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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 [1]

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The recording of sensory action potentials from peripheral nerves (Gilliatt and Sears 1958; Kaeser 1970) up to Erb's point and the averaging of the early cortical response recorded from the parietal scalp (Giblin 1964) can be combined to evaluate conduction along the somatosensory pathway and, in particular, the transit time between arrival at the spinal cord and cortex (Desmedt 1971; Desmedt et al. 1973). Attempts to analyse central conduction more directly have employed the averaging of spinal evoked potentials recorded over the neck (Liberson and Kim 1963; Cracco 1973; Matthews et al. 1974; Cracco and Cracco 1976) or intrathecal recording from the cervical spinal cord (Shimoji et al. 1972; Ertekin 1973, 1976). On the other hand, the use of non-cephalic reference electrodes helped identify several far-field components that precede the cortical somatosensory evoked potential (SEP) and presumably reflect subcortical events (Trojaborg and Jorgensen 1973; Cracco and Cracco 1976; Anziska et al. 1978; Cracco 1980). Various electrode montages have been in use which can reportedly enhance some of these waves (Dorfman 1977; Jones 1977; Abbruzzese et al. 1978; El-Negamy and Sedgwick 1978; Hume and Cant 1978; Kimura et al. 1978; Kritchevsky and Wiederholt 1978; Shimoji et al. 1978; Eisen and Odusote 1980; Wiederholt 1980). The identification of the different waves described in different papers is still uncertain and their interpretations in terms of possible neural generators remains obscure. For example, Cracco (1973) observed a latency shift of about 0.8 msec for a spinal wave recorded from the neck at the C7 and C2 levels whereas Matthews et al. (1974; cf. also Jones 1977; Small et al. 1980) did not observe any consistent latency change and therefore favoured fixed generator sites. There are in fact no satisfactory estimates of the conduction velocities up the cervical dorsal columns, the medial lemniscus and the thalamo-cortical radiations. These uncertainties are unfortunate in view of the anticipated clinical uses of the early SEP components for localizing focal spinal or brain stem lesions in patients (cf., Desmedt and Noël 1973; Noël and Desmedt 1975, 1980; Chiappa et al. 1980; Small et al. 1980; Stockard and Sharbrough 1980; see reviews in Halliday 1978; Desmedt 1980).

This paper presents findings in normal adults and attempts to resolve some of the current issues by comparing a larger-than-usual number of simultaneously recorded sites, and by using montages with non-cephalic as well as cephalic references. In addi-

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tion, the geometrical relationships between brain stem, spinal cord, and cranio-vertebral landmarks were studied on fixed anatomical specimens to reveal likely neural correlates of the successive early SEP components. Conduction times along the 3 afferent neurones of the somatosensory pathway are discussed in relation to the combined evidence available.

Material and methods

Eighteen experiments were carried out on 12 normal volunteers (8 males and 4 females) of 21—30 years (mean age 23 years). They were in good health, free from neurological disease, and had given informed consent. The subjects were mostly students selected from a larger group on the basis of good yields for SEP averaging and ability to relax fully in order to minimize muscle and eyeblink interference. They lay comfortably on a couch in a sound-proofed, electrically shielded and air-conditioned room at 24° C.

The stimuli were 0.2 msec square electrical pulses delivered through a pair of Beckman cup electrodes to the left median nerve just proximal to the wrist. The intensities, checked with a current probe, were 3—6 mA and elicited small thumb twitches. In a few experiments stimuli of 5—8 mA were delivered instead through silver ring electrodes to fingers I—II—III of the left hand, the stimulus to finger I being delayed by 0.5 msec to make up for the shorter distance to the spinal cord (Debecker and Desmedt 1964). The regular or random (Carmeliet et al. 1971) intervals between stimuli were chosen between 0.2 and 0.8 sec. The stimulation rate never exceeded 4/sec. The upper limb was warmed by infrared and its temperature was normal at 35—37° C.

Scalp, earlobes and upper neck sites were recorded from with unvarnished stainless steel needles of 0.2 mm diameter. Jelly-filled cups were placed on the lower neck and on the dorsum of the right hand (non-cephalic reference). Differential amplifiers with 10 MΩ input impedance were connected to one or two 8-channel Hewlett Packard FM magnetic recorder(s), model 3968A, operated at 7 in./sec, and to a Nicolet digital computer, model 1074, with 4096 words of 9 bits. The overall bandpass extended from 2.5 kHz to 0.5 Hz. The bin width was 80 μsec with 1024 points per channel. As a rule 1024 or 2048 samples were averaged off-line after editing the FM taped data to remove muscle and other interference (see Desmedt 1977 for details of methods). The traces were not smoothed and were written out on an X-Y plotter. The Nicolet averager allowed any 2 responses recorded with a common electrode to be electronically added or subtracted in order to display the wave form corresponding to a montage of the other 2 electrodes of the pair. With up to 14 recording channels available it was thus possible to make detailed topographical studies. Component profiles and latencies were found to be consistent in repeat runs on any given subject. The SEP components were labelled from their positive (P) or negative (N) polarity and their peak latency, as recommended by a recent international committee (Donchin et al. 1977). The actual peak latency measured for the component considered was used in the label.

Results

With a non-cephalic electrode on the right hand, the neck and scalp records all disclosed a positive far-field P9 with the same onset latency at about 6 msec (Figs. 1 and 2). This first far-field (FF1) was smaller over the lower neck (Figs. 1F, G and 2F) than over the upper neck or scalp. The SEP recorded from the spinous process of the seventh cervical vertebra presented thereafter a large negative component whose clear onset gradually shifted in latency from lower to upper neck (Fig. 1D—G). At C6-C7 this negativity appeared as an N11 component starting with a latency of about 10 msec (G). At more cephalic levels along the neck the negativity was preceded by

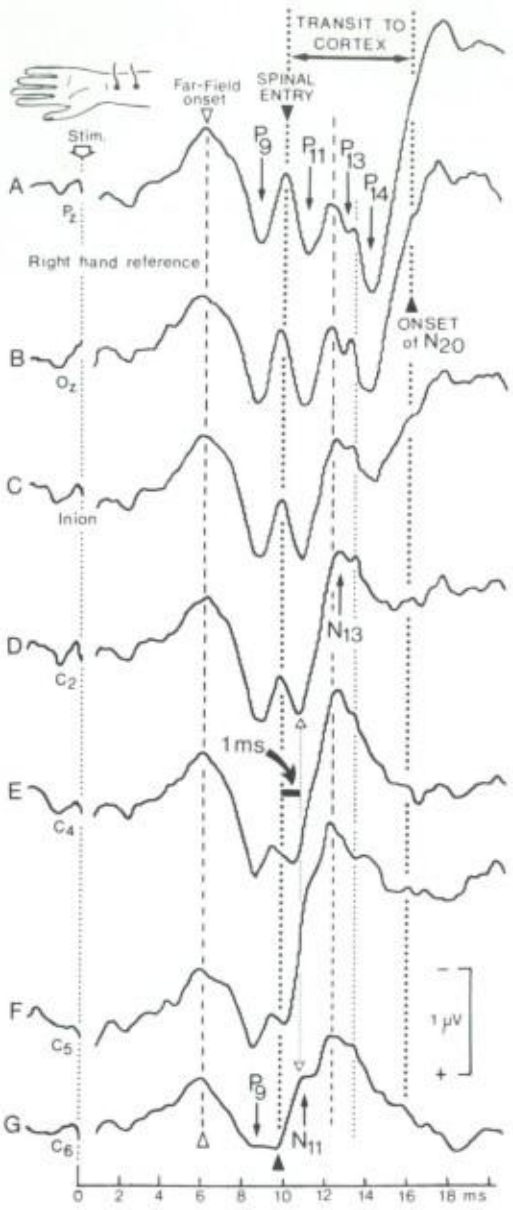

Fig. 1. Far-field SEP components recorded with non-cephalic reference (right hand) in a male subject of 22 years. Stimulation of the left median nerve at the time indicated by the vertical dotted line on the left side. Negativity of the active electrode records upwards in this and all other figures. Same calibration for all traces. Active electrode at the parietal midline Pz (A), occipital midline Oz (B), and 2 cm above the inion (C). The neck recording sites range from 2 cm below the inion (D) to the spinous process of the seventh vertebra (G). The actual spinal cord levels recorded from are discussed in Fig. 6. White arrowhead and vertical interrupted line, onset of first far field which is congruent in all the traces. Black arrowheads and thicker dotted lines, spinal entry time coinciding with onset of spinal negativity N11 at the lower neck (G) or onset of cortical N20 at contralateral parietal scalp. The transit time from spinal entry to cortex is 6.1 msec. Other vertical lines correspond to the onset of N13 (D), of P13 and of P14 (A–C). The latency shift of the negative spinal component is 1.0 msec from lower to upper neck records.

a small positive step (Fig. 1D–F) which could represent volume conduction of an ascending volley; this was not seen in all subjects (Fig. 2D, E). The onset of the N13 component in the upper neck corresponded roughly to the peak of the N11 recorded in the lower neck (Figs. 1D and 2D).

The onset of the neck N11 coincided with the onset of the positive-going second far-field (FF2) or P11 component recorded all over the scalp (Fig. 1A–C). None of these potentials was 'myogenic' since they were recorded in completely relaxed subjects with no residual electromyographic activity in the recorded channels. The N11 and N13 components at the neck were not followed by any well delineated wave. By contrast the scalp leads from 2 cm above the inion (Fig. 1C) disclosed a clear P14 component that increased in amplitude from inion to front (Fig. 1A–C) (cf., Cracco and Cracco 1976). The P14 was generally preceded by a smaller P13 (Fig. 1A). The subject of Fig. 2 showed interesting variations with a rather indistinct FF2 or P11, and a large third far-field (FF3) or P13 component that appeared to overlap and perhaps partially distort the fourth far-field (FF4) or P14 component. Indeed the P13 was small or missing in certain subjects but quite large in others. All these diffuse far fields preceded the cortical N20 component recorded from the contralateral parietal scalp (not illustrated in these figures) whose onset was indicated by the thick vertical dotted line on the right of Figs. 1 and 2.

The difference in onset latencies of N11 from lower neck and N13 from upper neck varied from 0.73 to 1.2 msec in 10 subjects,

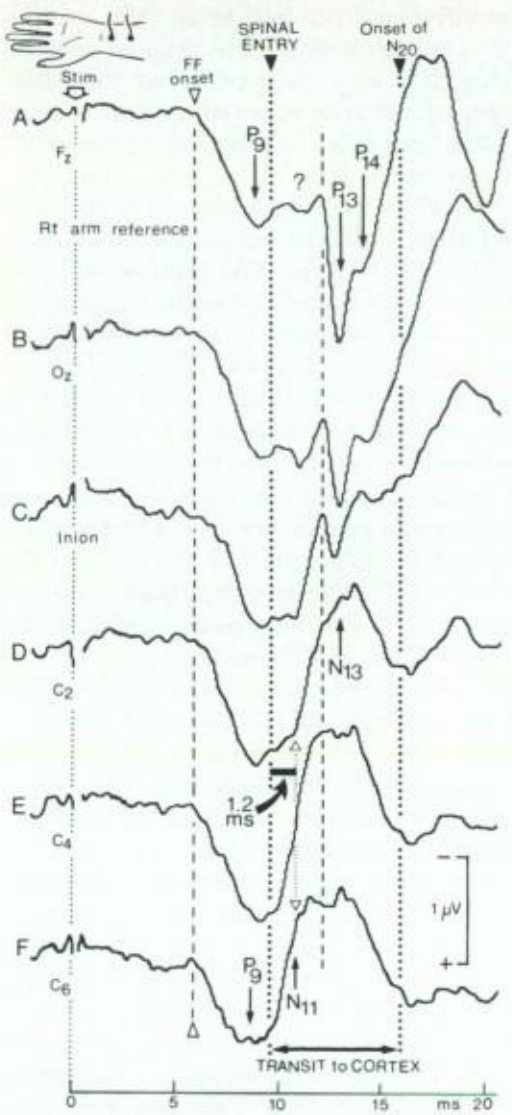

Fig. 2. Far-field SEP components in a female subject of 20 years. Stimulation of left median nerve. Same presentation as in Fig. 1. Notice the virtual lack of FF2 or P11 and the apparent preponderance of P13 over P14 in the scalp records. The transit time from spinal entry to cortex is 6.3 msec. The latency shift of the negative spinal component is 1.2 msec from lower to upper neck records.

and its mean was 0.95 ± 0.15 (S.D.) msec. This result substantiated the report of Cracco (1973) of an onset latency increase with progressively more rostral recording sites; however, it conflicted with the conclusions of Matthews et al. (1974), Jones (1977) and Small et al. (1980), who failed to observe any significant latency difference and concluded that the cervical SEP was 'generated at fixed sites.' We disagree with the latter view which was based on records distorted by the use of a frontal reference, as made clear from Fig. 3.

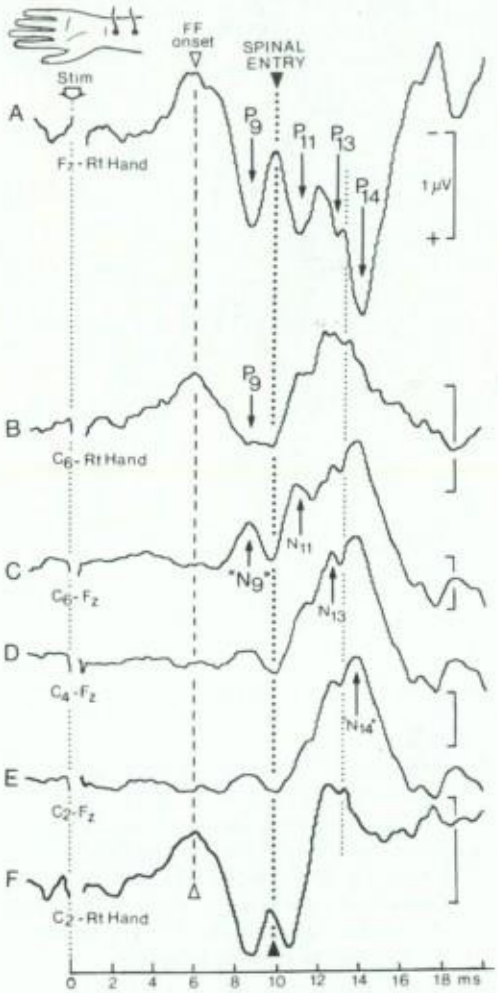

Fig. 3. Effect of using a scalp reference on the neck SEP components. Same subject as in Fig. 1. The reference is non-cephalic (right hand) for A, B and F, and scalp at Fz for B, C and D. The amplification is reduced to half in C, D and E. Spurious negative 'N9' and 'N14' are introduced by the Fz reference, and the latency shift of the negative spinal component is thereby obscured through subtraction of the scalp FF2.

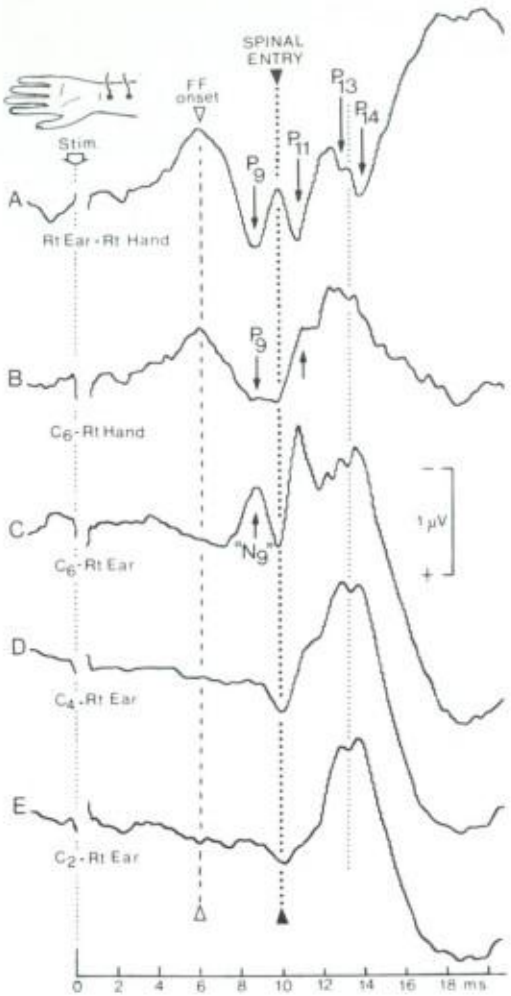

Fig. 4. Effect of using an earlobe reference on the neck SEP components. Same subject as in Fig. 1. The traces with non-cephalic reference (right hand) in A—B allow detailed comparisons. Same amplification throughout. The spurious 'N14' is less obvious in C—E than in Fig. 3B—D since the P14 is small at the earlobe (A). However, the latency shift of the negative spinal component is also obscured through subtraction of the FF2 of the earlobe reference.

With a non-cephalic reference, the midfrontal electrode Fz recorded a P9 (A) that was much larger than the P9 simultaneously recorded from the lower neck with the same reference (B). In the neck-to-Fz montage (C) the large P9 of Fz was thus injected into the grid 2 of the amplifier and resulted in an 'N9' component that substituted for the original neck P9 (B). This 'N9' may have obscured the interpretation of the true onset of the spinal negativity that normally follows the volume-conducted potentials from peripheral nerve. Furthermore, when Fz was connected to grid 2 of the amplifier, the large positive far fields P11 to P14 seen at Fz (Fig. 3A) resulted in negativities that were added to the neck SEP negativity: the latter thereby increased in size and acquired additional peaks (Fig. 3C) (cf., Abbruzzese et al. 1978; Hume and Cant 1978; Small et al. 1980). Such hybrid profiles introduced unnecessary complications into current issues about early SEPs at neck and scalp and their respective generators. For example, the addition of the far-field P11 from Fz to the upper neck SEP obscured the later onset of the negativity at that level as compared to the lower neck (compare E and F in Fig. 3). The use of an Fz reference also introduced a small 'N14' wavelet (Fig. 3C, D, E) which is not a feature of the SEP recorded from the neck with a non-cephalic reference (Fig. 3B, F) but is generated at a neural site above the foramen magnum (Figs. 1A, B, C and 6).

A somewhat similar though less severe distortion occurred when using the earlobe as a reference for the neck electrodes. The earlobe traces presented rather large P9 and P11 far fields whereas the P14 was definitely smaller than at Fz (Fig. 4A). Therefore the later parts of the SEP recorded with an earlobe reference are less distorted by the FF3 and FF4 (C). This is a reason why we used an earlobe reference for assessing the amplitude of the neck SEP in subjects in whom residual EMG activity prevented the use of a non-cephalic reference (Desmedt and Cheron 1980a, b).

Estimation of spinal entry time of the afferent volley

Current uncertainties about the relationships between spinal entry time and the early SEP components were resolved by comparing direct records of the sensory nerve potentials along the peripheral nerve (cf., Desmedt

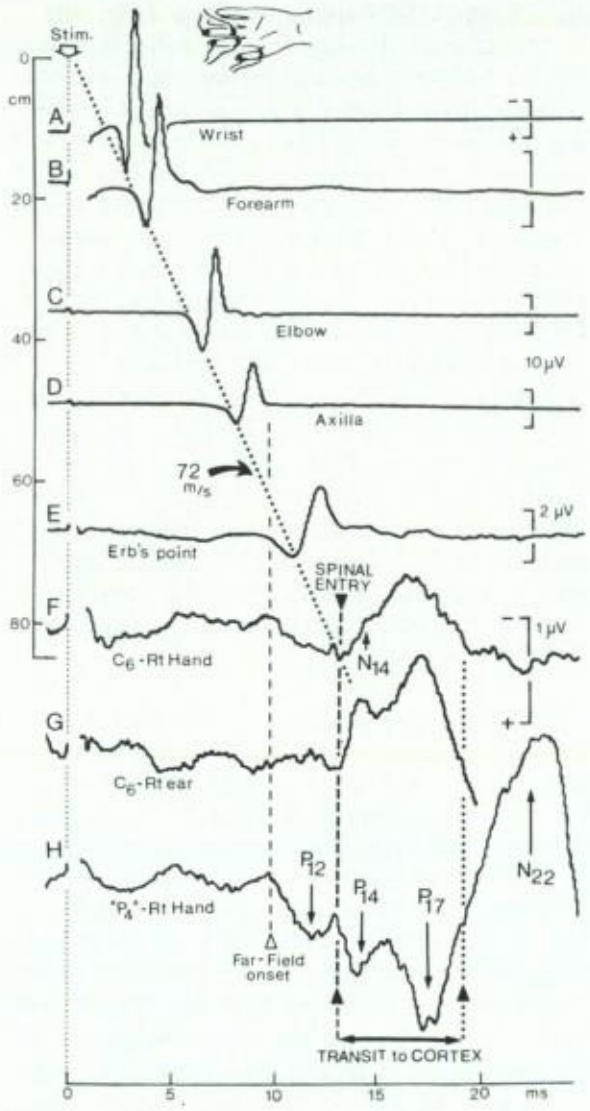

Fig. 5. Sensory nerve potentials recorded from the median nerve at wrist, forearm, elbow, axilla and Erb's point (A—E) with different amplifications. Stimulation at 3 times subjective threshold of fingers I—II—III of the left hand. The vertical separation of the averaged traces is proportional to the conduction distances between the recording sites. The calculated linear regression gives a CV of 72 m/sec. Extrapolation of CV to the lower neck recording site fits the onset of the negative spinal component N14 (F, G), and the onset of the positive scalp far-field FF2 or P14 (H). The onset of the cortical N22 recorded from the contralateral parietal scalp with non-cephalic reference (H) indicates a spino-cortical transit time of 6.0 msec. The onset of the first scalp far-field FF1 or P12 (white arrowhead and vertical interrupted line) is earlier than the Erb's point potential, but later than the nerve potential at the axilla.

1971). For consistent results it was necessary to stimulate fingers rather than mixed nerves in order to exclude group I muscle afferents and antidromic motor axon potentials. The sensory nerve potentials were recorded with fine uncoated needles inserted close to the nerve trunk (reference 3 cm at right angles to the nerve course) and averaged with a bin width of 20 μsec. The onset of the negative-going phase of the triphasic nerve potential indicated arrival of the volley under the recording electrode (Gilliatt and Sears 1958; Debecker and Desmedt 1964). The segment from finger to wrist presented a somewhat slower conduction velocity (CV) due to distal tapering of fibres. Latency data for sites between wrist and Erb's point over the brachial plexus consistently fitted a linear regression with a calculated CV of 72 m/sec (Fig. 5A—E). The mean afferent CV over this stretch was 71.1 ± 4.0 m/sec in 25 healthy adults of mean age 22 years (Desmedt and Cheron 1980b). Extrapolation of the regression to the lower neck recording site fitted remarkably with the onset of the spinal N14 and with the scalp FF2 or P14 (Fig. 5F—H). Notice that these N14 and P14 are homologous with the N11 and P11 observed for median nerve stimulation, the difference corresponding to the 3 msec conduction time from fingers to wrist. These data left little doubt that the onset of the neck *negativity* and of the scalp FF2 indeed corresponded with the spinal entry time of the afferent volley.

The first far-field FF1 was generally much smaller for finger stimulation (Fig. 5G) than for mixed nerve stimulation (Fig. 4C) and spinal entry time was unambiguously estimated in lower neck SEPs recorded with an earlobe reference (Desmedt and Cheron 1980b). The FF1 presented the same polarity and onset latency all along the neck and scalp when using a non-cephalic reference. The FF1 onset was about 7 msec for median nerve stimula-

tion at the wrist (Fig. 1), but about 10 msec for finger stimulation (Fig. 5H). In all experiments the F11 onset preceded the arrival of the peripheral nerve volley at Erb's point (Fig. 5E), but it was later than the onset of the nerve potential at the axilla (Fig. 5D). These data confirm that the positive FF1 is a volume-conducted potential generated by peripheral nerve activity just proximal to the axilla (Cracco and Cracco 1976; Cracco 1980; Wiederholt 1980).

Anatomical correlates

The mean latency shift of 0.95 ± 0.15 (S.D.) msec for the spinal SEP negativity could be related to conduction along the dorsal column (DC) fibres. An apparent difficulty arose from the separation of about 100 mm between the 'C7' and 'C2' electrodes since this would imply the rather unlikely CV of 100 : 0.95 = 105 m/sec. The proximal fibres of spinal root ganglia that travel up the DC are supposed to conduct slower, not faster, than the distal part of the same cells in the peripheral nerve (cf., Loeb 1976). We studied in the Department of Anatomy 4 fixed adult human heads cut sagittally *in toto* and 3 dissections of the medulla and spinal cord with its roots (Fig. 6A). It is known that the sensory innervation of finger I depends on the sixth cervical root and that of fingers II and III on the seventh root (Foerster 1933, 1936; Keegan and Garrett 1948). Thus the afferent volley to stimulation of these 3 fingers or of the median nerve arrived at spinal cord segments C7-C6.

When leaving the vertebral canal, each spinal nerve and its dorsal root ganglion are located *above* the posterior arch of the corresponding vertebra (Fig. 6A). The C6-C7 rootlets presented downward oblique courses so that the distance from the middle of the seventh vertebra was about 15 mm below the C7 spinal cord segment (at root entry) and about 20 mm below the level between the C6 and C7 spinal cord segments. Therefore the early spinal negativity picked up over the C7 spinous process was in fact generated more cephalad, in the C6-C7 spinal segments (Fig. 6B).

The site of the upper neck electrode over the nuchal ligament below the inion was also rather remote from the spinal cord. Considering the geometry of the region and the orientation of the base of the skull we think that this electrode was unlikely to pick up spinal activities above the second spinal segment identified by the entry of the second spinal root. The mean distance from spinal segments C6-C7 to C2 was 55 mm in 7 specimens and this was considered a rough approximation of the actual conduction distance of the negative spinal component seen at the lower and upper neck electrodes. Thus the CV in the dorsal column was taken as 55 : 0.95 = 58 m/sec. Since the mean distance measured from spinal segments C6-C7 to the middle of the cuneate DC nucleus was 70 mm, the mean conduction time from spinal entry to this nucleus was 70 : 58 = 1.2 msec.

Far fields at the right or left earlobes

Comparison of early SEP components at either earlobe with non-cephalic references had not hitherto been studied. They disclosed differential effects that appeared to be related to anatomical features of the pathway. Thus FF1 and FF2 were somewhat larger ipsilaterally in Fig. 7B–C, while FF4 was obviously larger contralaterally. The FF4 was still larger in the scalp records (at half amplification in Fig. 7D–E). Virtual far-field cancellation in the parietal to front montage clearly disclosed the onset of the contralateral cortical N23 (Fig. 7F).

Fig. 8 compares peak-to-peak amplitudes of FF1, FF2 and combined FF3-FF4 at the two earlobes. The calculated regression lines have high correlation coefficients. The ipsilateral-contralateral difference barely reached significance for FF1. FF2 was significantly larger ipsilaterally while FF3-FF4 were significantly larger contralaterally (Table I).

Scalp topography of SEP far fields

Scalp far fields are generally said to be widespread over the scalp, and usually some-

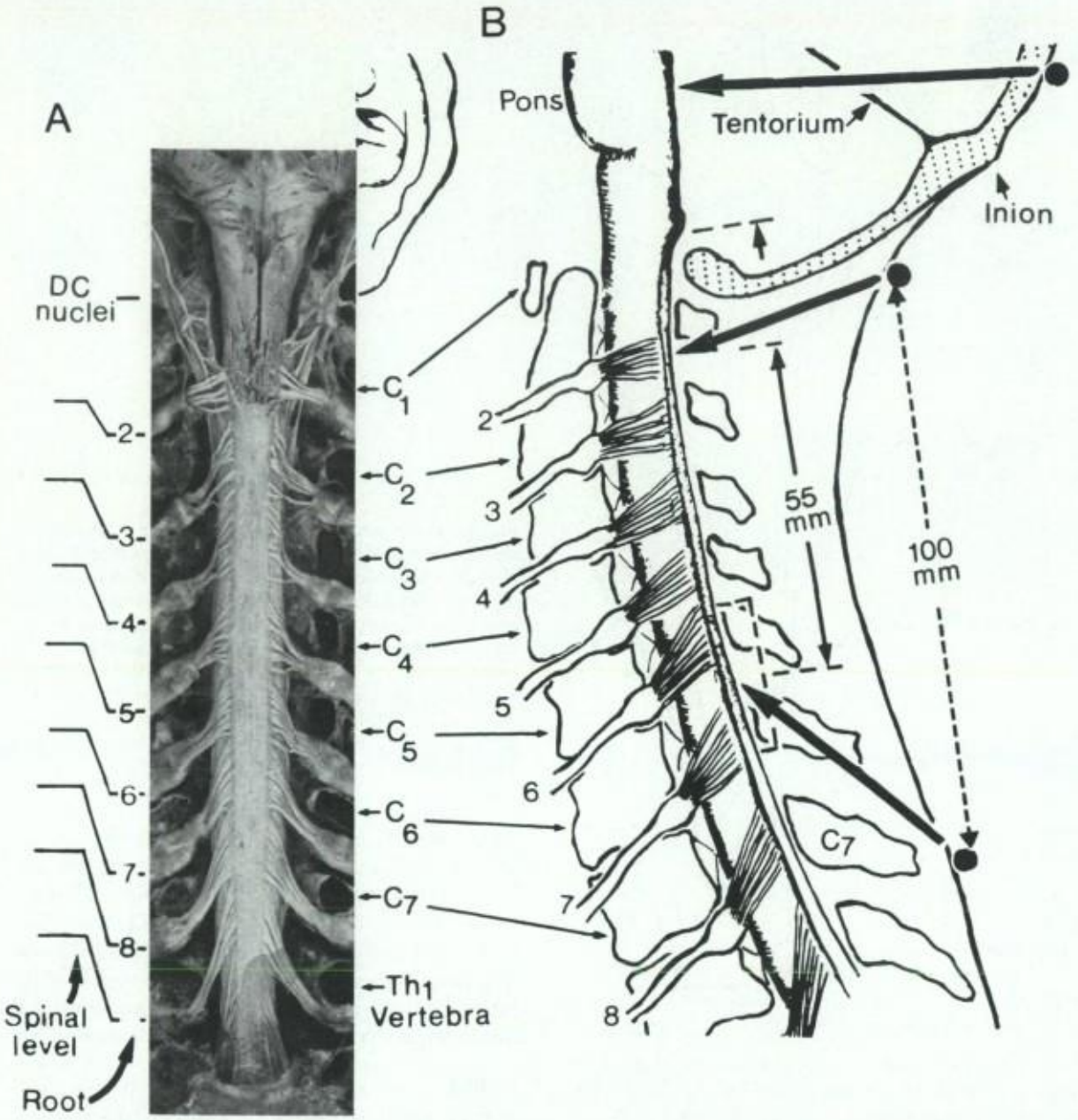

Fig. 6. A: photograph of a dissection of the posterior aspect of the medulla and cervical spinal cord with dorsal roots and spinal ganglia in a formalin-fixed adult man. Roots are labelled on the left side. Their levels at exit from the vertebral canal (above the corresponding vertebra) are clearly lower (more caudal) than the levels of the spinal segments where rootlets enter the cord. The posterior arch of each vertebra is labelled on the right side. B: sketch of a lateral view of the same region with the vertebrae and their spinous process, the skull base cut sagittally at the midline, and the neck skin contour. The positions of the (active) recording electrodes over the C7 spinous process, 2 cm below, and 2 cm above the inion are indicated. The large arrows point to the presumed zones of actual recording (see text).

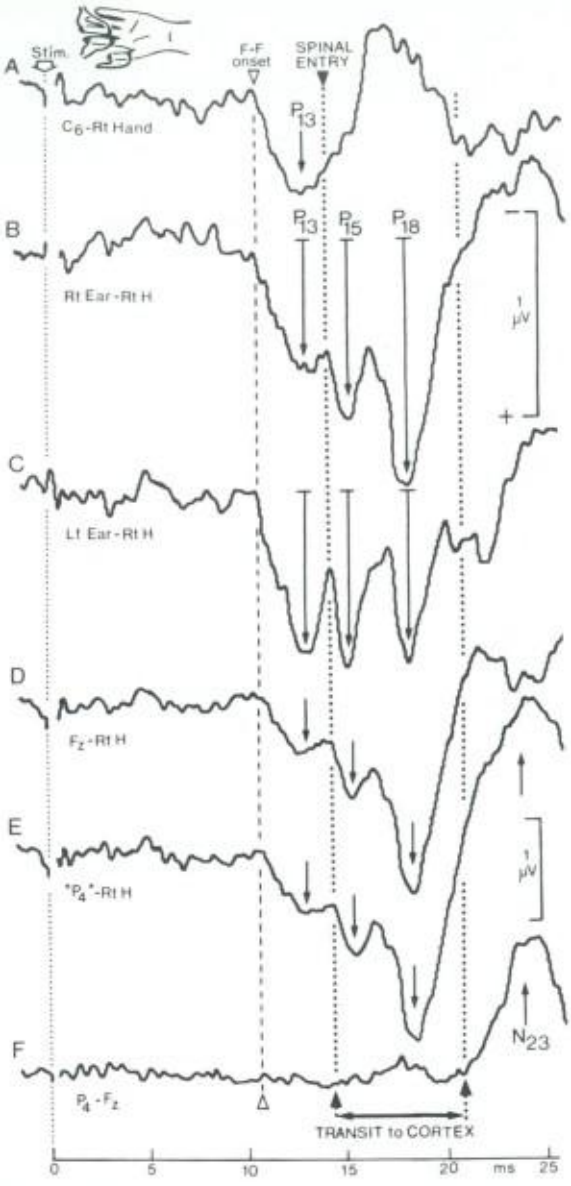

Fig. 7. Far-field SEP components to stimulation of fingers I—II—III in a male subject of 24 years. The reference is non-cephalic on the right hand in A—E. In F the contralateral parietal electrode is referred to the front whereby the far fields are nearly cancelled out and the onset of the cortical N23 is more clearly seen. The amplification is reduced to half in D—F since the far fields are larger on the scalp than at the earlobes. The transit from spinal entry to cortex is 6.4 msec. The peak amplitudes from baseline are indicated for P13 (FF1), P15 (FF2) and P18 (FF4) in the records from contralateral (B) and ipsilateral (C) earlobes.

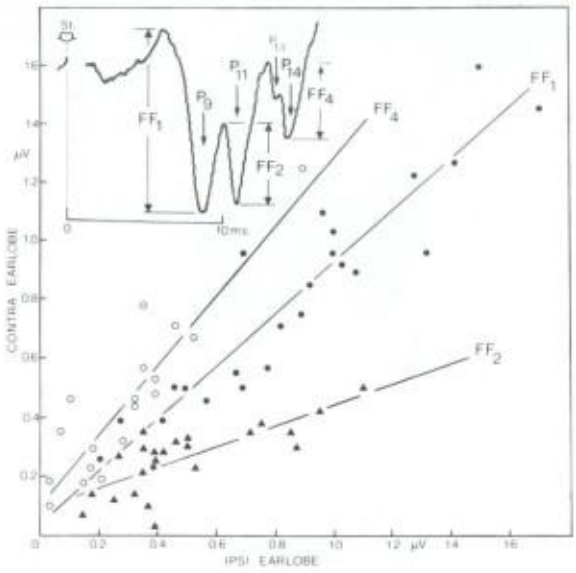

Fig. 8. Peak amplitudes of early SEP components simultaneously recorded from the ipsilateral (μV, abscissa) and contralateral (μV, ordinate) earlobes with non-cephalic reference on the right hand. Stimulation of fingers I—II—III or of median nerve at the wrist on the left side. Amplitudes were measured as depicted on the trace shown (insert). The calculated linear regressions were for FF1 (dots): $y = 0.89\ x + 0.04$ ($r_{xy} = 0.94$); for FF2 (triangles): $y = 0.35\ x + 0.09$ ($r_{xy} = 0.74$); and for the combined FF3 and FF4 (circles): $y = 1.17\ x + 0.10$ ($r_{xy} = 0.88$).

what larger contralaterally (Anziska et al. 1978). Our findings of asymmetries at the earlobes prompted a detailed mapping of scalp far fields in 3 subjects. The FF1 and FF4 were well delineated in Fig. 9, while FF2 was not consistent. FF1 presented a roughly similar amplitude at all sites and its variations did not appear to fit any consistent pattern. By

TABLE I

SEP far-field amplitudes at either earlobe recorded with non-cephalic reference (μV). Mean ± S.D. for 23 estimates in 5 subjects.

	Ipsilateral	Contra-lateral	*t* test by pairs
FF1	0.85 ± 0.40	0.79 ± 0.39	$P > 0.05$
FF2	0.49 ± 0.25	0.23 ± 0.13	$P < 0.001$
FF3-FF4	0.30 ± 0.21	0.45 ± 0.27	$P < 0.001$

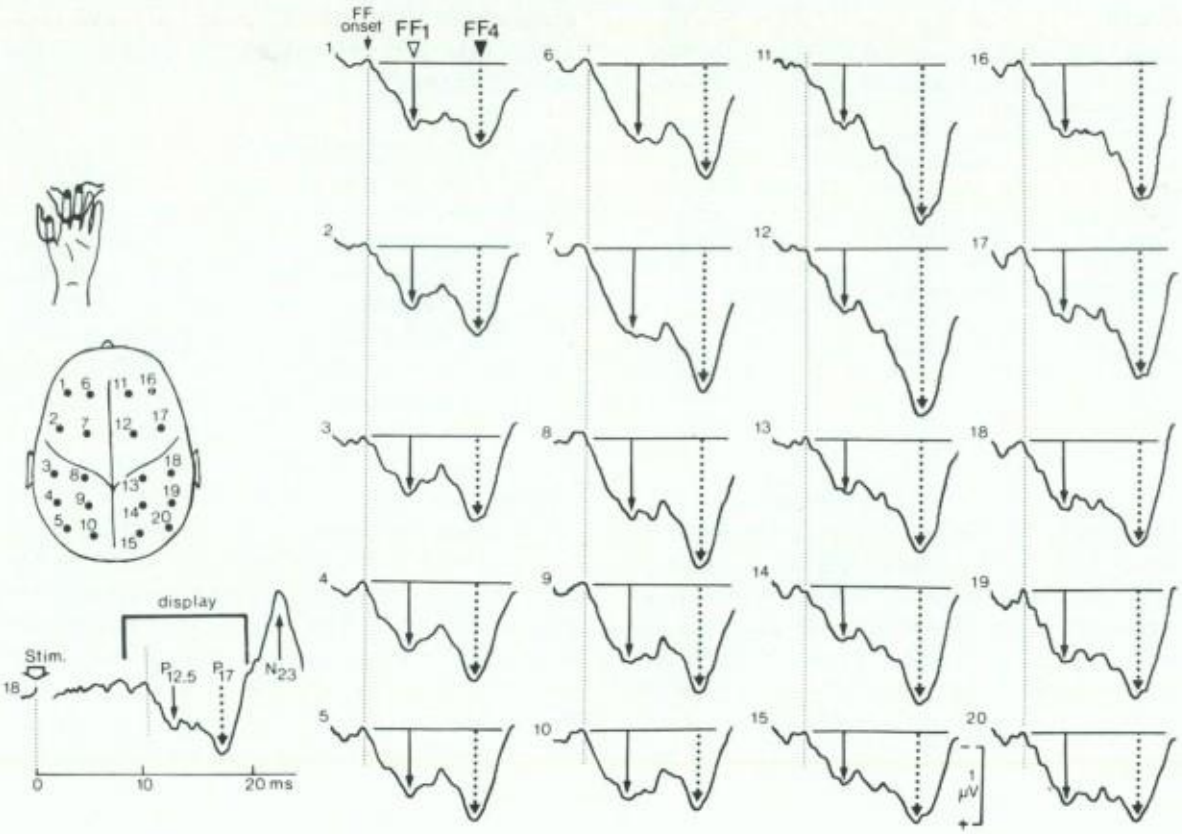

Fig. 9. Scalp topography of early SEP components in a male subject of 22 years. Stimulation of fingers I—II—III of the left hand. Non-cephalic reference on the right hand. The 20 electrode sites are indicated in the figurine. A complete trace recorded from electrode 18 is shown on the left to indicate the portion that is enlarged and displayed (from about 8 to 19 msec after stimulation) in the other traces. The peak amplitudes of FF1 (line) and FF4 (dots) are indicated.

contrast FF4 or P17 was larger contralaterally, and more so at 35 mm from the midline (traces 11—12) than at 70 mm from the midline (traces 16—17). The side differences were most obvious in the frontal region where the largest FF4 were recorded (Table II). Over the parieto-occipital scalp the side differences reached significance for the sites at 70 mm from the midline, but not for those at 35 mm from the midline.

TABLE II

Mean amplitude of FF4 (μV) at symmetrical electrode sites on the scalp in 3 subjects (mean ± S.D.).

	Ipsilateral	Contralateral	t test by pairs
Frontal 70 mm from midline	0.63 ± 0.06	0.90 ± 0.09	$P < 0.001$
Frontal 35 mm from midline	0.90 ± 0.08	1.06 ± 0.05	$P < 0.02$
Parietal 70 mm from midline	0.59 ± 0.07	0.66 ± 0.11	$P < 0.02$
Parietal 35 mm from midline	0.67 ± 0.08	0.83 ± 0.17	$P > 0.2$

TABLE III

Interpeak and onset-to-peak delays (msec) for SEP far fields and cortical N20 (mean ± S.D. for 11 estimates on 9 subjects).

	From onset FF2 (spinal entry)	From peak FF2
To onset FF3	2.26 ± 0.27	1.26 ± 0.20
To peak FF3	2.99 ± 0.33	1.86 ± 0.26
To onset FF4	3.20 ± 0.30	2.20 ± 0.30
To peak FF4	4.12 ± 0.36	2.97 ± 0.59
To onset N20	6.22 ± 0.31	5.22 ± 0.30
To peak N20	8.66 ± 0.68	7.73 ± 0.83

Interpeak latencies of SEP far fields

The onsets and peaks of far fields seemed better defined than usual in the present study and satisfactory measures were obtained in 4 experiments with finger stimulation and 7 experiments with median nerve stimulation (Table III). Both interpeak delays and transit times from the onset or peak of FF2 to subsequent peaks were estimated. Contrary to the absolute latency values, these interpeak measures were not related to the length of the subject's arm.

Onset of the cortical response

The first cortical component to the electrical stimulation of fingers or median nerve is commonly assumed to be the negative N20 which is largest over the contralateral parietal scalp (cf., Hirsch et al. 1961; Desmedt and Debecker 1964; Giblin 1964; Desmedt 1971; Matthews et al. 1974; Halliday 1978) and is consistently recorded with a mean peak voltage of about 6 μV directly from the exposed postcentral cortex (Domino et al. 1965; Papakostopoulos and Crow 1980). However, there is considerable disagreement about the exact time of arrival of the afferent volley in the cortex and, for example, the view has been entertained that N20 might originate in part in the thalamo-cortical radiation (Kritchevsky and Wiederholt 1978; Chiappa et al. 1980). Precise estimation of the onset time of the cortical SEP is essential both for clinical uses

TABLE IV

Evaluation of conduction times and velocities in the somatosensory pathway of normal adult man (afferent volley elicited by finger stimulation).

	Maximal diameter of axons (μm)	Conduction distance (mm)	Maximal CV (m/sec)	Time (msec)	Cumulated time (msec)
Finger-to-wrist	14 [a]		63.2 ± 3.2 [b]		
Wrist-to-cord	(16)		71.1 ± 4.0 [b]		
Cord entry-to-cuneate	—	70	58	1.2	1.2
Synaptic delay in cuneate	—	—	—	0.3	1.5
Medial lemniscus	9 [c]	70	40.5	1.7	3.2
Synaptic delay in thalamus	—	—	—	0.3	3.5
Synaptic delays in cortex (intracortical time)	—	—	—	0.6	4.1
Spinal cord-to-cortex transit	—	—	—	6.22 ± 0.31	
Conduction in thalamo-cortical axons	?	70	33	6.22 − 4.1 = 2.1	
Mean CV from cord entry to cortex	—	210	42	6.22 − 1.2 = 5.0	

[a] Desmedt (unpublished).
[b] Desmedt and Cheron (1980b).
[c] Verhaart and Schoen (unpublished).

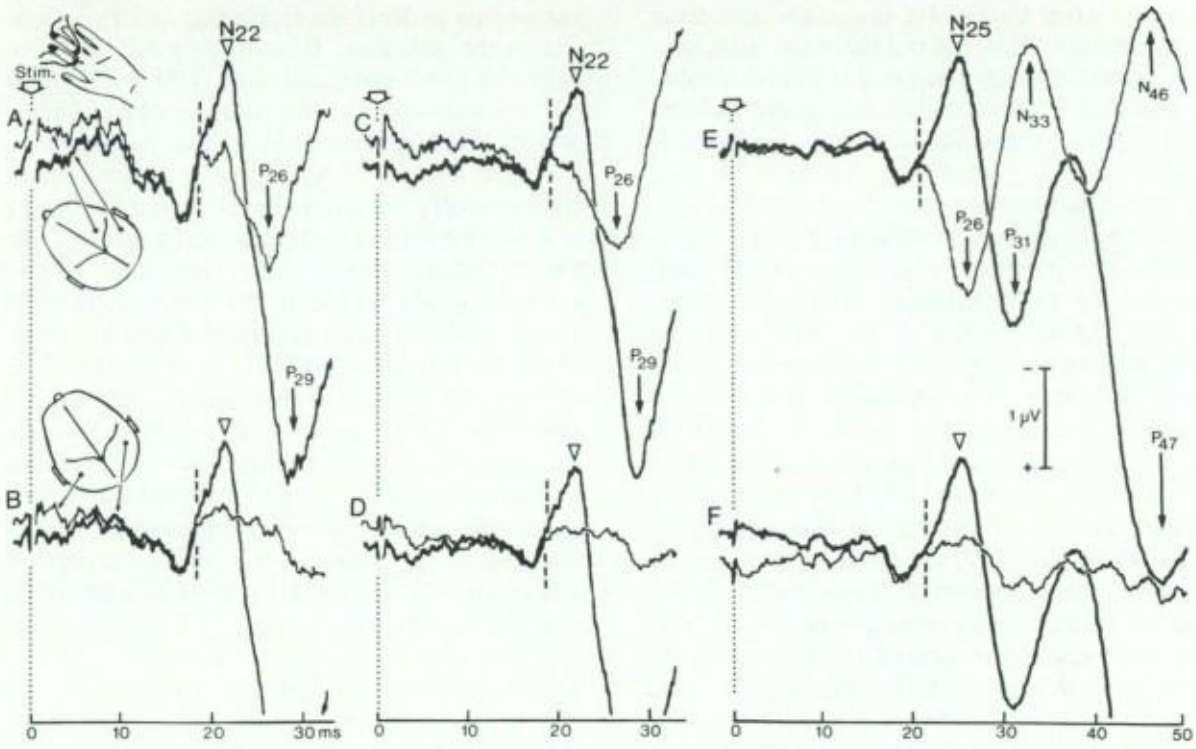

Fig. 10. Identification of the onset latency of the cortical SEP in a male subject of 22 years (A—D) and in a normal male subject of 87 years (E, F; same subject as illustrated in Desmedt and Cheron 1980b, Fig. 2). The thicker trace in each of the 6 superimpositions is the averaged SEP recorded from the postcentral scalp on the side opposite to the fingers stimulated. In A and B, a non-cephalic reference on the right hand is used. In C—F, the reference is the earlobe on the same side. The thinner trace is recorded from the precentral scalp on the side contralateral to the finger stimulated in A, C and E (see the figurines), and the postcentral scalp on the side ipsilateral to the finger stimulated in B, D and F. The parietal N22 peak is indicated by a hollow triangle. The time of divergence of the traces is indicated by a vertical interrupted line and this corresponds to the onset of the cortical negative response in the projection areas of the contralateral parietal cortex. The peaks of the precentral P26 and of the postcentral P29 or P31 and P47 are indicated.

(cf., Hume and Cant 1978; Desmedt 1980) and for discussing subcortical conduction times (see Discussion).

Our rationale in attempting to resolve the problem has been to superimpose for detailed comparison of wave forms the averaged SEP traces recorded from the contralateral parietal focus, with traces simultaneously recorded either from the symmetrical ipsilateral parietal scalp (Fig. 10B, D, F) or from the contralateral precentral scalp (Fig. 10A, C, E). The positive far fields were quite prominent in the SEPs recorded with non-cephalic hand reference (A, B), but reduced in size and of simpler profile in traces from the same active electrodes referred to earlobes (C—F) since the latter picked up early far fields to a significant extent (Figs. 3A and 8). The paired traces were superimposed as closely as possible for the P17 (FF4) components rather than for the pre-stimulus record.

In agreement with previous findings (Desmedt and Robertson 1977) the ipsilateral SEP had little, if any, significant negativity during the time of the contralateral N22, as well as little positivity during the contralateral P29 (Fig. 10B, D, F). This may be related to the lack of callosal connexions from primary

somatosensory cortex for the projection areas of the distal limb, as documented anatomically in the monkey (Jones and Powell 1969a; Pandya and Vignolo 1969; Karol and Pandya 1971). The divergence of traces corresponds roughly to the initial upgoing limb of N22 taking off at the time when the preceding P17 far field has reached baseline (Fig. 10).

Because thalamo-cortical radiation potentials are elicited from the contralateral thalamus (cf., Albe-Fessard et al. 1963) one can argue that the ipsilateral SEP provides no adequate reference for separating the possible subcortical contribution to N22. Therefore the superimposition of postcentral and precentral SEPs, both recorded contralaterally, appears to be very important. The positive far fields, including P17, are virtually identical at the two scalp locations (only about 5 cm apart) (cf., Fig. 9), as indeed expected for volume-conducted subcortical activities. However, the onset of N22 clearly diverges from the precentral trace (Fig. 10A, C, E) which presents little, if any, early negativity before the characteristic P26 component (cf., Desmedt and Cheron 1980b; Papakostopoulos and Crow 1980). In so far as the precentral and postcentral traces include similar subcortical volume-conducted potentials, one must conclude that the N22 is indeed the response of the cortical projection areas to the afferent volley. The upgoing limb of N22 rising from the baseline after the P17 far field can thus be taken as a titration point for estimating the cortical SEP latency.

Discussion

The increasing interest in early SEPs is no doubt sustained by their potentialities for improved non-invasive identification of focal brain stem lesions, much along the lines set forth by a number of recently disclosed diagnostic uses of brain stem auditory evoked potentials (BAEPs) (cf., Stockard and Sharbrough 1980). In this context it is necessary to understand better the far-field SEP components and the probable locations of their respective generators along the central somatosensory pathway. It is now agreed that the first far-field potential FF1 (P9 to median nerve stimulation at the wrist or P12 to finger stimulation) recorded as a positive wave of identical latency at all the neck or scalp electrodes (with non-cephalic reference) (Fig. 1) is a volume-conducted potential generated by the peripheral nerve volley proximal to the axilla (Fig. 5). However, the fairly consistent subsequent far fields recorded from the scalp (FF2 or P11, and FF3-FF4 or P14 to median nerve stimulation) raise many unresolved questions. One rationale of the present study has been to consider the conduction distances and anatomical features of each of the 3 neurones making up the central pathway in adult man, and to attempt to relate interpeak delays of SEP far fields to actual conduction times along these neurones.

Volume conduction of neural potentials

The sources of scalp-recorded far fields appear to be primarily related to the fairly synchronized volleys of action potentials travelling up in the corticipetal tracts (cf., Stockard and Sharbrough 1980) whereby an 'open-field' system (cf., Lorente de Nó 1947a, b) generates a coherent current field at a distance (Arezzo et al. 1979). The somatosensory relays, namely the cuneate nucleus of the dorsal column (DC) system and the ventrobasal thalamic nucleus, are unlikely generators for these brief SEP far fields (Table III) because of their 'closed-field' anatomical structure (Lorente de Nó 1947a, b; Klee and Rall 1977); electrical activities inside such structures with no consistent geometrical stereotypy generate little, if any, current flow outside their anatomical boundaries. In this respect it should be pointed out that the cortically generated potentials (N20, P30, . . .) that follow the early far fields are currently related, not to axonal volleys, but to postsynaptic potentials generated in the geometrically coherent dendrites of cortical pyramids ('open-field' system) (Eccles 1951; Towe 1966; Humphrey 1968; Klee and Rall 1977).

The sources of the spinal potentials recorded by electrodes placed on the skin of the neck appear somewhat more complex than those of the scalp far fields, and the spinal potentials indeed present longer durations (several milliseconds) than the latter (1—2 msec) (Fig. 1). Direct recording from the spinal cord surface in cats and monkeys disclosed that impulses in low-threshold cutaneous afferent fibres elicit cord dorsum potentials which are related to synaptic excitation of dorsal horn interneurones (Gasser and Graham 1933; Bernhard 1953; Lindblom and Ottoson 1953; Beall et al. 1977). These cord potentials present a sharp early negative component followed by a sequence of smaller, more prolonged, waves. Similar negative responses have been recorded epidurally from the cervical spinal cord in man (Shimoji et al. 1972, 1978; Ertekin 1973, 1976). Measurements by Ertekin indicate that the early negative potential at the lower cervical cord has a mean peak latency of 8.3 msec for stimulation of the median nerve at the elbow (which would correspond to about 11 msec for stimulation at the wrist).

These cord potentials no doubt contribute to the SEP profile recorded from the neck (Figs. 1F, G and 2F). It is interesting that 5 patients with complete traumatic spinal cord section at C5 or C6 presented no scalp SEP response, but an N11 component in the neck SEP (El-Negamy and Sedgwick 1978). On the other hand, a patient with an infiltrating astrocytoma invading the dorsal medulla and the right dorsal column funiculus down to the C5 level disclosed a negative wave peaking at 14 msec (frontal reference), but no far fields at the scalp (Mauguière and Courjon 1981). This might suggest that the neck SEP generated in the C6-C7 spinal cord levels includes, not only the early N11 potential of sharp onset, but also subsequent components of the spinal cord dorsum potentials. It is indeed difficult to differentiate in the neck SEP profile the respective contributions of the interneurone potentials of the dorsal horns and of the volley ascending in the dorsal column. Both are no doubt to be taken into account. We think that the abrupt front of negativity recorded from the neck with non-cephalic reference (Fig. 1D—G) corresponds primarily to the dorsal column volley, while the fixed generators in the dorsal horns result in enlarging and prolonging the neck SEP profile which indeed lasts several milliseconds, more than a spike volley. We suggest, however, that only the ascending dorsal column volley generates, by volume conduction, the P11 far field which lasts only 1—2 msec at the scalp (Fig. 1A—C). This working hypothesis accounts for the loss of scalp far fields following P11, but persistence of longer-lasting potentials at the neck, in patients with spinal cord transection at C5.

Spinal potentials and afferent conduction in dorsal columns

The present data have clarified the differentiation between potentials generated in the cervical cord and potentials generated above the foramen magnum. In this respect it is important to identify the distortions introduced in the neck SEP records by the use of a mid-frontal (Fz) scalp reference. The current popularity of the Fz reference no doubt relates to the increase of signal size that is obtained by the algebraic addition of the negative spinal potentials and the positive scalp far fields which are prominent at Fz (cf., Fig. 9) and are injected into grid 2 of the amplifier in this montage (Fig. 3). There is no doubt that the Fz reference is indeed valuable for practical diagnostic recording in patients under the adverse interference conditions of a busy clinic or operating theatre (Symon et al. 1979). For such uses the upper neck electrode referred to Fz is a better choice than the lower neck electrode since the upper neck-Fz montage more efficiently cancels out the first far-field FF1, thereby allowing the onset of spinal activities to be more easily estimated (Figs. 1 and 3D—E).

However, this montage, no matter how useful clinically, can create unnecessary confusion when it is used in isolation for discus-

sions about SEP generators (see above). For example, it seems now clear that the Fz reference montage used by Matthews et al. (1974), Jones (1977) and Small et al. (1980) obscured the latency shift of the spinal negative wave from lower to upper neck, that is demonstrated by non-cephalic reference recording (Figs. 1, 3 and 4). Cracco (1973) had claimed such a latency shift on the basis of traces recorded with an earlobe reference (not optimal for this study; see Fig. 4), and it is surprising that when subsequently using a non-cephalic reference, Cracco and Cracco (1976) did not mention this issue although an actual latency shift can be seen in their Fig. 3. The use of an Fz reference is also responsible for adding a 'N14' wavelet which does not belong to the neck SEP but is added in grid 2 of the amplifier by the scalp far-field P14 (Fig. 3) (see above).

The problem of the spinal SEP is thus made clearer by the use of a non-cephalic reference. The negative N11 component recorded from the lower neck starts upon the arrival of the peripheral nerve volley at the spinal cord (Fig. 5) which leaves little doubt about its being generated in the spinal cord rather than in the nerve or above foramen magnum. The N11 corresponds to the early negative component recorded intrathecally by Shimoji et al. (1978) and Ertekin (1973, 1976). The onset latency of this negative component increases from lower to upper neck by a mean of 0.95 ± 0.15 (S.D.) msec which we interpret as a conduction time along the dorsal column. Anatomical data discussed above (Fig. 6) suggest that the corresponding conduction distance is in fact shorter than the interelectrode separation, and can be estimated roughly as 55 mm from the level of spinal cord segments C6-C7 (where the nerve volley enters the cord) to spinal segment C2. The corresponding mean CV is calculated as 55 : 0.95 = 58 m/sec. This is 19% slower than the mean maximum CV from wrist to spinal cord, 71.1 ± 4.0 m/sec (S.D.), measured from sensory nerve action potential records (Desmedt and Cheron 1980b). The slower conduction in dorsal column than in peripheral nerve is in line with animal data (Lloyd and McIntyre 1950; Brown 1968; Uddenberg 1968; Loeb 1976). The arrival of the afferent volley from the fingers to the cuneate dorsal column nucleus is then calculated, on the basis of a (measured) mean conduction distance of 70 mm from the C6-C7 spinal segments in fixed brain specimens, as 70 : 58 = 1.2 msec (Table IV).

Conduction in medial lemniscus

Morphometric data are scarce or absent for central axons of known destination in man. Extrapolation from animal data is unwarranted in view of the known differences between man, primates and other mammals; for example, in the absolute values of peripheral nerve fibre CVs, which imply differences in fibre diameter or other properties (cf., Paintal 1973; Waxman 1980). Another point is that precise estimations must take into account the specialization of fibre groups in medial lemniscus in relation to the human hand capabilities and to the sensory submodalities involved by the stimulation.

For example, appropriate correlations for SEPs elicited by stimulation of the thicker nerve fibres of digital nerves should be made with the second-order axons that actually receive and relay the input from the distal hand. These correspond to the subgroup ml_3 of external arcuate fibres which has been identified anatomically in the human medial lemniscus (personal communication from Prof. W. Verhaart and Dr. J. Schoen of Leiden to J.E.D. in 1968). The largest ml_3 axons have a diameter of 9 μm, and are thus definitely smaller than the first-order axons.

The next problem is to decide on a conversion factor for roughly calculating the CV in these axons. It is indeed risky to apply any standard factor, especially for central axons that may present peculiarities of functional architecture. Since the study of Hursh (1939) on peripheral nerves, a range of factors from about 6 to 3 m/sec/μm has been proposed for different nerves in different animal species

(cf., Boyd 1964; McLeod and Wray 1967; Paintal 1973). One point is that such factors differ significantly with species so that only data from human nerves should be considered. The maximal diameter of sensory fibres in the digital nerves of the second and third fingers of normal adult man (autopsy material excluding subjects with neurological disease) was estimated as 14 μm in a large number of specimens (Desmedt, unpublished). The maximum sensory CV at 36—37°C between finger and wrist was measured as 63.2 ± 3.2 (S.D.) m/sec (Desmedt and Cheron 1978, 1980b) and these figures agree with those of Mayer (1963) and Nielsen (1973). On this basis a conversion factor was calculated as: 63.2 : 14 = 4.5 m/sec/μm. It is known that long axons such as those innervating fingers present some distal tapering and branching: it is thus not surprising that the maximal CV of the afferent volley elicited by finger stimulation was about 8 m/sec faster (71.1 m/sec) in the median nerve from wrist to spinal cord, and this would correspond to a maximal diameter about 2 μm larger for the proximal parts of these first-order axons (Table IV).

If the conversion factor of 4.5 is applied to the largest lemniscal axons of ml_3, a maximal CV of $9 \times 4.4 = 40.5$ m/sec is obtained. The distance from the cuneate nucleus to the ventro-basal nucleus in thalamus was measured with callipers at a mean of 70 mm in the fixed adult specimens. This includes allowance for the decussation detour under the obex which was defined from histological sections stained for fibres. The minimal conduction time in the faster lemniscal axons of ml_3 can thus be taken as 70 : 40.5 = 1.73 msec (Table IV). These are of course rough approximations but they provide a useful clue. Suppose the conduction distance was 65 mm instead of 70 mm, this would make the time 65 : 40.5 = 1.60 msec. Suppose the estimation of lemniscal maximal CV was 10% higher at 44 m/sec, this would make the conduction time as 70 : 44 = 1.59 msec. We feel the error on the lemniscal time is unlikely to exceed 0.2 msec.

Synaptic delays in cuneate and thalamic relays

Plausible figures for synaptic delays are now required to calculate the probable time at which thalamic ventro-basal neurones are activated by the somatosensory volley. A fundamental anatomical feature of the dorsal column-lemniscal system is the great density of axonal terminations and the large size of synaptic boutons around clusters of neurones and their dendrites (Cajal 1909; Kuypers and Tuerk 1964; Scheibel and Scheibel 1966; Valverde 1966; Walberg 1966; Keller and Hand 1970; Hand and VanWinkle 1976). The synaptic knobs on the cuneate neurones were described as among the largest in the nervous system (Roszos 1958). The lemniscal axon terminals are organized as synaptic glomeruli in conjunction with dendritic excrescences of the ventro-basal thalamic neurones (Jones and Powell 1969b; Ralston 1969; Spacek and Lieberman 1974). These features combine to produce fine grain somatotopy and powerful excitatory effects that are physiologically expressed in the short synaptic latency and high safety factor for transmission (Poggio and Mountcastle 1963; Andersen et al. 1964; Eccles 1966; Welker 1973; Krnjević and Morris 1976). Harwood and Cress (1954) noticed that the synaptic delay in cuneate in cat is less than 0.5 msec. In the absence of actual precise measures, we propose the figure of 0.3 msec for the synaptic delays in both cuneate and ventro-basal thalamus.

On this basis, the cumulative delays from spinal entry at C6-C7 would be for the fastest axons: 1.2 (dorsal column) + 1.7 (medial lemniscus) + 0.6 (synapses) = 3.5 msec (Table IV) at which time the ventro-basal neurones would first be activated. If one took 0.4 msec for the synaptic delays, this figure would be increased from 3.5 to 3.7 msec. Conversely if the synaptic delays could be as short as 0.2 msec in these high safety factor synapses, the cumulative delay would only be 3.3 msec. We admit that there is no compelling reason to choose between these possibilities since there

are no direct measures of the synaptic delays in man and we can only presume that they must be quite brief.

Conduction in thalamo-cortical axons

There are no measures available for the diameter of thalamo-cortical axons in man. A rough estimation of CV can be attempted by considering the probable transit time from ventro-basal nucleus to postcentral cortex. The onset of the cortical response can be estimated from the latency of the upgoing limb of the N22 SEP component as discussed above (Fig. 10). This response must be related to postsynaptic potentials elicited in apical dendrites of pyramidal neurones in areas 3-1-2 (cf., Eccles 1951; Purpura et al. 1964; Towe 1966; Klee and Rall 1977; Desmedt 1980). These potentials appear to be elicited after an intracortical relay because the ventro-basal axons terminate in discrete column-like clusters mainly on the spiny stellate cells or type 7 neurones of Jones (1975) whose axons will in turn act synaptically on the pyramidal neurone dendrites. As a rough guess we propose a time of 0.6 msec for these two synaptic delays, including the intracortical conduction from spiny stellate neurone to pyramidal cell (Table IV). The mean transit time from spinal entry at C6-C7 to onset of the parietal N22 SEP component was 6.22 ± 0.31 (S.D.) msec. If the ventro-basal neurones in thalamus initiate their action potential after a mean minimum delay of 3.5 msec (preceding section), the transit time in the thalamo-cortical pathway is: 6.22 − 3.5 − 0.6 = 2.12 msec. Since the mean distance from ventro-basal thalamus to postcentral cortex (following the course of radiation fibres) was measured as roughly 70 mm on fixed specimens, we take the maximum CV as: 70 : 2.12 = 33 m/sec (Table IV), thus somewhat slower than in the dorsal columns or medial lemniscus. The thalamo-cortical CV would turn out to be a few m/sec faster or slower if one preferred to consider slightly different figures for intracortical synaptic delays (say, with 0.8 instead of 0.6 msec, the CV would be 70 : 1.92 = 36.4 m/sec). We believe the data of Table IV should be valid to ±10 or 20%.

Neural generators of SEP far fields

The brief positive far fields occurred with identical latency at all scalp electrodes (non-cephalic reference) (Figs. 1, 2 and 9). Their brief duration of 1—2 msec appears remarkable since the corticipetal axons must conduct at a range of velocities and since bursts of action potentials can be elicited in the second- or third-order neurones by even a single peripheral nerve volley. The FF2 (or P11 to median nerve stimulation) has an onset latency identical with that of the negative component recorded from the lower neck and is interpreted as volume-conducted action potentials ascending the dorsal column. The scalp P11 is probably not influenced by the fixed generators of dorsal horn interneurones, which presumably contribute to the neck SEP (see above). The FF3 and FF4 (or P13 and P14 to median nerve stimulation) are generated above foramen magnum and do not occur at the neck when using a non-cephalic reference. We interpret them as volume-conducted action potentials in the medial lemniscus. All scalp electrodes 'see' the positivity related to this advancing front of depolarization. However, P13-P14 are smaller just above the inion, which is at right angles to the oblique caudo-rostral axis of the brain stem (Figs. 1, 2 and 6), while they are largest at the front which is just ahead in the axis of the propagated volley (Fig. 9).

While the input volley only begins to arrive at the thalamus 3.2 msec after spinal entry (Table IV), the mean onset of FF3 (or P13) occurs at 2.26 msec and its peak at 2.99 msec (Table III). Therefore the FF3 generator must indeed be located below the thalamus, but above foramen magnum. At the time of FF4 (or P14) onset, 3.2 msec, the lemniscal volley barely begins to activate the ventro-basal thalamic neurones (Table III). Clinical evidence showing that P14 can be recorded in patients with a thalamic lesion (Mauguière and Courjon 1980) is in line with the P14 generator being

located below the thalamus. Diencephalic lesions that abolished P14 were in fact quite diffuse and extended backwards into the brain stem (Noël and Desmedt 1980).

An interesting and hitherto unreported result is that FF2 (or P11) was significantly larger at the *ipsilateral* earlobe (Fig. 8, Table I) in agreement with the proposed relationship to the ascending volley in the dorsal column on the side stimulated, whereas FF3-FF4 (or P13—P14) were significantly larger at the *contralateral* earlobe (also with non-cephalic reference) as they indeed should be if they are related to a lemniscal generator located above the decussation of the somatosensory pathway. It is remarkable that the different far fields should differ in amplitude, but not in wave form or latency, at the two earlobes since the corresponding generators in the first- and second-order neurones are only a very few millimetres on either side of the midline. They are in fact closer to the midline than the auditory pathway studied with BAEP.

The detailed topography of scalp far fields also emphasized consistent patterns for FF3-FF4 (Fig. 9, Table II) and these can be related to geometrical relationships with respect to the oblique caudo-rostral axis of the brain stem generators and their location on one side of the midline. These findings suggest that electrode positions and montages used in SEP studies should receive somewhat more sophisticated attention than is currently the case.

Summary

Early somatosensory evoked potential (SEP) components to median nerve or finger stimulation were recorded with non-cephalic references in normal young adults. Detailed topographic data over scalp and neck were related to anatomical observations on the actual conduction distances in dorsal column, medial lemniscus and thalamo-cortical parts of the somatosensory pathway. The extrapolation of afferent conduction velocity (CV) measured from sensory nerve potentials along the peripheral nerve to the C6-C7 spinal segments identified the spinal entry time with the onset of the neck N11 or scalp P11 (far field 2 or FF2). The first far field (FF1) is generated in the nerve proximal to axilla. The definite latency shift of the spinal negativity along the neck indicates a CV of 58 m/sec. Data about the maximal diameter of lemniscal axons in man were used to calculate a CV of 40.5 m/sec. Consideration of transit times from spinal entry to cortex and of synaptic delays clarified the arrival times of the afferent volley at various relay nuclei, and also suggested a thalamo-cortical CV of about 33 m/sec. Interpeak and onset-to-peak measures on scalp far fields suggest that FF3-FF4 are generated in medial lemniscus rather than above the thalamus. Consistent differences in amplitude, but not in wave form, were recorded at right and left earlobes for FF2 (larger ipsilaterally) and FF3-FF4 (larger contralaterally). The scalp topography of far fields was analysed in detail.

Résumé

La conduction somesthésique centrale chez l'homme: générateurs nerveux et délais interpics des composantes 'far field' dérivées de la nuque, du cuir chevelu, et des oreilles droite et gauche

Les composantes précoces du potentiel évoqué somesthésique (PES) à la stimulation du nerf médian ou des doigts ont été dérivées avec référence non-céphalique chez des jeunes adultes normaux. Les données topographiques détaillées sur le cuir chevelu et la nuque ont été confrontées à des observations anatomiques sur les distances de conduction dans le cordon postérieur, le lemnisque médian et la partie thalamo-corticale de la voie somesthésique. L'extrapolation de la vitesse de conduction (VC) afférente mesurée par la dérivation des potentiels de nerf périphérique au niveau spinal C6-C7 a permis d'identifier le temps

d'entrée dans la moelle au début du N11 à la nuque ou P11 au cuir chevelu (far field 2 ou FF2). Le premier far-field FF1 est produit par le nerf périphérique proximalement à l'aisselle. La mise en évidence d'une augmentation régulière de latence de la négativité spinale le long de la nuque indique une VC de 58 m/sec. Des données sur le diamètre maximum des axones lemnisquaux ont permis de calculer une VC de 40,5 m/sec à ce niveau. La prise en considération des temps de transit depuis l'entrée dans la moelle jusqu'à l'arrivée au cortex ainsi que des délais synaptiques a permis d'evaluer les temps d'arrivée de la volée afférente aux divers relais nucléaires de la voie somesthésique; une VC de 33 m/sec environ a été trouvée pour les fibres thalamocorticales. Les mesures interpics et début au pic des far fields suggèrent que FF3-FF4 sont produits dans le lemnisque médian plutôt qu'au dessus du thalamus. Des différences systématiques d'amplitude, mais non de configuration, ont été enregistrées au niveau des oreilles gauche et droite pour FF2 (plus grand ipsilatéralement) et pour FF3-FF4 (plus grands contralatéralement). La topographie des far fields sur le cuir chevelu a été analysée en détail.

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