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# BEHAVIOUR OF SHORT AND LONG LATENCY REFLEXES IN FATIGUED HUMAN MUSCLES

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SUMMARY

1. The human abductor pollicis brevis (APB) and first dorsal interosseus (FDI) were fatigued by sustained maximal voluntary contractions and, in the case of the APB also by electrically induced (30 Hz) contractions, until the loss of force reached 50 % of control. The short latency or Hoffmann reflex (H reflex) and the long latency reflex (LLR) were evoked during weak voluntary contractions by the electrical stimulation of the median nerve at the wrist in control, during and after the fatigue experiments.

2. As compared to control, the normalized H reflex amplitude in the two fatigue modalities was found to have decreased by 30 % without any significant change in the LLR. This finding and the observation that the LLR was enhanced by 46 % in simultaneous recordings, in which the APB remained at rest during FDI fatigue, could be explained by a stronger descending fatigue-induced central drive which spreads to neighbouring non-fatigued muscles.

3. A comparison of the H reflex and the LLR behaviour during fatigue indicates that motoneurone activation threshold is not affected but that changes in peripheral drive are present, which possibly induce presynaptic inhibition of Ia afferents and/or inhibition of interneurones in the oligosynaptic pathways. Our observation of a rather slow time course for the H reflex decrease during fatigue supports the point of view that these inhibitions are activated by metabolic and/or chemical changes in the fatigued muscle.

4. It is concluded from the results of this study that muscle fatigue induces an enhanced descending supraspinal drive which compensates for a loss of excitation from the peripheral afferents on motoneurones.

## INTRODUCTION

Previous work has shown that muscle fatigue induced by maximal voluntary contraction (MVC) or electrical stimulation is associated with a decrease not only in force, but also in electromyographic (EMG) activity (for reviews see Bigland-Ritchie & Woods, 1984; Hainaut & Duchateau, 1989; Edwards & Gibson, 1991;

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Enoka & Stuart, 1992). Changes in EMG activity do not result mainly from neuromuscular junction failure in fatigue of short duration at the physiological frequency of motor unit activation, but from modifications to ionic muscle membrane processes (Merton, 1954; Bigland-Ritchie, Kukulka, Lippold & Woods, 1982; Duchateau & Hainaut, 1985; Milner-Brown & Miller, 1986). It has also been suggested that the excitability of the $\alpha$-motoneurone (MN) pool is depressed during fatigue (Kukulka, Moore & Russell, 1986; Garland & McComas, 1990) and thus contributes to the decline in the motor unit firing frequency recorded during sustained MVC (Bigland-Ritchie, Johansson, Lippold, Smith & Woods, 1983; Marsden, Meadows & Merton, 1983). In addition to the intrinsic adaptation of the MN firing frequency to a constant excitatory drive (Kernell & Monster, 1982*a*, *b*), changes in their activation may be reflexly modulated by afferents from the descending central drive and/or from the peripheral origin. At peripheral sites, it has been recently suggested that reduced facilitatory muscle spindle output contributes to the decreased excitation of the MNs (Bongiovanni & Hagbarth, 1990; Macefield, Hagbarth, Gorman, Gandevia & Burke, 1991). Another suggestion previously made is that the reduced excitation of a muscle MN pool during fatigue may be the result of reflex inhibition mediated by small diameter afferents from the exercising muscle (Bigland-Ritchie, Dawson, Johansson & Lippold, 1986; Woods, Furbush & Bigland-Ritchie, 1987; Garland & McComas, 1990).

The effects of fatigue on muscle afferents can be approached experimentally by means of a quick stretch which triggers short latency (SL) and longer latency EMG reflex responses. We recently recorded results in the first dorsal interosseous (FDI) which also support the point of view that afferents of peripheral origin induce a reduction in amplitude of the short latency reflex responses (Balestra, Duchateau & Hainaut, 1992), but they do not allow the question to be answered as to whether this decrease is due to reduced spindle output or to a decline in transmission at the spinal level.

In the present paper we examine this question in the FDI and abductor pollicis brevis (APB) by a study of reflex responses evoked by the electrical stimulation of the nerve (Upton, McComas & Sica, 1971; Deuschl, Schenk & Lücking, 1985). This method was used in order: (1) to analyse the behaviour and time course of the short and long latency reflex components during fatigue; and (2) to discuss the effects of decreased spindle sensitivity and afferent input into the MN pool. The results indicate that the decreased short latency reflex response is mainly controlled by changes in the peripheral drive to the MN pool. It also appears that muscle fatigue induces an enhanced descending supraspinal drive which compensates for changes in peripheral input into the MN pool.

## METHODS

### *Subjects*

Twelve healthy volunteers of both sexes (1 female and 11 male; 24–44 years old) well accustomed to the experimental conditions took part in this investigation. Most of them were submitted to various experimental protocols on several occasions at an interval of at least 1 week. This study was approved by the University Ethics Committee, and the subjects gave their informed consent to participation in the investigation.

*Stimulation*

The short latency or Hoffmann reflex (H reflex) and long latency reflexes (LLR) evoked by electrical stimulation were recorded during a weak sustained 10–15 % contraction of the muscle

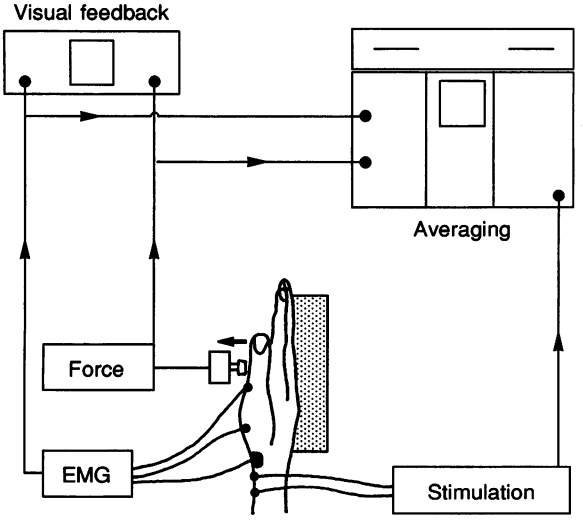


Fig. 1. Illustration of the experimental set-up when recording the EMG and force in the abductor pollicis brevis (APB).

MVC by using the method introduced by Upton *et al.* (1971) and adapted by Deuschl *et al.* (1985). This method consists of using two silver surface electrodes to electrically stimulate the median nerve at the wrist at a frequency of 2 Hz. The stimuli were rectangular pulses of 1 ms duration, and the intensity of the stimulation was set near the threshold response of the motor fibres. In order to normalize the H reflex changes during fatigue (cf. below), the maximal muscle compound action potential ($M_{max}$) was evoked by a supramaximal stimulation of the nerve. Pulses were delivered from a custom-made two-channel stimulator triggered by a digitimer (model 4030, Digitimer Ltd, Welwyn Garden City, UK). The muscle reflex responses and the $M_{max}$ were evoked through the same electrodes.

*EMG and force recording*

The EMG recordings (H reflex, LLR and $M_{max}$) of the abductor pollicis brevis (APB) and the first dorsal interosseous (FDI) were obtained by means of a pair of silver disc electrodes (8 mm in diameter), one fixed over the muscle motor point and the other over its distal tendon (belly-tendon derivation). The ground electrode was fastened to the skin between the stimulating and recording electrodes. The signal was AC amplified (1000 ×), filtered (bandpass, 10 Hz to 5 kHz), and full-wave rectified. The reflex responses were averaged (64–128 sweeps).

The recordings of the APB were made with the subject's arm placed on a horizontal board in a semi-supine position, with the back of the hand fixed against a vertical restraint (Fig. 1). The muscle abduction force was recorded by pushing with the middle of the thumb against a strain-gauge transducer (TC 100, Kulite, Ridgefield, NJ, USA). It was possible to record reflexes in five subjects' FDIs in response to the stimulation of the median nerve (heteronymous reflex response; cf. Deuschl, Michels, Berardelli, Schenck, Inghilleri & Lücking, 1991). To do so, the subject's arm was placed on a horizontal board with the palm of the hand turned downwards, the last three fingers strapped together, and the thumb immobilized in abduction and extension. The abduction force of the FDI was recorded by pushing with the middle of the fully extended index finger against the transducer (cf. Duchateau & Hainaut, 1990). In the experiments with the FDI, the reflex responses in the APB were recorded simultaneously. In this condition the abduction force

of the thenar muscles was monitored by pushing down with the middle of the thumb against a second transducer fixed to the table. The subjects were provided with visual feedback of the force and EMG signals in order to maintain a steady level of contraction.

*Muscle fatigue and testing procedure*

Muscle fatigue was induced by either a sustained MVC or an electrically evoked contraction in the APB, and only by sustained MVC in the FDI. In the electrically induced contractions the muscle was stimulated through the EMG recording electrodes, with supramaximal pulses at a frequency of 30 Hz. Due to the synergistic muscle contribution in the MVC, its maximal force was greater (by about 50 %) when compared to the maximal force evoked by electrical stimulation. Because of this, the contraction was interrupted in both fatigue tests when the force fell to 50 % of its initial value. The $M_{max}$ and reflex responses were recorded before and immediately after the fatigue tests. To avoid any recovery of the EMG activity during the post-fatigue recording, a blood pressure cuff wrapped around the arm was inflated (250 mmHg) just before the end of the fatigue-provoking contraction. The cuff (maintained for 45 s) was then removed and recovery was tested every 5 min for at least 15 min. In five experiments the cuff was maintained for 3–5 min in order to test the reflex recovery in the absence of blood circulation. In additional experiments performed on four subjects' APBs, the time course of $M_{max}$ and the changes in the reflex responses were analysed after various periods of sustained MVC. The EMG recordings were assessed before and after 5 and 10 s MVCs. An ischaemic cuff was inflated just before each MVC and maintained (total duration about 1 min) during the post-MVC recordings. Ten minutes rest was allowed between the two MVCs. Thereafter the subjects were asked to sustain four 20 s MVCs (under occluded blood supply), each separated by 40 s pauses. These pauses were necessary to test the $M_{max}$ and to average the reflex responses. In all the experiments the temperature of the skin overlying the muscle was continuously maintained at about 35 °C by means of an infra-red lamp.

*Measurements*

Whereas the force was continuously recorded on a paper chart (Graphtec, WX2400), the EMG data were recorded and averaged by a digital oscilloscope (Nicolet 4094c) before being stored on a floppy disk (Nicolet, XF44). For each reflex component and $M_{max}$ we measured: (1) latency as a time between the stimulus artifact and the beginning of the EMG responses; (2) duration and (3) peak amplitude. The size of each reflex was defined as the distance between the peak amplitude and the mean background level computed during the 15–20 ms following the stimulus. In order to exclude fatigue-induced changes in the muscle fibre membrane response each EMG component was normalized as a function of: (1) the peak size of the $M_{max}$; and (2) the percentage increment above the background level of the EMG activity. This last procedure is known to be independent of the recording conditions and the fluctuations of the force applied (Matthews, 1986). The data recorded during muscle fatigue and recovery were statistically tested by means of an analysis of variance (ANOVA) with repeated measures on one factor.

## RESULTS

### *Effects of fatigue caused by MVC*

In the control APB, the electrical stimulation of the median nerve with a stimulus intensity set near the threshold response of the motor fibres induced two EMG reflex responses (Fig. 2). The H reflex had a mean ($\pm$ S.D.) onset latency of $29{\cdot}6 \pm 1{\cdot}7$ ms, a duration of $10{\cdot}9 \pm 1{\cdot}3$ ms and a crude peak amplitude of $229 \pm 48\ \mu$V (which corresponded to $6{\cdot}2 \pm 2{\cdot}4$ % of $M_{max}$ or $3{\cdot}2 \pm 1{\cdot}5$ times the background activity). The LLR had a mean onset latency of $51{\cdot}5 \pm 2{\cdot}5$ ms and a peak amplitude of $124 \pm 49\ \mu$V. The effects of fatigue on the duration of the LLR were not considered because it is sometimes difficult to ascertain from our records the moment when this component returns to background EMG activity.

The sustained MVCs which were interrupted when the force of contraction dropped to 50 % of its control value, had a mean duration of $101{\cdot}0 \pm 16{\cdot}1$ s. When

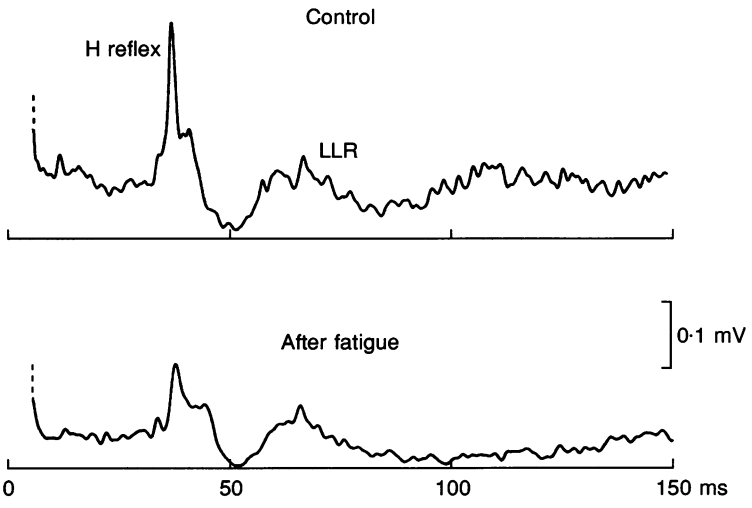


Fig. 2. H reflex and long latency reflex (LLR) recordings in the APB in response to the electrical stimulation of the median nerve at the wrist (averaging 64 sweeps). Typical traces recorded in control and after fatigue by a sustained MVC of 90 s are illustrated for one subject.

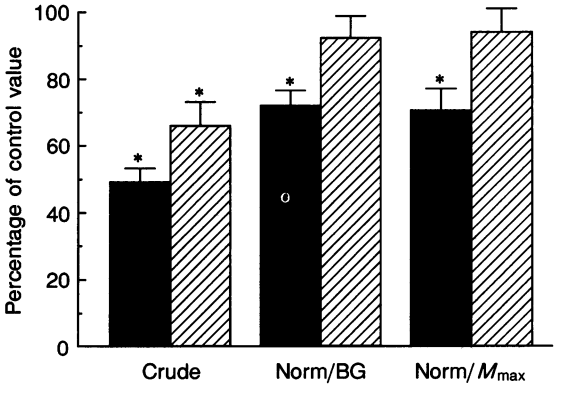


Fig. 3. Histogram showing the effect of fatigue induced by sustained MVC on the H reflex (■) and long latency reflex (▨) amplitudes. Crude and normalized EMG amplitudes with respect to mean background level (Norm/BG) and $M_{max}$ amplitude (Norm/$M_{max}$) are expressed as percentages of control. The values are means ± S.E.M. for 10 subjects. * $P < 0{\cdot}001$.

fatigue had set in (Fig. 2), the two reflex components showed a mean reduction in crude amplitude of 51·3 ± 13·2 and 34·4 ± 22·5 % for the H reflex and the LLR respectively. However, the H reflex only showed a significant ($P < 0{\cdot}001$) decrease in peak amplitude normalized as a function of the $M_{max}$ amplitude (30·2 ± 21·3 %). It should be noticed that there was no difference between normalized H reflex

values with respect to $M_{max}$ or to its background EMG activity induced by the voluntary 10–15 % contraction of the maximal force (Fig. 3). The results reported above were of the same magnitude ($n = 6$) when the absolute level of force of the sustained contraction was increased to 10–15 % of the control MVC during the

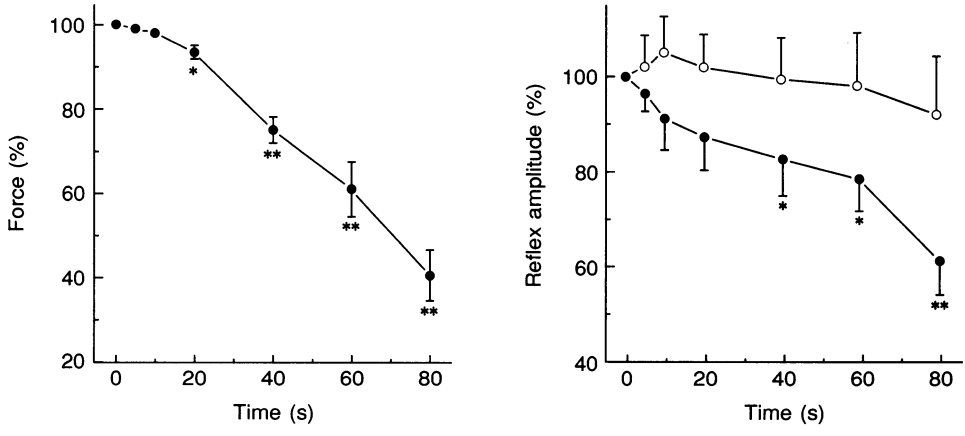


Fig. 4. Force (left-hand graph) and reflex amplitude (right-hand graph) time changes during fatigue by intermittent MVC in the APB. Values, expressed as percentages of control, are means ± S.E.M. for 4 subjects. ●, H reflex; ○, long latency reflex. * $P < 0{\cdot}05$, ** $P < 0{\cdot}01$.

TABLE 1. Onset latency and duration of reflex (H reflex and LLR) responses in the APB before and after fatigue by maximal voluntary and electrically evoked contractions

| | Latency (ms) | | Duration (ms) |
|---|---|---|---|
| | H reflex | LLR | H reflex |
| Voluntary contraction | | | |
| Before | 29·6 ± 1·6 | 52·1 ± 2·8 | 11·0 ± 1·3 |
| After | 30·3 ± 1·9 | 52·9 ± 2·6 | 13·3 ± 1·8 |
| *P* | n.s. | n.s. | 0·005 |
| Electrical stimulation | | | |
| Before | 29·5 ± 1·8 | 50·7 ± 1·8 | 10·8 ± 1·2 |
| After | 30·4 ± 2·2 | 51·9 ± 2·0 | 13·2 ± 1·7 |
| *P* | n.s. | n.s. | 0·005 |

Values are means ± S.D. for 10 subjects.

recording of the H reflex after fatigue. In these fatigue experiments the onset latencies of the H reflex and the LLR did not change significantly, while the duration of the H reflex increased by 21·2 ± 14·2 % ($P < 0{\cdot}005$). The means of the APB data recorded from ten subjects are reported in Table 1. These changes persisted as long as the cuff, inflated around the arm just before the end of the fatigue test, was maintained and returned to the control values within 5 min of the removal of the ischaemia.

*Effects of fatigue induced by electrical stimulation*

In the APB ($n = 10$) fatigue was also induced by sustained maximal electrical stimulation (30 Hz) at the motor point. This fatigue test was interrupted when the

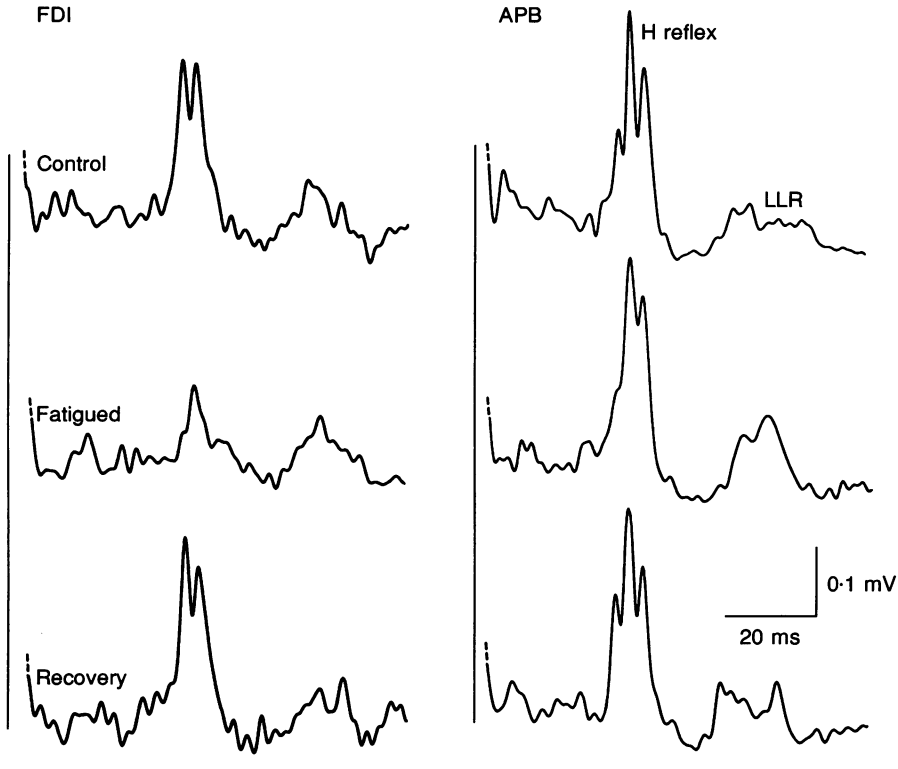


Fig. 5. Simultaneous recording of the H reflex and long latency reflex (LLR) in the FDI (left-hand traces) and the APB (right-hand traces) in response to the electrical stimulation of the median nerve at the wrist (averaging 64 sweeps) in one subject. In the FDI, the reflex responses are shown in control condition after fatigue provoked by a sustained MVC of 105 s and after 5 min of recovery. In the APB, the reflex responses are shown at comparable periods, but the muscle remained at rest throughout the fatigue test.

force of contraction fell to 50 % of its control value (mean duration of stimulation, $107{\cdot}0 \pm 21{\cdot}6$ s). The crude H reflex amplitude exhibited a mean reduction of $42{\cdot}0 \pm 19{\cdot}4$ % ($P < 0{\cdot}001$) and the normalized H reflex amplitude (with respect to $M_{max}$) decreased by $31{\cdot}4 \pm 21{\cdot}0$ % ($P < 0{\cdot}005$). While the crude LLR amplitude decreased by $30{\cdot}3 \pm 26{\cdot}5$ % ($P < 0{\cdot}05$) during electrically induced fatigue, its normalized amplitude (with respect to $M_{max}$) was not modified significantly ($-7{\cdot}8 \pm 29{\cdot}4$ %; $P > 0{\cdot}05$). The H reflex and LLR onset latencies did not change during fatigue, while the H reflex duration increased significantly (cf. Table 1). These data are not significantly ($P > 0{\cdot}05$) different from the results recorded during fatigue by voluntary contraction.

*Time course of force and EMG reflex changes during fatigue by MVC*

The time course of the force and EMG (H reflex and LLR) reflex changes during fatigue induced by intermittent MVC were analysed in the APB ($n = 4$) by testing

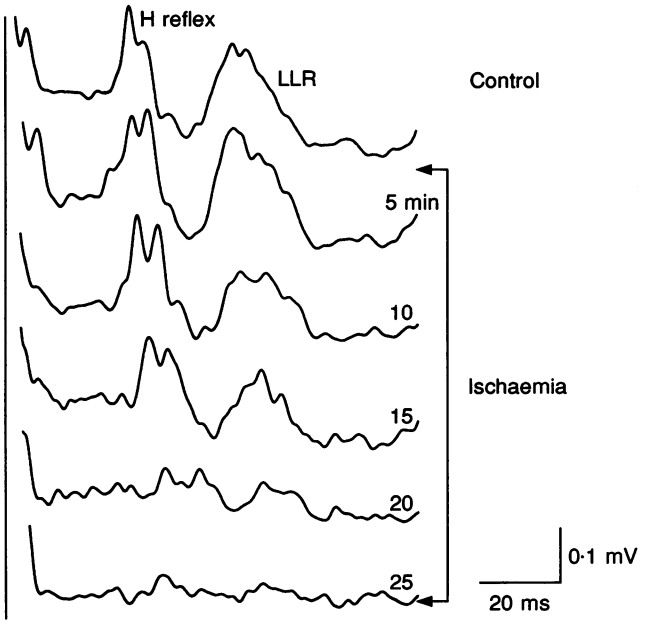


Fig. 6. H reflex and long latency reflex (LLR) recordings in the APB in response to the electrical stimulation of the median nerve at the wrist. Traces (averaging 64 sweeps) illustrate control and the effect of ischaemia recorded every 5 min in one subject.

their values after periods of 5, 10, 20, 40, 60 and 80 s duration. The maximal force dropped progressively during fatigue to mean values which were significantly different from control after 20 s, and decreased thereafter by about 60 % in 80 s (Fig. 4). During these fatigue tests, the normalized H reflex amplitude (with respect to $M_{max}$) decreased progressively by about 40 % in 80 s, but was only significantly different from control after 40 s contraction (Fig. 4). After fatigue tests of identical duration, the LLR showed no significant change in peak amplitude normalized to $M_{max}$ throughout the contractions.

*Behaviour of the LLR in the absence of fatigue*

In five of our subjects it was possible to elicit heteronymous FDI reflex responses via the electrical stimulation of the median nerve. This interesting possibility was used to study simultaneously the H reflex and the LLR in fatigued FDI and unfatigued APB. The FDI H reflex peak amplitude decreased by $32{\cdot}8 \pm 11{\cdot}9$ and $28{\cdot}8 \pm 11{\cdot}0$ % ($P < 0{\cdot}005$ in both cases) when normalized respectively to the $M_{max}$ obtained in response to the supramaximal stimulation of the ulnar nerve at the

wrist and to the EMG background activity. At the same time the H reflex duration increased by $16{\cdot}3 \pm 10{\cdot}5\,\%$ ($P < 0{\cdot}05$), while its onset latency did not change significantly. The LLR showed a normalized peak amplitude of $97{\cdot}7 \pm 13{\cdot}1\,\%$ (not significantly different from control), while its onset latency remained at control values throughout the fatigue test. There was no significant difference from control in the APB ($97{\cdot}9 \pm 11{\cdot}0\,\%$, Fig. 5) immediately after fatigue, when the H reflex amplitude was drastically reduced in the FDI. The changes in H reflex amplitude observed in the FDI during fatigue underwent a similar recovery time course when the fatigue test was interrupted, as in the case of the APB.

During the above-mentioned experiments involving the simultaneous recording from APB and FDI we also analysed the LLR in the APB, which remained at rest during the FDI fatigue. While $M_{max}$ did not change in the five subjects, the normalized (with respect to $M_{max}$) LLR showed a systematic enhancement ($46{\cdot}8 \pm 19{\cdot}7\,\%$; $P < 0{\cdot}01$) immediately after the FDI fatigue test without any significant change in the onset latency (cf. Fig. 5). The comparison of the LLR enhancement in the non-fatigued APB and the H reflex decrease in the neighbouring fatigued FDI indicates a close correlation ($r = 0{\cdot}89$; $P < 0{\cdot}05$), which means that the increase in LLR is proportionately larger in subjects showing greater H reflex drops in the fatigued muscle. The increased LLR response in the APB returned to control values within 5 min of the end of the FDI fatigue test.

*Effects of ischaemia on the H reflex and LLR*

Since the H reflex reduction after fatigue was tested using a cuff, the possible effects of ischaemia were examined in a complementary experiment. In this experiment a cuff was inflated around one subject's arm for 25 min in the absence of fatigue and the two reflex responses were recorded (Fig. 6). While there was no change in H reflex amplitude during the first 10 min of ischaemia, within the next 15 min the reflex response decreased almost to zero in comparison with its control value (Fig. 6). When the cuff was deflated, the H reflex recovered and returned to 85 % of its control amplitude in the following 10 min. The LLR showed a similar behaviour pattern during and after ischaemia.

## DISCUSSION

In the present investigation the H reflex and the LLR were evoked in the APB and FDI by electrical stimulation of the median nerve at the wrist. The major finding is that fatigue during sustained MVC and electrical stimulation induces a decrease in normalized H reflex amplitude with a rather slow time course in the progressive development during the MVC tests, but without any significant change in the LLR. While the LLR was enhanced in a neighbouring muscle which remained at rest, the H reflex was not changed.

In our experiments, the normalized H reflex EMG responses decreased by roughly 30 % and it should be pointed out that there was no significant difference between the normalization of the H reflex amplitude as a function of $M_{max}$ provoked by the supramaximal stimulation of the motor nerve and as a function of the EMG background induced by sustained voluntary contraction (10–15 % of MVC). A cuff

was inflated around the arm in order to avoid recovery during the averaging of the reflex activities after fatigue, but this did not affect our results. Indeed the reduction in the normalized H reflex amplitude should not be related to ischaemia since the simultaneously recorded H reflex in the unfatigued adjacent APB was not reduced. However, the ischaemia did induce a reflex decrease which started after 10 min in the unfatigued muscle, a latency which is too long to be of any importance in the interpretation of our results. Moreover, the pain associated with the ischaemic cuff inflated during the post-fatigue recordings should not affect the results since: (1) although the pain was more severe after fatigue by MVC, the drops in the H reflex were identical in the two fatigue tests; and (2) in the combined experiments, the H reflex was not modified in the unfatigued APB at a time when it was depressed in the fatigued FDI.

Since the decrease in the H reflex was observed during a small voluntary contraction (10–15 % of MVC) which kept the MNs' excitability rather constant, reduced central drive should not be the cause of this observation. The H reflex drop during fatigue may have resulted from other changes: (1) MN adaptation processes; (2) reduced muscle spindle output; or (3) transmission decline induced by increased presynaptic inhibition of the Ia terminals and/or inhibition of interneurones in the oligosynaptic pathways. Our observation that the H reflex was not significantly different from the control values during the first 40 s of the fatigue tests suggests that changes with a rather slow time course play a dominant role with respect to the H reflex decrease. The adaptation of the MN properties to constant excitatory drive as observed in anaesthetized cats by Kernell & Monster (1982*a*, *b*) does not appear to be preponderant with respect to the H reflex decrease recorded in our experiments because: (1) the time course of our observations was slower; (2) there was no recovery when ischaemia was maintained. Moreover, recent work suggests that the adaptation of the MN properties is not present during voluntary contractions (see Enoka & Stuart, 1992).

The mono- and oligosynaptic contributions to the short latency reflex evoked by stretch (SL) and electrical stimulation (H reflex) may differ slightly (cf. Burke, Gandevia & McKeon, 1984; Van Boxtel, 1986), but the main point is that the latter procedure bypasses the spindle. The comparison of normalized H reflex decreases in the APB and FDI with previously reported SL decreases in the FDI (Balestra *et al.* 1992) shows almost identical changes (−30 %) under comparable fatigue conditions. This suggests that the decrease in muscle spindle sensitivity should not play a key role in SL reduction during fatigue. Thus, in the present experiments, reduced spindle input into the MN pool should not explain the H reflex reduction, a point of view which is supported by the finding that changes in the spindle discharge frequency have a faster time course than the H reflex decrease (cf. Macefield *et al.* 1991). Moreover, one of the four subjects in which the normalized H reflex time course was tested even showed a slight increase in H reflex amplitude (11 % in the first 10 s of contraction) at a time when the muscle spindle discharge frequency should be reduced.

In these fatigue experiments, the H reflex reduction without any change in the LLR could be explained by a decline in transmission along neural elements between

the nerve stimulation and the H reflex responding MNs as a result of the activation of muscle afferents causing presynaptic inhibition of the Ia terminals and/or inhibition of interneurones in the oligosynaptic pathways. Because of the slow time course of the H reflex decrease during fatigue, it is suggested that this is induced by muscle metabolic or chemical processes which could be mediated by small diameter afferents from the fatigued muscle (Garland, 1991). This conclusion is supported by our finding that the H reflex decrease does not recover as long as ischaemia is maintained (cf. also Bigland-Ritchie *et al.* 1986; Woods *et al.* 1987; Garland & McComas, 1990).

In the present experiments the LLR was generated by the submaximal electrical stimulation of the median nerve at the wrist and its normalized amplitude did not change significantly during MVC and electrically induced fatigue. Moreover, in the experiments with simultaneous recordings from the fatigued FDI and the unfatigued APB, the normalized LLR amplitude was even increased in the APB without change of the H reflex. This LLR enhancement in the unfatigued muscle was closely correlated ($r = 0{\cdot}89$) with the H reflex drop recorded from the adjacent fatigued muscle. The absence of LLR decrease during fatigue should not be related to the observed H reflex drop and thus a reduced refractoriness of the MN pool to the LLR following the H reflex discharge since: (1) a large LLR was not correlated ($P > 0{\cdot}05$) with a small H reflex either in the control ($r = 0{\cdot}025$) or in the fatigued ($r = 0{\cdot}03$) muscles; and (2) in the APB, which remained at rest during FDI fatigue, the LLR was found to be enhanced without change of the H reflex.

It has been suggested that the H reflex and LLR evoked by the electrical stimulation of the median nerve at the wrist have the same origin and are both transmitted by Ia fibres, but that the LLR is routed transcortically (Deuschl *et al.* 1985, 1991; Mariorenzi, Zarola, Caramia, Paradiso & Rossini, 1991). If this is right, the different behaviour of the LLR compared to the H reflex in the sustained contraction could be explained by a stronger descending central drive induced by the electrical stimulation which elicits the reflex responses as a result from a higher central excitability at supraspinal levels during muscle fatigue. This proposition is in agreement with the observation of increased 'Bereitschaftspotential' or readiness potential and the suggestion of intensified central nervous activation required for sustained contractions (Freude & Ullsperger, 1987). The other possibility, that cutaneous facilitatory afferences might explain the different behaviour of the H reflex and the LLR, in the present work does not appear to be of any major importance because the LLR behaved similarly in the fatigue tests induced by voluntary contraction or transcutaneous electrical stimulation at the motor point (see also Maertens de Noordhout *et al.* 1992).

It is concluded that during sustained contractions muscle fatigue induces a peripheral afferent feedback which provides the MNs with less excitation and, as a reflex compensation, a stronger descending supraspinal drive which spreads to neighbouring non-fatigued muscles.

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