Torque decrease during submaximal evoked contractions of the quadriceps muscle is linked not only to muscle fatigue

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Matkowski B, Lepers R, Martin A. Torque decrease during submaximal evoked contractions of the quadriceps muscle is linked not only to muscle fatigue. J Appl Physiol 118: 1136–1144, 2015. First published March 12, 2015; doi:10.1152/japplphysiol.00553.2014.—The aim of this study was to analyze the neuromuscular mechanisms involved in the torque decrease induced by submaximal electromyostimulation (EMS) of the quadriceps muscle. It was hypothesized that torque decrease after EMS would reflect the fatigability of the activated motor units (MUs), but also a reduction in the number of MUs recruited as a result of changes in axonal excitability threshold. Two experiments were performed on 20 men to analyze 1) the supramaximal twitch superimposed and evoked at rest during EMS (Experiment 1, n = 9) and 2) the twitch response and torque-frequency relation of the MUs activated by EMS (Experiment 2, n = 11). Torque loss was assessed by 15 EMS-evoked contractions (50 Hz; 6 s on/6 s off), elicited at a constant intensity that evoked 20% of the maximal voluntary contraction (MVC) torque. The same stimulation intensity delivered over the muscles was used to induce the torque-frequency relation and the single electrical pulse evoked after each EMS contraction (Experiment 2). In Experiment 1, supramaximal twitch was induced by femoral nerve stimulation. Torque decreased by ~60% during EMS-evoked contractions and by only ~18% during MVCs. This was accompanied by a rightward shift of the torque-frequency relation of MUs activated and an increase of the ratio between the superimposed and posttetanic maximal twitch evoked during EMS contraction. These findings suggest that the torque decrease observed during submaximal EMS-evoked contractions involved muscular mechanisms but also a reduction in the number of MUs recruited due to changes in axonal excitability.

ELECTROMYOstimulation (EMS) is a technique that applied over a muscle or nerve, evokes muscle contraction via activation of both motor and sensory axons (10, 15, 19), thus generating contractions through peripheral and central pathways (5, 13, 19). This pattern via which motor units (MUs) are activated differs from voluntary contractions (17, 38), leading to exaggerated metabolic demand and greater force loss compared with voluntary exercise performed at the same intensity (21, 28, 37, 40).

The greater fatigability and discomfort associated with EMS have limited its clinical use. To improve its application, numerous researchers have focused on determining a stimulation protocol that minimizes fatigability and increases muscle performance (7, 8, 39). Most studies have been performed on able-bodied subjects in whom fatigability was quantified as the percent decline of the electrically evoked torque between the beginning and the end of the fatiguing exercise (start-end torque index) (7, 8, 17, 39). However, this torque decrease does not reflect the functional impact of the stimulation protocol on the force-generating capacity of an individual, as assessed by the maximal voluntary contraction (MVC). Indeed, greater declines in torque have been observed after EMS exercises evoked at constant and variable stimulation frequencies compared with MVC torque (30, 31). Moreover, similar results were observed when the maximal twitch was compared with one elicited at the stimulation intensity used during the EMS exercise (31). These contrasting results relating to torque losses between MVC and evoked contractions suggest that mechanisms, other than the fatigability of activated MUs, contribute to the decline in torque during an EMS protocol. Potential mechanisms that may contribute to greater torque decline during submaximal fatiguing EMS include an increase in the excitability threshold of the active axons due to repetitive stimulation, which leads to a decrease in the number of active axons, and thus contributes to torque decrease (4, 12, 23).

The twitch interpolation technique can be used to investigate the possible reduction in the number of MUs recruited during EMS fatiguing exercise, in association with a change in the excitability threshold of active axons. This technique, initially developed by Merton (29) for the adductor pollicis muscle, and subsequently widely used for other muscle groups such as the quadriceps femoris muscle (3, 27, 33), relies on the comparison of the maximal twitch force produced by a supramaximal nerve stimulation evoked at rest to that produced by the same stimulus superimposed on a voluntary contraction. The extra force developed by the interpolated twitch provides a quantification of the proportion of the muscle force that is not involved in the voluntary effort. This technique, previously used during a tetanic contraction of human adductor pollicis (16), can be applied during submaximal EMS fatiguing contractions to quantify the proportion of MUs that are not activated by electrical stimulation.

The aim of the present study was to evaluate the possible mechanisms involved in EMS-evoked fatigue by measuring 1) the maximal twitch evoked at rest and superimposed during EMS and 2) the mechanical response and torque-frequency relation of the activated muscle. We hypothesized that the torque decrease at the end of the EMS protocol would reflect not only the fatigability of the activated MUs, but also a reduction in the number of MUs recruited due to changes in axonal excitability threshold.
MATERIALS AND METHODS

Subjects

Twenty healthy men (Experiment 1: n = 9, age 27.4 ± 6.9 yr, height 176.0 ± 5.1 cm, weight 70.7 ± 7.1 kg; Experiment 2: n = 11, age 26.4 ± 5.7 yr, height 179.1 ± 5.8 cm, weight 76.3 ± 7.0 kg; mean ± SD) volunteered to participate in the present study and were tested before, during, and after fatiguing contractions elicited by EMS. None of them had any known neurological or neuromuscular disorder. Each individual was informed about the experimental procedures and possible risks and provided informed consent prior participating in the study. The University of Burgundy Committee on Human Research approved the protocol. The study adhered to the policies outlined in the Declaration of Helsinki.

Torque Measurements

Mechanical measurements were performed using an isokinetic dynamometer (Biodex, Shirley, NY) in an isometric mode. The axis of the dynamometer was aligned with the flexion-extension axis of rotation for the knee, and a strap attached the lever arm to the shank. The upper body was restrained with two crossover shoulder harnesses and a belt across the abdomen. Experiments were performed on the right (dominant) leg with knee and hip joint angles of 90°. Subjects were asked to cross their arms over the chest during the testing procedure.

Electromyostimulation

EMS of the quadriceps muscle was applied with a high-voltage (maximum 400 V), constant-current stimulator (DS7A; Digitimer, Hertfordshire, UK) to evoke repeated tetanic contractions. Two large electrodes were placed on the anterior aspect of the thigh. The cathode (10 × 5 cm; Compex, Ecublens, Switzerland) was placed distally at 10 cm above the upper border of the patella over vastus lateralis (VL), vastus medialis (VM) and rectus femoris (RF) muscles. The anode (10 × 5 cm) was placed ~5 cm below the inguinal ligament. A pulse duration of 1 ms was used and the current was set so that a 50-Hz stimulation elicited 20% of the MVC torque developed before the fatiguing exercise (see below). The required current (range: 26–68 mA) caused only mild discomfort and no noticeable voluntary contractions during EMS-evoked contractions. For the second experiment, this intensity was also used to induce a single twitch after each EMS-evoked contraction, and the torque-frequency relation was recorded before and after the fatiguing exercise.

Femoral Nerve Stimulation

The femoral nerve was stimulated with a second constant-current stimulator (DS7A; Digitimer, Hertfordshire, UK) to evoke maximal single twitches during (superimposed twitch) and after EMS-evoked contractions. The femoral nerve was stimulated with a 1-ms pulse using a cathode ball electrode (0.5 cm diameter) pressed into the femoral triangle by the same experimenter during all testing sessions. The anode was a 10 × 5 cm rectangular electrode (Compex, Ecublens, Switzerland) located in the gluteal fold opposite the cathode. The stimulus current was considered maximal when an increase in the current no longer increased the amplitude of the twitch torque or the peak-to-peak amplitude of the compound muscle action potentials (M-wave; see below). The current intensity was increased by 25%, and this supramaximal stimulus intensity was used throughout the experiment (range: 60–90 mA).

Electromyography

Electromyographic (EMG) signals were recorded in VL, VM, and RF muscles using pairs of silver-silver chloride electrodes (10 mm diameter; 20 mm between electrodes; Controle Graphique Medical, Brie-Comte-Robert, France) positioned over each muscle according to the European Union recommendations for surface electromyography known as SENIAM. The recording sites were adjusted in pilot testing to detect the greatest M-wave amplitude at a given intensity for each knee extensor muscle (33). The reference electrode was attached to the patella of the left knee. The skin was abraded and cleaned with alcohol to minimize resistance between the two electrodes (<5 kΩ). EMG signals were amplified with a bandwidth frequency ranging from 15 Hz to 2 kHz (common mode rejection ratio = 90 dB; impedance input = 100 MΩ; gain = 1,000). EMG signals and torque measurements were digitized online at a sampling frequency of 2 kHz and stored for analysis with commercially available software (Tida, Heka Elektronik, Lambrecht/Pfalz, Germany).

Experimental Protocols

Two different experimental protocols were used (Fig. 1).

Experiment 1. The first protocol (n = 9) was intended to determine the influence of EMS-evoked fatiguing contractions on superimposed and maximal posttetanic twitches evoked by stimulation of the femoral nerve at supramaximal intensity. Subjects performed a warm-up that included ~10 brief nonfatiguing submaximal contractions with the knee extensor muscles. After a 2-min rest, subjects began the experimental protocol. Before and after the fatiguing contractions, subjects performed 5-s MVCs with knee extensors. They were asked to contract their knee extensor muscles as forcefully as possible for 5 s. A supramaximal single pulse stimulus to the motor nerve was delivered when the torque plateau was reached (superimposed twitch), as well as 2 s after the end of the MVC (potentiated twitch). Investigators provided strong verbal encouragement during each MVC. Before the EMS exercise, subjects performed two MVCs separated by a 2-min rest period, whereas only one MVC was performed after the exercise.

The fatiguing task comprised 15 EMS-evoked contractions via electrodes applied over the quadriceps muscles. The current intensity used in the experiment produced 20% of the MVC developed prior to EMS exercise. This intensity was kept constant throughout the experiment. Each contraction lasted for 6 s interspaced by 6 s of rest. A single-pulse supramaximal stimulus of the femoral nerve was delivered in the middle of each evoked contraction, as well as and during each reducing period (2 s after the contraction) (Fig. 1A). The superimposed supramaximal nerve stimulation was evoked during the 20-ms interpulse interval at 7 ms after the beginning of the interval. This 7-ms interpulse interval avoided the collision between orthodromic and antidromic volleys generated by the supramaximal stimulus delivered over the motor nerve and the 50-Hz stimulation delivered over the muscle, respectively. At the muscle level, this 7-ms interpulse interval gives, depending on the muscle considered, a mean stimulation frequency of ~100 Hz for the MUs activated by EMS (see Methodological Considerations).

Experiment 2. The second experiment (n = 11) was designed to assess fatigability during EMS-evoked contractions and its influence on the posttetanic twitch and the torque-frequency relation evoked at the stimulation intensity used during the EMS exercise. Subjects began the experimental protocol 2 min after the warm-up as previously described (see Experiment 1).

Before and after the fatiguing contractions, neuromuscular tests composed of voluntary and evoked contractions were performed. As for Experiment 1, subjects performed MVCs of the knee extensors lasting 5 s. A supramaximal stimulus was delivered when the torque plateau was reached (superimposed twitch), as well as 2 s after the end of the MVC (potentiated twitch). In addition to the MVCs, the torque-frequency relation (Fig. 1B) was established by randomly evoking 1-s contractions with EMS at a stimulus frequency of 5, 10, 15, 20, 30, 40, 50, 60, 75, and 100 Hz, with 6-s rest periods between each frequency. The current intensity, applied via electrodes placed over the muscles, was set at the intensity that evokes a stimulation frequency of 50 Hz, 20% of the MVC.
recorded before EMS exercise. This stimulation intensity corresponded to that used for EMS exercise and was kept constant before and after the fatiguing exercise, as well as across the frequencies. As for the first experiment, the fatiguing task was induced by 15 EMS-evoked contractions lasting 6 s and interspaced by 6 s of rest (Fig. 1B). During this rest period and 2 s after the end of the contraction, a single electrical pulse was delivered at the current intensity that was used during EMS contraction, i.e., corresponding to a stimulation intensity that elicited 20% MVC prior to EMS exercise.

Data Analysis

Before and after the fatigue test. MVC torque was considered as the peak torque attained during the contraction. Before the fatiguing task, the MVC producing the highest torque was considered. The torque loss from the 1st to the 15th EMS contraction was considered as an index of muscle fatigability. Posttetanic twitch amplitude in response to nerve (Experiment 1) and muscle (Experiment 2) stimulation recorded during the 6-s rest period was analyzed after each of the 15 EMS-evoked contractions. For Experiment 1, the superimposed twitch evoked during each EMS contraction was analyzed as the amplitude of the electrically evoked twitch from the torque that developed just before supramaximal nerve stimulation (artifact stimulus on EMG trace) to the peak of the superimposed twitch. To obtain an estimation of the proportion of the muscle not activated by the stimulation during the EMS-evoked contraction, a muscle activation level (MAL) was estimated by the same formula used for VAL, but for MAL, the potentiated twitch was replaced by the posttetanic twitch, i.e., the twitch evoked after each EMS contraction.

For Experiment 1, peak-to-peak amplitude, duration, latency, and area of Mmax associated with the posttetanic twitch evoked by supramaximal nerve stimulation were analyzed for the 1st and the 15th EMS contraction for VL, VM, and RF muscles. For Experiment 2, the latency of the M-wave associated with the twitch evoked at EMS intensity was analyzed after the 1st EMS contraction. Due to the proximity of the site of stimulation and detection over the muscle, it was possible to analyze the M-wave latency only on a limited number of subjects (n = 6 for VL, n = 9 for VM, n = 8 for RF).

Methodological Considerations

One methodological issue with the use of superimposed supramaximal stimuli evoked at the nerve on submaximal EMS contractions evoked at the muscle, is to avoid the collision of the orthodromic volleys generated by the supramaximal stimulus with the antidromic volleys generated by EMS. To avoid this, the delay between EMS stimulation and nerve stimulation (7-ms interpulse interval) was chosen to be longer than the time needed for the antidromic volleys, initiated by EMS, to reach the site of femoral nerve stimulation (calculated to be 3.66 ± 2.67, 3.23 ± 1.11, and 1.51 ± 2.72 ms for VL, VM, and RF, respectively). This time is shorter than the 7-ms interpulse interval used here, suggesting that in the present study, the
supramaximal superimposed twitch to EMS contractions was not contaminated by antidromic collisions. The latency difference (added to the 7-ms delay between EMS and femoral nerve stimulation) was also used to estimate the stimulation frequency at which the MUs were activated by both nerve and muscle stimulations (117, 98, and 94 Hz, for RF, VM, and VL, respectively). These frequencies were close to the maximal frequency used to determine the torque-frequency relation induced by EMS.

**Statistical Analysis**

One-way repeated measures ANOVA and post hoc Tukey tests were used to assess fatigue-induced changes in the evoked torque, and amplitude of the superimposed and posttetanic twitches. Paired $t$-tests were used to compare the ratio between superimposed and posttetanic twitch amplitude, MVC torque, potentiated twitch amplitude, VAL, and M-wave amplitude, duration, latency, and area before and after the fatiguing stimulations. Paired $t$-tests were used to compare MAL and M-wave amplitude, duration, latency, and area between the 1st and 15th fatiguing stimulations. Two-way repeated measures ANOVA and post hoc Tukey tests were used to assess fatigue-induced changes in the torque-frequency relation. A Wilcoxon test was used to assess change in MVC torque and potentiated twitch amplitude across the two experiments. A significance level of $P < 0.05$ was used to identify statistical significance. Statistical analyses were performed using Statistica software for Windows (Statsoft version 6.1; Statistica, Tulsa, OK). Data are reported as means ± SD within the text and tables and as means ± SE in the figures.

**RESULTS**

**MVC and EMS-Evoked Changes**

The mean torque achieved in Experiment 1 during the 1st evoked contraction was 56.8 ± 18.8 Nm, and this decreased rapidly during the subsequent evoked contractions (Fig. 2). Torque was significantly depressed ($P < 0.001$) after the 3rd contraction and declined by ~60% of the initial value at the 15th contraction. A similar decrease in torque was observed in Experiment 2 (Fig. 4), when it was significantly ($P < 0.001$) depressed by the 4th contraction and decreased by ~62% of its initial value at the 15th contraction (Fig. 4C).

MVC torque (Table 1) decreased by 14.2 ± 6.9% of initial torque after the fatiguing contractions in Experiment 1 ($P < 0.001$) and by 19.4 ± 8.1% ($P < 0.001$) in Experiment 2. The decline in evoked torque was similar for the two experiments ($P = 0.15$). The EMS-evoked torque decreased by 61.1 ± 14.6% across the two experiments, compared with a reduction of 17.6 ± 8.0% in MVC torque. There was no association between the decrease in EMS-evoked torque and MVC torque in either experiment (Fig. 3).

VAL was estimated from the stimuli superimposed on the MVCs during both experiments (Table 1), and there were no changes after the fatiguing EMS-evoked contractions ($P > 0.05$). In contrast, potentiated twitch torque decreased by 12.2 ± 10.5% ($P < 0.01$) after the fatiguing contractions in...
of the 1st evoked contraction. The posttetanic twitch amplitude posed twitch was 26.6 ± 7.51 Nm after the 1st EMS-evoked contraction. The posttetanic twitch amplitude was calculated for the 1st and the 15th contractions evoked by EMS during the first experiment (at supramaximal intensity), MAL (M-wave latency) increased in posttetanic twitch amplitude and EMS-evoked torque between the evolution of EMS-evoked torque and posttetanic contractions in Experiment 1 (Table 1). The decrease in EMS-evoked torque after the 15th contraction was accompanied by a decrease in both MVC and EMS-evoked torque (18%) and the torque elicited by EMS (60%). The adjustments included a rightward shift of the torque-frequency relation and a decrease in muscle activation level recorded during the EMS exercise. These results suggest that the torque decrease observed during submaximal EMS-evoked contractions involved muscular mechanisms, but also a reduced number of MUs recruited by the stimulation, likely due to changes in axonal excitability.

**MVC and EMS-Evoked Changes**

The submaximal EMS used in the present study induced a marked torque decrease of ~60% after only 15 contractions. After the fatiguing contractions (Fig. 6A). The torque produced at 50 Hz was similar (P = 0.78) before and during the 1st contraction. However, the decrease in torque differed between the end of the fatiguing contraction (which decreased by ~60%) and the torque-frequency test (which decreased by ~40%, P > 0.001), indicating that some recovery had occurred before the torque-frequency test. Normalization of the data indicated a rightward shift of the torque-frequency relation after the fatiguing stimulation (Fig. 6B).

Statistical analysis of the \( M_{max} \) parameters associated with the posttetanic twitch evoked by supramaximal nerve stimulation at the 1st and 15th EMS contractions (Experiment 1) revealed a significant decline in amplitude for VM and RF muscles (~10.3 ± 12.0% and ~14.9 ± 14.3%, respectively; P < 0.05) and area for VM muscle (~16.0 ± 19.8%, P < 0.05), whereas M-wave duration increased in RF muscle (~17.6 ± 11.4%, P < 0.01). Regardless of the muscles considered, maximal M-wave latency did not differ between the start and end of the EMS exercise (Table 2).

**DISCUSSION**

The present study examined 1) the maximal twitch evoked at rest and superimposed during EMS, and 2) the mechanical response and torque-frequency relation of the active muscle. The results showed that fatigability elicited by EMS-evoked contractions was accompanied by a decrease in both MVC (18%) and the torque elicited by EMS (~60%). The adjustments included a rightward shift of the torque-frequency relation and a decrease in muscle activation level recorded during the EMS exercise. These results suggest that the torque decrease observed during submaximal EMS-evoked contractions involved muscular mechanisms, but also a reduced number of MUs recruited by the stimulation, likely due to changes in axonal excitability.

### Table 1. Maximal voluntary contraction torque, potentiated twitch amplitude at rest, and voluntary activation level of the knee extensors muscles before and after EMS-evoked contraction

<table>
<thead>
<tr>
<th></th>
<th>Experiment 1</th>
<th>Experiment 2</th>
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<tbody>
<tr>
<td></td>
<td>Before</td>
<td>After</td>
</tr>
<tr>
<td>MVC, Nm</td>
<td>242.7 ± 48.5</td>
<td>208.8 ± 46.6( ^\dagger )</td>
</tr>
<tr>
<td>Tw pot, Nm</td>
<td>65.4 ± 11.1</td>
<td>57.2 ± 10.1*</td>
</tr>
<tr>
<td>VAL, %</td>
<td>92.6 ± 5.8</td>
<td>93.4 ± 3.8</td>
</tr>
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MVC, maximal voluntary contraction torque; Tw pot, potentiated twitch amplitude at rest; VAL, voluntary activation level. Values are means ± SD. *P < 0.01, †P < 0.001; significantly different from the before value.

Experiment 1 and by 24.9 ± 8.9% (P < 0.001) after the fatigue contractions in Experiment 2 (Table 1). The decrease was greater in Experiment 2 (P = 0.02). There were small but statistically significant changes in \( M_{max} \) amplitude but not area for RF and for amplitude and area for VL and RF in Experiment 2 (Table 2). No changes were found in \( M_{max} \) duration and latency.

**Fatiguing Contractions**

The large decrease in EMS-evoked torque after the 15th contraction was accompanied by an increase in superimposed twitch amplitude and a decrease in posttetanic twitch. Mean data from the nine subjects participating in this set of experiments are shown in Fig. 2C. The amplitude of the superimposed twitch was 26.6 ± 4.9 Nm in the 1st evoked contraction. It then increased, being significantly larger (P < 0.001) from the 5th block until the last evoked stimulation, reaching ~120% of the 1st evoked contraction. The posttetanic twitch amplitude started at 42.1 ± 3.8 Nm after the 1st EMS-evoked contraction and significant decreased from the 5th until the last block (~10%, P < 0.001).

To characterize the MUs activated during the submaximal EMS protocol, a single electrical pulse delivered at the stimulation intensity used during the EMS protocol was delivered after each EMS-evoked contraction (posttetanic twitch). Figure 4C shows a large decrease in posttetanic twitch during the fatiguing contraction, which was accompanied by a decrease in EMS-evoked torque. The posttetanic twitch amplitude was 7.51 ± 3.83 Nm after the 1st contraction. It was significantly depressed from the 3rd block until the end of fatigue stimulation (64.9 ± 8.9%, P < 0.001). A linear relation was found between the evolution of EMS-evoked torque and posttetanic twitch, parallel to the line of identity, indicating similar declines in posttetanic twitch amplitude and EMS-evoked torque (r = 0.98; Fig. 5).

From the superimposed and posttetanic twitches obtained during the first experiment (at supramaximal intensity), MAL was calculated for the 1st and the 15th contractions evoked by EMS. MAL was 36.3 ± 9.0% for the 1st evoked contraction and decreased to 14.3 ± 7.8% for the 15th evoked contraction (~59.9%, P < 0.001).

The torque-frequency relationship was analyzed before and after the fatiguing protocol with the EMS parameters, i.e. the same pulse width and stimulation intensity used during fatiguing exercise. Torque decreased at all stimulation frequencies after the fatiguing contractions (Fig. 6A). The torque produced at 50 Hz was similar (P = 0.78) before and during the 1st contraction. However, the decrease in torque differed between the end of the fatiguing contraction (which decreased by ~60%) and the torque-frequency test (which decreased by ~40%, P > 0.001), indicating that some recovery had occurred before the torque-frequency test. Normalization of the data indicated a rightward shift of the torque-frequency relation after the fatiguing stimulation (Fig. 6B).
 Previous studies have already demonstrated comparable decreases in torque after submaximal evoked contractions when the stimulation intensity was kept constant during the entire exercise (e.g., 7, 8). This torque decrease is generally attributed to the unnatural order in which MUs are activated by EMS (25). Indeed, it is now well known that during EMS with a wide pulse, muscle fibers are recruited through motor axon depolarization, as well as through activation of spinal motor neurons by the afferent volley evoked by the stimulus (13, 19). Because EMS performed over the muscle belly generated contractions predominantly through the direct activation of the motor axons (6) and considering that faster contracting MUs tend to have lower axonal excitability threshold
compared with slower contracting MUs (35), it is tempting to suggest that the submaximal EMS used in the present study has mainly activated the fast-twitch MUs (see also 17, 20).

Despite the limited number of contractions (15 evoked contractions), the MVC torque loss (~18%) induced here by EMS is consistent with that reported in previously human quadriceps muscle (9, 14, 21, 24, 37, 41). In combination with this MVC torque loss, the decrease in potentiated peak twitch, the small but significant changes in maximal M-wave amplitude and area, and the unchanged VAL suggest that the mechanisms underlying the observed fatigue are due to the impairment of processes located in the muscle. Candidate mechanisms include altered muscle contractility related to Ca²⁺ kinetics beyond the membrane cell (2) and changes in muscle excitability linked to impaired Na⁺/K⁺ pump function (18).

Consistent with this interpretation, the reduction in twitch amplitude elicited at the stimulation intensity used during EMS was linearly related to the decrease in EMS-evoked torque (31). The decrease in twitch torque was accompanied by a shift in the torque-frequency relation. Our results demonstrate a torque decrease at all frequencies and a rightward shift in the torque-frequency relation after the fatiguing exercise. The decreased torque observed at the higher tested frequency (100 Hz) could be explained by 1) a reduced number of active cross-bridges and/or 2) a decreased in force produced by each one, whereas a rightward shift of the relation is more related to decreased myofilaments Ca²⁺ sensitivity (34, 36). These results indicate that physiological processes within the muscle are responsible for much of the fatigue induced by submaximal EMS and are in accordance with previous studies that have demonstrated that such exercise had a significant impact on the maximal torque-generating capacity of the subjects (11, 31, 32, 41).

The declines in EMS-elicited torque (~60%) and EMS twitch evoked during the fatiguing contraction (~62%) were greater than the reduction in MVC torque (~20%) and the potentiated twitch (~20%). Moreover, there was no correlation between the decreases in EMS-elicited torque and MVC torque or the maximal twitch evoked by nerve stimulation and the twitch elicited at muscle level at the stimulation intensity used during the EMS exercise. The lack of association between these different outcomes was likely due to the muscle mass that was activated, which was only 36% for EMS during the fatiguing contraction compared with 93% during the MVC.

**Fatiguing Contraction**

To estimate changes in the proportion of MUs activated during the fatiguing contraction, supramaximal stimulation was applied to the femoral nerve during (superimposed twitch) and after (posttetanic twitch) each EMS contraction. Superimposed and posttetanic twitches changed differently during the fatiguing contraction, the superimposed twitch was increased to ~120% of the initial value, whereas the posttetanic twitch decreased to ~90% of the 1st posttetanic twitch. As a consequence, the MAL demonstrated a significant decrease by ~60% from the start to the end of the fatiguing exercise, indicating that the number of MUs activated by EMS at the end of the exercise was reduced.

A possible mechanism to explain the opposing changes in the amplitudes of the evoked responses could be an increase in the excitability threshold of the motor axons due to repetitive activation. Trains of electrical stimuli can increase the excitability threshold of the motor axons due to repetitive activation. Trains of electrical stimuli can increase the excitability threshold of the motor axons due to repetitive activation. Trains of electrical stimuli can increase the excitability threshold of the motor axons due to repetitive activation. Trains of electrical stimuli can increase the excitability threshold of the motor axons due to repetitive activation. Trains of electrical stimuli can increase the excitability threshold of the motor axons due to repetitive activation.

**Fig. 5.** A linear correlation was found between EMS-evoked torque and posttetanic twitch (r = 0.98).

**Fig. 6.** Torque-frequency relation obtained by submaximal EMS obtained before and after fatiguing contractions. Data (means ± SE, n = 11) are presented as absolute (A) and relative (B; 100 Hz torque set to 100%) values. Filled circles, before fatigue; open circles, after fatigue.
ability threshold of motor axons at the site of stimulation (4, 12, 22, 23) as a result of axonal hyperpolarization (22, 23). However, these motor axons can be recruited by supramaximal nerve stimulation and thus contribute to an increased superimposed twitch. Such an interpretation is consistent with the greater decrease in posttetanic twitch amplitude elicited at the muscle level with the stimulation intensity used during the EMS exercise than that observed for the maximal twitch amplitude evoked by supramaximal nerve stimulation (~5.1 Nm vs. ~3.8 Nm).

However, another possible mechanism that may explain the divergent changes in the amplitude of the evoked responses could be the submaximal stimulus rate (50 Hz) used during EMS compared with the rate used for the superimposed twitch. Indeed, at the muscle level, superimposed nerve stimulation was elicited at 100 Hz (see MATERIALS AND METHODS). Considering the torque-frequency relation, the increase in the rate of stimulation from 50 to 100 Hz for the MUs that are both activated by EMS and supramaximal nerve stimulation leads to an increase in torque of ~15% before the EMS exercise. At the end of EMS, and due to the rightward shift in the torque-frequency relation, the difference in torque production between 50 and 100 Hz was ~22%. The ~7% difference (~0.9 Nm) explains only ~15% of the increase in superimposed twitch (~5.9 Nm) obtained at the end of the EMS-evoked fatigue. Thus the greater part of the MAL decrease (~85%) is likely due to activity-dependent changes in axonal excitability leading to a reduction in the number of MUs activated during the EMS exercise. This phenomenon, that reduced muscle activation from ~36% at the start to ~14% at the end of EMS, seems to be the main contributor to the torque decrease observed after the submaximal EMS exercise, thus explaining the greater declines in EMS-elicited torque (60%) compared with MVC torque (20%).

To conclude, the present study showed that the reduction in force produced by a submaximal EMS protocol (~60%) was accompanied by a lesser decrease in MVC torque (~18%). Despite a rightward shift in the torque-frequency relation, a reduction in the number of MUs activated due to changes in excitability threshold of motor axons is likely a major mechanism responsible for the greater torque loss during evoked contractions compared with voluntary contractions. These findings suggest that caution is required when interpreting decreases in EMS-elicited torque in terms of voluntary torque capacity. Indeed, changes in the excitability threshold of the active axons due to repetitive stimulation that lead to a decrease in the number of active MUs must be taken into account when comparing different EMS protocols, especially when stimulation parameters are modulated (width pulse, frequency).

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DISCLOSURES

No conflicts of interest, financial or otherwise, are declared by the authors.

AUTHOR CONTRIBUTIONS

B.M., R.L., and A.M. conception and design of research; B.M. and A.M. performed experiments; B.M. analyzed data; B.M., R.L., and A.M. interpreted results of experiments; B.M. prepared figures; B.M. and A.M. drafted manuscript; B.M., R.L., and A.M. edited and revised manuscript; B.M., R.L., and A.M. approved final version of manuscript.

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