Journal of Physiology (1990), **429**, *pp.* 17–27
With 4 figures
Printed in Great Britain

REFLEX INHIBITION OF HUMAN SOLEUS MUSCLE DURING FATIGUE

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(*Received 8 September 1989*)

SUMMARY

1. Human soleus muscles were fatigued under ischaemic conditions by intermittent stimulation at 15 Hz. When maximal voluntary plantarflexion was then attempted, the loss of torque was found to be associated with a reduction in voluntary EMG activity.

2. The decrease in EMG activity could not have been due to 'exhaustion' of descending motor drive in the central nervous system since fatigue had been induced by electrical stimulation of peripheral nerve fibres. Similarly, the decrease could not be explained by changes at the neuromuscular junction or muscle fibre membrane, since changes in the M wave (evoked muscle compound action potential) were relatively modest.

3. When the excitability of the soleus motoneurones was tested during fatigue, using the H (Hoffmann) reflex, it was found to be significantly reduced. Control experiments with ischaemia or electrical stimulation, but without fatigue, failed to demonstrate any significant effects on reflex excitability.

4. The findings in this study favour the concept of reflex inhibition of α-motoneurones during fatigue.

INTRODUCTION

Previous experiments have demonstrated that the EMG (electromyographic) activity associated with maximal voluntary contractions (MVCs) declines during muscle fatigue. It has been established that the origin of the declining EMG is largely central to the neuromuscular junction (Woods, Furbush & Bigland-Ritchie, 1987; Garland, Garner & McComas, 1988) and the suggestion has been made that it is the result of reflex inhibition of the motoneurone pool (Bigland-Ritchie, Dawson, Johansson & Lippold, 1986; Woods *et al.* 1987).

The H(Hoffmann) reflex, can be used to test the above possibility since, under conditions of constant input, variations in the size of the H reflex provide a reliable measure of the net excitatory and inhibitory influences on the α-motoneurones (Schieppati, 1987). It is known that brief voluntary contractions of many muscles enhance or reveal H reflexes (Upton, McComas & Sica, 1971; Ete-Okoro, 1982; Morin, Katz, Mazieres & Pierrot-Deseilligny, 1982; Bulbulian & Darabos, 1986) and that there is depression of the responses during and immediately following the relaxation phase (Enoka, Hutton & Eldred, 1980; Schieppati & Crenna, 1984). In

addition, Kukulka, Moore & Russell (1986) have shown that recurrent inhibition of motoneurones is enhanced during sustained isometric contractions of the soleus but we are unaware of other studies of the H reflex in prolonged exercise and fatigue. The present study sought to determine the maximal amplitude of the H reflex, and hence the excitability of the α-motoneurone pool, during fatigue of the human soleus muscle. In order to eliminate psychological effects associated with voluntary effort as well as reflex inputs from other exercising muscles, fatigue was induced by electrical stimulation of the soleus muscle alone. The design of the experiments was such that any depression of the H reflex would provide evidence consistent with reflex inhibition of motoneurones consequent to fatigue. A brief report of this work has appeared elsewhere (Garland & McComas, 1988).

METHODS

Subjects

Thirteen healthy volunteers of both sexes participated in this study; their ages ranged from 22 to 53 years (mean 30·0 years). Of these subjects there were ten who showed sufficient fatigue (see below) to justify complete investigation; partial data were obtained from the other three subjects. Approval for the study was granted by the University Ethics Committee.

Stimulating and recording arrangements

Subjects were seated in a semi-reclined position with the head and arms supported. Subjects were advised to keep their body position stationary and to relax as much as possible. The legs were placed inside two metal supporting frames and clamped in position, so that the knees and ankles were held at 90 deg; the right foot was strapped onto an aluminium foot plate which housed strain gauges to measure torque (Marsh, Sale, McComas & Quinlan, 1981). The legs were warmed by an infra-red lamp. A blood pressure cuff was wrapped around the right thigh and inflated to at least 350 mmHg during the experiment. The presence of the cuff was necessary to hasten and maintain the fatigue process in the soleus muscle (an inherently fatigue-resistant muscle).

After preparing the skin with alcohol and conducting cream, stimulating electrodes, constructed of lead plate, were positioned bilaterally for the H reflex testing; for each leg the cathode (4·5 cm × 2·5 cm) was placed in the popliteal fossa over the posterior tibial nerve and the anode (8 cm × 8 cm) was placed superior to the patella. Single rectangular pulses of 0·5 ms duration were delivered every 5 s from a stimulator (Devices Ltd, model 3072), itself controlled by a digital timing unit (Devices Ltd, Digitimer, model 3290). The stimulus intensity was increased to 15–40 V until a maximal H reflex was recorded. The absence of any background EMG activity and torque indicated that the subjects were relaxed at the time of administration of the stimuli; this precaution was important since the H reflex is known to change if the soleus muscle is not fully relaxed (Verrier, 1985). Maximal M waves (evoked muscle compound action potentials) were elicited with the same stimulating electrodes and stimulator (50 μs pulses of 200–320 V).

The recording electrodes for H reflexes, M waves and voluntary EMG were silver disc electrodes, 1 cm in diameter, placed over the soleus muscle of each leg approximately 6 cm above the superior aspect of the calcaneus. The ground electrodes were attached to the skin between the stimulating and recording electrodes while the two reference electrodes were fastened over the dorsum of each foot; all four electrodes were silver strips, 5 cm × 0·7 cm.

Voluntary EMG activity was passed through an amplifier with a bandpass of 10 Hz–1 kHz and was displayed on a variable persistence storage oscilloscope (Hewlett-Packard Ltd, model 141B). The EMG activity was also full-wave rectified and the integrated EMG over 1 s was computed by a programmable desk-top calculator (Hewlett-Packard Ltd, model 9810A). All recordings of torque and EMG were stored on FM tape for subsequent analysis.

Fatigue procedure

The right soleus was fatigued with submaximal indirect electrical stimulation (120–160 V) at 15 Hz under ischaemic conditions. This frequency was chosen to minimize peripheral fatigue

(Garland *et al.* 1988) and to be within the physiological firing range for soleus motoneurones (Bellemare, Woods, Johansson & Bigland-Ritchie, 1983). The stimuli were rectangular voltage pulses of 50 μs duration; the stimulator (Devices Ltd, model 3072) received triggering pulses from a digital timing device (Devices Ltd, Digitimer, model 3290) through a gated pulse generator

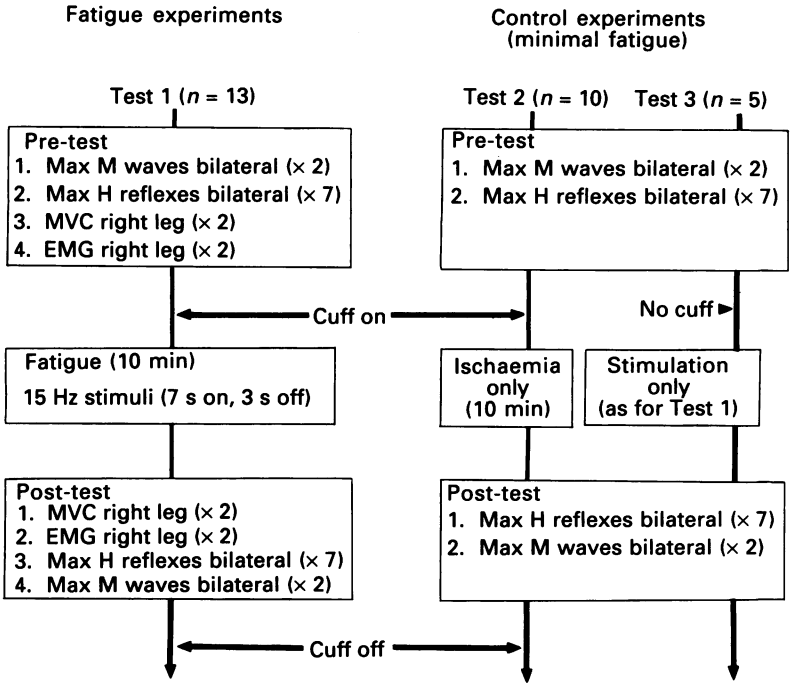

Fig. 1. Experimental protocol (see Methods).

(Devices Ltd, model 2521). The stimulating electrodes were two lead plates; the cathode (7·5 cm × 5·5 cm) lay over the soleus muscle belly 2 cm above the stigmatic recording electrode while the anode (12 cm × 7 cm) was slightly more proximal and over the gastrocnemius muscles. The trains of stimuli at 15 Hz were repeated every 10 s (7 s on, 3 s off) until the tetanic torque had fallen, in most experiments, by more than 60% of the control value.

Experimental protocol

Largely because of the various control procedures, the experimental protocols were rather complex and have therefore been summarized in Fig. 1.

Ten subjects were tested on two occasions with at least 1 week between tests (Fig. 1). The tests included: fatigue with ischaemia (test 1) and ischaemia without fatigue (test 2). Five of the subjects also volunteered for a further test: electrical stimulation without fatigue or ischaemia (test 3).

Pre-test measures included maximal M waves (two measurements) and maximal H reflexes (seven measurements) in each of the two legs, together with torque (two measurements) and soleus EMG (two measurements) during maximal voluntary contraction (MVC) of the plantarflexors of the right ankle (Fig. 1). The various types of measurement were averaged, except for torque, for which the larger of the two values was accepted. Interpolated stimuli (two pulses at 100 Hz) were administered during MVCs to assess the level of voluntary effort; in this test, the appearance of a twitch superimposed on the recording of voluntary torque is taken as evidence that the contraction is not maximal (Belanger & McComas, 1981).

In test 1, fatigue was induced under ischaemic conditions by electrical stimulation of the right soleus; the entire procedure took approximately 10 min. The left leg served as a non-ischaemic and non-fatigued control.

Duplicate measurements were made of maximal voluntary plantarflexor torque and soleus EMG activity in the right leg after fatigue had been achieved; as in the pre-test period, the degree of effort was determined using the interpolated twitch technique (see above). Following adjustments of the respective stimulus intensities seven maximal H reflexes and two maximal M waves were then recorded from both legs. The comparison of pre-test and post-test values for the right leg served to identify the effects of fatigue. Conversely, any generalized behavioural or environmental factors were assumed to affect the H reflexes similarly on the two sides, and the use of the left leg as a control enabled any such effects to be detected and eliminated in the calculation of 'reflex excitability' (see below). It is possible, however, that there may have been minor changes in the H reflex in the left leg due to crossed afferent inputs from the fatigued soleus muscles (cf. Baldissera, Hultborn & Illert, 1981). The complete sequence of post-test measurements occupied 90–120 s, during which time the arterial cuff remained inflated; within this period, at least 30 s were allowed to elapse between MVCs and H reflex recordings to avoid the problem of post-contraction depression of the H reflex (Enoka *et al.* 1980; Schieppati & Crenna, 1984). The maximal M wave served to identify any reduction in excitability at the neuromuscular junction or muscle fibre membrane as a result of fatigue.

In test 2, the effects of ischaemia alone on reflex excitability were determined. Pre-test maximal M waves and H reflexes were compared with those taken in the post-test period after 10 min of ischaemia.

In test 3, an attempt was made to study the effects of the electrical stimulation alone on reflex excitability. Pre-test maximal M waves and maximal H reflexes were compared with those taken in the post-test period after electrical stimulation had been delivered without the presence of ischaemia. This last protocol largely eliminated the development of fatigue.

Maximal H reflex amplitudes were expressed in relation to the respective maximal M wave amplitudes (Angel & Hofmann, 1963). Theoretically the H:M ratios, so determined, should not have been affected by any changes in the peripheral excitability of the muscle fibres consequent to fatigue (e.g. Miller, Giannini, Milner-Brown, Layzer, Koretzky, Hooper & Weiner, 1987). In practice, the rather distal location of the stigmatic recording electrode over the soleus may have permitted other (non-fatigued) muscles to contribute to the M wave in some subjects: when this happened the H reflexes and M waves had different configurations, as in Fig. 3. The difference between the pre-test and post-test H:M ratios was expressed as a percentage of the corresponding pre-test value. Any percentage change in H:M ratios from the left (control) leg was then subtracted from the percentage change on the right (experimental) side so as to give the 'reflex excitability' (RE). In this way the left leg served as a control for any generalized environmental factors affecting reflex excitability.

Statistical analysis

The statistical significance for the change in H reflex amplitude between tests and between legs was determined using analysis of variance (ANOVA) with repeated measures. After this Tukey's multiple comparisons tested for specific differences between the three tests. In test 1 changes during fatigue in MVC plantarflexor torque, voluntary EMG activity and maximal M waves were analysed with paired t tests; the α level of significance was set at $P = 0{\cdot}05$. Correlation coefficients were calculated to determine the relationship between post-test values of RE and EMG associated with maximal voluntary contractions. Throughout the text standard deviations have been given with arithmetic mean values.

RESULTS

Test 1

After 10 min of intermittent 15 Hz stimulation of the right soleus, performed under ischaemic conditions in test 1, the tetanic torque had fallen to less than half of the initial value in ten of the subjects (mean decline $66{\cdot}6 \pm 10{\cdot}7$ % S.D.). In these subjects the fatiguing stimulation was then stopped and measurements were made of

maximal voluntary plantarflexor torque, integrated soleus EMG activity, and the maximal M wave and H reflex amplitudes (see Methods); during this period the arterial cuff was left inflated. Both the voluntary torque and the integrated EMG activity were found to have decreased significantly ($P < 0{\cdot}001$), the mean reductions

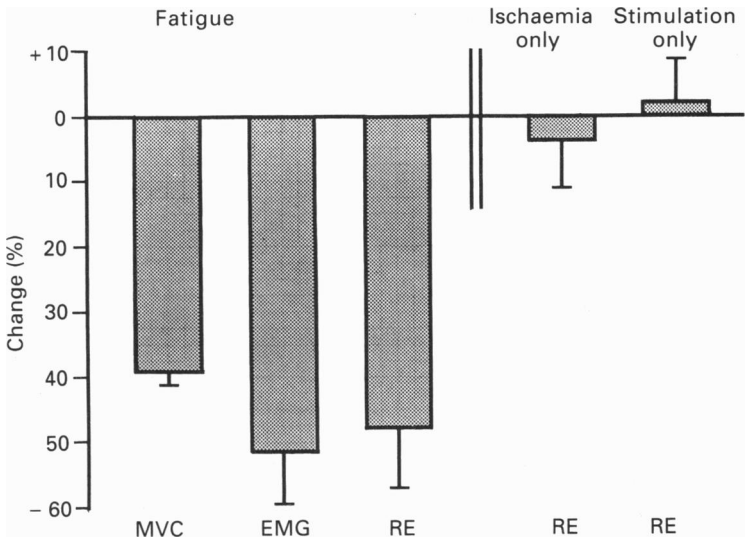

Fig. 2. Pooled results (means ± S.E.M.) showing effects of fatigue, ischaemia and stimulation on maximal voluntary torque (MVC), maximal voluntary EMG (EMG) and reflex excitability (RE) (see text).

being $38{\cdot}5 \pm 8{\cdot}6\%$ and $51{\cdot}9 \pm 17{\cdot}9\%$ respectively (Fig. 2). In addition, the maximal M wave showed a mean decline in amplitude of $8{\cdot}8 \pm 11{\cdot}9\%$ ($P < 0{\cdot}05$), indicating either some peripheral impairment of muscle fibre excitation or slowed impulse conduction in muscle fibres.

In the post-test condition H reflexes could still be elicited from the fatigued soleus muscles in nine of the ten subjects. However, the H reflexes were, on average, $47{\cdot}9 \pm 7{\cdot}6\%$ smaller than those in the resting (pre-test) state and this reduction in size exceeded any diminution in the M waves, causing the H:M ratios to fall (Fig. 2). The results of a typical experiment are shown in Fig. 3. In contrast to the findings in the fatigued legs, the H:M ratios in the left, non-fatigued, legs were not significantly altered, suggesting that emotional or other general factors were unimportant. When the H:M ratios for the two sides were compared, as described in Methods, the mean change in reflex excitability, due to fatigue, was $47{\cdot}3 \pm 27{\cdot}1\%$ ($P < 0{\cdot}001$).

To assess the reliability of the H reflex observations, seven measurements of amplitude were made for each leg in both the pre-test and post-test conditions. The pre-test variability, expressed as the coefficient of variation, ranged from 0·2 to 7·8% and the mean value was $4{\cdot}8 \pm 2{\cdot}9\%$. In the post-test condition (test 1) the corresponding values were $10{\cdot}0 \pm 9{\cdot}4\%$ in the left, control legs and $15{\cdot}8 \pm 13{\cdot}8\%$ in the right, fatigued legs (except one leg with consistently small but very variable responses). Inter-test variability was examined by repeating the entire experiment in

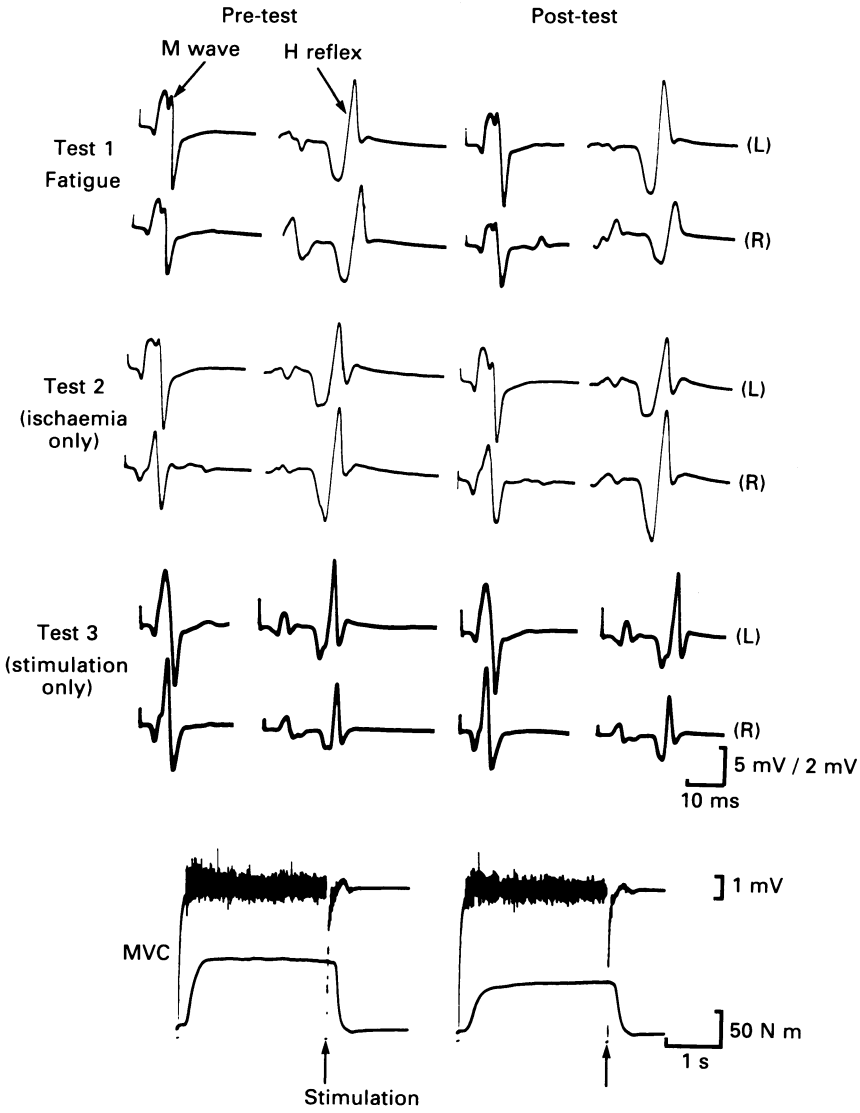

Fig. 3. Top, data from one subject illustrating maximal H reflex and M wave changes during the three tests. The first two columns from the left are pre-test values of M wave and H reflex amplitude, respectively; the third and fourth columns are post-test values of M wave and H reflex amplitude. Within each test (fatigue, ischaemia only and stimulation only), the electrical responses from the left control leg are above those from the right (test) leg. Any dissimilarities in the shapes of the H reflex and the M wave reflect the contributions of muscles other than soleus, but supplied by the tibial nerve, to the M wave. Calibrations of 5 and 2 mV refer to recordings for M waves and H reflexes, respectively. Bottom, maximal plantarflexion torque and associated EMG for the same subject before and after the fatiguing stimulation.

five subjects and in four of these the paired decreases in H:M ratio as a result of fatigue showed good correspondence (mean difference, 10·0 ± 7·5 %); no reason could be found for the one anomalous value.

Tests 2 and 3

The purpose of tests 2 and 3 (Fig. 1) was to establish to what extent ischaemia alone, or tetanic stimulation alone, might have been responsible for the depression in

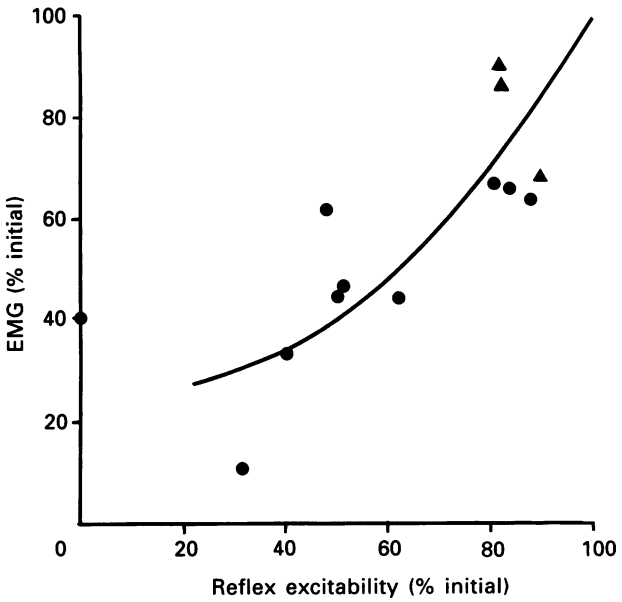

Fig. 4. Post-test EMG plotted against post-test reflex excitability (RE) for each of the thirteen subjects, using the equation, $V = 1{\cdot}75\,E = 0{\cdot}011\,E^2$, (where V and E represent EMG and reflex excitability respectively, both expressed as percentages of their initial values). The subjects with results denoted by ▲ experienced less than 30 % reduction in the MVC plantarflexion torque and hence were excluded from the remainder of the study (see Results).

reflex excitability. In these control experiments fatigue was minimal, the small reductions in mean tetanic torque being less than 10% and not statistically significant. The effect of ischaemia alone on the H reflex varied from one subject to another, but overall there was a small and insignificant reduction in reflex excitability (mean 3·7 ± 20·8 %; Fig. 2). In the post-ischaemic legs, the variability of the H reflex measurements was mostly between individuals; the mean coefficient of variation for the seven observations in each subject was 6·8 ± 4·4 %. In view of the variability between subjects, the change in reflex excitability following test 2 (ischaemia alone) was subtracted from that following test 1 (fatigue with ischaemia) for each person. Even when the contribution of ischaemia had been removed, the mean change in reflex excitability (43·6 ± 36·5 %) was still significant ($P < 0{\cdot}01$). The results of tetanic stimulation alone (test 3) were similar in that the mean change in reflex excitability was small and insignificant (1·8 ± 13·4 % increase; Fig. 2).

From the various control experiments, described above, it therefore appeared that the loss of H reflex excitability was a consequence of muscle fatigue rather than of any incidental factors in the experiments. Since the purpose of the study was to investigate whether, during fatigue, a decline in motoneurone excitability might underlie the progressive fall in EMG activity, these two variables were plotted against each other for each of the ten subjects (Fig. 4). Points have been added for the three other subjects in whom the soleus muscles proved particularly difficult to fatigue, and in whom the reductions in tetanic torque in test 1 were less than 30%. It was found that a significant relationship existed between integrated EMG activity and reflex excitability of soleus muscles such that a reduction in one, occurring during fatigue, was associated with a fall in the other (Fig. 4; $r = 0{\cdot}67$, $P < 0{\cdot}025$). In twelve of the thirteen subjects, the interpolated stimulus technique revealed that declines in EMG were not due to reduced effort during the voluntary contractions. In the remaining subject an interpolated twitch was evident prior to fatigue and an even larger one (17% of the control twitch torque) in the post-test trial.

DISCUSSION

The present study was undertaken to determine the electrophysiological mechanisms responsible for the decline in voluntary EMG activity observed following electrically induced fatigue of the soleus muscle (Garland *et al.* 1988). The decline in EMG could not be explained by 'exhaustion' of descending motor drive since these pathways had not been employed to induce fatigue. It was also evident that the decline in voluntary EMG activity could not be explained by loss of excitability of neuromuscular junctions or muscle fibre membranes. Although the small decline in the maximal M wave indicated the presence of some muscle fibre membrane inexcitability or of slowed impulse conduction, this was much less than the fall in voluntary EMG activity. To this extent, the results for the soleus muscle were consistent with those obtained by a similar experimental strategy for the human ankle dorsiflexors (Garland *et al.* 1988).

A more likely possibility was that the loss of voluntary EMG activity resulted from a decrease in α-motoneurone excitability; the testing of this possibility was the primary aim of the present study. A significant decrease in the mean level of reflex excitability could be demonstrated in the ten subjects; further, this fall was significantly associated with the decline in EMG activity.

A reduction in reflex excitability was also found by Kukulka *et al.* (1986), who employed a modified H reflex testing technique (Bussel & Pierrot-Deseilligny, 1977) to demonstrate increased recurrent inhibition, exerted by Renshaw cells, during maximal voluntary contractions of the soleus. Our experiments differed from those of Kukulka *et al.* in the use of electrical stimulation and ischaemia, rather than voluntary contraction, and in the production of significant fatigue. In view of the time elapsing between the end of the fatiguing stimulation and the start of the H reflex measurements (typically 30–40 s), the depression of the H reflex in the present experiments could not have been due to recurrent inhibition. The lack of any consistent changes in the H reflex, when electrical stimuli were delivered in the absence of fatigue (test 3), also pointed to recurrent inhibition being an unimportant

factor in the reduction of reflex excitability during fatigue. Rather, in keeping with the suggestion of Bigland-Ritchie *et al.* (1986), we have attributed the H reflex changes to inhibitory afferents projecting to motoneurones from receptors in the fatigued muscle.

The present study has not ruled out the possibility of reflexly induced depression of descending motor drive as a secondary factor in some individuals. Thus, even though interpolated twitches were absent during fatigue, it is still possible that there were some motor units which were no longer capable of either descending activation or of force generation in response to direct stimulation of motor axons (see also Garland *et al.* 1988). In keeping with such a possibility were observations in one subject in whom the depression of EMG activity was much greater than that in reflex excitability. In contrast to this situation, there was another subject in whom the H reflex was completely abolished while the EMG was reduced to 40% of original. It is possible that such a subject was able to overcome the reflex inhibition with increased descending excitatory drive to the motoneurone pool.

In experiments of this kind, there is no *a priori* reason why the declines in EMG and reflex excitability should be identical. Indeed, there are probably many important differences in the spinal circuitry involved in the H reflex and the descending drive onto motoneurones, and in the postsynaptic responses of the motoneurones to the two forms of excitatory command. Such differences could cause a fatigue-induced reduction in motoneuronal excitability to affect the EMG and H reflex excitability to unequal extents, particularly when the potential effects of presynaptic inhibitory circuits are taken into account.

In evaluating the present results, it was necessary to consider two possible confounding factors, ischaemia and electrical stimulation. The ischaemic cuff was necessary to induce fatigue in soleus within a reasonable period of time. However, the pressure exerted by the cuff on the sciatic nerve might have compromised impulse conduction in afferent fibres so that the H reflex would decrease in the absence of any fatigue effect (cf. Mayer & Mawdsley, 1965; Kots, 1977). Three additional subjects were found in whom this appeared to be the case, since their H reflexes were depressed with the ischaemic cuff inflated and in the absence of fatiguing stimulation; those subjects were excluded from the study. In the remaining ten subjects, the decreases in reflex excitability were greater in the fatigue protocol (test 1) than in the presence of ischaemia alone (test 2).

With regard to electrical stimulation, it is conceivable that stimulation of soleus motor axons could have depressed motoneurone excitability through antidromic activation of the Renshaw circuit; however, any such effect should have worn off by the time that the H reflexes were tested (see above). Cutaneous and muscle afferent input from the stimulation might also have affected motoneurone excitability but these effects were more likely to have been excitatory than inhibitory (Hagbarth, 1960; Delwaide, Crenna & Fleron, 1981; Crenna, Schieppati & de Curtis, 1982). To control for any such effects, electrical stimuli were delivered without ischaemia or fatigue; in this condition it was found that reflex excitability remained unaffected.

The presence of any discomfort during the experiment was also considered. The pain associated with the ischaemic cuff and with electrical stimulation was present in the control tests and yet depression of the reflex excitability was not evident.

Further, any effect of cutaneous pain would have been expected to be facilitatory rather than inhibitory (see above). In the present experimental protocol, there was also a deeply felt pain, which was presumably mediated by nociceptive endings within the fatigued muscle; this was regarded as an inevitable and natural part of the fatigue process and, as such, did not introduce any foreign variable. It is an open question as to whether or not these nociceptive afferents were responsible for the reflex inhibition observed in the present work.

It could be argued that use of the maximal H:M ratio biased the study towards finding depressive events (cf. Meinck, 1980). However, seven of ten subjects in this study demonstrated facilitation of the H:M ratio in the control experiments; hence, the ratio was sensitive to change in either direction and provided a meaningful measure of reflex excitability.

To conclude, both voluntary EMG and the reflex excitability of the α-motoneurone pool were significantly depressed during fatigue of the soleus muscle induced by electrical stimulation. The respective depressions could not be explained by peripheral failure, or reduced motor drive, or by the effects of ischaemia and stimulation alone. Instead the findings were consistent with the existence of a reflex whereby α-motoneurones were inhibited by afferents from the exercising muscle.

We wish to thank the Natural Sciences and Engineering Research Council of Canada and the Ontario Ministry of Health for support. We are also indebted to Doug Stuart for helpful comments and to Glenn Shine, Robin Roberts and Pat Holmes for technical, statistical and secretarial assistance.

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