©International Journal of Motor Learning & Sport Performance
ISSN: 2230-8628

# Low-Frequency Vibration of the Biceps Brachii Does Not Alter the Functional Properties of the Corticospinal Pathway

**Dawson J. Kidgell[1] and Alan J. Pearce[2]**

[1]*Centre for Physical Activity and Nutrition Research, Deakin University, Melbourne* ***(Australia)***
[2]*School of Sport and Exercise Science, Victoria University, Melbourne* ***(Australia)***

**Abstract**—*Several investigations have reported increased neuromuscular function following acute bouts of low-frequency vibration applied to the upper and lower limbs. Adjustments in neuromuscular function following vibration have been suggested to arise from modifications in the excitability of the descending neural pathways; however, no study has investigated acute corticospinal responses following low-frequency vibration applied to the upper limbs. The aim of this study was to determine the post-vibratory effects following a single bout of low-frequency vibration on corticospinal excitability and muscle torque of the biceps brachii muscle. Participants (n = 10) received two treatments in a balanced randomized order. Participants positioned themselves in a conventional push-up on a Whole Body Vibration platform (VibroGym) and were exposed to 3 sets of 60 s vertical sinusoidal vibration (frequency of 35 Hz and amplitude of 4 mm). One treatment was performing a static hold push-up whilst vibration was applied (V+, experimental condition), whilst the other treatment was without vibration (V-, sham) Transcranial Magnetic Stimulation (TMS) was applied prior to and immediately following V+ or V- over the participant's left motor cortex projecting to the right BB, to measure latency and corticospinal excitability. Elbow flexion torque was also measured before and after each treatment. TMS was delivered at 10% of the root mean square electromyographic (rmsEMG) signal obtained from a maximal voluntary contraction (MVC) at and 20% above active motor threshold. No significant differences for motor evoked amplitude at motor threshold (MT) (pre V- 0.71 ± 0.25 mV; pre V+ 0.69 ± 0.23 mV; p > 0.05) and 20% above motor threshold (post V- .2.2 ± 1.4 mV; post V + 2.0 ± .1.3 mV; p > 0.05) were observed between conditions. There was also no difference in elbow flexion torque between conditions. These results suggest that acute low-frequency vibration exercise applied to the upper limbs does not alter the efficiency of neural transmission along the corticospinal pathway or the post-synaptic excitability of the motoneuron pool projecting to the BB.*

**Keywords:** *Motor performance, strength, whole body vibration, transcranial magnetic stimulation*

## Introduction

The application of low-frequency (i.e. 10-50 Hz) vibration stimuli to the body through whole-body vibration (WBV) platforms or parts of the body (direct vibration) via high frequency (i.e. > 65 Hz) vibration has been shown to enhance corticospinal excitability in response to sensory receptor stimulation (Rosenkranz and Rothwell, 2003; Mileva, Bowtell, and Kossev, 2009). During vibration, the mechanical action of the oscillatory motion produces a rapid change in the length of the muscle-tendon complex, which stimulates the primary endings of 1a muscle afferents and to a lesser extent the Golgi afferent (1b), leading to excitation of á-motoneurons of the homonymous motor units (Martin and Park, 1997). Whilst segmental structures modulate the initial stages within the motor feedback loop in response to vibration,

central projections from supraspinal structures (such as the primary motor cortex and corticospinal pathway) are also important in generating the efferent neural response to afferent input (Lewis, Byblow, and Carson, 2001; Rothwell, and Rosenkranz, 2005; Chez, and Krakauer 2000).

Accordingly, modifications in 1a muscle afferent activity and corticospinal responses may be important corticospinal mechanisms that contribute to the observed effects of acute low-frequency vibration exercise. Increases in neural drive, motor unit synchronisation and recruitment have been reported during exposure to low-frequency vibration stimuli (Shinohara, 2005; Cormie, Deane, Triplett and McBride, 2006). This finding has been demonstrated in the applied context where low-frequency vibration has been used as a training intervention in athletic populations (Hazell, Jakobi, and Kenno, 2007). Acute low-frequency vibration has been shown to induce transient increases in the electrical activity of the muscle being vibrated (25–45 Hz) during submaximal isotonic and isometric contractions (Hazell, et al. 2007; Roelants, Verschueren, Delecluse, Levin, and Stijnen, 2006; Mileva, Naleem, Biswas, Marwood, and Bowtell, 2006). However, the acute use of low frequency vibration as a method to potentiate neuromuscular excitability remains unclear with limited study focused on the corticospinal pathway.

Changes in corticospinal excitability can be measured using transcranial magnetic stimulation (TMS) during voluntary contractions, making it possible to measure the corticospinal control (and efficacy of neural transmission) to a muscle (Hallet, 2007). If the intensity of the TMS stimulus is appropriate, it will depolarize the axons of presynaptic neurons that project onto corticospinal neurons that descend onto the spinal motoneuron pool to activate the muscle of interest. The subsequent transient muscle response can be recorded by surface electromyography (sEMG) and quantified as the motor evoked potential (MEP; Hallet, 2007).

It is well documented that afferent input via somatosensory stimulation (e.g. vibration) is capable of inducing organisational change within the sensory and primary motor cortex (Rothwell, JC, Rosenkranz 2005; Ridding, Brouwer, Miles, Pitcher, Thompson, 2000). Kossev et al. (1999) demonstrated that trains of 4 s high-frequency vibration (80 Hz, 05 amplitude), resulted in a significant increase in MEPs following single pulse TMS. Furthermore, Munte et al. (1996) also demonstrated increased corticospinal excitability during high-frequency vibration at 80 Hz whilst using the electroencephalography (EEG) technique. Based upon this data, sensory-stimulation via high-frequency vibration increases corticospinal excitability via acute adjustments in the activity of 1a afferents and changes in post-synaptic excitability of the motoneuron pool (as seen with increased MEPs during vibration). Therefore, the potential use of vibration may have important clinical implication in rehabilitation following neuromuscular injury.

Although several studies have demonstrated modifications in corticospinal excitability *during* high-frequency and low-frequency vibration (Mileva et al, 2009; Kossev et al, 1999), there is limited data available on the post-vibratory effect of low-frequency vibration applied to the upper limb on corticospinal excitability. In the only study to date, Mileva et al. (2009) assessed the acute effect of low-frequency vibration on corticospinal responses evoked by single and paired pulse TMS of a lower limb muscle and demonstrated facilitated MEPs only *during* vibration.

Therefore, the aims of the study were to determine the acute corticospinal responses from single pulse TMS following a single bout of low-frequency vibration exercise applied to the upper limb. Given that previous evidence suggests, increased corticospinal excitability *during* low-frequency vibration (Mileva et al, 2009), we hypothesized that acute exposure of the upper limb to low-frequency vibration would increase corticospinal excitability immediately *following* vibration exposure.

## Methods

### Participants

Ten healthy male participants (mean age 28.4 ± 5.9 years), with no history of neuromuscular impairment, participated in the study. Written informed consent was obtained from the participants and ethical approval was granted from the University Human Ethics Committee which conformed to the Declaration of Helsinki.

### Organisation of the Study

Participants performed two treatments, namely, a static hold in a push-up position with low-frequency vibration (V+) and without low-frequency vibration (V-), in a randomised balanced order with 48 h separating each intervention. Participants were familiarised with all procedures and equipment prior to their first testing session, undertook testing at the same time of day and were instructed to refrain from partaking in any vigorous physical activity 24 h prior to testing.

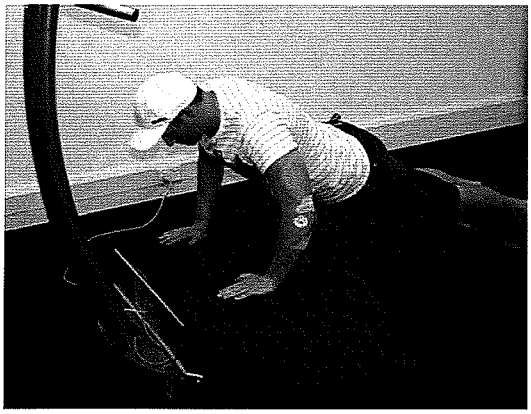
### Measurement of Maximal Voluntary Strength (MVC)

Measure of maximal elbow flexion torque was performed using a portable hand-held dynamometer (Microfet², USA), which has been previously shown to be a reliable measure of maximal voluntary strength, in adults, particularly in elbow flexion (Stuberg and Metcalf, 1988; $r$=0.74-0.99). Measurement of maximal voluntary contraction (MVC) strength required participants to be lying supine on a massage table with knees flexed to 90° and feet flat on the table. With the arm flexed to 90°, the dynamometer was positioned on the inside of the participant's forearm at the level of the wrist. The investigator's other arm was placed on the participant's bicep to ensure that the proximal segment of the upper limb remained in complete contact with the table during performance of each MVC. The participant was then asked to push against the dynamometer (and the examiner's hand) as forcefully as possible for 3 s. Following a rest period of 30 s (to ensure that the participant was not fatigued), two further attempts were performed. This procedure was repeated for the participant's other arm. If the result of the third trial was found to be the highest the test would be repeated, until the third test did not result in the highest test value. The highest acceptable value obtained was recorded for analysis and the participant was not provided feedback about the result.

### Whole-Body Vibration Intervention

Participants were required to position themselves on top of a commercial whole-body vibration (WBV) platform (vibroGym, Australia) in a conventional static push-up position (Figure 1a) with their hands positioned according to their bi-acromial width with the elbow flexed 10° (Fees, Decker, Snuder-Mackler and Axe, 1998). During the static push-up holds, the participants were provided with feedback of the elbow joint angle to ensure that no variation in joint angle occurred between sets and conditions. To negate the possibility of discomfort to the volar aspect of the hands a thin foam mat (10 mm) was placed over the machine. Whilst in this position, participants were exposed to three sets of 60 s bouts of vertical sinusoidal vibration, separated by a 60 s recovery period which involved the participant to sit and rest. In accordance with previous research, the vibration frequency was set at 35 Hz with a peak-to-peak amplitude of 4 mm (Mileva et al, 2006). Prior to and immediately after the third set vibration exposure, TMS was applied to the contralateral motor cortex (M1) projecting to the right biceps brachii (BB) muscle.

During the sham condition (V-), a custom made wooden box, with a 2 cm clearance was placed over the WBV platform to ensure that the vibration machine could be activated to the exact same parameters as for the vibration condition with the participants in the same static push-up position (Figure 1b) but without the vibration stimulus reaching the participants limbs. Similar to the V+ treatment, TMS was applied prior to and immediately (i.e. within 60 s) following the third set of V- condition.



A

B

**Figure 1: Participant Set Up Illustrating Push-up Position during the WBV+ (a) and WBV- (b) Sham Trials.**

### Electromyography (EMG) and Transcranial Magnetic Stimulation (TMS)

Prior to testing, the participant's maximum root mean square EMG (rmsEMG) was determined by a 3 s maximal voluntary contraction (MVC) of the right BB muscle which required the participants to be seated in a chair with the elbow flexed to 90°, measured by an electronic goniometer (Biometrics, USA), with their hand in a supinated position. Biofeedback of the participants' joint angle was provided on a computer screen 1.5 m away at eye level. A portable dynamometer (Microfet[2], USA) was positioned on a modifiable bench so the dynamometer was on the volar aspect of the participant's forearm at the level of the wrist. The participant was then instructed to flex the elbow against the dynamometer as forcefully as possible for 3 s. Three attempts, with a two-minute rest between each attempt were performed. The trial with the highest MVC and rmsEMG level was recorded and subsequently used to determine background muscle activity during the TMS protocol, pre and post vibration.

Surface EMG activity was recorded from the right BB muscle using bipolar Ag-AgCl electrodes. Two electrodes were placed 2 cm apart over the BB muscle, with the third reference electrode placed over the lateral epicondyle. EMG signals were amplified (1000×) with bandpass filtering between 10 Hz and 1 kHz and digitised at 1.5 kHz for 500 ms using custom-designed software (National Instruments V4.0). TMS was delivered using a Magstim 200[2] stimulator (Whitland, UK) with a 70 mm diameter, figure-of-eight coil. The handle of the TMS coil was positioned towards the back of the head at approximately 45° to the sagittal plane at the optimal coil position to elicit an MEP in the right BB muscle, thus inducing a posterior to anterior current flow. A snugly fitting cap, with pre-marked sites at 1 cm spacing was placed over the participant's head and positioned with reference to the nasion-inion and interaural lines. Sites near the estimated centre of the BB area (4–7 cm lateral to the vertex) were first explored to determine the site at which the largest MEP could be obtained. Active motor threshold (AMT) was defined as the intensity at which an MEP could be observed, at background contraction level of 10% of MVC rmsEMG, in at least five of ten stimuli (Pearce and Kidgell (2010). Once AMT was determined, stimulus intensity was set at 20% (of stimulator output), above AMT. TMS trials consisted of two sets five stimuli, for both conditions. A pause of 10 s was given between stimuli, with 30 s rest following each set of five stimuli. For all TMS trials, participants were required to maintain a voluntary isometric contraction of 10% rmsEMG.

All MEPs collected at AMT and 20% above AMT were displayed and averaged online for visual inspection and then stored off-line for further analysis. For more information and illustration regarding the cursoring of latency period and evoked potentials the reader is referred to Pearce and Kidgell (2010). Shapiro-Wilk tests showed non-normally distributed data, ($SW$ = 0.84 $p$ = 0.04), therefore, Wilcox on Signed Ranks tests and effect sizes ($ES$; trivial <0.20; small 0.21-0.49; moderate 0.5-0.79; large >0.80) were performed to compare corticospinal excitability between conditions (V+ vs. V-) and time (pre vs. post). Data is presented as means (± SD) and for all comparisons, with alpha set at $p < 0.05$.

## Results

### Maximal Strength

Figure 2 shows MVC elbow flexion torque pre and post intervention between conditions. There were no changes in MVC pre and post intervention for either the V+ (pre 254.1 ± 64.9 N vs post 248.7 ± 60.5 N; $p$=0.16; $ES$=0.08) or V- (pre 237.3 ± 49.3 N vs post 232.7 ± 50.8 N; $p$=0.31; $ES$=0.09)

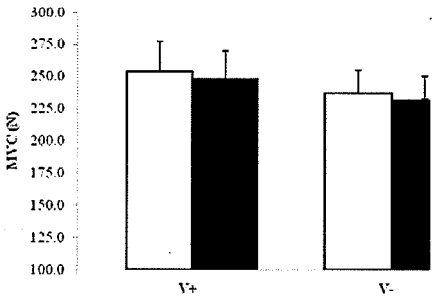



**Figure 2: Maximal Voluntary Contraction (MVC) Elbow Flexion Torque Pre (white bars) and Post (black bars) for the Whole Body Vibration (V+) and Sham (V-) Conditions. No Significant Differences in MVCs Where Observed between Conditions Pre and Post Intervention.**

### Corticospinal Latency Period

There were no significant differences in latency period at AMT between conditions at baseline (pre V- 13.2 ± 1.1 ms; pre V+ 13.2 ± 0.9 ms; $p$=1.0; $ES$<0.01) or 20% above AMT (pre V- 13.1 ± 1.3 ms; pre V+ 13.1 ± 0.8 ms; $p$=0.81; $ES$<0.01). Post testing between conditions also revealed no significant differences in latency duration at AMT (post V- 13.0 ± 1.0 ms; post V+ 13.2 ± 0.9 ms; $p$=0.7; $ES$<0.01) and at 20% above AMT (post BV- 13.2 ± 0.9 ms; post V + 13.1 ± 0.9 ms; $p$=0.3; $ES$=0.10).

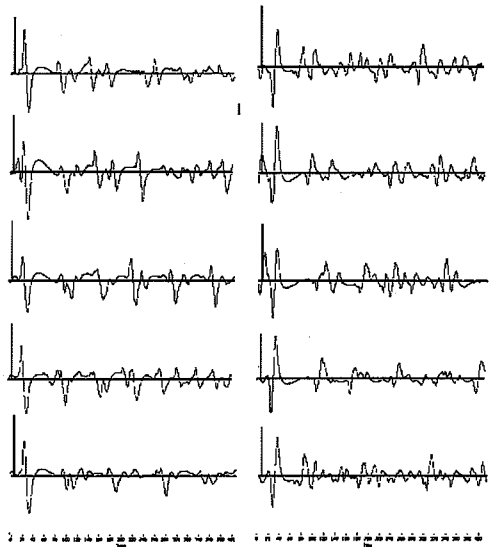


**Figure 3: Example of Five Raw MEP Sweeps (400 ms) from One Participant's Right BB during the TMS Trials Pre (Traces on the Left) and Post (Traces on the Right) Vibration.**

### Corticospinal Excitability

There were no significant differences in mean MEP amplitude at AMT between conditions at baseline (pre V- 0.71 ± 0.25 mV; pre V+ 0.69 ± 0.23 mV; $p$=1.0; $ES$=0.07). Further, there were no significant differences between conditions at baseline for MEP amplitude at 20% above AMT (pre V- 2.1 ± 1.3 mV; pre V+ 2.2 ± 1.4 mV; $p$=0.5; $ES$=0.07). Post testing analyses were performed between conditions (i.e. post V- vs. post V +), with no significant differences for MEP amplitude at AMT (post V- 0.70 ± 0.21 mV; post V + 0.69 ± 0.22 mV; $p$ = 0.65; $ES$=0.04; Figures 4 and 5) and 20% above AMT (post V- 2.2 ± 1.4 mV; post V + 2.0 ± 1.3 mV; $p$ = 0.88; $ES$=0.14; Figures 4 and 5 Figures 4 and 5) being observed.

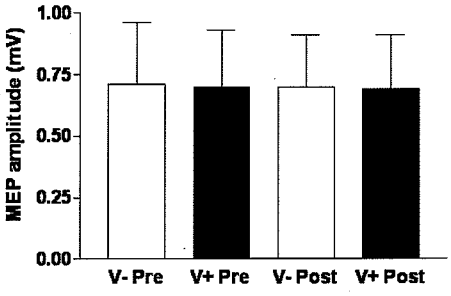



Figure 4: Mean (± SD) MEP Amplitudes ($n$ = 10) Obtained from Right BB Prior to and Immediately Following no WBV (V-, Light Bars) and with WBV (V+, Black Bars) Conditions at AMT. No Significant Differences in MEPs Where Observed between Conditions Pre and Post WBV.

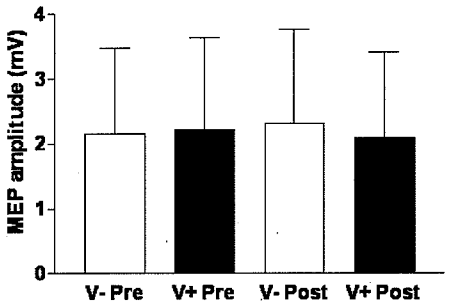



Figure 5: Mean (± SD) MEP Amplitudes ($n$ = 10) Obtained from Right BB Prior to and Immediately Following no WBV (V-, Light Bars) and with WBV (V+, Back Bars) Conditions at 20% above AMT. No Significant Differences in MEPs Where Observed between Conditions Pre and Post WBV.

## Discussion

With good evidence to demonstrate that afferent stimulation augments corticospinal excitability (Ridding et al, 2000; Rosenkranz and Rothwell, 2003), we hypothesised that a bout of low-frequency vibration exercise may also modulate corticospinal excitability through stimulation of the primary endings of muscle spindles, thus increasing muscle activation. To our knowledge, this is the first study to examine the post-vibratory effects of low-frequency vibration applied to the upper limbs on corticospinal excitability. The main findings of the study were when compared to a no vibration (V-) condition that required the same exercise to be performed with the vibration machine activated, acute exposure to low-frequency vibration did not modulate the excitability of the corticospinal pathway or the spinal motoneuron pool innervating BB. While it has been reported that acute exposure to low-frequency vibration may alter neuromuscular excitability (Cardinale and Bosco, 2003), we found no difference in the size of the descending corticospinal volley at AMT and at 20% above AMT immediately following low-frequency vibration, demonstrating that post-synaptic excitability of the motoneuron pool remained unaffected in the post-vibratory period. These findings are in agreement with previous studies that have examined the post-vibratory effect on corticospinal excitability using both high-frequency direct muscle and tendon vibration (Kossev et al, 1999; Munte et al, 1996; Steyvers, Levin, Van Baelen, and Swinnen, 2003) and low-frequency vibration on lower limbs (Mileva et al, 2009).

It has been suggested that a possible mechanism for no change in corticospinal excitability immediately following vibration may be due to changes in spontaneous discharge rate of 1a muscle afferents (Ribot-Ciscar, Rossi-Durand, and Roll; 1998) It has been observed that a decrease in spindle discharge rate occurs immediately following vibration and that this decrease is consistent with the decreases seen in the Hoffman reflex (H-reflex) following vibration (Hultborn, Meunier, Pierrot-Deseilligny and Shindo, 1987). Given the divergent projection of corticospinal axons and their monosynaptic connections onto the spinal motoneuron pool, the present findings demonstrate that vibration had no potentiating effect on the corticospinal pathway and spinal cord excitability.

Within the low-frequency vibration literature, theoretical considerations have suggested that the mechanisms for improved muscle activation (and corticospinal excitability) are due to increased excitatory input from 1a muscle afferents, as a result of the tonic vibration reflex (TVR). The findings

from this study and that of Mileva et al. (2009), demonstrate that the post-vibratory effect of low-frequency vibration are unlikely to be as a result of the TVR. Recent studies have tried to assess the effect of acute low-frequency vibration on motoneuron excitability (H-reflex) demonstrating acute exposure had no significant effect on the motoneuron pool (Hopkins, Fredericks, Guyon, Parker, Gage, Freland et al, 2009; Armstrong, Nestle, Grinnell, Cole, VanGilder, Warren et al, 2008; McBride, Nuzzo, Dayne, Israetel Nieman, and Tripplet, 2010). Since we have observed no difference in the size of the descending corticospinal volley immediately following low-frequency vibration, our findings support the notion that acute low-frequency vibration does not affect the post-synaptic excitability of the motoneuron pool during the post-vibratory period.

The results of this study can also be explained from the exposure time and intensity of the vibration protocol used. The exposure time used may have been too long, as previous studies have reported exposure times of greater than 30 s result in decreased muscle activation, most likely due to reduced 1a muscle afferent input (Shinohara, 2005; Hultborn, Meunier, Pierrot-Deseilligny, and Shindo, 1987; Ekland and Hagbarth, 1966). However, all parameters utilized in this study fall within the ranges routinely used by other acute low-frequency vibration studies (Mileva et al, 2009; Hazell et al, 2007) that have reported increased muscle activation. The recovery period allocated between bouts was also consistent with recent findings using the same low-frequency vibration parameters (30-35 Hz, 4 mm amplitude) as the present study (Da Silva-Grigoletto, Vaamonde, Castillo, Poblador, Garcia-Manso, Lancho, 2009). The number of sets prescribed is consistent with other studies, however, these variables were for the lower limb as currently, there are no studies that have assessed low-frequency vibration on the upper limbs (Nordlund and Thorstensson, 2007). Therefore, even though we have utilised a vibration protocol that has previously elicited positive effects (in the lower limb), it is likely that the vibratory response of upper limb muscles maybe different to the lower limbs and as such may require a different vibration protocol.

In light of this, most of the literature regarding low-frequency vibration exposure has established effects in lower limb muscles, with only one other study examining the upper-limb (whilst standing on the WBV machine; Hazell et al, 2007); therefore, employing targeted individualised vibration parameters (i.e. frequency and amplitude) may be required in order to obtain a more consistent finding. However, the novel aspect of this study was that a sham vibration intervention was used to reduce any placebo effect of vibration, by having participants complete the same exercises under the exact same conditions (i.e. with the vibration machine turned on and set to the exact same parameters). Previous studies have simply had participants to perform the control interventions with the machine turned off, thus not replicating a true experimental control condition.

## Conclusions

In conclusion, acute exposure to low-frequency vibration applied to the upper limb does not potentiate the efficiency of neural transmission along the corticospinal pathway in the post-vibratory period, therefore having little effect on the post-synaptic excitability of the motorneuron pool controlling the BB. Although, corticospinal excitability was not measured during vibration, previous low-frequency and high-frequency vibration studies have provided good evidence that corticospinal excitability is preferentially modulated during vibration. Therefore, the benefits of vibration appear to reside during vibration exposure and not during the post-vibratory period.

## References

1. Armstrong, JW, Nestle, HN, Grinnell, DC, Cole, LD, Van Gilder, EL, Warren, GS, Capizzi, EA. (2008) The acute effect of whole-body vibration on the hoffman reflex. J Strength Cond Res, 22: p. 471–476.

2. Cardinale, M, Bosco, C. (2003)The use of vibration as an exercise intervention. Exerc Sport Sci Rev, 31: 3–7.
3. Chez, C, Krakauer, J. (2000) The organization of movement., in Principles of Neural Science, E.R. Kandel, J.H. Schwartz, T.M. Jessell, Editors, McGraw-Hill: New York, USA. p. 653–673.
4. Cormie, P, Deane, RS, Triplett, NT, McBride, JM. (2006) Acute effects of whole-body vibration on muscle activity, strength, and power. J Strength Cond Res, 20: 257–61.
5. Da Silva-Grigoletto, ME, Vaamonde, DM, Castillo, E, Poblador, MS, Garcia-Manso, JM, Lancho, JL. (2009) Acute and cumulative effects of different times of recovery from whole-body vibration exposure on muscle performance. J Strength Cond Res, 23: 2073–2082.
6. Eklund, G., Hagbarth, KE. (1966) Normal variability of tonic vibration reflexes in man. Exp Neurol, 16: 80–92.
7. Fees, M, Decker, T, Snyder-Mackler, L, Axe, MJ. (1998) Upper extremity weight-training modifications for the injured athlete. A clinical perspective. Am J Sport Med, 26(5): p. 732–742 Hallett, M: Transcranial magnetic stimulation: a primer. Neuron 2007, 55:187–199.
8. Hazell, TJ, Jakobi, JM, Kenno, KA. (2007) The effects of whole-body vibration on upper- and lower-body EMG during static and dynamic contractions. Appl Physiol, Nutr & Metabol, 32: 1156–1163.
9. Hopkins, JT, Fredericks, D, Guyon, PW, Parker, S, Gage, M, Feland, JB, Hunter, I. (2009) Whole Body Vibration Does Not Potentiate the Stretch Reflex. Int J Sports Med, 30: 124–129.
10. Hultborn, H, Meunier, S, Pierrot-Deseilligny, E, Shindo, M. (1987) Changes in presynaptic inhibition of Ia fibres at the onset of voluntary contraction in man. J Physiol, 389: 757–772.
11. Kossev, A, Siggelkow, S, Schubert, M, Wohlfarth, K, Dengler, R. (1999) Muscle vibration: Different effects on transcranial magnetic and electrical stimulation. Muscle Nerve, 22: 946–8.
12. Lewis, GN, Byblow, WD, Carson, R. (2001) Phasic modulation of corticomotor excitability during passive movement of the upper limb: effects of movement frequency and muscle specificity. Brain Res, 900: 282–294.
13. McBride, JM, Nuzzo, JL, Dayne, AM, Israetel, MA, Nieman, DC, Tripplet, TN. (2010) Effect of an acute bout of whole-body vibration exercise on muscle force output and motor neuron excitability. J Strength Cond Res, 24: 184–189.
14. Mileva, KN, Bowtell, JL, Kossev, R. (2009) Effects of low-frequency whole-body vibration on motor-evoked potentials in healthy men. Exp Physiol, 94:103–116.
15. Martin, BJ, Park, HS. (1997) Analysis of the tonic vibration reflex: Influence of vibration variables on motor unit synchronization and fatigue. Eur J Appl Physiol, 75: 504–511.
16. Mileva, KN, Naleem, AA, Biswas, SK, Marwood, S, Bowtell, JL. (2006) Acute effects of a vibration-like stimulus during knee extension exercise. Med Sci Sports Exerc, 38:1317–28.
17. Munte, TF, Jobges, EM, Wieringa, BM, Klein, S, Schubert, M, Johannes, S & Dengler, R. (1996) Human evoked potentials to long duration vibratory stimuli: role of muscle afferents. Neurosci Lett, 216: 163–6.
18. Nordlund, MM, Thorstensson, A. (2007) Strength training effects of whole-body vibration? Scand J Med Sci Sports,17: 12–17.
19. Pearce, AJ, Kidgell, DJ. (2010) Comparison of corticomotor excitability during visuomotor dynamic and static tasks. J Sci Med Sport, 13: 167–171.
20. Ribot-Ciscar, E, Rossi-Durand, C, Roll, J. (1999) Muscle spindle activity following muscle tendon vibration in man. Neurosci Lett, 258: 147–150.
21. Ridding, MC, Brouwer, B, Miles, TS, Pitcher, JB, Thompson, PD. (2000) Changes in muscle responses to stimulation of the motor cortex induced by peripheral nerve stimulation in human subjects. Exp Brain Res, 131: 135–143.
22. Roelants, M, Verschueren, SM, Delecluse, C, Levin, O, Stijnen, V. (2006) Whole-body-vibration-induced increase in leg muscle activity during different squat exercises. J Strength Cond Res, 20: 124–9.
23. Rosenkranz, K, Rothwell, J. (2003) Differential effect of muscle vibration on intracortical inhibitory circuits in humans. J Physiol, 551: 649–60.
24. Rothwell, JC, Rosenkranz, K. (2005) Role of afferent input in motor organization in health and disease. IEEE Engineering Med Biol Magazine, 24: 40–44.
25. Shinohara, M. (2005) Effects of prolonged vibration on motor unit activity and motor performance. Med Sci Sports Exerc, 37: 2120–5.

26. Steyvers, M, Levin, O, Van Baelen, M, Swinnen, SP. (2003) Corticospinal excitability changes following prolonged muscle tendon vibration. Neuroreport, 14: 1901–1905.
27. Stuberg, W.A. and Metcalf, W.K. (1988) Reliability of quantitative muscle testing in healthy children and in children with duchenne muscular dystrophy using a hand-held dynamometer. Phys Ther, 68, 977–82.

**Dawson J. Kidgell**

**Employment,** Centre for Physical Activity and Nutrition Research, Deakin University, Melbourne, Australia, **Degree** Ph.D., **Research interests,** Neuromuscular aspects of exercise physiology.

E-mail: dawson.kidgell@deakin.edu.au

**Alan J. Pearce**

**Employment,** School of Sport and Exercise Science, Victoria University, Melbourne, Australia, **Degree,** Ph.D., **Research interests,** Applied physiology and physical performance of sport. Physiology of movement and motor control.

E-mail: alan.pearce@vu.edu.au