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Changes in Postural Control After a Ball-Kicking Balance Exercise in Individuals With Chronic Ankle Instability

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Context: Rehabilitation programs for patients with chronic ankle instability (CAI) generally involve balance-perturbation training (BPT). Anticipatory postural adjustments (APAs) and compensatory postural adjustments (CPAs) are the primary strategies used to maintain equilibrium during body perturbations. Little is known, however, about how APAs and CPAs are modified to promote better postural control for individuals with CAI after BPT.

Objective: To investigate the effect of BPT that involves kicking a ball on postural-control strategies in individuals with CAI.

Design: Randomized controlled clinical trial.

Setting: Laboratory.

Patients or Other Participants: We randomly assigned 44 volunteers with CAI to either a training group (TG; 11 women, 11 men; age = 24 ± 4 years, height = 173.0 ± 9.8 cm, mass = 72.64 ± 11.98 kg) or control group (CG; 11 women, 11 men; age = 22 ± 3 years, height = 171.0 ± 9.7 cm, mass = 70.00 ± 11.03 kg).

Intervention(s): The TG performed a single 30-minute training session that involved kicking a ball while standing on 1 foot. The CG received no intervention.

Main Outcome Measure(s): The primary outcome was the sum of the integrated electromyographic activity ($\sum\int$EMG) of the lower extremity muscles in the supporting limb that were calculated during typical intervals for APAs and CPAs. A secondary outcome was center-of-pressure displacement during similar intervals.

Results: In the TG after training, the $\sum\int$EMG decreased in both dorsal and ventral muscles during compensatory adjustment (ie, the time interval that followed lower limb movement). During this interval, muscle activity ($\sum\int$EMG) was less in the TG than in the CG. Consequently, center-of-pressure displacement increased during the task after training.

Conclusions: A single session of ball-kicking BPT promoted changes in postural-control strategies in individuals with CAI. These results should stimulate new and more comprehensive studies to investigate the effect of this and other BPT techniques on postural control in patients with CAI.

Key Words: recurrent sprains, reactive responses, balance training

Key Points

- After balance-perturbation training, postural sway increased during the ball-kicking activity and decreased during a static task in individuals with chronic ankle instability.
- Ventral and dorsal muscle activity decreased just before kicking the ball, and tibialis anterior and peroneus longus activity increased after the kick in the balance-perturbation–training group.
- Researchers should conduct more comprehensive studies to determine if balance-perturbation training improves postural control and simultaneously augments ankle stability in individuals with chronic ankle instability.

Lateral ankle sprain is one of the most common sports injuries and largely affects young adults who are involved in recreational or sporting activities, such as basketball, football, or soccer.[1] After the first injury, a substantial number of these individuals experience the sensation of ankle instability and recurrent sprains, a condition known as *chronic ankle instability* (CAI).[2] Balance deficits are among the main symptoms of CAI, usually verified through impaired standing stability[3] or increased displacement of the center of pressure (COP) when these individuals stand motionless on 1 lower extremity with their eyes open or closed.[4]

For this reason, a common treatment for rehabilitating patients with CAI is balance training.[5] This includes maneuvers such as balancing on 1 limb while postural disturbances are delivered in various ways (eg, standing on different unstable surfaces); physical perturbation via a push or pull; or throwing, catching, or kicking a ball.[6–10] Whereas some of these therapeutic techniques yielded positive outcomes for balance control and ankle-joint stability,[11,12] others have been less successful.[13,14] One potential explanation for these conflicting findings is limited understanding about how individuals with CAI react to these disturbances. Thus, measuring their

postural control during actual perturbation tasks might yield useful information because such tasks and conditions are often accompanied by a sensation of ankle instability.

In a recent study,[15] participants with CAI experienced a remarkable decrease in COP excursion relative to individuals without CAI while kicking a ball in a single-legged stance, which is anecdotally a perturbation training technique commonly used at athletic clubs and clinical sites to train and rehabilitate those with CAI. The authors suggested that individuals with CAI should increase the stiffness of their lower extremities via neuromuscular mechanisms during this type of activity to decrease the risk of recurrent ankle sprain. Therefore, postural sway during dynamic activities might increase after balance training as individuals become familiar with task demands and their anxiety levels about ankle instability decrease.

The postural-control strategies used while standing motionless on 1 limb versus those used while performing functional activities are quite different. In the former, one must restrict movement of the supporting limb's articulations, and, hence, one's center of mass or COP, as much as possible.[16] With the latter, one might need to achieve greater joint-angle excursions to enhance functional task performance. Typically, individuals with CAI exhibit greater postural sway when standing motionless on 1 limb than do healthy control participants, but this disparity decreases with balance training.[12,17] The opposite effect may occur in dynamic conditions, such as performing functional tasks; that is, with balance training, balance sway may increase. For instance, individuals with CAI improved their dynamic postural control (ie, increased excursion during a Star Excursion Balance Test) after training involving various balance activities in a single-legged stance[11] or balancing on an unstable surface.[10] Therefore, the results of these studies suggested that increased COP excursion while performing dynamic tasks might not necessarily indicate decreased postural stability, as observed in static conditions. This hypothesis needs to be tested, as it could affect the way postural instability is tested in patients with CAI.

When responding to balance perturbations, anticipatory postural adjustments (APAs) and compensatory postural adjustments (CPAs) are the main types of postural strategies used to preserve body equilibrium. *Anticipatory adjustments* consist of subtle contractions of postural muscles and slight body movement, both of which transpire before any perturbation occurs and are used to minimize any potential postural disturbance.[18] *Compensatory adjustments* are responses to the perturbation itself, with muscles activated and body movements enacted to counteract postural perturbation that has already occurred.[19] These APAs and CPAs can be observed and analyzed by measuring electromyographic (EMG) activity within the postural muscles,[20] COP and center-of-mass displacement,[15] articular movements,[21] or any combination of these. Investigating the effects of balance-perturbation training (BPT) on these postural strategies in individuals with CAI might explain how they alter the control of their body to perform given dynamic tasks.

Investigators have presented evidence that postural-control strategies can be modified by balance training[22] and physical activities.[23] For instance, exercises focused on balance improvement, such as the Tai Chi Chuan method, reduced the APAs of multiple muscles and improved postural stability.[22] Furthermore, BPT that involved a single session, such as standing on 1 limb on a Swiss ball,[24] throwing a ball,[25] or performing trunk-stabilization exercises,[26] appeared to promote changes in static and dynamic balance control. Whereas balance-training techniques that incorporate postural perturbations have been used widely in clinics and sports clubs to improve balance and potentially decrease recurrent sprains in patients with CAI, the effects of these training techniques on postural-control strategies remain unknown. Understanding how these strategies are modified by training could help researchers and clinicians improve existing techniques or develop new and more effective rehabilitation interventions to restore postural stability in patients with CAI.

Therefore, the purpose of our pilot study was to investigate the immediate effects of BPT on postural-control strategies (APA and CPA) in individuals with CAI during dynamic and static tasks. The EMG activity of lower extremity muscles and COP displacement were recorded and evaluated during the time intervals typical for APAs and CPAs in 2 CAI patient groups (trained, untrained) at 2 time points roughly 30 minutes apart. Based on the results of previous studies, we hypothesized that differences would exist between groups after training, including a decreased magnitude of APAs and CPAs in trained versus untrained participants and a consequent increase in COP displacement during the dynamic task. We also hypothesized that training would decrease COP displacement during the static task.

METHODS

Study Design

This was a controlled, single-blinded, randomized pilot study, with the data evaluator (J.C.) blinded to treatment arm allocations. An independent researcher (F.A.) randomly assigned participants to either a training group (TG) or control group (CG) via numeric randomization stratified by sex (2×2). This researcher had no knowledge of the relationship between the numeric codes and the experimental conditions, and participants were blinded to the study's outcomes of interest and a priori hypotheses. Participants in the TG received a 30-minute balance-training session, and participants in the CG received no training. The study's primary outcome was the sum of integrated EMG activity ($\sum\int$EMG) for all ventral and dorsal lower extremity muscles; the secondary outcomes were (1) variations in COP displacement and (2) the integrated EMG activity ($\int$EMG) for each muscle calculated individually.

Participants

The evaluator who was blinded to intervention allocation recruited 44 physically active individuals with CAI from Santa Catarina State University and the surrounding metropolitan area in the first semester of 2013. We defined

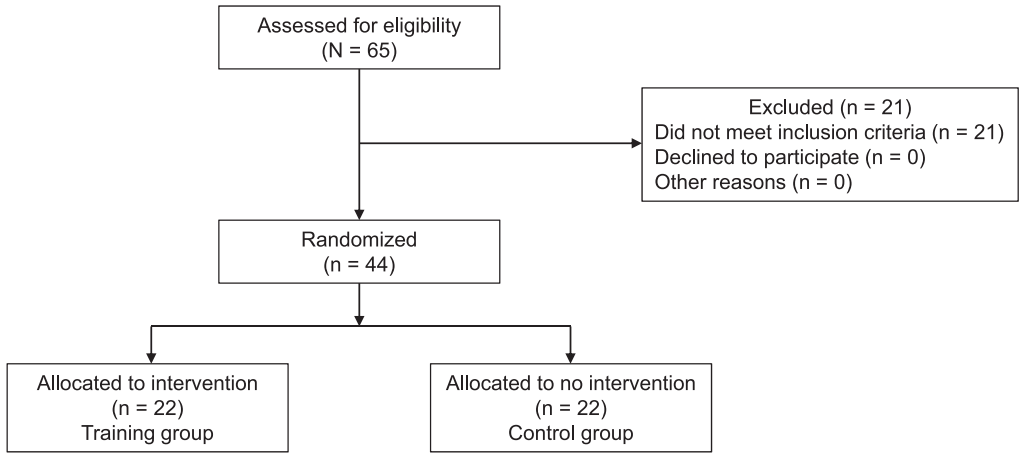

Figure 1. Participant flow chart.

physically active as exercising for at least 30 minutes per day for 3 days per week.[27] Twenty-two individuals were allocated randomly to the active TG (11 women, 11 men; age = 24 ± 4 years [range = 19–30 years], height = 173.0 ± 9.8 cm, mass = 72.64 ± 11.98 kg), and 22 participants were allocated to the CG (11 women, 11 men; age = 22 ± 3 years [range = 18–30 years], height = 171.0 ± 9.7 cm, mass = 70.00 ± 11.03 kg; Figure 1). We recorded each participant's sex; age; anthropometric measures; number of past sprains; and *limb dominance*, which was defined as the preferred limb for kicking a ball. The eligibility criteria for both groups were (1) age from 18 to 30 years; (2) history of 2 or more ankle sprains, with at least 1 sprain within the 6 months before the study; (3) a sensation of ankle instability (eg, feeling that the ankle was "giving way" during functional activities [work, leisure, or sports]); and (4) a score of less than 28 on the Brazilian-Portuguese validated version of the Cumberland Ankle Instability Tool (CAIT).[28] We used the most severely affected ankle, which was the one with the lowest CAIT score, for analysis in participants who reported bilateral instability. Exclusion criteria were (1) acute signs of inflammation (joint pain, redness, or swelling) in the ankle or (2) a history of fracture, rheumatic or neurologic problems or any other pathologic condition in the lower extremity that could interfere with the completion of the tasks required for the study. Potential participants were interviewed over the telephone and, once selected, were instructed to visit the laboratory where an evaluation (performed by J.C.) confirmed all eligibility criteria. Experimental procedures were performed only after volunteers agreed to participate in the research. An independent researcher with no other role in the study had access to the randomization results. The physiotherapist (facilitator; F.A.) responsible for the application of the intervention only learned which group a participant belonged to after pretrial data collection and before any training.

We calculated the sample size a priori using an effect size of 1.15 based on a previous study[23] and considering our primary outcome measurement ($\sum$EMG of the lower extremity muscles). Power analysis indicated the need for 18 participants in each group to detect differences between groups with 80% power and an α level of .05. We recruited 22 individuals to each group to compensate for possible dropouts. All participants provided written informed consent, and the study was approved by the Santa Catarina State University Ethics Committee in Research Involving Human Beings (protocol number 205/2011). We registered this study with the Registro Brasileiro de Ensaios Clínicos under protocol number RBR-8d67bt. Participants were instructed not to engage in any stressful physical activity during the 24 hours before testing.

Instruments

The CAIT was used to evaluate the severity of ankle-joint instability. Scores range from 0 to 30, with lower scores indicating more severe instability.

We used a force platform (model AMTI-OR 6-7; AMTI, Inc, Watertown, MA), which was positioned on the floor, to register ground reaction forces (Fx, Fy, and Fz) and associated moments (Mx, My, and Mz). Using these measurements, we calculated COP displacement. For the acquisition and monitoring of muscular electrical activity, we used an electromyograph (model 811C; EMG System of Brazil, São José dos Pinhais, Sao Paulo, Brazil) with a gain of 2000, an analog band-pass filter with a frequency from 23 to 500 Hz, a common mode rejection ratio greater than 80 dB, and a differential amplifier. To register the moment in which the posture was perturbed, we used an accelerometer (model ACL13000/03; EMG System of Brazil) with a tri-axial configuration. All signals reached the computer through an analog-to-digital conversion board (model PCI 6259; National Instruments, Austin, TX) with a frequency of 1000 Hz and resolution of 16 bits and were acquired in a LabVIEW environment (version 4.0; National Instruments).

Experimental Procedure

We cleaned disposable monopolar surface Ag/AgCl electrodes (Kendall Medi-trace 200; Medtronic PLC, São Sebastião do Paraíso, Minas Gerais, Brazil) with 70% isopropyl alcohol and affixed them to the participants' skin with a circular gel area diameter of 10 mm. These electrodes were placed at an interelectrode distance of 20 mm on the limb with CAI over the following muscles:

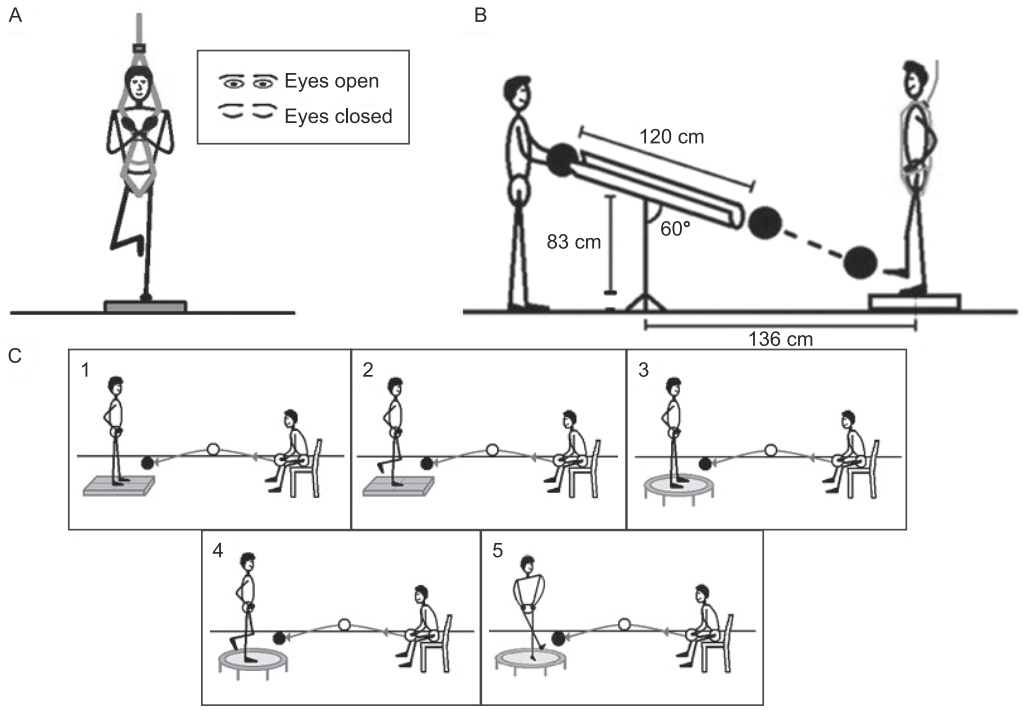

Figure 2. Representation of the experimental tasks and balance training protocol. A, Experiment 1: the single-legged stance during the static task. B, Experiment 2: the ball launcher and a participant performing the kicking task (dynamic task). C, The balance-training protocol.

tibialis anterior (TA), peroneus longus (PL), medial head of the gastrocnemius (GasM), lateral head of the gastrocnemius (GasL), soleus (SOL), rectus femoris (RF), and biceps femoris (BF). This limb was also the supporting limb during the experimental tasks. We positioned additional electrodes over the adductor longus muscle of the opposite limb (ie, the limb that performed the kicking task). We attached a reference electrode over the medial malleolus of the supporting limb and affixed the accelerometer to the lateral malleolus of the kicking limb. The same investigator who was single blinded to the intervention performed all described procedures in accordance with recommendations published for the Surface ElectroMyoGraphy for the Non-Invasive Assessment of Muscles (SENIAM) project.[29]

In the laboratory, we constructed a ball launcher to ensure homogeneity of the experimental tasks among trials and among participants. dos Santos et al[15] described this launcher in detail. In short, a 120-cm polyvinyl chloride pipe was cut in half longitudinally and coupled to an adjustable tripod. The pipe in which the ball rolled after being launched, was inclined 30° downward toward the participant's foot. Our preliminary tests indicated that using this device allowed the official 350-g futsal ball to reach each participant with similar latency, height, and speed when launched. In addition, dos Santos et al[15] observed that the kicking time was similar among participants and different experimental conditions (even on an inclined support surface).

Experiment 1 (Static Task). The goal of our first experiment was to investigate balance sway while participants were standing motionless on 1 limb. Authors[12,17,30–32] of multiple studies involving participants with CAI have shown changes in COP variables during this task resulting from BPT. Center-of-pressure variables have also been shown to be sensitive to change for athletes in quiet unipedal stance after 1 session of proprioceptive training using an unstable platform.[24]

Participants stood barefoot on their affected limb on the force platform. They positioned the opposite limb with the hip in neutral and knee flexed to 90°. We instructed them not to let the suspended limb touch the planted limb or the support surface during data collection. We also instructed them to stand still, cross their upper extremities in front of their chests, and focus their eyes on a small black circle located 4 m in front of them (eyes open [EO]). They also performed the same task with their eyes closed (EC). We determined the order of these 2 conditions randomly by a simple raffle for each participant. They attempted the task for 10 seconds 5 times in each condition while we collected data, with 10-second rests between attempts (Figure 2A). If the participant could not remain in a single-legged stance for 10 seconds, we discarded the results and repeated the trial.

Experiment 2 (Dynamic Task). The aim of this experiment was to investigate postural-control strategies (APA, CPAs) during external postural perturbations. We again positioned the barefoot participants in a single-legged stance with their affected limbs on the force platform and their hands on their waists. We instructed them to suspend the other limb in slight knee flexion with the hip laterally rotated and the ankle in a neutral position; in this position, the heel of the elevated foot was at the same height as the medial malleolus of the supporting limb. We placed the tripod (center) with the ball launcher at a distance of 136

cm from the center of the force-plate surface on which the participant stood (Figure 2B).

One researcher, who was not an author, positioned the ball at the top of the inclined ball launcher and released it toward the suspended foot. The experimenter then instructed the participant to kick the ball back toward him, aiming through 2 widely spaced wooden rods that were 39 cm high and 80 cm apart and positioned in front of the ball launcher (Figure 2B). The paired rods were intended to provide participants with a reference point rather than a target while kicking. Because the ball was always released from the same height and distance, participants received similar perturbations throughout the sequence of experimental tasks.

In this latter experiment, we subjected the participants to 2 successive perturbations: first, internal perturbation, which the individual generated by moving his or her own limbs, and second, external perturbation, which was the force of the ball during the generated impact. The time intervals APA, CPA1, and CPA2 corresponded to events before limb motion, before the kick, and after the kick, respectively. In particular, CPA1 was compensatory to limb movement but was anticipatory because of the perturbation caused by the ball's impact. We arbitrarily decided to name this interval *CPA*. Participants performed a series of 5 kicks while we collected data.

Before data collection, participants performed 3 trials to familiarize themselves with the procedure. We instructed them to say "Okay" at the exact moment they perceived stable balance for all attempts and conditions. After they said "Okay," we started data collection. Throughout the study, participants wore a harness to prevent falls (Figure 2A and B). The same researchers (J.C., F.A.) performed all procedures for both experiments.

Balance Training

For the TG, experiments 1 and 2 were conducted before and after 1 session of balance training. The CG also performed the tasks twice (before and after resting for 30 minutes) with no training between the 2 trials. Training sessions involved progressively increasing the level of neuromuscular demand over the 30-minute period because we intended to replicate perturbation training that is used widely in clinical practice by clinicians and in sports clubs, especially with active individuals and athletes, including soccer players. Participants followed the training protocol after a warmup, which included ankle dorsiflexion–plantar flexion and inversion–eversion movements for 2 minutes. Balance training consisted of kicking a ball launched by an investigator in the following, progressively altered conditions: (1) double-legged stance on a rigid surface, (2) single-legged stance on a rigid surface, (3) double-legged stance on an unstable surface (mini-trampoline [model Trampolim Semi-Pro; Polimet, Boituva, Sao Paulo, Brazil]), (4) single-legged stance on the mini-trampoline, and (5) as for condition 4 but with the participant positioned obliquely relative to the therapist with approximately 45° between the frontal and sagittal body planes (Figure 2C). For all these conditions, the *supporting limb* was the limb with CAI or the most severely affected limb when symptoms were bilateral, whereas the *kicking limb* was the uninjured or less affected limb. During balance training, an experimenter, who was not an author, stood close to the participant to prevent falls.

Participants in the TG performed a total of 15 kicks in each of the described conditions. The researcher who released the ball always sat 170 cm away from them, enabling homogeneous ball trajectories and speeds. During all training conditions, the investigator instructed participants to kick the ball back toward him, keeping their postural balance as stable as possible after the kick. Participants repeated trials in which they were unable to maintain their balance or kick the ball back to the experimenter. They rested for about 2 minutes between conditions; the total training period lasted approximately 20 minutes. After training was completed, participants rested for 10 minutes before posttraining data collection. We used this rest time to minimize any effect of muscle fatigue on postural control.[33]

Data Processing

We calculated COP displacement in both the anteroposterior and mediolateral directions, as described by Claudino et al.[34] In experiment 1, we calculated the 95% confidence interval (CI) for the sway area (ellipse) of COP displacement during the static task ($sCOP_A$) both with EO and EC over the entire 10-second interval of the task. This measurement represents postural stability throughout the static task.

For experiment 2, we combined a computer algorithm and visual inspection of the accelerometer signals to detect the timing of limb-movement onset and ball impact. We defined *limb-movement onset* as the time at which the signal reached 5% of its acceleration peak. We demarcated the impact time as the highest peak of the signal, which usually coincided with the reversal of acceleration.[15] We then calculated kicking time (KT) by subtracting the time of impact from limb-movement onset.

To calculate $\int$EMG, we first filtered the raw data through a digital Butterworth second-order band-pass filter with a frequency from 30 to 400 Hz.[35] Next, we aligned the time intervals for $\int$EMG during the APAs and CPAs using the onset of electrical activity of the adductor muscle (t), which we called the *focal muscle* and was the principal muscle used for the kicking task in this experiment. Adductor onset corresponded to the time at which the signal exceeded the average threshold plus 2 standard deviations of its baseline, lasting for at least 25 milliseconds.[20] From this point (t), we defined and integrated 3 time intervals, each lasting 200 milliseconds (x_0–x_1): (1) 200 milliseconds before t, referred to as *APA*; (2) 200 milliseconds after t, referred to as *CPA1*; and (3) 200 to 400 milliseconds after t, called *CPA2*. We corrected the $\int$EMG for each time interval by the $\int$EMG for baseline activity, which we calculated between 1000 and 800 milliseconds before adductor onset using the following formula:

$$\int \mathrm{EMG} = \int_{x_1}^{x_0} \mathrm{EMG} - \int_{1000}^{800} \mathrm{EMG},$$

where $\int$EMG represents integrated EMG activity during the established time intervals for anticipatory and compensatory adjustments ($\int_{x_1}^{x_0}$ EMG) minus the baseline integrated activity during 200 milliseconds ($\int_{1000}^{800}$ EMG). We selected

the time intervals for APA and CPA based on data from pilot studies and the literature.[15,18,19] After calculating $\int$EMG, we normalized it. The normalization process included obtaining absolute maximum $\int$EMG values for each muscle (TA, PL, GasM, GasL, SOL, RF, and BF) for each participant across all conditions and time intervals and dividing each $\int$EMG index by the absolute maximum value. Therefore, all possible values for $\int$EMG were between −1 and 1, with positive values indicating muscle activation and negative values indicating muscle inhibition.[34]

Given that synergy exists among certain muscle groups during dynamic tasks, we calculated the sum of $\int$EMG for the ventral ($\sum\int EMG_{VEN}$ = TA + RF) and dorsal ($\sum\int EMG_{DOR}$ = GasM + GasL + SOL + BF) muscles of the standing limb for the 3 time intervals. We determined the sum of these muscle groups based on the reciprocal muscular activity observed during the anticipatory phase for this task in pilot studies (ie, inhibition of ventral and activation of dorsal muscles).

For the dynamic task, we calculated and quantified the ranges of COP displacement (cm) in the anteroposterior ($dCOP_{AP}$) and mediolateral ($dCOP_{ML}$) directions during the time intervals corresponding to the APAs and CPAs. We moved the COP time intervals 50 milliseconds forward relative to the EMG activity time intervals due to electromechanical delay. Therefore, the APA time interval encompassed the period from 150 milliseconds before to 50 milliseconds after t; CPA1, from 50 to 250 milliseconds after t; and CPA2, from 250 to 450 milliseconds after t. We calculated the area of dynamic COP displacement ($dCOP_A$) as for experiment 1 but over the complete duration of the KT (3 time intervals) plus 2300 milliseconds after the CPA2 interval. We selected this time interval, including the postkick period, based on pilot and published studies[15] revealing substantial changes in COP displacement over this period, especially in $dCOP_{ML}$. This measure represents postural stability over the entire dynamic task. We processed all data using MATLAB (version R2010b; The MathWorks, Inc, Natick, MA).

Statistical Analysis

Statistical analysis was based on the intention-to-treat principle. We calculated descriptive statistics to summarize demographic data and the CAIT score (average, standard deviation, and minimum and maximum values). We identified baseline differences between groups with independent-samples *t* tests. The dependent variables were KT; COP displacement during static ($sCOP_A$ with EO and EC) and dynamic ($dCOP_{AP}$, $dCOP_{ML}$, and $dCOP_A$) tasks; and integrated EMG, which we calculated individually ($\int$EMGs) and together ($\sum\int EMG_{VEN}$ and $\sum\int EMG_{DOR}$). These variables were summarized similarly with descriptive statistics (average, minimum and maximum values, standard deviation, and standard error of the mean) and evaluated for data normality using the Shapiro-Wilk test.

Given that the distribution of these variables approached normality, factorial analysis of variance was used with a 2 × 2 mixed design. Training (pretraining, posttraining) was entered as a within-subject factor, and participant group (TG, CG) was entered as a between-subjects factor. When we identified main effects for group, training, or the interaction between them, we performed paired-samples *t* tests for training and independent-samples *t* tests for group. We also calculated η^2 and Hedges *g* effect sizes for all analyses of variance and *t* tests, respectively, thereby identifying the magnitude of differences. We interpreted effect sizes as *small* (η^2 = 0.01 or Hedges $g = 0.2$), *medium* ($\eta^2 = 0.06$ or Hedges $g = 0.5$), or *large* ($\eta^2 = 0.14$ or Hedges $g = 0.8$). The α level was set at .05 for all tests. We used SPSS statistical software (version 20.0; IBM Corporation, Armonk, NY) for all statistical analyses.

RESULTS

Participant Characteristics

We included all 44 participants in the analysis. Figure 1 shows the flow of these participants through the study. The TG had sustained an average of 8 (range, 2–20) ankle sprains during the 2 years before the study and had a CAIT score of 15.2 ± 5.0 points (range, 6–24 points) for the affected ankle. The CG averaged 7 (range, 2–15) ankle sprains during the 2 years before the study and had a CAIT score of 15.4 ± 4.6 points (range, 3–24 points). Before the intervention, we found no between-groups differences in demographic variables or any dependent variable. Therefore, we performed independent *t* tests for these dependent variables only posttraining.

Experiment 1

We observed an interaction between training and group for the $sCOP_A$ with EO ($P = .001$; $\eta^2 = 0.246$). The pretraining versus posttraining comparison revealed reduced $sCOP_A$ posttraining, indicating less balance sway ($P = .002$; Hedges $g = 0.53$; 95% CI = –0.07, 1.14; Table). We did not observe a main effect of group on $sCOP_A$ displacement for the EO ($P = .75$) or EC ($P = .51$) condition.

Experiment 2

Kicking Time. No difference in KT was observed between groups ($P = .79$; Table). Thus, we assumed that the groups performed the kicking task similarly. Mean KT values for the TG and CG were 192 ± 7 milliseconds and 199 ± 8 milliseconds, respectively, pretraining and were 195 ± 7 milliseconds posttraining and 193 ± 6 milliseconds after resting, respectively.

Integrated EMG Activity. The main effects of training and group and their interaction on $\int$EMG are summarized in the Table. An interaction between training and group occurred for the TA and SOL muscles during the CPA1 interval. However, the TG and CG were not different at the second evaluation for either the TA ($P = .87$; Hedges $g = –0.04$; 95% CI = –0.63, 0.55) or SOL ($P = .08$; Hedges $g = –0.55$; 95% CI = –1.15, 0.05). Training altered the TA during the CPA2 time interval from pretraining to posttraining ($P = .048$; Hedges $g = –0.53$; 95% CI = –1.13, 0.07), with the $\int$EMG magnitude higher posttraining (Table). An effect of training was also evident for $\int$EMG during the CPA1

Table. Normalized Integrated Electromyographic (EMG) Signals of All Tested Muscles and Center-of-Pressure Displacements for the Training and Control Groups With Chronic Ankle Instability (Mean ± SD)

	Training (n = 22)		Control (n = 22)		
Time Interval	Pretraining	Posttraining	Pretraining	Posttraining	Interaction Effect
Anticipatory postural adjustment					
Normalized integrated EMG signals, arbitrary units					
Tibialis anterior	0.00 ± 0.56	−0.11 ± 0.35	−0.02 ± 0.48	−0.15 ± 0.36	<.001
Peroneus longus	−0.38 ± 0.39	−0.17 ± 0.42	−0.29 ± 0.50	−0.37 ± 0.40	.07
Medial head of gastrocnemius	0.27 ± 0.37	0.22 ± 0.24	0.14 ± 0.49	0.10 ± 0.37	<.001
Lateral head of gastrocnemius	0.38 ± 0.39	0.36 ± 0.33	0.27 ± 0.44	0.09 ± 0.48	.03
Soleus	0.12 ± 0.19	0.06 ± 0.29	0.09 ± 0.35	0.22 ± 0.24	.08
Rectus femoris	0.05 ± 0.38	−0.08 ± 0.36	−0.09 ± 0.44	−0.09 ± 0.36	.02
Biceps femoris	0.28 ± 0.26	0.34 ± 0.35	0.27 ± 0.22	0.24 ± 0.41	.01
Sum of integrated EMG signals, ventral muscles	0.05 ± 0.66	−0.11 ± 0.41	−0.11 ± 0.72	−0.24 ± 0.56	<.001
Sum of integrated EMG signals, dorsal muscles	1.06 ± 0.78	0.98 ± 0.67	0.76 ± 1.02	0.65 ± 0.84	<.001
Center-of-pressure displacement during dynamic task, cm					
Anteroposterior	0.89 ± 0.64	0.72 ± 0.48	0.52 ± 0.32	0.70 ± 0.45	.10
Mediolateral	0.41 ± 0.27	0.36 ± 0.20	0.41 ± 0.33	0.39 ± 0.28	<.001
Compensatory postural adjustment 1					
Normalized integrated EMG signals, arbitrary units					
Tibialis anterior	0.39 ± 0.52	0.24 ± 0.41	0.46 ± 0.32	0.22 ± 0.40	.01
Peroneus longus	0.18 ± 0.53	0.35 ± 0.54	0.28 ± 0.55	0.26 ± 0.44	.02
Medial head of gastrocnemius	0.67 ± 0.35	0.42 ± 0.49	0.52 ± 0.50	0.46 ± 0.51	.03
Lateral head of gastrocnemius	0.54 ± 0.32	0.34 ± 0.39	0.50 ± 0.40	0.53 ± 0.44	.04
Soleus	0.66 ± 0.27	0.54 ± 0.35	0.49 ± 0.47	0.55 ± 0.30	.03
Rectus femoris	0.23 ± 0.39	0.15 ± 0.47	0.34 ± 0.53	0.24 ± 0.47	<.001
Biceps femoris	0.72 ± 0.30	0.55 ± 0.34	0.72 ± 0.27	0.57 ± 0.59	<.001
Sum of integrated EMG signals, ventral muscles	0.77 ± 0.34	0.47 ± 0.56[a]	0.88 ± 0.57	0.45 ± 0.67[a]	.01
Sum of integrated EMG signals, dorsal muscles	2.67 ± 0.61	1.69 ± 0.63[a,b]	2.22 ± 1.01	2.23 ± 0.99	.16
Center-of-pressure displacement during dynamic task, cm					
Anteroposterior	3.02 ± 1.19	2.58 ± 1.68	2.56 ± 1.03	2.36 ± 1.36	.01
Mediolateral	0.85 ± 0.73	0.70 ± 0.58	0.64 ± 0.43	0.72 ± 0.58	.06
Compensatory postural adjustment 2					
Normalized integrated EMG signals, arbitrary units					
Tibialis anterior	0.24 ± 0.47	0.52 ± 0.57[a]	0.71 ± 0.43	0.54 ± 0.47	.10
Peroneus longus	0.43 ± 0.34	0.64 ± 0.45	0.40 ± 0.41	0.55 ± 0.43	<.001
Medial head of the gastrocnemius	0.37 ± 0.49	0.35 ± 0.62	0.25 ± 0.68	0.30 ± 0.51	<.001
Lateral head of the gastrocnemius	0.32 ± 0.56	0.37 ± 0.52	0.27 ± 0.52	0.43 ± 0.49	.01
Soleus	0.67 ± 0.33	0.45 ± 0.43	0.48 ± 0.49	0.68 ± 0.39	.13
Rectus femoris	0.51 ± 0.53	0.55 ± 0.38	0.49 ± 0.53	0.56 ± 0.37	<.001
Biceps femoris	0.52 ± 0.36	0.38 ± 0.37	0.46 ± 0.26	0.34 ± 0.52	<.001
Sum of integrated EMG signals, ventral muscles	0.85 ± 0.53	1.07 ± 0.70	1.20 ± 0.79	1.10 ± 0.57	.03
Sum of integrated EMG signals, dorsal muscles	1.87 ± 1.22	1.55 ± 1.24	1.47 ± 1.34	1.75 ± 1.18	.03
Center-of-pressure displacement during dynamic task, cm					
Anteroposterior	1.49 ± 1.06	2.12 ± 1.25[a]	1.61 ± 0.92	1.71 ± 0.92	.06
Mediolateral	0.90 ± 0.98	0.96 ± 0.84	0.84 ± 0.96	0.72 ± 0.68	.01
Whole task					
Area-of-displacement during dynamic task, cm^2	78.07 ± 66.09	113.8 ± 110.8[a]	99.50 ± 69.12	96.70 ± 52.60	.10
Area-of-displacement during static task, cm^2					
Eyes open	8.70 ± 3.19	7.10 ± 2.66[a]	7.19 ± 1.90	8.14 ± 2.92	<.001
Eyes closed	24.93 ± 8.08	25.99 ± 8.93	24.19 ± 7.69	24.64 ± 7.33	.24
Kicking time, ms	192.40 ± 31.59	194.91 ± 35.00	198.83 ± 35.64	192.98 ± 26.33	.01

[a] Different from pretraining ($P < .05$).
[b] Difference between groups at posttraining ($P < .05$).

time interval for the TA and BF. However, subsequent testing did not identify differences in these muscles for any group.

Summed Integrated EMG. We observed an interaction between training and group for $\sum\int EMG_{VEN}$ during the CPA1 time interval (Table). Between-groups differences in the $\sum\int EMG_{DOR}$ were detected during CPA1 posttraining ($P = .04$; Hedges $g = -0.64$; 95% CI = −1.24, −0.03), with the magnitude of $\sum\int EMG_{DOR}$ less in the TG than in the CG. We also observed an effect of training for the $\sum\int EMG_{VEN}$ ($P = .01$; Hedges $g = 0.64$; 95% CI = 0.03, 1.24) and $\sum\int EMG_{DOR}$ ($P = .001$; Hedges $g = 1.55$; 95%

CI = 0.88, 2.23) during the CPA1 interval. Both $\sum\int EMG_{VEN}$ and $\sum\int EMG_{DOR}$ declined with training. In addition, $\sum\int EMG_{VEN}$ declined in the CG between the first and last assessment ($P = .04$; Hedges $g = 0.68$; 95% CI = 0.07, 1.29; Table).

Center-of-Pressure Displacement. An interaction was also apparent between training and group for $dCOP_{ML}$ during the APA time interval (Table). However, for this variable, no intergroup difference was noted ($P = .90$; Hedges $g = 0.04$; 95% CI = –0.55, 0.63). We also did not observe main effects of training or group during APA and CPA1 for either $dCOP_{AP}$ or $dCOP_{ML}$. However, a main effect of training occurred for $dCOP_{AP}$ during CPA2 (Table), with $dCOP_{AP}$ excursion greater at posttraining than at pretraining in the TG ($P = .03$; Hedges $g = –0.53$; 95% CI = −1.14, 0.07) but no difference between the 2 data points in the CG.

We observed an interaction between training and group for $dCOP_A$, with $dCOP_A$ greater at the time of the second assessment in the TG ($P = .03$; Hedges $g = –0.38$; 95% CI = –0.98, 0.21) but not in the CG (Table). In other words, the balance sway of the TG during the dynamic task was increased posttraining. No main effects of training or group were detected for this variable.

DISCUSSION

Our primary finding was that the BPT produced changes in postural-control strategies. These changes occurred mostly during the compensatory phase (CPA1: before ball impact) and in the ventral and dorsal muscles of the lower extremity, both of which decreased their activity posttraining. This was likely the reason for the observed increase in balance sway during the performance of the ball-kicking task.

Individuals with CAI may exhibit ligament laxity, decreased proprioception, and motor-reaction deficits.[3,36,37] Given these pathobiologic mechanisms, they experience the sensations of ankle instability and giving way and the fear of recurrent sprain. This condition may cause them to initially increase their muscular activity excessively, thereby increasing stiffness in their lower extremity joints and decreasing postural excursion during the experimental task.[15] Researchers[38] have shown that individuals with CAI react excessively, unloading their body weight, to painful electrical stimulation when balancing with their ankles in supination. Therefore, during pretraining in our experiment, they may have adopted this strategy to potentially decrease their risk of sustaining a recurrent sprain because they had to kick a ball while in an unstable condition (single-legged stance). With BPT, participants might have been more confident in their injured ankles and better able to address body disturbances, allowing them to complete the task or balance using higher sway amplitudes. Hence, the TG decreased the activity of their stance-limb muscles. These results coincide with those reported by Nagai et al,[39] who found that balance training in older adults led to decreased muscular activity while controlling postural balance in tasks involving upper extremity reaching in an orthostatic position. As such, those and our results suggest that balance training might decrease muscular activity during dynamic tasks, leading to increased postural sway.

Interestingly, the CG also exhibited decreased activity of the ventral muscles after resting, a phenomenon that might be explained by a learning effect acquired during the first trials of pretraining. However, contrary to the TG, the CG showed no changes in the dorsal muscles or any COP displacement variable due to EMG activity changes. Kicking a ball while standing on 1 limb on a stable floor might have preferentially targeted control of their ventral limb muscles, especially before the ball was struck (CPA1); thus, a few repetitions might have been enough to alter their activity. In contrast, the training protocol that involved more varied kicking conditions could have more thoroughly involved all the lower extremity muscles, inducing changes in both the ventral and dorsal muscles. This may be an informative focus of future studies.

When their muscles were evaluated individually, the TG experienced increased TA activity during the CPA2 interval, which followed ball impact, posttraining. In addition to ankle dorsiflexion, researchers[40,41] have shown that both the TA and PL have coupled activity during inversion and eversion of the ankle, which helps the ankle remain in a neutral position and maintains balance during the stance phase of gait. With our experimental task, this coupled activity was likely used to maintain mediolateral ankle and body stability, especially after the kick (CPA2). Whereas not different, PL muscle activity increased posttraining during the CPA2 interval. These results suggest that BPT involving kicking a ball increases the activity of muscles that control the mediolateral stability of the ankle postperturbation. This strategy might lower the risk of recurrent sprains after kicking.

Investigators[12,17,30–32] have shown that individuals with ankle instability usually have increased postural sway (COP displacement) while standing motionless on 1 limb[4] and that balance training decreases postural sway during this condition. For instance, individuals with CAI exhibit smaller COP displacements while standing motionless on 1 limb during BPT involving ball-catching tasks,[32] elastic-tubing exercises,[31] and balance exercises on unstable surfaces (eg, dura disc and mini-trampoline).[12] These results are consistent with our findings in which COP displacement (area of sway) during the static task decreased after training in the TG. Other authors[12,17] have interpreted this as improved postural stability. Our results, however, suggested that only 1 session of BPT that entails kicking a ball might modify postural sway when balance is evaluated in a single-legged static stance. This improvement was observed only in the EO condition and might be due to the type of training offered because no exercises with EC were included.

Contrary to the results obtained during the static task, balance sway increased posttraining for the dynamic task. Recently, dos Santos et al[15] showed that individuals with CAI experience drastically decreased COP excursion during a similar task of kicking a ball in a single-legged stance. During this task, the demand for postural control increases considerably, as participants must integrate control of balance with control of limb movement (kicking, internal perturbation) and the body disturbance

induced by impacting the ball (external perturbation). For this reason, dos Santos et al[15] suggested that the decrease in balance sway (COP excursion) observed in individuals with CAI during a ball-kicking task could be due to changes in preparation for a motor response (central set), which might be related to anxiety levels in these individuals. Therefore, the feeling of ankle instability, low confidence in postural control, and fear of recurring sprains during a challenging task could cause these individuals to increase the stiffness of their lower limb joints as noted, resulting in decreased COP excursion. Thus, the immediate effect of balance training possibly minimized our participants' anxiety levels, causing the COP to reach greater amplitudes during dynamic tasks. In fact, McKeon et al[11] identified enhanced dynamic balance (greater distances on the Star Excursion Balance Test) in individuals with CAI after 4 weeks of balance training that emphasized dynamic body stabilization while standing on 1 limb.

In contrast, standing still on a rigid floor decreases the demand for postural control relative to kicking a ball while standing on 1 limb; therefore, anxiety might not be relevant during a static task. Nonetheless, the balance-training protocol that we used involves controlling body posture in single-legged stance in its essence; hence, the learning effect of this training might have been transferred to static-task testing, decreasing postural sway in our participants. Han et al[31] reported similar results for the transfer effects of training. A resistance exercise program, practiced on 1 limb (the affected ankle) using elastic tubing, markedly decreased balance sway in healthy control participants and participants with CAI standing still on 1 limb.[31] These results of previous studies and ours suggest that balance training may promote decreased postural sway during static tasks but increased postural sway during dynamic tasks. The latter might represent better postural control during a functional (dynamic) task, in which individuals with CAI increase their limit of stability secondary to augmented control and trust in their ankle joint, albeit this is a hypothesis that needs to be tested.

In our study, in anticipation of limb movement, both groups generally activated paired agonist and antagonist muscles in a reciprocal manner. Therefore, we observed activation of the BF with inhibition of the RF in the thigh and activation of the dorsal muscles with inhibition of the ventral (TA) and lateral (PL) muscles in the lower limb (positive versus negative values; Table). However, before and after ball impact, participants used a strategy of muscular co-activation (all positive values; Table). They used the first (reciprocal) strategy to stabilize their posture in relation to limb motion toward the ball and the second (co-activation) to stabilize posture just before (CPA1) and after (CPA2) ball impact. The second perturbation was perhaps the most destabilizing; it caused individuals to use muscular co-activation, a strategy that demands greater energy expenditure but is more effective for further increasing joint stiffness and improving postural stabilization.[42] A previous study[20] in which researchers evaluated APA and CPA yielded similar results, with healthy individuals using reciprocal activation strategies in both APA and CPA after being disturbed by predictable (EO) postural perturbations (a moving pendulum). In contrast, during unpredictable perturbations (EC) that increased the demand for postural control, individuals chose a co-activation strategy, especially for lower leg muscles during the CPA phase. In our experiment, individuals with CAI maintained their co-activation strategy during CPA1 and CPA2 posttraining but decreased the activity of both their ventral and dorsal muscles before kicking. This change might indicate that they had improved their abilities to perform the task, allowing for greater balance sway during kicking. However, individuals with CAI may change to a reciprocal strategy of muscular activation with long-term training. This possibility is important to address in future studies of BPT.

Our study had limitations, as the number of interventions was small. However, investigating the immediate effect of 1 session of BPT allowed us to maintain precise locations of the EMG electrodes pretraining and posttraining, which might have decreased any potential intraparticipant variability in the EMG signals. In addition, the changes observed in the CPA muscle activity may have been due to fatigue. However, in a study involving fatigue, Corcos et al[43] showed that 10-minute rest intervals postfatigue allowed for recovery of muscle-fiber conduction velocity and performance (analyzed from kinematics). In addition, Kanekar et al[44] reported that the deltoid or hamstrings muscles recovered 89% of their maximal prefatigue force (100%) with 10 minutes of rest postfatigue and recovered 94% after 30 minutes of rest. During our experiment, no participant reported fatigue. For these reasons, we believe that the 2-minute breaks between treatment conditions and the 10-minute break between pretraining and posttraining were adequate to negate any meaningful effect of fatigue on APA and CPAs.

CONCLUSIONS

We observed that, after a ball-kick balance exercise, the activity of both the ventral and dorsal muscles decreased just before the kick among individuals with CAI, but the activity of muscles that control ankle inversion and eversion (TA and PL) increased after the kick. Overall, these changes were associated with increased postural sway during the ball-kicking task. In contrast, postural sway during the static task decreased posttraining. These results have implications for the clinical evaluation and treatment of postural control after balance training that uses postural perturbations involving kicking a ball. The observed changes in the strategies of postural adjustment might indicate an enhanced ability to address body disturbances and increase postural stability relative to task requirements. The results of our pilot study further support the use of ball-kicking BPT and should spur more comprehensive studies to evaluate and treat static and dynamic postural balance. More importantly, we need to discover whether these changes in postural control due to training actually result in improved ankle stability in individuals with CAI.

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