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# Adaptive changes in motor control of rhythmic movement after maximal eccentric actions

Reijo Bottas [a,*], Caroline Nicol [b], Paavo V. Komi [a], Vesa Linnamo [a]

[a] University of Jyväskylä, Neuromuscular Research Center, Department of Biology of Physical Activity, P.O. Box 35, 40014 Jyväskylä, Finland
[b] Department of Biology of Physical Activity, University of the Mediterranean, Marseilles, France

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**Abstract**

Effects of an exhaustive eccentric exercise (EE) on the motor control of maximal velocity rhythmic elbow extension/flexion movement (RM) were examined in eight male students. The exhaustive EE consisted of 100 maximal eccentric actions of the elbow flexor muscles. Movement range was 40–170° in EE at an angular velocity of 2 rad $s^{-1}$. A directive scaled RM of 60° with visual feedback was performed in a sitting position, with the right forearm fixed to the lever arm in horizontal plane above protractor. Surface electromyographic activity (EMG) was recorded from the biceps brachii (BB) and triceps brachii (TB) muscles. Maximal isokinetic eccentric and concentric tests and RM test were conducted before, after, 0.5 h, 2 days and 7 days after the exercise. Dynamic force production was deteriorated after EE ($P < .001$), and did not recover fully within 7 days. The delayed recovery phase was characterized by delayed onset of muscle soreness (DOMS) and elevated serum creatine kinase (CK) activity. The RM test revealed a delayed increase of the fatigued BB muscle EMG activity, but the maximal RM velocity could be preserved. The present results emphasize the capacity of the neuromuscular system to compensate for prolonged eccentric-induced contractile failure by optimizing antagonistic muscles coordination in a demanding rhythmic task. The underlying compensatory mechanisms could be related to increased sensitization of small diameter muscle nerve endings.


*Keywords:* Eccentric actions; Muscle damage; Rhythmic movement; Sensory feedback

## 1. Introduction

Rhythmic movements are normal in human locomotion, although their mechanisms and adaptations in special situations, such as exhaustive fatigue, are not fully understood. In rhythmic bi-directional movement the activity of antagonistic muscles changes reciprocally. It has been suggested that two central commands, reciprocal and unidirectional, may be operative simultaneously or separately in the movements showing reciprocal activity (Feldman, 1980; Yamazaki et al., 1994). Surprisingly, little attention has been paid to the significance of peripheral feedback in the control of rhythmic movement. It is well established, however, that muscle proprioception plays an important role in optimizing the motor control as the information conveyed by muscle afferents is assumed to contribute to position and movement sense (e.g. Roll et al., 1989). In this line, proprioceptive feedback has been shown to be necessary to maintain non-preferred rhythmic movement stationary (Bonnard and Pailhous, 1999). The absence of this feedback reportedly affects the fine control of antagonistic muscles activity (Nicol et al., 1997), and causes instantaneous fluctuations in rhythmic movement amplitude and frequency (Bonnard and Pailhous, 1999). When intensive enough, eccentric exercises typically lead to impaired sense of position (Skinner et al., 1986; Saxton et al., 1995; Brockett et al., 1997) and force (Gandevia and McCloskey, 1978; Saxton et al., 1995; Brockett et al., 1997; Carson et al.,

* Corresponding author. Fax: +358 142602071.
*E-mail address:* reijo.bottas@sport.jyu.fi (R. Bottas).

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2002; Weerakkody et al., 2003b) as well as to a reduced ability to discriminate movement velocity (Pedersen et al., 1999).

Based on the main contribution of the stretched (antagonist) muscles to the position acuity (Roll et al., 1989; Ribot-Ciscar et al., 2003), fatigue-induced deterioration of antagonist muscle proprioception could result in alterations of muscle activation pattern. Our recent study of goal-directed target movement (Bottas et al., 2005) revealed that eccentric fatigue of the elbow flexor muscles affected mostly the flexion movement. On the other hand, the extension movement performance was maintained despite modifications of the triphasic EMG activity pattern up to day 7 post-exercise. It was suggested that muscle fatigue-related mechanisms were operative in optimal modulation of the second agonist burst amplitude and timing. The performance defects may be attributed to various mechanical and neural changes induced by the eccentric exercise. Based on the potent supraspinal influence on the fusimotor system, a decreased neural activation following eccentric-induced muscle damage (for review, see Gandevia, 2001) is likely to evoke changes in γ-motoneurone excitability and, in turn, in muscle spindle afferent sensitivity.

Intensive and/or unusual eccentric exercises typically induce reversible ultrastructural muscle damage (Lieber et al., 2002). The occurrence of direct muscle spindle damage is more questionable (Gregory et al., 2004). The sequence of damage events has been postulated to include initial and secondary structural injury phases (Faulkner et al., 1993). The secondary phase of recovery reflects progressive inflammatory/regenerative process within the damaged muscles, with major structural disturbances and peak muscle soreness 2–3 days post-exercise (Clarkson and Newham, 1995; Jones et al., 1997; Fridén and Lieber, 2001; Cheung et al., 2003; Yu et al., 2003). Reductions in maximal static performance may last for several days (e.g. Clarkson et al., 1992; Howell et al., 1993; Sbriccoli et al., 2001) and even for weeks (Faulkner et al., 1993; Sayers and Clarkson, 2001), with an associated right shift of the active length-tension relation (e.g. Saxton and Donnelly, 1996; Jones et al., 1997; Whitehead et al., 2001). Additional mechanical changes may affect dynamic performances for several days such as increased passive stiffness (Howell et al., 1993; Whitehead et al., 2003), swelling and reduced range of motion (Clarkson et al., 1992; Whitehead et al., 2001).

The overall muscle recovery process, including its associated muscle pain, is very likely to influence muscle activation, both in its central and reflex components, and in different ways in its acute and delayed recovery phases (for a review, see Gandevia, 2001). The nature of eccentric fatigue is favouring the hypothesis of a delayed increased sensitivity of small diameter (group III and IV) afferents as they are sensitive to chemical, thermal and mechanical changes associated with exercise-induced muscle injury (Mense, 1977; Kniffki et al., 1978). In terms of their influence on neural activation, both facilitatory and inhibitory influences have been reported that may operate at different levels of the nervous system. Some convincing, although indirect, evidence exists in favour of a presynaptic inhibitory effect on α-motoneurons (e.g. Garland and McComas, 1990; Garland, 1991; Rossi et al., 1999) and reduced excitability of the motor cortex (e.g. Le Pera et al., 2001). In animal model, the sensitization of these afferents via intramuscular injections of either inflammatory or pain substances has been reported to affect the fusimotor system, and consequently the sensitivity of muscle spindles (e.g. Djupsjöbacka et al., 1995a,b; Pedersen et al., 1998; Thunberg et al., 2002). Activation of intramuscular chemonociceptors may be linked to the increases in γ-motor activity, thus leading to increased homonymous Ia and II spindle outputs and α-motoneurons (for a review see Ljubisavljevic and Anastasijevic, 1996). However, the validity of this hypothesis remains questionable in human subjects (Knutson, 2000). According to the recent study of Martin et al. (2006) on elbow muscles, inputs from small muscle afferents from homonymous or antagonist muscles would vary among muscles, with a trend to depress extensor motoneurons and to facilitate flexor ones. As suggested by these authors, rapid movements produced by coordinated flexor and extensor activity might be particularly disturbed by fatigue.

The aim of this study was to examine the influence of eccentric induced-fatigue of elbow flexor muscles on rhythmic extension/flexion movement (RM) performance. In addition to the examination of the actual performance, special emphasis was given to the fatigue-induced changes in the extensor and flexor muscles EMG activity.

## 2. Methods

### 2.1. Subjects

Eight physically healthy 21–33 year old right handed male students volunteered for this study. The subjects were physically active but not involved with regular weight lifting exercise. The subjects' mean height and body mass were 181.0 (SD 6.0) cm, and 77.3 (SD 7.3) kg, respectively. The subjects were not allowed to perform physically heavy activities acute before and during the study period. The subjects were aware of possible risks and discomfort. All of them gave their written informed consent to participate. The study was conducted according to the declaration of Helsinki and was approved by the ethics committee of the University of Jyväskylä, Finland.

### 2.2. Fatigue protocol

Using an isokinetic machine (Komi et al., 2000), the subjects performed with the elbow flexors 100 maximal eccentric actions (EE). Their supinated right forearm was fixed to the lever arm of the machine. Maximal force production of the elbow flexor muscles was measured all along the movement at the wrist level, using a strain gauge transducer attached to the lever arm of the machine. The angular displacement was from 40° to 170° in the eccentric exercise (EE) and the angular velocity was 2 rad $s^{-1}$. Thus, one repetition lasted 1.1 s. All actions were performed