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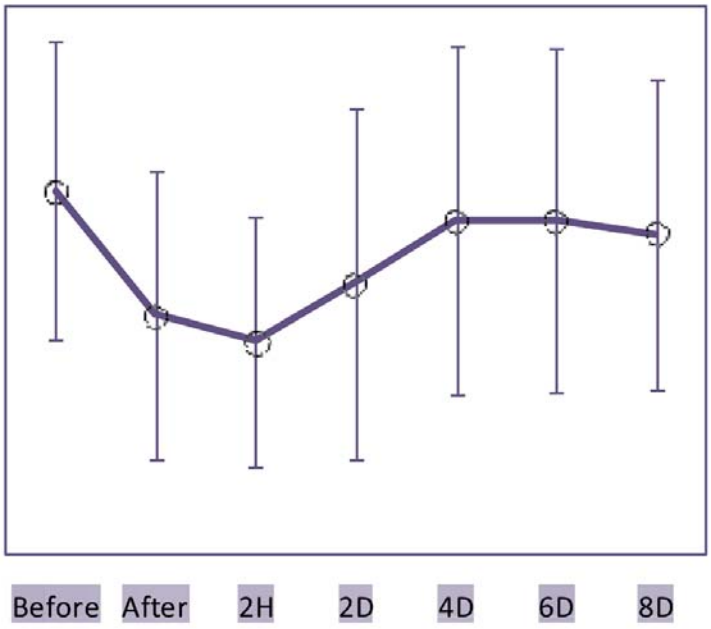
Reijo Bottas

Motor Control of Fast Voluntary Elbow Movements

Exercise-Induced Muscle Damage and Soreness and Learning Interventions



JYVÄSKYLÄN YLIOPISTO

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ABSTRACT

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The aim of the present series of studies was to examine if exercise-induced muscle damage would result in similar phenomena in young and healthy men than those observed in deafferented patients. Moreover, it was examined if voluntary movement range had an effect on responses. Modulations of associated electromyographic (EMG) activity patterns were compared to stretch reflex responses. Through the acute practice of target movement (TM), it was examined if performance improvement was related to decreased terminal oscillation and refinement of associated EMG activity patterns. The exercise did not disturb extension-TM, such as observed in deafferented patient. On the contrary, flexion-TM was acutely deteriorated while rhythmic movement (RM) preserved intact. The performances were changed the most at small elbow angles and at 2 h post-exercise. The changes were associated with the agonists' decreased EMG amplitudes and changed timing (flexion-TM), and with the drop in flexor EMG amplitudes (RM). The reduction of EMG amplitudes was parallel to reduced active stretch reflex amplitudes. The one exercise protocol led to delayed increase of flexor EMG activity in RM. During the acute practice of TM, the increase of antagonist and agonist 2nd bursts relation, the decreased co-activation of these bursts, and the decrease of movement terminal oscillation was associated to improvement of movement time. To conclude, the present eccentric exercises did not have clear joint angle (muscle-length) specific effect on fast voluntary movements' motor control. The central nervous system seemed to use EMG amplitude modulation, in order to optimize the performance during muscle fatigue, damage, and soreness. The acute EMG findings suggested the inhibitory reflex effect originated most likely from group III / IV mechanonociceptors. The delayed modulations of movements' kinetics, kinematics and associated muscle activities referred to adaptive changes in motor control. The practice related modulations of TM reciprocal terminal oscillation suggested the involvement of reflex control refining the co-ordination of antagonistic muscles to enhance joint stiffness.

Key words: muscle damage and soreness, eccentric exercise, motor control, target movement, rhythmic movement, learning

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ORIGINAL PAPERS

This thesis is based on the following papers, which will be referred to by their Roman numerals.

I Bottas, R., Linnamo, V., Nicol, C. & Komi, P.V. 2005. Repeated max imal eccentric actions causes long-lasting disturbances in move ment control. Eur J Appl Physiol 94, 62-69.

II Bottas, R., Nicol,. C., Komi, P.V. & Linnamo, V. 2009. Adaptive changes in rhythmic movement motor control after maximal eccen tric actions. J Electromyograph Kinesiol 19, 347-356.

III Bottas, R., Miettunen, K., Komi, P.V. & Linnamo, V. 2010a. Dis turbed motor control of rhythmic movement at 2 h and delayed after maximal eccentric actions. J Electromyograph Kinesiol 20, 608-618.

IV Bottas, R., Miettunen, K., Komi, P.V. & Linnamo, V. 2010b. Acute (0-2h) and delayed (2-8D) effects of exercise-induced muscle dam age and soreness on elbow target movements. Mot Contr (accepted for publication).

V Bottas, R., Piitulainen, H., Sääkslahti, A., Komi, P.V. & Linnamo, V. 2010c. Refined co-ordination of antagonist and agonists 2nd bursts associated to learning of target movement. Hum Mov Sci (submit ted).

ABBREVIATIONS AND DEFINITIONS

AOMS	acute onset muscle soreness
aEMG	average amplitude of the rectified surface electromyography
BB	biceps brachii
BR	brachioradialis
CE	exercise of elbow flexors 100 maximal concentric actions
CK	creatine kinase
CNS	central nervous system
DOMS	delayed onset muscle soreness
EE	eccentric exercise
EE1	exercise of elbow flexors 100 maximal eccentric actions
EE2	exercise of elbow flexors 50 maximal eccentric actions
EMG	electromyography
EMG area	total volume of the rectified surface electromyography
LA-RM	RM at angles 100-160°
LA-TM	TM at angles 100-160°
MA-RM	RM at angles 80-140°
MA-TM	TM at angles 80-140°
MCA	maximal concentric action
MEA	maximal eccentric action
MIA	maximal isometric action
MT	movement time of TM
peak EMG	peak amplitude of the rectified surface electromyography
RM	elbow rhythmic flexion-extension movement
TB	triceps brachii
TM	target movement
SA-RM	RM at angles 60-120°
SA-TM	TM at angles 60-120°

CONTENTS

1 INTRODUCTION

The exact significance of sensory peripheral feedback in movement control is still somewhat unclear. The importance of somatosensory afferent inputs in the performance of voluntary movements has been demonstrated by numerous deafferentation studies in humans (Nathan & Sears 1960, Rothwell et al. 1982) and in animals (Mott & Sherrington 1895, Taub & Berman 1963, Taub et al. 1975). On the other hand, it has been shown in man that movement can be controlled to a certain degree by central activation without sensory feedback (Lashley 1917, Polit & Bizzi 1979). The triphasic EMG activity pattern of a goal directed target movement (TM) (Hallett et al. 1975, Lestienne 1979) has been suggested to be produced by the central command based on the observation in humans who were lacking proprioceptive and cutaneus feedbacks (e.g. Rothwell et al. 1982, Sanes & Jennings 1984, Forget & Lamarre 1987). However, deafferented patients are known to have difficulties in controlling their TM amplitude due to an improper adjustment in both size and timing of the decelerating antagonist activity (Forget & Lamarre 1987). In addition, proprioceptive feedback has been shown to be necessary to maintain non-preferred rhythmic movement (RM) stationary (Bonnard & Pailhous 1999). The absence of feedback has affected the fine control of antagonistic muscles activity (Nicol et al. 1997), and RM amplitude and frequency (Bonnard & Pailhous 1999). Unaccustomed eccentric actions have been shown to induce reversible ultrastructural muscle damage (e.g. Lieber et al. 2002). The eccentric muscle damage has been connected to reductions both in maximal static (e.g. Clarkson et al. 1992, Howell et al. 1993) and dynamic actions lasting several days or even weeks (e.g. Faulkner et al. 1993, Sayers & Clarkson 2001). Fatiguing eccentric actions have also lead to impaired position (e.g. Skinner et al. 1986, Saxton et al. 1995, Brockett et al. 1997) and force (e.g. Gandevia & McCloskey 1978, Carson et al. 2002) senses and reduced ability to discriminate movement velocity (Pedersen et al. 1999). Impairments of senses have partly been attributed to fatigue-induced disturbance of proprioceptive sensors within both muscle and tendon. In this line, exhaustive SSC exercises - including eccentric muscle actions - have been reported to lead to significant reductions in both passive and active stretch-reflex

amplitude (Nicol et al. 1996, Avela et al. 1999). Within this context, the interest of the 1st phase of the present thesis project (experiment 1, I and II) was to examine if eccentric-type muscle fatigue would result in similar phenomena in TM and RM than those observed in deafferented patients.

It has been suggested that the magnitude of injury may be estimated from the decrease in force a few hours post-exercise (Faulkner et al. 1993). The secondary phase of recovery has been suggested to reflect progressive inflammatory and regenerative process within the damaged muscles, with structural disturbances and muscle soreness 2-4 days post-exercise (e.g. Clarkson & Newham 1995, Lieber & Friden 2002). In addition, muscle damage related mechanical changes such as swelling, reduced joint range of motion (e.g. Clarkson et al. 1992, Whitehead et al. 2001), and increased passive stiffness (e.g. Howell et al. 1993, Whitehead et al. 2003) are very likely to influence dynamic performance for several days. As a consequence of eccentric exercise, the elbow active angle-torque relationship has been found to shift to the right (e.g. Komi & Rusko 1974, Saxton & Donnelly 1996). The changes in triphasic EMG activity pattern of TM with different starting positions (Prodoehl et al. 2003) have been consistent with those seen when the available muscle force has decreased due to muscle fatigue (e.g. Corcos et al. 2002). Thereby, the present thesis project's 2nd phase (experiment 2, III and IV) eccentric exercise protocol was arranged to avoid acute metabolic fatigue and acidosis, where only the effects of muscle damage were expected to appear. Moreover, the aim was to study if the elbow angle had an effect on post-exercise responses.

The existing research on goal directed movement or TM has mainly been concentrated to investigate agonists' movement towards the target. However, the criteria for TM performance are the total movement time (velocity) and the final limb position related to an aimed target (accuracy). The performance improvement (i.e. learning) based on these criterions can be attained with very different profiles of movement kinetics and kinematics. In this regard, the sense of movement halting phase and limb terminal oscillation for the performance result has received a little attention. The triphasic EMG activity pattern of TM has been observed to develop in individuals making the fastest movements (e.g. Cooke & Brown 1990, Brown & Gilleard 1991). With practice the greatest change in performance has been demonstrated to appear over first 40-200 repetitions (e.g. Corcos et al. 1993, Flament et al. 1999). The instructions can accelerate learning (Flament et al. 1999), but using the instruction "always accurate" has been shown to delay the performance improvement (Kempf et al. 2001). It has been shown that training of SSC muscle action can modify a muscle activity pattern (Kyröläinen & Komi 1995, Kyröläinen et al. 1998). The increased muscle activation of power athletes was suggested to be related to enhanced muscle stiffness and elastic potentiation in a stretch shortening cycle task (SSC) (Kyröläinen et al. 1998). The practise of TM was found to be associated with increased bursts amplitudes (e.g. Darling & Cooke 1987b, Gottlieb et al. 1989) and decreased bursts durations (e.g. Brown & Cooke 1981, Gabriel & Boucher 2000). The triphasic EMG pattern involves the co-activation phases (i.e. overlap)

between the bursts (e.g. Berardelli et al. 1996). In general, decreased co-activation of antagonistic muscles is seen as a refinement of motor control. The changes in co-activation are suggested to occur due to spinal level modulations of the antagonistic muscles' reciprocal control (Leger & Milner 2001). In the 3rd and final phase of this thesis project (experiment 3, V), the aim was to examine the performance improvement and associated modulations of triphasic EMG activity pattern.

2 REVIEW OF THE LITERATURE

2.1 Feedback control of voluntary movement

Numerous deafferentation studies have reported various degrees of motor deficit in humans (Nathan & Sears 1960, Rothwell et al. 1982) and in animals (Mott & Sherrington 1895, Taub & Berman 1963, Taub et al. 1975), thus emphasizing the importance of somatosensory afferent input in the performance of voluntary movements. On the other hand, it has been shown in man that movement can be controlled to a certain degree by central activation without sensory feedback (Lashley 1917). Polit & Bizzi (1979) demonstrated that correct elbow movements could be performed by trained monkeys after surgical deafferentation of the upper limb. As the information of muscle afferents is supposed to contribute to position and movement sense (e.g. Roll et al. 1989), the muscle proprioception (e.g. Wadman et al. 1979), and especially the spindle messages of the lengthening muscle (antagonist) (e.g. Capaday & Cook 1983), are thought to play a role in optimizing motor control. In fact, the exact significance of sensory peripheral feedback in the control of voluntary movement remains unclear, even in a simple motor task such as rapid monoarticular movement towards a target.

2.2 Control models

In the length follow-up servo theory, γ- motoneurons had an important role drive through the muscle spindle and their afferents a motor output to achieve the required muscle length in voluntary contraction (e.g. Merton 1953). After that, the motor output was hypothesized to be servo-assisted by fusimoto-driven activity in muscle spindles (Ganit 1975). According to this α-γ co-activation scheme, voluntary movements are produced by combined feedforward command to α- motoneurons and to a servo that controls muscle length through the muscle spindle (e.g. Latash 1998). However, the servo action of the

muscle spindle is mediated by the tonic stretch reflex that needs to be very high to compensate for unexpected length changes by a reflex adjustment in the command to muscle. As muscle spindles monitor the change in muscle length and tendon organs monitor the tendomuscular force, Houk (1979) suggested the facilitative feedback of spindles (tends to increase force) and negative feedback of tendon organs (tends to decrease force) stabilize the stiffness of the muscle. Recently, however, a pure negative feedback effect of tendon organs in rhythmic movements has been questioned (e.g. Grey et al. 2002). In the servo-assistance and in stretch reflex control of voluntary movements several factors are suggested to be involved, i) overall fusimotor output, ii) spindle unloading vs. activation of spindles iii) muscle and contraction history effects on the length of intrafusal fibers iv) pre-motoneuronal excitability of spinal and supraspinal reflex arcs and v) the excitability level of α – motoneuron pool (rew. Hagbarth & Macefield 1995).

For the control of single-joint voluntary movements, an equilibrium-point (EP) hypothesis considers equally both the reflex feedback and the central control signals effects of EMG patterns that result from learning a specific target (joint equilibrium position) (e.g. Feldman 1986, Almeida & Latash 1995). Changes in muscle length activate the muscle (tonic stretch reflex) as a function of its length, and for a certain command the muscle length will change until the muscle force and the load are balanced (e.g. Feldman 1986).

One of the learning approaches (dual-strategy hypothesis) considers triphasic EMG pattern as a reliable reflection of control signals within the central nervous system, and that the brain learns an optimal pattern of muscle forces for a practiced task (Gottlieb et al. 1989, Corcos et al. 1989). More specifically, the "speed-insensitive" strategy is used when movements are made without speed restrictions (Gottlieb et al. 1989) while the "speed-sensitive" strategy is used when movement speed must change e.g. due to changes in accuracy demands (Corcos et al. 1989). The former can be characterized by duration modulation and the latter by amplitude modulation of an excitation to motoneuron pools. However, EMG reflects the activity of α – motoneurons, which is also affected by the reflex effects from proprioception. Therefore, the feedforward central command for a planned movement required to produce a certain torque, has been proposed to involve both a negative feedback component for reflex effects and a component that modulate effects of the reflex mechanisms (Gottlieb 1996).

2.3 Stretch shortening cycle muscle action

Both the TM and RM can be considered as a stretch-shortening cycle (SSC) (e.g. Komi 1984) type performance. In SSC actions, the reflex potentiation will strengthen the muscle activation and manifests itself as a segmented activity pattern (e.g. Dietz et al. 1981, Gollhofer et al. 1987, Nakazawa et al. 2001). In a SSC muscle action, the early muscle activity to decelerate the antagonistic

movement will store elastic energy in the muscle tendon complex, which gives possibilities for the utilization of elastic energy for the subsequent agonist movement acceleration phase. Through the recoil, the potentiated activity contributes to force production (e.g. Cavagna 1977, Komi & Gollhofer 1997). The amount of this reflex assistance will depend on the level of ongoing voluntary activity (e.g. Marsden et al. 1976, Matthews 1986), the load and velocity of active stretch (e.g. Stein et al. 1995, Nakazawa et al. 2001), and the muscle length or joint angle (Yamamoto et al. 2000, McClelland et al. 2001). Earlier observations of voluntary fast movement that include a reversal phase (SSC) have not put a large role on stretch reflexes, but supported the major role of muscle and tendon elasticity (Paulino et al. 2005). It has been suggested that the joint stiffness and damping (impedance) is modulated by use of intrinsic muscle properties and spinal reflexes to meet accuracy needs in target movements (Selen et al. 2006).

2.4 Target movement (TM)

2.4.1 Triphasic EMG activity pattern

A goal directed movement performed as fast and as accurately as possible is generated by a sequence of EMG bursts called a triphasic activity pattern (Hallett et al. 1975, Lestienne 1979). In this pattern the first agonist burst accelerates the movement, the antagonist burst decelerates it, and the second agonist burst makes the final correction to stabilize the limb on the target. In some individuals, the pattern consists of four or more bursts. These latter bursts are typically and clearly minor in amplitude and duration (e.g. Brown & Cooke 1981). In bidirectional movements the activity of antagonistic muscles changes reciprocally, and overlapping co-activity phases of antagonistic muscles are also present (e.g. Berardelli et al. 1996). In the late and post-movement phase the reciprocal activity is generally followed by co-contraction of antagonistic muscles, shown to vary with given instructions (e.g. Yamazaki et al. 1995). In other words, the final limb position is attained by more like tonic muscle activation whereas the movement path or trajectory is determined by the phasic muscle activity (e.g. Darling & Cooke 1987c).

2.4.2 Control of activity pattern

The triphasic activity has been suggested to be produced by the central command based on the observation of this pattern in humans who were lacking proprioceptive and cutaneus feedbacks (Hallett et al. 1975, Rothwell et al. 1982, Sanes & Jennings 1984, Forget & Lamarre 1987). On the other hand, deafferented patients are known to have difficulties in controlling their target movement amplitude due to an improper adjustment in both size and timing of the decelerating antagonist activity (Forget & Lamarre 1987). More recently, the

activity bursts are represented to be initiated serially from the primary motor cortex, but probably precisely timed elsewhere within the motor system (Irlbacher et al. 2006). During the movement execution, peripheral feedback is considered to modulate the two latter bursts (e.g. Ghez & Martin 1982, Shapiro et al. 2004, David et al. 2009) while evidence for the agonist 1st burst modulation is limited (e.g. Bennett 1994). As the antagonistic muscles are activated reciprocally, the control of the elbow antagonistic muscle pair is under reciprocal Ia inhibition (Katz et al. 1991), shown to be dependent on both the antagonistic muscle's activity turn and the joint angle (McClelland et al. 2001). It has been reviewed that, by using proprioceptive information, the CNS can adjust the antagonist excitation to an optimal level producing the torque to counteract the accelerating torque of the agonist, and in turn, antagonist excitation can be compensated by the agonists' 2nd burst (Berardelli et al. 1996).

2.4.3 Performance instructions and demands

The single-joint target movement performance is known to follow the principles of classical speed-accuracy trade off (Fitts 1954). When the task accuracy demands are great the movement velocity or time will be diminished. It has been shown that instructions can accelerate or delay the overall time course of learning of target movement (Flament et al. 1999, Kempf et al. 2001). The instruction to be both "fast and accurate" showed a similar time course of changes in learning both in the elbow (Flament et al. 1999) and in the wrist (Kempf et al. 2001), while the instruction "always accurate" led delayed progress in movement parameters (Kempf et al. 2001). However, the general order of changes that occurred during practice and skill acquisition was suggested not to be influenced by the instructions (i.e. time-elated parameters plateaued before magnitude-related parameters) (Kempf et al. 2001). In the study of Almeida and Latash (1995), where the instruction was to perform "as fast as possible" and the feedback was based on peak velocity and strongly encouraged subjects to move faster, no improvement was found in practiced (loaded) movement due to a very steep learning curve (Almeida & Latash 1995). In the study of Corcos et al. (1993), although improving movement "accuracy" was not the intention of the practice, subjects were able to maintain the accuracy in their movements, which were performed "as fast as possible" over several sessions. Results suggested that a decreased variability in performance can be attained despite increases in movement speed (Corcos et al. 1993).

2.4.4 Development of EMG pattern

The increased velocity of the movement has been found to associate to instruction related "intent" of a subject (Brown & Cooke 1981), and the triphasic EMG pattern has been observed to develop in individuals making the fastest movements (e.g. Gottlieb et al. 1989, Cooke & Brown 1990, Brown & Gilleard 1991, Flament et al. 1999). With practice the increases of measured myoelectric measures have been observed to co-vary with mechanical variables in the same way

as when subjects are asked to intentionally change the speed i.e. generating higher torques to accelerate and decelerate the arm (e.g. Corcos et al. 1989, Corcos et al. 1993). The agonist latency has been shown to be independent of instructions (e.g. Brown & Cooke 1981) and practice (e.g. Corcos et al. 1993). With the instruction-dependent increase of velocity or through practice, the antagonist latency has been observed to shorten (Brown & Cooke 1981, Corcos et al. 1989, Jaric et al. 1993, Almeida & Latash 1995) or elongate (Corcos et al. 1993). The late second agonist burst has been shown to occur progressively earlier, with a shortened duration, as instruction-dependent velocity increased (e.g. Brown & Cooke 1981). With increased peak velocity, the bursts (e.g. Brown & Cooke 1981, Darling & Cooke 1987b, Gottlieb 1998) and agonist early phase amplitudes have been observed to increase (e.g. Gottlieb et al. 1989, Flament et al. 1999) in parallel with the decrease of bursts durations (e.g. Brown & Cooke 1981, Corcos et al. 1993, Gabriel & Boucher 2000). The first two EMG bursts - especially the agonist burst – have been found to become more phasic (e.g. Brown & Cooke 1981). The progress of overlapping co-activation is somewhat less studied but it should be prominent in rapid small amplitude movements (Berardelli et al. 1996). The movement late phase diminished co-contraction of antagonistic muscles has widely been associated to advanced or learned movements. Especially, the increased accuracy has been shown to relate to low co-contraction (e.g. Gribble et al. 2003). On the other hand, during the antagonists' simultaneous isometric fatigue, Missenard et al. (2008) have found the decreased co-contraction to be associated with impaired accuracy of target extensions.

2.4.5 Improvement of performance

Motor learning is characterized by a set of permanent changes in performance parameters and changes in the muscles' neural activity generated to evoke the learned movement. The extensive practice of a fast target movement has commonly led to reduced movement time and improved kinematics (such as peak velocity, acceleration or deceleration) whereas the effect on movement accuracy has been remote. It has been well established that performance improvement follows a curvilinear function with the greatest gains in performance occurring earliest in practice. The practice gradually improves the performance of even the simplest movements over the course of a learning period and leads, eventually, to a plateau (Gottlieb et al. 1988, Gottlieb et al. 1989, Flament et al. 1999, Kempf et al. 2001). Improvement can continue throughout multiple experimental sessions, including hundreds of trials (Corcos et al. 1993), or be insignificant (Almeida & Latash 1995). However, the greatest change in performance has been demonstrated to appear over the first 40-80 (Corcos et al. 1993), 100 (Almeida & Latash 1995) or 200 movements (Flament et al. 1999, Kempf et al. 2001). The kinematic parameters (time-related) of the goal-directed movement have been found to plateau before the myoelectric parameters (Flament et al. 1999). The task outcome responses become more consistent as a result of practice (e.g. Darling & Cooke 1987a), but the variability of EMG associated to well-practiced

movements is controversial (e.g. Darling & Cooke 1987b, Corcos et al. 1993) and proposed to remain always more variable than movements kinematics (Flament et al. 1999).

2.4.6 Muscle force and TM performance

The target movement performance and control has been shown to be linked to force production capacity of muscles (e.g. Jaric 2000, Mirkov et al. 2002, Prodoehl et al. 2003). Firstly, elbow flexion movements made from a more extended starting position have been shown to be slower and to have longer acceleration and deceleration times than movements performed from a more flexed position (Prodoehl et al. 2003). However, the influence of joint angle has been different between flexion and extension movements (e.g. Mirkov et al. 2002).

Secondly, the isometric fatigue of the agonist or antagonist has been shown to increase or decrease, respectively, the movement symmetry ratio (Jaric et al. 1997, Jaric 2000). This has been connected to the muscle's ability to exert force, since the strengthening of muscles have been shown to have a similar but reverse effect on ratio (Jaric 2000). In support of this, both isometric (Corcos et al. 2002) and eccentric (Miles et al. 1997) fatigue protocols of the agonist muscle have been observed to increase the time to movement peak velocity. Moreover, agonist fatigue has led to increased movement time (Miles et al. 1997), lower movement velocity (Jaric et al. 1997, Jaric et al. 1999a, Corcos et al. 2002) and torque (Corcos et al. 2002). Isometric fatigue of antagonists has been shown not to affect (Jaric et al. 1999a) movement velocity, but to decrease peak deceleration (Jaric et al. 1997).

Thirdly, changes in the triphasic activity pattern of target movements with different starting positions (Prodoehl et al. 2003) have been consistent with those seen when the available muscle force has decreased due to muscle fatigue (e.g. Corcos et al. 2002).

Finally, practice induced changes in performance, together with modulations on triphasic activity pattern, have been found to be opposite of fatigue induced changes. Corcos et al. (2002) have concluded that, in the reduced peak agonist EMG, increased duration of agonist and delayed timing of the antagonist served as a partial centrally driven compensation ("central fatigue strategy") of muscle function. Berardelli et al. (1984) who observed the increased agonist 1st burst duration suggested that to be part of normal mechanism for provide needed impulse in fast contractions.

2.5 Rhythmic movement (RM)

Rhythmic movements of limbs 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 cen-

tral 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 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 line with this, proprioceptive feedback has been shown to be necessary to maintain non-preferred rhythmic movement stationary (Bonnard & 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 & Pailhous 1999). During rhythmic movement, the antagonistic muscles show reciprocal activity (e.g. Feldman 1980) with the overlapping co-activity phases between the activity bursts (e.g. Feldman 1980, Nicol et al. 1997). In the fastest movements, the reciprocally activated muscles are totally out of movement phase (Nicol et al. 1997). As with the muscles in TM, the control of antagonistic muscles in RM can be suggested to be refined with the reciprocal Ia inhibition (Katz et al. 1991).

2.6 Muscle fatigue

Muscle fatigue has been defined as any reduction in the force- or velocity-generating capacity of a muscle that is alleviated by the rest (Gandevia 2001). The site of muscle fatigue is due to a failure somewhere in the path that finally results in force production. The potential sites affected during fatiguing muscle actions lie within the central nervous system (CNS) and within the neural transmission from CNS to muscle (central mechanisms), and within muscle (peripheral mechanisms) (e.g. Bigland-Ritchie & Woods 1984). More specifically, the processes that can be impaired are 1) activation of primary cortex 2) CNS directed to the motoneurons 3) motor units and muscles that are activated 4) neuromuscular propagation 5) excitation-contraction coupling 6) availability of metabolic substrates 7) the intracellular milieu 8) the contractile apparatus and 9) muscle blood flow (Bigland-Ritchie 1981).

2.6.1 Central fatigue

A degree of central fatigue has been demonstrated, determined through the twitch interpolation technique, to develop during repeated muscle or sustained contractions (e.g. Gandevia et al. 1996, Smith et al. 2007). In general, the decrease in aEMG or in the discharge frequencies of the motor units has been interpreted as a reduction in neural drive to muscle. As the decline of neural activity likely serve the economy of fatiguing muscle it has been originally named as "muscle wisdom" (Marsden et al. 1983), which on the other hand, has been

questioned (e.g. Fuglevand & Keen 2003, Carpentier et al. 2004). In this regard, during the fatigue, twitch relaxation time has shown to increase with the fusion frequency to decrease. So a motor unit is capable to produce maximal force with lower firing rates (Bigland-Ritchie & Woods 1984). The most plausible mechanisms to cause central fatigue are i) supraspinal fatigue (e.g. Brasil-Neto et al. 1994, Gandevia et al. 1996, Smith et al. 2007), ii) peripheral reflex inhibition (e.g. Garland & McComas 1990) and iii) disfacilitation of the α-motoneuron pool through muscle spindle fatigue (e.g. Bongiovanni & Hagbarth 1990).

2.6.2 Supraspinal fatigue

The limitation of supraspinal centers to excitate muscles has been expressed as an increase in the effort of the task, the appearance of muscle tremor, and the activation of accessory muscles (e.g. Duchateau & Hainaut 1993). Through transcranial magnetic cortical stimulation, supraspinal fatigue has been demonstrated (e.g. Brasil-Neto et al. 1993) and even a very low force (Smith et al. 2007) submaximal (Sögaard et al. 2006), and maximal sustained (e.g. Gandevia et al. 1996) or intermittent contraction (Taylor et al. 2000) have been shown to produce supraspinal fatigue in elbow flexor muscles. Cortical humoral factors, such as neurotransmitter serotonin (5-HT), have been suggested to play a role in central fatigue. As it has been shown to be increased during exercise, its rising level due to diminished dopaminergic activity at some supraspinal site has been speculated to lead to the failure of the task (rev. Davis & Bailey 1997). Other possible candidates influencing fatigue by affecting directly and/or indirectly on neurotransmitter activity are neuromodulators such as cytokines and ammonia (Davis & Bailey 1997). It has been recently reviewed that the activity of fatigue sensitive muscle afferents affecting at a supraspinal level may impair the voluntary drive to muscles (Taylor et al. 2006).

2.6.3 Peripheral reflex inhibition

Metabolic products of muscular actions have been shown to stimulate group III and IV muscle afferents (Kniffki et al. 1978, Mense & Meyer 1988, Rotto & Kaufman 1988). These small muscle afferents are known to make input to inhibitory interneurons capable of inhibiting the Ia terminals (presynaptic inhibition) (Duchateau & Hainaut 1993) and / or the motoneuron pool (Bigland-Ritchie et al. 1986) causing reduced neural drive to muscles. Group III / IV muscle afferents are also stimulated by biochemical substrates which are known to be released following muscle damage (e.g. Rotto & Kaufman 1988).

2.6.4 Disfacilitation

Macefield et al. (1991) have shown, by microneurographic measurements, that the discharge of single muscle spindle afferents does decline during constant-force voluntary contraction lasting at least 60 sec. The indirect finding of altered reflex sensitivity, due to repeated and prolonged passive muscle stretching, has

led to a suggestion of potential mechanical modulation of intrafusal fibers (Avela et al. 1999a). Moreover, as the muscle vibration has been found to counteract the declined MVC motor unit firing rates caused by partial nerve block, the possible change in γ-motoneuron activation may well cause disfacilitation to play some role in the changes in neural drive to muscle (Bongiovanni & Hagbarth 1990).

2.6.5 Peripheral fatigue

The processes distally to the neuromuscular junction affecting the development and/or resumption of muscle fatigue can be called peripheral fatigue mechanisms. In this respect the well known processes associated with excitation-contraction coupling (ECC) are as follows: 1) propagation of the action potential along the sarcolemma and 2) down the T tubule 3) change in conductance of Ca^{2+} of the sarcoplasmic reticulum (SR) 4) transition of Ca^{2+} into SR 5) reuptake of Ca^{2+} by SR 6) binding of Ca^{2+} to troponin 7) interaction of myosin and actin and work done by cross-bridges. However, ECC is suggested not to be the initial site of origin where muscle fatigue develops. Especially in eccentric fatigue, the disturbances in Ca^{2+} movement and efficiency, and in cross-bridge force production, due to degradation of cellular components may play a role as fatigue progresses (Allen 2001). Thus, the ECC related mechanisms of fatigue can be classified as myofibrillar and activation failures. Metabolically, it seems that fatigue during high-intensity exercise is at least partly due to increased cellular H^+ and inorganic P acting at cross-bridges. Elevated H^+ may also have an effect on other sites, such as SR and regulatory proteins, while deletion of muscle glycogen most probably contributes to fatigue during prolonged exercise (e.g. Fitts 1994).

2.7 Eccentric exercise-induced muscle damage and soreness

Intensive and/or unaccustomed eccentric exercises induce ultrastructural muscle damage (e.g. Lieber et al. 2002). The possibility of selective damage of low oxidative capacity muscle fibers has been suggested (e.g. Lieber & Friden 2002). The sequence of damage events has been postulated to include initial and secondary structural injury phases (Faulkner et al. 1993). It has been suggested that the initial muscle injury is mechanical in nature (e.g. Morgan & Allen 1999) and that the magnitude of initial injury may be estimated from the decrease in force at 3 hours post-exercise (Faulkner et al. 1993). It is generally agreed that the muscle soreness is related to mechanical factors causing subcellular disturbances. The amount of strain associated with forced lengthening (e.g. Friden & Lieber 1992) and the disproportionately greater stress per active muscle fibers (e.g. Warren et al. 1993) during eccentric actions are the suggested factors behind the muscle injury.

Armstrong (1991) has categorized the muscle damage process into four stages, the initial damage, the initiation of membrane structure degradation process (following 3-4h), the phagocytic stage characterized by inflammation response, and the regenerative stage (muscle fibers' regeneration) beginning 4-6 days post-exercise.

The first acute inflammation responses, with associated acute soreness, during the first 2–6 h post-exercise have been shown to be related to a secondary injury phase (e.g. MacIntyre et al. 2001) and to be an underlying mechanism in delayed onset muscle soreness (Smith 1991). The secondary phase of recovery is known to reflect progressive inflammatory and regenerative process within the damaged muscles, with structural disturbances and peak muscle soreness 2-4 days post-exercise (e.g. Clarkson & Newham 1995, Friden & Lieber 2001, Lieber & Friden 2002, Cheung et al. 2003). Delayed-onset muscle soreness has been found to associate with increased plasma enzymes, e.g. creatine kinase, myoglobin and protein metabolites, and impairment of muscle function (Armstrong 1991). The muscle damage related acute and delayed mechanical changes causing functional impairments are; swelling, reduced joint range of motion (e.g. Clarkson et al. 1992, Whitehead et al. 2001), and increased passive stiffness (e.g. Howell et al. 1993, Whitehead et al. 2003).

2.8 Eccentric exercise-induced fatigue

As a consequence muscle damage and soreness, the eccentric exercise is shown to cause reductions in maximal static (e.g. Clarkson et al. 1992, Howell et al. 1993, Sbriccoli et al. 2001, Prasartwuth et al. 2006) and maximal dynamic forces that may last for several days or even for weeks (Faulkner et al. 1993, Sayers & Clarkson 2001). Reduced post-exercise force production has been associated with a right shift of the active angle-torque relationship (e.g. Komi & Rusko 1974, Saxton & Donnelly 1996, Jones et al. 1997, Morgan & Allen 1999, Prasartwuth et al. 2006). Both impairment in voluntary activation (Prasartwuth et al. 2005), and disturbances in excitation-contraction coupling (e.g. Warren et al. 1993) and myofibril disruption (e.g. Friden & Lieber 2001) have been suggested to contribute to the length-dependent reduction of voluntary force (Prasartwuth et al. 2006). Voluntary activation has been impaired in the early recovery phase post-exercise, between 2h and 1 day, particularly at the short muscle lengths. Thus, the fast voluntary movement performances and control at small elbow angles have been proposed to be prone to eccentric action impairment (Prasartwuth et al. 2006).

2.9 Exercise induced disturbance of sensory feedback

Fatiguing eccentric exercise may lead to impaired position (Skinner et al. 1986, Saxton et al. 1995, Brockett et al. 1997) and force (Gandevia & McCloskey 1978, Brockett et al. 1997, Carson et al. 2002) senses. Reduced ability to discriminate muscle stretches of varying amplitudes in animals (Pedersen et al. 1998), as well as movement velocity in humans (Pedersen et al. 1999), has been demonstrated. Part of the performance impairments have been attributed to fatigue-induced disturbance of proprioceptive sensors within both muscle and tendon.

The possibility of intrafusal fiber damage has been suggested (Komi & Nicol 1998) to explain disfacilitation, causing the significant reductions in both passive and active stretch-reflex amplitudes after SSC exercises including eccentric muscle actions, (Avela et al. 1999b, Nicol et al. 1996). On the other hand, the exercise-induced direct spindle (Gregory et al. 2004) or tendon organ (Gregory et al. 2002, 2003) damages in man are found to be questionable. The reliability and high sensitivity of Golgi tendon organs in signaling any change in muscle tension after fatiguing eccentric exercise has been demonstrated (Gregory et al. 2002, 2003).

On the other hand, isometric type exercises have been reported to deteriorate the sense of velocity (Jaric et al. 1997) and final position (Jaric et al. 1999) of rapid discrete movements as well. In these two experiments, the agonist fatigue was associated with prominent drops in performance, whereas the antagonist fatigue had no effect.

2.10 The role of group III / IV afferents in eccentric exercise-induced fatigue

The overall muscle recovery process, including its associated muscle pain, is very likely to influence muscle activation both at supraspinal and spinal levels during the acute and delayed recovery phases (review of Gandevia 2001). The nature of eccentric fatigue, favor the hypothesis of an increased sensitivity of small diameter (group III and IV) afferents. Some evidence exists to support their inhibitory (presynaptic) effect on α- motoneurons (e.g. Garland & McComas 1990, Garland 1991, Rossi et al. 1999) and on reduced excitability of the motor cortex (e.g. Le Pera et al. 2001, Martin et al. 2008). In animal models, the sensitization of these afferents via intramuscular injections of either inflammatory or pain substances have been reported to affect the fusimotor system (e.g. Djupsjöbacka et al. 1995a,b, Pedersen et al. 1998, Thunberg et al. 2002) and motoneuron excitation at the spinal level (Martin et al. 2008). Albeit this hypothesis remains questionable in human subjects (Knutson 2000), cheminociceptors activity may be linked to the increases in γ-motor activity, thus leading to increased homonymous spindle (Ia and II) outputs and α-motoneuron excitation

(Ljubisavljevic & Anastasijevic 1996). According to Martin et al. (2006), in 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 (Martin et al. 2006). Recently, the activity of group III and IV muscle afferents produced by hypertonic saline were found to facilitate both antagonistic muscles of the elbow but depress motor cortical cells directed to these muscles (Martin et al. 2008).

3 PURPOSE OF THE STUDY

Since fatiguing eccentric actions are known to cause reversible initial and secondary ultrastructural muscle damage, and lead to impaired position and force senses, the performance and control of fast voluntary movements were proposed to be disturbed by maximal eccentric actions. Moreover, as muscle damage has been associated with both acute and delayed muscle soreness and inflammation, the disturbances on TM and RM were expected to be long-lasting.

The movement range has shown to affect TM velocity and acceleration and the changes in EMG pattern of different starting positions have been consistent with those found in muscle isometric fatigue. Practice induced improvement of performance (e.g. increased velocity) and modulations on TM EMG pattern have been found to be reversed in agonists' by isometric induced fatigue. Moreover, as eccentric actions have been shown both to shift the optimal joint angle for force production and to disturb the performance at small angles, the angle specific effects of maximal eccentric actions on TM and RM performance and control were studied.

The earlier research has nearly bypassed the sense of antagonist and agonists 2nd burst roles for controlling the joint stiffness and TM terminal oscillation. Therefore, the aim was also to study fine modulations of the EMG bursts during the acute leaning.

The purposes of the series of experiments were to examine:

(1) if eccentric-type muscle fatigue would disturb the fast and accurate TMs and RM as has been previously observed in deafferented patients. To clarify the specific eccentric exercise effects, results concerning the TM were compared to those observed in the same subjects after an exhaustive concentric exercise. In addition to the examination of the actual RM performance, special emphasis was given to the fatigue-induced changes in elbow extensor and flexor muscles segmented EMG activity patterns.

(2) both acute and delayed effects of eccentric exercise when the fatigued muscle group worked in TM as an agonist or as an antagonist. Moreover,

the aim was to study if the elbow range of motion has an effect on TM and RM responses. Special emphasis was put on the dissection of the congruence between the elbow flexors activity pattern and their stretch reflex activity. It was hypothesized that the performance and control of movements would be disturbed the most at small elbow angles and especially acutely but also delayed after the exercises, and a reduction of elbow flexors EMG amplitude and change in timing associated to a parallel drop in their active stretch reflex amplitudes would be found acutely post-exercise.

(3) the performance improvement and associated modulations of triphasic EMG activity pattern. It was hypothesized that the decrease of movement terminal oscillation is associated with performance improvement, the co-activation (overlap) of antagonist and agonist 2nd EMG bursts is decreased as the performance develops and the increase of antagonist and agonist 2nd burst EMG relation is associated to acute learning.

4 RESEARCH METHODS

4.1 Subjects

In experiments 1 and 2, the subjects were not allowed to perform physically heavy activities acutely before and during the study period. The subjects were physically active but not involved with regular weight lifting exercise. The subjects in all three experiments were aware of the study ethics and possible risks and discomfort of the study protocol and they all 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.

4.1.1 Experiment 1

Eight physically healthy male students aged 21–33 years volunteered for the study. The subjects' mean height, body mass, and body fat were 181.0 (SD 6.0) cm, 77.3 (7.3) kg, and 11.9 (4.2) %, respectively.

4.1.2 Experiment 2

Ten physically healthy right handed males volunteered for this study. Selection criteria were employed as all testing apparatus were designed for right handed use and because the neuromuscular system of a subject had to be intact. The subjects' mean age, height, body mass, and body fat were 25.3 (SD: 1.7) years, 179.3 (SD: 7.4) cm, 78.0 (SD: 7.0) kg, and 15.8 (SD: 3.3) %, respectively.

4.1.3 Experiment 3

Altogether ten (10) physically healthy male students were recruited and volunteered for this study. Due to limitations of test apparatus design, all subjects were right handed. They were randomly divided into two groups, "accuracy"

the group (A-group) (26.4±3.6years, 182.0±6.5cm, 80.0±16.1kg) and the "velocity" group (V-group) (26.2±3.9years, 181.2±2.6cm, 77.2±8.0kg). The subjects were not allowed to perform physically heavy activities before the study.

4.2 The target movement (TM) protocol

4.2.1 Experiments 1 and 2

The TM task was performed with a specific apparatus equipped with a potentiometer constructed in the Neuromuscular Research Center of Jyväskylä University. As shown in Fig. 1, the subject was in a sitting position with the right forearm fixed to the lever arm of the apparatus in the horizontal plane above the protractor. Horizontal plane was chosen to avoid the effect of the gravity. The shoulder joint was abducted around 80° and the forearm was in a semi-supinated position (experiments 1 and 2) with the wrist stiffened by a spatula (experiment 2). As compared to the fully extended (180°) elbow joint position, the flexion movement was performed from 140 to 80°, and the extension movement from 80 to 140° (experiment 1). In experiment 2, the subject performed first the flexion-TMs followed by the extension-TMs. In experiment 2, TMs of 60° amplitude were performed in random order starting at small elbow angles (SA, 60 - 120°), followed by medium (MA, 80 - 140°), and then large elbow angles (LA, 100 - 160°). The subject performed TM with a visual online feedback of the movement from a television monitor (Fig. 1).

A total of 20 extension-TM followed by 20 flexion-TM (experiment 1) or 10 extension-TM followed by 10 flexion-TM (experiment 2) were performed at a preferred pace and to have a pause between movements as long as needed for the optimal performance. The subjects were instructed to perform the movement as fast and as accurately as possible. The TM tasks were practiced on the same day prior to experimental trials for as long as needed (approx. 30-50 repetitions) to achieve a plateau in the acute learning.

The TM tests were conducted before, immediately after, 0.5 h after, as well as 2 days and 7 days after EE1 and CE (experiment 1). In experiment 2, TM tests were conducted before, immediately after, 2 h after, as well as 2 days, 4 days, 6 days and 8 days after EE2.

4.2.2 Experiment 3

First the flexion-TM and then the extension-TM learning exercises were performed with the same apparatus as in experiments 1 and 2. The order of two TMs practice was not randomized due to possible learning effects (positive transfer) (e.g. Ilic et al. 1998) and due to the low number of subjects in both groups. The subjects' body and upper arm positioning were the same as in experiment 2.

The subject performed a directive scaled TM of 60° (at the middle of elbow joint operational centre) towards the 6° width target zone with visual feedback of the movement and its' success from a television monitor (Fig. 2). Both the flexion-TM and extension-TM consisted of 10 sets of 10 movements performed at the subject's own pace and with about two minutes rest between sets.

Subjects in both experimental groups were instructed that the final goal of the TM practice is to learn to move and stop the arm on the target zone as fast and as accurate as possible. Moreover, they were all instructed not to do any adjustment to the final position of the arm. The more detailed instructions to A-group (accuracy group) were as follows, "perform always accurate and after a successful movement you may speed up the performance without sacrificing accuracy" (Kempf et al. 2001). After an unsuccessful movement, the subject was reminded to perform always accurate. The detailed instructions to V-group (velocity group) were as follows, "perform always as fast and as accurate as possible". After an unsuccessful movement, depending on the reason for failure, the subject was reminded not to sacrifice either the TM velocity or accuracy. Before the practice all subjects received a demonstration of the TM task.

4.3 The rhythmic extension / flexion movement (RM) protocol

The RM test was performed in the same apparatus as TM (Fig. 1). Also, the subject's body and arm positioning were the same. In experiment 1, the subject performed a directive scaled RM of 60° between boundary marks at 80° and 140°. In experiment 2, the subject performed intentional constrained RM of 60° amplitude in random order at small, medium and large elbow angles; 60–120° (SA-RM), 80–140° (MA-RM) and 100–160° (LA-RM), respectively. Simultaneously with this accuracy demand, the subject was instructed to accelerate the bi-directional movement slowly to maintain correct rhythm and, eventually, perform it at the highest possible speed. The test was terminated after the subject had achieved and maintained the maximal tempo of his movement for at least five (experiment 1) or ten (experiment 2) cycles, and the movement was seen to become slower. RM test was conducted before, immediately after, 0.5 h later, as well as 2 days and 7 days after the fatiguing exercises (experiment 1). In experiment 2, RM test was conducted before, immediately after, 2 h after, as well as 2 days, 4 days, 6 days and 8 days after the exercise. Immediately post-exercise, other measurements and the set-up of the subject into RM apparatus took about 3 min, however, all participants were tested within an identical window of time.

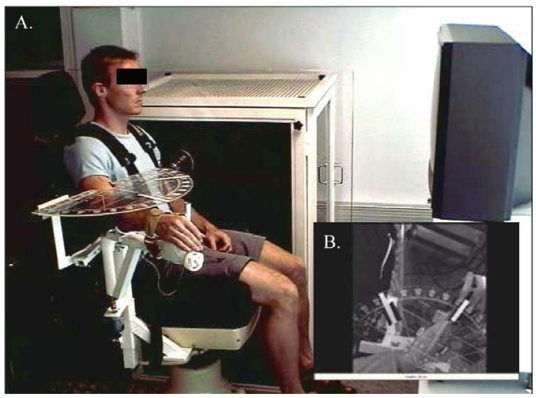

FIGURE 1 A: Arrangements of the tests for the Target (TM) and rhythmic movement (RM) (B). The subject performed RM with visual feedback of the movement from a television monitor

4.4 Eccentric exercise protocol

4.4.1 Experiment 1

At 4-week intervals, the subjects performed 100 maximal eccentric (EE1) and concentric elbow flexions (CE) in the vertical plane with an isokinetic machine (Komi et al. 2000) (Fig. 2). Their supinated right forearm was fixed to the lever arm of the machine. The selection of the used plane and the amount of arm supination were based on our earlier experience to optimally activate and load elbow flexors (e.g. Komi et al. 1972, Komi et al. 2000). The axis of machine lever arm corresponded to the rotational axis of the elbow joint. The force applied to the wrist for elbow flexion and extension was measured by a strain gauge transducer. The movement range was from 40° to 170° in eccentric exercise (EE1) and from 170° to 40° in concentric exercise (CE). The angular velocity was $120° \cdot s^{-1}$ ($2 \text{ rad} \cdot s^{-1}$) in both exercises. Thus, one repetition lasted ~1.1 s. All actions were performed maximally at 2 s intervals. From this time period, approximately 0.5 s was used for maximal isometric pre-activation, which was reached prior to the start of the next eccentric movement. Total duration of the maximal exercise averaged 5 min with almost 3 min of actual work.

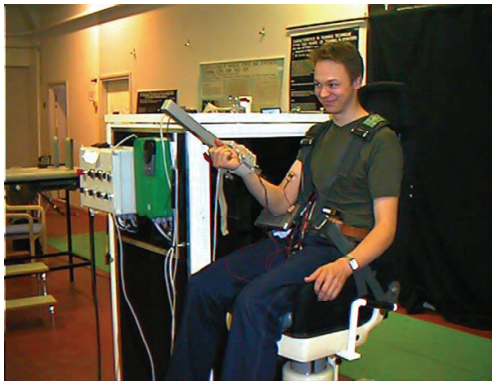

FIGURE 2 The eccentric exercise test arrangement in an isokinetic machine (Komi et al. 2000)

4.4.2 Experiment 2

After a detailed instruction and warm-up trials of the eccentric muscle actions, the subject performed 50 maximal eccentric elbow flexions (EE2) with an isokinetic (constant velocity) machine (Komi et al. 2000) in the vertical plane (Fig. 2). The movement range during eccentric exercise (EE2) was from 50° to 170°, wherein 180° position indicated full elbow extension. The angular velocity was $120° \cdot s^{-1}$ ($2 \text{ rad} \cdot s^{-1}$). Thus, one repetition lasted approximately 1.05 s. All actions were performed at 15 s intervals (duty cycle of 1:15). During this time period, the forearm was passively returned back to 50° starting angle and approximately 0.5 s was used for maximal isometric pre-activation before the next repetition. The subject was repeatedly encouraged to perform each action with maximal effort. The total exercise duration averaged about 13 min with about 1 min 20 s of actual work.

4.5 Maximal force tests

Maximal voluntary force tests of eccentric (MEA) (experiment 1 and 2), concentric (MCA) (experiment 1), and isometric (experiment 2) actions of the muscle group – including two to three actions per condition – were performed before, immediately after, half an hour, 2 and 7 days after (experiment 1), or before, immediately after, 2 h after, 2 days, 4 days, 6 days and 8 days (experiment 2) after the fatiguing exercise. The force of maximal isometric action (MIA) of elbow flexors was tested at 90°, 110° and 130° (experiment 2).

The measured action (eccentric, concentric or isometric) was always performed with full preactivation (maximal isometric phase) to follow the principles introduced by Edman (1978) for isolated muscle fibres (or sarcomeres), and by Komi & Rusko (1974) for human forearm flexors. The last maximal eccentric action of the fatiguing EE or the last maximal concentric action of the

fatiguing CE was used as a measure of the eccentric or concentric performance ''immediately after'' the exercise.

4.6 Stretch reflex measurements

In experiment 2, active and passive stretch reflex tests on elbow flexors were performed with the same machine as the exercise. Stretches of 20° amplitude with 0.110 s stretching time – 240° · s^{-1} (4 · $rads^{-1}$) velocity and 600° · s^{-2} (100 rad · s^{-2}) acceleration – were applied to the elbow at six joint angles, 50°, 70°, 90°, 110°, 130° and 150°. A pre-stretch force of 20% / MIA (maximal isometric force at that test point) was used in the ''active" stretch reflex tests. The subject was instructed to maintain constant force – which he saw from the oscilloscope screen – throughout the stretch perturbation. In the passive reflex test, the subject was blindfolded and he was instructed to keep the forearm relaxed. The stretches were applied with irregular intervals of 5-10 s starting from the most flexed joint position. The order of passive and active tests was kept constant, starting with the passive test.

4.7 Data recordings and analysis

4.7.1 Electromyography (EMG)

Surface electromyographic activity (EMG) was recorded from the biceps brachii (BB) (experiments 1, 2 and 3), brachioradialis (BR) (experiments 2 and 3) and triceps brachii (TB) (experiments 1, 2 and 3) muscles of the right upper arm using bipolar skin electrodes (Beckman miniature-size). According to recommendations of SENIAM (1999), the electrodes, with a 20-mm interelectrode distance, were placed longitudinally on the muscle belly and distally from the motor point, which in experiment 3 was assessed with a separate array electrode (Piitulainen et al. 2009). During the acute – 0.5 or 2 h – recovery period the electrodes were not removed. The electrode position was marked on the skin to ensure the same location during the whole experimental period.

EMG signals were recorded telemetrically (Glonner Biomes 2000, Germany, bandwidth 3–360 Hz) with a sampling frequency of 2000 Hz (experiment 1). In experiment 2, EMG signals were recorded (EISA 16-2, Freiburg, Germany) with bandwidth of 10–1000 Hz/3 db and a sampling frequency of 2000 Hz. In experiment 3, all EMG signals were sampled at 2000 Hz and converted to digital data by a 16-bit analogue to digital converter (Power 1401, CED Ltd. Cambridge, England) and stored on a computer hard disk. EMG signals were pre amplified (100-fold) and high-pass filtered (3-dB cutoff at 10 Hz) with a pre amplifier (NeuroLog 824, Digitimer Ltd. Hertfordshire, England).

4.7.2 TM analysis

In experiment 1, EMG signals were full-wave rectified and averaged over 20 repetitions at every test point. The averaged EMG curves and the movement trajectory curve were smoothed with a one direction sliding average of 50 ms for further analysis. The EMG variables included EMG burst peak amplitude and duration. The kinematic data included peak velocity and peak amplitude of main movement (extension in extension TM and flexion in flexion TM) and timing values, as well as movement oscillation amplitude over the target zone. The time delay of the EMG and kinematics was determined from the onset of movement (more detailed in paper I).

In experiment 2, for EMG analysis, signals were full-wave rectified and filtered (low-pass, Butterworth, with a 50 Hz cut-off frequency) (e.g. Ervilha et al. 2004a). The peak EMG (filtered data), activity timing (EMG burst onset and end) and durations of the all three EMG bursts were determined from the averaged (10 trials) curve (Fig. 3). In addition, co-activation (overlap) duration of antagonistic muscles and average EMG amplitude during the co-activation phase in both activity turns (BB > TB and TB > BB) were defined. Altogether ten movements with similar type movement trajectory were averaged for further analysis. The onset of the movement (0.5° increase from baseline) was taken as a reference point for analysis (Fig. 3). The arm arrival at a steady 2° width window marked the movement end. The determined movement performance-parameters were the time to target (movement time) and deviation from target (movement accuracy) (Fig. 3). Moreover, other determined parameters were as follows: arm total oscillation (travelled distance of the arm between the reversals), time to peak velocity, symmetry ratio (acceleration/deceleration time), and movement peak velocity (Fig. 3) (see details in original papers III and IV).

In experiment 3, for EMG analysis, signals were full-wave rectified and low pass smoothened (Signal, version 2.0 analysis program). Target movements were analyzed (Signal, version 2.0 analysis program) one by one and then averaged over ten trials. The beginning of the movement (0.5° increase from baseline) was taken as a reference point for analysis (Fig. 3). The arm trajectory plateau at a steady 2° width "window" marked the achievement of the target. The velocity curve of TM was applied in timing analysis. In the EMG timing analysis, the onset of a muscle EMG was the point beyond the value was continuously rising and the lowest EMG value between the bursts marked the end and onset of the next burst. The determined movement *performance-parameters* were time to target (s) and deviation from target (^{o}) (same as in Fig. 3). Moreover, other determined parameters were as follows: main movement (extension in Extension TM and flexion in flexion TM) overshooting of the target (^{o}), oscillation amplitude (^{o}) (travelled distance of forearm reciprocal movements) (*amplitude-parameters*), time to peak velocity (s), time of main movement (s), symmetry ratio of main movement, oscillation time (s) (time for forearm reciprocal movements) (*time-parameters*), and peak velocity of main movement ($^{o} \cdot s^{-1}$). EMG parameters were as follows; latency of activity onset (s), burst duration (s), peak

amplitude and area of EMG burst, relation of bursts EMG area and co-activation (i.e. overlap) (s) of the antagonistic muscle (partly seen in Fig. 3, see details in paper V).

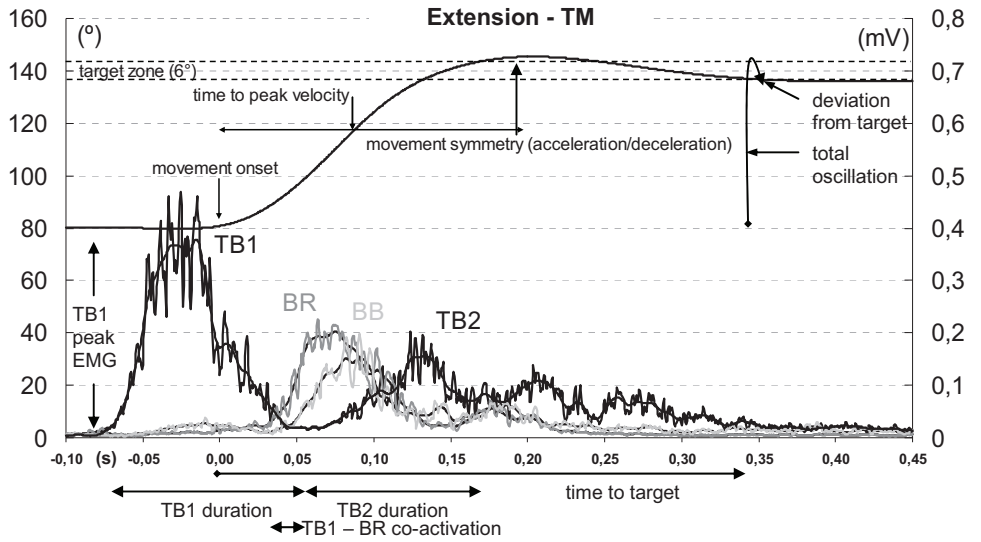

FIGURE 3 Average curve of ten (10) medium angle (80-140°) extension TM with associated EMG of agonist (TB1) and antagonists (BB and BR) muscles for one subject. Solid black lines = smoothed EMG curves, TB1 and TB2 = triceps brachii 1st and 2nd EMG bursts, BB = biceps brachii EMG burst, BR = brachioradialis EMG burst. TB1 and TB2 duration = duration of TB bursts. TB1-BR co-activation = duration of the overlap of these EMG bursts. Movement onset = this point served as a reference point for analysis. Deviation from target = amount of error (degrees) from target angle. Total oscillation = (the distance of the arm between the reversals). Time to reach peak velocity = point where movement achieved its maximum. Movement symmetry (acceleration/deceleration) = time relation of main movement (extension) acceleration and deceleration phases. Target zone (6°) = area and its wideness seen by a performer.

4.7.3 RM analysis

In experiment 1, for each subject, at the beginning of the steady phase of rhythmic movement, five successive cycles of approximately the same length (±0.05 s) were averaged for further analysis (Fig. 4). In experiment 2, from the beginning of the RM steady phase altogether ten successive cycles, of approximately the same length, were averaged for further analysis.

In both experiments, the beginning of the extension movement phase – minimum elbow angle – was taken as a reference point for analysis and was used with the end of the flexion movement to determine the whole extension/flexion cycle. Both extension and flexion movement phases were analyzed separately (Fig. 4).

In experiment 1 and 2, for the RM EMG analysis, signals were full-wave rectified and filtered (low-pass, Butterworth, with a 50 Hz cut-off frequency) (e.g. Ervilha et al. 2004a). In addition to the calculation of the average (rectified data) and the maximal values for the whole EMG burst, the same calculations were applied to visually separated EMG segments named as S0, S1, S2, and S3 (Fig. 4). The beginning of muscle activity – the S0 latency – was used as the onset for S1 latencies and durations, as well as for the total burst duration (Fig. 4).

In both experiments, the time duration that antagonistic muscles (flexor vs. extensor) EMG overlapped (overlap duration) with each other were used to quantify the level of co-activation. This overlap duration was determined for both activation phases, in which TB became activated either prior to BB or after it. The analyzed kinetic / kinematic variables for both movement phases were as follows: movement amplitude and actual elbow angles, movement peak velocity, time to peak velocity and movement deceleration time (acceleration and deceleration symmetry ratio), and movement duration.

In experiment 1, to reflect potential interaction between EMG bursts and the movement, the time delay from the onset of antagonist activity to the time to peak velocity (braking latency) and to the beginning of the subsequent agonistic movement (flexion or extension latency) were also determined (Fig. 4).

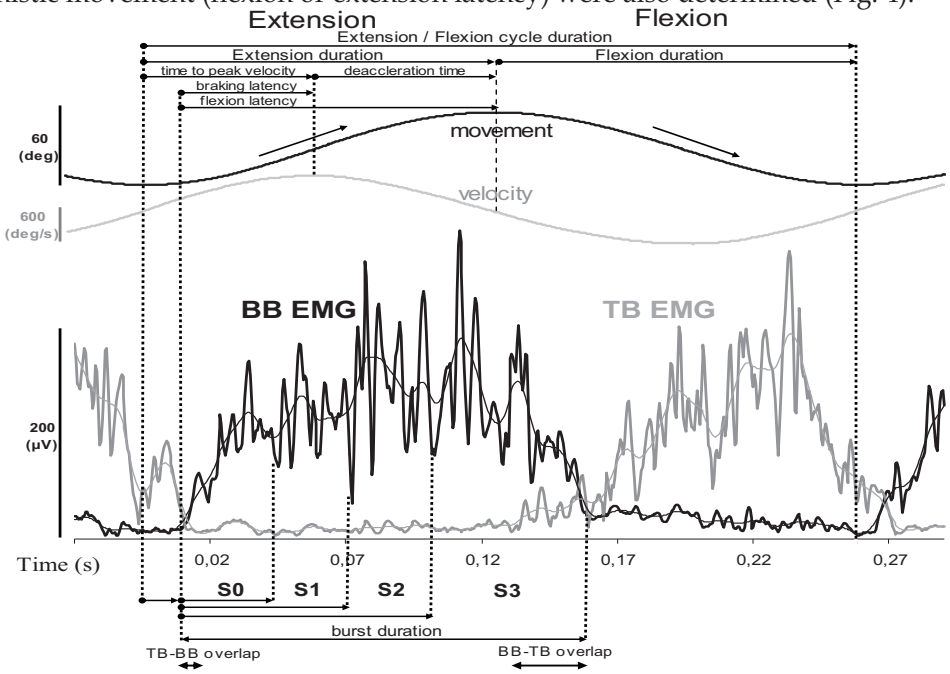

FIGURE 4 Variables of the elbow RM (average of 5 cycles, n = 1). BB = biceps brachii. TB = triceps brachii (Solid thick curves are corresponding filtered EMG curves). Arrows represent the latency of different variables. S0–S3 = segmen tation of EMG burst. TB-BB and BB-TB overlap = quantity of co-activation. Braking latency = time from onset of antagonist activity to time to peak velocity. Flexion latency = time to beginning of the subsequent agonistic movement (details in original papers II and III).

4.7.4 Maximal isometric and eccentric force

In experiment 1, both maximal eccentric and concentric force was analyzed together with associated muscle activity over five 26° sections throughout the movement range of 130°, and also averaged for the whole movement range.

In experiment 2, maximal eccentric force of MEA was analyzed for the whole movement range and over the six 20° sections (175 ms) throughout the movement range (50° - 170°). The associated muscle activity (aEMG) was determined only for the whole movement range. Maximal forces of MIA together with the associated aEMG were analysed for 1000 ms steady force production period (around peak maximal force).

4.7.5 Stretch reflex measurements

In the analysis of the active elbow flexors stretch reflex test, a 100 ms period of the aEMG before the onset of the stretch (1st arrow in Fig.5) was used as background activity (BGA) (Fig. 5). A clear EMG amplitude change served as the onset of the later reflex components (2nd arrow in Fig. 5). The genuine latency and average EMG amplitude of rectified reflex components M1 and M2 were determined from every stretch response. The average value of three stretches at 90°, 110° and 130° elbow angles in relation to BGA were calculated. In the passive condition, the responses of stretches at 50°, 70°, 90°, 110°, 130° and 150° were analyzed.

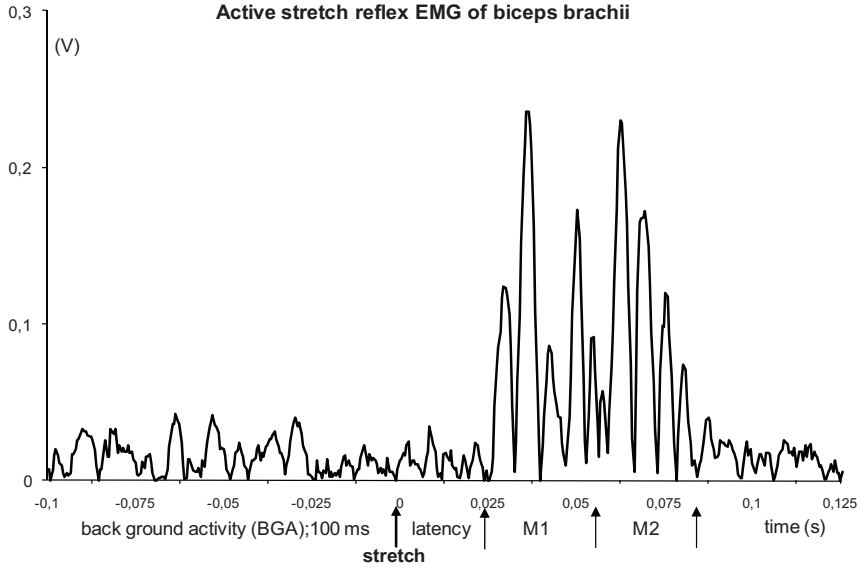

FIGURE 5 Example of biceps brachii active stretch reflex EMG activity for one subject at elbow angle of 90°. In the EMG analysis the genuine latencies (from stretch = 1st arrow) and average EMG amplitudes of reflex components (M1, M2) were determined. The average values of three stretches at in relation to BGA (at 20% force of MIA) were calculated.

4.7.6 Blood samples

Blood lactate concentration [B-La] was determined (Biochemica Boehringer GmbH, Germany, experiment 1 and Lactate Pro, Arkray, Inc. Kyoto, Japan, experiment 2) from fingertip blood samples drawn before and immediately after the exercise.

For the determination of the serum creatine kinase activity (s-CK) (a kit of Boehringer Mannheim, Germany), blood samples were drawn from the ulnar vein. In experiment 1, s-CK was determined before, immediately after, 0.5 h after, as well as 2 and 7 days after EE1 and CE. In experiment 2, s-CK blood samples were drawn before, immediately after, 2 h, 2 days, 4 days, 6 days and 8 days after EE2.

4.7.7 Muscle soreness

In experiment 1, subjects reported the daily soreness level (experienced in everyday life) of their exercised arm muscles. The pain sensation was rated on a scale from 0 (no pain) to 5 (intolerable pain).

In experiment 2, the subjectively experienced muscle soreness was determined by the Visual Analogue Scale (VAS) method (Nosaka & Clarkson 1996).

4.7.8 Muscle swelling

Arm circumference – for evaluating swelling – was measured medially and distally over the muscle belly, 80 mm and 40 mm, respectively, proximal to the elbow joint (Nosaka & Clarkson 1996). During the measurement, the subject stood upright and his relaxed arm hung loose on the side of the body with the forearm supinated.

4.7.9 Muscle passive stiffness

The elbow free relaxed joint angle (RANG) was measured when the subject stood with his relaxed arm hung loose on the side of the body with the forearm supinated. At the same body position, both the elbow free relaxed joint angle (RANG) and the flexed joint angle (FANG) during unloaded elbow flexion was determined to represent the passive stiffness (ROM=RANG-FANG; range of movement) of elbow joint (Nosaka & Clarkson 1996). ROM were determined and measured from both upper arms.

4.8 Statistics

In experiment 1 (I and II), data were analyzed by multivariate analyses of variance (MANOVA with method unique) with repeated measures for special cases using an SPSS computer analysis program. Paired t-tests, as post-hoc ana-

lyses, were conducted. Multiple comparisons were made with a significance level of $P < .05$.

In experiment 1 and 2 (III and IV), data were analyzed by a multivariate analyses of variance (MANOVA) by using an SPSS computer analysis program. The tests of within-subjects (contrast simple) and between-subjects (contrast repeated) effects were applied in the analysis of repeated measures for dependent variables. Multiple comparisons were made with a significance level of $P < .05$. The paired T-test was used as post-hoc test. The Pearson correlation r value was calculated between selected variables.

In experiment 3 (V), the analyses of variance (two-way ANOVA) using an SPSS computer analysis program method of General Linear Model with tests of within-subject and between-subjects factors for repeated measurements was used. Huynh-Feldt ẽ in tests of within-subject effects and Bonferroni adjustment for multiple pairwise comparisons was used with a significance level of $P < .05$. T-tests for independent variables were made as post-hoc tests. The Pearson correlation was calculated between appropriate variables.

5 RESULTS

5.1 Maximal voluntary actions

Experiment 1

As shown in Figure 6, eccentric exercise (EE1) led to a large acute decline in forces both of MEA (maximal eccentric action) (average -53 ± 10%, P < .001) and MCA (maximal concentric action) (average -38 ± 11%, P < .001), with a subsequent partial recovery 0.5 h later that remained incomplete until day 7 post-exercise. No significant change was observed in the optimal angle for maximal force production.

The concentric fatiguing exercise (CE) led to similar acute reductions in forces (MEA: -31 ± 6%; P < .001, and MCA: -50 ± 7%; P < .001), but only the recovery of the force of MCA was delayed by 7 days (-12 ± 7%, P < .05).

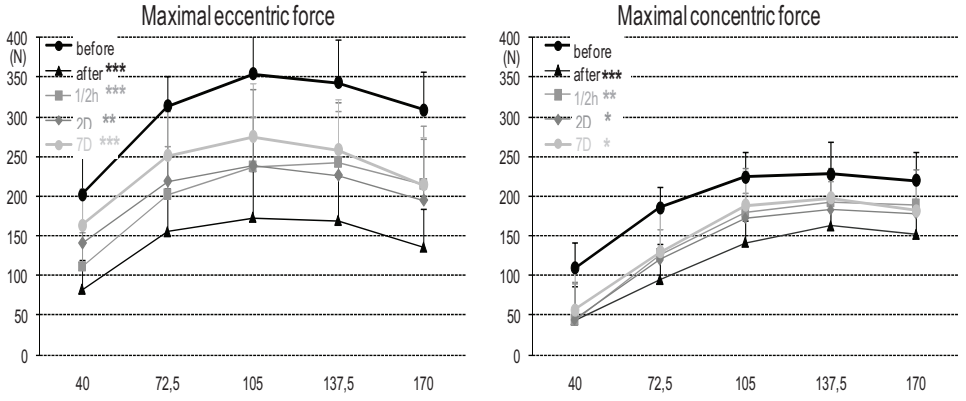

FIGURE 6 Changes in forces of MEA (maximal eccentric action) (left) and MCA (maximal concentric action) (right) of elbow flexors before the exercise (before), immediately after (after), 0.5 h after (1/2h), 2 days after (2D) and 7 days after (7D) EE1 (experiment 1). * P<.05, ** P<.01, ***P<.001.

The aEMG of the BB muscle in MCA was significantly decreased (-12 ± 12%, P < .05) immediately after EE1 for the whole movement range, while after CE no changes were observed in MEA.

Experiment 2

Eccentric exercise (EE2) caused long-lasting deterioration of elbow flexor maximal force. The acute drop in the force of MEA was -28 ± 10% (Fig. 7). Parallel and immediate decreases of force in MIAs (maximal isometric actions) at 90°, 110° and 130° angles were -23 ± 11%, -23 ± 14% and -25 ± 14%, respectively (Fig. 8). Hardly any recovery took place within the next 2 h. The MIA recovered within 4 days, but was again lower 8 days post-exercise compared to before value.

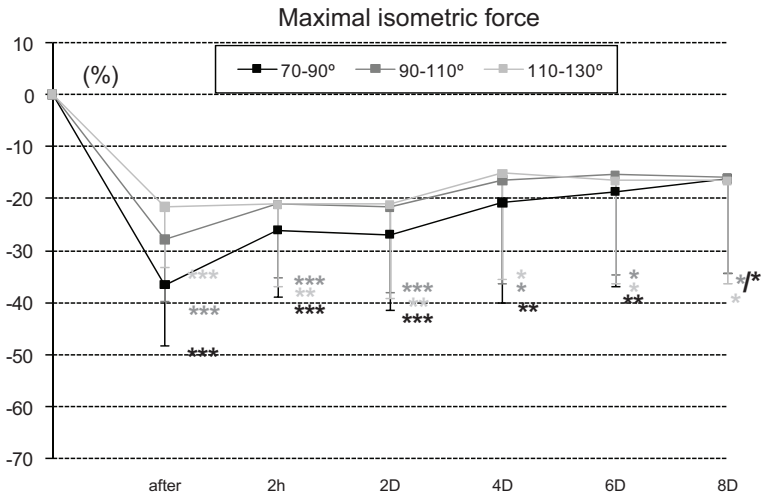

FIGURE 7 Relative change of MEA (maximal isometric action) force of elbow flexors at different elbow angles (90°, 110°, 130°) immediately after (after), 2 hours (2h), 2 days (2D), 4 days (4D), 6 days (6D) and 8 days (8D) after EE2. * P<.05, ** P<.01, ***P<.001.

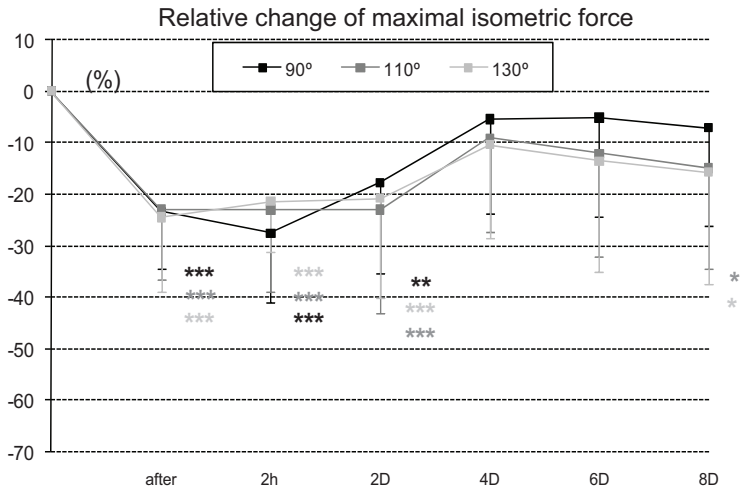

FIGURE 8 Relative change of MIA force of elbow flexors at different elbow angles (90°, 110°, 130°) immediately after (after), 2 hours (2h), 2 days (2D), 4 days (4D), 6 days (6D) and 8 days (8D) after EE2. * P<.05, ** P<.01, ***P<.001.

Fatigued BB muscle aEMG decreased immediately after EE2 at all three MIA at 90°, 110° and 130° angles. While it recovered at the 2 h point, BB aEMG was decreased again on day 2 (P<.05) in MIA-90° and MIA-110° (Fig. 9).

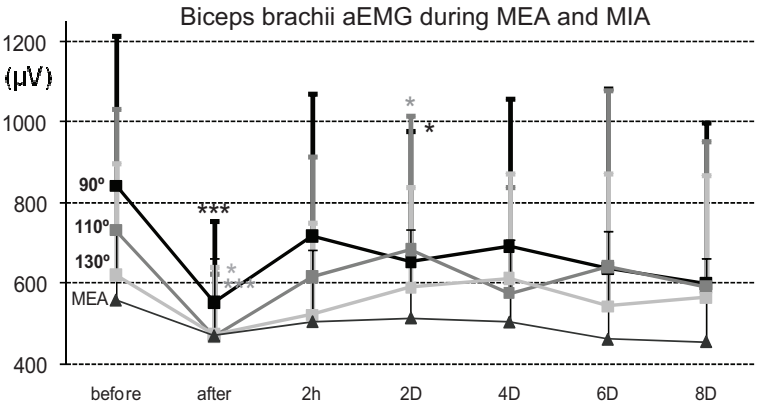

FIGURE 9 Biceps brachii aEMG during MEA and MIA (90°, 110°, 130°) before, imme diately after (after), 2 h (2 h), 2 days (2D), 4 days (4D) and 8 days (8D) after EE2. *P < .05, **P < .01, ***P < .001.

In experiment 1, the whole movement range BB aEMG/force ratio of MEA was significantly increased acutely and delayed (day 7) after EE1 (Fig. 10). The BB aEMG/force ratio of MCA over the whole movement was significantly in-

creased (P < .05) only in the acute recovery period, reflecting greater changes in the smallest elbow angles (Fig. 10).

In experiment 2, the BB EMG/force relation of MEA increased 2 h after EE2 (P<.05).

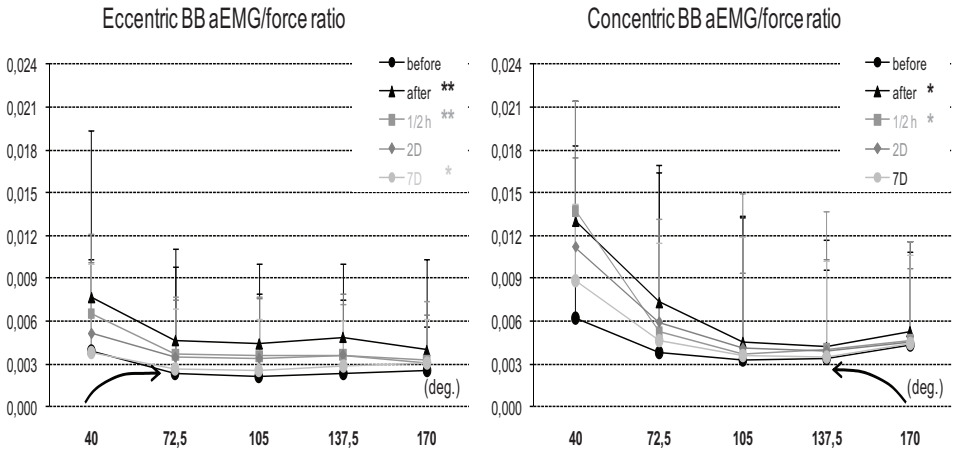

FIGURE 10 Biceps brachii aEMG during MEA (left) and MCA (right) before the exercise (before), immediately after (after), 0.5 h after (1/2h), 2 days after (2D) and 7 days after EE1 (7D) *P < .05, **P < .01, ***P < .001, different than before value.

In experiment 2, the maximal force of MIA of non-exercised elbow extensors at 110º was decreased 2 h (-12±13%, P<.01) and 2 days (-8±11%, P<.05) after EE2. A parallel drop of TB aEMG was 18±22% (P<.05) at the 2 h point and -23±28% (P<.05) on day 2.

5.2 Blood lactate and serum creatine kinase

In experiment 1, blood lactate concentrations were similar after EE1 and CE (P<.001) (Fig. 11). Only EE1 led to muscle soreness and elevated serum CK activity that peaked on day 2 and day 7 (Fig. 11).

In experiment 2, as expected, the present eccentric exercise protocol (EE2) of elbow flexors did not elevate the post-exercise blood lactate concentration which remained the same as the resting value (1.2 ± 0.4 mmol/l vs. 1.3 ± 0.5 mmol/l).

The averaged serum creatine kinase concentration peaked on day 4 after EE2 (Fig. 12).

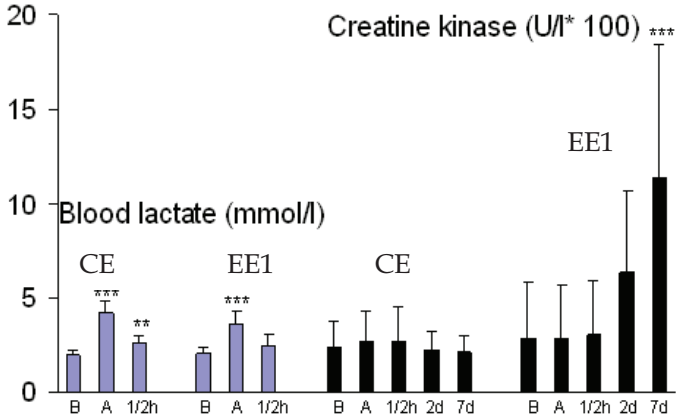

FIGURE 11 Blood lactate (left) concentration and serum creatine kinase (right) activity before and after EE1 and CE. Before (B), immediately after (A), 0.5 h later (1/2h), 2 days after (2d), 7 days (7d) after EE1. **P<0.01, ***P<0.001.

5.3 Muscle soreness

In experiment 1, differently to CE, only EE1 led to muscle soreness that peaked on day 2 post-exercise.

In experiment 2, acute onset muscle soreness (AOMS) was perceived in the exercised right arm elbow flexors during the early recovery period of 2 h and delayed onset muscle soreness (DOMS) peaked on day 2 after EE2 (Fig. 12).

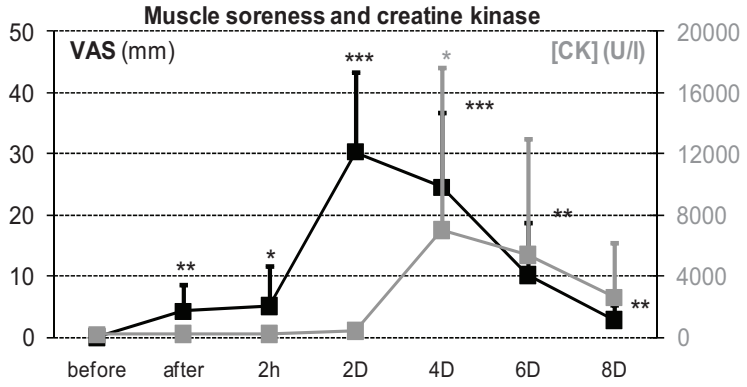

FIGURE 12 Muscle soreness (black) and serum creatine kinase (grey) concentration (CK) before, imme diately (after), 2 h (2 h), 2 days (2D), 4 days (4D), 6 days (6D) and 8 days (8D) after EE2. (n = 9). *P < .05, **P < .01, ***P < .001, different than before value. VAS = Visual Analogue Scale method (Nosaka & Clarkson, 1996).

5.4 Muscle passive stiffness

In experiment 2, the free range of movement (ROM) of the right elbow joint was the smallest 2 h after EE2 (118±11º) and it did not return to normal during the 8 day recovery period (132 ± 6º vs. 128 ± 7º) (Fig. 13).

5.5 Muscle swelling

In experiment 2, the exercised upper arm medial circumference was increased at 2 h point and, thereafter, both the medial and the distal circumferences were larger than before the EE2, reaching the highest values on day 4 and 6 (Fig. 14).

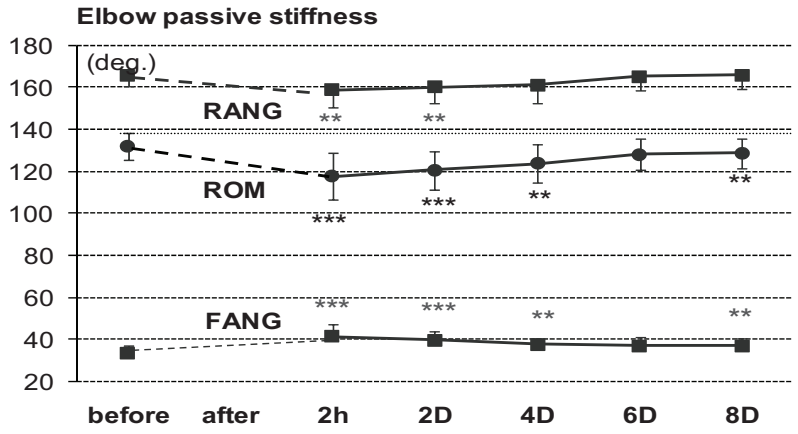

FIGURE 13 Elbow passive stiffness before, immediately (after), 2 h (2 h), 2 days (2D), 4 days (4D), 6 days (6D) and 8 days (8D) after EE2. (n = 9). *P < .05, **P < .01, ***P < .001, different than before value. RANG = relaxed arm angle, FANG = flexed arm angle and ROM = range of movement (Nosaka & Clarkson, 1996).

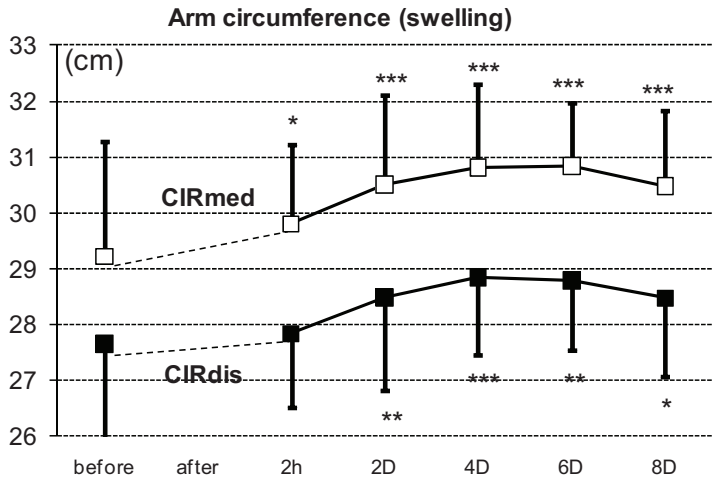

FIGURE 14 Arm circumference at distal (CIRdis) and medial (CIRmed) parts of the upper right arm muscle belly before, immediately (after), 2 h (2 h), 2 days (2D), 4 days (4D), 6 days (6D) and 8 days (8D) after EE2. (n = 9). *P < .05, **P < .01, ***P < .001, different than before value.

5.6 Stretch reflex

In experiment 2, the active BB and BR muscles' short latency reflex components (M1) were observable for every subject. They were decreased in all tested elbow angles 2 h after EE2 (Fig. 15). Responses did not differ between elbow angles.

The stretch applied to the elbow did not evoke a response in passive BB muscle before the exercise in any of the subjects. However, post-exercise BB EMG responses were observed from seven subjects depending on the elbow angle and the test session. The most responses were observed at 2 hours and 2 days after EE2. (details in original paper III).

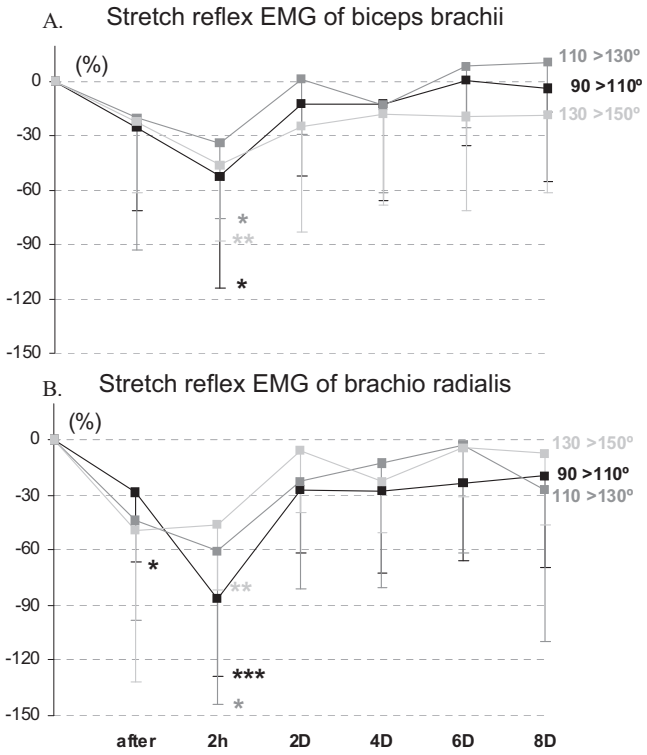

FIGURE 15 Relative post-exercise change of BB (A) and BR (B) EMG short latency reflex component relation to back ground activity (M1 / BGA) in active (20% of MIA) stretch reflex test at three different elbow angles (90°, 110°, 130°) before, immediately after (after), 2 hours (2h), 2 days (2D), 4 days (4D), 6 days (6D) and 8 days (8D) after EE2. (n=10). * P<.05, ** P<.01, different than before val ue. Vertical bars = S.D.

5.7 Flexion target movement (TM)

5.7.1 Velocity of performance

In experiment 1, movement time or time to target was not determined. In experiment 2 an average of all TM ranges, the time to target were increased – seen as a deterioration of the performance – immediately and 2 h after EE2 (P<.05 and P<.01, respectively).

5.7.2 Accuracy of performance

In experiment 1, the target degree was not changed when determined 600 ms after the movement onset. In experiment 2, no changes were found after EE2.

5.7.3 Main movement kinematics

In experiment 1, EE1 led to a significantly increased time to peak velocity acutely post-exercise. The time to peak velocity was not recovered on day 7 post-exercise (Fig. 16). The movement peak amplitude also occurred later at 0.5 hour (6±5%, P<.01) and 2 days (4±4%, P<.05) after EE1.

In experiment 2, the post-exercise progresses of any kinematic parameters were different between the elbow ranges (LA, MA, SA).

The acute performance reduction (time to target) immediately and 2 h after the exercise was paralleled with decreased peak velocity (P<.01 and P<.001) and increased time to peak velocity and symmetry ratio (P<.01 and P<.001). At 2 h post-exercise, in SA, the increase in time to target was associated with the decrease in peak movement velocity (r =.70, P<.05). On average, from all TM ranges, the peak velocity was decreased also 2 days (P<.001) and 4 days (P<.01) after EE2.

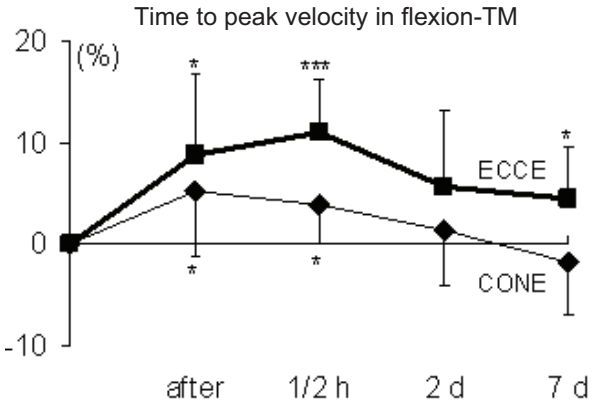

FIGURE 16 Relative changes in time to peak velocity in the flexion-TM after the EE1 and CE. Immediately after (A), 0.5 h (1/2h), 2 days (2d), 7 days (7d) after EE1. *P<.05, ***P<.001. Vertical bars = S.D.

5.7.4 Movement oscillation

In experiment 1, bi-directional oscillation over the target was increased immediately and 0.5 h after EE1 (Fig. 17).

In experiment 2, the average (all elbow ranges) total oscillation (movement trajectory) of the forearm was decreased during the delayed recovery phase (2 – 6 days) (P<.001) without any statistical relation to performance criteria.

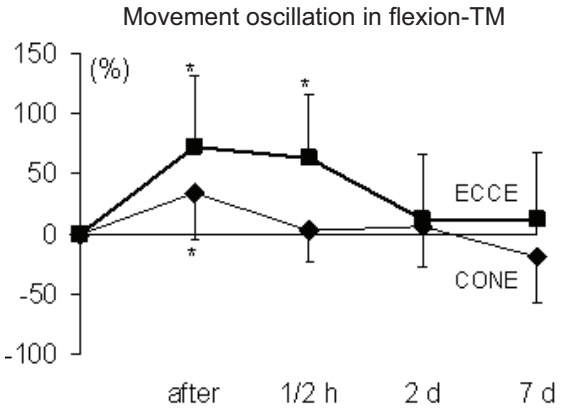

FIGURE 17 Relative changes of the movement oscillation over the target zone in flexion-TM after the EE1 and CE. Immediately after (A), 0.5 h (1/2h), 2 days (2d), 7 days (7d) after EE1. *P<0.05. Vertical bars = S.D.

5.7.5 Timing of agonists EMG

In experiment 1, the agonist BB 2nd burst was delayed immediately and 0.5 h after EE1 (Fig. 18).

In experiment 2, on average, the timing of the fatigued flexors was earlier immediately (BB1, P<.001 and BR1, P<.05) and 2 h after EE2 (BB1, P<.001 and BR1, P<.01) (Fig. 19). Moreover, the BR1 ended later immediately and 2 h post-exercise (P<.01), which was associated with increased duration of these bursts (P<.001). BB1 duration was increased immediately post-exercise as well (P<.01) (Fig. 19). Elbow flexor 2nd bursts ended later during the acute recovery phase of 2 h (P<.001), which was concurrent with the lengthened duration of these bursts (P<.01-.001) (Fig. 19). The post-exercise changes of burst timings were not different between the TM ranges (SA, MA, LA).

5.7.6 Timing of antagonist EMG

In experiment 1, the antagonist TB peak was delayed immediately and 0.5 h after EE1 (Fig. 18).

In experiment 2, no post-exercise change was observed in antagonist timing, however, timing was determined differently between the two studies (see methods).

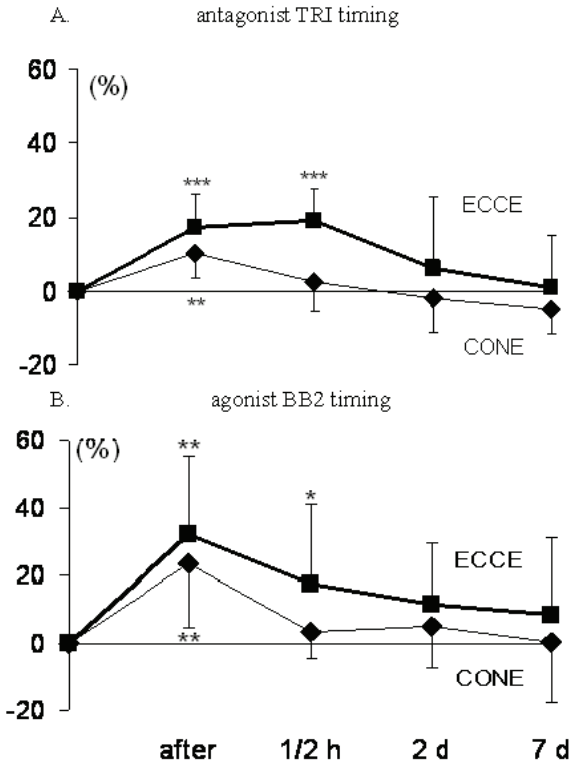

FIGURE 18 Relative changes in the timing of the peak EMG of the antagonist TB (A) and agonist BB2 (B) during the flexion-TM after EE1 and CE. Immediately after (A), 0.5 h (1/2h), 2 days (2d), 7 days (7d) after EE. **P<0.01, **P<0.01, ***P<0.001. Vertical bars = S.D.

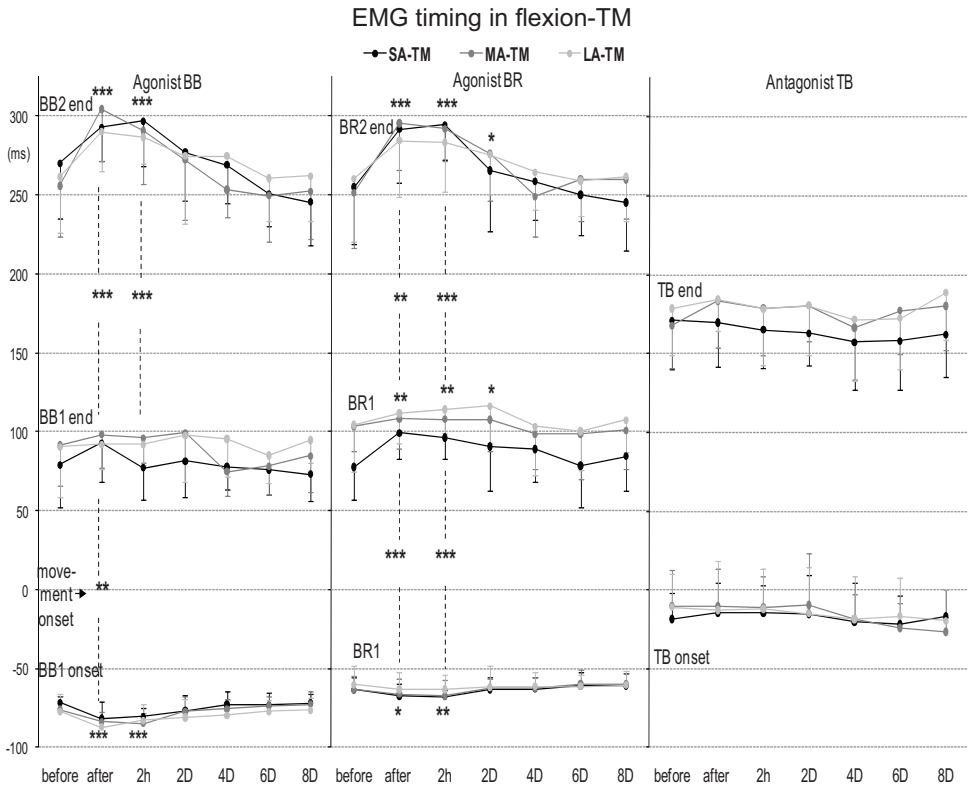

FIGURE 19 Agonist and antagonist muscles EMG timing in flexion-TM (SA, MA, LA) before, immediately after (after), 2 hours (2h), 2 days (2D), 4 days (4D), 6 days (6D) and 8 days (8D) after EE2. Broken vertical bar = change in burst duration. * P<.05, ** P<.01, *** P<.001. Vertical bar = S.D.

5.7.7 Amplitude of agonists EMG

In experiment 1, the agonist BB peak amplitude was reduced immediate after EE1 (–16±29%, P<.05).

In experiment 2, the fatigued flexor 1st burst peak amplitudes decreased immediately, 2 h (BB1 and BR1, P<.05-.001) and 2 days (BR1, P<.05) post-exercise (Fig. 20). In contrast, the flexor 2nd burst peak amplitudes increased during the acute recovery period of 2 h (P<.05-.01) (Fig. 20). The post-exercise changes of the agonists' burst peak amplitudes were not different between the TM ranges (SA, MA, LA).

5.7.8 Amplitude of antagonist EMG

In experiment 1, the antagonist TB peak showed a reduction 0.5 h after EE1 (–10±8 %, P<.05).

In experiment 2, the non-fatigued TB amplitude was decreased at 2 h (2-M, P<.01), 4 days and 6 days (P<.05) after EE2 (Fig. 20).

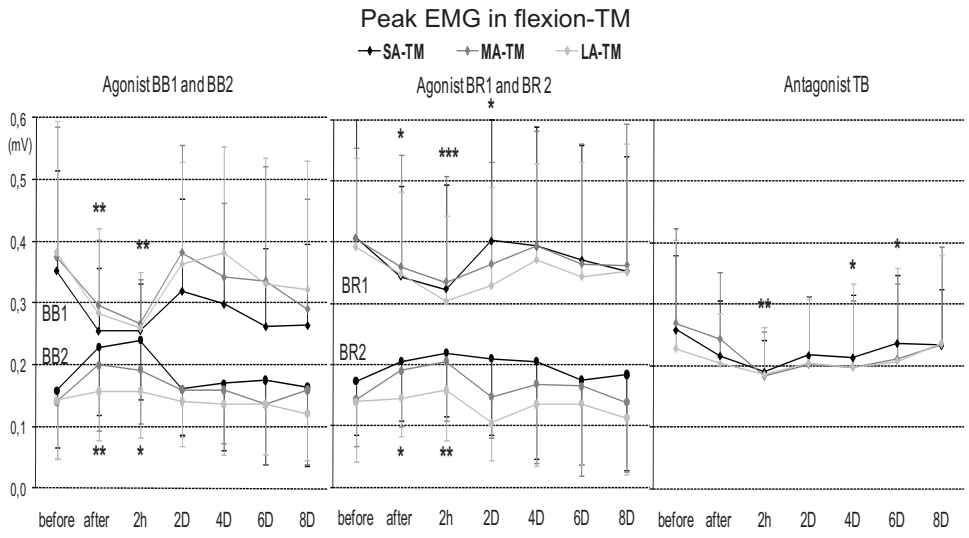

FIGURE 20 Peak EMG of agonist and antagonist muscles in flexion-TM (SA, MA, LA) before, immediately after (after), 2 hours (2h), 2 days (2D), 4 days (4D), 6 days (6D) and 8 days (8D) after EE2. * P<.05, ** P<.01, *** P<.001. Vertical bar = S.D.

5.7.9 Co-activation of agonist – antagonist

In experiment 1, co-activation was not determined. In experiment 2, from the average of all elbow ranges, co-activation of agonist BR1 and antagonist TB was increased immediately and 2 h after EE2 (P<.05). In parallel, aEMG of co-activation was decreased immediately (BR1, P<.05) and 2 h post-exercise (BR1 and TB, P<.05). The post-exercise changes of co-activations were not divergent between the TM ranges (SA, MA, LA).

5.7.10 Co-activation of antagonist – agonist 2nd

In experiment 1, co-activation was not determined. In experiment 2, from the average of all elbow ranges, TB and BR 2nd bursts' overlap was decreased both acutely (P<.05) and delayed (2D, 4D, 8D) after EE2 (P<.05) with a parallel drop in TB co-activation aEMG on day 2 and on day 4 post-exercise (P<.05). The post-exercise changes of co-activations were not divergent between the TM ranges (2-M) as they followed the changes in bursts timing.

5.8 Extension target movement (TM)

5.8.1 Velocity of performance

In experiment 1, target time (movement time) was not determined.

In experiment 2, in contrast to flexion - TM, seen as an improvement of the performance, the time to target was shortened at 2 h after EE2 ($P<.05$) and delayed post-exercise on days 4 ($P<.05$), 6 ($P<.01$) and 8 ($P<.001$). As in flexion-TM, the post-exercise changes of the performance parameters were not divergent between the elbow ranges (SA, MA, LA).

5.8.2 Accuracy of performance

In experiment 1, as in flexion-TM, target degree was not changed. In experiment 2, as in flexion TM, no changes were found after EE2.

5.8.3 Main movement kinematics

In experiment 1, no statistically significant changes were found after EE1.

In experiment 2, the acute performance enhancement, immediately and 2 h after EE2, was parallel to decreased peak velocity ($P<.001$ and $P<.001$). In addition, the delayed improvement of movement time was concurrent with the decreased peak velocity on days 2 ($P<.01$) and 4 ($P<.05$). Contrary to flexion-TM, symmetry ratio of extension TM remained unchanged. As in flexion-TM, the post-exercise changes of the kinematic parameters were not divergent between the elbow ranges (SA, MA, LA).

5.8.4 Movement oscillation

In experiment 1, oscillation was unchanged after EE1.

In experiment 2, the acute performance enhancement (velocity demand), immediately and 2 h after EE2, was parallel to decreased arm total oscillation time ($P<.01$). In addition, the delayed improvement of movement time was concurrent with the decreased arm oscillation time on days 2, 4, 6 and 8 ($P<.001$).

5.8.5 Timing of agonist EMG

In experiment 1, up to 0.5 h post-exercise, EE1 resulted in an earlier timing of the first agonist TB peak amplitude (–41±1 %, $P<.05$) compared to its delayed 2nd burst ($P<.01$). TB 2nd burst was delayed also on day 2 ($P<0.01$) and day 7 ($P<0.05$).

In experiment 2, the average of all elbow ranges showed that the non-fatigued agonist TB1 onset was timed later on day 4 ($P<.01$), 6 ($P<.001$) and 8 ($P<.001$) after EE2. The TB 2nd burst duration increased immediately post-exercise ($P<.05$). As in flexion-TM, the post-exercise changes of burst timings were not different between the TM ranges (SA, MA, LA).

5.8.6 Timing of antagonists EMG

In experiment 1, antagonist timing was not changed after EE1.

In experiment 2, the acute post-exercise (EE2) timing of fatigued antagonists was similar to flexion-TM, wherein they acted as agonists. The activity of BB and BR began earlier immediately post-exercise (P<.01). At 2 h after EE2 the BB end was timed later (P<.05). In parallel, BB duration increased immediately (P<.01) and 2 h (P<.001) after EE2. The post-exercise changes of burst timings were not different between the TM ranges (SA, MA, LA).

5.8.7 Amplitude of agonist EMG

In experiment 1, EE1 resulted in a decreased peak of the agonist TB 1st and 2nd bursts at 0.5 h (-10±8 % and -20±17 %, P<.05) and 2nd burst on day 2 (–28±16 %, P<.01) post-exercise.

In experiment 2, similarly to fatigued flexors in flexion-TM, non-fatigued TB peak amplitude decreased at 2 h after EE2 (P<.001). But in contrast to flexion-TM, its amplitude remained reduced (P<.05) up to day 6 post-exercise (Fig. 21). In parallel with TB decrement, peak amplitude of TB 2nd burst decreased acutely (P<.001) and delayed post-exercise on day 2 (P<.001) and day 4 (P<.05) (Fig. 21). As in flexion-TM, the post-exercise changes of bursts amplitudes were not different between the TM ranges (SA, MA, LA).

5.8.8 Amplitude of antagonist EMG

In experiment 1, the antagonist amplitude was not changed after EE1.

In experiment 2, in contrast to flexion-TM, the fatigued flexors' peak amplitudes decreased both at 2 h (BB, P<.05) and on day 2 (BB and BR, P<.01) after EE2 (Fig. 21). As in flexion-TM, the post-exercise changes of burst amplitudes were not different between the TM ranges (SA, MA, LA).

5.8.9 Co-activation of agonist – antagonist

In experiment 1, co-activation was not determined.

In experiment 2, as in flexion-TM, the average co-activation of agonist (TB1) and antagonist (BB) increased immediately (P<.01) and 2 h (P<.01) after EE2. The co-activation amplitudes of antagonistic muscles were not changed. The post-exercise changes of co-activations were not divergent between the TM ranges (SA, MA, LA).

5.8.10 Co-activation of antagonist – agonist 2nd

In experiment 1, co-activation was not determined.

In experiment 2, in contrast to flexion-TM, antagonist BB and agonist TB 2nd burst overlap increased 2 h after EE2 (P<.05). The co-activation amplitudes of antagonistic muscles were not changed. The post-exercise changes of co-activations were not divergent between the TM ranges (SA, MA, LA).

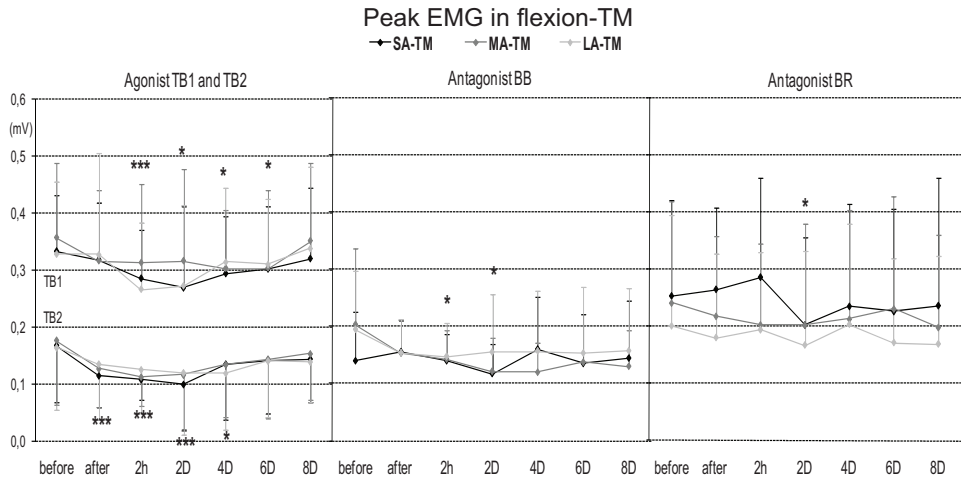

FIGURE 21 Peak EMG of agonist and antagonist muscles in extension-TM (SA, MA, LA) before, immediately after (after), 2 hours (2h), 2 days (2D), 4 days (4D), 6 days (6D) and 8 days (8D) after EE2. TB= triceps brachii, BB= biceps brachii, BR= brachioradialis. * P<.05, ** P<.01, ***P<.001. Vertical bar = S.D.

5.9 Rhythmic flexion – extension movement (RM)

5.9.1 Movement amplitude

In experiment 1, no changes were found after EE1.

In experiment 2, extension and flexion amplitudes of SA-RM were increased and delayed after EE2 (P<.05-.001). At these delayed test situations, the SA minimum angle decreased on average by -8 ± 20° (P < .05). Amplitude of phases did not differ between RM ranges (SA, MA, LA).

5.9.2 Time to peak velocity

In experiment 1, no changes were found after EE1.

In experiment 2, in SA-RM, the flexion phase relative time to peak velocity increased 2 h after EE2. Delayed post-exercise peak velocity was timed later also at other RM ranges (Fig. 22). The time to peak velocity did not differ between RM ranges (SA, MA, LA).

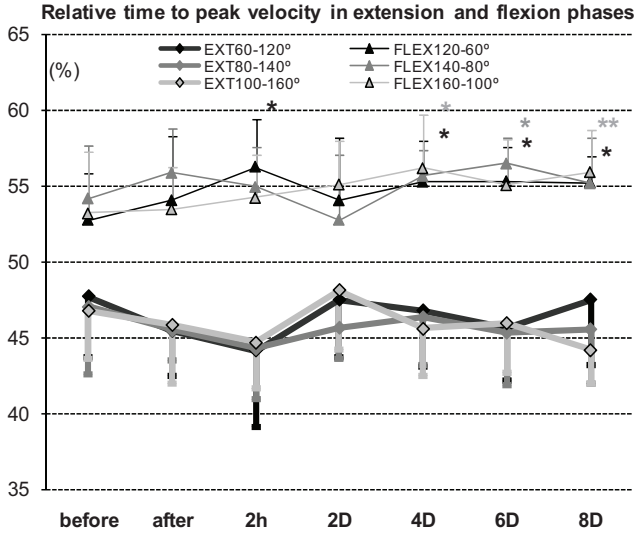

FIGURE 22 The time to peak velocity for extension- and flexion movement phases in RM at SA, MA and LA before, immediately (after), 2 hours (2h), 2 days (2D), 4 days (4D), 6 days (6D) and 8 days (8D) after EE2. EXT=extension phase, FLEX=flexion phase. (n=10). * P<.05, ** P<.01, *** P<.001, different than be fore value.

5.9.3 Cycle duration

In experiment 1, no changes were found after EE1.

In experiment 2, RM cycle duration increased on day 2 (SA, 8 ± 9%, P < .05), on day 6 (SA, 8 ± 8%, P < .01 and MA, 7 ± 9%, P < .05), and on day 8 (SA, 7 ± 7% and MA, 8 ± 9%, P < .05) after EE2.

5.9.4 RM velocity

In experiment 1, extension peak velocity was increased (+12 ± 10%, P < .01) on the 7th day after EE1 compared to pre-exercise value.

In experiment 2, in MA-RM both the extension and the flexion peak velocity decreased immediately after EE2 (P<.05). Contrary to this, delayed post-exercise phase peak velocities of RM phases increased at least at one test point (P<.05-01). As an average of all test sessions and all RM ranges, the extension peak velocity and flexion peak velocity correlated positively with the changes in movement amplitude (r = 0.66–0.73, P < .01 and r = 0.69–0.80, P < .01, respectively).

5.9.5 EMG patterns

Timing of EMG

In experiment 1, no post-exercise changes in antagonistic muscle timing was found after EE1. However, the braking latency increased 0.5 h post-exercise (+26 ± 26%, P < .05). On the other hand, the time delay from TB activity start to beginning of the extension phase (extension latency) decreased by -19 ± 22% (P < .05).

In experiment 2, the fatigued BB muscle activity began earlier immediately (SA, MA, LA) and 2 h (SA) after EE2 (P<.01-.001). In parallel, BB S1 and S2 (MA) and 2 h post-exercise S2 (LA) were timed earlier (P<.05-.001). Contrary to this, antagonist TB activity started later 2 h after EE2 (SA, P<.01) and was associated with parallel changes of S1 and S2 (P < .05).

Duration of EMG

In experiment 1, post-exercise changes were not found after EE1.

In experiment 2, the relative BB duration in RM cycle increased acutely (SA, MA, LA) (P<.001) and delayed (SA, LA) after EE2 (P<.05). Contrary to this, antagonist TB relative duration decreased delayed post-exercise (LA) (P<.05).

Amplitude of EMG

In experiment 1, the major fatigue-induced influences were seen in the 2D measurements, and they all concerned the profile of the fatigued BB EMG parameters (Fig. 23). The following changes (increments) were significant (P < .05 to P < .01): maximal amplitude, mean amplitude, S1 and S2 segment amplitude. Non-fatigued TB muscle EMG was unaltered by the EE1.

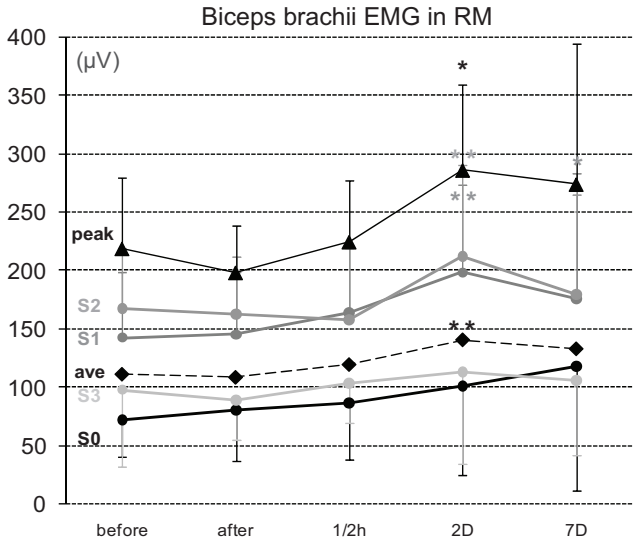

FIGURE 23 Biceps brachii EMG in elbow RM before, immediately after (after), 0.5 h after (1/2 h), 2 days (2D) and 7 days (7D) after EE1. peak = burst maximal value. ave = burst mean value. S0–S3 = mean values for different segments. *P < .05, **P < .01, different than the before value. Vertical bars= S.D.

In experiment 2, fatigued BB EMG burst peak amplitude decreased during the acute recovery period of 2 h after EE2 (Fig. 24). A closer EMG burst segment analysis revealed both the acute and delayed decrement of BB S1 and S2 amplitudes. In LA the BB S0 amplitude decreased immediately post-exercise, while in SA it increased acutely and delayed after EE2 (Fig. 24). The non-fatigued antagonist TB S0 amplitude was lower on day 2 post-exercise (LA, $P < .05$). There were no differences in EMG variables or in post-exercise changes between RM ranges. Contrary to our expectations, only a minor and statistically non-significant correlation was observed between the post-exercise changes of BB stretch reflex amplitude and BB amplitude in RM.

Co-activation

In experiment 1, after EE1, TB activation overlapped BB EMG more on day 2 (42 ± 12 ms vs. 37 ± 11 ms) ($P < .05$), but less on day 7 (35 ± 13 ms) ($P < .05$).

In experiment 2, in SA-RM, BB co-activation with TB in TB-BB activity turn increased immediately after EE2 (1 ± 1 ms vs. 8 ± 12 ms, $P < .05$). BB EMG amplitude increased during the co-activation phase of the TB-BB activity turn immediately ($P < .01$), 2 days and 6 days ($P < .05$) post-exercise.

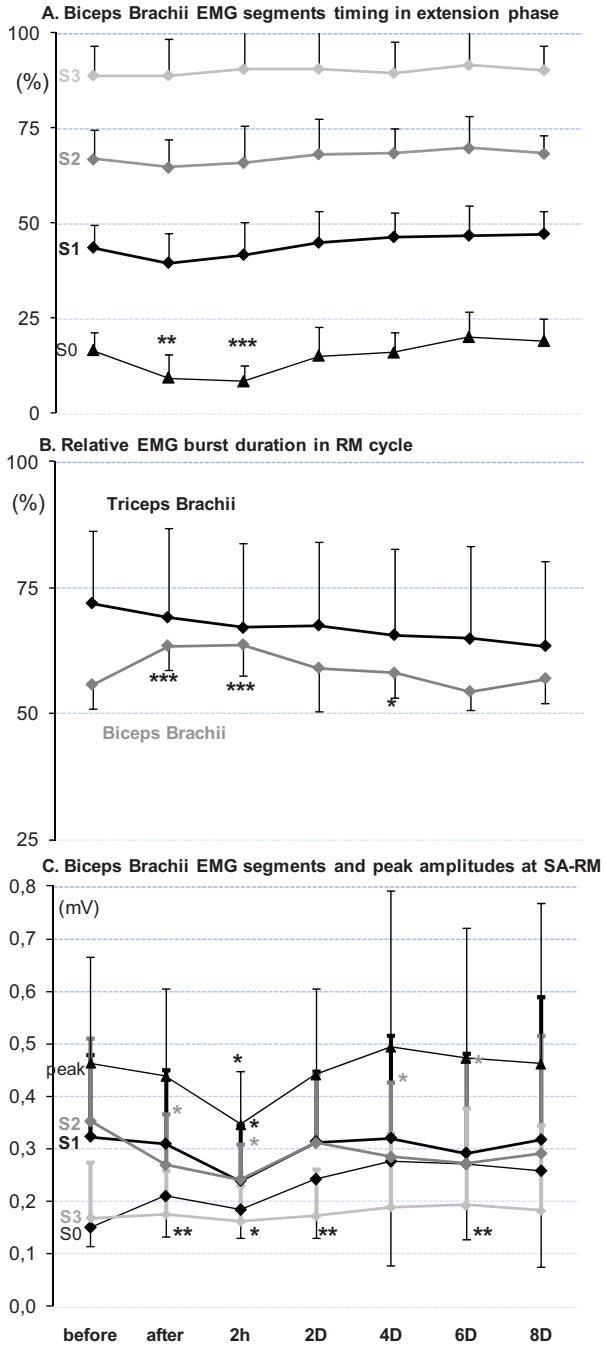

FIGURE 24 Biceps brachii EMG segments latencies, segments timing in extension phase (A), biceps brachii and triceps brachii EMG burst relative durations in RM cycle (B) and burst peak (peak) and segments average amplitudes (C) at SA-RM (60–120°) before, immediately (after), 2 h (2 h), 2 days (2D), 4 days (4D), 6 days (6D) and 8 days (8D) after EE2. (n = 10). *P < .05, **P < .01, ***P < .001.

5.10 Effects of acute learning on TM performance (experiment 3)

5.10.1 Velocity and accuracy

In flexion-TM, on average, the TM time was different between groups (P<.01, t-tests, P<.05-.001) and the development (P<.001), which was greater in A-group, continued up to end of the practice (Fig. 25). Movement deviation from the target (accuracy) was constant during the practice and did not diverge between the groups.

In extension-TM, in contrast to flexion-TM, the TM time did not differ between the groups. A similar total improvement (P<.01) with the greatest development within 30 reps were observed in both subject groups (Fig. 25).

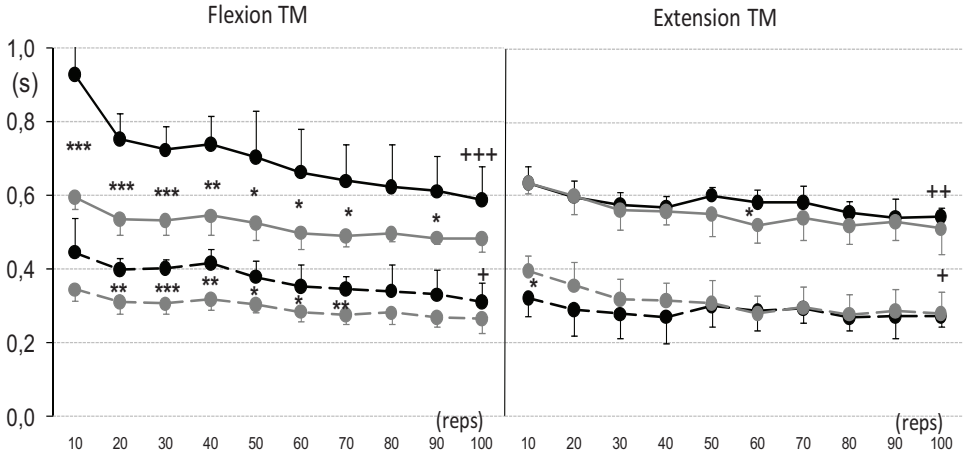

FIGURE 25 Movement time (solid line) and oscillation time (broken line) of accuracy group (●) and velocity group (●) during the practice of 100 repetitions (reps) of flexion-TM and extension-TM. * P<.05, ** P<.01, ***P<.001, statistical dif ference between the groups. + P<.05, ++ P<.01, +++P<.001, significant differ ence compared to first 10 repetitions set.

5.10.2 Movement kinematics

Peak velocity

In flexion-TM, on average, peak velocity was smaller in A-group compared to V-group (P<.01) (Fig. 26). The peak velocity of A-group showed the greater change (P<.001) during the practice sets, but remained smaller at the end of the practice than in the V-group (P<.05).

In extension-TM, as in flexion-TM, peak velocity of A-group was smaller than in V-group (P<.05, t-tests, P<.05-.01), and its development was slower than in the V-group (P<.001) (Fig. 26).

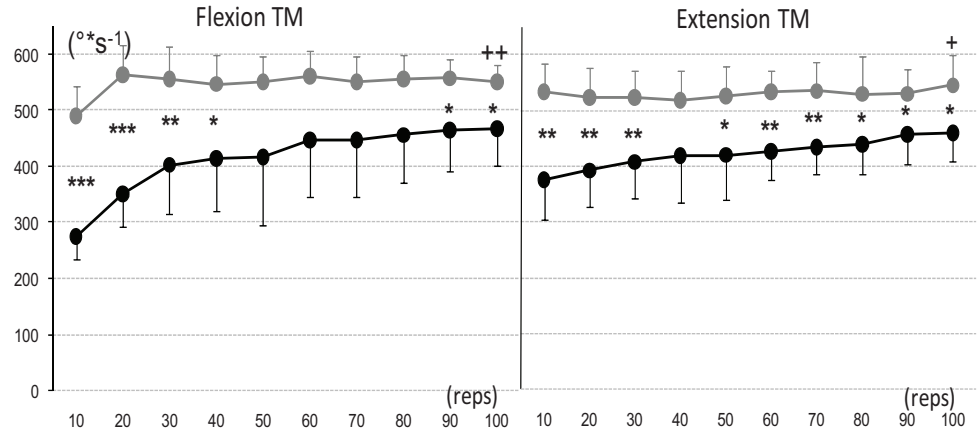

FIGURE 26 Peak velocity of accuracy group (●) and velocity group (●) during the prac tice of 100 repetitions (reps) of flexion-TM and extension-TM. * P<.05, ** P<.01, ***P<.001, statistical difference between the groups. + P<.05, ++ P<.01, +++P<.001, significant difference compared to first 10 repetitions set.

Time to peak velocity

In flexion-TM, the time to peak velocity was longer in A-group compared to V-group (P<.05) (Fig. 27). The change of peak timing was slower in A-group (P<.001), but the A-group values became equal to V-group at the 3rd set (Fig. 27).

In extension-TM, similarly to flexion-TM, the time to peak velocity was longer in A-group compared to V-group (P<.05). The A-group velocities became equal to the V-group's after the 5th set (Fig. 27).

Duration of main movement

In flexion-TM, the average time of the main flexion movement towards the target was longer in A-group than in V-group (P<.01). The total development of flexion time of A-group was slower than in the V-group (P<.001). However, the A-group flexion time closed in on the V-group's values, being equal after the 8th set (Fig. 27).

In extension-TM, similarly to flexion-TM, the average time of the extension movement towards the target was longer in A-group than in V-group (P<.05, t-tests, P<.05) (Fig. 27).

Symmetry ratio of main movement

In flexion-TM, after the practice, the symmetry ratio of the A-group was smaller than in the V-group (P<.05), but the ratios of both groups remained unchanged (Fig. 27).

In extension-TM, despite changes in peak velocity, the main movement symmetry ratios of both groups remained unchanged (Fig. 27).

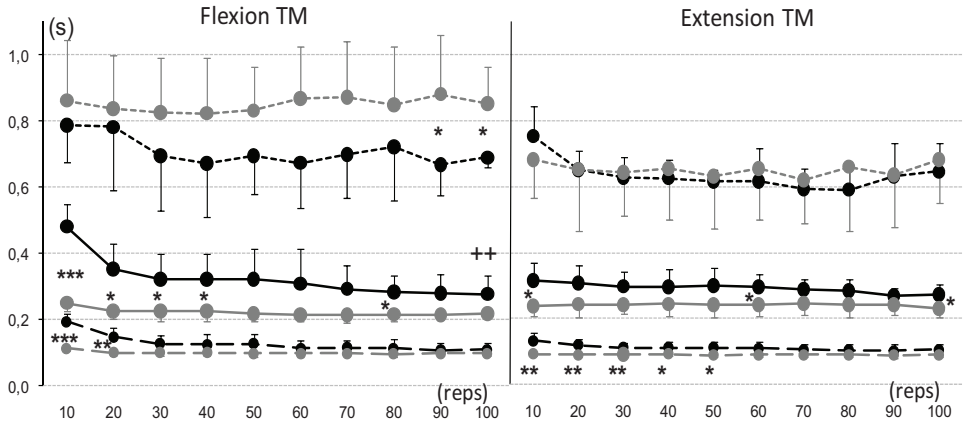

FIGURE 27 Time to peak velocity (broken line), main movement time (solid line) and symmetry ratio (dotted line) of accuracy group (●) and velocity group (◉) during the practice of 100 repetitions (reps) of flexion-TM and extension-TM. * P<.05, ** P<.01, ***P<.001, statistical difference between the groups. + P<.05, ++ P<.01, +++P<.001, significant difference compared to first 10 repetitions set.

Overshooting of target

In flexion-TM, on average (P<.05) and at the end of practice (10th set), the overshooting of the target was smaller in A-group than in V-group (5.4 ± 1.6° vs. 3.2 ± 0.7°) (P<.05). However, no statistically significant change was found in either group.

In extension-TM, in contrast to flexion-TM, the overshooting of the target was equal in both groups and did not change during the practice.

Amplitude of terminal oscillation

In flexion – TM, the oscillation amplitude decreased in both groups during the practice. At end of practice, it was smaller in A-group than in V-group (P<.05).

In extension-TM, the terminal oscillation did not differ between the groups. On average, it decreased during 100 repetitions (P<.05).

Duration of terminal oscillation

In flexion-TM, on average (P<.001) and up to the 7th set, the A-group oscillation time was longer than in V-group (P<.05-.001) (Fig. 25). The development of forearm oscillation time on the target zone of both groups followed the improvement of the TM time (r=.85, P<.01).

In extension-TM, the change of A-group oscillation time was different than in V-group (P<.05). At the beginning of the practice, the oscillation time of A-group was shorter (t-test, P<.05), thereby the development concentrated on

the V-group (Fig. 25). As in flexion-TM, the development of forearm oscillation time followed the improvement of the TM time (r=.63, P<.05).

5.10.3 Timing of agonists 1st and 2nd bursts

In flexion-TM, the agonists (BB, BR) onset (P<.05), and their change (P<.01), during the practice was slower in A-group than in V-group (P<.001). Up to the 2nd set (BB) and 6th set (BR), muscles in A-group were timed earlier (P<.05-.001) (Fig. 28).

In flexion-TM, the development of agonists' 2nd burst onsets and ends (P<.01) were slower in A-group compared to V-group (P<.001) (Fig. 28). At the beginning of the practice, bursts were timed later in A-group (P<.05-.001), however, the timing was congruent with V-group after two or seven sets (burst onset and end, respectively) (Fig. 28). On average, BB 2nd burst of A-group ended later than in V-group (P<.05).

In extension-TM, in contrast to flexion-TM, agonists' (TB) onset or its progress did not differ between the groups.

In extension-TM, as in flexion-TM, the development of TB 2nd burst onset and end were slower in A-group than in V-group (P<.01 and P<.05, respectively). At the first set (onset) and up to fourth set (end), TB 2nd was timed later in A-group than in V-group (P<.05).

Duration of agonists bursts

In flexion-TM, the shortening of the agonists' burst durations (P<.05-.01) followed the change of burst onsets and ends. During the early phase of practice, A-group burst durations were longer compared to V-group (P<.05-.001) (Fig. 28). The shortening of A-group burst durations were slower in A-group than in V-group (P<.001) (Fig. 28).

In extension-TM, similarly to flexion-TM, the duration of TB 1st and 2nd bursts were longer in A-group (P<.05), and they demonstrated the slower shortening than in V-group (P<.05).

5.10.4 Timing of antagonist burst

In flexion-TM, on average, the antagonist TB burst onset and end development (P<.05 and P<.01, respectively) were slower in A-group than in V-group (P<.05 and P<.001, respectively). On average, TB bursts ended later in A-group (P<.05), but after the 7th set TB end was equal between the groups (Fig. 28).

In extension-TM, the antagonists (BB and BR) timing developed similarly to the antagonist in the flexion-TM. The change of onsets and ends of BB and BR were slower in A-group than in V-group (P<.001 and P<.01, respectively). The end of both antagonists were timed later in A-group than in V-group up to 5th set (P<.05-.001).

Duration of antagonist burst

In flexion-TM, on average, the change of antagonist duration (P<.01) did not diverge between the groups, however, at the beginning of the practice the duration of TB was longer in A-group than in V-group (P<.05) (Fig. 28).

In extension-TM, the shortening of the durations were slower in A-group than in V-group (P<.05 and P<.01, respectively). Nearly up to end of practice the burst durations of A-group were longer compared to V-group (P<.05-.001).

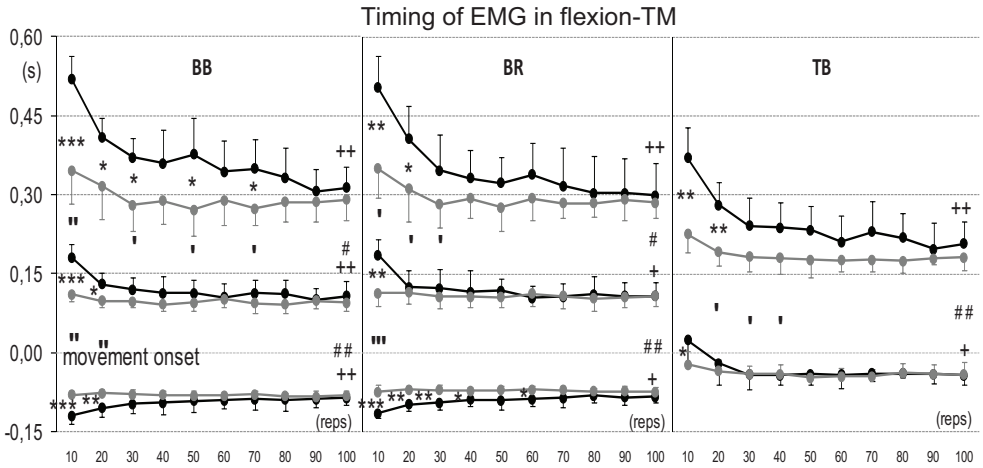

FIGURE 28 Timing of muscle activity (EMG) in flexion-TM. Onsets (down), 2nd bursts onsets (middle) and ends (up) of accuracy group (●) and velocity group (●) during the 100 repetitions (reps). *P<.05, **P<.01, ***P<.001, significant difference between the groups. +P<.05, ++P<.01, +++P<.001, significant dif ference compared to first 10 reps set.´P<.05,´´P<.01,´´´P<.001; significant dif ference of burst duration between the groups. #P<.05, ##P<.01, ###P<.001; significant difference of burst duration compared to first 10 repe titions set. BB=biceps brachii, BR= brachioradialis,TB= triceps brachii.

5.10.5 Amplitude and area of EMG bursts

In flexion-TM, the change of both agonists' (BB, BR) 1st EMG burst peak amplitudes differed between the groups (P<.05) since the A-group flexor amplitudes increased during the early sets of practice. The development of antagonist (TB) peak amplitude and TB burst areas and flexor 1st burst areas of A-group showed a similar increasing trend during the practice. The change of TB / BB 2nd EMG burst (area) relations differed between the groups (P<.05), since the A-group relation increased during the practice.

In extension-TM, in contrast to flexion-TM, the agonist (TB) 1st burst or the change of the other bursts peak amplitudes and areas did not differ between the groups during the practice. Similarly to flexion-TM, the antagonist / agonist (BB) 2nd EMG bursts (area) relation developed differently between the groups (P<.05), since A-group relation increased during the practice. At the end of practice, A-group relations were higher than in V-group (P<.05).

5.10.6 Co-activation of EMG bursts

In flexion-TM, the change of antagonist TB and agonists 2nd co-activation did not diverge between the groups, however, the co-activations in A-group were larger than in V-group up to 3rd set (P<.05-.01) (Fig. 29). On average, TB - BR 2nd co-activations were higher in A-group than in V-group (P<.05). The agonists and antagonist co-activations did not diverge between the groups, and no change of muscles co-activations were observed during the practice.

In extension-TM, on average, the antagonists and TB 2nd burst co-activations were higher in A-group than in V-group (P<.05). The co-activations of A-group showed a different decreasing change compared to V-group (P<.01). Up to 6th set, the co-activations of A-group were larger than in V-group (P<.05-.01). As in flexion-TM, the agonist and antagonist co-activations did not diverge between the groups, and no changes of muscle co-activations were observed during the practice.

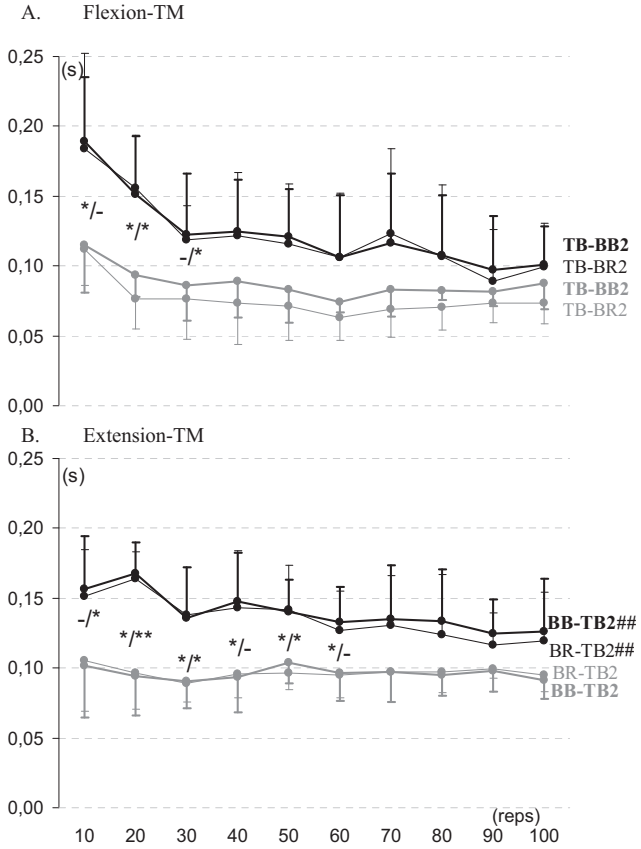

FIGURE 29 The co-activation of antagonistic muscles in flexion-TM (A) and extension-TM (B) of accuracy group (●) and velocity group (●) during the practice of 100 repetitions (reps). * P<.05, ** P<.01, statistical difference between the groups. ## P<.01, progress differed statistically between the groups. BB= BB antagonist burst, BB2= agonist BB 2nd burst, BR= BR antagonist burst, BR2= agonist BR 2nd burst, TB= TB antagonist burst, TB2= agonist TB 2nd burst.

6 DISCUSSION

The present eccentric exercise protocols disturbed the normal neuromuscular function especially a few hours post-exercise. However, the protocols did not disturb the extension-TM performance like observed in deafferented patients, who are deprived the sensory feedback. On the contrary, the performance was disturbed (flexion-TM) and modulated (TMs and RM) the most during the acute 2 h post-exercise recovery period when the drop of maximal force was the greatest. The post-exercise performance of TM and RM at small elbow angles were changed the most. The acute -2h- post-exercise changes in performance were concomitant with the agonists' decreased EMG amplitudes and changed timing (flexion-TM), and with the large drop in BB EMG burst amplitudes (RM). In addition, the acute post-exercise reduction of elbow flexors' EMG amplitudes associated with a parallel drop in their active stretch reflex amplitudes.

The development of TM performance during the acute practice was associated with increased EMG relation and decreased co-activation (overlap) of antagonist and agonist 2nd bursts. In addition, the decrease in TM terminal oscillation was associated to an improvement of TM time.

In the following Discussion the results concerning the exercise induced muscle damage and soreness will be discussed first followed by the results of the learning interventions.

6.1 Post-exercise indications of muscle fatigue and damage

After EE1, dynamic maximal forces dropped ~ 45% immediately post-exercise, while after EE2 the acute drop in maximal eccentric and static force was 23-28%. In EE1, 100 maximal eccentric actions were repeated with a duty cycle of 1:1, while EE2 consisted of 50 actions performed with a duty cycle of 1:15. A larger drop of forces after EE1 may have been partly due to a compound effect of both metabolic and mechanical factors, since a clear elevation of blood lactate was observed immediately post-exercise. It has been suggested that the magnitude of initial muscle injury may be estimated from the decrease in force around 3

hours post-exercise (Faulkner et al. 1993). At that time acute metabolic fatigue factors – not observed after EE2 - should have disappeared and no further injury has occurred. After EE1, force recovery began already within half an hour post-exercise, but during the acute 2 h follow up after EE2, no recovery was observed in maximal forces (Fig. 7 and 8). As expected, the EE1 and EE2 of the elbow flexors caused acute reductions in EMG/force ratio, as observed in earlier publications with similar fatigue protocols (e.g. Komi & Rusko 1974). After EE2, peripheral force production failure seemed to be the largest at 2 h post-exercise. After EE1 and EE2, neither the maximal dynamic force nor static force recovered within one week. A large decrement of maximal isometric force induced by eccentric exercise has been reported to need up to one month or more to recover (e.g. Faulkner et al. 1993, Sayers & Clarkson 2001). The present study showed that delayed recovery took place despite the muscle action type. It has been concluded that the secondary inflammatory response in exercise damaged muscle could contribute to this prolonged force recovery (Sayers & Clarkson 2001). DOMS and/or inflammation processes influencing muscle activation at the cortical and/or spinal level (e.g. Bigland-Ritchie et al. 1986, Duchateau & Hainaut 1993, Le Pera et al. 2001, Weerakkody et al. 2003, Ervilha et al. 2004b) might have affected the bimodal reduction in flexor activity (Fig. 9) that surely contributed to a long-term drop in isometric force in the present study.

An important observation was also that both eccentric exercises led to increased CK activity and delayed onset muscle soreness, known as indirect indicators of muscle damage. After both EE1 and EE2, soreness peaked on day 2 post-exercise. However, acute muscle soreness (AOMS) was perceived during the early recovery period of 2 h. Generally, the acute exercise-induced pain or soreness has not widely been observed after eccentric exercise. On the contrary, pain has been reported to develop several hours (24-48h) later (e.g. Jones et al. 1987, rev. Clarkson & Hubal 2002). Repeated eccentric actions of knee extensors have been reported to cause an acute inflammation response and acute soreness at 2 hours and 4 hours post-exercise (e.g. MacIntyre et al. 2001). After the present EE2, which did not develop metabolic fatigue, subjects experienced pain in their flexor muscles already immediately and 2 hours post-exercise (Fig. 12). As observed after the present EE2, CK usually peaks 2-4 days post-exercise. Therefore, the observed peak values on day 7 after the EE1 arose from the fact that it was not measured between days 2 and 7 post-exercise. On the other hand, the very late peaked CK could be explained by a difference in exercise model or by the level of subjects' fitness (Clarkson et al. 1992), as it has been suggested that the variability in CK response is partly related to the variability in exercise-induced muscle damage (e.g. Nosaka & Clarkson 1996). As widely observed, the timing of soreness and CK peak values was found to be different also in the present study. In contrast to earlier literature, after the present EE2, muscle swelling peaked about the same time as the soreness (cf. Clarkson & Hubal 2002). As the swelling was observed already at 2 h post-exercise, the AOMS may well have resulted from the swelling and pressure in the muscle. After the present EE2, elbow passive stiffness increased acute (2 h) post-exercise and re-

mained elevated for several days (Fig. 13), which was in line with earlier findings of others (e.g. Howell et al. 1993, Jones et al. 1987). Whitehead et al. (2003) have hypothesized that injury contractures of damaged fibres may influence muscle passive stiffness.

According to the present results, we interpret that, after EE2, the force loss was greater at short muscle lengths during the acute recovery period of 2 hours (Fig. 7). However, after EE1, we were unable to clearly show such a shift. One possibility could be that eccentric actions may have had a different effect on elbow flexor force production at different angles after that exercise, due to changes in EMG/force relation (see Fig. 10). After the EE1, regardless of an immediate decrease in BB EMG for the whole movement range, a relatively larger increase in concentric EMG/force ratio was demonstrated at the smaller elbow angles than at the larger ones. It has been indirectly shown that eccentric action may shift the angle-torque relationship towards longer lengths for several days (e.g. Saxton & Donnelly 1996, Prasartwuth et al. 2006). This shift of relationship has been considered as a sign of muscle damage (e.g. Whitehead et al., 2003). In an un-fatigued muscle, it has been shown that muscle voluntary activation is lower at shorter lengths (Rack & Westbury 1969, Prasartwuth et al. 2006). During the eccentric fatigue of elbow flexors (Prasartwuth et al. 2006) and knee extensors (Skurvydas et al. 2010) the muscle voluntary activation has been shown to be impaired particularly at short muscle lengths. Immediately after the present EE2, the neural activity of MIA was decreased, especially at the most flexed 90° angle (Fig. 9).

6.2 Effects of eccentric exercises on TM performance and control

In the present study, the subjects were instructed to perform TM "as fast and as accurate as possible". In respect of the task accuracy demands (deviation from target), neither the flexion-TM nor extension-TM performances were deteriorated after the eccentric exercise protocols. This contrasted earlier findings following isometric fatigue (Jaric et al. 1999). In respect of the task velocity demands, an impairment of flexion-TM performance (movement time) was found acutely after the EE2. This meant that the TM task, wherein fatigued elbow flexors acted as agonists, was deteriorated. Furthermore, after EE2, we observed a post-exercise decrease of the movement time of the extension-TM. That is, the TM performance, in which fatigued and damaged flexors worked as antagonists, was improved.

The effects of EE1 and EE2 on the main movement (movement towards the target) kinematics were seen primarily in the flexion-TM, but not in the extension-TM. During flexion-TM, agonist eccentric acute fatigue was associated with decreased peak velocity (EE2), delayed time-to-peak velocity (EE1 and EE2), and increased symmetry ratio (EE2). Similar phenomena have also been observed previously with isometric (Jaric et al. 1997, 1999) and eccentric fatigue (Miles et al. 1997). It has been suggested that deterioration of TM, including a

decreased movement peak velocity (Corcos et al. 2002) and/or a delayed movement peak velocity (Miles et al. 1997), may be the consequence of the reduced force production of the agonist muscles (e.g. Jaric 2000, Prodoehl et al. 2003). This suggestion was supported by the present finding when a slight shift of the peak force towards longer muscle lengths (EE1), and when the relative change of time to target and peak velocity were correlated negatively 2 h post-exercise (EE2) at SA-TM. Moreover, after EE2, the deterioration of the flexion-TM task seemed to affect only the performance at the small angles (SA) and at the time (immediately and 2 h post-exercise) when the drop in maximal forces was the greatest (Fig. 6, 7 and 8). Similarly to the present study, it has been shown that at the more extended elbow positions the durations of agonist 1st burst are higher, the movements are slower (Prodoehl et al. 2003), and the movement symmetry ratios are larger (Mirkov et al. 2002). This is consistent with the situation when the available muscle force is reduced e.g. in muscle fatigue. In the current study, after EE2, elbow flexor 1st bursts post-exercise timing and duration in flexion-TM at SA became closer to MA-TM and LA-TM values (Fig. 19). These findings assume that the force production failures, especially at small elbow angles, were responsible for the impairment of TM kinematics, especially at SA. An acute shift of the peak forces towards longer muscle lengths, possible due to sarcomere mechanical disruption, has recently been observed after elbow flexor eccentric exercises (e.g. Prasartwuth et al. 2006, Saxton & Donelly 1996). Shapiro et al. (2005) have suggested that the force-length property of the muscle contributes to force generation more when the muscle acts as the agonist than as the antagonist. This might also partly explain the current deterioration of flexion-TM (while extension-TM remained intact). After EE1, the recovery in flexor force production was slower than after CE. This delayed recovery is likely to be related to ultrastructural muscle damage known to take place after repeated eccentric actions (Lieber & Fridén 1988, Fridén & Lieber 1992). The minor delayed reductions in flexion-TM kinematics found after both EE1 and EE2 may have been associated with muscle damage secondary phase events (e.g. inflammation and regeneration) connected to delayed force production disturbances of the agonists (e.g. Armstrong 1991, Faulkner et al. 1993). However, whether the disruption of myofilaments plays a role in accurate TM, in which the force levels are submaximal, is not clear at present.

6.2.1 Extension-TM

In the current study, the post-exercise extension-TM performance was intact (EE1), or it was even improved (decreased movement time), in parallel with the decreased peak velocity (EE2). The change of peak velocity may have been part of a performance optimization strategy of the CNS for antagonists' fatigue after EE2. On the other hand, it may have resulted from the non-fatigued extensors' decreased force production capacity as a consequence of impaired neural activation. On day 8 after EE2, movement times of extension-TM performances were shorter than pre-exercise. At the same time, peak velocities of extension-

TMs were returned back to pre-exercise values. This phenomenon must be seen as adaptation to eccentric exercise and / or as part of the learning process associated to decreased arm oscillation on target zone.

Despite large – although nonparallel - modulations in triphasic EMG activity pattern after two exercise protocols, the extension-TMs were as sharp as pre-exercise. Therefore, factors other than activity changes must have contributed to success of extension-TM task. The increased stiffness of the elbow joint (narrowed ROM) found after EE2 - most likely originated from elbow flexors - may have assisted the flexors in halting the movement on the target zone (Jaric et al. 1998) also after EE1. Increased stiffness may have also affected the decrement of arm total oscillation (shorter movement trajectory) observed acutely and delayed after EE2. In addition, the present responses after EE1 and EE2 suggest that muscle-fatigue-related mechanisms were operative in optimal modulation of the second TRI burst amplitude and timing. Referring to the unsuccessful neural adjustments found in flexion-TM after both exercises, and the larger oscillation on the target zone found after EE1, this emphasizes the importance of a final adjustment on the target by a non-fatigued muscle group (agonist 2nd burst). The preserved movement performance in the extension-TM would suggest an adequate ability of the flexors' to decelerate the movement. Thus, it seems that the antagonist force production capacity does not play a major role in accurate TM performance.

After EE1, in the acute phase of recovery, an earlier burst of the non-fatigued agonist TB muscle was found, followed by its delayed and lengthened 2nd burst. These different EMG changes emphasize the possibilities of neural adjustments to contractile failure of one muscle group. As suggested by Jaric et al. (1999), different peripheral segmental reflex and/or central mechanisms may operate in agonist versus antagonist muscle fatigue. This would allow the CNS to adjust for fatigue of the antagonistic flexor muscles. The amplitude of non-fatigued TB 1st and 2nd burst was reduced after both EE1 and EE2. Since the rather identical acute and delayed decrease of extensor TB activity was also found in maximal isometric action after EE2, the likely reason for activity reduction (central fatigue) lies at the supraspinal and/or spinal level (Gandevia 2001). According to Gandevia (2001), "several possible routes for peripheral inputs (including III and IV) may affect the firing rates of motoneurons in fatigue". By utilizing cervical evoked motor potential (CMEP) and ischemia, Martin et al. (2006) have shown that, during elbow flexors fatigue, the direct and/or indirect inputs of small muscle afferents would inhibit extensor motoneurons in the human arm. More recently, the activity of group III and IV muscle afferents have been shown to facilitate both extensor and flexor motoneurones, but to have an inhibitory effect on cortical cells projecting to these muscles (Martin et al. 2008). In addition, it is possible that pain itself may cause inhibition of motor system excitability both at the cortical level and the spinal level (Le Pera et al. 2001). In the present study, BB muscle afferents could thus result in neural adjustments of both BB and TB muscles.

In extension-TM, the reduction of agonist TB amplitude (EE1 and EE2) was paralleled with a drop of the elbow flexor amplitudes (EE2) (Fig. 20 and 21). Contrary to our expectations, after EE2, the flexor amplitude changes did not evenly follow their active stretch reflex test amplitudes, which were decreased acutely during the 2 h post-exercise recovery period. In the extension-TM task, while activated during lengthening in the movement braking phase, flexors are expected to have a reflex potentiation in their activity pattern. Despite peripheral disturbances induced by the present repeated eccentric actions, this gain appeared to remain. On the other hand, it is also possible that the antagonists' feedback gain of this kind in TM is overrated.

After EE2, together with the acute amplitude reduction of the antagonist BB in extension-TM, the modulations of the fatigued flexors timing (earlier) and duration (increased) were observed as well. Modulations seemed to concentrate on activity patterns of SA-TM. This must be seen as indicative of peripheral force production failures, which have been suggested to be more sensitive to reductions in neural drive at shorter muscle lengths (Prasartwuth et al. 2006, Skurvydas et al. 2010). The delayed post-exercise improvement of the extension-TM performance after EE2 was coexistent with the later timed onset of TB. Thus, the shortened motor time at the end of study period may have been indicative of the delayed learning progress. Though there were some post-exercise timing and duration differences between the TM ranges, the changes of extension-TM performance and kinematics were not found to be angle specific.

6.2.2 Flexion-TM

The acute deterioration of flexion-TM performance (increased movement time after EE2), main movement kinematics (EE1 and EE2) and increased movement oscillation (EE1) were concomitant with the drop of flexor 1st bursts amplitudes (e.g. Fig. 20). According to Corcos et al's (2002) "central fatigue strategy", a reduction of agonist peak EMG may have been part of CNS compensation, in order to diminish the effects of peripheral fatigue and to prevent task impairment. In parallel to that strategy, antagonists TB peak was delayed acutely after EE1, but in contrast to that strategy, we did not observe a change in antagonist onset or in TM distance after EE2. In the flexion-TM task, force production deficit of agonists was likely compensated by modulations in the central motor program, e.g. by the elongated agonist burst (Fig. 19) (see also Miles et al. 1997, Corcos et al. 2002). The decrement of activity of BB after EE1 is suggested to be due to metabolic fatigue activating small muscle afferents. However, based on the constant blood lactate concentration, EE2 did not produce metabolic fatigue. After EE2, the flexors amplitudes were decreased the most at 2 h post-exercise when all metabolic fatigue factors should already have disappeared.

As already mentioned, BB muscle afferents could result in neural adjustments of both flexor and extensor muscles. The deterioration of the flexion-TM performance after EE1 and EE2 occurred and disappeared in parallel with the modulations in triphasic EMG pattern (Fig. 18 and 19). Referring to the potential mechanisms modulating the two latter EMG bursts of the triphasic pattern

in a deafferented patient, Forget & Lamarre (1987) have demonstrated that peripheral sensory information does modulate the timing and size of the antagonist activity in fast target movements. It is also well known that the second agonist burst is possibly affected by proprioceptive information during the movement (e.g. Hallett et al. 1975). In a fatigue situation, the functional role of proprioceptive information in position matching has been found to be disturbed after repeated maximal eccentric muscle actions (Saxton et al. 1995, Brockett et al. 1997, Pedersen et al. 1999). In the position-matching test performed without vision, the exercised arm was either flexed more (Saxton et al. 1995) or less (Brockett et al. 1997) as compared to the non-exercised arm. These authors explained the matching errors by the exercise-induced changes in muscle afferent discharge. On the other hand, Gregory et al. (2004), did not observe consistent changes in muscle spindle responses after series of eccentric contractions.

Acutely after present EE2, AOMS was perceived in elbow flexors and the joint stiffness was increased during the early recovery period of 2 h. The experimentally induced muscle pain has been observed to decrease agonist EMG and attenuate the acceleration profile of the fast elbow movements (Ervilha et al. 2004a, b). Our subjects described the AOMS to be sharp and cramp-like in nature and to diverge from the DOMS. The cramping type pain was experienced especially during the fast TM and when the elbow was passively extended. The AOMS was coupled to unexpected powerlessness during the muscle work, which refers to protective inhibition of the CNS. Moreover, acute inflammation response and acute soreness have been reported to develop within eccentrically exercised muscles at 2 h and 4 h post-exercise (MacIntyre et al. 2001, McIntyre et al. 2000, McIntyre et al. 1996). Thus, small group III / IV muscle afferents, which have been shown to be sensitized to mechanical, thermal, and chemical changes within muscle (Gandevia 2001), might well have been responsible for the current EMG amplitude reductions in flexion-TM.

The small group III / IV muscle afferents have been reviewed to have several routes to mediate inhibitory influence on both α - and γ- motoneurones (Gandevia 2001). Feedback of these afferents have shown to have an effect on planning of aimed movements, on supraspinal cortical, subcortical and propriospinal motor outputs, and on α - motoneurones at the spinal level (Gandevia 2001). While having a direct reflex inhibitory effect on motoneurone firing rates in muscle fatigue (Gandevia 2001), group III and IV muscle afferents may inhibit motoneurones through pre-synaptic inhibition of Ia afferents (e.g. Garland 1991, Garland & McComas 1990, Rossi et al. 1999). The agonist 1st burst in triphasic activity pattern of TM has been supposed to be pre-programmed (planned) and not to be under peripheral sensory feedback control (e.g. Forget & Lamarre 1987, Hallett et al. 1975). In this sense, the possible perturbation of fusimotor drive would not have influenced flexors activation in the flexion-TM. On one hand, the identical and parallel timed acute post-exercise drop of flexors amplitude, both in TM (EE1 and EE2) and in active stretch reflex test, (EE2) suggested that the agonist activation might have been supported by peripheral feedback. To find another explanation for the present agonist EMG drop, our

recent findings suggest that sarcolemmal function of the fastest MUs could be affected acutely up to 2 h and delayed after eccentric actions following the recovery of maximal force production (Piitulainen et al. 2010). Regardless of the original reason for possible sacrolemmal dysfunction (e.g. McBride et al. 2000, McNeil & Khakee 1992), it would have explained the current reductions of agonist EMG found both in flexion-TM and in maximal flexion actions after the present exercise protocols. In line with this reasoning, the acute 2 h increment of absolute BB and BR 2nd burst amplitudes in flexion-TM - coincided with an increment in their durations – after EE2 may have been central compensation in order to optimize the task completion.

The present post-exercise amplitude changes of a muscle were not congruent when it worked as an agonist or as an antagonist. In the present extension-TM and flexion-TM, antagonists' post-exercise amplitude changes seemed to follow the changes of agonists (Fig. 20 and 21). The antagonist motoneuron's excitability level would be matched to agonist activity by parallel segmental and suprasegmental inputs to antagonist motoneurons and to their corresponding Ia inhibitory interneurons (Lundberg 1970). The present behaviour of antagonist amplitude could well be linked to central reciprocal control and to reciprocal inhibition. After the EE2, both in extension-TM and in flexion-TM, the co-activation agonist and antagonist was increased during the 2 h recovery period. In parallel with the lengthened co-activation, EMGs of antagonistic muscles during the co-activation phase were decreased. Notably, the current EE2 seemed to modulate the triphasic EMG activity pattern co-activation phases of TM, wherein flexor force production decreased the most (SA-TM). The post-exercise modulations in co-activation have been suggested to relate to disturbances in reciprocal control (Leger & Milner 2001). In the present flexion - TM, the post-exercise changes in overlapping were connected to performance impairment, while extension-TM was improved. Thus, the post-exercise modulations of triphasic EMG activity pattern must, at least partly, be seen as an adjustment of reciprocal control of the CNS for the elbow flexors fatigue, damage and soreness.

6.3 Effects of eccentric actions on RM performance and control

6.3.1 Muscle fatigue

Despite the large and long-term decrease in maximal force production after both eccentric exercises, the RM performance remained rather intact. After the EE1, only the earlier timed BB (increased braking latency) at the 0.5 h point reflected a post-exercise fatigue effect. This was in contrast with our observations in fast TM showing reduced performance after eccentric fatiguing exercise. In the present TM, agonist fatigue reduced the acceleration towards the target and caused oscillation over the target zone. In the present RM task, the movement velocity was fairly close to the one measured in the target movement. However,

this type RM task does not necessary require as high precision as the TM which was performed as fast and as accurate as possible. Furthermore, the absence of clear changes in the RM test may be explained by the fact that this type of bi-directed movement task is controlled equally by both the elbow extensors (non-fatigued) and the flexors (fatigued). In line with this, the observed EMG activity of the BB muscle did not reach 50% of the maximal values measured in the dynamic testing conditions. Additionally, only one of the elbow flexors - however, the main flexor - involved in this RM test was recorded. After the present EE2, the observed decrement of peak velocity (MA-RM) and increased time to peak velocity (Fig. 22), i.e. the modulation of RM symmetry, suggested a deteriorated deceleration and acceleration ability of elbow flexors acutely post-exercise. Actually, it has been shown in rapid discrete (Jaric 2000, Mirkow et al. 2002) and oscillatory elbow movements (Mirkow et al. 2002) that antagonistic muscle fatigue, strengthening (Jaric 2000), or joint angle (Mirkow et al. 2002) (i.e. muscles ability to exert force), may affect the movement symmetry (acceleration/deceleration). It has been postulated that submaximal (neural activation) tasks, especially at short muscle lengths, may be difficult to perform after damaging eccentric actions (Prasartwuth et al. 2006). After EE2, regardless of the largest force loss at short muscle lengths and modulations in BB EMG burst (timing, amplitude or duration), a clear elbow angle specific deterioration in RM performance was not found.

6.3.2 Muscle damage and soreness

In the current study after EE2, in parallel with the delayed increment of the movement amplitude, decreased movement frequency was observed. It has been shown that RM amplitude is linked to movement frequency (Feldman 1980, Wallace 1989, Bonnard & Pailhous 1999). In addition, similarly to present findings, it has been shown in elbow goal directed movements (e.g. Brown & Cooke 1981) that there is also a positive correlation between the movement amplitude and peak velocity. Therefore, the present delayed changes in RM kinetic after EE2 cannot be fully explained by muscle fatigue. However, if the present delayed modulation in RM amplitude resulted from muscle damage or fatigue, the findings of deafferented patients (Bonnard & Pailhous 1999), performing without the support of proprioceptive feedback, may serve as an answer. Without the aid of vision, non-preferred (intentionally constrained) RM of patients showed increased instantaneous fluctuation and RM amplitude returned to normal natural preferred behaviour (subject freely chosen amplitude for certain frequency) (Bonnard & Pailhous 1999). In the present type non-preferred 60° maximal velocity RM, it was impossible for a subject to clearly see the forearm and to match the amplitude exactly. By contrast, the most important priority was to concentrate to maintain the movement rhythm. Actually, it has been suggested that there is a natural frequency for rhythmic movements. This natural frequency is claimed to be controlled by CNS tonic co-activation together with central generator accomplishing the reciprocal activation of the antagonistic muscles (Feldman 1980). In this respect, the present delayed RM amplitude

modulations after EE2 may have occurred due to changes in central control as a consequence of muscle damage or soreness. The movement amplitude increased without changes in antagonistic muscles activity burst amplitudes. However, increased BB duration and shortened activity of the TB muscle were observed. In un-fatigued muscle, both the flexor and extensor EMG amplitudes have been found to be decreased with decreasing frequency (increased duration) of the RM (Feldman 1980, Nicol et al. 1997). The current results after EE2 are in line with the findings after EE1, when exercised flexor muscle activity amplitude was increased while RM amplitude and performance was preserved. It seems that the neuromuscular system is capable to compensate prolonged eccentric-induced contractile failure by optimizing antagonistic muscles coordination.

After the present EE2, BB EMG burst peak amplitude (in parallel with segments aEMG) was decreased 2 h post-exercise in all three RM ranges (Fig. 24). The burst peak EMG value would be the most reliable indicator to describe the amount of potential reflex response in RM activation pattern resulting from the stretching of an active muscle. In particular at the 2 h point after EE2, the active BB stretch reflex amplitude decreased as well which suggested a weakened reflex-mediated excitation of the motoneuron pool. After the current EE2, the contradictory post-exercise BB activity changes in MIAs compared to BB activity changes in RM were probably due to different amount of cortical control of two different maximal tasks. While a decrease of stretch reflex response in an active condition was observed, the reflex response appeared to be facilitated in passive conditions. The pre-exercise reflex response in a passive condition could not be observed in any of the subjects, but after the exercise it was observable in most of the subjects. Possibly the reduced gamma co-activation overrode the facilitation, which was seen in the passive condition. Therefore, the post-exercise overall response in the active condition was reduced, even if the spindles were more sensitive to the stretch. As indicated from the increased passive stiffness and swelling after the present study, muscle damage may have affected receptor responses (cf. Saxton et al. 1995, Brockett et al. 1997). After the present eccentric actions, it is likely that the spindles and golgi tendon organs were intact and able to respond normally to stretching. In this respect, it is also possible that the classical protective Ib inhibition may have influenced the observed acute BB activity drop. Other candidates responsible for the decreased BB activity are the group III (A-delta) and IV (C) muscle mechano-nociceptors. In muscle fatigue, group III and IV muscle afferents are suggested to inhibit muscle activation by pre-synaptic inhibition of Ia afferents at the spinal level (e.g. Bigland-Ritchie et al. 1986, Garland & McComas 1990, Duchateau & Hainaut 1993). If, after the present EE2, the small muscle afferents were responsible for decreased flexors activation acutely post-exercise, their effect seemed to be the opposite compared to delayed situation.

After the current EE2, passive stiffness developed and peaked in elbow flexors in parallel with the drop of BB neural activity in RM at 2 h post-exercise. The increased passive stiffness may be associated with muscle soreness and

with sensitization of small muscle afferents. In muscle, both group III and IV fibres are known to transmit dull-aching or cramping type sensations of pain (O'Connor & Cook 1999). After EE2, which did not develop metabolic fatigue, subjects experienced pain in their flexor muscles already immediately and 2 h post-exercise. In the elbow cyclic (Ervilha et al. 2005) and flexion movements (Ervilha et al. 2004a, b), experimentally induced muscle pain has been shown to decrease agonist EMG and to attenuate the acceleration profile. The effects of pain on the movement strategy and on muscle activity have been found to be dependent both on task effort - accuracy demands and the pain intensity level (Ervilha et al. 2004a, b).

In the delayed recovery phase of EE1, while the RM kinematics was preserved, BB EMG activity was significantly enhanced. At the same time, no particular change was observed in the EMG activity of the antagonist TB muscle. These results seem to indicate that the RM performance was maintained despite indicators of muscle soreness/damage. The recent paper of Martin et al. (2006) could be helpful in explaining the associated increase in BB activity in the RM movement. These authors produced conclusive evidence that "during fatigue, inputs from group III and IV muscle afferents from homonymous or antagonistic muscles depress extensor motoneurons but facilitate flexor motoneurons". Thus, the inputs from these afferents are not uniformly dispersed. Especially relevant for the present results is the conclusion of Martin et al. (2006) that regardless of what muscles (extensors of flexors of the forearm) are being fatigued, the motoneurons innervating the flexors are likely to become facilitated. For the present study this would support the observed increased BB activation at day 2 (Fig. 23). Regarding the present maintained activity of the elbow extensors, this may be in line with the suggestion of Martin et al. (2006) that due to a more pronounced inhibitory influence of these afferents on extensor muscles, they may require greater cortical drive to generate force during fatigue.

There are several spinal level mechanisms that may cause delayed increment in RM muscle activation after EE1. Firstly, activity potentiation may be due to increased γ-activation increasing the sensitivity of muscle spindles. It has been suggested (Nicol & Komi 2003, Regueme et al. 2005) that increased neural adjustments result from feedback gain mediated through group III and IV muscle afferents ("vicious circle" theory of Travell et al. 1942). These small diameter afferents associated with muscle damage are known to be sensitive to inflammation substrates (Armstrong et al. 1991), as well as to intramuscular increases in pressure and temperature (Mense & Meyer 1988, Rotto & Kaufman 1988). Animal studies have shown that activated noci- and chemosensitive group III and IV afferents have an excitatory effect on γ-motoneurons (Appelberg et al. 1983, Jovanovic et al. 1990, Johansson & Sojka 1991), which can potentiate neural activation due to increased sensitivity of muscle spindles (Djupsjöbacka et al. 1995a, b, Matre et al. 1998). After the present EE1, this mechanism may have contributed to the observed rise of BB S1 and S2 activity at day 2 and day 7 post-exercise. It must be cautioned, however, that some controversy exists regarding the applicability of these animal results to human studies (see Knutson

2000). Secondly, supporting also BB S1 and S2 increased activity, in painful muscles Lund et al. (1991) observed a reduced activity when acting as agonist and an increased activity as antagonists during voluntary movements. This so-called pain adaptation has been confirmed in clinical pain (Graven-Nielsen et al. 1997). Controversy still exists, however, on the exact pain effects on motor system both at the cortical level and the spinal level (Le Pera et al. 2001, Matre et al. 1998, Weerakkody et al. 2003, Ervilha et al. 2004a, b). For instance, Matre et al. (1998) demonstrated a significant increase of the human stretch reflex with pain in the relaxed, but not in the contracted muscle, indicating that the muscle spindle system is most sensitive to pain without α-γ co-activation. In the present case of eccentric-induced muscle fatigue, it should also be taken into consideration that muscle pain was felt in given testing pain conditions, but not experienced as much during the RM task. Thirdly, it has been suggested that eccentric actions may have a direct effect on muscle spindles or on tendon organs (Saxton et al. 1995, Brockett et al. 1997, Pedersen et al. 1999), causing changes in their discharge. In cats, during stretch of the passive muscle and intrafusal contraction, Gregory et al. (2004) observed slightly increased mean sensitivity of both primary and secondary spindle endings after series of 20-150 eccentric actions. It was concluded that the intrafusal fibres are not damage-prone to eccentric actions similarily to extrafusal fibres. Tendon organs have also shown to be able to monitor passive tension after eccentric actions (Gregory et al. 2002, 2003).

It has been shown that the BB receives stronger short latency monosynaptic inhibitory projection from the TB than TB from BB (McChelland et al. 2001). In the present study after EE1, this might well have explained the shorter overlap of BB over the TB during the extension phase. With the enhanced BB activity on day 2 post-exercise, TB overlapped BB more in its braking activity during the flexion phase, which might reflect changes in reciprocal inhibition. However, due to opposed and decreased overlap on day 7 after EE1, it can only be assumed that repeated eccentric actions of elbow flexors caused modulation in the reciprocal control of antagonistic elbow muscles. According to Lundberg's original hypothesis (1970), and with the view of reciprocal control of antagonistic muscles (Crone & Nielsen 1994), the excitability level of antagonist motoneurons to match agonist activity would be ensured by a parallel segmental and suprasegmental input to antagonistic motoneurons and to their corresponding Ia inhibitory interneurons. In addition, it has been shown by ramp-and-hold reflex measurements that antagonistic muscles might be regulated so as to make the reflex responses comparable to a given joint rotational velocity (Nakazawa et al. 2001). The view of disturbed peripheral feedback acutely and delayed after eccentric exercise is also supported by the increased antagonistic muscles co-activation in SA-RM after EE2. The observed overlap (duration and amplitude) in TB-BB activity turn may have been due to central compensatory changes at elbow angles where the force production was especially deteriorated post-exercise. Leger & Milner (2001) have reported an increased post-exercise co-activation in a wrist tracking task, suggested to be due to the increased

common drive or decreased reciprocal inhibition of the antagonist muscles whenever the injured muscles were activated.

6.4 Acute learning of TM

The TM task improvement and the modulation process of associated triphasic EMG activity pattern were delayed with the instruction "perform always accurate". The EMG bursts determining the forearm terminal oscillation was modulated, that is, the antagonist and agonist 2nd burst EMG co-activation was decreased and the relationship of these bursts was increased during the acute practice. The improvement of TM performance (reduction of TM time) was connected to shorter terminal oscillation time.

6.4.1 Development of TM time and accuracy

Only the TM time (velocity demand) was improved during the practice. The improvement took place in both groups and both TM. However, the A-group (accuracy group) never reached the V-group (velocity group) level in flexion-TM. In extension - TM, MT did not diverge between groups. In this respect, it should be noted that flexion-TMs were performed before the extension-TMs, which may have influenced the extension-TM learning process. The greatest change in performance has been found to appear over the first 40 to 200 repetitions (Almeida & Latash 1995, Corcos et al. 1993, Flament et al. 1999, Kempf et al. 2001). In the present study, the greatest development of V-group MT appeared within 30 reps (Fig. 25).

The deviation of the arm final position (accuracy demand) was unchanged during the present practice. This result matched earlier findings after the practice of 660 elbow flexions (Almeida & Latash 1995) or of 1000 elbow flexions and extensions (Darling & Cooke 1987a). However, it contrasted the results of Ilic et al. (1998) as they found the variable error of final movement position to be decreased in extension-TM after the practice of flexion-TM. In the study of Corcos et al. (1993) although "accuracy" was not part of the stated learning task, the final position remained, which was found to oppose the speed-accuracy trade-off concept. The present results, within the framework of classical trade-off, favour the practice under the velocity criterion (TM time).

6.4.2 Progress of movement kinematics and terminal oscillation

In A-group, the peak velocity of TMs never reached the level of the V-group, which plateaued after 10 repetitions in both TMs (Fig. 26). The increased main movement velocity was not a consequence of increased movement amplitude, as it has been shown by earlier studies (e.g. Brown & Cooke 1981), since the amount of overshooting (main movement amplitude) remained constant during the present practice. The increased movement velocity has been shown to asso-

ciate with the shortened TM time (e.g. Corcos et al. 1993). However, in the present study this was true only within the A-group, since V-group TM time continued to shorten without remarkable change in peak velocity. When the subjects have performed with the similar instructions as the subjects in the present V- group, the development of TM time has not been related to movement maximal velocity (Liang et al., 2008).

In parallel with shortened time to main movement peak velocity, the symmetry ratios of the present TMs were not changed. This was indicative of constant relation of tensions produced by the antagonistic muscles (Jaric 2000). It is known that the produced dynamic tension, or torque produced by a certain velocity, is muscle action type dependent and that more tension is produced in eccentric (lengthening) action.

Noteworthy, we found the oscillation time to decrease (Fig. 25) and correlate with the shortened TM time. As recently found in wrist joint (Liang et al., 2008) the current development of TM time was associated with decreased movement oscillation over the target zone. This indicated that the ability of the neuromuscular system to damp down the forearm oscillation was enhanced.

6.4.3 Modifications of triphasic EMG activity pattern

In contrast to earlier findings (e.g. Flament et al. 1999), together with the current decrease of burst durations, the development of antagonist and agonist 2nd burst EMG peak amplitudes and areas were not statistically significant. Probably, the more powered activation of active MUs along with the muscle-tendon complex intrinsic elastic energy were exploited, since the movement kinematics and TM time were improved in the present TMs. The force–velocity property of the muscle force-generating elements is known to contribute to the generation of the contractile impulse (e.g. Shapiro et al. 2005). The EMG amplitude of the antagonist muscle has been found to be two- or threefold lower compared to amplitude when it worked as agonist. Therefore, less activation was needed when a muscle acted as antagonist (lengthening muscle) than as an agonist (shortening muscle) (Shapiro et al. 2005). Supported by the constant symmetry ratio found in the present study, the utilization of elastic energy of the muscle-tendon complex in fast voluntary movement, including stretch shortening cycle type muscle action (Paulino et al. 2005, Selen et al. 2006), may compromise the requirements of muscle activation.

In the present study, the A-group antagonist and agonist 2nd bursts EMG area relation was increased during the acute learning. If the 2nd bursts role is to stabilize the arm on target (e.g. Berardelli et al. 1996, Hallett et al. 1975), the increase of this EMG relation suggests a lesser need for stabilization with practice. Moreover, in the current study, the co-activation (activity overlap) of the antagonist and agonists 2nd bursts was observed to decrease in A-group during the practice of both TMs (Fig. 29). With practice, the A-group antagonist and agonist 2nd bursts overlap became closer to the values of V-group, being equal at the end of practice. The formation of the more reciprocal activity pattern has been taken as an indicator of practised or programmed movements (e.g. Brown

& Cooke 1981). However, the present modulation of two latter EMG bursts towards the more phased pattern refers to sharpened reciprocal reflex control (i.e. reciprocal inhibition) of these bursts, since they are suggested to be under peripheral sensory feedback control (e.g. Forget & Lamarre 1987). In this regard, as eccentric type muscle actions have been shown to disturb proprioception (e.g. Brockett et al. 1997, Carson et al. 2002, Saxton et al. 1995), we found in this thesis project the antagonist and fatigued agonists 2nd burst to overlap to a lesser extent post-exercise (original paper IV). The improved co-ordination of antagonistic muscles, detected as a refinement of phasic pattern in the present TMs, may also serve as a protective mechanism when the voluntary bidirectional movement gets faster, or in the case of muscle fatigue.

The co-activation of agonist and antagonist was not changed during the present practice. The reduction of these bursts co-activation has seen to be consistent with the skilled performance features (e.g. Corcos et al. 1993). If the modulation of co-activation is a consequence of improved central preprogramming, in the first place, a reduction of agonist and antagonist coactivation would have expected to appear. Noteworthy, in this thesis project after EE2, the co-activation of agonist and antagonist in flexion-TM and in extension-TM was modulated. The observed elongation of the co-activation, both in agonist and antagonist fatigue, referred to a disturbed central motor programming of the agonist 1st and antagonist bursts. In this sense, the present unchanged co-activation of the agonist and antagonist suggest that the control of this muscle pair is unaffected by the movement kinematics (e.g. increased main movement velocity). Thus, the main source of the agonist-antagonist, and the antagonist-agonist 2nd muscle pair, neural control is likely located differently in the CNS.

As an interesting finding, the present learning study showed for the first time the agonist (elbow flexors) motor time to be decreased, together with its increased EMG and with the increased peak velocity (Fig. 28). The agonist latency (motor time) has been shown to be independent of instructions and practice (e.g. Almeida & Latash 1995, Brown & Cooke 1981, Corcos et al. 1993, Darling & Cooke 1987b). However, it is well known that requirements of propulsive force production are fulfilled by increasing the duration and / or amplitude of the agonists (dual-strategy hypothesis) (Corcos e al. 1989, Gottlieb et al. 1989). Since, in the present TM, the agonist EMG duration was shortened and the amplitude increased, probably the recruitment of larger motor units with increased firing rate, or the more synchronous activation of the motor units (Raikova et al. 2005), was used as a control device for more powerful force production. Noteworthy, during the practice of extension-TM neither the agonist (TB) latency nor its EMG (amplitude and area) was changed significantly. Probably this resulted from the fact that flexion-TM was practiced before the extension-TMs.

7 PRIMARY FINDINGS AND CONCLUSIONS

The main findings and conclusions of the present study can be summarized as follows:

1) As expected, the present exercise protocols employing unaccustomed maximal lengthening actions on elbow flexors generated several well-known symptoms to muscle damage. It was shown that the recovery was faster after maximal concentric exercise, when muscle damage and soreness were absent. The present eccentric actions disturbed the normal neuromuscular function, especially a few hours post-exercise.

2) The current eccentric exercise protocols did not disturb the extension-TM performance like those observed in deafferented patient who are deprived of sensory feedback. The extension-TM was even improved (velocity demand) several days post-exercise. In contrast, the flexion-TM was acutely deteriorated (velocity demand). The delayed post-exercise modulations of TM and RM kinetics, kinematics, and associated muscle activities may be associated with adaptation in sensory (proprioceptive) feedback as a consequence of muscle damage and soreness.

3) Both exercise protocols led to alterations in flexion-TM performance, when the fatigued muscle group acted as agonist. As expected, the performance was disturbed (TM) and modulated (TM and RM) the most during the acute 2 h post-exercise recovery period when the drop of maximal force was the greatest. These changes reflected a reduced acceleration and deceleration capacity of elbow flexors.

4) As hypothesized, the post-exercise performance of TM and RM at small elbow angles were changed the most. However, this study was unable to demonstrate any clear joint angle (muscle-length) specific effect of eccentric exercise on the fast voluntary movements' motor control.

5) The acute - 2 h - post-exercise changes in performance were concomitant with the agonists' decreased EMG amplitudes and changed timing (flexion-TM), and with the large drop in BB EMG burst amplitudes (RM). In TM, the direction of the post-exercise EMG amplitude responses of the fatigued muscles varied when they acted as agonist, antagonist, or agonist 2nd, while the non-fatigued muscle amplitude responses were always in the same direction. In contrast, the timing responses of a muscle were in the same direction irrespective of the role it acted. Thus, the CNS seemed to use the amplitude modulation of the EMG bursts for optimizing the performance in damaged and sore muscles.

6) The acute post-exercise reduction of elbow flexor EMG amplitudes, associated with a parallel drop in their active stretch reflex amplitudes, suggests an inhibitory reflex effect originating most likely from group III / IV mechano-nociceptors. However, the effect of small muscle afferents acutely post-exercise seemed to be different compared to the delayed situation, as the EE1 led to an increase of BB EMG activity in RM.

7) Most probably, the improvement of TM time through practice was associated with changes in pre-programmed descending command. The performance development was associated to both timing and amplitude modulations of triphasic EMG bursts. The development of TM performance during acute practice was associated with antagonist and agonist 2nd bursts increased EMG area relation and decreased co-activation. The improvement of TM time was associated to decrease of TM terminal oscillation. The reduction of TM terminal oscillation suggested the involvement of proprioceptors reflex control refining the co-ordination of antagonistic muscles and enhancing joint stiffness.

8) An exercise that involves maximal eccentric muscle actions and cause muscle soreness leads to unigue responses and disturbances of neuromuscular system. If the exercise is exhaustive enough, the muscle length (joint angle) dependent perturbation of movements and the reflex arc supported control of movements may be disturbed. The influence is primarily seen in the very fast movements. This may have importance for many sport activities.

YHTEENVETO

Kyynärvarren nopeiden tahdonalaisten liikkeiden motorinen kontrolli harjoituksessa aiheutetun lihassoluvaurion ja lihaskivun sekä oppimisen interventiona

Tämän tutkimusprojektin tarkoituksena oli selvittää, aiheuttavatko harjoituksessa aiheutetut lihassoluvauriot samanlaisia vasteita nuorilla ja terveillä miehillä kuin on havaittu ihmisillä, joilta puuttuu hermolihasjärjestelmän kyky välittää sensorisia ärsykkeitä. Lisäksi tutkittiin, ovatko nämä vasteet sidoksissa liikkeen nivelkulmaan. Kyynärvarren target- (TM) ja rytmisen (RM) liikkeen lihasten EMG -mallin muutoksia verrattiin venytysrefleksin EMG -vasteisiin. Target -liikkeen oppimistutkimuksessa selvitettiin, oliko suorituksen paraneminen yhteydessä korjaavien liikkeiden vähenemiseen ja miten ne liittyivät EMG -mallin muotoutumiseen. Vastoin havaintoja, joita on saatu niillä, joilta puuttuu sensorinen palautejärjestelmä, tässä tutkimuksessa käytetty maksimaalinen eksentrinen väsytysharjoitus, ei häirinnyt ojennus- eikä rytmistä liikettä. Toisaalta koukistusliike oli akuutisti häiriintynyt. Suoritukset muuttuivat eniten pienillä kyynärvarren nivelkulmilla ja erityisesti kaksi tuntia harjoituksen jälkeen. Suoritusmuutokset olivat samanaikaisia vaikuttajalihaksen EMG:n (korkeus) laskun ja ajoituksen (koukistusliike) sekä kyynärvarren koukistajien EMG:n (korkeus) laskuun rytmisessä liikkeessä. EMG:n lasku oli samanaikaista myös aktiivisen venytysrefleksin EMG -vasteiden laskun kanssa. Yksi käytetyistä väsytysharjoituksista aiheutti viivästyneesti kyynärvarren koukistajien EMG kasvun RM -liikkeessä. TM -liikkeen akuutin oppimisharjoittelun aikana vastavaikuttaja- ja vaikuttajalihaksen EMG -aaltojen (EMG -mallin toinen ja kolmas aalto) suhde kasvoi, ja niiden päällekkäisyys laski, mikä oli yhteydessä korjaavien liikkeiden vähenemiseen sekä liikkeen nopeutumiseen. Yhteenvetona voidaan todeta, että käytetyt eksentriset harjoitukset eivät aiheuttaneet selviä nivelkulmasta riippuvia vaikutuksia nopeiden liikkeiden motoriseen kontrolliin. Hermolihasjärjestelmä näyttäisi käyttävän EMG:n korkeuden säätelyä optimoidakseen liikkeiden suoritusta lihasväsymyksen sekä lihassoluvaurioiden ja lihaskivun aikana. Akuutit EMG -löydökset viittaavat häiriintyneeseen - ryhmien III ja IV lihasreseptorialkuiseen - liikkeen refleksikontrolliin. Liikkeiden viivästyneet kineettiset, kinemaattiset ja EMG -löydökset viittaavat hermolihasjärjestelmän sopeutumismuutoksiin. Akuutin oppimisharjoittelun aikaiset muutokset liikeradassa ja EMG -mallissa viittaavat siihen, että liikkeiden aikainen lihasten refleksipalaute kontrolloi vastavaikuttajalihasten koordinaatiota lisäten niveljäykkyyttä.

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