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Reflex regulation during sustained and intermittent submaximal contractions in humans

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To investigate whether the intensity and duration of a sustained contraction influences reflex regulation, we compared sustained fatiguing contractions at 25 % and 50 % of maximal voluntary contraction (MVC) force in the human abductor pollicis brevis (APB) muscle. Because the activation of motoneurones during fatigue may be reflexively controlled by the metabolic status of the muscle, we also compared reflex activities during sustained and intermittent (6 s contraction, 4 s rest) contractions at 25 % MVC for an identical duration. The short-latency Hoffmann (H) reflex and the long-latency reflex (LLR) were recorded during voluntary contractions, before, during and after the fatigue tests, with each response normalised to the compound muscle action potential (M-wave). The results showed that fatigue during sustained contractions was inversely related to the intensity, and hence the duration, of the effort. The MVC force and associated surface electromyogram (EMG) declined by 26.2 % and 35.2 %, respectively, after the sustained contraction at 50 % MVC, and by 34.2 % and 44.2 % after the sustained contraction at 25 % MVC. Although the average EMG increased progressively with time during the two sustained fatiguing contractions, the amplitudes of the H and LLR reflexes decreased significantly. Combined with previous data (Duchateau & Hainaut, 1993), the results show that the effect on the H reflex is independent of the intensity of the sustained contraction, whereas the decline in the LLR is closely related to the duration of the contraction. Because there were no changes in the intermittent test at 25 % MVC, the results indicate that the net excitatory spinal and supraspinal reflex-mediated input to the motoneurone pool is reduced. This decline in excitation to the motoneurones, however, can be temporarily compensated by an enhancement of the central drive.

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The progressive decline in force that occurs during a sustained maximal voluntary contraction (MVC) is accompanied by adjustments in both the muscular and nervous systems (see Enoka & Stuart, 1992; Gandevia, 2001). Several studies have proposed that the central neural drive is controlled by reflex afferents that optimise force and thus prevent a loss of energy; that is, muscular wisdom (see Marsden *et al.* 1983; Bigland-Ritchie *et al.* 1986*b*; Garland & Gossen, 2002). In addition to a possible role for recurrent inhibition (Löscher *et al.* 1996*b*), three main mechanisms are suggested to modulate the neural drive to the muscle during fatigue: (1) motoneurone adaptation to a constant excitatory input (Kernell & Monster, 1982*a*, *b*); (2) decline in the group Ia excitatory input from muscle spindle afferents, which induces motoneurone disfacilitation (Bongiovanni & Hagbarth, 1990; Macefield *et al.* 1991); and (3) reflex inhibition of motoneurones that is mediated by feedback from group III and IV muscle afferents (Bigland-Ritchie *et al.* 1986*b*; Woods *et al.* 1987; Garland & McComas, 1990). Such reflex inhibition has been suggested to result from the accumulation of metabolites in the muscle fibres that then activate metaboreceptors and induce an inhibition of homonymous motoneurones via small diameter muscle afferents (Kaufman *et al.* 1983; Sinoway *et al.* 1993).

The viewpoint that metabolite accumulation in the muscle during fatigue provokes motoneurone reflex inhibition was supported in a previous study (Duchateau & Hainaut, 1993) that compared the behaviour of short- and long-latency reflexes in human abductor pollicis brevis (APB) and first dorsal interosseus muscles during sustained MVCs. The results also suggested that muscle fatigue is accompanied by an enhanced descending drive, which compensates for the loss of excitation from peripheral afferents on motoneurones. In contrast, the larger decrease in the interpolated twitch during a sustained submaximal contraction (Lloyd *et al.* 1991; Sacco *et al.* 2000) suggests that there are greater adaptations in the nervous system during fatiguing contractions sustained at submaximal compared with maximal force. This interpretation is supported by the observation of greater motor unit recruitment and

modulation of discharge rate during long-lasting fatiguing contractions (Bigland-Ritchie *et al.* 1986*a*; Carpentier *et al.* 2001).

Metabolic and chemical changes have been observed during sustained submaximal (30–40 % MVC) contractions, such as decreased pH and phosphocreatine content and accumulation of inorganic phosphate (P_i) and lactate (Béliveau *et al.* 1991; Vestergaard-Poulsen *et al.* 1992). However, intermittent contractions at a similar force level, at least prior to exhaustion, do not appear to involve similar metabolic changes (Bigland-Ritchie *et al.* 1986*a*; Moussavi *et al.* 1989). Consequently, feedback by group III and IV afferents (Bigland-Ritchie *et al.* 1986*b*; Woods *et al.* 1987; Garland & McComas, 1990) is likely to be different for sustained and intermittent contractions.

The purpose of the study was to compare the behaviour of the short-latency (H reflex) and long-latency (LLR) reflexes in the APB during contractions sustained at 25 % and 50 % MVC and during sustained and intermittent contractions at 25 % MVC. The sustained and intermittent contractions at 25 % MVC were performed for a similar duration.

METHODS

Thirteen healthy volunteers (3 women and 10 men; 21–46 years of age) who were familiar with the protocol took part in the study after giving informed consent. The University Ethics Committee approved the study and all experimental procedures were performed in accordance with the Declaration of Helsinki.

Force and EMG recordings

The force and EMG recordings of the abductor pollicis brevis (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 (Duchateau & Hainaut, 1993). The abduction force exerted by the thumb was recorded by pushing with the middle part of the first phalanx of the thumb against a strain-gauge transducer (Kulite, TC 2000-50, Basingstoke, UK; sensitivity 55 mV N^{-1}; linear range: 0–220 N). The EMG recordings of the APB were obtained with a pair of silver disc electrodes (8 mm in diameter), with one fixed over the muscle motor point and the other over its distal tendon. The ground electrode was placed on the wrist between the stimulating and recording electrodes. The EMG signal was AC amplified (× 1000), filtered (bandpass: 10 Hz–5 kHz), and full-wave rectified. Throughout the experiments, the subjects were provided with visual feedback of the force and EMG signals.

Stimulation procedure

The short-latency Hoffmann (H) reflex and long-latency reflex (LLR) evoked by electrical stimulation were recorded during a weak, sustained contraction at 25 % MVC by using the method introduced by Deuschl *et al.* (1985). This involves using two silver surface electrodes (8 mm in diameter) to electrically stimulate the median nerve at the wrist at a frequency of 3 Hz. The stimuli were rectangular pulses of 1 ms duration, and the intensity of the stimulation was set at or slightly above the threshold response of the motor fibres. To normalise the H reflex changes during fatigue (see below), the maximal compound muscle action potential (M-wave) was evoked by a supramaximal (20–30 %) stimulation of the nerve in the absence of muscular contraction. Pulses were delivered from a custom-made, two-channel stimulator triggered by a digital timer (Model 4030, Digitimer Ltd, Welwyn Garden City, UK). The reflex responses and the M-wave were evoked with the same electrodes.

Muscle fatigue and testing procedure

Each experiment began with the recording of the M-wave. Five responses were evoked at 5 s intervals. Thereafter, the subject performed three MVCs, which each lasted for 3–4 s. There was a 3 min rest between each MVC. After the last MVC, there was a 5 min rest and then reflex responses were recorded and averaged from 36 traces. Usually, 3–4 series of averaging were recorded.

In a first session, a fatiguing contraction was performed at 25 % MVC and sustained until the endurance limit. In the second session, the subject performed an intermittent contraction (6 s contraction, 4 s rest; Bigland-Ritchie *et al.* 1986*a*) at 25 % MVC for the same duration as the sustained contraction at 25 % MVC. In the third session, the subject performed a sustained contraction at 50 % MVC until the endurance limit. The two sustained contractions (25 and 50 % MVC) ended when the subject was unable to maintain the required level of force for a period of 5 s. Ten subjects took part in the first two experimental protocols (1–2 weeks between sessions). Seven of the original ten subjects participated in the third protocol (10–12 months later) in addition to another three subjects.

Voluntary EMG, M-wave, and reflex responses (H reflex and LLR) were recorded during the fatigue tests. For the sustained contractions (25 and 50 % MVC), the reflex responses were recorded during the last 12 s of every minute and at the end of the test, whereas for the intermittent contractions the responses were recorded and averaged during two successive contractions. These contractions are of a similar duration to those in the sustained contraction. For all fatigue conditions, reflex responses were recorded at 25 % MVC and averaged from 36 traces. The M-wave was recorded at the end of every minute of contraction during a brief rest period; the subject stopped the sustained contraction for 2–3 s for this purpose. An MVC was performed by the subject at the end of the fatigue test. The M-wave, reflex responses, and MVC were recorded once every 5 min for 15 min to assess recovery from fatigue. In all the experiments, the temperature of the skin overlying the muscle was maintained at approximately 35 °C with an infrared lamp.

Measurements

The force and corresponding EMG were continuously recorded on a computer and digitised at a frequency of 2 kHz (Biopac System Inc., Santa Barbara, CA, USA). The reflex responses were recorded and averaged with a digital oscilloscope (Model 4094c; Nicolet, Madison, WI, USA) at a sampling rate of 20 kHz and stored on floppy disk (Nicolet, XF44). The maximum force was determined from the trial that yielded the largest value. For the voluntary EMG (MVC and 25 % or 50 % MVC), the mean amplitude was determined for a 2 s duration. The reflex responses were characterised by (Fig. 1): (1) latency – the time between the stimulus artefact and the beginning of the EMG responses (small arrows); (2) duration – the time interval between the burst onset and the point at which it crossed the mean background EMG level; and (3) peak amplitude. The duration of the LLR was not considered due to the difficulty in identifying the moment at which this component returned to background EMG activity. The amplitude of each reflex was defined as the distance between the peak amplitude and the mean background level during the

Table 1. Peak amplitude, onset latency and duration of reflex (H reflex and LLR) responses before and after fatigue by sustained (50 and 25 % MVC) and intermittent (25 % MVC) contractions

	Sustained 50 % MVC		Sustained 25 % MVC		Intermittent 25 % MVC	
	Before	After	Before	After	Before	After
Peak amplitude (μV)						
H reflex	190.1 ± 10.9	98.1 ± 6.1 ***	176.9 ± 16.1	85.3 ± 9.9 ***	178.4 ± 8.2	185.3 ± 8.3
LLR	139.3 ± 13.7	91.7 ± 8.8 ***	158.3 ± 12.9	75.5 ± 5.2 ***	135.5 ± 11.8	139.8 ± 8.9
Latency (ms)						
H reflex	26.5 ± 0.6	27.1 ± 0.5	27.6 ± 0.5	29.8 ± 0.6	28.2 ± 0.4	29.1 ± 0.4
LLR	49.2 ± 1.2	49.8 ± 1.0	49.6 ± 0.8	51.3 ± 0.7	50.4 ± 0.6	51.4 ± 0.6
Duration (ms)						
H reflex	12.6 ± 0.8 ***	16.9 ± 1.7	12.5 ± 0.5	14.6 ± 0.8 **	12.3 ± 1.3	12.4 ± 1.1

Data are means ± S.E.M. for 10 subjects. ** $P < 0.01$, *** $P < 0.001$, after *vs.* before. The data did not differ significantly between groups prior to the fatiguing contractions.

10–15 ms preceding the onset of the H reflex response. To exclude fatigue-induced changes in the muscle fibre membrane response, each EMG response was normalised to the amplitude of the M-wave.

The data were statistically tested by an analysis of variance (ANOVA) with repeated measures on one factor (time). When a significant main effect was found, the Bonferroni test was used to locate significant differences between the means of each fatigue test and recovery compared with control values, and to compare the sustained (25 % and 50 % MVC) and intermittent fatigue tests.

RESULTS

Changes in reflex responses during fatigue

The electrical stimulation of the median nerve, with a stimulus intensity set at or slightly above the threshold response of the motor fibres, induced two EMG reflex responses (Fig. 1*A*): a short-latency (H reflex) and a long-latency (LLR) response. The onset latencies of the H reflex and LLR were about 30 ms and 50 ms, respectively (Table 1).

The behaviour of the H reflex and LLR were analysed during submaximal (25 % and 50 % MVC) fatiguing contractions sustained until exhaustion and compared with the reflexes that were recorded during an intermittent contraction at 25 % MVC for the same duration. The endurance limit ranged from 360 to 660 s (mean ± S.E.M.: 492 ± 39.6 s) for the sustained 25 % MVC test and from 150 to 240 s (205 ± 8.9 s) for the sustained 50 % MVC test. Figure 1 shows a typical recording from a subject before and after the sustained 25 % MVC test and after 5 min of recovery. The main observation was that at the end of the fatigue test the two reflex components showed a reduction in absolute amplitude (Fig. 1*B*). The mean decreases in H reflex and LLR amplitudes were 51.8 % and 52.3 % for the sustained 25 % MVC test and 48.4 % and 34.2 % for the sustained 50 % MVC test (Table 1). In contrast, there was no significant change in either the H reflex or the LLR amplitude for the intermittent fatigue test (Table 1). Similarly, the M-wave declined by 22.4 % ($P < 0.001$) at the end of the 25 % MVC test and by 11.8 % ($P < 0.05$) at the end of the 50 % MVC test (Table 2 and Fig. 4*B*), and was not different after the intermittent test.

In contrast to the absence of a significant change in the normalised amplitude of the H reflex during the intermittent

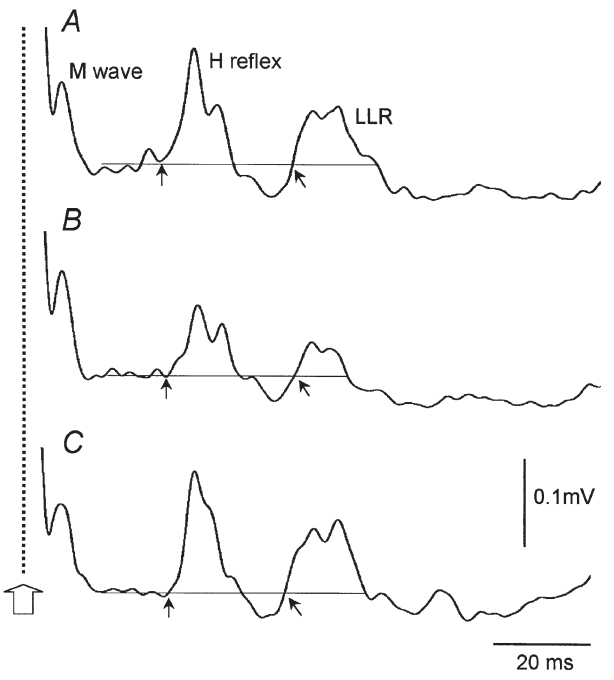

Figure 1. Hoffmann (H) reflex and long latency reflex (LLR) recordings in the abductor pollicis brevis (APB)

Typical traces (average of 36 sweeps) recorded in one subject in response to the electrical stimulation of the median nerve at the wrist before (*A*) and after (*B*) fatigue caused by a submaximal (25 % MVC) contraction which was sustained until the endurance limit, and after 5 min of recovery (*C*). The dotted vertical line represents the onset of the stimulation artefact. On each trace, the horizontal line denotes the average background EMG level and the small arrows indicate the onset of the two reflex responses.

Table 2. Force and EMG during the MVC, and M wave amplitude before and after fatigue by sustained (50 and 25 % MVC) and intermittent (25 % MVC) contractions

			Recovery		
	Before fatigue	After fatigue	5 min	10 min	15 min
Force					
Sustained 50 % MVC (N)	53.5 ± 6.8	39.5 ± 3.9 **	52.5 ± 4.4	52.3 ± 5.7	54.3 ± 5.1
Sustained 25 % MVC (N)	54.8 ± 7.0	36.1 ± 5.7 ***	49.8 ± 6.7	53.2 ± 6.7	54.3 ± 7.1
Intermittent 25 % MVC (N)	57.9 ± 10.3	55.3 ± 9.1	57.1 ± 9.1	57.1 ± 9.5	56.8 ± 9.6
Average EMG					
Sustained 50 % MVC (μV)	389.1 ± 38.7	252.1 ± 22.6 **	359.8 ± 33.6	377.7 ± 34.2	368.8 ± 36.5
Sustained 25 % MVC (μV)	428.0 ± 41.6	238.8 ± 34.0 ***	407.5 ± 38.8	427.1 ± 41.1	416.0 ± 39.9
Intermittent 25 % MVC (μV)	393.3 ± 34.8	368.1 ± 21.3	399.2 ± 34.2	391.3 ± 29.1	385.8 ± 34.6
M wave					
Sustained 50 % MVC (mV)	7.9 ± 1.0	7.0 ± 0.5 *	8.1 ± 1.0	8.1 ± 1.1	7.5 ± 1.0
Sustained 25 % MVC (mV)	7.7 ± 1.1	6.0 ± 0.5 ***	7.7 ± 1.3	7.7 ± 1.2	7.3 ± 1.1
Intermittent 25 % MVC (mV)	7.3 ± 0.9	7.1 ± 0.9	7.3 ± 0.8	7.4 ± 0.8	7.3 ± 0.9

Data are means ± S.E.M. for 10 subjects. The data did not differ significantly between groups prior to the fatiguing contractions. Significantly different from pre-fatigue at * $P < 0.05$; ** $P < 0.01$; *** $P < 0001$.

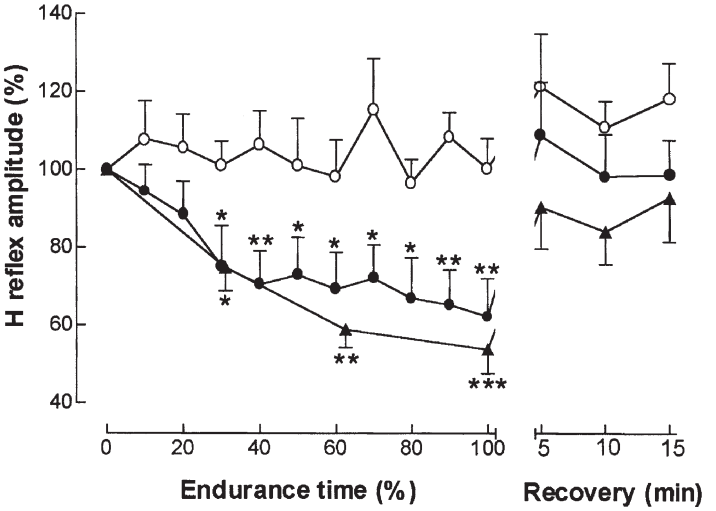

Figure 2. Changes in H reflex amplitude during submaximal contractions and recovery

Values (means ± S.E.M.; $n = 10$) obtained during sustained contractions at 25 % (●) and 50 % MVC (▲), and during intermittent contractions at 25 % MVC (○) as a function of endurance time. All data were first normalised to the corresponding M-wave amplitude and thereafter expressed as a percentage of the pre-fatigue value. Significant differences from pre-fatigue value: * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

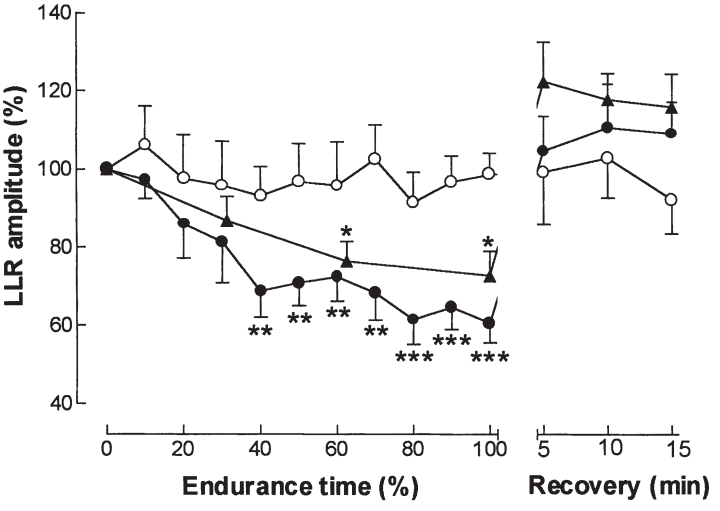

Figure 3. Changes in LLR amplitude during submaximal contractions and recovery

Values (means ± S.E.M.; $n = 10$) obtained during sustained contractions at 25 % (●) and 50 % MVC (▲), and during intermittent contractions at 25 % MVC (○) as a function of endurance time. All data were first normalised to the corresponding M-wave amplitude and thereafter expressed as a percentage of the pre-fatigue value. Significant differences from pre-fatigue value : * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

test, the sustained (25 and 50 % MVC) contractions induced a progressive decrease that reached statistical significance ($P < 0.05$) at 30 % of endurance time (Fig. 2). At the end of the test, there was a mean reduction in the amplitude of the H reflex of 37.8 ± 9.7 % ($P < 0.001$) for the 25 % MVC test and 46.1 ± 6.2 % ($P < 0.001$) for the 50 % MVC test. Similarly, the normalised amplitude of the LLR decreased by 39.4 ± 5.0 % for the 25 % MVC test and by 27.3 ± 6.3 % for the sustained 50 % MVC test (Fig. 3). In absolute terms, the decline in H reflex and LLR occurred more rapidly during sustained contractions at 50 % MVC than at 25 % MVC because the endurance time was shorter. In the three fatigue experiments, the onset latencies of the H reflex and LLR did not change significantly (Table 1). The duration of the H reflex did not change during the intermittent contractions, whereas it increased significantly during the first third of the two sustained fatigue tests. The changes that occurred in the reflex responses during the sustained contractions recovered completely within 5 min (Figs 2 and 3).

Changes in voluntary EMG during the submaximal contractions

The average EMG activity normalised to the M-wave increased progressively during the sustained submaximal contractions. For the 25 % MVC test, EMG increased slightly (not significant) during the first half of the fatigue test but rose more rapidly ($P < 0.05$) thereafter (Fig. 4*A*). At the endurance limit for the 25 % MVC test, the average EMG activity was enhanced significantly by 56 ± 8.8 % ($P < 0.001$). In contrast, the rise in EMG amplitude for the 50 % MVC test occurred more rapidly and it reached 127 ± 9.5 % ($P < 0.01$) of the initial value at about two-thirds of the endurance limit and then decreased slightly thereafter (Fig. 4*A*). In both sustained submaximal contractions, the EMG amplitude recovered to near its initial value after 5–10 min of rest. In contrast, no significant change (−4.6 ± 1.9 %; $P > 0.05$) occurred during the intermittent contractions and the recovery period (Fig. 4*A*).

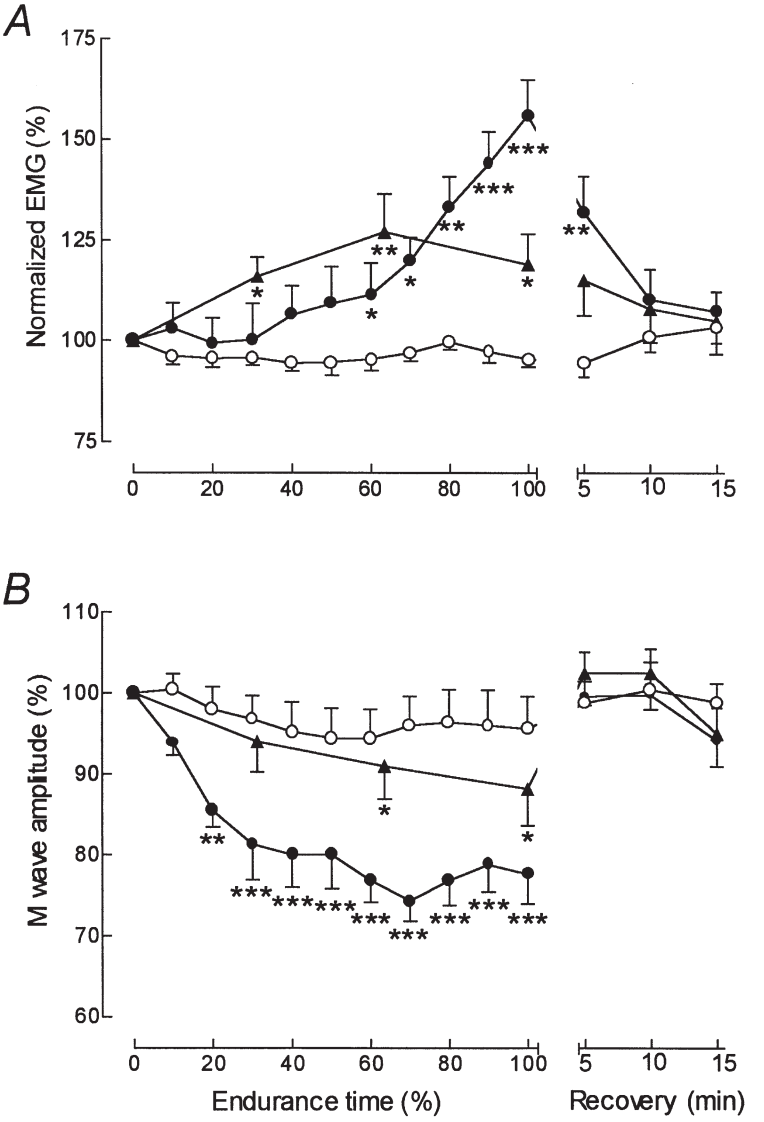

Figure 4. Changes in voluntary EMG and M-wave amplitudes during submaximal contractions and recovery

A, average EMG (means ± S.E.M.; $n = 10$) obtained during sustained contractions at 25 % (●) and 50 % MVC (▲), and during intermittent contractions at 25 % MVC (○) as a function of endurance time. All data were first normalised to the corresponding M-wave amplitude and thereafter expressed as a percentage of the pre-fatigue value. *B*, M-wave amplitude (means ± S.E.M.; $n = 10$), normalized to the pre-fatigue value during the three fatigue tests as a function of endurance time. Significant differences from pre-fatigue value : * $P < 0.05$; ** $P < 0.01$; *** $P < 0.001$.

Changes in maximal force and associated EMG after fatigue

MVC force declined by 34.2 % ($P < 0.001$) at the end of the sustained 25 % MVC test and by 26.2 % ($P < 0.01$) at the end of the sustained 50 % MVC test (Table 2). MVC force was not different at the end of the intermittent test (−4.5 %; $P > 0.05$). Similarly, the averaged EMG associated with the MVC was reduced after the sustained contractions at 25 % (−44.2 %; $P < 0.001$) and 50 % MVC (−35.2 %; $P < 0.01$) but not for the intermittent contractions (−6.4 %; $P > 0.05$; Table 2). These changes returned to control values within 5 min of recovery.

DISCUSSION

In a prior study on sustained MVCs, the findings suggested that metabolite accumulation in the muscle provoked motoneurone reflex inhibition and induced an increase in the descending supraspinal drive that compensated for the loss of excitation from the peripheral afferents onto motoneurones (Duchateau & Hainaut, 1993). Long-duration submaximal contractions appear to involve greater neural adaptations (Lloyd *et al.* 1991; Sacco *et al.* 2000), but it is unknown if the metabolic changes that occur during sustained submaximal and maximal contractions (Béliveau *et al.* 1991; Vestergaard-Poulsen *et al.* 1992) induce different reflex adaptations. The present study on reflex responses (H and LLR) demonstrates that fatiguing contractions sustained at 25 and 50 % MVC, but not an intermittent contraction at 25 % MVC, involve changes that depress the amplitude of the reflexes. In addition, short- and long-latency reflexes experienced differential effects during sustained efforts, which depended on the intensity, and hence the duration, of the contraction.

In the present study, only the two sustained contractions induced fatigue as indicated by the decrease in MVC force and EMG. In addition, the increased EMG activity during the submaximal fatigue tasks, which has been related to an increase in the central drive (Bigland-Ritchie *et al.* 1986*a*; Fuglevand *et al.* 1993; Garland *et al.* 1994; Löscher *et al.* 1996*a*), was only observed during the sustained contractions. In agreement with other studies (Fuglevand *et al.* 1993; Sacco *et al.* 2000), greater fatigue was associated with the longer duration contraction (25 % MVC). The likely explanation for the greater fatiguability of submaximal sustained contractions is the accumulation of metabolites (Béliveau *et al.* 1991;Vestergaard-Poulsen *et al.* 1992), which is probably enhanced by muscle ischaemia (Edwards *et al.* 1972; Crenshaw *et al.* 1997). This was not the case in our experiments during intermittent contractions at 25 % MVC because exhaustion was not reached (Bigland-Ritchie *et al.* 1986*a*; Moussavi *et al.* 1989).

Because M-wave amplitude decreased in our experiment during the two sustained contractions, an observation that is in agreement with others (Thomas *et al.* 1989; Fuglevand *et al.* 1993), voluntary and reflex EMG amplitudes were normalised as a function of the peak size of the M-wave. Such a procedure allows the exclusion of contamination of fatigue-induced changes of the voluntary and reflex neural activities by peripheral neuromuscular adaptations. Changes in M-wave amplitude during fatiguing contractions at submaximal forces appear to be related to impairment of neuromuscular propagation, largely due to alterations in neuromuscular transmission and variation in the amplitude of muscle fibre action potentials (Fuglevand *et al.* 1993; Jones, 1996). The relative importance of these alterations depends on the pattern of activation (Wise *et al.* 2001).

Although the non-linear summation of motor unit action potentials impairs the utility of using EMG magnitude as an index of muscle activation (Day & Hulliger, 2001), the increase in EMG activity during sustained submaximal contractions is usually considered to indicate an enhancement of the central drive (Bigland-Ritchie *et al.* 1986*a*; Fuglevand *et al.* 1993; Garland *et al.* 1994; Löscher *et al.* 1996*a*). This increase in the intensity of the central drive results in the recruitment of motor units and modulation of discharge rate (Bigland-Ritchie *et al.* 1986*a*; Enoka *et al.* 1989; Maton & Gamet, 1989; Carpentier *et al.* 2001) and contributes to the maintenance of a constant contraction force. In the present experiments, sustained contractions showed an increase in EMG amplitude that was greater during the fatigue test at the lowest force level (25 % MVC). Although the increase in EMG magnitude can be partly related to increased Na^+–K^+ pump activity (Hick & McComas, 1989), it is more likely that our observation is mainly explained by enhanced motor unit activity. Because motor unit recruitment is complete at about 50 % MVC in intrinsic hand muscles (Milner-Brown *et al.* 1973; Carpentier *et al.* 2001), the increase in EMG activity during sustained contraction at 50 % MVC must be due to rate coding. In the sustained contractions at 25 % MVC, both motor unit recruitment and rate coding would have contributed to the increase in EMG. Although the enhanced central drive compensates for a decrease in output of the activated motor units, the underlying mechanisms remain an open question. In this context, the study of the short- and long-latency reflexes provides insight into the functionality of the neural circuitries during fatigue (Duchateau & Hainaut, 1993).

The decrease in the amplitude of the short-latency or H reflex, which was only observed during sustained effort at 25 % and 50 % MVC, was not significant before 30 % of the endurance time. Because the decrease in the H reflex was observed when the EMG was relatively constant (see Fig. 4*B*), the decline in the amplitude of the H reflex was probably due to a decrease in transmission along the neural elements between the site of nerve stimulation and the motoneurones that responded to the stimulus (Duchateau & Hainaut, 1993; Kuwabara *et al.* 2002).

Despite an increase in the central drive during a sustained contraction, however, some motoneurones may have been derecruited and thus contributed to the decrease in the H reflex amplitude. Such motor unit deactivation during submaximal fatiguing contractions has been reported previously (Garland *et al.* 1994; Peters & Fuglevand, 1999; Carpentier *et al.* 2001).

The similar decrease in the normalised amplitude of the H reflex for the two levels of contraction, despite the greater increase in EMG for the sustained contraction at 25 % MVC compared with contraction at 50 % MVC, seems paradoxical. However, the H reflex reflects changes in only a fraction of the motoneurone pool (mainly low-threshold ones), whereas the voluntary EMG reflects the change in activation of the entire muscle. The observation of a similar decrease in H reflex amplitude after a sustained maximal contraction of short duration (60–80 s; see Duchateau & Hainaut, 1993; Fig. 5) suggests that either the responding motor units received comparable levels of inhibitory feedback or that the neural alterations in the spinal circuitry reached a saturation level.

Because of the rather slow time course of the decrease in the amplitude of the H reflex and the absence of recovery during ischaemic conditions (Bigland-Ritchie *et al.* 1986*b*; Garland & McComas, 1990; Duchateau & Hainaut, 1993), it has been suggested that this inhibition is induced by metabolic and chemical processes in the muscle that could be mediated by small-diameter group III and IV afferents from the fatigued muscle (Kaufman *et al.* 1983; Sinoway *et al.* 1993). The present work is consistent with this hypothesis. It has been shown that metabolic and chemical changes are induced during sustained submaximal (30–40 % MVC) contractions (Béliveau *et al.* 1991; Vestergaard-Poulsen *et al.* 1992), but not during intermittent contractions at a similar force level (Bigland-Ritchie *et al.* 1986*a*; Moussavi *et al.* 1989). Our observation that changes in reflex activities during sustained submaximal contractions returned to control within 5 min are consistent with the time needed to remove the accumulated metabolites from the fatigued muscle (Bigland-Ritchie *et al.* 1986*b*; Garland & McComas, 1990).

The normalised amplitude of the LLR decreased significantly during the two sustained submaximal contractions (25 and 50 % MVC), but not during intermittent contractions. Although there was a tendency toward a small decrease of LLR after a sustained MVC of 60–80 s, this decline was not statistically significant (see Fig. 4 in Duchateau & Hainaut, 1993). It has been suggested that the H reflex and LLR evoked by such electrical stimulation have the same origin and are both transmitted by Ia fibres, but that the LLR traverses a transcortical pathway (Deuschl *et al.* 1985; Mariorenzi *et al.* 1991). In the short-duration sustained MVC, the absence of an alteration in the LLR despite concurrent depression of the H reflex suggests that the supraspinal drive compensated for a loss of excitation from peripheral afferents on the motoneurones (Duchateau & Hainaut, 1993). Under the conditions of the current study, a similar reduction and time course in the LLR and the H reflex amplitudes during the sustained contraction at 25 % MVC suggests that this enhanced supraspinal drive was not maintained during long-lasting contractions. In contrast, the H reflex amplitude decreased to a greater extent than the LLR during the sustained 50 % MVC test, suggesting that the depression of the LLR cannot be due solely to a lesser excitability of the motoneurone pool. The observation that the decrease in the amplitude of the LLR was greater for higher intensity contractions (Fig. 5) is consistent with the hypothesis of a progressive reduction in the enhancement of the supraspinal drive with the duration of effort. Another possible explanation, however, is that the reduction in the amplitude of the LLR was related to a decline in transmission along the reflex pathway (Kuwabara *et al.* 2002). Regardless of the underlying mechanism, the concurrent decrease in LLR amplitude and increase in the voluntary drive is consistent with a suboptimal activation of some motoneurones in the pool during fatigue (Gandevia *et al.* 1996; Gandevia, 2001), probably low-threshold units (Carpentier *et al.* 2001).

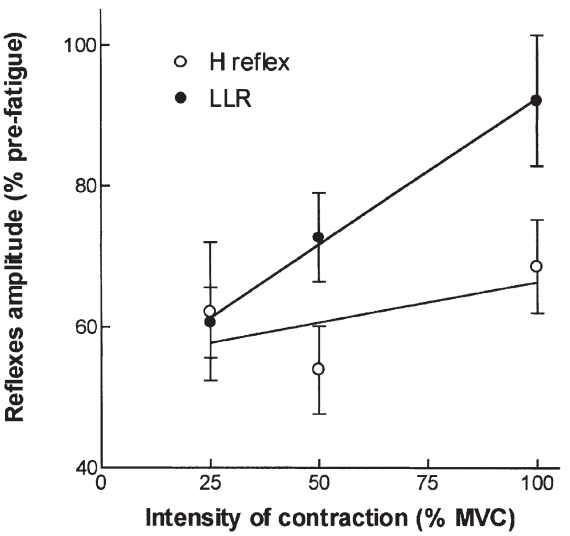

Figure 5. Changes in reflexes amplitudes as a function of the contraction intensity during the sustained fatigue tests

Data (means ± S.E.M.) for 25 % and 50 % MVC are from the present study, whereas those of the 100 % MVC are from a previous study (Duchateau & Hainaut, 1993). The equations of the linear regressions are : $y = 0.11x + 54.8$ ($r^2 = 0.35$) and $y = 0.42x + 50.8$ ($r^2 = 0.99$) for the H reflex and the LLR, respectively. The regression lines indicate a significant ($P < 0.001$) association between the changes in reflex amplitude and the intensity of the contraction for LLR but not for the H reflex ($P > 0.05$).

Our data, which were recorded during submaximal contractions, confirm previous reports that short- and long-latency reflexes are modulated differently during fatigue (Balestra *et al.* 1992; Duchateau & Hainaut, 1993). Moreover they indicate that during submaximal contractions, the central drive is only intensified during sustained contractions (see also Fuglevand *et al.* 1993; Garland *et al.* 1994; Löscher *et al.* 1996*a*) and not during intermittent contractions at 25 % MVC. Such enhancement of the central drive, possibly related to metabolic and chemical changes in the muscle, temporarily compensates for the decline in excitation from spinal and supraspinal reflex afferents onto the motoneurones.

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