000	−17	36

Abbreviation: CI, confidence interval.
*Muscle onset latency mean difference (SE) in milliseconds.
[†]P value; Familywise error $\alpha=.15$ with per comparison error adjusted to $\alpha=.007$.
[‡]95% CIs from mean difference at $P=.05$.

the general assertion that the central nervous system uses a parallel motor command to activate several trunk muscles in a feedforward manner during a self-initiated postural challenge. However, our findings do not exactly mirror those of the previously reported "normal" neuromotor response, and given the redundancy of the trunk muscle system, this is not a surprise finding.[13,15] Using a unilateral upper extremity flexion perturbation, we found that in our control group, the Cont

Table 4: Results of MLBP Noninstability Subgroup Post Hoc Pairwise Comparisons for Muscle Onset Latency Pattern

		Mean Difference*	$P^{\dagger}$	95% CI for Mean Difference[‡]	
				Upper Bound	Lower Bound
Isp IO/TrA	Cont IO/TrA	63 (51)	1.000	−120	245
	Isp EO	120 (36)	0.090	−10	249
	Cont EO	91 (66)	1.000	−144	327
	RA	−27 (46)	1.000	−192	138
	LM	205 (44)	0.004	50	361
	ES	226 (43)	0.001	74	378
Cont IO/TrA	Isp EO	57 (31)	1.000	−52	166
	Cont EO	29 (56)	1.000	−172	229
	RA	−90 (42)	1.000	−241	61
	LM	142 (37)	0.024	12	273
	ES	163 (41)	0.019	18	308
Isp EO	Cont EO	−28 (47)	1.000	−195	138
	RA	−147 (32)	0.005	−260	−34
	LM	86 (28)	0.166	−16	187
	ES	106 (25)	0.010	18	194
Cont EO	RA	−118 (40)	0.194	−262	25
	LM	114 (56)	1.000	−84	312
	ES	135 (51)	0.359	−47	316
RA	LM	233 (39)	0.000	93	372
	ES	253 (36)	0.000	125	381
LM	ES	20 (16)	1.000	−38	79

Abbreviation: CI, confidence interval.
*Muscle onset latency mean difference (SE) in milliseconds.
[†]P value. Familywise error $\alpha=.15$ with per comparison error adjusted to $\alpha=.007$.
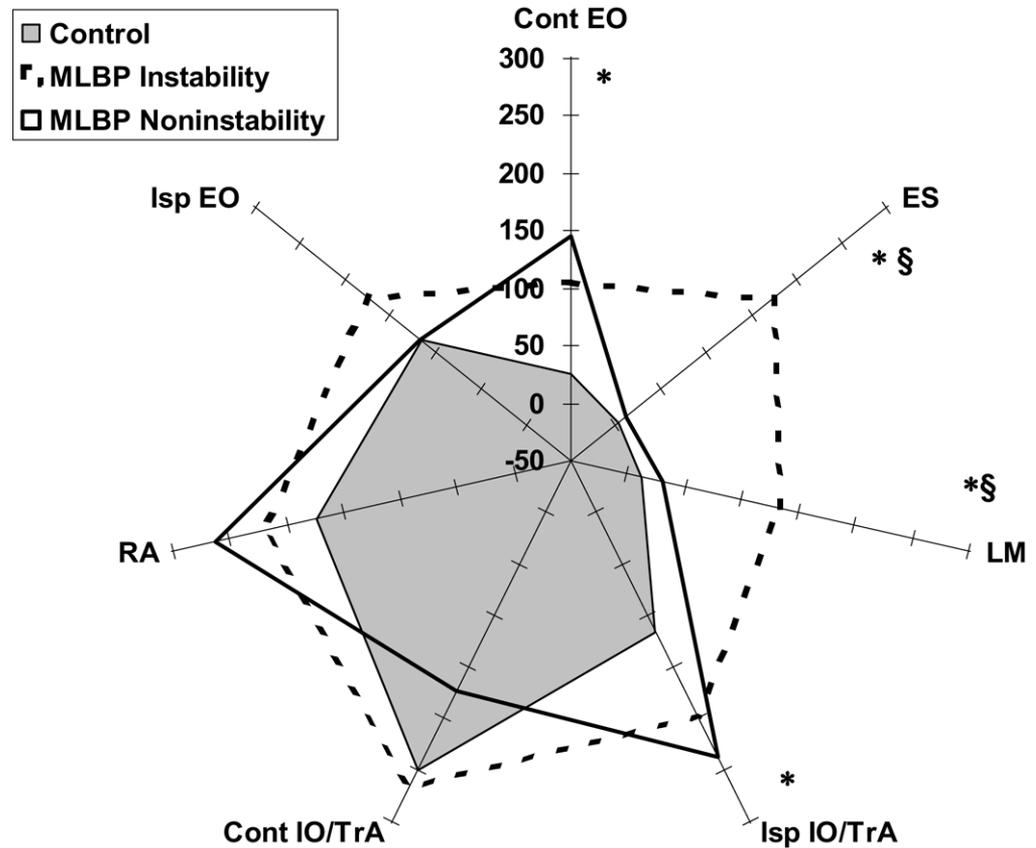[‡]95% CIs from mean difference at $P=.05$.

Fig 3. Group pattern of trunk muscle onset latency (mean) relative to deltoid onset (0ms). Plot demonstrates the latency of the MLBP groups relative to the control group (shaded region). *Significant difference (P≤.007) between control and combined MLBP groups. §Significant difference between the instability and noninstability MLBP subgroups.

EO, LM, and ES muscles responded predominantly in an anticipatory manner and were activated significantly earlier than the remaining trunk muscles. While the preparatory activation of the extensors (LM, ES) agrees with most of the previous literature, feedforward activation of the Cont EO has not been reported during a unilateral upper extremity flexion perturbation.[16,47]

Preparatory trunk motions in extension, contralateral bending, and contralateral rotation have been reported during a unilateral self-initiated flexion perturbation of the upper extremity, along with the presence of ES, IO, and TrA muscle feedforward activation.[17,47,67] Thus, it appears that the Cont EO, LM, and ES feedforward motor program in our control subjects was intended to maintain trunk alignment and mini-

mize displacement of the COM in all 3 planes simultaneously. However, in order to confirm this association, assessment of trunk kinematics would be required.

Most of the subjects in our control group activated neither the Isp (79%) nor Cont IO/TrA (67%) in a feedforward manner. While several authors have reported inconsistent demonstration of TrA feedforward onset,[60,68] others have reported Cont TrA feedforward onset in their asymptomatic subjects.[26,69] The inconsistency in findings could be accounted for by methodologic differences. Previous studies using this protocol recorded a greater number of perturbation trials to determine mean onset, and our use of 3 trials may not have captured the subjects' true activation pattern. In addition, earlier investigators used indwelling fine-wire electrodes to monitor the

Table 5: Results of the Planned Comparisons for Group Differences in Trunk Muscle Onset Latency

	Isp IO/TrA	Cont IO/TrA	Isp EO	Cont EO	RA	LM	ES
Control* (n=39)	115±102	248±232	115±103	26±83	173±145	12±58	3±53
Instability MLBP* (n=25)	195±179	266±349	173±223	105±191	219±137	134±188	174±269
Noninstability MLBP* (n=18)	236±177	173±169	116±115	144±217	263±156	30±61	10±53
Control vs MLBP†	P=.003	P=.628	P=.388	P=.006	P=.038	P=.008	P=.011
	−101 (33)	28 (59)	−29 (34)	−98 (34)	−68 (32)	−70 (26)	−89 (34)
	(−165, −36)	(−88, 144)	(−97, 38)	(−167, −29)	(−132, −4)	(−121, −19)	(−158, −21)
Instability vs noninstability MLBP†	P=.371	P=.257	P=.226	P=.414	P=.334	P=.005	P=.001
	−41 (45)	93 (81)	57 (47)	−40 (48)	−44 (45)	103 (36)	164 (48)
	(−131, 49)	(−69, 254)	(−36, 151)	(−136, 57)	(−133, 46)	(33, 174)	(68, 259)

*Data represent group mean ± SD in milliseconds.
†Planned comparisons; P value; mean difference (standard error); 95% confidence interval for mean difference at P =.05. Familywise error α=.15 with per comparison error adjusted to α=.011.

Table 6: Results of Planned Comparisons for Group Differences in the Number of Trunk Muscles Responding in a Feedforward Manner

	Total	Abdominal	Extensor
Control* (n=39)	4.3±2.2	2.2±1.7	2.1±0.8
Instability MLBP* (n=25)	2.9±2.5	1.5±1.7	1.3±1.2
Noninstability MLBP* (n=18)	3.4±2.0	1.3±1.7	2.1±0.8
Control vs MLBP†	P=.025	P=.040	P=.045
	1.15 (.50)	.80 (.38)	.44 (.22)
	(.15, 2.16)	(.04, 1.57)	(.01, .87)
Instability vs noninstability MLBP†	P=.505	P=.706	P=.017
	−.47 (.70)	.20 (.53)	−.74 (.30)
	(−1.86, .92)	(−.86, 1.26)	(−1.33, −.14)

*Data represent group mean ± SD.
†Planned comparisons; P value; mean difference (standard error); 95% confidence interval for mean difference at P=.05. Familywise error α=.15 with per comparison error adjusted to α=.025.

response of the abdominal muscles (TrA, IO, EO).[13,26] While use of surface electrodes does not allow individualized recording from deeper trunk muscles such as the TrA and IO, our surface electrode placement has been validated for combined activity of these muscles.[60] Furthermore, feedforward activation of the IO/TrA during rapid limb movement has been reported in control subjects using this surface EMG placement.[46,60,70] Therefore, we believe our IO/TrA data represent the combined function of these muscles.

The Cont EO, LM, and ES muscle groups activated significantly earlier than the other trunk muscles we monitored. However, not all the subjects within the control group used the same activation strategy, although each member of the group had at least 2 muscles act in a feedforward manner. Therefore, it seems plausible that during this perturbation, more than 1 sequence of trunk muscle activations may serve to stabilize the trunk adequately in persons with no history of LBP or spinal degenerative changes unusual for their age.

MLBP subgroups. Within the MLBP subgroup diagnosed with instability, neither feedforward activation nor individual muscle timing differences were found between mean muscle latencies. This subgroup demonstrated a predominantly reactive strategy with increased variability in the activation latency of the trunk muscles. The lack of significant timing differences between the instability subgroup's muscle latencies suggests a delayed general stiffening of the spine. While a trunk muscle coactivation response was predicted by Panjabi[29] and modeled by Cholewicki et al,[59] it may have unintended consequences when the strategy is delayed or used over the long term. This pattern may also result in the central nervous system perceiving a diminished demand for fine-tuning the response.[2] The lack of a feedforward postural response may result in an ineffective or suboptimal control of the forces associated with this postural perturbation, resulting in an inability to stabilize the spine adequately in a timely manner, thereby increasing the risk of further trauma to the spinal structures.

The noninstability MLBP subgroup demonstrated an activation timing pattern more like that of the control group, although with greater group variability. As a group, the noninstability subgroup activated the trunk extensors in a feedforward manner and demonstrated a more selective firing pattern of trunk muscles. The primary pattern difference between this subgroup

and our control group was fewer subjects activating the Cont EO in a feedforward manner. This subgroup's postural response to their unilateral arm movement is more consistent with that reported in the literature for patients with a history of chronic MLBP.[13,26] The altered trunk motor program and increased pattern variability in the noninstability MLBP subgroup suggest they are more capable of actively seeking an appropriate response strategy.

Group Differences

Control versus heterogeneous MLBP group. The take-home message from previously published postural control studies indicating that subjects with chronic LBP demonstrate delayed onset of trunk muscles compared with healthy controls is supported by our study.[7,26,71] The differences in activation timing patterns found in our study provide further evidence that patients with chronic MLBP use an altered, and presumably inadequate, neuromuscular control strategy for dynamic stabilization of the spine during a self-initiated postural challenge. Consistent with other studies, the deltoid reaction time was not significantly different between groups for fast upper extremity perturbation and was within the range reported in previous studies using a similar protocol.[13,27]

Results from the comparison between our heterogeneous MLBP group (combined subgroups) and asymptomatic control subjects partially agree with previous reports in the LBP literature. We also found significantly delayed onset of the IO/TrA (albeit the ipsilateral muscle group) and the Cont EO in our chronic MLBP group.[26] However, we did not find feedforward onset of the LM.[26] In fact, our heterogeneous MLBP group demonstrated significantly delayed trunk extensor muscle activation compared with the asymptomatic control group.

There are several plausible explanations for inconsistency in findings between this and previous studies. First, it is difficult to compare the results of our study directly to others because of methodology differences previously mentioned and differences in sample subjects. Our subjects were older (average difference=11y). Older persons are reported to demonstrate increased trunk muscle latency.[72] Unlike previous studies using a unilateral perturbation paradigm,[13,26,27,67] our subjects with chronic MLBP had pain and significant activity limitations at the time of testing. While use of subjects with current pain makes the determination of the underlying mechanism more difficult, the use of subjects with a history of LBP who are not having pain or having minimal symptoms at the time of testing may not epitomize the true clinical situation. While our subjects with MLBP underwent a course of physical therapy, the specifics of the individual interventions were not controlled as part of this study. Therefore, it may be that our patients with chronic MLBP as a group were unable to adequately compensate for their neuromuscular impairment (noncopers), thus reducing system performance. Overall, we believe that these data represent further evidence of an adapted generalized motor plan, the causes of which are hypothesized to be pain, injury, altered proprioception, and/or fear-avoidance.[2,73,74]

MLBP instability subgroup. To our knowledge, investigation of differences in trunk muscle latency responses to self-perturbation between subgroups of patients with MLBP has not been reported. Our findings suggest that not all subgroups of patients with chronic MLBP demonstrate the same postural response pattern, particularly related to the activation of the trunk extensors. The trunk extensors of the MLBP group with instability demonstrate both delayed onset and limited feedforward activation in comparison with the noninstability group. The reason for the differences in these subgroups is not readily explained from the perspective of clinical signs and symptoms

because their numeric pain rating scale, lumbar flexion range of motion, extensor strength, and movement patterns did not differ significantly from those of the noninstability subgroup. While the instability subgroup did have greater self-perceived disability on the RMQ (11.6) than the noninstability subgroup (8.1), the clinical significance of this is debatable given that the instrument's measurement error and minimally important clinical difference scores are 3 to 5 points.[75] We cannot rule out that the course of conservative care, not controlled as part of the study, may have influenced the patients' responses to perturbation and contributed to subgroup differences and large subgroup variability.

From a pathology or impairment perspective, it could be argued that these subgroups differ in the severity or amount of spinal tissue degeneration or injury. Although we did not directly measure intersegmental motion, our instability subgroup presents with significant change in a major stabilizing structure of the spine that has been associated with increased segmental mobility.[41,48] Over time, these impairments could have resulted in an altered generalized postural motor program specifically involving the trunk extensor response. Because the testing paradigm uses the neutral trunk position, which provides minimal passive restraint,[76,77] the stability of the spine in this situation is primarily dependent on muscle timing during sudden perturbation. Inability to stiffen the spine efficiently or effectively during a postural challenge may in turn allow continued trauma to spinal structures, resulting in sustained or repeated episodes of MLBP. Evidence suggests that even small rotations and translations can produce tissue injury.[78-80] The possibility of continued tissue injury from an inability to stabilize dynamically is further supported by recent findings of less preparatory trunk extension motion (presumably related to delayed extensor onset) and greater resultant lumbar flexion motion in subjects with recurrent MLBP after upper extremity flexion perturbation.[67]

MLBP noninstability subgroup. Although the overall pattern in the noninstability subgroup approaches that of the controls, this group still had pain and dysfunction. Possibly these subjects had not, as a group, maintained or fully reestablished appropriate abdominal muscle timing, and the delayed activation of the Cont EO resulted in insufficient stability or trunk control, particularly in the frontal and transverse planes. Consistent with the explanation for continued pain and dysfunction offered, the lack of this feedforward control could expose spinal structures to continued microtrauma. Another explanation is that the primary impairment resulting in their continued LBP is not that of impaired feedforward activation of trunk muscles, but is related to trunk muscle weakness, restricted motion, or fear avoidance.

In addition to those already discussed, our results should be interpreted in light of the following limitations. First, we did not directly measure arm velocity, lower extremity weight distribution, or postural alignment during data collection. These factors are reported to alter the trunk muscle responses significantly and may account for the differences in our findings, even in the activation pattern demonstrated by our control group.[26,81] However, we closely observed these variables and rerecorded trials that did not meet our performance criteria. Second, our use of only 3 trials may not have allowed us to obtain adequately the predominant postural response pattern to the self-perturbation. This may account for the increased variability in performance that is reflected in our within-session reliability findings. Further testing (test-retest, 7 days apart, healthy adults) in our laboratory using 6 repetitions resulted in improvements in these reliability estimations (range=.91–.61; $ICC_{2,6}$). Marshall and Murphy[60] have reported their labora-

tory's test-retest reliability of onset latency ranged from .90 to .33 ($ICC_{1,1}$) for surface EMG over abdominal muscles (IO/TrA, EO, RA) in young healthy adults. To date, the reliability of this measurement has not been widely reported or discussed in the literature. Generally a large number of practice trials and a mean of 10 repetitions are used, likely increasing measurement stability and perhaps more accurately representing the onset pattern. This issue should be pursued more thoroughly. Third, the medical imaging and testing used to subclassify our patients with MLBP should be kept in mind when interpreting the results. For this reason, we compared all of our subjects with MLBP to our control group to determine consistency of our results to previous studies where the patients with MLBP were defined primarily by verbal report of history of LBP. In addition, we have provided the results of our physical examination to assist with interpretation of our findings relative to other studies or to a specific patient population. Finally, the design of this study does not allow determination of which mechanisms might be responsible for the altered recruitment patterns nor whether the changes in trunk neuromuscular control were a cause or result of their LBP. Future studies designed to address the mechanism question in subgroups of patients with MLBP would be extremely helpful for customizing treatments to the patient's primary impairments. Exploration of other aspects of feedforward postural control, muscle response amplitude, and/or onset duration may further improve understanding of these motor program changes. Despite these limitations, we believe the study's design and statistical power adequately support our hypotheses.

CONCLUSIONS

Overall, our findings add to the evidence for an altered generalized trunk motor program in patients with chronic MLBP. In addition, we are the first to investigate and report differences in subgroups of patients with MLBP. The medical imaging findings and clinical signs and symptoms demonstrated by our patient subgroups further substantiate the characteristics of patients with MLBP who demonstrate impairments in the trunk feedforward motor program and support the notion that subgroups exist within the MLBP population. These data also introduce the notion of timing impairments in other trunk muscles (Cont EO, LM, ES) and suggest that the number of muscles functioning in feedforward may also be a marker of suboptimal trunk control. These unresolved impairments may indicate an inability of some patients with MLBP to adapt adequately (noncopers), which might ultimately contribute to their development of a recurrent and chronic condition. Better understanding of the impairments associated with subgroups of patients with MLBP is expected to improve our trunk neuromuscular training programs for individual patients, resulting in a reduction of severity or frequency of recurrent episodes of MLBP.

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