

Research article

## Repetitive peripheral magnetic stimulation (15 Hz RPMS) of the human soleus muscle did not affect spinal excitability

Martin Behrens<sup>1</sup>✉, Anett Mau-Möller<sup>1</sup>, Volker Zschorlich<sup>2</sup> and Sven Bruhn<sup>1</sup>

<sup>1</sup>Department of Exercise Science and <sup>2</sup>Department of Movement Science, University of Rostock, Rostock, Germany

### Abstract

The electric field induced by repetitive peripheral magnetic stimulation (RPMS) is able to activate muscles artificially due to the stimulation of deep intramuscular motor axons. RPMS applied to the muscle induces proprioceptive input to the central nervous system in different ways. Firstly, the indirect activation of mechanoreceptors and secondly, direct activation of afferent nerve fibers. The purpose of the study was to examine the effects of RPMS applied to the soleus. Thirteen male subjects received RPMS once and were investigated before and after the treatment regarding the parameters maximal M wave ( $M_{\max}$ ), maximal H-reflex ( $H_{\max}$ ),  $H_{\max}/M_{\max}$ -ratio,  $H_{\max}$  and  $M_{\max}$  onset latencies and plantar flexor peak twitch torque associated with  $H_{\max}$  ( $PT_H$ ). Eleven male subjects served as controls. No significant changes were observed for  $H_{\max}$  and  $PT_H$  of the treatment group but the  $H_{\max}/M_{\max}$ -ratio increased significantly ( $p = 0.015$ ) on account of a significantly decreased  $M_{\max}$  ( $p = 0.027$ ).  $H_{\max}$  onset latencies were increased for the treatment group ( $p = 0.003$ ) as well as for the control group ( $p = 0.011$ ) while  $M_{\max}$  onset latencies did not change. It is concluded that the RPMS protocol did not affect spinal excitability but acted on the muscle fibres which are part of fast twitch units and mainly responsible for the generation of the maximal M wave. RPMS probably modified the integrity of neuromuscular propagation.

**Key words:** Electromyography, repetitive peripheral magnetic stimulation, H-reflex, M wave, soleus, twitch torque.

### Introduction

During the last decades, increasing attention has been drawn to electromyostimulation (EMS) for rehabilitation in patients (Enoka, 1988; Morrissey, 1988) and strength training in healthy subjects (Dudley and Stevenson, 2003; Maffiuletti et al., 2002). It has been shown that the application of EMS results in maximal voluntary strength gains (Maffiuletti et al., 2002). Various studies have proposed that these strength gains can be attributed to neural adaptations (Colson et al., 2000; Maffiuletti et al., 2000a). When a muscle is artificially activated, by means of EMS, the stimulus evokes action potentials in different structures, for example in intramuscular nerve branches and cutaneous receptors. Thus force is generated directly by activation of motor axons and indirectly by "reflex" recruitment of spinal motoneurons (Collins et al., 2001). Another possibility to induce muscle activation artificially is peripheral magnetic stimulation (Lotz et al., 1989). The cellular mechanism of stimulation is the same for both techniques. In both cases, charge flows into an excitable cell membrane causing a change in transmembrane potential. If an adequate stimulus is applied, it can cause the

depolarization of the membrane and the initiation of an action potential (Barker, 1999).

Peripheral magnetic stimulation stimulates intramuscular motor axons which evoke muscle contraction (Machetanz et al., 1994) and, in contrast to EMS, peripheral magnetic stimulation is characterized by a great depth of penetration, the nonexistence of current flowing through the skin and the absence of local high current densities (Barker, 1999). This avoids the activation of cutaneous receptors like nociceptors and mechanoreceptors (Struppler et al., 2003). Repetitive peripheral magnetic stimulation (RPMS) applied to the muscle induces proprioceptive inflow to the central nervous system (CNS) in two different ways: Firstly, the indirect activation of mechanoreceptors due to stimulation induced rhythmic contraction and relaxation as well as vibration of the muscle. This includes the depolarization of fibre groups Ia, Ib and II. Secondly, the direct activation of sensorimotor nerve fibers with orthodromic and antidromic conduction (Struppler et al., 2004). It has been shown that low-intensity magnetic stimulation of gastrocnemius and soleus is able to activate terminal nerve afferents directly and is therefore capable to induce cerebral potentials (Zhu et al., 1996). Furthermore, it has been demonstrated that RPMS is a potential treatment option for spasticity (Krause and Straube, 2005; Marz-Loose and Siemes, 2009) and that it is able to activate modulatory as well as plastic processes in the CNS (Struppler et al., 2003). Therefore, the purpose of the present study was to examine the effects of RPMS of the m. soleus (SOL) on the amplitude and latency of its maximal H-reflex ( $H_{\max}$ ,  $H_{\max}$  onset latency) and maximal M response ( $M_{\max}$ ,  $M_{\max}$  onset latency) evoked by electrical stimulation of the posterior tibial nerve. Furthermore we investigated the potential RPMS-induced change of plantar flexor peak twitch torque associated with  $H_{\max}$  ( $PT_H$ ).

### Methods

#### Subjects

Twenty-four healthy male subjects with no history of neurological disorders or injuries participated in this double blind study. The subjects were randomly assigned to a treatment group (TG:  $n = 13$ , age  $24.1 \pm 2.0$  years, weight  $77.8 \pm 7.3$  kg, height  $1.81 \pm 0.07$  m, training status  $9.5 \pm 4.2$  h/week) and a control group (CG:  $n = 11$ , age  $23.0 \pm 1.6$  years, weight  $79.6 \pm 11.7$  kg, height  $1.82 \pm 0.08$  m, training status  $8.9 \pm 4.5$  h/week). All test persons signed informed consent prior to investigation. The study was conducted according to the declaration of Helsinki and

approved by the university ethics committee.

### Study design

All subjects were examined before and after RPMS. The dependent variables were peak-to-peak electromyographic amplitude of  $M_{\max}$  (mV) and  $H_{\max}$  (mV) for SOL, the  $H_{\max}/M_{\max}$ -ratio,  $H_{\max}$  onset latency (ms),  $M_{\max}$  onset latency (ms) as well as peak twitch torque associated with  $H_{\max}$  ( $PT_H$  in N·m).

The subjects were placed on the CYBEX NORM Testing & Rehabilitation System (Computer Sports Medicine<sup>®</sup>, Inc., Stoughton, MA) in a standardized prone position. The subjects were asked to relax their muscles and not to voluntarily contract during testing and stimulation. Their right foot was fixed to the device in order to assess peak twitch torque associated with the maximal H-reflex, produced by plantar flexion as a result of electrical stimulation of the posterior tibial nerve. The knee and ankle joint angles of 180° and 78° were consistently maintained during the experiment. The subjects remained in the same standardized prone position and stayed inactive during the whole experiment. In order to avoid the influence of acoustic signals on amplitude of the H-reflex (Palmieri et al., 2004) the test persons wore ear protection (Bilsom Thunder T3, signal-to-noise ratio = 36 dB).

### Magnetic stimulation

RPMS was delivered to SOL of the right leg using a MagProR30 stimulator (Medtronic, Denmark) and a MC-125 circular coil. In order to avoid overheating the circular coil was cooled in a water bath previous to muscle stimulation. RPMS took place once. The stimulation protocol included the application of symmetric biphasic pulses with an impulse duration of 290  $\mu$ s, a frequency of 15 Hz, 100 stimuli per train (train duration 6.6 s), 20 trains, an inter train interval of 2 s and 40 percent stimulator output (60 A/ $\mu$ s magnetic field gradient). A total of 2000 stimuli were applied to SOL which lasted 172 s. The stimulation was applied in a standardized manner to the posterior, lateral and medial part of SOL of TG to reach a deep penetration of the muscle. In this process the different parts of SOL were stimulated successively. This means that the posterior part of SOL was stimulated first, afterwards the lateral part, followed by the medial part. The 2 s inter train intervals were used to change the position of the magnetic coil. The stimulation site was changed till the 20 trains were completed. At the end the posterior, lateral and medial part of SOL received 7 trains, 7 trains and 6 trains, respectively. The stimulation evoked unfused tetanic contractions. CG received a placebo RPMS with 0 percent stimulator output.

### H-reflex recording

The percutaneous electrical stimulus was a square-wave pulse of 500  $\mu$ s duration delivered by a Digitimer<sup>®</sup> stimulator (DS7A, Hertfordshire, UK). Due to the subject position, which made unipolar stimulation not feasible, H-reflex was evoked using a bipolar setup (Pierrot-Deseilligny and Mazevet, 2000). SOL H-reflex was elicited with the cathode placed over the posterior tibial nerve proximal to the anode (both Ambu<sup>®</sup> Blue Sensor N surface electrodes, 2 cm diameter) in the popliteal fossa to

avoid anodal block (Fischer, 1992). In order to ensure constant inter stimulus intervals of 10 s (i.e. 0.1 Hz) a Digitimer<sup>®</sup> train/delay generator (DG2A, Hertfordshire, UK) was used.

The testing procedure included quasi-random stimulation with different current intensities (5-60 mA), resulting in a recruitment curve, until identification of peak-to-peak  $H_{\max}$  and  $M_{\max}$ . Afterwards  $H_{\max}$  was elicited ten times and then averaged in order to achieve reliable results.  $M_{\max}$  was elicited using a supramaximal stimulus (~110%). Testing was performed immediately before and 2 min after RPMS. The time between the treatment and the post test was used to refit the cables required for H-reflex stimulation and recording. Onset latencies of  $H_{\max}$  and  $M_{\max}$  were measured as the time interval between the stimulus artifact and the first deflection of the H or the M wave, respectively.

### Torques and EMG recordings

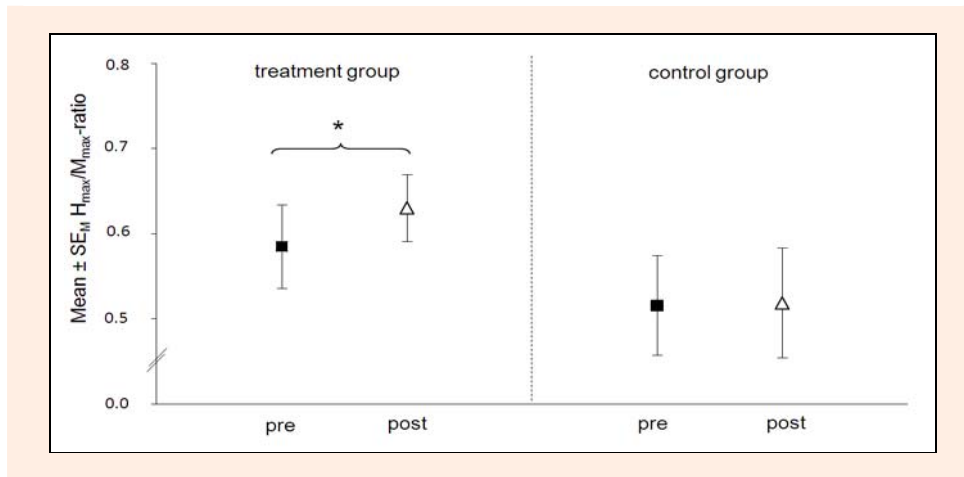
Torques induced by the ten stimulations of the posterior tibial nerve to elicit  $H_{\max}$  were recorded with a sampling frequency of 100 Hz. The generated peak torques were averaged offline. The bipolar EMG recording used Ambu<sup>®</sup> Blue Sensor N surface electrodes (2 cm diameter) which were firmly attached to the abraded and cleaned skin over SOL of the right leg. The electrodes were applied with a center-to-center distance of 2 cm, lateral to the calcaneal tendon, ~ 17 cm above the calcaneus and below the muscle fibres of the gastrocnemius. The recording electrodes were in line with the presumed direction of the underlying muscle fibres. The resistance between electrodes was kept below 5 k $\Omega$  to guarantee high quality EMG recordings. Signals were amplified (amplification factor 1500) with a band-width frequency ranging from 10 Hz to 1.3 kHz and simultaneously digitized online with a sampling frequency of 3 kHz.

### Statistical analyses

Differences between the values before and after RPMS were tested for significance by analysis of variance (ANOVA) for repeated measurements with groups (2) as factor. Differences in means between groups were tested with two-sided student's unpaired t-tests. In each case the level of significance was established at  $p \leq 0.05$ . SPSS 15.0 (SPSS Inc., Chicago, IL, USA) was used for statistical analysis. Furthermore, effect size ( $\epsilon$ , Cohen's d) was calculated. Data are presented as group mean values  $\pm$  standard error of the mean.

### Results

At pre-test, no significant difference was observed between the treatment and control group in terms of their variables. After RPMS  $H_{\max}/M_{\max}$  was significantly enhanced in the treatment group (TG:  $0.585 \pm 0.176$  vs.  $0.630 \pm 0.142$ ,  $p = 0.015$ ,  $\epsilon = 0.61$ ) whereas  $H_{\max}/M_{\max}$  of the control group remained unmodified (CG:  $0.516 \pm 0.195$  vs.  $0.519 \pm 0.214$ ,  $p = 0.843$ ) (Figure 1.). The increment of  $H_{\max}/M_{\max}$  of TG after RPMS was not generated by enhancement of the amplitude of  $H_{\max}$  ( $3.68 \pm 1.91$  mV vs.  $3.64 \pm 1.68$  mV,  $p = 0.758$ ,  $\epsilon = 0.09$ ) but on account of a significantly decreased maximal M wave



**Figure 1.** Group mean  $\pm$  standard error of the mean from pre- and post-measurement of all subjects of the treatment (TG) and control group (CG) are displayed. Following RPMS, the  $H_{\max}/M_{\max}$ -ratios of TG were significantly increased.

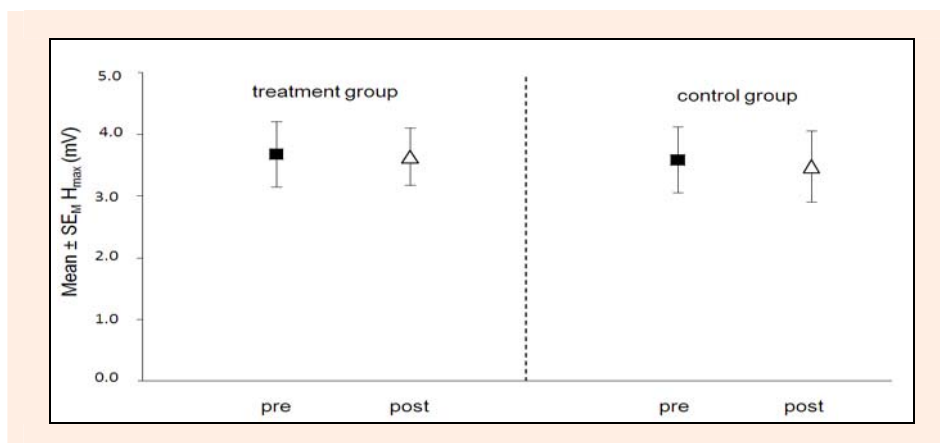
( $6.11 \pm 2.10$  mV vs.  $5.64 \pm 1.95$  mV,  $p = 0.027$ ,  $\varepsilon = 0.49$ ) (Figure 2. and 3.). The stable value for  $H_{\max}$  and the declined value for  $M_{\max}$  of TG are displayed exemplarily in Figure 4., where H-reflex recruitment curves of one subject before and after RPMS are displayed. For each subject, the amplitude of the small M wave preceding the  $H_{\max}$  response ( $M_{H\max}$ ) was comparable for both pre- and post-test (Table 1.).

CG revealed a slight decrease in the crude amplitude of  $H_{\max}$  ( $3.59 \pm 1.77$  mV vs.  $3.48 \pm 1.91$  mV,  $p = 0.505$ ), though not significant. Maximal M wave of CG showed no significant difference before and after placebo RPMS ( $6.78 \pm 1.53$  mV vs.  $6.49 \pm 1.51$  mV,  $p = 0.061$ ) but was slightly diminished as well (Figure 2. and 3.). Due to the simultaneous insignificant decline of the peak-to-peak amplitude of  $H_{\max}$  and  $M_{\max}$  the ratio of CG remained stable ( $0.516 \pm 0.195$  vs.  $0.519 \pm 0.214$ ,  $p = 0.843$ ) (Figure 1.). The onset latency of  $H_{\max}$  was significantly increased after RPMS for both groups, while  $M_{\max}$  onset latencies did not change significantly (Table 1.). Peak twitch torque values associated with  $H_{\max}$  were not significantly different before and after RPMS for the treatment group and the control group (Table 1.).

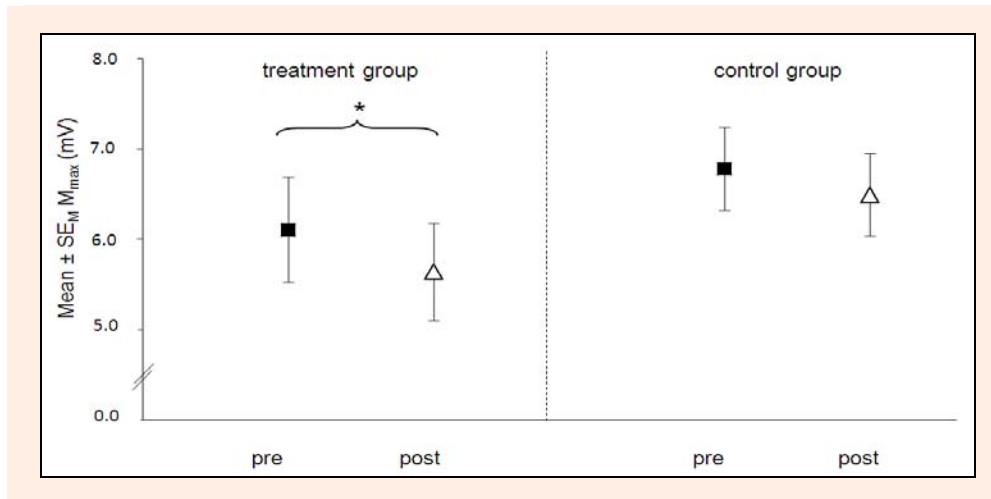
## Discussion

The 15 Hz RPMS protocol used in this study had no significant effect on the peak-to-peak amplitude of  $H_{\max}$  and the corresponding peak twitch torque. Alterations of H-reflex amplitude are normally ascribed to modifications in alpha motoneuron excitability and/or changes in pre-synaptic inhibition (PSI) of primary muscle spindle afferents (Zehr, 2002). However, we should take into account that H-reflexes evoked during actual voluntary contractions (Aagaard et al., 2002) and/or during performance (Voigt et al., 1998) probably represent a more functional assessment of treatment-induced adaptations at the spinal cord level. Nevertheless, these aspects were not investigated with the present methodology as H-reflex measurement was performed while muscles were relaxed.

The results indicate that 15 Hz RPMS did not have the ability to change the H-reflex amplitude of SOL significantly. However,  $M_{\max}$  of TG was significantly reduced after the intervention. The slight decline of  $H_{\max}$  and the significant reduction of  $M_{\max}$  led to a significant increment of the  $H_{\max}/M_{\max}$ -ratio. Similar results were produced by EMS treatment which was applied once as well and led to a significant decline of  $M_{\max}$ , while the  $H_{\max}/M_{\max}$ -ratio varied inconsistently (Tanino et al., 2003). The phenomenon of simultaneous decreasing maximal H-reflex and M wave amplitudes to the same



**Figure 2.** Group mean  $\pm$  standard error of the mean of peak-to-peak SOL H-reflex from pre- and post-measurement of all subjects of the treatment (TG) and control group (CG) are displayed. Following RPMS  $H_{\max}$  of TG revealed no significant difference.



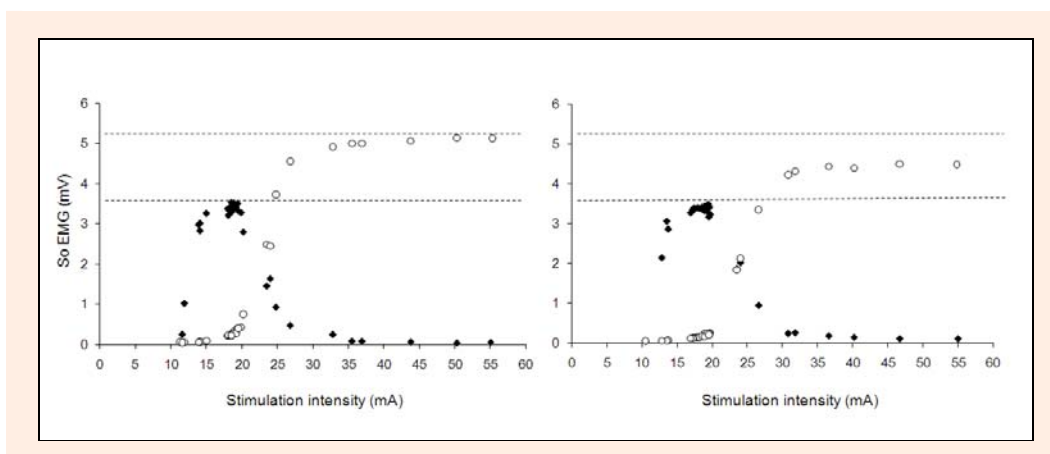
**Figure 3.** Group mean  $\pm$  standard error of the mean of peak-to-peak SOL  $M_{max}$  from pre- and post-measurement of all subjects of the treatment (TG) and control group (CG) are displayed. Following RPMS maximal M wave of TG was significantly decreased.

extent typically occurs during long lasting H-reflex experiments (Crone et al., 1999) and could be seen as an explanation for the results of CG but not for TG.

The decline of  $M_{max}$  can originate from various different sources and their associated physiology. The explanations include: (I) altered position of stimulation and recording electrodes, (II) temperature, (III) duration of the experiment and (IV) alterations in the peripheral structures below the point of stimulation (Enoka, 2008).

First, changes in the amplitude of  $M_{max}$  can be generated when the stimulating electrode shifted from its original position or the recording electrodes have moved relative to the muscle (Palmieri et al., 2004). However, these sources of error can be excluded because stimulation and recording electrodes remained in a constant position and were fixed to the skin. The electrode positions were not altered during entire testing and stimulation procedure. Furthermore, the subjects remained in the same standardized prone position and were inactive during the experiment. Therefore, the amplitude of the maximal M wave was probably not influenced by electrodes which have moved relative to the muscle due to changed muscle architecture or altered muscle length. The second explanation for a decreased maximal M wave is the al-

teration of tissue temperature. It has been shown that local cooling of the abductor digiti minimi induced a significant increase in maximal M wave (Denys, 1990). In contrast, Dewhurst et al. (2005) have revealed a decreased  $M_{max}$  of SOL while warming. The underlying mechanism is supposed to be a change in temperature which alters the opening and closing time of the sodium voltage-gated channels along the nerve axon. Cooling slows down the depolarization time and allows more sodium to enter the cell. This results in an increase of the depolarization amplitude, while warming has the opposite effect (Rutkove, 2001). The present decline of maximal M wave is probably not an effect of temperature change because the research environment was stable during the course of the experiment. Furthermore,  $M_{max}$  onset latencies of both groups were not altered. If RPMS had a significant effect on tissue temperature one would expect a visible discrepancy between the onset latencies of both groups as well as between pre- and post-measurement of TG which was not observable. Repetitive muscle actions typically increase intramuscular temperature (Enoka, 2008) but in accordance with Dewhurst et al. (2005) a rise in temperature had led to decreased onset latencies which were not evident. Therefore the temperature effect is not suitable to



**Figure 4.** H-reflex recruitment curves (◆ H-reflexes, ○ M waves) of one subject before and after RPMS. It becomes apparent, that  $M_{max}$  was reduced after RPMS.

**Table 1.** Soleus M wave associated with  $H_{\max}$  ( $M_{H_{\max}}$ ), peak twitch torque associated with  $H_{\max}$  ( $PT_H$ ),  $H_{\max}$  onset latency and  $M_{\max}$  onset latency before and after RPMS. Values are means ( $\pm$  SD).

	Treatment group (n = 13)			Control group (n = 11)		
	pre	post	p	pre	post	p
$M_{H_{\max}}$	.628 (.319)	.630 (.294)	.973	.731 (.426)	.697 (.405)	.754
$PT_H$ (Nm)	10.20 (2.67)	10.13 (2.81)	.794	10.53 (3.19)	10.70 (4.09)	.739
$H_{\max}$ onset latency (ms)	31.04 (1.01)	31.34 (1.14)	<b>.003</b>	30.22 (1.74)	30.38 (1.79)	<b>.011</b>
$M_{\max}$ onset latency (ms)	4.72 (.29)	4.78 (.33)	.366	4.67 (.37)	4.73 (.39)	.167

explain the difference in the amplitude of  $M_{\max}$  due to the treatment. Third, as mentioned before a decline  $H_{\max}$  and  $M_{\max}$  was observed during the course of an experiment (Crone et al., 1999). Therefore, it is important to look at the  $H_{\max}/M_{\max}$ -ratio in order to interpret results of H-reflex studies. In fact, this could be an explanation for the slight decline of  $H_{\max}$  and  $M_{\max}$  of CG but not a clarification for the significant reduction of  $M_{\max}$  and the continuance of  $H_{\max}$  of TG after RPMS. Fourth, a decline of maximal M wave is observable in relation with alterations in the peripheral structures below the point of stimulation which have the ability to modify neuromuscular propagation (Enoka, 2008). The alteration of neuromuscular propagation is probably an explanation for the results of this study. The relevant locations of concern are (I) impulse propagation along the motor axon into axon branches, (II) neuromuscular transmission and (III) propagation of action potential along the muscle fibre. Krnjevic and Miledi (1958; 1959) suggested that the failure of propagation regarding the motor axon might occur at either the point of branching of the fibre, or in the region where the fibre expands to form the nerve ending. The basic cause is probably the change of ionic gradients associated with loss of excitability (Sieck and Prakash, 1995). Another explanation is the modulation of neuromuscular transmission, due to lowered number of vesicles fused with the presynaptic membrane and quantal size of acetylcholin (ACh) per vesicle (Reid et al., 1999). In this context, the local calcium dynamic is of interest (Gardiner, 2001) because calcium plays an important role in transmitter release, probably by allowing the fusion of vesicles with the presynaptic membrane (Frotscher, 1996). Furthermore, at this level the desensitization of ACh receptors should be taken into account (Giniatullin et al., 1989; Thesleff, 1959). The third level, which is associated with a decline of maximal M wave, refers to changes in the muscle membrane (Shields et al., 1998). That means the alteration of conduction of the action potential along the sarcolemma due to changed ionic gradients (McKenna et al., 2008). The H-reflex response is mainly generated due to the activation of slow twitch units while the maximal M wave is the electrical counterpart of the activation of fast twitch units (Maffioletti et al., 2000b). As is known the different types of motor units include related types of muscle fibres and corresponding thresholds, respectively (Moritani, 2003). Based on our results it is conceivable that RPMS with the used stimulation protocol affected muscle fibres which are part of the fast twitch units. According to this RPMS probably acted on muscle fibres involved in high threshold motor units which are essential for the generation of the maximal M wave. After the treatment these fibres likely possess a RPMS-induced decline in their responsiveness to the supramaximal stimulus. Therefore we suggest that the

impairment of the maximal M wave is due to alterations in the peripheral structures below the point of stimulation which modified the integrity of neuromuscular propagation. In the future, RPMS could be an attractive alternative to EMS for rehabilitation and training applications. However, research is needed to further elucidate the effects.

## Conclusion

It is concluded that the RPMS protocol probably did not affect spinal excitability but acted on the muscle fibres which are part of fast twitch units and mainly responsible for the generation of the maximal M wave. RPMS probably modified the integrity of neuromuscular propagation.

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### Key points

- RPMS probably did not affect spinal excitability.
- Data suggested that RPMS likely acted on the muscle fibres which are part of fast twitch units and mainly responsible for the generation of the maximal M wave.
- RPMS probably modified the integrity of neuromuscular propagation.

### AUTHORS BIOGRAPHY



#### Martin BEHRENS

##### Employment

PhD candidate at the Department of Exercise Science, University of Rostock, Germany

##### Degree

Msc, PhD student

##### Research interests

Physiological adaptation, injury prevention, aging

**E-mail:** martin.behrens@uni-rostock.de



#### Anett MAU-MÖLLER

##### Employment

PhD candidate at the Department of Exercise Science, University of Rostock, Germany

##### Degree

Msc, PhD student

##### Research interests

Physiological adaptation, H reflex, aging

**E-mail:** anett.mau@uni-rostock.de



#### Volker ZSCHORLICH

##### Employment

Professor, Department of Movement Science, University of Rostock, Germany

##### Degree

PhD

##### Research interests

Athletic performance, physiological adaptation, aging

**E-mail:** volker.zschorlich@uni-rostock.de



#### Sven BRUHN

##### Employment

Professor, Department of Exercise Science, University of Rostock, Germany

##### Degree

PhD

##### Research interests

Knee joint stability, physiological adaptation, aging

**E-mail:** sven.bruhn@uni-rostock.de

#### ✉ Martin Behrens

Department of Exercise Science, University of Rostock, Ulmenstraße 69, D-18057 Rostock, Germany