Published in final edited form as:
Man Ther. 2014 October ; 19(5): 425–432. doi:10.1016/j.math.2014.04.007.

Altered postural responses persist following physical therapy of general versus specific trunk exercises in people with low back pain

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Abstract

Interventions that target trunk muscle impairments in people with LBP have been promoted; however, the treatment effects on muscle activation impairments during postural tasks remain unclear. Thus, our objective was to evaluate the effects trunk stabilization vs. general strength and conditioning exercises on the automatic postural response in persons with chronic low back pain (LBP).

Fifty-eight subjects with chronic, recurrent LBP (n = 58) (i.e., longer than six months) were recruited and randomly assigned to one of two, 10-week physical therapy programs: stabilization (n = 29,) or strength and conditioning (n = 29). Pain and function were measured at 11 weeks and 6 months post-treatment initiation. To quantify postural following support surface perturbations, surface electrodes recorded EMG of trunk and leg muscles and force plates recorded forces under the feet, to calculate the center of pressure.

Both groups demonstrated significant improvements in pain and function out to 6 months. There were also changes in muscle activation patterns immediately post-treatment, but not at 6 months. However, changes in COP responses were treatment specific. Following treatment, the stabilization group demonstrated later onset of COP displacement, while the onset of COP displacement in the strengthening group was significantly earlier following treatment.

Despite two different treatments, clinical improvements and muscle activation patterns were similar for both groups, indicating that the stabilization treatment protocol does not preferentially improve treatment outcomes or inter-muscle postural coordination patterns for persons with LBP.

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Keywords

Low back pain (LBP); physical therapy; electromyography; posture

1. Introduction

Impaired automatic postural responses (APR) are associated with chronic low back pain (LBP) (Henry et al., 2006, Mok et al., 2004). APRs reflect the nervous system's ability to rapidly organize and execute multi-segmental, functionally-relevant muscle activation patterns in response to externally induced perturbations. Compared to APRs in healthy subjects, persons with LBP demonstrate delayed and reduced EMG activity (Radebold et al., 2000, Stokes et al., 2006a), delayed centers of pressure (CoP), and reduced CoP excursion (e.g., margin of stability) (Henry, Hitt, 2006) in sagittal plane perturbations (Newcomer et al., 2002), suggesting that persons with LBP rely less on a hip strategy (Henry, Hitt, 2006, Mok, Brauer, 2004). The hip strategy combines trunk and hip motion to generate shear forces resulting from torques at the hip joint rather than ankle joint in order to maintain equilibrium (Henry, Hitt, 2006). Subjects with LBP may be (Radebold, Cholewicki, 2000) reluctant to use a hip strategy in response to sagittal perturbations due to anticipated pain or increased trunk muscle activity associated with these large trunk movements. Thus, impaired APRs may contribute to LBP recurrence and thus should be addressed in treatment

In healthy persons lateral surface perturbations are characterized by initial activation of the tensor fascia latae muscle (a hip abductor which aids in stabilizing the pelvis) of the loaded leg (Henry et al., 1998). In contrast, persons with LBP have demonstrated delays in CoP onset during lateral perturbations (Henry, Hitt, 2006), which are also indicative of an impaired hip strategy and trunk stiffening. None of these studies specifically examined the treatment response of the APRs to lateral perturbations, which typically rely on a hip strategy (Henry, Fung, 1998).

Whether these impaired APRs are the cause or consequence of LBP is unknown, presenting a treatment challenge for clinicians. Physical therapists (PT) design treatment programs to ameliorate impaired APRs for people with LBP (e.g., O'Sullivan & Allison, 1997, Richardson et al., 1999), based on the assumption that there is a causal relationship between motor control impairments and LBP (Hodges et al., 1999, Tsao & Hodges, 2008). One program, the trunk stabilization exercise (STB) program, focuses on ameliorating motor control impairments by improving control and strength of deep trunk muscles (Richardson et al., 2004). It is unknown whether STB influences the motor control responses to perturbations and/or reduces LBP compared to general strength/conditioning treatment (STC) programs or which treatment has the greatest potential to improve pain/function and ameliorate motor control impairments in persons with chronic LBP.

To develop effective LBP treatment, we must clarify the relationship between LBP and motor control impairments; thus, the aim of this study was to determine whether impaired APRs could be improved using either STC or STB exercises. This is the first study to examine treatment's influence on the APR up to 6 months post-treatment. We predicted that STB treatment would result in increased trunk muscle activation and a multi-segmental

postural response to perturbations post-treatment. We also predicted that STC treatment would increase trunk muscle strength and activation, but less specifically than STB, resulting in a rigid whole body postural response.

2. Methods

2.1. Subjects

Fifty-eight subjects met the inclusion criteria which were chronic, recurrent (Von Korff, 1994) LBP ≥ 6 months with an acute flare-up (McGorry et al., 2000), and current employment or active as a full-time student or homemaker. Subjects were excluded if they had: disc herniation, neurological signs, spinal or lower extremity disease, conditions or surgery, balance or cardiovascular disorders, a current pregnancy, involvement in litigation because of the LBP problem, or received worker's compensation for the LBP.

Subjects participated in a laboratory protocol and then were randomly assigned to treatment based on a covariate adaptive randomization scheme, with gender, age and smoking status as covariates (Pocock & Simon, 1975). Treatment assignments were transmitted to the study coordinator and the treating PT, neither of whom were masked to treatment; all other personnel were masked to treatment assignment. Thirty-eight subjects successfully completed the 10-week treatment program and returned for laboratory testing at 11weeks (Figure 1). Of the 38 subjects, 13 subjects returned for the 6 month laboratory protocol. Assuming a Type I error rate of 5%, a sample size of 13 subjects/group would provide greater than 80% power to detect differences between groups of 40% in EMG amplitude immediately post-treatment and an additional 20% difference at the 6-month time point. Sample size was increased by 20% to account for potential loss to follow-up. All subjects provided written informed consent to participate in the protocol, which was approved by the local institutional review board.

2.2. Laboratory Protocol

Testing occurred prior to treatment (at week 0), immediately following 10 weeks of treatment (at week 11), and 6 months after treatment initiation. The Numeric Pain Index (NPI) (Stratford & Spadoni, 2001) and the Oswestry Disability Index (OSW) (Fritz & Irrgang, 2001) were used to assess pain and functional levels (Table 1).

Trunk strength was assessed using: the Sorenson test (Biering-Sorensen, 1984), the trunk curl test (Nelson & Nestor, 1988), and the leg-lowering test (Nelson & Nestor, 1988). Muscle activation patterns were recorded during surface perturbations using bipolar surface EMG, 1-cm silver, silver-chloride electrodes (Norotrodes, 2-cm inter-electrode distance; Myotronics, USA) placed over the contracted belly of the following muscles: bilateral erector spinae at the first (EST), third (ESP), and eighth (ET8) lumbar spinal segments; external oblique (EOS); internal oblique (IOS); rectus abdominis (RAB) at the umbilicus, and; the left tibialis anterior (TIB) and gastrocnemius medialis (GAS) muscles. Electrode placement was standardized based on anatomical landmarks and distances were recorded to ensure consistent inter-session placements. Skin impedance was maintained under 10 kΩ.

EMG signals were sampled at 1000 Hz, pre-amplified by 1000 at the skin's surface, and then amplified for a total amplification of 2000–10,000.

Subjects then stood on a moveable platform (Compumotor, Parker Hannifin Corp., USA) with arms hanging at their sides and with each foot on a separate force plate (AMTI OR6-1000 ©, AMTI, Inc., USA) (sampled at 1000 Hz). Subjects were instructed to maintain standing balance in response to the platform movements. Each perturbation consisted of a 9-cm, ramp-and-hold waveform with a duration of 400 ms, peak velocity of 43 cm/s, and peak acceleration of 127 cm/s^2. Any trials in which the subjects stepped were discarded and immediately repeated.

After 2 practice trials, subjects received 3 trials in each of 6 directions of perturbations (separated by 30-degree increments; Figure 2A), presented randomly at unpredictable intervals. Data from trials were averaged across each of the perturbation directions for final statistical analyses. The subjects' self selected stance width and toe out angle are reported in Table 1.

2.3. Physical Therapy Treatment Protocols

After the laboratory protocol, subjects were randomly assigned to one of two treatment groups: STC (n = 20, 11 female) and STB (n = 18, 9 female) and underwent 10 weeks of their respective treatments. Both treatment groups were statistically similar in age, weight, and height (Table 1).

The STC program contained general trunk strengthening and endurance exercises administered in 3 phases: 1) initial strengthening of trunk flexors and extensors in single plane movements, 2) trunk and lower-extremity stretching (as determined by the treating PT) as well as progression of trunk-strengthening exercises in multi-planar trunk movements, and 3) trunk-strengthening exercises under dynamic conditions (e.g., unstable support surface and in multiple planes). There was no specific focus on the TA or multifidus muscle during any of these exercises, and exercises were progressed to tolerance without exacerbating symptoms more than 2 points on the NPI. Subjects received a home program and education on the anatomy and care of the back.

The STB subjects received specific instruction and exercises to improve their control of deep trunk muscles in 3 phases: 1) independent activation of the deep trunk muscle system (TA/internal oblique, multifidus), 2) refinement of any particularly painful movement (subject specific) while maintaining activation of the deep trunk muscle system, and 3) activation of the deep trunk muscles during the activities of daily living. All subjects were required to complete the first phase of exercises before moving to the next phase, and to be included in post-testing.

2.4. Data Processing

Using Matlab software (Matlab, Natick, MA, USA), force plate data were low-pass filtered at 10 Hz. The center of pressure (COP) for each foot was calculated using recorded forces and moments (Winter, 1996). Net COP displacements were calculated in the medial–lateral (COP_{ML}). COP data for each subject were normalized to the distance of the subject's foot

separation (distance between the midpoint of the right and left heel) (Mientjes & Frank, 1999). The latency of initial displacement, and the latency and magnitude of the peak displacement for COP were also determined (Figure 2B). A criterion of the mean baseline (1 s of data prior to platform movement) plus 3 standard deviations (with manual override) was used to determine the latency of initial COP displacement.

EMG signals were band-pass-filtered at 35–200 Hz, baseline-corrected by subtracting the mean of the signal, and full-wave-rectified. The integrated protocol method was then used with an option for manual override to identify EMG activation onset (Allison, 2003). EMG onsets were defined as in the automatic phase if they occurred from 80–120 ms after perturbation onset for the trunk and 80–150 ms for the leg muscles, based on the time estimated for voluntary response latencies (Chan et al., 1979, Jacobs & Horak, 2007) (Figure 2B). Activation incidence was then defined as the percent of trials with an identifiable onset of muscle burst activity within the automatic phase across the 6 directions of translations.

In addition, amplitudes of EMG activation were generated by integrating the rectified EMG signals across 2 phases: baseline (−75 ms prior to perturbation onset) and automatic (as described above). To facilitate subject group comparisons, each muscle's integrated EMG amplitudes were normalized to that muscle's maximum amplitude identified from any direction of 6 perturbation directions and from either of the 2 phases. This perturbation-generated reference contraction was most plausible for normalization, because people with LBP may be unwilling or unable to generate a maximum voluntary contraction (Larivière et al., 2003).

2.5. Statistical Analysis

All analyses were performed using SAS 9.2 Software (SAS Institute Inc., USA). Subject characteristics (age, height, weight, and heel-to-heel stance widths) were compared using independent samples t-tests. Repeated measures analyses of variance were used to assess the within-subject effects of visit (week 0, week 11, and month 6) and between-group effects of treatment group (STB, STC) on NPI and OWS scores, trunk strength scores, onset latency and peak displacement of COP. An additional repeated-measures factor of phase (baseline or automatic) was included for analysis of muscle activation incidence and EMG amplitude for all perturbations. An overall repeated measures multiple analysis of variance was initially performed. The final interactions terms, which involved differences across muscles over time, were statistically significant ($p < 0.0001$), suggesting that the group by time interactions of interest differed for each muscle. Thus, the incidence and amplitude of muscle activations were analyzed separately for each muscle. Results of Little's MCAR test suggested that data was missing completely at random (Chisq=135.37, df=144, p=0.68); thus, the "PROC MIXED" procedure was used for all analyses. This procedure relies on a maximum likelihood-based approach, which estimates the parameter values that would maximize the probability of observing the data collected. In the event of missing variables, the likelihood for a given individual is based on non-missing variables, without imputation (Wolfinger & Chang, 1998). Consequently, the analysis is an intention-to-treat analysis, including all subjects in the study. P-values < 0.05 were considered significant and where

appropriate interactions occurred, post-hoc analyses were performed using the least squares means method.

3. Results

Almost 700 people were screened by clinical exam or interview for the study, resulting in a study cohort of 58 (Figure 1). While several patients dropped out prior to the 6 month time point, a posteriori testing demonstrated that these drop outs did not change the baseline characteristics of the remaining group (i.e., pre-treatment OSW scores for the remaining sub-group were not different from the original subject group ($F = 1.35$, $p = 0.53$).

Both STC and STB groups demonstrated similar and significant decreases in OSW disability (visit main effect, $F = 21.20$, $p < 0.001$) and NPI (visit main effect, $F = 7.18$, $p = 0.002$) scores at 11 weeks and 6 months, compared to pre-treatment scores. There were no significant differences between treatment groups in OSW (group main effect, $F = 0.15$, $p = 0.70$) or NPI (group main effect, $F = 2.70$, $p = 0.08$) scores across all time points (Table 2).

Both treatment groups demonstrated similar, significant increases in trunk strength and Sorenson test times (visit main effects, range: $F = 11.91–34.9$; $p < 0.001$) at week 11 post-treatment (Table 2). No significant differences between treatment groups were found on any of the trunk strength or endurance measures (three tests: group main effects, range: $F = 0.00–1.11$; $p = 0.29–0.99$).

3.1. EMG Amplitude

3.1.1 Baseline phase (−75-0 ms pre-perturbation)—Prior to treatment, subjects in both groups exhibited similar normalized integrated EMG across all muscles. After treatment, there were significant group-by-visit interaction effects for the following muscles: TIB, GAS, bilateral RAB, ESP, EST, left EOS, and right IOS (range $F = 4.0–5.7$, $p = 0.006–0.02$), along with significant simple visit effects for each group (range $F = 3.96–28.52$, $p < 0.05$ and $F = 4.58–10.72$, $p < 0.05$, for the STB and STC groups, respectively). The STB group demonstrated significant increases in EMG activity in the left EOS and GAS, as well as in the right RAB, IOS, and EST muscles in the baseline phase (Figure 3, Table 3). These increases persisted 6 months following treatment for each of these muscles. The right EOS and ESP muscles also demonstrated significant increases in amplitude at month 6 after treatment, compared to 0 and 11 week post-treatment values (Figure 3). There was also a significant decrease in left ESP muscle activation after treatment in the STB group. In contrast, the STC group demonstrated significant increases in muscle activation across the following muscles during the baseline phase: bilateral EOS, EST, and ESP, and left RAB, GAS, and TIB. In contrast to STB group, amplitude increases in the STC group all returned to pre-treatment values at 6-months after treatment, with the exception of the left GAS, whose increase was maintained at 6 months.

3.1.2 Automatic phase (80–120/150 ms post-perturbation for trunk/leg muscles, respectively)—Automatic phase EMG activation in both groups showed similar EMG amplitudes prior to treatment; however, each presented a unique response after treatment. There were significant group-by-visit interactions for the following muscles: right

RAB, EOS, and ESP (range: F = 5.9–23.0, $p < 0.001$) with significant simple visit effects for the STB group (F = 7.18 and 18.27, $p < 0.05$, for each muscle respectively) and STC group (F = 5.81, $p = 0.005$ for the right RAB muscle). The STB group demonstrated increased EMG amplitude of the right EOS and RAB at week 11 (Figure 3, Table 3), and the EMG amplitude returned to pre-treatment values at month 6 after treatment. The right ESP muscle demonstrated increased activation at month 6, compared to the week 0 and week 11 values. Conversely, the STC group demonstrated significant decreases in EMG amplitude of the right RAB muscle post-treatment, which was observed at 6 months also (Figure 3).

3.2. Activation Incidence

During the automatic phase, activation incidence of the GAS muscle decreased at week 11 post-treatment in only the STB group (group-by-visit interaction: F = 3.67, $p = 0.03$). Visit main effects also indicated similar decreases in activation incidence in: left and right IOS and RAB muscles, and the left ESP muscle (visit effects: range: F = 4.45–17.31, $p < 0.01$) in both the STB and STC groups. Activation incidence was similar to post-treatment values across both treatment groups 6 months post-treatment for the left RAB muscle. However, activation incidence increased toward baseline values for the left ESP muscle. The activation incidence of the left and right IOS, and the right RAB muscle continued to decrease from week 11 to month 6 post-treatment, compared to pre-treatment values for both treatment groups.

3.3. Center of Pressure (COP)

The onset of COP displacement was similar between both groups pre-treatment, however, after receiving treatment, subjects responded differently, depending on the type of treatment (group-by-visit interaction: F = 11.1, $p < 0.001$). In the STB group, onset of COP displacement occurred significantly later at week 11 and persisted to month 6 after treatment (simple visit effect: F = 3.43, $p = 0.04$; Figure 4). The STC group's onset of COP displacement occurred significantly earlier post-treatment, but returned to pre-treatment values at month 6 (significant simple visit effect: range: F= 8.51, $p < 0.0001$; Figure 4).

The amplitude of COP displacement was similar between the STC and STB groups before treatment (group main effect: F = 0.01, $p = 0.9$) and following treatment, the COP displacement decreased across treatment groups (visit main effect: F = 24.08, $p < 0.001$; Figure 4) at week 11 and month 6 after treatment and remained reduced at 6 months post-treatment.

4. Discussion

General (STC) and specific (STB) programs are associated with changes in APR muscle activation patterns. STB subjects increased abdominal EMG amplitudes from baseline through the automatic-phase, decreased automatic-phase activation incidence in abdominal muscles, and had a later onset and reduced peak amplitude of COP displacement. This suggests that STB subjects modulated abdominal muscle perturbation responses from an activated baseline state rather than recruiting acute abdominal muscle burst activity from a quiescent state. Post-treatment, STB subjects adopted a pattern of stiffening the abdominal

region, resulting in a multi-segmental response which persisted up to 6 months post-treatment.

Conversely, post-treatment, the STC group increased EMG amplitudes across the abdominal, paraspinal, and leg muscles at baseline, but not during the automatic-phases following the perturbation. The STC group demonstrated decreased automatic-phase incidence of abdominal muscle activation, and earlier onset and reduced peak amplitude of COP displacement. Thus, suggesting that the STC group also attempted to modulate perturbation-related muscle activation from an activated baseline state across most muscles tested, rather than isolating abdominal muscles.

Neuromuscular measures suggest that neither treatment promoted healthy patterns of APRs; rather, subjects' response strategy remained impaired post-treatment (Henry, Hitt, 2006, Jacobs et al., 2011). Both groups used APRs with increased baseline EMG amplitude and a decrease in the activation incidence. Compared to healthy subjects, persons with LBP who were not in an active pain episode demonstrated: (1) increased baseline EMG amplitudes of the RAB, ESP, and GAS muscles prior to perturbation onset, and (2) fewer automatic-phase activations at the RAB, IOS, EOS, and ESP (Jacobs, Henry, 2011). However, the current study did not directly measure muscle activation from deep abdominal muscles (e.g., transversus abdominis and multifidus).

Persons with LBP demonstrated inhibited hip strategy in response to perturbations (Claeys et al., 2011, Mok, Brauer, 2004) and increased baseline EMG amplitude prior to perturbations (Jacobs, Henry, 2011, Stokes et al., 2006b). Reduced hip strategy may minimize forces and movement about the trunk (Jacobs, Henry, 2011), contribute to trunk-stiffening responses, and decrease overall trunk stability during perturbations (Henry, Hitt, 2006, Ishida et al., 2008) in persons with LBP. This is supported by increased GAS muscle activation across groups at both 11 weeks and 6 months post-treatment and reductions in both groups' COP peak amplitude displacement, suggesting a decrease in overall postural stability in both groups after treatment. Henry and colleagues (2006) demonstrated that reduced stability in persons with LBP suggests that they may be unable to produce adequate forces to restore equilibrium, possibly putting patients at risk for further injury or recurrence (Henry, Hitt, 2006).

5. Conclusion

Overall, instead of promoting normal APRs, both treatments promoted stiffening strategies, despite improving patient outcomes. This stiffening strategy may reinforce pathological movement patterns and further contribute to recurrence of LBP (Jacobs & Horak, 2007). A recent pain adaptation model (Hodges & Tucker, 2011) suggests that redistributing muscle activation results in rigid coordination of movement, representing an attempt to protect the injured site. Coordination changes may have short-term benefits to reduce pain but potential, long-term negative consequences (e.g., increased load, decreased movement, and decreased variability) (Hodges & Tucker, 2011). Patterns of muscle activation observed in this study suggest that clinical interventions should focus on APRs that address changes in central motor programming associated with LBP.

Acknowledgements

The authors wish to thank Drs. Jesse Jacobs and Sandra Oster for their valued comments on this manuscript. This study was supported by the National Institutes of Health NIH2R01HD040909 (PI: Henry SM).

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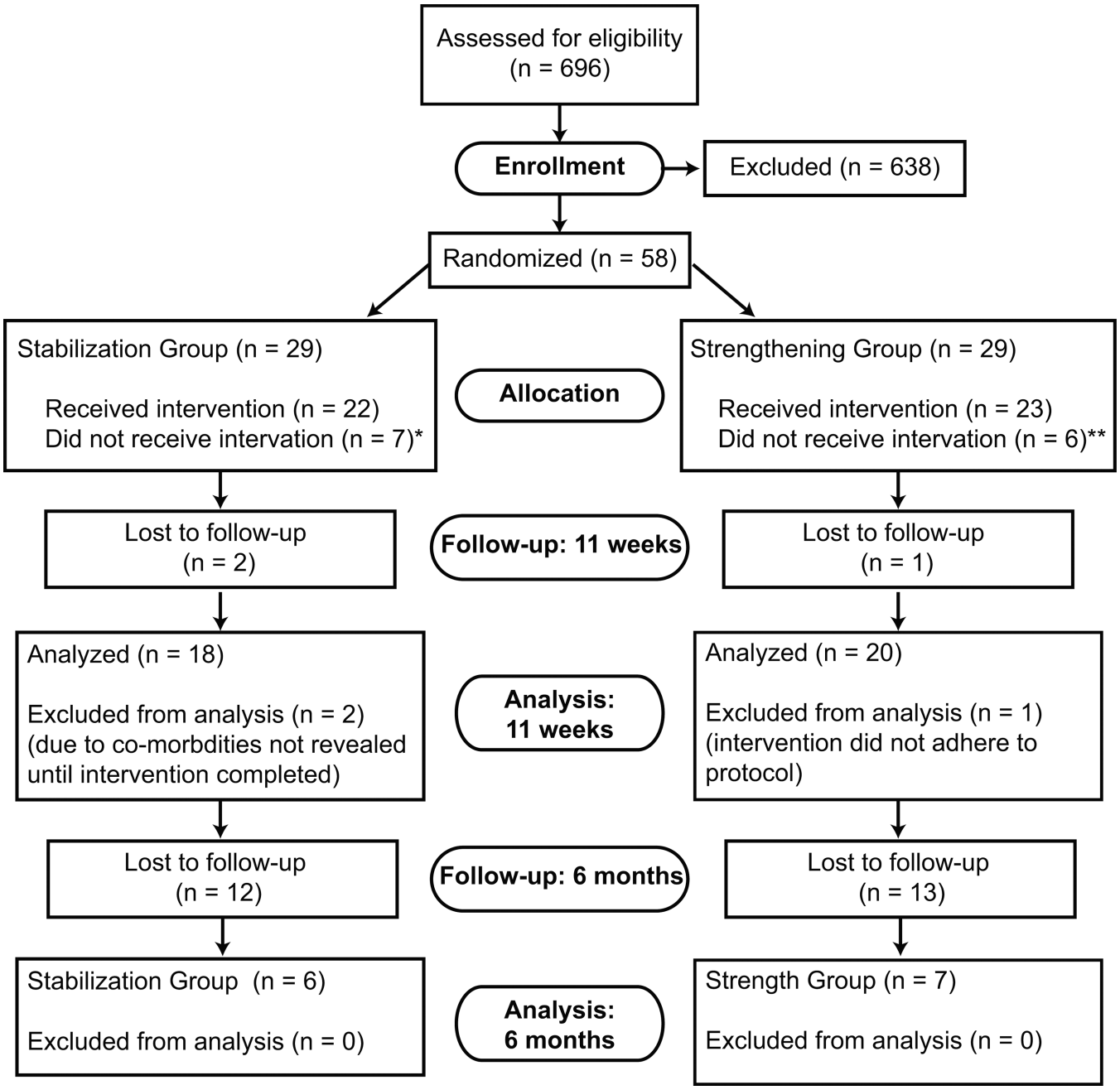

FIGURE 1.

Flow of subjects through recruitment, randomization and stage at which subjects were lost to follow-up for the STB group (*5 unable to schedule 10 consecutive appointments, 1 workers compensation claim, and 1 injured knee sustained outside of treatment) and STC group (**5 unable to schedule 10 consecutive appointments, and 1 broken collarbone sustained outside of treatment).

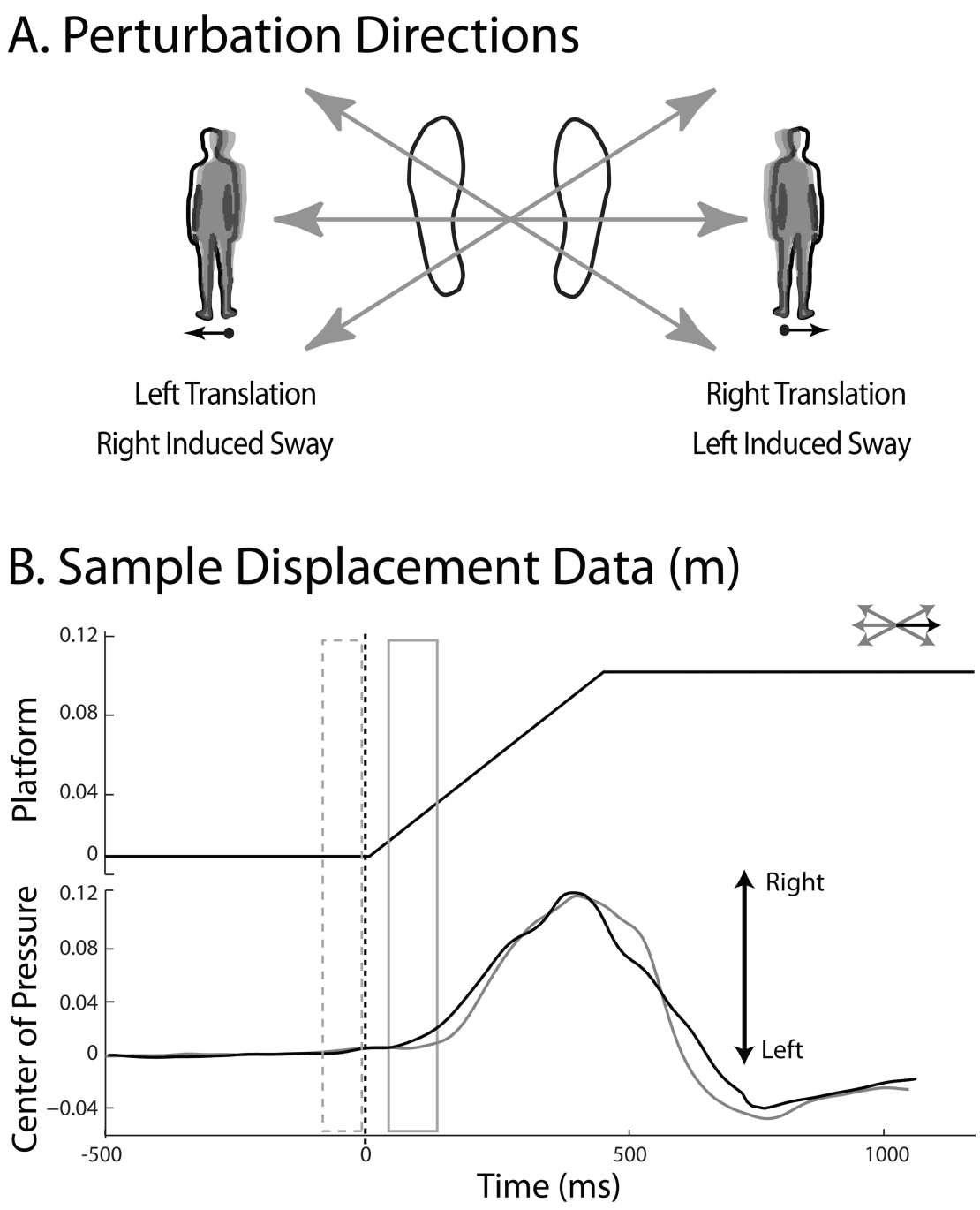

FIGURE 2.

Perturbation Directions and representative data. A) Schematic showing the 6 directions of translations with their induced sway direction, and; B) Representative platform displacement (upper) and net medio-lateral COP displacement (lower) for the STB (grey line) and STC (black line) during the baseline (grey dashed line box) and automatic (solid grey line) box), phases.

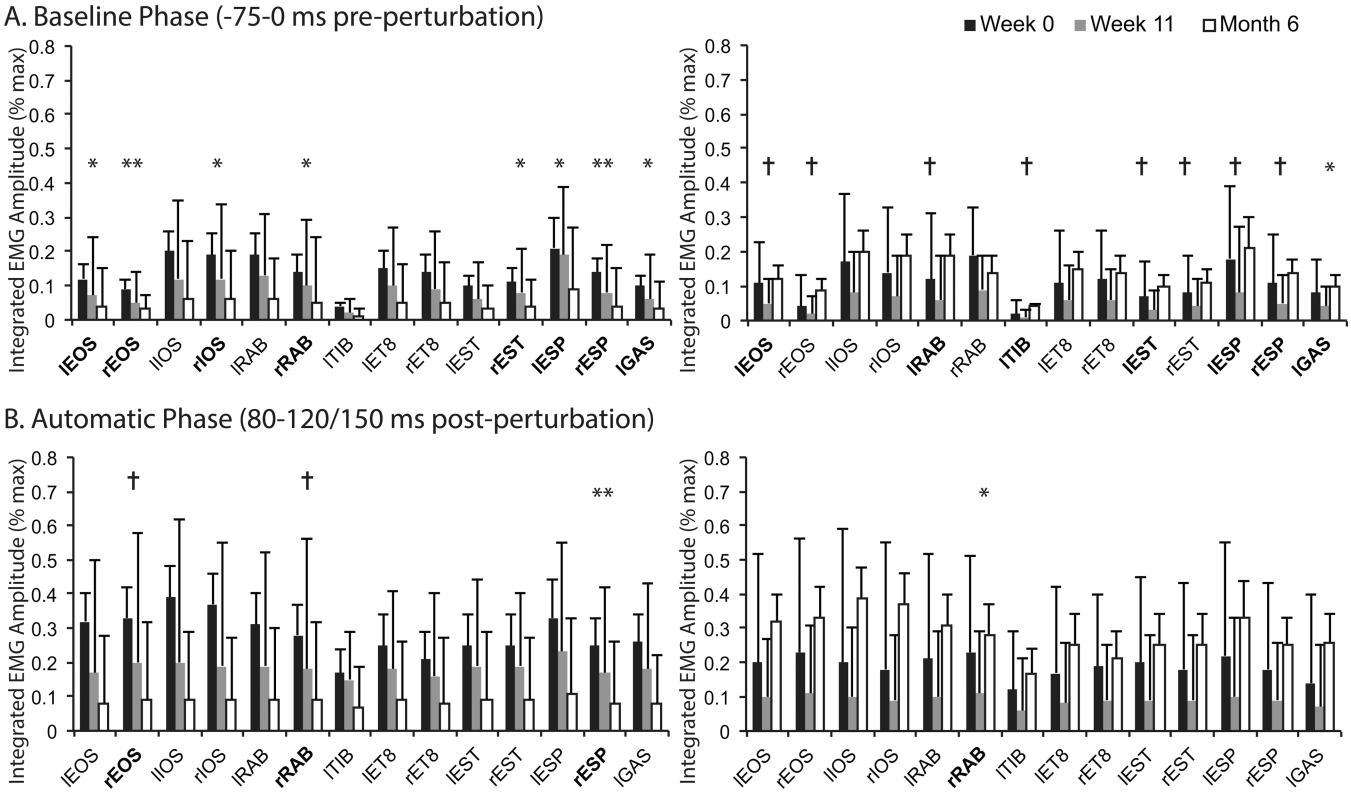

FIGURE 3.

Pre-/post-treatment changes in mean, normalized, integrated EMG activity from week 0 to week 11 and month 6. Results are presented for (A) baseline and (B) automatic phases. Increases from week 0 to week 11 are denoted with black fill and decreases are denoted with grey fill. Changes that continue from week 11 to month 6 are denoted by "#" signs. Increases from week 0 to month 6 are denoted with black outline and decreases are denoted with grey outline. Grey dotted circles represent no significant group-by-visit effects for that muscle. See Table 3 for further information.

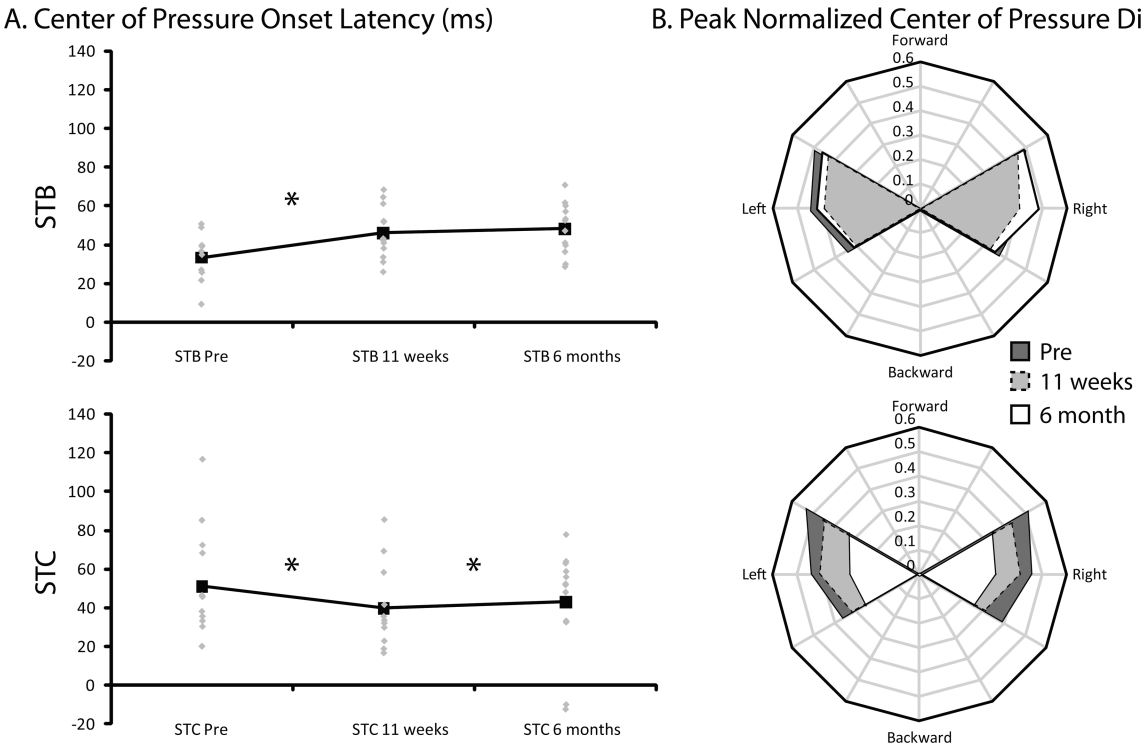

FIGURE 4.

Mean center of pressure onset latency (ms) (A) and peak normalized center of pressure displacement (arbitrary units) (B) for the STB (upper) and STC (lower) groups during the pre-treatment and 11-weeks and 6-months post-treatment visits. In (A) grey diamonds reflect means individual subjects, black squares indicate overall group means, and asterisks indicate significant group-by-visit effects. In (B), dark grey filled boxed indicates pre-treatment, while dashed light grey and white boxes indicate post-treatment (11 weeks and 6 months, respectively) displacements for each group.

TABLE 1

Comparison of subject baseline demographics, mean (standard deviation)

	STB*		STC†	
Number of Subjects	18		20	
Number of Females	9		11	
Age	40.1	(8.3)	41.2	(7.9)
Weight (kg)	75.2	(13.2)	76.4	(13.1)
Height (m)	1.7	(0.1)	1.7	(0.1)
Self-selected stance width (cm)	21.8	(3.1)	21.9	(3.9)
Self-selected foot angle (deg)	101.9	(6.6)	104.6	(9.4)
Oswestry Disability Index (/50)	22.3	(9.7)	20.1	(7.8)
Numerical Pain Index (/10)	3.7	(1.7)	3.2	(1.5)

*Stabilization exercise treatment program

†General trunk strength and conditioning exercise treatment program

TABLE 2

Difference in mean function and pain scores and trunk strength tests. Mean (95% CI) results are presented for each treatment group (stabilization and strength/conditioning) and visit (week 0, week 11, and month 6).

	Time	STB*-STC † mean difference
Oswestry Disability Index (%)	Week 0	1.8 (−3.6, 7.2)
	Week 11	−1.2 (−6.8, 4.4)
	Month 6	0.4 (−10.2, 11.0)
Numeric Pain Rating (/10)	Week 0	−0.9 (−1.9, 0.0)
	Week 11	0.4 (−0.9, 1.7)
	Month 6	−1.7 (−4.0, 0.7)
Sorenson Test (s)	Week 0	15.5 (−21.1, 52)
	Week 11	12.0 (−24.3, 48.3)
Trunk-curling Test (/4)	Week 0	0.0 (−0.6, 0.6)
	Week 11	0.0 (−0.2, 0.2)
Leg-lowering Test (/2)	Week 0	0.1 (−0.2, 0.4)
	Week 11	0.2 (−0.1, 0.4)

*Stabilization exercise treatment program

†General trunk strength and conditioning exercise treatment program

TABLE 3

Outcome measures for normalized, integrated EMG amplitude at week 0, 11, and month 6 for the stabilization and strength/conditioning treatment groups.

Analysis /Phase	Muscle	STB* Week 0	STC† Week 0	STB* Week 11	STC† Week 11	STB* Month 6	STC† Month 6
Outcomes**							
Baseline	lEOS	0.12 ± 0.04	0.11 ± 0.12	0.07 ± 0.17	0.05 ± 0.07	0.04 ± 0.11	0.12 ± 0.04
	rEOS	0.09 ± 0.03	0.04 ± 0.09	0.05 ± 0.09	0.02 ± 0.05	0.03 ± 0.04	0.09 ± 0.03
	lIOS	0.20 ± 0.06	0.17 ± 0.20	0.12 ± 0.23	0.08 ± 0.12	0.06 ± 0.17	0.20 ± 0.06
	rIOS	0.19 ± 0.06	0.14 ± 0.19	0.12 ± 0.22	0.07 ± 0.12	0.06 ± 0.14	0.19 ± 0.06
	lRAB	0.19 ± 0.06	0.12 ± 0.19	0.13 ± 0.18	0.06 ± 0.13	0.06 ± 0.12	0.19 ± 0.06
	rRAB	0.14 ± 0.05	0.19 ± 0.14	0.10 ± 0.19	0.09 ± 0.10	0.05 ± 0.19	0.14 ± 0.05
	lTIB	0.04 ± 0.01	0.02 ± 0.04	0.02 ± 0.04	0.01 ± 0.02	0.01 ± 0.02	0.04 ± 0.01
	lET8	0.15 ± 0.05	0.11 ± 0.15	0.10 ± 0.17	0.06 ± 0.10	0.05 ± 0.11	0.15 ± 0.05
	rET8	0.14 ± 0.05	0.12 ± 0.14	0.09 ± 0.17	0.06 ± 0.09	0.05 ± 0.12	0.14 ± 0.05
	lEST	0.10 ± 0.03	0.07 ± 0.10	0.06 ± 0.11	0.03 ± 0.06	0.03 ± 0.07	0.10 ± 0.03
	rEST	0.11 ± 0.04	0.08 ± 0.11	0.08 ± 0.13	0.04 ± 0.08	0.04 ± 0.08	0.11 ± 0.04
	lESP	0.21 ± 0.09	0.18 ± 0.21	0.19 ± 0.20	0.08 ± 0.19	0.09 ± 0.18	0.21 ± 0.09
	rESP	0.14 ± 0.04	0.11 ± 0.14	0.08 ± 0.14	0.05 ± 0.08	0.04 ± 0.11	0.14 ± 0.04
	lGAS	0.10 ± 0.03	0.08 ± 0.10	0.06 ± 0.13	0.04 ± 0.06	0.03 ± 0.08	0.10 ± 0.03
Automatic Phase	lEOS	0.32 ± 0.08	0.20 ± 0.32	0.17 ± 0.33	0.10 ± 0.17	0.08 ± 0.20	0.32 ± 0.08
	rEOS	0.33 ± 0.09	0.23 ± 0.33	0.20 ± 0.38	0.11 ± 0.20	0.09 ± 0.23	0.33 ± 0.09
	lIOS	0.39 ± 0.09	0.20 ± 0.39	0.20 ± 0.42	0.10 ± 0.20	0.09 ± 0.20	0.39 ± 0.09
	rIOS	0.37 ± 0.09	0.18 ± 0.37	0.19 ± 0.36	0.09 ± 0.19	0.09 ± 0.18	0.37 ± 0.09
	lRAB	0.31 ± 0.09	0.21 ± 0.31	0.19 ± 0.33	0.10 ± 0.19	0.09 ± 0.21	0.31 ± 0.09
	rRAB	0.28 ± 0.09	0.23 ± 0.28	0.18 ± 0.38	0.11 ± 0.18	0.09 ± 0.23	0.28 ± 0.09
	lTIB	0.17 ± 0.07	0.12 ± 0.17	0.15 ± 0.14	0.06 ± 0.15	0.07 ± 0.12	0.17 ± 0.07
	lET8	0.25 ± 0.09	0.17 ± 0.25	0.18 ± 0.23	0.08 ± 0.18	0.09 ± 0.17	0.25 ± 0.09
	rET8	0.21 ± 0.08	0.19 ± 0.21	0.16 ± 0.24	0.09 ± 0.16	0.08 ± 0.19	0.21 ± 0.08
	lEST	0.25 ± 0.09	0.20 ± 0.25	0.19 ± 0.25	0.09 ± 0.19	0.09 ± 0.20	0.25 ± 0.09
	rEST	0.25 ± 0.09	0.18 ± 0.25	0.19 ± 0.21	0.09 ± 0.19	0.09 ± 0.18	0.25 ± 0.09

Analysis /Phase	Muscle	STB* Week 0	STC† Week 0	STB* Week 11	STC† Week 11	STB* Month 6	STC† Month 6
	lESP	0.33 ± 0.11	0.22 ± 0.33	0.23 ± 0.32	0.10 ± 0.23	0.11 ± 0.22	0.33 ± 0.11
	rESP	0.25 ± 0.08	0.18 ± 0.25	0.17 ± 0.25	0.09 ± 0.17	0.08 ± 0.18	0.25 ± 0.08
	lGAS	0.26 ± 0.08	0.14 ± 0.26	0.18 ± 0.25	0.07 ± 0.18	0.08 ± 0.14	0.26 ± 0.08
Change from Week 0 levels #							
Automatic Phase	lEOS			0.015 (−0.02,0.051)	0.009 (−0.03,0.05)	0.030 (−0.02,0.07)	0.016 (−0.03,0.06)
	rEOS			0.046 (−0.001,0.09)	−0.009 (−0.05,0.03)	−0.115 (−0.16,−0.07)	0.045 (−0.0001,0.09)
	lIOS			0.033 (−0.04,0.07)	0.023 (−0.01,0.05)	−0.020 (−0.06,0.02)	−0.057 (−0.12,0.01)
	rIOS			−0.011 (−0.001,0.01)	−0.011 (−0.04,0.02)	0.074 (0.02,0.13)	0.058 (0.01,0.10)
	lRAB			0.022 (−0.01,0.06)	0.023 (−0.01,0.06)	−0.031 (−0.07,0.01)	−0.022 (−0.08,0.03)
	rRAB			0.110 (0.06,0.16)	−0.051 (−0.08,−0.02)	−0.035 (−0.10,0.03)	0.012 (−0.03,0.05)
	lTIB			−0.034 (−0.06,−0.01)	−0.026 (−0.06,0.003)	0.031 (0.01,0.06)	0.007 (−0.02,0.04)
	lET8			−0.020 (−0.06,0.01)	0.007 (−0.02,0.04)	0.049 (0.02,0.08)	0.003 (−0.04,0.05)
	rET8			0.034 (−0.01,0.07)	−0.008 (−0.04,0.02)	0.053 (0.01,0.10)	−0.004 (−0.05,0.04)
	lEST			−0.0003 (−0.04,−0.04)	0.012 (−0.02,0.05)	0.003 (−0.04,0.04)	−0.035 (−0.07,0.004)
	rEST			−0.032 (−0.06,−0.003)	−0.056 (−0.09,−0.02)	0.101 (0.06,0.14)	0.014 (−0.03,0.06)
	lESP			0.003 ± 0.14 (−0.03,0.04)	0.003 (−0.04,0.05)	−0.061 (−0.11,−0.01)	−0.167 (−0.26,−0.08)
	rESP			0.021 (−0.003,0.05)	−0.021 (−0.06,0.02)	0.067 (0.02,0.12)	−0.055 (−0.11,−0.002)
	lGAS			−0.0004 (−0.03,0.03)	0.004 (−0.02,0.03)	−0.027 (−0.07,0.01)	−0.005 (−0.04,0.03)

*Stabilization exercise treatment program

†General trunk strength and conditioning exercise treatment program

**Values are mean ± SD

#Values are mean (95% confidence interval)

§Week 11 compared to pre-treatment and month 6 compared to week 11