Change of α motoneurone excitability during fatigue by voluntary contraction

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It has been reported in a previous paper that the amplitude of the short and medium latency reflex components recorded in response to a sudden stretch in the human first dorsal interosseous are reduced after fatigue by voluntary contraction (Balestra et al. 1992). These changes can be the result of: (1) reduced spindle output (Macefield et al. 1991) and/or (2) decreased excitability of α motoneurones (MN) through inhibitory muscle afferences (Woods et al. 1987; Garland and Mc Comas 1990). The present study was designed to obtain further informations on the mechanisms whereby reflex activities are impaired during fatigue.

Experimental methods

Ten healthy subjects (9 males and 1 female) ranging between 24 and 44 years old took part to this investigation. The Hoffmann reflex (HR) was recorded during voluntary contractions (10-15% of MVC) of the thenar muscles by using the method introduced by Deuschl et al.(1985). This method consists of the electrical stimulation of the median nerve at the wrist with a stimulus strength set near the threshold response of the motor fibres. The electromyogram (EMG) of the abductor pollicis brevis was recorded by means of surface electrodes in a belly-tendon fashion. The signal was amplified (1000x), filtered (10-5000 Hz), rectified and averaged (64 sweeps). In order to exclude peripheral neuromuscular alterations due to fatigue in the interpretation of the results, the HR amplitude was normalized as a function of the maximal M wave amplitude and also as a percentage increment above the basal level of EMG activity. Muscle fatigue was induced by a sustained MVC which was interrupted when the force was reduced to 50% of its maximal value. To avoid any recovery of the EMG activity during the post-fatigue recording, a cuff placed around the arm was inflated (250 mmHg) just before the end of the fatigue test.

Results

In control condition, the mean (± SD, n = 10) latency and duration of HR are 29.6 ± 1.6 ms and 11.0 ± 1.3 ms respectively. After the fatiguing contraction (mean duration : 101 ± 16.1s), the duration of HR is significantly increased by 21.2 ± 14.2% ($P < 0.005$) whereas its latency is not statistically modified. HR amplitude is found to be drastically reduced by 51.3 ± 13.2% of the control value. When HR amplitude is normalized as a function of the M waves amplitude or of the basal EMG activity, its value is significantly ($P < 0.005$) reduced to 69.8 ± 21.3% and 71.8 ± 13.2% respectively. These changes persist as long as the cuff is inflated but return to control values within 5 min after the ischemic block is interrupted.

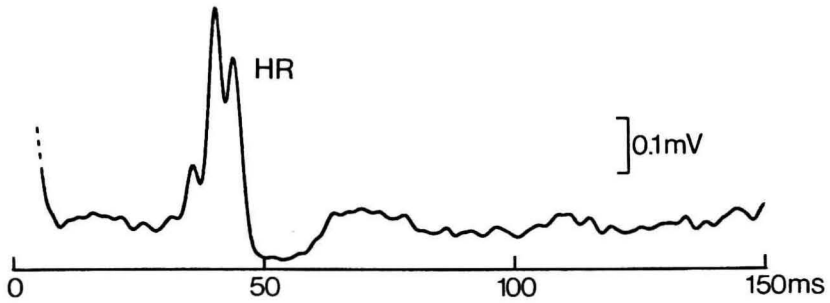

Figure 1. Typical example of an HR response in one subject.

Discussion

The main finding of this work is that the amplitude of HR is drastically reduced after fatigue by MVC. After normalization of the reflex amplitude, in order to avoid changes of peripheral neuromuscular alterations due to fatigue in the interpretation of the results, a significant deficit of about 30% persist which can only be related to decreased α MN excitability. This observation of a depressed excitability of the α MN pool after fatigue is in line with the recent results obtained by Garland and McComas (1990) on the soleus after electrically evoked fatigue tests. In the present study, the magnitude of the reflex inhibition is very similar to that recorded for the short latency stretch reflex after a comparable fatigue test (Balestra et al, 1992). Although this comparison is made in two different hand muscles, it suggests that spindle fatigue should not be the prime cause of the stretch reflex decrease after fatigue but that most of the reduction can be explain by inhibition of the α MN pool.

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