TERMINATION OF UPPER LIMB MOVEMENT BY CUTANEOUS AFFERENTS

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INTRODUCTION

Descending cortical signals commanding target-reaching movements guided by visual inputs impinge on spinal neurons located between C2 and C4 level (1,2). These neurons project to motoneurones of all muscles at the shoulder, elbow and wrist, and receive inhibition and excitation from cutaneous and muscle afferents in the moving limb (3).

These afferents usually carry feedback information. Those from the skin field close to the muscular region innervated by the central pathway are inhibitory. This peculiarity might be interpreted as being active because favouring termination of movement. In other words, cutaneous sensitivity activated by contact with the reached object would inhibit the motoneuron activity. On the other end, if this inhibitory pathway is controlled from the sensorymotor cortex we should expect that cortical facilitation be stronger at the end of movement than at the beginning.

In the present experiment, cutaneous inhibition to spinal neurons was compared at the onset and offset of a visually guided elbow extension movement. EMG activity of the triceps brachii was monitored for the effects of cutaneous stimulation.

MATERIALS AND METHODS

Experiments were performed on 8 normal subjects, mean age 21.8 years (5 women and 3 males). All subjects were informed about the procedure and gave their written consent. Surface EMG recording was taken from

the triceps brachii during tonic contraction and during visual tracking movements of elbow extension lasting 1 s.

The EMG activity was rectified and averaged against the cutaneous stimulation which was a train of 3 shocks (300 Hz) applied to the superficial radial nerve (SR). Trials with and without (control) stimulation were randomly alternated. Cutaneous stimulation was given during tonic contraction and either during the first (onset) or the last (offset) 100 ms of voluntary elbow extension, the amount of control EMG activity being about the same in the two situations. Because the Hoffman reflex (H-reflex) is usually too small in the triceps, the excitability of its motoneurone pool was tested by eliciting the tendon reflex. The tap was applied to the distal tendon of the muscle by an electromagnetic hammer. Reflex response was measured from peak to peak.

RESULTS

We establish at first whether superficial radial nerve stimulation inhibits the transmission of the descending excitation to triceps motoneurones. For this purpose, we investigated the effects induced by cutaneous stimulation on ongoing EMG activity. Figure 1 reports the effects of cutaneous stimulation on mean EMG and tendon reflex during tonic contraction. The responses conditioned by cutaneous stimulation are expressed as a percentage of their unconditioned value. The latency of the changes in EMG activity corresponds to a "central delay" calculated from the expected time of arrival of the cutaneous volley at the motoneurone pool. For the calculations, we evaluated the latency of the H-reflex plus about 5.5 ms (peripheral conduction times for cutaneous afferents). Open circles in figure 1 show that SR stimulations evoked clear inhibition in two periods (6-16 and 17-35 ms) with EMG reduction to about 60% of its control value at the 10 ms interval.

SR stimulation depresses the tendon reflex. The delay of the first EMG depression implies a spinal pathway, compatible with a C3-C4 spinal mechanism but, as previously suggested (4), also supraspinal mechanism could contribute to the second period of depression.

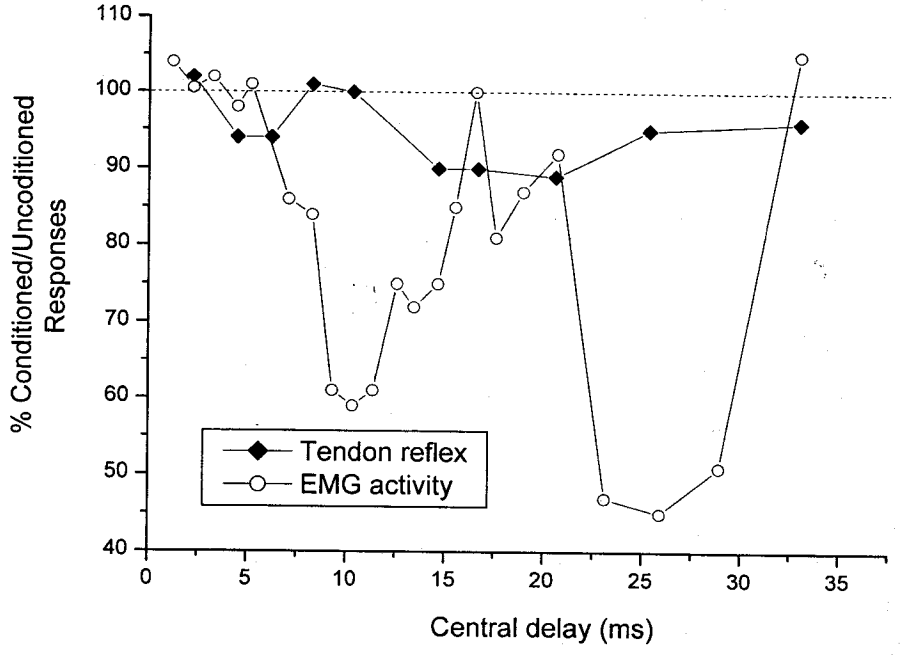

Figure 1- Changes in the EMG activity (O---O) and the tendon reflex of the triceps brachii (◆---◆) evoked by stimulation of the superficial radial nerve during triceps tonic contraction. Responses are expressed as a percentage of their unconditioned value. Each point represent the mean of 100 (O) or 10 (◆) points.

Fig. 2 illustrates the amount of cutaneous inhibition of triceps EMG occurring at the onset and at the offset of a reaching movement involving elbow extension. In each case, 300 trials (conditioned and unconditioned) were averaged and results obtained with cutaneous stimulation were expressed as a percentage of the control average EMG activity. At the onset, the depression occurred with a central delay of 5 ms, was moderate during its first 6 ms, reached its maximum at the 11 ms delay when the EMG was depressed to 55% of its control value and returned to normal at 30 ms.

At the offset, the depression had the same central latency and the same duration but was both significantly larger and much more abrupt, reaching a maximum at 8 ms where the EMG was depressed to 32% of its control value.

By contrast, the second part of the depression was similar (about 65% of its control) both at the onset and at the offset of the movement.

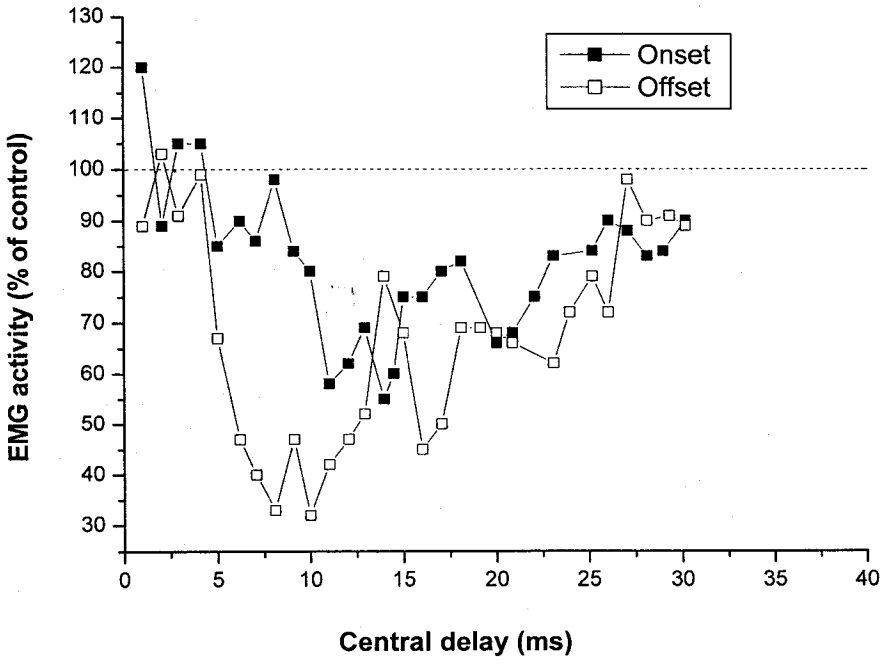

Fig. 2 - Cutaneous-induced changes in the voluntary EMG of the triceps recorded during the first (■) or the last (□) 100 ms of a visual-tracking movement of elbow extension. Each point represent the overage of 300 trials and the EMG conditioned by SR stimulation is expressed as a percentage of its unconditioned value.

DISCUSSION

In conclusion, during tonic contraction, at the onset and at the offset of a phasic isometric contraction SR stimulation had little effect on the

tendon reflex but depressed the ongoing EMG activity. Furthermore, this depression was more pronounced at the offset of the contraction. Possibly, this suggests that the depression of ongoing EMG activity reflects an inhibition of the descending excitation of triceps motoneuron at the spinal level. Being this hypothesis correct, the inhibition more marked at the offset of the movement suggests that this mechanism could be used to curtail a movement of target-reaching. As already emphasized (1), it would be a reasonable control strategy to delegate part of the termination of the movement to spinal chord mechanism, since termination must be one of the most difficult parameters of a movement for the brain to calculate. This might be of great relevance particularly if the movement meets an unexpected obstacle.

It can be speculated that it is advantageous that this integration takes place at a spinal neuron level and not directly at the motoneuron level. Of course, at the latter level the final inhibitory effect would be the same. However, because of the inertia related to the duration of the IPSPs in motoneurones, the movement could not start again before these IPSPs had vanished. For an inhibition at the premotoneuronal level the movement can start again, if necessary, immediately after activation of another excitatory pathway, since the motoneurones themselves are not inhibited.

We tested whether cutaneous afferents from the skin field close to an upper limb muscular region would carry information to spinal neurons at the onset or at the offset of a voluntary elbow extension movement lasting 1 s. We detected a depression of EMG activity both at onset and at the offset of the reaching movement but in the latter case depression was significantly larger and immediate. The marked depression of EMG activity suggests an inhibition, via spinal neurons, of the descending excitation to the motoneurones supplying the triceps brachii. This spinal control might be a very efficient mechanism for the termination of voluntary movement.

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KEY WORDS: triceps brachii, voluntary movement, spinal inhibition, EMG activity.

Lectured at the meeting held in Catanzaro on October 8, 1999.
Received: October 15, 1999; accepted: October 29, 1999.

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