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Nasopharyngeal recordings of somatosensory evoked potentials document the medullary origin of the N18 far-field

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Summary Because the nasopharyngeal electrode provides non-invasive access to the ventral brain-stem at the medullo-pontine level we used it for recording somatosensory evoked potentials (SEPs) to median nerve stimulation (non-cephalic reference). After the P9 and P11 far-fields, the nasopharyngeal SEPs disclosed a negative-going component which was interpreted as the near-field equivalent of the P14 scalp far-field generated in the caudal part of the medial lemniscus. Nasopharyngeal SEPs also revealed a large N18 with voltage and features strikingly similar to those of the scalp-recorded N18 far-field. These results suggest that N18 is generated in the medulla and not more rostrally in the brain-stem. The use of a nasopharyngeal electrode as reference for topographic brain mapping is discussed. The paper documents the feasibility and relevance of nasopharyngeal recordings for non-invasive analysis of short-latency SEPs.

Key words: Nasopharyngeal electrode; Somatosensory evoked potentials; P14 far-field; Negative far-field potential; N18 far-field; Non-cephalic reference; Cuneate nucleus

The nasopharyngeal electrode is currently used for recording mesial temporal EEG in patients with focal seizures (Gastaut 1948; MacLean 1949; MacLean and Arellano 1950; Mavor and Hellen 1964), but it is not currently appreciated that it provides a unique non-invasive access to the ventral brain-stem at about the level of the medullo-pontine junction (Fig. 1). In the present study, the short-latency nasopharyngeal somatosensory evoked potentials (SEPs) disclosed a large N18 with voltage and features similar to those of the scalp-recorded N18 far-field. We suggest on this basis that N18 is generated in the medulla, thus in a more caudal part of the brain-stem than currently believed.

Methods

Standard methods of recording SEPs were used in 12 normal adult humans (22–31 years, 7 males) who had given informed consent. The subjects were comfortably seated in a reclining chair with head rest. They were instructed to keep muscles relaxed and read a novel which was presented on short lines to minimize eye movements (Desmedt and Tomberg 1989). Data on patient 1 of Noël et al. (1992) are included.

A standard nasopharyngeal electrode with gold-plated ball tip and a bend of 20° about 25 mm from the tip (Grass Instruments Co.) was inserted into one nostril with its convex side upward. In a few experiments such an electrode was inserted into each nostril and the recordings were found to be essentially similar for both. When the electrode had reached the pharyngeal cavity, it was rotated through an angle of about 130° to bring its tip into firm contact with the upper posterior wall of the pharynx. Care was taken to achieve an electrode impedance below 5 kΩ.

Brief electric pulses were delivered to the median nerve at the wrist (thumb twitch threshold) at intervals of 2000, 800 or 400 msec. The temperature of the forearm skin was maintained at 33–35°C. Scalp SEPs were recorded with frontal and parietal steel needle electrodes. In 6 of the subjects, 41 scalp channels were used for topographic SEP mapping. A non-cephalic reference on the contralateral shoulder was used to record both near-field and far-field potentials (Cracco and Cracco 1976; Desmedt and Cheron 1980). The system bandpass was 10–1000 Hz and the bin width was 500 μsec. Each trial with interference exceeding 50 μV was automatically rejected from the average. A MicroVax-II (Digital Equipment Inc.) was used for programing, data acquisition, on-line quality controls and off-line data processing. When data from different

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subjects were pooled in scatter displays or in grand averages, the individual differences in SEP latency (mainly related to arm length) were compensated by appropriate horizontal shifts of the traces. Bit-mapped potential fields were computed by interpolation of averaged potential data to a radial projection of the head with about 10,000 surface points (pixels), using the 2-spline method (Perrin et al. 1987). Details of the methods have been published (Desmedt and Tomberg 1989; Tomberg et al. 1989; Desmedt et al. 1990).

Results

Nasopharyngeal electrode recording site

The nasopharyngeal electrode placed against the upper posterior wall of the pharyngeal cavity is seen in

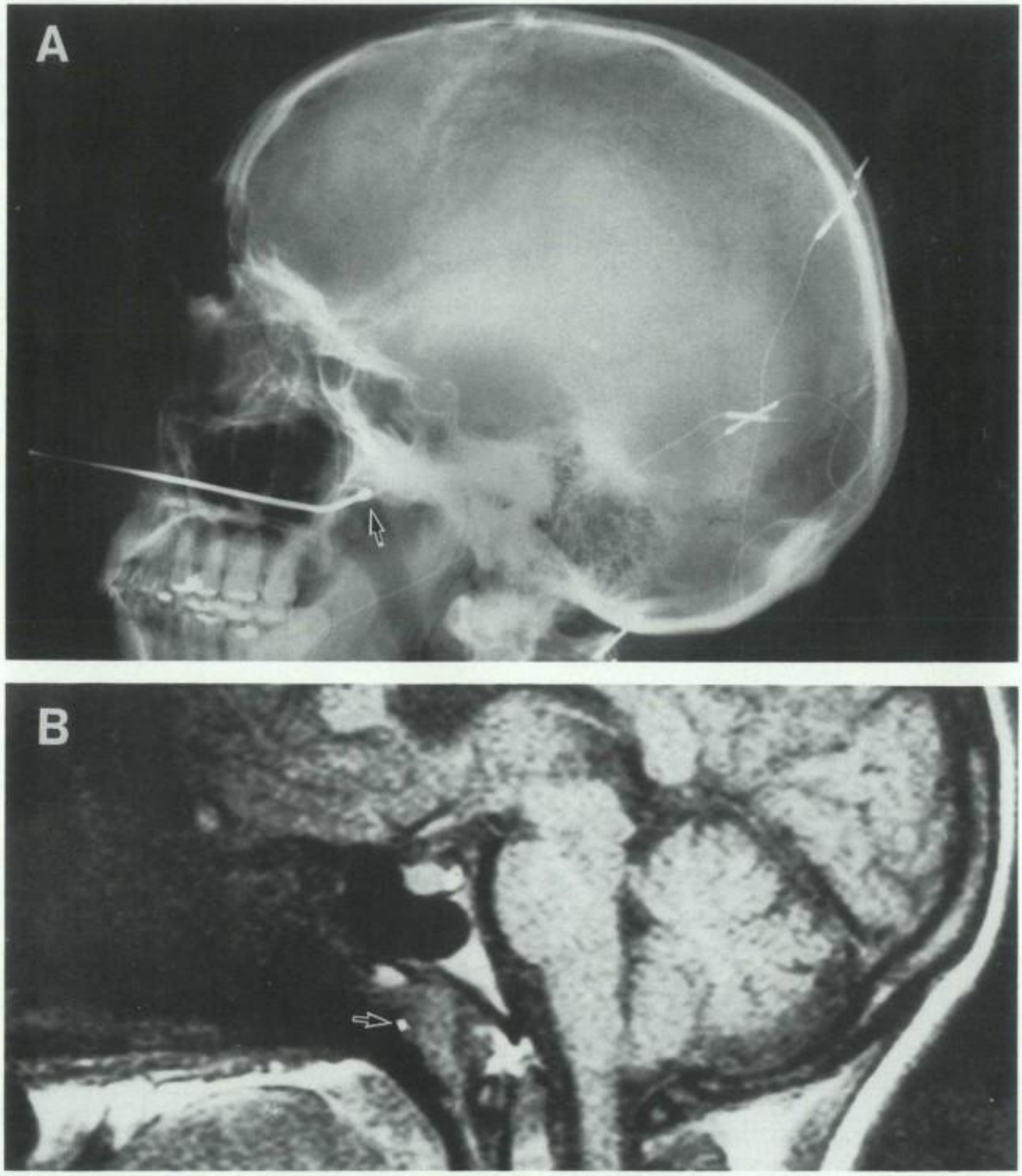

Fig. 1. Nasopharyngeal electrode in position against the upper posterior wall of the pharynx in X-ray (A) and magnetic resonance imaging (MRI) (B).

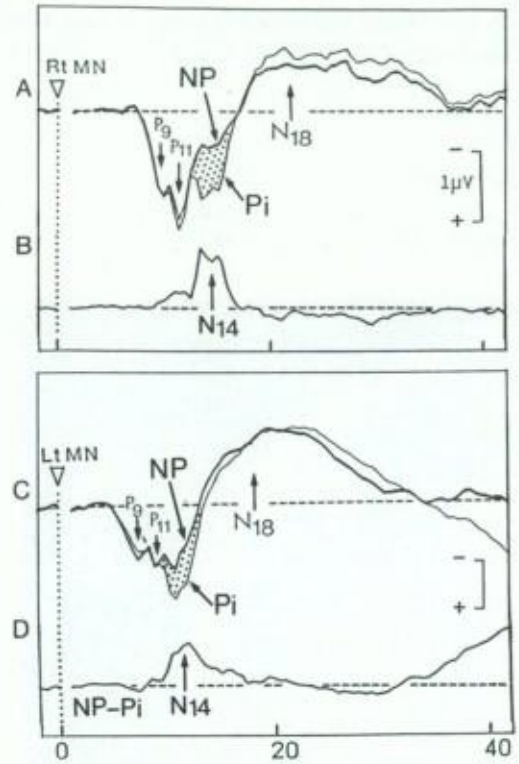

Fig. 2. SEPs recorded with non-cephalic reference from ipsilateral parietal (Pi) and nasopharyngeal (NP) electrodes. A–B: male subject of 23 years, right median nerve stimulation. C–D: pooled SEPs of 2 male subjects of 22 and 23 years, left median nerve stimulation. B and D: electronic subtraction of Pi from NP.

magnetic resonance imaging (MRI) to approach the ventral brain-stem at a level slightly below the medullo-pontine junction (Fig. 1B). The distance from the electrode tip to the brain-stem is about 36 mm which we consider to achieve near-field recording conditions. For comparison, when recording the spinal N11 and N13 SEP responses, the distance from the posterior (or anterior) neck skin to the high cervical spinal cord is about 60 mm (Desmedt and Cheron 1980, 1981a). When recording the N20 response at the parietal scalp, the distance from the skin surface to the N20 tangential generator located in area 3b within the central sulcus is about 30 mm (Okada et al. 1984; Allison et al. 1989; Desmedt et al. 1990, Fig. 12). Near-field SEPs have thus been recorded so far over a range of distances up to 30–60 mm from the active recording electrode.

Short-latency nasopharyngeal SEP

After the well-known P9 and P11 far-fields, the nasopharyngeal SEPs showed an unexpected negative-going response which rose towards the baseline ahead of the ascending limb of the scalp SEP; its latency corresponded to that of the positive-going onset of the scalp-recorded P14 far-field (Fig. 2). The ascending limb started from the positive level achieved by the P9-P11 far-fields and frequently presented a small notch. Both scatter displays and grand averages docu-

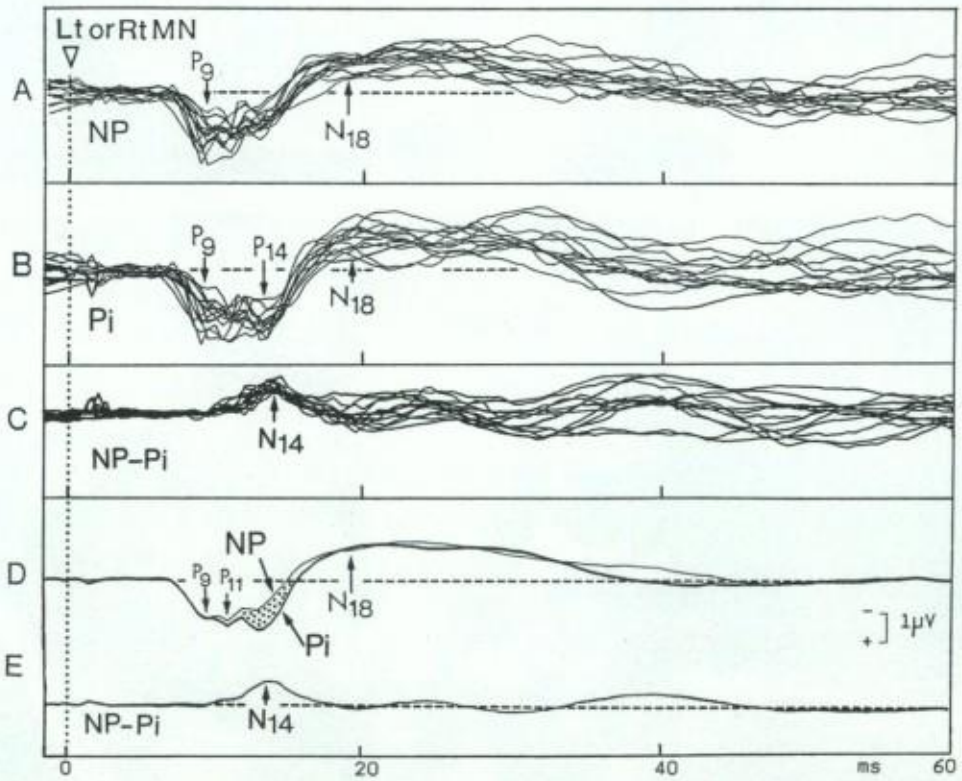

Fig. 3. Scatter displays of SEPs of all subjects (one trace per subject). Left or right median nerve stimulation. Non-cephalic reference. Adjustment of latencies. A: nasopharyngeal (NP) SEP. B: ipsilateral parietal (Pi) SEP. C: electronic subtraction of Pi from NP traces. D: grand average of traces of all subjects. E: electronic subtraction of Pi from NP grand average traces.

mented the overall consistency of the data for the normal subjects (Fig. 3). We present the frontal SEPs (Fig. 4) to illustrate the largest scalp P14 (followed by frontal SEP components) and the ipsilateral parietal SEPs which showed a slightly smaller P14 with virtually no focal cortical response thereafter (Desmedt and Cheron 1981b; Mauguière et al. 1983). The voltage difference between the nasopharyngeal and scalp SEPs, at P14 latencies, has been stippled (Figs. 2A,C, 3D, 4D) or displayed by electronic subtraction (Figs. 2B,D, 3C,E, 4C,E). Taking as time reference the onset of the scalp P11 far-field which reflects the onset of the dorsal column afferent volley at spinal entry (Desmedt and Cheron 1980), the mean onset latency of the scalp P14 was 1.87 +/− 0.35 msec. These transit time values compared with means of 2.26, 1.75 and 1.67 msec found respectively by Desmedt and Cheron (1980, 1981b) and Desmedt et al. (1983). The mean onset latency of the negative-going divergence of nasopharyngeal response was 1.74 +/− 0.2 msec (Table I), thus not significantly different.

TABLE I

Latencies (msec) of P14 scalp far-field and nasopharyngeal N14 near-field are not significantly different.

Transit times	to P14 onset	to N14 onset	*t* test
From P9 onset	4.76 ± 0.46	4.65 ± 0.51	$P > 0.2$
From P11 onset	1.87 ± 0.35	1.74 ± 0.57	$P > 0.2$

The voltage from prestimulus baseline was much less positive for the nasopharyngeal SEPs at the peak latencies of both the scalp P13 (early part of P14) and the scalp P14 (Table II). The mean voltage was 1.64

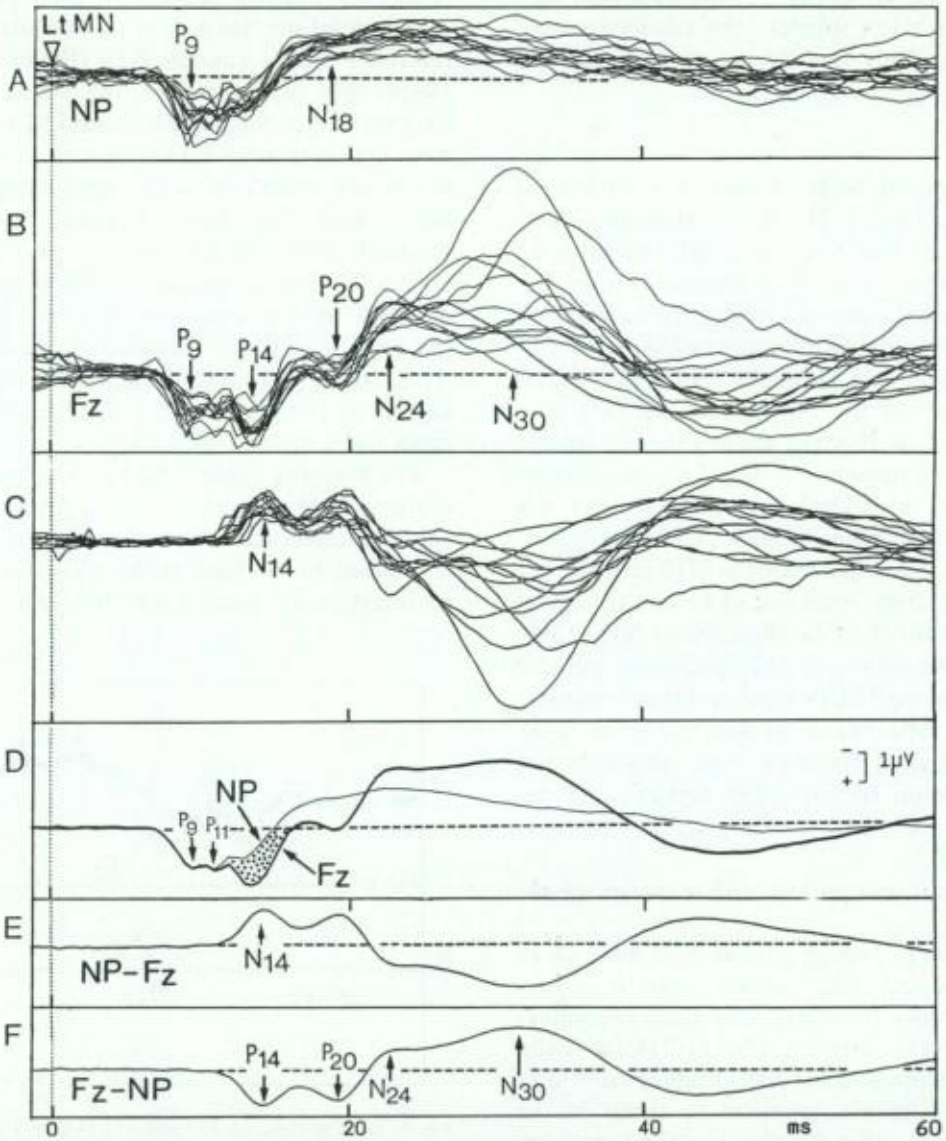

Fig. 4. Same presentation as Fig. 3 for frontal SEPs. A: nasopharyngeal (NP) SEP. B: frontal Fz SEP. C: electronic subtraction of Fz from NP. D: grand average of traces of all subjects. E: electronic subtraction of Fz from NP grand average traces. F: electronic subtraction of NP from Fz to display cortical components with usual polarities.

TABLE II

Total duration of N18 (msec) and voltage of SEP components measured from pre-stimulus baseline.

	Nasopharyngeal SEP	Scalp SEP	*t* test
N18 duration	18.05 ± 2.46	18.68 ± 1.81	$P > 0.2$
Voltage P9	1.51 ± 0.51	1.53 ± 0.52	$P > 0.1$
Voltage P11	1.52 ± 0.37	1.64 ± 0.36	$P < 0.001$
Voltage P13	0.96 ± 0.43	1.83 ± 0.54	$P < 0.001$
Voltage P14	0.66 ± 0.46	1.92 ± 0.52	$P < 0.001$
Voltage N18 (at 18 msec)	−0.97 ± 0.38	−0.79 ± 0.37	$P < 0.02$
Voltage N18 (at 27 msec)	−1.02 ± 0.53	−1.08 ± 0.48	$P > 0.5$

μV for the scalp P11 and 1.52 μV for the nasopharyngeal P11 and this slight difference appeared to be significant (Table II). It related to the somewhat surprising fact that, in a few subjects, the nasopharyngeal SEP diverged negatively slightly before the onset of scalp P14.

N18 response

The nasopharyngeal SEPs disclosed a prolonged negative response (Figs. 2–3) which was quite similar to the N18 recorded over the ipsilateral parietal scalp (Desmedt and Cheron 1981b) or bilaterally over the whole scalp in patients with a capsulo-thalamic lesion interrupting the somatosensory pathway (Mauguière et al. 1983; Mauguière and Desmedt 1988, 1989). The initial upgoing limb of the nasopharyngeal N18 rose earlier and tended to reach a slightly larger voltage initially (at 18 msec), namely −0.97 μV as compared to −0.79 μV for the ipsilateral parietal N18, but this difference did not reach significance (Table II). Variations occurred among subjects for the N18 profiles, but no significant differences were found for the voltage at 28 msec latency or for the total duration of N18 (Table II). Electronic subtraction of the ipsilateral parietal from the nasopharyngeal SEPs yielded difference traces that were indeed rather close to baseline (Figs. 2–3). The nasopharyngeal N18 profile was obviously not distorted by diffusion of any focal cortical SEP responses (Fig. 4D).

Nasopharyngeal SEP in a patient with a mesencephalic lesion

Cortical SEPs were lost in patient 1 of Noël et al. (1991) with a mesencephalic vascular lesion interrupting the somatosensory pathways, and scalp recordings only disclosed an N18 after the P9-P11-P14 far-fields. The profile and features of the nasopharyngeal N18 of this patient were virtually identical to those of the scalp N18 (Fig. 5A), also for the so-called wavelets (Mauguière and Desmedt 1989). Electronic subtraction of these traces canceled N18 (Fig. 5B).

Nasopharyngeal reference in scalp mapping

When mapping with a non-cephalic reference, the widespread N18 far-field drives all SEP traces negatively thereby unduly complicating the interpretation of focal cortical components that appear flooded for about 15 msec in a sea of negativity (Fig. 6A) (Desmedt et al. 1987; Mauguière and Desmedt 1989). When mapping with a nasopharyngeal reference, the scalp potential gradients did not change but the focal fields of the parietal N20-P24-P27, precentral P22 and frontal P20-N24-N30 SEP responses were zero-centered by removal of the underlying N18 (Figs. 4F, 6B). This can be done by electronically subtracting the nasopharyngeal channel from all other recording channels. The P9-P11 far-fields are then canceled while P14 is enhanced by algebraic addition of N14.

Because there are no ipsilateral short-latency SEP responses to distal upper limb stimulation, the ipsilateral parietal site has also been considered as a possible reference which cancels N18 (Desmedt and Cheron 1981b) (Fig. 6C). However, this reference may not be foolproof since focal contralateral SEPs (especially the frontal negativities) can diffuse to various extents towards the ipsilateral scalp, even after the ipsilateral hemisphere has been removed (Mauguière and Desmedt 1989). We consider the nasopharyngeal reference to be more consistent in this respect since it does not pick up any short-latency cortical potentials. For the same reason, the nasopharyngeal reference is obviously better than the frontal Fz reference which is known to introduce severe distortions of SEPs (Desmedt and Cheron 1980, 1981b).

For mapping *cortical* SEPs, the nasopharyngeal reference could conceivably be preferred over a non-cephalic reference at the shoulder on the ground that it is closer to the base of the head and thus less liable to interference from heart potentials. However, the

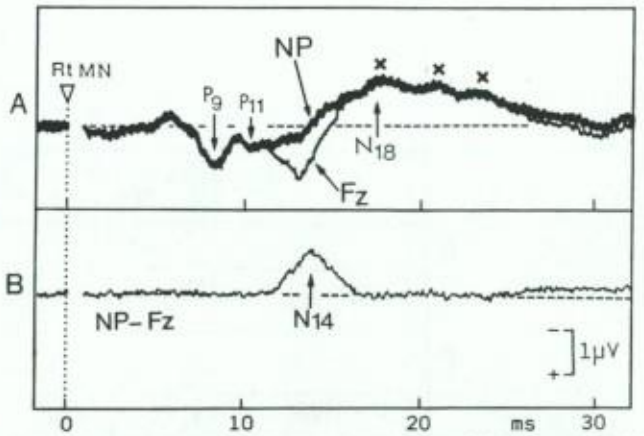

Fig. 5. SEPs to right median nerve in patient 1 of Noël et al. (1992) with a mesencephalic vascular lesion. Non-cephalic reference. A: superimposed Fz and nasopharyngeal (NP) recordings. B: electronic subtraction of Fz from NP.

non-cephalic reference is obviously essential for disclosing *subcortical far-field* SEP.

Discussion

The present paper documents the feasibility and relevance of using nasopharyngeal recordings for the non-invasive analysis of short-latency SEPs.

Origin of P14 far-field

It is now agreed that P14 has its source above the spinal cord and is abolished by lesions at the spino-medullary junction (Mauguière and Ibañez 1985; Yamada et al. 1986; García-Larrea and Mauguière 1988). Considerations of conduction distances and transit times along dorsal columns and cuneate nucleus suggested that the early part of the scalp P14 reflected the afferent action potential volley conducted in *caudal* lemniscal fibers (Desmedt and Cheron 1980, 1981b). Depth recordings along the fourth ventricle indeed disclosed a large negative N14 near-field (Suzuki and Mayanagi 1984) which changed over into a positive (far-field) P14 at about the level of the medullo-pontine junction (Urasaki et al. 1990). The caudal brain-stem origin of P14 has also been documented in patients in ongoing brain death (Buchner et al. 1988).

The present non-invasive nasopharyngeal recordings at a level slightly below the medullo-pontine junction (Fig. 1) disclosed a negative-going SEP response whose onset latency matched fairly well the onset latency of the scalp P14 far-field (Figs. 2–4, Table I). It must be appreciated that our nasopharyngeal lead is about 36 mm away from the ventral aspect of the medulla (Fig. 1B) which makes for a less efficient pick up than the depth electrodes inserted by Urasaki et al. directly into the fourth ventricle. Even though the negativity we recorded at P14 latencies did not actually cross the baseline, we interpret this response as identical to the 'N14' near-field seen in invasive recordings (Jacobson and Tew 1988; Urasaki et al. 1990) and propose to call it N14. This N14 near-field potential is thought to reflect the volley of afferent action potentials conducted in the caudal part of medial lemniscus fibers, just above the cuneate nucleus. At variance with an earlier suggestion of Lüders et al. (1983), the P14 source is thus located definitely above the foramen magnum which is caudal with respect to the cuneate nucleus (Desmedt and Cheron 1980, Fig. 6).

Origin of N18

N18 is lost after a lesion of the afferent somatosensory pathway at the spino-medullary junction (Mauguière and Ibañez 1985), but it persists after a lesion interrupting the somatosensory pathway at the thalamic level (Mauguière et al. 1983; Mauguière and Desmedt 1988). In patients with long-standing hemispherectomy, N18 is fully preserved while the scalp P14 is reduced in conjunction with retrograde degeneration of cuneo-thalamic (lemniscal) axons (Mauguière and Desmedt 1989). These results definitely exclude the spinal cord, diencephalon and cortex as the source of N18.

Depth recordings in the fourth ventricle did not seem to identify any precise N18 source and Urasaki et al. (1990) considered N18 to be generated 'between upper pons and midbrain.'

The present nasopharyngeal recordings disclosed large N18 responses with voltage and features quite similar to those of the concomitantly scalp-recorded N18 (Figs. 2–5, Table II). This robust evidence suggests that the N18 source must be located in the medulla caudally to the medullo-pontine junction, thereby eliminating as candidates a number of more rostrally located brain-stem nuclei that do receive direct lemniscal input, such as the pontine gray and nucleus raphe pontis, the basal and lateral parts of the inferior colliculus, the deep and intermediate layers of the superior colliculus, and the pretectal nuclei (Kuypers and Tuerk 1964; Hand and Van Winkle 1977; Baleydier and Mauguière 1978; Berkley et al. 1986). If N18 is indeed generated in the medulla, the cuneate nucleus and the medial and dorsal accessory nuclei of the inferior olive (Berkley 1975) are likely candidates for the source of N18 (Noël et al. 1992) because they receive short-latency somatosensory excitation from cuneate neurons.

Several important considerations are pertinent at this point. One is the fact that N18 is a widely distributed far-field characterized by a prolonged time course (Table II) and a negative polarity excludes spike volleys as its source. N18 rather reflects excitatory post-synaptic potentials (EPSPs) that generate extracellular negativities of several milliseconds' duration around the soma-dendrite pole of neurons. There is a good example of such an EPSP-related generator for the spinal N13-P13 non-propagated SEP response which reflects EPSPs in apical dendrites of layer IV–V neurons of the dorsal horn of the cervical spinal cord (Desmedt and Cheron 1981a; Desmedt and Nguyen 1984; Desmedt 1988).

On the other hand, we wish to emphasize that the N18 onset does not precede, but actually follows the P14 onset (Figs. 2–5). This implies that N18 generators only become manifest in human SEP recordings when an initial spike volley has actually been triggered in the caudal medial lemniscus axons. This essential boundary condition need not rule out cuneate neurons as a possible N18 source for several reasons: (1) the precise onset latency of N18 proper has not yet been measured with enough precision (to the split msec range) to resolve the issue of any possible overlap between the

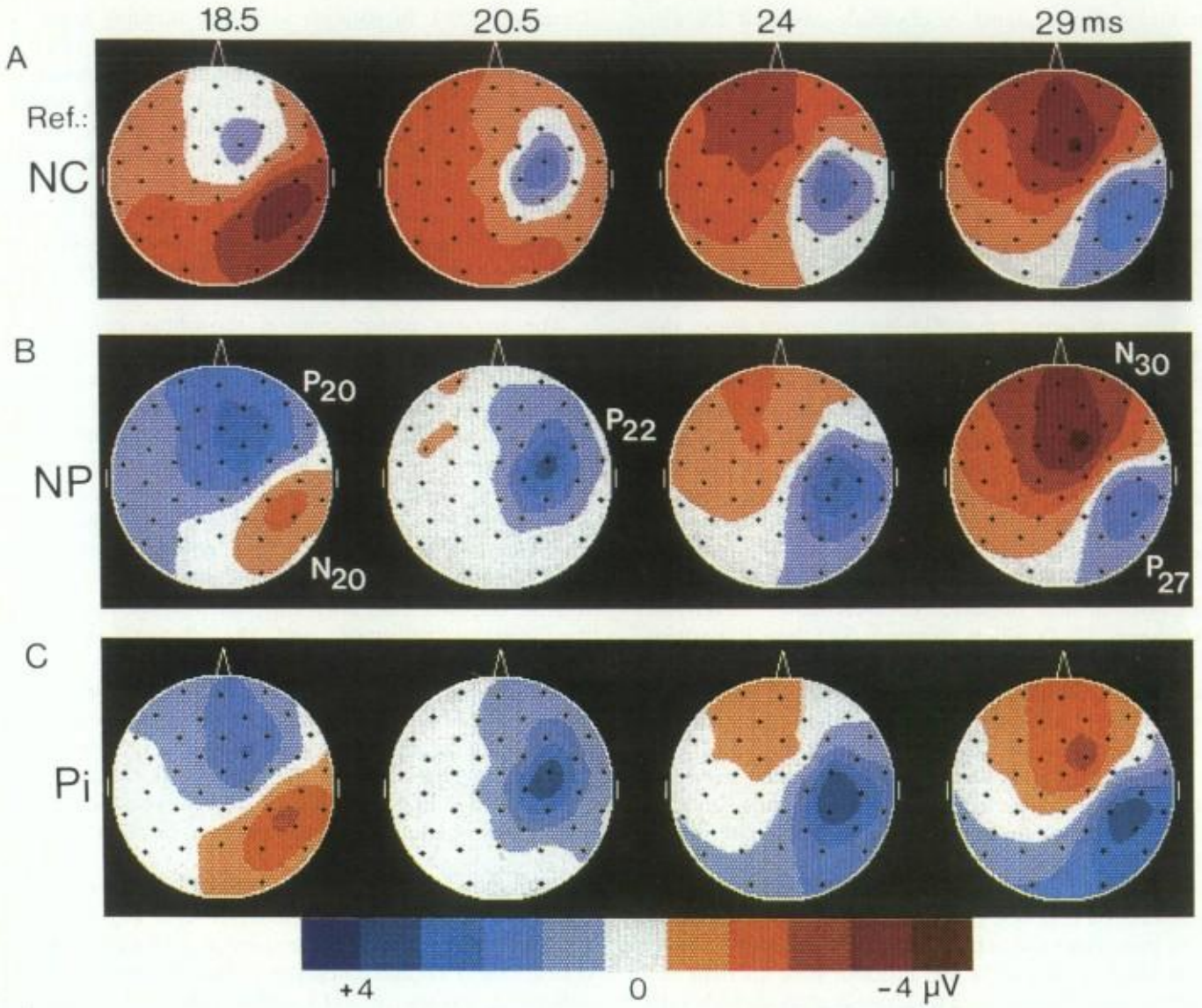

Fig. 6. Brain mapping with 41 electrodes and 2-spline interpolation. Same data as in Fig. 2C. Left median nerve stimulation. Voltages displayed in a linear color scale with red for negative and blue for positive. Latencies of frozen maps indicated above. A: non-cephalic reference on the right shoulder. B: nasopharyngeal reference. C: right (ipsilateral) parietal reference.

initial parts of N18 and P14 far-fields; (2) the dorsal column excitatory synapses on the soma-dendrites of cuneate neurons have a high safety factor (Andersen et al. 1964) so that the first lemniscal spikes (contributing to the scalp P14) must be triggered with a very short synaptic delay (0.5 msec or less). This early lemniscal volley could therefore precede the peak of the cuneate EPSPs which might conceivably generate at least in part the N18; (3) cuneate EPSPs are recorded as a surface negativity (so-called N wave) from the cuneate dorsal surface in the cat (Therman 1941; Andersen et al. 1964); they last for several milliseconds and elicit a burst of spikes in lemniscal axons. These data suggest that cuneate EPSPs have a rather long duration and thus persist beyond the initial spike burst which generates the (brief) P14 far-field.

Looking now beyond the cuneate nucleus, the axons of cuneate neurons elicit EPSPs in the neurons of the accessory inferior olives which can therefore also be considered a possible N18 source, pending more information about the likely open geometry and orientation of their extracellular potential fields (Noël et al. 1992). The fact that P14 actually precedes N18 should not raise any difficulty if the accessory olive neurons are considered the source of N18. These neurons must obviously generate their EPSPs shortly *after* (not before) the onset of action potentials in the caudal lemniscal axons.

Because it is diffuse, prolonged and of negative polarity, N18 has had the image of an unorthodox SEP component ever since its description (Desmedt and Cheron 1981b). N18 has been considered to reflect

multiple generators in the brain-stem nuclei that receive lemniscal input (Mauguière et al. 1983; Mauguière and Desmedt 1989), but the present nasopharyngeal recordings establish the medulla as the essential source of this challenging SEP response.

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