Chapter 8
Somatosensory Evoked Potentials

Jamison Beiriger, Varun Shandal, Josh Sunderlin, and Parthasarathy D. Thirumala

Somatosensory Evoked Potentials

Overview

Evoked potentials are electrical responses of the sensory pathways in the nervous system to sensory or electrical stimuli. Stimulation and subsequent recording of these potentials allows for evaluation of both peripheral sensory function and integrity of the central nervous system (CNS) sensory pathways. Evoked potentials are used to assess three main stimuli – visual, somatosensory, and auditory via pattern visual evoked potentials (PVEP), somatosensory evoked potentials (SSEP), and brainstem auditory evoked potentials (BAEP), respectively. This chapter will focus on somatosensory evoked potentials.

Somatosensory evoked potentials (SSEPs) were first described by Larson and Sances in the 1960s and are now the most widely utilized evoked potentials [1].

J. Beiriger
School of Medicine, Drexel University, Philadelphia, PA, USA

V. Shandal
Department of Neurological Surgery, Center for Clinical Neurophysiology, University of Pittsburgh, Pittsburgh, PA, USA

J. Sunderlin
Procirca, Clinical Neurophysiology, University of Pittsburgh Medical Center, Pittsburgh, PA, USA

P. D. Thirumala (✉)
Department of Neurological Surgery and Neurology, University of Pittsburgh, Pittsburgh, PA, USA
e-mail: thirumalapd@upmc.edu

S. M. Verst et al. (eds.), *Intraoperative Monitoring*,
https://doi.org/10.1007/978-3-030-95730-8_8

These potentials are useful for procedures that put any part of the somatosensory pathway at risk. Neurologic deficit is a common adverse event of certain procedures: Implanting instrumentation for scoliosis has a neurologic deficit incidence of 0.5–1.6%, decompression of spinal cord tumors or trauma has a 20% incidence of neurologic deficit, and thoracic aorta surgery has incidence of paraplegia up to 40% [2–5]. Monitoring SSEPs allow for real-time intraoperative assessment of spinal cord pathways and in some cases allows for correction of potentially harmful maneuvers. SSEP involves using an electrical impulse to generate an action potential propagation along the dorsal column and monitoring the functionality of the pathway at various points. The stimulus is typically applied distal to the site at risk and sites distal to the stimulation and proximal to the surgical site are subsequently recorded. Changes in amplitude or latency of waveforms representing a summation of hundreds of trials may indicate damage along the somatosensory pathway, which serves as a good surrogate marker for overall spinal cord integrity [6]. Surgeons may utilize this information to manipulate their technique to reverse the damage or prevent further injury. Monitoring of SSEPs is a quick and effective method commonly employed to reduce the incidence of neurologic deficits in at-risk patients intraoperatively.

Purpose

Intraoperative monitoring of SSEPs is an easy, effective technique for assessing somatosensory pathway function. Changes in SSEPs during surgery can result from either mechanical, thermal, or ischemic injury. Significant changes in SSEPs, indicative of warning for pending or completed intraoperative somatosensory pathway injury, allow for timely intervention to correct or limit the harm. SSEPs are an important neurophysiologic intraoperative monitoring technique critical to assessing the integrity of the somatosensory pathway (that includes dorsal column pathway and peripheral nerve) that can get injured or malfunction due to surgical manipulation or patient positioning.

Qualifications

The American Society for Neurophysiologic Monitoring (ASNM) categorizes proficiency in SSEP monitoring into two categories – professional and technical. For the professional standard, the ASNM recommends certification by the American Board of Neurophysiologic Monitoring, which entails having an advanced degree (Masters, PhD, M.D., or D.O.), clinical experience of at least 300 monitored cases over at least 3 years, surgeon attestations of experience, and passing scores on two

examinations (oral and written). An aspiring technician needs at least a high school degree with healthcare credential or a bachelor's degree, clinical experience with at least 100 monitored cases, a passing score on a written examination, and a supervising physician attestation to be eligible for the Certification in Intraoperative Monitoring by the American Board of Registry for Electroneurodiagnostic Technologists. In addition to these qualifications, individual institutions should have guidelines on the scope of practice and continuing education requirements [5].

Equipment and Settings

Electrodes

Three types of electrodes are used for SSEP generation and recording. Surface electrodes can be adhesive to the skin, bar electrodes, or cup electrodes filled with conductive gel. Bar electrodes are discouraged for intraoperative monitoring due to the risk for pressure necrosis [7, 8]. Surface electrodes can be applied preoperatively, reducing intraoperative setup time and allowing for early post-inductive parameter optimization and baseline measurements. With proper preparation, surface electrodes are safe and effective with <2 kΩ of impedance [9]. Surface electrodes are necessary for obtaining dermatomal SSEPs. One problem with surface electrodes is in long procedures the conductive paste can dry out, but as long as a constant current is maintained, the recording should remain accurate [5]. Also, adhesive electrodes are not reusable, so may be a costlier option. Popular for stimulation, needle electrodes are effective, quick to apply, and have <5kΩ of impedance. However, needle electrodes carry a risk of infection, hemorrhage, and burns due to accidental electrosurgical current passing through [7, 8, 10]. Needles should be secured with tape or self-securing as with corkscrew needles in the skull. Additionally, it is important to avoid subdermal structures at risk of injury including shunts. Surface cup electrodes and needle electrodes are good for recording from the body surface [11]. Lastly, invasive electrodes, either subdural or epidural, can be used in specific cases. Infection, hemorrhage, and trauma are potential risks when recording with these electrodes.

Channels

The evoked potential machine should be capable of supporting at least 8 channels. The machine should be able to record and display both a cortical and subcortical response from each limb simultaneously. In procedures with additional monitoring modalities such as spontaneous EMG or other evoked potentials, more channels may be required.

Filter

Filter settings are important for intraoperative monitoring. Diagnostic studies typically use consistent filter settings to compare individual patients against the normal, but for intraoperative monitoring it is more desirable to find a setting that gives easily interpretable results with the least trial numbers. The filter should be optimized at the beginning of the study and remain unchanged throughout the study so comparisons can be made to earlier time points. Initial filter settings should be standard with manipulation at the beginning of the case. Filters for cortical responses should begin with a system bandpass of 1–30 to 250–1000 Hz [11, 12]. Often, the SSEPs are above 30 and below 500, so narrowing the bandpass to 30–500 Hz may reduce higher frequency noise artifacts [5]. Subcortical filters should have a system bandpass of 30–100 to 1000–3000 Hz [11, 12]. Unlike cortical SSEPs, subcortical SSEPs can be up to 1000 Hz, so the high end of the bandpass range should extend up to 1000 Hz [5, 13]. The bandpass for all recording is often set narrow because the operating room has numerous sources of electrical signals producing extraneous artifacts. One common artifact in this setting is at 60 Hz, but a 60 Hz rejection filter is not advised because it can cause a "ringing artifact". The 60 Hz filter should only be used in situations where adequate responses are unobtainable without it [5].

Averager

The averager compiles electrical responses of numerous trials into a single output while simultaneously eliminating trials contaminated with artifact. The number of trials averaged for a given response depends on the given situation. Early guidelines suggested 500–2000 trials [12]. Fewer trials and therefore faster results come with increased error. In situations where fast results are desired, the bandpass is narrow, and there is low background noise, the number of samples may be as low as 100 for upper extremity SSEPs. Most systems, however, average about 300 to 500 trials for both upper and lower extremity potentials. One consideration for the necessary number of trials to be averaged is the signal to noise ratio. More trials maximize signal strength and reduce noise.

Timebase

The timebase accounts for the time it takes the signal to travel from the stimulation site to the recording site. It is usually set at 50 milliseconds for upper extremity SSEPs and 100 milliseconds for lower extremity SSEPs based on normal conduction time [5, 9]. This can be adjusted based on patient size, age, or conditions that affect conduction speed.

Generation of Potentials

Anatomy

Monitoring of SSEPs evaluates the functionality of the somatosensory pathway. Stimulation involves the peripheral nerves in the extremities and propagation of the signal is monitored throughout the pathway. First-order neurons in the somatosensory pathway have cell bodies in the dorsal root ganglion and axons that extend to the periphery. In addition to branching toward the periphery, these pseudo-unipolar neurons extend axons into the dorsal root entry zone and branch up and down through the ipsilateral dorsal column of the spinal cord. Some dorsal root ganglion fibers, instead of ascending the spinal cord, descend or synapse in the spinal cord gray matter. These branches are involved in reflexes, sensory modulation, and proprioception. Ascending fibers from the leg form the gracile fasciculus and fibers from the arm form the cuneate fasciculus. After ascending the spinal cord, these axons synapse with second-order neurons in the gracile and cuneate nuclei, respectively. From there, the axons of the second-order neurons decussate in the medulla as the internal arcuate fibers known as the sensory decussation. It is important to keep in mind that certain rare conditions can predispose patients to lack of decussation resulting in signals remaining ipsilateral, which should be screened for before monitoring [9, 14, 15]. After decussating, the second-order neuron fibers climb the brainstem in the medial lemniscus before synapsing with third-order neurons in the ventral posterolateral thalamic nuclei. Axons from the third-order neurons ascend in the posterior limb of the internal capsule before dispersing in the thalamocortical radiation to their respective areas of the primary sensory gyrus. Upper extremity fibers travel to the lateral convexity and lower extremity fibers go to the mesial parasagittal region. It is important to note that the stimulation technique is particular to the axons in the dorsal system and does not include anterolateral system axons due to differences in threshold and conduction [9].

Blood Supply of Somatosensory Pathway

Disruption in blood flow to various parts of the pathway can alter stimulus propagation. This allows for localization of lesions by observing which SSEPs are diminished. The somatosensory cortex is supplied by the anterior cerebral artery medially and by the middle cerebral artery laterally. This can help with localization of cerebral ischemia, since the tibial nerve pathway would be affected by anterior cerebral artery ischemia and the median nerve pathway by middle cerebral artery ischemia. Thalamocortical sensory axons are supplied by lenticulostriate branches of the

middle cerebral and anterior choroidal arteries. Branches of the posterior cerebral artery supply the thalamic sensory nuclei, while branches of the basilar and vertebral arteries supply the medial lemniscus. In the spine, the two posterior spinal arteries supply the dorsal column. The anterior spinal artery supplies the white matter and the remaining grey matter. It has anastomoses with the cervical, aortic, and iliac radicular arteries.

SSEP Waveforms

Recorded SSEPs are summated into waveforms that have a series of peaks and valleys. Nomenclature of the waveforms includes Ns and Ps which represent peaks and valleys respectively. N and P indicate the polarity of the waveform with N being negative (up) and P being positive (down). Different waves are of interest for different recording sites. When monitoring the sensorimotor cortex, the waveform peaks primarily analyzed are N20 and P20 (also referred to as P22). These waveforms originate from median nerve stimulation and are thought to be of thalamic and cortical origin. N20/P30 is recorded over the somatosensory cortex, while P20/N30 are of opposite polarity and recorded over the primary motor cortex. For the cervical spinal cord, Erb's potential (N9), P14, N18, and N20 are assessed. Erb's potential is a peripheral response recorded from Erb's point 2 cm above the mid-clavicle. P14 and N18 potentials are recorded from the centroparietal scalp ipsilateral to the stimulus and are most likely generated by subcortical structures including the caudal medulla, areas of the brainstem, and thalamus. Lastly, for thoracolumbar spinal cord monitoring, the popliteal fossa, P31, N34, and P37 are generally monitored. Recording in the popliteal fossa monitors peripheral responses, while P31, N34, and P37 are recorded from the forehead midline to monitor brainstem response. In each procedure the most readily apparent and robust waveforms should be used to analyze amplitude, morphology, and latency (Figs. 8.1, 8.2, and 8.3).

Stimulation and Recording Technique

SSEPs are used to check the functionality of the somatosensory pathway at various levels from the periphery to the cortex. The stimulation is applied to the distal portion of a peripheral nerve, most commonly the median or ulnar nerve at the wrist for upper extremity SSEPs and the posterior tibial nerve at the ankle or the peroneal nerve at the fibular head for the lower extremity. To monitor the sensorimotor cortex, the median nerve should be stimulated; however, the stimulated nerve should be contralateral to the portion of the cortex at risk. Alternatively, dermatomal SSEPs are elicited by stimulation to the skin of the dermatome at risk. Stimulation points

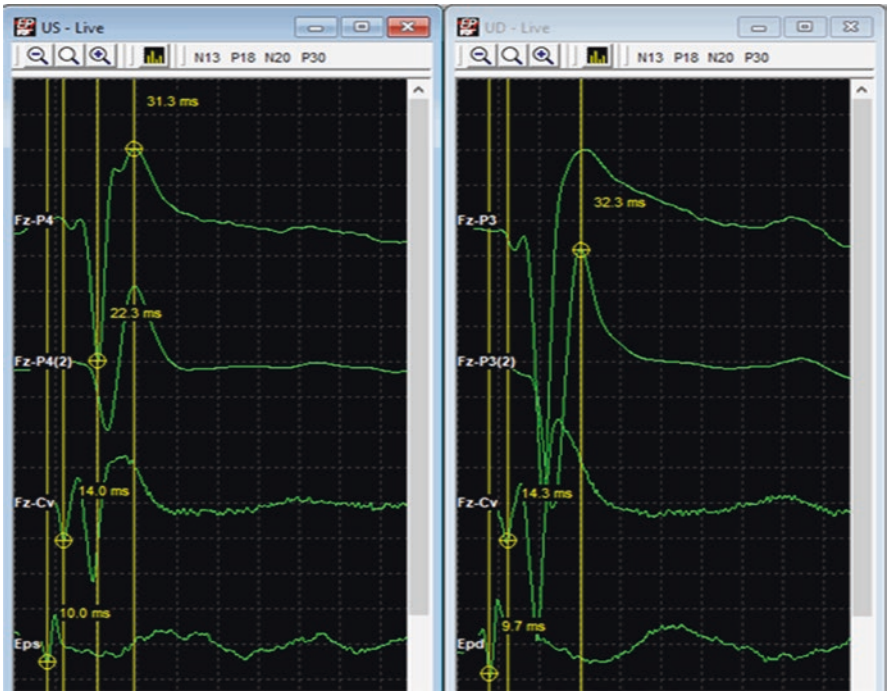

Fig. 8.1 Upper extremity SSEP data from ulnar nerve stimulation. In this dataset, the N9, N13, N20, and P30 waveforms are marked. Note that the active and reference electrodes are reversed to display N's as negative deflection waveforms, and P's as positive inflection waveforms. The Fz-P4 channel in the "US" waveform window shows an N20 with a latency of 22.3 ms and a P30 with a latency of 31.3 ms using a bandwidth of 10-250 Hz. The channel below uses the same two electrodes but has more aggressive filtering (30–100 Hz) which makes the N20 appear to have a longer latency and reduces some of the complexity in the P30. This channel is used primarily to allow for continuous monitoring during high-frequency noise. The Fz-Cv channel utilizes an electrode placed near the inion to record the N13 and P18 (if the inion isn't accessible the mastoid region can be used). Erb's point responses are collected from electrodes placed above the clavicle

are flexible and should be chosen based on ease of access to the site, easily identifiable landmarks, and ability to insert an electrode at the location. Typically for the upper extremities the default location is near the wrist, unless this site is unavailable. To stimulate the median nerve, the cathode of the pair of electrodes should be positioned between the palmaris longus and flexor carpi radialis tendons 2–4 centimeters proximal to the wrist [5, 11, 12]. The anode of the electrode pair should be placed 2–3 centimeters distal to the cathode to prevent an issue with action potential transmission known as anodal block. Anodal block is due to hyperpolarization of the axon under the anode leading to improper action potential initiation by the cathode if the two are in close proximity or the anode is proximal to the cathode. This placement guideline should be followed despite some evidence that anodal block

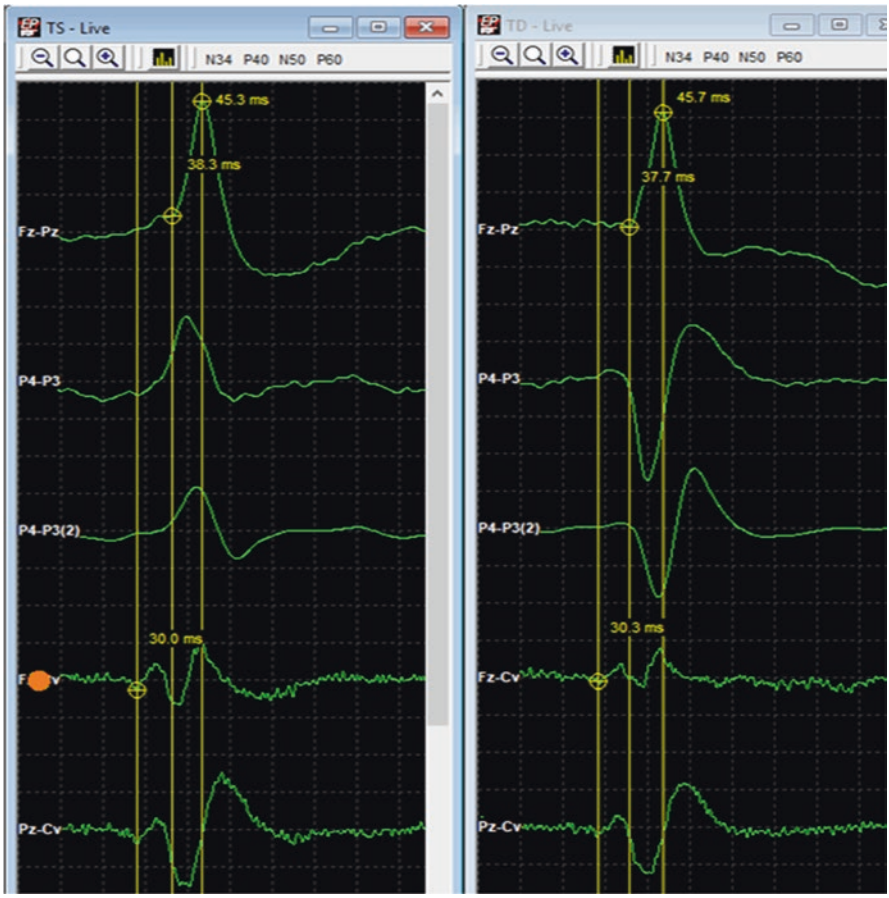

Fig. 8.2 Lower extremity SSEP data from posterior tibial nerve stimulation. In this dataset, the N30, N37, and P40 are marked. As with the upper extremity data, the active and reference electrodes are reversed to make N's negative and P's positive. The Fz-Pz channels show a downward pointing N37 followed by an upward pointing P40. The P4-P3 channel also shows the cortical P40 response but note that the left posterior tibial nerve P40 is upward pointing but the same channel in the right posterior tibial nerve waveform window shows a downward pointing waveform. This is by design and helps to identify left-right mix-up errors that can occur when plugging in the stimulating electrodes. The brainstem-generated responses are clearly present from the Fz-Cv and Pz-CV channels

will not play a significant role in stimulated action potentials [16]. The same setup with the cathode 2-4 cm proximal to the wrist and the anode distal to the cathode can be used for ulnar nerve stimulation, but on the medial side of the forearm next to the flexor carpi ulnaris muscle. The median nerve SSEP amplitude is usually larger than the ulnar nerve SSEP amplitude and is better for monitoring, but in some cases ulnar nerve SSEP monitoring can be more useful, especially cervical spine

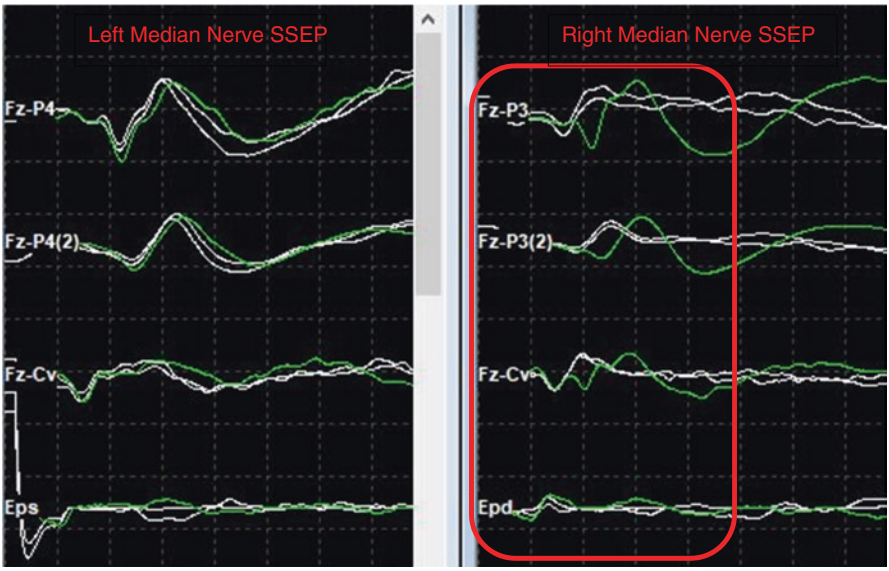

Fig. 8.3 Upper extremity SSEP data from a cardiac procedure showing the effects of a Left MCA occlusion (M1) on the cortically generated N20/P30 responses from the right median nerve. The baseline (green) shows data from the beginning of the procedure, and the two white traces show the data during the large vessel occlusion stroke. Note that the N9 (Erb's point) data remains unchanged, as does the brainstem generated N13. After the complete loss of the N20/P30, a far-field N13 can be seen from the Fz-P3 channels. This brainstem-generated response can mimic a cortical waveform in morphology, but marking the latency reveals that it is too early, and an ominous indicator of severe cortical dysfunction

surgeries depending on what cervical spine level is being operated on. Also, ulnar nerve SSEP monitoring is more sensitive in identifying position-related SSEP changes that can occur either from ulnar nerve compression around the elbow and/or stretch injury to the brachial plexus lower trunk from abnormal positioning. Placement of the cathode for posterior tibial nerve stimulation should be between the medial malleolus and the Achilles tendon with the anode 3 cm distal to the cathode. Alternatively, the peroneal nerve can be stimulated at the fibular head or the tibial nerve can be stimulated in the popliteal fossa. After stimulation, SSEPs are recorded from multiple locations along the sensory pathway. These locations can vary with both subcortical and cortical recording sites.

Certain stimulus parameters allow for accurate and consistent measurement. The stimuli should be constant-current rectangular pulses of 0.2–0.3 ms. [9] Supramaximal signal intensity is different for each patient and is estimated as 2 times motor or 3 times sensory threshold. Supramaximal intensity level is recommended for peripheral nerves, with the exception of proximal nerves at the knee, to avoid fluctuating stimulus to potential propagation and to avoid false alarms due to amplitude changes. In the unparalyzed patient, supramaximal intensity stimulation

at the knee may become problematic due to vigorous muscle contractions causing compartment syndrome in the lower leg, although more evidence is necessary to definitively identify SSEP as the cause [15, 17]. However, with neuromuscular blockade, the proximal supramaximal stimulation at knee does not pose any problem. Some evidence suggests this problem can be avoided by waiting to inflate the lower leg pressure cuffs until after the surgery has been completed [17]. Stimulus frequency also depends on the patient and the requirements of the procedure. Faster frequencies allow for more timely feedback but diminish cortical recording amplitude, potentially reducing effectiveness. The recommended optimal frequency is between 4.7 and 5.1 Hz, but ideally this should be adjusted to the individual procedure [13]. Importantly, frequencies that divide evenly into 50 or 60 can produce time-locked artifacts and should be strictly avoided. Interleaving, or alternating stimuli, between all four limbs can reduce the time for monitoring by 50%. Unfortunately, this time cannot be reduced further since there needs to be a decrease in frequency to accommodate all the stimuli; however, a benefit of the lower frequency is improved cortical recording amplitude.

Output of SSEP monitoring is in waveforms that represent the summation of multiple evoked potentials. The recommended number of trials to be averaged varies widely from 300 to 2000 trials, with most systems using 300–500 trials. As the number of trials increases, the time it takes to record increases much more than background noise is reduced. For instance, going from 300 to 1500 samples takes 5 times as long and only reduces the noise by a factor of 2.24. Noise reduction is equal to the square root of the factor of increased number of trials (in this case 5) [5]. Signal to noise ratio and the importance of a rapid alert to the surgeon for a particular surgery ultimately decide the number of trials that should be averaged.

Like stimulation, recording is also done with electrodes. Recording locations are variable, with options for both the upper and lower limb. For the upper limb, the peripheral recording electrode should be at the cubital fossa. The cortical recording is most commonly at the contralateral centroparietal with reference to the midline centroparietal (CPc-CPz) area. Other options include contralateral centroparietal with reference to ipsilateral centroparietal (CPc-CPi) or contralateral centroparietal with reference to the midline frontal area (CPc-Fz). For the lower limb, the peripheral recording electrode should be at the popliteal fossa. The primary cortical recording is midline centroparietal with reference to contralateral centroparietal (CPz-CPc). Other options include midline central with reference to contralateral centroparietal (Cz-CPc), midline parietal with reference to midline centroparietal (Pz-CPz), ipsilateral intermediate centroparietal with reference to contralateral centroparietal (iCPi-CPc), ipsilateral centroparietal with reference to contralateral centroparietal (Cpi-CPc), or midline central with reference to midline parietal (Cz-Pz). Patients with non-decussation should have the same peripheral recording sites and flipped ipsilateral and contralateral recording sites. For instance, the best cortical recording site from upper limb stimulation for a patient with non-decussation is ipsilateral centroparietal with reference to midline centroparietal (Cpi-CPz) [9]. Combining multiple pertinent recording sites into a montage in different channels may allow for more accurate and precise interpretation of the evoked potentials.

Warning Criteria

Classical warning criteria for SSEP monitoring is 50% reduction in peak amplitude and > 10% increase in latency from baseline [18, 19]. These criteria, developed in the 1970s, emphasized the impact of change in latency; however, we now consider amplitude change more diagnostic. Intraoperative pathologic processes mostly result in amplitude reduction while more chronic conditions such as demyelination have a greater effect on latency. Multiple studies corroborate the validity of these criteria [20, 21]. It is important to consider confounding factors when the warning threshold is reached. To account for other causes of amplitude or latency changes, peripheral recording sites or contralateral SSEPs should be used as controls. Factors such as anesthesia, hypothermia, edema, or changes in systemic blood flow may alter SSEPs without indicating the need for surgical intervention. Focal amplitude reductions can also result from nonsurgical causes like limb positioning. Despite not directly resulting from the surgery, the surgeon should still be informed in some cases so underlying issues may be addressed. Aside from confounding factors, other considerations need to be made for the warning criteria. Drift is the progressive change in amplitude over time and may affect the efficacy of the warning criteria. Downward drift to more than 50% of baseline amplitude can be noted without any obvious insult or pathology in up to 20% of scoliosis surgeries [22, 23]. Also, upward drift can reduce the sensitivity of the warning criteria because the amplitude fails to fall below 50% of baseline despite a noticeable decrease from the previous recording. In practice, drift should be accounted for and a less stringent reliance on the traditional warning criteria may allow for more accurate interpretation of the recording [9]. One recommendation for adaptive criterion is obvious reduction in amplitude from the previous values exceeding variability especially when sudden and focal. This recommendation has been demonstrated to reduce the likelihood of incorrect results compared to the traditional criteria. Additionally, the changes must be reproducible for an alarm to be given. This accounts for transient response variability and reduces unnecessary surgical interruptions.

Considerations

Anesthesia

Anesthetic agents and muscle relaxants are important to consider since SSEPs are being recorded intraoperatively. Anesthetic agents have the greatest effect on cortically generated responses and only minor effects on subcortical or peripheral responses. These effects are dose-related, vary by individual, and vary by type of anesthetic. Anesthetic lipid solubility and therefore potency correlate with the effect on SSEPs. These effects most commonly involve decreased amplitude and increased latency. Halogenated agents are particularly potent in the cortex, have some effect on the subcortical responses, and little effect on the peripheral responses. Of the

halogenated inhaled anesthetics, enflurane and halothane are least potent, while isoflurane is most potent. In procedures where cortical SSEP monitoring is critical, these agents should be restricted or avoided entirely. For subcortical and peripheral monitoring, low doses (< 0.5 MAC) could be viable as anesthetics. Propofol and opioid total intravenous anesthesia (TIVA) are the recommended anesthetics because of minimal SSEP depression and higher signal to noise ratio. Benzodiazepines, ketamine, or etomidate also may be acceptable alternatives. Unlike anesthetics, muscle relaxants can be beneficial for SSEP monitoring by reducing the interference from muscle groups near the recording electrodes. Additional relaxant can be useful in cases with excessive myogenic artifact [5].

Blood Pressure and Temperature

Blood pressure determines neural tissue perfusion. Decreased perfusion can result in decreased tissue functioning, affecting SSEPs. Cortical SSEPs begin to change when cerebral blood flow drops below 18 ml/100 g/min, resulting in amplitude decrease and latency increase. At cortical blood flow of less than 15 ml/100 g/min, ischemia results in a complete loss of cortical SSEPs [24–26]. This threshold is for tissue function and just above the perfusion threshold for permanent neurologic damage, serving as a warning sign for dangerously low blood pressure. The effect of blood pressure on SSEPs varies by individual; however, systolic pressure above 80 mm is generally sufficient for stable SSEPs [27]. Much of the variability is determined by the patient's normal blood pressure in the outpatient setting. Peripheral and subcortical SSEPs are less affected by changes in blood pressure. Body temperature can also affect SSEPs. Hypothermia tends to prolong latency and can even reduce scalp SSEP amplitude if severe [28].

Efficacy

The American Society of Neurophysiological monitoring positional statement on intraoperative monitoring of SSEPs to prevent postoperative neurologic deficits is strongly in favor, supported by Class II and III, strong Type A recommendation. SSEP works most effectively as a continuously run modality that captures data during nearly every minute of time spent in between surgical incision and closure. This intraoperative neuromonitoring modality (IONM) allows for timely therapeutic interventions to impending iatrogenic injury related to surgical maneuvers that cause time-dependent damage such as compression, traction, and ischemia. Some injuries, however, are irreversible and others may need more rapid identification and intervention than available. In these cases, SSEP still offers utility by helping the surgeon gauge the severity of the situation and take appropriate recourse. Additionally, preservation of SSEPs can serve as a surrogate for overall spinal cord integrity, but damage can occur in other regions without impacting the

somatosensory pathway. Damage to the ventral portion of the spinal cord may impact motor function without SSEP deterioration, so MEPs should be monitored simultaneously [3, 29, 30].

Limitations

As is true with any IONM modality, SSEP monitoring has limitations, the most notable of which is that it is a poor measure of nerve root integrity. This is due to the fact that multiple sensory nerve roots contribute to each of the peripheral nerves being stimulated to generate an SSEP. As mentioned, injury to the spinal cord may not impact the dorsal column, resulting in injury without changes in SSEPs. Alternatively, SSEP changes can occur after an irreversible injury resulting in postoperative neurologic deficits. Sometimes this occurs due to the nature of the injury and other times is due to a delay before the intervention. Also, SSEPs are less sensitive than MEPs at detecting spinal cord ischemia since white matter requires less blood flow than gray matter [31]. This can be useful for determining injury mechanism as abrupt SSEP deterioration is more likely a result of compression or another pathologic process rather than ischemia. Other monitoring techniques should be used in conjunction with SSEP monitoring when indicated to fully minimize the risk of an adverse event.

References

1. Larson SJ, Sances A, Christenson PC. Evoked somatosensory potentials in man. Arch Neurol. 1966;15(1):88–93. https://doi.org/10.1001/archneur.1966.00470130092010.
2. MacEwen GD, Bunnell WP, Sriram K. Acute neurological complications in the treatment of scoliosis. A report of the Scoliosis Research Society. J Bone Jt Surg - Ser A. 1975;57(3):404–8. https://doi.org/10.2106/00004623-197557030-00020.
3. Nuwer MR, Dawson EG, Carlson LG, Kanim LEA, Sherman JE. Somatosensory evoked potential spinal cord monitoring reduces neurologic deficits after scoliosis surgery: results of a large multicenter survey. Electroencephalogr Clin Neurophysiol Evoked Potentials. 1995. https://doi.org/10.1016/0013-4694(94)00235-D.
4. Coe JD, Arlet V, Donaldson W, et al. Complications in spinal fusion for adolescent idiopathic scoliosis in the new millennium. A report of the Scoliosis Research Society Morbidity and Mortality Committee. *Spine (Phila Pa 1976)*. 2006;31(3):345–9. https://doi.org/10.1097/01.brs.0000197188.76369.13.
5. ACNS. Guidline 11B: Recommended standards for intraoperative monitoring of somatosensory evoked potentials. Am Clin Neuophysiol Soc Guidel. 2009.
6. Guideline eleven: guidelines for intraoperative monitoring of sensory evoked potentials. J Clin Neurophysiol. 1994;11(1):77–87. https://doi.org/10.1097/00004691-199401000-00012.
7. Stecker MM, Patterson T, Netherton BL. Mechanisms of electrode induced injury. Part 1: theory. Neurodiagn J. 2006;46(4):315–42. https://doi.org/10.1080/1086508x.2006.11079592.
8. Netherton BL, Stecker MM, Patterson T. Mechanisms of electrode induced injury. Part 3: practical concepts and avoidance. Neurodiagnostic J. 2007;47

9. MacDonald DB, Dong C, Quatrale R, et al. Recommendations of the International Society of Intraoperative Neurophysiology for intraoperative somatosensory evoked potentials. Clin Neurophysiol. 2019;130(1):161–79. https://doi.org/10.1016/j.clinph.2018.10.008.
10. Patterson T, Stecker MM, Netherton BL. Mechanisms of electrode induced injury. Part 2: clinical experience. Neurodiagn J. 2007;47(2):93–113. https://doi.org/10.1080/1086508x.2007.11079617.
11. International organisation of societies for electrophysiological technology (OSET): guidelines for performing Eeg and evoked potential monitoring during surgery. Am J Electroneurodiagnostic Technol. 1999;39(4):257–77. https://doi.org/10.1080/1086508x.1999.11079270.
12. Celesia GG, Allison T, Bodis-Wollner I, et al. American electroencephalographic society guidelines for intraoperative monitoring of sensory evoked potentials. J Clin Neurophysiol. 1987;4(4):397–416. https://doi.org/10.1097/00004691-198710000-00005.
13. Nuwer MR, Packwood JW. Somatosensory evoked potential monitoring with scalp and cervical recording. Handb Clin Neurophysiol. 2008;8(C):180–9. https://doi.org/10.1016/S1567-4231(07)08011-2.
14. Vulliemoz S, Raineteau O, Jabaudon D. Reaching beyond the midline: why are human brains cross wired? Lancet Neurol. 2005;4(2):87–99. https://doi.org/10.1016/S1474-4422(05)00990-7.
15. MacDonald DB, Streletz LJ, Al-Zayed Z, Abdool S, Stigsby B. Intraoperative neurophysiologic discovery of uncrossed sensory and motor pathways in a patient with horizontal gaze palsy and scoliosis. Clin Neurophysiol. 2004;115(3):576–82. https://doi.org/10.1016/j.clinph.2003.10.029.
16. Wee AS, Leis AA, Kuhn AR, Gilbert RW. Anodal block: Can this occur during routine nerve conduction studies? Electromyogr Clin Neurophysiol. 2000;40(7):387–91.
17. Divani KG, O'Brien A, Molloy S, Trivedi J, Cowan J, Gibson A. A multicentre retrospective review of muscle necrosis of the leg following spinal surgery with motor evoked potential monitoring: a cause for concern? Eur Spine J. 2016;25(3):801–6. https://doi.org/10.1007/s00586-015-4063-2.
18. York DH, Chabot RJ, Gaines RW. Response variability of somatosensory evoked potentials during scoliosis surgery. Spine (Phila Pa 1976). 1987;12(9):864–76. https://doi.org/10.1097/00007632-198711000-00007.
19. Brown RH, Nash CL, Berilla JA, Amaddio MD. Cortical evoked potential monitoring: a system for intraoperative monitoring of spinal cord function. Spine (Phila Pa 1976). 1984;9(3):256–61. https://doi.org/10.1097/00007632-198404000-00006.
20. Wiedemayer H, Fauser B, Sandalcioglu IE, Schäfer H, Stolke D. The impact of neurophysiological intraoperative monitoring on surgical decisions: a critical analysis of 423 cases. J Neurosurg. 2002;96(2):255–62. https://doi.org/10.3171/jns.2002.96.2.0255.
21. More RC, Nuwer MR, Dawson EG. Cortical Evoked Potential Monitoring During Spinal Surgery. J Spinal Disord. 1988;1(1):75–80. https://doi.org/10.1097/00002517-198801000-00010.
22. MacDonald DB, Al Zayed Z, Al Saddigi A. Four-limb muscle motor evoked potential and optimized somatosensory evoked potential monitoring with decussation assessment: Results in 206 thoracolumbar spine surgeries. In: European Spine Journal. Vol 16. ; 2007. https://doi.org/10.1007/s00586-007-0426-7.
23. MacDonald DB, Al Zayed Z, Khoudeir I, Stigsby B. Monitoring scoliosis surgery with combined multiple pulse transcranial electric motor and cortical somatosensory-evoked potentials from the lower and upper extremities. Spine (Phila Pa 1976). 2003;28(2):194–203. https://doi.org/10.1097/00007632-200301150-00018.
24. Branston NM, Symon L. Cortical EP, blood flow and potassium changes in experimental ischaemia. In: Evoked Potentials. ; 1980:527–30. https://doi.org/10.1007/978-94-011-6645-4_61.
25. Nuwer MR. Intraoperative electroencephalography. J Clin Neurophysiol. 1993;10(4):437–444. https://doi.org/10.1097/00004691-199310000-00005.
26. Prior PF. Eeg monitoring and evoked potentials in brain ischaemia. Br J Anaesth. 1985;57(1):63–81. https://doi.org/10.1093/bja/57.1.63.

27. Cui H, Luk KDK, Hu Y. Effects of physiological parameters on intraoperative somatosensory-evoked potential monitoring: Results of a multifactor analysis. Med Sci Monit. 2009;15(5).
28. Markand ON, Warren C, Mallik GS, King RD, Brown JW, Mahomed Y. Effects of hypothermia on short latency somatosensory evoked potentials in humans. Electroencephalogr Clin Neurophysiol Evoked Potentials. 1990;77(6):416–24. https://doi.org/10.1016/0168-5597(90)90002-U.
29. Ben-David B, Haller G, Taylor P. Anterior spinal fusion complicated by paraplegia: A case report of a false-negative somatosensory-evoked potential. Spine (Phila Pa 1976). 1987;12(6). https://doi.org/10.1097/00007632-198707000-00005.
30. Minahan RE, Sepkuty JP, Lesser RP, Sponseller PD, Kostuik JP. Anterior spinal cord injury with preserved neurogenic "motor" evoked potentials. Clin Neurophysiol. 2001;112(8):1442–50. https://doi.org/10.1016/S1388-2457(01)00567-3.
31. Marcus ML, Heistad DD, Ehrhardt JC, Abboud FM. Regulation of total and regional spinal cord blood flow. Circ Res. 1977;41(1):128–34. https://doi.org/10.1161/01.RES.41.1.128.