Experimental Physiology

Fatigue-induced change in corticospinal drive to back muscles in elite rowers

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This study examined post-exercise changes in corticospinal excitability in five 'elite' rowers and six non-rowers. Transcranial magnetic stimulation (TMS) was delivered to the motor cortex and bilateral electromyographic (EMG) recordings were made from erector spinae (ES) muscles at L3/L4 spinal level and from the first dorsal interosseous (FDI) muscle of the dominant hand. Each subject completed two exercise protocols on a rowing ergometer: a light exercise protocol at a sub-maximal output for 10 min and an intense exercise protocol at maximum output for 1 min. A trial of ten magnetic stimuli was delivered before each of the protocols and, on finishing exercise, further trials of ten stimuli were delivered every 2 min for a 16 min period. Amplitudes of motor-evoked potentials (MEPs) in each of the three test muscles were measured before exercise and during the recovery period after exercise. The non-rowers showed a brief facilitation of MEPs in ES 2 min after light and intense exercise that was only present in the elite rowers after intense exercise. In the period 4–16 min after light exercise, the mean (± S.E.M.) MEP amplitude (relative to pre-exercise levels) was less depressed in the elite rowers (79.4 ± 2.1 %) than in the non-rowers (60.9 ± 2.5 %) in the left ES but not significantly so in the right ES. MEP amplitudes in FDI were significantly larger in the elite rowers, averaging 119.0 ± 3.1 % pre-exercise levels, compared with 101.2 ± 5.8 % in the non-rowers. Pre-exercise MEP latencies were no different in the two groups. After light exercise MEP latencies became longer in the elite rowers (left ES, 16.1 ± 0.5 ms; right ES, 16.1 ± 0.4 ms; dominant FDI, 23.4 ± 0.2 ms) than in the non-rowers (left ES, 15.0 ± 0.3 ms; right ES, 15.2 ± 0.3 ms; dominant FDI, 21.5 ± 0.2 ms). There were no differences in MEP depression or latency between elite rowers and non-rowers after intense exercise. We conclude that the smaller degree of MEP depression in the elite rowers after light exercise reflects less central fatigue within corticospinal control pathways than that seen in the non-rowers. The longer latency of MEPs seen in the elite rowers may reflect recruitment of more slower-conducting fatigue-resistant motor units compared with the non-rowers. These differences may be because the energy requirements for the non-rowers during light exercise are closer to their maximum capacity, leading to more fatigue. This notion is supported by the lack of any difference between groups following intense exercise when both groups were working at their own maximum. *Experimental Physiology* (2002) **87.5**, 593–600.

Merton (1954) defined motor fatigue as a reduction in the force generated by a muscle or a group of muscles after sustained or repeated contraction. Motor fatigue can be divided into peripheral and central components as defined by Bigland-Ritchie & Woods (1984). Peripheral motor fatigue is caused by reduced neural drive at or beyond the neuromuscular junction, while central fatigue is caused by reduced neural drive proximal to the anterior horn cell. It is difficult to separate the central components of motor fatigue from peripheral ones because central fatigue is usually measured through parameters such as electromyography (EMG) and muscle force that produce their outputs through the peripheral neuromuscular system. Although peripheral motor fatigue can be quantified separately, most evidence for central motor fatigue is indirect. After a fatiguing muscle contraction most of the reduction in force results from peripheral processes caused principally by fatigue in the muscle itself (Bigland-Ritchie & Woods, 1984; Boska *et al.* 1990). Although central motor fatigue has a more minor role in the reduction of force after fatiguing exercise, studies using transcranial magnetic stimulation (TMS) have shown that significant changes occur in cortical excitability during and after exercise (see Gandevia *et al.* 1996).

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Brasil-Neto *et al.* (1993) first used TMS and EMG recordings to investigate changes that occur in the motor system after exercise. Resting motor-evoked potentials (MEPs) were recorded from isolated forearm muscles before subjects completed an exhausting forearm exercise protocol. The results of this study showed that post-exercise MEP responses to TMS had decreased in amplitude relative to pre-exercise responses (post-exercise MEP depression). A follow-up study by Brasil-Neto *et al.* (1994) and studies from other groups (McKay *et al.* 1995; Liepert *et al.* 1996; Samii *et al.* 1996*a*,*b*) showed that the post-exercise MEP depression is often preceded by an initial short-duration increase in the post-exercise MEP amplitudes relative to pre-exercise responses (post-exercise MEP facilitation).

Brasil-Neto *et al.* (1994) demonstrated that MEP amplitudes were reduced to less than half pre-exercise values after an exercise protocol. Using the same exercise protocol they showed that post-exercise H-reflexes, M-waves and MEPs to transcranial electrical stimulation (TES) were not significantly different to pre-exercise responses. The authors concluded that post-exercise MEP depression to TMS was predominantly due to central motor fatigue. They also postulated that post-exercise MEP facilitation and depression were a result of the balance between neurotransmitter mobilisation and depletion.

One of the aims of this study was to investigate the exercise-induced changes that occur in the erector spinae muscles (at the level of L3/L4) using two different rowing ergometer exercise protocols. The erector spinae muscles are involved heavily in rowing making them suitable for investigation in a more typical exercise regime such as rowing on an ergometer; previous studies have generally investigated single muscles following isometric contractions. The studies conducted by Brasil-Neto *et al.* (1993, 1994) involved studying the post-exercise changes in isolated muscles in the forearm. Samii *et al.* (1996*a*,*b*) and Liepert *et al.* (1996) studied the post-exercise changes in the arm and in the small muscles of the hand. McKay *et al.* (1995) were the first group to look at the effects of exercise on TMS-induced responses in the lower limb. Whether or not lower and upper limb muscles respond to exercise in the same manner is still under debate. However, both show evidence of post-exercise MEP facilitation and post-exercise MEP depression.

The primary objectives of this work were to use TMS to: (1) test the post-exercise changes in amplitude of MEPs recorded from erector spinae muscles and to determine whether these muscles respond to a fatiguing exercise protocol in the same way as limb muscles, (2) compare post-exercise changes after two different levels of exercise, and (3) ascertain whether TMS can reveal differences in the post-exercise excitability between a group of elite rowers and a group of non-rowers.

METHODS

Experimental subjects

Five elite rowers (mean ± S.E.M.): age 22.37 ± 0.32 years; height 187.8 ± 1.7 cm; weight 82.7 ± 1.8 kg) and six non-rowers (mean ± S.E.M.): age 22.18 ± 0.26 years; height 178.0 ± 1.7 cm; weight 74.2 ± 6.0 kg) were recruited for this investigation. Seven subjects were right-handed and four were left-handed. The elite rowers were members of the Imperial College Boat Club top squad and had at least five years experience at top national or international level rowing. The elite rowers completed a 17 h training programme each week, the non-rowers completed an average of 5.17 ± 2.56 h of exercise per week. Ethical approval for the study was obtained from the Riverside Research Ethics Committee and all subjects gave their informed written consent to take part, in accordance with the Declaration of Helsinki.

Electrophysiological recordings

Surface electromyographic (EMG) recordings were made from the left and right erector spinae (ES) muscles and from the dominant first dorsal interosseus (FDI) muscle using self-adhesive electrodes (Arbo Neonatal Blue). Two recording electrodes were placed over ES muscles 3 cm and 5 cm lateral to the mid-line on both sides of the body and between the spinal processes of L3 and L4. Recordings were made from the dominant FDI with one electrode positioned on the belly of the muscle and the other placed over the metacarpo-phalangeal joint of the index finger. The EMG signal was amplified (× 1000 in FDI or × 10 000 in ES muscles) and filtered (± 3dB) below 100 Hz and above 2000 Hz. The EMG signals were then sampled (sampling rate 4000 Hz) by computer (Cambridge Electronic Design 1401/IBM-compatible PC) and analysed using signal averaging software (Cambridge Electronic Design Signal software).

Transcranial magnetic stimulation (TMS)

TMS of the motor cortex was achieved using a Magstim 200 stimulator (Magstim Company, Dyfed, UK) connected to an angled double cone figure-of-eight coil (coil diameter 12.5 cm) producing a maximum stimulus strength of 1.4 T. The coil was hand-held by an operator and stabilised with its cross-over over the vertex. The induced current in the brain under the cross-over flowed in a posterior to anterior direction. Before commencing exercise and while subjects were relaxed, the threshold intensity of transcranial magnetic stimulation (TMS) to produce MEPs in each muscle was determined. Threshold was taken to be the lowest stimulus strength that evoked at least five MEPs in each muscle in response to ten stimulus presentations. The experimental trials were conducted using a stimulus intensity of 1.2 times this threshold value with the muscles at rest before and after the exercise protocol. Using a constant TMS intensity allowed any change in corticospinal excitability after exercise to be identified by a corresponding change in MEP amplitude.

Experimental protocol

Before exercise, a trial of ten stimuli was given so that a pre-exercise level of excitability could be measured.

All five elite rowers and four of the non-rowers underwent two separate rowing exercise protocols separated by at least 24 h; the remaining two non-rowers did not agree to take part in the intense exercise protocol. A rowing ergometer (Concept II, model B, Concept II Inc., Vermont, USA) was used for both exercise protocols, as it mimics the rowing technique used in the boat. The basic rowing stroke, as performed by the elite rowers, was

explained to each of the non-rowers to ensure similar muscle usage by both groups. Before each exercise session subjects were given 3 min in which to warm up, during which time the non-rowers were coached on a safe and effective technique.

The first exercise protocol required the subjects to maintain a weight-adjusted split time for 10 min according to Abingdon Rowing Club recommendations (Martin, 1994). For example, an individual weighing 85 kg would be required to achieve a split time of 2 min 4 s per 500 m distance and the ergometer would be adjusted accordingly. This was deemed to be light exercise and both elite rowers and non-rowers were able to talk comfortably during the 10 min exercise period. The second intense exercise protocol required the subjects to row as fast as they could for 1 min.

Immediately after completion of the exercise protocol the subject was asked to lie prone and relaxed on a hospital bed and a stop-watch was started. The electrodes were reattached to the amplifiers and an initial post-exercise trial recording responses to ten magnetic stimuli was started as quickly as possible. This was between 1.5 and 2 min after cessation of exercise in every subject. Four minutes after completing the exercise another trial of ten stimuli was completed. Further trials of ten stimuli were carried out at 2 min intervals until the final trial 16 min after completion of exercise. The electrodes on FDI did not record stable data during the intense exercise protocol due to the strong mechanical interaction between the hand and the ergometer handle caused by the extra physical stress of the intense exercise protocol.

Analysis of EMG recordings

The peak-to-peak amplitudes of each unrectified MEP in each muscle were measured off-line and mean amplitudes for each trial of ten stimuli were calculated. MEPs from each trial were averaged off-line and latency and duration of the averaged MEPs were measured. Amplitudes and latencies of MEPs were normalised to pre-exercise values. Although the elite rowers and non-rower data sets were small, ANOVAs were used to examine changes in normalised MEP amplitude and latency within each group after exercise. Student's paired *t* tests were used to compare the mean normalised data between the elite rowers and the non-rowers in the post-exercise period.

RESULTS

Motor-evoked potentials

In all 11 subjects it was possible to evoke responses in both left and right ES muscles and in the dominant FDI. Figure 1 shows typical averaged MEP responses from all three muscles in a non-rower before exercise and at intervals after completion of the light exercise protocol. Note that the post-exercise MEPs recorded 2 min after completing exercise are larger than pre-exercise records (post-exercise MEP facilitation), while MEPs recorded 6–16 min post-exercise are reduced in amplitude compared with the pre-exercise records (post-exercise MEP depression).

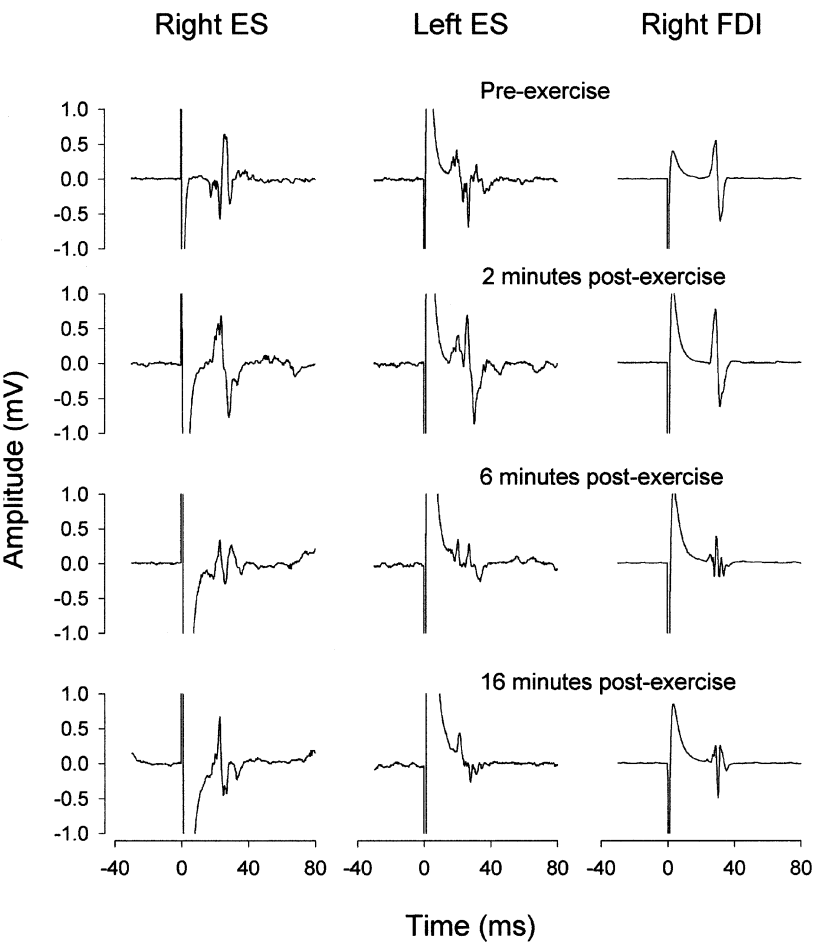

Figure 1

Averages of 10 MEP responses recorded from the left and right ES and the right FDI muscles in a non-rower. MEPs are shown pre-exercise and 2, 6 and 16 min post-exercise for the light exercise protocol. The magnetic stimulus was applied at an intensity of 72 % of the maximum stimulator output. Note that MEPs recorded 2 min post-exercise show facilitation, MEPs at 6 min show post-exercise depression, which has started to recover by 16 min post-exercise.

Amplitudes of MEPs

The bar charts in Fig. 2 show the mean MEP amplitudes relative to pre-exercise values in the left and right ES muscles in the elite rowers and the non-rowers.

In the non-rowers the mean amplitude of the MEPs showed an increase ($P < 0.05$) relative to pre-exercise level, immediately post-exercise (light exercise: left ES 121.4 ± 36 %, right ES 125.8 ± 47 %; intense exercise: left ES 195.7 ± 118 %, right ES 115.9 ± 33 %) followed by a reduction in amplitude in both exercise protocols. In the elite rowers there was a significant ($P < 0.05$) increase in MEP amplitudes immediately post-exercise only in the intense exercise protocol (left ES 128.6 ± 16 %; right ES 118.9 ± 7 %). In the period 4–16 min after light exercise, the mean MEP amplitudes in left and right ES, relative to pre-exercise levels, were reduced ($P < 0.05$) in both elite rowers (left ES 79.4 ± 2.1 %, right ES 80.9 ± 5 %) and in the non-rowers (left ES 60.9 ± 2.5 %, right ES 68.5 ± 6 %) in the left ES. In the left ES the amplitude reduction in the non-rowers was significantly greater than in the elite rowers ($P < 0.05$) but in the right ES, although the mean amplitudes of MEPs in the non-rowers were consistently lower than in the elite rowers, the difference was not significant ($P = 0.067$). In the intense exercise protocol the amplitudes were reduced in the period 4–16 min after exercise in both the elite rowers (left ES 89.6 ± 3 %; right ES 74.3 ± 3 %) and the non-rowers (left ES 88.0 ± 13 %; right ES 76.8 ± 7 %). There was no difference between degree of reduction of the amplitudes of MEPs in the two groups although the amplitudes were more reduced in left ES in both groups.

The bar charts in Fig. 3*A* show the mean MEP amplitudes relative to pre-exercise values in the dominant FDI muscle in elite rowers and non-rowers following the light exercise protocol. In both groups there was slight facilitation throughout the post-exercise test period with mean MEP amplitudes significantly larger ($P < 0.05$) in the elite rowers (119.0 ± 3.1 %) than in the non-rowers (101.2 ± 5.8 %) relative to pre-exercise levels. There was no evidence that this facilitation was greater immediately following exercise when post-exercise facilitation might have been expected.

Latency of MEPs

The bar charts in Fig. 4 show the mean MEP latency relative to pre-exercise values for the left and right ES muscles plotted before and after exercise; latencies for dominant FDI are shown in Fig. 3*B*. There was no difference ($P > 0.05$) in mean pre-exercise latencies between the elite rowers

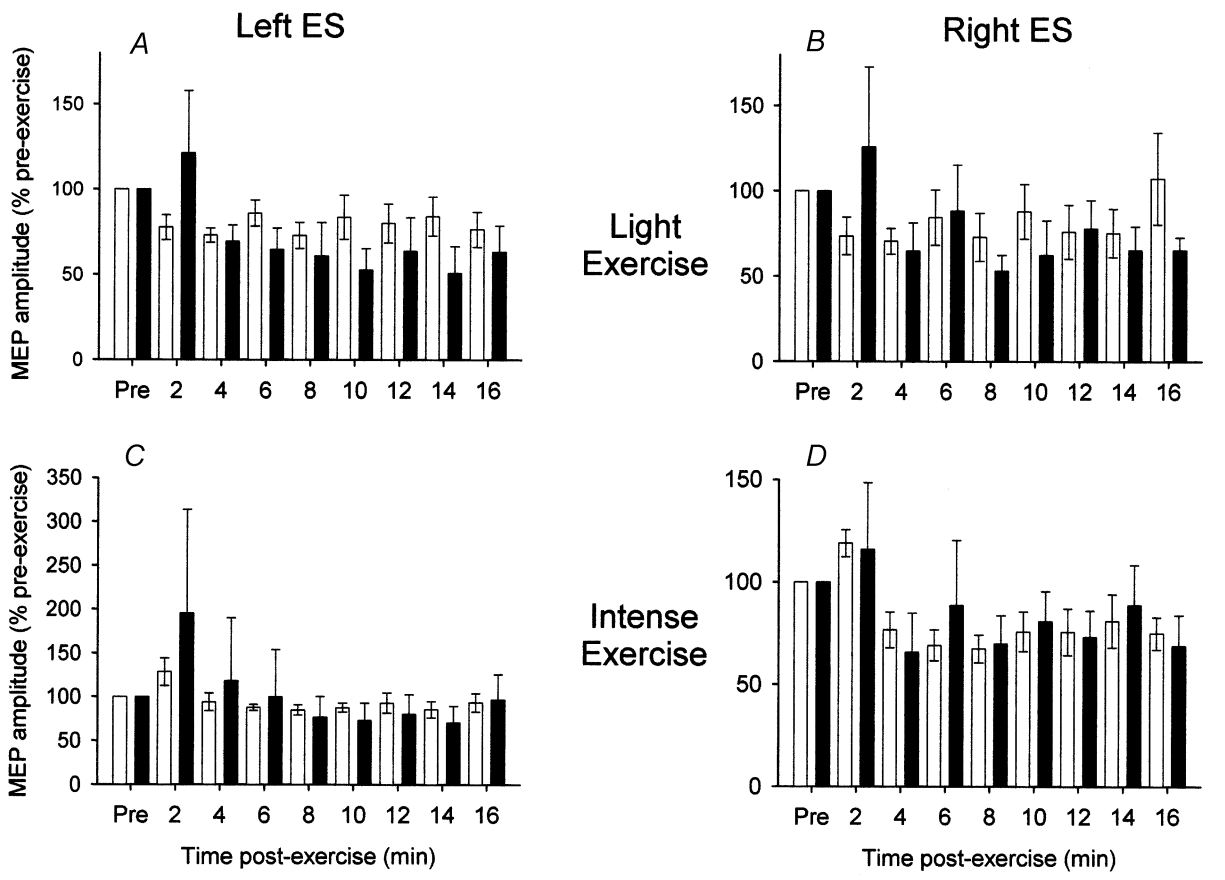

Figure 2

Mean peak-to-peak amplitudes of MEPs recorded from ES in the elite rowers (□) and the non-rowers (■) at different times post-exercise relative to pre-exercise amplitudes. *A* and *B*, light exercise protocol; *C* and *D*, intense exercise protocol; *A* and *C*, left ES; *B* and *D*, right ES. Pre-exercise amplitudes are normalised to 100 %. Error bars indicate 1 S.E.M.

(left ES 15.4 ± 0.9 ms, right ES 14.8 ± 1.2 ms, dominant FDI 23.4 ± 0.6 ms) and the non-rowers (left ES 17.3 ± 1.3 ms, right ES 16.5 ± 0.9 ms, dominant FDI 23.0 ± 0.7 ms).

However, in the light exercise protocol (Figs 4*A*, *B* and 3*B*) it is apparent that the latency is longer, relative to pre-exercise values, in the elite rowers (left ES 104.5 ± 3 %, right ES 108.5 ± 3 %, dominant FDI 100 ± 1 %) compared with the non-rowers (left ES 86.7 ± 2 %, right ES 92.2 ± 2 %, dominant FDI 93.4 ± 1 %) throughout the 4–16 min post-exercise testing period. During the 4–16 min post-exercise period, MEPs in ES on both sides of the elite rowers showed an increased mean latency while the non-rowers showed a decrease in the latency. Student's paired *t* test for all post-exercise values showed that the elite rowers had MEP latencies in left and right ES that were significantly ($P < 0.05$) longer than in the non-rowers. Although the effect in FDI is less pronounced, the elite rowers had mean MEP latencies that were significantly ($P < 0.05$) longer than those in the non-rowers throughout the post-exercise period.

In the intense exercise protocol (Fig. 4*C* and *D*) any latency differences between the elite rowers (left ES 99.2 ± 1 %, right ES 105.3 ± 2 %) and the non-rowers (left ES 100.5 ± 3 %, right ES 106.6 ± 2 %) were much more variable between subjects and were not significant ($P > 0.05$).

DISCUSSION

Post-exercise MEP facilitation

The post-exercise changes seen in erector spinae muscles of individuals in this study are similar to those seen in limb muscles in previous studies (see Brasil-Neto *et al.* 1993, 1994; Bonato *et al.* 1994; McKay *et al.* 1995; Samii *et al.* 1996*b*; Wassermann *et al.* 1996). We acknowledge that the number of subjects is small in this study and that the variance in the results may, in some cases, be rather large as a consequence. However, the study follows subjects serially after exercise, producing nine data points for each variable in each subject; this makes trends more easily identifiable. Most previous studies of central fatigue looked at exercise protocols involving continuous isometric contractions, often in only one muscle. The results of this study are encouraging since they suggest that studies of central fatigue can be extended into more dynamic exercise protocols of the sort used by athletes during training or performing their particular sport.

We saw evidence of post-exercise facilitation in left and right ES at both levels of exercise in the non-rowers and after intense exercise in the elite rowers. It was Brasil-Neto *et al.* (1994) who first described 'post-exercise facilitation' and the duration and decay time of this facilitation was investigated by Samii *et al.* (1996*b*). The extent and duration of post-exercise facilitation has been shown to vary according to duration and intensity of exercise (Liepert *et al.* 1996; Samii *et al.* 1996*b*). The fact that the elite rowers only showed post-exercise facilitation after intense exercise might indicate a training effect on the corticospinal control of these muscles. Brasil-Neto *et al.* (1994) concluded that the increased amplitude of post-exercise MEPs was the result of increased excitability in the motor cortex. This conclusion was supported by the fact that MEPs elicited by near-threshold transcranial electrical stimulation (TES) of the motor cortex or electrical stimulation at the cervico-medullary junction, which produce direct activation of the corticospinal tract, did not show an increased MEP amplitude post-exercise (Samii *et al.* 1996*b*; Wasserman *et al.* 1996; Taylor *et al.* 1997). Colebatch *et al.* (1990) presented evidence that more proximal muscles can exhibit long-latency MEP responses at a latency of around 30–40 ms longer than the conventional short-latency responses. In this study we had the opportunity to compare responses in distal FDI muscles with those in the proximal, axial ES muscles. We observed long-latency responses in the ES muscles in only one subject (latency 83 ms) and never in FDI muscles. No change was seen in the amplitude of this response after exercise supporting the notion that its origin might be non-cortical.

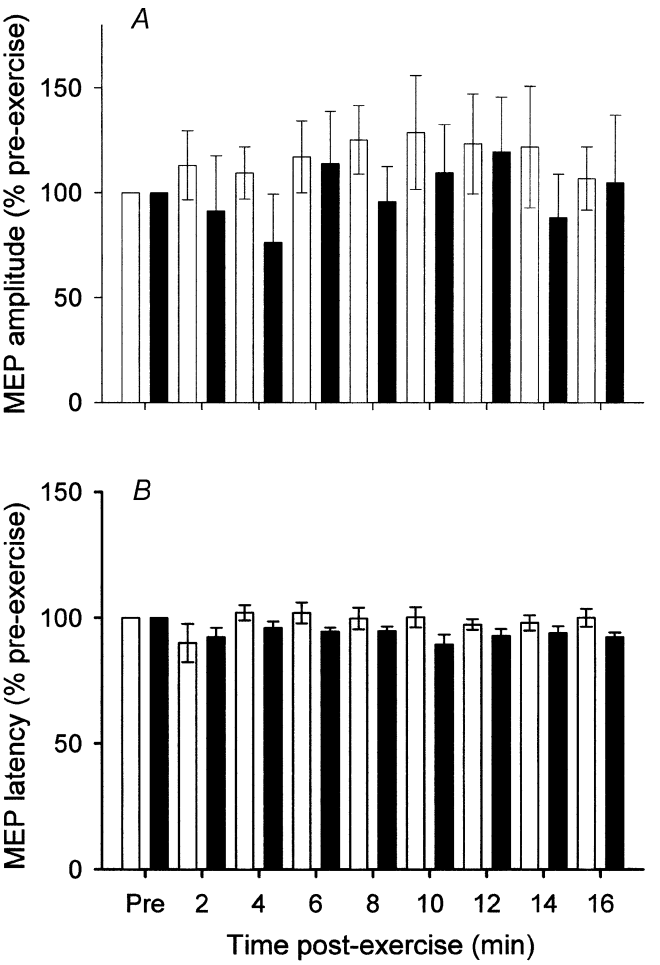

Figure 3

Mean peak-to-peak amplitudes (*A*) and latencies (*B*) of MEPs recorded from dominant FDI in the elite rowers (□) and the non-rowers (■) at different times after light exercise relative to pre-exercise amplitudes. Pre-exercise amplitudes are normalised to 100 %. Error bars indicate 1 S.E.M.

Rowing is an aerobic exercise that requires muscles throughout the legs, trunk and arms. The propulsive phase of the rowing stroke, described by McArthur (1997), relies predominantly on the leg muscles and the trunk muscles at initiation and the shoulder and arm muscles at completion. The upper leg and lower trunk muscles take the greatest load during this propulsive 'work' phase of the rowing stroke. Although respiration rate was not measured in this study, the subjects were breathing more heavily post-exercise. The erector spinae muscles are used as accessory muscles in respiratory movement. Nowicky *et al.* (2001) have shown that respiratory movement affects MEP amplitudes and that erector spinae muscles are active during maximal respiration. It is possible that increased respiratory movement from heavy breathing post-exercise may have affected MEP responses and caused more facilitation in the erector spinae muscles than might otherwise have been seen.

Post-exercise facilitation was not seen to the same degree in FDI muscles. In rowing the hand is used as a hook to hold the oar (or ergometer handle). Muscles of the hand (including the first dorsal interosseous) are not required to generate power, unlike the back muscles. Despite this, subjects reported that their hand muscles were sometimes co-contracted and might result in the post-exercise facilitation in non-exercising muscles that was occasionally seen in this study (see, for example, Fig. 1).

Post-exercise MEP depression

In the back muscles all subjects showed evidence of post-exercise fatigue (MEP depression) in both exercise protocols and for most subjects this lasted for the period of post-exercise testing (at least 16 min). As we saw with post-exercise facilitation, there was less evidence of post-exercise depression in the hand responses than in the back responses. Brasil-Neto *et al.* (1993) first described this post-exercise depression of MEP amplitude and called it an 'exercise-induced inhibitory phenomenon'. They showed that post-exercise MEPs were reduced to less than half pre-exercise values after an exercise protocol. Using the same exercise protocol they showed that post-exercise H-reflexes, M-waves and MEPs to TES were not significantly different from pre-exercise responses. The authors concluded that post-exercise MEP depression to TMS was predominantly due to central motor fatigue. In the present study, it is likely that the MEP depression seen post-exercise was mainly due to central motor fatigue. It was not possible to stimulate peripheral nerves to the ES muscles with present

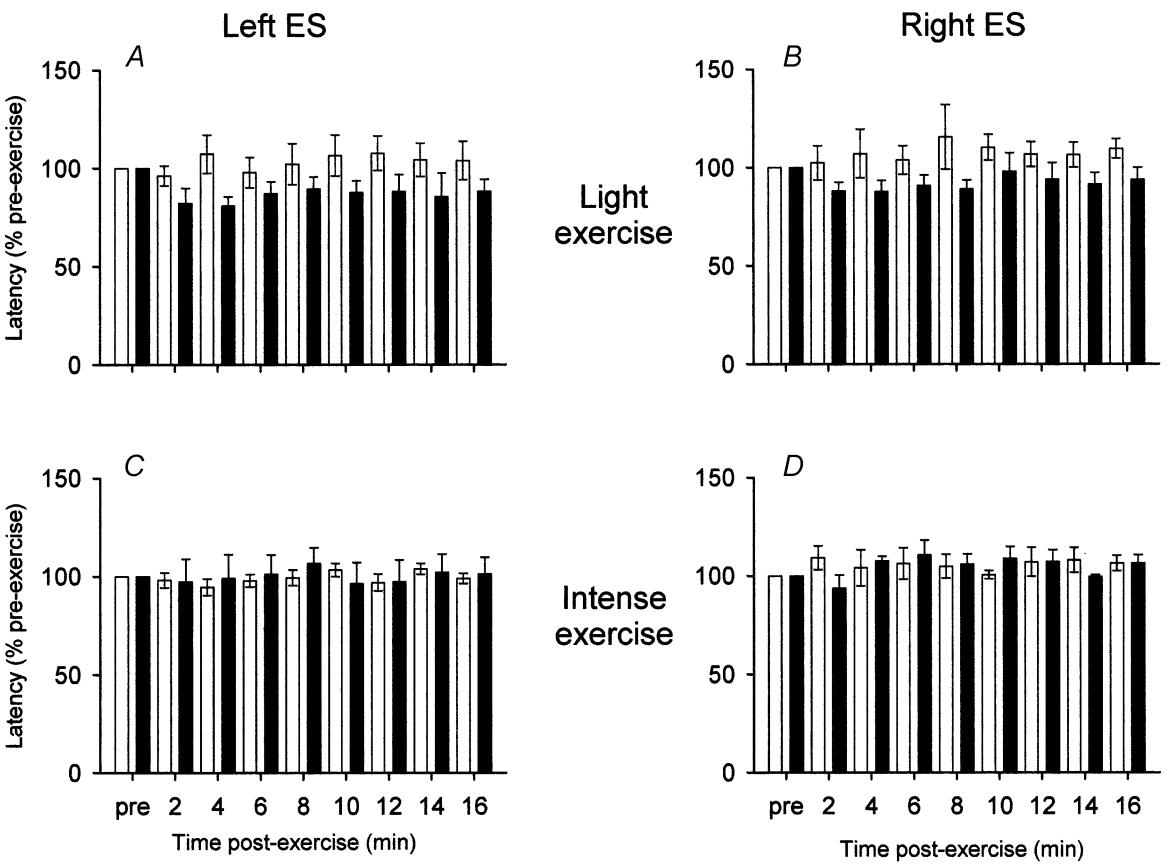

Figure 4

Mean latencies of MEPs recorded from left and right ES in the elite rowers (□) and the non-rowers (■) at different times post-exercise relative to pre-exercise amplitudes. *A* and *B*, light exercise protocol; *C* and *D*, intense exercise protocol; *A* and *C*, left ES; *B* and *D*, right ES. Pre-exercise latencies are normalised to 100 %. Error bars indicate 1 S.E.M.

investigative techniques as they lie too deeply below the skin to access non-invasively. Bonato *et al.* (1994) assessed corticospinal excitability to thenar muscles after 1 min of maximum voluntary contraction. They proposed a triphasic pattern for the time course of post-exercise MEP depression consisting of (1) a rapid decreasing phase up to the fifth minute of the recovery period, (2) a maximal depression for about 10 min, and (3) a slow recovery to baseline by about 35 min after the end of exercise. McKay *et al.* (1995) found that MEPs in leg muscles were depressed for about 20 min after exercise. In the present study, our post-exercise testing was terminated after 16 min but MEPs in ES muscles of most individuals had not recovered to pre-exercise values by the end of the final test. Although with hindsight we can postulate that a longer period of post-exercise testing might have allowed recovery to pre-exercise values, our data are not inconsistent with the results reported in arm and leg muscles. The post-exercise changes seen in the FDI responses, particularly in the non-rowers, suggest that exercise may lead to a general depression of motor cortical excitability, which could help recovery from the effects of localised areas of fatigue.

The degree of post-exercise depression was greater in the left ES and it is tempting to relate this to the asymmetrical use of back muscles in rowing on one side of the boat. Parkin *et al.* (2001) report an asymmetry of back muscle activity in rowers that correlates with the side of the boat on which they row; despite this, they reported no difference in muscle bulk between the two sides. In any case, the post-exercise depression in the present study was greater in the left ES in both elite rowers and non-rowers; suggesting that the reason is not related to rowing.

Rowers *versus* non-rowers

Our results showed significant differences between the elite rowers and non-rowers, with the elite rowers showing less post-exercise depression after the light exercise protocol. This difference was not seen after intense exercise.

Light exercise protocol. During the light exercise protocol the elite rowers were working at a low output relative to their maximum compared with the non-rowers. On average the elite rowers trained for about three times as long per week than the non-rowers. Furthermore, the non-rowers had not trained specifically for rowing. The apparent lesser level of MEP depression in the elite rowers could be simply a reflection of the fact that they were working proportionately further from their maximum capacity than the non-rowers, and presumably were recruiting a smaller percentage of corticospinal neurones projecting to the exercising muscles. In other words, the degree of central fatigue may be related to the level of exercise as a proportion of an individual's maximum exercise capacity. Clearly the maximum work output for the elite rowers has been increased by training; any given level of exercise must be further from their maximum capacity than an equivalent non-rower, this may in turn generate a lower degree of central fatigue than in a non-rower.

Samii *et al.* (1996*a*) conducted a study comparing the effects of exercise on the TMS-induced MEPs of normal subjects and patients with chronic fatigue. The post-exercise depression was more significant in the recovery period of the chronic fatigue patients than in the normal subjects. It is possible that there is a continuum whereby sports-trained individuals show the least amount of MEP depression in exercised muscles after a sub-maximal exercise protocol, normal subjects show slightly more MEP depression and chronic fatigue patients show the most evidence of MEP depression.

In the FDI after the light exercise protocol, the mean MEP amplitudes also showed significant differences between the groups. The elite rowers showed predominantly facilitated MEPs throughout the post-exercise testing period whereas the non-rowers showed little change from pre-exercise levels. FDI is not a muscle that is used to generate power in the rowing stroke. Individuals use muscles of the hand differently during the stroke depending on the way in which they hold the ergometer handle. Finally, the generation of central fatigue might have a more general component reflecting systemic metabolite levels. Clearly the non-rowers were generally more exhausted and the resulting change in systemic metabolite levels might influence overall corticospinal excitability.

Intense exercise protocol. In the intense exercise protocol there were no statistical differences in post-exercise depression between the groups. Despite the elite rowers producing more power than the non-rowers during this intense exercise protocol, both groups were working to their own voluntary maximum. In addition, the intense exercise protocol would, presumably, rely more on aerobic metabolism than the light exercise protocol. Aerobic and anaerobic metabolic changes might produce different cortical influences that could explain the lack of difference seen between the elite rowers and the non-rowers in this protocol. Another possibility is that, under these conditions of working to the exercise limit, it is possible that central fatigue processes are activated similarly.

Latency of MEPs. After light exercise MEP latencies became longer in the elite rowers (left ES 16.1 ± 0.5 ms; right ES 16.1 ± 0.4 ms; dominant FDI 23.4 ± 0.2 ms) compared with pre-exercise values (left ES 15.4 ± 0.9 ms; right ES 14.8 ± 1.2 ms; dominant FDI 23.4 ± 0.6 ms). However, in the non-rowers the MEP latencies after light exercise became shorter (left ES 15.0 ± 0.3 ms; right ES 15.2 ± 0.3 ms; dominant FDI 21.5 ± 0.2 ms) than pre-exercise values (left ES 17.3 ± 1.3 ms; right ES 16.5 ± 0.9 ms; dominant FDI 23.0 ± 0.7 ms). There was no difference in the pre-exercise latencies between the two groups. The altered patterns of latency seen after exercise in the two groups could be due to a different recruitment pattern of motor unit types or corticospinal neurones in the two groups after exercise. Thayer *et al.* (2000) have shown that long-term aerobic training decreases the proportion of fast fatiguable motor units in favour of non-fatiguable motor units that have slower transmission speeds. The benefits of

non-fatiguable motor units are that they increase aerobic capacity. The fatiguable motor units are used to produce short-lasting powerful muscle contractions and tend to be used in power movements like sprinting or weight-lifting. It is advantageous for an elite rower, in whom endurance is important, to have a greater proportion of non-fatiguable motor units.

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