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Muscle fiber conduction velocity is more affected after eccentric than concentric exercise

Harri Piitulainen · Alberto Botter · Roberto Merletti · Janne Avela

Abstract It has been shown that mean muscle fiber conduction velocity (CV) can be acutely impaired after eccentric exercise. However, it is not known whether this applies to other exercise modes. Therefore, the purpose of this experiment was to compare the effects of eccentric and concentric exercises on CV, and amplitude and frequency content of surface electromyography (sEMG) signals up to 24 h post-exercise. Multichannel sEMG signals were recorded from biceps brachii muscle of the exercised arm during isometric maximal voluntary contraction (MVC) and electrically evoked contractions induced by motor-point stimulation before, immediately after and 2 h after maximal eccentric (ECC group, $N = 12$) and concentric (CON group, $N = 12$) elbow flexor exercises. Isometric MVC decreased in CON by $21.7 \pm 12.0\%$ ($\pm$SD, $p < 0.01$) and by $30.0 \pm 17.7\%$ ($p < 0.001$) in ECC immediately post-exercise when compared to baseline. At 2 h post-exercise, ECC showed a reduction in isometric MVC by $24.7 \pm 13.7\%$ ($p < 0.01$) when compared to baseline, while no significant reduction (by $8.0 \pm 17.0\%$, ns) was observed in CON. Similarly, reduction in CV was observed only in ECC both during the isometric MVC (from baseline of 4.16 ± 0.3 to 3.43 ± 0.4 m/s, $p < 0.001$) and the electrically evoked contractions (from baseline of 4.33 ± 0.4 to 3.82 ± 0.3 m/s, $p < 0.001$). In conclusion, eccentric exercise can induce a greater and more prolonged reduction in muscle force production capability and CV than concentric exercise.

Keywords Electrical stimulation · Exercise-induced muscle damage · Sarcolemma · M-wave · Muscle fatigue

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Introduction

It is well established that repetitive eccentric muscle contractions cause exercise-induced muscle damage (EIMD), which is characterized by delayed muscle pain (Armstrong 1984; Jones et al. 1989), increased passive muscle stiffness (Stauber et al. 1990), muscle swelling (Clarkson et al. 1992) and muscle protein efflux to circulation (Clarkson et al. 1986; McNeil and Khakee 1992). It has been shown that, EIMD is associated with morphological changes such as disruption and disorganization of sarcomeres (Newham et al. 1983; Vijayan et al. 2001), sarcolemma (Jones et al. 1986; Lieber and Friden 1988) and transverse tubular system (Corona et al. 2010; Takekura et al. 2001). Most important functional consequence related to EIMD is a prolonged reduction in maximal muscle force production (Davies and White 1981).

Partly because of the complex, possibly multi factorial nature of EIMD, the detailed mechanisms responsible for the loss of force production capability have remained unclear. However, it appears that the primary mechanism involves failure in the excitation–contraction (E-C) coupling process. Based on animal experiments, the failure in the E-C coupling occurs in the events prior to the release of Ca^{2+} from the sarcoplasmic reticulum (SR), such as depolarization of transverse tubular system, but after action

potential conduction over the sarcolemma (Warren et al. 1999). Nevertheless, experiments with humans have shown an acute (≤2 h) reduction in mean muscle fiber conduction velocity (CV) during both maximal voluntary and electrically elicited contractions after eccentric exercise (Piitulainen et al. 2010). Therefore the impaired sarcolemmal action potential conduction could partly be related to the impaired E-C coupling and consequent force loss in the early stage of EIMD. It has been suggested that the initial events in EIMD are caused by mechanical disruption of cellular structures in muscle fibers (Morgan and Allen 1999), activation of stretch sensitive ion channels (McBride et al. 2000; Stauber 1989) and activation of proteolytic pathways (Belcastro et al. 1998). Nevertheless, any ruptures of the excitable membrane systems in the muscle fiber may affect ion concentrations and flow over the sarcolemma, and thus its excitability, and hinder the E-C coupling process and muscle force capacity.

The sensitivity of skeletal muscle to eccentric contractions could be related to higher force production (Katz 1939) with less muscle activation (Bigland and Lippold 1954; Westing et al. 1991) and thus higher mechanical stress to a smaller fiber area compared to concentric contractions. Therefore, it can be expected that sarcolemmal function may be more affected during maximal eccentric exercise as compared to the concentric one. Sarcolemmal function can be estimated in humans with multichannel surface electromyography (sEMG) during voluntary or electrically elicited contractions by measuring exercise-induced changes in sEMG variables, such as CV (Masuda and Sadoyama 1986), signal amplitude and its spectral content (Merletti et al. 1990).

Although eccentric exercise has been shown to impair sarcolemmal action potential conduction (Piitulainen et al. 2010) and sarcolemmal excitability (Piitulainen et al. 2008) in humans at the early stage (≤2 h post-exercise) of the progression of EIMD, no prolonged impairment (>2 h post-exercise) has been detected. In addition, applicability of the previous findings to exercises with other contraction types has not been evaluated in the previous experiments. For this reason, to further examine the mechanisms of EIMD and especially the importance of mechanical nature of the exercise on sarcolemmal action potential propagation, a comparison between eccentric and concentric exercises is required. Therefore, the aim of the present experiment was to examine whether the acute impairment of sarcolemmal function is limited only to repeated eccentric contractions or if exercise with concentric contractions will result in similar acute effects on the sarcolemmal action potential conduction and sarcolemmal excitability. To distinguish between possible central fatigue and peripheral muscle fatigue, the investigations were conducted both in voluntary and electrically evoked conditions for the biceps brachii muscle.

Methods

Subjects

Twenty-four healthy male volunteers participated in the experiment. Based on previous experience from our laboratory, this was adequate number of subjects to obtain statistical power of 80% (type one error $\alpha = 0.05$) for sEMG variables and markers of EIMD. The subjects were divided into weight and height matched pairs and were then randomly assigned either to concentric (CON, $N = 12$, age 27.2 ± 4.2 years, weight 78.9 ± 14.0 kg, height 182.9 ± 6.5 cm and subcutaneous tissue thickness 2.39 ± 1.12 mm) or eccentric (ECC, $N = 12$, age 27.8 ± 3.8 years, weight 80.2 ± 11.6 kg, height 182.6 ± 6.6 cm and subcutaneous tissue thickness 2.52 ± 1.02 mm) exercise groups. All subjects were healthy, right handed, non-smokers, did not drink caffeine-rich drinks within 12 h of the measurements, and avoided strenuous exercise for 2 days before the first measurement and throughout the whole experimental period. The subjects were not active competing athletes, but were physically active individuals and had some experience from strength training.

The experiment was conducted in accordance with the Declaration of Helsinki and approved by the Ethical Committee of the University of Jyväskylä. All participants were aware of the possible risks of the experiment, and provided written informed consent before inclusion.

Experimental protocol

The study consisted of three primary sessions of identical measurements: (1) before the exercise, (2) immediately after (<15 min) the exercise and (3) 2 h after the exercise (Fig. 1c). The 2 h time-point was chosen to ascertain recovery of immediate energy stores and muscle temperature back to baseline levels. Besides the primary sessions, additional tetanic motor-point stimulation was conducted 30 min after cessation of the exercise (Fig. 1c), since a late depression in sarcolemmal excitability has been observed at this time frame (McFadden and McComas 1996). Furthermore, a follow-up for subjective perceived muscle soreness was continued daily up to 7 days (before, immediately after, 2 h after and 1–7 days after the exercises) post-exercise. Moreover, venous blood samples were collected before, immediately after (<1 min), 2 h after and 1 day after the exercises.

The three primary sessions (before, immediately after and 2 h after the exercises) involved measurements in the following order: (1) collection of venous blood sample, (2) evaluation of subjective perceived muscle soreness, (3) recording of train of maximal M-waves during the monopolar motor-point stimulation and (4) tests for

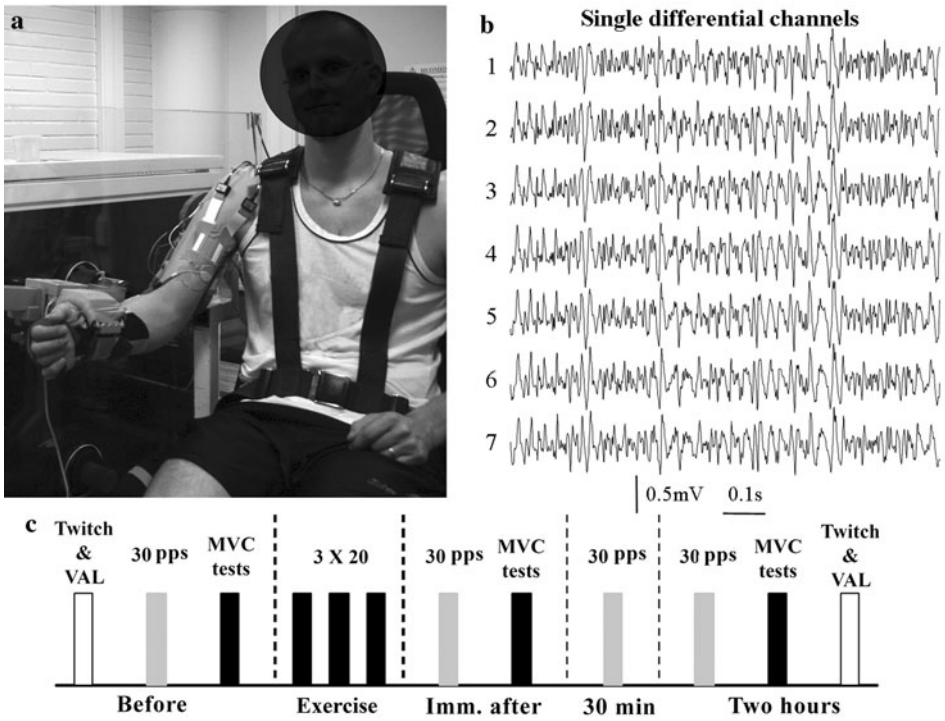

Fig. 1 **a** Measurement setup. **b** Raw signals during isometric MVC from the short head of the biceps brachii muscle in a single subject. **c** Schematic presentation of experimental protocol. Functional measurements included evaluation of passive twitch forces, voluntary activation level (*VAL*), monopolar motor-point stimulation (30 pulses per second) and maximal voluntary contraction (*MVC*) tests, before the exercise and immediately after, 30 min after and 2 h after exercises

isometric, concentric and eccentric maximal voluntary contractions (MVC; Fig. 1c). Additional measurements were conducted at the beginning (baseline) and at the end of the experiment (2 h post-exercise). These measurements included evaluation of (1) muscle and subcutaneous tissue thickness and (2) passive twitch force and superimposed twitch force during transcutaneous bipolar muscle stimulation (Fig. 1c).

Exercise protocol

Subjects performed either three sets of 20 maximal concentric or three sets of 20 maximal eccentric contractions with the elbow flexors of the right arm, on a motorized isokinetic dynamometer (angular velocity of 60°/s) (Komi et al. 2000). The range of motion at the elbow joint was 110°, between 65° and 175° (180° corresponds to full extension). The maximal contractions were performed with 15-s intervals and with maximal isometric pre-activation starting 1 s prior to the onset of the movement, giving a total duration of 2.8 s for each contraction. The exercise protocol was designed with sufficient rest periods between consecutive contractions and the three sets to avoid excessive metabolic loading and increase in muscle temperature.

During the exercises the subjects were seated and their half-supinated right forearm was attached to a strain gauge transducer, which was fixed to a lever arm (moving in the vertical plane) of the dynamometer to record the force applied by the elbow flexors (Fig. 1a). While the lever arm returned to the initial position (at an angular velocity of 60°/s), subjects were instructed to relax their arm muscles. The exercise lasted for 19 min with a total work time of 2 min and 50 s.

Force measurements

All elbow flexor forces were measured with the same device as the exercise was conducted with. The chair dimensions were individually adjusted for each subject, and in the case of isometric contractions an arm support was used to set the elbow angle to 120°. The highest value of three MVC trials (with 2-min rest periods in between) with less than a 5% difference from the second highest value was accepted as the true MVC value in each MVC test separately (isometric MVC, concentric MVC and eccentric MVC conducted in randomized order), and was used for further analysis. The concentric and eccentric MVCs were conducted similar to the concentric and eccentric exercise (see "Exercise protocol") and the MVC force was determined as average force between the elbow joint angles of 90° and 150°.

All force signals included in the analysis were sampled at 2,048 samples/s, converted to digital data by a 12-bit analog to digital converter (through the auxiliary inputs of EMG-USB 128 channel surface EMG amplifier, designed

by LISiN at Politecnico di Torino and manufactured by OT Bioelectronica, Torino, Italy) and stored on a computer hard disk along with the bipolar EMG signals. The force signals were also displayed in real-time on a computer screen (Signal software, CED Ltd.) as feedback to the subjects.

Electrical stimulation

A constant current stimulator (Digitimer Stimulator DS7, Digitimer Ltd., Hertfordshire, England) was used to stimulate the biceps brachii muscle with monophasic rectangular double pulses (100 μs duration, with 10 ms interpulse intervals) to measure (1) passive (relaxed muscle) twitch force and (2) superimposed (over MVC) twitch force of the elbow flexors (baseline and 2 h post-exercise). The same stimulator was used to stimulate the short head of the biceps brachii muscle with supramaximal monopolar motor-point stimulation, using a 30 s train of monophasic rectangular pulses (100 μs duration, 30 pulses per second stimulation frequency) to measure maximal M-wave properties (baseline, immediately after, 30 min after and 2 h after the exercises; Fig. 1c).

The isometric MVC trial with the superimposed twitch was used to calculate voluntary activation level based on the principle introduced by Merton (1954). Two (anode and cathode) adhesive electrodes (5 × 5 cm; V-Trodes, Metler Electronics corp., Anaheim, CA, USA) were placed over the proximal and distal ends of the biceps brachii muscle, and their positions were marked on the skin for accurate replacement at the 2 h post-exercise session. In voluntary activation level test, supramaximal double pulse electrical stimulation intensity was determined as the current level that was 30% above the level needed to elicit maximal passive twitch force in the first measurement session. During a single voluntary activation level test, two double pulse stimulations were applied: (1) during isometric MVC and (2) 3 s after relaxation from the isometric MVC. Voluntary activation level was calculated with the following formula: voluntary activation level (%) = (1 − ST/PT) × 100 (Babault et al. 2001), where ST = superimposed twitch amplitude (1st stimulation) and PT = post-activity potentiated passive twitch amplitude (2nd stimulation). The peak force of the second passive double stimulation was used to describe contractile properties of the biceps brachii muscle.

Monopolar motor-point stimulation was used to obtain information about maximal M-wave properties. First, the locations of the main motor points of short head of biceps brachii muscle were identified by scanning the muscle's surface with a ball pointed (cathode, 8 mm diameter) pen electrode while a large round (anode, diameter 7 cm) adhesive electrode (V-Trode, Mettler Electronics corp., Anaheim, CA, USA) was placed on the opposite side of the upper arm. The main muscle motor point was defined as the location of the negative electrode yielding the strongest mechanical response with the lowest pulse amplitude. After this, a round (diameter 3.2 cm) negative adhesive electrode (V-Trode) was placed over the main motor point. M-waves were detected with an 8-electrode array (see description below). The M-waves were monitored as the muscle was stimulated at two pulses per second with monophasic square pulses (duration of 100 μs) of increasing current intensity. The maximal current level was identified as the stimulation current yielding the maximal M-wave amplitude (no clear increment of peak-to-peak amplitude was evident when the current level was further increased). The stimulation intensity was set at 20% above the maximal current level. Electrical stimulation was then applied for 30 s with a frequency of 30 pulses per second.

Surface electromyography recordings

Surface electromyography (sEMG) signals were detected from short head of the biceps brachii muscle during isometric MVC and monopolar motor-point stimulation (analysis of M-wave properties) with a semi-disposable linear array consisting of 8-electrodes each (5 mm inter-electrode distance, model ELSCH008, designed by LISiN, Politecnico di Torino and manufactured by Spes-Medica, Battipaglia, Italy) in a single differential configuration (Fig. 1b). In addition, sEMG signals were recorded from the triceps brachii and brachioradialis muscles with bipolar surface electrodes (diameter 6 mm, inter-electrode distance 20 mm; Blue Sensor N-00-S/25, Medicotest, Olstykke, Denmark).

The subjects' main innervation zone was located individually prior to the 8-electrode array placement. This was done using a linear array of 16 electrodes (silver bar electrodes with 5 mm inter-electrode distance, LISiN, Politecno di Torino). During this search for the optimal location for the 8-electrode array, sEMG signals were recorded and displayed online on a computer screen while the subject produced submaximal isometric contraction of the elbow flexors. This was repeated from various parts of the short head of biceps brachii muscle until the sites with clear muscle fiber action potential propagation and main innervation zones were identified. The array was then placed on the optimal part of muscle parallel to its fibers, either proximally or distally from the main innervation zone location, depending on anatomical features of the subject.

Before the placement of the sEMG electrodes, the skin was lightly abraded and cleaned with alcohol. The 8-electrode array was attached to the skin using double

adhesive foam (1 mm thick), which included cavities under the electrodes between the skin and the electrode surface. To assure proper electrode–skin contact, each of the cavities was filled with 25 µl of conductive electrolyte gel (Spes-Medica, Battipaglia, Italy) with a pipette (Finnpipette 4540, Thermo Fisher Scientific, Inc., Waltham, MA, USA). The reference electrode (NI-4560, Ag/AgCl, Unomedical Ltd., Gloucestershire, Great Britain) was placed over the ipsilateral acromion of the scapula.

sEMG signals were amplified (128-channel surface EMG amplifier, EMG-USB, LISiN, OT Bioelettronica, Torino, Italy), band-pass filtered (3-dB bandwidth, 10–750 Hz), sampled at 2,048 samples/s per channel, converted to digital data (12 bit A/D converter), displayed in real time, and stored on the disk of a personal computer. In order to synchronize the stimulation and the M-wave acquisition, the trigger signal of the stimulator was sampled at 2,048 samples/s per channel, digitalized, and stored.

Surface EMG signal processing

All force and sEMG signals were analyzed with Matlab software (R2007a, ver. 7.4.0.287, The MathWorks Inc., MA, USA). Before any further calculations, the sEMG signals were digitally band-pass filtered (20–450 Hz, 4th order Butterworth filter) and the force signals were low pass filtered with a cut-off frequency of 20 Hz (4th order Butterworth filter).

Analysis of isometric MVC

Root mean square (RMS) was calculated separately for each bipolar channel both in the 8-electrode array and the conventional bipolar electrodes placed on triceps brachii and brachioradialis. Similarly, mean frequency of the power spectral density (MNF) was calculated for the array. Mean muscle fiber conduction velocity (CV) was estimated for the 8-electrode array with a method introduced by McGill and Dorfman (1984) based on three adjacent single differential channels (one triplet) in the longitudinal direction of the biceps brachii muscle. Channels with high interference and triplets with CV values beyond the physiological range (2–6 m/s) were excluded. In the case of 8-electrode array the values of RMS, MNF and CV were averaged among all the accepted channels or triplets (for CV estimation). The calculations were done within a fixed 1,000 ms epoch corresponding to the signal epoch with the highest force value.

Analysis of motor-point stimulation

For each single differential channel of the 8-electrode array, the 30 electrically elicited responses (M-waves) corresponding to each epoch of 1 s were averaged thus obtaining a sequence of 30 averaged M-waves during the 30-s contraction (Fig. 2). Thereafter, a sequence of 30 sEMG variable estimates (RMS, MNF and CV) was calculated from each available triplet, defined as a group of three single differential signals provided by adjacent electrodes. This was done because the signal to noise ratio increases by the square root of the number of M-waves averaged. Since for all the signals the stimulation artifact was almost completely separated from the M-wave, it was removed by offline blanking with average blanking window of 3 ms.

sEMG estimates used for further analysis were obtained from the best of the five triplets. The best triplet was determined to be the one showing the highest cross-correlation coefficient between its single differential signals (in any case greater than 0.70). RMS and MNF were computed as the average of the estimates of the two channels of the selected triplet.

For all subjects the time course of the sEMG variables showed a linear pattern for the first 10 s, therefore the first 10 sEMG variable estimations were fitted with a least mean square regression line whose intercept with the *Y* axis (at time = 0) was defined as the initial value. The slope of the regression line was used as an estimate of the rate of change over time and was adopted to assess myoelectric manifestations of fatigue.

Muscle soreness, muscle thickness and skin temperature

Subjective perceived muscle soreness of the right elbow flexors was assessed in each measurement session (baseline, immediately after, 2 h after and 1 day after the exercises) and was thereafter followed with self-assessment on a daily basis up to 7 days post-exercise. The assessment involved the use of a visual analog scale with a continuous line starting from 0 cm (no pain) and ending at 5 cm (worst possible pain) (Nosaka and Clarkson 1996). Each subject marked their subjectively perceived muscle soreness level on this line. The subjects were instructed and trained to perform a self-assessment of the muscle soreness during voluntary muscle activity over full range of motion in the elbow joint and during light palpation of the biceps brachii muscle.

Subcutaneous tissue thickness over short head of biceps brachii muscle and muscle thickness of the short head of biceps brachii muscle and brachialis muscle were measured with ultrasonography (model SSD-5500, Aloka; Tokyo, Japan) before and 2 h after exercise. The probe was positioned along the midline of the muscle, 2 cm proximal to the distal tendon. The probe position was marked on the skin in the first measurement. During the measurement,

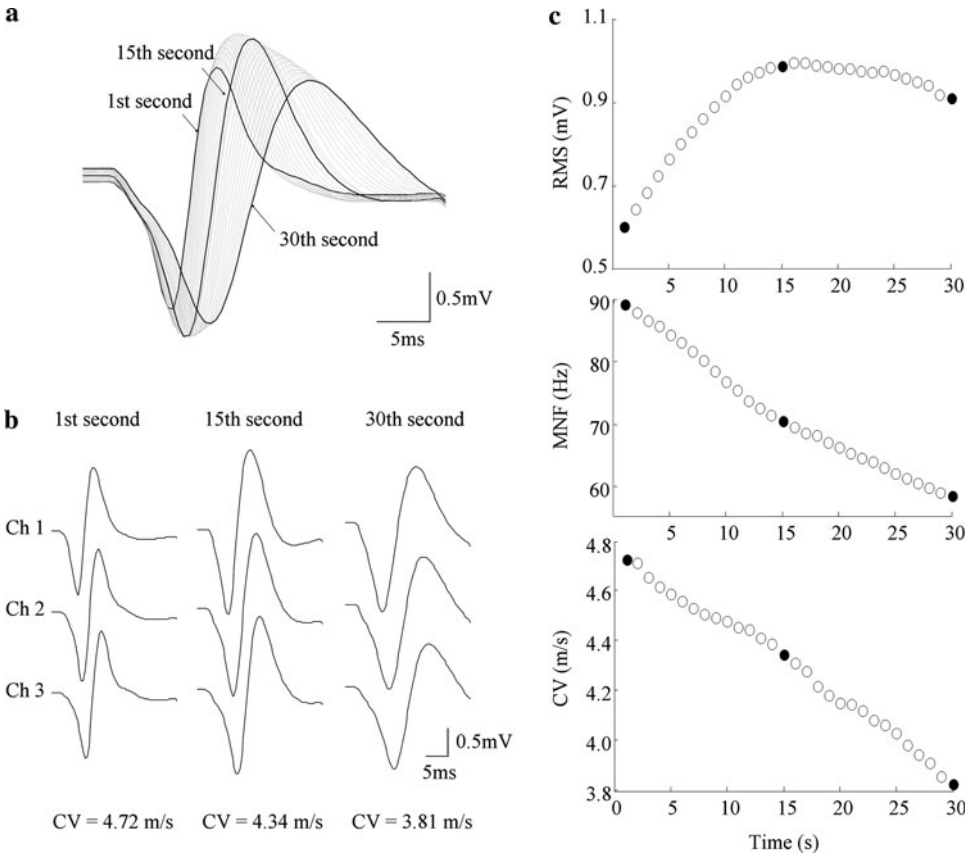

Fig. 2 **a** Single differential M-waves detected (from one subject) from biceps brachii muscle. M-wave time evolution over 30 s of sustained stimulation at 30 pulses per second is shown: *each curve* is the average of the 30 responses obtained during 1-s epochs. The *1st*, *15th*, and *30th* (averaged) M-waves are highlighted. **b** Single differential M-waves detected by three consecutive channels (corresponding to the *1st*, *15th*, and *30th* second of stimulation) and relative mean muscle fiber conduction velocity (*CV*) estimates calculated from the same three channels (triplet). **c** Time course of the sEMG variable estimations during the 30 s electrically elicited contraction. Reported variables are: root mean square (*RMS*); mean frequency of the power spectral density (*MNF*). The *black dots* correspond to the highlighted M-waves of panels **a** and **b**

subjects stood upright with the right arm relaxed at the side of the body.

Skin temperature was monitored throughout the experiment (YSI 4000, Advanced Industrial Systems, Inc., Kentucky, USA) from the central region of the muscle belly of biceps brachii since large changes in temperature may affect the magnitude of sEMG variables (Stalberg 1966).

Blood samples

A total of 10 ml blood sample was drawn from the ulnar vein of the non-exercised arm at baseline, immediately after (<1 min) cessation of the exercises, 2 h and 1 day post-exercise. Biosen C Line Sport (EKF-Daignostic GmbH, Madgeburg, Germany) was used to analyze blood lactate concentration. A Sysmex KX 21N-analyzer (Sysmex Co., Kobe, Japan) was used to monitor hematocrit to obtain blood plasma volume, which remained unchanged throughout the experiment. Both of the aforementioned analyses were done from a 5 ml sample collected to VenoSafe EDTA tube (VenoSafe, Terumo, Belgium). Plasma (centrifuged with 3,500 rpm) electrolyte (Na^+ and K^+) and myoglobin concentrations were analyzed with Konelab 20XTI (Thermo Electron Oy, Vantaa, Finland) from a 5 ml sample collected to VenoSafe Li-heparin tube (VenoSafe).

Statistical analysis

The possible changes in the variables between the groups were compared using multivariate analysis of covariance (MANCOVA) for repeated measurements, with one between-subject factor (group, CON and ECC) and with one within-subject factor (time, before, immediately after, 30 min after and 2 h after the exercises). The values measured before the exercises were used as a covariate. Where applicable, ANOVA for repeated measurements on one factor (time) and post hoc Holm–Bonferroni were used to determine significant differences between study variables separately in the CON and ECC groups. All statistical analyses were performed with SPSS software (ver. 1.4, SPSS, Inc. Chicago, IL, USA). Statistical significance was determined as $p < 0.05$ for all comparisons. The data was confirmed to be normally distributed with Shapiro–Wilk test and have equal variances with Levene's test. The values are presented as means ± SD.

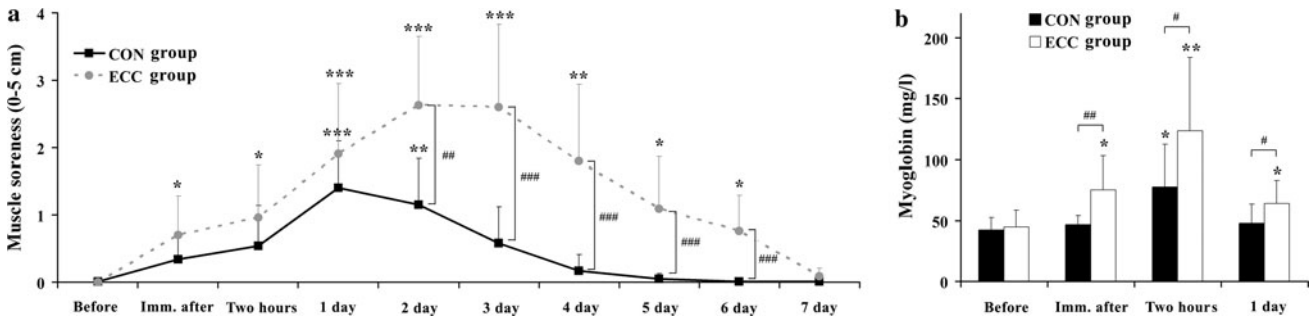

Fig. 3 Subjective perceived muscle soreness (**a**) and myoglobin (**b**) concentrations before, immediately after, 2 h after the exercises and during a follow-up period (days 1–7) in the concentric (*CON*) and eccentric (*ECC*) groups. ***$p < 0.001$; **$p < 0.01$; *$p < 0.05$ with respect to pre-exercise values. ###$p < 0.001$; ##$p < 0.01$; #$p < 0.05$ difference between groups. *Error bars* indicate standard deviation

Results

Muscle soreness, muscle thickness and skin temperature

The subjects did not perceive soreness in the biceps brachii muscle before the exercise. Subjective perceived muscle soreness increased after the exercise in both groups. Furthermore, muscle soreness was greater in the ECC than the CON group at 2 day ($p < 0.01$), 3, 4, 5 and 6 day ($p < 0.001$) post-exercise (Fig. 3a).

Muscle thickness of biceps brachii muscle did not change in CON (baseline, 11.6 ± 2.0 mm; 2 h post-exercise, 11.9 ± 2.1 mm) or ECC (baseline, 13.3 ± 2.6 mm; 2 h post-exercise, 13.8 ± 2.7 mm). Similarly, brachialis muscle thickness did not change in CON (baseline, 29.4 ± 3.6 mm; 2 h post-exercise, 30.5 ± 3.2 mm) or ECC (before, 27.7 ± 3.5 mm; 2 h post-exercise, 27.6 ± 3.1 mm).

There were no statistically significant changes in skin temperature in CON (baseline, 33.4 ± 0.8°C; immediately after, 33.7 ± 1.1°C; 30 min after, 33.2 ± 0.6°C; 2 h after, 33.2 ± 1.0°C) or ECC (baseline, 33.6 ± 0.7°C; immediately after, 34.2 ± 0.8°C; 30 min after, 33.5 ± 0.5°C; 2 h after, 33.3 ± 0.8°C) between the measurement sessions.

Blood variables

Blood lactate concentrations showed slight, but insignificant increases in both groups immediately after the exercise with respect to the baseline values (CON group, from 1.1 ± 0.6 to 1.7 ± 0.9 mmol/l; ECC group, from 1.4 ± 0.8 to 2.8 ± 2.1 mmol/l). Thereafter, blood lactate concentration was at the pre-exercise level at 2 h (CON group, 1.1 ± 0.4 mmol/l; ECC group, 1.5 ± 0.6 mmol/l) and 1 day post-exercise (CON group, 1.1 ± 0.4 mmol/l; ECC group, 1.1 ± 0.4 mmol/l).

Blood myoglobin concentration increased in both groups at 2 h post-exercise compared to pre-exercise levels (Fig. 3b). In addition, the ECC group showed an acute increase in myoglobin concentration immediately after the exercise and this elevation persisted longer. Furthermore, the increase in myoglobin concentration was higher in ECC than in CON immediately, 2 h and 1 day post-exercise ($p < 0.05$, Fig. 3b).

Blood K^+ concentration increased in ECC from a baseline value of 4.17 ± 0.44 to 4.42 ± 0.28 mmol/l immediately post-exercise ($p < 0.05$). No change was observed in the CON group (baseline, 4.25 ± 0.35 mmol/l; immediately post-exercise, 4.33 ± 0.25 mmol/l). However, the change in K^+ concentration was not significantly different between the groups. Blood Na^+ concentration showed no significant changes.

Blood Na^+ concentrations showed no significant changes in CON group (before, 139 ± 2.3 mmol/l; immediately after, 140 ± 2.2 mmol/l; 2 h, 139 ± 2.3 mmol/l; 1 day, 140 ± 1.0 mmol/l) or in ECC group (before, 140 ± 2.3 mmol/l; immediately after, 139 ± 1.7 mmol/l; 2 h, 140 ± 1.7 mmol/l; 1 day, 141 ± 2.2 mmol/l).

Voluntary force production

Isometric MVC was reduced from pre-exercise values (CON, 366 ± 74 N; ECC, 334 ± 108 N) in both groups immediately after the exercises (CON: 280 ± 40 N, $p < 0.01$; ECC: 234 ± 94 N, $p < 0.001$; Fig. 4). A prolonged reduction in isometric MVC was only observed in the ECC group at 2 h post-exercise (249 ± 93 N, $p < 0.01$). Furthermore, the reduction in isometric MVC was higher in ECC at 2 h post-exercise ($p < 0.01$) than in CON (Fig. 4).

Concentric MVC decreased in both CON (baseline, 277 ± 48 N; immediately post-exercise, 216 ± 50 N, $p < 0.01$) and ECC (baseline, 259 ± 79 N; immediately post-exercise, 171 ± 88 N, $p < 0.01$) immediately after the exercise. The concentric MVC also remained reduced at 2 h post-exercise in the ECC (185 ± 80 N, $p < 0.01$), but not in the CON (259 ± 57 N). In addition, the reduction in the concentric MVC was significantly greater ($p < 0.01$) in ECC than in CON at 2 h post-exercise (Fig. 4).

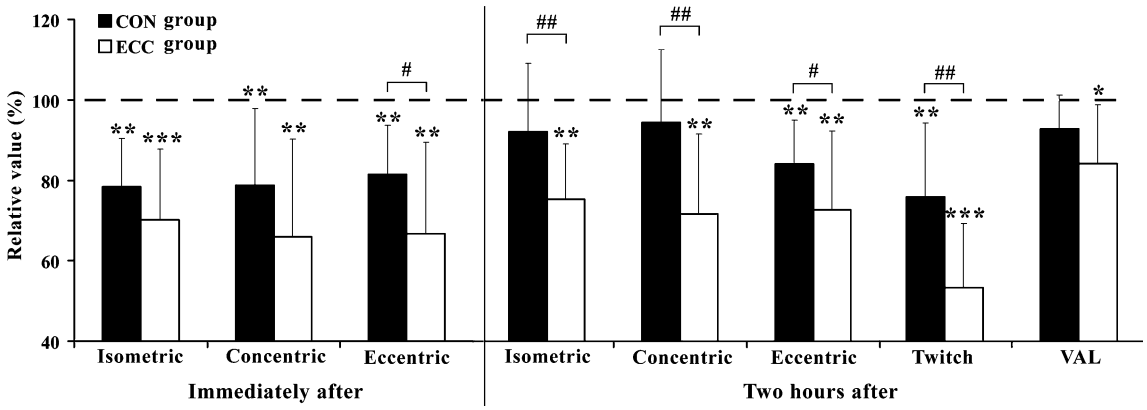

Fig. 4 Relative values of maximal voluntary contraction (MVC) trials with concentric, isometric and eccentric contraction types, passive twitch force and voluntary activation level (VAL) in the concentric (CON) and eccentric (ECC) groups immediately after (0-5 min after cessation of the exercise) and 2 h after the exercise. *** = $p < 0.001$, ** = $p < 0.01$ and * = $p < 0.05$ with respect to pre-exercise values (dashed line). ## = $p < 0.01$ and # = $p < 0.05$ difference between groups. Error bars indicate standard deviation

Eccentric MVC was reduced in CON (baseline, 336 ± 42 N; immediately post-exercise, 272 ± 39 N; 2 h post-exercise, 281 ± 40 N, $p < 0.01$) and in ECC (baseline, 318 ± 82 N; immediately post-exercise, 208 ± 88 N; 2 h post-exercise, 227 ± 74 N, $p < 0.01$) immediately after and 2 h after the exercises with respect to pre-exercise values. Furthermore, these reductions were more prominent ($p < 0.05$) in ECC than in CON in these measurement points (Fig. 4).

Passive twitch force and voluntary activation level

Passive twitch force was reduced from the baseline values in both groups at 2 h post-exercise (CON, from 92.0 ± 17.9 to 71.0 ± 17.3 N, $p < 0.01$; ECC, from 78.2 ± 20.4 to 42.0 ± 17.9 N, $p < 0.001$; Fig. 4). Furthermore, the reduction was significantly greater in the ECC group ($p < 0.01$, Fig. 4).

Voluntary activation level decreased in ECC after the exercises (baseline, 84.7 ± 11.8%; 2 h post-exercise, 73.4 ± 16.1%, $p < 0.05$; Fig. 4). However, this reduction was not significantly different from the CON group (baseline, 86.4 ± 10.1%; 2 h post-exercise, 82.9 ± 10.3%).

Surface EMG variables during isometric MVC

Bicpes brachii muscle

Both groups showed substantial reductions in RMS in biceps brachii muscle immediately after and 2 h after the exercises (see Table 1). These reductions did not differ between the groups.

During the isometric MVC, significant reduction of MNF was observed only in ECC group ($p < 0.01$) at 2 h post-exercise compared to pre-exercise values (Table 1; Fig. 5). Furthermore, this reduction was greater ($p < 0.05$) in ECC group than in CON group.

Table 1 sEMG during isometric MVC test

Session	CON group	ECC group
RMS (mV)		
Baseline	0.45 ± 0.17	0.37 ± 0.14
Imm. after	**0.29 ± 0.14*****	**0.24 ± 0.10*****
2 h	**0.30 ± 0.16***	**0.25 ± 0.14****
MNF (Hz)		
Baseline	105.4 ± 25.3	105.9 ± 27.4
Imm. after	114.9 ± 32.6	104.7 ± 22.7
2 h	**90.9 ± 14.9**$^{\#}$	**80.3 ± 12.7****$^{\#}$
CV (m/s)		
Baseline	4.21 ± 0.54	4.16 ± 0.34
Imm. after	**4.63 ± 0.47*****	4.24 ± 0.41
2 h	**4.10 ± 0.49**$^{\#,\#}$	**3.43 ± 0.44*****$^{,\#\#}$

All significant changes from baseline are highlighted with bold text

* $p < 0.05$; ** $p < 0.01$; *** $p < 0.001$ with respect to BEF. $^{\#}$ $p < 0.05$; $^{\#\#}$ $p < 0.01$ difference between the groups

The CON group showed an increase in CV immediately after the exercise ($p < 0.001$; Table 1; Fig. 5). However, this change was not significantly different from the ECC group. At 2 h post-exercise, significant reduction was observed in CV in ECC group ($p < 0.001$; Table 1; Fig. 5). Moreover, this reduction in CV of the ECC group was significantly greater ($p < 0.01$) than in the CON group.

Triceps brachii and brachioradialis muscles

There were no changes in triceps brachii muscle (antagonist) activity. In addition, brachioradialis muscle (synergist) showed a reduction in RMS in both groups

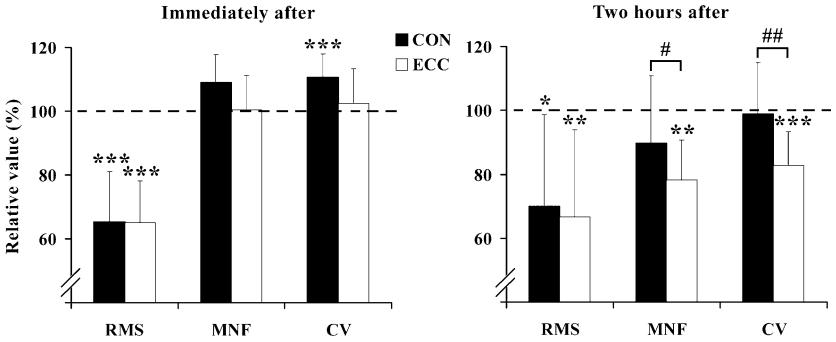

Fig. 5 Root mean square (*RMS*), mean power frequency (*MNF*) and mean muscle fiber conduction velocity (*CV*) in biceps brachii muscle during isometric MVC in concentric (*CON*) and eccentric (*ECC*) groups immediately after and 2 h after exercise normalized to pre-exercise values (*dashed line*). $***p < 0.001$; $**p < 0.01$; $*p < 0.05$ with respect to the pre-exercise values. $\#\#p < 0.01$; $\#p < 0.05$ difference between groups. *Error bars* indicate standard deviation

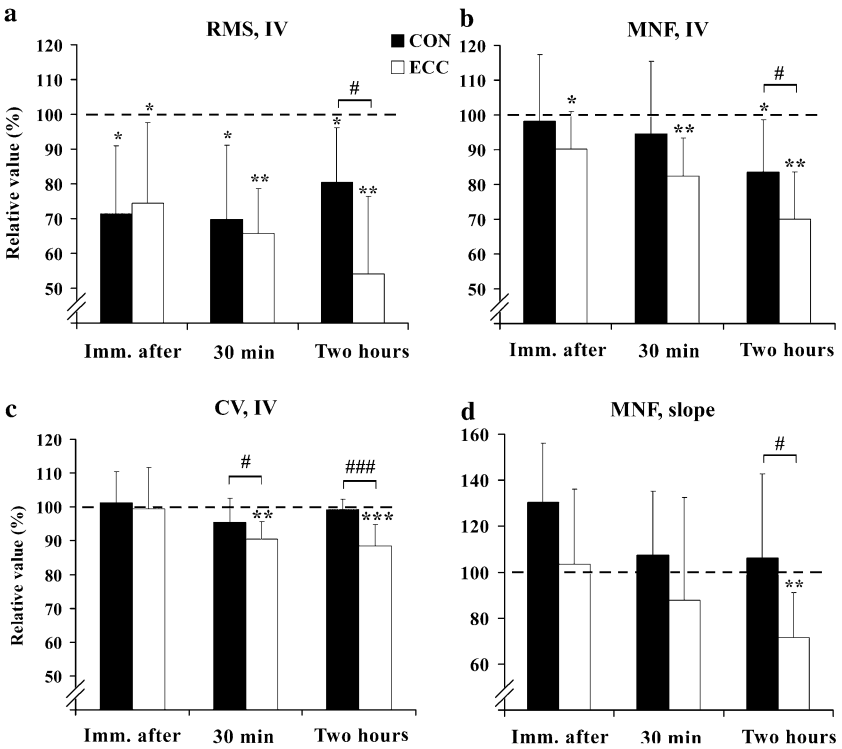

Fig. 6 Initial values (*IV*) of root mean square (*RMS*, **a**), mean power frequency (*MNF*, **b**) and mean muscle fiber conduction velocity (*CV*, **c**), and slope of MNF (**d**) measured from biceps brachii muscle during motor-point electrical stimulation in concentric (*CON*) and eccentric (*ECC*) groups immediately after, 30 min after and 2 h after exercises normalized to pre-exercise values (*dashed line*). $***p < 0.001$; $**p < 0.01$; $*p < 0.05$ with respect to the pre-exercise values. $\#\#\#p < 0.001$; $\#p < 0.05$ difference between groups. *Error bars* indicate standard deviation

immediately after the exercise (CON, from 0.80 ± 0.37 to 0.51 ± 0.21 mV, $p < 0.01$; ECC, from 0.80 ± 0.43 to 0.54 ± 0.28 mV, $p < 0.01$). However, the reduction in brachioradialis did not differ between the groups.

Maximal M-wave properties

Root mean square

Initial RMS values decreased in both CON (baseline, 0.63 ± 0.28 mV; immediately after, 0.45 ± 0.24 mV; 30 min after, 0.43 ± 0.22 mV; 2 h after the exercise, 0.51 ± 0.26 mV, $p < 0.05$) and ECC (baseline, 0.66 ± 0.35 mV; immediately after, 0.46 ± 0.23 mV, $p < 0.05$; 30 min after, 0.44 ± 0.25 mV, $p < 0.01$; 2 h after the exercise, 0.36 ± 0.25 mV, $p < 0.01$) at all post-exercise measurement points (Fig. 6a). In addition, the reduction in RMS was greater in ECC than in CON at 2 h post-exercise ($p < 0.05$).

Mean frequency of the power spectral density

MNF decreased in both groups at 2 h post-exercise (CON: baseline, 102 ± 24 Hz; 2 h post-exercise, 84.0 ± 20 Hz,

$p < 0.05$; ECC: baseline, 108 ± 30 Hz; 2 h post-exercise, 73.2 ± 17 Hz, $p < 0.01$), with a greater reduction observed in ECC than in CON ($p < 0.05$, Fig. 6b). Moreover, a significant reduction in MNF was also observed in the ECC group immediately after (96.3 ± 25 Hz, $p < 0.05$) and 30 min after (88.0 ± 24 Hz, $p < 0.01$) the exercise (Fig. 6b). The regression slope of MNF values during the length of monopolar motor-point electrical stimulation only showed a reduction in ECC (baseline, −2.64 ± 0.88 Hz/s; 2 h post-exercise, −1.86 ± 0.85 Hz/s, $p < 0.01$) at 2 h post-exercise (Fig. 6d). This change was significantly greater than in the CON group ($p < 0.05$).

Conduction velocity

A reduction of the initial value of CV from a pre-exercise value of 4.33 ± 0.36 m/s was only detected in ECC at 30 min (3.91 ± 0.24 m/s, $p < 0.01$) and 2 h (3.82 ± 0.3 m/s, $p < 0.001$) post-exercise (Fig. 6c). This reduction was significantly greater than in the CON group (30 min, $p < 0.05$; 2 h post-exercise, $p < 0.01$).

Discussion

Eccentric exercise caused typical symptoms of EIMD, such as a reduction of maximal force production capability, a delayed increase in muscle soreness and an increase in the permeability of the sarcolemma to myoplasmic proteins, indicated by an increase in blood myoglobin concentration. Similar, but significantly less evident symptoms were observed after concentric exercise. The main finding of the present experiment was that, in general greater reductions were observed in MNF and CV after eccentric than concentric exercise, both during maximal voluntary contractions and electrically elicited contractions. This difference was most evident at 2 h post-exercise, where the CON group showed either a recovery or less significant reduction in sEMG variables than the ECC group. RMS values were substantially reduced after both exercise modes, with a greater reduction after eccentric than concentric exercise, although this was only evident during electrically evoked contractions at 2 h post-exercise.

Force production capability and muscle activation

Many investigations have observed a greater and more prolonged reduction of maximal voluntary force production capability after repeated eccentric contractions compared to repeated concentric contractions (Jones et al. 1989; Smith and Newham 2007). The present results are well in line with these findings, since the ECC group showed greater reductions than CON regardless of contraction type applied in MVC test, as well as in electrically evoked twitch force at 2 h post-exercise (Fig. 4). Similar disparity in the reductions of twitch force and its evolution over time up to 4 h after eccentric and concentric exercise has been shown explicitly by Smith and Newham (2007). The greater reduction in MVC after eccentric exercise may involve central and peripheral factors. The central fatigue in the motor cortex and/or at the spinal level can be jointly estimated by measurement of voluntary activation level, which indeed showed reduction after the eccentric exercise at 2 h post-exercise, although this change was not different from the CON group (Fig. 4). Furthermore, electrically evoked passive twitch force, which bypasses the central contribution, was clearly reduced after both exercises and more substantially after the eccentric exercise than the concentric one. Therefore, the reduction in voluntary activation cannot solely explain the greater reduction of MVCs in ECC as compared to CON.

The peripheral factors explaining the greater loss of force production capability may involve a loss of excitability of some of the muscle fibers. This possibility is supported by simultaneous reduction of RMS amplitude (both voluntary and M-wave) and passive twitch force (Figs. 4, 5, 6a). Previous experiments have shown either an acute reduction (<1 day) in RMS (Hortobagyi et al. 1998; Michaut et al. 2002; Piitulainen et al. 2008), a more prolonged reduction (≥1 day) in RMS (Hortobagyi et al. 1998; Kroon and Naeije 1991), or no reduction in RMS (Chen 2003) after eccentric exercise comparable to the current exercise model. The acute (≤2 h) reduction of RMS after both eccentric and concentric exercise could be explained by (1) impaired neuromuscular transmission, although McFadden and McComas (1996) have argued that impairment in the neuromuscular junction is unlikely, since the safety factor for proper function of neuromuscular junctions is high due to a high concentration of postsynaptic Na^+ channels (Flucher and Daniels 1989), especially in the fast twitch fibers (Ruff and Whittlesey 1992); (2) reduced sarcolemmal excitability due to a change in resting membrane potential consequent to modified ion concentration gradients over the sarcolemma, as a result of increased sarcolemmal permeability (McNeil and Khakee 1992) caused by activation of stretch activated Na^+ and Ca^{2+} ion channels (McBride et al. 2000; Stauber 1989) and/or acute sarcolemmal damage (McNeil and Khakee 1992); (3) predominant damage of fast twitch fibers (Jones et al. 1986; Lieber and Friden 1988; Takekura et al. 2001; Vijayan et al. 2001), which are the main contributors to RMS values due to large size of MUs comprising of fast twitch fibers and thus their high compound action potential amplitude; and (4) a change in muscle fiber action potential shape due to an increase of the depolarization zone length

(Merletti et al. 1999) and/or slowing of action potential propagation velocity (Piitulainen et al. 2010). With the exception of the impaired neuromuscular transmission, most of these events are shown to occur after eccentric exercise, but not as clearly after concentric exercise. Therefore, it was surprising that no differences between the groups were observed in RMS during either the isometric MVC (Fig. 5) or in M-wave (Fig. 6a) immediately after and 30 min post-exercise. However, the initial value of RMS during the monopolar motor-point electrical stimulation showed a greater reduction of RMS in the ECC group than the CON group at 2 h post-exercise (Fig. 6a). The result from the electrically evoked contraction is probably the most valid indicator of mean muscle fiber excitability, since it is not affected by control strategies and possible fatigue of the central nervous system.

Function of sarcolemma

MNF and CV reflect the functional properties of the sarcolemma, but only MNF (of M-wave) showed reduction immediately after the eccentric exercise, but not after the concentric exercise (Figs. 5, 6b). It is known that both MNF and CV increase with function of muscle temperature (Stalberg 1966). However, the skin temperature showed only a moderate, non-significant increase (less than 0.6 ± 0.8°C) immediately after the exercises when compared to the pre-exercise values. Nevertheless, there might have been some post-activity potentiation present in these variables as the clear increase in CV in CON group may suggest (Fig. 5). It also appears that the sarcolemmal dysfunction is increasing with time in the ECC group during the follow-up period (Figs. 5, 6). It is also noteworthy that the additional 30 min post-exercise time-point revealed a significantly greater reduction in CV in the ECC group than the CON group (Fig. 6c). The 30 min time-point was added to the experimental protocol because a late depression of M-wave amplitude has been observed 16–150 min after fatiguing electrical stimulation (McFadden and McComas 1996). Such stimulation is known to induce a similar prolonged reduction in twitch force (Edwards et al. 1977; McFadden and McComas 1996) as is the case after eccentric exercise.

At 2 h post-exercise, both MNF and CV showed greater reductions after eccentric than concentric exercise (Figs. 5, 6b, c). These results are in line with the literature, since acute ($\leq$2 h) reductions of MNF (Linnamo et al. 2000; Piitulainen et al. 2010; Sbriccoli et al. 2001) and CV (Piitulainen et al. 2010) have been detected after eccentric exercise when measured during maximal voluntary and electrically evoked contractions. To our knowledge, the effect of maximal concentric exercise on CV has not been studied during MVC tests or M-waves. However, a reduction of MNF has been observed during MVC after maximal concentric exercise. These results could be associated with greater increase in sarcolemmal permeability in ECC group than CON group, as the increased blood myoglobin concentration indicated (Fig. 3b). This may disturb the normal ion concentrations (e.g., Na^+ and K^+) over the sarcolemma, and possibly slow down or even block the sarcolemmal or transverse tubular system action potential propagation. Indeed, sarcolemmal action potential amplitude and conduction velocity may be dependent on interstitial [K^+] (Hodgkin and Horowicz 1959; Juel 1988). For this reason, the increase in plasma [K^+] observed in the ECC group could partly explain the greater reductions of RMS, MNF and CV in the ECC group (Fig. 6a–c), although this issue remains unclear since interstitial [K^+] could not be measured in the current experiment.

The sensitivity of the sarcolemma to eccentric contractions could be related to lower MU activity during the maximal eccentric contractions than concentric contractions (Westing et al. 1991), despite the fact that eccentric contractions often show higher maximal force values (Katz 1939; Westing et al. 1991). Eccentric MVC was also significantly higher than concentric MVC in the current experiment (average concentric MVC, 268 ± 64 N; eccentric MVC, 327 ± 64 N, $p < 0.001$). Therefore, it may be that muscle fibers and their force transmitting sarcolemmas (Street and Ramsey 1965) are subjected to higher mechanical stress during eccentric exercise than during concentric exercise. This is supported by morphological observations of damage in membrane structures of muscle fibers after eccentric contractions (Jones et al. 1986; Lieber and Friden 1988; Takekura et al. 2001; Vijayan et al. 2001).

There is some evidence that extracellular [K^+] may increase due to leakage of K^+ from ATP and Ca^{2+}-sensitive channels (Burton et al. 1988; Pallotta 1985). Nevertheless, there seems to be a large flow of K^+ into the interstitial space during muscle activity (Sjogaard 1990). Furthermore, it may be that the increase in sarcolemmal permeability due to its initial wounding (McNeil and Khakee 1992) and activation of different stretch activated Na^+ and Ca^{2+} channels (McBride et al. 2000; Stauber 1989) preferentially loads the Na^+–K^+ pumps in the eccentrically exercised fast twitch muscle fibers. However, it seems that the loss of adenosine 5′-triphosphate and phosphocreatine are insignificant immediately after cessation of eccentric exercise (Bonde-Petersen et al. 1972) and the Na^+–K^+ pump seems to have a high safety factor, since only 2–6% of its capacity is in use under normal conditions (Clausen 1986). In contrast, the activation of stretch activated Na^+ channels has been observed to cause a depolarization of resting membrane potential 1 and 24 h after eccentric contractions (McBride et al. 2000), which may

cause a slow inactivation of voltage-gated TTX-sensitive Na^+ channels that are largely responsible for action potential propagation (Ruff 1999). Therefore, the inactivation of voltage-gated Na^+ channels could block or slow down the action potential propagation in the affected muscle fibers and thus result in a depression of the compound M-wave amplitude and power spectral variables.

Impairment in sarcolemmal action potential conduction has been observed earlier during sub- and maximal isometric contractions at high force levels (50–100% of MVC), where the MUs that comprise fast twitch fibers are likely to dominate the sEMG signals (Piitulainen et al. 2010). These are the same group of fibers known to be the most sensitive to eccentric contractions (Jones et al. 1986; Lieber and Friden 1988; Takekura et al. 2001; Vijayan et al. 2001). Since the current sEMG signals were recorded during maximal contractions involving most if not all MUs, it may be that eccentric exercise affects the gross sarcolemmal function of fast twitch fibers substantially more than concentric exercise. However, the effect of maximal concentric exercise on different threshold MUs should be studied in more detail to fully confirm this hypothesis.

The slope of MNF values during the motor-point stimulation showed a reduction at 2 h after the eccentric exercise (Fig. 6d). However, when the slope was normalized to the initial value of MNF, which was significantly lower after eccentric exercise (Fig. 6b), no significant changes were observed in the slope. In normal conditions with electrical stimulation of fresh muscle, a steeper slope would be interpreted as a higher rate of fatigue during the stimulation. The current eccentrically fatigued muscle showed the opposite, a slower rate of fatigue. This could be because of the fact that there was less "room" for additional fatigue to occur, since the fatigued state of the eccentrically exercised muscle and especially its fast twitch fibers are less fatigue resistant than the slow twitch fibers. This result further supports the greater effect of eccentric exercise on sarcolemmal action potential conduction than concentric exercise, which did not cause any significant changes in the MNF slope (Fig. 6d).

Conclusions

It is concluded that eccentric exercise can induce a greater and more prolonged reduction both in maximal force production of elbow flexors and action potential propagation over the sarcolemma of the affected muscle fibers than similar concentric exercise. Furthermore, the repeated maximal eccentric contractions appeared to induce a greater increase in sarcolemmal permeability, which may partly explain the impairment of sarcolemmal action potential propagation in the affected muscle fibers. Finally, the impaired sarcolemmal action potential propagation may partly explain the loss of muscle force particularly after eccentric exercise fibers in the early stage of EIMD ($\leq$2 h).

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