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Trunk Postural Muscle Timing Is Not Compromised In Low Back Pain Patients Clinically Diagnosed with Movement Coordination Impairments

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Abstract

Trunk muscle timing impairment has been associated with non-specific low back pain (NSLBP), but this finding has not been consistent. This study investigated trunk muscle timing in a subgroup of patients with NSLBP attributed to movement coordination impairment (MCI) and matched asymptomatic controls in response to a rapid arm-raising task. Twenty-one NSLBP subjects and 21 matched controls had arm motion and surface EMG data collected from 7 bilateral trunk muscles. Muscle onset and offset relative to deltoid muscle activation and arm motion, duration of muscle burst and abdominal–extensor co-contraction time were derived. Trunk muscle onset and offset latencies, and burst and co-contraction durations were not different ($p>0.05$) between groups. Patterns of trunk muscle activation and deactivation relative to arm motion were not different. Task performance was similar between groups. Trunk muscle timing does not appear to be an underlying impairment in the subgroup of NSLBP with MCI.

Keywords

Low back pain; motor control; trunk muscle function; feedforward postural adjustment

Introduction

Neural mechanisms that govern trunk postural control maintain dynamic equilibrium and alignment of trunk segments during voluntary movements of the extremities through

coordinated recruitment and timing of trunk muscles. Trunk postural control is accomplished using two strategies: anticipatory postural adjustment (APA) and compensatory postural adjustment (CPA). APA is the primary strategy that presets trunk stability prior to extremity movements through feedforward activation of trunk muscles to oppose the perturbing forces. The central nervous system also uses compensatory postural adjustment (CPA) to maintain trunk stability during extremity movements by modulation of feedforward activation of trunk muscles.

APA dysfunction appears strongly associated with chronic and recurrent low back pain (LBP) (Brumagne, Janssens, Janssens, & Goddyn, 2008; Hides, Richardson, & Jull, 1996; Hodges, 2003; Lariviere, Gagnon, & Loisel, 2000; Radebold, Cholewicki, Polzhofer, & Greene, 2001). A preponderance of literature suggests that patients with a history of or chronic LBP respond in a less timely manner (delayed activation or onset of trunk muscles) and lack feedforward activation (activation of trunk muscle prior to trunk perturbation) of specific trunk muscles as compared to asymptomatic subjects following self-generated arm or leg motions (Hodges & Richardson, 1997a, 1997b; Huang, Hodges, & Thorstensson, 2001; Mehta, Cannella, Smith, & Silfies, 2010). However, recent evidence has demonstrated no delay in trunk muscle activation/onset timing and no lack of feedforward activation in heterogeneous groups of patients with chronic LBP (Brooks, Kennedy, & Marshall, 2012; Gubler et al., 2010).

These conflicting findings could be related to methodological differences between studies, such as instrumentation used to capture muscle activity (e.g., ultrasound, surface EMG, indwelling EMG), method of onset identification (e.g., visual, computer and algorithms) and which trunk muscles were assessed (e.g., abdominal only, one side) (Gubler et al., 2010; Hodges & Bui, 1996; Hodges & Richardson, 1996; P. Marshall & Murphy, 2003). Reported differences in the trunk muscle onset timing could also be attributed to differences in protocols (in what direction the arm was moved) and how well the standardized task was performed (e.g., magnitude of perturbation, velocity of arm movement, base of support and instructions) (Hodges & Richardson, 1997c, 1999; Santos & Aruin, 2009). Therefore, standardizing, monitoring and comparing timing and protocol performance variables across a bilateral representation of key trunk muscles will strengthen our understanding of timing impairments and interpretation of findings. While, prior studies found muscle onset timing differences between groups, onset time has both a fairly large within subject variance (requiring averaging 5–10 trials to stabilize measurement) and between subject variance (Brooks et al., 2012; Hodges & Richardson, 1996). Therefore, with the absence of reported measurement error in many of the manuscripts true differences in performance may be undetermined within the prior literature. Vasseljen et al. 2012, is one of the few studies to report their protocol measurement error indicating that intramuscular EMG signal differences of less than 30ms were not distinguishable from measurement error (Vasseljen, Unsgaard-Tondel, Westad, & Mork, 2012).

Recent literature on LBP suggests that not all patients with LBP have the same level of dynamic trunk postural control dysfunction. Silfies et al. (2009) demonstrated that subgroups exist within the chronic LBP population based on the level of dynamic trunk postural control dysfunction (Silfies, Mehta, Smith, & Karduna, 2009). There is further

evidence for subgrouping based on research using the Treatment-Based Classification System (Childs et al., 2004; Delitto, Erhard, & Bowling, 1995; Fritz, Cleland, & Childs, 2007; Hicks, Fritz, Delitto, & McGill, 2005) and diagnostic systems developed by McKenzie (Hefford, 2008), Sahrman (Van Dillen et al., 2003), and O'Sullivan (P. O'Sullivan, 2005). Current low back pain clinical practice guidelines identify one subgroup of patients as those primarily demonstrating poor neuromuscular control and movement coordination impairment (MCI) that is related to their low back symptoms (Delitto et al., 2012; Hicks et al., 2005). This subgroup demonstrates exacerbation of symptoms with spinal movements and poor trunk muscle function when spine stability is challenged. The matched standard of care intervention focuses on exercises designed to improve muscle firing patterns and enhance stability of the lumbopelvic region. Theoretically, this clinical subgroup would have the most imprecise trunk postural control and therefore by specifically testing this subgroup we would expect to find significant trunk postural control dysfunction. In addition to the lack of subgrouping in previous studies, most previous investigators have only compared subjects with recurrent or chronic symptoms (Gubler et al., 2010; Hodges & Richardson, 1996; P. W. Marshall & Murphy, 2008; Mehta et al., 2010; Silfies et al., 2009). To date, no other investigators have compared trunk postural control between a homogenous subgroup of LBP patients during the acute to subacute phase of their symptoms. It is important to identify the existence of postural control impairments in this LBP subgroup during the acute/ subacute phase of symptoms, as the standard of care intervention used during these phases is based on the assumption of impaired muscle timing contributing to poor movement coordination and control during functional tasks (Delitto et al., 2012).

While much of the current research has focused on APA dysfunction in patients with LBP, other postural control strategies, such as CPA have not been as thoroughly evaluated. Therefore, the current literature on trunk postural control during self-perturbation provides only a partial picture of the trunk's muscle timing response (Hodges & Richardson, 1996, 1998, 1999). Santos, Kanekar and Aruin (2010) provided evidence of a relationship between the anticipatory and compensatory components of postural control (Santos, Kanekar, & Aruin, 2010). They suggested that while dealing with anticipated perturbations, the central nervous system (CNS) optimally uses the APA strategy, resulting in appropriate scaling down of the CPA. However, in conditions where APA is inadequate, sufficient CPA would be needed to preserve dynamic equilibrium and alignment of trunk segments (Santos et al., 2010). Both APA and CPA strategies are critical for postural control, because together they synchronize trunk muscle onset and deactivation/offset time based on the intensity of trunk perturbation to minimize displacement of the center of gravity and maintain whole body equilibrium (Friedli, Cohen, Hallett, Stanhope, & Simon, 1988; Friedli, Hallett, & Simon, 1984; Mochizuki, Ivanova, & Garland, 2004). The duration between onset and offset time of each trunk muscle (burst duration) also plays a major role in dynamic stability of the trunk by absorbing, dissipating, and counterbalancing the moments associated with the voluntary perturbation (Mehta et al., 2010). Burst duration of trunk muscles also influences the duration of agonist and antagonist co-contraction, which is associated with dynamic trunk stability. Since all of the trunk muscle timing parameters play an integral role in control of postural perturbation, knowing the onset latency alone does not allow us to accurately identify individuals with LBP who have a postural control dysfunction. Simultaneous

investigation of other parameters is important to explore trunk postural control differences between groups. To provide a perspective for all of these parameters, the kinematics of the self-initiated perturbation (i.e., extremity motion) also need to be considered. To date, we are unaware of reports of the trunk muscle timing response (onset and offset timing) relative to the extremity motion. It is plausible, that by simultaneously analyzing several postural control parameters with respect to extremity motion, we may begin to identify more specific dysfunctions in trunk postural control strategies.

Thus, the primary purpose of this study was to investigate the differences in several parameters of trunk muscle timing representing both APA and CPA strategies (onset latency, offset latency, burst and muscle co-contraction duration) across a robust representation of trunk muscles in a homogenous subgroup of patients with LBP and matched asymptomatic controls during a known, self-initiated postural challenge. Based upon previous studies and our preliminary findings, we hypothesized that patients in the MCI subgroup will demonstrate significantly delayed trunk muscle onsets and offset times, longer burst duration and decreased duration of muscle co-contraction in comparison to a control group, thus demonstrating timing impairments in trunk postural control. In addition, we expanded our analysis to assess trunk muscle timing data relative to the extremity motion and compared task performance between the groups using kinematic parameters of the arm motion, perturbation forces generated on a force plate, and deltoid reaction time.

Materials and Methods

Study Participants

Twenty one participants with acute to subacute non-specific LBP (NSLBP) who met the inclusion criteria for a clinical diagnosis of MCI were matched to asymptomatic controls by age (± 5 years), sex and body mass index (± 2 kg/m²). Their physical and clinical characteristics are listed in Table 1. NSLBP is defined as back pain that is not directly attributed to a known pathology or disease and encompasses approximately 85% of the low back pain cases (Balagué, Mannion, Pellise, & Cedraschi, 2012; Hart, Deyo, & Cherkin, 1995). A power analysis demonstrated that 19 subjects per group were needed based on effect size = 0.79, $p = 0.05$, power = 0.80. The effect size was determined from data obtained during a pilot study completed in our lab. The university's institutional review board approved this study and informed consent was obtained from all participants.

Inclusion criteria for the control group were no episodes of back pain: (1) that limited performance of daily activities for longer than 3 days, (2) for which they sought healthcare, and (3) with no regular exercise routine that consisted of Pilates or core stabilization exercises. Inclusion criteria for the NSLBP group were (1) less than 3 months duration of the current episode, (2) average pain intensity over past 2 weeks greater than 3 on an 11 point (0 = no pain, 10 = worst pain ever) numeric pain rating scale, (3) self-reported global function less than 80% (0–100 %, 100% = normal pain free function), (4) Oswestry Disability Score > 20%, and (5) no medical intervention for back pain within the 6 months prior to this episode. In addition, participants needed a diagnosis of MCI based upon specific clinical examination findings. Our operational definition of a MCI was at least 1 positive test in 2 or more of the following 4 categories: (1) abnormal active trunk movement patterns during

forward bending and multi-segmental movements, suggesting poor muscle activation coordination (Hicks et al., 2005), (2) hypermobility or pain during joint mobility tests suggesting clinical instability of the spine that may not be adequately controlled by reflex systems in passive motion or during muscle activation (Fritz et al., 2007; Hicks et al., 2005), (3) inability or difficulty with stabilizing the trunk during extremity motion tests suggesting altered or poor muscle activation patterns or timing (Van Dillen et al., 2003), and (4) inability or difficulty with specific activation of deep (TrA, IO, LM) stabilizing trunk musculature resulting in decreased spinal stability (P. B. O'Sullivan, Twomey, & Allison, 1998). See clinical practice guidelines for details regarding these examination procedures (Delitto et al., 2012). Physical therapists (n=3) with expertise in spine examination and treatment performed the clinical examination and determined the clinical diagnosis of MCI. Exclusion criteria included structural spinal deformity, spinal fracture, spinal tumor or infection, previous spinal surgery, or frank neurological signs.

Surface EMG Recordings

Muscle activity was recorded bilaterally from 7 trunk muscles (transversus abdominis/internal oblique [TrA/IO], external oblique [EO], external oblique 2 [EO2], rectus abdominus [RA], superficial lumbar multifidus [LM], lumbar erector spinae [LES], thoracic erector spinae [TES]) using surface EMG electrodes. Ag/Ag Cl surface electrodes (Ambu Inc., Glen Burnie, MD) with an inter-electrode distance of 2 cm and recording surface area of 13.2 mm² were used. Electrodes were aligned parallel to muscle fibers and placed in accordance with previous studies (Cholewicki et al., 2005; P. Marshall & Murphy, 2003; Mehta et al., 2010). Ability to capture deeper trunk muscle groups (TrA, IO) using surface electrodes has been previously validated (P. Marshall & Murphy, 2003; McGill, Juker, & Kropf, 1996). In addition, surface EMG signals provide a more representative picture of the muscle activation as a whole, but with the caveat of potential crosstalk. Taken together, our collection of 7 muscles groups bilaterally should robustly capture the trunk postural response to the perturbation. EMG signals were collected (2400 Hz) on lead wires (Noraxon Inc., Scottsdale, AZ), differentially amplified and filtered by preamplifiers (CMRR > 100 dB, bandwidth 6–29k Hz, 500 gain). The analog signal was further amplified (1000x gain; band pass 10–500 Hz, Butterworth low pass 2nd order and 4th order, range $\pm$ 5V) using a bioelectric amplifier (SA Instruments Company, San Diego County, California) and then converted to digital signal with a 16-bit A/D board (MIO-AT 16x; National Instruments, Austin, TX).

Electromagnetic Tracking System

An electromagnetic tracking system (Liberty, Polhemus, Inc., Colchester, VT) was used to record the position and orientation, each in 3 dimensions, of the dominant arm proximal segment with respect to the trunk using the global coordinate system. The arm sensor was mounted to an orthoplast cuff, which was strapped to the distal end of the dominant arm, just above the medial and lateral epicondyles (sensor positioned just above the Olecranon) and the trunk sensor was affixed to the T3 spinous process. The magnetic field generator was positioned 50 cm behind the subject at the level of the first sacral vertebra. Data were collected at 120 Hz during the self-perturbation task with the primary motion of the arm being within the sagittal plane.

Protocol - Standing Self - Perturbation Task

Subjects stood relaxed for at least 20s on a force plate (Kistler Instruments Corp., Amherst, MA, sampled at 2400 Hz) with the feet aligned under hips and weight equally distributed on feet prior to each trial. Subjects performed a quick flexion of the dominant arm through full range (>130 degrees) in the sagittal plane in response to an auditory signal presented randomly (1–3s). Thirty-second rest periods were provided between each trial to reduce fatigue. Surface EMG from trunk muscles and dominant arm anterior deltoid, dominant arm kinematics and force plate measurements were recorded simultaneously starting 1.5 s before and ending 5 s after the auditory stimulus. Subjects performed 6 repetitions of this task. Those tasks that did not meet the standards of alignment, quiet stance, quick motion or full arm flexion were re-recorded.

Torques

The rapid arm movement produced a change in the trunk torque along the vertical axis (T_z). Using force plate recordings, the maximum torque generated during the task was used to characterize the amount of trunk perturbation due to arm acceleration (Bleuse et al., 2005; Bleuse, Cassim, Blatt, Defebvre, & Guieu, 2002).

Data Processing and Reduction

Muscle Timing Variables—Heart rate was removed from the surface EMG data using fast independent component analysis (ICA) (Canella & Silfies, 2010; Mak, Hu, & Luk, 2010; Staude, 2001), data were then full wave rectified and low pass filtered (20Hz; RMS filter). A custom LabVIEW program (National Instruments, Austin, TX) was used to automatically identify muscle onset and phasic offset times (ms) (Figure 1). Anterior deltoid and trunk muscle onset and offset were defined using a threshold-based algorithm. Onset was determined as the time point where surface EMG amplitude exceeded 3 standard deviations of the median resting surface EMG signal for a minimum of 50 ms (Di Fabio, 1987; Hodges, 2001; Hodges & Bui, 1996). Similarly, phasic offset was defined as the time point where surface EMG amplitude dropped below 30% of the maximum activation for 100ms (Mehta et al., 2010). Baseline was set using the median amplitude of 500ms of data prior to auditory stimulus. All data were visually inspected for inaccurate picks and those values discarded.

Trunk muscle onset latency (ms) was defined as the time difference between the onset of individual trunk muscles and onset of the anterior deltoid. A negative number indicated the trunk muscle onset occurred before that of the anterior deltoid. Similarly, muscle offset latency (ms) was defined as the time difference between the phasic offset of each trunk muscle and the onset of the anterior deltoid. The muscle burst duration (ms) represents the time interval between the onset and phasic offset of each trunk muscle. This period of activity is representative of the phasic recruitment of the muscle associated with the rapid flexion of the arm. Trunk abdominal–extensor co-contraction (ms) was defined as the time spent in simultaneous activation of abdominal and extensor muscles. Co-contraction duration (ms) was determined by first plotting burst duration of each muscle and then calculating the overlap time between the abdominal and extensor phasic bursts (Mehta et al., 2010). These procedures were performed separately on each participant's data. Each

participant's onset latency (ms), phasic offset latency (ms), burst duration (ms) and co-contraction (ms) were averaged across the 6 trials and the mean values were used in the data analysis. Test-retest reliability and minimal detectable change (MDC) for muscle onset latency, phasic offset latency and burst duration were determined using (n=19) control subjects with a retest 8–10 weeks later under the same conditions. Test-retest reliability was calculated using an Intraclass Correlation Coefficient ($ICC_{(3, 6)}$) (Table 2).

In addition, each subject's mean onset latency (ms; average of 6 trials) for each trunk muscle was categorized as being under feedforward or feedback control. Feedforward control was defined as 150 ms prior to or within 50ms of the anterior deltoid onset. Muscle onset post 50 ms of the anterior deltoid onset was considered under feedback control. This criterion has been consistently used to dichotomize muscle onset responses into feedforward or feedback activation status (Gubler et al., 2010; Hodges & Richardson, 1996).

Task Performance Variables—The angular displacement (rotation [°]) and angular velocity ([°/sec]) of the dominant arm were determined with respect to the sagittal plane of the subject (trunk sensor reference). Onset of arm movement was determined by the first time after the auditory stimulus at which arm velocity exceeded the value of 3 standard deviations of the resting arm velocity 500ms prior to auditory stimulus. Maximum angular displacement and maximum angular velocity of the arm were used in data analysis of performance. To describe the timing of trunk muscle activation relative to arm movement, the angular velocity was segmented into 3 phases: Bin I - auditory trigger to onset of arm movement, Bin II - onset of arm movement to maximum arm velocity, and Bin III - maximum arm velocity to zero arm velocity (at maximum flexion) (Figure 1). Trunk muscle onset and phasic offset for each subject were classified with respect to arm movement phase (BIN) and analyzed based upon group. Classification definitions were feedforward, (Bin I) which characterized anticipatory response and feedback (Bins II, III) that characterized the response to the perturbation. Torque generated along the vertical axes of center of pressure occurring during arm flexion was calculated using Kistler's published equations (Bleuse et al., 2005; Bleuse et al., 2002) and normalized with respect to body weight. The maximum normalized torque value (NormTzMx [Nmm/kg]) in performing the task was used as a measure of perturbation (Bleuse et al., 2005; Bleuse et al., 2002).

Statistical Analysis

All statistical analyses were completed using SPSS (IBM SPSS Statistics v20). Data were checked for normality and group mean and standard deviation calculated. Our participants used the dominant arm to perform the self-perturbation; therefore we categorized each trunk muscle as ipsilateral (Ips) or contralateral (Cont) relative to the upper extremity perturbation. ANOVA tests were performed to compare task performance variables (deltoid onset, arm motion onset, maximum arm rotation, maximum arm velocity and NormTzMx) between groups. Repeated measures MANOVA was used to assess the main effect of between subject factors (group), within-subject factors (side) and interaction effect (group x side) separately for the different trunk muscle timing variables (onset latency, phasic offset latency, phasic burst duration). Effect sizes were calculated for each repeated measures MANOVA (η^2 = square root () of sum of square_{between}/ sum of square_{total}) and interpreted as small (.01),

medium (.24), large (.37) or very large (.45) (Leech, Barrett, & Morgan, 2011; Vaske, Gliner, & Morgan, 2002). Post hoc analysis (ANOVA) were performed where appropriate using Bonferroni adjustments. In addition, separate Chi-square analyses were completed on the dichotomous feedforward vs. feedback onset activation status of each muscle group. To describe each participant's pattern of trunk muscle onset and offset with respect to arm motion (BINs), we used Chi square analysis to determine whether the trunk muscle activation patterns were different between groups. Alpha level was set at 0.05 for all primary comparisons and MDC values used to interpret meaningful differences between groups.

Results

Data Quality

In each group, approximately 7% of the onset and offset picks had to be disregarded due to inaccurate determination of the computerized onset of activity. Only those trials where errors in timing were clearly associated with interference of motion or heart rate artifact, baseline noise or where onset time was not physiologically tenable (-200 ms to 200 ms) (P. Marshall & Murphy, 2010) were discarded. This did not result in the loss of any entire dataset for a given individual and was consistent with data loss reported in prior studies. Data losses of 5% were reported by Marshall and Murphy (2003) (P. Marshall & Murphy, 2003) and Hodges (2001) (Hodges, 2001) in similar study designs.

Task Performance Variables

Several parameters of task performance were compared between the groups. The only statistical differences found were greater maximum arm velocity and NormTzMx in the control group ($p < 0.05$). (Table 3)

Trunk Muscle Timing: Traditional Approach

The groups did not have significantly different onset latencies (Figure 2) ($F = 0.68$, $p = 0.78$, $\eta = 0.51$). Within each group an average of 11/14 trunk muscles was activated in a feedforward manner. The main effect of side was significant, $F = 6.15$, $p = 0.00$, $\eta = 0.87$ and post hoc side comparison showed statistically significant differences for LES and TES ($p < 0.001$) muscles, and trends in the TrA/IO ($p = 0.019$), EO ($p = 0.024$). Within each group (Cont) side trunk muscles activated prior to the (Ips) side. There was no significant interaction between group and side, $F = 0.57$, $p = 0.77$, $\eta = 0.56$.

The groups did not differ in their phasic offset latency (Figure 3) ($F = 1.01$, $p = 0.47$, $\eta = 0.58$). However, the phasic offset did differ based on side ($F = 14.77$, $p = 0.00$, $\eta = 0.93$). Post hoc comparison for side showed statistically significant differences for 6 of 7 trunk muscles (exception RA, $p = 0.037$). Trunk muscles on (Cont) side deactivated prior to (Ips) side. There was no significant interaction between group and side, $F = 1.33$, $p = 0.27$, $\eta = 0.68$ for phasic offset.

The groups did not differ in phasic burst duration (Figure 4) ($F = 10.57$, $p = 0.70$, $\eta = 0.51$). However burst duration did differ based on side ($F = 10.57$, $p = 0.00$, $\eta = 0.91$). Post hoc side comparison showed statistically significant differences in burst durations for 6 of 7

trunk muscles (exception RA, $p = 0.038$). Contralateral muscles had shorter burst duration. There was no significant interaction between group and side, $F = 0.81$, $p = 0.58$, $\eta = 0.62$ for burst duration. Duration of co-contraction between control (393 ± 143) and NSLBP (407 ± 156) group was not statistically different, $F = 0.09$, $p = 0.76$, $\eta = 0.04$.

In addition to comparing group averages for muscle onset latency, we further analyzed our findings by categorizing each participant's mean muscle response time (average of 6 repetitions, over 14 muscles individually) as either under feedforward or feedback control. We then performed 14 Chi-square analyses (Table 4). Similar to the group means, none of the 14 Chi-square analyses showed statistical differences between the groups ($p > 0.05$).

Trunk Muscle Timing and Pattern Relative to Arm Motion

Figure 5 shows the activation and deactivation patterns of the trunk muscles with respect to the preparatory (Bin I), acceleration (Bin II) and deceleration (Bin III) phases of rapid arm flexion. As evident from the figure, all trunk muscles activated before the onset of arm movement and all demonstrated feedforward activation (were within the deltoid BIN defined as -150 to 50 ms post deltoid onset) with exception of mean activation time for the Ips TrA/IO and Cont RA in both the groups, and Isp RA in the NSLBP group. Also the deactivation patterns were similar between the groups: practically all the extensors turned OFF during arm acceleration (Bin II), whereas the abdominals turned OFF during arm deceleration (Bin III). The pattern also demonstrated consistently earlier activation and deactivation of the contralateral muscles. None of the Chi-square analyses were statistically significant ($p > 0.05$), further supporting other results that EMG activation and deactivation patterns were not significantly different between the groups (Table 5,6).

Discussion

The results of this study did not support our primary hypothesis that patients in the MCI subgroup of NSLBP would demonstrate statistically significant and meaningful delays in trunk muscle onset and offset, longer burst duration or a shorter duration of co-contraction. Trunk muscle timing patterns relative to extremity motion also failed to support this hypothesis.

We compared the task performance between our groups objectively on several parameters. We found no statistical difference between the groups for deltoid onset times, arm motion onset times, and maximum angular displacement. Despite that maximum arm velocity (and NormTzMx) was significantly lower in our NSLBP group than controls, it can still be considered fast based on the results reported by Hodges and Richardson (1999) (Hodges & Richardson, 1999). In fact, both studies confirm that the NSLBP group has a lower maximum arm velocity with respect to the control group; however, the maximum arm velocity of our NSLBP group ($588 \pm 74 \text{ deg/s}$) is greater than the maximum arm velocity of the control group ($514.9 \pm 56.9 \text{ deg/s}$) reported by Hodges and Richardson (1999) (Hodges & Richardson, 1999). We believe that the arm velocity was sufficient in both groups to ensure the need for feedforward activation of the trunk muscles, as supported by our data. Other research supports the hypothesis that lower arm velocity and NormTzMx should not have an effect on the initial time response of the trunk muscles (Bleuse et al., 2002).

Onset latency

Delayed activation of trunk muscles in patients with NSLBP has been a hallmark for an altered anticipatory postural control strategy during self-initiated perturbation tasks. Most of previous studies have demonstrated delayed onset latencies and lack of feedforward activation of local (TrA, IO) and some reported delays in global trunk muscles (EO, RA, ES) in patients with heterogeneous NSLBP (Hodges, 2001; Hodges, Moseley, Gabrielsson, & Gandevia, 2003; Hodges & Richardson, 1996; Mehta et al., 2010). These studies reported latency differences ranging from 12–164 ms in the TrA and IO, 15–68 ms in the EO and RA, and 3–89 ms for the ES and LM. However, we did not find evidence of significantly delayed or prolonged trunk muscle activation timing (onset latency) in our homogenous MCI subgroup with acute to subacute NSLBP. In addition, mean group difference for each trunk muscle did not exceed our MDC. Interestingly, we found a statistically significant earlier activation of contralateral trunk muscles within each group, with no interaction between side and group. Earlier activation of trunk muscle contralateral to arm movement has been previously reported (Allison, Morris, & Lay, 2008; P. Marshall & Murphy, 2010). Though muscles onsets were statistically different between sides within each group, the side differences did not exceed our MDC. We recognize that our MDC was determined using our healthy cohort and may represent an under or overestimation of measurement error in our NSLBP group. However, most of the trunk muscles (average of 11/14 trunk muscles) in our homogenous NSLBP group were activated prior to initiation of arm motion, which represents their activation prior to the perturbation itself. Our ICC values were similar to those reported by Marshall and Murphy (2003) for comparable abdominal muscle surface EMG placements (P. Marshall & Murphy, 2003). It is evident from our data that onset latency variability between individuals within both the patient and control groups is high. However, our data is consistent with data presented in other studies (Hodges & Richardson, 1996, 1997a; P. Marshall & Murphy, 2010). Both within and between subject variability is a recognized problem (Vasseljen, Fladmark, Westad, & Torp, 2009; Vasseljen et al., 2012) making standardization of protocol procedures and reporting of protocol measurement error all the more important.

We suspect that differences between our findings and those who found delayed activation in individuals with NSBLP can be attributed to several factors. First, even though activation timing differences reported in previous studies were statistically significant, they might not have exceeded measurement error, as the reported activation timing differences were small (Hodges & Richardson, 1996, 1997b, 1998). Second, the subjects in this study had acute or subacute NSLBP rather than recurrent or chronic back pain (Hodges & Richardson, 1996, 1998; Mehta et al., 2010; Silfies et al., 2009). It is plausible that the muscle latency impairment is a function of duration of symptoms. Given our subjects had pain for less than 90 days, the duration of pain may have been insufficient to elicit timing changes in the trunk muscles. However, most of our NSLBP subjects had prior episodes of low back pain and as such, other studies have suggested that the individuals with recurrent episodes, but no current symptoms, demonstrate onset latency delays in TrA and IO (Hodges, 2001; Hodges & Richardson, 1999). Third, we compared a specific subgroup of NSLBP who were identified clinically to have signs of MCI. This increases external validity of our study, as these patients would most likely receive motor control exercises, based on their clinical

presentation. It is possible that there is a poor relationship between muscle activation timing to a self-perturbation and clinical signs of MCI. However, according to the literature this subgroup has their primary impairment in trunk motor control (Delitto et al., 2012). Fourth, methodological and task differences may have accounted for the variation in the results. In this study, we used a custom LabVIEW program to identify muscle onsets automatically. No manual override was allowed which eliminated the possibility of biasing the data. The data loss due to difficulty in accurately determining onset time (approx. 7%) was similar too (Hodges, 2001; P. Marshall & Murphy, 2003) or less than a previously reported study (approx. 26%) (Gubler et al., 2010). Lastly, use of indwelling electrodes in the deep muscles (TrA, IO) of previous studies could be a source of differing results compared to our findings. While the use of indwelling electrodes for deeper muscles groups provides accurate information about several motor units surrounding the electrode, this signal may not be representative the entire muscle bulk. Our surface EMG placement for deep abdominal muscles (TrA/IO) represents a combination of the TrA and IO (not individual muscles). This placement has been validated (P. Marshall & Murphy, 2003) and prior studies have demonstrated delayed deep trunk muscle activation in patients with NSLBP (P. Marshall & Murphy, 2010; Mehta et al., 2010; Silfies et al., 2009).

Our results are not the only ones to challenge the findings from previous literature indicating delayed activation of superficial muscles in response to self-perturbation. Recently Gubler et al. (2010) and Brooks et al. (2012) reported that patients with chronic LBP did not show a delayed onset of the abdominal muscles (TrA, IO, EO, RA) during rapid arm movements (Brooks et al., 2012; Gubler et al., 2010). Additionally, they also found side difference and feedforward activity of abdominal muscles in their LBP group. While Gubler et al. (2010) used ultrasound imaging to identify the onset time (Gubler et al., 2010); this method has been validated and compared to EMG in previous studies (Mannion et al., 2008; Vasseljen et al., 2009; Westad, Mork, & Vasseljen, 2010).

Offset latency

Voluntary (reactive) postural adjustments occur in conjunction with an external or focal perturbation and provide efficient but not necessarily safe postural control (Frank & Earl, 1990). The results from the present study also did not support our hypothesis that the MCI subgroup of NSLBP would demonstrate delayed deactivation (offset latency). This suggests that the compensatory postural adjustment timing is not impaired in our homogenous subgroup of NSLBP. The findings from the present study do not agree with the previous studies that demonstrated delayed offset of trunk muscles (Radebold, Cholewicki, Panjabi, & Patel, 2000; Radebold et al., 2001). However, those investigators used a sudden unloading paradigm to test muscle offset times in patients with LBP. Thus it is plausible that the methodological and task differences account for the variation in the results.

Burst Duration and Duration of Co-contraction

The lack of difference between burst duration and duration of co-contraction between the groups further suggest that trunk neuromuscular control, related to muscle timing, was not significantly affected by participants' clinical signs and symptoms found in our MCI subgroup of NSLBP. This finding also conflicts with previous finding that demonstrated

shorter burst and co-contraction duration in patients with heterogeneous chronic NSLBP (Mehta et al., 2010). It may be that our subjects, who were still in an acute to subacute phase, did not yet develop adaptations to timing reported in those with chronic symptoms. Similar to the onset and offset latency data, it is plausible that methodological and subgroup differences mentioned earlier account for the variation in the results.

Trunk Muscle Activity Pattern

The novelty of this study was the expansion of the analysis of trunk muscle timing based upon the actual kinematics of the self-initiated perturbation. Our results from trunk muscle pattern analysis further demonstrated that there is no significant difference in the trunk muscle activation and deactivation timing pattern within each phase of the extremity motion. Both groups demonstrated a common and coordinated pattern of activation and deactivation. The trunk extensor muscle deactivation during arm acceleration phase followed by abdominal deactivation during the deceleration phase of arm flexion was expected, as the subjects in our protocol moved their arms through 160° of shoulder flexion causing a gradual reversal from a flexion and axial rotation perturbation to the trunk to one of extension as the arm decelerates to stop the motion. We assessed these data from the perspective of both mean group differences and by classification of each subject's individual muscle activations as either as feedforward or feedback, and these results further strengthen our conclusions.

Limitations

Although this study has several strengths, there are some limitations and consequently our results should be interpreted with respect to the following. Our sample size was relatively small and between subject variance was relatively large. The effect sizes calculated (η) for group differences across onset, offset and duration are considered very large ($\eta = 0.51$ – 0.58); however, we did not find statistically significant group differences which may potentially be a consequence of the large variability. Even if we found statistically significant differences, which might be achieved by recruiting more subjects thus potentially reducing variability, we would still interpret findings as not meaningful based upon the fact that the group differences do not exceed the MDC values for our protocol. Using trunk muscle timing parameters to investigate differences in trunk postural control strategies may be a limiting factor because of the large individual and between subject variation in activation patterns that, in turn, increase measurement error and makes timing variables less sensitive to detection of the differences between groups. Further, trunk muscle timing represents a portion of the postural response and even while timing patterning may be preserved, relative EMG amplitude changes of significance may occur within bursts or muscle synergies. Future studies should consider assessing muscle amplitude to complete the picture of postural control and more sensitive variables to study the trunk postural control strategies.

Conclusion

In summary, both the groups examined in this study performed the arm raising task similarly and yet no statistical or meaningful differences were found in timing in their postural

response. A majority of the trunk muscles within each group activated in a feedforward manner and their group patterns of activation and deactivation were coordinated and similar. Based on these findings, the subgroup of NSLBP with MCI determined by clinical examination did not demonstrate trunk muscle timing impairment during a self-initiated perturbation. These findings have direct implications for selection of therapeutic interventions for this subgroup and may explain, in part, why RCT of motor control exercises (vs. general trunk strengthening exercises) have not consistently demonstrated superiority in outcomes (Costa et al., 2009; Macedo, Maher, Latimer, & McAuley, 2009; Vasseljen & Fladmark, 2010) or the ability to alter timing impairments (Brooks et al., 2012; Hall, Tsao, MacDonald, Coppieters, & Hodges, 2009; Vasseljen et al., 2012). Further, given we reported the measurement error for all the trunk muscle timing parameters, the use of activation timing as a robust measure of trunk postural control is in question. We acknowledge, there could be differences between our data and other groups in the amplitude of muscle recruitment or the clinical history and therefore, future studies are warranted to explore the robustness of these findings across similar tasks.

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Figure 1.

Schematic representation of automatic determination of muscle onset, offset and kinematic phases (BIN I-III) of arm perturbation for an individual trial. The filtered EMG signal is displayed for all 14 muscles in red with individual onset and phasic offset times; Ips, ipsilateral; Cont, contralateral (with respect to dominant arm); TrA/IO, transversus abdominis/ internal oblique; EO, external oblique; EO2, external oblique 2; RA, rectus abdominis; LM, lumbar multifidus; LES, lumbar erector spinae; TES, thoracic erector spinae; Arm DIS, arm displacement; Arm VEL, angular velocity segmented into 3 phases:

Bin I - auditory trigger to onset of arm movement, Bin II - onset of arm movement to maximum arm velocity, and Bin III - maximum arm velocity to zero arm velocity at maximum arm flexion angle.

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Figure 2.

Mean and standard deviation of individual trunk muscle onset latencies by group. Y-axis represents onset latency in ms and X-axis represents trunk muscles (TrA/IO, transversus abdominis/ internal oblique; EO, external oblique; EO2, external oblique 2; RA, rectus abdominis; LM, lumbar multifidus; LES, lumbar erector spinae; TES, thoracic erector spinae). 0 ms represents anterior deltoid onset time. Red line at 50ms denotes feedforward cut off. Any trunk muscle onset latency below 50ms is considered feedforward activation and above 50ms is considered feedback activation. Yellow line represents the start of arm movement for NSLBP group and Green line represents the start of arm movement for Controls with respect to anterior deltoid onset.

Figure 3.

Mean and standard deviation for trunk muscle offset latencies by group. Y-axis represents trunk muscle offset latencies in milliseconds and X-axis represents trunk muscles (TrA/IO, transversus abdominis/ internal oblique; EO, external oblique; EO2, external oblique 2; RA, rectus abdominis; LM, lumbar multifidus; LES, lumbar erector spinae; TES, thoracic erector spinae)

Figure 4.

Mean and standard deviation for trunk muscle phasic burst duration by group. Y-axis represents phasic burst duration in milliseconds and X-axis represents trunk muscles (TrA/IO, transversus abdominis/ internal oblique; EO, external oblique; EO2, external oblique2; RA, rectus abdominis; LM, lumbar multifidus; LES, lumbar erector spinae; TES, thoracic erector spinae).

Figure 5.

Illustration of phases (BIN I [feedforward], II, and III [feedback]) and individual trunk muscle onset (black circles) and offset (white circles) times, by group mean and standard deviation. X-axis represents time in seconds. (A) Control (B) NSLBP. Sagittal component of the angular velocity was segmented into 3 phases: BIN I, BIN II/acceleration phase, and BIN III/deceleration phase; Deltoid Bin (–150ms to +50ms from deltoid onset); (TrA/IO, transversus abdominis/ internal oblique; EO, external oblique; EO2, external oblique2; RA, rectus abdominis; LM, lumbar multifidus; LES, lumbar erector spinae; TES, thoracic erector spinae).

Table 1Group Characteristics (Mean $\pm$ SD)

Variables	Control (n=21)	NSLBP (n=21)	p
Sex (male, female)	5M, 16 F	5M, 16 F	1.00
Age (yr)	32.5 $\pm$ 12.5	32.7 $\pm$ 13.1	0.96
Body mass index (kg/m ²)	23.6 $\pm$ 3.5	25.2 $\pm$ 4.0	0.17
Current pain (0–10)	NA	4.1 $\pm$ 1.8	
Worst pain, last 2 weeks (0–10)	NA	6.7 $\pm$ 1.8	
Duration of current symptoms (days)	NA	42.4 $\pm$ 33.8	
ODS (0–100)	NA	23.4 $\pm$ 7.9	
PSFS (0–10)	NA	4.1 $\pm$ 1.9	
FABQ, physical (0–30)	NA	14.6 $\pm$ 7.9	
FABQ, work (0–66)	NA	14 $\pm$ 10.9	

ODS= Oswestry Disability Scale (0–100%; a higher score signifies greater disability)

PSFS = Patient Specific Functional Scale (0–10, lower score signifies greater difficulty with the task)

FABQ= Fear Avoidance Belief Questionnaire (higher score signifies greater fear of re-injury)

Table 2

Test Retest Reliability of Trunk Muscle Timing Variables

	Onset Latency (ms)		Phasic Offset Latency (ms)		Phasic Burst Duration (ms)	
	ICC	MDC	ICC	MDC	ICC	MDC
Trunk muscles						
Ips_TrA/IO	.25 *	66	.87	89	.63	83
Cont_TrA/IO	.76	48	.86	78	.90	79
Ips_EO	.73	45	.90	58	.92	62
Cont EO	.75	34	.83	88	.93	61
Ips_EO2	.02 *	68	.89	72	.82	96
Cont_EO2	.61	34	.91	61	.83	93
Ips_RA	.85	38	.52	98	.89	74
Cont RA	.66	75	.82	62	.68	115
Ips_LM	.70	24	.64	65	.82	86
Cont_LM	.47	31	.64	44	.76	52
Ips_LES	.25 *	38	.62	95	.67	37
Cont_LES	.45	34	.91	38	.67	86
Ips_TES	.36	30	.85	83	.95	27
Cont_TES	.23 *	35	.95	49	.86	78

Ips = ipsilateral and Cont = contralateral determined with respect to dominant arm.

TrA/IO= transversus abdominis/ internal oblique

EO =external oblique

EO2= external oblique second placement

RA= rectus abdominis

LM, lumbar multifidus

LES= lumbar erector spinae

TES = thoracic erector spinae

ICC= Intraclass Correlation Coefficient

SEM = standard error of measurement

MDC= minimal detectable change

* Represents those muscles with very low between subject variability that effected the ICC calculations.

Table 3Comparison of Task Performance Parameters Between Groups (Mean $\pm$ SD)

	Control	NSLBP	p
Deltoid Onset (ms)	225 $\pm$ 58	214 $\pm$ 52	0.45
Arm Onset (ms)	314 $\pm$ 69	279 $\pm$ 64	0.09
Max arm rotation (deg)	168 $\pm$ 11	160 $\pm$ 17	0.11
Max arm velocity (deg/s)	661 $\pm$ 69	588 $\pm$ 74	0.00 *
Normalized TzMx (Nmm/Kg)	92 $\pm$ 54	54 $\pm$ 38	0.01 *

deg =degree of rotational motion of the arm with respect to the trunk

deg/s, degree/second of maximum arm velocity

Nmm = Newton millimeter

TzMx = the maximum vertical torque at the center of pressure

*
p values less than 0.05 represented by an

Chi Square Analysis to Compare Trunk Muscle Feedforward Patterns Between Groups Using Each Subject's Mean Response for Each Trunk Muscle base on Deltoid Muscle Onset.

Table 4

Group	IpsTrA/IO		ContTrA/IO		Ips EO		Cont EO		Ips EO2		Cont EO2		Ips RA		Cont RA	
	FF	FB	FF	FB	FF	FB	FF	FB	FF	FB	FF	FB	FF	FB	FF	FB
NSLBP	9	12	15	6	10	11	19	2	18	3	18	3	10	11	11	10
Control	10	11	18	3	15	6	15	6	17	4	14	7	15	6	14	7
p	0.75		0.25		0.11		0.11		0.67		0.14		0.11		0.34	

Group	Ips LM		Cont LM		Ips LES		Cont LES		Ips TES		Cont TES	
	FF	FB	FF	FB	FF	FB	FF	FB	FF	FB	FF	FB
NSLBP	16	5	18	3	16	5	20	1	15	6	20	1
Control	15	6	16	5	17	4	17	4	16	5	16	5
p	0.72		0.43		0.7		0.15		0.72		0.15	

Ips = ipsilateral and Cont = contralateral determined with respect to dominant arm.

TrA/IO= transversus abdominis/ internal oblique

EO =external oblique

EO2= external oblique second placement

RA= rectus abdominis

LM, lumbar multifidus

LES= lumbar erector spinae

TES = thoracic erector spinae

FF = feedforward muscle activation defined as occurring –150 to 50 ms post deltoid activation.

FB= feedback muscle activation defined as occurring greater than 50ms post deltoid activation.

Table 5

Chi Square Analysis to Compare Trunk Muscle Onset Patterns Between Groups Using Each Subject's Mean Response for Each Trunk Muscle based on Initiation of Arm Movement

Group	IpsTrA/IO		ContTrA/IO		Ips EO		Cont EO		Ips EO2		Cont EO2		Ips RA		Cont RA	
	BinI	BinII	BinI	BinII	BinI	BinII	BinI	BinII	BinI	BinII	BinI	BinII	BinI	BinII	BinI	BinII
NSLBP	12	9	15	6	15	6	20	1	18	3	18	3	13	8	13	8
Control	16	5	19	2	19	2	20	1	19	2	18	3	16	5	17	4
p	0.19		0.11		0.11		1		0.63		1		0.31		0.17	

Group	Ips LM		Cont LM		Ips LES		Cont LES		Ips TES		Cont TES	
	BinI	BinII	BinI	BinII	BinI	BinII	BinI	BinII	BinI	BinII	BinI	BinII
NSLBP	18	3	19	2	18	3	21	0	18	3	20	1
Control	21	0	21	0	21	0	21	0	21	0	21	0
p	0.07		0.14		0.07		1		0.07		0.31	

Ips = ipsilateral and Cont = contralateral determined with respect to dominant arm.

TrA/IO= transversus abdominis/ internal oblique

EO =external oblique

EO2= external oblique second placement

RA= rectus abdominis

LM, lumbar multifidus

LES= lumbar erector spinae

TES = thoracic erector spinae

Bin I represents the time frame between auditory trigger to onset of arm movement.

Bin II represents the time frame between onset of arm movement to maximum arm velocity.

Table 6

Chi Square Analysis to Compare Trunk Muscle Offset Patterns Between Groups Using Each Subject's Mean Response for Each Trunk Muscle based upon Arm Motion

Group	Ips TrA/IO		Cont TrA/IO		Ips EO		Cont EO		Ips EO2		Cont EO2		Ips RA		Cont RA	
	BinII	BinIII	BinII	BinIII	BinII	BinIII	BinII	BinIII	BinII	BinIII	BinII	BinIII	BinII	BinIII	BinII	BinIII
NSLBP	12	9	17	4	3	18	15	6	6	15	12	9	2	19	6	15
Control	9	12	17	4	5	16	10	11	2	19	10	11	4	17	5	16
p	0.35		1		0.43		0.11		0.11		0.53		0.37		0.72	

Group	Ips LM		Cont LM		Ips LES		Cont LES		Ips TES		Cont TES	
	BinII	BinIII	BinII	BinIII	BinII	BinIII	BinII	BinIII	BinII	BinIII	BinII	BinIII
NSLBP	19	2	20	1	17	4	21	0	11	10	18	3
Control	20	1	20	1	17	4	20	1	7	14	18	3
p	0.54		1		1		0.31		0.21		1	

Ips = ipsilateral and Cont = contralateral determined with respect to dominant arm.

TrA/IO= transversus abdominis/ internal oblique

EO =external oblique

EO2= external oblique second placement

RA= rectus abdominis

LM, lumbar multifidus

LES= lumbar erector spinae

TES = thoracic erector spinae

Bin II represents the time frame between onset of arm movement to maximum arm velocity.

Bin III represents maximum arm velocity to zero arm velocity at maximum arm flexion angle.