ORIGINAL ARTICLE

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

Sheri P. Silfies, PT, PhD, Rupal Mehta, PT, MS, Sue S. Smith, PT, PhD, Andrew R. Karduna, PhD

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;90:1159-69.

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 ($P<.01$; $\eta=.50$; $1\text{-}\beta=.99$) and number of muscles functioning in feedforward ($P=.02$; $\eta=.30$; $1\text{-}\beta=.83$) 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.

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

From the Rehabilitation Sciences Research Laboratory, Drexel University, Philadelphia, PA (Silfies, Mehta, Smith); Department of Human Physiology, University of Oregon, Eugene, OR (Karduna).

Presented in part to the American Physical Therapy Association, February 14, 2007, Boston, MA.

Supported in part by grants from Orthopaedic Section of the American Physical Therapy Association and the US Department of Education, National Institute on Disability and Rehabilitation Research (H133F030024).

No commercial party having a direct financial interest in the results of the research supporting this article has or will confer a benefit on the authors or on any organization with which the authors are associated.

Device Status Statement: The devices are FDA approved for the indicated usage in the United States.

Reprint requests to Sheri P. Silfies, PT, PhD, Rehabilitation Sciences Research Laboratory, Drexel University, 245 N. 15th Street, MS 502, Philadelphia, PA 19102-1192, e-mail: *silfies@drexel.edu*.

Published online June 5, 2009 at *www.archives-pmr.org*.

0003-9993/09/9007-00543$36.00/0
doi:10.1016/j.apmr.2008.10.033

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,