Spine

SPINE Volume 37, Number 13, pp 1101–1108

RANDOMIZED TRIAL

Effect of Core Stability Exercises on Feed-Forward Activation of Deep Abdominal Muscles in Chronic Low Back Pain

A Randomized Controlled Trial

Ottar Vasseljen, PhD, PT,* Monica Unsgaard-Tøndel, MSc, PT,* Christian Westad, PhD,† and Paul Jarle Mork, PhD†

Study Design. A randomized controlled trial.

Objective. To investigate feed-forward activation or timing of abdominal muscle activation in response to rapid shoulder flexion after 8 weeks with core stability exercises, sling exercises, or general exercises in chronic nonspecific low back pain (LBP) patients.

Summary of Background Data. Delayed onset in abdominal muscles has been associated with LBP. Low load exercises to volitionally activate the transversus abdominis were introduced to restore trunk muscle activation deficits. More forceful co-contraction exercises have been advocated by others. This study explored whether abdominal muscle onset changed after low-load core stability exercises, high-load sling exercises, or general exercises.

Methods. Subjects (N = 109) with chronic nonspecific LBP of at least 3 months' duration were randomly assigned to 8 weekly treatments with low-load core stability exercises, high-load stabilizing exercises in slings, or general exercises in groups. Primary outcome was onset recorded bilaterally by m-mode ultrasound imaging in the deep abdominal muscles in response to rapid shoulder flexion.

Results. No or small changes were found in onset after treatment. Baseline adjusted between group differences showed a 15 ms (95% confidence interval [CI], 1–28; $P = 0.03$) and a 19 ms (95% CI, 5–33; $P < 0.01$) improvement with sling relative to core stability and general exercises, respectively, but on 1 side only. There was no association between changes in pain and onset over the intervention period ($R^2 \leq 0.02$).

Conclusion. Abdominal muscle onset was largely unaffected by 8 weeks of exercises in chronic LBP patients. There was no association between change in onset and LBP. Large individual variations in activation pattern of the deep abdominal muscles may justify exploration of differential effects in subgroups of LBP.

Key words: ultrasonography, neurophysiological recruitment, clinical trial, motor control. **Spine 2012;37:1101–1108**

From the *Department of Public Health and General Practice, Faculty of Medicine, Norwegian University of Science and Technology, Trondheim, Norway; and †Department of Human Movement Science, Faculty of Social Science and Technology Management, Norwegian University of Science and Technology, Trondheim, Norway.

Acknowledgment date: June 29, 2011. First revision date: September 30, 2011. Acceptance date: November 7, 2011.

The manuscript submitted does not contain information about medical device(s)/drug(s).

Foundation funds were received in support of this work.

No benefits in any form have been or will be received from a commercial party related directly or indirectly to the subject of this manuscript.

Address correspondence and reprint requests to Ottar Vasseljen, PhD, PT, Department of Public Health and General Practice, Faculty of Medicine, Norwegian University of Science and Technology, P.b. 8905, MTFS, 7491 Trondheim, Norway; E-mail: ottar.vasseljen@ntnu.no

DOI: 10.1097/BRS.0b013e318241377c

Low back pain (LBP) may affect motor control of trunk muscles that regulates spinal movements and stability.[1] One indicator of motor control for the low back is timing or onset of abdominal muscle activation when spinal stability is threatened by destabilizing forces. In response to expected postural perturbation, this activation is in pain-free subjects shown to occur in a feed-forward manner, that is, anticipatory muscle activation that occurs prior to the actual perturbation of balance.[1,2]

Core stability exercises (CSEs) in LBP rehabilitation have become popular due to observed changes in abdominal muscle activation patterns in the presence of LBP. Delayed feed-forward activation of deep abdominal muscles in response to postural perturbations induced by rapid shoulder flexion has been observed in chronic and recurrent LBP patients.[3–6] Because deep trunk muscles contribute to stabilization of the spine,[7] it is hypothesized that a deficit in feed-forward activation increases the susceptibility of injury to spinal structures. CSE, consisting of low-load exercises emphasizing voluntary and isolated control of deep trunk muscles, are assumed to help restore trunk muscle deficits in LBP.[8,9] However, evidence of clinical effects on pain and disability of CSE compared with other exercises is sparse, showing conflicting or at best small effects.[10–13]

Few studies have investigated the effect of CSE on feed-forward activation pattern. Shortening in onset latency in response to rapid shoulder flexion was found after 1 session with isolated transversus abdominis (TrA) exercise but significant effects were also observed after sit-up exercise.[14] The same research group found that onset improvement gained during

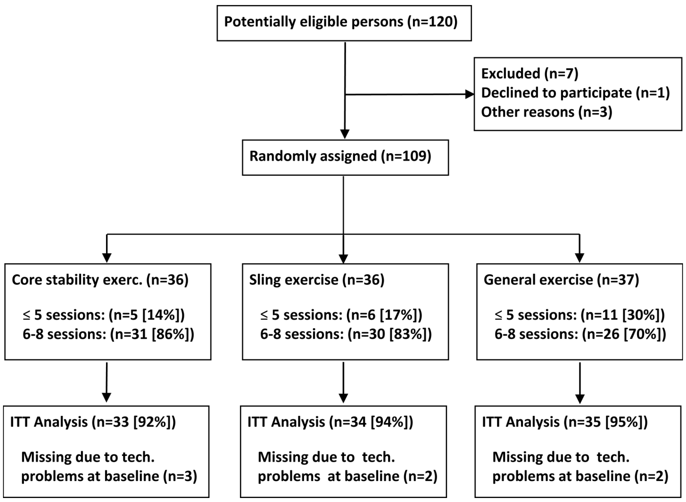

Figure 1. Treatment compliance and number of subjects included in the intention to treat (ITT) analysis. The number of subjects with 6 treatments or more who also showed up at post-test assessment was 31, 30, and 26 in the core stability exercise, sling exercise, and general exercise groups, respectively. Exerc. indicates exercise; tech, technical.

the initial 4 weeks with TrA exercise was partially maintained in a subset of the subjects at 6 months' follow-up.[15] However, improvement in onset did not correlate with changes in pain and function. Improvement in abdominal muscle latency time (feed-forward activation) was observed at 1-year follow-up after both specific LBP exercises (Swiss ball exercise) and home-based general exercises (GEs) but did not demonstrate any interaction effect between time and type of treatment or exercise.[16] The purpose of this randomized controlled trial was to investigate the effect of 3 different exercise interventions on feed-forward activation or timing of deep abdominal muscles in chronic LBP patients. Patients were randomized to CSE, sling exercise (SE), or GE.

MATERIALS AND METHODS

Settings and Participants

Subjects (n = 109) were recruited from general practitioners or physical therapist (29 of 109) or by announcement at a regional hospital (80 of 109; Figure 1) in 2006–2007. Inclusion criteria were ages 18 to 60 years, chronic nonspecific LBP for 3 months or more, and pain score of 2 or more on a 0 to 10 Numeric Rating Scale (NRS). Subjects were excluded in case of radiating pain below the knee or neurological signs from nerve root compression, former back surgery, systemic or widespread pain, adiposity preventing ultrasound imaging, pregnancy, psychiatric disease, sick leave for 1 year or more, receiving disability pension, unsolved social security or insurance problems, or insufficient comprehension of the native language. The study was approved by the Regional Ethics Committee and preregistered in ClinicalTrials.gov (NCT00201513).

Randomization

Written informed consent was signed by all participants before baseline assessment. Concealed random allocation to treatment groups was performed in permuted blocks of 3 to 9 after the baseline assessment, with equal number of subjects allocated between the 3 treatment groups. The randomization was administered by an independent study secretary who communicated treatment allocation to the researcher *via* telephone.

Interventions

Patients in the 3 intervention groups attended treatment once a week for 8 weeks and were not allowed to receive other LBP treatment during the intervention period. The patients were encouraged to stay active and all received an information booklet with general information on LBP. Attendance at weekly exercise sessions was recorded.

CSE was individualized according to protocols focusing on isolated activation of TrA during the abdominal drawing-in maneuver (ADIM).[9] The ADIM was performed in supine hook-lying position with real-time b-mode ultrasound feedback on performance.[17,18] The ADIM aimed to voluntarily activate TrA thickening and lateral slide with bilateral symmetrical activation, while obliquus internus (OI) and obliquus externus (OE) should remain relatively relaxed. When isolated TrA activation was achieved, pelvic floor and multifidus muscle co-contractions were included and the ADIM progressed to sitting and standing. The exercise program lasted 40 minutes and was carried out in a physiotherapy clinic. Written instruction to carry out the ADIM exercise at home was provided, and subjects were encouraged to perform 10 pain-free contractions 2 to 3 times per day, holding each contraction for 10 seconds.[9]

SE is described in detail elsewhere.[13,19,20] In brief, the exercises were adjusted to each patient's ability to keep the lumbar spine stable in neutral position throughout a range of leg/arm positions and movements. Elastic bands were attached to the pelvis to help the patient maintain a neutral spine position at all times and for exercises to progress without pain. Exercise progression was achieved by gradually reducing the elastic band support and placing the patients in progressively more demanding but pain-free positions to provide forceful activation of deep and superficial trunk muscles. The number of repetitions and sets was individually adjusted according to pain and fatigue. The SE was performed for 40 minutes in a physiotherapy clinic.

The GE group received general strengthening and stretching exercises as recommended in the treatment of nonspecific LBP.[21,22] Exercises were supervised by a physiotherapist and performed in groups of 2 to 8 patients for 60 minutes at a fitness center. Exercises included trunk extension/flexion/rotation in resistance apparatus (10 repetitions in 3 sets) and stretching of trunk and extremity muscles. Exercises were individually adapted when needed.

Outcome

The primary outcome was change in onset (before to after the intervention) of the deep abdominal muscles in response to unilateral rapid shoulder flexion, that is, a predictable perturbation of the trunk. The method was adopted from others[23,24] and has been used and described by our group previously.[25,26] Post-test was performed within 1 week after the 8-week intervention period. NRS was used to assess current back pain (NRS; 0–10, high score worse), Oswestry Disability Questionnaire to assess function or disability (0–100; high score worse),[27] Fear Avoidance Belief Questionnaire (FABQ) to assess fear of physical activity (FABQ physical activity subscale; 0–24, higher score worse),[28] and Body Mass Index (BMI) to assess body composition (body mass [kg] divided by square of height [m]). Pain and disability were reported before and after the intervention period and used to study associations with changes in onset, with adjustments for baseline fear (FABQ physical) and BMI.

Ultrasound recordings (see later) were stored and later analyzed to determine onset, in which case the researcher was blinded to group allocation. However, the researcher in charge of the onset recordings was aware of the group assignment. Subjects filled out the questionnaires without any influence of those involved with the interventions or research.

Experimental Procedure

Subjects performed rapid shoulder flexions from a standing position in response to a light signal switched on randomly within 3 to 10 seconds after a preparation command. The light signal was fed to both the electromyographic and ultrasound recording units (see later) allowing the physiological signals to be synchronized relative to the light onset. Subjects were told to flex the shoulder as quickly as possible from the side to approximately 60°. This was repeated 4 times, first on dominant and then on nondominant side, with a 10 to 15 seconds' pause between each repetition. Surface electromyography (EMG) was used to record onset in anterior deltoid, whereas ultrasound m-mode imaging was used to record the corresponding muscle deformation onset in the contralateral TrA, OI, and OE. The ultrasound operator was standing anterolateral to the patient supporting the probe by her hand at the predetermined location over the abdominal muscles contralateral to the moving arm. The electromyographic signal and ultrasound image were monitored to ensure that the muscles were relaxed before shoulder flexion was initiated.

EMG Recordings

EMG was recorded bilaterally from anterior deltoid, using bipolar surface electrodes (1-cm interelectrode distance). A ground electrode was placed over C7. The electromyographic signal was sampled at 2000 Hz, amplified 1000×, and bandpass filtered at 20 to 450 Hz (Bagnoli 16, Delsys, Inc., Boston, MA).

Ultrasound Recordings

A Vivid 7 scanner (GE-Vingmed Ultrasound, Horten, Norway) and a M12L linear probe set to 10 MHz were used for m-mode recording of abdominal muscle deformation onset in response to the rapid shoulder flexions.[25,26] The ultrasound probe was placed transversally, halfway between the 11th costal cartilage and the iliac crest, in a slightly oblique angle approximating the muscle fiber direction of TrA.[9,17,29,30] The position was adjusted so that the v-shaped midline border of TrA appeared toward one side of the image display, ensuring that TrA, OI, and OE were visible on the screen.[26]

The ultrasound recordings were stored and used to generate off-line m-mode traces to quantify onset of muscle deformation relative to the light signal. Structural displacements are depicted as changes in the grey-tone of the image, which can be used to construct root-mean-square (RMS) signals.[13,25] Muscle onset is determined by displacement of the RMS trace from selected depth intervals. A high-pass filter (4th order Butterworth filter, cutoff frequency 30 Hz) was used to reject slow probe movement artifacts, that is, signals with velocities of less than 2 mm/s. Muscle onset was determined as the point in time where the RMS trace clearly rose above baseline level.[31] Matlab-based software with a built-in calliper was used to assess time latency between the light signal and muscle onset (MathWorks Inc., Natick, MA). The operator chose the depth and vertical range for the RMS calculations, with separate ranges and calculations for TrA, OI, and OE. The earliest detected onset, irrespective of which muscle layer was activated first, was used for analyses.

Data Analysis

Based on pilot data and a previous study,[3] sample size was estimated to detect mean (SD) difference in onset between 2 groups of 40 (±50) ms, with alpha of 5% and power (1-β) of 90%. This required 31 patients in each group but 109 patients were included to account for withdrawals.

Before analysis, the quality of the EMG signal and the concurrent ultrasound trace were assessed. A researcher blind to the patients' group allocation qualified the difficulty in determining abdominal muscle onset in 3 levels: "no problem," "difficult," or "impossible/nearly impossible." Recordings classified as "impossible/nearly impossible" were due to technical difficulties in the instrumentation, weak muscle activity signal, or inadequate muscle relaxation and were excluded from further analyses.

Median onset of 2 to 4 repetitions was used for analysis. To use all available data, intention-to-treat analyses using linear mixed models were performed (IBM SPSS Statistics v18, Amonk, NY and Stata v 10, Stat Corp LP, College Station, TX). Mean group differences (95% confidence interval [CI]) at post-test were estimated with linear mixed models adjusted for baseline values and with patients as random effect (random intercept). The level of statistical significance was set to $P < 0.05$.

Association between changes in pain and onset from before to after the intervention was analyzed with linear regression and explained variance (R^2) used for interpretation. "Current pain" was basis for all analyses of pain. Logistic regression was used to estimate associations between clinical significant improvement in pain (≥2 on the 0–10 NRS scale) and change in onset after the intervention. Odds ratio (OR) for clinically significant pain reduction was compared in subgroups by dividing the patients in 2 groups by the median onset value at baseline and by improvement or deterioration in onset after the intervention (*i.e.*, resulting in a 2 × 2 contingency table). Similar analyses were performed for disability, with 10 points as cutoff for clinical significant improvement.[32] The analyses were adjusted for age, sex, BMI, pain duration, and baseline fear avoidance beliefs (physical subscale).

RESULTS

Because of technical problems, 7 of the 109 randomly assigned patients had no m-mode recording at either pre- or post-test and were omitted from analyses (Figure 1). Baseline characteristics for the patients included in the intention-to-treat analysis are presented in Table 1. The treatment groups were equal at baseline except for slightly younger age in the GE group ($P = 0.04$). Median duration since first LBP episode was 6.6 years (range, 0.3–38) and mean (SD) current pain intensity was 3.3 (1.6). Overall, 31, 30, and 26 subjects in the CSE, SE, and GE groups, respectively, attended at least 6 treatments.

No or only small changes were found in mean onset after treatment in the 3 intervention groups (Figure 2). The largest improvement was seen for dominant shoulder flexion in the SE group (12.4 ms). Comparisons of baseline adjusted between group differences for dominant shoulder flexion showed a 15 ms (95% CI, 1–28; $P = 0.03$) improvement in SE relative to CSE and a 19 ms (95% CI, 5–33; $P < 0.01$) improvement in SE relative to GE (Table 2). No group differences were observed for nondominant shoulder flexion.

Figure 3 illustrates change in abdominal muscle onset after the intervention relative to baseline onset for dominant and nondominant shoulder flexion, separately. The overall mean change was 0 ms with limits of agreement ±42 ms (±2 SD). The regression lines suggest a regression toward the mean effect; those with an early onset at baseline became slower and those with a later onset at baseline became faster.

There was no association between changes in pain and onset during the intervention period ($R^2 \leq 0.02$). ORs for clinically significant improvement in current pain were equal for those who were "slow" at baseline and became "faster" after intervention (OR = 1.03; Table 3) and for those who were "fast" at baseline and became "slower" after intervention (OR = 1.04). Similar analyses were attempted but not possible for disability as only 18 subjects reached a minimal important change of 10 points on the Oswestry Disability Index, as defined by Ostelo *et al*.[32]

DISCUSSION

This randomized controlled trial investigated the effect of CSE on deep abdominal muscle feed-forward activation in chronic LBP patients. No overall improvement in abdominal muscle onset was found after 8 weeks of different exercises. Change in onset differed less than 20 ms between the groups. A small but significantly shorter onset latency was found after SE compared with CSE and GE, but only on 1 side. No association was found between changes in pain and feed-forward onset in the intervention period.

Results Compared With Other Studies

The effect of isolated TrA training on feed-forward activation has been investigated in 2 small studies.[14,15] Significantly shorter TrA onset latency was observed after only 1 treatment in the former study.[14] Improvement in onset, gained after the initial 4 weeks of exercise, was partly retained at 6-month follow-up[15]; however, follow-up data were available for only 7 subjects and no control group was included.

Unlike isolated TrA training in the study by Tsao and Hodges,[14] no immediate effect on abdominal muscle onset was found after a single session with abdominal and back muscle cocontraction exercises in a small group of LBP patients.[33] Interestingly, in the study by Tsao and Hodges,[15] the positive

TABLE 1. Group Characteristics at Baseline. Values Are Mean ± SD Unless Otherwise Stated

Variable	Intervention Groups CSE (n = 33)	SE (n = 34)	GE (n = 35)	*P*
Sex (females, %)	26 (79%)	21 (62%)	24 (69%)	0.31
Age (yr)	41.5 ± 11.9	42.7 ± 10.0	36.3 ± 10.4*	0.04
BMI (kg/m^2)	24.7 ± 3.1	24.7 ± 2.9	24.2 ± 2.8	0.69
Current pain (0–10)	3.4 ± 1.3	3.5 ± 1.8	3.1 ± 1.6	0.49
Worst pain last 4 wk (0–10)	6.2 ± 2.1	6.3 ± 2.3	6.0 ± 1.9	0.83
Disability, Oswestry (0–100)	20.0 ± 7.2	19.9 ± 9.0	19.6 ± 8.3	0.98
FABQ, physical activity (0–24)	7.9 ± 5.3	8.4 ± 5.5	9.1 ± 5.3	0.69
Years since first LBP episode (median, range)	6.0 (0.4–38)	7.0 (0.5–26)	6.0 (0.3–27)	0.95
Sick leave at baseline (n, %)	3 (9%)	5 (15%)	3 (9%)	0.66

**GE significantly younger than SE, P = 0.02.*

CSE indicates core stability exercise; SE, sling exercise; GE, general exercise; BMI, body mass index; FABQ, Fear Avoidance Belief Questionnaire; LBP, low back pain.

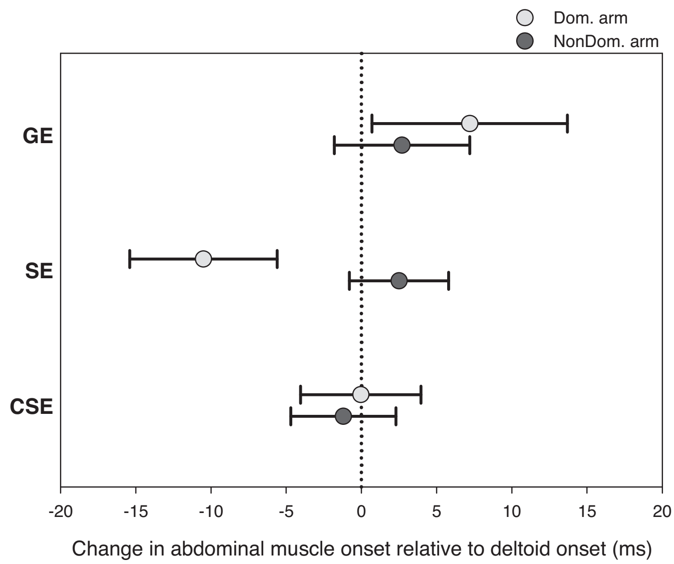

Figure 2. Change in mean (SE) onset (ms) during the rapid arm flexion test for dominant and nondominant arm (onset recorded from contralateral abdominal muscles) in the 3 intervention groups. Negative values represent faster onset at post-test relative to pretest. Only subjects with complete data were included (n = 78). GE indicates general exercise; SE, sling exercise; CSE, core stability exercise; dom, dominate; nondom, nondominant.

effect of isolated TrA exercise was found in subjects with considerably larger abdominal muscle onset delays at baseline (range, 5–75 ms after deltoid) compared with the patients included in our study and the study by Hall *et al*.[33] In a larger study, the effect of Swiss ball exercises was compared with that of GEs.[16] The study showed long-term improvement in deep abdominal muscle onset (which was recorded on the right abdominal side only), but the effect was unrelated to specific exercise interventions. Although the latter and our studies strongly indicate no clinically important short-term effects of exercises, the long-term effects and whether exercises are superior to natural recovery need further exploration.

Validity of Onset

The validity of deep abdominal muscle onset as an outcome measure in interventional studies may be questioned. Timing varies considerably in both healthy individuals[26,34] and LBP patients (Figure 3). A large study applying ultrasound recordings found no evidence of increased latency response in LBP patients.[35] Thus, delayed onset is not a consistent finding in LBP. Moreover, motor control characteristics in LBP vary considerably between tasks.[36–38] As opposed to upright trunk perturbations, TrA activation was not particularly early compared with other abdominal muscles in the absence of upright postural demands[37] or during multidirectional support-surface translations.[36] We have demonstrated that rapid shoulder flexion in standing generate various onset responses.[39] With ultrasound strain rate imaging, which can record onset in different muscle layers,[26] and based on several hundred contractions, the earliest onset was detected in TrA in 27% of the contractions and in deep OI in 53% of the contractions. In 12 of 24 subjects, the earliest onset alternated between different muscles in subsequent contractions. Within- and between-subject variations need to be acknowledged in future studies.

Strength and Limitations

Onset of muscle deformation is a proxy to onset of electrophysiological muscle activation. A critical question in this study is whether onset recorded by ultrasound m-mode is reliable and valid. Exploring onset in muscles situated in layers should ideally be recorded with intramuscular EMG.

TABLE 2. Abdominal Muscle Activity Onset (ms) Relative to Anterior Deltoid Onset in Shoulder Flexion With Dominant and Nondominant Arm. Mean (SD) Onset for the Intervention Groups at Pre- and Post-test and Estimated Mean Difference (95% CI) in Change Between the Groups (Post-test Minus Pretest) as Given by the Mixed Linear Models

Variable	Abdominal Muscle Onset			Adjusted Between-Group Differences in Onset*			P
	CSE	SE	GE	CSE *vs.* SE	CSE *vs.* GE	SE *vs.* GE	
Dominant shoulder flexion							
Pretest (ms)	−9 ± 22	3 ± 21	−8 ± 29				
Post-test (ms)	−5 ± 21	−11 ± 20	−3 ± 26	15 (1–28)†	−4 (−18–10)	−19 (−33 to −5)‡	0.02
Nondominant shoulder flexion							
Pretest (ms)	7 ± 21	−4 ± 19	5 ± 24				
Post-test (ms)	6 ± 17	0 ± 21	4 ± 25	−4 (−14–6)	−2 (−13–8)	2 (−9–12)	0.65

Results from intention to treat analysis by mixed model analyses with P value for overall between group differences. A negative difference means greater improvement in onset in the former relative to the latter group, and vice versa for positive differences.

†Significantly earlier onset in SE relative to CSE, P = 0.03.

‡Significantly earlier onset in SE relative to GE, P < 0.01.

CSE indicates core stability exercise; SE, sling exercise; GE, general exercise.

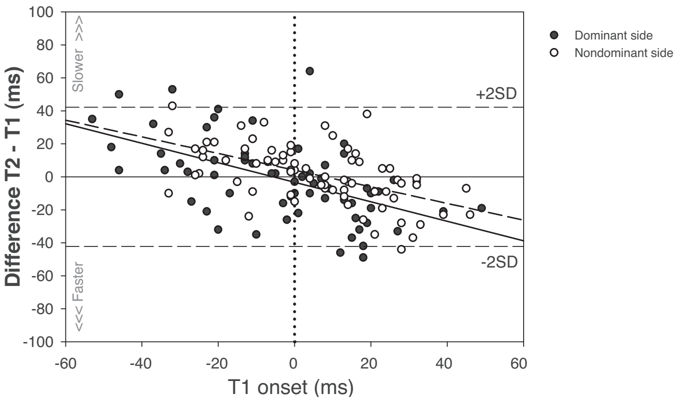

Figure 3. Association between baseline onset (T1) and change in onset (T2-T1) in the abdominal muscle onset relative to anterior deltoid onset during the rapid arm flexion test. The dotted vertical line represents deltoid onset at T1. Results are from dominant (solid circles and regression line; n = 64) and nondominant (hollow circles and dashed regression line; n = 71) arm flexion test. The overall mean change (solid horizontal line) was 0 ms and limits of agreement ±42 ms (2 SD). Positive values on the ordinate mean slower onset at T2 relative to T1, and vice versa. The data and the fitted regression lines indicate that subjects with an early response at T1 became slower at T2 (positive values on the ordinate), whereas those with a late response at T1 became faster at T2 (negative values on the ordinate), indicating a regression toward the mean effect.

However, intramuscular EMG is not optimal in clinical trials with repeated measures as it is invasive, often painful, has limited pick-up area, and the insertion location is difficult to reproduce. Ultrasound m-mode has shown to be highly reliable for detecting the first *or* earliest onset in deep trunk muscles,[25,26,34,39] but is not ideal for detecting sequential onsets in adjacent muscle layers.[26] However, high agreement has been found between intramuscular EMG and m-mode-detected onset in TrA and OI in response to rapid shoulder flexion with mean differences between the methods less than 10 ms.[26] Whether the earliest onset is a valid estimate of muscular control deficits depends on the underlying hypothesis for delayed onset. In terms of pathogenesis, delayed abdominal muscle onset may reflect a short time-window with spinal instability and increased risk of spinal injury. In this case, a general shortening of onset latency is likely beneficial irrespective of which muscle is responsible for the early activation. Alternatively, delayed onset may be an adaptation to pain that affects some muscles more than others, in which case the m-mode method lacks specificity to explore the different abdominal muscle layers independently. With strain rate imaging, we found indications for a functional subdivision of OI, that is, activation pattern of deep OI resembled TrA and were clearly different from superficial OI.[39] Furthermore, earliest onset was found to alternate between the muscles within the same individual and session, with earliest onset occurring equally or more frequently in deep OI than in TrA. With this complexity, it can be argued that the earliest onset, irrespective of which muscle is activated, is the most valid estimate to quantify muscle onset deficits. For that purpose, the ultrasound m-mode method has good reliability and validity.[25,26,34,39]

ADIM exercises have been proposed as a first step in correcting feed-forward delays and regaining control of the deep abdominal muscles.[9] To comply with these recommendations, ADIM exercises were included in the CSE intervention. In addition, both low (CSE) and high load (SE) motor control exercises were studied as they aim to improve spinal control and stability. We thus believe this study was adequately designed to investigate early effects of CSE on abdominal muscle feed-forward activation.

Regression toward the mean may have influenced the results in our study. Improvement in onset is typically observed in patients with considerable delay at baseline.[14–16] In subjects with no or little delay at baseline (*i.e.*, deep abdominal muscle onset equal to or earlier than the prime mover of the arm), no effect or even later onset was observed after a single session with trunk muscle cocontraction exercises.[33] In our study, marginal group differences at baseline made improvement in the SE group more likely, with slightly more delay in the SE group on the side showing significant treatment effects. The superior effect in the SE group may thus, at least partly, be explained by a regression toward the mean effect.

TABLE 3. Odds Ratio (95% CI) for Clinically Important Reduction in Pain (≥2 on the Numeric Rating Scale; NRS) After the Intervention Period in Subgroups Based on Baseline and Change in Deep Abdominal Muscle Onset. Results Based on Dominant Arm Shoulder Flexion for Participants With Complete Data Set

Muscle Onset at Baseline	Change in Abdominal Muscle Onset After Exercise					
	No Change or Slower			Faster		
		Pain Reduction ≥2 on NRS			Pain Reduction ≥2 on NRS	
	OR (95% CI)*	Yes (N)	No (N)	OR (95% CI)*	Yes (N)	No (N)
Slow (>0 ms)	Reference	3	5	1.03 (1.00–1.07)	13	16
Fast (<0 ms)	1.04 (0.99–1.09)	12	15	1.00 (0.96–1.05)	2	6

*Adjusted for age, sex, body mass index, pain duration since first episode, and initial fear avoidance beliefs, physical subscale.

NRS indicates Numeric Rating Scale; CI, confidence interval.

All but 1 patient in this study had abdominal onset (median of 2–4 contractions) within the feed-forward window, that is, −100 to +50 ms relative to onset in anterior deltoid.[23] Subjects in our study thus had little potential for improvement. Furthermore, the subjects had low pain and disability levels at baseline (Table 1). Although the levels probably reflect a large proportion of subjects with LBP in the population and in primary care, it is possible that patients with higher pain and disability levels or clearly reduced onset latency at baseline may experience more favorable exercise effects than observed in this study. The number of subjects dropping out and not presenting at post-test was larger in the GE group than in the other 2 groups (Figure 1). Although all dropouts were accounted for in the mixed models analysis, we cannot exclude that it may have affected the results. If dropout was due to lack of effect, this could overestimate the effect in the GE group. Dropouts were, however, not significantly distinguished on any of the baseline variables (Table 1) from those who completed the study.

The observed effects in this study are likely clinically insignificant. In terms of group averaged onset, we have previously reported that both ultrasound and intramuscular EMG give highly reliable estimates (test-retest differences of <5 ms).[26] However, on an individual level, the smallest detectable difference was 37 ms for ultrasound and 29 ms for intramuscular EMG onsets,[26] indicating that individual changes in onset of approximately less than 30 ms are not distinguishable from measurement error. The superior effects in the SE group on one side were within the limits of measurement error.

➢ Key Points

- ❑ This is the first randomized trial applying different loads and type of exercises, including CSEs, to investigate effects on abdominal muscle feed-forward activation (timing) in patients with chronic LBP.
- ❑ No or only small changes in abdominal muscle onset were observed during the 8-week intervention period.
- ❑ Large individual variations in activation pattern of the deep abdominal muscles may justify further exploration of differential effects in subgroups of LBP.

Acknowledgments

The study was financially supported by The Norwegian Fund for Postgraduate Training in Physiotherapy. We also thank Anne Fladmark, Line Helene Torsdatter, and Øystein Petter Nygaard for contributions in data acquisition and preparation.

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