Acute effects of whole body vibration during passive standing on soleus H-reflex in subjects with and without spinal cord injury

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Keywords

Spinal cord injuries, whole body vibration, neuromuscular plasticity, motoneuronal excitability, soleus H-reflex

Abstract

Whole-body vibration (WBV) is being used to enhance neuromuscular performance including muscle strength, power, and endurance in many settings among diverse patient groups including elite athletes. However, the mechanisms underlying the observed neuromuscular effects of WBV have not been established. The extent to which WBV will produce similar neuromuscular effects among patients with neurological impairments unable to voluntarily contract their lower extremity muscles is unknown. We hypothesized that modulation of spinal motorneuronal excitability during WBV may be achieved without voluntary contraction. The purpose of our study was to describe and compare the acute effects of WBV during passive standing in a standing frame on the soleus H-reflex among men with and without spinal cord injury (SCI). In spinal cord intact participants, WBV caused significant inhibition of the H-reflex as early as 6 s after vibration onset (9.0 ± 3.9 %) ($p<0.001$). The magnitude of the H-reflex gradually recovered after WBV, but remained significantly below initial values until 36 s post WBV (57.5 ± 22.0 %) ($p=0.01$). Among participants with SCI, H-reflex inhibition was less pronounced with onset 24 s following WBV (54.2 ± 18.7 %) ($p=0.03$). The magnitude of the H-reflex fully recovered after 60 s of WBV exposure. These results concur with prior reports of inhibitory effects of local vibration application on the H-reflex. Our results suggest that acute modulation of spinal motoneuronal excitability during WBV can be achieved in the absence of voluntary leg muscle contractions. Nonetheless, WBV has implications for rehabilitation service delivery through modulation of spinal motoneuronal excitability in individuals with SCI.

Introduction

Whole-body vibration (WBV) constitutes mechanical stimuli that enter the human body via the feet when standing on a vertical or oscillating platform. Nowadays, vibrating platforms are commercially available and typically utilize low-frequency and low-amplitude vibration, thereby delivering strong stimuli to the skeletal muscles of the lower body [9]. WBV is being extensively used to enhance sports training [9, 16, 25, 29]; improvements in muscle power and strength [17, 26], and force-velocity [29] can be achieved during a relatively short time period [6, 7, 9] with WBV. WBV may have similar positive attributes when used in the rehabilitation of patients with neuromuscular impairments such as spinal cord injury (SCI). The mechanisms underlying the observed neuromuscular benefits of WBV have not been established.

It has been assumed that the effects of WBV on muscle performance are elicited through increases in gravitational load leading to "neurogenic adaptation" [6, 7, 9], and/or hormonal changes [3, 8, 9]. However, acute changes to motor output due to WBV are most often associated with neural factors such as increases in sensitivity of the primary muscle spindle (Ia) afferent fibers, resulting in facilitation of homonymous alpha motor neurons [9, 25, 29]. The last, in turn, may lead to increased motor unit recruitment, increased firing frequency, and/or improved synchronization [29], followed by an increase in background muscle activity and resultant force (tonic vibration reflex) [5, 24]. At the same time, it has been demonstrated that a strong discharge in homonymous Ia fibres due to local application of vibration to a muscle or its tendon [8] results in depression of that muscle's stretch reflex and H-reflex [13, 15, 24]. Vibration induced enhancement of motor discharge along with the concurrent reflex depression is known as the "vibration paradox" [15, 24]. This phenomenon supports previously published data demonstrating that exposure to WBV enhances muscle performance [9, 16, 25, 29] on the one hand, and a lack of change in the patellar tendon stretch reflex [11, 20] or significant inhibition of the soleus H-reflex [3] on the other hand.

The neuromuscular effects of vertical WBV have been reported after vibration in a squat [9, 26] or standing posture with bent knees [1, 3, 14, 23] performed on the platform. However, the extent to which WBV without voluntary muscle activity will produce neuromuscular effects is unknown. We investigated the effects of WBV during passive standing on the H-reflex. H-reflex is routinely used to study the adaptive plasticity arising in spinal reflex pathways as a result of exercise training [30]. We hypothesized that modulation of spinal motorneuronal excitability during WBV may be achieved without voluntary contraction. Acute changes in motorneuronal excitability during WBV in passive standing would suggest that WBV has positive implications for utilization in individuals with neurological impairment. The purpose of our study was to describe and compare the acute effects of WBV during passive standing in a standing frame on the soleus H-reflex among spinal cord intact participants and individuals with SCI.

Materials and methods

Participants

Experiments were conducted in eight spinal cord intact and six SCI participants (Table 1). Participants provided written informed consent to the experimental procedures, which were approved by the local Research Ethics Board in accordance with the declaration of Helsinki on the use of human subjects in experiments.

EMG recording

Surface EMG signals were recorded via bipolar surface electrodes (Ag/AgCl, diameter of 9 mm, Bortec Biomedical Ltd., Canada) placed longitudinally on the right soleus muscle distal to the belly of the medial gastrocnemius, medial to the Achilles tendon, with an inter-electrode distance of 23 mm. A reference electrode (Ag/AgCl, diameter of 7 mm, Bortec Kendall Medi-Trace, Canada) was placed over the proximal fibular head. The EMG signals were amplified and acquired using AMT-8 EMG system (Bortec Biomedical Ltd., Canada) with a frequency bandwidth 10–1000 Hz, and a common mode rejection ratio of 115 dB (at 60 Hz). Finally, the EMG data of the right soleus were digitized at a sampling rate of 10 kHz.

Elicitation of soleus H-reflex

The tibial nerve was stimulated by a hand-held monopolar stainless-steel electrode with a diameter of 1 cm (cathode), placed on the skin above the posterior tibial nerve within the right popliteal fossa. The anode, a 45-cm^2 self-adhesive gel electrode (ValuTrode, Denmark), was placed superior to the patella. Having established the optimal location for the stimulation, the hand-held electrode was then replaced by a pre-gelled disposable electrode (Ag/AgCl, diameter of 7 mm, Bortec Kendall Medi-Trace, Canada), and a neoprene knee brace was used to hold the two electrodes in place during data collection.

The H-reflex was evoked by electrical stimulation (1 ms pulse width) using a constant voltage stimulator (Master-8, A.M.P.I., Israel). For normalization of the H-reflex, the amplitude of the soleus maximal M-wave (M_{max}) after supramaximal stimulation of the posterior tibial nerve was calculated by averaging the five highest peak-to-peak M-waves [30]. The stimulation intensity for the H-reflex was chosen to generate a control reflex with an amplitude equivalent to 20 to 30 % of M_{max}. With this stimulus intensity, the M-wave was continuously monitored to ensure that the same Ia afferents were excited during each stimulation [24]. For each test condition, ten soleus H-reflex responses were evoked every six seconds [24]. The reflexes were evoked under the same postural orientation and ‘‘set’’ of the participant to ensure the recording conditions.

Vibration intervention

Participants were exposed to 1 min of vertical WBV at 35 Hz and 1-mm peak-to-peak amplitude on a modified version of the WAVE® vertical WBV plate (WAVE Manufacturing Inc., Windsor, Canada). The modifications included two additional motors to provide more precise adjustment of vibration frequency and peak-to-peak amplitude. Additionally, a conventional standing frame EasyStand 5000 (Altimate Medical, USA) was mounted above the plate of the WAVE® platform. The standing frame provided external support enabling participants to passively maintain a standing posture while the frame secured their knees and pelvis (Fig. 1).

Experimental procedure

During the experiments, participants were supported by the standing frame with the feet strapped to the WAVE® platform. The participants’ hip and knee joint angles were fixed at 160º of flexion, and the ankle joints to a neutral position. The joint angles were confirmed

with a goniometer prior to administration of the WBV. Ten H-reflex responses were elicited before, during, and immediately after WBV exposure.

Data processing and analysis

The magnitude of the H-reflex responses was calculated by measuring the peak-to-peak amplitude of each response. Soleus H-reflexes were accepted once there was no statistically significant difference between the recorded M-wave amplitudes before, during, and after WBV exposure. The magnitudes of the H-reflex responses during and after WBV were expressed as percentages of the mean amplitude of the ten H-reflexes recorded prior to WBV for each participant. Then, a two-way ANOVA with repeated measures ($\alpha = 0.05$) and a post-hoc Tukey test were applied to the pooled data to identify significant differences in the magnitude of the H-reflex responses during and after WBV, and between non-SCI and SCI participants. Results of the pooled data are presented as mean values ± standard deviations (SD).

Results

Neither the spinal intact nor SCI participants showed any background EMG activity in the soleus muscle during passive standing prior and after WBV.

Figure 2, displays the soleus H-reflex responses immediately before, during, and after WBV in one spinal cord intact and one SCI participant. WBV resulted in a significant inhibition of the soleus H-reflex in the SCI and non-SCI participants.

The pooled effect of WBV on the magnitude of the H-reflex in spinal cord intact and SCI participants is illustrated in Fig. 3. The changes in the H-reflex magnitude in all trials occurred without significant changes in the M-wave: among spinal cord intact participants its values reached 5.1 ± 2.5 %, 5.1 ± 3.3 % (p=0.78), and 5.2 ± 2.4 % (p=0.44) of M_{max} before, during, and after WBV, respectively. Among SCI participants the M-wave reached 4.6 ± 1.7 %, 4.6 ± 1.6 % (p=0.85), and 4.6 ± 1.9 % (p=0.62) of M_{max} before, during, and after WBV, respectively. The effect of WBV in time on the soleus H-reflex was different in the two groups. Among participants with an intact spinal cord, the magnitudes of the reflexes obtained were significantly reduced during WBV, with pronounced inhibition within 6 s of vibration onset reaching 9.0 ± 3.9 % (p<0.0001) of the initial values. After 60 s of WBV exposure, the H-reflex remained suppressed, reaching 5.8 ± 3.8 % (p<0.0001) of the initial values. After WBV, H-reflex magnitudes gradually increased, remaining below the initial values until 36 s post WBV, reaching 57.5 ± 22.0 % (p=0.01) of the initial values. In contrast, WBV produced less inhibition of the H-reflex with significant changes at 24 s following WBV, reaching 54.2 ± 18.7 % (p=0.03) of the initial values among SCI participants. During WBV, the magnitudes of the H-reflex gradually decreased reaching a maximal inhibition of 49.2 ± 12.2 % (p=0.005) after 60 s of WBV. After WBV, the H-reflex remained suppressed for 6 s, reaching 41.2 ± 20.1 % (p=0.0002) of the initial values. However, the magnitude of the H-reflexes recovered to the initial values at 60 s after WBV 108.5 ± 19.5 % (p=0.99).

Discussion

In the present study, we found that WBV during passive standing produced inhibition of the soleus H-reflex in both spinal cord intact and SCI participants, thereby suggesting that acute modulation of spinal motoneuronal excitability during WBV can be achieved without voluntary contraction of the leg muscles. The inhibition of the H-reflex was less pronounced in individuals with SCI.

Activation of the vestibular system can be acknowledged as a potential factor leading to the H-reflex modulation during WBV. However, in research of Aiello et al. [2] the soleus H-reflex increased as a participant was inclined from supine to upright posture on a tilt table. These results suggest that the descending inputs from vestibular system have a facilitatory effect on the H-reflex pathway. The H-reflex result in the present study showed the opposite response, that is, the suppression of H-reflex magnitude during WBV. As such, it is unlikely that the revealed inhibition of the H-reflex is linked with activation of the vestibular system by WBV.

The observed effects support previous findings demonstrating inhibitory effects of WBV and/or local application of vibration to a muscle or its tendon on the H-reflex.

It has been shown, that the soleus H-reflex was significantly suppressed after 1 min of WBV during standing with the knees flexed approximately 10 degrees [3]. Armstrong et al. suggested that the H-reflex might be suppressed due to the muscle fatigue caused by WBV [3]. Indeed, it was reported that a prolonged superimposed vibration (> 10–20 s) accentuates the fatigue-induced decline in contraction force during sustained dorsiflexion [5]. However, in light of the current experimental protocol it is unlikely that muscle fatigue was the primary origin of the H-reflex inhibition observed in our study: First, the inhibition of the H-reflex was revealed as early as 6 s after WBV onset in spinal cord intact participants, and 36 s after WBV in individuals with SCI. Second, the H-reflex inhibition was less pronounced among participants with SCI, whereas there is the physiological evidence that the normally fatigue-

resistant soleus muscle transforms to faster fatigable muscle as a result of muscle fiber type transformation after SCI [27].

Additional mechanisms that may contribute to the vibratory-induced reflex depression were obtained from experiments in humans with local application of vibration to a muscle or its tendon [4, 8, 24, 28], and include an increased firing threshold of Ia afferent fibers [10, 19], presynaptic inhibition of Ia terminals with primary afferent depolarization [21, 24], and post-activation depression due to repetitive activation of the Ia motoneuron synapse [21, 24] followed by reduced probability of transmitter release [12].

An increase in threshold of Ia afferent fibers due to their selective activation was revealed after a *prolonged* (i.e., 20 min) high frequency, low amplitude vibration [19, 24], and are unlikely to explain the early acute depression of the H-reflex observed in this study.

It is arguable which mechanism, presynaptic inhibition with primary afferent depolarization or post-activation depression, is more feasible for the revealed WBV induced reflex depression. Interestingly, it has been shown that the intensity of both inhibitory mechanisms is reduced in individuals with SCI [4, 24, 28]. Following SCI, presynaptic inhibition of Ia terminals of the lower limbs decrease, due to interruption of descending pathways which help to maintain a tonic level of presynaptic inhibition of Ia terminals in the intact spinal cord [24, 28]. Additionally, it has been shown that post-activation depression following previous activation of Ia afferent fibers is reduced after SCI [22]. This reduction could be a consequence of altered muscle use due to motor impairment, and/or could be associated with spasticity [22]. Therefore, the less pronounced inhibition of the H-reflex due to WBV among participants with SCI supports the notion that either presynaptic inhibition with primary afferent depolarization or/and post-activation depression could result in WBV induced inhibitory effect.

The present results concur with prior findings that repetitive use of an afferent stimulus in the form of WBV induces persistent plastic changes in the neural circuits related

to spinal reflex excitability [18], and may result in a progressive decrease in spastic hypertonia during WBV intervention following SCI [23]. Thus, it can be suggested that WBV during passive standing may be used to decrease spasticity in individuals for whom gastrocnemius and soleus spasticity interferes with their functional abilities. It is unclear whether the revealed effects of WBV during passive standing on spinal motorneuronal excitability will persist for a sufficient period of time to produce corresponding improvements in muscle performance. Further research is needed to explicate the effects of WBV during passive standing on muscle functional properties among individuals with SCI.

Study Limitations

The study examined only male participants of similar body mass index, one mode of WBV, and limited periods of WBV exposure and recovery. The differences in age between the two groups may account for some of the observed variability in H-reflex between the two groups. Further research is required to determine to what extent the H-reflex might be modulated during WBV in participants with complete versus incomplete SCI.

Conclusion

The present study demonstrated that WBV during passive standing caused significant inhibition of the soleus H-reflex in male participants with and without SCI, suggesting that acute modulation of spinal motoneuronal excitability can be achieved during WBV without voluntary contraction. These findings concur with prior reports of inhibitory effects of vibration on the H-reflex after local application of vibration to a muscle or its tendon. Either presynaptic inhibition with primary afferent depolarization and/or post-activation depression could result in the inhibitory effects observed with WBV. The inhibition of the H-reflex

among participants with SCI during WBV was less pronounced. Nonetheless, WBV has the potential to modulate spinal motoneuronal excitability in individuals with SCI thereby opening the field for future related innovations in spasticity management and rehabilitation service delivery.

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Figure Legends

Figure 1. An overall view of the whole body vibration (WBV) system mounted with standing frame.

Figure 2. Raw soleus H-reflexes obtained for spinal cord intact participant and participant with SCI immediately before, during, and after WBV.

Figure 3. Pooled data showing the effect of WBV on the soleus H-reflex among spinal cord intact participants and SCI participants before, during, and after WBV. The values are presented as percentages of the mean values (mean ± SD) obtained prior to WBV (n = 10). Asterisks indicate statistically significant differences between the initial values and those during and after WBV (* $P < 0.05$). Cross-symbols indicate statistically significant differences between spinal cord intact participants and individuals with SCI († $P < 0.05$).