

Neuromonitoring

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Introduction

Neuromonitoring is used during surgery to assess the functional integrity of the brain, brain stem, spinal cord, or peripheral nerves. The aim of monitoring is to prevent permanent damage by early intervention when changes are detected in the monitor.^{1,2} Neuromonitoring is also used to map areas of the nervous system in order to guide management in some cases.

The best neuromonitor remains the awake patient. In the conscious state, the function of individual parts of the nervous system and the complex interactions of its different parts can be assessed more accurately. However, most surgical procedures involving the nervous system require general anaesthesia.

Procedures that require neuromonitoring can have changes in their monitored parameters corrected by modifying the surgical approach or by having the anaesthesiologist manipulate the parameters under their control.^{1,2} An ideal neuromonitor would be one that is specific for the parameter of interest, and gives reliable, reproducible, or continuous results.

Monitoring modalities

The most commonly used electrophysiologic monitoring techniques are:

- Electroencephalography (EEG)
- Processed electroencephalography (EEG)
- Electromyography (EMG)
- Somatosensory evoked potentials (SSEPs)
- Brainstem auditory evoked potentials (BAEPs)
- Visual evoked potentials (VEP)
- Motor evoked potentials (MEPs)

Electroencephalography

EEG records spontaneous electrical activity in the cerebral cortex and can detect seizure activity, inadequate blood flow, assess the anaesthetic agent's impact on the brain, as

well as assess conscious states. Electrodes are often placed on the scalp in a standardised radial and axial array as defined by the 10/20 system prior to surgery.¹ Intraoperatively EEG electrodes are placed on the surface of the brain or from microelectrodes placed transcortically during awake brain mapping, seizure surgery as well as for selected tumour resections.^{2,3}

A normal EEG has symmetric patterns with regards to frequency and amplitude for both asleep and awake patients. These are recorded from electrodes on each hemisphere. Asymmetric patterns reflect either metabolic or anatomical alterations in the brain and produce characteristic voltage spikes and waves. These can be seen in epilepsy, cerebral ischaemia or infarction, and sometimes produced by tumours. Reductions in cerebral blood flow produce rapid, characteristic changes in the EEG.^{1,4} With ischaemia, progressive decrease in synaptic activity results in loss of high-frequency activity, loss of power, and ultimately EEG silence. The severity of neurologic injury and expected EEG changes with decreasing cerebral blood flow are shown in a table (Figure 1).

Processed electroencephalography

This refers to translation of raw digitalised EEG signal from the time domain to the frequency domain, which is component sine waves of identifiable frequency and amplitude via fast

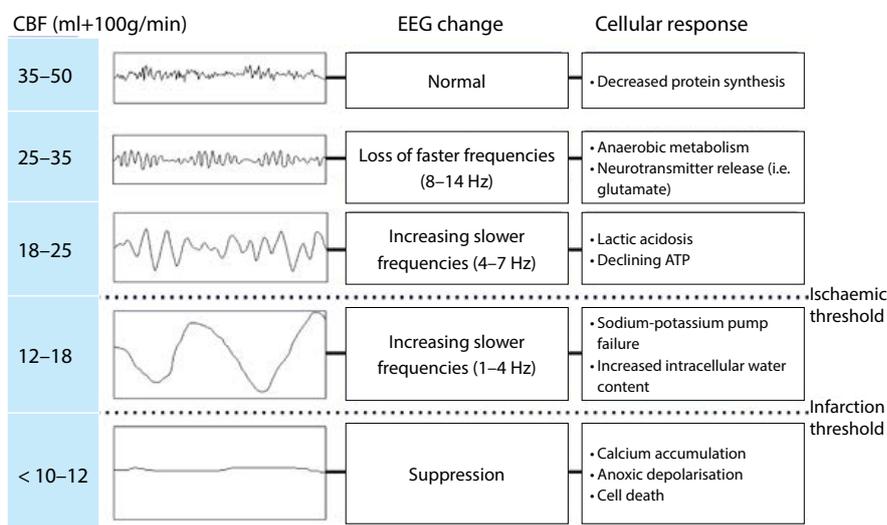


Figure 1: EEG changes with decreasing cerebral blood flow⁵

Fourier transformation.⁶ The analysis of the contribution of each frequency band results in a frequency spectrum displayed either as compressed spectral array or density spectral array. Characteristics of this spectrum are then analysed to assess anaesthetic depth. Anaesthesia and surgery may produce alterations which are reflected as changes in amplitude and frequency.^{1,7} Commercially available devices include bispectral index (BIS), density spectral array, entropy, and cerebral state index monitors, with continuing development of new models.

Electromyography

EMG is used to monitor muscle activity in muscles innervated by cranial or spinal nerves intraoperatively and allows assessment of nerve function and early detection of surgically created nerve damage.¹ Monitoring can be by continuous or evoked compound muscle action potentials (CMAPs). Recordings are either made from surface or needle electrodes, but sensitivity is best from needles directly innervating the muscle of interest. Responses are produced from significant nerve irritation and result in high frequency trains called neurotonic discharges which may indicate impending nerve damage in real time.^{1,2}

Stimulus-triggered EMG (often called "triggered EMG") can be used to assess functional integrity during tumour resection in order to avoid cutting or damaging the nerve. A mono- or bipolar stimulator is used within the surgical site to stimulate the nerve, and a resulting CMAP is recorded.⁶

EMG is commonly used to help prevent postoperative radiculopathy because of a malpositioned pedicle screw in spine surgery involving instrumentation (a more common complication than spinal cord injury), by identifying nerve irritation before injury.^{1,8,9}

Cranial nerve monitoring is only for cranial nerves with a motor component which can be monitored with EMG (cranial nerves III, IV, V, VI, VII, IX, X, XI, and XII). During intracranial tumour surgery, stimulus-triggered EMG can also be used for brainstem and motor-strip mapping. Stimulation can also be used to identify the motor cortex by recording EMG activity from the impacted area (e.g. upper extremity, lower extremity, or face).

Evoked potentials

Evoked potential monitoring is used to assess the integrity of the tested neural pathway by applying a stimulus to the neural tract and a response is evoked that indicates the integrity and functionality of the tract. Measurements are taken to monitor significant peak changes in latency and amplitude.⁹ Somatosensory, visual, and brainstem auditory evoked potentials are used to monitor neurologic structures between peripheral sites where specific stimulations are applied, and responses are recorded from central locations. Motor evoked potentials monitor such structures by stimulating the motor cortex and recording from the epidural space (D-wave) or, more commonly, from distal muscles. Changes in evoked responses

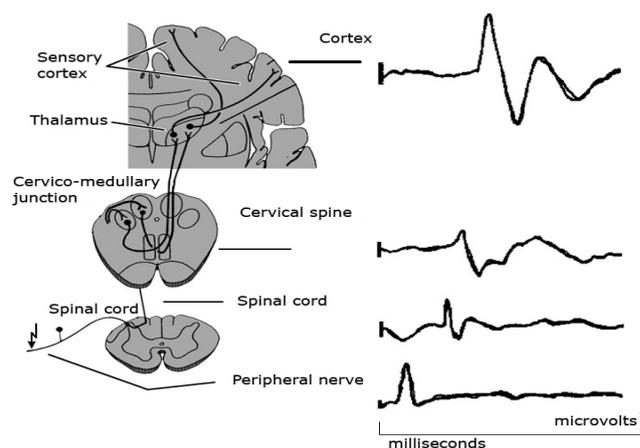


Figure 2: Sensory pathway for stimulation and monitoring of somatosensory evoked potentials¹²

can result from technical, positional, pharmacologic, physiologic, or surgical causes.^{2,10}

Morphology of the recorded waveform depends both on the site used for stimulation and the site used for recording. The amplitude and latency of the waveform can be analysed to provide functional neurologic assessment. Amplitude is measured peak-to-trough in microvolts, and latency is measured from stimulus application to peak appearance in milliseconds. Loss of or change in the waveform can indicate the need for modification of surgical strategy, patient positioning, and/or patient physiologic management in order to prevent or minimise neurologic system injury.^{7,10}

Somatosensory evoked potentials

SSEP is the most commonly used evoked potential monitoring modality in the operating room. An electrical stimulus is applied to a peripheral nerve, typically median or ulnar nerve and posterior tibial nerve for upper and lower limbs respectively to elicit a response.^{2,9} Motor and sensory components of these large, mixed nerves are stimulated. Activation of the motor components results in visible muscle twitches in distal musculature, these are large fast diameter, fast conducting group Ia muscle fibres, while activation of the sensory components, group II cutaneous nerve sensory fibres, results in responses that travel cephalad to the brain.^{6,7,9}

The monitored neural pathway includes the dorsal root ganglia and the dorsal or posterior column of the spinal cord. SSEP monitoring is particularly useful during posterior spine surgery, intracranial, cardiovascular and endovascular surgeries.⁸ Responses can be recorded at other points along the pathway, including over the peripheral nerves and spinal cord, however they are usually recorded over Erb's point, the popliteal fossa, the cervical spine, and the sensory cortex¹¹ (Figure 2).

Brainstem auditory evoked potentials

BAEPs, also known as brainstem auditory evoked responses or auditory brainstem responses, are specialised sensory evoked

potentials produced when the cochlea is stimulated by the sound delivered to the external ear. An acoustic stimulus (a loud, repetitive click) is delivered to the opposite ear. The sound is transduced via the eighth cranial nerve, with information conducted to the brainstem.^{6,9} The response is measured with recording electrodes placed at the top of and near the external ear. The response consists of five main short-latency peaks (I to V) which are usually seen within the first 10 milliseconds after stimulation.^{7,9}

Evaluation of BAEPs usually focuses on latency of peaks I, III, and V, and amplitude of peak V. Wave I is from extracranial portion of cranial nerve VIII, wave III from acoustic relay nucleus in the lower pons, and wave V from near the inferior colliculus (mesencephalon)² (Figure 3). BAEPs are used during posterior fossa surgeries, cerebellopontine angle of brainstem, midbrain and pons.

Visual evoked potentials

VEPs are specialised sensory evoked potentials of the visual pathway recorded from the visual cortex after flash stimulation of the retina through closed eyelids.¹³

Usefulness of VEP monitoring has not been established. Although VEPs are susceptible to effects from general anaesthetics, and technical problems with stimulators make monitoring difficult, newer stimulating methods have been used successfully.

Motor evoked potentials

Electrical stimulation of the motor cortex has emerged as the most commonly used technique that allows motor tract assessment. MEPs can be monitored by magnetic stimulation or, more usually, by transcranial electrical stimulation, either to the scalp utilising two electrodes or by direct stimulation of the surface of the brain directly through a craniotomy.^{6,9,14}

Stimulation of the pyramidal and internuncial cells in the brain generate D (direct) and I (indirect) waves recorded from the epidural space.¹⁴ Motor forms temporal summation of D-waves and I waves activate anterior cells to the lower motor neuron and send the signal to the neuromuscular junction recorded as compound muscle action potentials in peripheral muscle groups.^{2,7} MEPs monitor the corticospinal tract (i.e. motor cortex, corticospinal tract, nerve root, and peripheral nerve).

MEP response can also be monitored using epidural electrode as epidural D-waves.^{8,15} These are recorded from an epidural electrode and are specific for the corticospinal tract. This technique is most often used in intramedullary spinal cord tumour surgery but can also be utilised for mapping of the location of motor cortex and in subcortical tissue and brainstem.

For spine surgery, both MEPs and SSEPs are monitored together and used to monitor spinal cord function to increase sensitivity.⁶

MEPs are monitored frequently during critical surgical maneuvers and periodically throughout surgery. Complete loss of signal is always considered significant although several criteria have been proposed for identifying significant intraoperative change. MEPs are more effective than SSEPs for detecting motor injury since changes in the MEPs precede SSEP changes, usually allowing time to react in order to prevent neurologic damage.^{2,14}

Confounding patient factors like diabetic neuropathy, hypertension, age extremes, and pre-operative motor deficit can make MEP recording difficult. MEPs can however be reliably obtained even in very young paediatric patients with permissive anaesthetic and neuromonitoring stimulating techniques.^{6,9}

Hoffmann reflex

The Hoffmann reflex (H-reflex) is a true reflex with an afferent arc mediated by large, fast-conducting group 1a fibres, and an efferent arc mediated by alpha motor neurons. It is elicited by electrical stimulation of an afferent mixed peripheral nerve

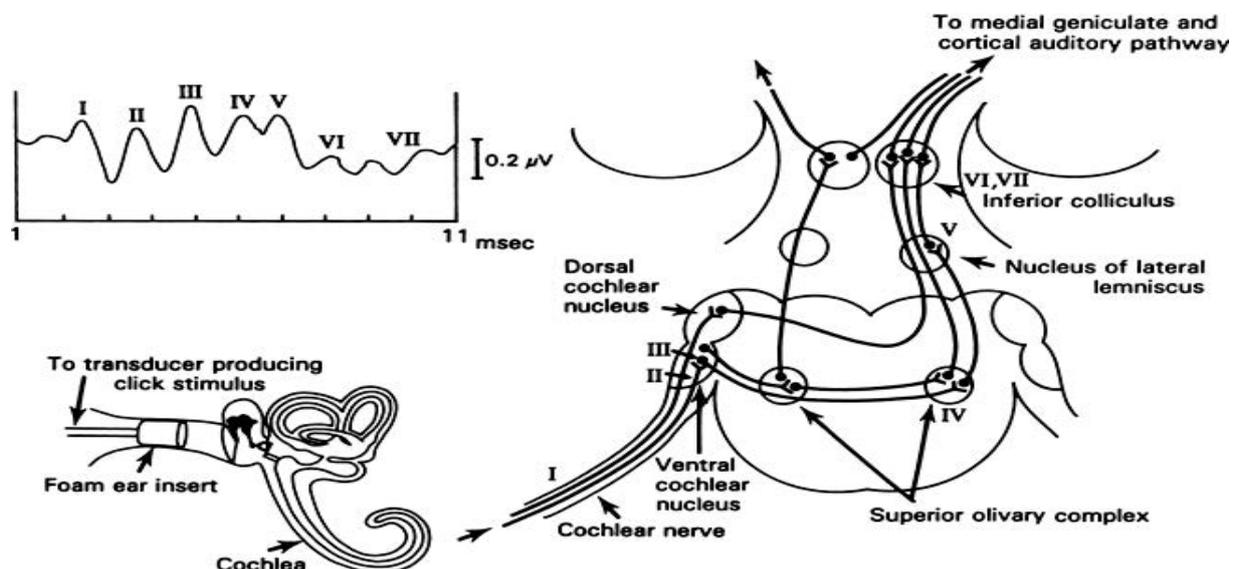


Figure 3: Waves representing evaluation of BAEPs ©Neurones.co.uk

and by then recording the muscle response.^{9,14} The H-reflex is dependent on normal cephalad spinal cord functioning. It monitors the pathway of the reflex and may be suppressed by injury of more cephalad motor tracts. The sensitivity and specificity of H-reflex monitoring have not been fully explored.

Anaesthetic effects on neuromonitoring

Most anaesthetic agents alter neural function by producing dose-dependent depression in synaptic activity.¹ All anaesthetic agents affect the EEG; this is the basis for the use of many monitors of the impact of anaesthesia on the brain.¹⁵ MEP muscle responses and cortical potentials are the most affected by anaesthetic agents. It is important for the neurophysiologist who interprets the EEG to know which drugs and doses are administered, thus the choice and dose of anaesthetic agents should be tailored to the modalities used.^{2,8,9} Effects of commonly administered anaesthetic medications on SSEP and MEP monitoring are shown in a table (Table I).

Table I: Effects of commonly administered medications on SSEP and MEP

Agent	Cortical SSEPs latency	Amplitude	MEPs amplitude
Volatile agents	↑↑↑	↓↓↓	↓↓↓
Nitrous oxide	↑	↓↓	↓
Barbiturates	↑↑	↓↓↓	↓↓
Propofol	↑↑	↓↓	↓↓
Benzodiazepines	↑	↓	↓↓
Narcotics/opioids	±	±	±
Ketamine	↑	↑	±
Etomidate	↑	↑↑	↑
Muscle relaxants	0	0	↓↓↓

↑↑↑ – Increase, ↓↓↓ – decrease, ± – little or no change, 0 – no change

Physiologic effects on neuromonitoring

Physiologic alterations can affect electrophysiologic (EP) signals, and the manipulation of physiological parameters can support the patient during surgically caused EP changes.⁶

Blood pressure

Reduction in systemic and regional or local blood pressure can affect EEG and cortical evoked potential monitoring. The basis for monitoring patients who undergo carotid endarterectomy is the changes associated with reduction in cerebral blood flow (CBF) and possible embolic episodes. Some patients may be at higher risk of ischaemia with any perturbation of blood flow, particularly those patients with baseline abnormal cerebral or spinal perfusion or autoregulation.^{7,9} Thus, when monitoring changes occur, we increase mean arterial pressure in order to increase tissue perfusion pressure.

Temperature

Body temperature has been shown to alter EEG, SSEPs, BAEPs, VEPs, and MEPs.¹⁶ It has been suggested that core body tem-

perature should be maintained within 2–2.5 °C of baseline temperature.⁹

MEP waveforms can be detected during hypothermia to core temperatures of 31–34 °C, with increasing latencies and stimulation threshold below 32 °C. These effects are relevant only for patients who are cooled.¹ Increasing latencies to component peaks may occur as temperature declines because nerve conduction velocity depends on temperature. A 50% reduction in amplitude is an acceptable warning sign.^{15,16} However, regional hypothermia can also affect evoked potential monitoring, such as that caused by a cool extremity after infusion of cold intravenous (IV) solution or by the spinal cord being exposed to cold irrigation prior to instrumentation.^{14,16}

Haematocrit

Anaemia can affect neuromonitoring by either reducing oxygen carrying capacity or changing the rheology of blood. The effect of anaemia on SSEPs and VEPs has been studied using progressive isovolemic haemodilution. Latencies increased with haematocrits of 10–15%, and with those below 10%, amplitudes fell and latencies increased further.⁶

Patient positioning effects

Patient positioning for surgery can cause neurologic and/or vascular compromise and can therefore affect neuromonitoring. In certain patients, when severe neck flexion is required with positioning, such as for certain spine or intracranial posterior fossa surgeries, baseline motor and somatosensory evoked potentials are ideally performed after induction with the patient supine, and then repeated after turning prone (or sitting). Similarly, for patients with unstable cervical spines, signals are ideally obtained pre- and post-positioning. However, in many patients, baseline evoked potentials can be obtained after the patient is positioned for surgery. If recorded potentials deteriorate or are absent, changes to the position can be made before surgery begins.⁶

Conflict of interest

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