Central and Peripheral Fatigue after Electrostimulation-Induced Resistance Exercise

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1INSERM ERM 207, Faculty of Sport Sciences, University of Burgundy, Dijon, FRANCE; 2Myology Institute, GH Pitié-Salpêtrière, Paris, FRANCE; and 3STAPS Department, University of Savoy, Chambéry, FRANCE

ABSTRACT

BOERIO, D., M. JUBEAU, R. ZORY, and N. A. MAFFIULETTI. Central and Peripheral Fatigue after Electrostimulation-Induced Resistance Exercise. Med. Sci. Sports Exerc., Vol. 37, No. 6, pp. 973–978, 2005. Purpose: To investigate central and peripheral fatigue induced by a typical session of electromyostimulation (EMS) of the triceps surae muscle. Methods: A series of neuromuscular tests including voluntary and electrically evoked contractions were performed before and immediately after 13 min of EMS (75 Hz) in 10 healthy individuals. Results: Maximal voluntary contraction torque of the plantar flexor muscles significantly decreased (−9.4%; P < 0.05), whereas soleus maximal Hoffmann reflex and tibialis anterior coactivation did not change significantly. Contractile properties associated with paired stimuli and maximal M-wave amplitude for both soleus and medial gastrocnemius muscles (−9.4 and −38.7%, respectively) were significantly affected by EMS (P < 0.05), whereas postactivation potentiation did not change. Conclusion: A single bout of EMS resulted in fatigue attributable to both central and peripheral factors. The most obvious alteration in the function of the central nervous system is a decrease in the quantity of the neural drive to muscle from the supraspinal centers. On the other hand, neuromuscular propagation failure was more evident for the muscle with the higher percentage of Type II fibers.

Key Words: TRICEPS SURAE, TORQUE, H REFLEX, EMG ACTIVITY, ACTIVATION LEVEL

Fatigue is defined as any exercise-induced reduction in force generating capacity of a muscle (4), and can originate from peripheral and/or central factors. Peripheral fatigue is typically associated with alterations at or below the neuromuscular junction, whereas central fatigue is defined as a progressive reduction in voluntary activation of muscle during exercise (10) mediated by intrinsic motoneuronal, spinal, and supraspinal factors. Many authors have documented the concomitant occurrence of central and peripheral fatigue after a protocol of maximal or submaximal voluntary contractions of the plantar flexor muscles (14,20,26,27). On the other hand, the acute adjustments in neuromuscular properties consecutive to a single bout of electrostimulation-induced resistance exercise, which is commonly used for muscle strengthening in sports medicine and rehabilitation settings, are poorly known. Electromyostimulation (EMS) is often considered a technique to supplement or to substitute for voluntary activation of muscle with no (apparent) involvement of the central nervous system. This assumption is, however, somewhat paradoxical because the electric stimuli applied at the skin level evoke action potentials in both motor and sensory fibers, thus generating force by direct activation of motor axons and by indirect recruitment of spinal motoneurons. Moreover, it is now well recognized that neural adaptations account for the strength gains associated with short-term EMS training programs, as increases in electromyographic (EMG) activity (6,21), voluntary activation level (21,32), and significant cross-education effect (12,25) have been reported after multiple sessions of EMS. It is therefore possible that a series of contractions triggered by EMS would result in fatigue not only at the muscle level (peripheral fatigue), as easily anticipated due to the artificial nature of the contraction, but also at the central nervous system level.

Central components of triceps surae fatigue can be assessed in maximal voluntary contractions (MVC) with the twitch interpolation technique (23), which involves applying supramaximal electrical stimuli to the posterior tibial nerve, and with surface EMG recordings, provided that raw signals are normalized to the maximal M wave (\(M_{\text{max}}\), that is, the EMG response due to the synchronous activation of most muscle fibers; (15)) for respective muscles. EMG recordings may also offer the possibility of investigating exercise-induced alterations in spinal reflexes, through the analysis of electrically evoked Hoffmann reflex (H reflex; (11)). However, to the best of our knowledge, twitch interpolation and EMG recordings have not been used concomitantly to study neural factors (spinal and supraspinal) pos-

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sibly associated with electrically induced fatigue of the plantar flexor muscles.

The aim of the present study was to investigate central and peripheral fatigue induced by a typical EMS session used for triceps surae muscle strengthening in healthy individuals. It has been hypothesized that decreased voluntary activation of the plantar flexor muscles (based on twitch interpolation and EMG results) due to acute EMS resistance exercise might accompany peripheral fatigue.

**METHODS**

**Subjects.** Ten healthy and physically active male subjects (mean ± SD age: 25 ± 4 yr; height: 176 ± 7 cm; weight: 71 ± 7 kg) volunteered to participate in this study. Subjects gave written informed consent before the experiment, and approval for the project was obtained from the local committee on human research. The study was conducted according to the Declaration of Helsinki.

**Neuromuscular tests.** A series of neuromuscular tests (duration less than 5 min) were performed before and immediately after 13 min of EMS-induced resistance exercise of the triceps surae muscle. Both pre- and postfatigue tests as well as EMS were performed on the right lower leg under isometric conditions. The tests consisted of single and paired stimuli to the posterior tibial nerve at different current intensities and MVC of the plantar flexor and dorsiflexor muscles with concomitant torque and EMG recordings. The techniques and methods adopted in this study have previously been used in our laboratory to investigate neural and mechanical properties of the plantar flexor muscles (21,22,24,29).

Torque during voluntary and evoked contractions was measured by means of an isokinetic dynamometer (Biodex Corporation, Shirley, NY). Subjects were seated comfortably on the dynamometer chair with both knee and ankle joints fixed at 90° and with the trunk inclined 20° with respect to the vertical. The right foot was strapped to the pedal of the dynamometer, and straps were also applied across the chest, pelvis, and midthigh to avoid the contribution of muscles other than the plantar flexors.

The warm-up consisted of submaximal EMS (5 min, frequency 5 Hz, pulses lasting 350 μs; see below for EMS details) and submaximal voluntary contractions of the dorsiflexor and plantar flexor muscles. Subjects were then familiarized with submaximal stimulations of the posterior tibial nerve that were delivered with a cathode ball electrode pressed in the popliteal fossa and an anode (5 × 10 cm) placed between the patella and the patellar tendon. The percutaneous electrical stimulation was a rectangular pulse (1-ms duration) delivered by a Digitimer DS7 stimulator (Digitimer, Herthfordshire, UK). For each individual, stimulation intensity was progressively increased by 4-mA increments from 20 mA until there was no further increase in peak-to-peak amplitude of soleus M wave (i.e., Mmax).

The recruitment curve of soleus H reflex was obtained at rest. For each subject, the stimulus intensity was gradually increased (every two stimuli) from below the threshold for an H-reflex response to the maximal H reflex (Hmax). Thirty reflex responses were thus recorded over a 90-s period, with a rather short repetition interval between stimuli (3 s). Nevertheless, according to Pierrot-Deseilligny and Mazevet (28) recommendations, H-reflex explorations at 0.2–0.3 Hz are allowed, considering that one needs to find a compromise between postactivation depression and the necessity of collecting a large number of reflexes because of variability. Three single and three paired stimuli (10-ms interval) were then delivered at rest at the Mmax intensity (range 40–60 mA), each separated by 3–4 s, to study twitch and doublet (nonpotentiated) contractile properties and Mmax characteristics. Two MVC of the plantar flexor muscles were performed to study the voluntary torque capacity and the associated neural activation, that is, EMG activity, activation level (1), and central activation ratio (17). The duration of these contractions was 4 s, and electrically evoked paired stimuli were delivered 1.5 s after the beginning of the contraction (i.e., superimposed doublet) and 1.5 s after the MVC (i.e., potentiated doublet). Finally, subjects completed one dorsiflexor MVC.

**EMG activity.** Silver chloride surface electrodes (10 mm in diameter) were used to record EMG activity of two plantar flexor muscles: the soleus and medial gastrocnemius (MG), and one dorsiflexor muscle: the tibialis anterior (TA). All the subjects were prepared for EMG electrode placement by shaving the skin at each electrode site, abrading with sandpaper, and cleaning with alcohol–ether–acetone mix (interelectrode impedance less than 5 kΩ). Electrodes were then fixed lengthwise over the middle of the muscle belly (interelectrode distance of 20 mm), with the reference electrode being placed on the left wrist. EMG signals were amplified with a bandwidth frequency ranging from 15 Hz to 2.0 kHz.

**EMS session.** Two square (5 × 5 cm) positive electrodes were placed over the superficial aspect of the soleus muscle, about 5 cm distal from where the medial and lateral heads of the gastrocnemius muscle join the Achilles tendon. The rectangular (10 × 5 cm) negative electrode was placed along the middorsal line of the leg, over both medial and lateral gastrocnemius. A portable stimulator (Medicompex SA, Ecublens, Switzerland) was used to deliver biphasic symmetric rectangular-wave pulsed currents with the following characteristics: frequency 75 Hz, pulse width 400 μs, duty cycle 24% (6.25 s on, 20 s off), rise time 1.5 s, fall time 0.75 s. Thirty contractions were completed (duration 13 min) at the maximal tolerated intensity, which was identified as the intensity of stimulation received when the subject said that he could no longer tolerate an increase in intensity. This is the most common method used for intensity determination in clinical settings, in line with the recommendations of Lake (19). Maximal tolerated intensity varied between 18 and 50 mA, depending on differences among subjects in pain threshold. The individual level of isometric torque developed at the beginning of the EMS session averaged 55% of the prefatigue MVC.

**Data analysis.** Mechanical and EMG traces were digitized online (sampling frequency 2024 Hz) and stored for
Neural activation. Raw EMG RMS values dropped at the end of the EMS session for both soleus (−21.4%; \( P < 0.001 \)) and MG muscle (−17.7%; \( P < 0.01 \), Table 1). When normalized to \( M_{\text{max}} \) amplitude, RMS values showed controversial results since a significant decrease was observed for soleus (−10.8%; \( P < 0.05 \), Table 1), but the RMS/\( M_{\text{max}} \) ratio of MG significantly increased (\( P < 0.05 \)). Both activation level and central activation ratio significantly decreased (−1.6 and −4.5% respectively; \( P < 0.05 \)) after EMS (Fig. 2; see also Fig. 1).

H reflex and coactivation. Soleus H-reflex amplitude (from 3.6 ± 2.8 to 3.5 ± 2.3 mV) and \( H_{\text{max}}/M_{\text{max}} \) ratio (Fig. 3) did not change significantly after EMS. In the same way, TA coactivation was not significantly modified by the present EMS bout (Fig. 3).

Contractile properties and \( M_{\text{max}} \) characteristics. Evoked torque decreased from 55 to 44% MVC during the EMS bout (first five vs last five contractions; \( P < 0.001 \)). All doublet contractile properties and twitch HRT were significantly lower after EMS (\( P < 0.05 \)), whereas no sizeable changes were observed for twitch PT and TPT (Table 2). This was true also if \( M_{\text{max}} \) amplitude of both soleus and MG significantly decreased (−9.4 and −38.7%, respectively; \( P < 0.05 \); Table 1). \( M_{\text{max}} \) duration for respective muscles were comparable between before (2.8 ± 0.5 and 3.4 ± 1.6 ms for soleus and MG, respectively) and after EMS (2.7 ± 0.4 and 3.7 ± 1.9 ms for soleus and MG, respectively). Postactivation potentiation was not modified by the present EMS bout (from 1.05 ± 0.10 to 1.08 ± 0.06).

DISCUSSION

The results obtained in this study indicated that both central and peripheral mechanisms contributed to the triceps surae muscle fatigue observed after a single bout of electrostimulation-induced resistance exercise, similar to those used in sports medicine and rehabilitation settings. The significant plantar flexor MVC torque reduction (−9%) observed after EMS was accompanied by decreased voluntary activation of the agonist muscles, as attested by twitch interpolation and normalized EMG results, and by maximal M-wave depression, which was more marked for the medial gastrocnemius than for the soleus muscle.

The MVC torque loss observed after EMS was associated with a significant reduction of the raw EMG activity of both soleus and MG muscle. Because EMG recorded at the skin

## RESULTS

**MVC torque.** MVC torque of the plantar flexor muscles significantly decreased 9.4% (from 112.0 ± 15.8 to 101.8 ± 19.1 N·m; \( P < 0.001 \)) after EMS (Fig. 1). Similarly, MVC of the nonstimulated dorsiflexor muscles was significantly lower after EMS (from 37.4 ± 7.2 to 35.2 ± 6.8 N·m; \( P < 0.05 \)).

**Neural activation.** Raw EMG RMS values dropped at the end of the EMS session for both soleus (−21.4%; \( P < 0.001 \)) and MG muscle (−17.7%; \( P < 0.01 \), Table 1). When normalized to \( M_{\text{max}} \) amplitude, RMS values showed controversial results since a significant decrease was observed for soleus (−10.8%; \( P < 0.05 \), Table 1), but the RMS/\( M_{\text{max}} \) ratio of MG significantly increased (\( P < 0.05 \)). Both activation level and central activation ratio significantly decreased (−1.6 and −4.5% respectively; \( P < 0.05 \)) after EMS (Fig. 2; see also Fig. 1).

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**TABLE 1.** Raw (RMS) and normalized (RMS/\( M_{\text{max}} \) ratio) EMG activity and \( M_{\text{max}} \) amplitude before and after the EMS bout.

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<tr>
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<td>RMS (mV)</td>
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<td>8.1 ± 2.2</td>
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<tr>
<td></td>
<td>post</td>
<td>7.3 ± 2.2*</td>
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MG, medial gastrocnemius; RMS, root mean square. Mean values ± SD.
* Post less than or greater than pre (\( P < 0.05 \)).
** Post less than pre (\( P < 0.01 \)).
*** Post less than pre (\( P < 0.001 \)).

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**FIGURE 1**—Recordings from one representative subject of plantar flexor MVC torque with superimposed doublet (arrow) followed by potentiated doublet at rest, before (gray line) and after (black line) the EMS bout. Note that the superimposed torque was higher whereas the resting doublet was lower (i.e., lower voluntary activation) in the fatigued state.

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level is inevitably affected by peripheral influences (e.g., changes in impedance, neuromuscular propagation failure), the signal was normalized to the maximal M-wave for respective muscles to better characterize neural drive. The RMS/M\text{max} ratio was indeed significantly lower for the soleus after EMS, but higher for the MG muscle. These results would confirm the occurrence of central fatigue for the former muscle, whereas for the gastrocnemius, the present normalization technique appears inappropriate in the presence of such an M\text{max} depression (~40%, see below). Because the twitch interpolation technique provides an estimation of the compound level of activation of the muscles innervated by the tibial nerve, the occurrence of central fatigue for the plantar flexor muscles was confirmed by the significant decrease of both activation level and central activation ratio after EMS. These results are in agreement with Avela et al. (2), who found that the interpolated double stimulation compensated for the force loss by 4.3% after 1 h with Avela et al. (2), who found that the interpolated double activation ratio after EMS. These results are in agreement with studies demonstrating no significant change in soleus H\text{max}/M\text{max} ratio after 10 min of intermittent EMS administered to healthy subjects. Although EMS may activate both motor and sensory fibers, the nonoccurrence of spinal fatigue would confirm earlier results obtained in our laboratory after multiple EMS sessions. It was indeed demonstrated that both the EMG activity and activation level significantly increased after 4 wk of EMS (21), whereas the soleus and gastrocnemius H\text{max}/M\text{max} ratios did not change significantly (22). Consequently, it seems that both chronic and acute adjustments associated with EMS of the triceps surae muscle would occur at a supraspinal level, therefore confirming the dose–response activation of supraspinal centers observed during EMS (31). Whatever the exact mechanism mediating the central fatigue observed in the present study, further research is warranted to analyze the behavior of the nervous system during contractions triggered by EMS.

In addition to central activation, force production is controlled by neuromuscular propagation and cellular mechanisms. The considerable M\text{max} reductions observed here indicate that a failure in the neuromuscular transmission–propagation and/or sarcolemmal excitation played a dominant role in the observed torque decline during both voluntary and stimulated contractions, for with transcutaneous stimulation it is likely that the muscle is excited via intra-muscular nerve endings (5). However, maximal M-wave duration was not significantly affected by EMS, therefore indicating that a slowing of conduction velocity along muscle cell membranes did not occur in our experimental conditions. In line with the current findings, a marked decrease in maximal M-wave amplitude during and after high-frequency (80–100 Hz) fatigue in both \textit{in vitro} (3) and \textit{in vivo} muscle preparations (7) has previously been demonstrated. It has also been shown that animal muscles with the higher percentage of fast-glycolytic fibers exhibited the greater alterations in muscle action potentials after electrically induced fatigue (13, 18), mainly because of potassium accumulation in

### Table 2. Contractile properties associated to twitch and doublet before and after the EMS bout.

<table>
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<th>PT (N.m)</th>
<th>TPT (ms)</th>
<th>HRT (ms)</th>
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<tbody>
<tr>
<td>Twitch</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>18.1 ± 4.1</td>
<td>109.8 ± 14.1</td>
<td>200.3 ± 10.4</td>
</tr>
<tr>
<td>Post</td>
<td>17.3 ± 3.7</td>
<td>110.5 ± 9.4</td>
<td>184.4 ± 11.0***</td>
</tr>
<tr>
<td>Doublet</td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>Pre</td>
<td>32.5 ± 7.5</td>
<td>131.6 ± 18.0</td>
<td>227.9 ± 9.7</td>
</tr>
<tr>
<td>Post</td>
<td>30.6 ± 6.5*</td>
<td>119.8 ± 10.8*</td>
<td>211.9 ± 10.2***</td>
</tr>
</tbody>
</table>

PT, peak torque; TPT, time to peak torque; HRT, half-relaxation time. Mean values ± SD. * Post less than pre (P < 0.05). *** Post less than pre (P < 0.001).
extracellular spaces (i.e., high-frequency fatigue; 8). The results obtained here extend these findings to human slow-twitch soleus and mixed gastrocnemius, as $M_{\text{max}}$ amplitude decreased, respectively, 9 and 39% in the fatigued state.

The analysis of the mechanical responses induced by single or double supramaximal electrical stimulation allows one to indirectly investigate the possible muscle intracellular changes responsible for the reduced mechanical performance. The more significant EMS exercise-induced alteration was the reduction of half-relaxation time for both twitch and doublet, suggesting that relaxation from contraction, which is an active process regulated by sarcoplasmic reticulum calcium and/or myosin ATP-ase (35), was accelerated after EMS. Although an increase in muscle temperature and/or changes in muscle stiffness could also explain the present results, these possibilities are purely speculative at this moment, and it can only be concluded that some changes occurred at the level distal to the sarcolemma. The fact that postactivation potentiation, which is closely related to the sensitivity of contractile proteins to calcium (34), was not significantly affected by the present session of electrostimulation-induced resistance exercise (pre-EMS: $1.05 \pm 0.10$ vs post-EMS: $1.08 \pm 0.06$) allows to conjecture that excitation–contraction coupling was not or little influenced by EMS, as also indicated by twitch and $M_{\text{max}}$ changes. Taken together, these findings suggest that the acute bout of EMS exercise led to high-frequency fatigue (8), but little, if any, low-frequency fatigue of the triceps surae muscle. This aspect deserves to be studied specifically in the future.

In conclusion, it has been shown that the decrease in plantar flexion torque after a single session of electrically evoked isometric contractions resulted in fatigue attributable to both central and peripheral factors. The most obvious change in the function of the nervous system is a decrease in the quantity of the neural drive to muscle from the supraspinal centers. On the other hand, neuromuscular propagation failure was attested for both soleus and gastrocnemius, the effect being maximal for this latter muscle. Future studies are needed to determine whether multiple EMS sessions will serve to minimize central and/or peripheral fatigue of the plantar flexor muscles.

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REFERENCES


ELECTRICALLY INDUCED NEUROMUSCULAR FATIGUE

Medicine & Science in Sports & Exercise一位助手。


