Journal of Athletic Training 2012;47(4):379–389
doi: 10.4085/1062-6050-47.4.12

www.nata.org/journal-of-athletic-training

original research

Cocontraction of Ankle Dorsiflexors and Transversus Abdominis Function in Patients With Low Back Pain

Seung-Chul Chon, PhD, PT*; Joshua H. You, PhD, PT†; Susan A. Saliba, PhD, PT, ATC, FNATA‡

*Department of Physical Therapy, Konyang University, Seo-Gu, Daejeon, Republic of South Korea; †Department of Physical Therapy, Yonsei University, and Movement Healing Laboratory (Wellness, Fitness, and Prevention/Movement Impairment/Sports Medicine and Movement Science), Wonju City, Kangwon-do, Republic of South Korea; ‡Department of Kinesiology (Sports Medicine and Athletic Training), Physical Medicine & Rehabilitation, University of Virginia, Charlottesville

Context: The abdominal draw-in maneuver (ADIM) with cocontraction has been shown to be a more effective method of activating the transversus abdominis (TrA) in healthy adults than the ADIM alone. Whether such an augmented core stabilization exercise is effective in managing low back pain (LBP) remains uncertain.

Objective: To determine the effect of 2 weeks of ADIM and cocontraction training on abdominal muscle thickness and activation timing and to monitor pain and function in patients with LBP.

Design: Case-control study.

Setting: Local orthopaedic clinic and research laboratory.

Patients or Other Participants: Twenty patients with mechanical LBP (age = 27.20 ± 6.46 years, height = 166.25 ± 8.70 cm, mass = 58.10 ± 11.81 kg) and 20 healthy, age-matched people (age = 24.25 ± 1.59 years, height = 168.00 ± 8.89 cm, mass = 60.65 ± 11.99 kg) volunteered for the study.

Intervention(s): Both the LBP and control groups received ten 30-minute sessions of ADIM and cocontraction training of the tibialis anterior (TA) and rectus femoris (RF) muscles over a 2-week period.

Main Outcome Measure(s): A separate, mixed-model analysis of variance was computed for the thicknesses of the TrA, internal oblique (IO), and external oblique muscles. The differences in mean and peak electromyographic (EMG) amplitudes, onset time, and latency were compared between the groups. The visual analog pain scale, Pain Disability Index, and LBP rating scale were used to assess pain in the LBP group before and after the intervention.

Results: We found an interaction between the LBP and control groups and a main effect from pretest to posttest for only TrA muscle thickness change ($F_{1,38} = 6.57$, $P = .01$). Reductions in all pain measures were observed after training ($P < .05$). Group differences in peak and mean EMG amplitudes and onset time values for TrA/IO and TA were achieved ($P < .05$). The RF peak ($t_{38} = -3.12$, $P = .003$) and mean ($t_{38} = -4.12$, $P = .001$) EMG amplitudes were different, but no group difference was observed in RF onset time ($t_{38} = 1.63$, $P = .11$) or the cocontracted TrA/IO peak ($t_{38} = -1.90$, $P = .07$) and mean ($t_{38} = -1.81$, $P = .08$). The test-retest reliability for the muscle thickness measure revealed excellent correlations (intraclass correlation coefficient range, 0.95–0.99).

Conclusions: We are the first to demonstrate that a cocontraction of the ankle dorsiflexors with ADIM training might result in a thickness change in the TrA muscle and associated pain management in patients with chronic LBP.

Key Words: core stability, electromyographic sequencing, ultrasound imaging

Key Points

- The abdominal draw-in maneuver followed by the cocontraction technique stimulated the selective recruitment of the transversus abdominis and internal oblique muscles, possibly leading to reduction of low back pain.
- Because the cocontraction was associated with improved activation of the transversus abdominis muscle, this novel technique might have therapeutic efficacy for the management of individuals with mechanical low back pain and core instability.
- Abdominal draw-in maneuver training is beneficial for the selective recruitment of the transversus abdominis muscle and its central mechanism of action of the lumbopelvic region, and the mechanism of deep musculofascial onset might be augmented further by the cocontraction technique.

Mechanical low back pain (LBP) is a common musculoskeletal impairment that often is associated with neuromuscular dysfunction of the transversus abdominis (TrA) muscle and spinal instability, affecting activities of daily living and physical activity.[1–3] Epidemiologic evidence has indicated that up to 70% of patients with acute LBP ultimately develop chronic LBP.[4] Delayed onset time of TrA feed-forward activation during shoulder movement[5] and altered muscle-activation patterns during locomotion[6] have been identified in patients with LBP as important pathologic markers of abdominal neuromuscular dysfunction. Normally, the neuromuscular system is believed to maintain stability of the lumbar spine by increasing the active and passive stiffness of the deep abdominal and multifidus muscles or modulating muscle cocontraction, which increases the compressive loads.[7] This

Table 1. Demographic and Clinical Characteristics of Participants (N = 40), Mean ± SD

Characteristic	Group		t_{38} Value	*P* Value
	Low Back Pain (n = 20)	Control (n = 20)		
Age, y	27.20 ± 6.46	24.25 ± 1.59	−1.98	.06
Height, cm	166.25 ± 8.70	168.00 ± 8.89	0.67	.51
Mass, kg	58.10 ± 11.81	60.65 ± 11.99	0.69	.50
Sex				
Male	7	9	NA	NA
Female	13	11	NA	NA
Onset, mo	15.3 ± 9.03	NA	NA	NA
Visual analog scale score[a]	6	NA	NA	NA
Pain Disability Index[b]	30	NA	NA	NA
Low back pain rating scale[c]	70	NA	NA	NA

Abbreviation: NA, not applicable.
[a] The visual analog scale ranges from 0 to 10.
[b] The Pain Disability Index ranges from 0 to 70.
[c] The low back pain rating scale ranges from 0 to 130.

lumbar spinal stability offsets the deleterious effects of stress imposed on the spine during lifting.[8–10]

Core stabilization exercises, including the abdominal draw-in maneuver (ADIM), lateral bridging, pelvic tilting, and abdominal bracing,[2,11,12] have been used widely to improve lumbopelvic stability.[5,13] Core stabilization exercises often incorporate a low degree of TrA activation loading (less than 30% maximal voluntary isometric contraction [MVIC]) with minimal activity of the superficial muscles, such as the external oblique (EO) and rectus abdominis, during the initial phase of rehabilitation.[9,14] One important mechanism by which core stabilization exercise increases the neuromuscular function of the TrA and associated lumbar spinal stability is neuromechanical stiffening of the thoracolumbar fascia (TLF).[10] Specifically, the synergistic contraction of the TrA and posterior fibers of the internal oblique (IO) increases the posterolateral lumbar tension on the TLF that connects to the spinous and transversus processes of the lumbar spine.[10] When the ADIM is performed, the activated TrA draws the abdominal wall inward while concurrently forcing the viscera upward into the diaphragm and downward into the pelvic floor. Coactivation of the TrA and IO (TrA/IO) together with the TLF generates intra-abdominal pressure, which transforms the abdomen into a mechanically rigid cylinder, providing spinal stability.[15]

Administering core stabilization exercises to patients who have LBP and severe pain might result in a substitution or compensatory movement (eg, rotation and extension of the lumbopelvic complex) associated with neuromuscular inefficiency in the deep core muscles. Therefore, researchers[5,13] have suggested that abdominal or core stabilization exercise without proper pelvic stabilization might increase intradiscal pressure, anterior shearing, and compressive forces in the lumbar spine, accentuating LBP. A method to enhance the activation of the deep abdominal muscles might be advantageous.

Resisted ankle dorsiflexion to augment the TrA/IO via cocontraction is a technique for improving the selective activation of deep core muscles, such as the TrA/IO, in populations without pain.[16] This approach was derived from the concept of irradiation in proprioceptive neuromuscular facilitation, which emphasizes the important contribution of the relatively stronger distal muscle group by increasing the number of potential motor-unit recruitments involved or weakened. Chon et al[16] reported that the coactivation of the ankle dorsiflexors and rectus femoris (RF) muscles effectively augmented the selective activation of the TrA muscle, as demonstrated by an increased mean electromyographic (EMG) amplitude of the TrA/IO muscles after the resisted ankle dorsiflexion. The EMG analysis showed that a strong contraction of the dorsiflexion muscles, specifically the tibialis anterior (TA), improved motor recruitment of the TrA/IO muscles during the ADIM.[16] This finding suggests that cocontraction of the dorsiflexion muscles increases recruitment of the active motor units of the TrA/IO muscles.[6,16,17] Researchers have found that enhanced TrA neuromuscular control patterns in people with LBP play an important role in functional spinal mobility and back pain.[8,18,19]

Although evidence that core stabilization exercises can contribute to deep abdominal contraction exists,[8] little information on effective ways to improve TrA muscle activation and timing in the population with LBP is available. Therefore, the purpose of our study was to determine the effect of 2 weeks of ADIM and cocontraction training on abdominal muscle thickness and activation timing and to monitor pain and function in people with LBP.

METHODS

Participants

A convenience sample of 40 people volunteered for this study. Twenty patients with LBP (age = 27.20 ± 6.46 years, height = 166.25 ± 8.70 cm, mass = 58.10 ± 11.81 kg) were recruited from a local orthopaedic clinic, and 20 healthy people (age = 24.25 ± 1.59 years, height = 168.00 ± 8.89 cm, mass = 60.65 ± 11.99 kg) were recruited for the control group from a university community (Table 1). The flow chart is presented in Figure 1. The data we collected for the patients with LBP included onset time, nature and location of symptoms, aggravating and relieving factors, medication, history of surgery, history of back pain or injury, and pain measurements. The inclusion criteria for the LBP group were clinical assessment of mechanical LBP, periods of LBP within the 6 to 12 months before the study, and a current pain level ranging from 4 to 8 of 10 on the self-reported visual analog scale (VAS). Patients who

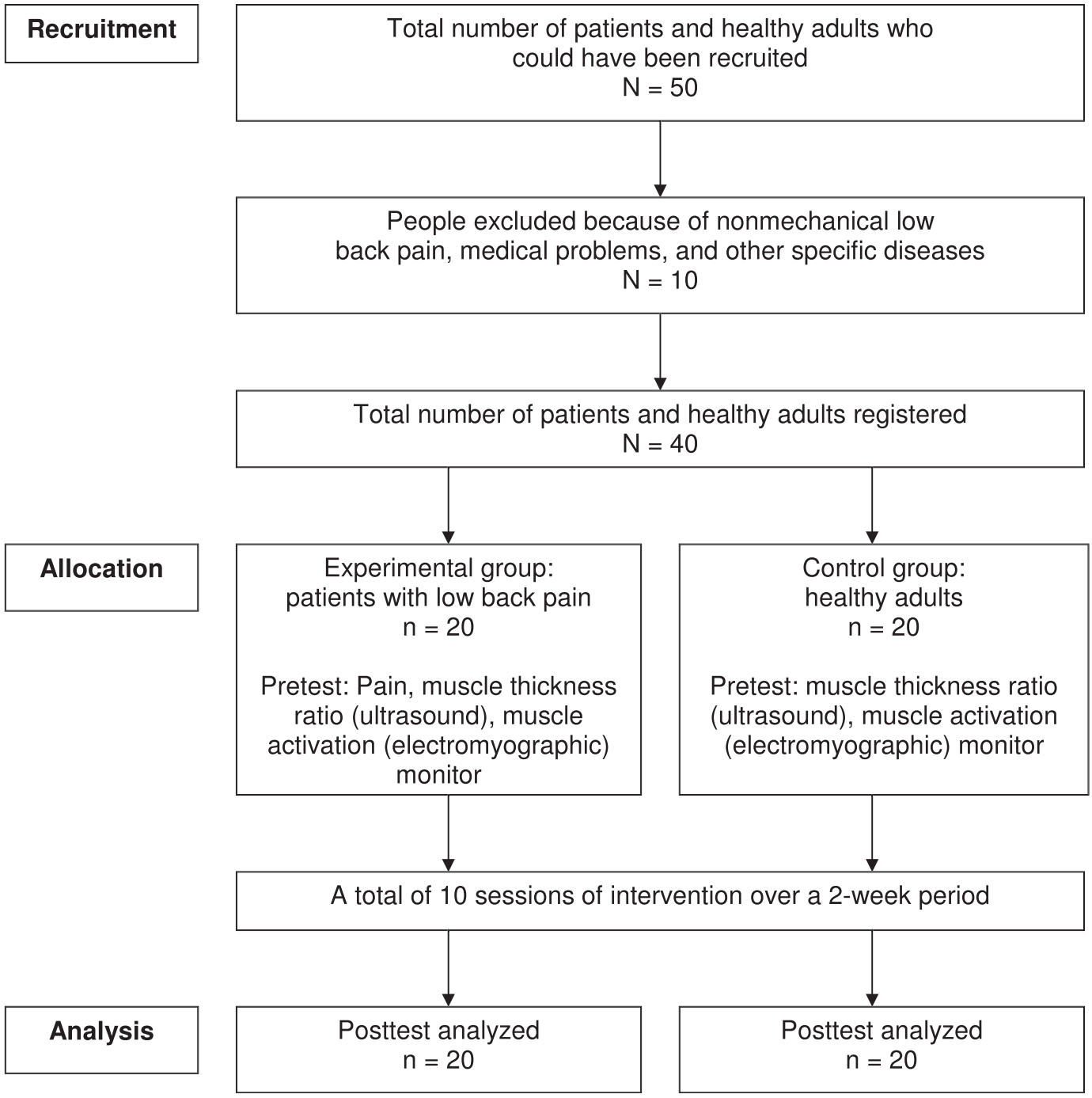

Figure 1. Flow diagram for the study.

had LBP and had received nonoperative therapy (ie, hydrocollator, ultrasound, transcutaneous electric nerve stimulation, interferential current therapy, range of motion, and Williams' flexion exercises) with limited therapeutic effects were observed. No participant had knowledge of or experience with ADIM training.

The clinical assessment criteria for mechanical LBP were intermittent pain that gradually develops later in the day, pain when standing or sitting for a long time, pain upon trunk flexion (or occasionally extension),[20,21] and pain when driving long distances or getting in and out of a car. A physical therapist (S.C.C.) with 10 years of experience made the diagnosis of mechanical LBP according to the clinical assessment criteria. Medical diagnosis of LBP was made by an attending orthopaedist or a physician who was not an author. The exclusion criteria included osteoporosis, structural deformity, systemic inflammatory disease, nerve-root compression, facet osteophytes, prolonged severe pain, problems with the neuromusculoskeletal system, and history of spinal surgery. These exclusion criteria were confirmed by reviewing each patient's medical record, which was completed by the physician. The control group comprised healthy young adults with no known medical problems or history of LBP.

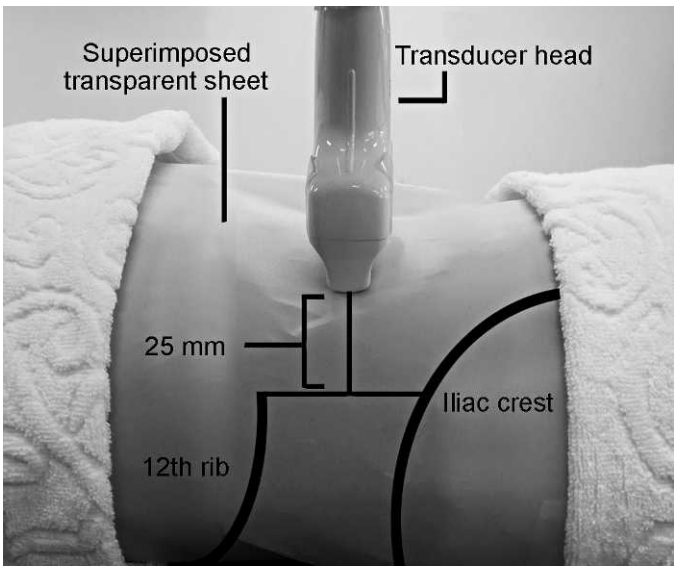

Figure 2. Placement of ultrasound transducer on abdominal muscle.

All participants provided written informed consent, and the study was approved by the Woosong University Ethics and Institutional Review Board.

Experimental Procedures

All assessments were made by researchers (S.C.C., J.H.Y., S.A.S.) who were blinded to participants' clinical status (healthy or LBP) and all measurements. Both the control and LBP groups underwent a pretest, then participated in a training program (cocontraction treatment) 5 days per week for 2 weeks and a posttest after the training (Figure 1). The dependent variables measured included the VAS; Pain Disability Index (PDI); LBP rating scale (LBPRS); muscle thickness for TrA, IO, and EO; EMG mean and peak amplitudes; onset time; and latency for TrA/IO, TA, and RF.

Pain and Function Assessment

Standardized pain and associated functional activity-based pain measurements included the VAS, PDI, and LBPRS for the LBP group only. The VAS consists of a 10-cm straight line on which the participant scores his or her pain on a scale ranging from 0 (*no pain*) to 10 (*pain as bad as it could be*).[22,23] The test-retest reliability of this scale ranges from 0.60 to 0.77, and its validity ranges from 0.64 to 0.84.[24] The PDI is a brief self-report instrument that provides information to complement the evaluation of physical functional impairment. It comprises 7 subitems of physical activities: recreation, occupation, sexual behavior, family/home responsibilities, social activity, self-care, and life-support functions.[25,26] The scoring system allows the patient to rate these activities on a scale ranging from 0 to 10, with a total possible score of 70.[25] The test-retest reliability of the PDI ranges from 0.73 to 0.91.[26] The LBPRS includes the 3 separate clinical illness components that constitute LBP in point scales: back and leg pain (range, 0–60 points), disability index (range, 0–30 points), and physical impairment (range, 0–40 points).[27,28] The scale was designed to monitor outcomes after therapeutic intervention. A higher score indicates a greater level of disability and impairment, and the maximum point value is 130. The intraclass correlation coefficient (ICC) of the LBPRS is 0.61,[28] with a high level of interrater reliability (97.7%).[27]

Ultrasound Imaging

A Logiq sonography system (model α 200; Samsung-GE Medical Systems Inc, Seongnam, Republic of South Korea) with a 7.5-MHz linear transducer was used to assess muscle thickness during the test. The thicknesses of the abdominal wall muscles, including the TrA, IO, and EO muscles, were measured, and changes in the TrA ratio were calculated. Muscle thickness was an indicator of muscle function or activity. The change in TrA thickness represents the relative change in the thickness ratio of the TrA contracted to TrA rest, which typically involves examination of the relative change in TrA muscle thickness (TrA contraction ratio = TrA contracted/TrA relaxed).[29] Participants were instructed to assume a relaxed hook-lying position.[5] Their hip- and knee-joint angles were maintained at approximately 40° to 80° to eliminate lumbar lordosis. The inferior borders of the rib cage and iliac crest on the dominant side were palpated as reference points.[30] We determined the *dominant side* of the control participants by observing the limb with which they kicked a ball, whereas the *dominant side* of the participants with LBP was determined by asking them which was the more painful side. Ultrasound gel (AQUASONIC 100; Parker Inc, Orange, NJ) then was applied to the transducer head, which was positioned transversely 25 mm anteromedial to the midway point between the 12th rib and the iliac crest (Figure 2).[30,31] The head of the transducer was maneuvered until the sharpest images of all lateral abdominal muscles (EO, IO, and TrA) had been obtained.[19] Three scans were taken on the dominant side of the abdominal muscles in their relaxed states.[19] The pretest scanning location was marked on a transparent sheet to ensure identical placement throughout the experiment, including the posttest.[32] Specifically, the anatomic reference locations for the iliac crest and the 12th rib first were palpated to identify them and mark them with a permanent marker. Second, we superimposed the transparent sheet over these locations and made corresponding markings on it with a permanent marker for consistent measurement (Figure 2). The images were acquired at the end of the exhalation phase.[30] The image data were stored, and the measurements of the muscle-thickness dimension in millimeters were determined with an on-screen caliper. The thicknesses of all 3 muscles were defined by drawing a vertical reference line that was located 25 mm from the left edge (muscle-fascia junction) of the TrA (Figure 3).[30]

Based on this protocol, we conducted a test-retest reliability study to determine the degree of reliability between our pretest and posttest use of the ultrasound measurements of abdominal muscle size in patients with LBP, including those of the TrA, IO, and EO muscles.

EMG Measurement

We carefully prepared the skin of each participant to reduce skin impedance to less than 5 kΩ by dry shaving hair with a disposable razor, abrading the skin with fine

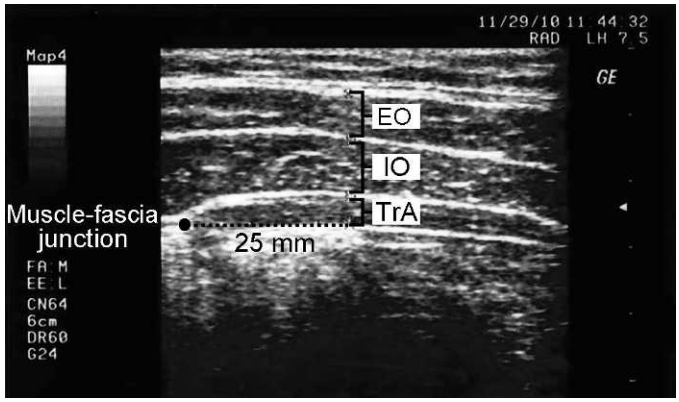

Figure 3. Abdominal muscle-thickness measurement. Abbreviations: EO, external oblique; IO, internal oblique, TrA, transversus abdominis.

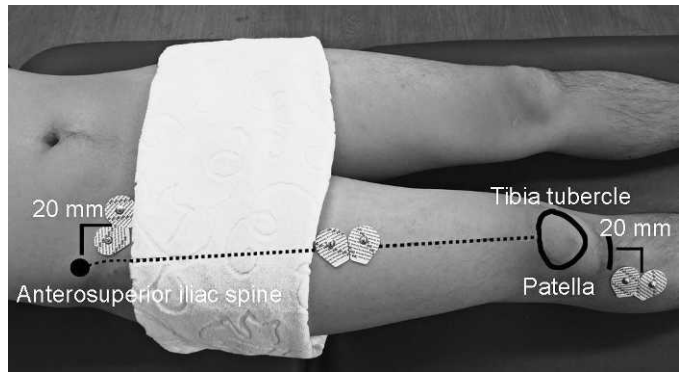

Figure 4. Placement of electromyography electrodes.

sandpaper, and cleansing the skin with a 2% alcohol swab. After the skin was dry, pairs of circular Ag/AgCl surface electrodes with a contact diameter of 19 mm were attached at an interelectrode distance of 20 mm to the following locations (Figure 4). A reference electrode was positioned over the lateral malleolus. The electrode placement for the TrA/IO was approximately 20 mm medial and inferior to the anterosuperior iliac spine (ASIS).[33] For the TA, it was 20 mm distal and lateral from the tibial tubercle, and for the RF, it was halfway between the ASIS and the superior part of the patella (Figure 4).[34]

The surface EMG system (Laxtha Inc, Sinil-dong, Daejeon, Republic of South Korea) comprised 8 electrodes, a preamplifier for initial processing, a second amplifier, an analog-to-digital converter with 16-bit resolution, a universal serial bus connection, and a WEMG-8–type cable. This EMG system was used to record the onset times and mean and peak amplitudes of the TA, RF, and TrA/IO muscles. These EMG data were used to provide the proper muscle-activation sequence during the cocontraction training.

Because approximately 30% MVIC has been reported to be the best activation level for the TrA/IO muscles,[9] we used this criterion during our EMG biofeedback training for effective cocontraction of the target muscles. After the MVIC for each TA, RF, and TrA/IO muscle was reached, participants were instructed to sustain 30% MVIC of the TrA/IO[9] followed by 50% MVIC of the TA, RF, and TrA/IO during cocontraction training for 3 seconds, and then to rest for 5 seconds. The EMG monitoring was used to ensure consistent muscle activation at each target MVIC for the corresponding muscle. An automatic auditory cue that lasted for 3 seconds over a 20-second period signaled each participant to start contracting the muscle at the proper time interval (Figure 5).

The raw EMG signal was processed using Telescan software (version 2.89; Laxtha Inc) at a sampling frequency of 1024 Hz with a 60-Hz notch filter for noise reduction associated with electric interference arising from the usual sources, including 60-Hz power lines or radio frequencies and electric or magnetic devices. The root mean square EMG amplitude for each TA, RF, and TrA/IO muscle was calculated for 3 seconds (seconds 3–6) during the ADIM and for 3 seconds (seconds 12–15) during the cocontraction (Figure 5). The identification of the onset time of EMG for each TrA/IO, TA, and RF muscle was determined as the onset point at which the mean of 51.2 consecutive samples (50 milliseconds) exceeded the baseline activity (threshold level) by 3 standard deviations (SDs). The raw EMG signal was full-wave rectified and filtered using a band-pass filter at 8 to 480 Hz with a rejection factor of –3 dB. *Baseline activity* was defined as a period of approximately 3 seconds before ADIM movement or 6 seconds before ankle dorsiflexion. Each onset time was checked visually to ensure that EMG onset was not misrepresented or confounded by motion artifact or environmental interference. Fewer than 5% of all trials were discarded after visual inspection because of an inability to differentiate the muscle onset from environmental interference or activity. The latency between the onset of the TrA/IO and TA muscles, of the TA and RF muscles, and of the TrA/IO and RF muscles was analyzed for both groups.

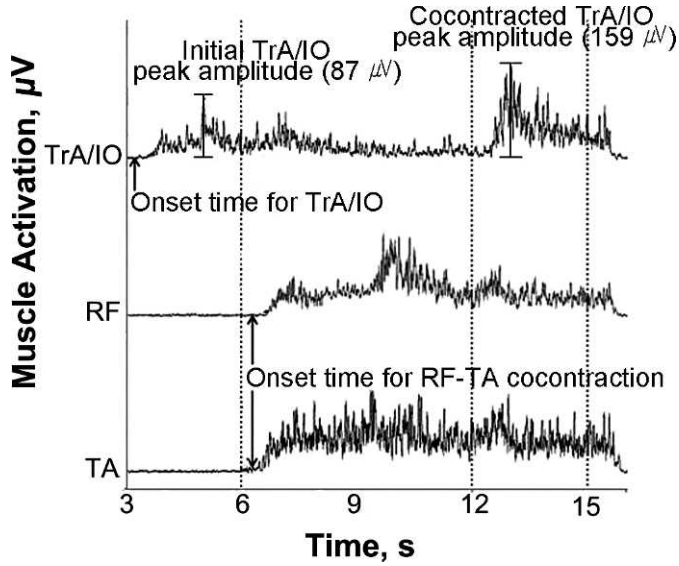

Figure 5. Electromyographic biofeedback during the resisted dorsiflexion training to augment transversus abdominis (TrA) muscle contraction. Electromyographic biofeedback was used to provide visual feedback about muscle activation of the corresponding transversus abdominis and internal oblique (IO), tibialis anterior (TA), rectus femoris (RF), and transversus abdominis and IO in sequence. The vertical arrow indicates the time at which the automatic auditory cue from the electromyography software was given sequentially for the initial TrA and IO contraction, cocontraction of TA-RF-augmented TrA and IO muscles, and release (or rest).

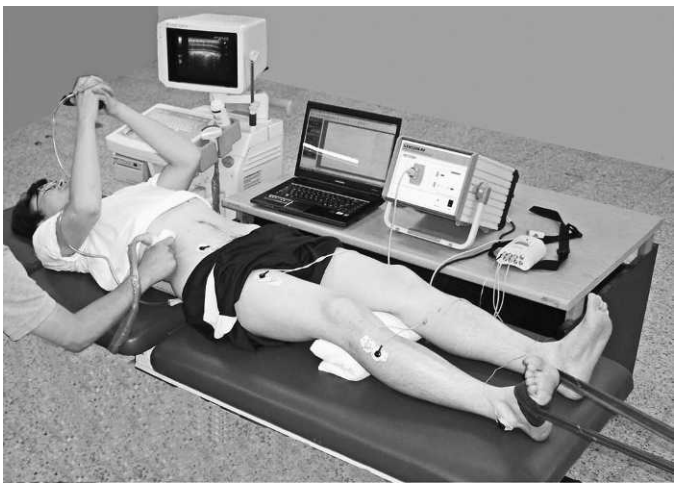

Figure 6. Participant positioning during cocontraction biofeedback training.

The EMG data for the pretest and posttest were not recorded. Initially, we intended to use EMG primarily to provide visual biofeedback and monitor consistent mean and peak amplitudes and sequences for the TrA/IO, RF, and TA to maximize our ADIM training effect during the cocontraction training. The EMG activity was recorded in 2 sessions in the first week of the training and another 2 sessions in the second week to facilitate a proper sequence of muscle activation.

Intervention

Both the control and LBP groups received a combination of ultrasound-guided and EMG-guided visual biofeedback for 30 minutes each day for 5 days each week over a 2-week period. The outcomes and performance of the ADIM and cocontraction to augment TrA/IO were determined using visual and tactile feedback. As illustrated in Figures 5 and 6, visual feedback information about EMG cocontraction and change in muscle thickness was presented in the respective EMG and ultrasound computer monitors and used for augmented feedback during ADIM and cocontraction training. Correct electrode placement for TrA/IO was ensured with ultrasound imaging, which was used to identify the proper location of these muscles during ADIM.

For the ADIM training, each participant was instructed to lie in a hook-lying position. A Stabilizer pressure biofeedback unit (model 9296; Chattanooga Group Inc, Hixson, TN) was placed under the fifth lumbar vertebra and was inflated to 40 to 70 mm Hg.[35,36] Next, the participant was instructed to draw in his or her navel gradually and maintain the target pressure without any pelvic motion. For ADIM and added cocontraction training, the participant was instructed to perform ADIM and then to cocontract the TA and RF muscles against static resistance (with 50% MVIC of the TA), which was induced by a fixed-strap band. If the participant correctly performed ADIM and cocontraction training without pelvic rotation or compensatory upper chest elevation with overexertion, the training was considered *successful*. The proper performance of ADIM and cocontraction was confirmed by visual inspection and concurrent ultrasound and EMG measurements, which were used to carefully monitor changes in TrA/IO muscle thickness and activity sequence. Additional tactile feedback was provided if necessary.

Statistical Analysis

Standard statistical analysis included computations of means and SDs, a mixed 2×2 analysis of variance (ANOVA), a 2-tailed paired-samples t test, ICC, and standard error of measurement (SEM). The independent variables included group and time factors. The dependent variables included VAS, PDI, LBPRS, muscle thickness, EMG peak and mean amplitude, onset time, and latency. Three separate 2 (group) $\times$ 2 (time) mixed-model ANOVAs were performed to evaluate the effect of cocontraction training on increasing TrA muscle thickness using the resisted ankle dorsiflexion technique, with time (or intervention) as a within-subjects factor and 2 independent groups as a between-groups factor. Post hoc comparison using a Tukey honestly significant difference test was performed if interactions were obtained. Independent-samples t tests were used to determine differences in muscle thickness and muscle thickness ratio (contraction to rest) for TrA, IO, and EO between groups at the pretest. Additional analysis was implemented using an independent-samples t test to assess the differences in mean and peak EMG amplitudes, onset time, and latency between the control and LBP groups. A paired-samples t test also was used to assess the differences in mean and peak EMG amplitudes between baseline TrA/IO and cocontracted TrA/IO. Differences from pretest to posttest in the VAS, PDI, and LBPRS were used to assess pain in the LBP group using a paired-samples t test.

An ICC analysis was used to examine the test-retest reliability of the ultrasound measurements of abdominal muscle thickness.[37] An ICC (3,1) (2-way mixed single measure) was performed at a 95% confidence interval (CI) of the difference between the repeated ultrasound measurements of muscle thickness at 2 separate occasions (48–72 hours apart).[38,39] The *SEM* was defined as $\text{SEM} = \text{SD}(1 - \text{ICC})^{0.5}$, where SD is 1 SD of all measurements. We used SPSS (version 12.0 for Windows; SPSS Inc, Chicago, IL) The α level was set at .05 for all analyses.

RESULTS

Clinical Data

The independent-samples t test revealed no differences in age ($t_{38} = -1.98$, $P = .06$), height ($t_{38} = 0.67$, $P = .51$), or mass ($t_{38} = 0.69$, $P = .50$), which confirmed the similar demographic characteristics of the 2 groups (Table 1). Separate paired-samples t tests showed a difference in pain measurements, VAS ($t_{19} = 3.59$, $P < .001$), PDI ($t_{19} = 3.24$, $P < .001$), and LBPRS ($t_{19} = 1.98$, $P = .02$) between pretests and posttests in the LBP group (Table 2).

Ultrasound Imaging Data

A separate mixed 2×2 ANOVA showed a group-by-intervention interaction ($F_{1,38} = 6.57$, $P = .01$) and an intervention main effect for the TrA ratio ($F_{1,38} = 17.44$, $P = .001$), but this was not the case for the IO ($F_{1,38} = 0.05$, $P = .83$) and EO ($F_{1,38} = 0.40$, $P = .53$) ratios (Table 3). Further analyses using Tukey post hoc tests showed that the

Table 2. Comparison of Pain Data Obtained From the Visual Analog Scale, Low Back Pain Disability Index, and Pain Rating Scale Between Pretest and Posttest in the Low Back Pain Group (N = 20), Mean ± SD

Measure	Pretest	Posttest	Mean Difference	95% Confidence Interval	t_{19} Value	*P* Value
Visual analog scale[a]	6.15 ± 0.29	4.65 ± 0.25	−1.50	−2.15, −0.85	3.59	.001
Pain Disability Index[b]	30.95 ± 5.94	23.90 ± 4.77	−7.05	−9.63, −4.47	3.24	.001
Low back pain rating scale[c]	69.60 ± 4.59	61.60 ± 4.27	−8.00	−10.07, −5.93	1.98	.02

[a] The visual analog scale ranges from 0 to 10.
[b] The Pain Disability Index ranges from 0 to 70.
[c] The low back pain rating scale ranges from 0 to 130.

LBP group had greater improvement in the TrA muscle thickness ratio after the training than the control group ($P = .03$). No between-groups effects were observed ($P > .05$). Independent-samples *t* tests showed differences in baseline (rest) muscle thickness for TrA ($t_{38} = -7.64$, $P = .001$), IO ($t_{38} = -2.39$, $P = .02$), and EO ($t_{38} = 3.45$, $P = .001$) between groups at the pretest, but no changes in muscle thickness (muscle thickness ratio = contraction to rest) were observed ($P > .05$) (Table 4).

EMG Data

The independent-samples *t* test showed differences in the mean peak EMG amplitudes for TrA/IO ($t_{38} = -5.46$, $P = .001$), TA ($t_{38} = -3.06$, $P = .004$), and RF ($t_{38} = -3.12$, $P = .003$) but not for the cocontracted TrA/IO ($t_{38} = -1.90$, $P = .07$) between the control and LBP groups (Table 5). Differences in the mean EMG amplitudes were observed for TrA/IO ($t_{38} = -4.32$, $P = .001$), TA ($t_{38} = -3.21$, $P = .003$), and RF ($t_{38} = -4.12$, $P = .001$), but not for the cocontracted TrA/IO ($t_{38} = -1.81$, $P = .08$), between the control and LBP groups (Table 5). Differences in the mean onset time were observed for the TrA/IO ($t_{38} = 2.79$, $P = .009$) and TA ($t_{38} = 2.87$, $P = .007$) but not for the RF ($t_{38} = 1.63$, $P = .11$) between the control and LBP groups (Table 5). No differences in the mean latencies for TrA/IO-TA ($t_{38} = -0.72$, $P = .48$), TA-RF ($t_{38} = -1.49$, $P = .14$), and TrA/IO-RF ($t_{38} = -1.92$, $P = .06$) were found between groups (Table 6).

Test-Retest Reliability

The test-retest reliability analysis revealed ICCs (1,3) of 0.99 (95% CI = 0.98, 0.10; SEM = 0.02), 0.95 (95% CI = 0.82, 0.99; SEM = 0.06), and 0.96 (95% CI = 0.85, 0.99; SEM = 0.03) for the TrA, IO, and EO muscles, respectively.

DISCUSSION

Our clinical evidence demonstrated the potential efficacy of the combined cocontraction and ADIM technique for sequential motor recruitment and muscle thickness in the abdominal muscles of healthy adults and adults with chronic LBP. Treatment with the combined technique (cocontraction) effectively increased TrA muscle thickness in the LBP group. Our findings suggest that the ADIM followed by the cocontraction technique stimulates the selective recruitment of the TrA. Previously, the cocontraction technique had been studied only in healthy people rather than injured people.[16]

We used ultrasound imaging to determine a participant's ability to activate or contract the TrA using changes in the muscle thickness. McMeeken et al[31] investigated the relationship between muscle activity and thickness changes of the TrA during the ADIM using fine-wire EMG and ultrasound imaging techniques and reported a strong correlation of the 2 measures ($R^2 = 0.87$, $P < .001$). Our ultrasound imaging data are consistent with the findings of Chon et al,[16] who investigated the effect of core stabilization on muscle thickness during ADIM combined with resisted ankle dorsiflexion treatment. In our study, the TrA ratio increased 13% (from 12 to 13.6) for the patients with LBP and 3% (from 14.7 to 15.1) for healthy control participants. Independent-samples *t* tests showed differences in baseline muscle thickness for TrA, IO, and EO between groups at the pretest but no changes in muscle thickness (muscle thickness ratio = contraction to rest). The pretest differences in baseline muscle thickness between groups implied that patients with LBP had either atrophy or neuromuscular inhibition in the abdominal muscles. However, increased activation of the previously inhibited TrA after training suggests the positive benefits of ADIM and the cocontraction technique in patients with LBP.[1,5,8,40] Moreover, the effect of adding cocontraction to ADIM training seemed to be more advantageous for patients with LBP than for the control participants. As shown in Figure 6, the second TrA/IO EMG peak amplitude was greater after the cocontraction was applied. This finding suggests that the cocontraction was associated with improvements in the TrA activation, supporting the potential therapeutic efficacy of this novel technique. Researchers have shown that increases in TrA muscle thickness were associated with improved lumbar stiffness or spinal stability, contributing to pain reduction in people with LBP.[8,16]

Investigators have proposed that the recurrence of LBP is associated with delayed timing of the TrA.[6,9,14,41] Our EMG

Table 3. Comparison of Abdominal Muscle Thickness Ratio Data Between Groups (N = 40), Mean ± SD

	Low Back Pain Group (n = 20)		Control Group (n = 20)			
Abdominal Muscle	Pretest	Posttest	Pretest	Posttest	$F_{1,38}$ Value	*P* Value
Transversus abdominis, mm	12.0 ± 11.7	13.6 ± 12.0	14.7 ± 7.2	15.1 ± 7.5	6.57	.01
Internal oblique, mm	10.2 ± 1.2	9.7 ± 1.0	9.8 ± 2.8	9.5 ± 4.3	0.05	.83
External oblique, mm	9.8 ± 0.4	9.0 ± 1.1	9.9 ± 2.4	9.5 ± 2.6	0.40	.53

Table 4. Comparison of Baseline Abdominal Muscle Thickness and Ratio Data Between Groups at the Pretest (N = 40), Mean ± SD

Abdominal Muscle	Low Back Pain Group (n = 20)	Control Group (n = 20)	*t* Value	Degrees of Freedom	*P* Value
Rest					
Transversus abdominis, mm	3.0 ± 9.0	6.0 ± 1.5	−7.64	38	.001[a]
Internal oblique, mm	7.2 ± 2.6	9.3 ± 2.9	−2.39	38	.02[a]
External oblique, mm	6.1 ± 1.7	4.4 ± 1.5	3.45	38	.001[a]
Ratio					
Transversus abdominis	12.0 ± 11.7	14.7 ± 7.2	−1.66	20	.11
Internal oblique	10.2 ± 1.2	9.8 ± 2.8	0.50	26	.62
External oblique	9.8 ± 0.4	9.9 ± 2.4	−0.18	20	.86

[a] Indicates independent-samples *t* test showed differences in baseline (rest) muscle thickness data for the transversus abdominis, internal oblique, and external oblique between groups at the pretest, but no changes in muscle thickness ratio were observed.

onset-time data confirmed that initial TrA/IO, TA, and RF onset times in the LBP group were slower than those in the control group. Similarly, patients with LBP had delayed EMG latency. The mean EMG amplitudes of the patients with LBP were smaller than those of the control participants. These findings suggest that patients with LBP had altered motor-activation patterns compared with the control participants. This altered neuromuscular response has been identified as an important marker or a characteristic associated with mechanical LBP.[5,6,35,41] However, this assumption needs to be validated. After cocontraction with ADIM in our study, the impaired neuromuscular responses (peak amplitude and mean amplitude) improved more in the cocontracted TrA/IO than in the initial TrA/IO, suggesting that the cocontraction might be useful in treating activation timing factors. Our findings are consistent with those of previous investigators who demonstrated increased EMG amplitude after cocontraction training.[6,16]

Neurophysiologically, cocontraction involves motor synergies or coordinative structures whereby groups of muscles are recruited to work together as a functional unit.[18] Hence, a facilitation of the impaired TrA function in patients with LBP can be achieved by integrating the TA, quadriceps, and abdominal groups to work together as a functional core. The lumbopelvic unit was trained to demonstrate a motor pattern more similar to that of healthy people when cocontraction was added to the ADIM, as observed by the improved sequencing of the EMG-activation pattern during the cocontraction training. Researchers[8,42] have demonstrated that a combination of the isolated training of delayed TrA activation and nonisolated functional training (involving abdominal curl-ups, side bridges, and bird dogs) was beneficial for pain and functional improvement in patients with LBP. Cowan et al[43] found that delayed feed-forward activation of the medial quadriceps muscle in people with patellofemoral pain was enhanced with comprehensive isolated and nonisolated contraction training. A combination of isolated training (initial ADIM of delayed TrA/IO) and nonisolated training involving cocontraction of the TA and RF helped to restore delayed TrA activation, which is a consistent promarker of abdominal neuromuscular dysfunction in LBP. Hence, earlier activation of the TrA during the cocontraction training as reflected in our EMG onset-time data can be considered an important indicator of improved neuromuscular control. This improved neuromuscular re-

Table 5. Electromyographic Peak Amplitude, Mean Amplitude, and Mean Onset Time Data (Root Mean Square) Between Groups During Cocontraction Training, Mean ± SD[a]

Abdominal Muscle	Low Back Pain Group (n = 20)	Control Group (n = 20)	t_{38} Value	*P* Value
Transversus abdominis and internal oblique				
Peak amplitude, μV	87.27 ± 19.76	151.23 ± 48.54	−5.46	.001
Mean amplitude, μV	65.08 ± 20.07	106.71 ± 38.14	−4.32	.001
Onset time, s	4.15 ± 0.69	3.68 ± 0.34	2.79	.009
Tibialis anterior				
Peak amplitude, μV	71.20 ± 16.71	89.83 ± 21.55	−3.06	.004
Mean amplitude, μV	43.79 ± 15.86	63.81 ± 22.99	−3.21	.003
Onset time, s	6.72 ± 0.39	6.39 ± 0.33	2.87	.007
Rectus femoris				
Peak amplitude, μV	76.77 ± 23.41	99.42 ± 22.54	−3.12	.003
Mean amplitude, μV	44.29 ± 17.71	68.63 ± 19.65	−4.12	.001
Onset time, s	6.89 ± 0.35	6.73 ± 0.25	1.63	.11
Cocontracted transversus abdominis and internal oblique				
Peak amplitude, μV	159.18 ± 30.94[c]	181.73 ± 43.01[c]	−1.90	.07
Mean amplitude, μV	111.38 ± 37.59[c]	134.09 ± 41.54[c]	−1.81	.08
Onset time, s[b]	NA	NA	NA	NA

Abbreviation: NA, not applicable.

[a] The tibialis anterior and rectus femoris muscles were cocontracted and were followed by the initial onset of transversus abdominis and internal oblique muscle activation.

[b] Indicates onset time was not determined because of additive contraction.

[c] Indicates paired-samples *t* test showed a difference between transversus abdominis and internal oblique and cocontracted transversus abdominis and internal oblique ($P < .05$).

Table 6. Electromyographic Latency Between Groups During Cocontraction Training, Mean ± SD

Latency	Low Back Pain Group (n = 20)	Control Group (n = 20)	t_{38} Value	*P* Value
Transversus abdominis and internal oblique-tibialis anterior, ms	2.57 ± 0.74	2.72 ± 0.52	−0.72	.48
Tibialis anterior-rectus femoris, ms	0.16 ± 0.42	0.34 ± 0.28	−1.49	.14
Transversus abdominis and internal oblique-rectus femoris, ms	2.73 ± 0.57	3.06 ± 0.45	−1.92	.06

sponse has greater force-generating potential and an enhanced ability to increase spinal stiffness, resulting in improvements in pain, function, and recurrence rates in patients with LBP.[8,12,44] Perhaps EMG could be used to provide accurate information about patterns and sequences of motor activation.

The LBP group included people who had recurrent mechanical back pain and had not improved with nonoperative treatments. In participants with LBP, we observed a reduction of pain and improvement in function—specifically improvements in VAS, PDI, and LBPRS—after the intervention. Our findings are consistent with those of O'Sullivan et al,[8] who showed that engaging in ADIM exercise for 15 minutes each day for 10 weeks reduced the VAS scores of patients with spondylolysis or spondylolisthesis from 6 to 2. Kumar et al[40] reported that the administration of the ADIM in combination with various core exercises for 5 weeks in patients with chronic LBP reduced VAS scores from 7 to 1.

Our results have several important clinical implications. They show that ADIM training is beneficial for the selective recruitment of the TrA and its central mechanism of action on the lumbopelvic region and that the mechanism of the deep musculofascial corset might be augmented further by the cocontraction technique. Evidence of the clinical management of LBP that Hides et al[3] reported suggests that the support and protection of the spine is essential to stiffening the lumbosacroiliac joints during selective core stabilization training of the TrA, minimizing clinical reports of LBP and lumbar spinal instability.

Our test-retest reliability data suggested a good degree of reliability in our repeated ultrasound measurements, which is in contrast to the relatively poor degree of reliability that Mannion et al[45] and Hodges et al[46] reported. Others have demonstrated a good to high degree of reliability.[31,47,48] Our higher degree of test-retest reliability might be due to our consistent use of a transparent sheet and static position measurement to control for potential errors associated with the inconsistent location of ultrasound applications and movement artifacts. Our findings corroborate existing evidence showing that the abdominal thickness measurements obtained from ultrasound imaging are accurate and reliable. Hence, such measurements are good indicators of intervention-related thickness changes and associated motor-control mechanisms.

Our study had several shortcomings that should be addressed in a more robust and large-scale clinical study. First, ultrasound-guided visual feedback at pretest might have affected outcome results in muscle-thickness measures. Hence, in the future, visual feedback should be excluded in the pretest. Second, the permanent changes in muscle thickness are unlikely to occur within such a short duration of strength training. Authors of the motor-learning literature have shown that corticospinal excitability occurs within the first 2 weeks of training, when the main improvement in motor performance is achieved, and reaches a significant level after 4 weeks of training.[49–52] The long-term effect of such intervention needs further exploration. Third, the function of the multifidus, which provides segmental stability, was not measured. Further probing the mechanisms of action in these muscles would be of great interest.[53] Fourth, our results cannot be generalized because of limited sample size and our case-control study design. A larger clinical trial involving a true control group with LBP is needed to investigate the therapeutic effects of the resisted dorsiflexion-contraction training to augment TrA/IO in clinical practice.

CONCLUSIONS

We highlighted the potential application of ADIM along with cocontraction in a group of healthy participants and a group of participants with LBP. We demonstrated increased muscle thickness and the associated reduction of LBP after the intervention. The added cocontraction training could be integrated as a part of a core stabilization regimen for the management of patients with LBP, but further study is needed to validate its therapeutic efficacy.

ACKNOWLEDGMENTS

This work was supported in part by Yonsei University Research Fund of 2005. We thank our research assistants, Won-Ju Lee and Sung-Jae Kim; Yong-Han Kim, PT; and Yong-Ho Cho, MD (orthopaedist), for technical support in data collection and participant recruitment. We also thank the patients at Daejeon Orthopedic Clinic for their participation in our study.

REFERENCES

1. Cairns MC, Foster NE, Wright C. Randomized controlled trial of specific spinal stabilization exercises and conventional physiotherapy for recurrent low back pain. *Spine (Phila Pa 1976)*. 2006;31(19):E670–E681.
2. Standaert CJ, Herring SA. Expert opinion and controversies in musculoskeletal and sports medicine: core stabilization as a treatment for low back pain. *Arch Phys Med Rehabil*. 2007;88(12):1734–1736.
3. Hides J, Wilson S, Stanton W, et al. An MRI investigation into the function of the transversus abdominis muscle during "drawing-in" of the abdominal wall. *Spine (Phila Pa 1976)*. 2006;31(6):E175–E178.
4. Pengel LH, Herbert RD, Maher CG, Refshauge KM. Acute low back pain: systematic review of its prognosis. *BMJ*. 2003;327(7410):323.
5. Hodges PW, Richardson CA. Inefficient muscular stabilization of the lumbar spine associated with low back pain: a motor control evaluation of transversus abdominis. *Spine (Phila Pa 1976)*. 1996;21(22):2640–2650.
6. Hall L, Tsao H, MacDonald D, Coppieters M, Hodges PW. Immediate effects of co-contraction training on motor control of the trunk muscles in people with recurrent low back pain. *J Electromyogr Kinesiol*. 2009;19(5):763–773.
7. Vera-Garcia FJ, Elvira JL, Brown SH, McGill SM. Effects of abdominal stabilization maneuvers on the control of spine motion and stability against sudden trunk perturbations. *J Electromyogr Kinesiol*. 2007;17(5):556–567.

8. O'Sullivan PB, Phyty GD, Twomey LT, Allison GT. Evaluation of specific stabilizing exercise in the treatment of chronic low back pain with radiologic diagnosis of spondylolysis or spondylolisthesis. *Spine (Phila Pa 1976)*. 1997;22(24):2959–2967.
9. Butler HL, Hubley-Kozey CL, Kozey JW. Changes in trunk muscle activation and lumbar-pelvic position associated with abdominal hollowing and reach during a simulated manual material handling task. *Ergonomics*. 2007;50(3):410–425.
10. Stanton T, Kawchuk G. The effect of abdominal stabilization contractions on posteroanterior spinal stiffness. *Spine (Phila Pa 1976)*. 2008;33(6):694–701.
11. Akuthota V, Nadler SF. Core strengthening. *Arch Phys Med Rehabil*. 2004;85(3 suppl 1):S86–S92.
12. Kavcic N, Grenier S, McGill SM. Quantifying tissue loads and spine stability while performing commonly prescribed low back stabilization exercises. *Spine (Phila Pa 1976)*. 2004;29(20):2319–2329.
13. McGill SM. The biomechanics of low back injury: implications on current practice in industry and the clinic. *J Biomech*. 1997;30(5):465–475.
14. Ferreira PH, Ferreira ML, Hodges PW. Changes in recruitment of the abdominal muscles in people with low back pain: ultrasound measurement of muscle activity. *Spine (Phila Pa 1976)*. 2004;29(22):2560–2566.
15. Nordin M, Weiner SS, Lindh M. Biomechanics of the lumbar spine. In: Nordin M, Frankel VH, eds. *Basic Biomechanics of the Musculoskeletal System*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins; 2001:256–285.
16. Chon SC, Chang KY, You SH. Effect of the abdominal draw-in maneuver in combination with ankle dorsiflexion in strengthening the transverse abdominal muscle in healthy young adults: a preliminary, randomised, controlled study. *Physiotherapy*. 2010;96(2):130–136.
17. Eccles JC, Sherrington CS. Reflex summation in the ipsilateral spinal flexion reflex. *J Physiol*. 1930;69(1):1–28.
18. Torres-Oviedo G, Macpherson JM, Ting LH. Muscle synergy organization is robust across a variety of postural perturbations. *J Neurophysiol*. 2006;96(3):1530–1546.
19. Teyhen DS, Miltenberger CE, Deiters HM, et al. The use of ultrasound imaging of the abdominal drawing-in maneuver in subjects with low back pain. *J Orthop Sports Phys Ther*. 2005;35(6):346–355.
20. Walker BF, Williamson OD. Mechanical or inflammatory low back pain: what are the potential signs and symptoms? *Man Ther*. 2009;14(3):314–320.
21. Brown A, Snyder-Mackler L. Diagnosis of mechanical low back pain in a laborer. *J Orthop Sports Phys Ther*. 1999;29(9):534–539.
22. Love A, Leboeuf C, Crisp TC. Chiropractic chronic low back pain sufferers and self-report assessment methods, part I: a reliability study of the Visual Analogue Scale, the Pain Drawing and the McGill Pain Questionnaire. *J Manipulative Physiol Ther*. 1989;12(1):21–25.
23. Jensen MP, Chen C, Brugger AM. Postsurgical pain outcome assessment. *Pain*. 2002;99(1–2):101–109.
24. Boonstra AM, Schiphorst Preuper HR, Reneman MF, Posthumus JB, Stewart RE. Reliability and validity of the visual analogue scale for disability in patients with chronic musculoskeletal pain. *Int J Rehabil Res*. 2008;31(2):165–169.
25. Tait RC, Chibnall JT. Factor structure of the pain disability index in workers compensation claimants with low back injuries. *Arch Phys Med Rehabil*. 2005;86(6):1141–1146.
26. Grönblad M, Hupli M, Wennerstrand P, et al. Intercorrelation and test-retest reliability of the Pain Disability Index (PDI) and the Oswestry Disability Questionnaire (ODQ) and their correlation with pain intensity in low back pain patients. *Clin J Pain*. 1993;9(3):189–195.
27. Manniche C, Asmussen K, Lauritsen B, Vinterberg H, Kreiner S, Jordan A. Low Back Pain Rating scale: validation of a tool for assessment of low back pain. *Pain*. 1994;57(3):317–326.
28. Childs JD, Piva SR, Fritz JM. Responsiveness of the numeric pain rating scale in patients with low back pain. *Spine (Phila Pa 1976)*. 2005;30(11):1331–1334.
29. Mannion AF, Pulkovski N, Toma V, Sprott H. Abdominal muscle size and symmetry at rest and during abdominal hollowing exercises in healthy control subjects. *J Anat*. 2008;213(2):173–182.
30. Whittaker JL. Ultrasound imaging of the lateral abdominal wall muscles in individuals with lumbopelvic pain and signs of concurrent hypocapnia. *Man Ther*. 2008;13(5):404–410.
31. McMeeken JM, Beith ID, Newham DJ, Milligan P, Critchley DJ. The relationship between EMG and change in thickness of transversus abdominis. *Clin Biomech (Bristol, Avon)*. 2004;19(4):337–342.
32. Rankin G, Stokes M. Reliability of assessment tools in rehabilitation: an illustration of appropriate statistical analyses. *Clin Rehabil*. 1998;12(3):187–199.
33. Marshall P, Murphy B. The validity and reliability of surface EMG to assess the neuromuscular response of the abdominal muscles to rapid limb movement. *J Electromyogr Kinesiol*. 2003;13(5):477–489.
34. Cram JR, Kasman GS, Holtz J. Electrode placement. In: *Introduction to Surface Electromyography*. Gaithersburg, MD: Aspen Publishers; 1998:360–371.
35. Roussel N, Nijs J, Truijen S, Vervecken L, Mottram S, Stassijns G. Altered breathing patterns during lumbopelvic motor control tests in chronic low back pain: a case-control study. *Eur Spine J*. 2009;18(7):1066–1073.
36. Hodges P, Richardson C, Jull G. Evaluation of the relationship between laboratory and clinical tests of transversus abdominis function. *Physiother Res Int*. 1996;1(1):30–40.
37. Bland JM, Altman DG. Statistical methods for assessing agreement between two methods of clinical measurement. *Lancet*. 1986;1(8476):307–310.
38. Hopkins WG. Measures of reliability in sports medicine and science. *Sports Med*. 2000;30(1):1–15.
39. Shrout PE, Fleiss JL. Intraclass correlations: uses in assessing rater reliability. *Psychol Bull*. 1979;86(2):420–428.
40. Kumar S, Sharma VP, Negi MP. Efficacy of dynamic muscular stabilization techniques (DMST) over conventional techniques in rehabilitation of chronic low back pain. *J Strength Cond Res*. 2009;23(9):2651–2659.
41. Hodges PW. Changes in motor planning of feedforward postural responses of the trunk muscles in low back pain. *Exp Brain Res*. 2001;141(2):261–266.
42. Stuge B, Veierod MB, Laerum E, Bragelian M, Vollestad N. The efficacy of a treatment program focusing on specific stabilizing exercises for pelvic girdle pain after pregnancy: a two-year follow-up of a randomized clinical trial. *Spine (Phila Pa 1976)*. 2004;29(10):E197–E203.
43. Cowan SM, Bennell KL, Hodges PW, Crossley KM, McConnell J. Simultaneous feedforward recruitment of the vasti in untrained postural tasks can be restored by physical therapy. *J Orthop Res*. 2003;21(3):553–558.
44. Cholewicki J, McGill SM. Mechanical stability of the in vivo lumbar spine: implications for injury and chronic low back pain. *Clin Biomech (Bristol, Avon)*. 1996;11(1):1–15.
45. Mannion AF, Pulkovski N, Gubler D, et al. Muscle thickness changes during abdominal hollowing: an assessment of between-day measurement error in controls and patients with chronic low back pain. *Eur Spine J*. 2008;17(4):494–501.
46. Hodges PW, Pengel LH, Herbert RD, Gandevia SC. Measurement of muscle contraction with ultrasound imaging. *Muscle Nerve*. 2003;27(6):682–692.
47. Hides JA, Miokovic T, Belavý DL, Stanton WR, Richardson CA. Ultrasound imaging assessment of abdominal muscle function during drawing-in of the abdominal wall: an intrarater reliability study. *J Orthop Sports Phys Ther*. 2007;37(8):480–486.

48. Hebert JJ, Koppenhaver SL, Parent EC, Fritz JM. A systematic review of the reliability of rehabilitative ultrasound imaging for the quantitative assessment of the abdominal and lumbar trunk muscles. *Spine (Phila Pa 1976)*. 2009;34(23):E848–E856.
49. Jensen JL, Marstrand PC, Nielsen JB. Motor skill training and strength training are associated with different plastic changes in the central nervous system. *J Appl Physiol*. 2005;99(4):1558–1568.
50. Legg SJ. The effect of abdominal muscle fatigue and training on the intra-abdominal pressure developed during lifting. *Ergonomics*. 1981;24(3):191–195.
51. Abe T, DeHoyos DV, Pollock ML, Garzarella L. Time course for strength and muscle thickness changes following upper and lower body resistance training in men and women. *Eur J Appl Physiol*. 2000;81(3):174–180.
52. MacDougall JD, Gibala MJ, Tarnopolsky MA, MacDonald JR, Interisano SA, Yarasheski KE. The time course for elevated muscle protein synthesis following heavy resistance exercise. *Can J Appl Physiol*. 1995;20(4):480–486.
53. MacDonald DA, Moseley GL, Hodges PW. The lumbar multifidus: does the evidence support clinical beliefs? *Man Ther*. 2006;11(4): 254–263.

Address correspondence to Joshua H. You, PhD, PT, Department of Physical Therapy, Yonsei University, and Movement Healing Laboratory (Wellness, Fitness, and Prevention/Movement Impairment/Sports Medicine and Movement Science), 234 Heoungup-Myon, MaeJi-Ri, Wonju City, Kangwon-do, 220–710 Republic of South Korea. Address e-mail to neurorehab@yonsei.ac.kr.