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Trunk Motor Control Deficits in Acute and Subacute Low Back Pain are Not Associated with Pain or Fear of Movement

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Abstract

Background Context—A subgroup of patients with acute/sub-acute low back pain (LBP) presenting with trunk movement control deficits, pain provocation with segmental testing, and segmental hypermobility have been clinically identified as having movement coordination impairments (MCI) of the trunk. It is hypothesized that these patients have proprioceptive, postural and movement control impairments of the trunk associated with LBP. While, trunk control impairments have been identified in patients with chronic LBP, they have not been investigated in this subgroup or closer to symptom onset.

Purpose—To identify trunk motor control (postural control and movement precision) impairments in a subgroup of patients with acute/sub-acute LBP who have been clinically identified to have MCI and determine association of these impairments with pain and fear of movement.

Study Design/Setting—Observational design; University biomechanics lab and clinical practice.

Patient Sample—Thirty-three patients with acute/sub-acute LBP identified with trunk MCI and 33 gender, age, and BMI matched healthy controls.

Self-report Measures—Numeric Pain Rating Scale, Oswestry Disability Questionnaire, Fear Avoidance Beliefs Questionnaire.

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Physiologic Measures—Postural control, Movement precision

Methods—Center of pressure movement was measured while subjects attempted to volitionally control trunk posture and movement while sitting on a platform with a hemisphere mounted underneath. This created an unstable surface that required coordinated trunk control to maintain an upright-seated posture. Postural control was tested using eyes-open and eyes-closed balance protocols. Movement precision was tested with a dynamic control test requiring movement of the center of pressure along a discrete path. Group trunk motor control performance was compared with ANOVA and t-Test. Performance association with pain and fear of movement were assessed with Pearson's Correlations. Funding for this study was provided by the National Institutes of Health (xxxxxxx; \$xxx,000), with no study specific conflicts of interest to report.

Results—Patients' postural control in the eyes closed condition ($P=.02$) and movement precision ($P=.04$) were significantly impaired compared to healthy controls, with moderate to large group difference effect sizes. These trunk motor control impairments were not significantly associated with the patients self-reported pain characteristics and fear of movement.

Conclusions—Patients with clinical identification of trunk MCI demonstrated decreased trunk motor control, suggesting impairments in proprioception, motor output, or central processing occur early in the back pain episode. This information may help to guide interventions to address these specific limitations, improving delivery of care.

Keywords

Low back pain; postural control; movement control; movement coordination impairment; motor control

Introduction

Patients with recurrent and chronic low back pain (LBP) present with several types of motor control impairments including: altered muscle timing [1, 2], changes in muscle quality [3, 4], altered proprioception of trunk movements [5, 6], and altered trunk stiffness [7]. However, in some cases, interventions that were successful in improving pain and function did not affect these motor control variables [8–10]. The success of these interventions in the absence of improvements in the above physiologic variables may be associated with the heterogeneous nature of LBP in which the treatment does not match the primary neuromuscular impairment or the specific facet of impaired motor control. Studying individual components of motor control (e.g., muscle timing, proprioception) in isolation may not capture the broader clinical picture of motor control impairments these patients present with, making translation of biomechanical findings to direct clinical care difficult.

Posturography using center of pressure (COP) data allows investigation in to the integration of sensory input, motor output and central processing [11], which may allow for a more comprehensive method of studying motor control in patients with NSLBP. Previous studies have shown that patients with chronic LBP demonstrate increased sway compared to healthy subjects during standing challenges to upright postural control, suggesting impaired motor control [12, 13]. Motor control impairments are greater when visual input is altered [14, 15], possibly due to dependence on vision once proprioceptive feedback is reduced. Patients may

also become more dependent on the lower extremity to adapt to motor control deficiencies in the trunk [16]. Many studies of motor control in patients with LBP compared patients' standing balance [12–14, 17]. This approach does not provide direct information on trunk motor control deficits as standing balance uses different motor control strategies [18] and the control effects of the limbs distort assessment of trunk motor control [19]. Seated balancing tasks may isolate motor control of the trunk and provide better insight into impairments directly affecting the trunk [20]. However, few studies have taken this approach to assessing active trunk motor control [21, 22].

While studies above have demonstrated changes in motor control in patients with LBP, these studies involved mainly small samples sizes, poorly defined and heterogeneous patient populations and primarily included patients with chronic LBP whose symptoms were continuously present for 6 months or greater. Changes in motor cortex organization have been associated with altered postural control in patients with long term LBP [23] and deficits noted in the studies above could be an expression of cortical changes associated with chronicity of the condition. Chronicity of LBP has also been associated with increased influence of risk factors for avoidance behaviors including pain catastrophizing and fear of movement may lead to greater levels of disuse and inactivity [24]. Catastrophizing behaviors can result in fear of pain within 6 months, if risks for these behaviors are present [25]. Neural pathway reorganizations have also been noted in chronic low back pain patients with persistent pain over 12 months, as alterations between pleasure centers of the pre frontal cortex and the nucleus accumbens [26]. In some patients with chronic LBP, impairments related to movement may be a result of behavior changes including deconditioning and fear of movement, rather than a reflection of some impairment in the sensorimotor pathway.

It is unknown if trunk motor control changes are present in those with a more recent onset of LBP. This would be valuable information as components of many rehabilitation paradigms for patients in the acute/sub-acute phase focus on activities related to trunk motor control with emphasis on performing exercises that require maintaining control of the trunk while performing extremity or whole body movements [27–29]. Prior studies also cluster patients together making no distinction of patient subgrouping based on clinical presentation. While LBP is considered to be heterogeneous in nature [30], treatment has been found to be more effective when patients are subgrouped through examination and interventions are matched to these subgroups [31, 32].

One of these subgroups has been identified as having trunk movement coordination impairments (MCI) associated with acute/sub-acute LBP and impaired muscle function [33]. A key physiologic predictor in these patients has been associated with impairments in lumbar multifidus (LM) function [34], a muscle that may play a large role in lumbar stabilization and proprioception [35] and has been noted to atrophy rapidly in patients with LBP [36, 37]. Based on clinical definition of the subgroup [38, 39] and physiologic changes that have been shown in those patients, trunk motor control impairments should be particularly evident in this clinical subgroup of patients. Based on pathoanatomical changes that occur rapidly in these patients, these control deficits should present early. These deficits should be present in static and dynamic conditions and identifiable using tasks that selectively and actively challenge the trunk muscles, allowing study into motor coordination.

Two examination pathways currently exist to clinically identify MCI. Identification of patients who would benefit from trunk stabilization exercises within the Treatment-based Classification (TBC) system [39], and the Movement Systems Impairment (MSI) system [40] have shown to be reliable methods, with promising validity [39, 41–44]. TBC has identified that patients younger than 40 years old with straight leg raise motion greater than 91 degrees, presence of aberrant motion during forward bending assessment, positive prone instability test, and segmental hypermobility [39, 45] can accurately identify patients who would benefit from this trunk stabilization exercises to improve pain and function. An RCT validation using these identifiers, demonstrates that presence of aberrant motion during forward bend and prone instability test, clinical examinations thought to assess trunk motor control, can better predict this group [42]. The MSI uses the quality and pain response of standardized functional movements that challenge the sagittal, transverse, and frontal plane along with progressive challenges on the trunk through extremity movements to categorize patients' trunk movement impairments [40]. Both of these methods allow for clinicians to use examination techniques to identify patients with MCI of the trunk.

The aim of this study was to identify trunk motor control (postural control and movement precision) impairments in a subgroup of patients with acute/sub-acute LBP who have been clinically identified to have MCI associated with their low back pain. It was hypothesized that these patients will: 1) have greater static balance deficits compared to healthy control subjects particularly when visual input is removed; and 2) demonstrate smaller regions of movement precision in dynamic conditions compared to healthy subjects. We also sought to determine if there was a strong association between motor control impairment in patients with acute to sub-acute LBP and their pain characteristic and fear of movement.

Methods

Subjects

Subjects in this study were part of a cross sectional study, (NCTxxxxxxx) and funded by a grant from the National Institute of Child Health and Human Development of the National Institutes of Health (xxxxxxx). Thirty-three patients, (subject demographics in Table 1) with acute to sub-acute LBP (duration of less than 3 months by self report of history) that were identified to have MCI through clinical examination were analyzed as part of this study. They were recruited from physical therapy clinics and the community through newspaper advertisements and flyers. Inclusion criteria for the study were: bilateral or unilateral low back pain in the region between T12 and sacrum, average pain greater than 3/10 on a numeric pain rating scale (NPRS; 0–10) [46], and Oswestry Disability Index (ODI) [47] of 20% or greater. Patients reporting rehabilitation intervention for the current episode of LBP were excluded. If patients had a prior history of low back pain, they were included in the study if their previous symptoms had gone into remission for at least 6 months, they had returned to premorbid levels of activity, and this episode was a recent exacerbation of their pain. Patients were excluded if they presented with low back pain attributed to (e.g., fracture, osteoporosis, tumor), frank neurological signs, a body mass index (BMI) of greater than 30, reported spinal surgeries, pregnancy, or lower extremity injury that would interfere with testing. Subjects were screened for study eligibility through two

methods. Subjects referred to the study following an initial visit to study associated therapy clinics were screened at the clinics for exclusion criteria including chronicity of symptoms, frank neurologic signs, surgery and membership into other low back pain subgroups. Subjects recruited through self-referral were screened through telephone conversations for exclusion criteria of age, chronicity, prior therapy and surgery. If study participants were screened as eligible, they were offered the opportunity to participate in the study. Those who completed informed consent to participate were further evaluated for inclusion criteria by undergoing a clinical examination performed by the study investigators.

Thirty-three gender, age (± 5 years), and BMI ($\pm 2 \text{ kg/m}^2$) matched subjects who did not participate in regular core stabilization exercises were used as a control group. Control subjects could not have a history of low back pain that required medical or allied health intervention or reduced their functional activities for greater than 3 days. All subjects read and signed a written informed consent approved by the University's Human Research Protection Program.

Clinical Examination, Pain, Functional Limitations and Fear of Movement

All subjects screened for study eligibility underwent clinical examination prior to biomechanical testing. NSLBP subjects were identified into the MCI subgroup through a comprehensive clinical examination performed by study physical therapists with over 10 years of experience in orthopaedic practice. Past medical history and thorough subjective history was taken by the clinical examiners to ensure the patients' symptoms were acute to subacute in nature. MCI characteristics for acute/sub-acute LBP include: coordination impairment during sagittal plane trunk movement, pain provocation with spinal segmental testing, segmental hypermobility, and coordination impairments during functional tasks [33]. The clinical exam was designed to ensure that the patient fit into the MCI subgroup, which included examination procedures used to determine if a patient would benefit from trunk stabilization exercises (TBC)[39]. The examination also assessed coordination impairments during functional tasks using selected items from the movement system impairment classification system (MSI) tests [40].

Clinical Examination

Clinical examination began with a neurologic screen to test dermatome and myotome integrity as well as lower quarter reflex testing. Straight leg raise and slump tests were also performed to rule out possibility of nerve root compression [48, 49]. Following the screen, patients underwent repeated motion testing in standing into flexion, extension and side gliding to determine if there were any changes to symptoms in regards to directional preference [50]. During this step, patients' forward bending was observed to determine the presence of aberrant movement including out of plane deviations, instability catch/juddering, reversed lumbopelvic rhythm or Gowers sign, described elsewhere, as presence of any of these aberrant movements would suggest membership into the MCI group [39, 42, 51]. Pain characteristics during these movements were also noted for use in MSI testing. Patients then underwent testing in supine and prone positions to assess trunk movement control, segmental mobility testing, and prone instability tests [39, 45]. This was followed by hook lying, prone and quadruped tests from the MSI that are described elsewhere [40,

44]. Movement tests were considered to be positive for impairment if there was asymmetry of movement or difficulty controlling movement within the plane [44].

The purpose of the MSI in this case was not to diagnose a patient with a specific movement pattern dysfunction, but to identify that there was some fault with trunk coordination during functional movement. MSI is typically used to match patients treatments based on their responses and results to a standardized movement battery, but it does so by identifying trunk movement impairments[40]. Clinical tests from both approaches were used to identifying MCI in this study. All patients with NSLBP subgrouped to the MCI for the purpose of the study met at least 2 of 3 clinical examination findings mentioned above [38, 39] and demonstrated at least 3 positive MSI tests. These clinical examination findings are consistent with current clinical practice guidelines for subgrouping patients presenting with primary impairment in motor coordination of the trunk and pelvis [52].

NPRS was measured to determine pain intensity on the day of testing. ODI was used to determine level of perceived disability and functional limitations. The physical activity subset of the Fear Avoidance Beliefs Questionnaire (FABQ-PH) (Table 1) [53] was collected to determine a subject's fear of movement. The FABQ-PH (0–24 scale) can help identify a person's beliefs of movement's effect on their low back pain. An FABQ-PH score of 15 or greater has been associated with high fear avoidance beliefs in patients seeking medical care for their pain [54].

Instrumentation and Procedures—A seated testing apparatus was developed that isolated trunk motor control by minimizing the contribution of the lower extremities [15, 20]. Subjects sat on a seat with a hemisphere mounted underneath (Figure 1) to create an unstable surface requiring active trunk control to maintain an upright-seated posture. The seating system itself was balanced without a subject seated on it and was adjustable to allow the location of the hemisphere and starting posture to be standardized across subjects. Subjects sat in a neutral spine position with their hips and knees flexed to 90 degrees. Isolated trunk postural control and movement precision were measured using a force plate (Kistler, Novi, Michigan, USA) that was situated under the hemisphere. For all tests, subjects were asked to sit with their arms across the chest, with the hands just under the clavicle. The following protocol was developed through separate pilot testing that focused on stabilizing performance, determining testing tolerance and reducing muscle fatigue. This resulted in subjects being provided practice trials and rests breaks in between trials (30 seconds) and tests (5 minutes) by stabilizing the seat through holding onto the safety bar located in front of them. The order of testing was designed to gradually increase the trunk control challenge. Biomechanical testing of trunk motor control lasted approximately 30 minutes. In an attempt to limit bias, different members of the research team completed the screening of all subjects, the clinical examination of the acute/sub-acute LBP subjects and the biomechanical testing.

Subjects sat on the testing apparatus with visual display directly in front of them at eye level. This display provided real time feedback of their COP. They were given 2 practice trials of free multidirectional movement with real time COP displayed on the monitor to allow familiarization with the apparatus and how movement of the trunk effected on the COP.

After this, the display was turned off and seated postural control testing was performed. Subjects actively controlled upright posture by balancing themselves for 60 seconds with the instruction to “balance themselves and move as little as possible”. Three trials with eyes open (EO) followed by 3 trials with eyes closed (EC) were recorded, after a 30 second practice trial in each condition. Breaks (30 seconds) were given between each trial. Trials in which the subject completely lost control of the chair were eliminated and the trial retested.

Once postural control testing was completed, the monitor was turned on and movement precision was tested. At the start of the test, lines were displayed on the monitor and subjects were required to move a dot representing their COP, along the path of the line. Figure 2 contains the 8 directions and order in which the subjects were required to move their COP. The order was not randomized; 1-practice trial was followed by 2 test trials with 30-second rest breaks. To accomplish this task and move their COP along a particular path, the subject had to maintain upright-seated control, while reconfiguring the trunk segments (lower thoracic, lumbar spine and pelvis [see insert, Figure 1]), and maintaining their upper trunk/head over their pelvis. The LBP subjects did not report pain intensity changes during the seated protocol.

Data Reduction

Center of pressure time series was calculated from force plate data collected at 2400Hz, filtered and down sampled to 400Hz with custom written software (Labview 8.6, National Instruments, Austin, TX, USA). For seated postural control, a 95% confidence ellipse area (CEA_{95}) (mm^2) of the COP was calculated during EO ($EOCEA_{95}$) and EC ($ECCEA_{95}$) conditions and averaged across 3 trials. CEA_{95} represents the area that contained 95% of the COP tracing during testing [55]. Larger CEA_{95} measures indicate greater deviation from the balance position and therefore decreased postural control. Dynamic control regions (DCR) were derived as a measure of movement precision. For DCR, the maximum distance the subject maintained precise directional control was calculated. For each direction, the distance at which the subject's COP crossed a defined boundary to the left or right of the directional control line, determined loss of directional control. These boundaries were standardized to 10% of the distance the COP moved from the center position. This allowed the boundary (absolute error off the line) to proportionally increase as the subject displaced their COP further from the center balance point (Figure 2). The maximum distance each subject traveled in the 8 directions was used to form octants. The area of each octant was calculated using a known side/angle/side triangular formula, with the following equation where $Area_{octant} = 1/2 bc \sin A$, where A is the angle of the octant, and b and c are either sides of the known distance, forming a triangle (Figure 2). The areas from all octants were added to determine the DCR and averaged across 2 trials. This represents the area boundaries that a subject would be able to demonstrate precisely controlled trunk movements away from a trunk neutral starting point.

Reliability and Protocol Measurement Error—Within-session reliability was performed using 10 controls and 10 acute/sub-acute LBP patients with clinical findings of MCI to determine minimal detectable change (MDC) for static and dynamic tests. Trial 1 and 3 were used for EO/EC CEA, while the 1st and 2nd trials were used for DCR. ICC (3,1)

were calculated, and used to determine the MDC with 95% confidence (MDC_{95}) for trunk control variables. Using this approach, the interaction between subjects and repeated trials is reflected in the reliability coefficients [56], therefore any changes in performance due to learning effect negatively impacted these values. Incorporating the standard deviation between trials in calculation of the MDC_{95} produces an estimate of the smallest amount of change that reflects a measurable change in performance factoring in learning effect and instrument error with 95% confidence [57]. The MDC_{95} was used to determine if group differences exceeded protocol measurement error and reflected true differences in performance. ICC (3,1) and MDC_{95} were as follows: EOCEA₉₅ was 0.94 with a MDC_{95} of 48.8 mm², ECCEA₉₅ was 0.90 with a MDC_{95} of 198.5 mm², and DRC was 0.95 with a MDC_{95} of 21.9 cm².

Data analysis

Statistical analysis was performed using SPSS 21 (IBM) and alpha was set at 0.05 for all analyses. Comparison of seated postural control in EO/EC (condition) and DCR was made between the subgroup of acute/sub-acute LBP patients and controls. A mixed model ANOVA (between groups: LBP/control; within condition: EO/EC) was used to compare CEA₉₅ during the seated postural control. Eta squared (η^2) was calculated to determine effect size or strength of association. For DCR, an independent t test was used to compare directional control region area between groups. Cohen's d was calculated to determine effect size or expression of the magnitude of standardized difference between group means. Statistically significant differences were interpreted as meaningful if the effect size was at least medium ($\eta^2 > .13$ and $d > .50$) [58] and the group differences exceed our protocol MDC_{95} . Pearson correlations were performed to explore if there were any associations between motor control variables and pain intensity, number of previous low back pain episodes, duration of current episode, and fear of movement, with the intention of performing further regression statistics if there were any significant correlations. The strength of the correlation was interpreted based upon the following: no or little relationship (0.00–0.25), fair (0.25–0.50), moderate to good (0.50 – 0.75) and excellent (> 0.75) [59].

Sample Size

A priori power analysis was performed to estimate sample size based upon a pilot of study ($n = 10$), which compared LBP and control subjects during the seated postural control protocol in both EO and EC conditions. The effect size from this pilot was considered to be medium ($f = .29$). Using $\alpha = .05$, power = .80, and this effect size; the projected sample size was 37 subjects pre group.

Results

Total of 195 patients with low back pain were screened from October 2009 to November 2013. Fifty-two acute/sub-acute LBP subjects met the initial inclusion criteria. Forty-one subjects met the criteria for MCI, but 2 withdrew from the study prior to data collection. Thirty-nine acute/sub-acute LBP subjects who met MCI criteria completed the testing protocol. Complete loss of seated control occurred in the EC condition in less than 10% of the subjects and all but 1 was able to complete the 3–60 second trials. An additional 5 LBP

subjects' biomechanical data were eliminated during post-processing due to discovery of the technical malfunction of the force plate and were not included in the study. This resulted in full data on 33 subjects with acute/sub-acute LBP and their matched (age, gender, body mass index) control subjects ($n = 33$) being used for group difference analysis. Total number of subjects was 66. See flow diagram (Figure 3).

Static Postural Control

Motor control variables were tested for multivariate normal distribution and outliers using Mahalanobis distance with a critical value of 13.8 and all assumptions were met. Mean EOCEA₉₅ was 114.7 mm² (SD: 123.7) for the control group and 169.3 mm² (SD: 120.3) for the subgroup of patients with acute/sub-acute LBP. Mean ECCEA₉₅ was 327.7 mm² (SD: 454.4) for the control group and 777.4 mm² (SD: 935.4) for patients. Comparison of seated balance in EO/EC CEA₉₅ between LBP and controls demonstrated a significant main effects of group $F(1,64)=6.4$, $P<0.014$, effect size (η^2)= 0.30 and condition $F(1,64)=24.4$, $P<0.001$, $\eta^2=0.53$. There was also a significant interaction between group and condition $F(1,64)=5.6$, $P=0.02$, $\eta^2=0.28$. The LBP patients had greater COP area (less trunk postural control) than the healthy subjects. Both patients and controls demonstrated less postural control in the EC condition. The greatest deviation of the COP (impairment) was seen in the LBP group in the EC condition. Group differences for EOCEA₉₅, and ECCEA₉₅ exceeded MDC₉₅.

Dynamic Directional Control

For the DCR, the control group had a significantly larger precision control region with a mean of 90.1 cm² (SD: 85.9) compared to LBP patients' mean control region of 67.6 cm² (SD: 39.5), $T(1,64)=2.04$, $P=0.04$, effect size (Cohen's d)=0.51 (Figure 4). Group differences for DCR exceeded MDC₉₅, and therefore were considered to exceed testing and apparatus error.

Correlation to Symptoms

There were no significant correlations (Table 2) between trunk motor control parameters and pain intensity (NPRS), number of prior episodes of back pain, duration of pain, or fear of movement (FABQ6-PH). Given little to no relationship between these variables and trunk motor control, we did not move forward with regression analysis.

Discussion

Study findings support the hypothesis that trunk motor control is impaired in the subgroup of patients with acute/sub-acute LBP presenting with clinical signs of motor coordination impairment. Our findings suggest that both isolated trunk postural control and precision of movement are compromised in this subgroup of patient during an acute to sub-acute episode of LBP. The strength of this study lies in both the homogenous subgrouping of the LBP subjects and the use of seated static and dynamic methods to compare isolated trunk motor control supported by group differences that demonstrated large effect sizes and exceed variance due to protocol error and learning effect. In addition, our sample represents a clinical subgroup of acute/sub-acute LBP that is frequently seen in clinical practice. Our

findings are consistent with those reported by Radebold et al (2001) [21] in patients with chronic low back pain, but not with van Dieen et al (2010) [22] whose patients population was based on a reported history of back pain, some still reporting current symptoms. Both of these studies used a similar seated paradigm to assess isolated trunk postural control, where the leg and foot support was attached to the seat to prevent lower extremity influence. Other investigators have studied trunk motor control impairments in patients with LBP in the standing position [17, 60] and also reported motor control impairment. Again, those studies were also performed on patients with chronic low back pain. We believe our data are robust as the protocol for testing active trunk control is based on subject performance with limited tester influence. In addition, efforts were made to decrease any bias by separating clinical testing from lab-based sessions and using different researchers to complete data collection for the clinical examination and seated testing protocols. Based on this, blinding the seated protocol tester was accomplished for 85% of the subjects.

An underlying assumption of this study was that the custom seating apparatus isolated movements of the trunk and provides a reliable and valid measure of motor control that is more focused to the trunk. This approach allows a more specific look at trunk motor control. Greater upright-seated postural control impairments in this acute/sub-acute LBP subgroup when the eyes were closed suggests an increased dependence on visual information to maintain postural control in this subgroup of LBP patients. While performance of the LBP subgroup is decreased in the eyes open condition compared to healthy controls, the availability of visual information may help to offset other system impairment in our seated paradigm. Prior work has pointed to a greater reliance to the lower extremities for proprioceptive information in patients with recurrent LBP [14, 61]. When vision and unimpaired lower extremity sensory information are eliminated, the trunk becomes the focal point of somatosensory input. It is likely that without supplemental visual information, there is a reduction in information that can be used for processing positional awareness, leading to the greater reduction in postural control seen in EC condition. This is suggestive of reduction in trunk proprioception in this subgroup of acute/sub-acute patients, but confirmation of this requires further investigation. Altered trunk proprioception has been reported in patients with LBP [16, 62, 63]. Reduced somatosensory input from the spinal structures may hamper overall ability to detect situations that require changes in motor activity and affect the ability to stabilize the spine during changes in demand.

The dynamic control region (DCR) comparison also suggests that this subgroup of patients with acute/sub-acute LBP have a significant reduction in the area they are able to demonstrate precise, controlled movements. DCR testing required the subject to use isolated movements of the trunk to move a real time target, representative of their COP, along a clearly defined line. This test combines the motor efferent function necessary to displace the COP, as well as processing somatosensory input of current COP location, to make changes in the trajectory of movement in order to stay on the path. The test is more likely an indicator of movement accuracy as the subject is required to process visual information regarding COP position on the visual display, use somatosensory information to determine current position of the trunk, and use motor efferent activity to guide the COP along the path. Our subgroup of patients with acute/sub-acute LBP demonstrated reduced ability to move the COP away from the starting position while staying within the boundary,

demonstrating a reduction in ability to accurately coordinate somatosensory input and motor output. The large effect size ($d=0.51$) suggests that impairment in the processing of the somatosensory information or specific muscle control needed to produce smooth coordinated movement may play a large role in separating out those who are healthy from those in a current episode of LBP.

Altered somatosensory processing in patients with LBP has been noted previously in the literature with reports of as greater reliance on lower extremity afferent information [61]. Brumagne et al (2008)[14] demonstrated that patients with LBP had greater postural disturbance with vibration stimulation of the triceps surae than with vibration to the lumbar multifidus, as is typically seen in healthy subjects. This suggests a shift from proximal proprioceptive input of intrinsic spinal musculature to the more distal calf muscles in these patients. Since a shift to lower extremity control is not a productive option in this protocol, these patients may use a different strategy to maintain postural control that de-emphasizes the intrinsic proprioceptive mechanism of the spine [60]. The effect of changes in somatosensory and motor organization is demonstrated by the significantly larger COP in our subgroup of patients with acute/sub-acute LBP in the EC condition of the seated balance test. The combination of altered sensory and motor organization in these patients would reduce their ability to maintain postural control on an unstable surface.

One concern of testing subjects with acute or sub-acute symptoms is that their pain intensity may have an effect on measures of trunk control. However our findings demonstrated a weak and non-significant correlation between pain intensity and performance. In addition, our LBP subjects did not report high pain intensity at the time of testing, nor did they report symptom increase during the protocol. However, this does not eliminate the possibility that just experiencing pain alters trunk motor control and this should be considered in interpreting our findings. This interpretation is supported by experiments that induced pain in non-painful subjects and found changes in motor control [64, 65].

Another potential confounder is prior episodes of low back pain. While our subject reported they recovered from those episodes, prior injury or higher rate of recurrence may signal residual motor control deficits that have influenced our findings. Findings of residual motor control deficit when pain is no longer present is supported by the work of Lomond et al (2014) [66]. Given that 76% of our subjects had prior episodes of low back pain, our findings may reflect residual trunk motor control impairment, which may be a risk factor for recurrence of symptoms. However, the weak and non-significant correlations between the duration of the current low back pain episode or history of recurrence did not significantly effect the magnitude of the motor control impairments in these subjects. While fear of movement could also influence our findings, 67% of our LBP subject's FABQ-PH scores were below the threshold (15 points) that indicates elevated fear of movement [54]. In addition, there was little to no relationship between trunk motor control performance and the FABQ-PH. Given this we believe that fear of movement did not have a significant influence on our findings.

Our subject's self-reported pain intensity and fear of movement were similar to the patient samples used in the derivation (NPRS 4.5 ± 2.4 ; FABQ-PH: 14.6 ± 5.9)[39] and validation

(NPRS 4.9 ± 1.7 ; FABQ6-PH: 16.2 ± 4.4)[42] phases of the clinical prediction rule (CPR) that identified clinical criteria for the TBC stabilization subgroup. A detailed description of the clinical tests and criteria used in the CPR can be found in the appendix of the Hicks et al (2005) study [39]. These clinical criteria were used in the determination of eligibility for our LBP sample, suggesting impairment in controlling the motor system to stabilize the spine, may be a factor in this acute/sub-acute LBP subgroup. However, our criteria also included tests that directly assessed movement coordination and control during simple extremity movement tasks including: active straight leg raise, side lying hip abduction, quadruped rocking, supine active hip abduction, prone knee flexion and prone hip rotation and extension. Several of these tests were borrowed from the MSI classification system developed by Sahrmann and we refer the reader to her work [40] and that of her colleagues [43, 44] for more information regarding these assessments. The study results highlight the possibility of a different or expanded cluster of clinical exams that identify the presence of trunk motor control impairments. This is supported by a recent published randomized control trial of patients with LBP that compared matched to unmatched treatment using TBC vs. MSI approaches which yielded significant improvements in pain and function with no differences between matched and unmatched group outcomes [67]. Given that the current research indicates that approximately 30% of patients who meet the current CPR criteria alone, do not adequately respond to lumbar stabilization exercises [39, 42], perusing the possibility of an expanded cluster of clinical examinations opens the path to further research into identifying clinical exam items that may better predict impaired trunk motor control and potentially improve the response rate to stabilization exercises.

Our findings suggest that patients presenting with clinical examination findings consistent with our LBP subgroup demonstrate impaired motor control in the acute/sub-acute phase of back pain. This supports the underlying premise of the primary impairment in this LBP subgroup and suggests that treatment approaches designed to address trunk motor control impairment (e.g., lumbar or core stabilization exercises) are appropriate for these patients. However, whether or not trunk motor control impairment is unique to this subgroup of LBP requires further investigation. When considering specific treatment exercises, our findings may support the need for emphasizing not only maintenance of posture (control of neutral trunk and pelvic position) during rehabilitation exercises, but also the importance of movement precision. Thus, adding movement precision tasks using proprioceptive challenges and movements outside of a neutral trunk position, such as moving away from a set control point, to the rehabilitation regimen may also be an important component to restoring trunk motor control. However, confirmation of these recommendations requires systematic intervention studies. This protocol provides a mechanism to systematically test the ability of specific low back interventions to changes trunk motor control, as well as clinical outcomes, and should be considered a viable model for future investigations.

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Figure 1.

Custom designed apparatus to test isolated trunk motor control. Subject's legs are constrained to prevent lower extremity compensation. Seat is balanced on a hemisphere designed to tip forward if lower extremities are involved in moving the trunk. Graphic demonstrates the trunk motions (dashed lines) performed while the patient is seated in the chair and making adjustments in the sagittal and frontal planes. The chair allows for movements in the sagittal, frontal, and transverse planes.

Figure 2.

The order and the 8 directions in which the subjects were required to move their center of pressure during the directional control test. The directional control region was calculated summing the area of the 8 octants, with each octant defined by an angle (a) and two known sides (b and c). The precision of the COP trajectory in the 8 directions was determined by a standardized stability boundary. This boundary (lines to the left or right of the directional line) was calculated in real time, at 10% of the distance the subject moved their COP away from the center position. Thus, at a distance of 10 mm along the line, the boundaries would be set 1 mm. Between 10 and 20 mm from the center, the boundaries would gradually increase to 2 mm. Maximum distance for each of the direction was the maximum excursion the subject was able to move away from the center along the directional line without his or her COP crossing the stability boundary. This provided the length of sides b and c.

Figure 3. Flow diagram outlining the screening process, clinical examination and trunk postural and dynamic control testing of subjects with low back pain and the matching healthy control subjects.

Figure 4.

This graph provides an example of a directional control region for a matched healthy control subject and patient with non-specific low back pain. The patient has a smaller area and less symmetry in their directional control region.

Table 1

Subject demographics and characteristics.

	LBP Group	Healthy Controls
Subjects	33	33
Gender (# females)	20	20
Age (years)	32 (14)	34 (13)
Height (cm)	170.4 (7.9)	169 (9.3)
BMI	25.6 (4.4)	23.8 (3.8)
Numeric Pain Rating (0–10)	4.2 (1.7)	NA
Oswestry Disability Index (0–100%)	23.9 (8.9)	NA
FABQ-PH	12 (6.4)	NA
Duration of Pain (days)	47 (33)	NA
No Prior Episode (# subjects)	8	33
2–3 Prior Episodes (# subjects)	13	NA
4 Prior Episodes (# subjects)	12	NA

Data represents frequency and mean (standard deviation).

Oswestry Disability Index: higher score represent greater functional limitations.

BMI: Body mass index

FABQ-PH: Fear Avoidance Belief Questionnaire Physical Activity Subset; higher score represents greater fear of movement.

Table 2

Pearson correlations between measures of trunk motor control and pain characteristics and fear of movement.

	Eyes Open CEA	Eyes Closed CEA	Directional Control Region
Pain Intensity (NPRS)	−0.1	0.05	−0.1
Number of Prior Episodes	0.05	−0.05	−0.05
Duration of Pain (days)	−0.11	0.04	0.11
Fear of Movement (FABQ-PH)	0.09	0.24	−0.16

No correlations were significant at $P < .05$

NPRS: Numeric Pain Rating Scale

FABQ-PH: Fear Avoidance Belief Questionnaire Physical Activity Subset