More than 40 years have elapsed since the first somatosensory evoked potential (SEP) was recorded from humans, and 15 years have passed since the clinical role of SEPs was reviewed as an American Association of Electrodiagnostic Medicine (AAEM) monograph. At that time, interest in SEPs was reaching its zenith, and a vast literature had already accumulated. The subsequent development of sophisticated imaging techniques has impacted on the role of SEPs in the clinical setting, making it timely to review this aspect as the end of the century is approached. This monograph considers current concepts of the physiologic basis of the SEP, discusses the different techniques available to elicit and record it, and critically analyzes its clinical utility.

The peripheral electrical stimulus routinely used to elicit an SEP activates predominantly—if not entirely—the large-diameter, fast-conducting group Ia muscle and group II cutaneous afferent fibers. Selective intracerebral stimulation has provided evidence for a direct muscle afferent fiber (Ia) projection to the human somatosensory cortex. However, when a mixed nerve is stimulated, both group Ia muscle afferents and cutaneous group II afferents contribute to the resulting SEP. Its amplitude is almost maximal when the peripheral nerve action potential is only 50% of its maximum. This translates into a requisite stimulus intensity of about twice sensory threshold. It is also possible to elicit SEPs using a variety of mechanical stimuli. This allows selective activation of specific sensory modalities, but the SEPs elicited are often of small amplitude and may require many hundreds of responses to be averaged. This limits the clinical utility of mechanically elicited SEPs.

The SEP is greatly attenuated or abolished when the dorsal columns are selectively ablated in animals, indicating that within the spinal cord the SEP is mediated predominantly via these tracts. Conversely, cord lesions that do not interrupt the dorsal columns are associated with a relatively normal SEP. Loss of posterior column function in humans is almost invariably accompanied by a grossly abnormal SEP. Some SEP components may, however, reflect extralemniscal activity; they have been evoked in cats after selective dorsal column transection by stimuli that are sufficient to excite small-diameter peripheral fibers, and tourniquet-induced ischemia in humans abolishes short-latency before long-latency SEP components, suggesting that they are mediated by different centrally conducting tracts.

Although in general the SEP is best recorded over the somatosensory cortex, topographic mapping indicates that several of its components are widely distributed over the scalp, and some are maximally recorded outside the somatosensory cortex. Because the SEP monitors more than just the somatosensory pathways, abnormalities recorded in certain primary diseases of the motor system (such as amyotrophic lateral sclerosis) should not cause concern.
STIMULATION TECHNIQUES

**Mixed Nerve Stimulation.** Because electrical stimulation of a mixed nerve initiates a relatively synchronous volley that elicits a sizable SEP, it has become the standard for clinical use. The stimulus intensity required to elicit an SEP of maximum amplitude need not be supramaximal. The ideal stimulus induces a mixed nerve action potential that is just greater than 50% of its maximum amplitude and clinically produces a slight muscle twitch. Too high a stimulus is counterproductive, producing occlusion of Ia impulse traffic by other converging afferent impulses. The occurrence of occlusion may depend on the limb that is stimulated. When occlusion occurs, a percentage of the initial volley is ineffective, and the SEP is accordingly lower in amplitude. A stimulus of short duration (200–300 µs) is popular, but longer duration (1000 µs) pulses of appropriately lower intensity may be appropriate because they preferentially activate the type Ia and II afferents. A repetition rate of 5.1 Hz is convenient. Faster rates may be tolerable to the patient and do not alter the SEP until they exceed 15 Hz. Troublesome electrocardiographic artifact, particularly relevant with noncephalic referential recordings of SEPs elicited from the legs, can be obviated by triggering the stimulus off the electrocardiogram. In general, SEPs elicited by lower limb stimulation have the added advantage of testing the functional integrity of much of the length of the spinal cord, an important consideration in suspected multiple sclerosis and other myelopathies that may not be detected by imaging techniques.

**Cutaneous Nerve Stimulation.** Virtually any accessible cutaneous nerve can be stimulated to elicit an SEP. With cutaneous nerve stimulation, however, the potentials are smaller than those evoked by mixed nerve stimulation, and the small far-field components are difficult to record. Use of cutaneous nerve stimulation should be considered when it is necessary to: (1) assess the integrity of specific cutaneous nerves, such as the lateral femoral cutaneous nerve, which are not readily studied by conventional techniques; (2) measure peripheral sensory conduction when this is not otherwise possible because the sensory nerve action potential (SNAP) is either absent or very small; (3) evaluate isolated root function, because of the increased segmental specificity of cutaneous stimulation, and (4) assess dubious patchy numbness for medicolegal reasons, by stimulating homologous areas of “involved” and normal skin supplied through cutaneous terminals.

**Dermatomal Stimulation.** Dermatomal stimulation is even more segmentally specific than cutaneous nerve stimulation, because cutaneous nerve stimulation invariably activates fibers from more than one dermatome. However, with dermatomal stimulation the ascending volley is very desynchronized, and this sometimes makes the SEP difficult to interpret. Dermatomal stimulation has been used most often to assess function of the lumbosacral roots. For L5, the medial side of the first metatarsophalangeal joint or the dorsal surface of the foot between the first and second toes is stimulated. For S1, the lateral side of the fifth metatarsophalangeal joint is stimulated. Care must be taken to avoid stimulus spread to neighboring dermatomes, underlying muscle (which induces activity of Ia afferents), and digital cutaneous nerves. This can be achieved if the stimulus is kept at 2.5 × sensory threshold, which gives about 80% of the maximum amplitude. Normative data for the L5 and S1 dermatomes are well established.

**Motor Point Stimulation.** A single muscle motor point can be stimulated to elicit SEPs. This is achieved by using a monopolar needle electrode for stimulation. A long-duration (1.0 ms), low-intensity stimulus preferentially activates the Ia afferents that exit in the same bundle as the alpha motor fibers at the motor point. This method allows stimulation of proximal (large) muscle Ia afferent input.

**Paraspinal Stimulation.** The paraspinal region at sequential levels along the vertebral column can be stimulated to elicit SEPs. This method “obviates” peripheral input from long nerves in the limbs and more readily identifies lesions of the spinal cord. The stimulus is applied simultaneously to both sides, 2 cm lateral to the midline, at an intensity that induces a small visible muscle twitch. Potentials are recorded over the scalp (Cz–Fz). The afferent volley is primarily initiated in the cutaneous branches of the primary dorsal root rami, with some contribution from the paraspinal Ia afferents. In normal subjects, the value for spinal cord conduction velocity between T12 and T1 is approximately 64 m/s. However, it is better to refer to conduction time, which over the same segments is 5.4 ± 1.6 ms.

RECORDING AND FILTERING OF SEPs

Surface or needle electrodes can be used for recording SEPs. There is no difference in the resultant SEP. The latter, although easily inserted into the scalp, are not popular because of their higher impedance, the discomfort of their insertion, and the theoretical
risk of infection. Recording montages are either “cephalic bipolar” or “referential.” In a cephalic bipolar montage both electrodes are placed on the head, while in a referential montage the reference electrode is placed off the head. A cephalic bipolar montage is relatively noise-free and is satisfactory for routine clinical use. However, there is cancellation of the small-amplitude, far-field potentials, which can only be recorded with noncephalic references (e.g., the opposite mastoid, shoulder, arm, hand, or knee, or the linked mastoids or earlobes). Recording with both types of montage is advantageous (see Fig. 1).

The number of channels (recording derivations) used should be dictated by the specific reason for performing the SEP. For example, in field distribution studies, 16 or more channels may be useful, whereas one channel is sufficient when the SEP is being used to measure peripheral conduction velocities.

With a bipolar cephalic derivation, near-field potentials, which are characteristically of negative polarity and of relatively large amplitude, are recorded. Their amplitude falls rapidly when the electrode is moved only a short distance from the generator source. Certain small-amplitude far-field potentials may be recorded with bipolar cephalic derivations, but referential recording is required for proper identification. Far-field potentials are small in amplitude, recorded with equal ease and amplitude over a wide area of the scalp, and usually positive and monophasic at the active electrode, reflecting a moving front approaching the recording electrode. Kimura and coworkers showed that under some circumstances far-field potentials may be biphasic and of either polarity. The posterior tibial near-field potential is also positive in polarity.

Small-amplitude components of the SEP are composed of both high and low frequencies, and filtering can be problematic. Too wide a bandpass results in a “noisy” SEP, while a restrictive bandpass attenuates either the high- or low-frequency components, depending on the settings chosen. There is no “correct” filter setting—the choice is best related to the particular task. For general purposes, a relatively broad bandpass (10–2500 Hz) is suitable. Restrictive filtering of between 150 and 300 Hz to 3000 Hz enhances high-frequency, small-amplitude, near- and far-field components, but does so at the expense of the low-frequency components.

Analog-restrictive filtering induces phase shift of

\[
\begin{align*}
C4' - C3' & \quad N20 \\
C3' - EPC & \quad P14 \\
CV5 - EPC & \quad N13 \\
CPI - EPC & \quad NA \\
\end{align*}
\]

**Figure 1.** Normal SEP elicited by stimulation of the left median nerve and recorded over the Erb’s point region, fifth cervical spine, and scalp. EPI, ipsilateral Erb’s point; EPC, contralateral Erb’s point; CV5, fifth cervical spinous process; C3’ and C4’, scalp placements halfway between C3 and P3, and C4 and P4, respectively. The bottom trace shows the Erb’s point potential, the second channel shows mainly the stationary cervical potential, the third channel shows the subcortical far-field potentials (especially P14 and N18), and the top channel shows the N20 potential.
components and may induce distortion of components. Digital filtering, designed for zero phase shift, does not induce phase shift or distortion of components and does not create "new" peaks. Digital filtering does however greatly enhance peaks that are normally present. This is of particular promise for SEPs evoked by leg stimulation, when the small-amplitude components, usually difficult to recognize, become easily identifiable.

**MEASUREMENT OF THE SEP**

The latency, interpeak latency, amplitude, morphology (presence or absence of components), and dispersion of the SEP can be measured, and side-to-side comparisons can be made. Latency is easily measured and standardized, whereas other characteristics (e.g., morphology or dispersion) may be difficult to assess. Latency varies with limb length. Interpeak transit (conduction) times are reliable parameters independent of limb length and usually independent of peripheral nerve disease. Central afferent pathways do not mature at the same rate as peripheral pathways, and adult values for conduction velocity are not attained until 7 or 8 years of age. 

Aging is associated with prolongation of SEP latencies. This is not simply a reflection of slowed peripheral conduction, because central conduction times are also slowed significantly.

Absolute and interpeak latency are considered abnormal when they are more than 3 SDs rather than 2 or 2.5 SDs above the normal mean. Less statistical strictness is valid if the results are related to age and height. In fact, however, latency is seldom related to age and height in clinical laboratories. Absolute and interpeak latencies are easily measured but frequently are normal in the face of obvious clinical impairment. Care is required to distinguish between a delayed and an absent component; when a component is absent, the subsequent component may be mistaken for the missing potential.

Absolute amplitude of SEP components is variable, but a side-to-side difference exceeding 50% is usually abnormal, providing the clinician can be certain that the disease is unilateral. An excessive interside amplitude difference is nonspecific and indicates either central conduction block or considerable neuronal/axonal loss. However, complex facilitatory and occlusive interactions leading to "central gain" can prevent amplitude reduction in the SEP, masking axonal or neuronal loss. SEP amplitude increases in the elderly, and "giant" potentials typify hereditary myoclonic epilepsy. SEPs evoked by dermal—as opposed to nerve—stimulation are much more variable in amplitude.

The morphology (shape) and dispersion of the SEP are difficult to quantitate, but both may be abnormal before or in the absence of latency prolongation or amplitude reduction. Computer-assisted methods for quantifying both dispersion and morphology of the SEP have been described.

**NEURAL GENERATORS OF THE SEP**

It is generally accepted that the different components of the SEP predominantly reflect sequential activation of neural generators excited by the ascending volley. This concept is appealing because it provides a rational base for interpreting an absent or abnormal component in relation to a specific anatomic lesion. However, factors other than neural generators (synapses in relay nuclei) are important in the origin of some SEP components, especially the small far-field potentials recorded using noncephalic references. In particular, the stationary far-field peaks, for example P9, P11, P13, and P14, evoked by median nerve stimulation reflect propagated volleys of action potentials traveling in axons and can be recorded as the traveling volley approaches, but before it actually reaches, the active recording electrode. This results from physical changes in the surrounding volume conductor, including the resistance or impedance of the volume conductor, sites of axonal branching, and anatomic orientation of the traveling impulse.

Of the far-field potentials in the median and tibial SEPs, it seems likely that relay nuclei are responsible only for the generation of P13/14 (median) and P31 (tibial) components, with the other potentials probably reflecting electrophysical change in the surrounding volume conductor. The near-field N20 (median) and P38 (tibial) components are neurally generated, but it is likely that they reflect multiple and even independent thalamocortical projections. Certainly, from studies using digital filtering, it appears that there are "too many components" for available "generators." SEP components are designated by their polarity and normal latency. The numerical latency values ascribed to each component are summarized in Table 1, which also indicates current views of the origin of specific, early latency, SEP components elicited by median and tibial nerve stimulation and recorded over the scalp.

Little is known about the anatomic substrates of the middle (and longer) latency components of the
SEP. They exhibit considerable intertrial variability, and their amplitude and latency are affected by sleep, level of consciousness, habituation, and cognitive function. Nevertheless these components may have some clinical relevance. Isolated abnormality of the contralateral P40–N60 in the median SEP has been described in multiple sclerosis and with deep subcortical lesions involving thalamic nuclei other than the primary sensory nuclei.117

Long-latency components are distributed bilaterally, being of greatest amplitude at the vertex. These components relate to nonspecific thalamocortical projection systems involved in habituation, adaptation, and arousal. These components are of large amplitude, being between 10 and 40 µV, so that as few as 50 averaged epochs are required to obtain a measurable response. Unfortunately their recovery time takes several seconds, necessitating a stimulus repetition rate that is lower than once every 5 s to avoid significant attenuation of the response.

Spinal SEPs. Using a vertical and horizontal array of electrodes placed over the neck and referred to a noncephalic reference, the cervical SEP elicited by median nerve stimulation consists of three distinct components41: (1) the proximal plexus volley (PPV), with a latency of about 10 ms; (2) the dorsal column volley (DCV), reflecting the volley in the dorsal column, with a latency of about 12 ms; and (3) the cervical N13/P13, which is possibly generated at the level of the dorsal gray of the cervical cord. With a cephalic reference these three components become fused, but the latency difference between its peak and the N20 cortical SEP gives a “central conduction time” between the lower brain stem and cortex that measures about 5.5 ms.

Using a knee or iliac crest reference, SEPs elicited by tibial nerve stimulation can be recorded over the thoracolumbar spine. The components of the SEPs correspond to those described above for the cervical SEPs elicited by median nerve stimulation. Posterior tibial nerve stimulation at the ankle evokes a traveling wave, N18, that can be recorded over the lower lumbar spine immediately after the volley has passed the sacral plexus and is the scalp-recorded far-field P18 potential. This plexus-propagated volley is the equivalent of the PPV recorded over the neck with median nerve stimulation. A second distinct potential, N22, can also be recorded over the lower spine. It is a stationary peak and its latency remains constant. Its amplitude is maximal over the lower thoracic spine (T10–L1). N22 probably represents postsynaptic activity in the lumbar gray matter generated in response to inputs from axon collaterals.40 A third negative traveling wave, N24, whose latency shortens from caudal to rostral thoracic cord, is evident over the lower thoracic cord. It reflects a propagated volley in the dorsal columns and is equivalent to the DCV potential recorded over the neck with median nerve stimulation. Spinal SEPs may not be

<table>
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<th>Table 1. Origin of far-field and near-field SEP components.</th>
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<td><strong>Generator</strong></td>
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<td>Median nerve</td>
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<td>P9</td>
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This reflects the authors’ view; differing opinions exist.
recordable in all normal subjects, and their absence must be interpreted with caution.

**SEPS IN DISORDERS OF THE PERIPHERAL AND CENTRAL NERVOUS SYSTEM**

Although SEPs may reveal or localize a lesion involving the somatosensory pathways, they are simply an extension of the clinical examination and do not indicate the underlying disease process. Normal findings do not exclude an organic basis for symptoms. Despite these limitations, SEP studies may be helpful diagnostically and to determine the extent of pathologic involvement in different disorders.

**Peripheral Nervous System.**

**Disorders of the Peripheral Nerves.** SEPs may be helpful in evaluating the peripheral nervous system when nerve conduction studies (NCSs) are inapplicable because of the proximal site or severity of pathology. Peripheral sensory conduction velocity can be measured by SEPs with comparable results to those from conventional NCSs. The nerve is stimulated at two or more sites and the responses are recorded over the scalp. Scalp-recorded SEPs may be absent or delayed in the presence of polyneuropathies and mononeuropathies. Scalp-recorded SEPs may be important for identifying peripheral nerve lesions when SNAPs are unrecordable peripherally. Parry and Aminoff recorded robust SEPs on stimulation of each of 15 nerves from which SNAPs are not recordable in all normal subjects, and their absence must be interpreted with caution.

**Plexus Lesions.** The prognosis of brachial plexus injuries influences clinical outcome; a postganglionic lesion—unlike root avulsion—sometimes requires operative treatment and may be followed by recovery. Conventional NCSs can suggest the site of injury. Preserved SNAPs despite clinical sensory loss indicate a preganglionic lesion. Recording over the scalp and spine of SEPs following stimulation of upper limb nerves has been suggested by some physicians to improve the electrophysiologic evaluation of plexus lesions, based on the belief that N13 attenuation reflects the total damage, whereas N9 attenuation reflects the extent of postganglionic damage.

Jones and coworkers compared the preoperative electrophysiologic findings with the site of the lesion at surgery in 16 patients with unilateral traction lesions of the brachial plexus. The electrophysiologic findings correctly localized the lesion in only 8 instances; in 3, the findings suggested a purely peripheral SNAP findings, perhaps because of the misleadingly normal values for conduction velocity sometimes obtained by the SEP technique.

Focal nerve lesions can be diagnosed by scalp-recorded SEPs, but this is only helpful when the lesion is too proximal to be recognized by conventional techniques, such as in meralgia paresthetica. Some physicians have also used the SEP technique to follow recovery after peripheral nerve transaction injuries—the SEP may be present when peripheral SNAPs cannot be elicited.

The SEP findings have been used to diagnose Guillain–Barré syndrome when distal conduction is normal. The aim is to demonstrate conduction slowing in the proximal segments of peripheral nerves. In this regard, some physicians have found SEPs to be more sensitive than F waves, whereas others have found the converse. Aminoff and associates, in a study of 44 nerves in 15 patients with Guillain–Barré syndrome, found F-wave abnormalities more frequently than SEP abnormalities, especially when peripheral conduction was normal. The authors therefore believe that SEP studies are best reserved for occasions when both peripheral and F-wave studies are normal in patients with suspected Guillain–Barré syndrome.

SEPs may have some role in the evaluation of patients with distal axonopathies, but this remains to be established.
taneous nerve stimulation are especially likely to be attenuated or absent with C5 and C6 root lesions. A preganglionic lesion may be partially or completely obscured electrophysiologically by coexisting postganglionic pathology, thereby limiting the utility of SEPs for evaluating plexus lesions. Moreover, the information derived from SEP studies concerning the site and extent of plexus injury can usually be obtained by needle electromyography (EMG). Nevertheless, the finding of attenuated (but present) peripheral SNAPs and absent spine and scalp SEPs is important in suggesting that multiple root avulsions have occurred in addition to more peripheral lesions.

At operation, the appearance of the brachial plexus may be misleading, especially when disrupted nerve fascicles appear intact. Intraoperative stimulation of specific roots, with recording of the responses from the contralateral scalp, is therefore helpful in indicating whether there is functional continuity with the cord. Similarly, when ruptured nerves are to be repaired by grafting, cortical responses to stimulation of the proximal stump indicate that a second more rostral lesion is unlikely, and an absent response suggests a poor outcome.

Thoracic Outlet Syndrome. Characteristic electrophysiologic changes may occur in patients with neurogenic thoracic outlet syndrome. This is an uncommon disorder. In many patients suspected to have neurogenic thoracic outlet syndrome, there is no clinical or electrophysiologic abnormality in conventional studies. The SEP findings in such patients are variable. Both the authors' findings, and those reported from the Mayo Clinic, indicate that SEP studies are of limited value for the diagnosis of the neurogenic variety of thoracic outlet syndrome. In some patients there may be abnormalities of the ulnar-derived SEP, such as an absent or markedly attenuated N13 response despite a relatively normal N9 peak, or a small delayed N9 peak with or without abnormalities of the N13 or N20 or prolonged interpeak intervals. In one instance, the authors found the only abnormality was marked attenuation of the N20 component.

In patients with the nonneurogenic syndrome, the authors and others have found normal median, ulnar, or radial SEPs. However, in one study, 12 of 18 patients, most of whom had no clinical deficit, were found to have abnormal ulnar SEPs, with small N9 potentials despite preserved distal ulnar SNAPs, a finding difficult to explain on pathophysiologic grounds. In another study, 13 of 19 patients with suspected thoracic outlet syndrome had abnormal SEPs evoked by median or ulnar nerve stimulation, but the clinical and electrophysiologic findings were not detailed, and interpretation is further complicated because diagnostic criteria for the SEP abnormalities were not provided.

The published evidence is thus conflicting concerning the role of SEPs in the evaluation of suspected thoracic outlet syndrome, but in the authors' view, SEPs are of limited utility in diagnosing the neurogenic disorder and of no established value in diagnosing the nonneurogenic variety.

Cervical Spondylotic Myeloradiculopathy. SEPs do not distinguish spondylosis from other cervical lesions. Patients with pain and paresthesias but no neurologic signs frequently have normal median or ulnar SEPs, whereas those with objective clinical deficits may have abnormal SEPs, with delayed or lost components, regardless of whether there is a myelopathy. The nature of the SEP abnormality does not indicate either the severity or long-term prognosis of the neurologic disorder, and some patients with a spondylotic myelopathy have normal SEPs. Thus the findings are no better than careful clinical examination in determining severity and prognosis of cervical spondylosis, and do not help in the selection of patients for surgery.

It has been suggested that fibers ascending from the lumbosacral region are more likely to be affected by cervical compression than fibers arising from the upper limbs. It is not yet established, however, whether recording sural SEPs in patients without a myelopathy helps to indicate those at risk of a clinical cord deficit.

Epidurally recorded SEPs have been recorded in patients with cervical spondylosis but should not be used routinely for evaluating patients or in selecting patients for surgery.

Radiculopathy. There are reports that peroneal or tibial SEPs are abnormal in many patients with an isolated lumbosacral radiculopathy, but this is surprising, because stimulation of a multisegmental nerve should not lead to an SEP abnormality in the presence of an isolated root lesion. Aminoff and colleagues have found normal peroneal SEPs in all of their patients with a compressive L5 or S1 root lesion.

Eisen and coworkers stimulated cutaneous nerves to improve the segmental specificity and recorded the SEPs in patients with myelographically proven cervical or lumbosacral radiculopathies. Among 28 patients, only 16 (57%) had abnormal SEPs, particularly amplitude reductions and abnormal morphology; a prolongation in latency was uncommon. Others subsequently reported that SEPs elicited by cutaneous nerve stimulation were more reward-
Thus, they correctly identified 25 of 34 roots from their patients, most of whom had extensive disease.3 In fact, they accurately and completely predicted on electrophysiologic grounds the operative findings in only 4 of 20 patients, a yield similar to that of Aminoff and associates.4,5 A more recent study by Dumitruc and Dreyfuss also suggested that dermatomal SEPs are of limited diagnostic utility in patients with suspected unilateral L5 or S1 radiculopathy, because they do not have both a high sensitivity and a high specificity.28 They appear to be less accurate and sensitive than EMG and anatomic (imaging) studies.95

Dermatomal SEPs have also been used to evaluate patients with lumbar spinal stenosis, and may be more helpful in this context than when used to evaluate isolated compressive root lesions (W.C. Stolov, personal communication). A detailed account of this work and a comparison of the yield of dermatomal SEPs and needle EMG, however, has yet to be published.

Central Nervous System. The SEP findings may detect and localize clinical somatosensory lesions but are not pathognomonic of specific diseases.1 Detection of Lesions in Central Somatosensory Pathways, Multiple Sclerosis. The presence of SEP abnormalities may reveal subclinical somatosensory lesions and thereby establish that multiple lesions are present. An SEP abnormality may also indicate that vague sensory complaints have an organic basis, especially when the findings on clinical examination are equivocal.

The likelihood of finding an SEP abnormality is greater in patients with definite multiple sclerosis (MS) than in patients with possible MS. The incidence of abnormalities in patients with definite MS is about 80% regardless of whether there is any clinical sensory disturbance, but among patients with possible MS the incidence of subclinical SEP abnormalities is only about 25–35%. The yield is highest from SEPs elicited by stimulation of a nerve in the legs. An SEP abnormality is more likely if there is a clinical sensory disturbance in the stimulated limb, although the electrophysiologic finding then provides little further information than obtained by clinical examination. SEP abnormalities are also more common when there are pyramidal signs in the stimulated limb or legs.103 In patients with a pyramidal deficit, however, an SEP abnormality may not reflect a separate pathologic lesion but merely an extensive lesion involving both motor and sensory pathways.

Among the different evoked potential techniques for detecting subclinical involvement of afferent pathways in patients with suspected MS, the highest yield is with SEPs; brain stem auditory evoked potentials (BAEPs) are the least useful.52,65,93,111 The basis of such differences is unknown but may reflect the length and extent of the tracts being tested or differential susceptibility to pathologic involvement.

Abnormalities of the SEP may include delay or absence of various components or an altered response-morphology. In the median SEP, a common abnormality is loss of the cervical (N13) component with preservation of the later components. This work and a comparison of the yield of dermatomal SEPs and needle EMG, however, has yet to be published.
sidered as well. The corresponding number for patients with definite MS was 87% and 73%, respectively. The cervical SEP abnormalities encountered most often were absence, attenuation, and increased dispersion of the response; only very occasionally was there an increase in latency. Central conduction time, as calculated from the interpeak latency of various components of the SEP, commonly shows significant side-to-side difference in patients with MS. In normal subjects, there is no significant change in the median SEP when body temperature is raised by 1°C, apart from a slight reduction in latency resulting from increased peripheral conduction velocity. Among patients with MS, by contrast, an initially normal cervical response may become abnormal, or—less commonly—an initially abnormal response may normalize. In many patients, especially when the N13 is already abnormal, the cervical SEP abnormalities encountered most often were absence, attenuation, and increased dispersion of the response; only very occasionally was there an increase in latency. Central conduction time, as calculated from the interpeak latency of various components of the SEP, commonly shows significant side-to-side difference in patients with MS. In normal subjects, there is no significant change in the median SEP when body temperature is raised by 1°C, apart from a slight reduction in latency resulting from increased peripheral conduction velocity. Among patients with MS, by contrast, an initially normal cervical response may become abnormal, or—less commonly—an initially abnormal response may normalize. In many patients, especially when the N13 is already abnormal, the central nervous system, other than MS, that interrupts the somatosensory pathways. They are normal, however, when only spinothalamic function is impaired.

Spinal cord dysfunction. Patients with spinal cord tumors or malformations or with spinal arteriovenous malformations may have abnormal SEPs if the posterior columns are involved. In patients with repaired spinal dysgraphic lesions, serial SEP studies have sometimes been used to facilitate early detection of clinically significant retethering, but the findings do not correlate well with clinical status and are of questionable utility.

An upper cervical myelopathy occurs in achondroplasia due to a small foramen magnum, and may lead to abnormal median or peroneal SEPs. In one study, SEPs were abnormal in all patients with neurologic symptoms or signs and in 44% of patients without neurologic dysfunction. In several of these patients, CT scans showed significant stenosis at the foramen magnum. Thus SEPs may have some use as a monitor of cord function in achondroplasia.

SEPs have also been used to monitor the effects of radiation on the spinal cord. The findings have suggested that subclinical cord dysfunction may follow radiotherapy for lung cancer even at conventional dose schedules.

A variety of SEP abnormalities has been described in Friedreich’s ataxia, including mild latency prolongation, and broadening or fractionation of the cortical response. Nuwer and colleagues found in Friedreich’s ataxia that SEPs elicited from upper-limb nerves generally have delayed N20 peaks, but that the Erb’s point potential is either normal or only minimally delayed when present. In some cases the Erb’s point potential may be markedly attenuated.

In patients with hereditary spastic paraplegia or hereditary cerebellar ataxia, there may be loss of spinal or cortical components or marked cortical delay, even when there is no clinical sensory loss. In these disorders and Friedreich’s ataxia, there may...
also be abnormality of the visual and auditory evoked potentials. In olivopontocerebellar atrophy, SEPs may be normal, attenuated, or absent.

SEPs may be abnormal in patients infected with the human immunodeficiency virus (HIV). Patients with acquired immune deficiency syndrome (AIDS) may have delayed cortical responses to tibial nerve stimulation, suggesting slowed spinal conduction. In non-AIDS patients, spinal latency and conduction time from gluteal crest to T12 tend to increase over time. Again, others have reported that in some patients with subclinical HIV infection, SEPs are abnormal and may suggest a conduction defect in central somatosensory pathways. Whether SEP studies have any clinical or prognostic relevance in this context is not established. In human T-cell lymphotropic virus (HTLV)-I-associated myelopathy/tropical spastic paraparesis, lower-limb SEPs may provide important information; central sensory conduction time correlates with disability and may reveal subclinical lesions of afferent pathways. Lower-limb SEPs may be unrecordable in patients with Pott’s paraplegia.

Brain stem lesions. SEPs are normal in Wallenberg’s syndrome, but are usually abnormal when the medial lemniscus is involved. They may be abnormal in the “locked-in” syndrome due to pontine infarction. The SEP findings do not distinguish reliably between intra- and extra-axial lesions of the brain stem.

Thalamic lesions. The SEP findings in patients with thalamic pathology tend to parallel the clinical findings, usually but not invariably being abnormal when there are significant clinical deficits. Lateral lesions affect particularly the tibial SEP, and more mesial lesions affect the median SEP. The SEP is sometimes preserved with slowly growing noninfiltrating extrinsic tumors involving the thalamus, whereas extrinsic mass lesions compressing the thalamus acutely may abolish both median and tibial SEPs. The SEP is generally markedly abnormal in patients with intrinsic thalamic tumors and a clinical sensory deficit.

Hemispheric lesions. In patients with predominantly unilateral cerebral lesions, there is a correlation between the clinical and electrophysiologic findings. SEPs are often normal in patients with no sensory deficit and abolished in those with moderate or severe cortical sensory loss. In some patients, however, there is a discrepancy between the clinical and SEP findings—SEPs are normal despite cortical sensory deficits or absent despite preserved sensation.

Mauguiere and coworkers found that complete parietal lesions produced contralateral hemianesthesia without pyramidal signs, and eliminated the parietal N20–P27–P45 complex but not the prerolandic P22–N30 complex, indicating that the N20 and P22 components have separate generators. Small postcentral lesions causing astereognosis (with otherwise preserved sensation) reduced or abolished the N20 and P27 components without affecting the P22–N30 complex.

Recent work suggests that the electrophysiologic findings are no better than a detailed neurologic examination in predicting outcome from stroke, despite earlier suggestions to the contrary.

SEPs as a Guide to Prognosis. Coma. In 36 comatose patients with preservation of some brain stem function, Goldie and colleagues found the Erb’s point response and the N13/N14 cervical response to median nerve stimulation were preserved. Patients with bilateral loss of the N20–P22 response either died or remained in a vegetative state. Unilateral loss of the N20–P22 responses was associated with a variable clinical outcome: death, a persistent vegetative state, or recovery of a functional level. Among patients with bilaterally preserved N20–P22 responses, 56% became functional, 25% remained in a vegetative state, and 19% died. Loss of the cortical N20–P22 responses therefore suggests a poor prognosis.

Among patients in posttraumatic coma, there is an association between the degree of abnormality in the median SEP and clinical outcome. Hume and Cant examined the scalp-recorded SEP findings soon after onset of posttraumatic coma. Bilateral loss of median SEPs was believed to suggest a fatal outcome, and consistently asymmetric responses (in amplitude and latency) or a unilaterally absent SEP were regarded as indicating the probable development of a severe residual deficit. The SEP findings predicted the outcome correctly in 38 of the 49 patients studied in the first 84 h after coma onset. However, the SEP findings sometimes changed when recorded serially, and the outcome did not correlate well with the SEP results in every test period. For example, 44 patients ultimately did badly (i.e., died or were left with a severe deficit), and 10 of them sometimes had normal SEP findings, while 3 had normal SEPs on every recording. Similarly, among the 31 patients who did well, 15 had abnormal SEPs on at least one occasion. Thus the SEP findings may not be as useful as originally hoped in providing a satisfactory prognostic guide in individual cases.

Among patients in anoxic–ischemic coma, the findings in one study revealed that none of 30 patients with absent cortical SEPs recovered cognition. The presence of normal SEPs did not reliably predict recovery, however. Some patients with normal corti-
cal SEPs several hours after cardiac arrest recovered cognition, whereas others did not.11 The SEP findings corresponded to some extent with brain stem function. One or several brain stem reflexes were absent on initial examination in a majority of patients with lost cortical SEPs but in only 20% of those with preserved SEPs. In another study, involving 60 patients in coma for more than 6 h after cardiac arrest, the association of a Glasgow coma score of less than 8 at 48 h with abnormal or absent early cortical components of the median SEP was highly predictive of a bad outcome.8

In locked-in syndrome, the SEP findings vary considerably in different patients and are of little diagnostic help.54

Brain Death. SEPs are more important than BAEPs in evaluating patients with suspected brain death, because all responses to auditory stimulation are often absent, possibly for technical reasons or from deafness.50 With median nerve stimulation, by contrast, most patients have cervical (N13/N14) but not later responses, indicating that input reaches the cord-medulla but does not generate cerebral activity more rostrally. This suggests an important role for SEPs in the evaluation of suspected brain death. The use of special recording derivations (midfrontal to median nasopharyngeal) for obtaining the median SEP may be helpful in distinguishing between coma and brain death, but this requires further study.114

Spinal Injuries. SEP changes may occur with spinal injury.27,97,99 Responses to stimulation of a nerve in the legs may be normal, delayed, small, or lost, depending on the extent and severity of the lesion and the timing of the examination.

It may not be possible to record any cortical response during the acute stage after incomplete traumatic cord lesions,99 so an absent SEP does not reliably permit early distinction of complete from incomplete lesions. Preserved responses or their early return after injury, however, indicates an incomplete lesion and therefore a better prognosis than otherwise, although functional recovery may still be poor.97

Use of SEPs to Prevent or Minimize Neurological Problems. SEPs have been used intraoperatively to monitor cord function, but their utility in this context is unclear and beyond the scope of the present monograph.

Defining the Extent of Neuropathologic Involvement in Neurologic or Neuromuscular Disorders. SEP abnormalities have been found in patients with amyotrophic lateral sclerosis,53,94 and this accords with previous clinical and pathologic reports of sensory involvement in this disorder. Polo and colleagues have found the SEP elicited from both upper and lower limbs to be abnormal in patients with bulbar spinal neuronopathy (Kennedy's syndrome).92 SEP studies have suggested that in diabetics, central afferent transmission—as well as peripheral nerve conduction—may be affected.14 Median SEPs may be abnormal in dystrophia myotonica, reflecting peripheral or central conduction delays in about one third of cases.7 There seems to be no relationship, however, between the SEP findings and severity of the clinical disorder.

Some patients have sensory symptoms but no objective clinical deficits, suggesting that symptoms are not organic in origin. In such a circumstance, an abnormal SEP indicates that symptoms have an organic basis. Normal SEPs and peripheral sensory NCSs may support suspicions that symptoms are not organic, but do not establish this with certainty. Indeed, SEPs may be normal in patients with pure sensory stroke due, for example, to lacunar infarcts.

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