

Subject Review:**The Somatosensory Evoked Potential**

ANDREW EISEN

Three decades have elapsed since Dawson (1947) recorded the first somatosensory evoked potential (SEP). Simple superimposition of individual responses was possible because the patient had progressive myoclonic epilepsy. In this disease the SEP amplitude is much enhanced (Shibasaki et al, 1978; Kelly et al, 1981). Subsequently Dawson (1951, 1954) presented his averager to the Physiological Society, thereby initiating the present-day explosive growth of evoked potentials.

SEPs are made up of components with varying latencies. The components are best identified by latency and polarity as recorded at the scalp (P = positive and N = negative). Nevertheless, the nomenclature of somatosensory evoked potentials can be extremely confusing, mainly because the same component can have a different polarity depending on the electrode montage used. Generally speaking (but this is not a firm rule), far-field (subcortical) potentials are positive in polarity when a non-cephalic reference is used, whereas these same components have a negative polarity when the reference is on the scalp. It is therefore useful to always indicate the recording montage being employed. In addition, use of absolute latencies in the terminology can cause confusion because they are dependent upon length and body height. For example, the brachial plexus component usually occurs at about 9 msec, but may extend to as long as 11 or more msec in a very tall individual. Subsequent components then become difficult to identify in relation to normal means. In this regard, Donchin et al (1977) suggested use of an overlined latency terminology (\overline{P}_5), indicating that although in this individual the latency of the brachial plexus response was in fact 11 msec, it

corresponded to the normal mean usually at 9 msec.

Short latency components are considered as those with latencies of under 25 msec when stimulating an arm, and less than 45 msec when stimulating a leg. Such components are remarkably stable within the normal milieu of the recording laboratory, being unaffected by drowsiness, sleep, or light anaesthesia (Abrahamian et al, 1963; Goff et al, 1966). They are, however, affected by deeper, especially barbiturate, anaesthesia (Angel, 1977; Shaw and Cant, 1981). Medium, and particularly long latency components, are much less stable. They will not be considered further in this review.

The somatosensory evoked potential is largely mediated via large-diameter peripheral sensory fibres and the dorsal column-lemniscal systems centrally. Abnormal SEPs are particularly associated with position sense loss (Giblin, 1964; Halliday, 1967). Mixed nerve stimulation evokes a cerebral potential having a latency that is about 5 msec shorter than that evoked by cutaneous nerve stimulation using comparable stimulation sites (Eisen and Elleker, 1980; Burke et al, 1981), (see figure 1). The difference is probably due to activity of the faster group I muscle afferents compared to group II cutaneous afferents (Burke et al, 1981). This has relevance when trying to correlate clinical cutaneous sensory deficit with SEPs evoked by mixed nerve stimulation, since these reflect muscle as well as skin afferents (Desmedt and Brunko, 1980). Experimentally, excitation of delta (Alpsan, 1981) and C fibres (Simpson, 1981) also can evoke SEPs with proportionately longer latencies. It is likely that some components of the human SEP are also related to delta fibre activity (Yamada et al, 1978) and this could be used to

From the Division of Neurology, The University of British Columbia.

Reprint Requests to: Andrew Eisen, M.D. Division of Neurology, The University of British Columbia and Vancouver General Hospital, 855 West 12th Avenue, Vancouver, B.C., Canada V5Z 1M9.

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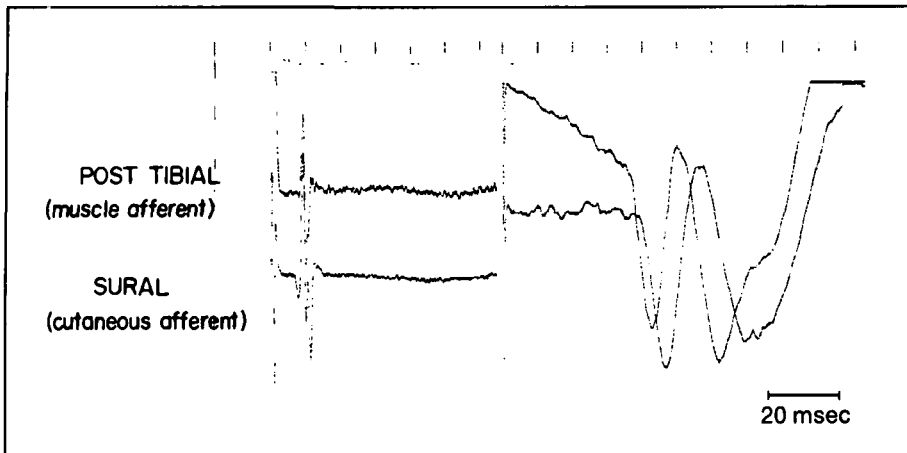


Figure 1 — Stimulation of the posterior tibial and sural nerves, which are equi-distant from the scalp, elicit SEPs of different latencies. P40 is about 5 msec shorter stimulating the posterior tibial as compared to the sural nerve. The latency of the mixed nerve and sensory nerve action potentials shown on the left are identical.

evaluate slower conducting sensory systems not amenable to routine methods.

That the SEP is mediated centrally via the dorsal columns is supported by the fact that it is markedly attenuated or abolished following experimental selective destruction of these tracts. On the other hand, the SEP remains relatively normal when the dorsal columns are isolated but kept intact (Cusick et al, 1979). Some SEP components may, however, reflect extralaminar activity, for they have been recorded in cats after dorsal column transection using stimuli sufficient to excite small as well as large diameter fibres (Martin et al, 1980). In man, tourniquet induced ischemia abolishes the short latency SEP components earlier than the later components, which may be recordable for long periods post-ischemically (Yamada et al, 1981). This dissociation between early and late latency components of the SEP has also been noted in various clinical conditions (Tsumoto et al, 1973; Yamada et al, 1978; Yamada et al, 1982).

Non-Cephalic (Far-Field) Versus Scalp Bipolar Recording

Accurate identification of the neural generators of the SEP components is paramount for optimal clinical use. Non-cephalic referential recording in

which the reference electrode is placed over the shoulder, arm, or hand contralateral to the side stimulated has identified four far-field potentials following median nerve stimulation at the wrist (Cracco and Cracco, 1976; Kritchinsky and Wiederholt, 1978; Anziska et al, 1978; Desmedt and Cheron, 1980(a)). They have been labelled P9, P11, P13, and P14 respectively. Their small amplitude, short latency, widespread distribution and positivity at the scalp are all consistent with them being far-field potentials. P9 reflects activity in the brachial plexus. P11 is a travelling wave within the dorsal columns (Desmedt and Cheron, 1980(a); 1981). The shortening of the P11 latency between dorsal and rostral cervical spinal cord was originally disputed (Matthews et al, 1974). This has now, however, been clearly documented by use of an esophageal recording electrode enabling accurate positioning at sequentially rostral cervical segments (Desmedt and Cheron, 1981). P13 and P14 have fixed generators and reflect activity in the cuneate nucleus and medial lemniscus respectively (Kritchinsky et al, 1978; Anziska et al, 1978; Desmedt and Cheron, 1980(a); 1981).

A large negative potential, N20, follows the small subcortical positivities (P9 through P14). This reflects arrival of impulses at the somatosensory cortex (Allison et al, 1980). It is, however, doubtful that N20 reflects activity of a

single cortical generator, since the thalamic somatosensory relay is distributed to several post-central parietal areas (areas 3b, 1 and 2). Little is known of the neural generators involved in the subsequent SEP peaks, P25, N35, and P40. The parietal "W" completed by these peaks is only recordable in about 50% of young adults but becomes very consistent in the elderly (Desmedt and Cheron, 1980(b)). These SEP components might represent sequential activation of cortical modules through cortico-cortical and/or thalamo-cortical connections (Desmedt and Cheron, 1980(b)).

SEPs can also be recorded from the pre-central region; some components (P22, N30) have onset and peak latencies that differ from those recorded post-centrally. The differences have been explained as resulting from a dipole in the depth of the central sulcus whose anterior half gives rise to P22 and whose posterior half gives rise to N20 (Broughton, 1969). Alternatively, the difference has been ascribed to a travelling wave across the central sulcus (Cracco, 1980). Recent evidence derived from direct human cortical recording (Papakostopoulos and Crow, 1980) contradicts both the deep dipole and travelling wave hypotheses and suggests that the frontal SEP components are related to distinct cortical generators (Desmedt and Cheron, 1980(b)).

The advantages bestowed by non-cephalic referential recording in accurate identification of subcortical generators must be weighed against the inherent technical difficulties involved. EMG and other interference necessitates the averaging of several thousand sweeps and all components may not be universally recordable in normal subjects. On the other hand, scalp bipolar recordings in which the "reference" is cephalic are technically much easier to achieve. Averaging of 256 or 512 sweeps is often sufficient. The cephalic reference may, however, produce a cancellation effect so that identification of all the subcortical components may not always be possible. Use of a bipolar montage of a frontal scalp electrode (Fpz; International 10-20 system) against an electrode over

the lower cervical spine has identified four negativities, N9, N11, N13, and N14 (Matthews et al, 1974; Jones, 1977; Abbruzzese et al, 1978(a); Sedgwick and Soar, 1980). There is good evidence that these mirror the positive potentials P9 through P14 obtained with non-cephalic recordings and that they share the same neural generators. The scalp SEP recorded using a bipolar electrode pair of Fpz and the hand area C3 or C4 (International 10-20 system) contralateral to the side of stimulation has the same "W" sequence N20, P25, N35, and P40, as described above.

It is interesting that although the thalamus (ventro-basal complex) and thalamo-cortical projections are intricate parts of the somatosensory pathway, little mention has been made regarding their expression in the scalp recorded SEP. It has been suggested that N20 reflects thalamic activity (Kritchevsky and Wiederholt, 1978; Chiappa et al, 1980). P15 (see figure 2) has alternatively been considered to be due to a thalamic generator (Allison et al, 1980). However, direct thalamic recordings (Pagni, 1967; Larsen and Sances, 1968; Goto et al, 1968; Fukushima et al, 1976; Celesia, 1979) and recent clinical studies correlating the SEP and CT scan (Mauguière and Caurjon, 1981) do not support either of these views. Following median or ulnar nerve stimulation a monophasic positive potential with peak latency at about 16 to 17 msec is recordable from the thalamus (Celesia, 1979). Furthermore, thalamic hemorrhage visualized through the CT scan is associated with an intact P15 peak, indicating its generator must be sub-thalamic (Mauguière and Caurjon, 1981).

Using bipolar scalp recordings, Abbruzzese and colleagues (1978(b)) drew attention to two additional negative waves, N16 and N17, occurring between P15 and N20. They suggested N16 and N17 are generated by independent dipoles, the thalamic relay, and thalamo-cortical radiation respectively. N14 and P15 components have a constant opposition in polarity irrespective of cranial electrode placement or combination. Therefore, N14 - P15 can be considered as a biphasic complex due to a single dipole gen-

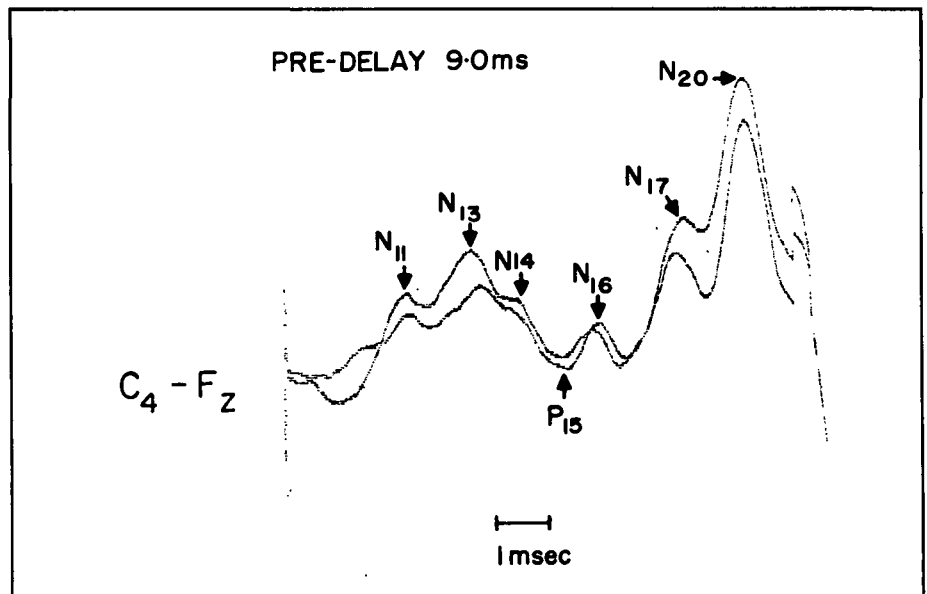


Figure 2 — This normal SEP was recorded using a total analysis time of 10 msec coupled to a 9 msec pre-delay. The new components N16 and 17, as well as earlier subcortical components N11, 13, and 14, are easily recognizable. The median nerve was stimulated at the wrist.

erator — the medial lemniscus (Abbruzzese et al, 1978(b)).

In this laboratory, using high gain amplification employing a special sensory amplifier with a band-pass of between 200 and 1000 Hz, we have recently been able to consistently and easily record N16 and N17, (figure 2, table 1). Use of a short analysis time such as 10 msec coupled to an appropriate pre-delay (figure 2), has facilitated recording of these new SEP components. In figure 3, it is shown how the use of various recording montages attenuates or enhances the various SEP components. The presumed neural generators of the different SEP components obtained by mixed median nerve stimulation using bipolar cephalic recording is shown in figure 4. The overall evoked potential now begins to approach the brainstem auditory evoked response with activation of successive brainstem and more

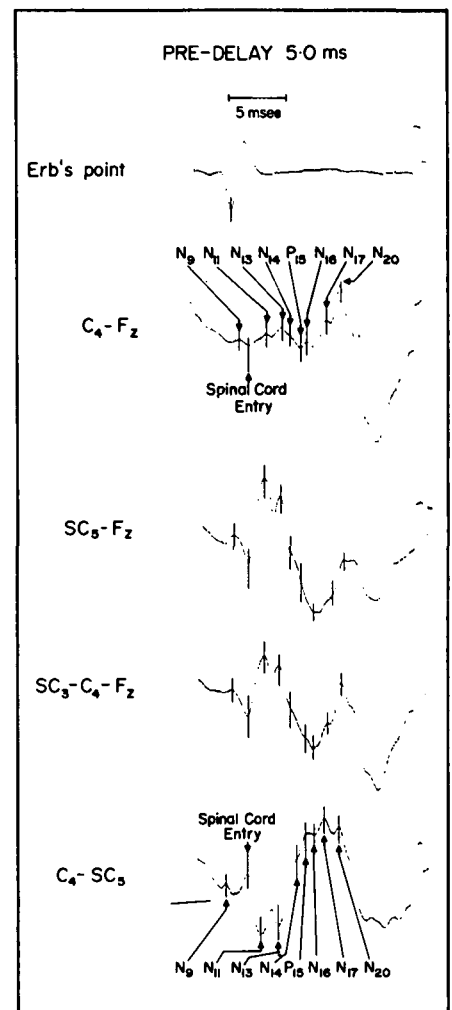


Figure 3 — The effects that various recording montages have upon the SEP components are shown. N16 and 17 are best seen when the recording is between C4 and Fz. Earlier subcortical potentials (N9 through N15) are best seen when the recording is between the cervical cord and Fz. SC5 is the 5th cervical vertebrae. In all the recordings the reference is cephalic.

TABLE 1
SEP COMPONENTS AND CENTRAL CONDUCTION TIMES ELICITED BY MEDIAN NERVE STIMULATION

Component	$\bar{x} \pm SD$ (msec)	Central conduction times (msec)
N11	11.6 ± 0.5	N11-N13 1.2 ± 0.3
N13	13.2 ± 0.6	N13-N14 1.1 ± 0.25
N14	14.2 ± 0.7	N14-N15 0.8 ± 0.2
P15	15.0 ± 0.6	P15-N16 0.8 ± 0.3
N16	16.1 ± 0.9	N16-N17 1.5 ± 0.3
N17	17.6 ± 0.8	N17-N20 1.3 ± 0.2
N20	19.2 ± 0.9	*N13-N20 5.4 ± 0.3

* See also Dorfman, 1977; Hume and Cant, 1978; Ganes, 1980; Eisen and Odusote, 1980; Desmedt and Cheron, 1980a).

rostral structures becoming delineated (Stockard et al, 1977). It will be important to verify the new SEP components N16 and N17 and their presumed neural generators by careful clinical correlation and CT correlation.

Lower Limb and Lumbar-Spinal SEPs

Cerebral potentials evoked through stimulation of the leg have the advan-

tage of monitoring conduction through the whole neuraxis. Their use is gaining popularity (Tsumoto et al, 1972; Dorfman, 1977; Kimura et al, 1978; Jones and Small, 1978; Eisen and Nudleman, 1979; Eisen and Odusote, 1980; Vas et al, 1981; Beric and Prevec, 1981). With tibial nerve stimulation at the ankle a prominent positive wave occurs at about 40 msec (P40). However, activity of the primary somatosensory cortex can be ascribed to an earlier, smaller amplitude, negative peak, N32. This is the equivalent of N20 evoked by median nerve stimulation (figure 4, table 2). It and earlier subcortically generated peaks (gracile nucleus amongst others) have been recently recorded using both non-cephalic references (Vas et al, 1981) and bipolar cephalic montages (Beric and Prevec, 1981; Leandri et al, 1981; Lueders et al, 1981). Figure 4 schematically shows the early latency SEP components and their presumed neural generators evoked by lower extremity stimulation.

A spinal SEP having maximum amplitude over the lower thoracic-upper lumbar spine can be recorded by use of either a common (iliac crest) reference or bipolar (T12 against T10) derivative (Cracco, 1973; Cracco et al,

1975; Jones and Small, 1978; Delbeke and McComas, 1978; El Negamy and Sedgwick, 1978; Dimitrijevic et al, 1978; Ertekin, 1978; Phillips and Daube, 1980; Eisen and Odusote, 1980). The potential is triphasic (figure 5). The initial negative peak (the cauda potential) is a travelling wave reflecting conduction through the cauda equina and root (Phillips and Daube, 1980). It has a mean latency of 19 msec which increases between S1 and L1 segments. The second negative peak is of larger amplitude and has a fixed latency over the lower thoracic and upper lumbar cord of about 21 msec. It represents volume conducted post-synaptic activity in the dorsal columns (Cracco, 1973; Delbeke and McComas, 1978;

TABLE 2
SEP COMPONENTS ELICITED BY POSTERIOR TIBIAL NERVE STIMULATION AT THE ANKLE

Component	$\bar{x} \pm SD$ (msec)
N24	23.5 ± 1.6
N27	27.8 ± 1.7
P30	30.0 ± 2.2
N32	31.9 ± 1.7
P40	38.6 ± 2.2

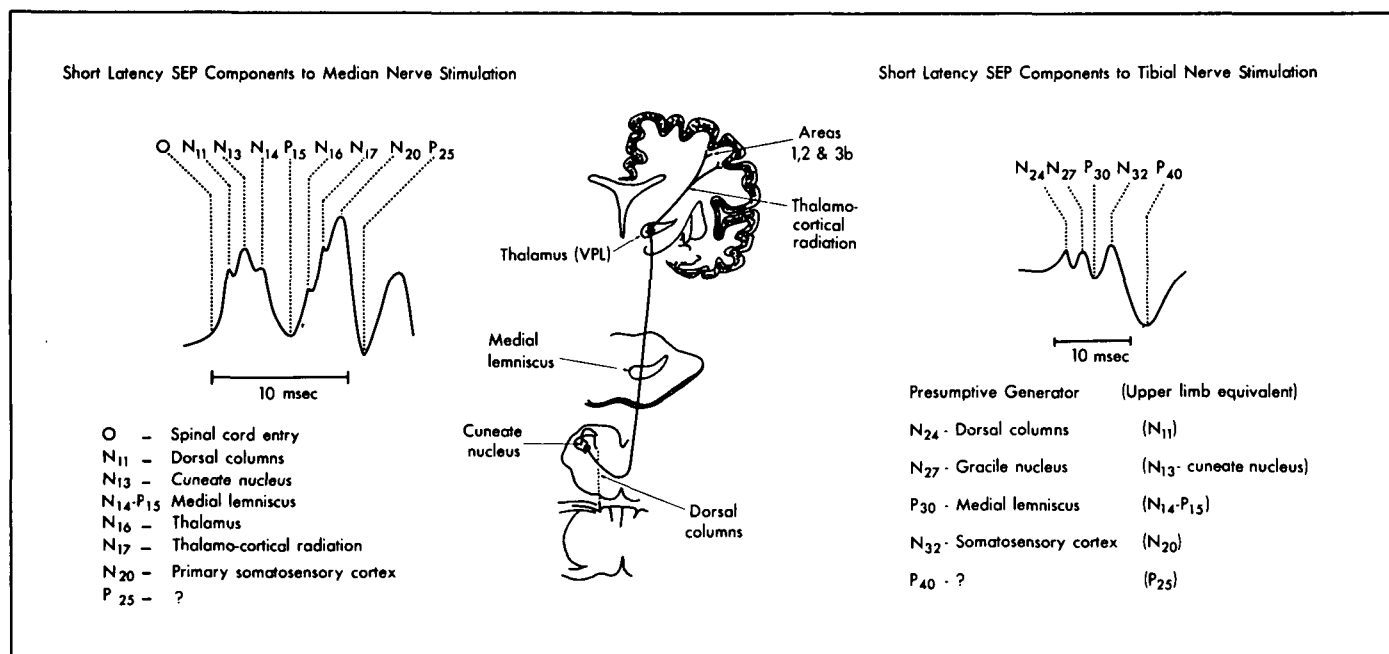


Figure 4 — The early latency SEP components and their presumed neural generators following stimulation of the median nerve at the wrist are compared with similar components obtained on stimulating the posterior tibial nerve at the ankle. The somatosensory pathway is shown for reference.

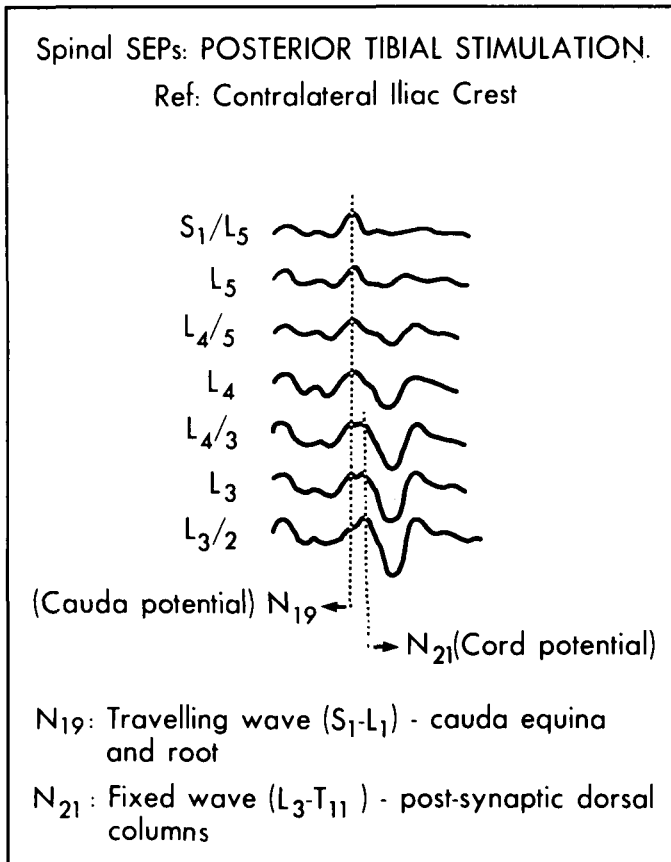


Figure 5 — The latency of N₁₉ shortens between S₁ and L₁, whereas that of N₂₁ is of fixed latency between the lower thoracic and mid lumbar spinal cord. A post-synaptic travelling wave can, however, be detected especially in children and infants, between the lower thoracic and mid or upper cervical cord.

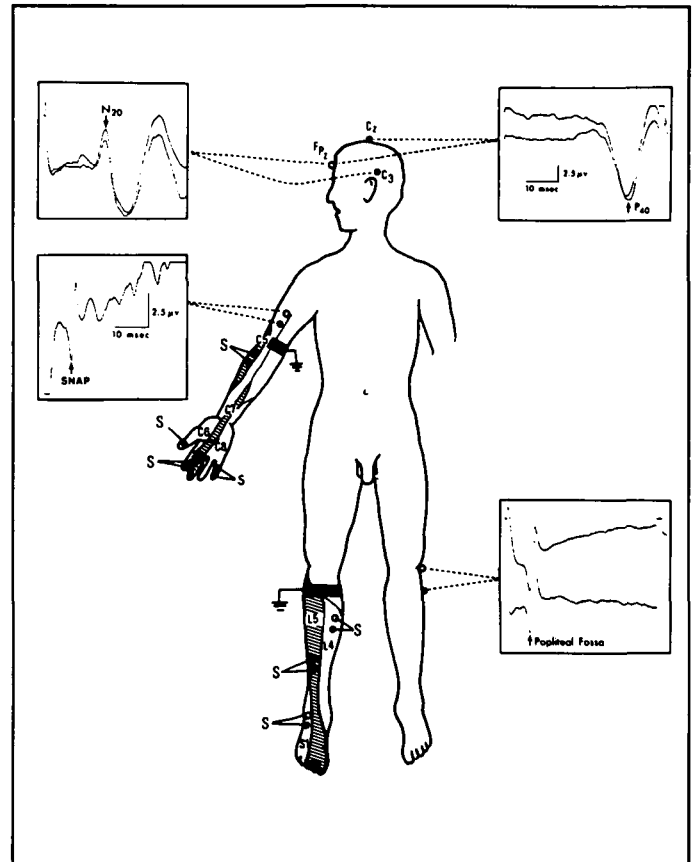


Figure 6 — Schema for segmental sensory stimulation using cutaneous nerves representative of one, or at most two, segmental levels or dermatomes. The peripheral sensory nerve action potentials were recorded using a near-nerve needle recording technique.

Ertekin, 1978; Phillips and Daube, 1980). In infants and young children, it is possible to record the spinal (but not cauda) potential as a travelling wave as it crosses the dorsal columns (Cracco et al, 1975). Dorsal column conduction velocity reaches adult values of about 65 M/sec after age four years. Unfortunately in the adult it is much more difficult to record a spinal potential evoked by leg stimulation at cervical levels. Summation of many thousands of responses is required (Cracco, 1973). An approximation of spinal transit time can be obtained more easily by simultaneously recording spinal and cortical SEPs. The difference in latencies for example of P₄₀ (the cortical SEP) and N₂₁ (the spinal SEP) measures about 17 msec. This conduction time also includes transit through the brainstem, thalamus, and thalamo-cortical projections (Eisen and Odu-sote, 1980).

Other SEPs

Cerebral responses can be evoked by stimulation of any accessible cutaneous nerve. This way segmental specificity (figure 6) can be achieved which is not possible by stimulating a mixed nerve trunk such as the median or posterior tibial nerves. The cutaneous branch of the musculocutaneous nerve (Trojaborg, 1976), the thumb, adjoining surfaces of the 2nd and 3rd fingers, and the 5th finger, are representative of the 5th, 6th, 7th, and 8th cervical dorsal roots respectively. The saphenous, superficial peroneal and sural nerves are representative of the 4th and 5th lumbar and 1st sacral roots respectively (see table 3). Segmental sensory stimulation is proving potentially useful in evaluating radiculopathies and plexopathies (Eisen and Elleker, 1980; Eisen and Hoirsch, 1982). In addition, when relating clinical cutaneous sen-

sory deficit with abnormal SEPs, cutaneous stimulation is more relevant than is mixed nerve stimulation, which also excites faster conducting muscle afferents (Burke et al, 1980). The cutaneous nerves referred to contain relatively few axons compared to a mixed nerve and many more responses need to be summated to record adequate subcortical SEP components. The scalp SEP is, however, comparable to that obtained using mixed nerve stimulation requiring the same amount of averaging (figure 6).

Trigeminal somatosensory evoked potentials (TSEPs) were originally used as an objective correlate for acute experimental pain (Chatrian et al, 1975), but have recently found wider appeal (Stöhr and Petruch, 1979; Bennett and Jannetta, 1980; Drechsler, 1980; Stöhr and Petruch, 1981; Eisen et al, 1981(a)). The general wave form of the TSEP is similar to that obtained with

TABLE 3
SEP COMPONENTS (N20/P40)
ELICITED BY SEGMENTAL
SENSORY STIMULATION

Nerve	Segment	Latency: $\bar{x} \pm SD$ (msec)
Musculocutaneous	C5	17.4 \pm 1.2
Median (finger 1)	C6	22.5 \pm 1.1
Median (finger 2/3)	C7	21.2 \pm 1.2
Ulnar (finger 5)	C8	22.5 \pm 1.1
Lateral femoral cutaneous	L3	31.8 \pm 1.8
Saphenous	L4	43.4 \pm 2.2
Superficial peroneal	L5	39.9 \pm 1.8
Sural	S1	42.1 \pm 1.4

arm stimulation. N13 is the N20 equivalent and measures between 11.5 and 15 msec. The proximity of the recording and stimulating electrodes makes for a troublesome stimulus artefact in which shorter latency components are buried. The artefact can be reduced by using mucosal stimulation (cathode over the inside of the lip, Bennett and Jannetta, 1980; Eisen et al, 1981(a)).

Terminal divisions of the pudendal nerve which supplies the penis or clitoris can be stimulated to evoke cerebral potentials. Preliminary data suggests a useful role of these SEPs in evaluating sexual or sphincter dysfunction (Haldeman et al, 1981). It is also possible to record adequate SEPs subsequent to stimulating small areas of skin. This is potentially useful in the medicolegal setting or when trying to validate organicity of dubious areas of cutaneous sensory deficit. SEPs can be compared following stimulation of homologous patches of skin (Desmedt, 1979).

Electrical stimulation is convenient because it is easily controlled. It is, however, non-physiological in terms of daily experience and excludes sensory receptor mechanisms. These problems have drawn recent attention to the feasibility of recording SEPs elicited by mechanical stimuli. Tapping (Pratt et al, 1979(a) and (b); Pratt and Starr, 1981), muscle stretch (Starr et al, 1981) and vibration (Johnson et al, 1980; Abbruzzese et al, 1980(a)) have all been used to evoke cerebral potentials. In general they are of smaller amplitude

and have fewer components than are recognizable using electrical stimulation, presumably because of the difficulty of inducing a totally synchronous volley. This problem can be approached from a somewhat different angle; that is, by using an interfering non-electrical stimulus of one sort or another superimposed upon routinely used stimuli to, for example, the median nerve (Jones, 1981; Abbruzzese et al, 1981; Rushton et al, 1981). Modulation of the electrically evoked SEP by interfering stimuli such as touch, movement, or vibration, may well prove to be a more sensitive indicator in disease than is presently available.

The Clinical Application of SEPs

There are several SEP characteristics that are either routinely measured or could be usefully measured. These are latencies of the various components, central conduction times, (i.e., the difference between component latencies), amplitude of individual components, morphology (are all normally occurring components present?), and dispersion (smoothness of the SEP). In addition to these characteristics, side to side differences of any or all of them can be very useful. Clearly latencies, particularly peak latencies, are the easiest to measure. If absolute values are used, they have to be correlated with arm length and body height. Unlike peripheral conduction velocities, central conduction times usually do not vary appreciably with either age (Desmedt and Cheron, 1982) or sex (Abbruzzese et al, 1980(b)). However, Green et al (1982) would disagree with this and have recently described significant effects of both age and sex on central conduction times. Although latencies, or central conduction times, are the easiest characteristic of the SEP to measure, they should not be expected to be abnormal in all clinical situations. So long as there are sufficient centrally conducting fast fibres,

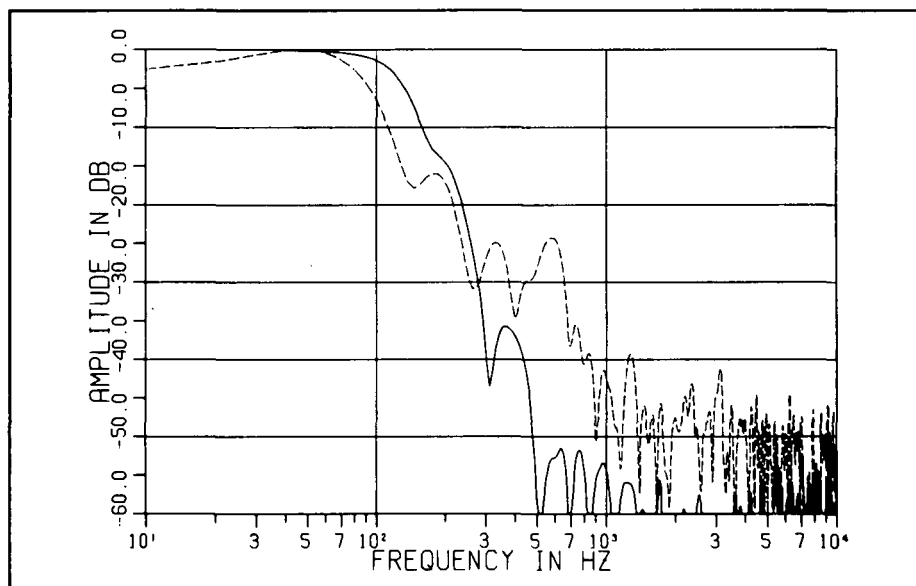


Figure 7 — The curves represent the combined data from 20 normal subjects (uninterrupted line) and 20 patients with multiple sclerosis (dotted line). The Fast Fourier transform used to look at the SEP dispersion shows that in the normal subjects there was very little energy above 380 Hz, as compared to the patients. The energy above 1000 Hz is mainly noise.

the onset of the response will still be normal. Amplitude is too variable to be meaningful in absolute terms, however, a 50% or greater side to side difference does indicate significant central conduction block or axonal loss, or both. Absence on one side of a normally occurring SEP component easily recordable on the other side might indicate disease, but insufficient data is available on this point to come to any firm conclusions. Theoretically, dispersion of the SEP reflected in its smoothness could be the most useful characteristic to measure. This is difficult however, and must not be confused with loss of smoothness due to technical factors such as a noisy recording. Using a Fast Fourier transform technique, it has been possible to quantitate dispersion of the SEP elicited by median nerve stimulation (Eisen et al, 1982(a)). A control group was compared with a group of patients having definite or suspected MS. Only those records were included in which noise, because of poor recording technique, had been carefully excluded. Normal wave forms had very little energy above 380 Hz compared to the multiple sclerosis patients (figure 7). The various aspects of the SEP should be considered abnormal only when three or more standard deviations outside their normal means.

Peripheral Nerve Disease

A sizable, often fairly normal looking SEP can be recorded even when the peripheral sensory nerve action potential (SNAP) is too small and presumably too desynchronized to be recognizable (Desmedt and Noel, 1963; Giblin, 1964; Eisen and Elleker, 1980; Assmus, 1980). This finding can be used to follow early return of peripheral sensory function post-traumatically (Assmus, 1980), and to measure sensory conduction velocity in chronic neuropathies when SNAPs are unrecordable. The ability to record a SEP in the absence of a SNAP indicates central amplification of the incoming peripheral volley. Central amplification is a normally occurring phenomenon doubling the response when stimulus intensities used to evoke SEPs and SNAPs are between 50% and 70% above threshold (Eisen et al,

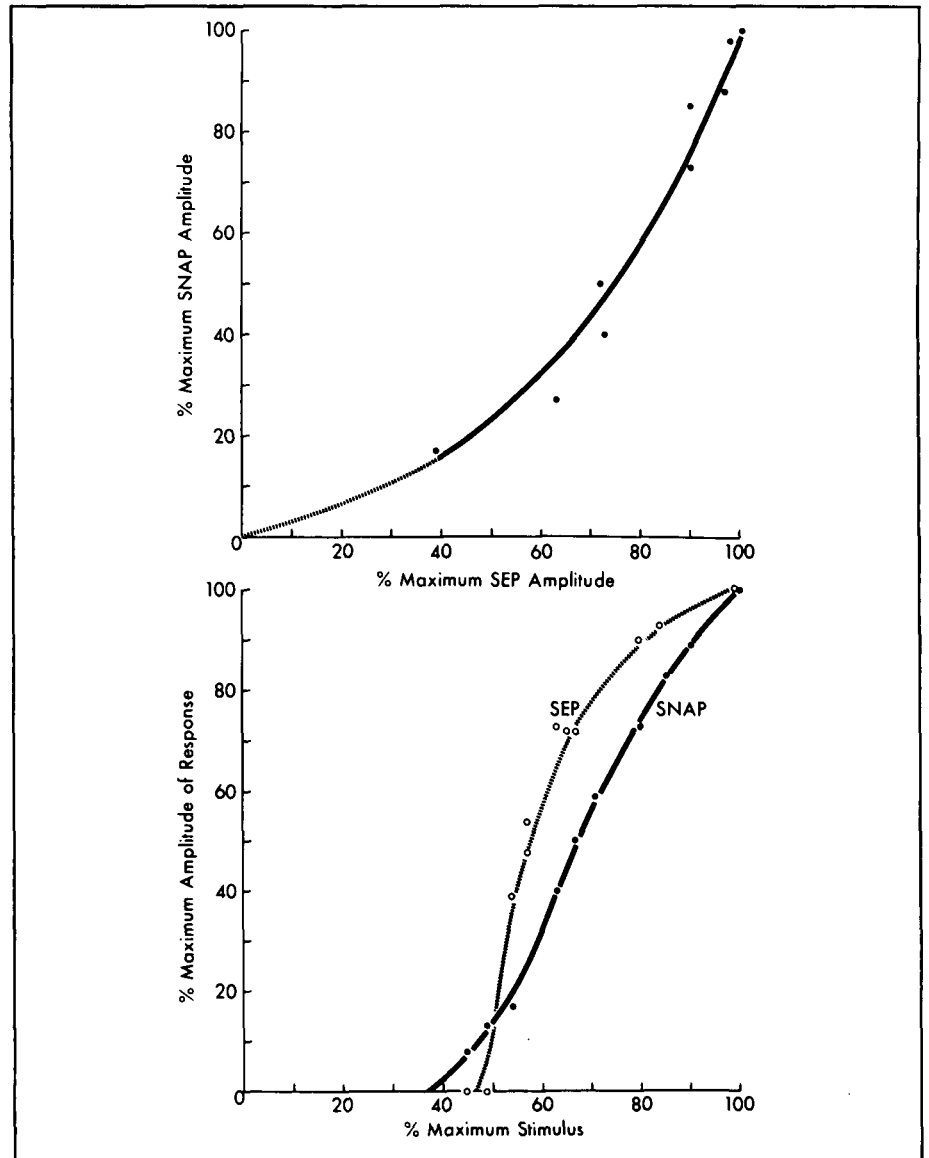


Figure 8 — The top half of the figure shows the increment of SNAP versus SEP amplitude occurring with increasing stimulus. 100% stimulus is defined as 2.5 times threshold, so that threshold stimulus is 40% of maximum. Below this the curve is interrupted, indicating no points are experimentally obtainable. Had the SNAP and SEP amplitudes changed to the same degree, the relationship would be a linear one. In the bottom half of the figure, plotted from the same normal subject, is shown the relationship of the SEP and SNAP amplitudes expressed as a percentage of their maxima, and how they increase with increasing stimulus. The difference between these two is representative of the central gain which between 40 and 70% of maximum stimulus is roughly about 2.

1982(b)). This is shown in figure 8. SEPs can also be utilized to investigate peripheral nerve disease in certain nerves, for example, the lateral femoral cutaneous, which normally are difficult to evaluate using routine clinical neurophysiological methods. Figure 9 illustrates SEPs obtained from a patient having unilateral meralgia paresthetica. These potentials were evoked by

stimulating the lateral femoral cutaneous nerve.

Simultaneous recording of SNAPs and SEPs has revealed unexpected slowing of central in addition to peripheral conduction in diabetes (Cracco and Castells, 1980; Gupta and Dorfman, 1981), Guillain-Barre syndrome (McLeod, 1981), and Charcot-Marie-Tooth disease (Halliday et al,

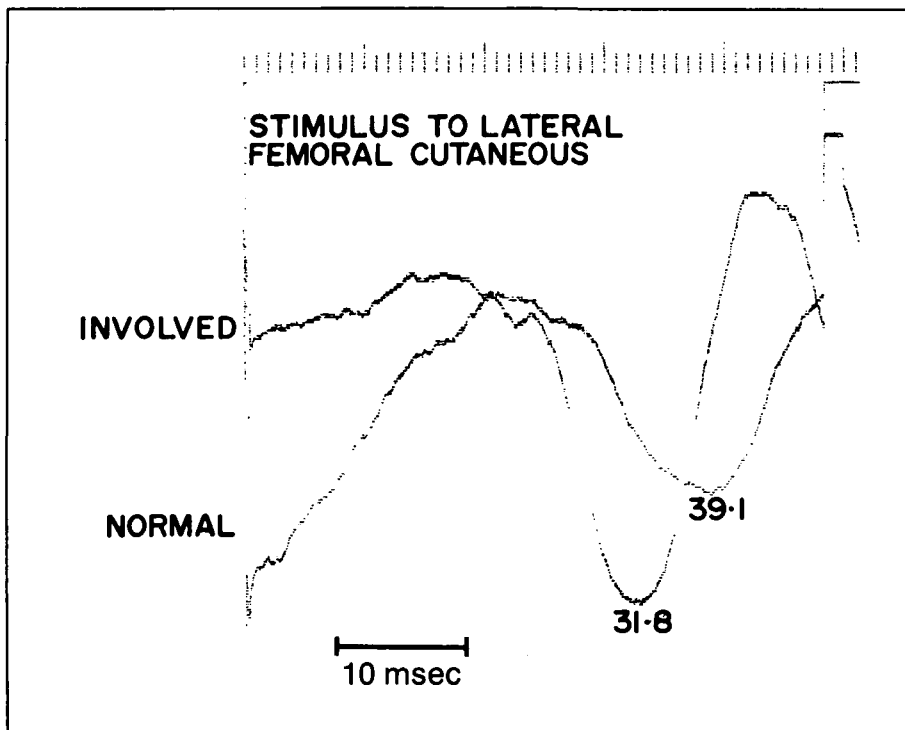


Figure 9 — The SEPs were recorded from a patient with meralgia paresthetica and elicited by stimulating the lateral femoral cutaneous nerve. On the involved side the latency of P40 was prolonged and the amplitude of the response was reduced compared to the normal side, indicating conduction block and slowing along this nerve.

1981). In contrast, slowed central but normal peripheral conduction may occur in vitamin B-12 deficiency (Krumholz et al, 1981). The peripheral component of subacute combined degeneration is controversial and may simply reflect concurrent disease or deficiency predisposing to neuropathy (Swash and Schwartz, 1981).

Radiculopathies and Plexopathies

Radiculopathies may be difficult to evaluate electrophysiologically. F-wave and needle electromyographic abnormalities, including those encountered in paraspinal muscles, reflect only dysfunction of the motor root. H-reflex abnormalities are limited to disease of the S1 sensory root (Tonzola et al, 1981). However, SEPs elicited by segmental sensory stimulation (Eisen and Elleker, 1980) may provide valuable information in disc disease involving common cervical and lumbosacral levels, with predominant or even isolated sensory abnormalities, see figure 10 (Eisen and Hoirsch, 1982). A good correlation between the SEP and

myelographic and clinical abnormalities (or absence of these) has been demonstrated (Eisen and Hoirsch, 1982). SEPs evoked by mixed stimulation are less likely to be abnormal in a single root lesion because of the multisegmental input. However, myelopathy due to cervical disc disease has resulted in abnormal cervical spinal and scalp recorded SEPs following median nerve stimulation (El Negamy and Sedgwick, 1979; Ganes, 1980; Siivola et al, 1981).

Following traumatic plexopathies, recordable SEPs in the face of attenuated or absent SNAPS indicate continuity between peripheral and central structures. This finding is helpful because modern microsurgical techniques can aid axonal regeneration when the lesion lies distal to the dorsal root ganglion cell. Comparison of SEPs evoked through median versus ulnar nerve stimulation, or better still using segmental cutaneous nerve stimulation, are helpful in anatomical localization of the part of the plexus involved (Jones, 1979; Eisen and Hoirsch, 1982).

Multiple Sclerosis

In definite multiple sclerosis (MS) visual (VEP), auditory (BAEP), and somatosensory (SEP) evoked potentials are virtually always abnormal when the system tested is clinically involved (Chiappa, 1980). The most useful role of evoked potentials in MS is documenting a second clinically silent lesion in suspects (McDonald, 1980). For example, finding abnormal VEPs (Halliday et al, 1973; Asselman et al, 1975; Hennerici et al, 1977; Matthews et al, 1977; Paty et al, 1979) or trigeminal SEPs (Eisen et al, 1981(a)) in progressive spinal MS indicates a second silent lesion "above the neck". Similarly, abnormal SEPs which have been found in patients with optic neuritis but without other neurological abnormalities would indicate a more widespread disease (Eisen et al, 1981(b)). Based upon such findings, the patient's category of MS can be changed from suspected to probable, or probable to definite (Trojaborg et al, 1981). Although each type of evoked potential has a role, multimodality studies in MS indicate that BAEPs give a much lower diagnostic yield than do either VEPs or SEPs (Chiappa, 1980; Green et al, 1980; Purves et al, 1981; Khoshbin and Hallett, 1981). The best yield is given by SEPs using leg stimulation, presumably reflecting the large extent of neuraxis screened (Trojaborg and Peterson, 1979; Green et al, 1980). Routinely a combination of VEPs and SEPs using leg stimulation is recommended and is not overly time consuming. The addition of arm SEPs and cervical spinal SEPs and BAEPs is unlikely to add significantly to the overall diagnostic yield.

Demyelination is not a prerequisite for an abnormal SEP. Abnormal SEPs have been recorded in hereditary spastic paraplegia (Thomas et al, 1981; Pedersen and Trojaborg, 1981), Friedreich's and other hereditary cerebellar ataxias (Pedersen and Trojaborg, 1981) and subacute combined degeneration due to vitamin B-12 deficiency (Fine and Hallett, 1980; Krumholz et al, 1981). In these diseases degeneration of the centrally directed axon from the first sensory neuron is the primary pathological process (Thomas et al, 1981).

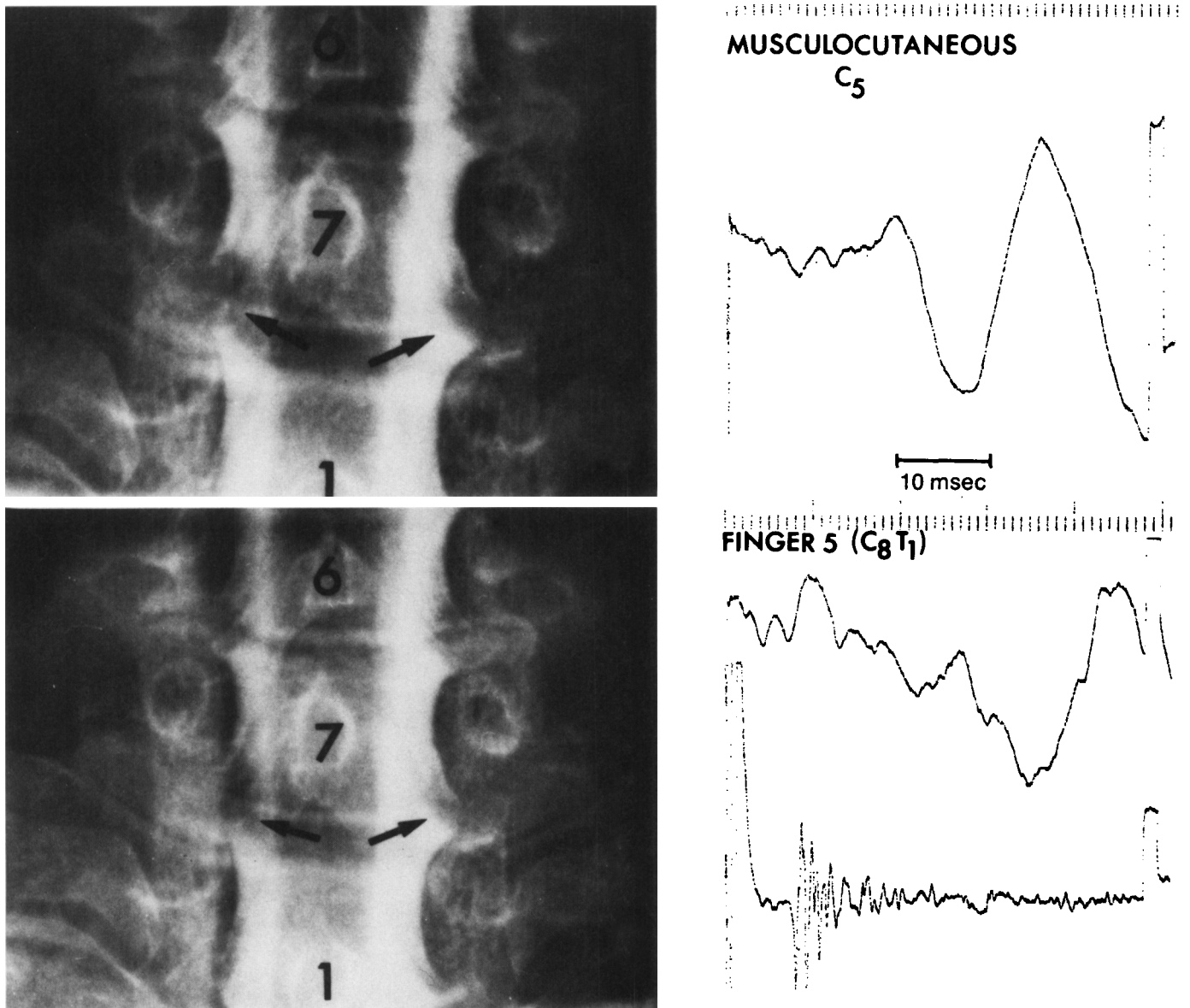


Figure 10 — This patient's right C8 root was compressed by a disc protrusion (compare arrows on myelogram). The corresponding SEPs shown on the right side of the figure show that on stimulating finger 5 no real recordable SEP was seen despite a normal peripheral sensory action potential. A segment or two higher, at C5, the SEP is normal.

Spinal Cord and Cerebral Trauma and Ischemia

Following spinal cord transection which is considered clinically complete, SEPs are and remain unrecordable. In contrast, their preservation or return in the early post-traumatic period is associated with a generally good prognosis (Perot, 1973; Rowed et al, 1978; Spielholz et al, 1979; Dorfman et al, 1980). SEPs have also been valuable in monitoring the course of coma from head injury (Greenberg et al, 1977; Hume and Cant, 1981) and other

causes (DeLaTorre et al; Cant, 1980). Central conduction time (N20 - N14), which is independent of sedative or paralyzing drugs, is a useful indicator of outcome. If central conduction time is initially normal or rapidly becomes so, a favourable prognosis can be anticipated (Hume and Cant, 1981). On the other hand, a progressively lengthening conduction time or one that remains prolonged for over 30 days signals little hope of functional recovery after head injury.

There has been recent interest in us-

ing SEPs to monitor spinal cord function during intraoperative manipulation (Brown and Nash, 1979). This would negate the need to awaken the patient during surgery. Experimentally, the cortical SEP is attenuated or abolished by ischemia before the spinal SEP. However, they are both affected to a similar degree and with the same time course following spinal cord manipulation (Larson et al, 1980). This suggests that the cortical SEP might be safely used as a mirror of spinal cord dysfunction and, furthermore, that

simultaneous recording of both spinal and cortical SEPs could help differentiate between ischemic and/or mechanical dysfunction occurring intra-operatively.

SEPs have been used thus far in a limited way in the investigation of cerebral ischemia and cerebrovascular disease as a whole. Studies relating to cerebral blood flow (CBF) and SEPs have demonstrated that when CBF falls below 12 ml/100 gm/min, the SEP is unrecordable. Below 16 mg/100 gm/min, the SEP amplitude is attenuated by greater than 50%. This has been termed "the flow threshold for failure of neuronal electrical function" (Symon, 1980). A normal SEP is recordable when the CBF is over 20 ml/100 gm/min. SEPs could therefore be useful in detecting borderline cerebral ischemia and the test could complement the presently utilized method of monitoring of EEG during carotid endarterectomy. An interesting aspect for the future is the correlation of SEP abnormalities in cerebrovascular disease and those detected through positron emission tomography.

Despite the fact that the first SEP was recorded quite some time before the first visual evoked potential or auditory brainstem evoked potential, the initial promise of somatosensory evoked potential recording was not fulfilled and the test fell into disrepute. This review has however, demonstrated how, in the last several years, there has been considerable revival in the value of the SEP. Indeed Broughton (1967) concluded his Ph.D. thesis with the following: "These results all tend to indicate that the evoked potential technique will become a very useful ancillary diagnostic tool and over the years should yield much interesting data on normal and abnormal functioning of sensory systems in man". The clairvoyance of this statement has in the intervening 15 years been fully validated.

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