Chapter 2.4

Somatosensory evoked potentials

F. Mauguière (France)*, T. Allison (USA), C. Babiloni (Italy),
H. Buchner (Germany), A.A. Eisen (Canada), D.S. Goodin (USA),
S.J. Jones (UK), R. Kakigi (Japan), S. Matsuoka (Japan), M. Nuwer (USA),
P.M. Rossini (Italy) and H. Shibasaki (Japan)

General description of the method and physiological background

Somatosensory evoked potentials (SEPs) are the electrical potentials generated in sensory pathways at peripheral, spinal, subcortical and cortical levels of the nervous system. SEPs can be elicited from almost any nerve, although the median and posterior tibial nerves are usually chosen in clinical practice. Recently up-dated reviews on SEP recording, normal waveforms, normative data and clinical applications can be found in Jones (1993) and Mauguière (1999). Recommendations from an IFCN committee published in 1994 (Nuwer et al. 1994) have been updated in this chapter. The short latency responses occur within the first 60 ms after an electrical stimulus. Later middle latency and long latency SEPs show a wider range of normal variability making their clinical use more difficult. SEPs are usually evoked by bipolar transcutaneous electrical stimulation applied on the skin over the trajectory of peripheral nerves. Electrical stimuli used in clinical practice (see below) produce a twitch in the muscles innervated by the stimulated nerve when it contains a contingent of motor fibers. At this intensity the rapidly conducting large myelinated fibers, including fibers subserving touch and joint sensation but also muscle afferents, are activated because of their higher resistance. Muscle afferents have been shown to elicit cortical SEPs

* Correspondence to: Dr. François Mauguière, Department of Functional Neurology and Epileptology, Hôpital Neurologique, 59 boulevard Pinel, 69003 Lyon (France).
in particular after stimulation of lower limb sensori-motor nerves such as the posterior tibial nerve.

Mechanical stimuli can also produce SEPs and have a potential advantage over the conventional electrical stimuli which bypass the peripheral receptors. Nevertheless, these responses are not used routinely because of their low voltage and the technical difficulties in obtaining a short and stable stimulus.

As performed routinely, SEPs to electrical stimuli do not assess function in the small myelinated or unmyelinated afferents subserving temperature and pain sensation. Selective activation of pain and temperature fibers can be achieved by brief heat pulses delivered by a CO₂ laser beam applied to the skin surface.

In clinical practice SEP recording is mostly used to assess conduction in the somatosensory pathways and, particularly, in the spinal and intracranial segments. Therefore analysis of latencies and time intervals between components is emphasized in these recommendations.

Technical requirements

Stimulation

Monophasic square wave electrical pulses of 100 to 500 μs are generally delivered at a constant voltage or intensity two disk or needle electrodes connected to the negative (cathode) and positive (anode) pole of the stimulator. Electrical stimuli depolarize nerve fibers directly by generating a potential difference in the medium adjacent to the nerve trunk and thus across the nerve fiber membrane, causing a depolarization close to the site of the cathode. Care should be taken to be sure that the cathode is proximal to the anode in order to prevent anode block.

Stimulus intensity

In most clinical studies the intensity of electrical stimuli is set slightly above motor threshold, or at motor plus sensory threshold, for stimulation of mixed sensory-motor nerves, and at three or four times the sensory threshold for stimulation of sensory nerves. At these stimulus intensities all SEP components peaking before 50 ms post stimulus reach their maximal amplitude.

Stimulus rate

Stimulus rates of 1–10/s (1–10 Hz) are commonly used in clinical neurophysiology laboratories with no significant changes in the latencies of subcortical or primary sensory cortex (SI) responses. The amplitude of cortical potentials peaking after the initial SI response, however, can be reduced at rates over 3 Hz and middle and long latency SEPs reach the saturation level at stimulus rates below 1 Hz. A stimulus rate below 5 Hz is recommended for routine recording of early cortical SEPs.

Ground electrode

To minimize the electrical artifact produced by the stimulation, the ground electrode should be placed on the stimulated limb between the stimulation site and the recording electrodes. Flexible metal strips covered with saline soaked cloth wrapped around the limb close to the stimulus site are recommended. Electrically isolated stimulators allow the use of a ground electrode on the head which is adequate for eliminating artifact related to the electrical main and radiofrequency interference.

Analysis time and sampling rate

Most of the clinically useful SEP components peak before 50 and 100 ms respectively for upper and lower limb stimulation. Consequently there is no necessity to extend the analysis time for SEP recordings beyond 100 ms in most clinical settings. Most commercially available recording devices allow 500 or 1000 sampling points over analysis times of 10–100 ms, resulting in a bin width of 100 or 50 μs for an analysis time of 50 ms. These bin widths are appropriate for the recording of subcortical and short-latency cortical SEPs without aliasing.

Filters

A high-pass filter set at less than 3 Hz and a low pass filter set over 2000 Hz are optimal to record all SEP components without distortion. The roll-off of an analog filter should not exceed 12 and 24 dB/
octave for low and high frequency filters, respectively. Digital filtering of responses acquired with a broad band pass filter offers the possibility of selecting off-line frequencies of clinical interest. The 50 or 60 Hz notch filter should be turned off.

Gain and number of sweeps
Optimal preamplifier gains depend on the input range of the AD converters; gains of 20 000 to 100 000 are typical. The number of sweeps that need to be averaged depends upon the initial signal-to-noise ratio of the different SEP components. In practice 500 sweeps are generally enough to obtain the peripheral responses at Erb’s point, as well as to define the early cortical and subcortical components of the SEP used in clinical practice. For the recording of spinal and sub-cortical EPs often up to 1000–2000 sweeps must be averaged.

Quality control
EP tracings must be replicated (at least once) and superimposed in order to demonstrate the reproducibility of the components measured. Muscle artifact should be eliminated by making the patient as comfortable as possible. Movement occurring during nerve stimulation can cause attenuation of cortical SEPs. The supine position may help the patient to relax and, in some cases, a light sedative such as an oral benzodiazepines may be helpful. Automatic artifact rejection should be employed to eliminate occasional high amplitude artifactual transients. Repeated or, preferably, continuous examination of the raw data during collection can identify artifacts, particularly continuous low amplitude artifacts that may be present. Latency values measured on the separate repetitions should be within 0.25 ms of each other (0.5 ms for the tibial nerve P39 potential). Amplitude values should be within 20% of each other. Latencies and amplitudes should be measured in the grand average of 2 or 3 runs, provided the repetitions are judged to be reasonably consistent.

Factors affecting the quality of the investigation

Maturation
The development of somatosensory pathways from birth to adult life is dominated by myelogenesis which causes a progressive increase in conduction velocities and synchronization of potentials with age, and by body growth which increases latencies and desynchronization. During the first 4–5 years of life SEP maturation is marked by a progressive synchronization and a latency reduction of all potentials. Conduction velocities reach adult values before the age of 3 years in the peripheral nervous system; spinal cord conduction in lower limb fibers reaches adult values at the age of 5–6 years. Later on changes in conduction velocity related to fiber maturation interact with those of body growth and peak latencies progressively increase, so that adult values are reached at the age of 15–17. Several studies of SEP normative data in children have been published recently using cephalic (Taylor 1993) or non-cephalic (Boor et al. 1998a, b) reference montages. Normal latency values from these two studies are given in Tables 1 and 2.

Ageing
The effects of age on SEP latencies mainly reflect conduction slowing in the peripheral nerves evidenced by the increase of the N9 component after median nerve stimulation. Most of this change occurs after the age of 55 years. The central conduction time (CCT) was reported as unaffected or slightly increased during normal ageing. Regression equations for calculation of the upper limits of normal SEPs from age and body height are provided in Allison et al. (1983). The amplitude of median nerve SEPs recorded in the frontal region tends to decrease with age while that of the parietal N20 tends to increase (Desmedt and Cheron 1980).

Body height and gender in adults
The absolute latencies of SEPs varies according to the distance between the stimulated site and the SEP sources. This effect is naturally more pronounced for lower than for upper limb SEPs and is less for the interpeak intervals than for absolute latencies. However the upper normal values of the N22-P39 interval of tibial nerve vary between 18 and 21.5 ms for body heights of 1.50 and 1.90 m respectively. Shorter CCTs in females relative to
TABLE 1  
MEDIAN NERVE SEPs, LATENCIES IN CHILDREN (Mean (SD))

<table>
<thead>
<tr>
<th>Age</th>
<th>Height (cm)</th>
<th>N9</th>
<th>N13</th>
<th>P14</th>
<th>N20</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Peak latencies (ms)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>2–6 weeks</td>
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<td></td>
<td></td>
</tr>
<tr>
<td>7–13 weeks</td>
<td></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4–8 months</td>
<td>113 (8.1)</td>
<td>7.1 (0.7)</td>
<td>9.4 (0.8)</td>
<td>11.0 (0.9)</td>
<td>15.7 (0.8)</td>
</tr>
<tr>
<td>9–15 months</td>
<td>135 (5.9)</td>
<td>7.7 (0.4)</td>
<td>10.2 (0.5)</td>
<td>11.6 (0.5)</td>
<td>16.0 (0.6)</td>
</tr>
<tr>
<td>16–22 months</td>
<td>155 (9.7)</td>
<td>8.8 (0.8)</td>
<td>11.4 (0.8)</td>
<td>13.0 (0.8)</td>
<td>17.4 (1.0)</td>
</tr>
<tr>
<td>2–4 years</td>
<td>166 (6.6)</td>
<td>9.1 (0.4)</td>
<td>12.1 (0.6)</td>
<td>13.6 (0.6)</td>
<td>17.8 (0.6)</td>
</tr>
<tr>
<td>4–6 years</td>
<td></td>
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<td></td>
<td></td>
</tr>
<tr>
<td>7–9 years</td>
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</tr>
<tr>
<td>10–12 years</td>
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<td></td>
</tr>
<tr>
<td>13–15 years</td>
<td></td>
<td></td>
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</tr>
</tbody>
</table>

**Intervals (ms)**

| Age           |     |     |     |     |     |

| 4–8 months    | 2.3 (0.3) | 3.9 (0.3) | 6.3 (0.6) |
| 9–15 months   | 2.5 (0.2) | 3.9 (0.3) | 5.8 (0.3) |
| 16–22 months  | 2.7 (0.3) | 4.2 (0.3) | 6.0 (0.5) |
| 2–4 years     | 3.0 (0.2) | 4.5 (0.2) | 5.7 (0.3) |

| N9-N13        |     |     |
| N9-P14        |     |     |
| N13-N20       |     |     |

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1 From Taylor (1993): Fz reference recordings.  

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males has been reported by some authors but not by others.

**Skin and core temperature**

Peripheral nerve conduction velocities are known to be slowed by a reduction in limb temperature and, therefore, the patient’s limbs should be kept warm during testing. Markedly decreased body temperature, as can be observed during drug-induced hypothermia, increases the absolute and interpeak latencies (Guérin et al. 1990). In addition, SEP changes related to hypothermia deserve special attention in comatose patients because they can combine with those induced by central nervous system (CNS) depressant drugs.

**Attention, sleep and vigilance**

Attention has relatively little impact on the early cortical SEPs recorded in routine conditions, i.e. using runs of 500 stimuli or more with no instruction given to the subject. Nevertheless, some changes in the amplitude, waveform and latency of the parietal N20 have been reported in normal subjects during natural sleep and a slight latency asymmetry may appear when one limb is tested with the patient awake and the other with the patient asleep. Therefore, it is recommended to monitor the state of consciousness during the recording when the patient’s vigilance level is fluctuating.

**Drugs**

Most medications have little effect on early SEPs, and sedation with a benzodiazepine or similar medication can be used to aid relaxation of the subject. This procedure does not produce false-positive SEP changes. Only serious overdoses of CNS depressant drugs cause abnormally prolonged N13-N20 central conduction time and this effect is only clinically relevant to SEP interpretation in the context of coma or brain death (see
TABLE 2
POSTERIOR TIBIAL NERVE SEPS LATENCIES IN CHILDREN (Mean (SD)) (from Boor et al. 1998b)

<table>
<thead>
<tr>
<th>Age</th>
<th>N8</th>
<th>N22</th>
<th>P30</th>
<th>P39</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Peak latencies (ms)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>4–6 years</td>
<td>5.4 (0.5)</td>
<td>13.9 (0.8)</td>
<td>20.6 (1.4)</td>
<td>33.3 (3.3)</td>
</tr>
<tr>
<td>7–9 years</td>
<td>6.2 (0.5)</td>
<td>16.1 (0.8)</td>
<td>22.7 (1.6)</td>
<td>33.0 (2.5)</td>
</tr>
<tr>
<td>10–12 years</td>
<td>7.5 (0.9)</td>
<td>19.1 (1.5)</td>
<td>26.7 (2.2)</td>
<td>35.9 (3.1)</td>
</tr>
<tr>
<td>13–15 years</td>
<td>8.1 (0.8)</td>
<td>20.7 (1.0)</td>
<td>29.1 (1.6)</td>
<td>37.7 (2.3)</td>
</tr>
<tr>
<td><strong>Intervals (ms)</strong></td>
<td></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td></td>
<td>N8-N22</td>
<td>N22-P30</td>
<td>P30-P39</td>
<td>N22-P39</td>
</tr>
<tr>
<td>4–6 years</td>
<td>8.5 (0.6)</td>
<td>6.7 (1.2)</td>
<td>12.6 (2.7)</td>
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<td>9.8 (0.5)</td>
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<tr>
<td>13–15 years</td>
<td>12.6 (0.6)</td>
<td>8.3 (0.8)</td>
<td>8.6 (0.9)</td>
<td>16.9 (1.7)</td>
</tr>
</tbody>
</table>

Mauguière 1999, for a recent review). Also, a latency increase of N13 and N20 components of the median nerve SEPs has been reported after a single intravenous injection of a loading dose of phenytoin.

**Clinical protocols for the investigation**

**Median nerve SEPs**

**Stimulation.** Stimulation is applied to the median nerve at the wrist. Disk electrodes should be placed on the skin overlying the nerve at the wrist. The cathode (negative stimulating electrode, designated with a black connector) should be placed 2 cm proximal to the wrist crease. The anode (positive stimulating electrode, designated by a red connector) should be placed on the wrist crease. The technologist should alter the stimulus intensity as needed to maintain a constant thumb twitch throughout the test. Stimulation should not be applied over or adjacent to catheters or wires whose central end lies in the heart or great vessels.

**Recording.** Median nerve SEPs are recorded on an analysis timebase of 30–50 ms. Recording electrodes with an electrical impedance of less than 5000 Ω should be placed over Erb’s point bilaterally, skin overlying the cervical spine and in the parietal and frontal scalp regions. In each case, negative electrical potentials at the first electrode designated below should be displayed as upgoing deflections or peaks.

Erb’s point is located within the angle formed by the posterior border of the clavicular head of the sternocleidomastoid muscle and the clavicle, 2–3 cm above the clavicle. Erb’s point electrodes are designated as EPc (contralateral to stimulation) and EPI (ipsilateral to stimulation).

The posterior spinal cervical electrode should be located over the fifth (C5) cervical spinous process, i.e. two spines above the C7 process which is identified as the most prominent spinous process at the base of the neck when the neck is flexed. Some users place the posterior cervical electrode over the C6 process; this position is also adequate. The anterior cervical electrode (AC) is attached on the skin surface of the supra-glottal region on the midline.

The locations of scalp electrodes are specified using the 10–20 international system of EEG electrode placement. Parietal scalp electrodes are placed 5 cm posterior to Cz and 7 cm lateral to midline. These locations are designated as Pi and Pc (P Parietal, i ipsilateral to stimulation, c contralateral to stimulation). In some studies these electrode-sites are designated as Ci and Ce (C Central) in spite of their clear parietal position.

The frontal scalp electrode is usually placed at the site Fz of the 10–20 system.
For evaluation of pathways up to the level of the cortex, the suggested recording montage is:

- Channel 1: Pc-Fz, the scalp channel
- Channel 2: Pc-EpC, the scalp-non-cephalic channel
- Channel 3: Cv5-AC, the spinal or cervical channel
- Channel 4: Epi-EpC, the Erb’s point channel.

It is noteworthy that bipolar recordings can be computed off-line using simple algorithms and that variations related to reference electrode can be cancelled from scalp potentials using surface Laplacian derivation, which can be computed at one scalp site on a minimum of three surrounding electrodes.

**Variants.** Some users prefer Pc-Pi in place of Pc-Fz for the scalp channel.

To study separately the cortical SEPs culminating in the contralateral parietal and midline frontal regions the two recommended scalp channels are: channel 1 Fz-EpC and channel 2 Pc-EpC. When the non-cephalic EPC reference scalp recordings show a high noise level the reference electrode can be placed at the earlobe ipsilateral to the stimulus (Ei) for the recording of cortical and P14 potentials.

Some users prefer to place the non-cephalic reference electrode at the shoulder (acromial process) contralateral to stimulus rather than at EpC in the scalp-non-cephalic channel.

The cervical active electrode may be placed slightly higher or lower than Cv5 on the neck. The EpC electrode can be used to record the cervical response, however identification and amplitude measurement of the N13 potential is facilitated by the use of the AC supraglottal reference electrode. The use of an Fz reference for the cervical channel improves the signal to noise ratio by causing early positive scalp potentials recorded at Fz to be injected as negatives in the resulting waveform. The Cv5-Fz derivation is not recommended whenever it is necessary to discriminate cervical cord potentials from similar latency potentials generated at, or above, the foramen magnum (Mauguère and Restuccia 1991).

Additional recording electrodes can help to separate the cortical potentials occurring between 18 and 50 ms, using either EpC, shoulder or earlobe (see above) as reference electrode sites. For example: (i) an additional central electrode contralateral to stimulus situated 2 cm in front of Cz and 5 cm laterally is necessary to pick up the P22 potential, (ii) a midline electrode at halfway between Fz and Cz is optimal for the recording of the frontal N30 and (iii) the Pi electrode referred to a non-cephalic site is optimal to record the subcortical N18 potential.

When only two channels are available the minimal montage for assessing central conduction time is: channel 1 Pc-Fz and channel 2 Cv5-AC. However it is recommended to record the Erb’s point and the scalp-non-cephalic channels on separate runs.

**Recognition of components.** A series of positive and negative potentials are recorded from the shoulder, neck and scalp. These can be recognized by their typical distributions, as determined by comparing the several recording channels (see Fig. 1).

The Erb’s point potential is designated N9. It is the principal negative peak seen in the Epi-EPc channel. When the Erb’s point channel shows a double negativity, the first peak is chosen. (This doubling is seen most often in children.) N9 arises from the brachial plexus trunks.

The N11 is a small negative potential seen in the cervical channel, preceding the N13 peak. N11 is often difficult to differentiate from the N13 component; this seriously hampers its use in clinical practice. In children, N11 may be quite prominent, especially at ages 1–4 years. N11 probably reflects the ascending volley in dorsal column fibers at the cervical level.

The cervical N13 potential is recorded at the posterior neck, with a maximum voltage at the level of Cv5-Cv7 spinous processes, and decreases in amplitude at more rostral or caudal electrode positions. At the anterior aspect of the neck the cervical response is recorded as a P13 positivity, suggesting a transverse dipolar generator oriented perpendicular to the spinal axis. Consequently the N13 potential is recorded with maximal amplitude using the Cv-AC channel. The most likely generator of the N13/P13 cervical potentials is the
compound segmental post-synaptic potential triggered in the dorsal horn gray matter by the afferent volley in fast conducting myelinated fibers. N13 is preceded by a P9 positivity or by a N9/P9 deflection (see Fig. 1) reflecting the dorsal root volley.

In scalp-non-cephalic channel recordings 3 or 4 positivities preceding the N20 potential are consistently observed. These potentials have a wide distribution and a mediofrontal predominance on the scalp. In normal adults these potentials peak with mean latencies of 9, 11, 13 and 14 ms respectively and are labelled P9, P11, P13 and P14. The P9 potential is picked up at the neck as well as on the scalp; it reflects the afferent volley in the trunks of the brachial plexus in axilla and supra-clavicular fossa. The P11 potential is supposed to reflect the ascending volley in the fibers of dorsal columns at the cervical level; it begins in synchrony with the cervical N11 potential at the Cv6 level, which is close to the dorsal root entry zone in the cervical cord. The P11 potential is not recorded in about 20% of normal controls. The P13-P14 potentials are consistently recorded in normals. P14, but not P13, peaks later than the cervical segmental N13 potential. In some subjects P14 is hardly visible as a notch on the ascending phase of P13, in others P13 and P14 cannot be differentiated. In the same individual the morphology of the P13-P14 complex can display some degree of side-to-side difference. For sake of clarity the P13-P14 complex is often labeled as 'P14'. The P14 potential is picked up at the earlobe with a lesser amplitude than in the frontal region of the scalp, thus it can be recorded with a scalp-earlobe montage, it is usually much smaller or absent in the Pc-Fz scalp channel. The P14 potential arises from the lower brain-stem close to the cervico-medullary junction.

The N18 potential (Desmedt and Cheron 1981) is a long-lasting scalp negative shift which immediately follows P14. In normals N18 can only be identified using non-cephalic reference montage in parietal region ipsilateral to stimulation, where there is no or minimal interference with cortical potentials. Lesion studies suggest that N18 has a brain-stem origin situated below the thalamus and above the foramen magnum.

N20 is localized to the parietal scalp region, showing a polarity reversal across the central fissure. N20 represents the largest early negative deflection at Pc, although it may have several small peaks riding on top of it. The N20 peak is usually identified as a portion of the negative potential just preceding the sharp drop-off toward the succeeding cortical positive peak P25. N20 is generated from the primary somatosensory cortex in the posterior wall of the central fissure (SI area).

The P25 (or P27) component is recognized as the main prominent positive peak succeeding the N18-N20 complex at Pc. It is supposed to be generated in area SI. In non-cephalic (or earlobe) reference scalp channels a P22 positive potential and a N30 negative potential are consistently recorded in contralateral central and medio-frontal regions, respectively. The exact origins of these potentials are still a matter of debate. N30 changes have been reported during execution and programming of voluntary movements in normals.

Fig. 1. Median nerve SEPs. Abbreviations for derivations and peak labels are described in the text.

**Measurements of peak latencies and amplitudes.** Arm length should be measured
from the stimulation cathode to the Erb’s point electrode. Typical latency and amplitude values in young adults are given in Table 3.

The latency of the N9 Erb’s point negativity is measured to its peak on the Epi-EPc channel, not to its onset as is usual for some nerve conduction velocity techniques. The amplitude of N9 is measured on the Epi-EPc channel from the peak of N9 to that of the initial or succeeding positive deflections.

The latency to the N13 peak is measured on the Cv-AC channel; its peak amplitude is evaluated from the peak of P9, or by calculating the ratio between its amplitude and that of the P9 deflection on the Cv-AC montage (see above and Fig. 1).

The latency of the P14 peak is measured on the scalp-non-cephalic channel (Pc-EPc), to the maximal positive point. Its amplitude can be measured from baseline or by calculating the P14/ P9 amplitude ratio.

The latency of N20 is measured to the point of maximal negativity just preceding the sharp drop-off into the P25 trough. The N20 onset latency can be measured on Pc-Fz traces, or by superimposing traces recorded at Pc and Fz sites with a non-cephalic reference electrode, the N20 onset then corresponds to the point where the N20 ascending slope diverges from the Fz trace (Ozaki et al. 1994). The amplitude of the cortical potentials can be measured from the N20 peak to the trough of the P25 on the Pc-Fz channel. For evaluating independently the amplitude of each cortical potential at a given scalp location, measurement is done from baseline to peak on traces recorded using the Pi scalp electrode as reference. Most users consider that side-to-side amplitude differences of the cortical components over 50% are abnormal. The N20-P25 deflection can be considered giant when its amplitude is more than 15 μV. The amplitude ratio between the parietal N20 and the frontal N30 can be helpful in interpreting a selective reduction of the latter (Rossini et al. 1995).

Measurement of conduction velocities and conduction times. Peripheral conduction velocity (in m/s) is calculated by dividing the arm length (in mm) by the N9 latency (in ms).

Plexus-cord conduction time is calculated by subtracting the N9 and N13 latencies. This corresponds to conduction through the proximal plexus, roots up to the dorsal horn of the cervical cord. The N9-P14 (or P9-P14 in the non-cephalic scalp channel) reflects the conduction time (CT) from proximal plexus through roots and spinal cord, up to the cervico-medullary junction.

Cord-cortex conduction time, sometimes referred to as the central conduction time (CCT), is usually calculated by the peak-to-peak N13-N20 interval. This CCT can also be evaluated by measuring the interval between N13 and N20 onset latencies; a correlation between CCT and body height has been reported with this procedure (Ozaki et al. 1994). The brain-stem to cortex CT (intracranial conduction time) is measured by the P14-N20 interval on the non-cephalic scalp channel.

The plexus-cortex interpeak latency is the difference between the N20 and N9 latencies. Typical criteria for normal limits are shown in Table 3.

**Posterior tibial nerve SEPs**

**Stimulation.** Stimulation should be delivered to the posterior tibial nerve at the ankle. Surface electrodes should be placed on the skin overlying the nerve as it passes posterior to the medial malleolus. The cathode should be placed midway between the medial border of the Achilles tendon and the posterior border of the malleolus. The anode electrode should be located off the nerve 3 cm distal to the cathode. The electrical pulse should be delivered with a sufficient intensity to cause a 1–2 cm plantar flexion of the toes. The technologist must observe this movement throughout the time of testing, adjusting the stimulus intensity as needed to maintain a constant degree of toe movement.

**Recording.** Posterior tibial nerve SEPs are usually recorded on an analysis timebase of 60–80 ms, which should be extended to 100–200 ms if a significant delay or absence of P39 is anticipated, or if no scalp potentials are detected during the first 60 ms.

Recording electrodes with an electrical impe-
TABLE 3
MEDIAN NERVE SEPs, TYPICAL VALUES FOR YOUNG ADULT TESTING (BODY HEIGHT 1.70 ± 0.1 m)

<table>
<thead>
<tr>
<th>Peaks</th>
<th>Mean</th>
<th>Upper limit of normal mean +3 SD</th>
<th>Upper limit of normal side-to-side difference</th>
</tr>
</thead>
<tbody>
<tr>
<td><strong>Latencies (ms)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N9 (EPi-EPc)</td>
<td>9.8</td>
<td>11.5</td>
<td>–</td>
</tr>
<tr>
<td>P9 (Pc-EPc)</td>
<td>10.1</td>
<td>12.0</td>
<td>–</td>
</tr>
<tr>
<td>N13 (Cv5-AC)</td>
<td>13.3</td>
<td>14.5</td>
<td>–</td>
</tr>
<tr>
<td>P14 (Pc-EPc)</td>
<td>14.3</td>
<td>16.7</td>
<td>0.8</td>
</tr>
<tr>
<td>N20 (Pc-EPc)</td>
<td>19.8</td>
<td>23.0</td>
<td>1.4</td>
</tr>
<tr>
<td><strong>Intervals</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N9-N13</td>
<td>3.5</td>
<td>4.5</td>
<td>1.3</td>
</tr>
<tr>
<td>P9-P14 (Pc-EPc)</td>
<td>4.5</td>
<td>6.0</td>
<td>1.1</td>
</tr>
<tr>
<td>P14-N20 (Pc-EPc)</td>
<td>4.6</td>
<td>6.6</td>
<td>1.2</td>
</tr>
<tr>
<td>N9-N20</td>
<td>9.3</td>
<td>10.8</td>
<td>0.9</td>
</tr>
<tr>
<td>N13-N20</td>
<td>5.7</td>
<td>7.2</td>
<td>1.0</td>
</tr>
<tr>
<td><strong>Amplitudes (μV)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N9</td>
<td>4.8</td>
<td>1.0</td>
<td>50%</td>
</tr>
<tr>
<td>N13</td>
<td>2.3</td>
<td>0.5</td>
<td>–</td>
</tr>
<tr>
<td>N20 (Pc-Pi, baseline to peak)</td>
<td>2.2</td>
<td>0.6</td>
<td>47%</td>
</tr>
<tr>
<td>N20-P25 (peak to peak)</td>
<td>3.2</td>
<td>0.8</td>
<td>–</td>
</tr>
<tr>
<td><strong>Amplitude ratios (%)</strong></td>
<td></td>
<td></td>
<td></td>
</tr>
<tr>
<td>N13/P9 (Cv5-AC)</td>
<td>1.1</td>
<td></td>
<td></td>
</tr>
<tr>
<td>P14/P9 (Pc-EPc)</td>
<td>1.3</td>
<td></td>
<td></td>
</tr>
</tbody>
</table>

dance of less than 5000 Ω should be placed on the ipsilateral knee, the low back, cervical region and on the scalp.

The recording electrode at the knee should be placed over the tibial nerve in the popliteal fossa, 4–6 cm above the popliteal crease, midway between the combined tendons of the semimembranous-semitendinous muscles medially and the tendon of the biceps femoris muscle laterally. It can be helpful to identify the best position by stimulating the nerve to determine the site of lowest threshold. This electrode should be designated PF. A reference electrode should also be placed on the medial surface of the knee, over the medial femoral condyle. This electrode should be designated K.

Recording electrodes over the low back should be placed on the skin overlying the spinous processes of vertebrae L3, L1, T12 or T10. Level L4 can be estimated from a line connecting the posterior inferior iliac crests and levels T12 and L1 are optimal for the recording of the segmental dorsal horn response. For the second montage suggested below, the supra-umbilical region (Um) or iliac crest (Ic) contralateral to stimulation can be used as a reference.

The principal scalp recording electrode is placed at site Cz', located 2 cm behind Cz with a reference electrode located on the forehead at Fz, Fpz or Fpz' (2 cm above Fpz) sites. Acceptable alternative sites are CPz located half way between Cz and Pz for the recording electrode and earlobe ipsilateral to stimulation (Ei) for the reference. There are two suggested montages. The following peripheral montage is suggested for use when differentiating peripheral from central problems. The suggested peripheral montage is:
• Channel 1: Cz'-Fz, the scalp channel
• Channel 2: T12-T10, the thoracic channel
• Channel 3: L1-L3, the lumbar channel
• Channel 4: PF-K, the knee channel.

For those patients in whom the test is meant to investigate primarily the central (spinal and intracranial) conduction times (CT), the following central montage is suggested:
• Channel 1: Cz'-Ei (or Cpz-Ei) the scalp channel
• Channel 2: Fz-Cv5 (or Fpz-Cv5) the scalp-non-cephalic channel
• Channel 3: L1-Um (or L1-Ic), the lumbar channel
• Channel 4: PF-K, the knee channel.

It is recommended to record the knee channel in the two montages to assess the latency and amplitude of the peripheral volley. If additional channels are available it can be useful to record scalp potentials from the same locations used for median nerve SEPs, Pc and Pi, referenced to Fz or to one another.

In each case, negative electrical potentials at the first contact designated above should be displayed as upgoing deflections or peaks. It is often advantageous to combine these two montages together to form a 6–8 channel combined montage if sufficient recording channels are available. When only two channels are available the minimal montage for assessing the central conduction time is: channel 1 Cz'-Fz (or Cz'-Ei) and channel 2 L1-Um (or L1-Ic).

Recognition of peaks. Normal responses recorded with the central montage are shown in Fig. 2.

The sequence of negative and positive potentials recorded from the knee, low back and scalp can be recognized by their distribution, time course and their relation to other peaks (Fig. 2). The knee potential, N8, is the principal negative peak seen in the PF-K channel. It arises from the tibial nerve or its parent sciatic nerve.

N22 is the principal negative peak occurring at sites L1 and T12. It may be preceded by a smaller negativity occurring around the L3 sites. As all segmental spinal SEPs the N22 reverses its polarity at the anterior aspect of the lumbo-sacral cord. It reflects the post-synaptic response in the dorsal gray matter of the lumbo-sacral cord. When the reference electrode is not situated on the axis of propagation of the peripheral ascending volley (Ic or Um reference sites) the N22 potential is preceded by a small P17 positivity peaking around 17 ms originating in lumbo-sacral plexus trunks. Care must be taken to distinguish the N22 from the slightly earlier N19 negativity reflecting the volley in spinal ascending somatosensory pathways. The N22 is usually more dominant, broader and later.

A P30 positive potential is recorded on the scalp-non-cephalic channel (Fpz-Cv5) of the central montage. The P30 potential is widely distributed on the scalp with a predominance in the frontal region (Desmedt and Bourguet 1985). The utility of the P30 potential has been recently validated for clinical applications of tibial SEPs (Tinazzi and Mauquiere 1995). The P30 potential is likely to have the same origin as the median nerve P14 potential at the cervico-medullary junction. It must be recorded for measuring the spinal conduction time in patients with spinal cord lesions.

The P39, also labeled P37 or P40 by some users, is the major positive peak located in the post-central scalp region. The P39 may be preceded by a slight negative N33 potential equivalent to the N18 median nerve potential (see above). It is consistently followed by N50 and P60 potentials, these three waves forming the ‘W’ profile (P39-N50-P60)

![Posterior Tibial Nerve SEPs](image)

**Fig. 2.** Tibial nerve SEPs recorded with the central montage. Abbreviations for derivations and peak labels are described in the text.
of the cortical response on a 100 ms analysis time. The P39 is generated from the post-central somatosensory cortex. When the P39 is low amplitude or indistinct in the Cz'-Fz or Cz'-Ei scalp channels, the recording electrode should be moved 2 cm apart from Cz' ipsilaterally to stimulation. P39 may often be found better there. This paradoxical scalp localization ipsilateral to stimulus is due to the orientation of P39 source in the interhemispheric fissure, as suggested by the dipolar scalp distribution of the P39 field, with a maximum of positivity ipsilateral to stimulus in the parietal region and a maximum of negativity (N39) in the fronto-central region contralateral to stimulus. The P39 can sometimes be dwarfed by the P60 and care should be taken not to confuse a P60 of normal latency with a delayed P39.

Using the same montage as for recording the tibial nerve SEPs, a spinal negative N15 potential equivalent to the tibial nerve N22 potential can be recorded on the skin surface after electrical stimulation of the dorsal nerve of the penis or clitoris. Stimulation of the pudendal nerve also evokes a consistent, W-shaped response on the scalp. The earliest positivity of this cortical response peaks at about 40 ms after stimulation.

**Measurements.** The N8 latency is measured to the latency of the maximal negative deflection in the PF-K channel. The amplitude of this peak is measured from the negative peak to the succeeding positive trough. A conduction velocity (in m/s) can be derived by dividing the N8 peak latency (in ms) by the distance (in mm) between the PF and cathode stimulating electrode. Subject height should also be measured.

Latency to the N22 can be measured on the T10-T12, L1-L3, L1-Um or L1-Ic channels by noting the time of the greatest simultaneous negative activity at the L1 and T12 electrode sites. Amplitude of N22 is measured from the N22 to the succeeding positive peak.

The latency of P39 is measurable in any of the scalp channels of the two montages described above. The amplitude of the cortical peak is measured either from P39 to N50, or from baseline.

The central interpeak latency is measured as the difference between the N22 and P39 latencies. This corresponds to the central conduction time (CCT) in the entire extent of the central sensory pathway from lumbosacral cord level to primary somatosensory cortex. The spinal CCT is measured by the N22-P30 interpeak interval and the P30-P39
interval measures the intracranial conduction time from lower brainstem to cortex. The peak latencies of N22, P30 and P39 are correlated with body height in normal subjects. Normal values of latencies and amplitudes in young adults are given in Table 4.

References


