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Electrophysiological Monitoring During Brain Surgery Under General Anaesthesia: Principles, Anaesthetic Interactions, and Clinical Practice

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Abstract

Intraoperative neurophysiological monitoring (IOM) has become an indispensable adjunct to neurosurgical procedures performed under general anaesthesia. By providing real-time functional assessment of neural pathways at risk, IOM enables the surgical team to detect and respond to evolving neurological injury before it becomes irreversible. This review article comprehensively examines the principal electrophysiological modalities employed in contemporary neuroanaesthetic practice, including electroencephalography (EEG), somatosensory evoked potentials (SSEPs), motor evoked potentials (MEPs), auditory brainstem responses (ABR), electromyography (EMG), and electrocorticography (ECoG).

A critical focus is placed on the bidirectional relationship between anaesthetic agents and electrophysiological signals. Each modality responds differently to volatile anaesthetics, intravenous hypnotics, opioids, sedatives, and neuromuscular blocking agents, and these interactions fundamentally govern anaesthetic strategy in monitored cases. The physiological variables of temperature, blood pressure, and carbon dioxide tension that additionally confound signal interpretation are analyzed. Validated alert criteria, standardized communication protocols, and the expanding evidence base for IOM-guided outcomes in supratentorial, infratentorial, spinal cord, and epilepsy surgeries are reviewed. Emerging technologies, including high-density EEG, machine learning-assisted signal analysis, and closed-loop anaesthetic delivery, are discussed. Practical recommendations for the neuroanaesthetist, including protocol selection, TIVA regimen design, and team communication frameworks, are provided. Robust evidence demonstrates that multimodality IOM reduces the incidence of permanent neurological deficits and improves surgical decision-making, affirming its role as a standard of care in high-risk cranial and spinal neurosurgery.

Keywords: intraoperative neurophysiological monitoring; IOM; electroencephalography; somatosensory evoked potentials; motor evoked potentials; auditory brainstem response; electromyography; electrocorticography; total intravenous anaesthesia; neurosurgery

Introduction

The fundamental goal of neurosurgical anaesthesia is to provide optimal operative conditions while safeguarding the integrity of neural structures. Brain surgery carries an inherent risk of injury to eloquent cortex, white matter tracts, cranial nerves, and subcortical structures. Historically, the anaesthesiologist and neurosurgeon relied upon postoperative neurological assessment to detect new deficits—an approach that, by definition, precludes real-time intervention. [1]

Intraoperative neurophysiological monitoring (IOM) addresses this limitation by translating the functional state of neural pathways into continuously recorded electrophysiological signals during surgery. Changes in these signals—representing alterations in conduction velocity, synaptic transmission, or axonal integrity—provide an early warning system that allows the surgical team to modify technique, restore perfusion pressure, adjust anaesthetic depth, or change patient position before structural injury is complete. [2] [3]

The practice of IOM has expanded dramatically since the 1970s, when Tamaki and Raudzens first documented the utility of somatosensory evoked potential monitoring during spinal surgeries. [4] Subsequent decades witnessed the development of motor evoked potentials, high-resolution EEG, electrocorticography, and multimodality monitoring platforms. Concurrent advances in anaesthetic pharmacology—particularly the introduction of propofol infusion and short-acting opioids—enabled the design of IOM-compatible total intravenous anaesthesia (TIVA) regimens that maintain signal quality without compromising hypnosis or analgesia. [5]

Today, IOM is considered a standard of care in a broad spectrum of neurosurgical procedures including resection of supratentorial tumors in or near eloquent areas, posterior fossa surgery, cerebrovascular surgery, skull base procedures, and epilepsy surgery. [6] The American Society of Neurophysiological Monitoring (ASNM), the American Clinical Neurophysiology Society (ACNS), and the International Federation of Clinical Neurophysiology (IFCN) have each published practice guidelines endorsing multimodality IOM for these high-risk cases. [7]

This review is directed at the practising neuroanaesthetist and aims to provide a comprehensive, clinically oriented account of each monitoring modality, the mechanisms by which anaesthetic agents influence signal parameters, the physiological confounders that must be considered, validated alert criteria, and the evidence base linking IOM to improved patient outcomes. Emerging technological developments and practical protocols are also discussed.

1. Electrophysiological Monitoring Modalities in Neurosurgery

1.1 Electroencephalography

Electroencephalography (EEG) records spontaneous electrical activity generated by the summated postsynaptic potentials of cortical pyramidal neurons. In the intraoperative context, EEG serves three principal functions: monitoring depth of anaesthesia, detecting cerebral ischemia, and guiding the management of intraoperative seizures. [8]

The cortical EEG undergoes characteristic changes as anaesthetic depth increases: initial paradoxical high-frequency activation is followed by progressive slowing, the appearance of delta waves, and ultimately

burst suppression, in which periods of electrical activity alternate with isoelectric silence. [9] Commercially available processed EEG parameters—the Bispectral Index (BIS), Patient State Index (PSI), and Narcotrend—derive numerical indices from the raw EEG that correlate with depth of hypnosis, though none is entirely immune to the confounding effects of specific agents. [10]

In carotid endarterectomy and other vascular procedures, a new onset of ipsilateral delta activity or EEG suppression following vessel occlusion signals critically reduced perfusion and may necessitate shunt insertion. [11] During epilepsy surgery, raw EEG identifies seizure onset zones and guides resection margins. Dense-array EEG systems with 128–256 channels provide superior spatial resolution for source localization compared with standard 16–32-channel configurations. [12]

1.2 Somatosensory Evoked Potentials

Somatosensory evoked potentials (SSEPs) are recorded in response to repetitive electrical stimulation of peripheral nerves—most commonly the median nerve at the wrist and the posterior tibial nerve at the ankle. The generated signals travel through the posterior column of the spinal cord, ascend via the medial lemniscal pathway through the brainstem, and terminate at the primary somatosensory cortex (S1). [13]

Key waveform components include the N9 (brachial plexus), N13 (cervical dorsal horn), N20 (cortical response to median nerve stimulation), and P37 (cortical response to tibial nerve stimulation). The critical monitoring parameters are cortical amplitude—the voltage difference between peak and trough—and peak latency. The widely accepted alert criterion is a greater than 50% decrease in amplitude or a greater than 10% prolongation in latency relative to baseline. [14]

SSEPs monitor the integrity of the dorsal somatosensory pathways but do not directly assess motor tract function. In practice, combined SSEP and MEP monitoring provides complementary coverage of both major long-tract systems. SSEPs are used routinely in supratentorial surgery near the central sulcus, posterior fossa surgery, aneurysm surgery, and spinal cord procedures. [15]

1.3 Motor Evoked Potentials

Motor evoked potentials (MEPs) assess the functional integrity of the corticospinal tract. Transcranial MEPs (tcMEPs) are elicited by high-voltage electrical stimulation of the motor cortex through scalp electrodes overlying the hand or leg motor areas (typically C1/C2 or C3/C4 positions). The descending electrical volley traverses the internal capsule, brainstem, and corticospinal tracts, and the compound muscle action potential (CMAP) is recorded from contralateral limb muscles. [16]

MEPs are exquisitely sensitive to anaesthetic suppression, particularly volatile halogenated agents, which abolish responses at concentrations exceeding 0.5 MAC. Total intravenous anaesthesia is therefore mandatory during MEP monitoring. [17] Neuromuscular blocking agents must be either avoided or maintained at a partial block (train-of-four ratio 2–3 twitches) to permit recordable CMAPs. [18]

Validated alarm criteria for MEPs include an amplitude decrease exceeding 50%, complete loss of response, or a stimulus voltage increase greater than 100 V required to achieve threshold. [19] Direct cortical MEPs, elicited by stimulation through subdural strip electrodes placed on the motor cortex, circumvent transcranial impedance and yield more stable, lower-stimulus responses particularly valuable in paediatric patients and those with preoperative motor deficits. [20]

1.4 Auditory Brainstem Responses

Auditory brainstem responses (ABR), also termed brainstem auditory evoked potentials (BAEPs), are elicited by repetitive acoustic click stimuli delivered through insert earphones. The resulting far-field potentials reflect sequential activation of the cochlear nerve (Wave I), cochlear nucleus (Wave II), superior olivary complex (Wave III), lateral lemniscus (Wave IV), and inferior colliculus (Wave V). [21]

ABRs are particularly valuable in posterior fossa surgery involving the cerebellopontine angle, including vestibular schwannoma resection, microvascular decompression for trigeminal neuralgia, and posterior fossa meningioma surgery. Monitoring focuses on the latency of Wave V and the amplitude of the Wave I–V complex. A Wave V latency prolongation exceeding 1 ms or an amplitude decrease exceeding 50% relative to baseline constitutes a clinically significant alert. [22]

The principal advantage of ABR monitoring is its relative resistance to anaesthetic agents; responses are generally preserved throughout the normal range of surgical anaesthesia, making ABRs among the most robust signals in the IOM repertoire. [23]

1.5 Electromyography

Electromyography (EMG) in the intraoperative setting serves two distinct roles. Free-running (spontaneous) EMG detects mechanical irritation of motor nerves as neurotonic discharges that arise from ion channel disruption at the site of injury. Triggered EMG uses electrical stimulation through a surgical probe to identify and map cranial or peripheral motor nerve anatomy. [24]

EMG monitoring of cranial nerves is particularly valuable in skull base and posterior fossa surgery. Facial nerve (CN VII) EMG monitoring during acoustic neuroma, parotid, and skull base surgery has been associated with significantly higher rates of anatomical nerve preservation compared with unmonitored cases. [25] Monitoring of the vagus, glossopharyngeal, accessory, and hypoglossal nerves during jugular foramen and foramen magnum surgeries employs the same methodology. [26]

A critical requirement for EMG monitoring is the avoidance of complete neuromuscular blockade. While patients may be intubated with succinylcholine, subsequent neuromuscular blockade must either be omitted or maintained at a partial level permitting clear spontaneous and evoked EMG responses. [27]

1.6 Electrocorticography

Electrocorticography (ECoG) involves direct recording of electrical activity from the cortical surface using subdural grid, strip, or depth electrodes. In epilepsy surgery, ECoG provides the highest-resolution mapping of epileptiform discharges, guiding resection of the seizure onset zone while preserving functionally critical cortex. [28]

ECoG is also employed during awake craniotomy for cortical language mapping and during tumor resection in eloquent areas, where it can identify epileptic activity within or adjacent to the resection field. Interpretation of intraoperative ECoG requires careful anaesthetic titration; many agents suppress or alter interictal activity, and protocols typically involve a period of reduced anaesthetic depth or brief inhalational agent washout to unmask spontaneous or stimulated epileptic discharges. [29]

Table 1. Summary of Intraoperative Electrophysiological Monitoring Modalities

Modality	Neurological Function	Anaesthetic Sensitivity	Key Clinical Use	Limitations
EEG	Global cortical activity, seizure detection	Highly sensitive; suppressed by most agents	Depth of anaesthesia, cerebral ischemia	Limited spatial resolution, artifact-prone
SSEP	Somatosensory (dorsal column)	Amplitude reduced; latency prolonged	Spinal cord, brain stem, cortical monitoring	Motor pathway not monitored directly

Modality	Neurological Function	Anaesthetic Sensitivity	Key Clinical Use	Limitations
MEP	Voluntary motor (corticospinal tract)	Highly sensitive; abolished by volatile agents	Motor cortex, internal capsule surgery	TIVA required; movement risk
ABR	Auditory brainstem pathway	Relatively resistant to anaesthetics	Posterior fossa, CPA tumor surgery	Only auditory pathway; limited by noise
EMG	Peripheral and cranial nerve motor units	NMB abolishes; partial paralysis needed	Cranial nerve preservation in skull base	Requires expert interpretation
ECoG	Direct cortical epileptic activity	Requires reduction of anaesthetic depth	Epilepsy surgery, cortical mapping	Invasive; limited coverage area

Abbreviations: EEG, electroencephalography; SSEP, somatosensory evoked potential; MEP, motor evoked potential; ABR, auditory brainstem response; EMG, electromyography; ECoG, electrocorticography; CPA, cerebellopontine angle; NMB, neuromuscular block; TIVA, total intravenous anaesthesia.

2. Anaesthetic Effects on Electrophysiological Signals

2.1 Mechanisms of Anaesthetic Interference

Anaesthetic agents exert their neurophysiological effects primarily through modulation of ligand-gated ion channels: enhancement of inhibitory GABA-A receptor-mediated chloride conductance and inhibition of excitatory NMDA, AMPA, and nicotinic acetylcholine receptors. [30] These mechanisms uniformly reduce neuronal excitability and synaptic transmission, translating into attenuated signal amplitude, prolonged conduction latency, or complete suppression of evoked responses.

The sensitivity of different IOM modalities to anaesthetic effects varies considerably and reflects the complexity of the underlying neural pathway. Multisynaptic pathways with extensive cortical representation—such as those generating MEPs—are far more vulnerable than oligosynaptic pathways with predominantly subcortical generators—such as those generating Wave I of the ABR. [31]

2.2 Volatile Halogenated Agents

Volatile anaesthetic agents (isoflurane, sevoflurane, desflurane) produce dose-dependent suppression of all cortically generated IOM signals proportional to their MAC fraction. [32] At 0.5 MAC, SSEPs show modest amplitude reduction; at 1.0 MAC, amplitude decreases of 50% are common, particularly in paediatric patients. MEPs, which require robust polysynaptic corticomotoneuronal transmission, are practically abolished above 0.5 MAC of any volatile agent. [33]

Importantly, the effect of volatile agents on EEG follows a characteristic progression: initial activation at subanaesthetic doses, followed by progressive slowing, delta activity, burst suppression, and ultimately isoelectric silence. The burst suppression ratio—the proportion of time the EEG is isoelectric—provides a quantitative measure of anaesthetic depth that is of particular importance in cerebral protection protocols

during temporary vessel occlusion in aneurysm surgery. [34]

2.3 Total Intravenous Anaesthesia

Total intravenous Anaesthesia (TIVA), typically combining a propofol infusion with a short-acting opioid such as remifentanil, is the preferred technique for cases requiring MEP monitoring. [35] Propofol at clinically relevant plasma concentrations (2–5 mcg/mL) produces less cortical suppression than equipotent volatile anaesthetic concentrations, permitting reliable MEP recording. [36]

Dexmedetomidine, an alpha-2 adrenoceptor agonist, has emerged as a valuable adjunct in TIVA regimens. Through inhibition of noradrenergic neurons of the locus coeruleus, dexmedetomidine produces sedation and analgesia with minimal suppression of cortical IOM signals, making it particularly valuable for procedures requiring combined neurophysiological monitoring and a cooperative patient, such as awake craniotomy. [37]

Ketamine, an NMDA receptor antagonist, uniquely augments SSEP amplitude and may partially overcome propofol-induced MEP suppression when used as a low-dose infusion adjunct. [38] Its sympathomimetic and cerebral metabolic stimulating properties require careful consideration in patients with raised intracranial pressure, but at subanaesthetic infusion rates (0.1–0.5 mg/kg/h) ketamine can reliably improve signal quality in patients with difficult-to-record SSEPs or MEPs.

2.4 Neuromuscular Blocking Agents

The interaction between neuromuscular blocking agents (NMBAs) and IOM is primarily relevant for MEP and EMG monitoring. Succinylcholine may be used for endotracheal intubation without residual IOM consequence, provided sufficient time is allowed for complete recovery (approximately 15 minutes). [39] Non-depolarizing NMBAs should be avoided entirely when MEP or EMG monitoring is planned, or limited to a standardized partial block titrated by train-of-four (TOF) monitoring to maintain 2–3 twitches of a four-twitch response. [40] Complete neuromuscular blockade abolishes MEP CMAPs and renders triggered and free-running EMG uninterpretable.

2.5 Opioids

Opioids at clinically used infusion rates exert only modest effects on IOM signals. Remifentanil, a rapidly metabolized mu-opioid receptor agonist, produces minimal MEP and SSEP suppression at infusion rates up to 0.2–0.3 mcg/kg/min and is the opioid of choice in TIVA regimens for IOM. [41] High-dose opioid infusions may produce mild amplitude reduction and latency prolongation in SSEPs, but these effects are clinically less significant than those produced by hypnotics or volatile agents.

Table 2. Effects of Anaesthetic Agents on Intraoperative Electrophysiological Monitoring Modalities

Agent	EEG	SSEP	MEP	ABR	EMG/NMB
Propofol (TIVA)	Burst suppression at high dose	Mild amplitude ↓, latency ↑	Best preserved	Minimal effect	No NMB effect
Isoflurane / Sevo	↓ amplitude, burst suppress >1.5 MAC	Significant amplitude ↓	Marked suppression	Mild effect	No NMB effect

Agent	EEG	SSEP	MEP	ABR	EMG/NMB
Ketamine	↑ gamma power; paradoxical excitation	↑ Amplitude (adjunct)	May augment	Minimal	No NMB effect
Dexmedetomidine	Spindle activity, stage 2 sleep-like	Minimal effect	Well preserved	Minimal	No NMB effect
Opioids (remifentanil)	Mild suppression	Minimal at usual doses	Well preserved	Minimal	No NMB effect
Neuromuscular blockers	No direct effect	No direct effect	Abolish if complete NMB	No direct effect	Abolish EMG
Nitrous oxide	Activates high-freq EEG	↓ amplitude significantly	Marked ↓ amplitude	Minimal	No NMB effect

Abbreviations: ↑, increase; ↓, decrease; MAC, minimum alveolar concentration; NMB, neuromuscular block; TIVA, total intravenous Anaesthesia.

3. Physiological Variables Affecting IOM Signal Fidelity

3.1 Temperature

Hypothermia exerts a powerful effect on all electrophysiological signals. A reduction in core temperature prolongs conduction velocity in peripheral and central neural pathways, resulting in progressive latency increases of approximately 1 ms per degree Celsius decrease in body temperature. [42] Simultaneous amplitude reduction occurs as hypothermia reduces neuronal excitability and synaptic transmission. Mild intraoperative hypothermia (core temperature 34–36°C), a frequent occurrence during prolonged craniotomies, must therefore be identified and corrected before attributing latency changes to surgical injury.

Conversely, mild therapeutic hypothermia (32–34°C) is sometimes deliberately induced during cerebrovascular procedures to provide neuroprotection during temporary arterial occlusion. In this setting, the monitoring team must anticipate and account for the expected temperature-dependent signal changes when interpreting IOM data. [43]

3.2 Blood Pressure and Cerebral Perfusion

Cerebral perfusion pressure (CPP) is a critical determinant of IOM signal quality. Arterial hypotension below the lower limit of cerebrovascular autoregulation leads to ischemic suppression of evoked potentials, EEG slowing, and ultimately signal loss. [44] In neurosurgical patients, particularly those with pre-existing cerebrovascular disease or intracranial hypertension, autoregulatory thresholds may be shifted, requiring higher MAP targets to maintain adequate cortical perfusion.

When IOM signals deteriorate in the absence of a clear surgical cause, restoration of MAP to baseline or higher is the first priority in the anaesthetic management algorithm. [45] Vasopressor support with phenylephrine or norepinephrine is frequently employed, though the differential effects of pure alpha agonists versus agents with beta-adrenergic activity on IOM signals are generally negligible.

3.3 Carbon Dioxide Tension

Hypocapnia, frequently employed to reduce intracranial pressure via cerebral vasoconstriction, may paradoxically impair IOM monitoring by reducing cerebral blood flow below the critical threshold for normal electrophysiological function. [46] PaCO2 values below 30 mmHg are associated with reproducible SSEP amplitude decreases and should be avoided unless the clinical indication for hyperventilation clearly outweighs the monitoring compromise.

3.4 Anemia and Hemodilution

Significant intraoperative anemia (hemoglobin < 8 g/dL) reduces oxygen delivery to neural tissues and may produce ischemic IOM changes indistinguishable from surgical or perfusion-related causes. [47] Maintenance of hemoglobin above 9–10 g/dL is recommended during high-risk IOM cases, particularly when prolonged retraction or temporary vessel occlusion is anticipated.

4. Alarm Criteria, Communication Protocols, and Response Algorithms

4.1 Standardized Alert Criteria

The reliability of IOM as a clinical tool depends upon adherence to validated, standardized alert criteria that discriminate signal changes caused by surgical injury from those arising from physiological fluctuation or anaesthetic variation. [48] The most widely adopted criteria are summarized in Table 3.

A critical distinction exists between warning alerts—which call for heightened vigilance and non-surgical interventions—and critical alerts—which require immediate surgical action. [49] The fundamental principle guiding alert interpretation is a systematic stepwise exclusion of non-surgical causes (anaesthetic depth change, NMBA level, temperature, blood pressure, positioning) before attributing signal deterioration to surgical injury.

4.2 Communication Framework

Effective IOM requires seamless real-time communication among the neurophysiologist (or technologist), anaesthesiologist, and surgeon. The ASNM and ACNS recommend a standardized verbal reporting format in which the monitoring team immediately calls out: (1) the affected modality; (2) the laterality; (3) the magnitude of change; and (4) the presence or absence of preceding anaesthetic changes. [50]

Systematic implementation of structured communication—analogous to the World Health Organization (WHO) surgical safety checklist—has been associated with improved team response times to IOM alerts and reduced rates of failure to respond. [51] Prospective documentation of all IOM changes, anaesthetic adjustments, and surgical responses in a dedicated intraoperative neurophysiology record is essential for postoperative clinical review and medicolegal documentation.

Table 3. Validated Alert Criteria and Recommended Anaesthetic and Surgical Responses

Modality	Warning Criterion	Critical Criterion	Recommended Action
SSEP	>50% amplitude decrease or >10% latency increase	Complete loss of response	Notify surgeon; check BP, anaesthetic depth, temperature; consider repositioning

Modality	Warning Criterion	Critical Criterion	Recommended Action
MEP	>50% amplitude decrease (absolute threshold approach)	Loss of response or threshold increase >100 V	Immediately alert surgeon; increase BP; switch to TIVA; avoid NMB
EEG	New asymmetry, focal slowing, or burst suppression ratio change	Electrocerebral silence or isoelectric EEG (unanticipated)	Rule out anaesthetic causes; check perfusion; alert surgeon to possible ischemia
ABR	>1 ms Wave V latency prolongation	Loss of Wave V	Halt retraction; irrigate with warm saline; limit cautery near cochlear nerve
EMG (cranial)	Repetitive neurotonic discharges	Sustained bursts >1 s; silence after stimulation	Warn surgeon of mechanical irritation; reduce traction; avoid electrocautery near nerve

Abbreviations: SSEP, somatosensory evoked potential; MEP, motor evoked potential; ABR, auditory brainstem response; EMG, electromyography; BP, blood pressure; NMB, neuromuscular block; TIVA, total intravenous Anaesthesia.

5. Clinical Applications by Surgical Procedure

5.1 Supratentorial Tumor Surgery

Resection of brain tumors involving or adjacent to eloquent cortex—primary motor cortex, Broca's and Wernicke's areas, primary somatosensory cortex, and the underlying white matter tracts—carries significant risk of new neurological deficit. Multimodality IOM combining SSEPs, MEPs, and if appropriate ECoG or direct cortical stimulation mapping can reduce permanent motor deficit rates from approximately 10–15% to less than 5% in expert centers. [52]

Phase reversal of the N20/P20 SSEP component over the central sulcus provides reliable intraoperative identification of the primary somatosensory cortex and, by inference, the motor strip, enabling safe placement of resection margins. [53] Direct cortical stimulation mapping during awake craniotomy provides the highest-resolution functional localization; however, when awake surgery is not feasible or appropriate, combined asleep SSEP/MEP monitoring with subcortical stimulation offers a clinically validated alternative. [54]

5.2 Cerebrovascular Surgery

In aneurysm surgery, temporary parent vessel occlusion required for aneurysm dissection and clipping can lead to ischemic stroke in 5–15% of cases. Combined SSEP and MEP monitoring provides continuous assessment of the cortical territory at risk. [55] A significant body of evidence demonstrates that IOM-guided management—including shortening of occlusion time, pharmacological brain protection, and clip readjustment—reduces permanent ischemic deficit rates.

Carotid endarterectomy is the vascular procedure most strongly supported by randomized evidence for EEG monitoring. Several randomized controlled trials have demonstrated that EEG-triggered shunting, employed selectively in patients with significant intraoperative EEG changes, achieves equivalent stroke

protection to routine shunting while avoiding the mechanical complications of unnecessary shunt insertion. [56]

5.3 Posterior Fossa and Skull Base Surgery

The posterior fossa and skull base present the highest density of IOM-relevant neural structures per surgical field. Facial nerve EMG monitoring during cerebellopontine angle tumor surgery has been shown in large prospective studies and a meta-analysis to significantly improve anatomical and functional nerve preservation rates. [57] Facial nerve stimulation thresholds at the end of resection correlate with postoperative facial function outcomes and guide surgical decision-making regarding the extent of tumor removal.

ABR monitoring during microvascular decompression for hemifacial spasm and trigeminal neuralgia protects the cochlear nerve from traction and cautery injury. The risk of postoperative hearing loss in CPA surgery is reduced by approximately 50% with ABR monitoring compared with unmonitored controls in pooled analyses. [58]

5.4 Epilepsy Surgery

In resective epilepsy surgery, intraoperative ECoG provides high-resolution mapping of the epileptiform zone, enabling the surgeon to extend resection margins to achieve complete resection of the ictal onset zone while simultaneously preserving functionally critical cortex. [59] Residual intraoperative ECoG spike activity following resection has been shown in multiple cohort studies to predict postoperative seizure recurrence, guiding decisions regarding additional resection.

Extraoperative invasive monitoring with subdural grids or stereoelectroencephalography (sEEG) has largely supplanted intraoperative ECoG for complex cases, though the latter retains an important role in straightforward mesial temporal lobe epilepsy and lesional cases where the epileptic zone is clearly defined preoperatively. [60]

6. Emerging Technologies and Future Directions

6.1 High-Density EEG and Source Localization

Advances in electrode manufacturing and signal processing have enabled high-density EEG systems with 128–512 channels to be deployed intraoperatively. These systems facilitate source localization algorithms—including equivalent dipole fitting, distributed source models, and beamforming—that can localize epileptiform and ictal sources with spatial resolution approaching that of invasive recording, potentially reducing the need for chronic intracranial monitoring in selected patients. [61]

6.2 Machine Learning and Artificial Intelligence

Machine learning algorithms applied to intraoperative IOM data have demonstrated the ability to automatically detect significant signal changes, classify artifact from true deterioration, and predict postoperative neurological outcomes from intraoperative signal trajectories with accuracy exceeding that of experienced human neurophysiologists in retrospective studies. [62] Deep learning architectures trained on large multimodality IOM datasets are under active clinical validation and may in future provide real-time decision support for the monitoring team.

6.3 Closed-Loop Anaesthetic Delivery

The integration of EEG-based depth of Anaesthesia indices with automated anaesthetic delivery systems represents a clinically implemented technology in some specialized centers. Closed-loop propofol delivery systems titrating infusion rate against BIS or entropy maintain more stable depth of Anaesthesia with lower total drug consumption than manual control in controlled trials. [63] Future systems may incorporate

multimodality IOM signal quality as an additional feedback variable, automatically adjusting anaesthetic depth to optimize both patient unconsciousness and signal fidelity.

6.4 Ultrasound-Guided Neuronavigation and Intraoperative MRI

The integration of IOM with intraoperative imaging modalities—3T intraoperative MRI, ultrasound, and fluorescence-guided surgery—provides a multimodal framework for surgical decision-making that combines anatomical imaging with functional neural monitoring. [64] High-field intraoperative MRI has been shown to improve the extent of resection in glioma surgery; when combined with IOM, it enables real-time reconciliation of imaging and functional boundaries.

7. Practical Anaesthetic Protocol for Intraoperative Monitoring

7.1 Preoperative Assessment

The anaesthetic management of IOM cases begins with a dedicated preoperative assessment that identifies: (1) the monitoring modalities planned; (2) the patient's neurological baseline and any pre-existing signal abnormalities; (3) medications that may affect IOM signals (antiepileptics, chronic opioids, benzodiazepines); and (4) patient suitability for TIVA (allergy history, vascular access). [65]

7.2 TIVA Regimen Design

The recommended TIVA regimen for combined SSEP/MEP monitoring consists of: propofol target-controlled infusion (TCI) targeting a plasma concentration of 2–4 mcg/mL (Schnider or Marsh model); remifentanil TCI at 0.2–0.4 ng/mL effect-site concentration; and an adjunct selected on the basis of case requirements. [66] Dexmedetomidine infusion (0.3–0.7 mcg/kg/h) reduces propofol requirements and provides superior intraoperative analgesia without MEP suppression. Ketamine infusion (0.1–0.3 mg/kg/h) can augment SSEP amplitude and should be considered in patients with baseline signal abnormalities.

Neuromuscular blockade should be limited to the intubation dose of succinylcholine or a single intubating dose of rocuronium, with reversal confirmed by TOF monitoring before commencement of MEP or EMG recording. Sugammadex provides rapid, reliable reversal of rocuronium-induced neuromuscular blockade and is the agent of choice when early establishment of MEP monitoring is required. [67]

7.3 Intraoperative Management Algorithm

A stepwise algorithm governs the anaesthetic response to IOM alerts. Upon receiving a monitoring alert, the anaesthetic team should:

- Immediately communicate the alert to the surgeon and neurophysiologist.
- Verify and document the most recent anaesthetic changes (propofol or volatile agent concentration, opioid dose, adjuncts administered).
- Check and optimize mean arterial pressure: target MAP ≥ baseline or ≥ 80 mmHg, whichever is higher.
- Confirm core body temperature: correct hypothermia if present.
- Verify TOF ratio (for MEP/EMG monitoring): ensure partial block ≤ 3/4 twitches.
- Check PaCO2: avoid hypocapnia below 35 mmHg.
- If non-surgical causes are excluded and alert persists, call a critical alert and request surgical intervention (halting retraction, repositioning, or restoring blood flow).

Documentation in the anaesthetic record should include time-stamped entries for all IOM alerts, anaesthetic interventions, and return of signals. This record constitutes a medically and legally important chronological account of intraoperative neural function. [68]

8. Evidence Base for IOM in Improving Patient Outcomes

The evidence supporting IOM in neurosurgery spans prospective cohort studies, retrospective analyses, systematic reviews, and meta-analyses, though large randomized controlled trials—feasible only in selected procedural contexts—remain limited. [69]

In spinal surgery, a landmark prospective multicenter study by the Scoliosis Research Society found that the combined use of SSEPs and MEPs identified 100% of patients who developed new neurological deficits intraoperatively, compared with 43% detected by SSEP alone. [70] This study provided the methodological foundation for current multimodality monitoring guidelines.

In supratentorial tumor surgery, a systematic review of 25 studies comprising 5,854 patients reported that IOM-guided surgery was associated with a 60% reduction in permanent neurological deficit rate compared with surgery without monitoring, with absolute risk reduction of approximately 5–8 percentage points. [71]

In posterior fossa surgery, a prospective study of 1,006 patients undergoing vestibular schwannoma resection demonstrated that continuous facial nerve EMG and triggered MEP monitoring was associated with a 78% anatomical nerve preservation rate compared with 63% in historical unmonitored controls. [72]

The economic analysis of IOM further supports its adoption. A cost-effectiveness model demonstrated that IOM in spinal deformity surgery reduces the incidence of permanent paraplegia at a cost per quality-adjusted life year (QALY) well below accepted willingness-to-pay thresholds in high-income healthcare systems. [73]

9. Limitations and Controversies

Despite its widespread adoption, IOM is not without limitations. False-positive alerts—signal changes that do not reflect true neural injury—occur with a frequency of 10–20% across modalities and can prompt unnecessary surgical interruption, potentially increasing operative duration and exposure. [74] False-negative monitoring—where new postoperative deficits occur despite stable intraoperative signals—is less common (< 2%) but represents the most serious monitoring failure and reflects the inability of current modalities to detect all mechanisms of neural injury.

The heterogeneity of monitoring protocols, alarm criteria, and reporting standards across institutions limits the comparability of IOM outcome data. International consensus initiatives—including the joint ASNM/ACNS position statements—have sought to standardize practice, but significant institutional variation persists. [75]

The infrastructure requirements for IOM—dedicated neurophysiology equipment, trained personnel, and multidisciplinary team coordination—present logistical and cost challenges for lower-resource health systems. Models incorporating trained neurophysiology technologists supervised remotely by neurophysiologists using telemedicine platforms offer a promising approach to extending IOM access in resource-limited settings. [76]

Conclusion

Intraoperative neurophysiological monitoring has undergone a transformation from an experimental adjunct to an evidence-based standard of care in high-risk neurosurgical procedures. The successful application of IOM depends upon a thorough understanding of the physiological basis of each monitoring

modality, the pharmacological effects of anaesthetic agents on electrophysiological signals, validated alarm criteria, and effective multidisciplinary communication.

The anaesthesiologist occupies a central and indispensable role in IOM-monitored cases. Selection of an IOM-compatible anaesthetic technique—principally TIVA with propofol, remifentanil, and appropriately chosen adjuncts—requires specific pharmacological knowledge and careful intraoperative management. Recognition and systematic exclusion of anaesthetic and physiological causes of IOM signal changes are prerequisites for accurate alert interpretation.

Emerging technologies including high-density EEG, artificial intelligence-assisted signal analysis, and closed-loop anaesthetic delivery promise to enhance the sensitivity, specificity, and automation of IOM in the near future. Robust evidence demonstrates that IOM-guided surgery reduces permanent neurological deficits, and its continued development and wider adoption are strongly justified by clinical outcomes data. The neuroanaesthetist who combines mastery of IOM pharmacology with effective team communication contributes directly and measurably to improved neurological outcomes for patients undergoing the most complex procedures in surgical medicine.

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