The effect of whole-body vibration on spasticity in post-stroke hemiplegia: A prospective, randomized-controlled study

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Received: December 27, 2021 Accepted: May 05, 2022 Published online: November 22, 2022

ABSTRACT

Objectives: This study aims to investigate whether whole-body vibration (WBV) has an anti-spastic effect on the ankle plantar flexors.

Patients and methods: This single-blind, prospective, randomized-controlled clinical study included a total of 48 patients with chronic stroke (33 males, 15 females; mean age: 60.7±10.9 years; range, 25 to 80 years) between May 2019 and February 2020. They were randomized into two groups: WBV group (n=24) and sham WBV group (n=24). A training program of 12 sessions (three days a week for four weeks) was applied regularly in both groups. The spasticity degree of the plantar flexors was evaluated by using both a subjective assessment method (modified Ashworth scale [MAS]) and several objective assessment methods (Hmax/Mmax, homosynaptic post-activation depression [HPAD], and torque) before and after the training program.

Results: There were no significant changes in the torque values, Hmax/Mmax, and HPAD level after the training program in both groups (p>0.05). However, the MAS score in the WBV group significantly decreased (-9.0%), but no change in the control group was observed (0.7%) (p=0.027, effect size = 0.32).

Conclusion: The objective assessment methods for spasticity show that WBV has no anti-spastic effect.

Keywords: Hemiplegia, rehabilitation, spasticity, stroke, whole-body vibration.

Stroke is one of the most common causes of adult mortality and morbidity worldwide. Post-stroke spasticity is present in about 20 to 40% of stroke survivors. Spasticity is defined as a phenomenon of velocity-dependent increase in tonic stretch reflex (muscle tone) with exaggerated tendon jerks, resulting from hyperexcitability of the stretch reflex. It should be treated, if it restricts daily living activities and has a downstream effect on the quality of life. Recently, whole-body vibration (WBV), as a non-pharmacological treatment, has become popular in spasticity management. Using this method, vibration stimuli of various amplitudes and frequencies are transmitted to the body through the feet that are in contact with the vibration platform. It has been suggested that WBV inhibits muscle spindle Ia afferent activity by making presynaptic inhibition and, thus, increased muscle spindle and gamma motor neuron activity can be controlled. The post-stroke effects of WBV have attracted the interest of many researchers in the rehabilitation area. Chan et al. reported a reduction in ankle spasticity after a single WBV session. Also, in the study of Pang et al., improvements in knee spasticity were observed.
On the other hand, some researchers have reported that WBV has no significant effect on spasticity.\cite{10,11} The most likely explanation for these controversial results includes the subjectivity and the low accuracy of the Modified Ashworth Scale (MAS), although it is widely used in studies.\cite{12} It has been already reported that MAS has low intra- and inter-rater reliability to assess spasticity of the lower extremity.\cite{13,14} Clinicians need reliable measures of spasticity to assess the new rehabilitative interventions, such as WBV. Considering that spasticity is a common complication and strongly associated with difficulties in performing daily living activities, WBV therapy can be a good choice, a safe method in alternative or combined treatment with other anti-spastic therapies. In previous studies, it was recommended that studies with larger sample size and more objective measurements should be conducted in terms of reliability and generalizability of the results.\cite{3-9} In the present study, we aimed to investigate the anti-spastic effect of WBV using objective methods in a large sample size.

**PATIENTS AND METHODS**

This single-blind, prospective, randomized-controlled clinical study was conducted at the Department of Physical Medicine and Rehabilitation of Istanbul Physical Medicine and Rehabilitation Training and Research Hospital between May 2019 and February 2020. The study was registered at ClinicalTrials.gov (NCT03916770). A total of 69 hemiplegic patients were evaluated for study eligibility. Forty-eight patients (33 males, 15 females; mean age: 60.7±10.9 years; range, 25 to 80 years) who met the inclusion criteria were enrolled to the study. Inclusion criteria were as follows: ischemic/hemorrhagic stroke with duration after stroke ≥1 month, plantar flexor spasticity, the ability of standing for more than 5 min and have a good static balance. Exclusion criteria were as follows: a cardiac pacemaker, lower extremity fracture, recent thromboembolism and infectious diseases, polyneuropathy, epilepsy, panic attacks, botulinum toxin A injection in the last six months, absence or highly variable H-reflex response, noncompliant patients. The participants were randomized into two groups: the WBV exercise group (WBV group, n=24) and the sham WBV group (control group, n=24) (Figure 1). Two investigators (Z.R.Y and I.K.) provided the randomization process. A permuted block design of six was used, created by a computer random-number generator, with an allocation ratio of 1:1.

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**Figure 1.** Study flowchart.

WBV: Whole-body vibration.
Procedures

The spasticity degree of the plantar flexors was objectively quantified by using both an electromechanical and electrophysiological method before and after the training program. In addition, MAS, a subjective clinical method, was used for spasticity. All measurements were performed by a single investigator (Z.R.Y.) who was not blinded to the treatment groups to which the patients were assigned. However, the patients had no knowledge of which group they were allocated.

Whole-body vibration exercise protocol

The WBV exercises were applied to both groups in a semi-squat position, three days a week for four weeks, a total of 12 sessions using the Power Plate® Pro5 (Volkel, Netherlands). In the WBV group, the frequency and acceleration of vibration were 30 Hz and 18.0 m/sec², respectively. The WBV exercise intensity was progressively increased throughout the 12-session exercise program (Appendix 1). In the control group, the same procedures were followed. However, unlike the WBV group, a vibration was given of which acceleration was attenuated by 99.5%. All participants completed all exercise sessions. In addition to the WBV, the same conventional rehabilitation program was applied in both groups.

Outcome measures

Measurement of the degree of spasticity

The MAS and the electromechanically measurement were performed simultaneously to evaluate the spasticity degree of the plantar flexors using a custom-made device (Figure 2). Then, soleus H-reflex recordings were obtained and the homosynaptic post-activation depression (HPAD) level and H_{max}/M_{max} ratio were determined.

Modified Ashworth Scale: The MAS tests resistance to passive movement about a joint. The ankle joint in a maximally extended position is placed and moved to a position of maximal flexion over 1 sec. The tester feels the resistance with the hand and scores the magnitude of the resistance between 0 and 4.

Electromechanical method: The electromechanical method measures the resistance to passive movement about a joint. Subjects were requested to lie in the supine position on the examination table and their affected foot was secured to the force plate with Velcro tape. Force plate has a piezoelectric force sensor (FC2211-0000-0100-L Compression Load Cell, TE Connectivity company, Toulouse Cedex, France) placed close to the first metatarsophalangeal joint. The accuracy of the force transducer is ±1%. The resistance was measured as torque and expressed as a kilogram-force meter (kgf.m). The electromechanical method enabled concurrent MAS evaluation (Figure 2). Torque data were recorded with the PowerLab data recording system at a sampling rate of 10 kHz.

Electrophysiological method: The degree of spasticity was evaluated by both H_{max}/M_{max} and HPAD level. Subjects were requested to lie in the supine position on the examination table and relax his/her lower legs throughout the procedure. Before measurement, it was questioned whether any additional condition such as pain, urinating, or defecation would increase spasticity. To record surface electromyography (SEMG), self-adhesive bipolar Ag/AgCl electrodes (KENDALL Coviden, MA, USA) placed with the distance of 4 cm, on the belly of the soleus muscle of the hemiplegic leg, were used. For skin preparation, the area over the soleus muscle was shaved first. Then, a sandpaper was used for rubbing. Finally, the area was cleaned with an alcohol swab and the skin preparation was completed. The ground electrode was placed on the medial malleolus and

![Figure 2. The evaluation of ankle plantar flexor spasticity with custom-made device. The arrow shows a piezoelectric force sensor placed close to the first metatarsophalangeal joint.](image)
the skin on the medial malleolus was prepared also in the same way. To minimize motion artifact, the electrode cables were fixed to the skin with a hypoallergenic plaster. Soleus H-reflex and the M-wave were elicited via electrical stimulation of the tibial nerve in the affected leg. Square pulse stimuli with a width of 1 ms were delivered by a constant current stimulator (FE155 Stimulator HC, AD Instruments, Oxford, UK). An anode (5×10 cm) was placed just above the patella, while a cathode (5×5 mm) was placed at the midpoint of the popliteal fossa. The data acquisition system PowerLab (AD Instruments Co, Oxford, UK) was recorded to obtain SEMG and force data with a sampling rate of 10 kHz and filtered with a 5 to 500 Hz bandpass filter. Recorded data were processed and analyzed offline using LABCHART® Software Version V7.3.3 (AD Instruments, Oxford, UK).

$H_{\text{max}}/M_{\text{max}}$ measurement: The H/M recruitment curves were mapped for the soleus muscle by increasing stimulus intensity gradually until the maximal M-wave ($M_{\text{max}}$) and H-reflex ($H_{\text{max}}$) were obtained. The $M_{\text{max}}$ represents activation of the entire motoneuron pool and, thus, total muscle activation. On the other hand, H-reflex is an estimate of alpha motoneuron excitability. The amplitude of the H-reflex varies among subjects; therefore, we normalized $H_{\text{max}}$ to the $M_{\text{max}}$ ($H_{\text{max}}/M_{\text{max}}$) to compare H-reflexes between subjects and conditions. Nielsen et al. reported that the higher the $H_{\text{max}}/M_{\text{max}}$ ratio, the higher the level of spasticity and, therefore, we use this ratio to evaluate spasticity. When trains of stimuli are delivered with a short time interval, a decrease in H-reflex amplitude occurs. It is called HPAD. In our previous study, the optimal stimulus rate for eliciting the affected side soleus H-reflex without causing HPAD was determined to be 0.1 Hz for stroke patients. Therefore, in this study, we used a frequency of 0.1 Hz to elicit the soleus H-reflex without HPAD.

HPAD level measurement: The stimulus intensity necessary to generate 50% of $H_{\text{max}}$ on the ascending part of the recruitment curve was determined and used to reveal the H-reflex while determining the HPAD level. At this current intensity, two stimuli were given consecutively with a 0.5 s interstimulus interval. These double stimuli were applied 10 times with 10 sec intervals. The peak-to-peak amplitude of the first H-reflex response ($H_1$) was used as the control, and peak-to-peak amplitude of the second H-reflex response ($H_2$) was used as the test to determine HPAD level. Then, $H_2/H_1$ ratio (HPAD level) was calculated. This ratio was determined separately for each of the ten double stimulus pairs and, then, averaged.

Statistical analysis

A prior power analysis was used to estimate minimum required number of participants. (G*Power, version 3.1.9.6, Franz Faul, Universität Kiel, 2019, Germany). The primary outcome variable of the present study was the effect of WBV on motor neuron excitability levels (e.g., $H_{\text{max}}/M_{\text{max}}$ ratio) which reflect spasticity severity. Using data from Chan et al.’s study, the effect size for the motor neuron excitability level ($H_{\text{max}}/M_{\text{max}}$ ratio) of the intervention group was determined as 0.77. A sample size of 24 data pairs achieves 95% power to reject the null hypothesis of zero effect size, when the population effect size is 0.77 and the significance level (alpha) is 0.05 using a two-sided paired t-test for the intervention group. It was also planned to include 24 participants in the control group. Therefore, the total number of participants was determined as 48.

Statistical analysis was performed using the PASW version 17.0 software (SPSS Inc., Chicago, IL, USA). The Shapiro-Wilk test was used to test, if the data were normally distributed. Normally distributed data were presented in mean ± standard deviation (SD), while non-normally distributed data were presented in median and interquartile range (IQR). Categorical variables were presented in number and frequency. For the comparison of the mean of torque data, $2 \times 2$ [Time (before and after Treatment) × Group (WBV group and control group)] two-way mixed analysis of variance (ANOVA) was used. The homogeneity of variance-covariance matrices between groups was confirmed using the Box’s M test. Inter-group comparisons of the mean of normally distributed data were made using the unpaired t-test. Intragroup and inter-group comparisons of the mean of non-normally distributed data were made using the Wilcoxon test and Mann-Whitney U test, respectively. To obtain information regarding the magnitude of between-group differences or within-group differences, non-parametric effect sizes were calculated based on the Z-values obtained from the Mann-Whitney U test or Wilcoxon test using following formula: effect size = $z/\sqrt{n}$. The Yates corrected chi-square or Fisher exact test was used to analyze categorical variables. A $p$ value of <0.05 was considered statistically significant.
RESULTS

Duration of hemiplegia was longer in the WBV group (median: 16.5 [range, 6.5 to 34.5] months) than the control group (median: 8.5 [range, 3.8 to 16.8] months) (p=0.035). Except for the duration of hemiplegia, two groups were found to be homogeneous in terms of their clinical characteristics (Table 1). The mean pre-treatment degree of resistance to passive ankle dorsiflexion in the WBV group and control group was 2.21±0.55 kgf.m and 1.86±0.57 kgf.m, respectively. There was no time effect and no group-by-time interaction (Table 2). The median pre-treatment H_max/M_max ratio in the WBV group and control group was 0.54 (range, 0.44 to 0.73 and 0.55 (range, 0.39 to 0.65), respectively (p=0.585). The median pre-treatment HPAD in the WBV group and control group was 0.66 (range, 0.53 to 0.82) and 0.65 (range, 0.50 to 0.79), respectively (p=0.585). There was no significant change in the H_max/M_max and HPAD level after the training program in both groups. The median pre-treatment MAS score in the WBV group and control group was 3.0 (range, 2.0 to 3.0) and 2.0 (range, 2.0 to 2.0), respectively (p=0.006). The MAS scores decreased significantly after training in the WBV group, but did not change in the control group. The percentage

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**TABLE 1**
Demographic and clinical characteristics of the groups

|                      | WBV group (n=24) | Control group (n=24) | p   |
|----------------------|------------------|----------------------|-----|
| Age (year)           | 59.6±10.1        | 61.8±11.9            | 0.491^a|
| Body mass index (kg/m²) | 27.9±4.6        | 27.1±3.3             | 0.458^a|
| Duration of hemiplegia (month) | 16.5 6.5-34.5 | 8.5 3.8-16.8 | 0.035^b|
| Sex                  |                  |                      |     |
| Male                 | 17               | 16                   | >0.999^c|
| Female               | 7                | 8                    |     |
| Dominant hand        |                  |                      |     |
| Right                | 23               | 22                   | >0.999^d|
| Left                 | 1                | 2                    |     |
| Ischemic             | 19               | 19                   | >0.999^c|
| Hemorrhagic stroke   | 5                | 5                    |     |
| Paretic side         |                  |                      | 0.244^e|
| Right                | 8                | 13                   |     |
| Left                 | 16               | 11                   |     |
| Use of anti-spastic drugs | 18             | 19                   | >0.999^d|
| Yes                  | 18               | 19                   |     |
| No                   | 6                | 5                    |     |
| Brunnstrom stage     | 3.0              | 3.0-4.0              | 0.486^p|
| Functional ambulation score | 3.0 3.0-3.0 | 3.0 2.3-4.0 | 0.876^p|
| Timed up and go test (s) | 36.5 25.3-61.5 | 45.0 26.3-88.8 | 0.261^p|

WBV: Whole-body vibration; SD: Standard deviation; a: Unpaired t-test; b: Mann Whitney U test; c: Yate’s corrected chi-square; d: Fisher exact test.

**TABLE 2**
The severity of spasticity measured in torque (kgf.m)

|                      | WBV (n=24) | Control (n=24) | Time effect | Time × group interaction |
|----------------------|------------|----------------|-------------|--------------------------|
|                      | Mean±SD    | Mean±SD        | F(1, 46)    | Partial η²  | p  | F(1, 46) | Partial η²  | p  |
| Pre-treatment        | 2.21±0.55  | 1.86±0.57      | 0.008       | 0.032       | 0.227 | 0.008     | 0.002       | 0.779 |
| Post-treatment       | 2.11±0.48  | 1.80±0.50      |             |             |       |           |             |     |

WBV: Whole-body vibration; SD: Standard deviation.
Effect of WBV on spasticity

Effect of WBV on spasticity of change for MAS score was significantly higher in the WBV group (-9.0%) than in the control group (0.7%) (p=0.027, effect size (d) = 0.32, power = 17.1%) (Table 3).

**DISCUSSION**

In the present study, we investigated the anti-spastic effect of WBV, which is gaining popularity as a modality for rehabilitation in both types of research and clinical practice. Although quantifying spasticity is difficult, it is important to determine the efficacy of a therapeutic intervention. An objective measure of spasticity is limited by the variability in spasticity from day to day in response to physical or emotional stimuli. Electrophysiological tests and biomechanical methods, such as torque measurement, are helpful to measure spasticity objectively. The main finding of this study is that objective quantitative measurements (Torque, Hmax/Mmax, and HPAD) showed that WBV exercises had no anti-spastic effect. However, MAS evaluation, a subjective clinical method, indicated that WBV exercises might have an anti-spastic effect.

Several studies have previously examined the anti-spastic effect of WBV. Ahlborg et al. reported that eight weeks of intervention with WBV improved knee extensor spasticity. In contrast, the remaining muscle groups showed no significant decrease in MAS scores. Similarly, Pang et al. reported that the knee extensor MAS scores showed a significant reduction. However, no improvement was observed in plantar flexors MAS scores after 24 sessions of WBV in post-stroke patients. Furthermore, Schyns et al. investigated the anti-spastic effect of WBV in multiple sclerosis and showed no improvement in MAS scores. The discrepancy in the results can be attributed to the subjectivity and low reliability of MAS. Despite being commonly used in the measurement of spasticity, MAS has been reported to lack temporal and inter-examiner reproducibility. Moreover, MAS may not detect minute changes due to its qualitative analysis. As Pandyan et al. reported, it can be quite challenging to distinguish between MAS 1, 1+ and 2 scores while measuring. To overcome this problem and evaluate the spasticity more objectively, we used electrophysiological (Hmax/Mmax ratio and HPAD) and electromechanical (torque) parameters in addition to MAS in our study. It is known that post-stroke spastic individuals have a higher Hmax/Mmax ratio, an earlier recovery of the second wave (H2), and a greater second/first wave ratio (H2/H1 ratio) in the double electric pulse examination than healthy

**TABLE 3**

|                         | WBV group (n=24) | Control group (n=24) | Effect size |
|-------------------------|-----------------|----------------------|-------------|
|                         | Mean±SD Median | 1st-3rd quartile     | Mean±SD Median | 1st-3rd quartile | p | p** |
| MAS (Pre-treatment)     | 3.0 2.0-3.0     | 2.0 2.0-2.0          | 0.40 0.006 |
| MAS (Post-treatment)    | 2.0 2.0-3.0     | 2.0 2.0-2.0          | 0.21 0.150 |
| Effect size             | 0.50            | 0.00                 | 0.32 0.027 |
| P value*                | 0.014           | >0.999               |             |
| Change of MAS score (%) | -9.0±16.3       | 0 -25 – 0 0.7±12.5   | 0.03 0.027 |
| Hmax/Mmax (Pre-treatment)| 0.54 0.44-0.73 | 0.55 0.39-0.65       | 0.08 0.585 |
| Hmax/Mmax (Post-treatment)| 0.53 0.39-0.69 | 0.52 0.33-0.76       | 0.01 0.942 |
| Effect size             | 0.16            | 0.06                 |             |
| P value*                | 0.432           | 0.764                |             |
| HPAD (Pre-treatment)    | 0.66 0.53-0.82  | 0.65 0.50-0.79       | 0.08 0.585 |
| HPAD (Post-treatment)   | 0.77 0.57-0.84  | 0.66 0.53-0.78       | 0.15 0.284 |
| Effect size             | 0.34            | 0.11                 |             |
| P value*                | 0.094           | 0.607                |             |

WBV: Whole-body vibration; SD: Standard deviation; MAS: Modified Ashworth Scale; HPAD: Homosynaptic post-activation depression; * Wilcoxon test, ** Mann Whitney U test.
subjects. These are the consequences of complex mechanisms including disinhibitions of peripheral and central origin.[26-28] Whole-body vibration induces a presynaptic inhibition of Ia afferents. Through the mechanism of presynaptic inhibition, the effects of Ia-afferents on motoneurons weaken by reducing the release of neurotransmitters to the motoneurons, resulting in inhibition of the H-reflex amplitudes during vibration.[5,8,29,30] In the light of this information, in our study, we would have expected a significant decrease in $H_{\text{max}}/M_{\text{max}}$ ratio after 12 sessions of WBV. According to our results, although the $H_{\text{max}}/M_{\text{max}}$ ratio and HPAD showed no significant improvement, MAS scores decreased in the WBV group, but not in the control group. Furthermore, Chan et al.[8] reported no significant differences in the $H_{\text{max}}$ amplitude or $H_{\text{max}}/M_{\text{max}}$ ratio after a single session of WBV. However, MAS scores decreased significantly in the WBV group. This discrepancy between MAS scores and H-reflex parameters is similar to that in our study.

In their study, Sayenko et al.[29] reported that WBV caused significant inhibition of the H-reflex in both spinal cord intact and spinal cord injured participants. They also observed that the magnitude of the H-reflex fully recovered after 60 sec of WBV exposure. Kipp et al.[30] demonstrated that the $H/M$ ratio decreased after a 1-min single session of WBV, but this effect was temporary. In our study, H-reflex responses were evaluated not immediately after the intervention, but a day after the 12-session intervention. Considering our results and the literature data, when 12 sessions of WBV are applied, the acute-transient effect of WBV on the H-reflex does not turn into a long-term permanent effect. Simply put, 12-session WBV has no cumulative effect on the H-reflex.

Post-activation depression is a presynaptic mechanism regulating the excitability of the stretch reflex. Decreased presynaptic inhibition and homosynaptic depression are also thought to play a role in the pathophysiology of spasticity.[31] In our opinion, if the vibration is effective in treating spasticity, we would expect HPAD to increase. Although HPAD has been reported to be lower in patients with spasticity in previous studies, we did not observe any significant difference in HPAD after all sessions.[31,32] Following the $H_{\text{max}}/M_{\text{max}}$ assessment, HPAD showed that 12-session WBV had no cumulative effect on spasticity.

In the current study, plantar flexors spasticity was also evaluated electromechanically as resistance to passive dorsiflexion, as a torque. No significant decrease in torque values was observed and, therefore, we concluded that no improvement was found in spasticity biomechanically. Our findings, no improvement in torque values, are in line with the results we obtained with the electrophysiological method.

Similar to our study, Annaswamy et al.[33] reported a discrepancy between the MAS and torque measurements, both of which are mechanical methods. They examined the usefulness of the torque measurement in quantifying spasticity by comparing its use with MAS. The authors demonstrated that the torque seems to be a fair quantitative correlate of the MAS in assessing spasticity and attributed this reasonable level of correlation to the lower reliability of MAS as shown in the previous study.[14] According to the authors, the mechanically imposed stretch (torque measurement) has the advantage of being more reproducible and more consistent across clinical trials than a manually applied stretch. However, due to technical difficulties, the use of torque measurements in the assessment of spasticity is limited.[34]

Although the electrophysiological results of this study are consistent with electromechanical results, there may be some limitations of the H-reflex measurements. First, polyneuropathy was only attempted to be clinically ruled out by performing sensory and motor examinations, but not with electrophysiological examinations priorly. Second, it is unclear that the patients remained calm enough during the procedure.

In conclusion, our study results demonstrate that WBV at the frequency of 30 Hz and the amplitude of 2.2 mm has no anti-spastic effect on the plantar flexors in post-stroke hemiplegia, using the electrophysiological and biomechanical methods. We further suggest that future studies should further examine these suggestions using different frequencies and amplitudes of vibration to obtain precise information on the anti-spastic effect of WBV.

Ethics Committee Approval: The study protocol was approved by the Bakırköy Dr. Sadi Konuk Training and Research Hospital Ethics Committee (no: 2019/117). The study was conducted in accordance with the principles of the Declaration of Helsinki.

Patient Consent for Publication: A written informed consent was obtained from each patient.
Data Sharing Statement: The data that support the findings of this study are available from the corresponding author upon reasonable request.

Author Contributions: Idea, design, data collection, writing of manuscript, references and fundings, literature review: Z.Y.; Data collection and processing and materials: D.E.Z.; Control/Supervision: A.N.B.; Analysis and interpretation, design, critical review: K.T.; Idea, design, analysis and interpretation, critical review: I.K.

Conflict of Interest: The authors declared no conflicts of interest with respect to the authorship and/or publication of this article.

Funding: The authors received no financial support for the research and/or authorship of this article.

REFERENCES

1. Zorowitz RD, Gillard PJ, Brainin M. Poststroke spasticity: Sequelae and burden on stroke survivors and caregivers. Neurology 2013;80(3 Suppl 2):S45-52.
2. Lance JW. Symposium synopsis. In: Feldman RG, Young RR, Koella WP, editors. Spasticity: Disordered motor control. Chicago, IL: Year Book Medical Publishers; 1980. p. 485-94.
3. Huang M, Liao LR, Pang MY. Effects of whole body vibration on muscle spasticity for people with central nervous system disorders: A systematic review. Clin Rehabil 2017;31:23-33.
4. Ribot-Ciscar E. Cutaneous and muscle mechanoreceptors: Sensitivity to mechanical vibrations. In: Rittweger J, editor. Manual of vibration exercise and vibration therapy. Cham: Springer; 2020. p. 87-108.
5. Gillies JD, Lance JW, Neilson PD, Tassinari CA. Presynaptic inhibition of the monosynaptic reflex by vibration. J Physiol 1969;205:329-39.
6. Desmedt JE, Godaux E. Mechanism of the vibration paradox: Excitatory and inhibitory effects of tendon vibration on single soleus muscle motor units in man. J Physiol 1978;285:197-207.
7. Ritzmann R, Kramer A, Gollhofer A, Taube W. The effect of whole body vibration on the H-reflex, the stretch reflex, and the short-latency response during hopping. Scand J Med Sci Sports 2013;23:331-9.
8. Chan KS, Liu CW, Chen TW, Weng MC, Huang MH, Chen CH. Effects of a single session of whole body vibration on ankle plantarflexion spasticity and gait performance in patients with chronic stroke: A randomized controlled trial. Clin Rehabil 2012;26:1087-95.
9. Pang MY, Lau RW, Yip SP. The effects of whole-body vibration therapy on bone turnover, muscle strength, motor function, and spasticity in chronic stroke: A randomized controlled trial. Eur J Phys Rehabil Med 2013;49:439-50.
10. Brogårdh C, Flansbjer UB, Lexell J. No specific effect of whole-body vibration training in chronic stroke: A double-blind randomized controlled study. Arch Phys Med Rehabil 2012;93:253-8.

11. Alp A, Efe B, Adali M, Bilgic A, Demir Türe S, Coşkun Ş, et al. The impact of whole body vibration therapy on spasticity and disability of the patients with Poststroke Hemiplegia. Rehabil Pract Res 2018;2018:8637573.
12. Pandyan AD, Johnson GR, Price CI, Curless RH, Barnes MP, Rodgers H. A review of the properties and limitations of the Ashworth and modified Ashworth Scales as measures of spasticity. Clin Rehabil 1999;13:373-83.
13. Bohannon RW, Smith MB. Interrater reliability of a modified Ashworth scale of muscle spasticity. Phys Ther 1987;67:206-7.
14. Allison SC, Abraham LD, Petersen CL. Reliability of the Modified Ashworth Scale in the assessment of plantarflexor muscle spasticity in patients with traumatic brain injury. Int J Rehabil Res 1996;19:67-78.
15. Tucker KJ, Türker KS. A new method to estimate signal cancellation in the human maximal M-wave. J Neurosci Methods 2005;149:31-41.
16. Hwang IS. Assessment of soleus motoneuronal excitability using the joint angle dependent H reflex in humans. J Electromyogr Kinesiol 2002;12:361-6.
17. Nielsen JB, Crane C, Hultborn H. The spinal pathophysiology of spasticity--from a basic science point of view. Acta Physiol (Oxf) 2007;189:171-80.
18. Yurtturutmu Z, Ekcici Zincirci D, Bardak AN, Topkara B, Aydin T, Karacan I, et al. A stimulus rate that is not influenced by homosynaptic post-activation depression in chronic stroke. Somatosens Mot Res 2020;37:271-6.
19. Fritz CO, Morris PE, Richler JJ. Effect size estimates: Current use, calculations, and interpretation. J Exp Psychol Gen 2012;141:2-18.
20. Rittweger J. Vibration as an exercise modality: How it may work, and what its potential might be. Eur J Appl Physiol 2010;108:877-904.
21. Burry HC. Objective measurement of spasticity. Dev Med Child Neurol 1972;14:508-10.
22. Ji Q, He H, Zhang C, Lu C, Zheng Y, Luo XT, et al. Effects of whole-body vibration on neuromuscular performance in individuals with spinal cord injury: A systematic review. Clin Rehabil 2017;31:1279-91.
23. Ahlborg L, Andersson C, Julin P. Whole-body vibration training compared with resistance training: Effect on spasticity, muscle strength and motor performance in adults with cerebral palsy. J Rehabil Med 2006;38:302-8.
24. Scyons F, Paul L, Finlay K, Ferguson C, Noble E. Vibration therapy in multiple sclerosis: A pilot study exploring its effects on tone, muscle force, sensation and functional performance. Clin Rehabil 2009;23:771-81.
25. Blackburn M, van Vliet P, Mockett SP. Reliability of measurements obtained with the modified Ashworth scale in the lower extremities of people with stroke. Phys Ther 2002;82:25-34.
26. Bakheit AM, Maynard V, Shaw S. The effects of isotonic and isokinetic muscle stretch on the excitability of the spinal alpha motor neurones in patients with muscle spasticity. Eur J Neurol 2005;12:719-24.
27. Burke D, Wissel J, Donnan GA. Pathophysiology of spasticity in stroke. Neurology 2013;80(3 Suppl 2):S20-6.
28. Cho SH, Lee JH. Comparison of the amplitudes of the H-reflex of post-stroke hemiplegia patients and normal adults during walking. J Phys Ther Sci 2013;25:729-32.
29. Sayenko DG, Masani K, Alizadeh-Meghrazi M, Popovic MR, Craven BC. Acute effects of whole body vibration during passive standing on soleus H-reflex in subjects with and without spinal cord injury. Neurosci Lett 2010;482:66-70.
30. Kipp K, Johnson ST, Doeringer JR, Hoffman MA. Spinal reflex excitability and homosynaptic depression after a bout of whole-body vibration. Muscle Nerve 2011;43:259-62.
31. Lamy JC, Wargon I, Mazevet D, Ghanim Z, Pradat-Diehl P, Katz R. Impaired efficacy of spinal presynaptic mechanisms in spastic stroke patients. Brain 2009;132:734-48.
32. Aymard C, Katz R, Lafitte C, Lo E, Pénicaud A, Pradat-Diehl P, et al. Presynaptic inhibition and homosynaptic depression: A comparison between lower and upper limbs in normal human subjects and patients with hemiplegia. Brain 2000;123:1688-702.
33. Annaswamy T, Mallemati S, Allison SC, Abraham LD. Measurement of plantarflexor spasticity in traumatic brain injury: Correlational study of resistance torque compared with the modified Ashworth scale. Am J Phys Med Rehabil 2007;86:404-11.
34. Powers RK, Campbell DL, Rymer WZ. Stretch reflex dynamics in spastic elbow flexor muscles. Ann Neurol 1989;25:32-42.

| Session | 1. Set (sec) | 2. Set (sec) | 3. Set (sec) | 4. Set (sec) |
|---------|-------------|-------------|-------------|-------------|
| 1.       | 10          | 30          | 30          | 30          |
| 2.       | 30          | 30          | 30          | 30          |
| 3.       | 30          | 30          | 30          | 30          |
| 4.       | 30          | 30          | 30          | 60          |
| 5.       | 30          | 30          | 45          | 60          |
| 6.       | 30          | 30          | 60          | 60          |
| 7.       | 30          | 45          | 60          | 60          |
| 8.       | 30          | 60          | 60          | 60          |
| 9.       | 30          | 60          | 60          | 60          |
| 10.      | 60          | 60          | 60          | 60          |
| 11.      | 60          | 60          | 60          | 60          |
| 12.      | 60          | 60          | 60          | 60          |