Anesthesia and Neuromonitoring:
Electroencephalography and Evoked Potentials

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Anesthesia and Neuromonitoring (EEG & EP)

Patients undergoing neurologic/orthopedic procedures involving the peripheral and central nervous system may be at increased risk from hypoxia/ischemia to vital neurologic structures. Intraoperative neuromonitoring may improve patient outcome by:

    a. Allowing early detection of ischemia/hypoxia before irreversible damage occurs

    b. Indicating the need for operative intervention (shunts placed in carotid surgery) to minimize nerve damage

The role of anesthesiology in neuromonitoring is one of understanding the appropriate anesthetic techniques, applying knowledge of medicine, surgery, physiology and pharmacology to get the best possible outcome. This monograph will discuss the various clinically important neuromonitors and offer solutions as they apply to clinical anesthesia. It is divided in 3 broad sections: Electroencephalography, sensory evoked potentials and motor evoked potentials.

Electroencephalography (EEG)

Electroencephalography or EEG is the summation and recording of postsynaptic potentials from the pyramidal cells of the cerebral cortex. The EEG is typically classified by frequency. The EEG can be recorded off the scalp and forehead using surface and needle electrodes. EEG can take the following forms:

1. Raw EEG
2. Computer processed EEG
    a. CSA (Compressed spectral array): we have this at Upstate
    b. Density Spectral array
    c. Aperiodic analysis
3. Bispectral Analysis (BIS)

Indications for EEG

1. Craniotomy for cerebral aneurysm clipping when a temporary clip is used
2. Carotid Endarterectomy (under general anesthesia)

3. Cardiopulmonary bypass
4. Extracranial-intracranial bypass procedures
5. Pharmacologic depression of brain for "cerebral protection"

What does the EEG tell us:

The EEG reflects metabolic activity of the brain. Metabolic activity of brain cells requires energy. Problems or alterations with energy production (increased demand or reduced supply) by brain cells can profoundly affect EEG activity. Depression of cerebral blood flow, depressed oxygen or glucose delivery will depress the EEG. Other factors adversely affecting the EEG are hypotension, hypothermia as well as all volatile anesthetics, N_2O and most IV anesthetics.

Table 1: EEG waves

Wave	Frequency	Explanation
Beta	13-30HZ	High frequency, low amplitude, awake state
Alpha	9-12HZ	Medium frequency, higher amplitude, awake but eyes closed (EEG seen in occipital lobes)
Theta	4-8HZ	Low frequency; seen under general anesthesia
Delta	0-4HZ	Very low frequency, depressed functions (coma, deep anesthesia, hypoxia, ischemia, infarction, poor metabolism)

Table 1 shows the frequency and characteristic of different EEG waves. Awake EEG (beta activity) can rapidly progress to alpha, theta and delta with onset of ischemia/hypoxia or other factors cited above. Continued insult can cause suppression of electrical activity with an occasional burst of activity (burst suppression). This will eventually lead to electrical silence (flat EEG).

Figure 1 Alpha, Beta and Delta waves
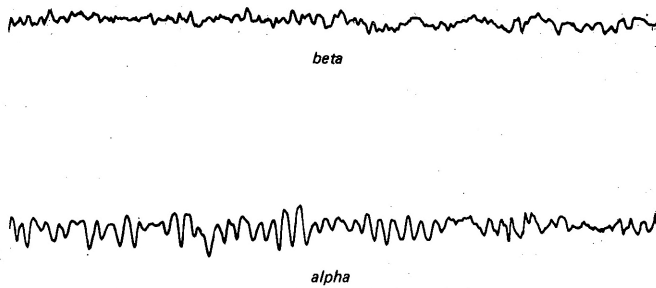

beta

alpha

FIGURE 14.1   Alpha activity (relaxed) and beta activity (aroused, alert) from the EEG.

Example of beta and alpha waves on the EEG [i]

Below are delta waves[ii]

Table 2	Inhalation Anesthesia and EEG	
Dose (MAC)	Anesthetic Agent	EEG Pattern
1.0	Isoflurane, Desflurane, Sevoflurane, Halothane	Limited Beta activity
1.5	Isoflurane ------------> Desflurane, Sevoflurane, Halothane ------------>	Burst Suppression Limited Alpha activity
2.0	Isoflurane ------------> Desflurane ------------> Sevoflurane ------------> Halothane ------------>	Electrical Silence Burst Suppression, Electrical Silence Delta &Burst Suppression Theta and delta

Sudden development of delta, burst suppression (See figure 2) or electrical silence warrants immediate investigation by the anesthesiologist and surgeon to address any immediate correctable risk factors. As we have seen, ischemia and hypoxia can cause severe depression of the EEG but the same changes can be seen with anesthetics. The anesthetic (see Table 2) and hypothermic effects on EEG are reversible. On the other hand, the patient can be at risk from a surgical intervention such as improper instrument placement causing real neural injury. Burst suppression can be recognized on the EEG as 3 seconds of brain activity followed by 7 seconds of electrical silence.

It is vital that we strive for a constant level of anesthetic to avoid misinterpreting EEG depression caused by boluses or rapid changes in anesthetic level from true physiologic/pathologic insults to the cortex.

Figure 2: Burst Suppression
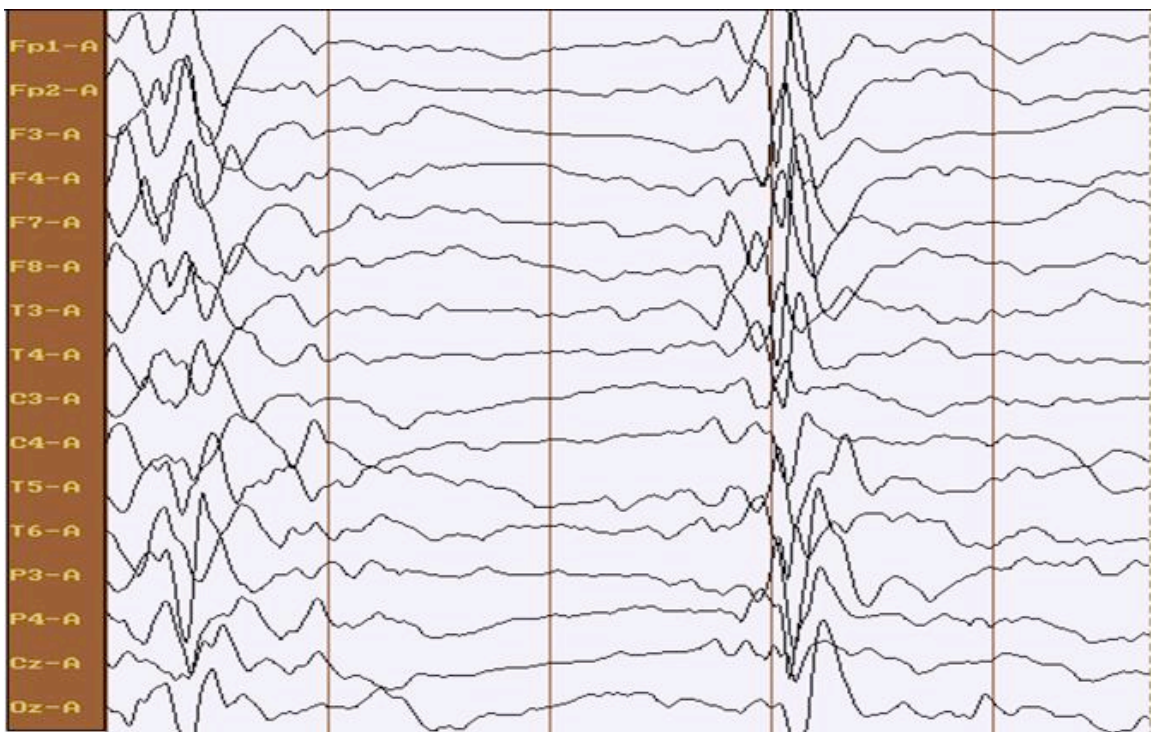
Example of burst suppression[iii]

Click on http://www.gorji.com/neuro/burstsupression.mov to view an example of burst suppression.

Bispectral Index (BIS monitor)

The BIS monitor is a derived EEG parameter used to measure (crudely) the depth of hypnosis under anesthesia. A number above 80 indicates emergence from anesthesia. A value between 40-80 usually implies adequate hypnosis without possible recall. The BIS monitor is placed on the forehead, thus one of its limitations: it is NOT good for detecting regional ischemia except perhaps in the frontal lobe. It is the author's belief that in cases where immobility of the patient is critical and muscle relaxants cannot be used, a BIS value of 30-40 is needed. Many other institutions follow this guideline as well.

Anesthetic technique for a patient requiring EEG

It is not possible to provide one single anesthetic regimen for a patient being monitored by EEG. Most anesthetics (except Ketamine) cause dose dependant decrease of EEG frequency and increase in amplitude. This includes volatile agents, propofol, barbiturates, benzodiazepines and narcotics. The anesthetic goals for a patient undergoing a carotid endarterectomy are very different from a patient having an aneurysm clipped. However most patients who are being monitored by EEG can benefit from an stable anesthetic level that comprises some or all following in various combinations:

1. Isoflurane, desflurane, sevoflurane: 0.5-1.0 MAC*
2. N_2O can be used (up to 70%) or avoided all together
3. Fentanyl infusion of 0.5-3 mcg/kg/hr
4. Morphine loading dose 0.1-0.3 mg/kg
5. Propofol (50-300 mcg/kg/min)*
   - Titrate to EEG state desired

   Avoid rapid changes in anesthetic levels. If such is unavoidable, inform surgeon and neuromonitoring personnel ASAP.

Anesthetic drug dosing during clamping of major vessels such as during carotid endarterectomy and cerebral aneurysms requires close timing and monitoring of the drug effects on the EEG. Currently the 2 drugs used most commonly for burst suppression during cerebral aneurysm clipping are propofol and thiopental. Doses for these drugs are:

      a. Propofol: 50-300 mcg/kg/min drip or 10-50 mg boluses

      b. Thiopental 25-150 mg boluses to keep the EEG silent (doses as high as 30mg/kg have been administered over 1-2 hours)

For example: You begin to administer propofol or thiopental to get burst suppression. You can administer a loading dose of the drugs (thiopental 50-100 mg, propofol 50-100 mg). At the same time you observe the EEG slowing but still exhibiting delta waves. A smaller dose is given and burst suppression is achieved. As time passes by, you begin noticing an EEG pattern reflecting more brain activity. You give a smaller dose of thiopental (50 mg) or propofol (30-50 mg) and EEG burst suppression is achieved once again. This pattern of dosing continues until burst suppression is no longer needed. As more drug is given to the patient, the frequency and dose of thiopental and propofol will likely decrease. The use of etomidate has not been well studied so its use as an infusion is not recommended. In addition, repeated long-term etomidate use has been associated with adrenal suppression.

Close communication between surgeon, anesthesiologist and neuromonitoring personnel ensure correct timing and avoidance of excessive dosing of medications. Overdosing a patient secondary to poor timing can lead to excessive dosing producing postop respiratory depression, which will require ventilatory support.

Evoked Potentials

There are many types of evoked potentials. There are sensory evoked potentials as well as motor evoked potentials.

Sensory Evoked Potentials (SEP)

SEP can be subdivided as follows:

      a. SSEP: Somatosensory Evoked Potentials

      b. Auditory Evoked Potentials (AEP)

      c. Visual Evoked Potentials

SSEP: SSEP **(**example figure 3) is a very common form of neuromonitoring. It is recorded in response to stimulation of a cranial or peripheral sensory nerve. Common nerves stimulated are median, ulnar and posterior tibial nerves. SSEP attempts to ascertain integrity of the sensory pathway specifically the dorsal columns of the spinal cord. SSEP can also be used during carotid endarterectomy surgery to evaluate subcortical ischemia (remember EEG looks at cortex only).

Figure 3 Common SSEP.[iv]
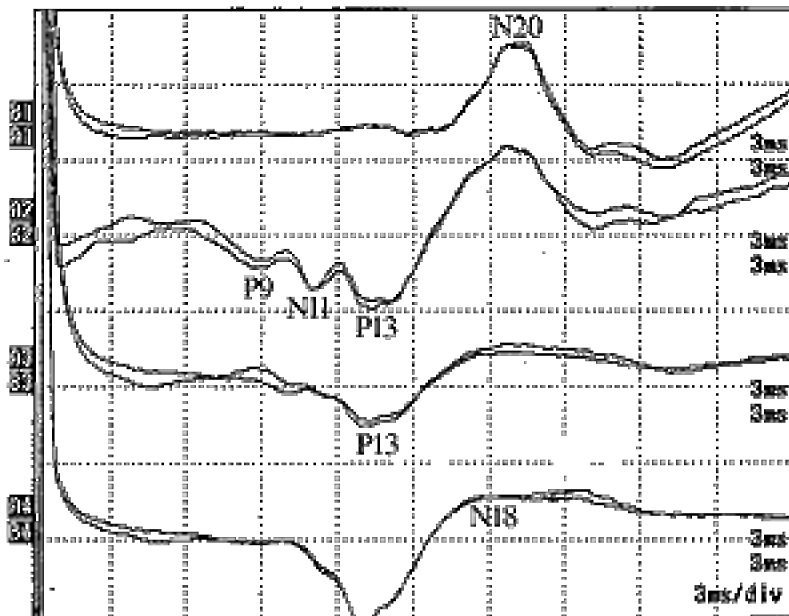
The anesthetic technique for a patient undergoing SSEP usually involves 0.5-1.0 MAC of volatile agent and a narcotic infusion for longer cases. Narcotic boluses can profoundly affect the SSEP- at least transiently. Informing the

neuromonitoring techs is important if boluses are planned during the operation. N_2O up to 50% can be used if baseline SSEP waves are not severely compromised. If the baseline SSEP is poor, the addition of N_2O may make the SSEP uninterpretable.

AEP:

A typical AEP[v] is shown:
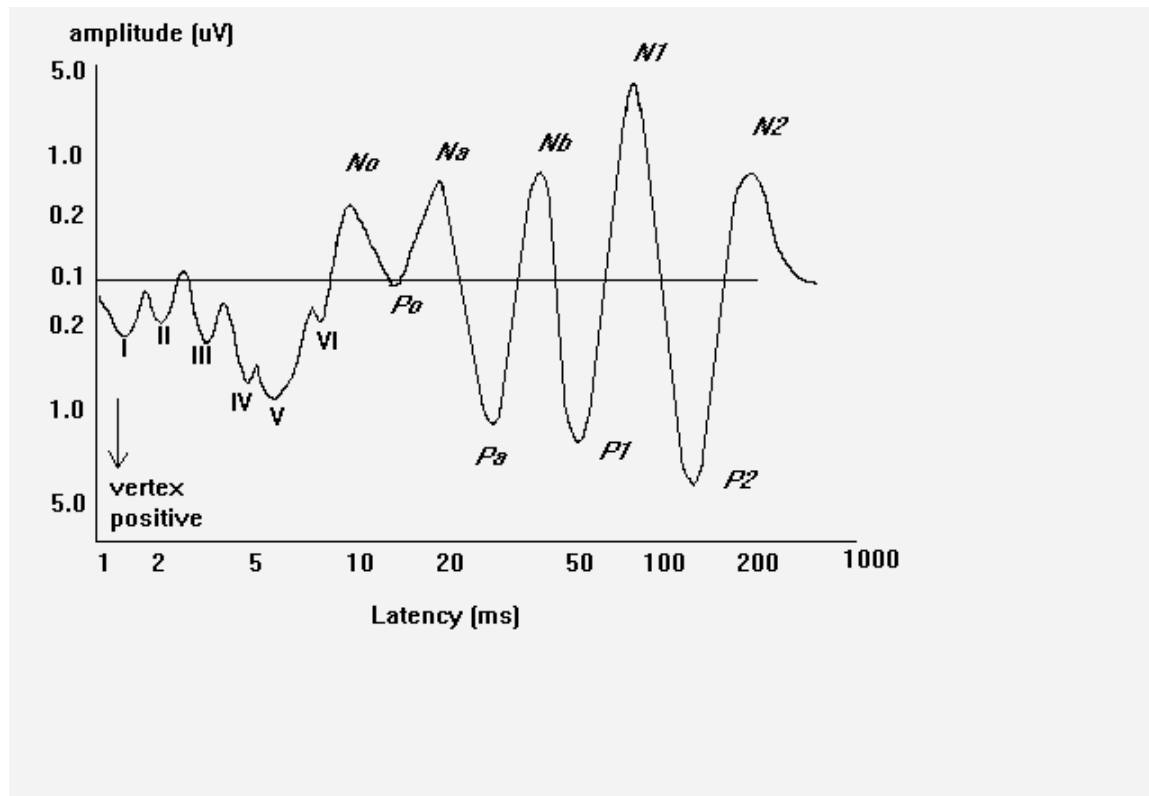
AEPs are used to monitor the integrity of cranial nerve 8 such as during resection of acoustic neuromas. Fortunately, AEPs are resistant to the effects of anesthetic agents so there a no special anesthetic requirements during these procedures.

VEP:

VEPs are seldom used clinically. During VEP monitoring, light is flashed into the patient's eyes, and recordings are taken off the occipital area. VEPs are sensitive to anesthetic agents. An anesthetic similar to SSEP would be appropriate.

Motor Evoked Potentials (MEP)

Motor evoked potentials (MEPs) are useful when the common sensory and somatosensory evoked potentials (SSEP) fall short of adequate monitoring as

well as for specific situations where pure motor function needs to be monitored. In MEP monitoring, motor pathways are assessed directly and avoid one of the major limitations of SSEPs; the inability of SSEPs to determine the integrity of motor neurons. In a strict sense, MEPs measure the integrity of the motor neuron output. MEPs are an outcome measured: ie: A nerve is stimulated and an outcome is measured from a muscle or a group of muscles. The following areas of motor monitoring are relevant clinically and will be discussed below:

    a. Direct spinal cord stimulation
    b. Transcranial motor evoked potentials (tcMEP)
    c. Cortical motor evoked potentials (cMEP)
    d. EMGs
    e. Other motor monitoring

Basic Theory of motor evoked potentials

Theoretically, motor evoked potentials are obtained and monitored using the same methodology as SSEPs. Thus the equipment used for MEP is similar to SSEPs. Stimulus sites include a peripheral nerve, spinal cord or the motor cortex (either directly or thru the scalp). Stimulation of the motor cortex thru the scalp is called transcranial motor evoked potential (tcMEP). Cortical motor evoked potentials (cMEP) are when the motor cortex is stimulated directly (ie: thru a craniotomy). This distinction is not that important for anesthesiologist, as the anesthetic management does not change. Evoked responses may be recorded over many sites such as the spinal cord, peripheral nerve (including cranial nerves) or muscle tissue. Potentials collected from the muscle are called myogenic potentials and are part of a pure electromyogram (EMG). An EMG can be a MEP but more often it is not. Some EMG potentials are spontaneous (spontaneous EMG) while some are triggered by a response (triggered EMG). The EMG will be discussed in the following sections.

Direct spinal cord stimulation

Direct spinal cord stimulation is rarely used at our institution and will not be discussed here. Virtually all anesthetic agents can be used when direct or indirect stimulation of the spinal cord occurs. The spinal cord is resistant to the effect of anesthetics during recording and stimulation.

Transcranial and Cortical motor evoked potentials (TcMEP and cMEP)

TcMEP and cMEP are some of the newest modalities used in neuromonitoring and will be discussed at the same time. In this type of monitoring, the cortex is stimulated directly (cMEP) or thru the scalp (tcMEP) over the motor cortex and responses are elicited in the appropriate area. For example the cortical area involving the upper extremity is stimulated and motor potentials are recorded from the appropriate hand or fingers.

Anesthetia for tcMEPs and cMEP monitoring

Not all anesthetics can be used with MEPs. Transcranial motor evoked potential stimulation as well as cMEPs are incompatible with virtually ALL commonly used volatile anesthetic agents.[vi], [vii] In fact, 0.2-0.3 MAC of volatile agent can abolish both tcMEP and cMEP. The solution lies in the use of total intravenous anesthetics, namely propofol or etomidate. The use of these two drugs in combination with narcotics allows for stable recording of MEPs.[viii], [ix] Thiopental infusion is not favored as it causes profound postop respiratory depression. The use of etomidate as an insfusion has not been studied well and is not currently recommended. In addition, repeated long term etomidate use has been associated with adrenal suppression. Below are some basic guidelines for clinical anesthesiologists taking care of patients undergoing neuromonitoring. Each case has to be individualized, as patient's responses are variable to anesthetics. Please use good clinical judgment and communicate early and often with the surgeon and neuromonitoring staff.

Suggested Anesthetic agents for TcMEP* and cMEP

1. Induction: All commonly used induction agents
2. Paralysis for intubation: succinylcholine, mivacurium or small dose of an other short acting nondepolarizer
3. Maintenance:

    a. TIVA: Propofol Infusion (75-300 mcg/kg/min)

    b. N_2O allowed up to 70%

    c. Narcotic Infusion

        i. Fentanyl 1-3 mcg/kg/hr (titrate to lower range with time as drug will accumulate over time)

        ii. Remifentanil 0.05-0.2 mcg/kg/min

        iii. Alfentanil 10-30 mcg/kg/hour

        iv. Morphine sulfate 0.1-0.3 mg/kg does according to length of operation and other anesthetic/patient factors

   v. Avoid ALL Neuromuscular blockers unless patient safety necessitates use

4. Adjuncts:

    a. The use of the BIS monitor is highly recommended for cases that will take longer than 4 hours. It can be used for shorter cases as well. The BIS monitor is used to titrate the infusion of drugs to appropriate levels facilitating awakening in a timely manner. If you look at the context specific graphs below you will see that intravenous agent accumulation occurs with most drugs as infusion duration increases. The same is true with volatile anesthetics.[x] The BIS monitor can be used to help maintain a constant hypnotic level of propofol during the operation. Please refer to the discussion of the BIS monitor above. As the operation progresses, one can see that the infusion needs to be titrated to a lower level in order to avoid the problem of prolonged awakening times. For further information about context specific half-lives, please consult a textbook of anesthesia.

Comparison - anaesthetics
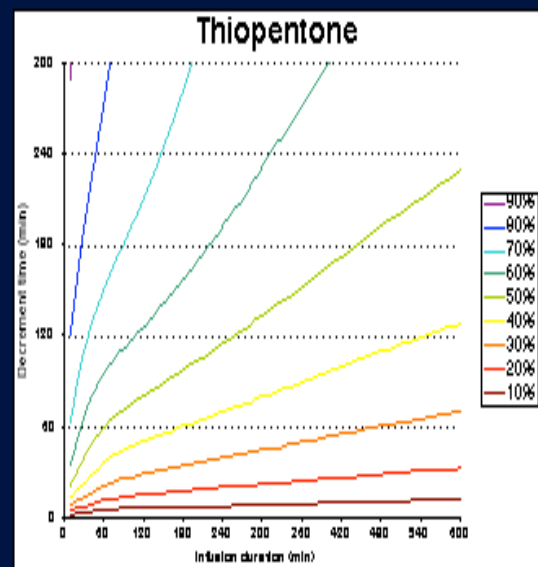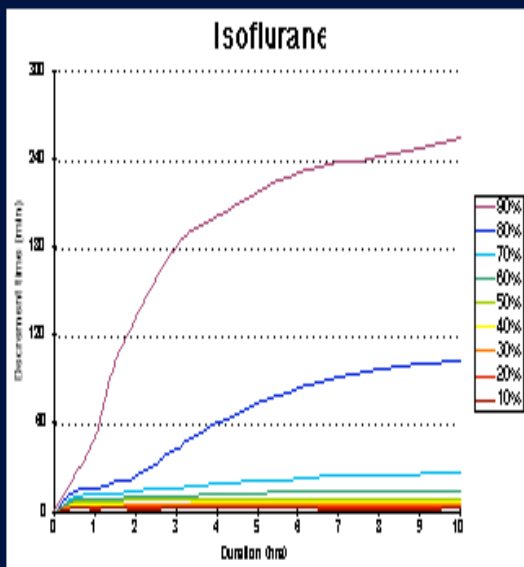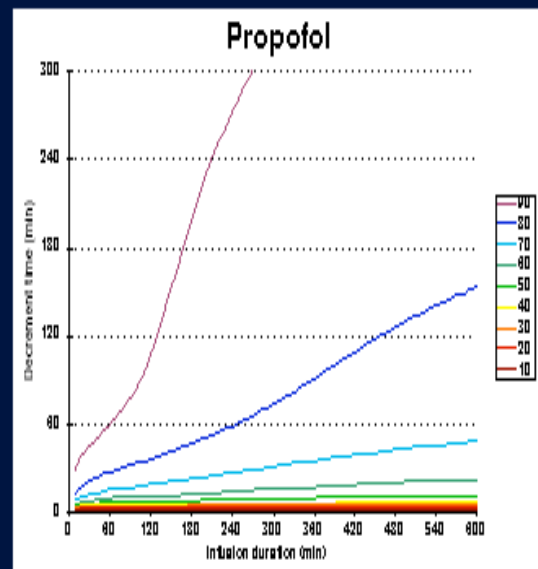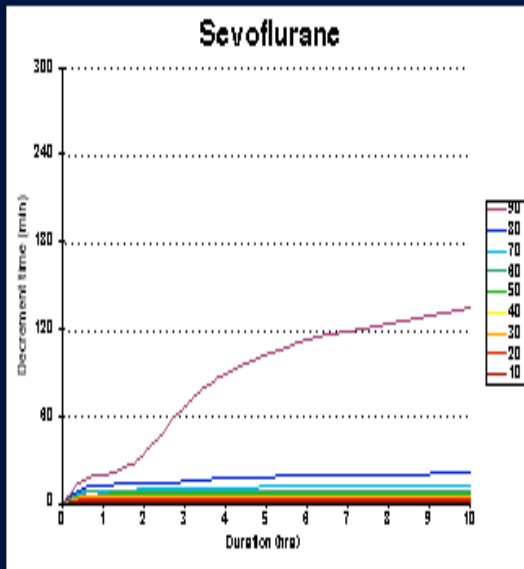
Comparison of narcotics

Alfentanil

Fentanyl

Remifentanil
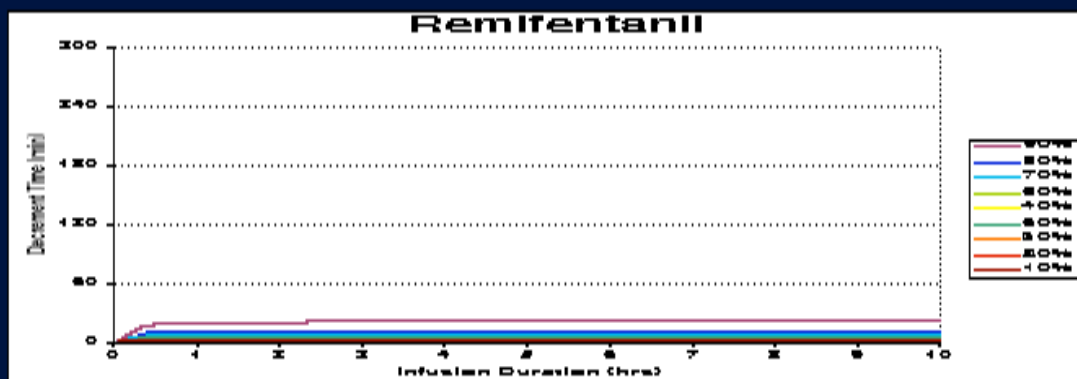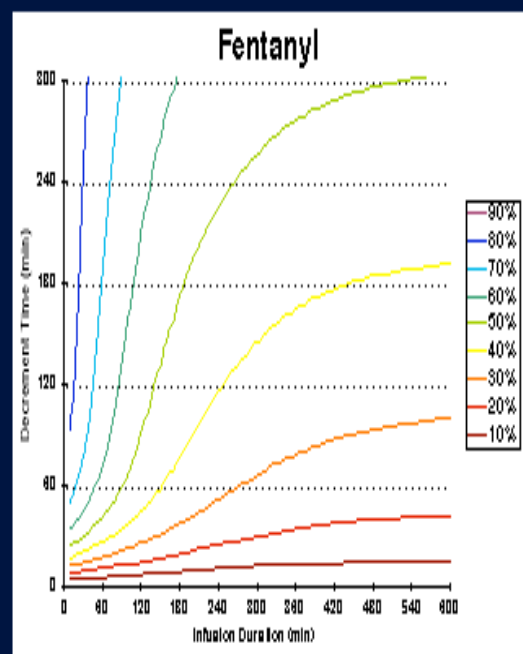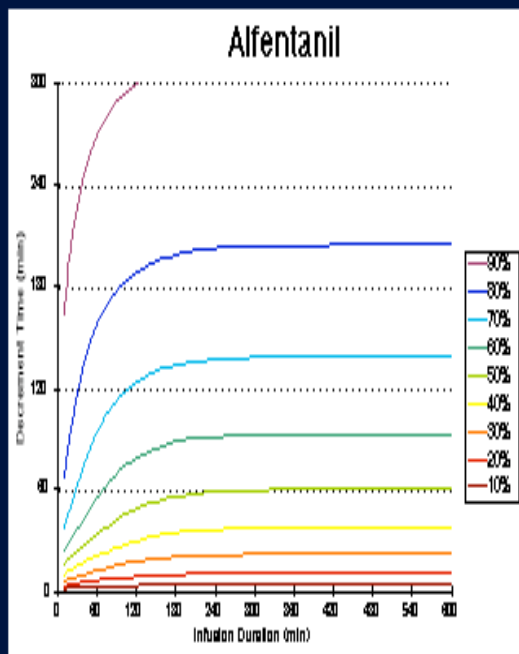
5. Emergence

    a. Timely emergence from anesthesia is very important for surgeons. Facilitation of neurologic monitoring at end of case helps prevent potential spinal cord/nerve damage

    b. Decision to have the patient awake at the end of the case should be based upon standard anesthetic criteria. If these criteria are not met (acidosis, large blood loss and transfusions), then awakening does NOT take priority and should be delayed.

* Please remember that patient safety takes priority over any type of monitoring but the monitoring is done to increase patient safety.

EMG

Electromyography (EMG) is a special type of motor monitoring. EMGs are further classified into 2 distinct categories. They are:
1. Spontaneous EMG (Not an MEP)
2. Triggered EMG (ie: nerve root stimulation; a MEP). As you can see the definitions between a MEP and EMG can be unclear.

When spontaneous potentials are measured from a muscle we are not dealing with a MEP. There is no stimulus so we can't measure a response. ON the other hand when a nerve root or facial nerve is stimulated by the surgeon, one can measure and quantitate a response and gauge this to a response comparing it to a baseline. Fortunately it is not important for the anesthesiologist to know the distinction the different types of EMGs. In summary, EMGs (either type) are outcome measurements. An EMG is measurement, while the MEP is a measured response. Thus a triggered EMG is a MEP.

Suggested Anesthetic agents for EMG (both types of EMG)
1. Induction: All common drugs used for induction
2. Intubation: Short acting muscle relaxants
3. Maintenance:
    a. Volatile agent (no upper limit of MAC) or TIVA

b. N_20 allowed up to 70%

c. Narcotic infusion or boluses (same precautions as the section on SSEPs)

d. Neuromuscular blockers: Ideally none is used past intubation. If that is not possible, titrate muscle relaxant to a TOF of > 3/4.

Please remember that patient safety takes priority over any type of monitoring but the monitoring is done to increase patient safety.

Facial nerve Monitoring (FNM) as an example of EMG

Operations involving the posterior fossa especially, excision of acoustic neuromas and operations at the base of the skull may result in damage to the facial nerve leading to facial weakness or worse paralysis. For facial nerve monitoring, EMG electrodes are placed in the obicularis oculi and obicularis oris muscles. The facial nerve is stimulated and EMG activity is recorded from the muscles mentioned. Often the EMG response is also displayed/converted to audio signals that provides immediate feedback to the surgeon. In addition surgical manipulation of the nerves (for example retraction of he nerve) will cause electrical activity in the nerve and help the surgeon avoid potential damage to the nerve.

The seventh nerve is not the only nerve that can be monitored. Other cranial nerves can be monitored in a similar fashion. Anal sphincter tone is one such example.

Other motor monitoring

Finally I want to mention that the field of neuromonitoring is blossoming. Newer forms of monitoring will be introduced which will require us to adapt to situations we are not familiar with at present. For example, recently we have started monitoring the vagus nerve with specially designed endotracheal tubes. These tubes have electrodes on their surface that monitor the vagus nerve innervation of the airway.

Quick Reference Guide to Neuromonitoring and Anesthesia*

Monitoring		Type of Anesthesia	
		Agent	**Dose**
Auditory Evoked Potentials	No limitations	-	-
Visual Evoked Potentials	Similar to SSEP	-	-
Somatosensory Evoked Potentials (SSEP)		Volatile Agent	0.5-1.0 MAC acceptable
		N_2O	50-70% if baseline SSEP not compromised
		IV anesthetics	Fentanyl 1-2 mcg/kg/hr
		Neuromuscular Blockers	No limitations
Spinal Cord Stimulation	Similar to SSEP	-	-
Motor Evoked Potentials (EMG)		-	-
		Volatile Agent	No limitations
		N_2O	No limitations
		IV anesthetics	No limitations
		Neuromuscular Blockers	Limited use; try to avoid**
TcMEP and cMEP (BIS recommended esp. in long cases)		Volatile Agent	Limited use; 0.3 MAC maximum
		N_2O	50-70 % acceptable
		IV anesthetics	Propofol 50-300 mcg/kg/min
		Neuromuscular Blockers	Very limited use*

<u>* Tailor the anesthetic to patient clinical needs and safety concerns</u>
<u> Use only if patient safety outweighs monitoring needs</u>**

Summary

The care of patients requiring neuromonitoring can be simple or complex and taxing. Close communications with the surgeons and neuromonitoring staff will facilitate an atmosphere of mutual respect and understanding. I hope that by reading, understanding and applying the materials in this paper, you will be able to improve the quality of care to patients requiring neuromonitoring. By providing specialized monitoring to our patients we are at the forefront of excellence in patient care. If you have suggestions on improving the contents of this paper, please do not hesitate to contact me at anytime.

Reza Gorji 9.29.2005

[i] http://cti.itc.virginia.edu/~psyc220/alpha-beta.jpg

[ii] http://www.epilepsyhealth.com/delta.gif

[iii] http://www.dx-telemedicine.com/rus/publications/eeg_paternu/image021.jpg

[iv] http://www2.oninet.ne.jp/ts0905/onepoint/oitem/nbssep.gif

[v] http://www.cf.ac.uk/biosi/staff/jacob/teaching/sensory/aep1.gif

[vi] Zenter J, Albercht T, Heuser D: Influence of halothane, enflurane and isoflurane on motor evoked potentials. Neurosurgery 31:298, 1992.

[vii] Zenter J, Kiss I, Ebner A: Influence of anesthetics-nitrous oxide in particular – on electromyographic response evoked by transcranial electrical stimulation of the cortex. Neurosurgery 24:253, 1989.

[viii] Jellinek D, Platt M, Jewkes D et al: Effects of nitrous oxide on motor evoked potentials under total anesthesia with intravenously administered propofol. Neurosurgery 29:558, 1991.

[ix] Kallman CJ, Drummond JC, Ribbernick AA et al: Effects of propofol, etomidate, midazolam, and fentanyl on motor evoked responses to transcranial electrical and magnetic stimulation in humans. Anesthesiology 76:502, 1992.

[x] http://www.usyd.edu.au/su/anaes/lectures/contextsens/noframe.htm