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Dissociation in changes in EMG activation during maximal isometric and submaximal low force dynamic contractions after exercise-induced muscle damage

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ABSTRACT

Exercise induced muscle damage (EIMD) is characterized by muscle pain, symptoms of swelling, muscle shortening, increased serum creatine kinase activity, decreased force output and altered neuromuscular function. The aim of this study was to determine how the symptoms of EIMD influence the neuromuscular recruitment patterns during maximal and submaximal isometric and submaximal flexion–extension movements. Twenty-five right-handed males were recruited for this study, and were allocated to a control (n = 13) or experimental group (n = 12). In contrast to the control group, the experimental group participated in an EIMD protocol. Subsequently, symptoms of both groups were closely monitored for 132 h. After the EIMD protocol, muscle pain scores in the experimental group peaked after 36 h with creatine kinase activity peaking after 108 h. Twelve hours after the EIMD protocol, EMG activity had decreased by 20% during a maximal voluntary contraction in the experimental group and decreased by a further 10% until the end of the study was reached 132 h after the EIMD protocol. In contrast EMG during a submaximal flexion–extension movement increased significantly until the end of the study (132 h). This dissociated EMG activity at submaximal and maximal intensity, suggests that central regulation influences the neural firing patterns and motor unit activity.

1. Introduction

Exercise-induced muscle damage (EIMD) is a well described phenomenon which often occurs after exercise during which the muscle is lengthened under tension. The symptoms of EIMD include a lowered pain threshold in the affected area, a decreased range of motion of the affected joint, increased circumference of the affected area due to swelling and increased serum creatine kinase activity. The clinical symptoms usually appear 12 h after the exercise, peak at 48 h and subside about 7 days later (Armstrong et al., 1991). In addition to these symptoms there is also impaired performance during the recovery from muscle damage, especially during low force shortening and lengthening actions of the biceps brachii (Turner et al., 2008). Several studies have shown that EMG activity during a submaximal isometric contraction increases with EIMD (Dartnall et al., 2008; Semmler et al., 2007; Turner et al., 2008; Weerakkody et al., 2003). Similarly, increased EMG activity also occurs during isokinetic elbow flexion under constant load (Turner et al., 2008) with the greatest increase in EMG activity occurring during muscle shortening (90–60°) compared to isometric or lengthening action (90–120°). The changes in the submaximal EMG can be ascribed to changes in motor unit or muscle fiber recruitment, changes in neuromuscular firing rates or increased synchronization of the firing units (Sayers and Clarkson, 2001). Muscle fiber recruitment changes are possibly a result of the type II muscle fibers being more prone to damage causing a recruitment shift towards type I fibers (Fridén and Lieber, 1992; Fridén et al., 1983). Therefore to achieve the same submaximal force output when the muscle is damaged, compared to before the damage, more type I fibers are recruited increasing EMG activity.

While EMG activity during submaximal exercise increases after EIMD, the activity during maximal exercise is reduced (Komi and Rusko, 1974). This could either be due to changes in recruitment patterns as a result of damage to the muscle fibers or as a compensatory mechanism designed to protect the muscle fibers from further damage which might occur during maximal exercise (Linnamo et al., 2003; Prasartwuth et al., 2005).

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The time course of the changes and recovery of the neuromuscular function during submaximal and maximal exercise is not as clearly defined as the manifestation of the clinical symptoms described earlier. It has been shown that maximal EMG and neuromuscular function can be altered during a maximal contraction for up to seven to ten days after an EIMD exercise (Bottas et al., 2007; Deschenes et al., 2000), while the symptoms of EIMD subsided seven days after the protocol (Deschenes et al., 2000). However most EMG studies following EIMD have been conducted up to 24 h (Semmler et al., 2007; Turner et al., 2008) or 48 h (Weerakkody et al., 2003) after the exercise protocol, therefore the time course of changes have not been well described.

Therefore, the aim of this study was to determine how the symptoms of EIMD influence the neuromuscular recruitment patterns, measured by the means of EMG, during maximal and submaximal isometric and submaximal flexion–extension contractions over a 132 h period after the muscle damage was induced.

2. Methods

Thirty-two right-handed male participants, aged 21–40 years, were recruited for this study. Handedness was determined by the Edinburgh handedness inventory (Oldfield, 1971). Participants were recruited in groups of six and allocated to the control or experimental group matched for age, height, weight, body fat and skinfold thickness measured at the biceps and triceps as the thickness of the fat layer can influence the EMG signal (Nordander et al., 2003). None of the participants had any chronic or acute upper body pain or injuries and did not do any upper body training, which included lengthening the muscle under tension, within the 12 weeks before the study. They were informed about the study design, familiarized with the equipment and signed the consent form before starting the study. The study was approved by the Human Ethics Committee of the Faculty of Health Science, University of Cape Town, while the principles outlined by the Declaration of Helsinki were adopted in this study (2002).

2.1. Study design

Before the start of the study all participants were familiarized with the different testing protocols. Data collection occurred over seven consecutive days (Fig. 1). To minimize the effect of circadian rhythm on any of the outcome measures, all testing was scheduled at the same time of the day (within 60 min), except for the 12 h measurements.

Twelve hours before the exercise protocol, stature, body mass, body fat percentage (Durnin and Womersley, 1974) and skinfolds (Ross and Marfell-Jones, 1991) of each participant was measured. In addition a blood sample was taken and elbow muscle function, pain scores, biceps girth and resting elbow angle were measured for comparative reasons. During the different muscle function tests (maximal voluntary contraction (MVC), isometric low force contraction test and active flexion and extension movement test) electromyography activity was captured of the musculus (M.) biceps brachii, M. triceps brachii and M. trapezius. Except for EMG measurements (at 0, 12, 36 and 132 h) all other measurements occurred at 0, 12, 36, 60, 84, 108 and 132 h (Fig. 1).

2.2. Exercise protocol

The experimental group participated in an exercise intervention 12 h after baseline testing. As the aim of the study was to induce muscle soreness, a previously described exercise protocol, which has been shown to induce EIMD (Lambert et al., 2002), was adopted in this study. In brief, participants were asked to resist the lengthening movement of the left biceps (5 sets of 25 movements; see more details in 'muscle function tests' section). The resistance to these movements was set on a Biodex dynamometer at 80% of each subject's maximum isometric contraction torque (Lambert et al., 2002).

2.3. Muscle functions tests

Maximal voluntary contraction (MVC) for the left elbow flexion was determined by a Biodex dynamometer (Biodex pro 3, New York, USA). To assess MVC, participants sat on the Biodex chair with their upper body and left upper arm securely strapped to the machine while the left forearm rested freely on the arm rest. In this position, participants were able to freely flex and extend their elbow over a range of approximately 120°, without hyperextending the elbow. The rotation axis of the dynamometer was aligned with the lateral epicondyle of the humerus, while the forearm was fixed into a fully supinated position. This ensured that the flexion/extension movement was carried out in the transversal axis and sagittal plane. Participants were asked to perform three 5 s isometric elbow contractions at maximal effort, with a fixed dynamometer arm angle of 45°. This set-up will result in an elbow angle at about 60°, which is within the optimal length tension curve range (Chang et al., 1999; Saxton and Donnelly, 1996), while also allowing for optimal EMG activity (Linnamo et al., 2006). The three measurements were interspaced with 60 s recovery periods,

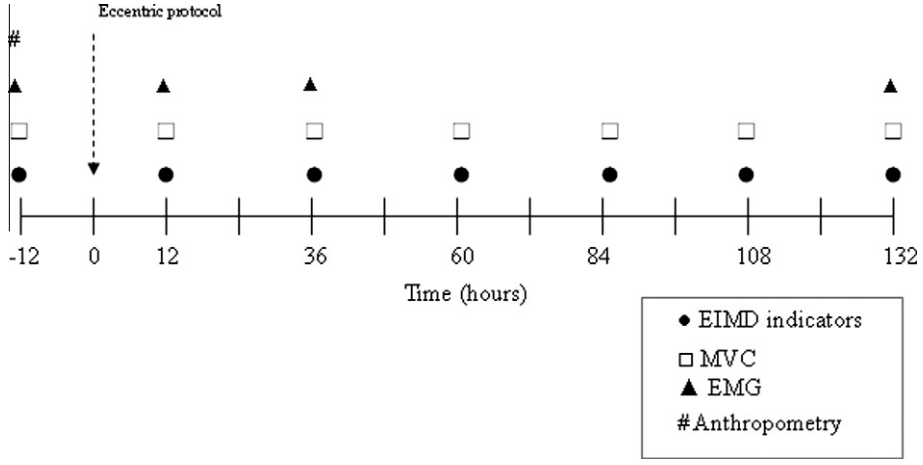

Fig. 1. Timeline of measurements. The EIMD indicators include, pain, arm circumference, elbow angle, creatine kinase activity.