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Trunk Muscle Reflex Amplitudes Increased in Patients With Subacute, Recurrent LBP Treated With a 10-Week Stabilization Exercise Program

Anand Navalgund [Senior Scientist],

Research and Development, G-Tech, Palo Alto, CA.

John A. Buford [Associate Professor],

Physical Therapy Division, College of Medicine, The Ohio State University, Columbus, OH.

Mathew S. Briggs [PhD student], and

Health and Rehabilitation Sciences, School of Allied Medical Professions, The Ohio State University, Columbus, OH.

Deborah L. Givens [Professor]

Department of Physical Therapy, School of Pharmacy and Health Professions, Creighton University, Omaha, NE.

Abstract

Altered trunk muscle reflexes have been observed in patients with low back pain (LBP). Altered reflexes may contribute to impaired postural control, and possibly recurrence of LBP. Specific stabilization exercise (SSE) programs have been shown to decrease the risk of LBP recurrence in a select group of patients with acute, first episode LBP. It is not known if trunk muscle reflex responses improve with resolution of subacute, recurrent LBP when treated with a SSE program. A perturbation test was used to compare trunk muscle reflexes in patients with subacute, recurrent LBP, before and after 10 weeks of a SSE program and a group of matched control subjects (CNTL). The LBP group pre therapy had delayed trunk muscle reflexes compared with the CNTL group. Post therapy reflex latencies remained delayed, but amplitudes increased. Increased reflex amplitudes could limit excessive movement of the spine when perturbed; potentially helping prevent recurrence.

Keywords

stabilization exercise; low back pain; sudden loading; reflex amplitude; multifidus

Low back pain (LBP) affects 60–80% of individuals during their lifetime and most are managed with conservative treatments by their primary care provider (Patel and Ogle, 2000). However, a majority of people go on to experience persistent and/or recurrent LBP, and up to one-third report moderate to severe pain one year later (Croft, Macfarlane, Papageorgiou, Thomas, and Silman, 1998; Dunn, Jordan, and Croft, 2006; Von Korff and Saunders, 1996). These issues have motivated interest in identifying unresolved impairments and effective interventions for preventing low back injury or reinjury.

Motor control deficits are among the impairments in body structure and function observed following low back injury. People with LBP respond differently to perturbations induced by sudden changes in external loading conditions affecting the torso compared with people without a history of LBP. Studies have demonstrated that LBP is associated with delayed trunk muscle responses to perturbations (Hodges, 2001; Radebold, Cholewicki, Panjabi, and

Patel, 2000; Wilder et al., 1996), delayed muscle shut-off times after an external load has been removed (Radebold, Cholewicki, Polzhofer, and Greene, 2001), and increased coactivation during complex tasks (Granata and Orishimo, 2001). An increase in the displacement of the trunk in response to sudden loads resulting in excessive motion in the spinal segments may strain or deform peri-articular tissues (Brown and McGill, 2009; Solomonow, Zhou, Baratta, Lu, and Harris, 1999). Hence, it is proposed that persistent impairments in the trunk muscle responses to sudden perturbations may predispose individuals to reinjury (Cholewicki et al., 2005).

There are three components in the motor response that may be observed following a rapid perturbation applied to the trunk; a short latency reflex (M1), a medium latency reflex (M2), and a long latency component (M3; Wilder et al., 1996). M1 is dominated by the monosynaptic stretch reflex, initiated by the muscle spindle afferents (Hammond, 1955). M1 onset times as fast as 12.0 (SD 2.9) ms have been elicited by tapping directly on the erector spinae of healthy subjects (Hjortskov, Essendrop, Skotte, and Fallentin, 2005). M2 is thought to be preprogrammed and mediated by several circuits which include the brain stem (Grey, Ladouceur, Andersen, Nielsen, and Sinkjaer, 2001), cerebellum (Nashner, 1976), and primary motor cortex (Marsden, Merton, and Morton, 1976). There is also evidence that the M2 reflex may depend heavily on signals originating in mechanoreceptors of the interspinous ligaments (Solomonow, Zhou, Harris, Lu, and Baratta, 1998). The M2 response requires more neural processing and longer pathways than the M1; hence, longer latencies are observed. M2 onset times as fast as 44.6 (SD 2.5) ms have been recorded from the erector spinae in healthy controls (Zedka, Prochazka, Knight, Gillard, and Gauthier, 1999). M3 has onset times in the ranges of 120–180 ms and is considered a voluntary response initiated by cortical motor areas (Petersen, Christensen, Morita, Sinkjar, and Nielsen, 1998).

Investigations of trunk muscles using sudden unexpected perturbations applied to the trunk (Cholewicki et al., 2005; Radebold et al., 2001; Granata, Slota, and Bennett, 2004; Rogers and Granata, 2006) have reported onset times of 60–120 ms for trunk muscle reflex responses, which correspond to M2 response times. Radebold et al. (2001) reported average onset times of 85 (SD 25) ms in the back muscles in patients with LBP in reaction to a sudden load as compared with an average onset time of 69 (SD 8) ms in the control subjects. In a 2 year prospective study, the authors (Cholewicki et al., 2005) demonstrated in 292 college athletes that the athletes who went on to sustain a low back injury (LBI) had, on average, a 14 ms longer muscle shut-off time after an external load was removed as compared with athletes who did not sustain an LBI. There were no significant changes in the muscle shut-off times before and after the LBI in the athletes. The authors concluded that delays in reflex latencies were a significant predictor of and a preexisting condition for LBP.

Pain (Ervilha, Farina, Arendt-Nielsen, and Graven-Nielsen, 2005), damage to the nerves / receptors (Solomonow et al., 1998), muscle wasting leading to loss of type II fibers (Hides, Stokes, Saide, Jull, and Cooper, 1994), fat infiltration (Kjaer, Bendix, Sorensen, Korsholm, and Leboeuf-Yde, 2007), and reduced endurance in the muscles (Holmstrom, Moritz, and Andersson, 1992) may all contribute to the greater variability seen in the reflex response in patients with LBP compared with healthy subjects (Danneels et al., 2002). These altered muscle response patterns are thought to contribute to the impaired postural control and poor balance also observed in persons with LBP (Luoto et al., 1996; Madigan, Davidson, and Nussbaum, 2006; Radebold et al., 2001). Moreover, a persistent imbalance in muscle response patterns may alter the motor control and be a potential reason for recurrent LBP (MacDonald, Moseley, and Hodges, 2009).

Is it possible to modify these trunk muscle responses to sudden loads to make them quicker and stronger? In theory, this may reduce the risk of LBI or reinjury. In the specific

segmental exercise (SSE) approach of Richardson and colleagues (Richardson, Hodges, and Hides, 2004), patients with LBP practice conscious, preactivation of selected trunk muscles before movement to enhance stability of the spine. The motivation for this approach was based on evidence that LBP leads to altered feedforward control of the transverses abdominis (Hodges, 2001) and focal atrophy of the ipsilateral multifidus near the involved spinal segment (Hides et al., 1994). The thought was to target these same muscles for exercise intervention, and by treating the impairment, to improve outcomes. Promising reductions in pain and increases in function have been demonstrated with this approach (O'Sullivan, Phytty, Twomey, and Allison 1997). These changes have been associated with better symmetry in the multifidus muscles between sides (Hides, Richardson, and Jull, 1996). However, there is little evidence to document the changes, if any, in the neuromotor control of these muscles after the SSE program. We hypothesized that in addition to effects on muscle bulk, the SSE program would also lead to improved neuromotor control. As one means to study motor control, we used the perturbation protocol to study trunk muscle response patterns to sudden unexpected loads following an intervention that incorporates the SSE approach for patients with LBP. Understanding what happens to muscle response patterns with resolution of LBP after treatment using the SSE approach may provide insight as to how and why this program could be beneficial to patients at risk for LBI or reinjury.

The purpose of this pilot study was twofold. The first was to quantify differences in the reflexes of the back muscles between the subjects with subacute, recurrent LBP and healthy controls (CNTL). The second was to examine the changes in reflexes associated with a 10-week physical therapy (PT) program incorporating the SSE approach. Based on the expectation that muscle bulk and recruitment would both be improved, and the preexisting presence of longer-than-normal latencies in the LBP patients, our expectation was that the LBP group before therapy (PRE) would demonstrate delayed reflexes and reduced amplitude as compared with the CNTL group; and a resolution of LBP accompanied by a PT intervention aimed at altering the neuromuscular control in the LBP group would lead to quicker reflexes and increase in the reflex amplitudes after therapy (POST).

Methods

Subjects

13 subjects (6 female) with a primary complaint of subacute, recurrent LBP, and 13 subjects (6 female) with no history of significant LBP in the past 2 years were recruited for the study. Subjects with LBP were included if the duration of pain in the current episode was less than or equal to 8 weeks, and had experienced at least 1 separate episode in the past year that had resolved. Subjects were excluded if they had a prior history of lumbar surgery and presence of sciatica or medical conditions that might affect spinal control such as cauda equina syndrome, neurological disorders, fracture, cancer, infection, or systemic disease. The subjects in the control group were matched to the subjects in the group with LBP by their age, gender, height and weight. Informed consent was obtained from each subject following procedures approved by the Biomedical Institutional Review Board at the University.

Experiment Protocol

All subjects provided demographic information and underwent a baseline screening examination by one of the investigators in the laboratory before testing. Subjects with LBP completed additional questionnaires at both the pre therapy and post therapy data collection session to characterize their current pain and function. An 11 point numeric pain rating scale (NPRS) with the range of 0 (no pain) to 10 (worst imaginable pain) was used to measure pain by rating the current pain intensity and the best and worst pain intensity during the previous 24 hr (Jensen, Turner, and Romano, 1994). NPRS half scores were rounded up and

the 3 ratings were averaged to represent the 24-hr intensity of the pain. When used in this way, the NPRS has shown good sensitivity and reliability (Childs, Piva, and Fritz, 2005). The Modified Oswestry Disability Index (ODI), used to characterize self-reported function, has good reliability with test-retest intraclass correlation coefficients (ICCs) between 0.83 and 0.94 (Fritz and Irrgang, 2001; Kopec et al., 1995).

Trunk muscle activity was recorded bilaterally with surface electromyography (EMG) using the MA3000 system and MA-411 EMG preamplifiers from Motion Laboratories (Baton Rouge, LA). A bipolar recording was obtained using the MA-411 EMG preamplifier with two 12 mm disks of medical grade stainless steel at an interelectrode distance of 17mm. Before recording, the skin at the location was cleaned with alcohol and hair was shaved if necessary. The surface electrodes were taped on the site without any gel. Muscle activity was recorded from the L3 erector spinae (ES S) at 3 cm from the midline at the L3 spinous process (Ng and Richardson, 1994) and for the L5 multifidus (Mult S) at 3 cm from midline at the L5 spinous process (Bogduk, Macintosh, and Pearcy, 1992). Previous experiments have observed differences in fine wire and surface EMG recordings from the multifidus (Moseley, Hodges, and Gandevia, 2002; Stokes, Henry, and Single, 2003). Moseley, et al. (2002) suggested that the deep and superficial fibers of the multifidus are differentially activated, with the deep fibers contributing to intersegmental motion and the superficial for spine orientation. In this study, along with the surface EMG recording the L5 multifidus activity, fine wire EMG recorded activity bilaterally from the deep fibers of the L5 multifidus (Mult FW) using two sets of paired fine wire electrodes ($0.002 \times 8''$ Nickel alloy insulated wires, Chalgrin Inc, CA). The fine wire electrodes had 6 mm bare-wire terminations, which were inserted using a 30 mm, 27 gauge hypodermic needle and placed in the muscle adjacent to the lamina of the L5 vertebrae. The appropriate depth for insertion was based on measurements made using ultrasonography (Titan, Sonosite, Bothell, WA). The paired wires were connected to the MA-416 preamplifiers to obtain a bipolar recording.

For normalization of the EMG signals from the multifidus and erector spinae, subjects performed the Biering-Sorensen test while EMG was recorded (Biering-Sorensen, 1984). For the test, subjects were prone with the legs and hips secured by straps to the test bed and the upper body extended over the edge of the table. The endurance test was terminated when subjects could no longer hold the torso in a horizontal position or at the 3 min endpoint of the test. A separate Biering-Sorensen test was conducted for the post therapy data collection for the subjects with LBP to accommodate any changes in the EMG due to the training program. For the reflex perturbation test, the subject was seated in a kneel chair (Figure 1) with a belt across the lower torso to restrict pelvic motion. A metal bar was placed across the thorax at the inferior angles of the scapulae (T6-T7). A harness from the bar was attached to a cable from the servomotor (Pacific Scientific, Rockford, IL). Tension in the cable provided a constant isotonic flexion preload of 100 N. Pseudorandom force perturbations of ± 30 N were superimposed on the 100 N preload. A single trial, as indicated in Figure 2, consisted of 3 s where the load was ramped up to 100 N followed by 3 s of flexion preload at 100 N and subsequently 10 s of perturbations. For the 10 s of perturbations, a random number generator produced numbers between 0 and 1 every 0.2 s (Gollhofer and Rapp, 1993; Stein and Kearney, 1995). If the number was greater than 0.5 then a perturbation of +30N was allowed/sustained. If it was less than 0.5 then a -30 N perturbation was allowed/sustained. On average during a single trial the subject experienced 12 such perturbations. Six such trials were performed for each subject and in all on average around 72 individual perturbations. The loads were measured by a torque transducer (Omega TQ301 series, 0–45 Nm, Stamford, CT) attached to the shaft of the motor. The LBP group was tested PRE and POST the 10-week SSE program, while the CNTL group was tested on one occasion using the same protocol.

The SSE intervention was modeled using the program by Richardson and colleagues (Richardson and Hides, 2005). Subjects attended 10 visits at the physical therapy clinic, each visit lasting 45 min, which were scheduled twice per week for 2 weeks (weeks 1 and 2), once per week for four weeks (weeks 3–6), and then once every 2 weeks (weeks 8 and 10) for the 10-week SSE program.

The initial phase of the SSE program involved exercising the abdominal and low back muscles with specific exercises and biofeedback via rehabilitation ultrasound imaging for the multifidus and the transverse abdominis (Herbert, Heiss, and Basso, 2008; Hides, Jull, and Richardson, 2001). Subjects first learned abdominal hollowing exercise in supine (Henry and Westervelt, 2005) and isometric recruitment of the multifidus in prone (Herbert, Heiss, and Basso, 2008). Each contraction was held for 10 s and repeated 10 times. Subjects then learned to cocontract these muscle groups and perform the holding contractions while in quadruped, sitting, standing, walking, and daily activities (Richardson and Jull, 1995). Subjects were progressed to more challenging exercises when the physical therapist judged that a previously painful or difficult movement could be performed with good form (cocontraction and breathing) and minimal to no pain.

Subjects performed gentle stretching exercises for the trunk (single plane movements) and lower extremities (hamstrings and hip flexors). Subjects were instructed to avoid extreme stretching movements and that there should be no detrimental change in symptoms during or after the 30 s holds that were performed 3 times. Subjects also performed an aerobic activity such as walking, biking, or swimming. Activities were selected based on patient interest and access to equipment/facilities. Patients were coached regarding use of neutral spine position and good form. Subjects were encouraged to work up to a total of 15 min of continuous activity and educated to work at level that allowed good form and did not result in a detrimental change in symptoms. The subjects were also instructed to carry out a home exercise program for 30 min per day during the 10-week program. The home exercises were similar to those that were practiced in the clinic with the physical therapist. Compliance was monitored using patient self-reported exercise logs. Attendance was required for 70% of the physical therapy sessions and subjects could not miss more than 2 appointments in a row.

Data Analysis

The raw EMG and torque transducer signals (Figure 2) were collected and sampled at 2000 Hz using LabVIEW (National Instruments, Austin, TX) and processed using Datapac (RunTech Inc., Mission Viejo, CA). All the EMG signals including the Biering-Sorenson test were low pass filtered at 100Hz using a fifth order zero lag Butterworth filter and full-wave rectified. A 300 msec window, starting 5 s from the initiation of the Biering-Sorenson test, was chosen for normalization of the EMG data. The mean of the processed data from this 300 msec window was used to normalize the reflex amplitude to provide a standardized comparison between muscles, times, and subjects.

For the reflex trials, a single file was created by concatenating the six trials for each subject. The onset times of the perturbations were detected whenever the force increased 2 standard deviations from the baseline of 100 N. A –100msec to +400msec window was created around each of these perturbation onset times. Each of the corresponding 500msec windows was selected then for the EMG channels. The processed EMG activity during each of these windows were summed and averaged to result in the muscle reflex responses for analysis (Figure 3). As indicated in Figure 3, the beginning and end of the M2 reflex was detected as a change of 2 standard deviations from the baseline activity. The baseline activity was defined as the period of activity between –1.0 s and 0.0 s. The reflex latency was calculated as the time between 0.0 s and the beginning of the M2 reflex. The area under the reflex peak

was calculated as the reflex amplitude. The reflex latencies and amplitudes were averaged over both the left and right muscle sides.

Statistical Analysis

Subject characteristics (age, height, and weight) were compared between groups using an independent t test (Excel 2007, Microsoft, Redmond, WA). The ODI scores were converted to a percent score. The NPRS and OPI scores were compared PRE and POST therapy in the group with LBP using a paired samples t tests.

Mixed effects regression-modeling framework (SAS, v9.1, SAS Institute Inc., Cary, NC) was used to model the reflex latency and amplitude computed for each muscle. A random intercept for each subject was included to separate within-subjects and between-subjects variability. Empirical —sandwich— estimators of the variance-covariance matrix for the fixed effects parameter of group (PRE versus CNTL and PRE versus POST) were used to alleviate the impact of any departures from the defined covariance structure. Data are presented as means and standard deviations. For all statistical tests, a $p \leq 0.05$ was considered significant.

Results

Subjects

The PRE analysis included thirteen subjects for the LBP group and thirteen matched CNTL subjects, who were similar in age, height and weight (Table 1). Nine subjects were included for the LBP PRE therapy versus POST therapy analysis. Of the thirteen subjects in the LBP group, two attended only the first PT session and one attended all PT sessions but could not be contacted for the POST test. Reflexes were not elicited in one of the remaining subjects with LBP, so that subject's data were not included in the final comparison. This phenomenon of absent reflexes in some individuals has also been observed in other studies (Cholewicki et al., 2005; Granata, Rogers, and Moorhouse, 2005), but the clinical significance of this is unknown. Pain was reduced ($p = .0039$) from an average PRE pain rating of 3.6 (SD 1.30) to 1.7 (SD 0.92) POST therapy. A 2.0 point change is considered clinically important, so the average change of 1.9 almost met this criterion (Childs, Piva, and Fritz, 2005). The subjects with LBP reported improved function ($p = .0014$) with a PRE average ODI score of 26.5% (SD 10%) and a POST score of 13.0% (SD 11%) for a change in function of 13.5%, which exceeded the minimal clinically important difference of 12% (Fritz and Irrgang, 2001).

LBP Group at PRE Therapy vs. CNTL Group

The averaged reflex responses for a representative control subject and subject with LBP at PRE therapy are shown in Figure 3. Responses with latencies above 120 ms were considered M3 or voluntary responses. Responses below 100 ms were considered reflex responses.

The average latencies for the LBP group at PRE therapy were in the range of an M2 response. These reflex latencies were delayed in the LBP group at PRE therapy for all the three measurements by 7.1 ms in Mult FW ($p = .014$), 5.4 ms in Mult S (0.016) and 6.7 ms in ES S ($p = .001$) compared with the CNTL group (Figure 4A). The mean reflex latencies were 44 ms (SD 9.5 ms), 42.7 ms (SD 3.0 ms) and 43.1 ms (SD 4.7 ms) for the LBP group at PRE therapy and 36.9 ms (SD 6.8 ms), 37.3 ms (SD 5.1 ms) and 36.4 ms (SD 5.2 ms) for the CNTL group for Mult Fw, Mult S and ES S respectively.

The mean reflex amplitudes were lower by 3 units for Mult FW ($p = .030$) for the LBP group therapy compared with the CNTL group but not for Mult S ($p = .071$) or ES S (0.098). The mean reflex amplitudes were 6.1 (SD 4.1), 7.1 (SD 3.9), and 5.3 (SD 3.8) for the LBP

group at PRE therapy and 9.1 (SD 5.1), 9.7 (SD 4.1) and 8.6 (SD 4.4) for the CNTL group for Mult FW, Mult S and ES S respectively.

LBP Group at PRE Therapy VS. LBP Group at POST therapy

There was no change in the reflex latencies for Mult FW ($p = .915$), Mult S ($p = .561$) and ES S ($p = .549$), between the two sessions at PRE and POST therapy (Figure 4B). The latencies remained in the range of an M2 response. The mean reflex latencies were 41 ms (SD 7.4 ms), 42.6 ms (SD 3.4 ms) and 42.7 ms (SD 5.3 ms) for the LBP group at PRE therapy and 41.4 ms (SD 3.4 ms), 42 ms (SD 4.4 ms) and 41.4 ms (SD 5.1 ms) for the LBP group at POST therapy for Mult Fw, Mult S and ES S respectively.

A significant increase was observed in the reflex amplitudes by 5.4 in Mult FW ($p < 0.0001$), 3.2 in Mult S ($p = .0056$) and 4.3 in ES S ($p = .0018$) at the POST therapy session for the LBP group compared with the PRE therapy session. The mean reflex amplitudes were 3.8 (SD 4.6), 6.5 (SD 4.8), and 4.1 (SD 3.1) for the LBP group at PRE therapy and 9.2 (SD 4.3), 9.7 (SD 6.8) and 8.4 (SD 3.6) for the LBP group at POST therapy for Mult FW, Mult S and ES S respectively.

Discussion

The findings of this study show that the subjects with LBP exhibited delayed latencies in the M2 reflex in the Mult S, Mult FW and ES S as compared with the healthy controls. These results follow similar trends observed in previous studies. In addition, the reflex amplitudes were lower for the Mult FW in the LBP subjects. The subjects with LBP after completing the SSE program and resolution of pain demonstrated strengthened M2 reflexes with increased reflex amplitudes but no changes in the reflex latencies.

The amplitude of the M2 response is known to be modifiable in response to immediate pretask instructions (Hammond, 1956; Marsden, Rothwell, and Day, 1983; Matthews, 1991; Rothwell, Traub, and Marsden, 1980; Strick, 1983; Strick, 1978). The locus of this modified response may include the cerebral cortex (Cheney and Fetz, 1984; Evarts and Tanji, 1974; MacKinnon, Verrier, and Tatton, 2000; Shemmell, An, and Perreault, 2009) as well as subcortical systems in the brainstem, which may be influenced by cortical and cerebellar activity in preparation for the movement (Jacobs and Horak, 2007; Shemmell, Krutky, and Perreault, 2010; Strick, 1978). Segal, Wolf, and colleagues have demonstrated that the M2 reflexes in the arm are also affected by training (Wolf, Segal, Heter, and Catlin, 1995), and subjects can learn to modulate the M2 independently of the H-reflex (M1) component (Segal et al., 2000). There is also evidence of changes in the M2 component of the stretch reflex in the arm after a long term training paradigm in monkeys (Christakos, Wolf, and Meyer-Lohmann, 1983).

Brainstem centers may be particularly important for automatic postural responses and recruitment of low back muscles (Cottingham, Femano, and Pfaff, 1988; Femano, Schwartz-Giblin, and Pfaff, 1984; Jacobs and Horak, 2007). Voluntary motor control areas in the cerebral cortex are a significant source of cortical projections to brainstem motor centers (Buford 2008; Keizer and Kuypers, 1984[AUQ1]), and voluntary recruitment of these muscles could in part work via relays from the cortex through the brainstem to the spinal cord. If so, practice recruiting these low back muscles in a voluntary exercise for the SSE program might lead to strengthened corticobulbar projections. With those pathways strengthened, subjects could have been better able to subconsciously prepare brainstem circuits for these perturbations, which could explain why the M2 response was strengthened.

Pederson et al. (2007) trained healthy individuals to react to a variety of sudden trunk loading activities. The authors measured reflex latencies in the ES and trunk stopping time before and after the training. The training did not alter the reflex latencies in the ES but did reduce the trunk stopping time by 7.8% as compared with a control group that did not receive any training. The reduction in the trunk stopping time was achieved by a large burst seen in the ES response, reflecting the increased reflex amplitude seen in the subjects with LBP post therapy in the current study. In a more recent study, the authors (Pedersen, Randers, Skotte, and Krustrup, 2009) observed that a group of healthy individuals who underwent a recreational soccer- training program had significantly decreased their trunk stopping time and trunk stopping distance during sudden unexpected loading tests compared with a group that underwent a simple running training program. The authors attributed the improved performance of the soccer trained group to the training, which involved frequent changes in loading directions and movements brought on as a result of the nature of the game. The outcomes of these studies suggest that training programs apart from general strengthening and flexibility exercises should also perhaps incorporate reacting to sudden unexpected perturbations, which might influence to strengthen the reflex response. Our prediction for the current study was that the LBP patients would not only improve their amplitudes, but also their latencies. Our results, however, showed only a change in amplitude. Our reasons for expecting a change in latency were simply based on the fact that they were likely to be too slow in association with low back pain, and an effective treatment might restore both amplitude and latency. However, given the lack of data in the literature showing a change in M2 data, it is not surprising in retrospect that amplitude was the only significant change.

Trunk exercise programs such as the SSE program are directed toward training recruitment of muscles and restoring the cross-sectional area, particularly in the multifidus (Richardson and Hides, 2005), which is considered to be one of the important stabilizers for the intersegmental control of the spine (Kim, Gottschalk, Eng, Ward, and Lieber, 2007). The increased amplitudes of the trunk muscle reflexes in subjects with LBP after the SSE program suggest a training-induced effect similar to that reported in the arm (Wolf, Segal, Heter, and Catlin, 1995). Hypothetically, this increased amplitude would produce a more effective response to an external perturbation, limit excessive movement and excessive strain on the peri-articular tissues, and prevent injury. In contrast, a modeling study by the authors (Franklin and Granata, 2007; Franklin, Granata, Madigan, and Hendricks, 2008) found that large reflex delays required smaller reflex amplitudes in order for the system to remain stable. However, these authors only examined reflex delays greater than 60 ms. The need for smaller reflex amplitudes to maintain stability became pronounced only as the delays approached 100 ms. The reflexes observed in the subjects with LBP in the current study were in the ranges of 39–57 ms, which may not necessarily represent the same phenomenon modeled by Franklin et al.

The limitations of this study include the small sample size, the number of subjects who did not complete the SE program, and the lack of a group of subjects with LBP who did not receive the intervention. The impact of the SSE program on a cohort of CNTL subjects was not evaluated and may be worth exploring in the future. These factors limit the ability to translate the findings to a larger population. Of the thirteen subjects with LBP, eight of them reported pain on the left side, Handedness has been demonstrated to influence trunk muscle reflexes (Mullington, Klungarvuth, Catley, McGregor, and Strutton, 2009), but handedness was not analyzed in the current study and differences between sides due to pain was not accounted for in the current study. A much larger cohort of patients with left v. right sided pain and left v. right hand dominance would be required to test these relationships. In addition, similar to the previous studies, M1 reflexes were not elicited. The possible benefits of the SSE program on the segmental reflexes (M1) might be worth exploring in future.

In conclusion, longer latencies for the M2 reflex were observed in subjects with LBP at PRE therapy compared with CNTL subjects. A 10-week SSE rehabilitation program that was associated with clinical improvement in these subjects was also associated with increased amplitudes in the M2 response for lumbar extensors, but reflex latencies were not affected. These results indicate changes in the neuromuscular control system associated with rehabilitative exercises for LBP and the nature and significance of these neural control changes warrants further investigation.

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Figure 1.
Subject's position in the reflex perturbation device

Figure 2.
Raw EMG response patterns to the pseudo-random perturbations for a CNTL group subject

Figure 3.

Reflex responses in the individual low back muscles to the perturbations for A) a CNTL subject; B) a LBP subject at PRE therapy; C) and the same LBP subject at POST therapy. The M2 reflex onset latency was determined as the time from 0.0 s when the magnitude increased 2 standard deviations from the baseline activity seen in the - 0.1– 0.0 s window. The M2 reflex amplitude was determined as the shaded area under the curve.

Figure 4. Comparing mean and standard deviations for reflex latencies and reflex amplitudes for the low back muscles between A) CNTL group subjects and LBP subjects at PRE therapy; and between B) LBP subjects at PRE therapy and POST therapy.

Table 1

Subject Characteristics for the Sudden Perturbation Test Experiment

	CNTL n = 13 (6 female)	LBP n = 13 (6 female)	
Age (yrs)	35 (SD 10.1)	32.3 (SD 8.2)	p = 0.31
Height (m)	1.75 (SD 0.12)	1.75 (SD 0.08)	p = 0.43
Weight (kg)	84.5 (SD 19.9)	81.2 (SD 19.6)	p = 0.32
BMI (Kg/m ²)	27.3 (SD 4.2)	26.4 (SD 6.0)	p = 0.31