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THE EFFECTS OF A PERTURBATION-BASED BALANCE TRAINING ON THE REACTIVE NEUROMUSCULAR CONTROL IN COMMUNITY--DWELLING OLDER WOMEN: A RANDOMIZED CONTROLLED TRIAL

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ABSTRACT
Purpose. The purpose of this study was to evaluate the effects of short-term perturbation-based balance training and a detraining period on postural control in older adults. **Methods.** A group of healthy older women were recruited and divided into two groups: an exercise group (EG, *n* = 21, age = 67.0 ± 2.0 y) that performed balance-based exercises three times a week over a six-week period and a control group (CG, *n* = 20, age = 67.9 ± 3.1 y). Center-of-pressure displacement (CoP) and electromyographic data (EMG onset, time-to-peak and amplitude) were assessed during forward and backward perturbations for six leg muscles. All variables were analyzed before the training program began, at its end, and after a six-week period of detraining. A mixed ANOVA model was used to analyze the within- and between-subject results. **Results.** A decrease in backward CoP displacement, EMG onset and time-to-peak of the ankle muscles, especially the tibialis anterior (TA) and gastrocnemius (MG), was observed. Improvement in muscle EMG amplitude for the ankle muscles (TA, MG and Soleus – SO) at the early phase (0–200 ms) of the perturbation test, with the SO also showing an increase in amplitude at the intermediate phase (201–400 ms). After the detraining period, only the TA muscle maintained an improvement in reaction time. **Conclusions.** Perturbation-based balance training improved neuromuscular responses such as muscle reaction time and ankle muscle activation and consequently aided the body's ability to maintain correct center of pressure, although after a period of detraining this gain was not maintained for most of the assessed variables.

Key words: elderly, exercise, postural balance, electromyography, reaction time

Introduction

Senescence is associated with a general deterioration of many biological systems, mainly in the locomotor system. The deterioration of postural control and the decreased ability to preserve body balance during unpredictable perturbations, such as those that occur during activities of daily living, may contribute to an increase in the incidence of falls among elderly persons [1, 2]. Changes in the sensory and motor systems are also suspected to play a role in the decreased ability to maintain adequate postural adjustment when standing upright [3].

In an attempt to maintain postural control during unpredictable perturbations, excessive movement of the center of mass can be quickly corrected by generating muscle torques at the ankles, hips, and other joints, whereby a stabilizing effect can be achieved by rapidly moving the base of support [4]. However, elderly people have greater difficulty in detecting the direction and magnitude of such disturbances, which limits the generation of appropriate neuromotor action that would be quick and forceful enough to counteract the effects of such perturbations [5].

Older adults show larger horizontal center-of-pressure displacement and require more time to reverse the direction of this displacement than younger adults when exposed to unpredictable balance perturbations [3, 6]. This could be explained, at least in part, by the increased time to activate and reach peak muscle activation in response to postural changes [1], as response time has been shown to play an import role in the recovery from a loss of balance. This slowed response causes greater acceleration during a fall, therefore demanding a greater force to counteract the fall [7]. In addition, Van den Bogert [8], through the use of an inverted pendulum model, showed that variations in response time are more important than variations in walking velocity when determining the successful recovery of avoiding a fall after tripping.

Understanding the changes in the postural control of elderly individuals is important to help prevent the loss, reduction, or reversal of specific age-related impairments and help improve reactive postural control [9]. Several systematic reviews and clinical trials have recommended the use of exercise programs that include a balance-training component to improve body balance [10] to prevent falls in elderly adults [11–13]. However, current exercise programs are frequently criticized for a lack of standardization of specific training protocol such as type of exercise and duration [13], and those

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studies who did perform analysis of such programs did not involve testing a perturbation situation [10, 13].

A perturbation-based balance training program that is arbitrarily administered (variations in time, magnitude, and direction) works against the central nervous system's ability to predict the specific characteristics of perturbation or its ability to use such information to counteract the effects of a loss of balance [12]. The introduction of new dynamic postural control strategies might therefore help better restore balance against abrupt postural disorders [9, 14, 15] and could possibly be used in the future to predict falls and improve balance in the elderly.

Unstable devices such as the mini-trampoline have been used to improve the elderly's abilities to recover their balance during forward falls [16]. However, the effects of perturbation-based balance training, such as in a circuit set that includes the use of such unstable devices, on reactive neuromuscular responses and center-of-pressure displacement in the elderly have not yet been studied.

Thus, the aim of this study was to conduct a randomized clinical trial to verify the effects of a six-week perturbation-based balance training program followed by a six-week period of detraining in a group of healthy elderly women by measuring electromyographic (onset, time-to-peak, and mean amplitude of EMG signals at 0–200, 201–400 and 401–600 ms) and stabilometric (forward and backward center-of-pressure displacement) variables in situations with unpredictable perturbation. Our hypothesis was that a regularly performed six-week (three times per week) perturbation-based balance training program may be sufficient in ameliorating reactive postural control and maintain improvements in balance control after a detraining period of six weeks.

Material and methods

The study population consisted of elderly women living in a local community who were invited to participate in the study by leaflet or word of mouth. Seventy-three women attended the initial meeting, of whom 27 were excluded after the following exclusion criteria were applied: pain in the lower extremities; having had orthopedic surgery; history of fractures within the past year; inability to walk unaided; occurrence of neurological diseases; diagnosed acute inflammatory disease (due to possible interference in performing the training exercises), uncontrolled mellitus diabetics or arterial hypertension; use of medications that can affect balance; or featuring cognitive impairment based on a score of < 24 on the mini-mental state examination. The remaining 46 women were selected for inclusion. All were community-dwelling individuals, of mixed race and social class, aged between 65 and 80 years old, and classified as active (score > 53) according to the Brazilian version of the Human Activity Profile criteria [17].

The study was approved by the Ethics Committee of the State University of Centro-Oeste (#200/2011) and was registered at The Brazilian Clinical Trials Registry (#RBR6HP5H9).

The volunteers were randomly divided into two groups: a Control Group (CG, $n = 20$, age = 67.9 ± 3.1 y, BMI = 28.5 ± 3.2 kg m^{-2}) and Exercise Group (EG, $n = 21$, age = 67.0 ± 2.0 y, BMI = 27.6 ± 2.4 kg m^{-2}). In order to reduce the chance of having unbalanced groups, the participants were classified in quartiles based on the Timed-up & Go test (TUG) [18]. The participants of each quartile were randomly assigned to either the EG (TUG = 10.2 ± 1.3 s) or CG (TUG = 10.1 ± 1.8 s). An independent t test ($p > 0.05$) found no differences between groups for age, BMI, and TUG scores. The study design is shown in Figure 1.

All participants were evaluated by use of a perturbation test based on a sliding apparatus–constructed specially for this study, which was similar to the one described by Freitas et al. [1]. Data were collected three times, before the training program began (pre-training), after completing six weeks of balance training (post-training), and after a six-week detraining (post-detraining).

The purpose of the specially-built apparatus (see Fig. 2) was to produce a sudden perturbation as a closed kinetic chain (e.g., trip and slip) by instigating forward and backward balance loss. The apparatus consisted of a sliding steel platform (45 cm × 45 cm × 5 cm) built so as to allow the measurement of center-of-pressure displacement during postural change and to collect electromyographic data in accordance with previous studies [4, 7].

To capture center-of-pressure displacement during perturbation, a baropodometric platform was placed on top of the sliding platform. The platform moved either forwards or backwards at a distance of 12 cm in a time frame of 0.47 s at a mean velocity of 24.8 cm/s and mean acceleration of 54.7 cm/s^2. The force generated to move the platform (abrupt perturbation) was generated by dropping a weight of 5 kg from a height of 25 cm that was connected to the platform by a steel cable. During the perturbation test, participants were instructed to stand on the baropodometric platform with their feet parallel and 5 cm apart, their arms relaxed close to their sides, head upright, and eyes open. A strain gauge was used to measure the "initial moment" of the platform's displacement in order to determine the start of reaction time. To avoid the risk of falling the participants were secured by an upper-body safety harness [12].

To evaluate for intra-observer variability, ten measurements made with the strain gauge (peak strength) and platform speed on two separate days three days apart were collected. The Intra-class Correlation Coefficient (ICC) and a confidence interval (CI) of 95% were adopted. Good repeatability for peak strength with the strain gauge was found (ICC = 0.785; 95% CI 0.37, 0.94) and moderate to good repeatability (ICC = 0.73, 95% CI 0.24, 0.92) for sliding platform speed. ICC and IC analysis were performed using SPSS ver. 20.0 (IBM, USA).

To assess concomitant electromyographic data, six pairs of bipolar surface electrodes were placed on the participants' dominant side's rectus femoris (RF), vastus

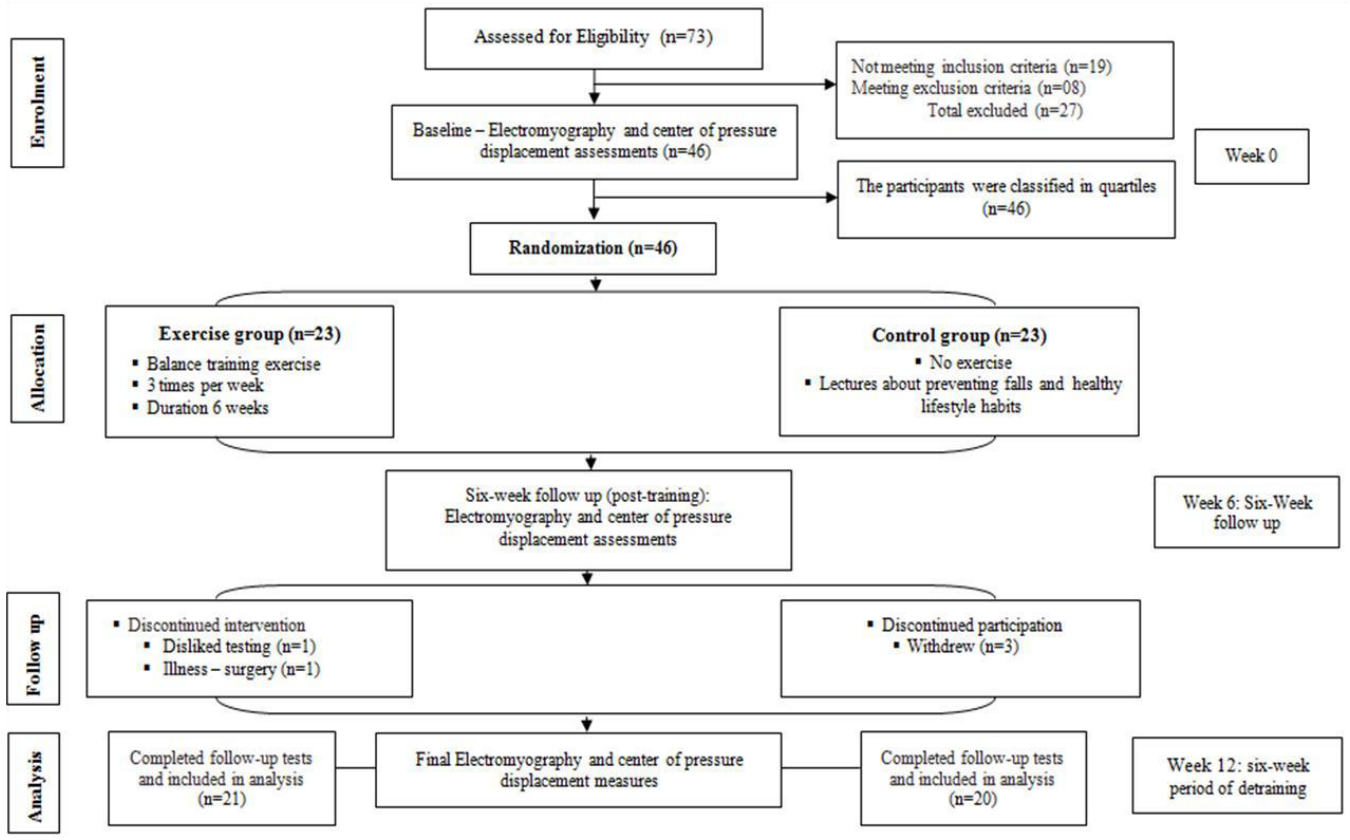
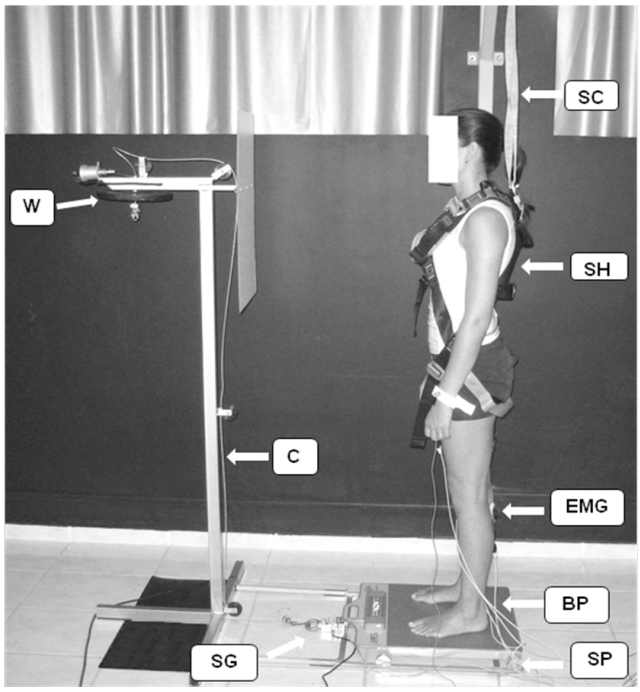


Figure 1. Flow diagram of the study design



SC – security cable BP – baropodometric platform
SH – safety harness SP – sliding platform
EMG – electrodes C – steel cable
SG – strain gauge W – 5 kg weight

Figure 2. Experimental perturbation-based apparatus using a sliding platform

medialis oblique (VMO), tibialis anterior (TA), semitendinosus (ST), medial head of gastrocnemius (MG), and soleus (SO) muscles (leg preference was chosen by asking the participants to climb up a 40 cm platform; the leading leg instinctively chosen by the participants was considered the dominant leg). Capture of EMG signals for the RF, VMO and TA muscles was performed during forward displacement of the platform, while the ST, MG and SO muscles were analyzed when the platform moved backwards. EMG signals were recorded at 2000 Hz amplified with a gain of 1000x, then band-pass filtered (20–500 Hz) and converted from analog to digital by a 16 bit A/D converter. The EMG signals were full-wave rectified and low-pass filtered by a 3rd order zero-lag Butterworth filter.

EMG data includes measurements of EMG onset (representing how fast a muscle is activated after the introduction of the perturbation), EMG time-to-peak (representing how quickly a muscle reaches its maximum level of activation), and the amplitude (intensity) of the EMG signal. These variables provided important information on muscle function and force production.

Statistical analysis was performed using a customized version of Matlab (Mathworks, USA) to process and analyze the EMG signal data. EMG onset for each muscle was automatically determined when the intensity of muscle activity crossed 7% of the electromyographic signal peak after perturbation. All EMG onsets were visually confirmed by the experimenter with graphs generated by

the Matlab program. EMG time-to-peak was computed as the difference between the instant that the muscle reached maximum level of activation and EMG onset time. The amplitude of EMG activity for each muscle was estimated by calculating the root mean square (RMS) at three time intervals: 0–200 ms (early phase), 201–400 ms (intermediate phase), and 401–600 ms (late phase) with respect to EMG muscle onset.

The exercise training program consisted of sessions lasting 40 min held three times per week [13] for a period of six consecutive weeks [13, 19, 20]. They were conducted in groups of 12 and supervised by a physiotherapist. The first 10 min consisted of a warm-up performed on a stationary bicycle at 40–60% of heart rate reserve. The remaining 30 min consisted of exercises designed specifically for balance training with the use of balance devices simulating unstable ground [20, 21]. During each training session, six unstable devices were used: a proprioceptive disk [21], a rocker, a balance board [20, 22], a Bosu ball (Bosu, USA), an inverted Bosu ball, and a mini-trampoline [16]. During all balance exercises the participants were instructed to stand upright, look forward, and maintain a knee flexion of 15 degrees. The types of training exercises performed were based on the suggestions of researchers [19, 20], beginning with bipodal exercises for the first three weeks which advanced to unipodal exercises for the remaining three weeks. The exercise protocol consisted of performing four 1-min repetitions [19, 22] on each device (completing the circuit) with 1 min rest between each repetition.

For statistical analysis, distributions of the stabilometric data and EMG onset, time-to-peak, mean amplitude signals were tested for normality using the Shapiro-Wilk test while Levene's test was used to determine homogeneity. Two-way ANOVA (2 groups × 3 measures) was used to compare the groups versus their results. When the relationship was determined to be statistically significant, one-way ANOVA with repeated measures was used. For the mean amplitude of the EMG signals, the Friedman test was used to compare measures at pre-training (PRE) × post-training (POST) × 6 weeks after training (POST6). When statistical significance was observed, the Wilcoxon test with Bonferroni correction (α/number of comparisons) was used as a post-hoc test. Comparisons between groups were performed with the Mann-Whitney *U* test. In addition, the Minimal Detectable Change (MDC) was adopted in order to detect the smallest amount of change by a measure that corresponds to a noticeable change in ability, calculated by the equation MDC = 1.96 * √2 * SEM, where SEM (Standard Error of Measurement) = standard deviation * √(1 – ICC).

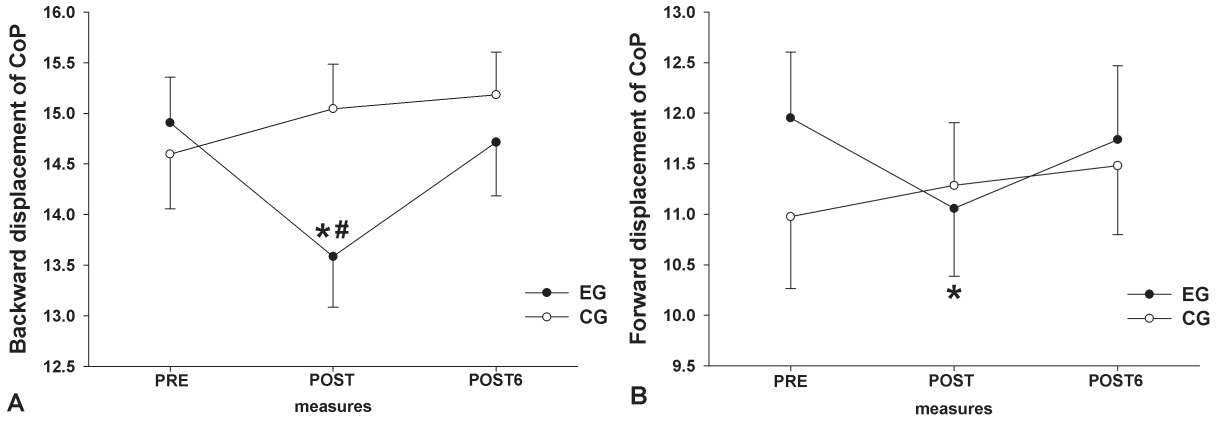
A significance level of $p < 0.05$ was adopted for all statistical procedures, which were performed using SPSS ver. 20.0 (IBM, USA).

Results

Stabilometric measurement of center-of-pressure (CoP) displacement found backward CoP displacement having a significant group × measure ($F_{2,78} = 9.17$; $p < 0.0001$) interaction. The backward CoP displacement measured after the training program was significantly less than measures tested at pre-training ($p < 0.0001$) and post-detraining ($p = 0.003$). Additionally, the exercise group's (EG) backward CoP displacement was significantly smaller than the control group's (CG) before beginning the training program ($F_{1,39} = 4.60$, $p = 0.038$) but not after the detraining period ($F_{1,39} = 0.46$, $p = 0.50$).

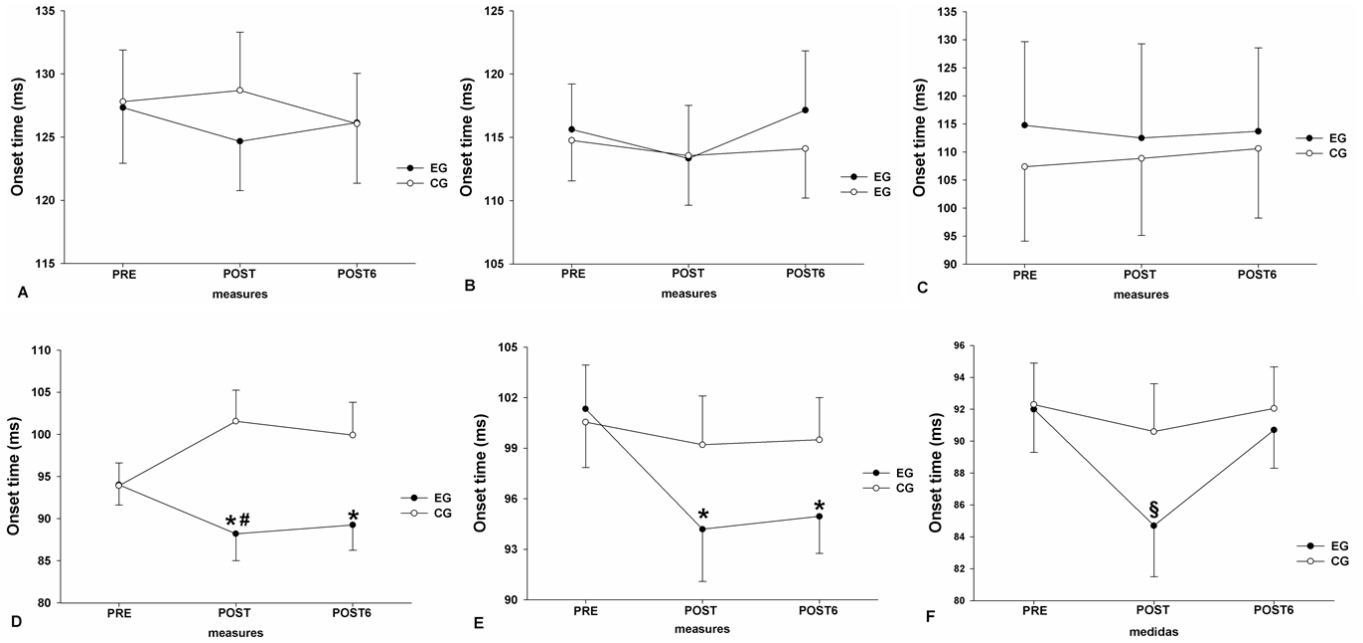
In contrast to the results on backward displacement, forward CoP displacement did not show significant group × measure ($F_{2,78} = 3.01$; $p = 0.056$) interaction, although repeated measures performed separately for each group showed a reduction in the CoP for the EG ($F_{2,40} = 5.28$, $p = 0.009$) after completing the training program (Fig. 3).

Analysis on the collected electromyographic data found that EMG onset for RF, VMO and ST after the balance training program did not show significant differences or interaction for measures and groups ($p > 0.05$,



Exercise Group (EG) and Control Group (CG)
* – significantly different from PRE of the same group ($p < 0.05$)
# – significantly different from POST of the CG ($p < 0.05$)

Figure 3. Backward (A) and forward (B) CoP displacement measured at intervals before (PRE), after (POS), and six weeks after (POS6) the balance training program



* – significantly different from PRE measures of the same group ($p < 0.05$), § – significantly different from PRE and POS of the same group ($p < 0.05$)
# – significantly different from POS of the CG ($p < 0.05$)

Figure 4. EMG onset of rectus femoris (A), vastus medialis oblique (B), semitendinosus (C), tibialis anterior (D), medial head of gastrocnemius (E) and soleus (F) muscles after sudden perturbation

Table 1. Mean ± SD of stabilometric and EMG time variables (EMG onset and time-to-peak) measured at intervals before (PRE), after (POST), and six weeks after (POST6) the balance training program showing Intraclass Correlation Coefficient (ICC), Standard Error of Measurement (SEM) and Minimal Detectable Change (MDC) for each variable

Group		Variable	PRE	POST	POST6	ICC	SEM	MDC
Exercise group	Stabilometry (cm)	Forward displacement	11.96 ± 3.06	11.05 ± 3.13[a]	11.73 ± 3.44[b]	0.97	0.55	1.54
		Backward displacement	14.91 ± 2.11	13.58 ± 2.37[a,c]	14.71 ± 2.50[b]	0.95	0.45	1.44
	Onset (ms)	RF	127.33 ± 19.99	124.66 ± 17.86	126.14 ± 21.88	0.87	7.21	19.97
		VMO	115.61 ± 21.88	113.33 ± 19.35	117.14 ± 21.37	0.75	9.5	26.32
		ST	114.76 ± 14.87	112.47 ± 16.81	113.66 ± 14.90	0.91	4.65	12.88
		TA	94.00 ± 11.22	88.19 ± 14.52[a,c]	89.23 ± 13.94[d]	0.93	3.49	9.67
		MG	101.33 ± 11.78	94.19 ± 14.21[a]	94.95 ± 10.12	0.84	4.8	13.3
		SO	92.00 ± 12.16	84.66 ± 14.48[a,b]	90.66 ± 10.79	0.90	3.95	10.95
	Time-to-peak (ms)	RF	115.38 ± 17.29	107.28 ± 23.27	112.57 ± 18.30	0.88	6.79	18.81
		VMO	131.14 ± 22.08	130.09 ± 26.54	132.52 ± 19.49	0.90	7.18	19.88
		ST	110.85 ± 20.19	103.08 ± 19.70	107.90 ± 21.30	0.86	7.63	21.14
		TA	144.71 ± 23.73	127.71 ± 20.21[a,c]	130.09 ± 17.08[d]	0.92	5.66	15.67
		MG	128.52 ± 14.65	111.71 ± 13.55[a,b,c]	117.61 ± 13.96[d]	0.95	3.13	8.67
		SO	129.66 ± 17.64	126.95 ± 13.85	127.95 ± 15.42	0.84	6.24	17.28
Control group	Stabilometry (cm)	Forward displacement	10.97 ± 3.15	11.28 ± 2.76	11.48 ± 3.05	0.93	0.79	2.2
		Backward displacement	14.60 ± 2.40	15.04 ± 1.94	15.18 ± 1.85	0.91	0.63	1.75
	Onset (ms)	RF	127.80 ± 18.22	128.70 ± 20.65	126.05 ± 18.02	0.72	10.05	27.85
		VMO	114.75 ± 14.09	113.55 ± 17.60	114.10 ± 17.48	0.80	7.33	20.32
		ST	107.35 ± 13.24	108.85 ± 13.72	110.60 ± 12.36	0.82	5.56	15.4
		TA	93.90 ± 11.88	101.55 ± 16.63	99.90 ± 11.52	0.72	8.1	22.43
		MG	100.55 ± 12.29	99.20 ± 13.08	99.50 ± 11.00	0.80	5.41	14.99
		SO	92.30 ± 11.78	90.60 ± 13.52	92.05 ± 11.77	0.91	3.72	10.3
	Time-to-peak (ms)	RF	123.86 ± 18.23	121.30 ± 21.31	122.20 ± 18.47	0.93	5.11	14.14
		VMO	131.15 ± 23.02	132.40 ± 19.07	133.75 ± 17.39	0.88	6.86	19.00
		ST	111.60 ± 16.80	113.55 ± 14.58	112.80 ± 12.29	0.90	4.62	12.79
		TA	142.20 ± 18.72	144.35 ± 21.67	143.05 ± 15.31	0.85	7.2	19.95
		MG	124.35 ± 16.26	125.45 ± 17.37	127.70 ± 15.33	0.91	4.89	13.55
		SO	124.30 ± 16.96	123.25 ± 13.83	123.00 ± 12.45	0.90	4.55	12.61

[a] significantly different (repeated measures ANOVA/post-hoc Bonferroni, [b] significantly different between POST × POST6 of the same group ($p < 0.05$), [c] significantly different from POST of the CG ($p < 0.05$), [d] significantly different from POST6 of the CG ($p < 0.05$), $p < 0.05$) between PRE × POST of the same group ($p < 0.05$)

Table 2. Mean ± SD of EMG amplitude at early (0–200 ms) and intermediate (201–400 ms) phase of activation measured before (PRE), after (POST), and six weeks after (POST6) the balance training program

Group	EMG amplitude		PRE	POST	POST6
			Mean ± SD		
Exercise group	0–200 ms	RF	57.47 ± 19.08	58.19 ± 19.14	55.90 ± 16.95
		VMO	51.52 ± 19.94	56.52 ± 22.12	53.09 ± 17.47
		ST	58.61 ± 17.84	73.47 ± 15.50	62.71 ± 14.73
		TA	150.04 ± 32.61	166.85 ± 30.06[a, c]	164.42 ± 26.01[b]
		MG	74.57 ± 18.83	87.52 ± 16.24[a, c]	79.14 ± 18.03[b]
		SO	66.90 ± 20.25	84.47 ± 16.13[a, c]	70.14 ± 16.17[b]
	201–400 ms	RF	32.33 ± 21.77	34.57 ± 20.51	33.71 ± 18.84
		VMO	33.57 ± 14.09	36.57 ± 17.89	35.09 ± 14.87
		ST	34.42 ± 14.98	35.14 ± 13.91	33.19 ± 9.66
		TA	80.52 ± 24.92	90.19 ± 21.04	88.23 ± 28.90
		MG	35.42 ± 15.81	43.23 ± 19.33[a]	39.57 ± 18.39
		SO	40.00 ± 12.91	49.90 ± 19.99[a, c]	42.04 ± 15.09[b]
Control group	0–200 ms	RF	56.10 ± 15.61	55.15 ± 15.19	54.30 ± 14.50
		VMO	48.15 ± 14.41	50.15 ± 13.16	48.55 ± 11.28
		ST	69.80 ± 19.31	71.85 ± 15.42	68.60 ± 16.29
		TA	152.55 ± 30.43	149.05 ± 28.85	150.80 ± 32.81
		MG	76.75 ± 18.62	74.70 ± 16.20	74.30 ± 16.73
		SO	67.95 ± 18.25	65.35 ± 14.62	64.10 ± 13.30
	201–400 ms	RF	32.50 ± 18.47	32.85 ± 14.46	31.30 ± 15.59
		VMO	36.85 ± 12.81	34.95 ± 16.25	33.90 ± 13.32
		ST	33.10 ± 11.32	31.00 ± 10.55	31.10 ± 8.68
		TA	81.15 ± 31.63	83.55 ± 26.42	80.05 ± 31.09
		MG	34.50 ± 14.56	36.15 ± 14.49	36.10 ± 14.38
		SO	38.60 ± 10.48	39.55 ± 17.59	38.05 ± 12.19

[a] significantly different in PRE × POST, [b] POST × POST6 (Friedman test, [c] significantly different (Mann Whitney *U* test, $p < 0.05$) between means of groups in POST, $p < 0.05$/Wilcoxon as post hoc α/3)

see Fig. 4A–C). In contrast, EMG onset for TA post-training showed a significant group × measure interaction ($F_{2,78} = 6.52$; $p = 0.002$) (see Fig. 4D). Further analysis indicated that EMG onset for the TA was reduced after the training period only for the EG ($p < 0.05$) and that this reduction continued to be present even after the 6-week detraining period ($p < 0.05$). The EG group also showed earlier EMG onset times compared with the CG at measurement period after the training ($F_{1,39} = 7.525$, $p = 0.009$) and detraining period ($F_{1,39} = 4.67$, $p = 0.03$).

As observed with the TA muscle, EMG onset for the MG and SO muscles post-training found that the EG showed significant differences among the measures (MG: $F_{1.40, 28.12} = 6.04$, $p = 0.013$, SO: $F_{2.40} = 20.61$, $p = 0.000$, see Fig. 4E–F). Both muscles exhibited a significant reduction in EMG onset time after undergoing balance training, although this reduction was sustained after the six-week detraining period only for the MG ($p < 0.05$). There was no significant group × measure interaction for both MG ($F_{1.55, 60.58} = 1.80$, $p = 0.18$) and SO ($F_{2.78} = 2.43$, $p = 0.09$) and no difference between the CG and EG for all analyses ($p > 0.05$).

Similar to what was observed with the time of EMG onset, EMG time-to-peak after training for RF, VMO, and ST did not show significant differences or interaction for measures and groups ($p > 0.05$), while the TA showed a significant group × measure interaction ($F_{1.74, 67.99} = 10.30$, $p = 0.0001$) (see Tab. 1) with differences found only for the EG ($F_{1.39} = 20.61$, $p = 0.000$), which indicated that EMG time-to-peak for the TA was reduced after training only for the EG ($p < 0.05$) and that this reduction was kept even after the detraining period ($p < 0.05$). These values were 16% lower for the EG at post-training and 13% lower at post-detraining than the CG ($p < 0.05$).

The EMG time-to-peak for the GM post-training showed a significant difference between measures ($F_{2,78} = 14.29$; $p < 0.001$) and significant group × measure interaction ($F_{2,78} = 20.73$; $p < 0.001$). The GM muscle exhibited a significant reduction in EMG time-to-peak post-training and post-detraining ($p < 0.05$) and these values were lower than the CG's only at post-training ($p < 0.05$).

The EMG amplitude at the early phase of activation (0–200 ms) found significant differences between measures for the EG for the TA, MG and SO muscles as identified by the Friedman test ($p < 0.0001$ for all muscles, see Tab. 2). Post hoc analysis indicated an increase in EMG amplitude post-training for the MG, SO and TA muscles at 13%, 17%, and 17%, respectively ($p < 0.0001$), although only the TA muscle sustained this significant increase at post-detraining (POST × POST6, $p > 0.05$).

EMG amplitude at the intermediate phase (201–400 ms) showed an increase in muscle activation for the TA, MG, and SO muscles ($p < 0.05$) for the EG at post-training (TA: 10%, $p = 0.005$; MG: 8%, $p = 0.002$; and SO: 10%, $p = 0.001$). However, only the TA and MG muscles sustained this increase after the detraining period (POST × POST6, $p > 0.05$). Comparison between groups showed greater amplitude for the EG than the CG post-training for muscles: TA (17%, $p = 0.04$), MG (13%, $p = 0.007$), and SO (19%, $p = 0.000$) at the early phase and for SO (10%, $p = 0.047$) at the intermediate phase. EMG amplitude at the late phase (401–600 ms) had no statistically significant differences within- and between-groups for all of the evaluated muscles ($p > 0.05$).

No clinical changes were observed in the analyzed variables when tested by the Minimal Detectable Change (MDC), finding that the balance training program did not generate statistically significant differences in this regard.

Discussion

The purpose of the present study was to investigate the role of perturbation-based balance training on postural control. Two hypotheses were formulated: (1) would elderly women show an improvement in the reactive neuromuscular response of the lower limb muscles and a decrease in center-of-pressure (CoP) displacement after completing a six week perturbation-based balance training course based on a circuit set of unstable devices and (2) would these changes still be present following a six-week period of detraining?

It was observed that CoP displacement was reduced following the balance training program, although significant differences were observed only for backward displacement when compared with control group. Nonetheless, this suggests an improvement in balance control as it would help prevent excessive horizontal displacement of center-of-mass (CM). Indeed, the reduction of CoP displacement is the main goal of postural response immediately after perturbation so as to avoid a fall [1]. This was also confirmed in the present study by the decreased temporal muscle activity and increased EMG activity of the ankle muscles after completing the balance training program, which may have prevented excessive CoP displacement through quick and appropriate muscle activation [1, 4].

Furthermore, the exercise group (EG) showed faster reaction times in the anterior and posterior ankle muscles after completing the balance training program, in particular with the tibialis anterior (TA), which maintained this reduction even after the detraining period. The results were greater for TA possibly due to more frequent activation by unstable surfaces such as when standing on a mini-trampoline or balance board [23], equipment used in this study's training regime.

Regarding EMG time-to-peak, both TA and MG had a 17% average reduction in the EG post-training compared with the CG, although this improvement was maintained post-detraining only for the TA muscle. Nonetheless, this indicates that improvement in muscle reactivity can be achieved in the elderly with short-term balance training (six weeks). This outcome shows how quickly the muscles of older adults can reach their maximum level of activation and generate torque after perturbation [1]. This was additionally confirmed by other studies, which found that maximum TA strength is preserved in the sixth decade of life [24] and that older adults can generate ankle joint torque similar to that of younger adults after platform translation [25].

In addition, early and appropriate muscle activation diminishes the chances of excessive CM displacement and can help obtain quicker time-to-peak intensity [1], which is particularly important in the elderly when the central and peripheral nervous systems suffer age-related degradation. Demyelination, loss of axonal fibers, degeneration of fast fibers' motor neurons are all common problems that can lead to a decrease in nerve conduction velocity [26] and peak activation, especially after the fifth decade of life [1]. Furthermore, slowed motor time reaction is representative of reduced excitation–contraction coupling, including slowed calcium release or reuptake from the sarcoplasmic reticulum and decreased activity of metabolic enzymes such as creatine kinase and actomyosin ATPase [27, 28].

Several studies had shown changes in the reaction time of the peroneus longus and tibialis anterior muscles with balance training in young adults [21, 29]. Our study showed that both EMG onset and EMG time-to-peak can also be trained and improved in the elderly with balance training. For older individuals, the improvement of these variables can help prevent a fall as a fast muscular reactive response is essential in maintaining balance during a slip or trip.

Concerning the results on EMG amplitude, this study showed improvement among the distal muscles after balance training. However, this improvement was expressive only in the EMG amplitude at the early phase of activation (0–200 ms), which may possibly be the result of the unstable apparatuses used in the training exercises that may have stimulated a faster muscle reactive response. According to Maki et al. [14], compensatory reactive responses are decisive reactions for preventing falls. These reactions are much more rapid than volitional limb movements and are effectual in controlling the CM motion generated by sudden, unpredictable perturbations.

Our results confirm, at least in part, the first hypothesis on the effectiveness of a balance training exercise regime in reducing CoP motion, EMG time-to-peak, and onset and increasing muscle activity (EMG amplitude). However, these improved EMG parameters (EMG onset, time-to-peak, and amplitude) were found only in the distal muscles, which may have resulted from the use of unstable surface training that would more actively recruit the ankle muscles, especially the TA [23].

It is known that older individuals decrease their use of ankle-based strategies and prefer hip postural strategies to remain upright [30], and this may be related to the slowness in stabilizing the ankles in perturbation tests that simulate a fall [4] and to delayed muscle activation [1]. Therefore, the neuromuscular enhancement of the distal muscles obtained here may be important in improving the postural control in older adults.

For the second hypothesis, on the effects of detraining, we found that the balance training exercise regime was not able to maintain all improvements (except for the TA muscle, which maintained improved EMG onset and time-to-peak levels), which may be related to the continuously debilitating effects of senescence. The deterioration observed among the majority of the studied electromyography variables after the detraining period, particularly in the MG and SO muscles, might also explain why the improvement in CoP displacement was not retained. Thus, six weeks of no exercise can generate significant reductions in muscle reactive capacity and postural control, indicating that a progressive loss of postural and muscle capacity can occur in the elderly in a relatively short timeframe [2]. This highlights the need for the elderly to participate in regularly-held exercise programs (i.e., without long pauses) so as to sustain the benefits obtained after completing an initial balance training program.

There were some limitations in the study that need to be addressed in future research. First, the training regime adopted in the present study used static balance training. The use of dynamic balance training might have generated more indicative results that may have also been maintained after the detraining period. The evaluation of a perturbation while walking would have served as a more pertinent guide as most falls occur during walking [15]. Another limitation was the absence of kinematic analysis, which could have shown an improvement in other balance strategies other than neuromuscular. It is also suggested that future studies consider the evaluation of mediolateral displacement and those muscles corresponding to this movement direction as well as perform weekly evaluations after the detraining period in order to analyze exactly when and why the losses arising from detraining happen.

Conclusions

Disturbance-based balance training with the use of multidirectional unstable devices improved ankle muscle reactive capability (EMG onset and EMG time-to-peak). This was best demonstrated by EMG amplitude at the early phase of activation during the perturbation test especially for the TA muscle and at the intermediate phase for the MG and SO. However, no changes among the analyzed variables were observed for the hip and knee muscles or for EMG amplitudes of all muscles in the late phase of activation in response to the disturbance.

The improvement in muscle reactive capability may explain better postural control during the perturbation test. However, a six-week period of detraining was enough to reverse the observed improvements in postural control and reactive muscular response, except for the increase in temporal activation of the tibialis anterior muscle.

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