Effects of fatigue on the stretch reflex in a human muscle

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Summary

The effects of fatigue on the electromyographic (EMG) reflex activities were compared during sustained voluntary contractions and contractions evoked by electrical stimulation (30 Hz) in the human first dorsal interosseus (FDI). Short latency (SL), medium latency (ML) and long latency (LL) reflex responses to a ramp-and-hold stretch of the muscle were recorded and analysed in 27 healthy subjects of both sexes. The amplitude of the reflex components was normalized as function of the amplitude of the surface action potential (SAP) recorded in response to the supramaximal stimulation of the motor nerve.

The results indicate that for a similar reduction of force, SL and ML are significantly reduced after fatigue induced by voluntary contractions but they are not when the fatigue test is performed by electrical stimulation at the motor point. In voluntary fatigue experiments, the LL component showed no significant decrease below control values, but an enhancement was observed during electrically evoked contraction. This enhancement remained above control values for at least 15 min during the recovery period, whereas SL and ML decreased returned to control within 5 min after the fatigue tests. The electrical stimulation applied to the skin overlying the FDI at an intensity lower than the motor threshold did not affect SL and ML, but enhanced LL for about 15 min. On the contrary, the anaesthesia of the skin overlying the FDI induced a decrease in LL without significant change of SL and ML.

It is concluded that muscle reflex fatigue is present during sustained voluntary contractions and decreases SL and ML responses to quick stretches. The different behaviour of LL suggests that the long latency reflex activity is a complex response mediated by different peripheral afferents.

Key words: Electromyography; Muscle reflexes; Fatigue

In movements controlled by reflexes, short and long latency electromyographic (EMG) activities were proposed to contribute to the regulation of muscle stiffness (Marsden et al. 1976; Houk 1978). There is a consensus that Ia afferents are involved in short latency EMG activity (SL or M₁) (Matthews 1984), but the aetiology and significance of the long latency EMG responses remain exciting topics of discussion. It has been proposed that the long latency responses (M₂—M₃) result from muscle spindle oscillation (Hagbarth et al. 1981), or are transmitted by other spinal (Gottlieb and Agarwal 1980) or supraspinal (Desmedt and Godaux 1978; Lee and Tatton 1978; Marsden et al. 1978) pathways. It has also been suggested that long latency responses originate totally or partly from other receptors such as cutaneous (Darton et al. 1985; Matthews 1989) or cutaneous and joint receptors (Marsden et al. 1985).

The study of fatigue has proved to be an interesting approach to the understanding of muscle contraction (for reviews, see Bigland-Ritchie and Woods 1984; Edwards 1984; Hainaut and Duchateau 1989). The effects of fatigue on the EMG reflex activities have been studied mostly during SL responses (Hakkinen and Komis 1983) and limited data are available on the effects of fatigue on the long latency EMG complex, which is considered as a whole by some authors (Desmedt and Godaux 1978; Darton et al. 1985; Noth et al. 1985) while others distinguish different components (Lee and Tatton 1978; Marsden et al. 1978; Matthews 1987). Among these, at least two, medial latency (ML) and longer latency (LL), activities are discussed (cf., Gollhofer et al. 1987).

The present investigation compares the effects of fatigue on short and long latency EMG reflex activities after voluntary contractions and contractions evoked by electrical stimulation at the motor point of the human first dorsal interosseus (FDI).

Methods

Subjects

The experiments were carried out on the FDI muscle of 27 healthy subjects of both sexes. They were
between 17 and 34 years old. All were fully informed regarding the nature of the experimental method and agreed to participate in this investigation.

Mechanical stimulation and recording

The subject’s arm was placed on a horizontal board with the palm of the hand turned down. The last 3 fingers were strapped together in flexion and the thumb was immobilized in abduction and extension by a per-
sperse plate covered with soft rubber. The index was fully extended, and its middle part was held firmly in a clamp attached to an electromagnet. The stretches applied were ramp-and-hold movements. The ramp duration and amplitude were respectively 15 msec and 9 mm, corresponding to a velocity of 600 deg/sec. During the recording, the subject was required to exert an abduction torque (10% of his maximal voluntary isometric contraction before the fatigue tests and at any particular time during the recovery from fatigue) against the stretcher in order to maintain a steady mean level of EMG activity (cf., Davis et al. 1985). The subject was aided in this task by watching the EMG signal on an oscilloscope. The stimuli were deliv-
ered at intervals of 2–3 sec so that the initial position was restored at least 1.5 sec before the next stimulus. In general, 16–32 responses were averaged.

The index movement was monitored by a displace-
ment transducer (Kulite, Type AC 50). An accelerome-
ter (Brüel and Kjaer, Type 4375) linked to the connec-
tion between the electromagnet and the finger was used to trigger the sweep of the averager ( Nicolet, Type 4094C). The isometric muscle torque was recorded by means of an electro-mechanical strain gauge transducer (Kulite, Type TC 100).

Electromyogram (EMG) recording

The EMG of the FDI obtained in response to mechanical and electrical stimuli was recorded by a pair of surface electrodes, one fixed over the muscle motor point and the other over the metacarpo-
phalangeal joint of the index. The signal was AC amplified (1000–2000 × ), filtered (bandpass: 10 Hz–5
kHz), full wave rectified and averaged. The maximal direct motor response or muscle surface action potential (SAP) was obtained by delivering supramaximal pulses of 0.2 msec duration to the ulnar nerve at the wrist via two steel needle electrodes (cf., Hainaut and Duchateau 1989).

Muscle fatigue and testing procedure

Muscle fatigue was induced either by a sustained maximal voluntary contraction (MVC) or by an electrically evoked one. In the latter condition, the muscle was electrically stimulated through the EMG recording electrodes, with supramaximal square pulses at a frequency of 30 Hz. In both fatigue tests, the contraction was interrupted when the force fell to 50% of its maximal value. The reflex activities and the SAP were recorded before and after the fatigue tests. To avoid recovery of the EMG activity during the post-fatigue recording, a cuff was inflated around the arm for 1 min just before the end of the fatigue tests (250 mm Hg) (Milis 1982). The cuff was then removed and recovery was tested every 5 min for at least 15 min.

In complementary experiments, the skin overlying the FDI was electrically stimulated just below the motor threshold via the same electrodes used to record the EMG reflex responses. In another series of experiments 2 ml 1% lidocaine (without vasopressin and noradrenaline) was subcutaneously injected into the same area of the skin as described above and control EMG reflex responses were compared before and during 60 min following the injection.

During the experiment, the skin temperature was continuously controlled and maintained at ± 35°C.

Measurements and statistics

All data were recorded on a digital oscilloscope (Nicolet, Type 4094C) before storing on floppy disk. On the EMG trace, the following were measured for each reflex component and SAP: (1) latency as the time between the stimulus and the beginning of the EMG response; (2) duration; (3) peak amplitude; and (4) area of the EMG response. The latency and the duration of the reflex responses were determined by visual observation of the EMG trace on the oscillo-
scope by means of a cursor. The size of each EMG component was defined as the distance between the peak amplitude of the EMG reflex response and the mean background EMG activity computed during the 50–100 msec preceding the onset of the mechanical stimulus (excluding the artifact related to the electo-
magnet). The area of each reflex component was com-
puted by integrating the EMG response above the mean background activity. In order to exclude peripher-
al neuromuscular alterations due to fatigue from changes of the sensitivity of the reflex loop, each reflex response amplitude was normalized as a function of the peak size of the muscle SAP. This approach showed similar results to those observed by the expression of the reflex response amplitude as a percentage incre-
ment above the basal level of EMG activity. The data were analysed by a Friedman’s 2-way analysis of var-
iance by ranks or by Student’s t test when appropriate.

Results

EMG responses in control

When the FDI was stretched by an imposed stan-
dard abduction of the second finger, two EMG re-
sponses of different latencies were induced (cf., Fig.
Fig. 1. EMG responses of FDI to imposed finger displacement (A); before (B) and after (C) muscle fatigue induced by electrical stimulation (15 sec at 30 Hz) in one subject. During the recording, the finger maintained a voluntary constant abduction force equal to 10% of its maximum. Each rectified EMG trace is the average of 16 individual responses. SL: short latency; ML: medium latency; LL: long latency. The initial deflection of the EMG traces prior to the stretch is an artifact induced by the electromagnet. The displacement of the finger remained unchanged during fatigue.

1B). The first response had a mean onset latency of 30.9 ± 3.0 msec (mean ± S.D.; n = 22). This SL EMG activity was followed by a second response of longer duration (48.2 ± 6.6 msec vs. 16.8 ± 7.3 msec in SL) and of longer onset latency (46.2 ± 4.9 msec). In 17 of the 22 subjects this second EMG activity is clearly divided into two parts, i.e., a medial latency (ML) and a long latency (LL) burst. The LL EMG activity had a mean onset latency of 75.5 ± 8.8 msec. The mean durations of the ML and LL bursts were respectively 31.9 ± 8.2 msec and 16.2 ± 5.3 msec. In all the subjects, a later voluntary activity appeared with a mean onset latency of 119 ± 18 msec.

Table 1

<table>
<thead>
<tr>
<th>Latencies (msec)</th>
<th>SL</th>
<th>ML</th>
<th>LL</th>
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<tbody>
<tr>
<td><strong>Electrical stimulation</strong></td>
<td></td>
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<tr>
<td>Before (n = 10)</td>
<td>30.7 ± 2.6</td>
<td>47.6 ± 5.8</td>
<td>76.8 ± 11.0</td>
</tr>
<tr>
<td>After</td>
<td>31.0 ± 2.8</td>
<td>46.7 ± 6.7</td>
<td>75.9 ± 9.1</td>
</tr>
<tr>
<td><strong>Voluntary contraction</strong></td>
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<td></td>
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<tr>
<td>Before (n = 10)</td>
<td>31.5 ± 2.8</td>
<td>45.2 ± 3.9</td>
<td>74.8 ± 5.0</td>
</tr>
<tr>
<td>After</td>
<td>30.3 ± 3.5</td>
<td>44.7 ± 3.7</td>
<td>74.5 ± 5.6</td>
</tr>
</tbody>
</table>

**Effects of fatigue.**

In voluntary and electrically evoked contractions, the mean durations of the fatigue tests were 108 ± 25 sec and 117 ± 32 sec (mean ± S.D.) respectively. These durations were not significantly different (P > 0.05, Student’s t test). After fatigue, the EMG responses were not as well individualized as in the control muscles, but the latencies and durations of the different components did not change significantly (P > 0.05; cf. Fig. 1 and Table I). On the other hand, the amplitude of SL and ML EMG components triggered by the stretch showed an overall reduction after both fatigue tests, whereas LL did not and was found to be enhanced in 5 subjects after electrically evoked contrac-
tions. This observation is illustrated in Fig. 1 with respect to one subject after fatigue induced by electrical stimulation. Fig. 2A illustrates the mean changes of the crude value in the 2 reflex components in all the subjects after both fatigue tests. The amplitudes of SL and ML responses were significantly reduced in both tests whereas the LL response was not significantly different in the control and fatigued muscles. The comparison of the two fatigue tests indicated a larger (P < 0.05) effect of voluntary contractions as compared to electrically triggered contractions in the SL and ML EMG activities. Similar results were obtained when the area of each reflex component was measured: the areas of SL, ML and LL were reduced to 58.0 ± 6.9, 62.8 ± 4.7 and 84.4 ± 5.8% and to 77.7 ± 8.1, 78.1 ± 9.6 and 91.9 ± 10.4% respectively after voluntary and electrically evoked contractions.

After voluntary contractions and electrically evoked contractions, the muscle SAP amplitude recorded in response to a supramaximal electrical pulse was reduced to 80.9 ± 7.1% and to 63.5 ± 7.0% respectively in the fatigued muscles. These mean decreases in SAP amplitudes were significantly (P < 0.001) different in the two cases.

Whereas the LL reflex component was not significantly different from control, the comparison of the normalized amplitude of the EMG reflex components as a function of the muscle SAP amplitude (cf., Fig. 2B) showed a significant decrease in SL and ML responses after voluntary fatigue. Conversely, after electrically evoked fatigue, SL and ML were not significantly different from control whereas LL was found to be enhanced.

After both fatigue tests, the muscle SAP and the normalized amplitude of the SL and ML EMG components returned to control values within 5 min (Fig. 3A).

On the other hand, after electrically evoked fatigue, the LL response remained enhanced during the 15 min recovery test (Fig. 3B).

**Cutaneous stimulation and anaesthesia**

In 6 subjects, electrical stimulation (30 Hz for 60–100 sec) of the skin in the area of the muscle belly and below the motor threshold did not significantly change SL and ML (−4% ± 7% and +7% ± 5% respectively) but increased (+47% ± 17%) the LL component (Fig. 4A). This enhancement of the LL response reached its maximum within 1–5 min following the electrical stimulation of the skin and then decreased slowly 13 min after the interruption of stimulation the LL component was still 19% ± 15% above the control values.

In one subject, a subcutaneous injection of 2 ml 1% lidocaine, in the same area that was previously stimu-
lated, decreased after 5 min the control LL response by 48% with little change of the SL and ML responses (Fig. 4B). After 60 min, the effect of the lidocaine was completely dissipated.

Discussion

The major finding of the present investigation, which compares muscle fatigue induced by voluntary contractions and by electrical stimulation of the muscle is the observation of a decrease of SL and ML during voluntary fatigue without significant change of LL and of enhanced LL without significant change of SL and ML during electrically induced fatigue. The amplitude of an EMG reflex activity can be modified by: (1) a change in the excitability of the reflex loop; (2) a change in the synchronization of the fibre action potentials (APs). In our experiments the second possibility should not play a key role because the latencies and the durations of the reflex components and their total duration were not significantly different in control and during fatigue. In previous studies (Darling and Hayes 1983; Gollhofer et al. 1987), the effects of fatigue on reflex responses were discussed on the basis of changes in crude EMG. This approach does not enable changes to be distinguished in the excitability of the reflex pathway from peripheral neuromuscular processes because the muscle SAP is also sometimes reduced during fatigue performed by MVC or electrical stimulation (Müller-Brown and Miller 1986; Hainaut and Duchateau 1989). When the EMG reflex responses are normalized as functions of the muscle SAP evoked by supramaximal stimulation of the motor nerve, it is possible to approach the effects of fatigue on the sensitivity of the reflex loop more specifically.

The SL reflex component has been studied for a long time and it is well known that it involves the myotatic segmental loop. The observation that the normalized SL reflex response decreases during voluntary fatigue, but not in fatigue induced by electrical stimulation, suggests that the recruitment of the MNs is reduced during sustained voluntary contractions in response to the standard stretch. This point is coherent with the results of Kukulka et al. (1986) and Garland and McComas (1990) who report a reduction in the H reflex amplitude during fatigue. Decreased MN recruitment after fatigue may at first appear contradictory because it has been observed that repetitive stimulation increases the sensitivity of the muscle spindle (Nelson and Hutton 1985) and decreases the sensitivity of the Golgi tendon organs (Hutton and Nelson 1986). In fact, these authors stimulated the muscle at 100 Hz for short durations (9−15 sec) and these experimental conditions are not comparable with our fatigue tests performed for 10 times as long and at a lower frequency closer to the motor unit discharge frequency during voluntary contraction (Duchateau and Hainaut 1990). Our finding of a decrease of SL during MVCs appeared at first in line with the observation of a decreased muscle spindle activity at low pH (cf., Fukami 1988) and the suggestion that sustained MVC induces fatigue of the intrafusal muscle fibres (cf., Bongiovanni and Hagbarth 1990). In recent experiments (n = 6, unpublished) using the Deuschl technique in human abductor pollicis brevis (Deuschl et al. 1985), we observed a 31 ± 5% decrease of the normalized amplitude of the H reflex (as a function of SAP) after sustained MVC performed in identical conditions to the fatigue experiments. This decrease, which is nearly identical to the decrease of SL after voluntary fatigue (28 ± 6%), thus suggests that spindle fatigue is not the prime cause of SL decrease in our experimental conditions and that it should be related to a change in MN excitability. A possible explanation for a change in excitability is that sustained MVC induces exhaustion of the central drive, but in fact, when the cuff is maintained after the fatigue test for 3−5 min, SL does not return to control at a time when the central drive should have recovered from fatigue (cf., Garland et al. 1988). Thus changes of SL appear to be more closely related to changes of peripheral mechanisms such as inhibitory afferents from group III and IV fibres (cf., Bialland-Ritchie et al. 1986; Garland and McComas 1990) and/or presynaptic inhibition, which could lower the frequency of discharge of α MNs and reduce their recruitment during the stretch. The absence of SL decrease during electrically induced fatigue cannot be directly discussed on the basis of our experimental results, but an interpretation is that the electrical stimulation favours the recruitment of large motor units (MUs) via cutaneous afferents (Garnett and Stephens 1980; Kanda and Desmedt 1983) and their SAP of larger amplitude masks a decreased MU recruitment.

Long latency reflexes have been intensively studied, but still are controversially interpreted. They are discussed as a unique complex (Desmedt and Godaux 1978; Darton et al. 1985; Noth et al. 1985) or as different long latency loops originating from different receptors or involving different routes (Lee and Tatton 1978; Marsden et al. 1978; Matthews 1987, 1989). In our experiments, ML decreases during sustained voluntary contractions whereas LL remains at control level, but in electrically induced fatigue, ML remains at control level and LL is now enhanced. These observations suggest that ML and LL reflex components are different entities in the long latency complex. The significant decrease of ML during voluntary fatigue without change during electrically evoked fatigue could at first indicate that ML is controlled by the central drive, but when the cuff is maintained after the fatigue test (cf. above), ML (as well as SL) does not return to
control at a time when the central drive should have recovered from fatigue. Thus changes of ML, as well as those of SL, appear to be more closely related to changes of peripheral mechanisms, as discussed above. Anyhow, the observation that the ML component of the long latency complex behaves like SL in our fatigue experiments and is of longer latency supports the proposition of a similar origin (muscle spindle) for SL and ML and of different pathways (cf. also Desmedt and Godaux 1978; Lee and Tatton 1978; Marsden et al. 1978).

The suggestion that ML and LL are different parts of the long latency complex is also supported by the finding that ML returns to control values in about 5 min while LL is still above control 15 min after electrically induced fatigue. The enhancement of the LL component during electrically induced fatigue could be explained by excitatory afferents from cutaneous receptors. This point of view is coherent with the finding that electrical stimulation of the digital nerve endings and of the skin evokes a long latency (above 50 msec) reflex component (Jenner and Stephens 1982; Deuschl et al. 1985) and also with the observation of a long latency (60–80 msec) reflex response induced by the activation of tactile receptors (Johansson and Westling 1987). More recently, Matthews (1989) also provided experimental evidence, by cooling the peripheral pathway, that fast cutaneous afferents contribute to the long latency stretch reflexes. In our experimental conditions, a long lasting (15 min) enhancement of the LL component was observed without any significant change in SL and ML, when the skin was electrically stimulated below the motor threshold at the belly of the muscle. On the other hand, the subcutaneous injection of anaesthetic reduced LL drastically and had only little effect on SL and ML.

It is concluded that during sustained voluntary contractions of the human FDI, fatigue of reflex components is present and induces decreases of SL and ML via an inhibition of α MN excitability. The different behaviour of LL during fatigue suggests that the long latency reflex complex is composed of at least two entities which are mediated by different peripheral afferents.

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