Fatigue and recovery of voluntary and electrically elicited dynamic force in humans

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1. Percutaneous electrical stimulation of the human quadriceps muscle has been used to assess the loss of central activation immediately after a bout of fatiguing exercise and during the recovery period.

2. Fatigue was induced in eight healthy males by a maximal effort lasting 25 s performed on an isokinetic cycle ergometer at a constant pedal frequency of 60 revolutions per minute. The cranks of the ergometer were driven by an electric motor. Before and after the sprint, subjects allowed their legs to be passively taken round by the motor. During the passive movement the knee extensors were stimulated (4 pulses; 100 Hz). Peak voluntary force (PVF) during the sprint and peak stimulated forces (PSF) before and in recovery were recorded via strain gauges in the pedals. Recovery of voluntary force was assessed in a series of separate experiments in which subjects performed a second maximal effort after recovery periods of different durations.

3. Peak stimulated forces were reduced to 69·8 ± 9·3% immediately after the maximal effort, ($P < 0·05$), but had returned to pre-exercise values after 3 min. The maximum rate of force development (MRFD) was also reduced following fatigue to 68·8 ± 11·0% ($P < 0·05$) of control and was fully recovered after 2 min. PVF was reduced to 72·0 ± 9·4% ($P < 0·05$) of the control value following the maximal effort. After 3 min voluntary force had fully recovered.

4. The effect of changing the duration of the fatiguing exercise (10, 25 and 45 s maximal effort) resulted in an increased degree of voluntary force loss as the duration of the maximal effort increased. This was associated with an increased reduction in PSF measured immediately after the exercise.

5. The close association between the changes in stimulated force and voluntary force suggests that the fatigue in this type of dynamic exercise may be due to changes in the muscle itself and not to failure of central drive.

During voluntary isometric contractions, most human subjects are able to activate maximally their limb muscles (Merton, 1954; Belanger & McComas, 1981; for review see Bigland-Ritchie, 1981), despite some reports to the contrary (Ikai, Yabe & Ishii, 1967; Bigland-Ritchie, Kukulka, Lippold & Woods, 1982). Moreover, the decline in force during prolonged isometric contractions, (i.e. fatigue), can usually not be overcome by supramaximal stimulation of the motor nerve or muscle itself (Merton, 1954; Bigland-Ritchie, 1981; Bigland-Ritchie *et al.* 1982), although in recent studies some central fatigue has been documented even in well-motivated subjects (Lloyd, Gandevia & Hales, 1991; McKenzie & Gandevia, 1991). Although failure of transmission of action potential can be demonstrated in electrically stimulated contractions (e.g. Krnjevic & Miledi, 1959; Bigland-Ritchie, Jones & Woods, 1979), it is unlikely that such failure plays a role in fatigue of voluntary contractions (Bigland-Ritchie, 1981). Thus, fatigue in voluntary isometric contractions is most probably due to changes in the contractile apparatus with reduced muscle activation by the CNS being a minor factor.

In everyday life, however, many activities involve dynamic contractions in which speed as well as force are important and the extent to which subjects can activate their muscles during shortening contractions has received relatively little attention. To our knowledge there are only two studies in which the extent of voluntary activation of limb muscles during fatiguing dynamic contractions was investigated using direct electrical stimulation (Newham, McCarthy & Turner, 1991; James, Sacco & Jones, 1995). James *et al.* (1995) showed that only in one of two subjects

was there a small reduction in central drive during fatiguing dynamic contractions of the knee extensors. Newham *et al.* (1991) found evidence of activation failure in the fatigued state during slow (20 deg s^{-1}) but not fast (150 deg s^{-1}) isokinetic contractions. In both these studies a single-joint movement at relatively low angular velocities ($\leqslant$ 150 deg s^{-1}) was used. Clearly, in complex tasks such as cycling, which involve changes in velocity and the co-ordinated activity of many muscle groups, there are more possibilities for changes in central activation to affect overall performance.

In the present study we have examined this possibility during a complex fatiguing multi-joint exercise (cycling) using percutaneous electrical stimulation of the quadriceps muscle group.

Initially we attempted to apply the twitch interpolation technique (superimposing electrical stimulation on a voluntary contraction) to maximal voluntary effort during cycling. However, during the cycling movement we studied, we found that single twitches and even short trains of high frequency stimulation could not be detected reliably and no additional force from electrical stimulation could be detected above about 50% of a maximal voluntary effort.

We therefore adopted an approach in which the electrically evoked force was measured whilst the quadriceps was relaxed but being taken passively through the full range of movements by the motor of the cycle ergometer. Changes in the electrically evoked force were compared with the force of voluntary efforts before and after bouts of maximal exercise and during the recovery phase.

If the changes in electrically stimulated and voluntary force were similar, this would strongly suggest that they were both reflecting impairment of the muscles' capability to generate force, rather than a failure of central nervous system drive.

METHODS

Subjects

Experiments were performed on eight healthy male subjects (mean age 27·1 $\pm$ 2·6). The nature of the study and the techniques to be used were fully explained to the subjects before their voluntary consent was obtained. The study was approved by the local ethics committee.

Force recording

To measure leg forces generated at a constant pedal frequency an isokinetic cycle ergometer was used (Beelen, Sargeant & Wijkhuizen, 1994). In this ergometer system the cranks are driven by a 2·2 kW electric motor at a constant preselected pedal frequency through a variable gearbox. Due to the characteristics of the motor–gear system, pedal frequency remained constant despite variable forces generated on the pedals.

The subjects were seated on the ergometer with their feet strapped to the pedals by means of toeclips (that is, with the ball of the foot over the pedal spindle). Seat height was adjusted such that a slight knee flexion occurred at bottom dead centre of each revolution.

Two restraining straps were placed around the waist, one which was anchored to the bicycle frame in front of the saddle, the other to the frame behind the saddle. This ensured that the subject maintained a standard seating position even during maximal exercise.

Forces exerted on the pedals were measured by means of strain gauges mounted in the pedals. Vertical and horizontal forces acting at the pedal surface were measured at a sample frequency of 150 Hz. Pedal frequency was set at 60 r.p.m.

Electrical stimulation

The quadriceps muscles of the right leg were stimulated percutaneously through two 5 × 20 cm aluminum foil electrodes applied on dampened absorbent paper towel and bandaged proximally and distally to the antero-lateral thigh. A train of four stimuli at 100 Hz was used consisting of square-wave pulses of 50 μs duration and 200 V. Stimulation of the resting quadriceps in an isometric situation generated a force that was 36 $\pm$ 9% of the isometric maximum voluntary contraction (MVC) at the same knee angle (60 deg knee flexion).

Experimental protocols

Three series of experiments were performed. The protocols for these are given below.

Series 1: fatigue and recovery of voluntary force. Fatigue was induced in eight subjects by a maximal effort lasting 25 s. Subjects were instructed to make a maximal effort for 25 s in an attempt to increase pedal frequency, which was not possible due to the characteristics of the isokinetic ergometer. During the 25 s maximal effort the forces generated on the pedals were continuously monitored. Maximum voluntary force was calculated at the beginning and at the end of this fatiguing exercise as described in the following section on data analysis.

Recovery of voluntary force was assessed in five subjects in a series of four separate experiments. In each experiment subjects performed, after a standard warm-up, a maximal effort of 25 s (at $t = 0$) followed by a second maximal effort (25 s) after a recovery period of either 0·5 (at $t = 1$), 2·5 ($t = 3$), 4·5 ($t = 5$) or 11·5 min ($t = 12$ min). During the maximal efforts forces generated on the pedals were continuously recorded. Experiments were performed at least one day apart and in random order. A schematic representation of the protocol used in Series 1 is given in Fig. 1*A*.

Series 2: fatigue and recovery of electrically stimulated force. Fatigue was generated during a standard 25 s maximal effort as described for Series 1. Before the 25 s maximal effort and throughout the 20 min recovery period, subjects allowed their legs to be passively taken round by the motor. During this passive movement the quadriceps muscles were stimulated in selected revolutions in order to generate peak stimulated force at 90 deg past top dead centre. The timing of the stimulation was chosen such that the peak stimulated force occurred at the same knee angle (and presumably the same muscle length) as the maximal voluntary force during cycling exercise. Forces generated by electrical stimulation were measured before and at

intervals during and after the exercise period as well as during the 20 min recovery phase. Each measurement consisted of a recording of pedal forces during sixteen revolutions and on every fourth revolution the (relaxed) quadriceps were electrically stimulated.

In six subjects blood samples were taken from the fingertip before, and at intervals after, the 25 s maximal effort (at $t = 3{\cdot}5$, 8·5, 13·5 and 21 min). Blood samples were analysed for whole blood lactate using a lactate analyser (model 23L; Yellow Springs Instruments, Yellow Springs, OH, USA).

Electromyogram records were obtained from five subjects during passive cycling, before and during the recovery from 25 s of maximal voluntary exercise. Activity in the vastus lateralis, vastus medialis and rectus femoris was found to be very low at all times during the passive movements.

A schematic representation of the protocol used in Series 2 is given in Fig. 1*B*.

Series 3: effect of different exercise duration. To investigate the effect of the duration of exercise on fatigue and recovery of the electrically stimulated force, four subjects performed two additional experiments using the protocol described in Series 2 except that the duration of the maximal effort lasted 10 or 45 s. Experiments were performed at least one day apart and in random order.

Data analysis

From the horizontal (F_h) and vertical forces (F_v), resultant forces (F_r) were calculated:

$$F_r = \{\sqrt{[(F_h)^2 + (F_v)^2]}\}.$$

From the force recording during the maximal effort a peak voluntary force (PVF) was calculated for each revolution as the highest force seen in each revolution and corrected for limb weight in the same way as the electrically stimulated force. Maximum PVF was calculated as the highest mean of two consecutive revolutions within the first five revolutions. Peak voluntary force at the end of the 25 s (or 10, 45 s; Series 3) maximal effort was calculated as a mean of the PVF of the last two revolutions, expressed as a percentage of the maximum PVF.

The highest resultant force generated by electrical stimulation (PSF) was corrected for limb weight by subtracting from it the force on the pedal at the same moment in the pedal cycle during passive cycling. From each force measurement four values of peak stimulated force were averaged.

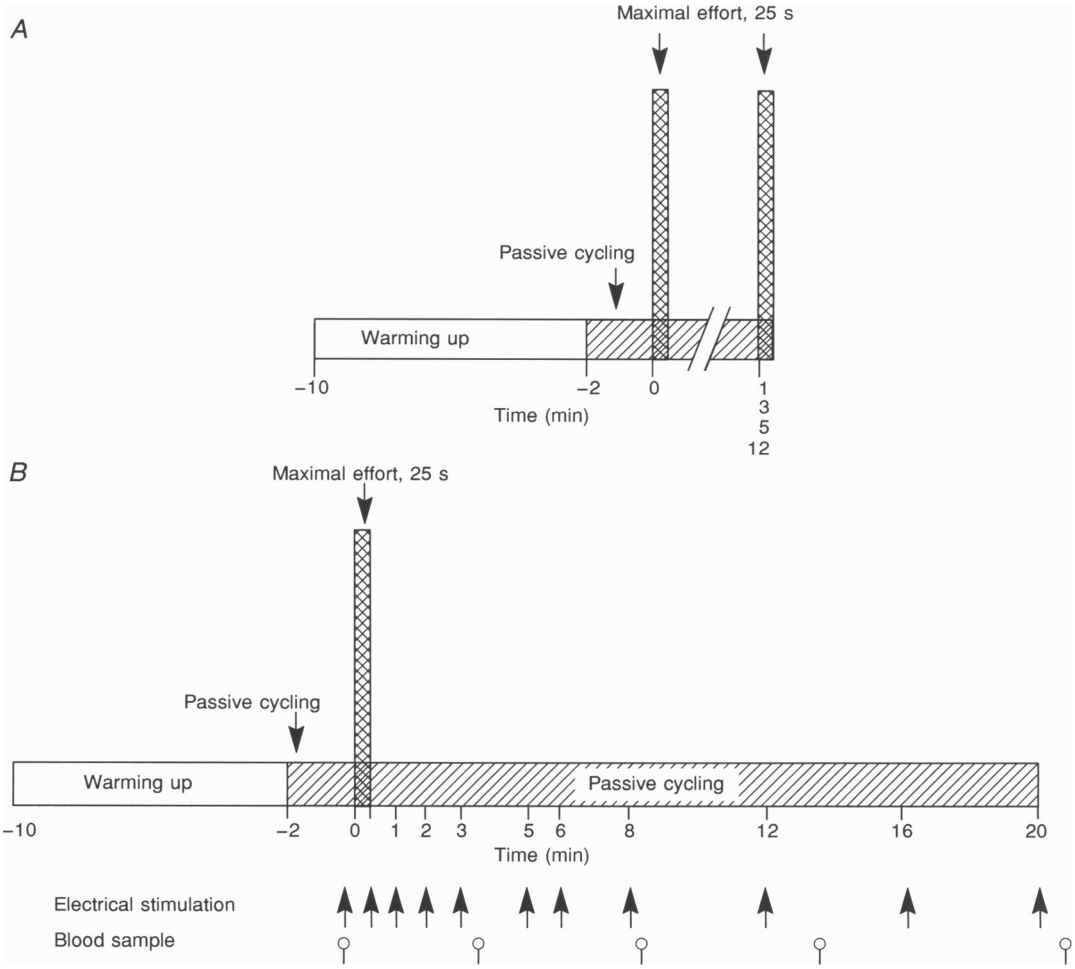

Figure 1
Protocols for Series 1 (*A*) and Series 2 (*B*).

The maximum rate of force development (MRFD) was calculated as the maximum dF/dt (Fig. 2) for each force trace and the four values from each test were averaged.

Force measurements made before the maximal effort (Series 2 and 3) were taken as control (100%). Force measurements made after the maximal effort were expressed as a percentage of the control value.

Statistics

The data are presented as means ± 1 S.D. Significance was determined by Student's t test for paired data and set at $P < 0.05$ (Series 2).

RESULTS

Series 1. Fatigue and recovery of voluntary force

In the five subjects studied, peak voluntary force (PVF) during the first 25 s maximal effort was reduced to 76·9 ± 5·7% of the initial value (maximal PVF). Recovery of PVF was very rapid (Fig. 3). In the second maximal effort performed at $t = 1$ min, that is after a recovery of 35 s, 92·2 ± 8·1% of the initial maximum PVF was reached. In one subject voluntary force had already recovered completely. At $t = 3$ min, peak voluntary force had fully recovered in all subjects.

Series 2. Fatigue and recovery of electrically stimulated force

The effect of fatigue induced by 25 s maximal voluntary effort and the recovery of the forces generated by electrical stimulation is shown in Fig. 4*A*. Mean data (± S.D.) for eight subjects are given. The mean value of the peak force generated by electrical stimulation of the relaxed muscle before the maximal effort was 204 ± 31 N. Peak stimulated force (PSF) immediately after the 25 s maximal effort reduced significantly to 69·8 ± 9·3% of the pre-exercise value. The reduction was not significantly different from that of the peak voluntary force (PVF) at the end of the 25 s maximal effort: 72·0 ± 9·4% of the pre-exercise maximal PVF. At $t = 3$ min (that is a recovery of 2·5 min after the exercise) PSF had returned to pre-exercise values (99·1 ± 5·3%). At $t = 5$ min, significantly higher values of PSF were reached (109·7 ± 5·7%) which remained significantly higher up to the measurement at 8 min inclusive. Thereafter PSF

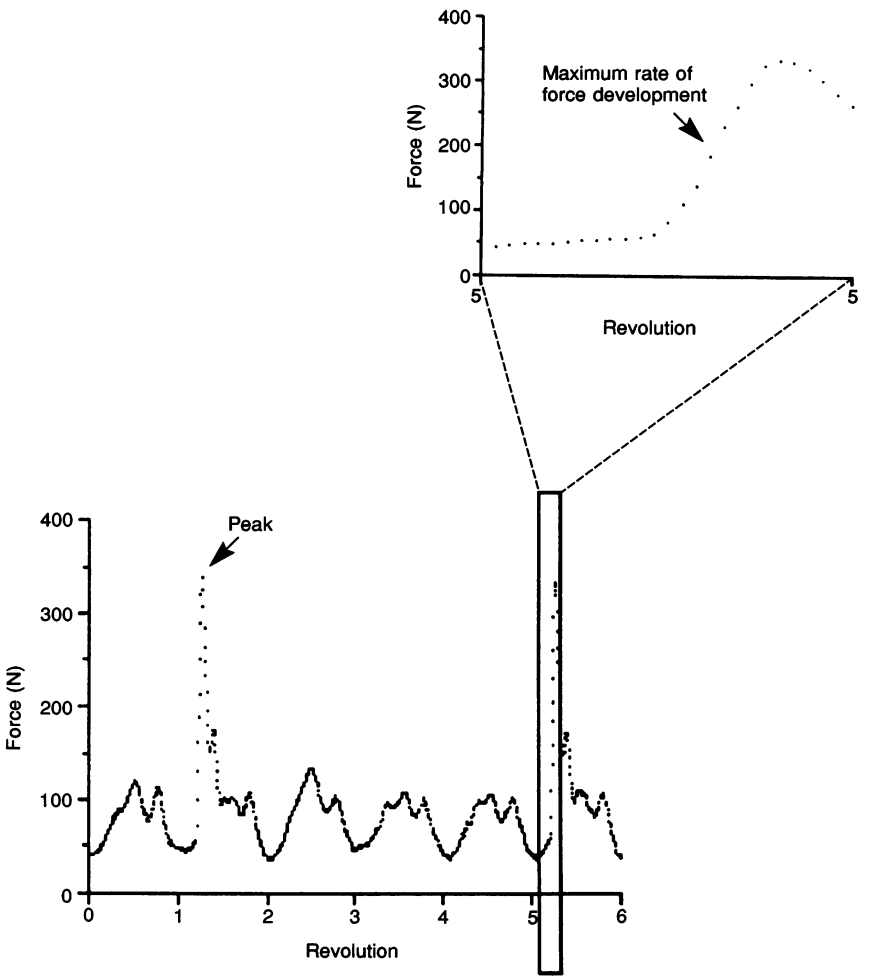

Figure 2. Example of a force recording made during six revolutions

Peak stimulated force and maximum rate of force development were calculated.

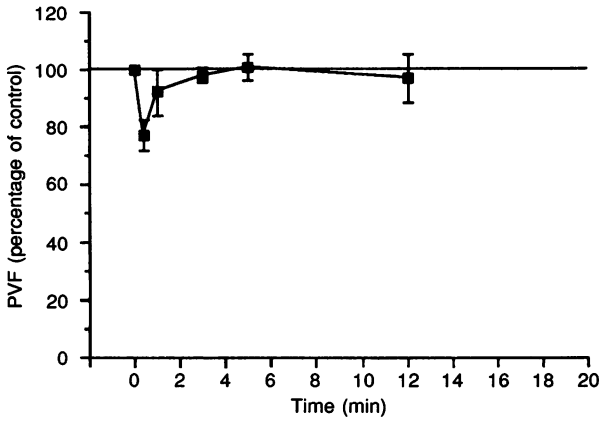

Figure 3. Peak voluntary force
PVF expressed as the percentage of control before and at intervals during recovery after a 25 s maximal effort (mean ± S.D., $n = 5$).

returned to pre-exercise values. This pattern differs from that of the PVF where no increase above the initial maximal PVF was found.

The maximum rate of force development (MRFD) was also significantly reduced following the 25 s maximal effort to 68·8 ± 11·0% and recovered with a similar time course as PSF (Fig. 4*B*). The potentiation (at $t = 5$–8 min) seen in the MRFD was, however, more marked than the potentiation in PSF. Up to the measurement at $t = 3$ min PSF was highly correlated with MRFD. The linear relationship is given by: PSF = −0·98 + 1·04 MRFD (coefficient correlation, $r = 0·94$, $P < 0·05$).

The mean concentrations of blood lactate before and at intervals during recovery are illustrated in Fig. 5. The first measurement made after the 25 s maximal effort (at $t = 3·5$ min) resulted in a mean value of 7·6 ± 0·4 mM. Even after 20 min recovery, blood lactate concentration was still elevated.

Series 3. Effect of different exercise duration

The effect of different exercise duration (10, 25 and 45 s) on fatigue and recovery of PSF is illustrated by Fig. 6, which shows data for one subject. A 10 s maximal effort hardly affected PSF measured immediately after the exercise. Also, PVF at the end of the maximal effort hardly differed from the initial value (97·9% of maximum PVF). At $t = 5$ min, a potentiation was observed in PSF of 4%. At $t = 12$ min, PSF returned to pre-exercise values. After a maximal effort of 25 s duration in which PVF reduced to 89·5% of the maximum PVF, a marked reduction in PSF was observed (to 61·5% of the pre-exercise value) which had recovered completely at $t = 3$ min. Again, a marked potentiation of ~10% was seen at $t = 5$ min which remained for the duration of the test. The maximal effort of 45 s duration, which reduced PVF to 67·8%, resulted in a large reduction in PSF to 42·3% of the pre-exercise value. The time course of recovery and potentiation was similar to that seen after a 25 s maximal effort.

Table 1 summarizes the changes in voluntary force during a 10, 25 and 45 s maximal effort together with the changes in PSF after the three maximal efforts for the four subjects studied. As the duration of the maximal voluntary effort increased so the PSF decreased immediately after the exercise. The greater reduction in

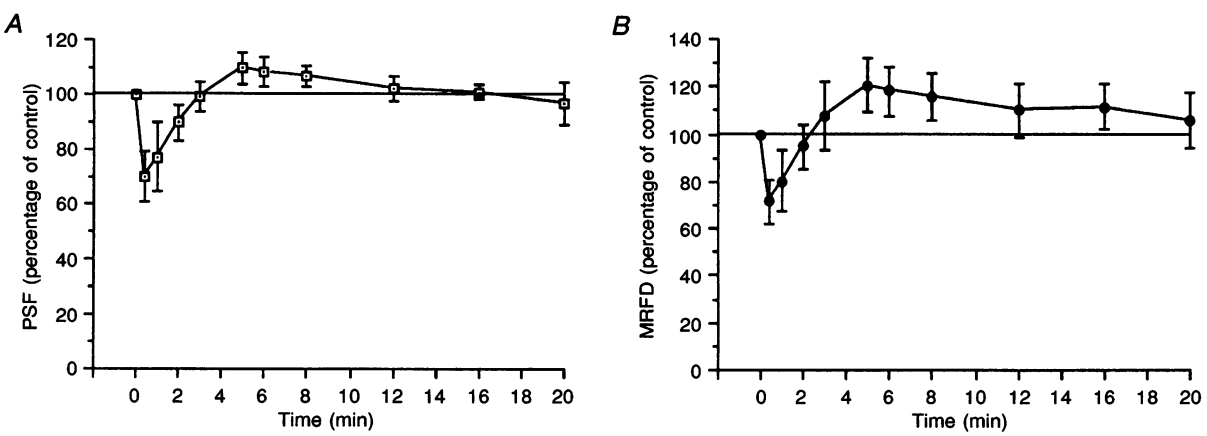

Figure 4. Peak stimulated force and maximum rate of force development
PSF, (*A*) and MRFD, (*B*) expressed as percentage of control, before and at intervals during recovery after a 25 s maximal effort (mean ± S.D., $n = 8$).

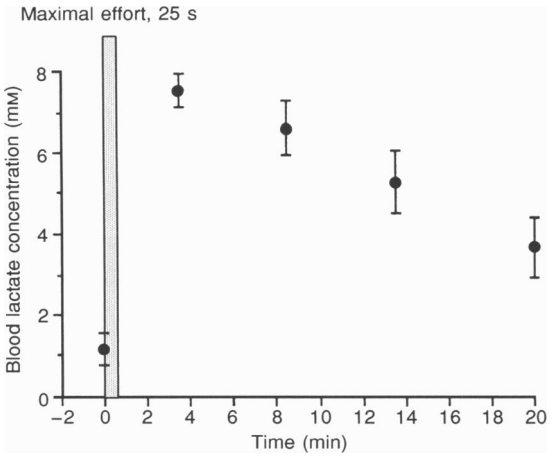

Figure 5. Blood lactate concentration
Blood lactate concentration at rest and during recovery from a 25 s maximal effort (mean ± S.D., $n = 6$).

voluntary force as the duration of the maximal effort increased was associated with a greater reduction in PSF, measured immediately after exercise. Recovery of PSF after a maximal effort of 45 s tended to be somewhat slower than after a maximal effort of 25 s duration. In one subject, in which a large reduction in PSF immediately after exercise was found (to 21·8% of the pre-exercise value), 20 min was not enough for full recovery of PSF.

Virtually identical results were obtained for both PSF and MRFD (Table 1).

DISCUSSION

In the present study we stimulated the relaxed quadriceps muscles with a short train of stimuli at 100 Hz during passive cycling, since single twitches or even short trains of high frequency stimulation superimposed on a voluntary dynamic contraction during cycling could not be detected at force levels above 50% of the maximal dynamic force. Only when a short train of high-frequency stimulation was applied to the relaxed muscle could the additional force be measured in a reliable manner. By comparing changes in voluntary force with changes in stimulated force after fatigue induced by dynamic contractions, we gain some insight into whether a failure of central drive plays a major role in maximal short-term cycling exercise.

During pedalling at 60 r.p.m. the time for electrical stimulation is very short and with a train of four pulses the force did not reach a plateau. The peak force achieved will be a function of the maximum force that could have been reached if the stimulation were to have continued for longer, and the rate of rise of force during the short interval of stimulation. The fact that both PSF and MRFD changed in so similar a fashion, as a result of the fatiguing exercise and during recovery, makes it difficult to decide whether the loss of performance was due to a change in intrinsic force-generating capacity or a slowing of the rate of activation of the muscle.

Series 1 and 2

There was a large reduction in PSF (31 ± 9%) due to a 25 s maximal effort which matched well the reduction in voluntary force (28 ± 9%). MRFD was also reduced to the same extent. Voluntary force recovered to pre-exercise values at $t = 3$ min. This was similar to the electrically stimulated force recovery. The initial kinetics of the voluntary force recovery seemed somewhat faster, however, so that at $t = 1$ min, that is after 35 s recovery only, voluntary force had returned to 92% of the initial control value (maximum PVF; Fig. 3), whereas PSF and maximum rate of force development had only recovered to 78 ± 12 and 80 ± 13%, respectively (Fig. 4).

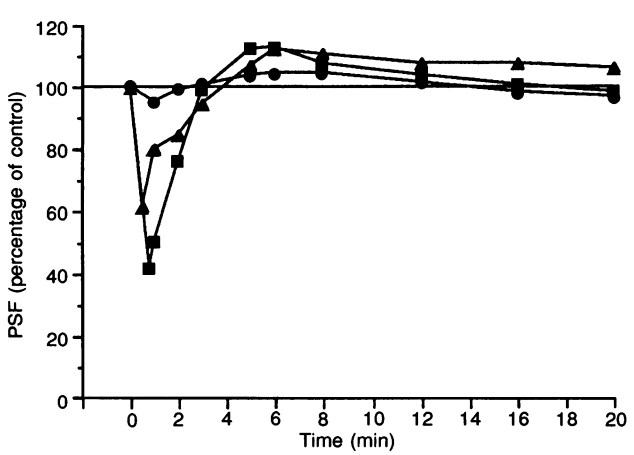

Figure 6. Peak stimulated force
PSF expressed as the percentage of control, before and during recovery from a 10 (●), 25 (▲) and 45 s (■) maximal effort. Data are for one subject.

Table 1. Peak stimulated force, maximum rate of force development and peak voluntary force

	Peak stimulated force (%)			Maximum rate of force development (%)			Peak voluntary force (%)		
	10 s	25 s	45 s	10 s	25 s	45 s	10 s	25 s	45 s
End	94·6 ± 4·5	72·2 ± 13·0	62·8 ± 12·3	104·1 ± 9·4	70·2 ± 12·1	44·7 ± 17·5	102·1 ± 7·1	67·3 ± 12·3	44·5 ± 15·6
t (min)									
1	98·3 ± 7·0	77·0 ± 14·1	53·2 ± 20·2	97·9 ± 11·3	74·2 ± 14·1	47·0 ± 15·2	—	—	—
2	102·3 ± 4·6	88·1 ± 7·2	71·8 ± 21·0	104·5 ± 7·9	89·6 ± 10·1	68·5 ± 21·7	—	—	—
3	100·6 ± 6·9	98·9 ± 4·6	86·7 ± 16·3	101·6 ± 9·1	101·2 ± 8·9	84·8 ± 18·7	—	—	—
5	105·4 ± 3·7	108·7 ± 7·9	97·9 ± 12·1	106·7 ± 6·3	114·6 ± 11·8	99·3 ± 19·2	—	—	—
6	108·1 ± 3·6	107·8 ± 8·2	101·5 ± 12·1	108·4 ± 7·5	112·3 ± 8·9	102·3 ± 16·0	—	—	—
8	108·1 ± 5·4	106·4 ± 5·1	100·1 ± 7·4	109·6 ± 4·8	110·8 ± 6·3	99·6 ± 9·7	—	—	—
12	101·3 ± 6·2	101·9 ± 4·0	93·6 ± 11·4	102·6 ± 3·7	102·7 ± 4·4	94·4 ± 9·7	—	—	—
16	98·1 ± 6·8	101·4 ± 4·4	87·0 ± 9·5	98·4 ± 3·7	105·1 ± 4·9	88·0 ± 13·1	—	—	—
20	98·1 ± 7·2	93·7 ± 10·4	87·6 ± 11·9	96·7 ± 5·6	96·7 ± 3·5	89·1 ± 15·1	—	—	—

The peak stimulated force and maximum rate of force development values before maximal effort are taken to be 100%; the values immediately after the maximal effort, for durations of 10, 25 or 45 s, and during recovery (t = 1–20 min) are shown. Peak voluntary force is the force generated at the end of the maximal effort, for durations of 10, 25 and 45 s, expressed as the percentage of the peak force generated at the beginning of the maximal effort (taken to be 100%).'End' signifies the force measured at the end of maximal effort. All values are expressed as a percentage of the value before the maximal effort (means ± S.D.).

Series 3

In Series 3 we induced different levels of fatigue by changing the duration of the maximal effort. The increased reduction in peak voluntary force at the end of the 45 s maximal effort compared to the maximal effort of 25 s was associated with an increased reduction in stimulated force (PSF as well as MRFD) although the reduction in stimulated force was more marked. Despite the greater reduction in voluntary and stimulated force (and hence the greater fatigue) the time course of recovery was almost the same. In two of the four subjects studied, complete recovery of stimulated force after a 45 s maximal effort had occurred at t = 3 min. In one subject stimulated force remained below control values during the whole 20 min recovery. During the 10 s maximal effort, no reduction in PVF was found, which was reflected by an unchanged PSF and MRFD compared with control values.

The close association between the changes in electrically stimulated force and voluntary force due to fatigue, together with a similar time course of recovery of stimulated force (PSF and MRFD) and voluntary force suggest that fatigue seen in this type of high-intensity exercise was most likely attributable to changes within the muscle itself, rather than a failure of central drive.

A few studies have examined voluntary activation during dynamic exercise by superimposing electrical stimulation. Although most studies have shown that in fresh muscles full voluntary activation during concentric contractions can usually be reached (Westing, Seger & Thorstensson, 1990; Newham *et al.* 1991; James *et al.* 1995), there is some disagreement about the extent of activation failure during fatiguing dynamic contractions. James *et al.* (1995) showed a minimal reduction in central drive during fatigue. This is in contrast to the results of Newham *et al.* (1991) who reported considerable activation failure (of ~30%) at the slow isokinetic contraction (20 deg s^{-1}) but not at the fast isokinetic contraction (150 deg s^{-1}). In the present study knee angular velocities during cycling at 60 r.p.m. are somewhat higher than the highest velocity used by Newham *et al.* (1991). The angular velocity in the knee at the moment of peak force production is ~200 deg s^{-1}. If the mechanism for an activation failure during dynamic contractions is related to the absolute force generated as has been suggested by Westing *et al.* (1990) then it may be expected that at the relatively high velocities and hence low forces generated in the present study, no activation failure occurred.

As previously pointed out in relation to the recovery of dynamic voluntary force (Sargeant & Beelen, 1993), the rapid recovery of stimulated and voluntary force follows a similar time course to that shown for the resynthesis of phosphocreatine after exhaustive cycle ergometer exercise (Harris, Edwards, Hultman & Nordesjö, 1976), which suggests that the decrease in force at the end of a maximal effort is associated with a reduction in phosphocreatine concentration. This may reflect a reduction in the maximum rate of ADP rephosphorylation as argued by Sahlin (1986). It is notable that in the present experiments

blood lactate concentration was still elevated after 20 min of recovery even though voluntary and stimulated force had fully recovered after 3 min. This observation is in agreement with more direct measurements of muscle lactate and pH during recovery of voluntary and stimulated isometric force (Sahlin, Harris, Nyland & Hultman, 1976).

These findings indicate clearly how in human muscle *in vivo*, there can be an absence of fatigue even under acidotic conditions, supporting the suggestion that the link between hydrogen ion accumulation and fatigue is probably not a simple cause and effect, but rather indirect or masked by other events (Sahlin & Ren, 1989; Cady, Jones, Lynn & Newham, 1989).

The potentiation seen in peak stimulated force after a 25 s maximal effort at $t = 5$ min (by about 10%) remained up to the measurement at $t = 8$ min. This elevation above control values was also seen in the MRFD although for the latter parameter greater increases were found (at $t = 5$, 6 and 8 min).

Although electrically stimulated force increased above control values at $t = 5$ min, this was not reflected by an increase in peak voluntary force. This may be due to a change in skin resistance as a result of an increased skin blood flow and sweat rate during, and subsequent to, the 25 s maximal effort. This makes percutaneous stimulation more effective without altering the voluntary force. Although a standard warm-up was performed prior to the control measurements this is probably not enough to increase skin blood flow to a level comparable with that after a 25 s maximal effort.

Potentiation was also seen in the subjects who performed in experiments in which the duration of the maximal exercise was varied. After a maximal effort of 10 s which had no effect on peak voluntary force, potentiation of stimulated force occurred in all subjects at $t = 5$ min. In two of the four subjects who performed the 45 s maximal effort a marked potentiation of stimulated force occurred. Factors that could contribute to the potentiation in PSF and MRFD might include muscle temperature (Sargeant, 1987). In one subject we measured muscle temperature (vastus medialis at 3 cm depth) immediately after the 25 s maximal effort and found an increase of 1 °C. Based on earlier experiments, however, (Sargeant, 1987), this could only account for an increase in (voluntary) force generated during cycling at 60 r.p.m. of ~2%. One reason for the relatively small increase in voluntary force compared with the marked increase in stimulated force (PSF and MRFD) may relate to the fact that, although the force builds up faster due to the increased muscle temperature, the actual plateau force is not increased.

In interpreting these results it should be recognised first that the stimulation was sufficient to activate a significant proportion of muscle force (36 ± 9% MVC), but that the duration was, of necessity, too short to achieve a plateau. Thus PSF and MRFD are normally related. Following full recovery after 3 min there was, however (as already pointed out) more marked potentiation of the MRFD than of the PSF. It is not clear why this should occur, but it may be due to subtle changes in the kinetics of force development.

Functional consequences

The rate of rise of tension in a tetanus is approximately directly proportional to the maximal speed of shortening (Close, 1964). Thus, the present findings of a slowing in MRFD could be seen as suggesting a slowing of the muscle. Indeed this would be consistent with both animal experiments (Crow & Kushmerick, 1983; Jones and Bigland-Ritchie, 1986; De Haan, Jones & Sargeant, 1989) and human experiments. In the latter, fatigue resulted in a greater reduction in power at fast, compared with slow, pedalling rates in a maximal cycling exercise (Beelen & Sargeant, 1991), or a greater reduction of knee extension isokinetic compared with isometric force (James *et al.* 1995). In the present study we had hoped to investigate fatigue during this complex dynamic task at different movement speeds and hence at different muscle shortening velocities. We tried cycling at 90 r.p.m. but our subjects were not able to let their legs be passively taken round by the motor-driven cranks. Due to the much higher inertia of the leg segments compared with cycling at 60 r.p.m. (Widrick, Freedson & Hamill, 1992) the amount of muscular activity varied considerably. It was not possible to make accurate and reproducible measurements of the force generated by electrical stimulation.

In conclusion, the close association between the changes in stimulated force and voluntary force consequent upon, and in recovery from, fatigue induced by short-term maximal dynamic exercise suggests that fatigue in this type of exercise may be due to changes in the contractile apparatus and is probably not a result of a reduced muscle activation by the CNS.

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