Editorial

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Intraoperative neurophysiologic monitoring and anaesthetic implications

It was in 1973 that the intraoperative wake-up test was described by Pierre Stagnara along with the anaesthesiologist Mme Vauzelle. Over a period of 65 years, the intraoperative wake-up test has been replaced by intraoperative neurophysiologic monitoring, which has become a real-time monitor of the functional integrity of neural structures. If appropriate manoeuvres (such as correction of hypotension and hypoxia and timely decompression from surgical retractors, pressure from bony structures, or haematomas) are taken, there may be a reduction of the neurological ischaemia and decreased morbidity.

Both sensory and motor evoked potentials (EPs) are used in the operating room to assist in the surgical decision making. In the operating room, both sensory evoked potentials (SEPs) and motor evoked potentials (MEPs) are used as surrogates for postoperative clinical endpoints of motor and sensory wellbeing, as their true endpoints (with the exception of the "wake-up" test or awake cranial surgeries) are not available in the operating room. Holdefer[3] et al. did a literature search to investigate the causal links between surgical events and EP changes. They found that although the monitoring of EPs was not fully validated, their continued use in the intraoperative period helps to avoid or reduce the neural injury.[3] The American Society of Neurophysiological Monitoring came out with a position statement that evoked potentials are an established practice option for cortical and subcortical mapping and for monitoring during surgeries that may cause injury to the brain, brainstem, spinal cord, or facial nerve.[4] During monitoring of somatosensory, visual, and brainstem auditory EPs, the specific stimulations are applied to the peripheral sites and the desired responses are recorded from the central locations. MEP monitoring for intraoperative monitoring requires transcranial stimulation of the motor cortex by electrical or magnetic means to produce a descending response that traverses the corticospinal tracts and eventually generates a measurable response. The desired recordings may be obtained from the epidural space (D-wave) or from the distal muscle.

The amplitude (measured in microvolts) is defined as the distance from the peak to adjacent trough. The time from the stimulation to the peak in milliseconds is defined as the latency. Loss of or change in the waveform can indicate the need for modification of surgical strategy, patient positioning, or patients physiological management in order to prevent or minimise neurologic injury. Specifically, a 50% reduction in amplitude or a 10% increase in latency of SSEPs, MEPs, and brain stem auditory evoked potentials (BAEPs) is considered to be of pathological significance.

ANAESTHETIC EFFECTS ON NEUROMONITORING

Most anaesthetic agents cause a dose-dependent depression in synaptic activity. It has been seen that all anaesthetic agents affect MEP muscle responses and cortical potentials. Inhalational agents affect the EPs to a greater extent than the intravenous (IV) anaesthetic agents.^[5] The effects are much greater on cortical responses than on subcortical responses.[6,7] Nitrous oxide (N₂O) also causes a reduction in the amplitude and an increase in the latency of both cortical sensory responses and MEP when used alone or in combination with halogenated inhalational agents or opioids.[8] It has been observed that opioids cause mild depression of sensory and motor responses, with a loss of late sensory evoked response peaks (>100 msec) at doses, which cause sedation. Fentanyl, remifentanil, or sufentanil infusions are commonly used for TIVA to facilitate neuromonitoring. Koht et al.[9] observed the effects of etomidate, midazolam, and thiopental on median nerve somatosensory EPs. Etomidate increased both amplitude and latency. Thiopental decreased amplitude and increased latency, whereas midazolam had no effect on amplitude but increases the latency. Schubert and Licina^[10] observed that ketamine enhanced the cortical SSEP and MEP amplitude and partially reversed the depressant effect of N2O on SSEPs. Hence, an anaesthetic regimen with induction of anaesthesia by ketamine (2 mg/kg IV) followed by continuous infusion at a rate of 30 mcg kg-1 min-1 may be desirable for neuromonitoring. Variable effects have been reported with dexmedetomidine during SSEP or MEP monitoring. Mahmoud et al. found that dexmedetomidine as an anaesthetic adjunct to propofol-based TIVA significantly attenuated the amplitude of transcranial electric MEPs.[11] However, Rozet et al. observed that there was no difference in SEPs (latency and amplitude) and MEPs (amplitude and latency) between dexmedetomidine versus placebo groups.[12]

A balanced anaesthesia approach may be followed using a low-dose inhalation anaesthetic agent (up to 0.5 MAC) and low to medium dose propofol (50 to 100 mcg kg⁻¹ min⁻¹ intravenously) with a relatively high-dose opioid (fentanyl, remifentanil). However, inadequate generation of EPs may need a modification of the anaesthetic techniques. There is still a paucity of ideal anaesthetic techniques, that can be utilised for the optimal generation of EPs.

Over the last few decades, there has been an increase in the number of centres using intraoperative neuromonitoring across India in an attempt to reduce the neurological morbidity. This issue of Indian Journal of Anaesthesia features two articles on intraoperative neuromonitoring.[13,14] In a pursuit for an ideal anaesthetic technique for patients undergoing surgery for spinal cord tumours, Parthiban et al.[13] have made an attempt to study the effects of IV anaesthesia (propofol) and inhalational anaesthesia (isoflurane) on the intraoperative motor evoked potentials (iMEPs). Authors observed that propofol anaesthesia lead to the generation of more successful baseline iMEPs (74%) when compared with isoflurane anaesthesia (50%). In addition to the anaesthetic effects, the authors found interesting findings that age and duration of symptoms could have a significant influence on elicitation of baseline iMEP. These possible effects of aging may be a result of a decrease in nervous tissue mass, neuronal density, and concentrations of various neurotransmitters. [15,16] In patients over 40 years under propofol anaesthesia, iMEP responses could be recorded in 60% of the muscles whereas under isoflurane group responses, it could be recorded in only 40% of the muscles. The correlation trend line shows that in patients with >12 months of duration of symptoms, 42% iMEP responses could be recorded under propofol anaesthesia whereas it is lesser under isoflurane (35%) anaesthesia. Lesser stimulus strength to elicit baseline iMEPs and less potential fading was observed with propofol as compared with isoflurane anaesthesia.

Before interpreting the changes in EP during intraoperative neurophysiological monitoring, physiological factors such as hypoxia, hypotension, hypothermia, and anaemia should be corrected. Positional changes as extreme head position, peripheral nerve compression, spine flexion or extension, and technical faults (as lead failure and electromagnetic interference) should be ruled out before interpreting the EPs.

In another article published in this issue of Indian Journal of Anaesthesia, Nitin *et al.*^[14] describe the rare experience of successful intraoperative neurophysiological monitoring during pregnancy with no adverse foetal or maternal effects. Normal foetal heart rate and viability was confirmed with continuous cardiotocography and foetal echocardiography during and after the completion of the surgical procedure. No significant changes in MEP amplitude were observed intraoperatively, with no new onset motor or sensory deficits in postoperative period. This case report is valuable as we have a limited experience of intraoperative neuromonitoring during pregnancy.

We hope that these two studies published in the present issue of Indian Journal of Anaesthesia may help physicians to better understand the use of IONM and to improve the safety of patients undergoing spine surgery. EPs may act as biomarkers of neurological injury in the same way as the blood pressure and pulse oximetry monitors are monitors of tissue perfusion and oxygenation. Although we do not have any level I evidence, favouring the use of neurophysiological monitoring, its judicious use as surrogate markers in the intraoperative period (to reduce the neurological morbidity) may be justified by its ability to detect the neural injury and the absence of "second best options" in such scenarios.

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