Electroencephalography and clinical Neurophysiology, 1983, 56: 283–292
Elsevier Scientific Publishers Ireland, Ltd.

NEURAL GENERATORS OF N18 AND P14 FAR-FIELD SOMATOSENSORY EVOKED POTENTIALS STUDIED IN PATIENTS WITH LESION OF THALAMUS OR THALAMO-CORTICAL RADIATIONS [1]

F. MAUGUIÈRE, J.E. DESMEDT [2] and J. COURJON

EEG Department, Hôpital Neurologique, Faculté de Médecine Lyon Nord, 59 Boulevard Pinel, 69394 Lyon Cedex 3 (France) and Brain Research Unit, University of Brussels, 115 Boulevard de Waterloo, Brussels 1000 (Belgium)

(Accepted for publication: May 4, 1983)

Short-latency components of the somatosensory evoked potentials (SEPs) to median nerve stimulation have increasing uses in clinical diagnosis (Desmedt 1971; Noel and Desmedt 1975, 1980; Colon et al. 1978; Hume and Cant 1978, 1981; Kimura et al. 1978; El Negamy and Sedgwick 1979; Jones 1979; Anziska and Cracco 1980; Chiappa et al. 1980; Mauguière and Courjon 1981; Siivola et al. 1981; Chiappa and Ropper 1982; Eisen 1982; Mauguière et al. 1983). Current advances rely on better differentiation of standard SEP components and their generators. For example, the use of a non-cephalic reference (Cracco and Cracco 1976) helped resolve the neck SEP negativities into an N11 component propagated with a measurable velocity up the dorsal columns (Desmedt and Cheron 1980a; cf. also Lesser et al. 1981) and an N13 component related to a fixed generator in the spinal cord dorsal horn (Desmedt and Cheron 1981a). The scalp-recorded SEP shows a P9 (volume-conducted brachial plexus volley), a P11 (far-field potential equivalent of the spinal N11), and a P14 (volume-conducted lemniscal volley). These positive scalp far-field potentials are followed by a negativity whose generator source is still debated (see Desmedt and Cheron 1981b). Kritchevsky and Wiederholt (1978) using a non-cephalic reference drew attention to the apparently bilateral distribution of the early SEP negativity, while Chiappa et al. (1980, page 270) found an early negative SEP deflexion to persist (with loss of the subsequent P27) in a patient with extensive radiolucent lesions of the centrum semioval white matter at CT scan. This suggested the early SEP negativity to be generated in thalamus or in thalamo-cortical radiation fibers rather than in the cortex as previously believed (Giblin 1964; Desmedt and Manil 1970; Desmedt and Robertson 1977; Small et al. 1980). A way out of this dilemma was proposed by Desmedt and Cheron (1981b) who emphasized the dual nature of the early SEP negativity; they differentiated an N18 component with a widespread symmetrical scalp distribution from the classical N20 component which is only recorded over the contralateral postrolandic scalp. Furthermore Mauguière et al. (1983) found the parietal N20 and P27-P45 SEP components to be lost in patients with a postcentral *cortical* lesion producing hemianesthesia on the opposite side of the body and they concluded that N20 indeed indexes the cortical SEP response of the contralateral parietal receiving areas.

The present study looks into the generators of the widespread N18 and shows that it persists in patients with an extensive thalamic or suprathalamic lesion which eliminates both the postcentral N20-P27-P45 and the prerolandic P22-N30 SEP components.

Materials and Methods

Four patients with a severe unilateral subcortical vascular lesion were selected from a larger group. The lesions were documented at the time of

[1] This research has been supported by grants from the Fonds de la Recherche Scientifique Médicale, Belgium

[2] Correspondence to: Prof. J.E. Desmedt, Brain Research Unit, 115 Boulevard de Waterloo, 1000 Brussels, Belgium.

SEP study by CT scans and by clinical signs of hemianesthesia with hemiplegia. The normal side of each patient served as his or her own control. An extensive series of normals of various ages also provided comparative data (cf. Desmedt and Cheron 1980b, 1981b). For SEP recording, the subjects (who had given informed consent) were lying supine on a couch, with closed eyes, in a warm, quiet, semi-darkened room. The stimuli were 0.2 msec square electrical pulses delivered to the median nerve at wrist (cathode proximal; intensity just above thumb twitch threshold). Intervals between stimuli were 500–600 msec. Several runs of 500 trials each were carried out by stimulating in turn the right or the left median nerve. The SEPs were recorded with 8 derivations referred to the opposite hand or shoulder (non-cephalic reference). Active electrodes were placed over the Cv6 and Cv2 spinous processes at the neck, and bilaterally on scalp at 6–7 cm from midline at parietal, central and frontal positions. All channels were averaged simultaneously, each with 136 μsec bin width. The overall bandpass extended from 1 or 3.2 to 3000 Hz (see Desmedt 1977). Samples with excessive muscle potentials interference were automatically rejected. The figures only illustrate the parietal and frontal derivations, as unsmoothed traces drawn on paper by the averaging computer (negativity of the active electrode registers upwards). SEP traces simultaneously recorded from symmetrical left and right scalp sites are superimposed for more accurate analysis of components (see Desmedt and Cheron 1981b) (thicker trace for SEP contralateral to the side stimulated).

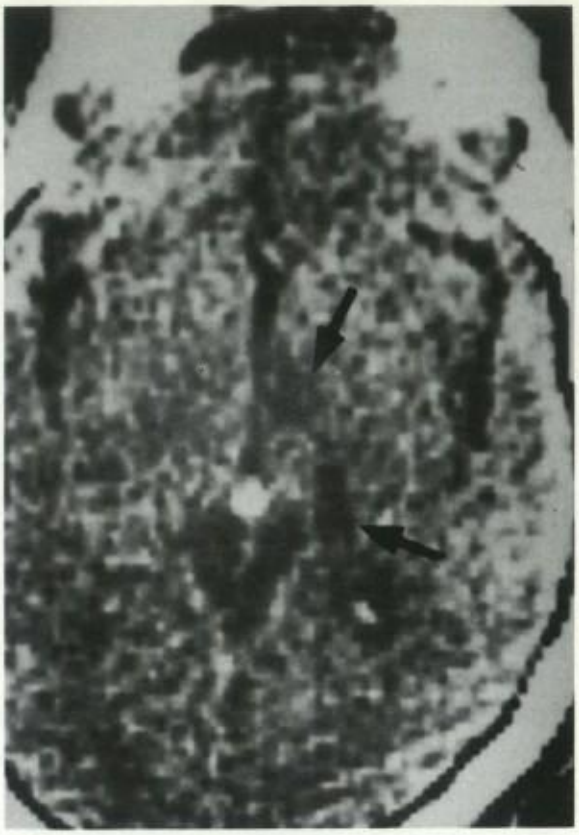

Fig. 1. Enlarged horizontal CT scan section showing the right capsulo-thalamic vascular lesion (arrows) in patient no. 1.

Results

Patient no. 1, a right-handed male of 60 years, suddenly developed a left hemiplegia with hemianesthesia. In the acute phase he was aware of his deficit (no anosognosia), but he had the feeling of having lost his left hemibody and he was unable to identify his own left limbs with his right hand (hemiasomatognosia). At the time of SEP recording 6 weeks later, the CT scan disclosed hypodense areas in the posterior and median right thalamus, in the territory of the right posterior cerebral artery (Fig. 1). There was a clinical loss of tactile, vibration, joint position, cold and warm sensations on the left hemibody (including the face). The patient did not react to painful cutaneous stimuli to the left side, but had a mimic of pain for passive forceful extension of his left fingers. No painful overreactions were elicited to repetitive cutaneous stimuli on the left side. There was also a left hemiparesis with brisk tendon reflexes and a left homonymous hemianopia. The EEG showed frequent right temporal bursts of delta waves at 3/sec.

Stimulation of the median nerve on the normal side elicited normal SEP components, with P9-N11-N13 at the neck (Fig. 2A) and P9-P11-P13-P14 far fields all over the scalp (B, C). The contralateral postrolandic N20-P27-P45 clearly diverged from the superimposed ipsilateral trace

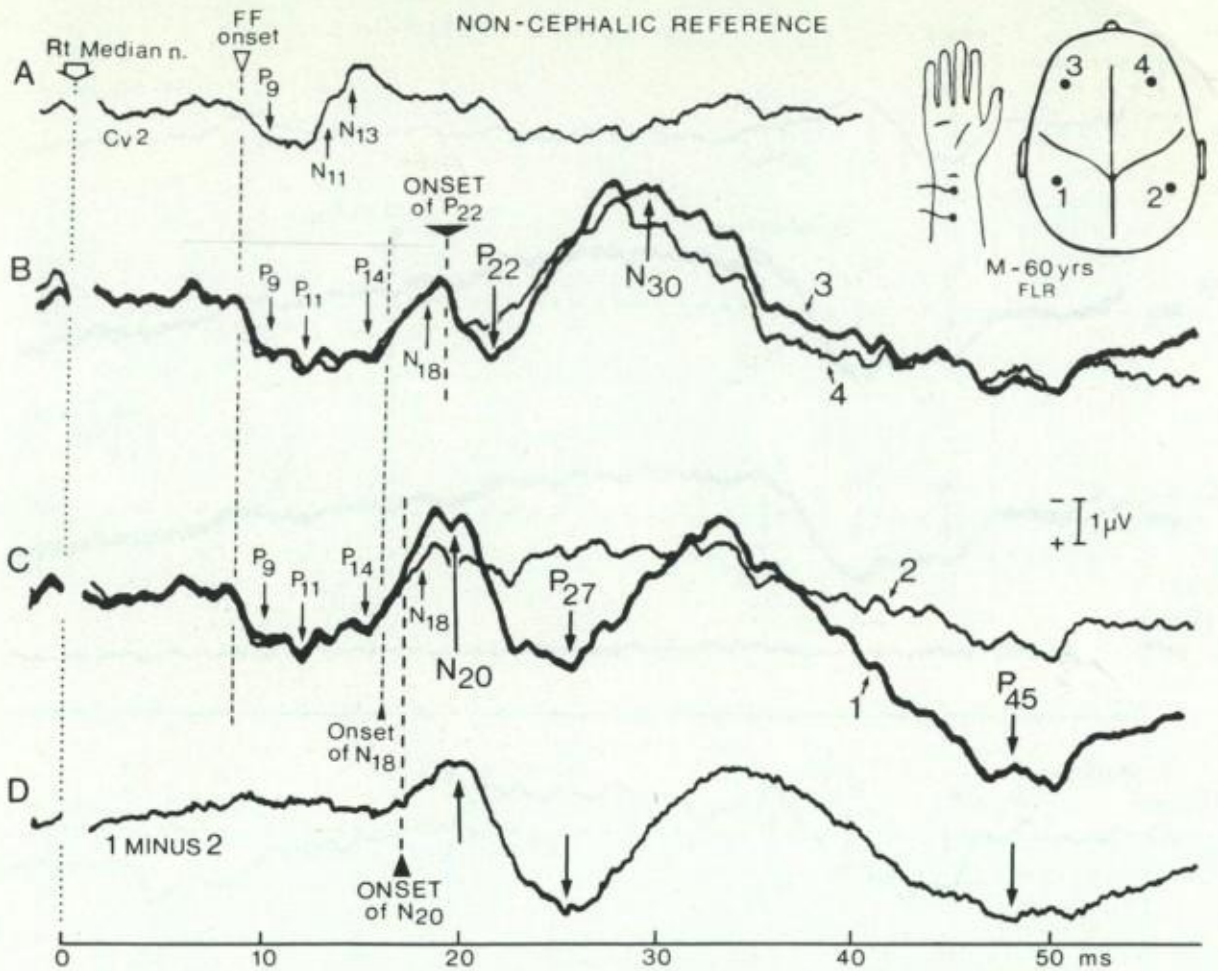

Fig. 2. Non-cephalic reference recording of SEPs to stimulation of the right median nerve on the normal side in patient no. 1. All components are identified by their polarity (P for positive and N for negative) and mean peak latency in subjects of standard size, as recommended by Donchin et al. (1977). A: active electrode over the spinous process of the second cervical vertebra (the onset of N11 at that level is slightly later than the spinal entry time; see Desmedt and Cheron 1980a, Figs. 1 and 2). B: superimposed traces recorded from the contralateral (thicker trace) and ipsilateral frontal scalp. C: superimposed traces recorded from the contralateral (thicker) and ipsilateral parietal scalp. D: algebraic subtraction of the ipsilateral parietal trace from the contralateral parietal trace. This virtually eliminates all positive far fields and the N18. The vertical interrupted line indicates onset of far field (FF). The onset of prerolandic P22 is slightly later than that of the parietal N20 as previously described (see Desmedt and Cheron 1981b; Mauguière et al. 1983).

which only showed a prolonged negativity N18. Algebraic subtraction of the ipsilateral from the contralateral parietal trace removed the P9-P11-P13-P14-N18 components (with fairly equal bilateral size) and left the contralateral cortical N20-P27-P45 components (Fig. 2D). At the front, a symmetrical N18 effect was detected before the prerolandic P22-N30 components which were slightly larger contralaterally (Fig. 2B) (Desmedt and Cheron 1981b).

Stimulation of the left median nerve on the affected side elicited rather similar P9-N11-N13 at the neck (Fig. 3A) and P9-P11-P13-P14 far fields at the scalp (B, C); the latter were followed by a rather prolonged N18 seen as a negative dome extending to about 35 msec which was symmetri-

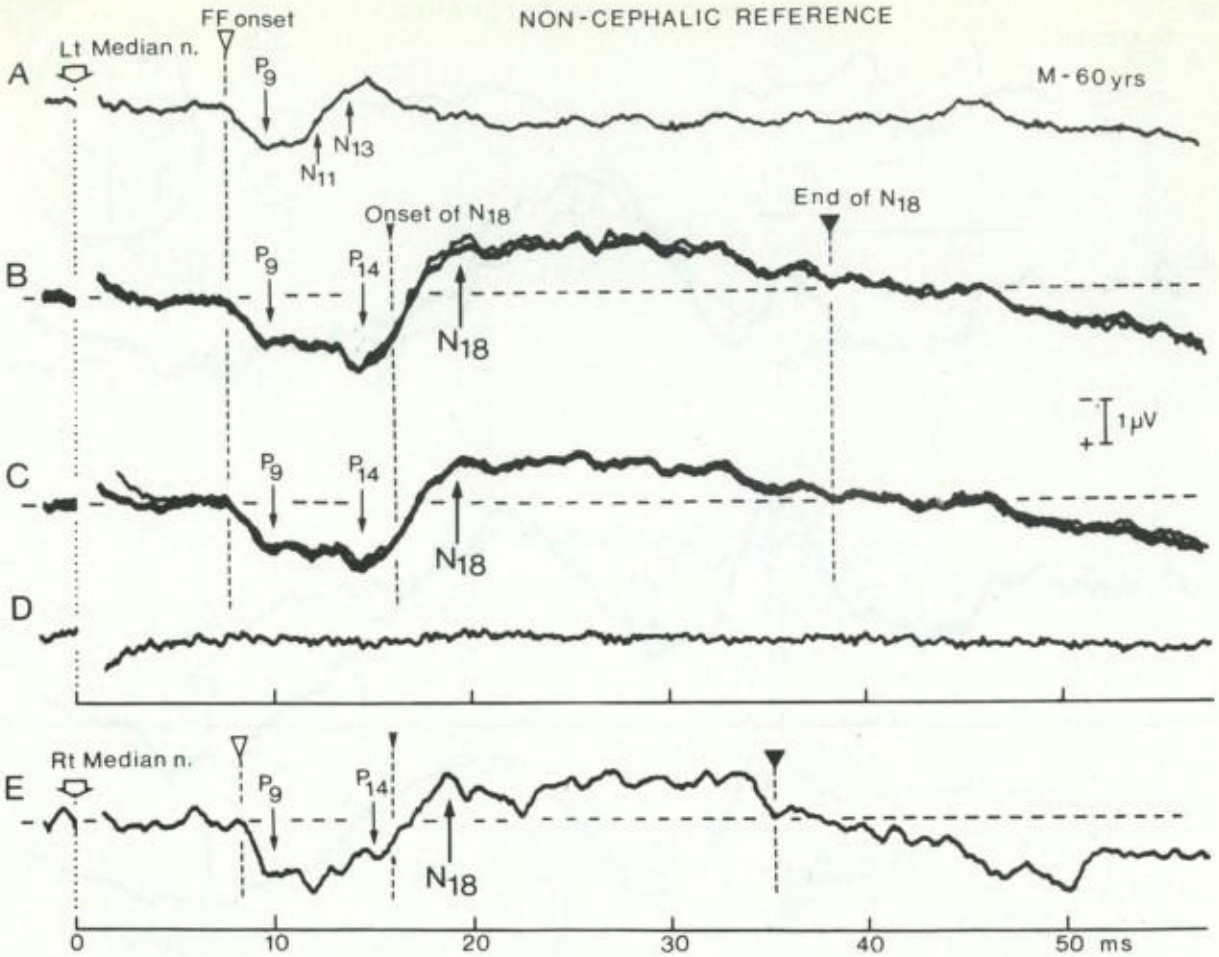

Fig. 3. SEPs to stimulation of the left median nerve on the affected side in patient no. 1. Same presentation of the traces in A–D. E: the ipsilateral parietal response to right median nerve stimulation is reproduced from Fig. 1C for comparison. The presumed onset and end of N18 are indicated by vertical interrupted lines.

cal. Indeed algebraic subtraction of the parietal traces resulted in a fairly straight line (D). The absence of any postcentral N20-P27-P45 or prerolandic P22-N30 cortical components was in line with the massive deafferentation by the thalamic lesion and with the clinical signs. The N18 looked rather similar to the N18 recorded ipsilaterally for stimulation on the normal side (Fig. 3E).

Patient no. 2, a right-handed male of 77 years, presented a sudden right hemiplegia with transient aphasia and a complete right hemianesthesia (including the right hemiface). At the time of SEP study 9 months later, the CT scan disclosed a small lacunar hypodensity in the left posterior thalamus and posterior limb of the internal capsule (Fig. 4). Besides the right hemiparesis, there was a complete loss of joint position sense on the right side. Tactile thresholds were nearly symmetrical, but the two-point discrimination was drastically impaired on the right. Astereognosis and loss of graphesthesia were prominent on the right side. Cold or warm stimuli were correctly discriminated, but perceived as unpleasant or even painful on the right side. Repeating pinpricks gradually triggered painful overreactions. The EEG was normal. Stimulation of the left median nerve on the normal

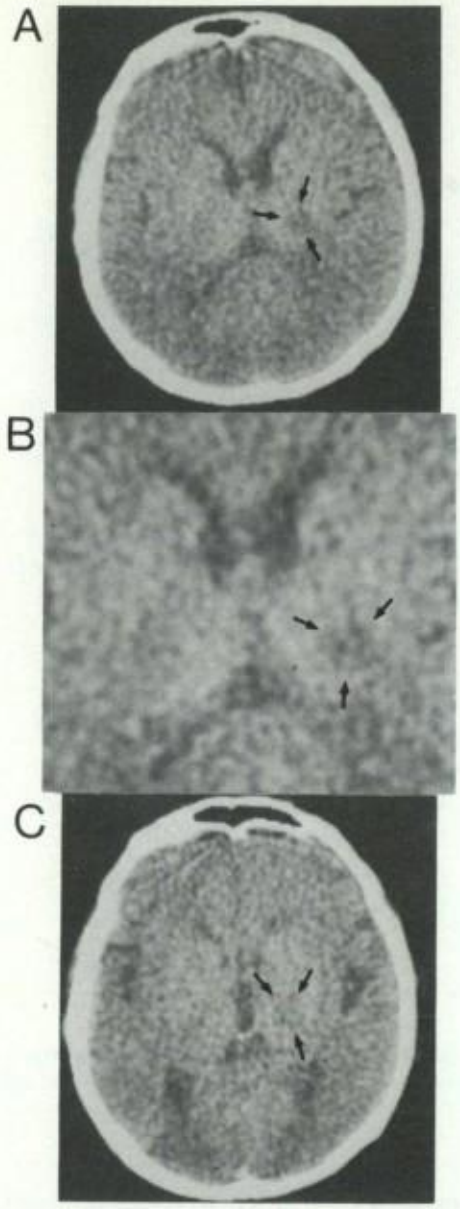

Fig. 4. Horizontal CT scan sections showing the left thalamic vascular lesion in patient no. 2. A: small laminar hypodensity in the left posterior thalamus and posterior limb of the internal capsule. B: enlarged view of the lesion site in A. C: CT scan section at a slightly higher level. The small arrows point to the lesion.

side elicited fairly normal SEPs with P9-P14 far fields and N18 (Fig. 5A, B). The P11 was not clearly delineated in this patient, as occasionally occurs in normals (see Desmedt and Cheron 1981b, page 563). The large postcentral P27-P45 and (contralateral) prerolandic P22 contrasting with the rather small N30 in this patient of 77 years was in line with the aging trends reported for normal octogenarians (Desmedt and Cheron 1980b, 1981b). Stimulation of the right median nerve on the affected side only elicited positive P9-P14 far fields and a prolonged N18 (Fig. 6A, B). Algebraic subtraction of the parietal traces yielded a fairly straight line (C).

Patient no. 3, a right-handed female of 60 years, suddenly developed a right hemiplegia with aphasia while on anticoagulant therapy. At SEP study 1 month later, the CT scan disclosed a large hemorrhagic lesion in the left fronto-parieto-temporal areas and subcortical white matter. Besides an upper motoneuron deficit in the right upper limb and hemiface, there was a complete loss of joint position and two-point discrimination on the right side, as well as astereognosis and loss of graphesthesia. Vibration, cold and warm sensations were preserved. The EEG showed a steady delta slowing at 3/sec over the right parieto-temporal scalp. The SEPs to left median nerve stimulation (normal side) presented the characteristic profile. The SEPs to stimulation of right median nerve on the affected side only showed the P9-P14 far fields and a symmetrical N18, with no subsequent cortical SEP components.

Patient no. 4, a right-handed female of 79 years, suddenly developed a right hemiplegia and hemianesthesia with transient aphasia. At the time of SEP study 3 months later, the CT scan showed two lacunar hypodensities in the caudal part of the left thalamus and in the white matter of the left occipital lobe (left posterior cerebral artery thrombosis). The upper motoneuron deficit had virtually cleared, but there was a massive hemianesthesia with loss of tactile, pinprick, vibration, joint position and thermo-algesic sensations on the right side and hemiface. There was no spontaneous pain, nor any overreaction to repeated nociceptive stimulation. There was also a right homonymous hemianopia. The SEPs to left median nerve stimulation were within normal range for age. Stimulation of the right median nerve on the affected side elicited only the P9-P14 far fields and a symmetrical N18, as in the previous patients.

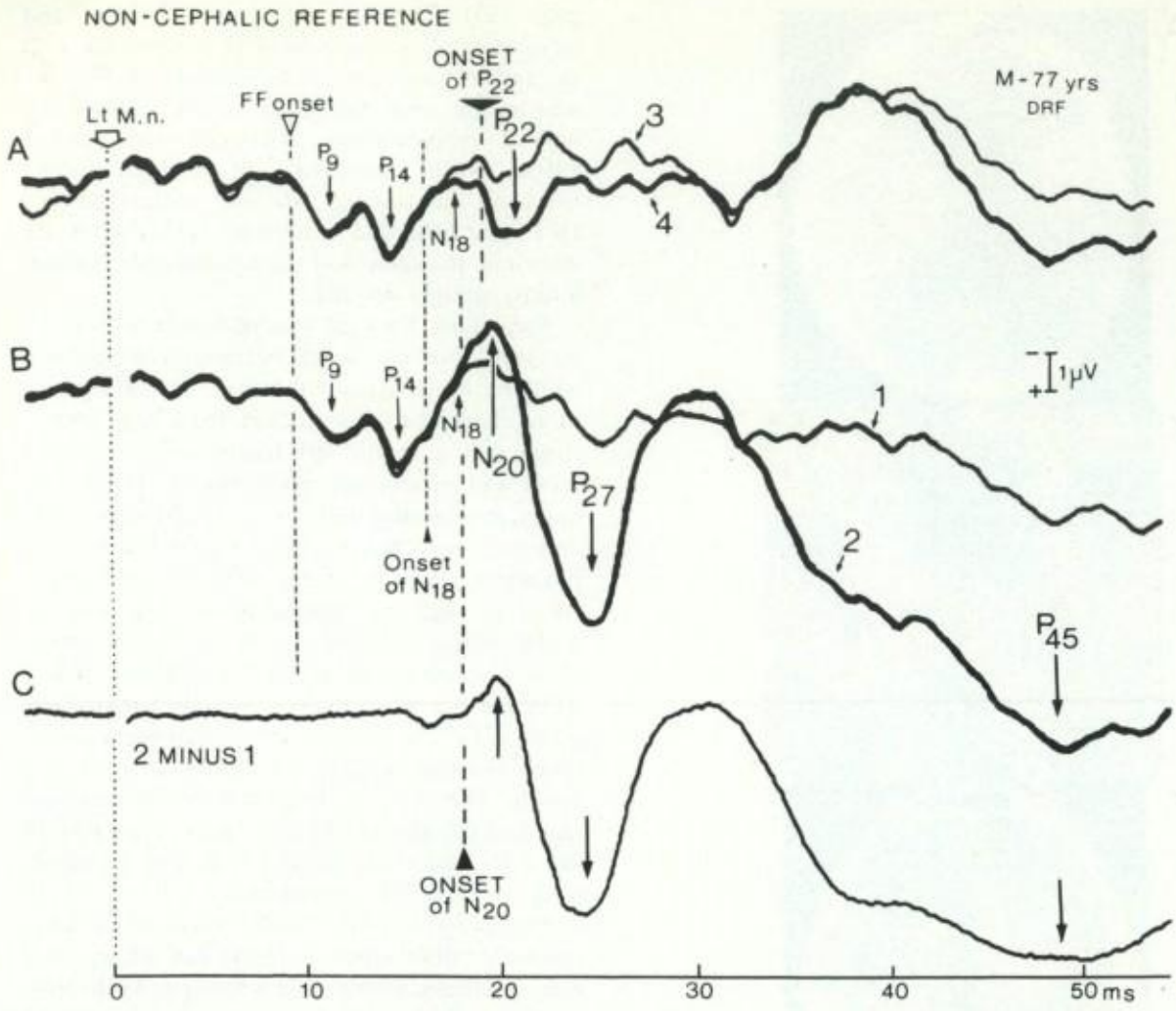

Fig. 5. SEPs to stimulation of the left median nerve on the normal side in patient no. 2. A: frontal traces. B: parietal traces superimposed (contralateral trace thicker). C: algebraic subtraction of the ipsilateral parietal trace from the contralateral parietal trace. The frontal N30 component is small in this old patient.

The peak voltages of the P9 or P14 far fields were measured from baseline at the frontal or the parietal derivations in the 4 patients (Table I). The voltage of N18 measured from its onset inflexion just after P14 (see Figs. 3, 5, 6) averaged 1.6 μV. These values were not significantly different whether the SEPs were elicited by stimulation of the normal or the affected side (Table I). The peak voltages of N18 were somewhat larger than the mean 0.6 μV reported by Desmedt and Cheron (1981b, Table I), while our mean voltage of 1.2 μV for N20 (measured by difference from N18; see Figs. 2C and 5B) roughly agreed with their mean of 0.92 ± 0.3 μV in healthy octogenarians. The loss

TABLE I

Mean voltage of early SEP components (μV) ± S.D. (n = 8).

	Normal side	Affected side	*t* test by pairs
P9	1.41 ± 0.17	1.24 ± 1.9	$P > 0.8$
P14	1.76 ± 2.21	1.74 ± 1.77	$P > 0.9$
N18	1.55 ± 0.42	1.70 ± 1.36	$P > 0.7$

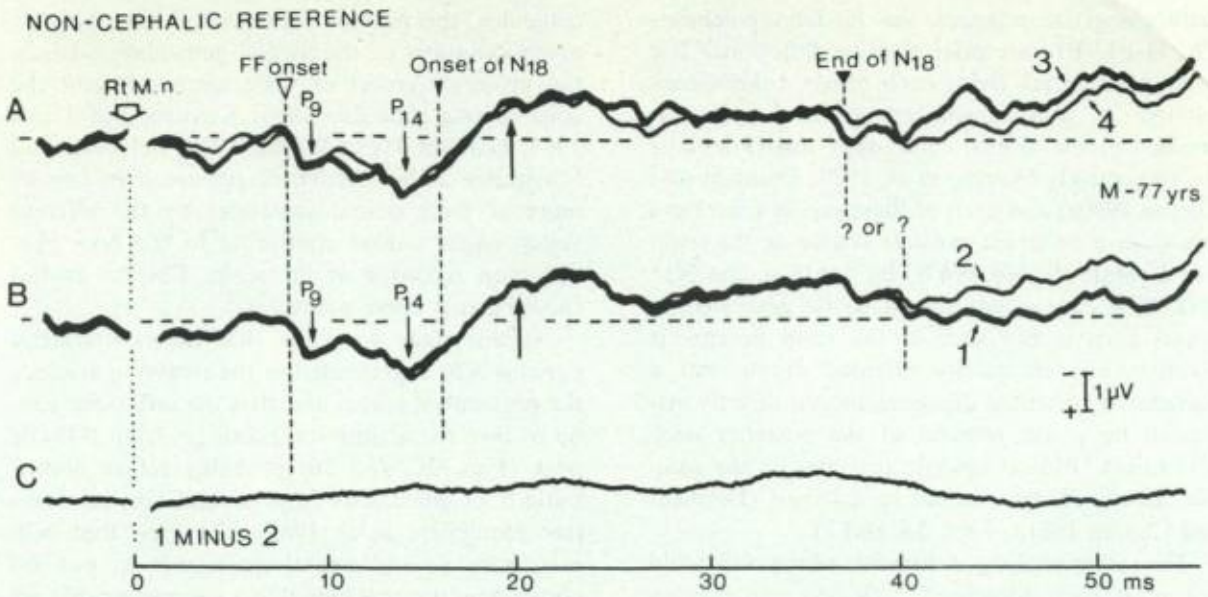

Fig. 6. SEPs to stimulation of the right median nerve on the affected side in patient no. 2. Same presentation of the traces. The presumed onset and end of N18 are indicated by vertical interrupted lines.

of the cortical SEP components for stimulation on the affected side of our patients uncovered surprisingly prolonged N18 whose total mean duration was estimated as 19 msec (range 17–24 msec) (Figs. 3B, C, E and 5A, B).

Discussion

Early SEP negativities to median nerve stimulation in man have been raising issues that must be resolved for a correct interpretation of the abnormal profiles recorded in patients with central nervous system lesions. A better understanding of these issues resulted from the proposal that several distinct neural generators are involved and that the classical contralateral parietal N20 should be differentiated both from the prerolandic P22-N30 SEP components (Desmedt and Cheron 1980b; Mauguière et al. 1983) and from the bilateral N18 SEP component (Desmedt and Cheron 1981b). SEP components can be distorted or cancelled in montages with a scalp reference (Figs. 2D, 3D and 6C), and N18 is best displayed by using a non-cephalic reference. In normals, N18 seems to occur in virtual isolation at the ipsilateral parietal derivation, whereas other SEP components superimpose at the contralateral parietal derivation (N20-P27-P45) or at the front (P22-N30): the underlying N18 is suggested by the inflexions with bilateral congruence on the rising limb of SEP just after the P14 far field (Desmedt and Cheron 1981b).

The present clinical data support this view by showing that virtually identical N18 components occur on both sides in patients with a unilateral thalamic and/or suprathalamic lesion (Figs. 1 and 4) that eliminates the parietal N20-P27-P45 and the prerolandic P22-N30 SEP components (Figs. 3 and 6). Such a lesion does not affect the earlier P9-P11-P14 scalp far fields (Nakanishi et al. 1978; Anziska and Cracco 1980; Mauguière and Courjon 1981) which are generated below the thalamus, the P14 peak probably representing the arrival of the lemniscal volley at the thalamic VPLc relay nucleus (Arezzo et al. 1979; Desmedt and Cheron 1980a, 1981a, b).

The persistence of an unreduced N18 after such a lesion suggests that this component cannot be generated in either thalamo-cortical radiations or cortex. Why then should the N18 polarity be

surface-negative whereas the far-field potentials P9-P11-P13-P14 are brief positive deflexions? The latter positive far fields each reflect volume-conduction of a synchronized afferent volley at brachial plexus, dorsal columns or medial lemniscus respectively (Arezzo et al. 1979; Desmedt and Cheron 1980a) and each of these dipole generators results in a coherent positive source at the scalp (see Lorente de Nó 1947). By contrast, the N13 neck SEP component shown to be generated in dorsal horn is not seen at the scalp because it involves, not an axially oriented dipole, but a horizontally oriented dipole as indeed directly evidenced by phase reversal of the posterior neck N13 into a 'P13' at oesophageal sites on the anterior aspect of the cervical spinal cord (Desmedt and Cheron 1981a; Figs. 5A and 7).

The rather prolonged duration of the N18 could not have been identified with any certainty in normals (Desmedt and Cheron 1981b) and it represents an intriguing finding in our patients (Figs. 3 and 6). We do not see N18 as a unitary phenomenon, and the small inflexions seen in the N18 profiles would suggest several generators. N18 appears to start at about 15 msec and shows one or more inflexions on its rising limb. N18 generally presents a positive dip at 21–25 msec and it appears to terminate at a mean latency of 34 msec, thus about 19 msec after its onset.

If N18 is not generated above the thalamus as the present lesion data strongly suggest, it must represent a far-field potential whose neural generators would not be a conducted spike volley, but rather activity in 'open-field systems' with coherent geometry (Humphrey 1968; Klee and Rall 1977) in the brain stem (cf. Hashimoto, personal communication) and/or in some uninvolved parts of the thalamus (cf. Fukushima et al. 1976). While the classical projection of the dorsal column (cuneate) nucleus is directed to the contralateral VPL somatosensory relay nucleus in thalamus, there is anatomical evidence in cats that the cuneate also sends fibers to several brain stem nuclei, such as: the medial and dorsal accessory olives, the basal and lateral regions of the rostral inferior colliculus, the ventrolateral parts of the superior colliculus, the nucleus suprageniculatus, the pars magnocellularis of the medial geniculate nucleus, the posterior group of thalamic nuclei and the zona incerta (Bowsher 1961; Kuypers and Tuerk 1964; Hand and Van Winkle 1977; Baleydier and Mauguière 1978). Activities generated in one or more of these neural structures by the afferent volley might indeed contribute to the N18 phenomenon recorded at the scalp. Further studies should clarify these points.

At this stage we think that the contralateral parietal N20 is generated in the receiving areas of the postcentral cortex and that the inflexions seen on its own rising limb (that diverges from N18) or peak (Figs. 2C and 5B) probably reflect contributions of generators from several cortical areas (see Mauguière et al. 1983). The view that N20 may have several neural generators in parietal cortex does not preclude that a completely distinct N18 phenomenon accounts for the earlier portion of the SEP negativity. The N18 starts earlier than the N20, and it features a prolonged wave form rising from the end of the P14 far field. The widespread distribution of N18 and its sizeable voltage at the right and left scalp sites (Table I) suggest that it corresponds to deeply situated neural generators. The issue raised by Kritchevsky and Wiederholt (1978) thus appears genuine and it can indeed be resolved as proposed by Desmedt and Cheron (1981b) by dissociating the widespread N18 from the contralateral parietal N20 SEP components.

This distinction is important for practical clinical uses of SEP in patients with focal central nervous lesions. The use of a non-cephalic reference electrode is valuable for differentiating the various cortical components (Desmedt and Cheron 1981b; Mauguière et al. 1983). Furthermore it is necessary to differentiate the distinct negative SEP components that have now been shown to be related to separate neural generators, such as the postcentral cortical N20, the prerolandic cortical N30 or the bilateral far-field N18 of subcortical origin. One of these negative components can persist while others are lost as a result of a focal lesion, and this finding is clinically relevant.

Summary

Somatosensory evoked potentials (SEPs) to electrical stimulation of the right or left median nerve were studied in 4 patients with hemianesthesia and a severe thalamic or suprathalamic vascular lesion on one side. The SEPs were recorded with a non-cephalic reference. The normal side of each patient served as his or her own control. The lesion consistently abolished the parietal N20-P27-P45 and the prerolandic P22-N30 SEP components. It did not significantly affect the P9-P11-P14 positive far fields, nor the widespread bilateral N18 SEP component. This allowed N18 features to be studied without interference from cortical components. It is proposed that N18 reflects several deeply located generators in brain stem and/or thalamus whereas N20 represents the earliest cortical response of the contralateral postcentral receiving areas.

Résumé

Générateurs des composantes far field P14 et N18 des potentiels évoqués somesthésiques: malades avec lésion du thalamus ou des radiations thalamo-corticales

Les potentiels évoqués somesthésiques (PES) à la stimulation électrique du nerf médian gauche ou droit ont été étudiés chez 4 malades avec une lésion vasculaire grave thalamique ou suprathalamique unilatérale et une hémianesthésie clinique. Les PES ont été enregistrés avec référence non-céphalique. Le côté normal a servi de contrôle. La lésion a chaque fois aboli les composantes pariétales N20-P27-P45 et prérolandiques P22-N30. Elle n'a pas modifié significativement les far fields P9-P11-P14, ni la composante diffuse bilatérale N18. Il a donc été possible d'étudier le N18 sans interférence des composantes corticales. Le N18 est produit par plusieurs générateurs profonds situés dans le tronc cérébral et/ou le thalamus, alors que le N20 représente la réponse corticale précoce des aires postcentrales de projection somesthésique.

References

Anziska, B. and Cracco, R.Q. Short latency somatosensory evoked potentials: studies in patients with focal neurological disease. Electroenceph. clin. Neurophysiol., 1980, 49: 227–239.

Arezzo, J., Legatt, A.D. and Vaughan, H.G. Topography and intracranial sources of somatosensory evoked potentials in the monkey: early components. Electroenceph. clin. Neurophysiol., 1979, 46: 155–172.

Baleydier, C. and Mauguière, F. Projections of the ascending somesthetic pathways to the cat superior colliculus visualized by the HRP technique. Exp. Brain Res., 1978, 31: 43–50.

Bowsher, D. The termination of secondary somatosensory neurons within the thalamus of *Macaca mulatta*: an experimental degeneration study. J. comp. Neurol., 1961, 117: 213–227.

Chiappa, K.H. and Ropper, A.H. Evoked potentials in clinical medicine. New Engl. J. Med., 1982, 306: 1205–1211.

Chiappa, K.H., Choi S.K. and Young, R.R. Short-latency somatosensory evoked potentials following median nerve stimulation in patients with neurological lesions. In: J.E. Desmedt (Ed.), Clinical Uses of Cerebral, Brainstem and Spinal Somatosensory Evoked Potentials, Progr. Clin. Neurophysiol., Vol. 7. Karger, Basel, 1980: 264–281.

Colon, E.J., Joosten, E. and De Weerd, J. Somatosensory evoked responses in controlled A-alpha sensory fiber disease. J. Neurol., 1978, 219: 273–278.

Cracco, R.Q. and Cracco, J.B. Somatosensory evoked potentials in man: far-field potentials. Electroenceph. clin. Neurophysiol., 1976, 41: 460–466.

Desmedt, J.E. Somatosensory cerebral evoked potentials in man. In: A. Rémond (Ed.), Handbook of Electroencephalography and clinical Neurophysiology, Vol. 9. Elsevier, Amsterdam, 1971: 55–82.

Desmedt, J.E. Some observations on the methodology of cerebral evoked potentials in man. In: J.E. Desmedt (Ed.), Attention, Voluntary Contraction and Event-Related Cerebral Potentials, Progr. Clin. Neurophysiol., Vol. 1. Karger, Basel, 1977: 12–29.

Desmedt, J.E. and Cheron, G. Central somatosensory conduction in man: neural generators and interpeak latencies of the far-field components recorded from neck and right or left scalp and earlobes. Electroenceph. clin. Neurophysiol., 1980a, 50: 382–403.

Desmedt, J.E. and Cheron, G. Somatosensory evoked potentials to finger stimulation in healthy octogenarians and in young adults: wave forms, scalp topography and transit times of parietal and frontal components. Electroenceph. clin. Neurophysiol., 1980b, 50: 404–425.

Desmedt, J.E. and Cheron, G. Prevertebral (oesophageal) recording of subcortical somatosensory evoked potentials in man: the spinal P13 component and the dual nature of the spinal generators. Electroenceph. clin. Neurophysiol., 1981a, 52: 257–275.

Desmedt, J.E. and Cheron, G. Non-cephalic reference recording of early somatosensory potentials to finger stimulation in adult or aging man: differentiation of widespread N18 and contralateral N20 from the prerolandic P22 and N30 components. Electroenceph. clin. Neurophysiol., 1981b, 52: 553–570.

Desmedt, J.E. and Manil, J. Somatosensory evoked potentials of the normal human neonate in REM sleep, in slow wave sleep and in waking. Electroenceph. clin. Neurophysiol., 1970, 29: 113–126.

Desmedt, J.E. and Robertson, D. Differential enhancement of early and late components of the cerebral somatosensory evoked potentials during fast sequential cognitive tasks in man. J. Physiol. (Lond.), 1977, 271: 761–782.

Donchin, E., Callaway, E., Cooper, R., Desmedt, J.E., Goff, W.R., Hillyard, S.A. and Sutton, S. Publication criteria for studies of evoked potentials: report of a committee. In: J.E. Desmedt (Ed.), Attention, Voluntary Contraction and Event-Related Cerebral Potentials, Progr. Clin. Neurophysiol., Vol. 1. Karger, Basel, 1977: 1–11.

Eisen, A. The somatosensory evoked potential. J. Canad. Sci. Neurol., 1982, 9: 65–77.

El-Negamy, E. and Sedgwick, E.M. Delayed cervical somatosensory potentials in cervical spondylosis. J. Neurol. Neurosurg. Psychiat., 1979, 42: 238–241.

Fukushima, T., Mayanagi, Y. and Bouchard, G. Thalamic evoked potentials to somatosensory stimulation in man. Electroenceph. clin. Neurophysiol., 1976, 40: 481–490.

Giblin, D.R. Somatosensory evoked potentials in healthy subjects and in patients with lesions of the nervous system. Ann. N.Y. Acad. Sci., 1964, 112: 93–142.

Hand, P.J. and Van Winkle, T. The efferent connections of the feline nucleus cuneatus. J. comp. Neurol., 1977, 171: 83–110.

Hume, A.M. and Cant, V.R. Conduction time in central somatosensory pathways in man. Electroenceph. clin. Neurophysiol., 1978, 45: 361–375.

Hume, A.L. and Cant, B.R. Central somatosensory conduction after head injury. Ann. Neurol., 1981, 10: 411–419.

Humphrey, D.R. Re-analysis of the antidromic cortical response. On the contribution of cell discharge and PSPs to the evoked potentials. Electroenceph. clin. Neurophysiol., 1968, 25: 421–442.

Jones, S.J. Investigations of brachial plexus traction lesions by peripheral and spinal somatosensory evoked potentials. J. Neurol. Neurosurg. Psychiat., 1979, 42: 107–116.

Kimura, J., Yamada, T. and Kawamura, H. Central latencies of somatosensory cerebral evoked potentials. Arch. Neurol. (Chic.), 1978, 35: 683–688.

Klee, M. and Rall, W. Computed potentials of cortically arranged populations of neurons. J. Neurophysiol., 1977, 40: 647–666.

Kritchevsky, M. and Wiederholt, W.C. Short-latency somatosensory evoked responses in man. Arch. Neurol. (Chic.), 1978, 35: 706–711.

Kuypers, H.G.J.M. and Tuerk, J.D. The distribution of cortical fibers within the nuclei cuneatus and gracilis in the cat. J. Anat. (Lond.), 1964, 98: 143–162.

Lesser, R.P., Lueders, H., Hahn, J. and Klem, G. Early somatosensory potentials evoked by median nerve stimulation: intraoperative monitoring. Neurology (Minneap.), 1981, 31: 1519–1523.

Lorente de Nó, R. A study of nerve physiology. Stud. Rockefeller Inst. 1947, 132: Ch. 16.

Mauguière, F. and Courjon, J. The origin of short-latency somatosensory evoked potentials in man. Ann. Neurol., 1981, 9: 707–710.

Mauguière, F., Desmedt, J.E. and Courjon, J. Astereognosis and dissociated loss of frontal or parietal components of somatosensory evoked potentials in hemispheric lesions: detailed correlations with clinical signs and computerized tomographic scanning. Brain, 1983, 106: 271–311.

Nakanishi, T., Shimada, Y., Sakuta, M. and Toyokura, Y. The initial positive component of scalp-recorded somatosensory evoked potentials in normal subjects and in patients with neurological disorders. Electroenceph. clin. Neurophysiol., 1978, 45: 26–34.

Noel, P. and Desmedt, J.E. Somatosensory cerebral evoked potentials after vascular lesions of the brainstem and diencephalon. Brain, 1975, 98: 113–128.

Noel, P. and Desmedt, J.E. Cerebral and farfield somatosensory evoked potentials in neurological disorders. In: J.E. Desmedt (Ed.), Clinical Uses of Cerebral, Brainstem and Spinal Somatosensory Evoked Potentials, Progr. Clin. Neurophysiol., Vol. 7. Karger, Basel, 1980: 205–230.

Siivola, J., Sulg, I. and Heiskari, M. Somatosensory evoked potentials in cervical spondylosis and herniated disc. Electroenceph. clin. Neurophysiol., 1981, 52: 276–282.

Small, D.G., Beauchamp, M. and Matthews, W.B. Subcortical somatosensory evoked potentials in normal man and patients with central nervous system lesions. In: J.E. Desmedt (Ed.), Uses of Cerebral, Brainstem and Spinal Somatosensory Evoked Potentials, Progr. Clin. Neurophysiol., Vol. 7. Karger, Basel, 1980: 190–204.