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Evoked Potentials during Cardiac and Major Vascular Operations

Mark M. Stecker, MD, PhD

Somatosensory evoked potentials are widely used in spine surgery to prevent injury to the spinal cord. However, their application in cardiac and major vascular surgery is largely unappreciated. This paper will review the unique stresses placed on peripheral nerves, spinal cord, and brain during these operations. In addition, the potential benefits of peri-operative somatosensory evoked potentials monitoring are described in detail.

Over 200,000 coronary bypass operations (CABG) are performed each year in the United States yet even for this common surgical procedure, the 30-day risk of death remains 3.0%, with a 1.6% risk of stroke.^{1,2} Because of the continued risk of neurologic complications, many techniques have been used to evaluate the nervous system during cardiac operations with a goal of detecting and potentially treating nervous system injuries in real time.

In general, these neuromonitoring techniques can be divided into three categories. Transcranial Doppler quantification of cerebral arterial blood flow velocity, transcranial near-infrared spectroscopic assessment of cerebrocortical oxygen balance, and invasive measurement of jugular venous oxygen saturation fall into the first category. They provide indirect evidence of neurologic status by measuring basic substrates required for brain function and the metabolites produced by cellular activity. The techniques in the second category measure spontaneous activity generated in the nervous system such as the EEG. The third category consists of those methodologies such as evoked potentials that measure the response of the nervous system to external stimuli.

Although each of these techniques has advantages and limitations, the beneficial attributes of evoked potentials in comparison with other techniques include (1) simplicity in the identification and measurement of critical responses, (2) uncomplicated characterization of the effects of temperature and anesthesia on responses, and (3) selectivity in the functional assessment of subcortical structures and pathways. This latter point is especially relevant to the measurement of intra-operative peripheral nerve and spinal cord function.

This review will primarily deal with somatosensory evoked potentials (SSEP), because this modality is most commonly used during cardiac operations. It will first address some important technical issues in the recording of evoked responses during cardiac operations, including the effects of temperature and anesthesia. Subsequently, the role of SSEP monitoring in detecting peripheral nerve, brachial plexus, spinal cord, and brain injury will be discussed in both cardiac and aortic operations.

The two most commonly used types of SSEP recording in cardiac/aortic surgery involve the upper or lower extremity. Because the information that can be gained from these studies is highly dependent on the recording methodologies, their review is important prior to a discussion of the clinical applications.

Somatosensory Evoked Potential Methodology

In order to monitor upper extremity evoked responses, stimulating electrodes are typically placed over the surface of the skin on the medial side of the wrist over the ulnar nerve. This minimizes the interference with the placement of radial artery catheters. It also allows for monitoring of both the ulnar nerve and the lower brachial plexus, which are more likely to be injured than the median nerve and upper plexus. Subdermal needle electrodes may also be used, but are less than optimal because of additional risks of bleeding and burns.

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Lower extremity evoked potentials are most typically obtained after stimulation of the posterior tibial nerve at the medial malleolus. However, in cases where the possibility of ischemia to the legs is likely, it is also advantageous to place stimulators more centrally over the peroneal nerve at the fibular head. The responses to more central stimulation are somewhat less affected by peripheral ischemia and may allow differentiation between combined leg and cord ischemia and pure extremity ischemia during surgical procedures on the aorta.

Recording electrodes are generally placed to record peripheral, brainstem, and cortical potentials. These typically involve electrodes placed over peripheral locations such as the Erb point to record potentials at the brachial plexus, or the popliteal fossa to record peripheral potentials at the level of the knee. Electrodes placed either in a longitudinal or transverse orientation on the scalp are best used to record the cortical potentials, while subcortical potentials generated in the brainstem and the cervico-medullary junction are best recorded in a derivation in which a scalp electrode is referenced to a noncephalic locus.

Once appropriate stimulating and recording electrodes are placed, it is important to identify as many of the waveforms as possible (Table 1) and establish a reliable baseline (Figure 1). The acquisition settings used to acquire evoked potentials during cardiac operations are similar to those used in recording other intraoperative evoked potentials. The major exception is that the sweep duration should be roughly twice that used during procedures that do not involve hypothermia so that the prolonged latency of the potentials during the period of hypothermia may still be recorded.

Temperature Effects on SSEP

Unlike other surgical procedures, in cardiac operations there are often major changes in body temperature. It is well known that as temperature declines, the rate of conduction along an axon declines as well,^{9,10} and at temperatures of 2.7°C in unmyelinated and 7.2°C in myelinated axons,¹¹ complete conduction block occurs. In evoked potential studies, this implies that as the temperature is lowered the latency of the various potentials increases (Figure 2).¹² With small temperature changes, the amplitude of the waves does not

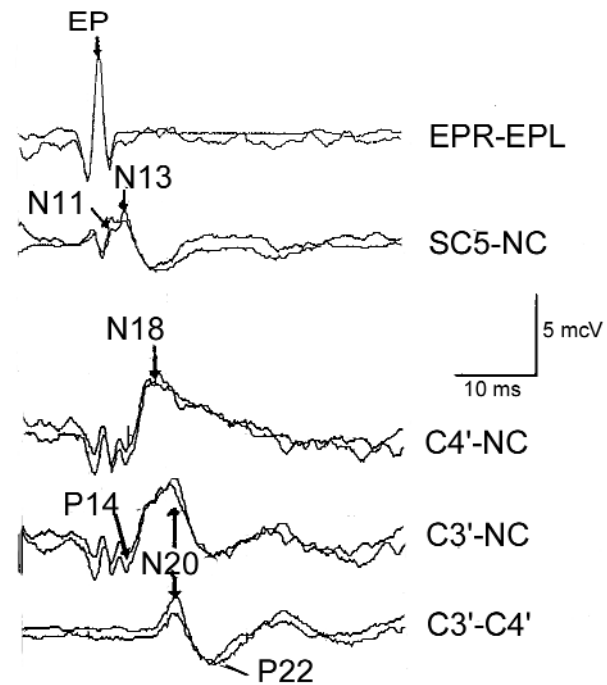


Figure 1. This figure illustrates normal somatosensory evoked potential (SSEP) waveforms produced by repetitive pulsatile electrical stimulation of the right median nerve at the wrist. The first (top) trace indicates the response obtained from electrodes placed on right (EPR) and left (EPL) supraclavicular Erb points. The resultant N9 Erb potential represents the passage of afferent volleys through the brachial plexus ipsilateral to the stimulation site. The second trace was obtained from electrodes placed over the fifth cervical vertebra and a noncephalic (NC) reference. The N11 and N13 components indicate afferent volley passage through the dorsal columns of the cervical spinal cord near the brainstem. Signals in the third trace were obtained from a NC reference and an active electrode located over sensory cortex (C4 is 2 cm posterior to the C4 electrode location in the standard international 10-20 system of electrode placement) ipsilateral to the stimulation site. The N18 component is thought to represent volley passage through the thalamic sensory relay nucleus. The N20 component in waveforms four and five, recorded contralateral to the side of stimulation, indicate afferent activation of sensory cortex.

change significantly, but at sufficiently low temperatures, the waves disappear (Figure 3). The N20 potential disappears at roughly 21.4°C and the N13 disappears at 17.3°C.¹³

A major difficulty in understanding temperature effects on the SSEP in individual patients is that the body is not in thermal equilibrium during cooling. As a result, significant differences in the tissue temperature often occur at different body locations.¹⁴ The thermal gradients depend on the

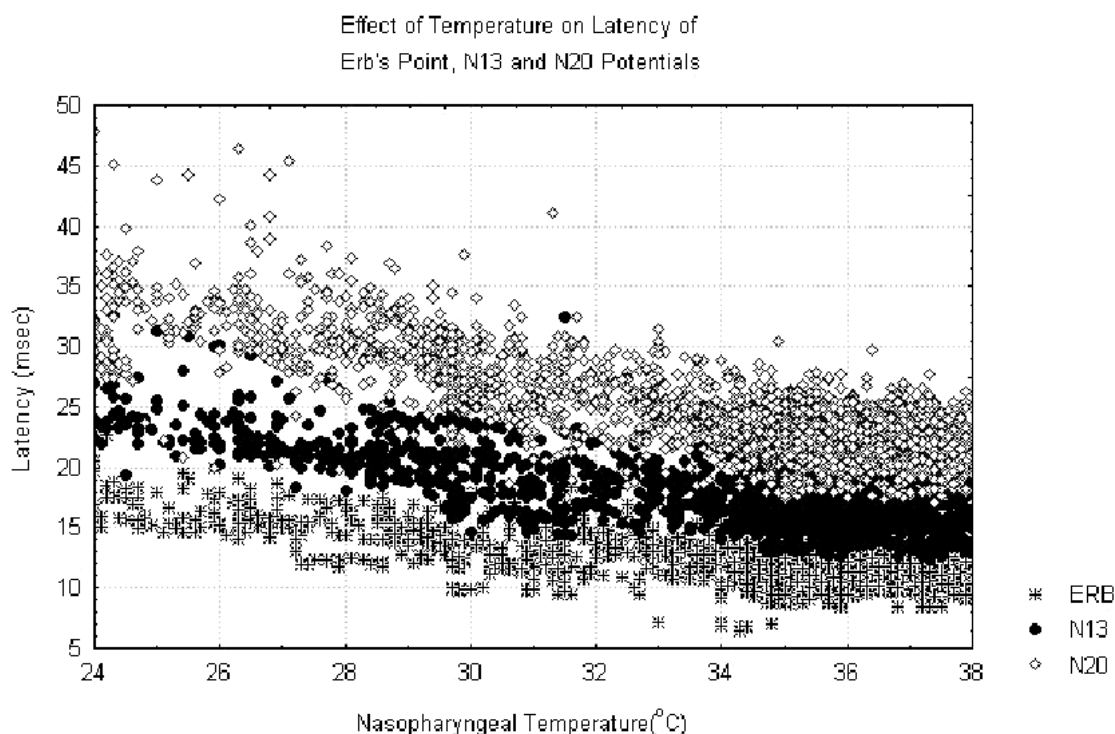


Figure 2. This figure shows the differential sensitivity to cooling of various components of the neuraxis. Note the relatively uniform and limited sensitivity of brachial plexus transmission (ERB) to deep cooling. In contrast, responsiveness in the cerebral cortex during cooling is more markedly and variably affected, as shown by the prolonged latencies.

rate of cooling, body habitus, and regional blood flow. In addition, the relationships between temperature and SSEP waveform component latencies and amplitudes demonstrate significant and variable hysteresis.^{15,16}

Thus, the latency of a particular SSEP component at a specified temperature may differ between cooling and re-warming phases. For example, the cortical N20 component disappears at 21.4°C during cooling, but reappears at 18.8°C during re-warming.¹⁶ On the other hand, the shorter latency brainstem N13 component reappears during re-warming at nearly the same temperature at which it disappeared during cooling.¹⁶

A third factor complicating the interpretation of evoked responses during hypothermia is the effect of temperature on the recovery of the excitability after stimulation. It is well known¹⁷ that the rate of change in the state of sodium channels that is required to generate an action potential is

reduced at low temperatures. Thus, the time during which an axon remains refractory after a stimulus will increase at cold temperatures. This is important because the evoked potential recordings typically cannot be distinguished from noise without averaging the responses to multiple stimuli presented at a regular interval.

Studying the effects of paired-pulse stimuli can provide important insight into the recovery of excitability after stimulation. As expected, the time to recover after stimulation is greater at low temperatures and for the longer latency multisynaptic cortical components such as the N20 and P22. In fact, with an interstimulus interval of 50 milliseconds, the N20-P22 amplitude ratio of the second to the response of the pair decreases tenfold from 2.0 at 37°C to 0.2 at 24°C.¹⁸ Practically, this suggests that the use of very low stimulus rates with interstimulus intervals of at least 100 milliseconds (rate < 10 Hz) may be helpful in eliciting clear responses at low temperatures.

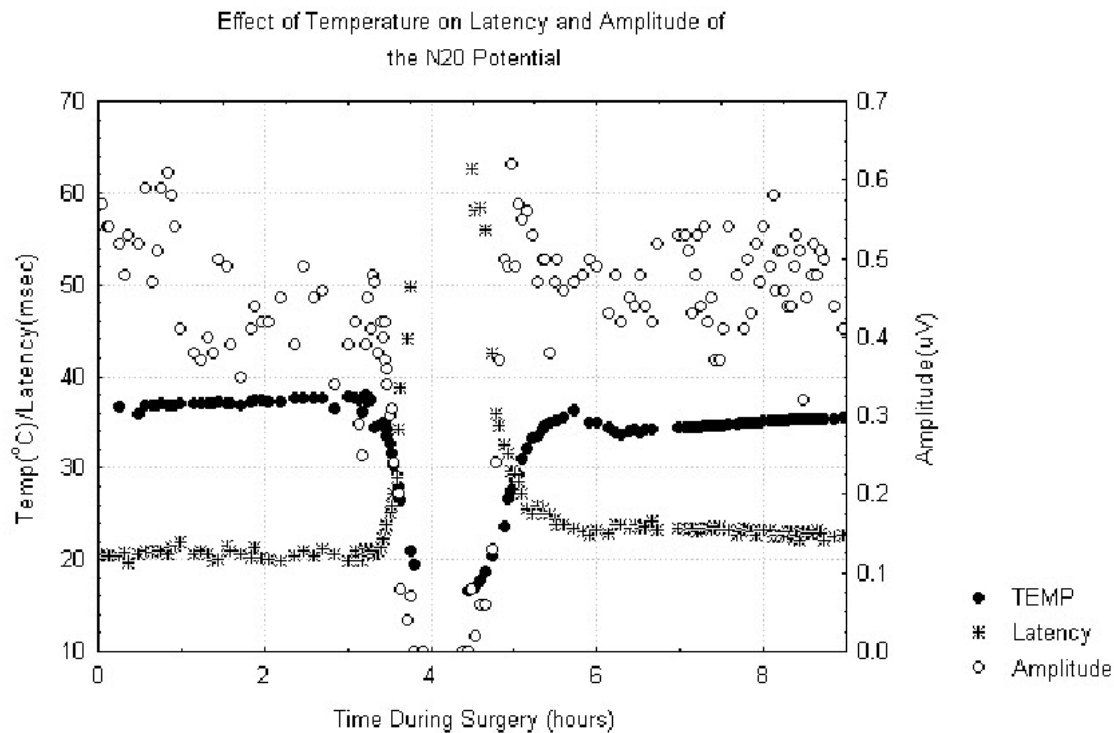


Figure 3. This figure describes the effect of cooling and rewarming on SSEP latency and amplitude. The marked variability in SSEP amplitude makes it an impractical method to assess the effect of cooling on cerebral function. In contrast, the stable latency measure seems ideal as a method to functionally characterize temperature effects on the brain.

Anesthesia Effects on SSEP

General anesthetic agents work at both the cortical and spinal levels,¹⁹ a fact that is clinically evident in the extreme sensitivity of motor-evoked responses that are elicited by transcranial electrical stimulation. Although anesthetic agents do have direct effects on axonal membranes,²⁰ much of their effect is exerted as synapses. Evoked responses that are separated from the stimulus by multiple synapses are thus much more sensitive to anesthetic effects than responses with shorter latency that are separated from the stimulus by fewer synapses (Table 1).

Anatomically, the impulses that form the SSEP travel along single axons from the point of stimulation through the posterior columns to its first synapse at the posterior column nuclei at the cervicomedullary junction. Thus, potentials generated distal to this point are relatively independent of anesthesia. Of the potentials typically recorded during cardiac and aortic operations, the N20–P22 responses after stimulation of the

upper extremity and the P40 cortical responses from the lower SSEPs are the most sensitive to the effects of anesthesia.

Since different anesthetic agents have distinct mechanisms of action, they also have unique effects on the SSEP. Particularly important is the fact that halogenated inhalational agents such as isoflurane have the greatest effects on the SSEP,^{21,23,24,26} although nitrous oxide²⁶ also exerts potent effects. Intravenous opioid analgesics and benzodiazepines amnestics have the least effects.²⁷ The hypnotics (propofol,²³ etomidate), the barbiturates,²⁵ and dissociative anesthetics (ketamine) produce much less SSEP suppression than the halogenated anesthetics and nitrous oxide. In addition to the inherently complex effects of anesthetic agents on the SSEP, these effects may also be strongly influenced by the temperature changes occurring during cardiac operations.²⁸⁻²⁹

Certain indicators can help to distinguish between SSEP alteration caused by ischemia and that caused by changes in anesthesia or temperature. First, temperature- and anesthesia-related

Table 1. Properties of Important SSEP Potentials

Potential	Stimulation		Latency (msec)	Origin ³	Anesthesia Sensitivity	Sensitive to Nerve Injury	Sensitive to	Sensitive to	Sensitive to	Ease of Recording
	Site	Montages					Spinal Cord Injury	Brainstem Injury	Cortical Injury	
Erb's	Wrist ^a	Erbs-Any ^b	10	Brachial Plexus	-	++	-	-	-	++
N13	Wrist	Fpz ^d -Cervical	13	Cervico-Medullary Junction ⁴	-	++	+ ^c	-	-	+
N18	Wrist	Ic ^e -Non Cephalic	18	Brainstem ^{5,6}	-	++	++	+	-	±
N20	Wrist	Cc-Ic ^f Or Fpz-Cc	20	Thalamus/ Cortex ^{3,7}	+	++	++	+	±	++
P22	Wrist	Cc-Ic Or Fpz-Cc	22	Cortex ^{3,7}	++	++	++	++	++	+
Popliteal	Ankle	Popliteal	10	Sciatic Nerve ⁸	-	+	-	-	-	+
N21	Ankle	Lumbar-Iliac Crest	21	Cauda Equina ⁸	-	+	-	-	-	-
N34	Ankle	Fpz-Cervical	34	Brainstem ⁸	±	++	++	±	-	+
P40	Ankle	Cc-Ic ^f Or Fpz-Cc	40	Cortex ⁸	+++	++	++	++	+	+

^aEither the median or ulnar nerve at the wrist or the superficial radial nerve at the wrist.

^bTypical montages include Left Erbs-Right Erbs or Erbs to a cervical or scale electrode.

^cThe N13 has components that arise out of the cervico-medullary junction as well as more distal structures in the cord thus high cervical injuries may not always cause large changes in the N13 (Maugierre).

^dFpz is an electrode position over the prefrontal area in the midline.

^eIc is an electrode over that part of the scalp over the somatosensory cortex ipsilateral to the stimulus (C3' or C4').

^fCc is an electrode over that part of the scalp over the somatosensory cortex contralateral to the stimulus (C4' or C3').

changes are typically bilateral, although they may be accentuated on the side of a previous injury or may vary with local variations in blood flow. Second, changes produced by anesthesia and temperature are often gradual,²⁴ while acute ischemic change is often rapid.

Again, measurement of the unconditioned and subsequent conditioned SSEP responses to paired stimulus patterns may aid interpretation. The different effects of falling temperature and increasing volatile anesthetic dose on the conditioned responses are separable through variation in the interstimulus interval.¹⁸ However, neurologic injuries may also result in reductions in the relative amplitude of the conditioned response.³⁰ It is hoped that studies of the SSEP paired responses to variable interstimulus intervals in humans may clarify the situation.

Ulnar Nerve Monitoring

Ulnar nerve injuries can be seen during of all types of surgery³¹⁻³³ including cardiac opera-

tions.³⁴ Stimulation of the ulnar nerve at the wrist produces impulses that traverse the critical segment of the ulnar nerve at the elbow prior to their arrival at the brachial plexus, so that injury of the ulnar nerve may produce changes in the latency or amplitude of the Erb point potential and following waves.³⁵ No studies have been performed using this technique to detect ulnar neuropathy during cardiac operations, although changes in the SSEP have been demonstrated to be strongly associated with clinical symptoms related to the ulnar nerve during positioning of awake patients.³⁶

One problem in using the SSEP to detect ulnar nerve injury is that with the standard montage as described, the first recorded wave is generated in the brachial plexus. Thus, it is impossible to distinguish ulnar nerve injury from plexopathy. Use of other electrode placements to record more distal potentials, including electrodes just above the elbow or near the axilla,³⁷ may provide helpful information in high risk patients.

Brachial Plexus Monitoring

Brachial plexopathies occur in up to 15% of patients undergoing cardiac operations and are especially prevalent in patients undergoing dissection of the internal mammary artery.³⁸⁻⁴¹ Because of the frequency of plexopathies, a number of studies have addressed the ability of SSEP monitoring to detect brachial plexus injuries.

Hickey et al used the amplitude of the cortical component of the upper extremity SSEP (N19-P22) to study 30 patients.⁴² They found that noteworthy changes occurred in waveform alteration during insertion of the central venous catheter (13%), Favolaro retractor application for internal mammary artery dissection (57%), and symmetrical sternal retraction (57%). All patients with absent (or >3 standard deviation amplitude reduction from baseline) potentials (20% of study population) at the end of surgery had a clinical brachial plexus injury. Patients who experienced transient changes in the evoked potentials, with only one exception, had no evidence of brachial plexopathy.

Jellish and colleagues^{43,44} recorded Erb point potential as well as a subcortical far-field potential (N13) and cortical N20 potentials in 44 patients who were undergoing elective myocardial revascularization, while trying to maintain constant temperature and anesthesia. They found that both Rultract and Pittman IMA retractors were frequently (85% and 65%, respectively) associated with greater than 50% decreases in the cortical SSEP amplitude. The authors did not comment on the subcortical responses, but their figures suggest that major changes did not occur in these potentials even when the cortical potentials did show changes.

In still another study, Seal and colleagues⁴⁵ found statistically significant changes in the SSEP in relation to the use of a sternal retractor, but not the Favolaro retractor. However, the SSEP changes were not reliably predictive of postoperative neurologic deficit. My personal experience with SSEP monitoring during cardiac operations mirrors that of Porkkala and colleagues,⁴⁶ who found no significant changes during sternal retractor placement.

The markedly different results have a number of potential explanations:

- First, significant differences may exist in surgical technique among the surgeons involved in these studies.

- Second, because of the slow time course of anesthesia related effects on the SSEP, progressive changes may be seen in the cortical evoked potentials even 20 to 30 minutes after a change in anesthetics. This effect could cause a neurophysiologist to falsely attribute changes in the SSEP caused by a prior change in anesthesia to a current surgical event. The complicating effects of anesthesia could be minimized by looking carefully at the Erb point potential, as well as subcortical potentials such as the N13, which clearly originate central to the brachial plexus.

- A third complicating factor is that the criteria for a significant change used in some of these studies may have been too liberal. If this were the case, there would be many false-positives and wide interstudy variation.

- Finally, inconsistencies in the technical production and measurement of the SSEP (especially the stimulus amplitude) also may represent an important source of variability.

Given the confusing evidence in the literature, the conservative approach to this problem may be the most appropriate until more information is available. Significant SSEP changes should only be attributed to a plexus injury if both subcortical responses generated central to the plexus and cortical responses show at least a 50% amplitude decrease and a 10% latency increase in the absence of other potential explanations.

This approach is supported by Lorenzini et al,⁴⁷ who recorded the SSEP during positioning of awake patients. Criteria for change in the SSEP were selected as 60% amplitude reduction or 10% latency increase. All evoked potential changes that met these criteria were associated with immediate clinical symptoms. However, 21% of patients had some symptoms such as tingling, numbness or aching that were not associated with any SSEP changes.

Spinal Cord Monitoring

The potentials that produce the SSEP after stimulation of either the ulnar or median nerves at the wrist are generated by the impulses traveling along the fastest conducting (ie, largest diameter) sensory axons. These axons traverse the brachial plexus before ascending in the posterior columns (fasciculus cuneatus) of the spinal cord to the posterior column nuclei. The N13 and all later waves will thus be sensitive to any injury to the posterior columns of the spinal cord.

Because the dorsal spinocerebellar tract⁴⁸⁻⁵¹ in the anterior part of the cord is also involved in conducting impulses that generate the SSEP, injury to the anterior cord may also produce changes in the SSEP, but to a smaller degree. In a like manner, the impulses producing the SSEP from the lower extremity also ascend in the posterior columns (fasciculus gracilis) and to a lesser extent the dorsal spinocerebellar tract, but follow a longer course throughout the spinal cord and so are also more sensitive to posterior than anterior spinal cord injury. This provides the anatomic substrate for the use of SSEP to monitor the spinal cord. The main utility of spinal cord SSEP monitoring during cardiac surgery is in operations that also involve the descending aorta. This specific application is discussed in detail in an accompanying article.

Brain Monitoring

Compared with other electrodiagnostic techniques used to monitor brain function during cardiac operations such as the EEG, the SSEP requires relatively little signal processing and interpretation is more straightforward. Despite this, surprisingly few studies have addressed the potential role of the SSEP as a monitor of brain function during cardiac operations.

Existing studies have, however, suggested at least three separate SSEP applications: (1) detection of cerebral ischemia during cardiopulmonary bypass, (2) determination of the appropriate temperature for hypothermic circulatory arrest, and (3) measurement of cerebral function during systemic circulatory arrest with supplementary cerebral perfusion.

Cerebral Ischemia Detection

Cheung et al⁵² demonstrated in a single case that the SSEP could be sensitive to the appearance of intraoperative cerebral ischemia (Figure 4). A subsequent study⁵³ of 25 patients undergoing cardiac operations demonstrated that SSEP monitoring was able to correctly identify 2 patients who experienced intraoperative stroke without any false-positive results.

These authors, recognizing that variations in temperature and anesthesia can produce sub-

stantial changes in the evoked responses, attempted to determine rational criteria for making the decision as to whether a given change in the SSEP indicates cerebral ischemia. They found that even in patients with normal preoperative and postoperative neurologic examinations, it was not uncommon to observe 20-fold changes in the amplitude of the N20-P22 complex during the course of cardiac operations.

In addition, the absolute latency difference between the right and left N20 and P22 potentials often varied by more than 4 milliseconds intraoperatively. Both of these are relatively larger than the typically used criteria of a 50% decrease in amplitude or a 10% change in latency. In fact, the only criterion which distinguished the group of patients that experienced an intraoperative stroke from the other patients was a greater than 21-fold change in the right/left N20-P22 amplitude ratio during surgery.

This study also noted that the variations in this right/left N20-P22 amplitude ratio could be strongly dependent on technical factors such as the stimulus intensity. The use of higher stimulus intensities tended to reduce the effects of any variations in the threshold of the peripheral nerve to stimulation during the surgical procedure and hence the variability. This emphasizes the fact that SSEP methodology may critically affect the clinical significance of waveform changes.

These small sample studies are suggestive of SSEP benefit, but larger patient populations must be examined in order to provide reliable estimates of the sensitivity and specificity for ischemia detection. When ischemia is identified by SSEP monitoring, a number of actions may be taken to prevent injury. These include blood pressure elevation, transesophageal echocardiographic determination of an embolic or occlusive source, and the administration of neuroprotectants. Although no prospective studies have addressed the utility of this approach, my experience suggests that SSEP monitoring is beneficial.

Indirect Monitoring of Brain Temperature

Guérit et al⁵⁴ recorded the N20 potential as well as the subcortical P14 potential in 32 patients undergoing deep hypothermic circulatory arrest. All patients in whom the disappearance of P14 (mean temp 16.9°C) was taken as a measure of adequate cooling prior to circulatory arrest suffered no neurologic sequelae. However, all pa-

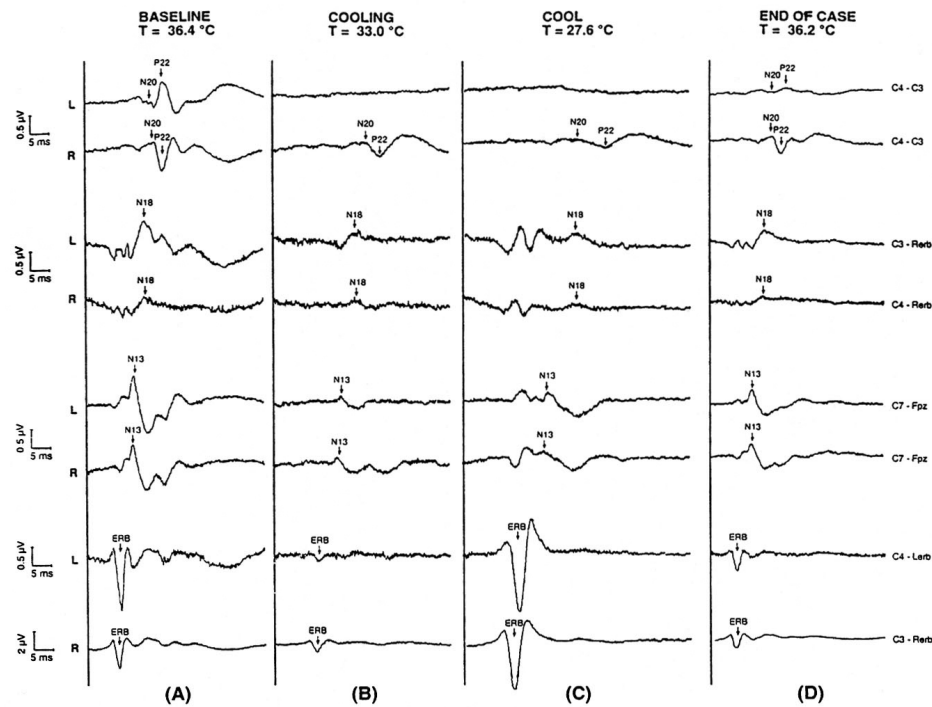


Figure 4. Vertical panel (A) represents the “normal” SSEP morphology observed during cardiac surgery. L and R refer to left and right ulnar nerve stimulation. In panel (B), generated after initiation of cardiopulmonary bypass, the N20-P22 complex arising from left arm stimulation has disappeared, whereas that arising from right arm stimulation remains intact. Latencies are prolonged as a result of cooling and note bilateral retention of the subcortical N18 and spinal N13 potentials. In panel C shows the continued loss of response to left arm stimulation and further suppression of the cerebral and spinal cord responses to additional cooling. Panel D shows only slight recovery of the response to left arm stimulation at the end of the case. Post-operative CT demonstrated an infarct in the posterior limb of the right internal capsule. (Figure reproduced with permission of the publisher⁵²).

tients in whom SSEPs disappeared because of a nontemperature-related cause showed demonstrated postoperative neurologic abnormalities.

On the basis of these observations, these authors suggested using the temperature of the disappearance of the P14 potential as the appropriate temperature for circulatory arrest. Other authors^{55,56} have suggested using the temperature at which electrocerebral silence appears on the EEG as the marker of the best temperature for hypothermic circulatory arrest. However, the temperature at which electrocerebral silence appears (17.8°C)¹³ is very close to the temperature at which the P14 disappears, and so either choice is probably appropriate.

Both Guérit,⁵⁴ Coles,⁵⁷ and later Stecker¹⁶ demonstrated that, in addition to its use as a “brain

thermometer”, the SSEP can also yield important information on the effects of circulatory arrest on brain function. In particular, each study demonstrated that during rewarming after a period of circulatory arrest, the time to recovery of the N20 potential increased by 0.3 to 1.0 minutes for every minute of circulatory arrest. Stecker¹⁶ further demonstrated that the risk of neurologic deficit after circulatory arrest (with retrograde cerebral perfusion) is dependent upon the temperature at which the N20–P22 complex returns during rewarming after circulatory arrest. Specifically, the risk of neurologic deficit increases by a factor of 1.27 for every degree increase in the nasopharyngeal temperature at which the N20 potential returns during rewarming. This effect was independent of the effects of the temperature at which continuous EEG returns dur-

ing rewarming and so indicates that the SSEP provides information in addition to that provided by the EEG, as suggested by Guérit.⁵⁴

SSEP monitoring has a role not only in determining optimal hypothermic circulatory arrest temperature and the cerebral response to rewarming, but also in characterizing brain function during hypothermic circulatory arrest. Cheung et al,⁵⁸ using electrocerebral silence as the criterion for hypothermic circulatory arrest, demonstrated that subcortical SSEP waveforms often persisted during hypothermic circulatory arrest. Although present, the key SSEP waveform components gradually declined in amplitude over time. The thalamocortical N18 potential amplitude halved within 16 minutes, while similar decreases in the brainstem N13 and brachial plexus N11 components required 19 and 30 minutes, respectively. More important, the rate at which the N18 declined was dependent on the fraction of time that retrograde cerebral perfusion was used during circulatory arrest. Greater use of retrograde cerebral perfusion was associated with a longer time to disappearance of the N18. In that study,⁵⁸ the N18 persisted almost twice as long when retrograde cerebral perfusion was used throughout the period of hypothermic circulatory arrest as when retrograde cerebral perfusion was not used at all. Further demonstration of the significance of these changes is found in the observation that the time course of N18 amplitude reduction mirrored the temporal changes in oxygen extraction during circulatory arrest with retrograde cerebral perfusion.⁵⁹

Finally, it should be noted that the SSEP may also be very helpful in other ways during complex cardiac and aortic operations. In particular, the SSEP can aid in determining optimal circulation management. This is of particular interest during aortic operations. The SSEP may, for example, be used to detect the inadvertent placement of a clamp that eliminates flow to either the right or left carotid artery. Another example is that SSEP monitoring during aortic dissection repair can quickly detect malperfusion through the false lumen that compromises the cerebral circulation.

Conclusion

SSEP monitoring during cardiac and major vascular surgery provides vital information about the functional status of peripheral nerve, brachial

plexus, spinal cord, and brain. This technique can detect cerebral ischemia or stroke and provides an indirect functional measure of temperature effects on the central nervous system. Additionally, it appears to offer new insight into the dynamics of deep hypothermic circulatory arrest and supplementary cerebral perfusion. However, evidenced-based medicine demands a series of appropriately controlled and adequately powered prospective studies to objectively determine the cost-benefit of this monitoring modality.

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