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Neuromonitoring syllabus

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INTRODUCTION

THE EVOLUTION OF MONITORING FOR SPINE AND BRAIN SURGERY

The potential for neurologic injury during surgery of the brain and spinal cord has caused a strong interest in the early recognition of iatrogenic compromise. In 1973, Vauzelle and colleagues published a paper on the practice of awakening patients during critical phase of their spine surgery (i.e. after placement of rods during scoliosis surgery) and asking them to move their extremities[1]. If the patient was able to move then the spinal cord was considered to be intact and surgery was allowed to proceed. If the patient did not move, then the hardware was removed and the surgery was terminated. This first attempt at intraoperative monitoring of the spinal cord had some obvious disadvantages: it was time consuming, it could cause physical or psychological harm to the patient, and it was impractical to perform multiple times. Hence, the need was recognized for a test of spinal cord integrity that was less dangerous and could be repeated throughout the surgery.

The ankle clonus reflex test addressed some of the issues of the wake up test and was primarily used in children during scoliosis surgery[2]. This test assesses whether the lower extremity stretch reflex remains intact; passive dorsiflexion of the foot stimulates the stretch reflex and produces rhythmic contraction of the calf muscles (clonus). This reflex is absent in normal awake patient because of central reflex inhibition but is present during periods of light anesthesia in neurologically intact patients due to the return of lower motor neuron function before the appearance of inhibitory upper motor neuron impulses. Presence of the ankle clonus reflex signifies intact spinal cord pathway along the reflex arc. However, absence of the reflex may be due to an anesthesia plane that is too light, too deep, or injury to the cord at the surgical site. This lack of specificity was proven in many studies, and the test has fallen out of favor[3-5].

In the late 1970 and early 80's, studies in animal models suggested that changes in somatosensory evoked potentials of the hind limbs during distraction of the spinal cord correlate with loss of motor and sensory function[6,7]. Subsequently SSEP signal changes have been found to correlate well with clinical outcomes and serum markers of cell damage[8,9]. This information is the basis of modern intraoperative neuromonitoring. In this chapter we will consider the major types of intraoperative neuromonitoring currently in use: somatosensory evoked potentials (SSEPs), motor evoked potentials (MEPs), electromyography (EMG), brainstem evoked auditory responses (BAER), and visual evoked potentials (VEPs). We will discuss the utility/indication, anesthetic considerations, and limitations of each modality. This chapter is not meant to be an exhaustive discussion of the techniques of monitoring. For a more in depth discussion, the reader is referred to many excellent texts and review articles[10-12]. Rather, this chapter will discuss the fundamental basis of neuromonitoring for the purpose of helping anesthesia practitioners understand the rationale for choosing a particular test, suggestions of how to facilitate acquisition of signals through anesthetic technique, and the significance of intraoperative deterioration in evoked potentials.

SOMATOSENSORY EVOKED POTENTIALS

Definition: Somatosensory evoked potential (SSEP) monitoring involves peripheral stimulation of a mixed motor/sensory nerve, which then initiates sensory and motor transmissions. These transmissions are recorded as an EEG response monitored by electrodes over the sensory cortex, and peripherally as muscle contractions. The most commonly monitored nerves are superficial and large enough to be stimulated

easily. Examples include the median (C6-T1), ulnar (C8-T1), common peroneal (L4-S1) and posterior tibial (L4-S2). A disturbance of transmission can be caused by an injury anywhere along the peripheral nerve, plexus, spinal cord or cortex. The neural tract for the upper extremity ascends the ipsilateral dorsal column, synapses at the nucleus cuneatus, crosses at the cervico-medullary junction, continues to ascend via the medial lemniscus, and projects to the contralateral parietal sensory cortex. Sensory evoked responses from the lower extremity ascend along a similar path; additionally some of the response travels via the antero-lateral spino-cerebellar pathways. Signals are usually recorded at 3 to 4 sites along the sensory pathway from the periphery to the cortex. For example, in the upper extremity a stimulus at the median nerve would be recorded over Erb's point, cervical vertebrae, and cortex.

Utility

In animal studies, depression of SSEP signals correlates with loss of sensory and motor function during spine distraction[7]. In the absence of malfunctioning monitoring equipment, these changes are caused by loss of blood supply to the cord from direct compression or disruption of the blood supply to the cord by secondary factors (e.g. low blood pressure). The largest study to examine the utility of SSEP responses included 51,000 scoliosis patients and was conducted by the Scoliosis Research Society and European Spinal Deformities Society. In this study, patients who underwent surgery with SSEP monitoring had a significantly lower (0.55%) incidence of neurologic injury than patients who did not have SSEP monitoring (0.7-4%)[13]. Based on this data, the SRS issued a position statement that «neurophysiological monitoring can assist in the early detection of complications and possibly prevent postoperative morbidity in patients undergoing operations on the spine». Subsequently, surveys suggest that most surgeons in the United States use SSEP monitoring for most of their spine surgery cases[14].

SSEP monitoring has utility in many other types of surgery where direct injury to a neural structure is possible. For example, SSEPs can be used during resection of spinal cord tumors or to find the optimal area for transection during dorsal root entry zone lesioning. Some uses of SSEPs outside of spinal surgery include prevention of positioning injuries through evaluation of peripheral nerves and brachial plexus. SSEPs can be used to identify areas for surgical repair when there has been nerve injury. In combination with auditory and cranial nerve monitoring, SSEPs may be used to assess the integrity of the brainstem during surgery on the posterior fossa. Direct monitoring of the sensory cortex using bipolar recording strips can be used to identify the gyrus separating the motor and sensory strip.

Like EEG, SSEPs are sensitive to changes in blood flow below a threshold (20 cc/min/100 g) and are lost entirely during ischemia (15-18 cc/100 g). This information can be used to determine the critical threshold for induced hypotension or anemia, which may occur at unanticipated levels in predisposed individuals[15]. Systemic blood pressure is an imprecise surrogate marker of oxygen delivery to the spinal cord because of regional compromise of flow due to spinal pathology or surgical compression. Neural tissue sensitivity to changes in blood flow can be important in open craniotomy and interventional radiology procedures for arteriovenous malformation, intracranial aneurysms. SSEPs can also detect changes in blood flow during vascular surgery procedures such as carotid endarterectomy. Postoperatively, SSEPs can be used to identify cerebral vasospasm after subarachnoid hemorrhage.

Anesthetic considerations

All anesthetics affect conduction along neural pathways, as demonstrated by their effect on EEG, and will therefore affect acquisition of evoked potentials to varying degrees. Anesthetics may affect the evoked responses by either direct inhibition of synaptic pathways or indirectly by changing the balance of inhibitory and excitatory influences. The magnitude of the effect increases with the number of synapses in the pathway.

Most anesthetics depress evoked response amplitude and increase latency. Inhaled anesthetics accomplish this by altering specific receptors or through nonspecific effects on cell membranes that alter the conformation structure of the receptor or ion channel. The effect of halogenated agents on SSEP signals directly correlates with potency; isoflurane > enflurane > halothane. Studies suggest that sevoflurane and desflurane are similar to isoflurane[16]. Nitrous oxide reduces SSEP cortical amplitude and increases latency alone or with halogenated inhalational agents or opioid agents. The greatest effect is seen in the waveforms recorded over the cortex, and less in the Erb's point and cervical waveforms. When compared to equipotent halogenated anesthetic concentrations, nitrous oxide produces more profound changes in cortical SSEPs[16].

Intravenous agents have their effects on evoked potentials by their affinity for neurotransmitter receptors (e.g. GABA, NMDA, glutamate, etc). The effect varies with the specific receptor and pathways affected. Propofol, benzodiazepines and barbiturates cause significant depression of the amplitude of the waveforms. Benzodiazepines and barbiturates infusions are no longer commonly used for maintenance anesthesia during spine surgery for various reasons, including their extremely long context sensitive half life, barbiturate's potential to cause hyperalgesia, and prolonged depression after a bolus induction dose. While

an induction dose of propofol cause depression of SSEPs, its context sensitive half time is significantly shorter, and therefore its titratability makes it an important component of a maintenance anesthetic during a monitored spine surgery especially in combination with other favorable drugs. Opioids affect SSEP signals less than inhalational agents, making them an important component of evoked potential monitoring. Bolus doses of opioids can be associated with mild decrease in amplitude and an increase in latency in responses recorded from the cortex. Opioid infusions are generally conducive to monitoring and many neuroanesthesiologists have taken advantage of remifentanyl, which has an extremely short half life, to supplement an intravenous maintenance anesthetic. Some intravenous anesthetics which do not depress SSEP waveform amplitude include etomidate and ketamine. These drugs increase signal amplitude, potentially by attenuating inhibition[16]. In addition to its beneficial effects on neuromonitoring signals, ketamine is a powerful analgesic and may be especially helpful for controlling pain in opioid tolerant patients[17]. Recent studies have examined the effect of dexmedetomidine on the acquisition of neuromonitoring signals. Several studies have found that the use of dexmedetomidine is compatible with acquisition of SSEPs[18].

Neuromuscular blocking agents have their affect at the neuromuscular junction and therefore do not negatively affect the acquisition of SSEP signals. If anything, this class of drugs improves the acquisition of signals by decreasing movement artifact.

Limitations

Although SSEPs are effective for monitoring the integrity of the dorsal columns, the technique is limited in its ability to predict overall clinical outcomes. Most importantly, the presence of SSEPs does not guarantee an intact motor pathway. This is because the motor and sensory tracts are located in different regions of the spinal cord and have distinct blood supplies. Although early reports of SSEP preservation in patients with postoperative paraplegia was considered a failure of SSEP monitoring, this was inappropriate because preserved SSEPs are only considered to be false negative when there is a postoperative sensory deficit[19]. While it is likely that stretch injury, e.g. due to distraction during scoliosis surgery might affect both pathways; it is possible that surgery may directly injure one of the tracts (e.g. placement of a strut graft during an anterior cervical diskectomy. In patients having scoliosis surgery Nuwer et al. found that 0.063% of patients with preserved SSEPs had permanent neurologic deficits[14]. In comparison 0.983% of patients in the same study had SSEP changes without deficits (false positive). Therefore, the best use of SSEPs is either to detect injury to the sensory tracts when this is the primary concern or in combination with other modalities (e.g. motor evoked potentials).

MOTOR EVOKED POTENTIALS

Definition: Motor evoked potentials refers to the use of direct electrical or magnetic stimulation of the motor cortex to produce electrical activity which can be recorded as D waves and I waves, or with epidural electrodes on the surgical field, or as compound muscle action potentials (CMAPs) measured by pairs of needles over the corresponding muscle. The electrical stimulation to evoke the response results in a volley of activity that descends the anterior horn of the corticospinal tract. After synapsing in the anterior horn, the impulse travels via peripheral nerve and crosses the neuromuscular junction, resulting in a muscle response.

As mentioned above, the sole use of SSEP monitoring to determine integrity of the spinal cord during surgery eventually gave rise to multiple reports of unchanged signals associated with postoperative motor deficit and normal sensory function. This indicated that the anatomic isolation of the motor tracts required the addition of a monitoring modality that would monitor them directly. The ventral portion of the spinal cord is particularly vulnerable to injury because of its relatively tenuous blood supply; a single anterior spinal artery supplies 75% of the entire cord which includes the motor tracts. Therefore the anterior portion of the cord is more susceptible to hypoperfusion injury due to anemia, hypotension, and blood vessel compression.

MEPs can be recorded rapidly by a single brief stimulation or with several pulses to facilitate smaller responses, in comparison SSEP requires multiple stimulations and signal averaging. Common muscles monitored include: adductor pollicis brevis, biceps, triceps, dorsal intraosseus, tibialis anterior and anal sphincter. The required pattern of stimulation and milliamperes required for stimulation is highly variable between individuals and even within the same individual during an uncomplicated surgical procedure.

Utility

Motor evoked potential monitoring can be used to detect injury whenever the motor portion of the cord or cortex may be at risk during a surgical procedure. A common use is during scoliosis surgery where studies have suggested a high correlation of motor evoked potential recordings with neurologic outcome. In the largest study, transient changes were relatively common (11.3%), however, permanent MEP changes were associated with neurologic injury[20]. Another use of MEP monitoring is for spinal surgery when the anterior portion of the cord is particularly at risk, e.g. anterior cervical diske-

ctomy. Hildebrand et al described several cases of anterior cervical surgery in which SSEPs were maintained while MEPs were decreased during the placement of an anterior strut graft. The MEPs recovered after removal of the graft[21]. MEPs are well validated for prevention of motor injury during resection of spinal cord tumors[22]. It has been demonstrated that MEPs are more sensitive to spinal cord ischemia than SSEPs[23,24]. However, in both deformity surgery and in tumor surgery, several studies have demonstrated that no single modality monitors the entire spinal cord[25,26]. When used in combination with SSEP, motor evoked potentials are associated with a higher sensitivity and specificity of motor tract injury than single modality monitoring[27].

MEP monitoring has been used in craniotomy to prevent injury to the motor cortex. For example, direct motor cortex stimulation can be used to define the edge of motor cortex tumors[28]. MEP is a sensitive indicator of hypoperfusion of the motor cortex, and permanent changes in MEPs during aneurysm clipping are associated with postoperative paresis[29]. Similar to monitoring of the spinal cord, multimodality monitoring is also recommended during craniotomy. Multiple studies have described cases of preserved responses of either SSEP or MEP without changes in the other modality. Brainstem surgery for tumor resection is a good example where multimodal monitoring is utilized; MEP may be used in combination with SSEP, EMG, and auditory responses to map anatomic landmarks and facilitate the surgical approach.

Anesthetic considerations

Motor evoked potentials are extremely sensitive to anesthetics at the level of the synaptic transmission to the alpha motor neurons to produce CMAP responses. However, responses recorded prior to this synapse (e.g. D and I waves) are relatively insensitive to anesthetic technique. While some stimulation techniques (e.g. multiple pulses of stimulation) are used to facilitate acquisition of signal, MEPs remain much more sensitive to anesthetic agents and are more difficult to obtain and maintain than SSEP[30].

All of the volatile agents are associated with a significant and dose dependent depression of MEP responses. Inhaled anesthetics suppress pyramidal activation of spinal motor neurons at the level of the ventral horn. Studies suggest that relatively low doses of volatile anesthetics (0.25-0.5 MAC) suppress single pulse transcranial stimuli[31,32]. While more aggressive stimulation patterns can produce CMAP responses in some patients, others are entirely unattainable[33]. This may be a function of preexisting myelopathy due to the spinal disease process, neuropathy secondary to diabetes, or more insidiously to subclinical neuropathy associated with chronic hypertension[34]. When the concentration of inhaled anesthetic approaches 1 MAC, less than 10% of patients have appreciable signals[35]. It is controversial whether nitrous oxide is associated with less depression than other agents, and can be supplemental to other agents (e.g. ketamine, opioid or low dose propofol). Up to 50% nitrous oxide has been shown to be compatible to adequate myogenic response especially when intravenous drugs with minimal effects are used (ketamine, opioid)[36,37]. However, higher doses and use with propofol may be associated with significant (> 50%) reduction in CMAP amplitude.

Barbiturates and propofol are associated with a dose dependent decrease in CMAP amplitude and need for more aggressive stimulation patterns to produce similar CMAP waveforms. Propofol has become ubiquitous during surgery for which motor evoked potential monitoring is planned because of its more favorable context sensitive half time, titratability and side effect profile. An exact dose response curve plotting serum propofol concentrations and MEP signal strength has been difficult to determine due to heterogeneity of anesthetic technique across studies, lack of control for variation in blood pressure, and patient comorbidities. Opioids can suppress cortical excitation, but have minimal effect when used as an infusion, and as such are often used in combination with propofol or low dose inhalational agents. However, care must be taken because bolus doses of opioids can be associated with prolonged CMAP depression, with longer acting narcotics having prolonged effects[38,39]. Benzodiazepines can also cause CMAP amplitude depression, but have been shown to have minimal effects when used for premedication, or used as an infusion. Because of their less favorable context sensitive half life, benzodiazepines are generally not used as a maintenance anesthetic.

Etomidate causes minimal and transient suppression of CMAP amplitude without increasing latency, even with induction doses. It is likely that etomidate accomplishes this by disinhibition of subcortical structures, resulting in increased excitability of the motor system. Administration of etomidate may cause adrenocortical suppression. It also frequently causes nausea, which limits its utility as a maintenance anesthetic.

Ketamine can be a useful adjunct in the anesthetic of patients with chronic pain. Low to moderate dose ketamine (1 mg/kg or 0.25-0.5 mg/kg/h has a minimal effect on MEP responses, both as bolus dose and as an infusion. High dose ketamine (4-8 mg/kg) can be associated with moderate depression of CMAP amplitudes. A recent study showed that a loading dose of 0.5 mg/kg followed by an infusion of 10 μg/kg/min was associated with a lower 48 hour postoperative narcotic consumption, and no increase in side effects[17]. Previous studies have suggested that ketamine when used with narcotic infusion and nitrous oxide is associated with a 40% incidence of psychedelic side effects however another

study have suggested that this is significantly reduced with the addition of propofol[40].

Neuromuscular blockade, while not completely incompatible with MEP monitoring, is unpredictable and is therefore not recommended. Studies suggest that use of neuromuscular blocking drugs when carefully monitored using single twitch M- responses to maintain twitch height 20-50% of baseline allows CMAP responses[41]. However, this is not necessarily true if clinically monitoring twitch height, and if the patient has preexisting neurologic dysfunction[42]. If neuromuscular blockade is required, therefore, the patient should not have any existing neurologic deficits and an accelerometer should be as an objective measure of twitch height. In general, if there is a question regarding whether absence of MEP signal in a patient who has received a neuromuscular blocking agent is due to neuromuscular blockade or neurologic injury then the pattern (single limb *vs* complete loss of responses) and twitch height should be considered.

Limitations

Beyond its limitations as a single modality monitor, MEPs can be more technically difficult to obtain and maintain than SSEP. Chen et al. studied over 300 high risk spine surgeries and found that the success rate for upper extremity MEP signals was 94.8%, but only two thirds of patients had consistent signals for the lower extremity[30]. The likelihood of success was lower in children under 7 years old and adults over 64 years of age, and this was compounded by the presence of preexisting neurologic deficits. In comparison the same study demonstrated SSEP success rates greater than 98% in the upper extremities, and 93% in the lower extremity. In children with cerebral palsy scheduled for scoliosis surgery, SSEPs are attainable in greater than 80% of patients, whereas MEPs are reliably present only in 40-60%[43]. MEPs are also exquisitely sensitive to anesthetic technique (see above) and the patient's neurophysiologic status. It has been demonstrated that MEPs cannot be reliably obtained in patients undergoing aortic aneurysm surgery which involves lumbar epidural cooling[44].

Use of MEPs have significant safety considerations, some of which due to the inability to use neuromuscular blocking agents (i.e. the patient may move), and others that are inherent to the technique itself. The most common safety consideration is the possibility of bite injuries, which can occur because of direct activation of the temporalis muscle during cortical stimulation[45]. Bite injuries vary in severity and include a tongue hematoma, buccal lacerations, injury to the dentition, and jaw fracture. When MEP monitoring is planned, use of a bite block is mandatory. The bite block is placed between the molars on the side opposite to the endotracheal tube and secured in place. Oral airways are generally not large or solid enough to be sufficient for this purpose. In a patient without molars, it is acceptable to use a rolled gauze in the front of the oropharynx, however it is important that the roll is not so bulky that it precludes venous drainage of the tongue[46].

Relative contraindications to MEP monitoring include a history of seizure disorder, increased intracranial pressure or cortical lesions, defects of the skull or skull convexity, implanted deep brain stimulator leads, cardiac pacemakers or automated internal defibrillators. MEP monitoring should be used with caution in patients with epilepsy because pulses of electrical current delivered to the brain may cause seizures in susceptible patients. This phenomenon, called «kindling» has been described a single case report[47]. While the most conservative approach is to avoid MEP in patients with seizure history, limited MEP testing may be used in certain high risk procedures if the risk of a neurologic injury outweighs the relatively small possibility of inducing seizures. In this case, EEG activity is monitored during surgery, and use of TIVA generally serves to suppress seizure activity, which did resolve spontaneously with cessation of monitoring in the single report in which it was described.

The issue of electrical interference with implanted cardiac devices is somewhat more complex. The proximity of the stimulus to the device makes interference more likely than during SSEPs. However, the presence of a pacemaker or automated implanted defibrillator (AICD) is a relative contraindication and in procedures that carry a high risk of neurologic injury, MEP monitoring may be indicated. In this case, the patient, surgeon, monitoring team, and cardiologist should discuss the relative risks of MEP monitoring and decide upon a plan in advance of the scheduled surgery. If MEPs are strongly indicated, pacemaker dependence should be assessed. If the patient has an implantable cardiac defibrillator (ICD), antitachyarrhythmia properties should be disabled and external pacing/defibrillation pads should be placed on the patient. Pulse oximetry and invasive arterial monitoring should be used to throughout the procedure to allow continuous assessment of the patient's cardiac rhythm. The external pads should remain in place until the procedure is finished and the device has been interrogated in order to ensure that it is fully active and functional. Placement of a magnet on the patient's chest is not recommended, especially during prone positioning due to the potential for dislodgement and injury due to compression of the skin between the device and the magnet.

EMG

Definition: Electromyography (EMG) is a measurement of electrical activity generated by muscle contraction. EMGs are recorded by two electrodes in or near a muscle. Two major types of EMG recorded during surgery. *Passive,* or *free run* EMGs, reflect spontaneous activity and are continually recorded during the procedure. *Stimulated EMG* is generated

by applying an electrical stimulus either directly to a nerve or in its immediate vicinity. The response is recorded as a waveform or an audible tone.

The pattern of EMG response can be used to determine the difference between normal muscle activity during light anesthesia, reversible insult or injury. Normal EMG waveforms associated with light anesthesia are characteristically low amplitude and high frequency. Extremely deep general anesthesia is associated with lack of spontaneous activity and may even result in difficulty eliciting a stimulated response. Less ominous but abnormal activity is characterized by short asynchronous polyphasic waves called burst activity. This type of activity can be caused by fluid irrigation, nerve traction, or brief trauma. This type of activity is generally not sustained, resolves with cessation of the stimuli, and is not associated with permanent injury[11]. The presence of neurotonic activity, which consists of prolonged (minutes to hours) presence of synchronous waveforms, is more ominous[11]. This type of injury is associated with more significant stretch injury (spinal distraction with hardware placement) or compression with retractions. If this type of activity is not addressed by relieving the traction on the nerve, the likely result is a postoperative deficit.

Utility

While SSEP and MEP monitor the integrity of a tract, they may not be sensitive for injury of a single nerve root. EMG allows the identification of a single nerve especially in cases where the anatomy is abnormal (e.g. scar tissue or tumor). EMG is extremely sensitive and can detect activation of only 1-2% of a muscle's fibers[48]. Commonly monitored nerves include cervical nerve roots during spine surgery (C2-7), lumbrosacral (L2-S2) during spine surgery, facial nerve during acoustic neuroma surgery or parotid surgery, recurrent laryngeal nerve during anterior cervical surgery, and cranial nerves during brainstem surgery.

EMG monitoring is considered to be the standard of care for many surgerical procedures in which the facial nerve is at risk. Examples include: acoustic neuromas, parotid tumors, and cerebellar pontine angle tumors. The integrity of branches of the trigeminal nerve may also be identified with EMG recordings of the orbicularis oculi, oris, mentalis, and temporalis. Both the facial and trigeminal nerves may be monitored by intraoperative stimulation during decompression for trigeminal neuralgia with improved results[47,49]. Cranial nerve monitoring has become commonplace during radical neck surgery, thyroid, partiod, auditory surgery, and skull base tumor resection. In the lower extremity, free run EMG can be used to avoid sciatic nerve injury during hip arthroplasty.

During spine surgery, both free run and stimulated EMG can be useful to identify correct placement of pedicle screws. Without use of EMG stimulation, the incidence of neurologic complications associated with pedicle screw placement is estimated to be 2-10%[50-52]. In one study using pedicle screw stimulation, this complication was avoided entirely[53]. To perform pedicle screw stimulation, an electrode is attached to a screw that has been placed into a pedicle. If the cortex has been breached then the current required to cause the nerve root to fire and generate EMG activity is one-tenth less than if the bone is intact. In real terms, screw placements with thresholds greater than 10 mA are almost always in the correct position[53]. EMG response at less than 10 mA suggests the need for further inspection of the screw by the surgeon. Likelihood of false positive response correlates positively with stimulation intensity.

Anesthetic considerations

EMG response is generally not dependent on anesthetic technique, except for extremely deep general anesthetics– beyond what is used in clinical practice. Since the response depends on muscle activity, paralytic administration should be avoided or titrated with care. Studies suggest that EMG activity can be recorded with as few as 2 train-of-four responses[54]. However, this is assuming that the muscle recording TOF has the same sensitivity to muscle relaxant as the group where EMG monitoring is desired. Also, care must be taken that the TOF electrodes are over a nerve, and not causing direct muscle stimulation, which would cause an underestimation of the neuromuscular blockade.

Limitations

EMG activity may not be present in the face of real injury the following cases: chronically compressed nerve roots (which may require much higher stimulation to fire), sharp complete transection of a nerve root, excessive pharmacologic blockade, and recording electrodes not placed into the myotome corresponding to the nerve in question. For these reasons it is advisable to test a positive control (i.e. the exposed nerve root) before stimulating a screw[50].

BRAINSTEM AUDITORY EVOKED POTENTIALS

Definition: Auditory evoked responses are generated by direct stimulation of the cochlea with click noises.

Utility

BAER are used to monitor cranial nerve VIII function during acoustic neuroma surgery and is associated with better preservation of hearing in patients with good hearing prior to surgery[55]. BAER can also be used to monitor CN VIII during CP angle tumor resection, decompression of CN VII, V, and

aneurysm clipping in the brainstem where small case studies have suggested its ability to detect hypoperfusion[56]. It can also be used to detect brainstem function in comatose patients.

Anesthetic considerations

There is minimal effect of anesthetic technique on BAER response. Even planes of anesthesia causing an isoelectric EEG are compatible with BAER response[56]. BAER are mildly sensitive to extremes of physiology: temperature, hypoxia, and hypotension.

Limitations

Inadequate responses may be the result of soaking or dislodgement of the insert which sits in the auditory canal or use of ultrasound aspiration devices[56]. Auditory evoked potentials are also unusable in patients who are deaf. They are affected by *auditory masking* which occurs when there is a loud noise near the patient (i.e., drilling) that prevents the patient's brain from processing the auditory stimulus.

VISUAL EVOKED POTENTIALS

Definition: Visual evoked potentials (VEPs) are elicited by flash stimulation of the retina via plastic goggles or contact lenses and recorded over the scalp centrally and over the occipital and parietal regions.

Utility

VEPs vary with stimulus, part of the retina stimulated, degree of pupil dilation, and the patient's attention level in awake patients. While VEPs are seldom used in the operating room, they are sensitive for compression of the optic nerve[57]. Outside of the operating room this monitoring technique can be used to confirm a diagnosis of multiple sclerosis[58].

Anesthetic implication

Similar to other potentials recorded over the cortex, visual evoked potentials are extremely sensitive to anesthetic agents.

Limitations

The technical aspect of performing visual evoked potentials can be difficult and previously VEPs have been considered highly variable and therefore intraoperative changes not specific for injury. Hence VEPs are the least commonly used evoked response monitoring technique intraoperatively. However, recent studies have asserted that VEPs may be more reliable than previously thought[57].

OTHER FACTORS AFFECTING MONITORING

Maintenance of a steady neurophysiologic condition is the primary way in which the anesthesiologist can facilitate neuromonitoring. Maintenance of blood flow is extremely important for SSEP and MEP monitoring. Similar to cortical EEG, signals are depressed at 20 cc/100 g/min and lost between 15-18 cc/g/min. In the operating room, the anesthesiologist generally do not directly measure blood flow or tissue oxygenation. Blood pressure is a crude surrogate of blood flow and may be influenced by systemic factors, like a right-shift of autoregulation curves secondary to poorly treated hypotension or local factors like compression by positioning (e.g. brachial plexus injury), spinal hardware, retractors, or clamps. Therefore it is not always possible to define a «safe» intraoperative blood pressure. A decrement in MEP/SEP signals in the face of a decline in blood pressure without a change in anesthetic technique should be considered to be due to a clinically significant drop in blood flow. It these cases, the surgeon should be informed about the change, the blood pressure raised by use of vasopressors, patient positioning optimized, and any contributing surgical factors considered. Increased intracranial pressure is another reason for decrement of signals which may be due to decrease in perfusion.

Evoked signals are also sensitive to anemia. Paradoxically an increase in amplitude is observed in mild anemia, moderate anemia is associated with an additional mild prolongation in latency. Significant depression of SSEPs occurs at extremely low hematocrits (<10 mg/dL)[59]. Hypoxia results in signal decrement, as does hypocarbia through a mechanism involving vasoconstriction. The effects of hypothermia (increased latency, slowing of conduction) can be a result of systemic hypothermia or because of local irrigation with cold solution. Significant electrolyte disturbance could also change neural conduction, which would affect evoked potential signals.

CONCLUSION

In summary, modern neuromonitoring may consist of multimodal use of SSEP, MEP, EMG, BAER, and VEP. Tests are generally chosen to monitor the structures at risk during a surgical procedure. Although not mandatory, use of intraoperative neuromonitoring is associated with improved surgical outcomes for many procedures. The anesthesiologist should strive to understand the indications for test selection, how to optimize signal attainment, and how to respond to an intraoperative change in signals. Through communication with the neuromonitoring team and the surgeon the anesthesiologist can play an important role in using these tests to maximize postsurgical outcomes.

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