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The Effects of Eccentric Exercise on Neuromuscular Function of the Biceps Brachii

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This study investigated the effects of a bout of exercise-induced muscle damage on strength and motor skill of the elbow flexor muscles. Eight subjects performed 35 maximal isokinetic eccentric elbow flexions at $90°$ sec[-1] and maximal voluntary contraction (MVC) force, surface electromyography (EMG), plasma creatine kinase (CK) and tracking error associated with a one-dimensional elbow flexion/extension visuomotor pursuit task were studied at intervals up to 28 days after exercise. Subjects showed a post-exercise decline in MVC (mean = $63 \pm 11\%$ (s.d.) of pre-exercise after 1 day, $p<0.02$) and were still significantly weaker at 21 days. The delayed-onset plasma CK rise and the absence of any quantitative change in surface EMG suggest that the observed weakness was related to muscle fibre damage. Tracking performance decreased in all subjects with the greatest tracking error occurring 1 day post-exercise (mean = $127\% \pm 20\%$ of control value, $p<0.02$). There was a significant negative correlation between strength and tracking performance following exercise ($r^2 = 0.724$). The results demonstrate that performance in activities requiring fine motor control will be impaired for a number of days following a bout of damaging exercise.

Introduction

Delayed onset muscle soreness (DOMS) is a commonly experienced phenomenon associated with vigorous physical activity at all levels of pursuit and occurs when the exercise is unaccustomed or involves an increase in training intensity. Activities that involve a high component of eccentric contractions are more likely to result in DOMS (Schwane et al, 1983; Jones et al, 1989). Typically subjects complain of muscle tenderness (pain associated with contraction or pressure) and stiffness starting 8-12 hours after exercise which is most acute by 24-48 hours and subsides within 3-5 days (Armstrong, 1984). The mechanical, biochemical and pathological manifestations of DOMS have been well documented and include prolonged weakness (Newham et al, 1987), muscle swelling/loss of range of motion (Howell et al, 1993), an efflux of intramuscular proteins into the blood stream (Clarkson et al, 1992), and muscle fibre necrosis (Jones et al 1989).

Most studies of muscle function and DOMS have concentrated on changes in maximal voluntary isometric force and contractile properties elicited by electrical stimulation (Newham et al, 1983; Sargeant and Dolan, 1987). Few studies have examined the effect of DOMS on other aspects of neuromuscular control. Miles

et al (1993) showed a disruption of the pattern of agonist and antagonist muscle activity following eccentric exercise of the elbow flexors, whilst Saxton et al (1995) demonstrated an increase in muscle tremor and impaired proprioception of joint position after a bout of eccentric exercise. As yet no studies have investigated the effects of DOMS on the ability to execute a skilled task.

The aim of the present study was to investigate the effects of a bout of eccentric exercise of the elbow flexors on the performance of a visuomotor tracking task at intervals up to 1 month post-exercise, and to relate motor performance to alterations in muscle strength, plasma creatine kinase (CK) and voluntary electromyographic (EMG) activity.

Methods
Subjects
Eight healthy subjects (5 male, 25-40 years of age) who did not participate in specific upper body strength training were studied. All subjects were right handed as assessed by a standard handedness questionnaire (Bryden, 1976). Procedures were carried out with approval from the Local Human Ethics Committee and all subjects gave written informed consent prior to testing.

Exercise protocol and post-exercise testing
The eccentric exercise protocol used to induce DOMS consisted of 35 maximal voluntary eccentric contractions of the non-dominant elbow flexors, performed through $130°$ range of motion so that the subject finished the contraction in full extension. Contractions were performed at a constant angular velocity of $90°sec^{-1}$ using an isokinetic dynamometer (Kin-Com, Chattex Corp.) The protocol consisted of 7 sets of 5 repetitions with 4 sec between repetitions and a 2 minute passive recovery between each set. Subjects were given visual feedback via the force signal and encouraged to provide maximal efforts throughout each contraction. Assessments of maximum voluntary contraction (MVC) force, maximum voluntary EMG, plasma creatine kinase (CK), and visuomotor co-ordination were carried out prior to exercise and at 1, 3, 7, 14, and 28 days following the bout of eccentric exercise.

Muscle strength and plasma CK measurements
Three isometric MVC's of 3 sec duration were measured at 1 minute intervals at $90°$ elbow flexion using the Kin-Com dynamometer. MVC force was taken as the highest force level which could be sustained for 1 sec.

A 50 micro-litre capillary blood sample was drawn from a fingerprick into a heparinised capillary tube. Plasma CK activity was measured using a portable spectrophotometer (Reflotron, Boehringer Manheim, Australia).

Electromyographic (EMG) activity
EMG activity was recorded using 1 cm diameter gold disc electrodes (Grass) placed over the belly of the biceps brachii muscle with the active electrode just distal to the motor point of the muscle and inactive electrode a further 4 cm distal. The signals were amplified by 1000 and pass filtered below 1 KHz and above 1 Hz. In order to standardise the electrode placement for each testing session, upper arm 'maps' were used, consisting of landmarks on the surface of the skin marked on clear plastic wrap. The root mean square of the EMG signal (rmsEMG) during the period of MVC measurement was calculated off-line.

Skilled-tracking task

Visuomotor co-ordination was assessed using a one-dimensional visual pursuit elbow flexion/extension tracking task. Subjects were seated with the upper arm in 90° of flexion resting on a table just below shoulder height and held a lightweight lever arm attached to a rotary potentiometer positioned about the axis of rotation at the elbow. A target cursor was displayed on a PC monitor and computer driven in a semi-random sine wave pattern in the vertical plane. Subjects used the lever arm to control the position of an indicator cursor and were instructed to keep the (indicator) cursor aligned with the target cursor through 130° of flexion/extension. The amplitude of the target cursor varied with the frequency fixed at 5 cycles in 10 seconds and the target and indicator positions were digitised at 10 ms intervals. Accuracy in motor skill tracking was determined by the total sum-of-squares error between the target and indicator cursor waveforms expressed as a percentage of the total sum-of-squares amplitude of the target cursor waveform.

Each subject was allowed adequate time for familiarisation with the set-up and two practice tests (10 trials) prior to the first test measurement. Each test consisted of 5 trials of the 10 second tracking task and the mean of the best 3 trials was taken as the tracking score. In order to account for improvements in tracking score occurring as a result of learning, a group of 5 control subjects were tested on the same number of occasions as those subjects who performed the eccentric exercise bout, but performed none of the other tests.

Test reliability and Data Analysis

In order to determine the reliability of the test measurements the test-retest error from the results of two repeat tests (separated by 7 days) was measured in 5 subjects who did not form part of the study group. Coefficients of variation (CV's) of repeat tests were calculated as described by Thorstensson (1976) for MVC force, plasma CK and maximal voluntary rmsEMG. The mean CV across subjects for the test/retest measurements was 2.4% for MVC, 6.3% for CK and 19% for rmsEMG.

Results of strength, CK, and rmsEMG activity were analysed by using the Wilcoxin Signed Ranked Test (p<0.05). For inter-group comparisons of motor skill tracking data, the Wilcoxin Mann Whitney Test were used with significance set at p<0.05.
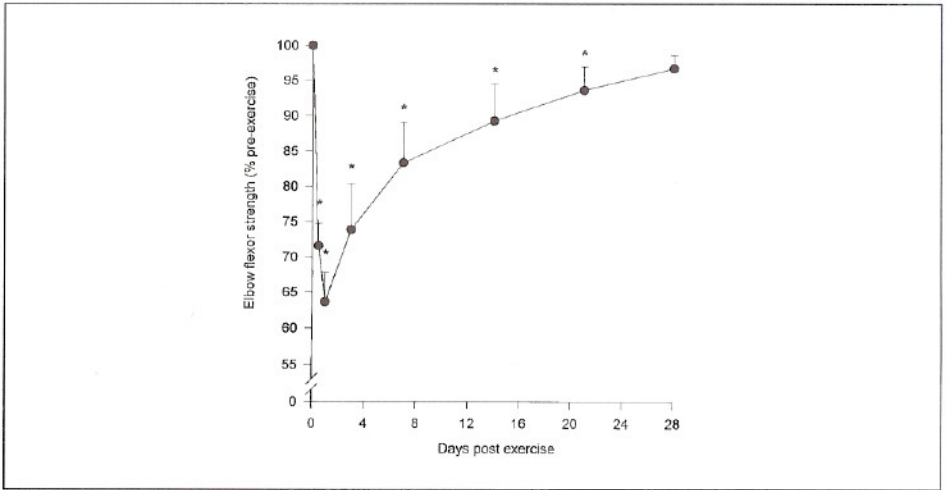
Results
Symptoms

All subjects reported some tenderness of the biceps, which was greatest 1-3 days post exercise. However, no subject indicated that muscle pain affected the ability to produce a maximal voluntary effort when required. Four subjects experienced difficulty fully extending the exercised elbow for up to 5 days post exercise. When these subjects came to complete the tracking task it was noticeable that full elbow flexion was difficult at first but adequate stretching of the biceps alleviated the problem for long enough to complete the tracking task without apparent difficulty.

Muscle Strength and EMG

All subjects showed a reduction in MVC force following eccentric exercise (Fig 1) which was most pronounced after 1 day (averaging $63.3 \pm 11.4\%$ (mean ± s.d.) of pre-exercise values, p<0.02). Strength recovered gradually over the time course

Figure 1   Changes in elbow flexor strength following eccentric exercise. Results are mean + s.e.m. for 8 subjects (*p<0.05)

Figure 2   Changes in plasma creatine kinase concentration following eccentric exercise. Results are mean + s.e.m. for 8 subjects (*p<0.05)
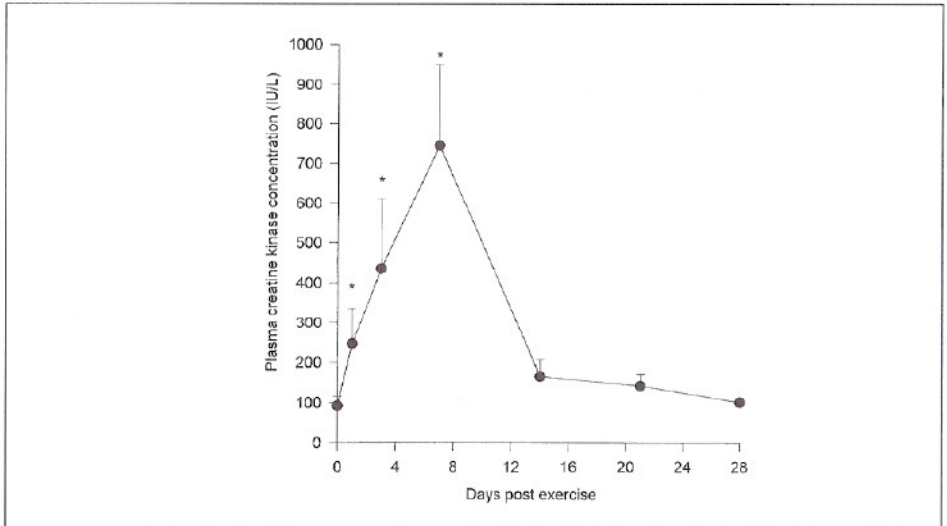
of the study, but remained significantly below pre-exercise values for up to 21 days. In contrast, the rmsEMG activity recorded during MVC's showed no consistent change following eccentric exercise. At 1 day post-exercise the mean maximal rmsEMG activity had declined by only 12.2 ± 15.6 % which was within the coefficient of variation for test-retest of this variable.

Plasma Creatine Kinase

Plasma CK levels increased following a bout of eccentric exercise. Mean CK levels were slightly raised 1 day post-exercise and showed significant increases above baseline levels at 3 and 7 days post-exercise (Fig 2). However, the responses were quite variable between individuals, with peak CK elevations ranging from 120% to 1864% of pre-exercise concentrations. After 14 days of recovery CK values had returned to the control range (Fig 2).

Tracking task

Immediately following the exercise protocol the mean tracking error increased by 14.1 ± 13.5% of pre-exercise values, and by 1 day tracking error had increased to 27.7 ± 20.1% of pre-exercise (Figs 3 & 4). Some recovery in tracking task performance occurred 3 days post-exercise and this trend continued over the time course of the study with tracking scores returning to pre-exercise levels by 14 days (Fig 4). However the control subjects, despite having very similar tracking scores as the test subjects prior to exercise, showed consistently superior performances over the exercised group and this was reflected in noticeable improvements in tracking score (mean 15.1%) over the first four test measurements (Fig 4).

The relationship between changes in MVC force and tracking score following eccentric exercise are shown in Fig 5. The time course of each variable was very similar (compare Figs 1 and 4) with a significant negative correlation coefficient ($r^2 = 0.724$) between strength and tracking error. However the relationship is not linear over the damage and recovery periods (compare lines in Fig 5) since

Figure 3   *Typical tracking trace of elbow flexors/extensors pre and one day post exercise (solid lines = target; dotted lines = elbow position)*
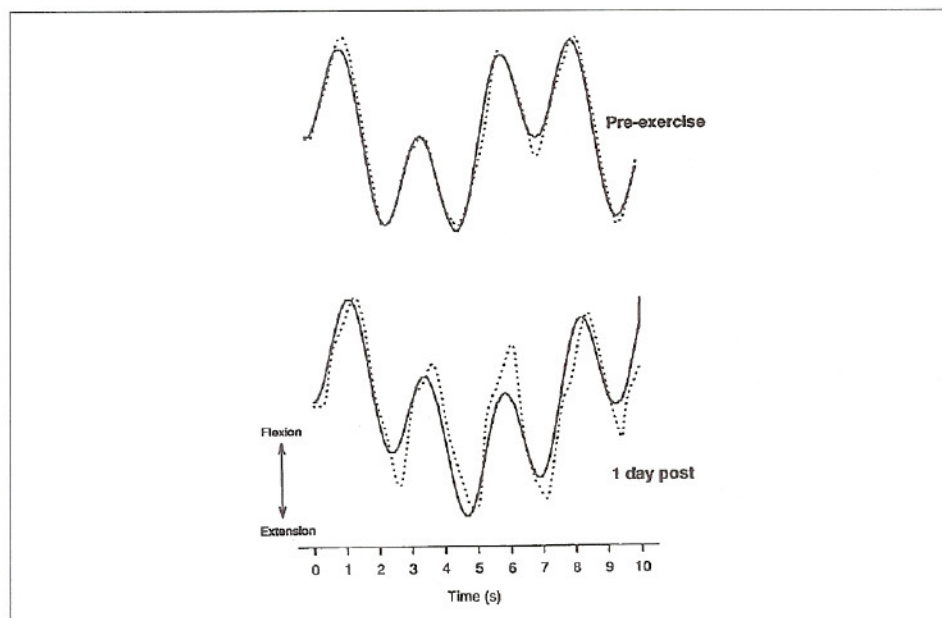
Figure 4   *Changes in tracking task error following eccentric exercise. Mean + s.e.m. (*p<0.05 for difference between groups)*
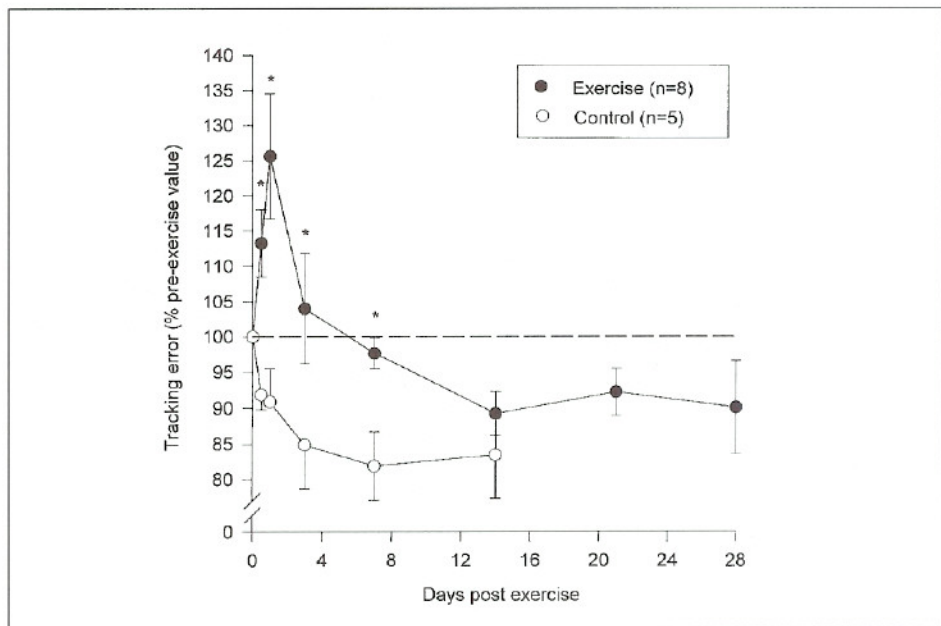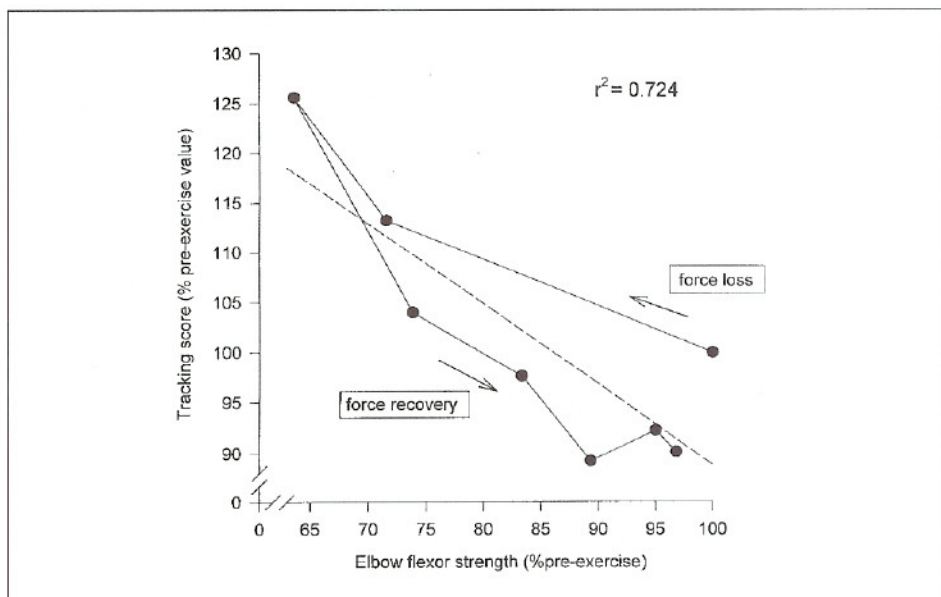

Figure 5   *Relationship between tracking score and strength following eccentric exercise (dotted line = line of best fit)*

subjects showed an improved performance in the skilled tracking task during recovery attaining a reduced tracking error 7 days after exercise compared with the pre-exercise values. The non-exercised control group also showed significant improvements in tracking task over time but it is interesting to note that the exercised group were still improving in tracking score long after the control group had reached a plateau (Fig 4).

Discussion

Isometric muscle strength and tracking ability both declined within hours after a bout of eccentric exercise of the elbow flexors and subjects still showed some muscle weakness up to 21 days after exercise. These strength losses agree with those found by other groups using similar paradigms, eg. Newham et al (1987) reported decrements in elbow flexor force of around 50% with recovery being incomplete two weeks after a bout of eccentric exercise. Likewise the plasma CK values observed in our subjects 3-5 days post-exercise are similar in magnitude and time-course to that previously reported (Clarkson et al, 1992).

Although most subjects complained of some discomfort in the muscle 2-3 days after exercise, they did not report that this affected their ability to perform any of the testing procedures. Muscle tenderness is a common consequence of eccentric exercise (Armstrong et al, 1974) and it has been suggested that DOMS may be associated with inflammatory responses in the muscle or connective tissue compartments (Jones & Round, 1990; Smith, 1991). It is well known that activation of pain receptors can have a negative effect on the ability to achieve full muscle activation (Stokes & Young, 1984) but several studies using transcutaneous electrical muscle stimulation have shown that sufficiently motivated individuals are capable of complete muscle activation despite considerable muscle soreness (Davis & White, 1981; Newham et al, 1983). The strength losses observed in our study can best be explained by a lowering of the inherent force-producing capability of the muscle resulting from exercise-induced damage to the contractile elements. The finding that rmsEMG values associated with maximal efforts did not show any significant decline in the days following eccentric exercise further supports this explanation, although the poor reliability of the voluntary rmsEMG prohibits conclusions to be made regarding this measurement.

We found that motor skill was impaired in the week following eccentric exercise, and although skill had recovered to pre-exercise values within 7 days, performance was significantly inferior to that of the control group. It is unlikely that DOMS was primarily responsible for the observed decline in motor skill since subjects did not report that muscle pain impaired their ability to perform the tracking task. Furthermore, motor skill was still impaired long after DOMS had resolved (3-5 days). A number of factors might be responsible for the loss in motor skill observed in this study. Firstly the pronounced weakness may have impaired force judgement during the tracking task, and this is supported by the close correlation between tracking error and strength loss over the course of the study. Saxton et al (1995) found that force proprioception was impaired following eccentric exercise such that subjects judged the isometric force of the exercise arm to be a similar percentage of maximum force despite a reduced force-generating capacity and we would envisage similar effects to have occurred in our subjects. However, the tracking task required very low forces to be generated,

little more than that necessary to flex the arm.

A second potential contributing factor to the loss in motor skill could be disruption of proprioceptive feedback from muscle spindle and cutaneous afferents. These receptors relay information to the central nervous system regarding muscle position and movement (Rothwell, 1994). There is evidence that eccentric exercise does not impair the ability to replicate joint position directly, but only in relation to using an external reference such as the contralateral arm (Saxton et al, 1995). It is possible that muscle spindle or tendon organ receptors may have undergone mechanical damage as a result of exercise but, to date, no studies have reported the effect of exercise-induced damage on discharge of the spindle or other afferent fibres. A further influence on motor skill could be corticomotor reorganisation secondary to altered feedback to the sensorimotor cortex. This could cause an alteration in the pattern of corollary discharge to the sensory portions of the brain which may provide a kinaesthetic reference by which the central nervous system can monitor sensory feedback resulting from a motor action (McCloskey, 1981). Changes in limb representations of the motor cortex have been described in a number of situations involving changes in peripheral input (Cohen et al, 1993; Pascual-Leone, 1993), and also following exercise-induced muscle weakness (Thickbroom et al, 1995).

In conclusion we have found a prolonged impairment in motor skill following a bout of eccentric exercise which may have been related to a loss in force-generating capacity, an alteration in proprioceptive feedback from the moving limb, or a secondary alteration in corticomotor control. It may be that alterations in proprioception and sensorimotor integration (even in the absence of muscle soreness) could account for the delayed rate of improvement in the skilled motor task following eccentric exercise. These findings have important implications in sports which require a combination of strength and fine control (eg. racquet sports) and suggest that motor skill and learning may be impaired for a considerable period of time subsequent to activities that result in exercise-induced damage to the muscle/tendon complex.

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