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Sarcolemmal excitability as investigated with M-waves after eccentric exercise in humans

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Abstract

It has been shown that intensive eccentric muscle actions lead to prolonged loss of muscle force and sarcolemmal damage. This may lead to a reduction in the excitability of the sarcolemma and contribute to the functional deficit. Experiments were carried out to test sarcolemmal excitability after eccentric elbow flexor exercise in humans. Electrically elicited surface compound muscle action potential (M-wave) properties from 30 s stimulation trains (20 Hz) were analyzed in biceps brachii muscle immediately after, 1 h and 48 h after the exercise. M-wave area, amplitude, root mean square and duration were reduced immediately after the eccentric exercise. However, no such reduction could be observed 48 h after the exercise, although the maximal voluntary isometric and eccentric torques were still depressed by $12.2 \pm 9\%$ ($P < 0.001$) and $17.7 \pm 9\%$ ($P < 0.001$), respectively. Acute increase in plasma concentrations of K^+ and Ca^{2+} were also observed after the eccentric exercise. These findings suggest that eccentric exercise may acutely decrease sarcolemmal excitability, which seems to be partially related to increased extracellular ion concentrations. However, disturbance of sarcolemmal excitability is not the major factor determining eccentric exercise induced prolonged loss of muscle strength, because no prolonged impairment was observed in any of the studied M-wave parameters.

Keywords: M-wave; Electromyogram; Muscle fatigue; Delayed-onset muscle soreness; Sarcolemma

1. Introduction

It is well known in the literature that eccentric exercise is accompanied by a prolonged loss of muscle force (Davies and White, 1981), muscle pain (Armstrong, 1984), increased joint stiffness (Stauber et al., 1990), muscle swelling (Clarkson et al., 1992), a shift in optimal joint angle for torque generation (Jones et al., 1997), muscle protein degradation and efflux to circulation (Belcastro et al., 1998; Clarkson et al., 1986). All these effects are well known symptoms of exercise induced muscle damage (EIMD) (Allen, 2001). The cause for the loss of muscle strength is not fully understood, but has usually been attributed to disturbance of excitation–contraction (EC) coupling after eccentric actions (Warren et al., 2001), however, failure of activation has also been suggested (Linnamo et al., 2004). EC coupling could be disturbed due to (a) defect of Ca^{2+} kinetics in the sarcoplasmic reticulum (SR) (Warren et al., 2001), (b) sarcomere level damage in the contractile machinery (Friden et al., 1981; Roth et al., 1999) and (c) ultrastructural selective damage of force bearing proteins such as subsarcolemmal dystrophin (Komulainen et al., 1998; Koskinen et al., 2006) or the intermediate filament desmin (Barash et al., 2002; Friden et al., 1981). Selective damage of force bearing proteins may disturb lateral force transmission in the sarcolemma, and thus impair muscle strength (Monti et al., 1999). The sarcolemma itself is often found to be damaged after eccentric actions in animal studies (Friden et al., 1981; Komulainen et al., 1998), but not always in human studies (Yu et al., 2002). Recently, Koskinen et al. (2006) have observed some loss of dystrophin also in some human subjects after eccentric actions. This is an important finding, since any damage

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in the excitable membranes of the muscle cell (sarcolemma or *T*-tubular system) could lead to a failure in action potential (AP) generation and/or propagation, and thus impair EC coupling (Allen, 2001).

It could therefore be hypothesised that reduced excitability of the sarcolemma could lead to a failure in generation and propagation of AP after intensive contractile activity. This could be due to increased extracellular K^+ concentration (Sejersted and Sjogaard, 2000) or direct damage of the sarcolemma (Friden et al., 1981; Koskinen et al., 2006). Sarcolemmal excitability is defined as the inward current that is required to depolarize the sarcolemma enough to reach the threshold potential that triggers a sufficient amount of Na^+ channels to elicit an AP (Sejersted and Sjogaard, 2000). If the sarcolemma is damaged, as indicated by dystrophin negative immunohistochemical stained fibers after eccentric actions (Koskinen et al., 2006), the sarcolemmal ion permeability may increase. This could possibly lead to ion concentration disturbance over the muscle cell membrane and affect the resting and threshold membrane potentials. This will naturally also affect the membrane excitability. In addition to direct microscopic observations of damaged sarcolemma, there are changes in sarcolemmal function after eccentric actions. McBride et al. (2000) observed that in animals, eccentric exercise caused prolonged membrane depolarization due to increased cation conductance via stretch-activated ion channels in the sarcolemma. Thus, eccentric actions may decrease the sarcolemmal excitability due to increased sarcolemmal ion permeability and, therefore, decrease the force production capability of the muscle.

In muscle fatigue experiments, sarcolemmal excitability has been studied with electrically elicited electromyography (EMG) signals (Bellemare and Garzaniti, 1988; Michaut et al., 2002; Milner-Brown and Miller, 1986; Pasquet et al., 2000; Stephens and Taylor, 1972). Electrical stimulation has the advantage of standardized conditions as compared to voluntary muscle actions, since it controls motor unit (MU) firing frequency, MU recruitment, eliminates cross-talk from nearby muscles and is independent of subject motivation to perform muscular contraction (Merletti et al., 1990). Motor point electrical stimulation recruits only a portion of the muscle, thus accurate repositioning of the electrode is very critical to obtain repeatable results. In motor point electrical stimulation, all the activated MUs are recruited at the same time. Thus, simultaneously measured EMG signals enable analysis of the M-wave, which contains information about membrane properties of the active MUs (Merletti et al., 1992).

Even though disturbances in EC coupling have been suggested to be responsible at least partly for the strength loss after eccentric actions (Warren et al., 2001), it is not certain if sarcolemmal excitability impairment contributes to EC coupling disturbance after eccentric actions. Therefore, the aim of the present study was to verify if sarcolemmal excitability is decreased during the two day post-exercise period after eccentric exercise, and whether it could further explain the EC coupling dysfunction.

2. Methods

2.1. Subjects

One female and eleven male volunteers (age 25.4 ± 2.8 yr; height 179.7 ± 5.8 cm; weight 76.9 ± 11.8 kg) participated in the study. One female and two males were only included for the reliability measurements of the electrically elicited EMG parameters, and nine male subjects carried out both the exercise protocol and reliability measurements. Subjects were physically active students and had no known symptoms of neuromuscular disorders. The subjects were non-smokers, did not drink caffeine rich drinks 12 h before the measurements, and avoided strenuous exercise two days before the first measurement and throughout the whole study period. The study was conducted in accordance with the Declaration of Helsinki and approved by the ethics committee of the University of Jyväskylä. All participants were aware of the possible risks and discomfort of the experiments and signed a written informed consent form before inclusion.

2.2. Study protocol

The measurements consisted of three different sessions on different days and five identical measurements. During the familiarisation session (PRE, 1–2 day before exercise session) subjects practised submaximal eccentric elbow flexor actions, electrode locations were determined and measurements for reliability analysis were conducted. The effect of a few eccentric actions was checked during the familiarisation session and no changes were observed in any of the measured variables. Baseline measurements were performed before (BEF) the eccentric exercise and followed up to 48 h post-exercise [immediately after (IA), one hour (1 h) after and two days after (2D) the eccentric exercise]. The measurements were identical and had the same order in each session. Tetanic electrical stimulation (+10 s post-exercise) was conducted first and was followed by a collection of blood samples (+40 s post-exercise), maximal voluntary contraction (MVC) measurements (+60–80 s post-exercise), measurement of elbow joint angle and assessment of subjective muscle soreness (+5 min post-exercise).

The tetanic electrical stimulation was repeated during the PRE measurement to analyze trial-to-trial reliability. Day-to-day reliability was acquired by comparing the PRE and BEF measurements. Reliability was tested to ascertain the consistency of the used M-wave parameters. The warm-up protocol before each session consisted of three submaximal eccentric and isometric elbow flexions.

2.3. Exercise protocol

Subjects performed two sets of twenty maximal eccentric contractions (2×20) with the elbow flexors of the right arm, on a motorized isokinetic dynamometer (Komi et al., 2000). The exercise protocol included a moderate number of eccentric actions to avoid extensive swelling of the soft tissues (Hesselink et al., 1996; Nosaka et al., 2002), but high enough to induce muscle damage (Nosaka and Clarkson, 1996). The supinated right forearm was attached to a strain gauge transducer, which was fixed to the lever arm of the dynamometer for recording of the force