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Changes in tibialis anterior corticospinal properties after acute prolonged muscle vibration

Adrien Farabet¹ · Robin Souron¹ · Guillaume Y. Millet² · Thomas Lapole¹

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Abstract

Purpose Prolonged local vibration is known to impair muscle performance. While involved mechanisms were previously evidenced at the spinal level, changes at the cortical level were also hypothesized. The aims of the present study were to investigate the effects of 30 min of 100-Hz tibialis anterior muscle vibration on force production capacities and to further identify the respective changes in spinal loop properties, descending voluntary drive and corticospinal properties.

Methods Thirteen subjects were tested before and after a vibration condition, and before and after a resting control condition. Maximal voluntary contraction (MVC) in dorsiflexion was measured. Transcranial magnetic stimulation was superimposed during MVCs to assess cortical voluntary activation (VA_{TMS}), motor-evoked potential amplitude (MEP) and cortical silent period length (CSP). MEP and CSP were also measured during 50 and 75 % MVC contractions. Spinal excitability was investigated by mean of H-reflex.

Results There were no vibration effects on MVC ($p = 0.805$), maximal EMG activity ($p = 0.653$), VA_{TMS} ($p = 1$), and CSP ($p = 0.877$). Vibration tended to decrease MEP amplitude ($p = 0.117$). H-reflex amplitude was depressed following vibration ($p = 0.008$).

Conclusions Dorsiflexion maximal force production capacities were unaffected by 30 min of tibialis anterior muscle vibration, despite spinal loop and corticospinal excitabilities being reduced. These findings suggest that acute prolonged vibration has the potential to modulate corticospinal excitability of lower limb muscles without a concomitant functional consequence.

Keywords Vibration · Transcranial magnetic stimulation · Cortical voluntary activation · H-reflex

Abbreviations

CSP	Cortical silent period
CSP ₅₀	CSP recorded during 50 % MVC contraction
CSP _{MVC}	CSP recorded during MVC
ERT	Estimated resting twitch
MEP	Motor-evoked potential
MEP ₅₀	MEP recorded during 50 % MVC contraction
MEP _{MVC}	MEP recorded during MVC
M _{max}	Peak to peak amplitude of the maximal M-wave
MVC	Maximal voluntary contraction
RMS	Root mean square of electromyography
rMT	Resting motor threshold
SIT	Superimposed twitch
SIT _{MVC}	Superimposed twitch during MVC
TA	Tibialis anterior muscle
TMS	Transcranial magnetic stimulation
VA_{TMS}	Cortical voluntary activation

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Introduction

Prolonged local vibration (i.e. applied directly to a muscle or a tendon) is known to induce a short-term decrease in force-generation capacity of the corresponding muscle.

This has been reported following 20–30 min of local vibration of knee extensors (Jackson and Turner 2003; Konishi et al. 2009; Kouzaki et al. 2000), plantar flexors (Herda et al. 2009; Ushiyama et al. 2005) or first dorsal interosseus muscle (Shinohara et al. 2005). Reduced motor performance was suggested to be due to impaired ability of the central nervous system to activate motoneurons.

Changes in the motor drive may come from spinal and/or supraspinal factors (Gandevia 2001; McNeil et al. 2011). At the spinal level, the main mechanism involved in the vibration-induced central drive impairment is thought to be a decreased alpha motoneurons activity through attenuation of Ia afferent efficacy (Bongiovanni et al. 1990), Ia fibers being largely solicited by vibrations (Eklund and Hagbarth 1966). Indeed, most studies that reported a vibration-induced force decrement suggested an altered spinal loop excitability as the main hypothesis (Jackson and Turner 2003; Kouzaki et al. 2000; Konishi et al. 2009; Ushiyama et al. 2005; Herda et al. 2009). Spinal loop excitability is commonly assessed through the Hoffmann reflex (H-reflex) (McNeil et al. 2013; Zehr 2002). Its amplitude is known to depend directly on the motoneuron excitability (Schieppati 1987), but also on the level of presynaptic inhibition exerted on Ia afferents (Misiąszek 2003). Accordingly, soleus H-reflex amplitude was reported to decrease following 30 (Eklund and Thorstensson 2011; Ushiyama et al. 2005) and 60 min (Lapole et al. 2012b) of Achilles tendon vibration. Similar results were recently reported for the vastus medialis muscle following 30 min of infrapatellar tendon vibration (Fry and Folland 2014). From these studies, only the study of Ushiyama et al. (2005) reported decreased H-reflex with concomitant motor performance impairment. Altered spinal excitability due to prolonged local vibration is not the only mechanism that could reduce motor voluntary drive following vibration: a decrease in motor cortex excitability was also suggested to be a potential mechanism (Jackson and Turner 2003; Ushiyama et al. 2005). Corticospinal excitability may be tested through the analysis of motor-evoked potentials (MEPs) recorded in response to stimulation applied over the motor cortex (Mills 1999). Steyvers et al. (2003) reported an attenuation of the motor cortex excitability of the flexor carpi radialis muscle during the first minutes after prolonged vibration. Such an attenuation of the corticospinal excitability may potentially be a factor in vibration-induced motor performance decrement (Ushiyama et al. 2005) but this remains to be tested.

TMS may also be used to evoke superimposed twitches during contractions, in a similar way as the classical interpolated twitch technique (Merton 1954). While the latter method cannot distinguish adaptations at the spinal and

cortical levels, TMS can be used to assess cortical voluntary activation level (VA_{TMS}), i.e. to highlight the presence of suboptimal drive from the motor cortex to the involved muscles during maximal voluntary contraction (MVC) (Goodall et al. 2014). Tibialis anterior muscle (TA) is known to have the strongest corticospinal projections among all leg muscles (Perez et al. 2004; Brouwer and Ashby 1992). Thus, to study the role of motor cortex in the vibration-induced MVC impairment in this muscle is of major interest. Moreover, when applied during a voluntary contraction, TMS may also inform about intracortical inhibition through the assessment of cortical silent period (CSP) (Terao and Ugawa 2002). CSP results from the activation of inhibitory cortical neurons projecting onto the pyramidal cells in the motor cortex (Inghilleri et al. 1993), and is thought to be mainly influenced by GABA (gamma aminobutyric acid) neurotransmission (Werhahn et al. 1999).

Therefore, the aim of the present study was to investigate the effects of prolonged vibration of the TA muscle on force production capacities, and to further identify the respective changes in spinal loop properties (i.e. H-reflex), descending voluntary drive (i.e. VA_{TMS}) and corticospinal properties (MEP and CSP). It was hypothesized that prolonged TA vibration would impair motor performance through reduced spinal loop excitability and decreased cortical voluntary activation.

Methods

Subjects

Thirteen young and healthy subjects (9 males; age: 23 ± 4 years; height: 177 ± 8 cm; body mass: 69 ± 10 kg) participated in this study. Written informed consent was obtained from all subjects prior to their participation and this study conformed to the standards from latest revision of the Declaration of Helsinki and was approved by the local ethics committee. All participants were free of lower-limb injury during the previous 3 months, contraindications to TMS, acute and chronic neurological disorders and trauma. They were instructed to abstain from caffeine for a minimum of 12 h before each session.

Design of the study

Subjects were tested twice in random condition (crossover design) on two separate days with a 1-week interval. Prior to the first testing session, subjects were familiarized to the whole experimental protocol. The subjects were then randomly divided into two groups: one group performed the

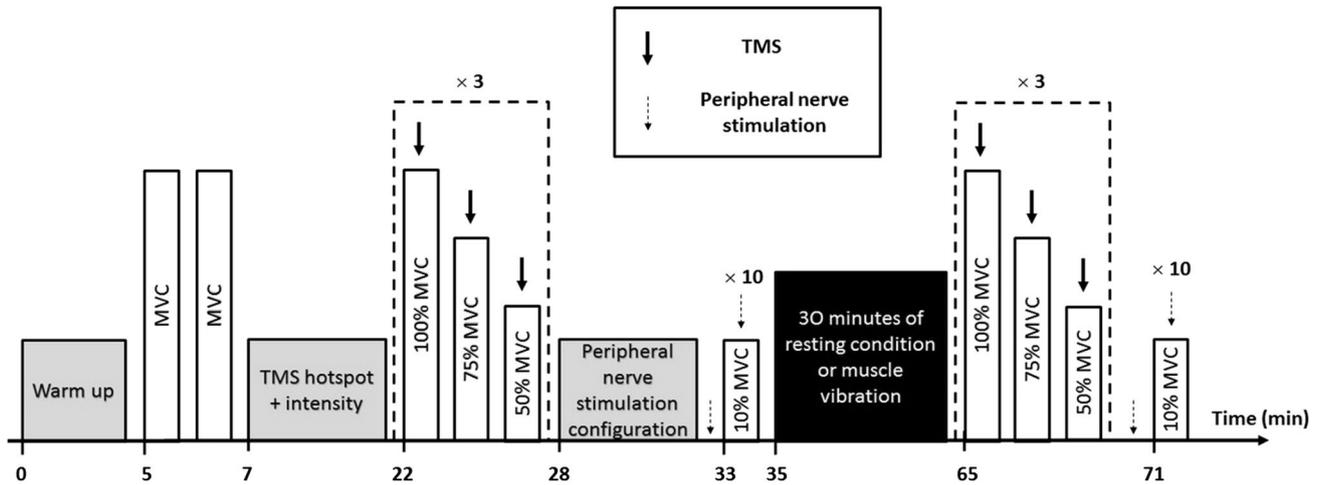


Fig. 1 Illustration of the experimental protocol



Fig. 2 Illustration of the vibratory device strapped on the tibialis anterior muscle belly

first test with the control condition and the other started with the vibration condition. This was inverted for the second session. Both sessions took place at the same time of the day, to avoid circadian variations. A testing session consisted in neuromuscular measurements performed before (PRE) and immediately after (POST) a 30-min resting period (control condition) or 30 min of muscle vibration (vibration condition) (Fig. 1). The application of vibration (1 mm amplitude and 100 Hz frequency) was performed with local vibrator (VB 115, Techno Concept, Mane, France) strapped on the tibialis anterior muscle belly (Fig. 2).

Torque and electromyographic recordings

Dorsiflexion torque was measured during voluntary contractions by a calibrated instrumented pedal (CS1060 300 Nm, FGP Sensors, Les Clayes Sous Bois, France). Subjects were seated upright in a custom-built chair with pelvis, knee and ankle angulations of 90, 120 and 90°, respectively. Electromyographic activity (EMG) of the right TA and soleus was recorded with pairs of self-adhesive surface electrodes (Meditrace 100, Covidien, Mansfield, USA) in bipolar configuration with a 30 mm interelectrode distance and the reference on the internal malleolus. Soleus electrodes were placed 2 cm below the insertion of the gastrocnemii on the Achilles tendon and TA electrodes were placed on the muscle belly according to SENIAM recommendations (Hermens et al. 2000). Low impedance (<5 kΩ) between electrodes was obtained by shaving, gently abrading the skin and then cleaning it with isopropyl alcohol. Signals were analogue-to-digitaly converted at a sampling rate of 2000 Hz by PowerLab system (16/30—ML880/P, ADInstruments, Bella Vista, Australia) and octal bio-amplifier (ML138, ADInstruments; common mode rejection ratio = 85 dB, gain = 500) with bandpass filter (5-500 Hz) and analyzed offline using Labchart 7 software (ADInstruments).

Transcranial magnetic stimulation

Single TMS pulses of 1-ms duration were manually delivered to elicit MEPs and superimposed twitches (SITs) during voluntary isometric dorsiflexion. The motor cortex contralateral to the right TA was stimulated by a magnetic stimulator (Magstim 2002, The Magstim Company Ltd, Whitland, UK) with a 110-mm double-cone coil (maximum output of 1.4 T). Subjects wore a cervical collar during all

TMS measures to stabilize the head and neck. The coil was positioned over the leg area of the motor cortex along the nasal-inion axis to induce a posteroanterior current. After a standardized warm-up, subjects performed two maximal dorsiflexion (MVCs) lasting approximately 3 s, with a 30-s resting period between trials. Optimal coil position was then determined as the site eliciting the largest SIT and TA MEP amplitude with minimal soleus MEP amplitude in response to stimulation at 50 % maximal stimulator output during brief voluntary dorsiflexion at 10 % MVC. This position was marked on a swim cap secured to the scalp. Before the delivery of each stimulus, the coil position was verified with regard to the marks on the swim cap. Stimulus intensity for VA_{TMS} measurement was determined at PRE from an input-output curve determined during brief voluntary dorsiflexion at 20 % MVC. The same intensity was used at POST. Input-output curve was drawn from the average of four consecutive contractions performed at 15-s intervals at each of the following randomly-ordered stimulus intensities: 20, 30, 40, 50, 60, 70 and 80 % of maximal stimulator output. Optimal stimulus intensity was then defined as the lowest stimulus intensity eliciting maximal TA MEP and SIT amplitude (i.e. 65.4 ± 7.8 and 65.4 ± 8.8 % of maximal stimulator output for control and vibration conditions, respectively). If a plateau was not confirmed from these intensities, higher TMS intensities were used (i.e., 90 and 100 % of maximal stimulator output). Such method has been recently reported to be appropriate for determining TMS intensity in the quadriceps (Temesi et al. 2014a). During voluntary contractions, TMS was delivered once the subject had stabilized the force on the target level. Subjects were instructed to re-contraction as strong and as fast as possible immediately after TMS delivery to permit accurate measurement of the CSP.

Neuromuscular measurements

Corticospinal properties

VA_{TMS} during maximal effort was measured by a modified version of the interpolated twitch technique. Measurement consisted of three series of three contractions performed at 100, 75 and 50 % MVC with real-time visual feedback (Fig. 3) (3 to 5 s of contraction, 30 s between contractions and 1 min between sets). In each series, contraction at 100 % MVC was always performed first while 50 and 75 % MVC contractions were randomized. TMS was delivered at each target force to record the evoked SITs. Corticospinal excitability increases substantially during the transition from relaxed to contracted muscle states (Ugawa et al. 1995), thus underestimating TMS evoked twitch in the relaxed muscle. Hence, estimated resting twitch (ERT) was determined by a linear regression of the relation between SIT amplitude evoked when TMS was delivered at 100,

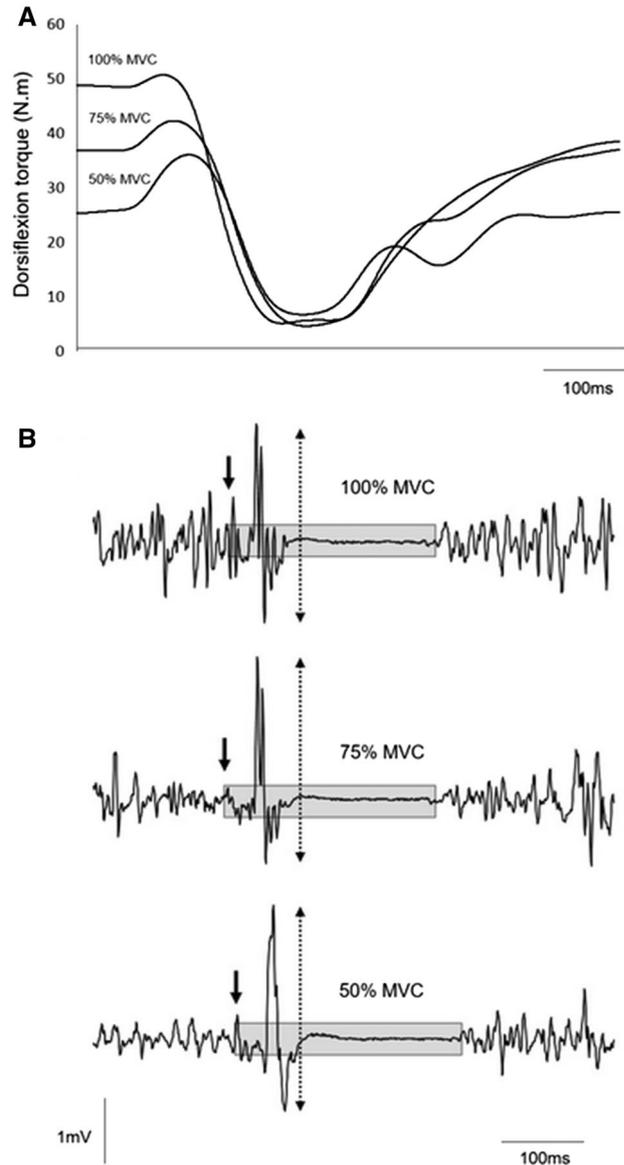


Fig. 3 Example of torque (a) and eletromyographic (b) recordings in response to TMS for a representative subject at 100, 75 and 50 % MVC. *Bold arrows* indicate the time point of TMS. *Dashed arrows* and *grey rectangles* show how MEP amplitudes and CSP duration were calculated, respectively

75 and 50 % MVC and voluntary force (Todd et al. 2003). This relation was extrapolated and the y-intercept was interpreted as the ERT amplitude. VA_{TMS} during MVCs was calculated as follows:

$$VA_{TMS} = (1 - SIT_{MVC}/ERT) \times 100$$

where SIT_{MVC} is the superimposed twitch evoked during MVC. Mean R^2 of the linear regression was 0.93 ± 0.10 and 0.90 ± 0.10 before and after control condition, respectively; and 0.93 ± 0.08 and 0.97 ± 0.05 before and after

vibration, respectively. MVC was determined as the highest torque value achieved during maximal effort. TA EMG root mean square (RMS_{MVC}) was calculated as the mean over a 500-ms period after maximal force had reached a plateau. EMG–RMS was then normalized to the maximal peak-to-peak amplitude of the M-wave (M_{max} ; see below) obtained at each time point. MEPs and CSPs elicited by TMS during contractions were also calculated and averaged over the three sets. Peak-to-peak amplitude of MEPs were measured and normalized to M_{max} (MEP_{MVC} , MEP_{75} and MEP_{50} , respectively) obtained at each time point. The duration of the CSPs were determined visually and defined as the duration from the TMS stimulus to the return of continuous voluntary EMG (CSP_{MVC} , CSP_{75} and CSP_{50} , respectively) (Sidhu et al. 2009).

Spinal excitability

Single electrical stimuli of 0.5-ms duration were delivered to the right peroneal nerve via constant-current stimulator (DS7A, Digitimer, Welwyn Garden City, Hertfordshire, UK) and bipolar bar stimulating electrode with 30 mm anode–cathode spacing (Bipolar Felt Pad Stimulating Electrode Part number E.SB020/4 mm, Digitimer) placed next to the fibular head. The optimal site of stimulation was determined as the location eliciting the greatest TA M-wave for a given stimulus intensity. This site was marked with semi-permanent ink. Single electrical stimuli were delivered incrementally (5-mA step) at rest until maximal TA M-wave plateaued (M_{max}). The amplitude of the H-reflex was then recorded during low contraction intensity of 10 % MVC to ensure easily identifiable TA H-reflex with background EMG (Tallent et al. 2012). In H-reflex recordings, it is essential to ensure that effective stimulus intensity remains constant (Aagaard et al. 2002; Lapole et al. 2013). Thus, H-reflex measurements were performed

with a stimulation intensity producing concomitant TA M-waves equal to 15–20 % of M_{max} (Tallent et al. 2012). Once the appropriate target stimulus intensity was found, 10 H-reflexes were recorded, measured peak-to-peak, and expressed relative to M_{max} . Ten seconds intervals separated the stimulations. Only recordings that met the criteria of a concomitant M-wave 15–20 % of TA M_{max} were averaged (i.e., 8.2 ± 1.1 and 7.8 ± 0.9 recordings kept before and after control condition, respectively; 7.6 ± 1.4 and 8.1 ± 1 recordings before and after vibration, respectively).

Statistics

Statistical analyses were performed using Statistica software (StatSoft Inc., Tulsa, Oklahoma, USA). All variables were normally distributed (Kolmogorov–Smirnov normality test). For ANOVA analyses, homogeneity of variance was verified by Levene’s test. A two-way (condition \times time) repeated measures ANOVA was performed on M_{max} , MVC, RMS_{MVC}/M_{max} , SIT_{MVC} and VA_{TMS} . A three-way (conditions \times time \times force levels) repeated measures ANOVA was performed on MEP and CSP. When the ANOVA identified significant interaction effects (condition \times time), post hoc Student–Newman–Keuls testing was performed. Data are presented as mean \pm SD. Statistical significance was set at $p < 0.05$.

Results

P values from all ANOVA analyses are presented in Table 1. Vibration did not influence MVC or TA maximal EMG activity. Similarly, no interaction effect was reported for SIT_{MVC} , VA_{TMS} and CSP. Figure 4 presents the results for MEP amplitude during 50, 75 and 100 % MVC contractions. Although there was a significant condition \times time

Table 1 p values from all ANOVA analyses

	Condition	Time	Condition \times time	Force level	Condition \times force level	Time \times force level	Condition \times time \times force level
M_{max}	0.692	0.168	0.255				
H-reflex	0.133	0.237	0.006				
MVC	0.770	0.344	0.805				
RMS_{MVC}/M_{max}	0.950	0.894	0.653				
SIT_{MVC}	0.225	0.810	0.967				
VA_{TMS}	0.094	0.372	1.000				
MEP	0.005	0.041	0.040	0.017	0.721	0.417	0.891
CSP	0.427	0.122	0.877	0.905	0.916	0.187	0.897

M_{max} the amplitude of the maximal M-wave evoked in the relaxed muscle, *H-reflex* amplitude of the H-reflex expressed as a percentage of maximal M-wave amplitude, *MVC* Maximal voluntary contraction in dorsiflexion, RMS_{MVC}/M_{max} Tibialis anterior maximal EMG activity expressed as a percentage of maximal M-wave amplitude, SIT_{MVC} superimposed twitch during MVC, VA_{TMS} Cortical voluntary activation, *MEP* Motor-evoked potential, *CSP* Cortical silent period

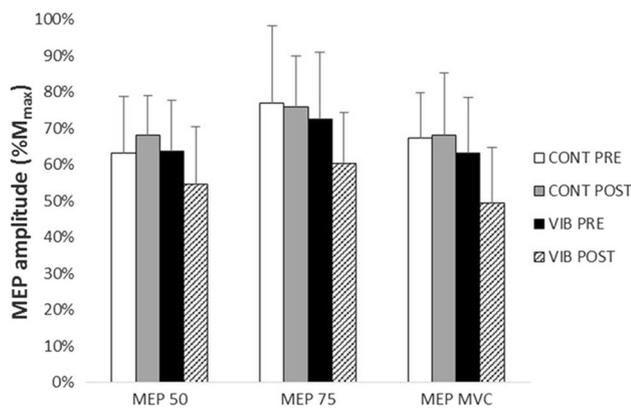


Fig. 4 MEP amplitude recorded during dorsiflexion at 50, 75 and 100 % MVC (MEP₅₀, MEP₇₅ and MEP_{MVC}, respectively) for PRE/POST vibration and control conditions. MEP amplitude is expressed as a percentage of M_{\max} , the amplitude of the maximal M-wave evoked in the relaxed muscle

interaction for MEP, Post-hoc testing only demonstrated a trend for decreased MEP amplitude after vibration ($p = 0.117$; -13.7 ± 20.3 , -14.8 ± 22.0 , and -19.7 ± 26.1 % for MEP₅₀, MEP₇₅ and MEP_{MVC}, respectively). There was a significant condition \times time effect for H-reflex with decreased H-reflex amplitude following vibration (-21.0 ± 23.0 %; $p = 0.008$) while no changes were apparent in the control condition ($p = 0.387$) (Fig. 5). M_{\max} remained stable over time and conditions (Table 2).

Discussion

This is the first study investigating the effects of vibration on maximal force production capacities and its effects at both the spinal and cortical levels. Although there was significant TA H-reflex and a trend toward MEP amplitude decrease after 30 min of 100-Hz vibration applied over the TA muscle, there were no effects on motor performance as indicated by unchanged MVC and VA_{TMS} .

The vibration exposure performed in the present study was effective in altering Ia afferent efficacy, as suggested by the 21 ± 23 % decrease in H-reflex amplitude. This is in agreement with the previously reported decreased soleus H-reflex amplitude following 30 (Eklom and Thorstensson 2011; Ushiyama et al. 2005) and 60 min (Lapole et al. 2012b) of Achilles tendon vibration, as it is with decreased vastus medialis H-reflex following 30 min of infrapatellar tendon vibration (Fry and Folland 2014). H-reflex amplitude depends on both motoneuron excitability (Schieppati 1987) and the level of presynaptic inhibition exerted on Ia afferents (Misiąszek 2003). Since previous studies reported unchanged motoneuron excitability following prolonged vibration, as indicated by unchanged F-waves (Lapole

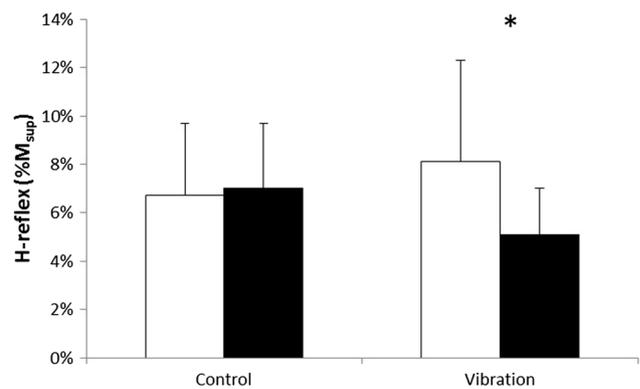


Fig. 5 Mean amplitude of the H-reflex PRE (open bars) and POST (filled bars) in vibration and control conditions. H-reflex amplitude is expressed as a percentage of M_{\max} , the amplitude of the maximal M-wave evoked in the relaxed muscle. Significant difference between PRE and POST values: $*p < 0.05$

et al. 2012b; Christova et al. 2011), changes in the level of presynaptic inhibition exerted on Ia afferents may more likely have resulted in the attenuation of Ia afferent efficacy (Misiąszek 2003). This attenuation has been previously considered to result from several mechanisms including presynaptic inhibition of Ia afferents (Hultborn et al. 1987), an increased firing threshold of Ia afferents (Hayward et al. 1986), and transmitter depletion at the Ia afferents terminals (Curtis and Eccles 1960). This latter mechanism has been suggested to mainly impair force production capacity (Bongiovanni et al. 1990) but not H-reflex although this has not been evidenced (Fry and Folland 2014). Reduced excitability of the Ia afferents has been recently rejected to contribute to the observed vibration-induced vastus medialis H-reflex decrease, as indicated by the constancy of the current at which maximal H-reflex was elicited (Fry and Folland 2014). As a result, and although transmitter depletion cannot be totally ruled out, it is likely that increased presynaptic inhibition of Ia afferents largely contributed to the post-vibration decrease in H-reflex amplitude (Lapole et al. 2012a), in response to the high activation of these Ia afferents by the vibratory process (Roll et al. 1989; Burke et al. 1976). In other words, H-reflex depression following prolonged vibration may essentially result from similar mechanisms as during the vibration paradox, i.e. with presynaptic inhibitory interneurons under supra-spinal control inhibiting Ia afferents (Gillies et al. 1969; Delwaide 1973).

Despite decreased spinal loop excitability after prolonged TA vibration, the present study failed to demonstrate any motor performance impairment (i.e., unchanged MVC, VA_{TMS} and maximal EMG activity). This contrasts with several studies that reported force production decrement and altered EMG activity following 20–30 min of local vibration of knee extensors (Jackson and Turner 2003; Konishi et al. 2009; Kouzaki et al. 2000), plantar

Table 2 Neuromuscular measurements PRE and POST vibration and control conditions

	Control		Vibration	
	Pre	Post	Pre	Post
M_{\max} (mV)	4.8 ± 1.4	4.7 ± 1.3	5.1 ± 1.2	4.8 ± 1.0
MVC (Nm)	54.0 ± 13.1	54.4 ± 13.3	53.4 ± 12.7	54.3 ± 13.0
RMS_{MVC}/M_{\max} (%)	10.7 ± 3.4	10.5 ± 2.1	10.3 ± 3.0	10.7 ± 3.6
SIT_{MVC} (Nm)	0.94 ± 1.05	0.92 ± 1.10	0.66 ± 0.74	0.62 ± 0.49
VA_{TMS} (%)	95.4 ± 5.6	95.9 ± 5.3	96.9 ± 3.8	97.5 ± 2.9
CSP_{50} (ms)	254.7 ± 52.4	260.9 ± 67.5	251.6 ± 49.6	252.8 ± 58.6
CSP_{75} (ms)	261.4 ± 64.5	264.5 ± 60.3	259.6 ± 54.5	264.6 ± 63.0
CSP_{MVC} (ms)	249.1 ± 52.5	267.3 ± 54.0	225.7 ± 63.5	233.0 ± 70.5

M_{\max} the amplitude of the maximal M-wave evoked in the relaxed muscle, MVC Maximal voluntary contraction in dorsiflexion, RMS_{MVC}/M_{\max} Tibialis anterior maximal EMG activity expressed as a percentage of maximal M-wave amplitude, SIT_{MVC} superimposed twitch during MVC, VA_{TMS} Cortical voluntary activation, CSP_{50} Cortical silent period recorded during 50 % MVC dorsiflexion, CSP_{MVC} Cortical silent period recorded during MVC

flexor muscles (Herda et al. 2009; Ushiyama et al. 2005) or first dorsal interosseus muscle (Shinohara et al. 2005). However, our results are in agreement with other studies on plantar flexor (Eklblom and Thorstensson 2011) and knee extensor muscles (Fry and Folland 2014). The causes of discrepancies between studies remain unclear. The lack of observed MVC impairment in the present study despite decreased spinal excitability may be explained by the fact that there is no evidence that the adaptations observed during a low intensity contraction reflect the functional adaptations during MVC (Taylor and Gandevia 2008). Nevertheless, it has been previously reported that the soleus muscle maximal H-reflex was similar at rest and during MVC (Pensini and Martin 2004). H-reflex recording may also not reflect the efficacy of the entire γ -loop (Fry and Folland 2014), stretch reflexes being paradoxically reported to increase after vibration despite decreased H-reflex (Van Boxtel 1986). However, in the study of Van Boxtel (1986), the vibration exposure only lasted 2 min and different results may be expected following prolonged vibration (i.e. 20-30 min). Hence, unchanged TA force production capacities despite reduced spinal loop excitability remain to be further investigated.

The main originality of the present study was to investigate the effects of prolonged vibration on corticospinal properties by using transcranial magnetic stimulation in TA muscle. Indeed, since decreased MEP amplitude has been reported immediately after vibration exposure (Steyvers et al. 2003), vibration-induced altered voluntary drive was suggested as a potential mechanism explaining MVC impairment after vibration (Jackson and Turner 2003; Ushiyama et al. 2005). Consistent with MVC and TA-EMG activity results, VA_{TMS} remained unaffected by prolonged vibration. Hence, the hypothesis of impaired voluntary drive from the motor cortex following vibration exposure

as it has been reported in fatigue conditions (Temesi et al. 2014b; Gruet et al. 2013) has not been validated. Despite the lack of VA_{TMS} change, MEP amplitude, a marker of corticospinal excitability (Mills 1999), tended to decrease after vibration. This would be in agreement with findings of Steyvers et al. (2003) who reported an immediate attenuation of resting flexor carpi radialis muscle MEP amplitude following 30 min of tendon vibration. While a reduced synchrony of motor units action potentials evoked by TMS could potentially explain the decreased MEP amplitude, this hypothesis could be rejected since MEP duration did not change (data not shown). Hence, decreased MEP amplitude may be explained here by a lower number of activated motor units by TMS. Since MEP amplitude depends on the synaptic relays of the corticospinal projections at both the cortical and spinal levels, spinal mechanisms (i.e. reduced motoneuronal excitability) can contribute to the vibration-induced MEP depression (Devanne et al. 1997), which is consistent with the reported decreased H-reflex. In the future, the use of cervicomedullary stimulation (McNeil et al. 2013) in conjunction with TMS would enable the determination of differential effects of vibration on both cortical and motoneuronal excitability. The functional significance of decreased corticospinal excitability remains unclear since motor performance was unaffected.

While MEP amplitude tended to be altered by vibration, no effects were found in CSP. To our knowledge, only one study investigated vibration effect on CSP. Binder et al. (2009) reported during extensor carpi radialis muscle vibration an increased CSP in the antagonist flexor carpi radialis, suggesting enhanced intracortical inhibition in the non-vibrated antagonist muscle, a parameter which was not measured in our study. Still, our results suggest that acute prolonged muscle vibration did not alter GABA neurotransmission (Werhahn et al. 1999) of inhibitory cortical

neurons projecting onto the pyramidal cells in the motor cortex (Inghilleri et al. 1993).

In conclusion, while TA spinal loop and corticospinal excitabilities were reduced by 30 min of muscle vibration, force production capacities and maximal voluntary activation were unaffected. Although the functional link between the recorded parameters and motor performance remain to be elucidated, these findings suggest that acute prolonged vibration has the potential to modulate corticospinal excitability of lower limb muscles.

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