infants. Handbook Electroenceph. clin. Neurophysiol., 1975, 6B: 6–23.
Gibbs, F.A. and Gibbs, E.L. Atlas of Electroencephalography, Vol. 1. Addison Wesley, Cambridge, Mass., 1950.
Havlicek, V., Childiaeva, R. and Chernick, V. EEG frequency spectrum characteristics of sleep states in full-term and preterm infants. Neuropädiatrie, 1975, 6: 24–40.
Joseph, J.P., Lesèvre, N. and Dreyfus-Brisac, C. Spatio-temporal organization of EEG in premature infants and full-term new-borns. Electroenceph. clin. Neurophysiol., 1976, 40: 153–168.
Kiloh, L.G., McComas, A.J. and Osselton, J.W. Clinical electroencephalography. 3rd Ed. Appleton-Century-Crofts, New York, 1972.
Kooi, K.A. Fundamentals of electroencephalography. Harper and Row, New York, 1971.
Petersén, I., Selldén, U. and Eeg-Olofsson, O. The evolution of the EEG in normal children and adolescents from 1 to 21 years. Handbook Electroenceph. clin. Neurophysiol., 1975, 6B: 31–68.
Schulte, F.J. and Bell, E.F. Bioelectric brain development. An atlas of EEG power spectra in infants and young children. Neuropädiatrie, 1973, 4: 30–45.

Electroencephalography and Clinical Neurophysiology, 1977, 43: 853–863

SHORT LATENCY POTENTIALS RECORDED FROM THE NECK AND SCALP FOLLOWING MEDIAN NERVE STIMULATION IN MAN

S.J. JONES *

Institute of Neurology, The National Hospital, Queen Square, London W.C.1. (England)

(Accepted for publication: May 5, 1977)

The waveform and distribution of the human scalp-recorded SEP following median nerve stimulation have been described by several authors (Goff et al. 1962; 1977; Giblin 1964) with little essential disagreement, although a number of theories have been put forward concerning the identity of the afferent pathways involved and the location of the cortical areas responsible for generating the various components (Allison et al. 1974). There have been fundamental differences, however, between accounts describing the short latency (up to 15 msec) potentials recorded from the cervical spine and elsewhere (Cracco 1973; Cracco and Cracco 1976; Matthews et al. 1974), with correspondingly little agreement as to whether these potentials originate in the cerebrum, cerebellum, spinal cord or peripheral nervous system. Clearly there is a need for clarification, which should not be beyond the capacity of established surface-recording techniques provided the sites for stimulating, recording and reference electrodes are chosen with regard to neuroanatomy and to the likely distribution of potentials throughout the body.

This report presents findings from normal subjects, obtained with percutaneous stimulation of median nerve fibres at elbow, wrist and fingers, which enable the generator sites of the short latency SEP components to be deduced within a narrow range of possibilities. It is hoped that this may extend the clinical applications of a technique which provides a non-invasive means of investigating the integrity of the afferent somatosensory pathways in diseases of the central nervous system (for example Small 1976).

Method

Responses were recorded to the stimulation of 33 arms in 23 normal subjects, 5 male and 18 female, whose ages ranged from 19 to 33 years with a mean of 24 years. The stimulus was a capacitative discharge, time constant 50 μsec, delivered through an isolating transformer to the skin overlying the median nerve at the wrist via a saddle-type bipolar electrode with the cathode 3 cm distal to the anode. In 4 subjects stimuli were also delivered to the median nerve at the elbow, and in 4 subjects to the base of the first and second fingers using ring electrodes. The stimulus strength was adjusted to between 3 and 4 times the sensory threshold voltage, so as to produce (for stimulation at the wrist or elbow) a moderate twitch of the hand. Stimuli were delivered at 2/sec (1/sec for the first 6 subjects) and could be tolerated without discomfort for long periods. Subjects sat in an armchair or lay supine, and were instructed to relax and sleep if they wished.

Electrodes were conventional 9 mm silver/silver chloride discs attached to the skin overlying the spine between the sixth thoracic

* Member of the External Staff of the Medical Research Council.

(T6) and second cervical (C2) vertebrae. Recordings were also made from sites overlying the clavicles, mastoid processes, and the hand area of the somatosensory cortex ipsilateral and contralateral to the stimulated arm. A common mid-frontal reference electrode (Fz, 10-20 system) was employed, and additionally in 5 subjects a midline lower thoracic, lumbar or sacral reference. The frequency response of the amplifiers was better than −3 dB at 5 kc/sec with a time constant of 1 sec. Four hundred responses were summed with sampling rates between 5 and 8 points/msec (16 or 17 points/msec for 4 subjects), recording from up to 16 'active' electrodes simultaneously. To improve the signal/noise ratio further any number of summed responses could be added together using a PDP12 computer. Summed responses recorded from two sites simultaneously with a common reference could also be subtracted in order to simulate the waveform which would have been obtained from the first site with reference to the second.

Results

The general waveform of responses produced by stimulation at the wrist and recorded over the spine with a mid-frontal reference electrode consisted of a complex of mainly negative-going components (Fig. 1) with latencies directly related to arm length. In the majority of subjects it was possible to distinguish at least 4 negative components ($N\bar{9}$, $N\bar{1}\bar{1}$, $N\bar{1}\bar{3}$, and $N\bar{1}\bar{4}$) in recordings made over the seventh cervical (C7) vertebra where amplitudes were generally near-maximal. Equivalent components were recorded at sites above and below C7 with little change in peak latency, although the distribution of amplitudes differed considerably between components (Table I). Components were distinguished from each other mainly on grounds of differing amplitude distributions as described below. Components $N\bar{1}\bar{1}$ and $N\bar{1}\bar{3}$ were sometimes resolved into two of more sub-components, with the subcomponents of $N\bar{1}\bar{1}$ differing in their amplitude distributions but those of $N\bar{1}\bar{3}$ essentially similar. For example in Fig. 1, subject S.J., $N\bar{1}\bar{1}$ is resolved into three subcomponents with differing amplitude distributions, and $N\bar{1}\bar{4}$ (not clearly seen at recording sites over the spine) is distinguished from two $N\bar{1}\bar{3}$ subcomponents by its near-synchrony with a small positive potential recorded over the hand region of the somatosensory cortex contralateral to the stimulated arm (demonstrated by subject J.P.). In line with previous classifications (for example Goff et al. 1977)

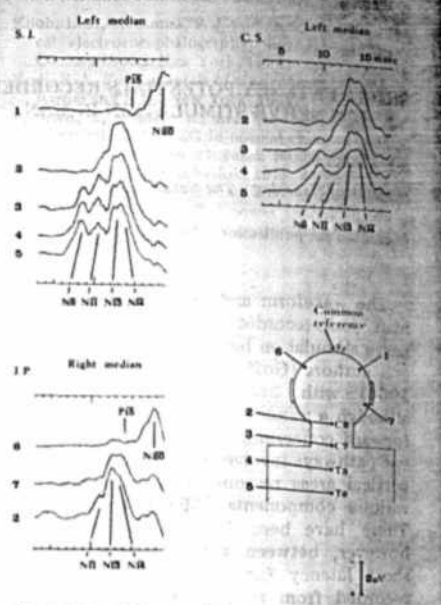

Fig. 1. Potentials recorded over the somatosensory cortex hand area, cervical and thoracic vertebrae and mastoid process following median nerve stimulation at the wrist. The common reference electrode was at Fz and the potentials are the averaged responses to 2400 (S.J., J.P.) or 2000 (C.S.) stimuli at 2/sec. The average sampling rate was 16 (S.J., J.P.) or 17 (C.S.) points/msec.

TABLE I

Means and standard deviations (σ_{n-1}) of response amplitude and latency following stimulation at the wrist

Negative components were recorded over the sites indicated with a reference electrode at Fz. Mas 1 and 2 are the mastoid processes ipsilateral and contralateral to the stimulated arm. Amplitude information was rejected from 2 subjects owing to uncertainty of calibration.

Component	Recorded over	Identified in (proportion of subjects)	Amplitude (μV) Mean ± S.D.	Latency (msec) Mean ± S.D.	Latency after $N\bar{9}$ (msec) Mean ± S.D.
$N\bar{9}$	T3	14/16	1.5 ± 0.4	8.7 ± 0.5	
	C7	21/23	0.8 ± 0.4	8.7 ± 0.6	
$N\bar{1}\bar{1}$	T3	16/16	1.6 ± 0.7	10.7 ± 0.5	1.9 ± 0.4
	C7	23/23	1.9 ± 0.7	11.2 ± 0.6	2.4 ± 0.4
	C2	22/22	1.5 ± 0.5	11.5 ± 0.5	2.6 ± 0.3
$N\bar{1}\bar{3}$	T3	16/16	2.7 ± 0.9	12.8 ± 0.6	4.1 ± 0.5
	C7	23/23	3.3 ± 0.8	12.7 ± 0.6	4.0 ± 0.4
	C2	22/22	3.1 ± 0.7	12.9 ± 0.5	4.1 ± 0.5
$N\bar{1}\bar{4}$	C2	22/22	2.1 ± 0.7	14.2 ± 0.6	5.4 ± 0.5
	Mas 1	8/ 9	1.5 ± 0.5	13.9 ± 0.7	5.1 ± 0.3
	Mas 2	8/ 9	1.5 ± 0.5	14.0 ± 0.7	5.2 ± 0.3

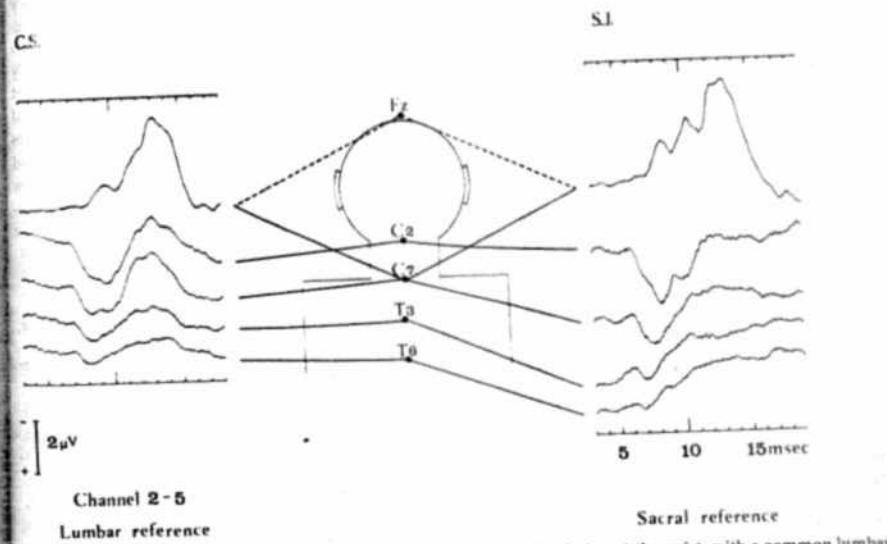

Fig. 2. Potentials recorded over the spine following median nerve stimulation at the wrist, with a common lumbar (C.S.) or sacral (S.J.) reference electrode. Channel 1 shows the potential simultaneously recorded over C7 with the reference at Fz. 2000 (C.S.) or 1200 (S.J.) responses were averaged to stimulation at 2/sec with sampling rates 17 or 16 points/msec respectively.

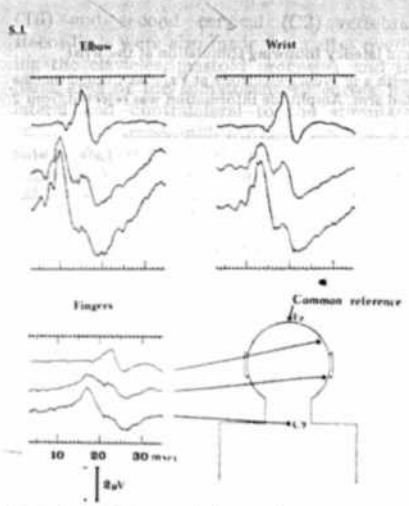

Fig. 3. Potentials recorded over the somatosensory cortex hand area, mastoid process and seventh cervical vertebra (in descending sequence) following stimulation at the elbow, wrist and fingers. The common reference electrode was at Fz and 1200 (elbow, wrist) or 2400 (fingers) responses were averaged at 2/sec, sampling rate 8 points/msec.

the scalp-recorded positive potential will be called $P\overline{15}$ and the following negative peak $N\overline{20}$.

With a lower thoracic, lumbar or sacral reference (Fig. 2) the response was usually resolved into components of similar latency to those seen in mid-frontal reference recordings, although the earlier components were inverted in polarity and succeeding negative components were sometimes of considerably smaller amplitude (for example subject S.J.). The sacral reference response was maximal between C7 and C2, and below C7 the latency of the initial positive peak was slightly shorter than that of $N\overline{9}$.

Responses recorded over the spine, mastoid process and contralateral somatosensory cortex with a mid-frontal reference were of broadly similar waveform when the stimulus was delivered at the elbow or fingers (Fig. 3), although latencies were correspondingly reduced or increased relative to responses following stimulation at the wrist. Components equivalent to $N\overline{9}$, $N\overline{11}$ and $N\overline{13}$ were clearly identifiable in the response to stimulation at the elbow, as were the subcomponents of $N\overline{11}$ and $N\overline{13}$. The latency differences between equivalent components recorded with stimulation at the wrist and the elbow were calculated for each component or subcomponent, including $P\overline{15}$ and $N\overline{20}$ from the scalp-recorded response, and the values for one subject are given in Table II. Latency differences between equivalent features all fell in the range 3.2—3.4 msec, indicating that all the major early components of the response depended on activity in median nerve fibres with a conduction time of 3.2—3.4 msec between stimulation sites at the wrist and the elbow. This gave a value of between 65 and 75 m/sec for the forearm conduction velocity of the median nerve fibres concerned.

TABLE II

Mean latencies of components recorded from the sites indicated following stimulation at the wrist and the elbow, subject S.J. $N\overline{14}$ was not clearly resolved with stimulation at the elbow.

Component	Recorded over	Mean latency (msec) Stimulation at		Difference
		Wrist	Elbow	
$N\overline{9}$	T3, C7	8.7	5.3	3.4
$N\overline{11}$	T3, C7	10.8	7.5	3.3
	C7, C2	11.4	8.1	3.3
$N\overline{13}$	C7, C2	12.6	9.2	3.4
	C7, C2	13.2	10.0	3.2
$N\overline{14}$	C2, Mastoids	14.2	10.8	3.4
$P\overline{15}$	Scalp	14.6	11.4	3.2
$N\overline{20}$	Scalp	18.3	15.1	3.2

Components corresponding to $N\overline{13}$, $P\overline{15}$ and $N\overline{20}$ (possibly $N\overline{11}$ and $N\overline{14}$ also) were identified when the stimulus was delivered at the base of the first and second fingers (Fig. 3), although amplitudes were smaller and $N\overline{11}$ and $N\overline{14}$ appeared only as shoulders to $N\overline{13}$, at C7 and the mastoid process respectively. The temporal relationship between components was similar for finger stimulation as for nerve trunk stimulation at the wrist or elbow, although the peak of $N\overline{20}$ appeared to be slightly delayed relative to $N\overline{13}$ with stimulation of the fingers. It was concluded that none of the components from $N\overline{11}$ to $N\overline{20}$ can have been wholly due to antidromic conduction in motor fibres, since such fibres are not present at the base of the fingers and stimulation of the fingers was not seen to produce a direct motor response.

$N\overline{9}$ was recorded in 21 out of 23 subjects at all sites between T6 and C4, with maximal amplitude at or below T3. The component was not normally seen in recordings made over C2 with a mid-frontal reference electrode. No progression in peak latency was seen in recordings from the spine with a mid-frontal reference, but a progression in onset latency was occasionally observed between T6 and C7, onset at the rostral site being delayed by 1—1.5 msec (Fig. 1, subjects S.J. and C.S.). With a reference electrode over the lower spine (Fig. 2) the component corresponding to $N\overline{9}$ was positive in polarity, maximal at C2, and exhibited an increase in peak latency of 1—1.5 msec between T6 and C2. In some subjects (for example C.S.) the shift in peak latency was apparently due to the presence of two subcomponents with the later predominating at rostral recording sites, but in others (for example subject S.J.) the progression appeared to be more gradual. These properties of $N\overline{9}$ appear to resemble those of a rostrally moving dipolar generator located between T6 and C2 in the vertical plane, with the positive pole oriented towards the head and the later, rostral, part of the generator contributing a fairly large positive potential equally at the midfrontal reference electrode and at C2. The subcomponents sometimes seen in lumbar or sacral reference recordings might be due to a discontinuity in the movement of the generator, for example an angular change of direction, or to an early negative potential recorded by the reference electrode.

A large amplitude positive—negative biphasic potential was recorded from the region of the clavicle on the stimulated side, with a reference electrode at C2 or Fz (Fig. 4). The major negative peak was of similar latency to that of $N\overline{9}$ when the active electrode was situated at the lateral extremity of the clavicle, and exhibited a progressive increase in latency of up to 1 msec as the electrode was moved medially by approximately 8 cm. At the medial extremity the latency of the negative peak was similar to that of a small negativity recorded over the mastoid process ipsilateral to the stimulated arm with a reference electrode over the contralateral mastoid. This latter peak underwent an inversion of polarity when the stimulus was applied to the other arm. It is likely on grounds of latency and distribution, therefore, that the biphasic potential and $N\overline{9}$ were both due to the electric field of a diagonally oriented, proximally moving dipolar generator, located in the region of the brachial plexus on the stimulated side. This location is consistent with a latency of 7—10 msec, given that the median nerve fibres responsible are those which conduct at 65—75 m/sec in the forearm.

In mid-frontal reference recordings $N\overline{11}$ was usually identified at all sites between T6 and C2, with maximal amplitude in the cervical or upper thoracic region. In most subjects

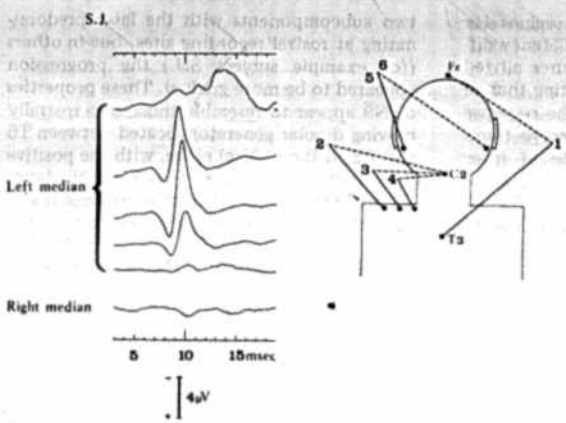

Fig. 4. Potentials recorded over the third thoracic vertebra, left clavicle and left mastoid process with the reference electrode at Fz, C2 or the right mastoid process respectively. 1200 responses were averaged at 2/sec to stimulation of the left (Channels 1–5) and right (Channel 6) median nerve at the wrist, sampling rate 16 points/msec. The channels are numbered in descending sequence.

$N\overline{11}$ recorded at C2 was not seen as a distinct peak, but merely as a shoulder to the following component which was invariably of larger amplitude. In many subjects (for example Fig. 1, subject C.S.) $N\overline{11}$ recorded at C7 was again seen only as a shoulder to the following component, but in the majority of subjects (for example Fig. 1, subject S.J.) was present as a distinct peak.

There was a highly significant increase in $N\overline{11}$ peak/shoulder latency between C7 and C2 ($t = 4.863$, $P < 0.001$, 22 subjects), probably due to the presence of two or more distinct subcomponents which were resolved at C7 in 9 out of 23 subjects. In Fig. 1 subject S.J. 3 $N\overline{11}$ subcomponents are resolved, with the second predominating at C7 and the third at C2, although these two subcomponents are both present in more caudal channels also.

Further evidence for the splitting of $N\overline{11}$ at C7 is provided by the standard deviation of the latency after $N\overline{9}$. The major subcomponent of $N\overline{11}$ at C7 occurred with a mean latency of 2.5 msec after $N\overline{9}$ for the 9 subjects in whom $N\overline{11}$ was demonstrably bifid, with a standard deviation of 0.4 msec. When two $N\overline{11}$ subcomponents were distinguished the first was found to occur at a mean of 2.0 msec after $N\overline{9}$ and the second at 2.6 msec. The standard deviation for each of these values was approximately 0.25 msec. The reduction in standard deviation when $N\overline{11}$ subcomponents were computed individually indicates that an additional variable was introduced when only the larger of the two (usually but not invariably the second) was considered, and suggests therefore that the resolution of two subcomponents was real and not an artefact due to residual high-frequency noise.

In lower thoracic, lumbar or sacral reference recordings (Fig. 2) $N\overline{11}$ was ill-defined at C7 and T3 but remained negative in polarity. At C2, however, the peak was sometimes prominent and positive-going with respect to the baseline and to later components (for example subject S.J.). This reversal of polarity suggests that at least the earlier subcomponent(s) of $N\overline{11}$ may be due to the field of a dipolar generator, with the dipole oriented in a vertical plane and situated in the cervical region between the C7 and C2 vertebrae, positive pole rostrally.

$N\overline{13}$ was the major peak recorded from the spine with a mid-frontal reference electrode, and was seen in all subjects at all sites between the T6 and C2 vertebrae. $N\overline{13}$ amplitude was maximal in the region of C7, falling off fairly rapidly towards T3 ($t = 5.025$, $P < 0.001$, 15 subjects) and fractionally towards C2 ($t = 2.414$, $P < 0.05$, 20 subjects). Such fine distinctions are of doubtful validity, however, owing to the possibility of distortion by overlap with earlier and later components. In 12 out of 23 subjects $N\overline{13}$ was bifid at C7 and/or C2, with mean peak latencies of 12.6 and 13.5 msec at C2 (9 subjects). The earlier peak was of slightly greater mean amplitude (3.2 μV compared with 3.0 μV) although in individuals the reverse was frequently the case. At C2 the standard deviation of $N\overline{13}$ latency after $N\overline{9}$ was reduced by about one-third when the first and second subcomponents were computed individually (compared with the value obtained by considering only the larger subcomponent in the same group of 9 subjects), suggesting that the distinction between subcomponents was real and not an artefact due to residual noise.

In recordings made with reference to the lower spine (Fig. 2) the component corresponding to $N\overline{13}$ was of smaller amplitude but negative in polarity and maximal at C4–C7. This suggests, first, that a negative potential was detected by 'active' electrodes over the neck, second that a simultaneous positive potential was recorded by the mid-frontal 'reference' electrode, presumably due to a generator situated rostral to C2. Empirically, therefore, the distribution of $N\overline{13}$ was also consistent with the potential field of a dipolar generator, situated between the cervical spine and the cerebrum with the positive pole rostrally.

$N\overline{14}$, also seen in all subjects, occurred as a shoulder to $N\overline{13}$ in recordings from the spine, and could usually be distinguished at all sites between T6 and C2 in mid-frontal reference

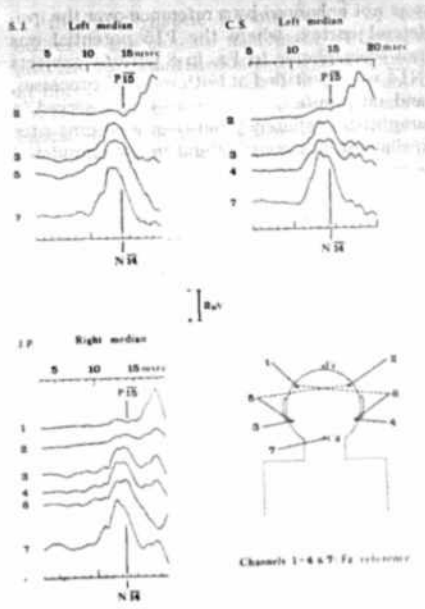

Fig. 5. Potentials recorded over the somatosensory cortex hand area, mastoid processes and C2 following median nerve stimulation at the wrist. Cortical and cervical electrodes were referred to Fz, and the mastoid processes to Fz or the somatosensory cortex hand area contralateral to the stimulated arm. Responses were averaged to 3200 (S.J.), 2000 (C.S.) or 2400 (J.P.) stimuli at 2/sec with sampling rate 16 (S.J., J.P.) or 17 (C.S.) points/msec.

recordings. The peak was enhanced relative to $N\overline{13}$ (with an apparent slight increase in latency) when recorded over the mastoid process on either side, and further enhanced when the reference electrode was located over the somatosensory cortex contralateral to the stimulated arm (Fig. 5). This latter enhancement was due to the near-synchrony of $N\overline{14}$ recorded at the mastoids with the small positive potential ($P\overline{15}$) frequently seen preceding $N\overline{20}$ over the contralateral somatosensory cortex in mid-frontal reference recordings. $N\overline{14}$

was not enhanced by a reference over the ipsilateral cortex, where the $\overline{P15}$ potential was not seen relative to Fz. In 8 out of 9 subjects $\overline{N14}$ was identified at both mastoid processes, and no significant difference was observed in amplitude or latency between recording sites ipsilateral and contralateral to the stimulated arm.

In lower thoracic, lumbar or sacral reference recordings $\overline{N14}$ was of very small amplitude but negative in polarity at C2 (for example Fig. 2, subject C.S.). This suggests that, as for $\overline{N13}$, simultaneous positive and negative potentials may contribute to $\overline{N14}$ in mid-frontal reference recordings, with the source of the negative field situated in the upper cervical region or above, and the positive source situated further rostrally and best detected by an electrode over the parietal cortex contralateral to the stimulated arm.

Discussion

The potentials recorded over the spine, clavicles, mastoid processes and cortex with electrical stimulation at the elbow, wrist or fingers may be ascribed with some confidence to the activation of sensory afferent fibres within a narrow band of conduction velocities. The value of 65–75 m/sec, obtained for the forearm of one subject, corresponds to the conduction velocity of large myelinated fibres of Group II, which may make synaptic connections in the spinal grey matter (Wall 1967), or alternatively pass directly into the dorsal columns. The dorsal column/lemniscal sensory pathway projects to the contralateral somatosensory cortex via synapses in the cuneate nucleus of the brain stem and the nucleus ventralis lateralis posterior (VPL) in the thalamus. Sensory modalities mediated by Group II afferent fibres include joint-position sense, vibration sense and light touch. The dorsal column/lemniscal pathway is particularly associated with the first of these, and a correlation has been demonstrated between loss of joint-position sense and abnormality in the scalp-recorded somatosensory response (Halliday and Wakefield 1963). There is considerable evidence, therefore, that the responses recorded to median nerve stimulation as described in this account were somatosensory responses in the true sense.

On grounds of latency and distribution $\overline{N9}$ appears to be generated in the region of the brachial plexus. The dipolar nature of the generator, apparently of diagonal orientation with the positive pole proximal, and the increase in peak or onset latency towards medial and rostral recording sites are consistent with the volume-conducted electric field properties of an afferent nerve volley propagating in the brachial plexus. It has been argued (for example Woodbury 1960) that an advancing wavefront of depolarisation in a nerve trunk recorded at a distance through a homogeneous conducting medium will be analogous to a dipole oriented parallel to the direction of propagation with the positive pole in advance and the negative in arrears. The following edge of repolarisation will be analogous to a similarly oriented dipole rotated through 180°, but will contribute a smaller potential as the event is less abrupt. The distribution and properties of $\overline{N9}$ appear to be entirely consistent with generation by an advancing wavefront of depolarisation in the brachial plexus (Jones 1977), with the contribution due to repolarisation possibly obscured by larger potentials generated elsewhere. A similar peak recorded in response to median nerve stimulation at the wrist has been described by Cracco and Cracco (1976), who similarly proposed an origin in the peripheral nervous system.

Having established a probable generator site for $\overline{N9}$, and knowing that subsequent components of the response depend on activity in the same group of afferent fibres, the possible generator sites of $\overline{N11}$ to $\overline{N14}$ become more circumscribed. The earlier subcomponent(s) of $\overline{N11}$, occurring approximately 2 msec after $\overline{N9}$ and no more than 1 msec after the negative peak recorded from the medial end of the clavicle, most probably arise from a region immediately adjacent to or just within the spinal cord, with no synapse intervening. Furthermore the dipolar properties of $\overline{N11}$ suggest an origin due to the rostral propagation of a wavefront of depolarisation in a vertically oriented nerve trunk situated between the C7 and C2 vertebrae. Since there is little evidence for a continuous latency increase ascending the spine the most likely generator sites appear to be the lower cervical and T1 roots, which are vertically oriented for a short distance within the spinal column before entering the cord itself, but the last subcomponent might alternatively be due to the propagation of activity in the dorsal columns.

The $\overline{N13}$ generator appears also to be substantially dipolar in nature, with the negative field maximally recorded over C7 and the positive at the mid-frontal 'reference' site. By inference the positive pole was also recorded at other cerebral locations (for example over the hand area of the somatosensory cortex) which were at a negligible potential relative to Fz at $\overline{N13}$ latency. The relatively large amplitude of $\overline{N13}$ compared with $\overline{N11}$ suggests that $\overline{N13}$ may not be generated in primary afferent fibres but may arise as a result of postsynaptic depolarisation in the cervical region or above, with the positive field distributed rostral to an advancing wavefront of depolarisation in second or higher order somatosensory afferent fibres.

Taking into account the latency of $\overline{N13}$ after $\overline{N9}$ and $\overline{N11}$, and the probability of its generation via the same group of afferent fibres, it is likely that if $\overline{N13}$ originates in the spinal cord adjacent to the root of entry one or possibly two synapses might intervene. If the potential is generated more rostrally (for example in the brain stem) the model suggests there must be no more than one intervening synapse, and therefore it is proposed that $\overline{N13}$ may arise possibly in the spinal grey matter but no further rostral than the cuneate or external cuneate nuclei of the brain stem.

Similar arguments may be invoked to propose a generator site for $\overline{N14}$. An event such as the postsynaptic depolarisation of cell bodies in the cuneate nucleus or VPL might give rise to instantaneous source/sink activity in fibres projecting ultimately to (or oriented towards) the somatosensory cortex contralateral to the stimulated arm. In this manner a negative field arising at the generator site and detected by an electrode over the cervical region or mastoid process might be complemented by a positive field distributed further rostrally and best recorded over the parietal cortex on the contralateral side. Some properties of $\overline{N14}$, including its enhancement by recording from the ipsilateral mastoid process with reference to the scalp overlying the contralateral somatosensory cortex, have already been described by Matthews et al. (1974), and the widely distributed $\overline{P15}$ component recorded by Allison et al. (1974) using a linked ear reference would be expected, on the basis of the findings described above, to be composed mainly of the positive field of $\overline{N13}$ and $\overline{N14}$ appearing at the scalp electrodes.

An apparently analogous positive potential has been recorded from the scalp in the cat (Iragui-Madoz and Wiederholt 1976), with maximal amplitude contralateral to the stimulated forepaw. This latter potential was abolished along with the cortical somatosensory response by high brain stem transection. Present evidence, however, does not permit $\overline{N14}$ or $\overline{P15}$ to be attributed with certainty to events arising uniquely in the brain stem or uniquely in the thalamus.

There is little evidence to associate the major short latency components of the median nerve somatosensory evoked response (except possibly $\overline{N11}$) with activity in the dorsal columns, as originally proposed by Cracco (1973). Rather the present recordings resemble those of Matthews et al. (1974) who distinguished three components (waves 1, 2 and 3) which correspond in latency and distribution to $\overline{N11}$, $\overline{N13}$ and $\overline{N14}$ described above, and concluded that all three were probably due to activity at localised generator sites as opposed to the propagation of afferent activ-

ity in the long tracts of the spinal cord. Cracco and Cracco (1976) have now revised earlier views, and have proposed generator sites in the brain stem and thalamus to account for positive potentials (apparently corresponding to the positive constituents of N$\overline{11}$, N$\overline{13}$ and N$\overline{14}$) recorded by electrodes on the ear, nose and scalp. Particularly in the case of N$\overline{11}$, however, one should take account of latency information provided by the conduction velocity of nerve fibres mediating the response, and the dipolar nature of the generators. Such information leads one to propose generator sites which are generally located more distally than those proposed by Cracco and Cracco (1976), and support is lent to this view by the properties of intrathecal potentials recorded following stimulation of the median nerve at the elbow (Ertekin 1973, 1976). Two major potentials recorded in the cervical region and attributed to generators in the dorsal columns ('CD' potential) and dorsal roots ('DR' potential) appear on grounds of latency, polarity and relative amplitude to correspond with N$\overline{11}$ and N$\overline{13}$ respectively. The 'DR' potential was thought to arise in the dorsal roots as a result of activity in smaller myelinated afferent fibres, but the present findings suggest that had the stimulus alternatively been delivered at the wrist it might have transpired that the 'CD' and 'DR' potentials were mediated by the same group of afferent fibres, as was found to be the case for N$\overline{11}$ and N$\overline{13}$.

Applications of SEP recording techniques in the investigation of diseases of the central nervous system have been greatly extended by the recognition of the early 'spinal' potentials (Small 1976). It is to be hoped that further advances may ensue once there is general agreement as to which of the conceivable peripheral, spinal and cerebral structures might be responsible for generating these short latency components.

Summary

Short latency evoked potentials were recorded from sites overlying the cervical and thoracic vertebrae, the clavicles, mastoid processes and cerebral cortex, following percutaneous stimulation of median nerve fibres at the elbow, wrist and fingers in 23 normal subjects. At least four major early components each with simultaneous positive and negative constituents, plus the first component (N$\overline{20}$) of the cortical response, were all found to be mediated by sensory afferent fibres with conduction velocity 65—75 m/sec in the forearm of one subject.

Study of the distribution of these potentials, using reference electrodes located at Fz or over the lower part of the spine, has led to the proposal of generator sites in the brachial plexus (N$\overline{9}$), spinal roots or dorsal columns (N$\overline{11}$), spinal grey matter or brain stem (N$\overline{13}$), and brain stem or thalamus (N$\overline{14}$). Comparison with intrathecal recordings in man lends support to the view that N$\overline{11}$ and N$\overline{13}$ are generated in or adjacent to the spinal cord. It is hoped the findings may extend the clinical applications of a non-invasive technique for investigating the afferent sensory pathways in man.

Résumé

Potentiels à courte latence enregistrés à partir du cou et du crâne après stimulation du nerf médian chez l'homme

Des potentiels évoqués de courte latence ont été enregistrés à des emplacements susjacents aux vertèbres cervicales et thoraciques, aux clavicules, aux apophyses mastoïdes et au cortex cérébral, après stimulation de fibres du nerf médian au niveau du coude, du poignet et des doigts chez 23 sujets normaux. Au moins 4 des composantes principales à latence brève, chacune avec une composante soit positive soit négative, ainsi que la première composante (N$\overline{20}$) de la réponse corticale, étaient transmises par des afférences à vitesse de conduction de 65—75 m/sec dans l'avant-bras.

L'étude de la distribution de ces potentiels, à l'aide d'électrodes de référence situées en Fz ou à la partie inférieure de la colonne vertébrale, a permis d'identifier des sites générateurs dans le plexus brachial (N$\overline{9}$), dans les racines dorsales ou les colonnes dorsales (N$\overline{11}$), dans la substance grise spinale ou du tronc cérébral (N$\overline{13}$), et dans le tronc cérébral ou le thalamus (N$\overline{14}$). La comparaison avec des enregistrements intrathécaux chez l'homme suggère que les composantes N$\overline{11}$ et N$\overline{13}$ sont engendrées dans la moelle ou en son voisinage. Ces résultats permettront peut-être d'étendre les applications cliniques d'une technique non-pénétrante aux recherches sur les voies sensorielles centripètes chez l'homme.

I am indebted to Mr. J. Pitman and Mrs. E. Halliday for technical support, and to Drs. D.G. Small and A.M. Halliday for valuable ideas and discussion.

References

Allison, T., Goff, W.R., Williamson, P.D. and Van Gilder, J.C. On the neural origin of early components of the human somatosensory evoked potential. Proc. Int. Symp. Cerebral Evoked Potentials in Man, Belgium 1974 (in press).

Cracco, R.Q. Spinal evoked response: peripheral nerve stimulation in man. Electroenceph. clin. Neurophysiol. 1973, 35: 379—386.

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

Ertekin, C. Human evoked electrospinogram. In J.E. Desmedt (Ed.) New Developments in Electromyography and Clinical Neurophysiology. Karger, Basel, 1973, 2: 344—351.

Ertekin, C. Studies on human evoked electrospinogram. 1, The origin of the segmental evoked potentials. Acta Neurol. Scandinav. 1976, 53: 3—20.

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

Goff, W.R., Rosner, B.S. and Allison, T. Distribution of cerebral somatosensory evoked responses in normal man. Electroenceph. clin. Neurophysiol. 1962, 14: 697—713.

Goff, G.D., Matsumiya, Y., Allison, T. and Goff, W.R. The scalp topography of human somatosensory and auditory evoked potentials. Electroenceph. clin. Neurophysiol. 1977, 42: 57—76.

Halliday, A.M. and Wakefield, G.S. Cerebral evoked potentials in patients with dissociated sensory loss. J.N.N.P. 1963, 26: 211—219.

Iragui-Madoz, V.J. and Wiederholt, W.C. Brain stem somatosensory evoked responses in the cat. Neurology. 1976, 26: 365—366P.

Jones, S.J. The origin of the first component (N9) of the human spinal evoked response. Electroenceph. clin. Neurophysiol. proceedings (in press).

Matthews, W.B., Beauchamp, M. and Small, D.G. Cervical somatosensory evoked responses in man. Nature (Lond.), 1974, 252: 230—232.

Small, D.G. Peripherally evoked spinal cord potentials in neurological diagnosis. In J.P. Nicholson (Ed.), Scientific Aids in Hospital Diagnosis. Plenum, London, 1976: 155—163.

Wall, P.D. The laminar organisation of the dorsal horn and effects of descending influences. J. Physiol. (Lond.) 1967, 188: 403—423.

Woodbury, J.W. Potentials in a volume conductor. In T.C. Ruch and J.F. Fulton (Eds.), Medical Physiology and Biophysics. Eighteenth Edition. Saunders, London 1960, 83—91.