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Short Communication

Is the Onset of the Premotion Silent Period Temporally Fixed to the Initiation of Phasic Discharge?

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We studied the timing of PSP of triceps in human subjects during ballistic elbow extension responding to visual stimulation. The latency of PSP, regardless of EMG-RT, was close to the shortest EMG-RT presented when PSP was absent. The time interval between the PSP onset and the phasic discharge was varied depending on the EMG-RT, and the PSP onset was not coupled to the initiation of phasic discharge.

INTRODUCTION

A transient cessation of activity (called premotion silent period, PSP) is observed on agonist EMG prior to voluntary bursts when a pre-tensed muscle is rapidly contracted. It is considered that PSP is the first overt change on EMG in making an intended movement. Although PSP can be observed in a self-paced movement as well as a reaction movement, the reaction time situation is of advantage to examining time course of PSP within the central process for execution of voluntary movement. The present study focused on the question of whether PSP is temporally coupled to the stimulus or to the onset of the EMG bursts.

Methods

The subjects were nine healthy volunteers, ranging in age from 27 to 43.

The ballistic elbow extension was performed as a reaction time task responding to a flashing light. The subjects were seated with the elbow supinating and resting on a table which was set before them and was beveled so that they could fully extend the elbow. The gravitational load (adjusted to 10% of the maximal voluntary force) was transmitted to the distal end of forearm through a lever which was fixed to a shaft of a gear (Fig. 1). After a warning (oral), the subjects pushed down the lever to hold elbow angle at 60 degrees. This preparatory position required the triceps activity to balance the external load. The response signal was presented 2–3 sec after completion of preparatory position. The subjects were instructed to extend the elbow as rapid as possible responding to the stimulus. Each subject has 60 trials at a minimum.

EMGs were recorded from the triceps and biceps muscles using surface electrodes, and were digitized with a sampling frequency of 2 kHz. The latency of onset of PSP and onset of phasic discharge were measured on the digitized records.

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Results

PSP was seen in all subjects, but the frequency of occurrence showed a large interindividual variation. The occurrence of PSP tended to increase with lengthening in EMG-RT. The mean EMG-RT in five subjects who showed higher frequency (2/3 or more) of PSP occurrence was 149.8 ± 22.57 msec, and significantly longer than that (104.6 ± 16.17 msec) in four subjects who showed lowr frequency of occurrence ($t=3.36$, $df=7$, $p<0.05$). The mean EMG-RT in subjects with lower frequency of PSP occurrence was rather near to the mean latency of PSP (95.6 ± 13.59 msec) in subjects with higher frequency of PSP occurrence ($t=0.91$, $df=7$, ns).

The temporal relationship between the onset of PSP and initiation of voluntary EMG bursts was examined for five subjects who presented enough number (2/3 of trials) of trials with PSP. Fig. 2 shows typical pattern of temporal relationship between PSP and initiation of voluntary EMG bursts. Trials were sorted in order of the latency of onset of phasic discharge (EMG-RT), and were synchronized on the response signal onset (time 0). PSP is presented as sparse area just before the most dense area of dots in Fig. 2. PSP was not found in the trials with the shortest EMG-RT. PSP occurred more frequently in the trials with longer EMG-RT. The duration of PSP was not constant among trials, but it lengthened with prolonging in EMG-RT. The duration of PSP distributed between 20 and 120 msec, and have high correlation coefficient with EMG-RT ($r=0.95$, $p<0.01$). On the other hand, the latency of onset of PSP regularly lay in the range of 90–120 msec in spite of variability in EMG-RT. Such papallelism that the onset of PSP prolonged with the onset of phasic discharge was not found. The onset of PSP was at level of the shortest EMG-RT (90 msec). The same tendency was observed in other four subjects who showed PSP in more than 2/3 of trials.

The comparison of variability confirmed that the duration of PSP was more variable than the latency of PSP. For all of five subjects, the coefficient of variation of the latency of PSP was smaller than that of the duration between the onset of PSP and phasic discharge. The significant difference between two variances was shown for three of five subjects by F-test.

Discussion

The main result of this study shows that PSP does not precede the initiation of voluntary EMG bursts with a constant time difference, and rather PSP tends to begin with a constant latency in spite of variability in EMG-RT. A constant latency might suggest a reflex mechanism. PSP, however, occurs in response to stimulus in various modalities and in self-paced movement. The sensory stimulus by itself cannot evoke PSP.

Task-related neurons in which the activity is mainly related to the triggering stimulus have been observed in the supplementary motor area and the cerebellum (Tanji & Kurata, 1982, Lamarre & Chapman, 1986). Responses of these neurons are observed only when the stimulus calls for a motor response, though these responses are time-locked to a stimulus. It is assumed that such neurons are involved at early stages in transforming a imperative sensory input into a motor command. Neuronal events which are involved in PSP initiation may have close time-relation with these responses. The variability in the time between the PSP onset and the phasic discharge suggests that PSP is produced by some purposeful needs rather than time difference passing separate hard-wired pathways.

There was a positive relation between the duration of PSP and EMG-RT in agreement with

Conrad et al (1983). Disagreement with Conrad et al exists however. We find that the latency of PSP is at level of the shortest EMG-RT, though Conrad et al have reported that the latency of PSP is far shorter than the EMG-RT. But they only compare their resulting PSP latency with the reaction time, perhaps mean reaction time, reported by other authors.

The latency of PSP in this study was longer than that reported by Conrad et al (1983). This is mostly due to difference in stimulus modality, that is difference between visual and auditory reaction signal. The latency of SMA and cerebellar dentate neurons as well as the conventional reaction time responding to visual stimulus are consistently longer than those responding to auditory stimulus.

PSP was absent when EMG-RT was very short. It consists with Yabe & Murachi (1976). The frequency of PSP occurrence was also low in subjects who had mean EMG-RT close to the latency of PSP. To shorten EMG-RT is disadvantage for PSP occurrence. On the other hand, PSP frequently occurs when the briefest force impulses are performed (Ghez & Gordon, 1987). The positive relation between the duration of PSP and the peak velocity of movement (and angular displacement) has been reported by Conrad et al. It is suggested that one of factors which induce PSP in reaction time situation is intention to execute rapid build-up of force rather than rapid initiation of movement.

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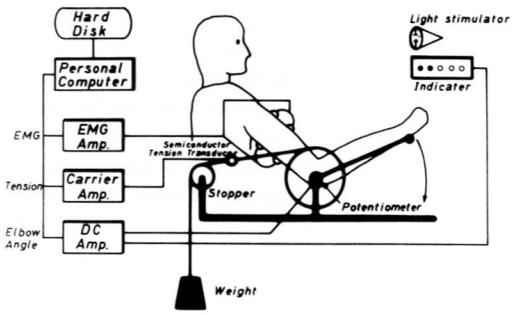

Fig. 1. Experimental arrangement. Subject is seated with the elbow flexed at 60 degree holding the gravitational load. A row of LEDs indicates the elbow angle so that the subject can flex the elbow at the requested angle. The reaction signal is presented by flashing light placed 1.5 mm away at eye level.

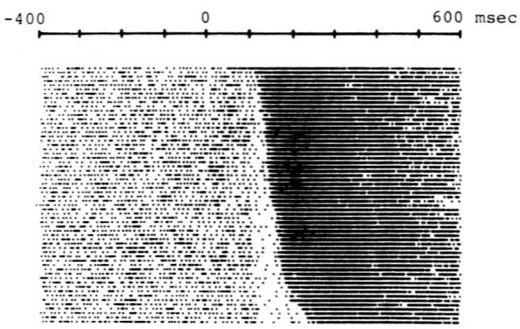

Fig. 2. Relation of the onset of PSP to EMG-RT within one subject. Trials are sorted in order of lengthening EMG-RT (top to bottom) and synchronized on the onset of stimulation. The rectified and integrated EMG of each trial is displayed ás a series of dots in which the density of dots is proportional to EMG activity.

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