Research article

Muscle activity, cross-sectional area, and density following passive standing and whole body vibration: A case series

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Objective: To investigate the effects of intermittent passive standing (PS) and whole body vibration (WBV) on the electromyography (EMG) activity, cross-sectional area, and density of lower extremity muscles in individuals with chronic motor complete spinal cord injury (SCI).

Design: Case series.

Methods: Seven adult men with chronic (≥2 years), thoracic motor complete (AIS A–B) SCI completed a 40-week course of thrice-weekly intermittent PS-WBV therapy, in a flexed knee posture (160°), for 45 minutes per session at a frequency of 45 Hz and 0.6–0.7 mm displacement using the WAVE® Pro Plate, with an integrated EasyStand™ standing frame. EMG was measured in major lower extremity muscles to represent muscle activity during PS-WBV. The cross-sectional area and density of the calf muscles were measured using peripheral quantitative computed tomography at the widest calf cross-section (66% of the tibia length) at pre- and post-intervention. All measured variables were compared between the pre- and post-intervention measurements to assess change after the PS-WBV intervention.

Results: PS-WBV acutely induced EMG activity in lower extremity muscles of SCI subjects. No significant changes in lower extremity EMG activity, muscle cross-sectional area, or density were observed following the 40-week intervention.

Conclusions: Although acute exposure to PS-WBV can induce electrophysiological activity of lower extremity muscles during PS in men with motor complete SCI, the PS-WBV intervention for 40 weeks was not sufficient to result in enhanced muscle activity, or to increase calf muscle cross-sectional area or density.

Keywords: Whole body vibration, Spinal cord injury, Muscle

Introduction

Individuals with spinal cord injury (SCI) frequently experience motor disability that can adversely affect their independence and ultimately, their quality of life. In addition, motor impairments contribute to muscle disuse, atrophy, and increased intramuscular fat.[1] This muscle atrophy is due to a decrease in the number and size of motor units and changes in muscle fiber type, specifically a preponderance of Type IIb fibers.[2] The observed declines in muscle size and increases in intramuscular fat content have been predominantly attributed to the lack of voluntary muscle activity below the neurological level of injury.[3] Various rehabilitation interventions have been proposed to counteract muscle atrophy following motor complete SCI such as functional electrical stimulation, body-weight-supported treadmill training, PS, and walking in a exoskeleton. To date, no tool has succeeded in demonstrating

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DOI 10.1179/2045772314Y.0000000255

substantial, sustained increases in lower extremity muscle function, cross-sectional area (CSA), and density.[1]

Whole body vibration (WBV), which provides a mechanical stimulus to the entire body using a vibrating platform, has been proposed as a potential method to improve musculoskeletal health to facilitate musculoskeletal performance and to augment bone density.[4] WBV therapy has been shown to improve muscular performance such as muscle strength after a long-term application in sedentary able-bodied individuals,[5,6] in untrained females,[7–9] and in individuals with a history of stroke.[10] Among individuals with SCI, Ness and Field-Fote[11] applied 4 weeks of WBV therapy in individuals with motor incomplete SCI, and demonstrated that walking speed significantly increased after WBV therapy.

WBV may increase muscle activity by invoking the tonic vibration reflex or stretch reflex during voluntarily standing.[12,13] PS-WBV has also been shown to induce acute modulation of spinal motor neuron excitability in the absence of voluntary muscle contraction[14] and may induce muscle activity in resting muscles of individuals with motor complete SCI.[15] It is reasonable to hypothesize that muscle activity can be enhanced through repetitive application of WBV, since repetitive short-term exposure to WBV may enhance the stretch-reflex loop and subsequently improve neuron excitability and motor unit recruitment within a specific muscle. Indeed, Lapole and Pérot[16] reported that the application of a local vibration using a vibrator on the Achilles tendon during rest for 14 days induced an increase in maximum voluntary contraction and muscle activity during maximum voluntary contraction of the triceps surae in able-bodied individuals.

Thus, PS-WBV therapy has the potential to improve lower extremity muscle function in individuals with SCI. However, the effects of the PS-WBV intervention during standing on muscle electromyography (EMG) activity, area, and density in individuals with motor complete SCI are unclear. Therefore, we sought to explore the potential effects of WBV on muscle activity, size, and density among individuals with motor complete SCI (WAVE® project). During the WAVE® project, we developed a customized WBV platform that incorporated EasyStand™ frame such that individuals with motor complete SCI who are unable to stand independently were able to stand passively on the platform; we refer to this procedure as PS-WBV. We provided intermittent thrice-weekly PS-WBV therapy for 40 weeks to adult men with chronic motor complete paraplegia to investigate the effect of PS-WBV therapy on indices of bone strength as well as body composition. In the current study, we report on the effects of PS-WBV therapy on muscle activity, area, and density.

Methods

Subjects

The inclusion criteria were the following: male, 20–60 years of age at enrollment; with chronic (≥2 years), motor complete paraplegia (T2–T10, AIS A–B) of traumatic etiology with a stable neurological deficit. Subjects included adult men with paraplegia, those with motor complete AIS A impairment and an absence of sensory and motor function in the sacral segments, S4–S5, and those with AIS B impairment with preservation of sensory but not motor function below the neurological level including the sacral segments.[17] In order to eliminate subject heterogeneity and ensure staff and subject safety, exclusion criteria included weight <55 kg or >113 kg; height <168 cm or >189 cm; subjects with a history of, or development of medical conditions which could make WBV exposure unsafe, such as uncontrolled autonomic dysreflexia, untreated orthostatic hypotension; subjects with conditions which make it difficult to stand safely in the device (e.g. hip and knee flexion contractures of >30°); concurrent participation in another intervention study which would confound interpretation of the study results; subject unable to pass the Postural Retraining Protocol to ensure safe standing;[18] and subjects with anemia or vitamin D deficiency which fails to come within the normal serum range after 90 days of supplementation.

All subjects provided their written informed consent to study participation after having received a detailed explanation of the study purpose, and potential benefits associated with study participation. The experimental procedures used in this study were approved by the local research ethics board.

Procedures

Subjects underwent the PS-WBV therapy for 45 minutes three times weekly for 40 weeks at the Lyndhurst Centre, Toronto Rehabilitation Institute – University Health Network. The WBV protocol included intermittent 45 Hz vibration, with a displacement of 0.7 mm on a modified WBV platform (WAVE® Manufacturing Inc., Windsor, Canada)[19] with an integrated EasyStand 5000 standing frame (Altimate Medical Inc., Morton, MN, USA) at a knee angle of 160° of flexion. Each intermittent vibration cycle consisted of 60 seconds of vibration followed by 120 seconds of PS. This cycle was presented 15 times over 45 minutes. The pre-

intervention measurements were conducted prior to or during the first week of the PS-WBV exposure. The post-intervention measurements were conducted within a week of completing the study intervention.

Measurements

Muscle activity during WBV

Surface electromyograms (EMG) of tibialis anterior (TA), soleus (SO), gastrocnemius (GM), the rectus femoris (RF), and vastus lateralis (VL) were measured during PS-WBV to assess the muscle activity. An EMG amplifier was used with a gain set at 5000, a frequency bandwidth of 10–1000 Hz, and a common mode rejection ratio of 115 dB (at 60 Hz) (AMT-8 EMG amplifier, Bortec Biomedical, Ltd, Calgary, Canada). Bipolar silver–silver chloride surface disposable electrodes (10 mm diameter, 18 mm inter-electrode distance) were placed over each muscle following the recommendations of, "Surface Electromyography for the Non-Invasive Assessment of Muscles (SENIAM)".[20] Data were recorded with a data acquisition unit PowerLab 16SP (ADInstruments, Dunedin, New Zealand) at a sampling rate of 2000 Hz.

After the subject was positioned and erect in the standing frame, a 10-second PS EMG sample was taken. During the 15 cycles of the vibration session, EMG was sampled for 10 seconds at four timepoints: 1st, 6th, 11th, and 15th cycle. Sampling commenced after 20 seconds of vibration to ensure steady-state vibration was reached.

The recorded data was analyzed in MATLAB (R2008a, Mathworks, Natick, MA, USA). When recording EMG during PS-WBV exposure, a motion artifact component was picked up by the electrodes. To quantify the muscle EMG activity with this motion artifact eliminated, we introduced a novel signal processing technique that estimated the power of the EMG waveform, while minimizing interference and artifacts from the plate vibration.[15] In brief, the artifact frequency and its harmonics were identified by examination of the EMG signal's power spectral density (PSD) ± 1.5 Hz intervals around those frequencies were then set to zero in the PSD, and the remaining power in the PSD was used to quantify the amount of EMG activity. Signal content above 300 Hz was discarded. Normalization was used to compensate for the different numbers of non-zero entries in the processed PSD functions of each signal. The resulting total power represented the activity of each muscle without the motion artifact. The total power during PS-WBV was normalized by the total power during PS at the same timepoint for comparison among subjects.

Muscle CSA and density

Muscle and subcutaneous adipose tissue CSA and muscle density were calculated from peripheral quantitative computed tomography (pQCT) scans at the site of the widest calf cross-section (66% of the tibia length, measuring distal to proximal) acquired using a Stratec XCT 2000 scanner (Stratec Medizintechnik, Pforzheim, Germany). The right tibia was scanned except in cases of severe spasticity, calf circumference greater than the size of the gantry, presence of metal or prior fracture in the region of interest. Bony landmarks at the medial condyle and medial malleolus were palpated, and a measuring tape was used to measure the distance between the two points to obtain tibia length. The scanner was manually positioned at a line marked on the leg corresponding to 66% of the tibia length, measured proximally from the distal landmark. All images had a slice width of 2.2 mm and voxel size of 0.5 mm. To ensure consistency, the same technologist performed all the scans.

Analysis of pQCT scans was performed using SliceOmatic software (v.4.3 for PC, Tomovision, Magog, Canada). Tissue boundary identification was performed using the watershed tool and manually traced by a single investigator. The calibration equation used by the Stratec software (v6.0) was used to convert units of X-ray attenuation (Hounsfield Units) into density values (mg/cc). The scans of two subjects were excluded due to severe motion artifacts.[21] Muscle CSA and muscle density were calculated from the area and mean volumetric density of the pixels corresponding to the soft tissue beneath the epimysium. Subcutaneous adipose tissue CSA was defined as the pixel area corresponding to the tissue outside of the epimysium. We have established the precision for these assessments in our setting in individuals with and without SCI, i.e. the root mean square coefficient of variation was $<1.5\%$ for muscle CSA and $\leq 2.0\%$ for muscle density.

Statistical analysis

All statistical analyses were performed using Kaleida Graph ver 4.1 (Synergy Software, Reading, PA, USA). Descriptive statistics (mean (standard deviation (SD)) for continuous outcomes and count (%) for categorical outcomes) were used to describe the subjects demographic and impairment characteristics. As data of the total power of EMG for each muscle was not normally distributed, we applied Wilcoxon signed-rank test for each muscle. One sample *t*-tests and Wilcoxon signed-rank test were also used to compare the sample with 1 for muscle activity and 0 for increase percentages of muscle CSA and density. $P < 0.05$ served as the level

of statistical significance. To explore whether PS-WBV resulted in changes in muscle area and density between baseline and post-intervention measurements, paired *t*-test comparisons were performed for each variable.

Results

Subjects

We obtained consent from 14 men who completed screening; one subject withdrew due to an unrelated lower extremity fracture prior to the study intervention; two subjects withdrew consent prior to completing the baseline testing. During the study intervention, four subjects were withdrawn by the investigators: one due to a lower extremity fracture unrelated to the study intervention, one due to sepsis likely related to the study intervention (stone blocking ureter), one subject withdrew consent due to a study-related adverse event (increased back pain) during the PS-WBV intervention, and one subject was withdrawn due to prolonged absence from study intervention. In total seven subjects completed the 40-week intervention, we analyzed the data obtained from these seven subjects as a case series (Table 1).

Muscle activity during WBV

Table 2 shows the results of muscle activity for TA, SO, GM, RF, and VL at pre-intervention and post-intervention. There were no significant differences between the baseline and post-intervention measurements for TA ($P = 0.578$), SO ($P = 0.938$), GM ($P = 0.688$), RF ($P = 0.469$), and VL ($P = 0.109$). At each timepoint (i.e. baseline and post-intervention), the muscle activity during WBV was significantly larger than 1 ($P = 0.016$–0.031) except for TA at the post-intervention ($P = 0.078$), indicating that EMG was larger during WBV compared to PS in most of the muscles.

Table 1 Demographic and anthropometric characteristics of subjects

Subject	Age	DOI (years)	NLI (years)	AIS	Height (cm)	Weight (kg)
A	42	21	T6	A	169	105.0
B	24	7	T4	A	168	55.0
C	38	16	T4	A	170	65.1
D	58	25	T4	A	171	71.7
E	39	17	T6	B	178	97.1
F	54	29	T3	A	169	87.1
G	29	3	T5	A	171	60.8
Mean	40.5	16.9	–	–	171	77.4
SD	12.3	9.3	–	–	3	19.2

DOI, duration of injury; NLI, neurological level of injury; AIS, American Spinal Injury Association Impairment Scale.

Muscle CSA and density

Table 3 shows the pre-intervention and post-intervention values for muscle CSA and muscle density, and the percentage change for each of these variables. There were no significant differences between the baseline and post-intervention measurements in the muscle CSA ($P = 0.687$) and the muscle density ($P = 0.495$). The increase percentages of muscle CSA ($P = 0.644$) and density ($P = 0.473$) were not significantly different from zero, respectively.

Discussion

Our exploratory study suggests that the PS-WBV intervention induced lower extremity muscle activation in the study subjects including those with motor complete paraplegia. In addition, we found that the induced lower extremity muscle EMG activity during PS-WBV did not measurably change after the 40-week application of intermittent PS-WBV intervention. Furthermore, we also noted no significant measurable differences in calf muscle CSA or density following the PS-WBV intervention.

Table 2 Results from muscle EMG activity for TA, SO, GM, RF, and VL

	TA		SO		GM		RF		VL	
Subject	Pre	Post	Pre	Post	Pre	Post	Pre	Post	Pre	Post
A	1.60	3.25	1.19	48.70	1.21	19.39	1.16	7.60	1.23	14.03
B	1.20	2.25	2.95	3.77	1.42	2.65	8.65	30.70	30.07	2.41
C	36.64	1.05	95.13	1.02	34.60	1.14	1005.89	1.40	188.49	1.28
D	1.03	1.14	1.01	1.12	0.87	272.75	1.10	9.99	1.52	1.55
E	1.57	1.15	1.68	1.21	6.00	2.24	4.38	1.17	1.55	1.14
F	222.73	1.01	136.00	1.01	708.69	1.08	186.12	1.18	36.64	1.01
G	1.78	1.30	0.41	1.14	9.99	1.99	26.57	3.88	20.20	1.20
Median	1.60	1.15	1.68	1.14	6.00	2.24	8.65	3.88	20.20	1.28
P-value (vs. 1)	0.016	0.078	0.031	0.016	0.016	0.016	0.016	0.016	0.016	0.016
P-value (Pre vs. Post)	0.578		0.938		0.688		0.469		0.109	

Each subject's value and the median value are presented for each timepoint, i.e. pre-intervention (Pre) and post-intervention (Post). Each P-value at a row of "P-value (vs. 1)" indicates the result of Wilcoxon signed-rank test comparing each sample with 1. Each P-value at a row of "P-value (Pre vs. Post)" indicates the result of Wilcoxon signed-rank test comparing values at the timepoints.

Table 3 Muscle area and density measured using pQCT, and their percentages of change

	Muscle area		Muscle density		%Change	
Subject	Pre	Post	Pre	Post	Muscle area	Muscle density
A	53.7	62.2	63.8	65.1	15.83	1.99
B	32.7	35.5	67.7	68.5	8.69	1.14
C	24.6	25.7	26.6	29.3	4.38	9.95
D	28.9	27.1	41.7	41.5	−6.24	−0.55
E	56.6	54.9	64.1	64.2	−3.02	0.19
F	47.3	42.9	47.1	46.6	−9.47	−0.98
G	28.8	29.1	44.5	43.0	0.87	−3.44
Mean	38.9	39.6	50.8	51.2	1.58	1.19
SD	13.2	14.3	15.0	14.9	8.81	4.24
P-value (pre vs. post)	0.687		0.495			–
P-value (vs. 0)	–	–	–	–	0.644	0.473

For muscle area and density, each participant's value, the mean value, and the SD are presented for each timepoint, i.e. pre-intervention (Pre) and post-intervention (Post); each P-value indicates the result of paired *t*-test comparing Pre and Post. For the percentages of change, each participant's value, the mean value, and the SD are presented for each variable; each P-value indicates the result of one sample *t*-test compared to 0.

Muscle activity during WBV

We hypothesized that the long-term application of PS-WBV would increase muscle lower extremity EMG activity during PS-WBV. However, the results did not support this hypothesis. Acute changes in motor output due to WBV are most often associated with neural factors such as increases in sensitivity of the primary muscle spindle afferent fibers, resulting in facilitation of homonymous alpha motor neurons.[22] These mechanisms, in turn, may lead to increased motor unit recruitment, increased firing frequency, and/or improved synchronization,[23,24] and ultimately result in non-voluntary muscular contraction, i.e. tonic vibration reflex.[25,26] In this study, we examined the chronic and not the acute effects of PS-WBV. Chronic adaptations to a local vibration, in the form of increased maximal voluntary contraction, were reported by Lapole and Pérot[16] for able-bodied individuals. The current results suggest that the application of WBV therapy during PS was insufficient to result in reorganization of the spinal network and to enhance muscle activity during WBV. Further investigation using other WBV intensities (i.e. vibration amplitude and/or frequency) in the subacute stages after injury might yield different results. However, since the WBV intensity was almost at the maximum level tolerable without participant discomfort,[19] using WBV at a higher intensity may not be feasible without adverse effects.

Muscle lower extremity EMG activity was induced by WBV in resting muscles of men with motor complete injury at both timepoints, except for TA post-intervention. Furthermore, the amount of muscle activity was maintained at the post-intervention measurement (i.e. no difference between the baseline and the post-intervention in all muscles). This suggests that acclimation of tonic vibration reflex did not occur over the course of this long-term (40 week) intervention. Because the effectiveness of WBV for acutely eliciting activity in the lower extremity muscles does not appear to decrease over time, and the response is specific to the WBV parameters (direction of oscillation, frequency, amplitude, and plate type), it remains possible that PS-WBV may be beneficial in the future for inducing muscle activity in paralyzed muscles using alternate or refined WBV parameters and or an alternate plate.

Muscle CSA and density

The mechanical stimuli provided with PS-WBV 40-week intervention were insufficient to elicit an increase in muscle CSA, contrary to our hypothesis. There are several published studies reporting the benefits of WBV on increasing muscle CSA or reducing muscle atrophy.[8,27–29] For example, Roelants *et al.*[8] reported that WBV therapy for 24 weeks for untrained females increased fat-free body mass. However, the WBV therapy in these previous studies[8,27–29] involved active voluntary exercise such as performing squats while on the WBV platform. Therefore, it is likely that the muscle stimuli and anticipated muscle response were much larger in these studies, compared to the PS-WBV intervention we studied. Thus, WBV may have the potential to increase muscle CSA with recruitment of a greater proportion of the motor unit using higher intensity WBV stimulation parameters or continuous vibration. In summary, PS-WBV therapy with the current protocol was not sufficient to increase muscle size in individuals with complete SCI.

Muscle density is a proxy for fatty infiltration of muscle,[30] and may not be altered by PS-WBV in individuals with motor complete SCI. Muscle that has increased fatty infiltration will have lower muscle density, or attenuation. There are reports that reduced muscle density is associated with impaired glucose tolerance,[31] incident,[32] disability,[33] and even mortality.[34] Few studies have examined whether interventions targeting muscle can alter muscle density, and those that have been conducted have had small sample sizes, or have included muscle density as one of the number of measures. A study of a weight loss intervention in able-bodied sedentary men and women reported decreased CSA of low density muscle.[35] A few reports of exercise interventions conducted in small samples of

healthy older adults suggest that exercise interventions can increase calf muscle density, however the observed changes were quite variable.[36,37] We did not observe changes in muscle density in response to thrice-weekly PS-WBV in this exploratory pilot study; our stimulus was passive and intermittent, and did not progressively increase in intensity, and our sample size was small. Similar to our findings, an exploratory analysis of data from a large randomized controlled trial (RCT) in post-menopausal women indicated no significant change in muscle density or area in the distal musculature of the lower extremity distal muscles in response to regular standing on a WBV platform.[38] Both studies were short in duration and evaluate the benefit of intermittent vs. continuous vibration. Therefore, intermittent short-term WBV applied during PS may not provide a sufficient stimulus to increase muscle size or density.

Study limitation

Our study was largely exploratory, and has several limitations. We did not have a control group, which limits our ability to make conclusions about the effect of PS-WBV relative to no intervention. It is possible that muscle size or density may decrease over time, and that PS-WBV could prevent that change, but we cannot address that hypothesis with this study. Our sample size was very small, and it is likely that we did not have the statistical power to detect small changes in muscle size or density, if PS-WBV had any effect on these outcomes. Although the method error is <2% for these outcomes, changes in response to intervention in individuals with SCI may be very small, necessitating a large sample size and longer duration of follow-up. For example, a recent RCT revealed that walking assisted by functional electrical stimulation (FES) may result in preservation of muscle size after 12 months, but the between-group differences were very small.[39] That intervention required active muscle contractions stimulated by FES, so it is not surprising that our less intense intervention did not result in significant changes. That said, it is important to conduct pilot studies even if findings are negative, so that realistic expectations about the effect of potential therapies can be generated. It is important to note that the vibration parameters chosen for this PS-WBV intervention were selected for their tolerance by patients with SCI.[40]

Conclusion

Our exploratory study revealed that the 40-week PS-WBV intervention induced EMG activity in the lower extremity muscles (TA, SO, GM, RF, and VL) during PS among individuals with motor complete SCI, but the lower extremity EMG activity did not change after 40 weeks of intermittent PS-WBV therapy. In addition, we failed to find a measurable change in calf muscle CSA and density in response to the PS-WBV intervention. The results of this study suggest that, although acute exposure to PS-WBV can induce muscle activity during PS in paraplegic men, PS-WBV for 40 weeks is an insufficient stimulus to enhance lower extremity muscle activity, CSA, or density.

Acknowledgment

We thank to Mr Jude Delparte and Ms Maggie Szeto for their assistance. The authors acknowledge the support of the Toronto Rehabilitation Institute – UHN which receives funding under the Provincial Rehabilitation Research Program from the Ministry of Health and Long-Term Care in Ontario.

Disclaimer statements

Contributors KM, LG, MRP, and BCC designed the study. MRP and BCC obtained funding. MAM, DGS, and CM collected the data. KM, MAM, CM, and LG analyzed the data. KM, LG, MRP, and BCC interpreted the data. KM, DGS, JZ, LG, MRP, and BCC wrote the article. All authors reviewed and provided input into the manuscript.

Funding The study was supported by Ontario Neurotrauma Foundation (ONF-SCI-2006-WAVE-445) and Rick Hansen Foundation (SCISN Ref# 2010-94S).

Study attribution Toronto Rehabilitation Institute – University Health Network.

Conflicts of interest None.

Ethics approval The experimental procedures used in this study were approved by the Research Ethics Board at Toronto Rehabilitation Institute – University Health Network (REB#08-051).

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