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CONTRACTILE IMPAIRMENT AFTER QUADRICEPS STRENGTH TRAINING VIA ELECTRICAL STIMULATION

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ABSTRACT

Zory, RF, Jubeau, MM, and Maffiuletti, NA. Contractile impairment after quadriceps strength training via electrical stimulation. *J Strength Cond Res* 24(2): 458–464, 2010—The purpose of this study was to investigate the neural and muscular changes associated with electrical stimulation (ES) training and subsequent detraining. Twenty healthy active men were randomized to receive (intervention group) or not (control group) 4 weeks of ES strength training followed by 4 weeks of detraining. Quadriceps ES training sessions (20 minutes per session, 4 sessions per week) were completed under isometric loading conditions. Quadriceps maximal voluntary contraction (MVC) strength, activation level, maximal electromyographic (EMG) activity, and excitation-contraction coupling properties were assessed before training, after training, and after detraining. Maximal voluntary contraction strength did not change after training but significantly increased after detraining (+21.5%; $p < 0.05$). Activation level (+7.3%) and maximal EMG activity (+27.9%) increased significantly after training and remained elevated after detraining ($p < 0.05$). Vastus lateralis M-wave amplitude did not change during the study period, whereas quadriceps contractile properties were significantly impaired after training but then recovered to pre-training values after detraining. We conclude that the maximal force-generating capacity of the quadriceps was unchanged after 4 weeks of ES strength training because of the interplay between neural (increased activation) and muscular (contractile impairment) changes. On the other hand, recovered contractile function and preserved activation after 4 weeks of detraining resulted in significant MVC strength increases. Quadriceps strength training via ES may induce overreaching and delayed adaptations and therefore should be used with caution. These

findings may help in conceiving effective ES strength training programs for physically active subjects.

KEY WORDS knee extensors, muscle activation, excitation-contraction coupling, contractile properties

INTRODUCTION

Neuromuscular electrical stimulation (ES) is adopted in sports medicine and in rehabilitation settings as a complement and as a substitute to voluntary strength training modalities (8,14), in particular for the quadriceps femoris muscle. The typical ES settings involve the application of intermittent stimuli at frequencies eliciting fused tetani (>30–50 Hz), to maximize muscle tension and therefore to mimic strength training.

Short-term (3–4 weeks) programs of ES training could promote strength gains of up to ~30% for healthy quadriceps (15,16), which are generally accompanied by increased neural drive, as witnessed by twitch interpolation and surface electromyographic (EMG) activity results (6,19). Cross-education effect (9) and observations on the specificity of ES training effects (18) further confirmed the occurrence of neural adaptations to artificial strength training.

On the other hand, some lines of evidence suggest that ES strength training programs could result in transient peripheral impairment, probably linked to overreaching. For example, alterations in whole-muscle contractile properties (19), severe muscle injury (7), and delayed training effects (15,20) have been reported after multiple sessions of ES. More recently, it has been demonstrated that the acute application of electrical stimuli to elicit isometric quadriceps contractions could result in exaggerated muscle fatigue (13,26,30), muscle damage, and soreness (13), so that ES has been classified as an aggressive modality of activation from a muscular point of view (19).

In line with recent results obtained in our laboratory (19), we hypothesized antagonist influences of neural and muscular changes on maximal voluntary strength after short-term ES strength training. Specifically, it was hypothesized that the transient peripheral impairment induced by ES training would have masked the influence of increased neural drive on maximal voluntary quadriceps strength. Therefore, this study aimed to investigate the neural (muscle activation, EMG activity) and muscular (excitation-contraction

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coupling properties) changes associated with ES training for 4 weeks and consecutive detraining for further 4 weeks.

METHODS

Experimental Approach to the Problem

We conducted a randomized controlled trial to evaluate the effect of ES training and detraining (vs. no training) on maximal quadriceps strength and on the underlying neural and muscular adaptations. The independent variable was time or treatment, and the dependent variables were quadriceps maximal voluntary contraction (MVC) torque and neuromuscular characteristics (muscle activation, EMG activity, and excitation-contraction coupling properties). After a familiarization session and after signing the informed consent form, subjects were randomized (simple random sample) by computer to receive (intervention group) or not (control group) 4 weeks of ES strength training followed by 4 weeks of detraining. Subjects in the intervention group were tested 3 times: before training (3–4 days before the first session), after training (5–7 days after the last session), and, then again, after 4 weeks of detraining (i.e., no training). Control group subjects were tested and retested after 4 weeks to assess the reliability of the measurements. Due to scheduling difficulties, we did not include a third testing session for this group.

Subjects

Twenty healthy men without any known orthopaedic, neuromuscular, or cardiovascular problems volunteered to participate in this investigation. They were undergraduate and postgraduate physical education students in our department at the time of the experiment (spring 2003). All participants were moderately active (5–8 hours per week of physical activity, mainly in team sport activities at a recreational level) but not specifically trained for any one sport or engaged in systematic strength training (including ES). They were asked not to modify their usual lifestyle (including physical activity) during the study period. Age and anthropometric characteristics were similar between the intervention group ($n = 12$; age: 24 ± 3 years; height: 176 ± 4 cm; mass: 78 ± 6 kg) and the control group ($n = 8$; age: 25 ± 4 years; height: 177 ± 4 cm; mass: 75 ± 4 kg). Subjects were informed of the experimental risks and signed an informed consent document before the investigation. The investigation was approved by an institutional review board for use of human subjects.

Procedures

Electrical Stimulation Training. The unilateral ES resistance training program was completed under isometric loading conditions, with 4 sessions per week. Each training session lasted ~20 minutes and incorporated 40 stimulated intermittent contractions (time on: 6.25 seconds, with a rise phase of 1.5 seconds and a fall phase of 0.75 seconds; time off: 20 seconds). Subjects were seated on a leg extension machine (Multi-Form, La Roque d'Anthéron, France), with both hip and knee joints at $\sim 90^\circ$. The quadriceps muscle of the

nondominant (nonkicking) limb was stimulated using a portable device (Compex Sport-P; Medicompex SA, Ecublens, Switzerland) and three, 2-mm-thick, self-adhesive electrodes. Two small electrodes (5×5 cm) were placed as close as possible to the motor point of the vastus medialis and vastus lateralis muscles. One large electrode (10×5 cm) was placed ~ 10 cm below the femoral triangle. The stimulator discharged biphasic symmetric rectangular wave pulses (pulse duration: 400 μ s) at a frequency of 75 Hz. During the stimulation, subjects were consistently reminded to fully relax their quadriceps muscle. The stimulation parameters of the present study were selected according to the recommendations of several recent reviews on ES (8,10). Previous ES training studies completed in our laboratory have also confirmed the effectiveness of these stimulation characteristics for quadriceps muscle strengthening (6,16).

Each session was preceded by a standardized warm-up, consisting of 5 minutes of low-intensity quadriceps ES (frequency: 5 Hz; pulse duration: 350 μ s; self-selected and comfortable current level) and 2 quadriceps MVCs. Then, subjects were asked to progressively increase current amplitude (range: 0–120 mA) during 5–8 contractions, until a maximal tolerated level (pain threshold) was reached. Maximal current amplitude increased regularly throughout the training program (first session: 58 ± 14 mA; eighth session: 90 ± 21 mA; last session: 106 ± 21 mA). The level of electrically evoked isometric force was measured by means of a strain gauge transducer mounted on the leg extension machine (Allegro, Sallanches, France) and was generally in the order of 45% of the MVC of the day (first session: $43 \pm 15\%$ MVC; eighth session: $49 \pm 11\%$ MVC; last session: $42 \pm 13\%$ MVC). Each training session was supervised by the same experimenter (R.Z.).

Neuromuscular Assessments. The testing session included a series of electrically evoked contractions (via femoral nerve stimulation) and MVC of the nondominant quadriceps (i.e., the trained muscle for the intervention group), with concomitant recording of vastus lateralis EMG activity and quadriceps torque output. Individual ergometer adjustments and EMG and stimulating electrodes positioning were consistently recorded and reproduced during the different testing sessions. The present experimental procedures are commonly adopted in our laboratory for the study of neural (muscle activation, EMG activity) and muscular (excitation–contraction coupling) properties of the quadriceps muscles. Both intra- and intersession reliability have been shown to be very good (6,17,21).

All the contractions were performed under isometric conditions by means of an isokinetic device (Biodex; Shirley Corporation, New York, NY, USA). The subject was seated on the dynamometer chair with 90° at the knee and hip joints. To minimize hip, trunk, and thigh motion during the contractions and therefore to avoid the contribution of muscles other than the quadriceps, straps were applied across

TABLE. Dependent variables before and after the 4-week period in the control group ($n = 8$).^{*†}

	Before	After
MVC torque (N·m)	222.5 ± 9.8	232.4 ± 14.5
Activation level (%)	94.2 ± 1.4	93.0 ± 1.5
Normalized EMG activity	0.048 ± 0.006	0.055 ± 0.005
M-wave amplitude (mV)	10.0 ± 1.1	9.6 ± 1.4
Doublet peak torque (N·m)	101.2 ± 6.4	105.1 ± 6.0
Postactivation potentiation (%)	111.7 ± 3.0	111.7 ± 2.6

*Mean values ± SEM.

†EMG = electromyographic; MVC = maximal voluntary contraction.

the chest, pelvis, and mid thigh. The arms were positioned across the chest with each hand clasping the opposite shoulder. A strap also secured the leg of the subject to the ergometer arm lever, and the alignment between the center of rotation of the dynamometer shaft and the axis of the knee joint was checked at the beginning of each trial.

Before each testing session, subjects warmed up the quadriceps muscle by performing 5 minutes of low-frequency ES (see ES training). Then, 10–15 submaximal isometric contractions were completed before determining the optimal intensity for single and paired stimuli. The femoral nerve was stimulated using a cathode ball electrode (diameter: 0.5 cm) manually pressed in the femoral triangle, 3–5 cm below the inguinal ligament. The anode was a large electrode (10 × 5 cm) located in the gluteal fold. Rectangular pulses (duration: 1 milliseconds) were delivered by a constant-current high-voltage stimulation unit (Digitimer DS7A, Digitimer Ltd,

Hertfordshire, United Kingdom). Stimulation intensity was progressively increased by 10 mA increments until there was no further increase in quadriceps twitch torque and vastus lateralis M-wave amplitude. This intensity was further increased by 10% (i.e., supra-maximal) and then maintained for single and paired stimulations (range: 40–90 mA). Recordings started with 3 single pulses, each separated by 3 seconds, and 3 paired stimuli (interval: 10 milliseconds), each separated by 4 seconds, during which subjects were asked to relax. Then, subjects were instructed to produce their maximal quadriceps force for ~5 seconds without any concern for the rate of force development, to evaluate quadriceps MVC torque (3 trials). Paired stimuli were also delivered over the isometric plateau of each MVC (superimposed doublet) and 1.5 seconds after each MVC (potentiated doublet), to assess the level of voluntary activation according to the twitch interpolation technique. A 2-minute rest period was allowed between MVCs to minimize the effects of fatigue.

The EMG signal from the vastus lateralis muscle was recorded with silver chloride circular bipolar electrodes. The electrodes, having a recording diameter of 10 mm, were fixed lengthwise over the middle of the muscle belly with an interelectrode (center to center) distance of 20 mm. The reference electrode was attached to the wrist. Low resistance between the 2 electrodes was obtained (<5 kΩ) by abrading the skin with emery paper and cleaning with alcohol-ether-acetone mix. Electromyographic signals were amplified with a bandwidth frequency ranging from 15 Hz to 5 kHz (common mode rejection ratio: 90 dB; impedance: 100 MΩ; gain: 1000).

Electromyographic and torque traces were digitized online (sampling frequency: 2024 Hz), and stored for analysis with a commercially available software (Tida; Heka Elektronik, Lambrecht/Pfalz, Germany). Peak-to-peak amplitude of the vastus lateralis M-wave and peak torque of the potentiated and unpotentiated doublets

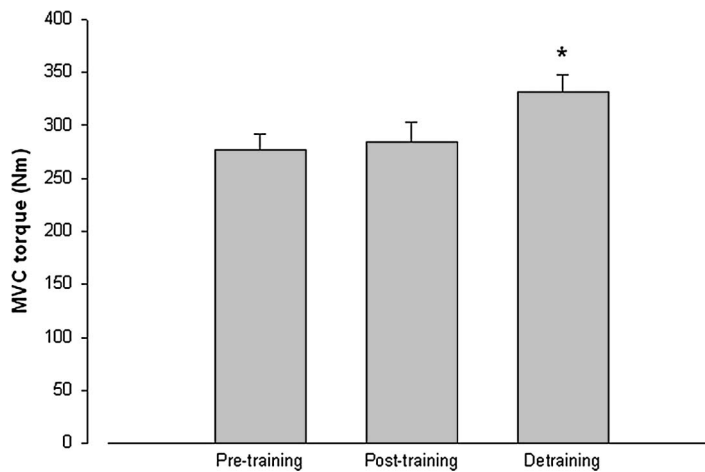


Figure 1. Quadriceps maximal voluntary contraction (MVC) torque before training (pre training), after training (post training), and detraining in the intervention group ($n = 12$). Mean values and SEM. *Detraining values are higher than pretraining and posttraining at $p < 0.05$.

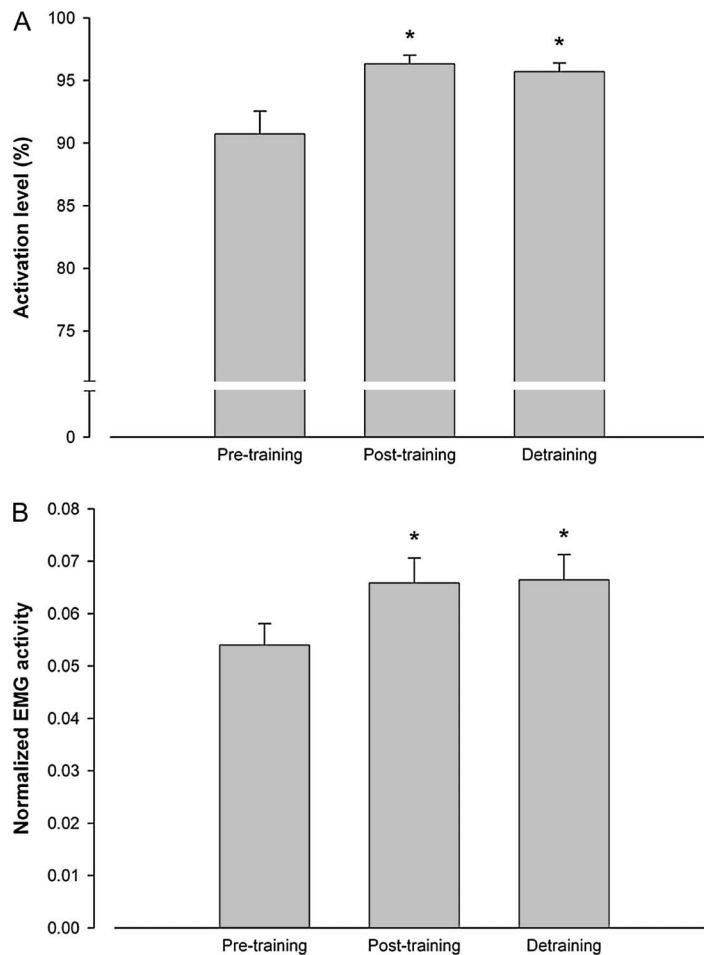


Figure 2. Quadriceps muscle activation level (A) and vastus lateralis normalized electromyographic (EMG) activity (B) before training (pre training), after training (post training), and detraining in the intervention group ($n = 12$). Mean values and *SEM*. *Posttraining and detraining values are higher than pretraining at $p < 0.05$.

and detraining). If significant main time effect was observed, Tukey post hoc tests were conducted. Within the control group, dependent variables obtained before and after the 4-week period were compared using paired Student's *t*-tests, and the associated intraclass correlation coefficients (model 2,1) ranged from 0.754 to 0.911. Between-group differences at baseline were analyzed using unpaired Student's *t*-tests. For all the procedures, alpha was set at 0.05. Data are presented as mean and *SEM*.

RESULTS

At baseline, the only between-group difference observed was for MVC torque, which was lower in control than in ES subjects ($p < 0.05$). In the control group (Table), no significant changes over time were observed for the ensemble of the dependent variables.

Maximal Voluntary Contraction Torque

Maximal voluntary contraction torque was unchanged after the ES training program, whereas a significant increase was observed after the detraining period (Figure 1). The percent increase from baseline to detraining was $21.5 \pm 5.1\%$.

were determined. The root mean square EMG amplitude of vastus lateralis muscle was calculated over a 500-millisecond period around the MVC torque and subsequently normalized to the amplitude of the M-wave of the session (6,17,30). Activation level was estimated according to the following formula: $(1 - \text{superimposed doublet} \times \text{potentiated doublet}^{-1}) \times 100$. Postactivation potentiation was computed as: $(\text{peak torque of the potentiated doublet} \times \text{peak torque of the unpotentiated doublet}^{-1}) \times 100$. All data were considered as the average of 3 trials.

Statistical Analyses

Within the intervention group, dependent variables (MVC torque, activation level, normalized EMG activity, M-wave amplitude, potentiated doublet peak torque and postactivation potentiation) were analyzed with a 1-way analysis of variance with repeated measures (pretraining, posttraining,

Neural Changes

Both activation level (Figure 2A) and normalized EMG activity of vastus lateralis (Figure 2B) significantly increased after ES training, by $7.3 \pm 2.0\%$ and $27.9 \pm 12.9\%$, respectively. These increases were maintained after detraining, as witnessed by the significant difference between pretraining and detraining values for both activation level and normalized EMG activity.

Muscular Changes

Vastus lateralis M-wave amplitude did not change significantly during the experimental period (pretraining: 11.3 ± 1.2 mV; posttraining: 11.0 ± 0.7 mV; detraining: 12.2 ± 1.0 mV). On the other hand, both doublet peak torque (Figure 3A) and postactivation potentiation (Figure 3B) decreased significantly

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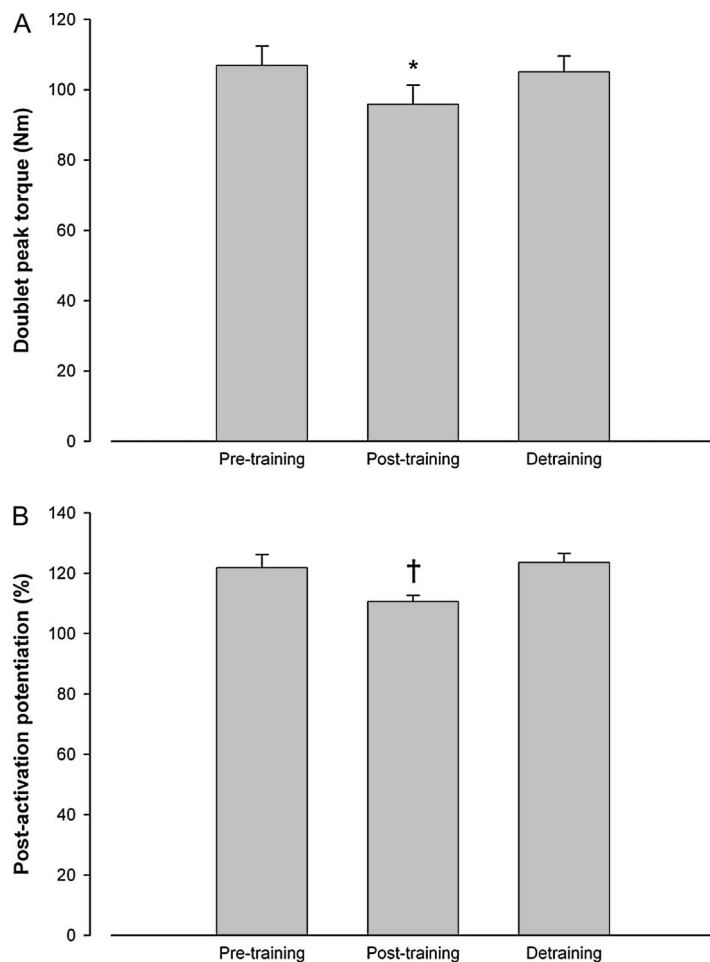


Figure 3. Quadriceps doublet peak torque (A) and postactivation potentiation (B) before training (pre training), after training (post training), and detraining in the intervention group ($n = 12$). Mean values and SEM. *Posttraining values are lower than pretraining at $p < 0.05$. †Posttraining values are lower than pretraining and detraining at $p < 0.05$.

after training, by $-8.3 \pm 4.2\%$ and $-8.5 \pm 2.5\%$, respectively, and then recovered to baseline values after detraining. In addition, postactivation potentiation after training was lower than after detraining ($p < 0.05$).

DISCUSSION

The key findings of this study were that quadriceps MVC strength was unchanged after a short-term ES training program because of the interplay between neural (increased activation) and muscular (contractile impairment) changes. On the other hand, the recovery of contractile properties and the preservation of muscle activation after 4 weeks of detraining resulted in significant quadriceps MVC strength gains.

The significant increase in quadriceps muscle activation after ES strength training, as witnessed by EMG and twitch

interpolation results, is not a new finding. Others and (6,25) we (11,18,19) already reported that multiple sessions of ES significantly increased the root mean square amplitude of the interference EMG signal and reduced the superimposed torque amplitude during MVC. These changes are usually ascribed to increased spatial recruitment or firing rate of motor units and could occur at different levels of the neuraxis. Electrical stimulation strength training has long been believed to promote muscular adaptations only because of the peripheral origin of the stimulated contraction. However, the central effects of ES—both acute and chronic—have been increasingly acknowledged in the last few years. A series of fatigue (1) and training studies (9,11) have provided experimental evidence that ES could evoke widespread activity within the central nervous system that is capable of mediating a range of acute and chronic neural adaptations. The findings of the present study add support to this concept. In trying to understand the mechanisms underlying ES training-induced neural adaptations, researchers should consider that ES—which activates intramus-

cular nerve branches and not the muscle fibres directly—could also activate selected brain regions in a dose-response manner (23) and spinal motoneurons via reflex inputs to the spinal cord (3), so that the torque evoked by ES results from both peripheral and central contributions.

Besides neural adaptations, for the first time, we demonstrated that ES strength training can result in peripheral impairment as witnessed by the decreased doublet peak torque and postactivation potentiation, without changes in compound muscle action potential amplitude, that is, contractile or excitation-contraction coupling failure. Interestingly, such contractile failure was only transient and moderate (i.e., a sort of overreaching effect) because doublet peak torque and postactivation potentiation recovered to baseline levels after 4 weeks of detraining. Before undertaking the present investigation, several lines of evidence suggested

the possibility of ES exercise-induced peripheral impairment, which were used to construct our hypothesis. First, acute studies showed exaggerated muscle fatigue (13,26,30) and muscle damage/soreness (4,13) after one single bout of ES exercise. Second, chronic application of ES exercise was associated to severe muscle injury, that is, rhabdomyolysis (7) and delayed training effects on neuromuscular function in healthy individuals (11,15,20). Third, using the same ES training paradigm adopted in this study, we recently demonstrated significant alterations in quadriceps muscle contractile properties, which were associated to the appearance of neonatal myosin heavy chains in one subject (19). This phenomenon was seen as an indicator of regeneration (29), probably subsequent to ES training-induced muscle damage.

One single bout of quadriceps ES exercise—as used in this present study—has been demonstrated to induce significant muscle fatigue (~20% MVC torque loss) that was mainly explained by neuromuscular transmission failure (~17% depression of M-wave amplitude), whereas excitation-contraction coupling was not or little influenced (30), that is, high-frequency fatigue (see also 1)). Such effects are generally observed immediately after (0–45 minutes post exercise) the completion of an ES exercise session, as confirmed by previous quadriceps ES studies performed in our laboratory (13,26,30). On the other hand, considerable muscle damage and soreness have been observed 48–72 hours after quadriceps ES (2,4,13), which were accompanied by a significant decline in the 20 to 100 Hz tetanic force ratio (i.e., low-frequency fatigue) (2). Interestingly, low-frequency fatigue has been suggested to be caused by muscle fiber damage or impairment in the excitation-contraction coupling mechanism (12). It seems therefore that the application of ES could result in 2 different kinds of acute adjustments: the first—occurring immediately after the completion of exercise—that affects one or more of the processes involved in converting the axonal action potential into a sarcolemmal action potential. The second—taking place in a few days after the exercise bout—that impairs some steps of the excitation-contraction coupling beyond membrane ionic processes.

These physiological considerations are in harmony with the findings of the present ES training study. Indeed, we observed transient alterations in contractile properties (doublet torque and postactivation potentiation) without changes in the M-wave, therefore providing experimental evidence that recovery of excitation-contraction coupling process after multiple ES exercise sessions can be quite slow, requiring several days to be completed. Although low-to-high frequency ratio was not experimentally assessed in our investigation, we are justified in assuming that muscle damage (see above) and excitation-contraction coupling failure played a dominant role in low-frequency fatigue occurrence. Indeed, the originally described low-frequency fatigue can be produced by repeated high-frequency contractions (28),

a phenomenon termed “delayed low-frequency recovery” by Westerblad and Allen (28). It remains to be investigated in future studies whether excitation-contraction coupling impairment is associated to low-frequency fatigue and muscle damage after single and repeated ES exercise bouts.

Impairments in excitation-contraction coupling that have been suggested to contribute to low-frequency fatigue include a reduction of calcium release or reuptake by the sarcoplasmic reticulum or a decrease in the calcium sensitivity of troponin (14). The ~8% decrease in doublet peak torque was associated with significant alterations in the maximal rate of doublet torque contraction and relaxation (data not presented), therefore confirming that phasic cytosolic calcium movements during contraction played a role. Moreover, because postactivation potentiation changes seem to be more closely related to the sensitivity of contractile protein to calcium (27), our results taken as a whole indicate that different intracellular calcium-controlled processes were affected by the ES training program. Whatever the exact physiological mechanisms, contractile failure was not observed after 4 weeks of detraining and this—together with the neural adaptations previously discussed—certainly contributed to the significant MVC strength improvement.

PRACTICAL APPLICATIONS

A short-term ES training program for quadriceps muscle strengthening may induce overreaching and delayed adaptations in healthy active men. The observed peripheral impairment confirms that the present ES modality (high frequency and high force) represents a very strong stimulus for musculoskeletal structures. In the context of sport training, our findings indicate that ES intervention programs should be carefully supervised and implemented early in the season. As previously suggested by our laboratory, a detraining period with no ES lasting 10 days (20) to 1 month (this study) should follow an ES program before beneficial effects on muscle performance could be optimized. We confirm that ES is an effective modality for increasing strength of unimpaired quadriceps muscle. However, because the present ES modality is largely applied to impaired muscles, for example, spinal cord injury individuals and orthopedic and cardiorespiratory patients (5,22,24), the contractile impairment reported in our study should caution against the use of high-frequency and high-force ES with these frail populations. Further randomized-controlled studies are required to determine the effectiveness of ES vs. voluntary strength training programs for unimpaired and impaired muscles and for muscle groups other than the quadriceps.

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