

Intraoperative Neurophysiologic Monitoring

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ANESTHESIA CONSIDERATIONS

Establishing intraoperative neurophysiologic protocols must involve close cooperation with the anesthesiologist. Anesthesia optimized to facilitate neurophysiologic monitoring will, nevertheless, produce drug effects that alter sensory and motor evoked responses that must be appreciated and appropriately interpreted by the monitoring neurophysiologist. Although some generalizations exist about anesthesia drug effects, the relative potency and specific location of drug actions differ between agents, so that some discussion of each agent is necessary to better understand the alternatives and their implications.

Some of the differences between anesthetic agents relate to the anatomic site of the anesthetic effect.³²⁹ Other differences relate to the neurophysiologic site of drug action. The major target for anesthetic action appears to be at the gamma aminobutyric acid (GABA) and *N*-methyl-*D*-aspartate (NMDA) receptors mediating electrolyte channels (Na⁺, Cl⁻, CA²⁺) at central nervous system synapses.

In the sensory system, the response generated by synapses in cortical structures will be the most affected, with less effect occurring at more peripheral structures, where fewer synapses are involved.³⁷¹ Because the most prominent anesthetic effects for the sensory system is on the synaptic-rich cortically generated responses, it is not surprising that anesthetic effects on cortical evoked potentials parallel the drug effects described for the EEG, which is also a cortical synaptically mediated response (Fig. 12-2).⁴²⁸ A schema is proposed for anesthesia effects on cortical sensory evoked potentials (Fig. 12-3).⁴²⁷ Unfortunately, most commonly used anesthetic drugs today produce a dose-related depression of the recordable EEG, as well as decreased

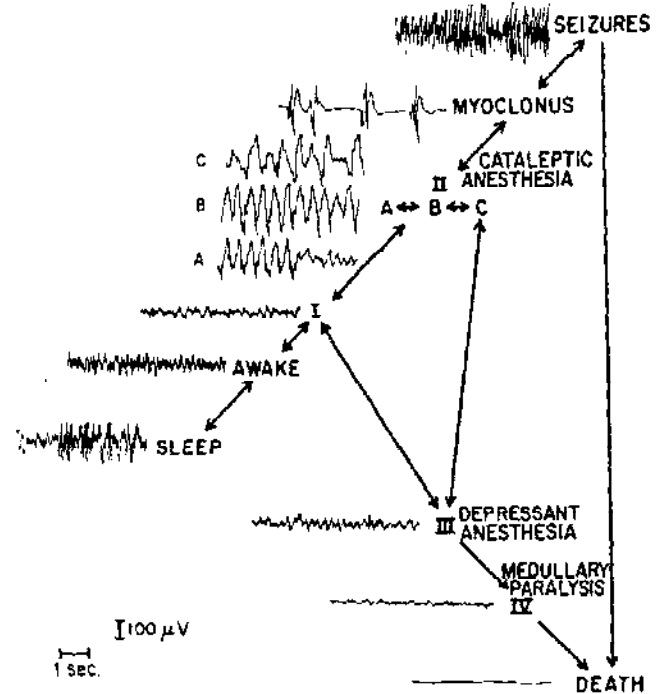


Figure 12-2. Cortical EEG stages typical of anesthesia. (From Winters WD: Effects of drugs on the electrical activity of the brain: Anesthetics. *Ann Rev Pharm Toxicol* 16:413-426, 1976, with permission.)

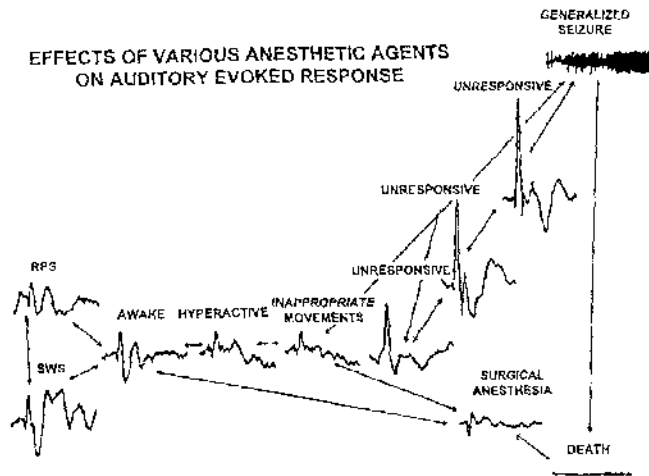


Figure 12-3. Cortical evoked potentials stages typical of anesthesia. (From Winters WD, Mori K, Spooner CE, Bauer RO: The neurophysiology of anesthesia. *Anesthesiology* 1967;28:65, with permission.)

amplitude and increased latency of cortical SEP and myogenic motor evoked potential (MEP), making the anesthetic choice for monitoring with these electrophysiologic modalities particularly challenging.³⁷²

Based on the major effect of anesthetic drugs occurring at the synapses, three locations in the motor pathways will be the most susceptible to anesthesia. The first location is within the motor cortex where internuncial neurons and synapses participate in activation of the motor cortex by transcranial stimuli. When electrical or magnetic pulses activate pyramidal cells, they produce a direct activation of the cells producing a “D” wave and activation via the internuncial pathways (dependent upon synapses) producing a series of I waves (Fig. 12-4). Weaker magnetic impulses appear to depend on synaptic activation for production of a response. The implication of anesthetic effects on these internuncial synapses is that the production of D waves will be relatively immune to anesthetic effects whereas the production of I waves will be reduced with anesthetic agents that depress synaptic function. Of further consideration is that synaptic function may be a delicate balance of inhibitory and excitatory influences. The second major sites of anesthetic action in the motor system are the synapses at the anterior horn cell. At this location, the summated D and I waves bring the anterior horn cell to threshold with a resulting peripheral nerve action potential leading to a muscle response. Anesthetics at this

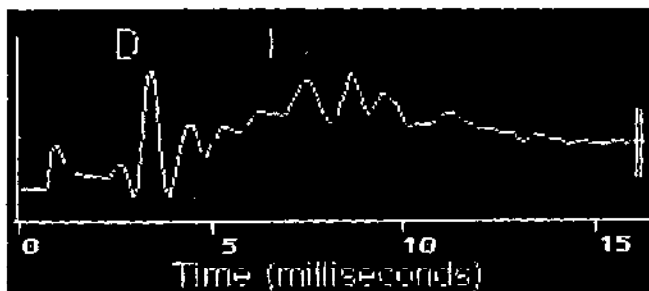


Figure 12-4. D wave and I waves with MEP stimulation.

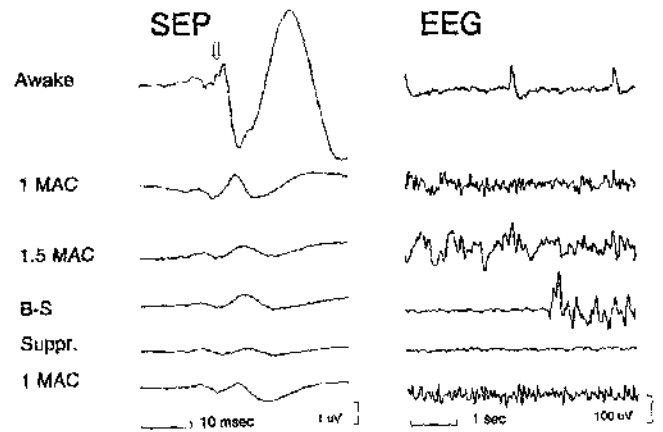


Figure 12-5. Cortical SEP and EEG recorded at various doses of isoflurane. (From Porkkala T, Jantti V, Kaukinen S, Hakkinen V: Somatosensory evoked potentials during isoflurane anaesthesia. *Acta Anaesthesiol Scand* 1994;38:206–210, with permission.)

site may have one of two effects. First, partial synaptic blockade may compound a loss of I waves, making it more difficult to bring the anterior horn cell to threshold. At higher doses, synaptic blockade may inhibit synaptic transmission at this site regardless of the composition of the descending spinal cord volley of activity. The third major synaptic location for anesthetic effects in the motor pathway is at the neuromuscular junction. Fortunately, with the exception of neuromuscular blocking agents and drugs, which alter acetylcholine transmission, anesthetic drugs have little effect at the neuromuscular junction. Similarly, neuromuscular blocking agents have little effect on central nervous system synaptic transmission and axonal conduction in motor pathways other than at the neuromuscular junction.

Finally, it should be noted that anesthetic drugs may have an effect on evoked responses indirectly by altering other physiologic factors that influence the provision of nutrient supply to the neural tracts. This is discussed in the sections that follow.

INHALATIONAL AGENTS

Halogenated Inhalational Agents

Perhaps the most common anesthetics in use today are the **halogenated inhalational agents** (desflurane, enflurane, halothane, isoflurane, sevoflurane). Paralleling their effects on the EEG, all halogenated inhalational agents produce a dose-related increase in latency and reduction in the amplitude of the cortically recorded evoked potential responses (SEP, VEP, BAEP). Although the effects of halogenated inhalational agents appear to be dose related, the changes observed in some studies appear to plateau at low concentrations (0.5–1% inspired concentration).³³⁹ Figure 12-5 shows the effects of isoflurane on the EEG and on the cortical SEP, demonstrating a parallel effect.³¹⁵

Studies support differences in the potency of the halogenated inhalational agents on the cortical SEP. The relative order seen is isoflurane (most potent), enflurane, and halothane (least potent).³⁷¹ Studies with sevoflurane and desflurane suggest that they are similar to isoflurane at steady state, but owing to their more rapid onset and offset of effect (because of their relative insolubility), they may appear to be more potent during periods when concentrations are increasing.

EFFECT OF ISOFLURANE ON BAEP

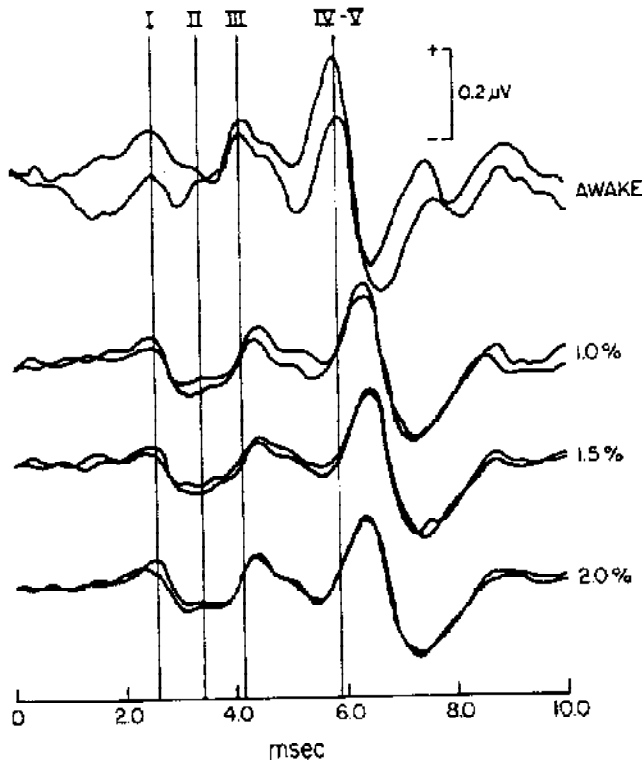


Figure 12-6. Influence of isoflurane alone on BAEP. Latency of peaks III and IV-V increased at 1.0% but plateaued with increasing anesthetic depth. (From Manninen PH, Lam AM, Nicholas JF: The effects of isoflurane and isoflurane-nitrous oxide anesthesia on brainstem auditory evoked potentials in humans. *Anesth Analg* 1985; 64:43, with permission.)

The most prominent effect of halogenated inhalational agents is on cortical responses, with markedly less effect on subcortical structures. Studies of recordings at Erb's point (over the brachial plexus) and over the cervical spine show minimal changes (0-9%), which are not dose related. As a subcortical response, the BAEP is minimally affected by halogenated inhalational agents. The more prominent latency changes occur in wave V, with III being less affected and wave I being little affected, having amplitude changes that are minimal (Fig. 12-6).²³⁸

MEPs recorded in muscle (myogenic) are the most easily abolished by halogenated inhalational agents. Single pulse stimulation transcranial motor evoked myogenic potentials (tcMEP) appear to be so easily abolished by inhalational agents that they are often unrecordable in the presence of these agents. When recordable, the major effect may occur at low concentrations (e.g., less than 0.2-0.5% isoflurane) (Fig. 12-7).^{129,171,442} This effect is likely a result of the combination of the anterior horn cell synapse depression as well as loss of I waves due to anesthetic effects on the internuncial synapses.¹⁴⁷ Changes in the H-reflex confirm an effect of halogenated inhalational agents at the spinal level.²¹⁶

In contrast to myogenic responses, the D response seen in the epidural space is highly resistant to the effects of these agents and is easily recordable at high volatile anesthetic concentrations¹²⁷ and can be used for monitoring (Fig. 12-8). It has been

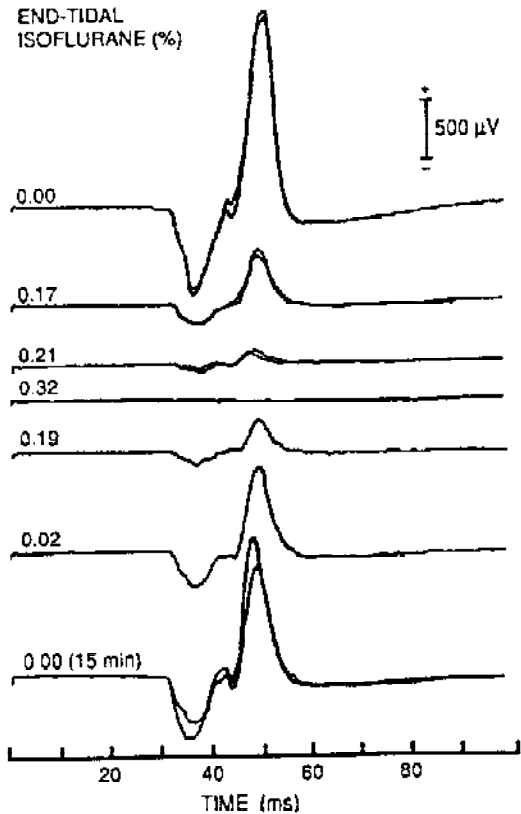


Figure 12-7. Motor evoked responses to transcranial electrical stimulation during nitrous oxide/sufentanil anesthesia before, during, and after administration of isoflurane (0.3% end-tidal). (From Kalkman CJ, Drummond JC, Ribberink AA: Low concentrations of isoflurane abolish motor evoked responses to transcranial electrical stimulation during nitrous oxide/opioid anesthesia in humans. *Anesth Analg* 1991;73:410, with permission.)

suggested that the most prominent anesthetic effect on the MEP is at the anterior horn cell level. However, the loss of I waves from a cortical effect may be sufficient to block myogenic responses, even without significant anesthetic effects at the anterior horn cell. This is because a series of I waves appear to be

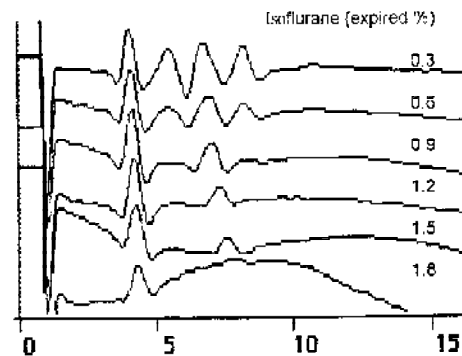


Figure 12-8. Effect of isoflurane on epidural recordings following transcranial electrical stimulation of the motor pathways. The first wave (D wave) remains intact as the concentration of isoflurane is increased, but there is a progressive loss of I waves.

necessary for producing myogenic responses in the unanesthetized state.

Studies comparing **transcranial magnetic motor evoked potentials (tcMMEP)**, using an externally applied magnetic field, and **transcranial electrical motor evoked potentials (tcEMEP)**, using an electrical voltage applied across the cranium, suggest that the magnetic technique can be more sensitive to the inhalational agents,³⁶⁷ probably because magnetic stimulation relies more on transsynaptic activation. High magnetic strength tcMMEP (which can produce D waves) appears to minimize this cortical difference.

Because the D wave is resistant to anesthetic depression, the anesthetic effect at the anterior horn cell can be partially overcome at low concentrations by high-frequency (multiple-pulse) transcranial stimulation.³⁷⁰ In this circumstance, the multiple D waves formed (and I waves, if produced) summate at the anterior horn cell resulting in a peripheral nerve activation and subsequent motor response (Fig. 12-9). Low concentrations of inhalational agents appear acceptable when high-frequency transcranial stimulation is used (trains of stimuli with interstimulus interval [ISI] of 2–5 milliseconds).^{177,309,370} As predicted, higher concentrations of these agents eliminate myogenic responses from this stimulation. Clinical experience suggests that anesthetic plans avoiding the inhalational agents may still be desirable for optimal MEP monitoring, even with the high-frequency stimulation technique.³⁰⁹

Studies with direct spinal or epidural stimulation show minimal effects of anesthesia on neurogenic or myogenic responses.³⁰⁹ However, the above described effects at the anterior horn cell suggest that depression may change the mixture of orthodromic motor and antidromic sensory contributions to the recorded responses. A study of the responses in the peripheral nerve and muscle following epidural stimulation in the cat revealed that single-pulse stimulation produced a response that was eliminated by pentobarbital, low-dose isoflurane, or by posterior column transection (but not lateral column transection).⁷⁵⁴ This suggests the response recorded from the peripheral nerve was largely mediated by sensory pathways, especially those of the posterior column. When a pair of stimuli were used (interstimulus interval 1–5 milliseconds), a new complex in the peripheral nerve response was seen. This complex and the compound muscle action potential (CMAP) were eliminated only by high-dose isoflurane or lateral spinal cord transection (lysing the descending motor pathways). Therefore, the type of spinal cord stimulation and the anesthetic agents used may alter the balance of sensory and motor contributions to the peripheral nerve and muscle response from direct spinal stimulation. Recent studies suggest that with isoflurane anesthesia, the motor component is preferentially blocked, perhaps by interaction at the synapses in the anterior horn cell or by differential effects on conduction in the spinal tracts in humans.⁷¹ These studies do not, however, clearly allow a recommendation of anesthesia that will preferentially promote monitoring of motor pathways with direct spinal, epidural, or paraspinal stimulation.

Nitrous Oxide

Nitrous oxide produces SEP cortical amplitude reductions and latency increases when used alone or when combined with halogenated inhalational agents or opioid agents (Fig. 12-10). Studies of nitrous oxide in a hyperbaric chamber confirm the depressant nature of its effect at higher doses as well.³³⁴ When compared at equipotent anesthetic concentrations, nitrous oxide produces more profound changes in cortical SEP and muscle recordings from transcranial motor stimulation than any other

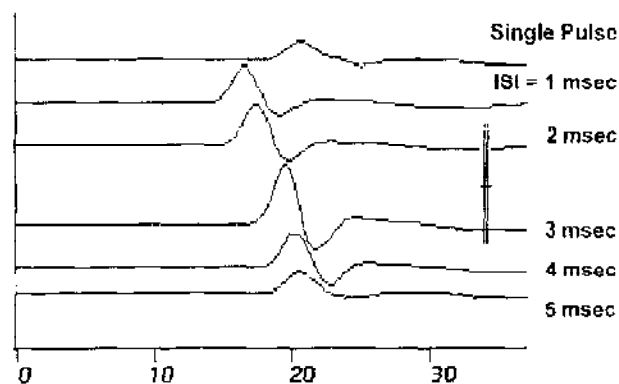


Figure 12-9. Effect of multiple pulse transcranial stimulation on compound muscle action potentials (CMAPs) during 0.9% isoflurane. The amplitude of the CMAP increases as a second stimulation pulse is added, with a maximum effect in this study when the interstimulus interval (ISI) is 3 milliseconds.

inhalational anesthetic agent.^{161,359,371} Like halogenated agents, effects on subcortical and peripheral sensory responses and on epidurally recorded MEP are minimal.

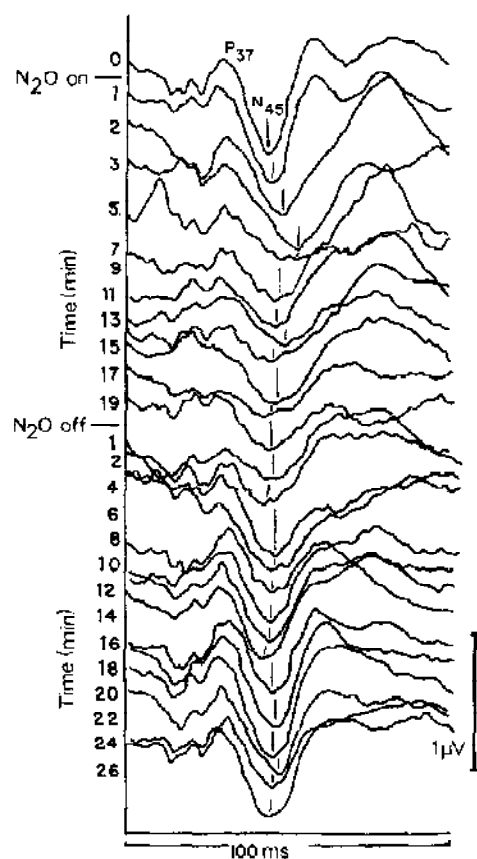


Figure 12-10. Effect of nitrous oxide on cortical recordings of posterior tibial nerve somatosensory evoked potentials. The amplitude of the response is markedly reduced over the 10–15 minutes following the introduction of nitrous oxide, and a response returns after agent is removed. (From Sloan TB, Koht A: Depression of cortical somatosensory evoked potentials by nitrous oxide. *Br J Anaesth* 1985; 57:850, with permission.)

Despite the depressant effect of nitrous oxide, it has been used with recording of responses, particularly when combined with opioids ("nitrous-narcotic" anesthetic technique). When combined with other agents, nitrous oxide may be "context-sensitive" in its effects, similar to its effects on the EEG (i.e., the actual effect may vary depending on the other anesthetics already present).^{249,269}

As with sevoflurane and desflurane, nitrous oxide is relatively insoluble. Therefore, anesthetic effects can change rapidly when concentrations are varied intraoperatively. Since a decrease in concentration will be associated with a rapid increase in amplitude and decrease in latency, it may "mask" amplitude and latency changes that may be occurring from concurrent neural compromise. Therefore, such changes should be avoided during critical portions of the surgery when the monitored structures may be at higher risk.

Also, nitrous oxide can increase middle ear pressure and hearing threshold, thereby presenting the possibility for disproportionate effects on BAEP and cortical auditory evoked potential (AEP) responses when eustachian tube dysfunction occurs. This could result in false-positive monitoring deficits occurring in the BAEP. Therefore, avoidance of an increase in nitrous oxide during critical portions of surgery requiring BAEP monitoring is also important. Changes in such anesthetic agent concentrations should be relayed from the anesthesiologist to the clinical neurophysiologist because they are required to help with correlating electrophysiologic monitoring changes to the operative environment.

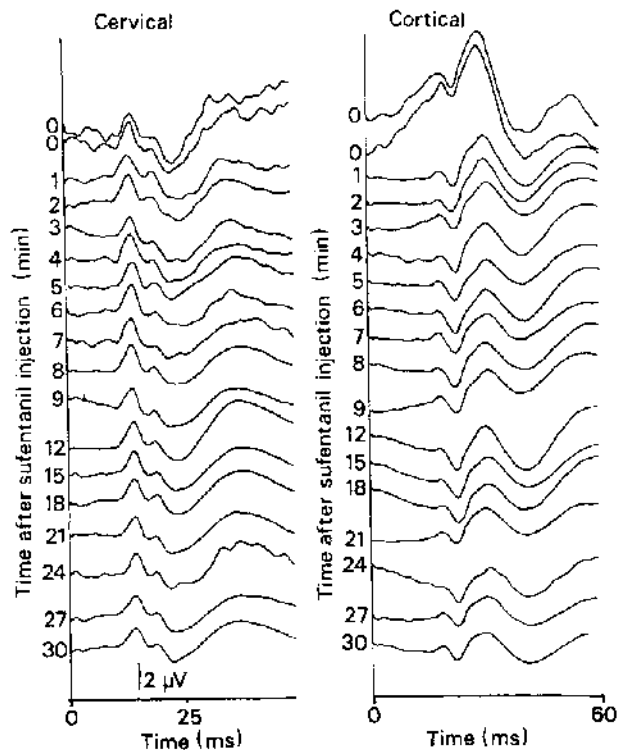


Figure 12-11. Changes in median nerve cervical and cortical SEP recording with time in one patient after sufentanil 5 g/kg. Two baseline recordings at time zero are shown. (From Kimovec MA, Koht A, Sloan TB: Effects of sufentanil on median nerve somatosensory evoked potentials. *Br J Anaesth* 1990;65:169, with permission.)

INTRAVENOUS ANALGESIC AGENTS

Most anesthesia techniques utilize a mixture of different anesthetic agents such as supplementation with inhalational agents (halogenated agent or nitrous oxide) with opioids or intravenous sedatives (e.g., benzodiazepines, etomidate, droperidol, or propofol). If the inhalational agents need to be completely avoided, intravenous agents can be combined to produce a **total intravenous anesthetic (TIVA)**.

Opioid Agents

The effects of **opioid analgesics** (fentanyl, sufentanil, alfentanil, remifentanyl) on sensory and motor evoked responses are less adverse than inhalational agents, making them important components of anesthetic planning for monitoring evoked responses.³⁰⁶ Effects are similar for most evoked sensory modalities. Minimal changes in spinal or subcortical recordings are noted with some amplitude depression and latency increases in cortical responses, especially loss of late cortical peaks (over 100 ms) at doses sufficient to produce sedation (Fig. 12-11).¹⁸⁴ As with systemic opioids, the spinal application of morphine or fentanyl for postoperative pain management produces minimal changes in the SLP and fails to alter the H-reflex.³⁴⁵

Opioid-based anesthesia is frequently used when cortical SEP responses and transcranial motor evoked potentials are monitored.³⁰⁵ Studies with myogenic responses from tcMEP with electrical and magnetic methods show only mild amplitude decreases and latency increases that permit good recording.^{112,212} With respect to the latter, fentanyl has been suggested to be useful in reducing background spontaneous muscle contractions and associated motor unit potentials, which may further improve muscle recordings. Since the opioids do not guarantee sedation or amnesia, opioid-based anesthesia must include an additional sedative agent to produce TIVA.

Ketamine

The effects of ketamine on the evoked responses also differ from those of inhalational agents. Ketamine can produce central nervous system excitement with associated enhancement of cortical sensory and myogenic responses.³⁶⁹ Thus, an increase in cortical SEP amplitude³⁴⁵ and an increase in amplitude of muscle and spinal recorded responses following spinal stimulation

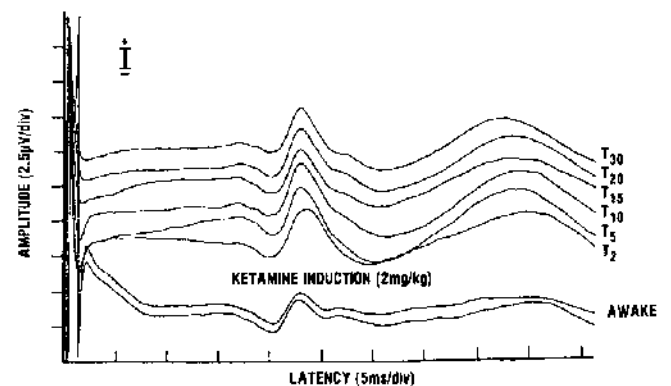
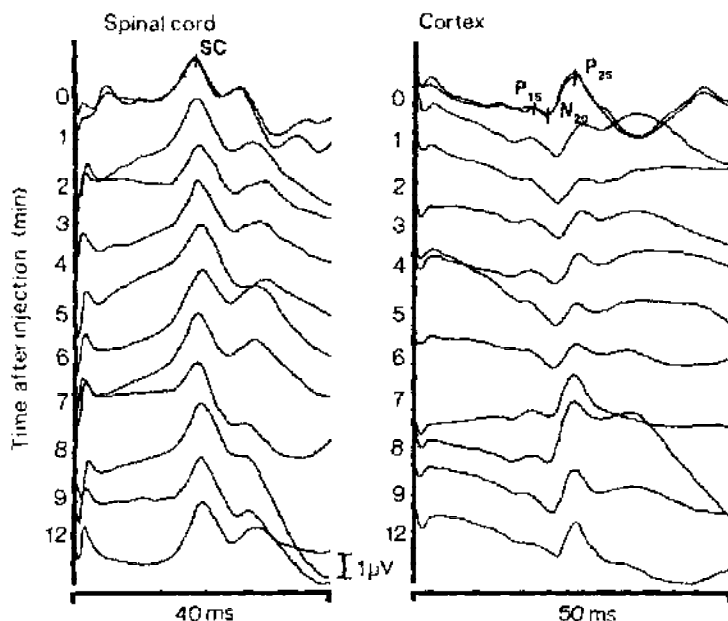


Figure 12-12. Example of SCEP waveforms before and after induction with ketamine at times 2, 5, 10, 15, 20, and 30 minutes. (From Schubert A, Licina MG, Lineberry PJ: The effect of ketamine on human somatosensory evoked potentials and its modification by nitrous oxide. *Anesthesiology* 1990;72:33, with permission.)

Figure 12-13. SEP responses recorded from the cervical and cortical electrodes before (0) and at several times up to 12 minutes following the injection of thiopentone (4 mg/kg). (From Sloan TB, Kimovec MA, Serpico LC: Effects of thiopentone on median nerve somatosensory evoked potentials. *Br J Anaesth* 1989;63:51, with permission.)



has been seen.¹⁷⁴ This latter effect on muscle responses may be mediated by the same mechanisms that potentiate the H-reflex.³⁵⁴ However, effects on subcortical and peripheral sensory responses are minimal (Fig. 12-12). Minimal effects are also observed in myogenic tcMSEP with ketamine.¹¹²

Because of these effects, ketamine is a desirable agent for monitoring responses that are usually difficult to record under anesthesia (e.g., dermatomal evoked responses and transcranially elicited muscle motor evoked responses). However, its hallucinatory potential and known increase in intracranial pressure with intracranial pathology have led to a reluctance to utilize this agent routinely.

Sedative-Hypnotic Drugs

Intravenous sedative agents are frequently used to induce or supplement general anesthesia. If inhalational agents must be avoided, sedative-hypnotic agents are routinely combined with opioids or ketamine to ensure adequate sedation, anxiolysis, and amnesia. Although ketamine doses produce some dissociative effects in addition to analgesia, supplementation can reduce the risk of excitatory events including hallucinations.

Droperidol

When combined with fentanyl ("neurolept anesthesia"), droperidol appears to have minimal effects on SEP, VEP, and MEP.¹⁷³ However, since its effect is quite long-lasting, many anesthesiologists would prefer to utilize a more rapidly metabolized sedative hypnotic for TIVA.

Barbiturates

Thiopental remains a popular drug for anesthesia induction, though transient decreases in amplitude and increases in latency of cortical sensory responses occur. Longer latency cortical waves are most affected, while minimal effects are seen on the subcortical and peripheral responses. Studies with another barbiturate, phenobarbital, demonstrate that the BAEP is virtually unaffected at doses that produce coma; changes are not seen until doses sufficient to produce cardiovascular collapse are reached.²⁴² Similarly, the SEP is unaffected at doses that produce

a silent EEG. For this reason, sensory evoked responses have been used successfully to monitor neurologic function during barbiturate-induced coma (Fig. 12-13).³⁶²

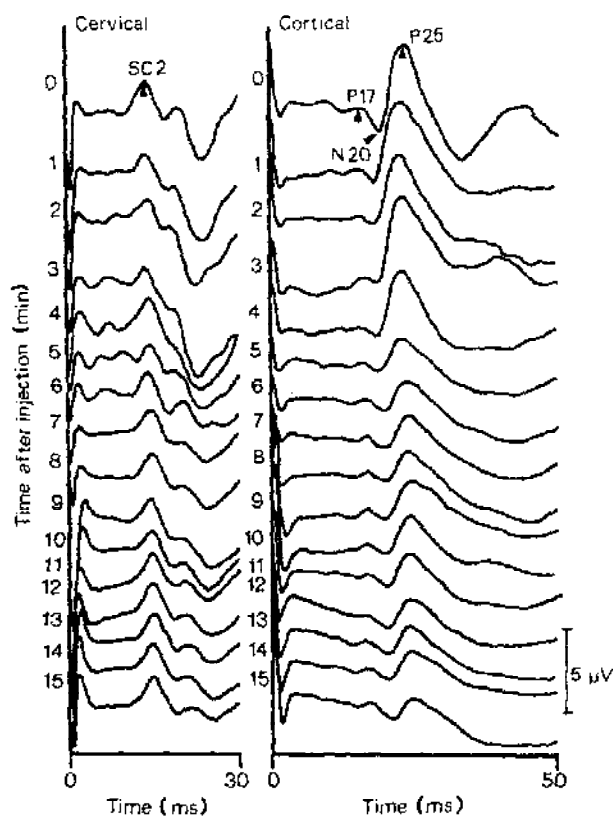


Figure 12-14. SEP responses recorded from the cervical and cortical electrodes are shown before and at 1-minute intervals after the injection of midazolam (.2 mg/kg). (From Sloan TB, Fugina ML, Toleikis JR: Effects of midazolam on median nerve somatosensory evoked potentials. *Br J Anaesth* 1990;64:590, with permission.)

Barbiturates are not commonly used during recording of tcMEP because the CMAP responses are particularly sensitive to barbiturates. Further, the effect appears quite prolonged; in one study, the induction bolus eliminated the CMAP from tcMEP for a period of 45–60 minutes,⁴¹⁷ suggesting that barbiturates are a poor induction choice when monitoring with this modality is needed. For this reason, most anesthetic protocols do not use induction with thiopental. One exception, methohexital, has been used in one TIVA protocol with opioids and ketamine.⁴¹⁷ Fortunately, this drug is more rapidly metabolized and appears to have excitatory properties (low doses can be used to identify seizure foci during cortical mapping of epilepsy).

Benzodiazepines

Midazolam has desirable properties of amnesia and has been used for monitoring cortical SEPs. However, because of its significant effects on myogenic MEP and its slow metabolism, it has been replaced by other agents. Midazolam, in doses consistent with induction of anesthesia (0.2 mg/kg) and in the absence of other agents, produces a mild depression of cortical SEP³⁶³ and minimal effects on subcortical and peripheral sensory evoked responses (Fig. 12-14). As with thiopental, midazolam produces prolonged marked depression of tcMMEP, suggesting that it also may be a poor induction agent for MEP recording.¹⁷² This effect has been interpreted as due to inhibition of cortical pyramidal cell neurons. In addition to possible cortical locations for the benzodiazepine effect, an effect at the spinal cord dorsal root has been suggested by a study of posterior tibial stimulation,¹⁷⁰ which revealed a marked decrease in the amplitude of the H-reflex with no effect on the stimulated CMAP (M-wave).

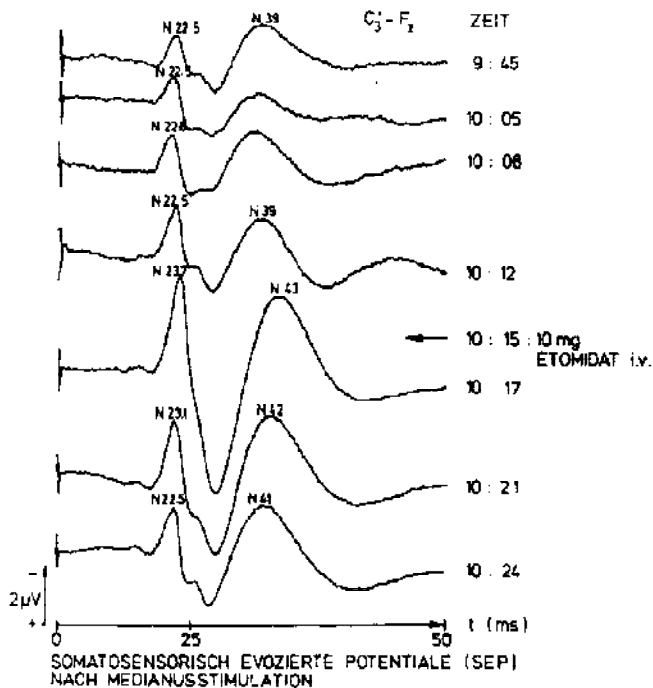


Figure 12-15. Cortical SEP from median nerve stimulation before and following 10 mg etomidate. (From Russ W, Thiel A, Schwandt H, Hempelmann G: Somatosensorisch evozierte Potentiale unter thiopental und etomidat. *Anaesthesist* 1986; 35:679, with permission.)

Etomidate

Like ketamine, etomidate has the unusual effect of response enhancement. Hence it has become a desirable agent for some monitoring uses. Etomidate increases the amplitude of cortical SEP components following injection¹⁹⁴ with no changes in subcortical and peripheral sensory responses (Fig. 12-15). This amplitude increase appears coincident with the myoclonus seen with the drug, suggesting a heightened cortical excitability (no evidence of seizure activity, however, was seen). A sustained amplitude increase with constant drug infusion has been used to enhance SEP cortical recordings that were otherwise not monitorable.³⁶¹ This effect has also been used to enhance amplitude in motor evoked responses.^{194,361} Fortunately, the enhancing activity occurs at doses that are consistent with the desired degree of sedation and amnesia needed for TIVA.

Studies with tcMEP have suggested that etomidate is an excellent agent for induction and monitoring of these modalities.³⁶⁸ Of the several intravenous agents studied, etomidate had the least degree of amplitude depression after induction doses or continual intravenous infusion.¹¹² Thus, etomidate has been used for induction of anesthesia and as a component of TIVA, combined with opioids.^{141,203,204,434}

Propofol

Propofol has a very rapid metabolism, making it an excellent component of TIVA. Although propofol is a depressant agent, adjustment of infusions often will allow adequate monitoring. Propofol induction produces amplitude depression in cortical AEP (Fig. 12-16) and cortical SEP with rapid recovery after termination of infusion.⁵¹ When the SEP is recorded in the epidural space, propofol has no significant effect. This is consistent with the postulated site of anesthetic action of propofol on the cerebral cortex.¹² Studies with transcranial electrically or magnetically elicited motor evoked potentials have demonstrated a depressant effect on response amplitude, also consistent with a cortical effect.^{160,172,181} Propofol does not appear to enhance cortical responses, but its rapid metabolism allows the depth of anesthesia and effects on evoked responses to be adjusted rapidly.

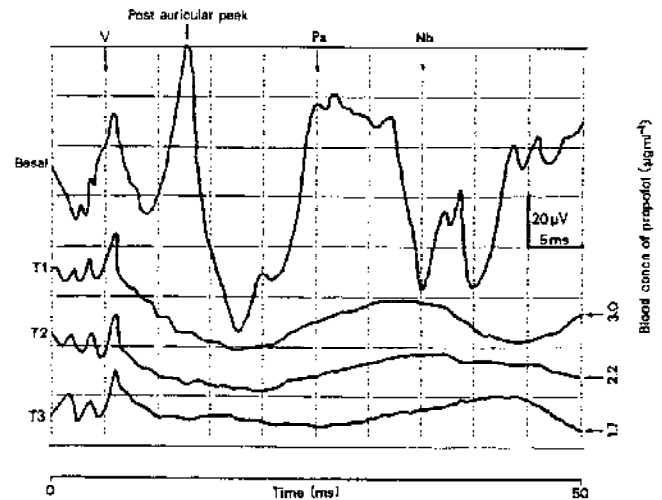


Figure 12-16. Cortical AEP before anesthesia and at different concentrations of propofol. Arrows indicate the position of waves V, Pa, and Nb. (From Chassard D, Joubaud A, Colson A, et al: Auditory evoked potentials during propofol anaesthesia in man. *Br J Anaesth* 1989;62:522, with permission.)

However, as a component of TIVA, infusions of propofol have been combined with opioids to produce acceptable conditions for myogenic MEP monitoring.^{47,166,308,309}

Regional Anesthesia

Major conduction anesthesia (e.g., epidural or spinal) does not appear to be an acceptable alternative to general anesthesia for monitoring evoked responses that depend on the tracts anesthetized.³⁷¹ This effect has also been seen with intravenous regional block and specific nerve blocks. However, local anesthesia placed away from the neural pathway mediating the monitored evoked response (such as scalp anesthesia for awake craniotomy) is satisfactory for monitoring unless systemic absorption is substantial.

Muscle Relaxants

Since muscle relaxants have their major site of action at the neuromuscular junction, they have little effect on electrophysiologic recordings such as the SEP that do not derive from muscle activity. In fact, they may improve, or be essential for, some types of recordings where the muscle activity near the recording electrode may create unwanted noise. This is true for epidural or peripheral nerve recordings where the activity of overlying muscle obscures the response from transcranial stimulation. For recording of these responses, complete or near-complete neuromuscular blockade is highly desirable.

Certainly, complete neuromuscular blockade will prevent recording of CMAP responses during MEP. However, partial neuromuscular blockade has the benefit of reducing a substantial portion of the movement that accompanies the testing. Some degree of neuromuscular blockade may facilitate some surgical procedures where muscle relaxation is needed for adequate tissue retraction.

Two methods are customarily utilized to assess the degree of neuromuscular blockade. The method that best quantitates the blockade involves measuring the amplitude of the CMAP (T1) or M-wave produced by supramaximal peripheral motor nerve stimulation. When neuromuscular monitoring is conducted this way, most monitoring protocols use neuromuscular blockade of T1 that is 10–20% of baseline. Clinically, anesthesiologists often assess neuromuscular blockade by counting the number of twitches remaining when four motor nerve stimuli are delivered at a rate of 2 Hz. Measured this way, acceptable CMAP monitoring has been conducted with only 2 of 4 responses remaining⁴⁷ (this corresponds to a T1 response in the range of 10–20%). When intense neuromuscular blockade is required (e.g., recording of epidural or neurogenic responses), T1 response of less than 10%, or no more than 1 of 4 twitches, is generally recommended.

When using neuromuscular blockade, tight control of the blockade is necessary so that excessive blockade does not eliminate the ability to record or mimic the loss of the response with neural injury (Fig. 12-17), creating a false-positive result.³⁶⁶ Because of varying muscle sensitivity to muscle relaxants, the neuromuscular blockade may need to be evaluated continuously in the specific muscle groups used for monitoring. It is important to note that the use of neuromuscular blockade is controversial during monitoring of muscle responses from mechanical stimulation of nerves, as partial paralysis may reduce the ability to detect these responses (e.g., facial nerve monitoring or monitoring for pedicle screw placement). Often muscle relaxants are avoided when monitoring these latter mechanically evoked muscle EMG neurotonic responses.

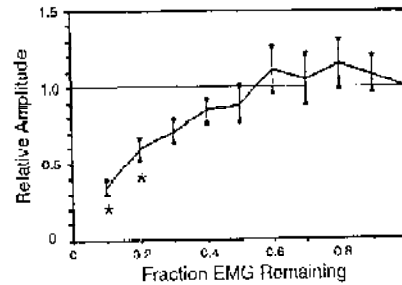


Figure 12-17. Plot of tcMMEP amplitude as recorded from thenar muscles as plotted versus the fraction of a single twitch remaining following median nerve stimulation. (From Sloan TB, Erian R: Effect of atracurium induced neuromuscular block on cortical motor evoked potentials. *Anesth Analg* 1993;76:979, with permission.)

OTHER INTRAOPERATIVE PHYSIOLOGIC FACTORS

BLOOD FLOW

As measures of neural function, the evoked responses are responsive to a variety of physiologic factors that alter neuronal function and viability. Numerous studies^{14,28,29,33,392,394} have demonstrated a threshold relationship between regional cerebral blood flow and cortical evoked responses. The cortical SEP remains normal until blood flow is reduced to about 20 ml/min/100 gm.³⁶⁷ At more restricted blood flows of between 15 and 18 ml/min/100 gm of tissue, the SEP is altered and lost (Fig. 12-18).^{31,32,64,119,134,193,212} Subcortical responses appear less sensitive, though global hypotension, affecting both subcortical and cortical blood flow, is associated with cortical SEP loss at higher rates of cerebral blood flow than with middle cerebral artery occlusion, which more selectively impairs cortical than subcortical blood flow.^{119,213} The difference in sensitivity to ischemia between cortical and subcortical structures may explain why the **central conduction time (CCT)** of the SEP bears a parametric relationship to cerebral blood flow (Fig. 12-18).^{103,134}

Regional factors may produce focal ischemia not predicted by systemic blood pressure. For example, during spinal surgery, the effects of hypotension may be aggravated by spinal distraction, such that an acceptable limit of systemic hypotension cannot be determined without monitoring.^{35,82,120,172} Other examples include peripheral nerve ischemia from positioning,

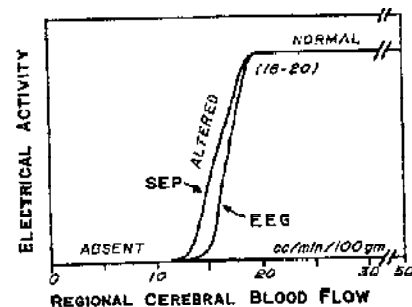


Figure 12-18. Relationship between the SEP and EEG electrical response and regional cerebral blood flow. (From Sloan T: American Society of Anesthesiologists: Refresher Courses, October 1985, Lecture 211, with permission.)

tourniquets, or vascular interruption,^{98,780,432} spinal cord ischemia from aortic blood flow compromise, carotid artery interruption,³³¹ vertebrobasilar insufficiency aggravated by head extension, cerebral artery constriction by vasospasm, and cerebral ischemia due to retractor pressure.³⁹⁴

MEPs and SEPs are sensitive to spinal cord events produced by vascular ischemia (aortic cross-clamping) or mechanical compression (epidural balloon). However, because these tracts are topographically removed from one another, the MEP and SEP may show differential sensitivity to an ischemic event.¹⁴⁹

MEP studies using transcranially generated MEPs and recorded epidurally are sensitive to ischemia but not to anterior horn cell injury. This is postulated to be due to persistent conduction in the corticospinal tracts.⁹² This is in contrast to the recording of peripheral nerve or muscle response with MEPs, in which anterior horn cell injury can destroy the anterior horn cell function that is required to translate the descending neural signal into a peripheral nerve or muscle response.³⁸⁹ As with the SEP, myogenic MEP is sensitive to spinal cord ischemia associated with thoracic aortic clamping,^{149,208,389} and a decrease in response has been shown to correlate with reduced spinal cord blood flow.^{92,149}

INTRACRANIAL PRESSURE

Another factor leading to regional (cortical) ischemia is **elevated intracranial pressure (ICP)**. Elevated ICP is associated with reductions in amplitude and increases in latency of cortically generated visual, somatosensory, and brain stem auditory evoked responses. The BAEP is altered as uncus herniation occurs.²⁷⁰ The relationship of the VEP to ICP has suggested the VEP as a means of noninvasive ICP testing (Fig. 12-19).⁴³⁵

Increased ICP, probably by virtue of its effect on cortical structures, produces a gradual increase in onset of the tcMMEP until a response can no longer be detected (i.e., threshold exceeds the capacity of the stimulator). The increase in latency suggests that the central component of the motor pathway has slowed conduction velocity.³⁶³

HYPOXEMIA

Hypoxemia can also cause evoked potential deterioration. This has been recorded in one case when the P_aO_2 reached 41 mmHg¹²¹ before other clinical parameters had changed.

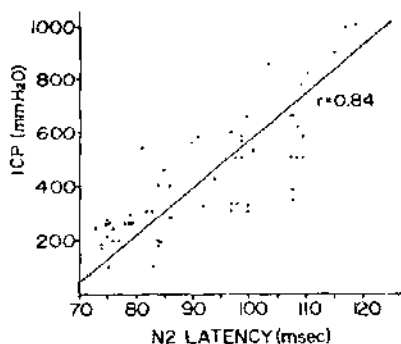


Figure 12-19. Relationship of latency of VEP with intracranial pressure. (From York DH, Pulliam MW, Rosenfeld JG, Watts C: Relationship between visual evoked potentials and intracranial pressure. *J Neurosurg* 1981;55:909, with permission.)

BLOOD RHEOLOGY

Since changes in hematocrit can alter both oxygen carrying capacity and blood viscosity, the maximum oxygen delivery is often thought to occur in a mid-range hematocrit (30–32%). Evoked response changes with hematocrit are consistent with this optimum range. In a study of VEPs and upper limb SEPs in the baboon, Nagao²⁶⁹ observed an increase in amplitude with mild anemia, an increase in latency at hematocrits of 10–15%, and further latency changes and amplitude reductions at hematocrits below 10%. These changes were partially restored by an increase in the hematocrit.

VENTILATION

In addition to changes in oxygenation, alterations in ventilation can alter blood carbon dioxide, thus altering spinal cord and cortical blood flow (hypocapnia producing vasoconstriction and hypercapnia producing vasodilation). The most significant changes in cortical SEP occur when the carbon dioxide is extremely low, suggesting excessive vasoconstriction may produce ischemia (carbon dioxide tensions below 20 mm Hg). Hypocapnia may aggravate hypotension as a result of arterial vasoconstriction. This effect has been suggested to contribute to alterations in SEP during spinal surgery¹²⁴ or in BAEP during posterior fossa surgery in the sitting position.¹²⁴

TEMPERATURE

Hypothermia can also alter evoked responses by changing nerve depolarization (increased action potential duration,¹⁸⁷ reduced conduction velocity,^{73,197} and decreased synaptic function⁴¹⁸), resulting in latency increases and decreases in evoked response amplitude.⁸³ These changes have been observed in visual,^{323,431} brain stem auditory,^{85,168,380} cortical auditory,¹⁸³ and somatosensory evoked potentials.^{21,83,135,316} Contrarily, induced hyperthermia can reduce SEP latencies,⁸⁷ but this is less often an intraoperative concern. Hypothermia appears to affect central nervous system synaptic function more than conduction,⁴¹ probably by interference in the postsynaptic membrane.³⁹⁹ Thus, changes are more prominent at the cephalic end of long neural tracts (such as the SEP) or in components of responses associated with multiple synaptic elements (Fig. 12-20). Hence, responses recorded from peripheral nerves are minimally affected, whereas those produced by cortical structures are markedly affected for the same degree of cooling.^{53,155} Core temperatures

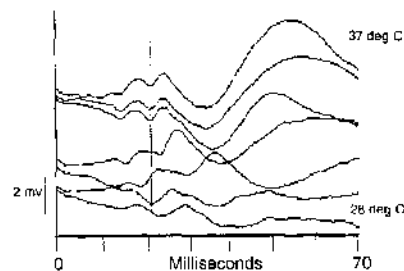


Figure 12-20. Changes in the cortical SEP (median nerve) with whole body hypothermia as esophageal temperature is lowered from 37 to 28 degrees Centigrade. (From Sloan TB: Evoked potentials. In Albin MS (ed): Neuroanesthesia. New York, McGraw-Hill, 1997, pp 221–276, with permission.)

frequently drop by greater than 1°C, but peripheral nerves lifted from the body for focal stimulation may be as cool as 20°–30°C.³⁸

Whole body hypothermia, either inadvertent or intentional, is the most obvious temperature change that occurs during surgery. Changes in regional temperature can also occur, resulting in evoked response alterations that would not be otherwise predicted based on unchanged body (core) temperature. For example, cold irrigation solutions applied to the spinal cord,⁶¹ brain stem, or cortex routinely cause evoked response changes. These cold irrigation solutions may also irritate the nerve, causing increased muscular activity if the nerve has motor components.²⁵⁶ Similarly, limb cooling (as from cold intravenous solutions) can alter the SEP originating from stimulation to a nerve from that limb.

With hypothermia, the tcMMEP demonstrates a gradual increase in onset as temperature decreases from 38°C to 32°C esophageal. An increase in stimulation threshold has also been observed at lower temperatures. This is consistent with both cortical initiation and peripheral conduction being affected by the temperature drop.³⁶⁴

OTHER PHYSIOLOGIC VARIABLES

Changes in a variety of other physiologic variables may produce alterations in the evoked responses during surgical monitoring. Significant reduction in blood volume can alter evoked responses as a result of changes in blood flow distribution, despite absence of significant blood pressure changes (e.g., limb ischemia altering the SEP as blood flow to central organs is spared). An increase in superior vena caval pressure during cardiopulmonary bypass has been associated with SEP changes.¹⁴⁸

Other physiologic events may occur too slowly to be noted as changes in the evoked response. For example, changes in glucose,⁷⁴ sodium, potassium, and other electrolytes important in the neurochemical environment are likely to also result in evoked response changes over time.

INTRAOPERATIVE NEUROPHYSIOLOGIC MONITORING: SPECIFIC APPLICATIONS

As previously noted, **intraoperative monitoring (IOM)** can be described as the application of neurophysiologic, usually electrophysiologic, techniques to detect changes in the functional state of the nervous system consistent with ischemia or injury. Electrophysiologic techniques may also assist in localizing neural structures, identifying specific cortical functional areas, and delineating an epileptogenic cortex. The information obtained may also help determine the mechanism of injury, correlated with the surgical proceedings, and serves to prevent damage by detecting cellular dysfunction prior to its reaching an irreversible cell death stage. Outcome studies have in general been promising in that intraoperative electrophysiologic studies do make a positive difference in patient care.^{69,215,257,290,293,399,414}

Intraoperative neurophysiologic monitoring for surgical procedures where the central and peripheral nervous systems are at risk has become increasingly popular over the past several years. IOM of facial nerve activity, utilizing EMG, was first performed in the late 19th century, and EEG was subsequently used in 1965.³¹⁰ Other electrophysiologic techniques such as SEPs, BAEPs, VEPs, and peripheral nerve compound action potentials

gradually came into use. The intraoperative application and clinical utility of EEG, BAEPs, SEPs, and LMG in a variety of different surgical procedures are discussed below. The clinical utility of VEPs is very limited; therefore, VEPs will be only briefly discussed.

The cortical EEG is produced by the spontaneously generated electroencephalographic brain wave activity and is easily recorded, requiring no stimulus. However, evoked potential (EP) recordings can only be generated by applying an external stimulus. In general, stimulation is provided peripherally using peripheral nerve electrical stimulation for SEPs, auditory clicks for BAEPs, and light flashes for VEPs. In addition to recording EPs, spontaneous or stimulus triggered EMG can be recorded from muscle using either intramuscular or surface electrodes. Electrical or magnetic stimulation of peripheral nerves, nerve roots, spinal cord, cranial nerves, or cortical structures can generate a motor evoked response that also can be recorded from a muscle or nerve.

ISCHEMIA AND ELECTROPHYSIOLOGIC STUDIES

Different techniques have been used in order to identify and attempt to prevent damage caused by cerebral ischemia. The most extensively studied and commonly used techniques are EEG and SEPs. The rationale for employing electrophysiologic techniques as a marker for ischemia is the good correlation between these techniques and **regional cerebral blood flow (rCBF)**. As previously noted, studies demonstrated that in patients undergoing carotid endarterectomies (CEA), major EEG changes occurred with rCBF < 10 ml/100 gm/min, and less severe EEG changes were seen with rCBF between 10 and 18 ml/100 gm/min, with a critical level defined as 15 ml/100 gm/min.^{352,386,387} In contrast, primate studies show that SEPs are maintained at levels of rCBF ≥ 16 ml/100 gm/min but absent at levels below 12 ml/100 gm/min. At rCBF levels between 14 and 16 ml/100 gm/min, there is a sharp decline in the evoked response amplitude, with a 50% amplitude reduction corresponding to a rCBF of 16 ml/100 gm/min.^{28,39,32,391} In addition to altering the SEP amplitude, ischemia also appears to prolong the CCT, with a threshold rCBF (<15 ml/100 gm/min) similar to those previously reported for EP amplitude reduction.^{134,213} Interestingly, there is experimental evidence suggesting a differential susceptibility to local ischemia as one descends the neuraxis, demonstrated by increasing resistance of electrophysiologic function to systemic hypotension.³²

There clearly seems to be a threshold relationship between cerebral blood flow and alteration of the EEG and SEP. There is also a good deal of experimental evidence for a rCBF threshold and cellular membrane failure.^{15,391} An investigation has found that at rCBF levels between 12 and 16 ml/100 gm/min, the SEP was abolished and small, self-limiting increases in extracellular potassium activity were detected.³⁰ This study further demonstrated that the rCBF threshold range for a massive irreversible rise in extracellular potassium, associated with structural changes of infarction, occurred between 7.6 and 11.4 ml/100 gm/min. In baboon chronic stroke models, following middle cerebral artery (MCA) occlusion, areas of infarction corresponded to blood flow levels of 10 ml/100 gm/min or less.^{399,399} In acute stroke primate models, infarction occurred only in areas where rCBF measured ≤ 12 ml/100 gm/min (Table 12-12).^{167,186,260} Thus, these findings suggest that significant (50%) reduction in amplitude of the SEP, which corresponds to a rCBF of 14–16 ml/100 gm/min, is indicative of ischemia and is a

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