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Title
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Abstract
Widespread N18 potential to **median nerve** stimulation was preserved in a patient who had profound unilateral disturbance of deep sensation and a lesion of the pontine medial lemniscus confirmed by MRI. It was concluded from this result that at least a significant part of the N18 potential was generated caudal to the pontine level or at higher levels via extralemniscal pathways. Careful review of studies in man with intraoperative recordings seemed to support that the N18 potential already exists at the medullary level. We suggested that the potential generated at the cuneate nucleus which was described in cats may correspond to part of the N18 potential.

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Short communication

Widespread N18 in median nerve SEP is preserved in a pontine lesion

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Summary Widespread N18 potential to median nerve stimulation was preserved in a patient who had profound unilateral disturbance of deep sensation and a lesion of the pontine medial lemniscus confirmed by MRI. It was concluded from this result that at least a significant part of the N18 potential was generated caudal to the pontine level or at higher levels via extralemniscal pathways. Careful review of studies in man with intraoperative recordings seemed to support that the N18 potential already exists at the medullary level. We suggested that the potential generated at the cuneate nucleus which was described in cats may correspond to part of the N18 potential.

Key words: Somatosensory evoked potential; Median nerve; Cuneate nucleus; Widespread N18; Medial lemniscus

A widespread N18 potential to median nerve stimulation was first recognized by Desmedt and Cheron (1981). The N18 potential was widely distributed over the scalp, could be differentiated from the contralateral parietal N20 that is the first response of the primary sensory cortex, and was first thought to be generated by the thalamus. However, Mauguière et al. (1983) demonstrated that the N18 potential is preserved in patients with thalamic lesions and ascribed its origin to postsynaptic potentials of several brain-stem nuclei which receive collaterals from the medial lemniscus. In this article, the SEPs of a patient with a pontine lesion involving the medial lemniscus is reported, and the origin of the widespread N18 potential is discussed.

Case presentation

The patient was a 51-year-old female who had suddenly developed headache, rotatory vertigo and transient disturbance of conciousness 7 years before; brain CT scan had revealed a hemorrhage in the left pontine tegmentum. Four years after the attack, brain MRI showed a low intensity area in the pontine tegmentum which nearly corresponded to the left medial lemniscus (Fig. 1); no other lesion was found in the brain. On recent examination, the patient had a sensory disturbance on the right side of the body; deep sensation was most severely impaired, and sensory ataxia and pseudoathetosis of the right-side extremities were profound. In addition, she had a mild right hemiparesis, left Horner's syndrome and right palatal myoclonus.

Fig. 1. MRI (T1 intensified image) showing an axial section at the middle to upper pons level. There was a low intensity area almost corresponding to the left medial lemniscus.

Method

The method of recording SEPs was the same as described previously (Sonoo et al. 1990). The median nerve was stimulated at the wrist at a rate of 5 Hz. Stimulus intensity was adjusted so that the amplitude of the antidromic sensory nerve action potential (SNAP) would exceed half the maximum amplitude. Disk electrodes were placed on several points on the neck and scalp including CV6, CV2, inion, hand-sensory area (HS) and Fz. A non-cephalic reference was placed on the shoulder contralateral to the stimulation. Evoked potentials were amplified and filtered between 5.3 and 1500 Hz (−3 dB). 2000–2500 responses were averaged over an analysis time of 50 msec using a 7T18 signal processor (NEC Sanei Co.). Two averages were superimposed. Some bipolar montages were calculated by subtraction between two records with non-cephalic references.

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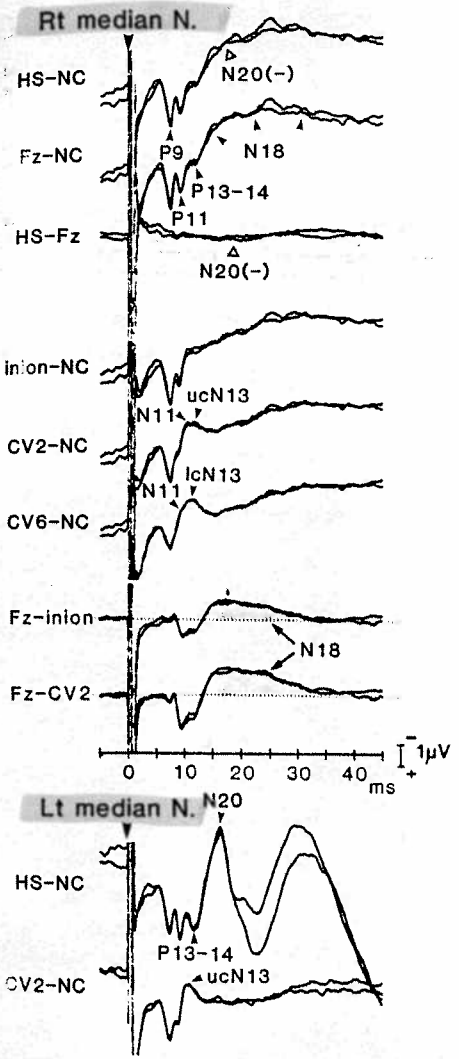

Fig. 2. The upper 8 traces were obtained by right median nerve stimulation; lower 2 traces by left median nerve stimulation. When the right median nerve is stimulated, scalp P9, P11, lcN13 at CV6 and ucN13 at CV2 were normally preserved, but P13–14 was diminished in amplitude compared to the non-affected side. Cortical components including N20 were eliminated, which was confirmed because the HS-Fz lead registered no significant potential. Widespread N18 was preserved in scalp electrodes as a broad elevation of baseline following P13–14. Bipolar montages, such as Fz-inion of Fz-CV2, clearly demonstrated N18 which lasted about 20 msec, because gradual inclines of all the traces with non-cephalic references due to slow ECG artifacts were canceled.

Results (Fig. 2)

When the right median nerve was stimulated, both CV6 and CV2 electrodes registered normal P9, (P11)-N11 and N13. N13 at CV6 was labeled lcN13 (lower cervical N13), and N13 at CV2 was labeled ucN13 (upper cervical N13; Sonoo et al. 1990). Scalp electrodes registered normal P9 and P11, but P13-14 was smaller than that of the unaffected side. Scalp N20 and succeeding cortical components were completely lost, which was proved by the fact that the HS-Fz bipolar lead registered no significant components. Widespread N18 potential was preserved at scalp electrodes as a broad elevation of the baseline after P13–14, which was somewhat difficult to judge in records with non-cephalic reference since the slow ECG artifact gave a uniform incline to baselines of all the cephalo-cervical leads. These inclines were canceled in calculated bipolar montages such as Fz-inion and Fz-CV2, and N18 potential was clearly demonstrated as a broad negativity which lasted about 20 msec.

Discussion

The patient had a profound disturbance of deep sensation, and MRI revealed a lesion involving the medial lemniscus in the pons. Although her various symptoms indicate that the lesion was not confined to the medial lemniscus, it is obvious that the lemniscal pathway has been interrupted at (or caudal to) the pontine level since no lesion was detected at other levels in the sensory pathway. The fact that the scalp P13-14 which is usually attributed to the medial lemniscus was diminished in amplitude and that cortical components including N20 were eliminated corresponds to this lesion.

The widespread N18 potential which lasted about 20 msec was preserved in this case; therefore, at least a significant part of the N18 potential is generated caudal to the pontine level or at higher levels via extralemniscal pathways. Of course, we cannot deny the possibility that another part of the N18 potential is generated at the rostral brain-stem via lemniscal pathways and is lost in this case. In our previous study (Fig. 6a in Sonoo et al. 1990), the widespread N18 potential was lost when the dorsal column was interrupted at the C1–2 level, which indicates that N18 potential is generated rostral to the C1–2 level.

A few authors (Urasaki et al. 1985; Suzuki et al. 1988) have reported that the N18 potential was delayed or lost in pontine lesions. Some of their patients had massive pontine hemorrhage; thus, damage to more caudal structures cannot be ruled out. Moreover, confusion has resulted because these authors paid attention to the 'latency of N18' in frontal electrodes, which is in fact the onset latency of the frontal P20, the first cortical component. As Desmedt and Cheron (1981) and Mauguière et al. (1983) have demonstrated, the essential attribute of the N18 potential is a broad elevation from the baseline which follows the P13–14 potential and lasts approximately 20 msec.

Several brain-stem nuclei which receive collaterals from the medial lemniscus, such as medial and dorsal accessory olives, inferior colliculus, superior colliculus, red nucleus and medial geniculate nucleus, are possible generators of the N18 potential (Mauguière et al. 1983). Mauguière and Desmedt (1989) have suggested that tectal and pretectal nuclei are the most important generators of the N18 potential. In the present case, however, the cuneo-tectal pathway which leaves the medial lemniscus at midbrain level (Björklund and Boivie 1984) must have been interrupted, and probably only the accessory olives among the nuclei listed above have been able to receive inputs from the medial lemniscus.

Intraventricular recordings in the human brain-stem showed that a large negativity which lasts around 5 msec follows small positive far-field potentials (Hashimoto 1984; Urasaki et al. 1990). Urasaki et al. (1990) concluded that the N18 potential is generated at the rostral brain-stem because they observed that the peak latency of this negativity at this region coincided with 'the latency of scalp N18.' It

should be noted that these intraventricular recordings have never conclusively shown a broad negativity lasting about 20 msec like the scalp N18 potential. We think that intraventricular electrodes may be considerably influenced by the near-field potentials of the medial lemniscus which runs close to the fourth ventricular floor. Direct recording in animals in the vicinity of the medial lemniscus revealed that repetitive firings last about 5 msec after the first response (Arezzo et al. 1979).

Recordings from the ventral surface of the brain-stem were performed by Suzuki and Mayanagi (1984). Although they did not mention the N18 potential, broad elevation of the baseline which resembles the N18 potential was clearly recognized in their Fig. 4 from the ventral medulla to ventral midbrain. The amplitude of this negativity was largest at the medulla. In addition, it should be noted that there appeared to be a broad positivity corresponding to the ventral negativity at the dorsal medulla in their recordings (Fig. 6 of Suzuki and Mayanagi 1984). Broad positivity following the first negativity was also recorded by other authors at the dorsal surface of the cuneate nucleus (Møller et al. 1986). Thus, a positive-negative dipolar potential whose time-course resembles the scalp N18 potential appears to be distributed around the medulla, dorso-ventrally and probably also caudo-rostrally. As has already been mentioned, the possibility that the remaining N18 potential in the present case was generated at the rostral brain-stem via the extralemniscal pathway could not be excluded by our result alone. However, our result combined with the information from intraoperative studies suggests that at least a significant part of N18 potential is generated at the caudal brain-stem.

When we review animal studies, Andersen et al. (1964a) showed in cats that the second phase of the potential, generated at the cuneate nucleus following peripheral nerve stimulation, has dorso-caudal positive and ventro-rostral negative polarity. They demonstrated that its origin is the depolarization of presynaptic terminals of the dorsal column fibers terminating in the cuneate nucleus (primary afferent depolarization; Andersen et al. 1964b). The distribution, time-course and wave form of the ventro-rostral negative pole of this dipolar potential in cats are similar to those of the N18 potential in man. We can find no other well described potential of brain-stem origin in animal studies which can be related to the N18 potential. Therefore, we suggest the possibility that some part of the N18 potential corresponds to the potential generated at the cuneate nucleus in cats.

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