Slow cord dorsum potentials elicited by descending volleys in man

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SUMMARY Human cord dorsum potential (CDP) was recorded from the posterior epidural space at the levels of cervical and lumbar enlargements, using epidural catheters as the recording electrodes, in 15 normal volunteers and six patients suffering from amyotrophic lateral sclerosis. In normal subjects the CDP elicited by descending volleys consisted of spike potentials followed by sharp negative and slow positive waves which were very similar to the P1, N1, and P2 potentials, respectively, of the CDP evoked segmentally. By contrast, in all patients with amyotrophic lateral sclerosis both the sharp negative and slow positive waves were absent in the CDP elicited by descending volleys, while the pattern of each component of the segmentally evoked CDP remained unchanged. Thus, the sharp negative and slow positive waves of human CDP elicited by descending volleys might reflect the activity of interneurons and primary afferent depolarisation respectively, produced by impulses through the corticospinal tract.

It is well known that the cord dorsum potential (CDP) evoked by stimulation of dorsal rootlets or peripheral nerves can be recorded from the cord surface in spinal animals (Gasser and Graham, 1933; Eccles, 1964; Schmidt, 1971). Human CDP has also been recorded intrathecally with needle electrodes (Magladery, 1951; Erterkin, 1976). This method, however, involves many risks, and has not been applied for routine clinical testing. Although small and short latency potentials recorded over the spine have been interpreted to be of spinal origin (Cracco, 1973; Matthews *et al.*, 1974; Jones, 1977), several excitable tissues underneath an electrode impede analysis of their origin. In addition, the slow potentials sometimes have proved difficult to record with a surface electrode (Shimoji *et al.*, 1978). A simple, safe method of recording human CDP has been developed using an epidural catheter as a recording electrode (Shimoji *et al.*, 1971), and its potential pattern has been shown to resemble that of CDP directly recorded from the cord surface in animals (Shimoji *et al.*, 1976, 1977). Here we report slow negative-positive potentials recorded from the posterior epidural space at the

lumbar enlargement, elicited by volleys descending through the cord in normal man, while these potentials are absent in patients suffering from amyotrophic lateral sclerosis. The finding suggests that these slow negative-positive potentials might correspond to interneuronal activities and primary afferent depolarisation (PAD) respectively, produced by descending volleys in the corticospinal tract.

Method

Fifteen normal volunteers (11 men, four women, aged 20-52 years) and six patients suffering from amyotrophic lateral sclerosis (four men, two women, aged 42-69 years) participated in this study, having given informed consent. The CDP was recorded from the posterior epidural space at the levels of cervical and lumbar enlargements, using epidural catheters as both the recording and stimulating electrodes (Fig. 1). The recording methods resembled those used in previous investigations (Shimoji *et al.*, 1971, 1977) on human CDP with in addition a weak epidural stimulation of the spinal cord. The subjects were placed in the lateral decubitus for fixation of the electrodes. The epidural catheters were first inserted into the

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Fig. 1 Schematic representation of introduction of electrode into the posterior epidural space of the spine (Shimoji *et al.*, 1971).

posterior epidural space, and then stainless steel wires (230 μm in diameter) were slid inside the catheters up to 5 mm beyond the tip of the tube (Fig. 1). The reference electrodes (needles, 230 μm in diameter) were placed nearby in the supraspinous ligament of the same spinal level. The peripheral stimulating electrodes, paired non-polarisable silver needles, were inserted into the popliteal fossa or wrist of both sides as close as possible to the peripheral nerves. Stimulus pulses of 0.5 ms duration were generated by a constant current stimulator with an isolation unit for both the peripheral and epidural stimulations. The evoked response was amplified with a biophysical amplifier having frequency response more than 3 kHz and led to an averaging computer. The time constant used was 0.3 s for all responses. The background potential changes were also monitored on the pen-writing system. The evoked responses were so small and easily distorted by the electrocardiographic (ECG) artefacts that signal averaging was made at isoelectric intervals between T and P waves of the ECG. The sampling rate was 5–50 points/ms and 10–50 responses were summed. At the end of each experiment the position of the electrodes in the epidural space was confirmed by radiography.

Results

The CDP elicited by segmental nerve stimulation

consisted of the initially positive spikes (P1) and subsequent slow and sharp negative wave (N1), followed by a slower positive wave (P2) which sometimes had two peaks (Fig. 2a). Several characteristics of these potentials are similar to those of spikes, negative (N wave) and positive (P wave) potentials of the CDP in spinal animals (Shimoji *et al.*, 1976, 1977). Therefore, P1, N1, and P2 waves probably reflect the incoming volleys along the roots, interneuronal activities and primary afferent depolarisation respectively. The difference between intact human CDP and CDP in spinal animals has been shown to be the appearance of the second peak in the P2 wave (Shimoji *et al.*, 1976, 1977).

Weak epidural stimulation of the lumbar enlargement, which caused a slight segmental muscle twitch but was not unpleasant, produced polyphasic spikes in the cervical epidural space. On the other hand, weak epidural stimulation of cervical cord produced not only spikes but slow negative-positive complexes in the posterior epidural space of the lumbar enlargement in all normal subjects. These “suprasegmentally evoked” slow potentials were very similar in configuration to those produced segmentally (Fig. 2a). The central latent periods to the peaks of the negative wave and the first component of the slow positive wave in the CDP produced by descending volleys (calculated from the first positive peak of the spike) were approximately 1 ms shorter than those of the N1 and P2 waves evoked segmentally (Table). These slow potentials evoked by descending volleys, however, could not be demonstrated in any of the six patients who all showed typical signs of amyotrophic lateral sclerosis (muscle weakness, muscular atrophy, fasciculation, hyperreflexia) (Fig. 2b). There were, however, no significant changes in conduction velocities of ascending and descending impulses along the cord in these patients (Table).

Discussion

The polyphasic spikes recorded from the posterior epidural space elicited by both ascending and descending volleys probably represent the orthodromic or antidromic impulses or both travelling through several pathways in the spinal cord. The subsequent slow negative and positive potentials produced by descending volleys might represent the synchronised potential of interneurons and PAD respectively (Andersen *et al.*, 1964; Fetz, 1968; Schmidt, 1971). In fact, there is evidence that volleys in the corticospinal tract produce PAD in the cat (Andersen *et al.*, 1964; Fetz, 1968;

PERIPHERAL NERVE STIMULATION

DORSAL COLUMN STIMULATION

Fig. 2 Cord dorsum potentials recorded from the posterior epidural space in a normal subject (a) and in a patient suffering from amyotrophic lateral sclerosis (b). UN→C7, CDP recorded from the C7 vertebral level, corresponding to the C8 spinal segment, in response to ulnar nerve stimulation at the wrist. P1, N1, P2F, and P2S denote the initially positive spike, the sharp negative wave, the first (P2F) and second (P2S) peaks of the slow positive wave, respectively. Note the prolonged negative wave which sometimes occurs (Shimoji et al., 1976) after the P2 wave in recording from a normal subject. TN→T12, CDP recorded from the T12 vertebral level, corresponding to the S1 spinal segment, elicited by tibial nerve stimulation at the popliteal space. T12→C7, CDP recorded from the C7 vertebral level evoked by weak epidural stimulation at the T12 vertebral level. C7→T12, CDP recorded from the T12 vertebral level by weak epidural stimulation at the C7 vertebral level. Note the appearance of slow potentials, similar to the N1 and P2 waves evoked by peripheral nerve stimulation in the normal subject but not in the patient suffering from amyotrophic lateral sclerosis. Positivity is upward in all tracings. Each tracing is the average of 50 individual responses, obtained with an averaging computer (sampling rate of 10–50 points/ms), and plotted on an X–Y recorder. The reference electrodes in recording from cervical and lumbar levels were placed on the skin surface of C7 and T12 spines, respectively. The time constant used was 0.3 s for all responses.

Table Central peak latency of each component in human CDP and approximate conduction velocity through the cord. Central latency was measured from the initial positive peak of the spike. An approximate conduction velocity was calculated from the distance between stimulating and recording electrodes situated epidurally, and the onset time of the first spike. All values represent mean $\pm$ SEM; values in parentheses indicate number of subjects

	Segmentally evoked CDP (ms)			Suprasegmentally evoked CDP (ms)			Conduction velocity (m/s)	
	N1	P2F	P2S	Sharp negative wave	First peak of positive wave	Second peak of positive wave	Ascending	Descending
Normal subjects	3.50 $\pm$ 0.28 (11)	7.32 $\pm$ 0.31 (11)	21.30 $\pm$ 0.53 (11)	2.55 $\pm$ 0.32 (11) *P < 0.05	6.25 $\pm$ 0.22 (11) P < 0.05	18.9 $\pm$ 0.6 (11) P < 0.05	85.5 $\pm$ 6.6 (6)	105.8 $\pm$ 3.8 (6)
Amyotrophic lateral sclerosis patients	3.31 $\pm$ 0.27 (6) NS†	6.92 $\pm$ 0.38 (6) NS	22.08 $\pm$ 0.54 (6) NS				85.6 $\pm$ 9.8 (5) NS	112.3 $\pm$ 9.8 (5) NS

* Values were compared by using Student's *t* test between the similar components in segmentally and suprasegmentally evoked CDPs.
† Values were also compared between normal subjects and patients with amyotrophic lateral sclerosis (NS = not significant).

Schmidt, 1971; Wall, 1967). The finding of peak latency differences between the slow potentials evoked suprasegmentally and segmentally suggests that there are some different interneuronal pathways to produce PAD in human spinal cord. Absence of these slow potentials with preservation of the initial spike in the CDP elicited by descending volleys in patients with amyotrophic lateral sclerosis, in which the pyramidal tract is affected (Oppenheimer, 1976), may support this thesis. The spikes produced by descending volleys in the spinal cord of patients with amyotrophic lateral sclerosis, therefore, are presumably the antidromic impulses along the dorsal column. Thus, epidural recording of human CDPs evoked suprasegmentally may be useful in the study of neurological diseases which affect spinal cord function.

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