

THE ROLE OF THE ANESTHESIOLOGIST IN NEUROMONITORING DURING CRANIOTOMY: RESULTS BETWEEN 2005-2008 AND REVIEW OF THE LITERATURE

Original Article

KRANİYOTOMİ OPERASYONLARI SIRASINDA NÖROMONİTÖRLEMEDE ANESTEZİSTİN ROLÜ: 2005-2008 YILLARINDAKİ SONUÇLARIMIZ VE LİTERATÜRÜN GÖZDEN GEÇİRİLMESİ

Hatice Ture

*Yeditepe University Hospital
Department of Anesthesiology.*

Sevgi Bilgen

*Yeditepe University Hospital
Department of Anesthesiology.*

Ozge Koner

*Yeditepe University Hospital
Department of Anesthesiology.*

Serap Karacalar

*Okmeydanı Education and Research Hospital
Department of Anesthesiology.*

Geysu Karlıkaya

*Yeditepe University Hospital Department of
neurology.*

Canan Aykut Bingol

*Yeditepe University Hospital Department of
neurology.*

Ugur Ture

*Yeditepe University Hospital Department of
neurosurgery.*

Corresponding Author

Hatice Ture

*Yeditepe University Hospital
Department of Anesthesiology.
Devlet Yolu, Ankara Cad, No:102-104, Kozyatağı,
34752 İstanbul, Turkey
Tel: 90 (216) 578 4000
Fax: 90 (216) 578 4266
E-mail: htcture@yahoo.com*

ABSTRACT

Objectives: The whole surgical team carries out the important tasks of neuromonitoring during craniotomy to prevent neurological damage. We examined the role of the anesthesiologist in intraoperative neuromonitoring in light of experience between 2005-2008.

Methods: We gathered the files of patients who underwent craniotomy with neuromonitoring from November 2005 to 2008. The neuromonitoring data were analyzed, details of demographic characteristics, neuromonitoring methods, and anesthesia were recorded.

Results: During 3-year period, 204 patients who underwent craniotomy were monitored with the following techniques: SSEP with phase transformation (n:16), motor cortex localization (n:31), corticospinal tract localization (n:51), direct cortical and subcortical stimulation (n:27), motor speech center (Broca) localization (n:3), cranial nerve (n:60), MEP (n:84), SEP (n:92), EEG (n:24), BAEP (n:35), and cranial nerve monitoring (303 cranial nerves). Total intravenous

anesthesia with propofol and remifentanyl was used in all patients. Patients having EMG and MEP were not given muscle relaxants after anesthesia was induced. The dosages of intravenous anesthetic agents were reduced during subcortical stimulation and EEG monitoring.

Conclusion: If anesthesiologists guide the use of anesthetic agents (depending on the neuromonitoring method used) and inform the team of changes in the patient's hemodynamic status, the information gained through neuromonitoring can be obtained and interpreted more accurately.

Keywords: *Anesthesia; craniotomy; neuromonitoring.*

ÖZET

Amaç: Kraniyotomi operasyonlarında, nörolojik hasarın önlenmesi için kullanılan nöromonitörlemede tüm ekibe önemli görevler düşmektedir. Bu çalışmada, 2005-2008 tarihleri arasında yapılan intraoperatif nöromonitörleme uygulamalarımızın ışığında anesteziistlerin rolü irdelenmeye çalışılmıştır.

Yöntemler: Bu çalışmada, 2005 kasım-2008 aralık tarihleri arasında, nöromonitörleme ile kraniyotomi operasyonu geçiren hastalara ait dosyalar ve nöromonitörleme kayıtları incelenmiştir. Bu hastaların demografik özellikleri, uygulanan nöromonitörleme yöntemleri ve anestezi detayları kaydedilmiştir.

Bulgular: 3 yıllık periyotta, kraniyotomi operasyonu geçiren 204 hastaya: faz dönüşümlü SSEP (n:16), direkt kortikal stimülasyonla motor korteks lokalizasyonu (n:31), subkortikal stimülasyonla kortikospinal trakt lokalizasyonu (n:51), direkt kortikal ve subkortikal stimülasyon (n:27), motor konuşma merkezi (Broca) lokalizasyonu (n:3), kranial sinir (n : 60), MEP (n :84), SSEP (n:92), EEG (n: 24), BAEP (n:35) ve kranial sinir

monitörlemesi (303 kranial sinir) yapılmıştır. Tüm hastalara propofol ve remifentanyl ile total intravenöz anestezi uygulanmıştır. EMG ve MEP yapılan hastalara anestezi indüksiyonunu takiben kas gevşetici ajan kullanılmamıştır. Subkortikal stimülasyon ve EEG monitorizasyonu sırasında intravenöz anestezi ajan dozları azaltılmıştır.

Sonuç: Anestezi doktorlarının; nöroloji ekibinin uygulayacağı nöromonitörleme yöntemine göre, anestezi ajanların kullanımını yönlendirilmesi ve hastanın hemodinamik durumundaki değişikliklerden nöroloji ekibini haberdar etmesi, nöromonitörlemede daha doğru bilgi elde edilmesine ve elde edilen bilgilerin doğru olarak yorumlanmasına yardımcı olacaktır.

Anahtar Sözcükler: *Anestezi; kraniyotomi; nöromonitörleme.*

INTRODUCTION

Intraoperative neuromonitoring (IONM) procedures are used frequently to track the status of the nervous system, which is at risk of hypoxic, ischemic and mechanical trauma during surgery (9,27). IONM helps identify changes in the nervous system during the early intraoperative period and helps the surgeon prevent irreversible damage and determine surgical margins. IONM methods can be categorized into two main groups: monitoring techniques, which include electroencephalography (EEG), sensory and motor evoked potentials (SSEP, MEP), and electromyography (EMG), and mapping techniques, which include cortical and subcortical stimulation and brainstem mapping (9,19).

To acquire accurate and adequate information from the surgical area and evaluate the information properly during IONM, different applications of anesthesia can be used, depending on the method of neuromonitoring (4). Since the results of IONM applications in some ways guide,

perhaps even limit the surgical attempt, the anesthesia team undertakes highly significant tasks to acquire information from the patient and ensure the reliability of the interpretations. These tasks start with the induction of anesthesia and continue throughout surgery. Unfortunately, all studies currently available in the literature focus only on the agents to be used in anesthesia (5-7,28), and the role of the anesthesia team during neuromonitoring, as part of the team in the operating room, has been referred to in only one publication (19).

The objective of this study was to determine the role of the anesthesia team during craniotomy operations done with neuromonitoring. We also outlined points to consider for the use of anesthesia based on applications done in our clinic over a 3-year period (November 2005 – November 2008) as well as those reported in the literature.

MATERIALS AND METHODS

Upon receiving approval from the local ethics committee, we collected the files of patients who underwent elective craniotomy with neuromonitoring at Yeditepe University School of Medicine over a 3-year period (November 2005 – November 2008) and we reviewed the neuromonitoring records kept by the neurology team. We recorded the demographic characteristics of the patients, the surgical diagnoses, details of the anesthesia procedures used (agents, perioperative hemodynamic changes, body temperature, the number of patients not given muscle relaxants, changes in the dosage of anesthetic agents during EEG), neuromonitoring methods, and adverse effects. Data are presented as mean values with the standard deviation (SD).

RESULTS

The following procedures were used for 204 patients during craniotomy whose preoperative electrophysiological evaluations were done in our clinic: SSEP

with phase transformation (n:16), motor cortex localization with direct cortical stimulation (n:31), corticospinal tract localization with subcortical stimulation (n:51), direct cortical and subcortical stimulation (n:27), motor speech center (Broca) localization (n:3), cranial nerve monitoring (n:60), MEP monitoring (n:84), SSEP monitoring (n:92), EEG monitoring (n:24), BAEP monitoring (n:35), and cranial nerve monitoring (303 cranial nerves). The ISIS monitoring system was used for recording and the Osiris neurostimulator was used for alerts (both units from Inomed, Teningen, Germany). The demographic characteristics of the patients appear in **Table 1**.

Age (year)	54±13
Gender (F/M) (n)	106/98
Weight (kg)	67±10
Placement of the Surgical Lesion (n)	
Supratentorial	176 (86%)
Infratentorial	28 (14%)
Neuromonitoring method (n)	
SSEP	108
MEP	84
EMG	303
EEG	24
BAEP	35

Table 1. Demographic Characteristics (n:204)

Of these patients, 27% had a diagnosis of major motor deficits, sensory deficits, or both, that interfered with their daily activities preoperatively.

Of the total 204 patients, 176 (86%) monitored preoperatively were operated on in the supine position. The remaining

28 (14%) were operated on in the sitting position.

The heart rate, non-invasive blood pressure and oxygen saturation were monitored for all patients and anesthesia was induced with thiopental sodium and remifentanyl (93%) or fentanyl (7%). After anesthesia induction, muscles were relaxed with *sis-atracurium* (89%) or *rocuronium* (11%), and an endotracheal intubation was done. The anesthesia was maintained in all patients with a propofol (75-200 mcg/kg/min) and remifentanyl (0.15-0.25 mcg/kg/min) infusion and an oxygen-air mixture. Patients were ventilated mechanically to keep the end-tidal carbon dioxide values at 25-30 mmHg. Venous pressure for all patients operated on in the sitting position (n: 28) was tracked through a central vein catheter placed in the subclavian vein and the air inlet was monitored with perioperative transesophageal echocardiography. The invasive blood pressure, end-tidal carbon dioxide level, esophageal temperature (84%) or rectal temperature (31%), and skin temperature were tracked. The mean arterial pressures were stable (76 ± 7 mmHg) intraoperatively. Neither bolus dosage anesthetic agents nor muscle relaxants were administered during the recording. Muscle relaxants were not given to patients having either EMG or MEP monitoring after anesthesia was induced. Propofol dosages for patients having EEG or cortical or subcortical stimulation were reduced (n:32) or discontinued (n:19) just before stimulation. No problems related to anesthesia were noted during the recording of signals. All but 3 patients were successfully extubated. These 3 were either taken to the operating room already intubated or had a tracheostomy preoperatively.

An analysis of adverse effects showed no skin hematoma or skin infection as a result of the needle electrodes. Eight of 27 patients having a needle electrode for vocal cord stimulation experienced a sore throat. Treatment with steam inhalation

and anti-inflammatory lozenges was necessary for 2 of these 8 patients. None of the patients had dysphonia due to neuromonitoring. One patient who underwent posterior fossa surgery needed a tracheostomy on the second day due to dysphagia. All patients were discharged from the hospital without any problem.

DISCUSSION

During the study period, total of 204 patients were given total intravenous anesthesia using by propofol and remifentanyl and signals were recorded with neuromonitoring techniques. With these procedures, the surgeon could be notified about changes in neurologic function early and patients were released with no residual neurological sequelae.

Patients undergoing anesthesia for a craniotomy who are not monitored neurologically are vulnerable to neurological damage during surgery (4,9). Intraoperative neuromonitoring (IONM) was first used in 1898 for the facial nerve. Intraoperative SSEP and EEG were first used in 1966 and 1967, respectively, and since then IONM has come into regular use for all craniotomy operations (17). With neuromonitoring today, procedures that could damage the nervous system can be done more safely (17).

To carry out IONM, a needle is inserted into or surface electrodes are placed on the patient after the induction of anesthesia. All electrodes are sterilized and prepared under the supervision of the neurological team before use. These electrodes may be any of the following: electrodes placed in peripheral nerves (typically the median nerve and posterior tibial nerve) and into the scalp for SSEP monitoring to track the structural and functional integrity of sensory pathways; needle or threaded electrodes inserted into the scalp for MEP monitoring to track the structural and functional integrity of motor pathways; subdermal electrodes inserted into extremities; needle electrodes inserted into relevant muscles

for cranial nerve monitoring; tampon electrodes inserted into the external ear pathway to protect the integrity of the brainstem and auditory pathways during the recording of auditory evoked potentials of the brain stem; and a sterile electrode inserted into the area of a lesion after the surgical incision during electrocorticography. Anesthesiologists and the neurology team, who are all present for the insertion of the electrodes, must act together during preparation of the patient, the induction of anesthesia, and anesthesia maintenance before the craniotomy, all of which is done at an intensive tempo. The working area and times generally overlap before the long surgical attempt. But for the benefit of the patient, the preparatory period, which is prolonged by neuromonitoring, can be shortened by starting the IONM preparations as soon as possible after anesthesia is induced and with the approval of the anesthesiologist. Care must be taken not to dislodge the catheters or fixations during the insertion of the electrodes, especially when areas, such as the forearm and face, which may be used by the anesthesia team for catheterization and catheter fixation, are also being used by the neurology team (**Figure 1**).

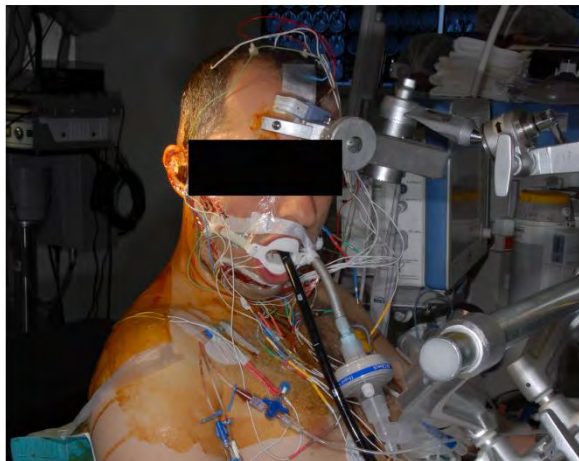


Figure 1. Intraoperative neuromonitoring.

Furthermore, if vocal cord stimulation is to be done during posterior fossa surgery, it is crucial to correctly insert the needle

electrodes into the vocal cord and mouth and secure the fixations during endotracheal intubation (or afterwards by the anesthesiologist) (**Figure 2**).

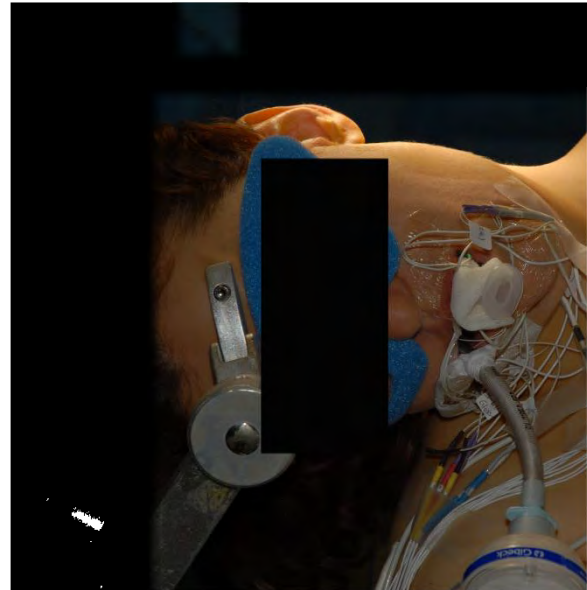


Figure 2. The lower cranial nerve monitoring using needle electrodes for brainstem lesion

The period can also be shortened if the needle electrodes are handed properly to the anesthesiologist performing the intubation, marked immediately, and by working as a team.

If the patient is moved for any reason, the anesthesia team must notify the neurology team of any possible problems with respect to the safety of the electrodes fixed before the patient was placed in the surgical position and from which base values were recorded after positioning. In addition, because the electrodes fixed on the patient, the connected cables, the mobile systems carrying these cables, and the monitors are located in the same area with the anesthesia team, all equipment must be placed so that it does not block access to the patient and enables the anesthesiologist to work freely. The placement of equipment must also allow the neurology team to move easily and

gain quick control of the electrodes and connections whenever necessary.

Depending on the neuromonitoring techniques to be used during surgery, a number of choices must be made with regard to the anesthetic agents administered to the patient.

Motor Evoked Potential (MEP)

Monitoring

Studies of the agents used during MEP monitoring show that all anesthetic agents affect MEP signals negatively, but with the use of inhalation agents, especially those used in high alveolar concentrations, the MEP responses disappear (23,35). With the use of etomidate, ketamine and nitrous oxide, increasing the amplitude during the recording of evoked potentials is not favored for patients undergoing craniotomy (34).

A decrease in the MEP amplitude with inhalation agents at 1 MAC levels, such as halothane, sevoflurane and isoflurane, is observed in patients having a partial neuromuscular blockade. However, the negative effect of halothane on the MEP response at a 0.5 MAC level is less than the effects of isoflurane and sevoflurane (8). This effect is related to the depth of neuromuscular blockage (26). The general opinion is to keep the MAC level of the inhalation agents below 0.5. Administering intravenous anesthesia with propofol and opioids is always the first choice. Depending on the dose, however, propofol decreases the MEP amplitude but does not affect the latency (18). Studies in the literature that compared the effects of propofol to the effects of inhalation agents such as isoflurane and nitrous oxide on the multipulse stimulation concluded that propofol provides better conditions for recording (20).

Opioids have limited effect on the MEP, and are commonly used to supplement anesthetic agents during motor monitoring. They also have only minimal

effects on myogenic potentials when used as a low-dose, continuous infusion (15). However, most of the reported results are limited to the authors' experience and only a few publications mention the depressing effects of alfentanil, fentanyl, remifentanyl and sufentanil (12,21). Nevertheless, fentanyl, sufentanil and remifentanyl are commonly used during and after surgery.

Sekimoto and colleagues concluded that, although the inhalation agent added during intravenous anesthesia with propofol and fentanyl causes hypotension, it affects MEP signals positively (26). Nonetheless, the general opinion among published studies is that, when MEP signals are monitored preoperatively, intravenous anesthesia should be done with propofol and opioids (especially remifentanyl due to its wide range of dosing) and muscle relaxants should not be used after the induction (19,24,25,28,30). It has also been reported that dexmedetomidin, which is often used as an anesthetic agent, can be used safely as a component of total intravenous anesthesia during neuromonitoring (1,31). Since a stable anesthesia depth is recommended during neuromonitoring, infusion of the drugs through target-controlled devices may help (12).

When a patient's MEP responses from motor cortex stimulation are to be monitored, a muscle relaxant should not be used after anesthesia is induced. These patients are given an anesthetic drug more often (22). Each of the stimulations may cause the patient to move; therefore, safety precautions (such as securing the airway and immobilizing arms and legs with a safety belt) must be taken. To avoid injuring the tongue during direct stimulation of the masseter muscle during stimulation of the motor cortex or motorways, the inner mouth must be prepared and the teeth and tongue protected. A recent report describes new stimulation techniques that prevent movement during MEP stimulation without

interrupting the operation, especially during critical procedures such as microsurgery, and we use these techniques in our clinic (33). Thus, the patient can be safely stimulated and the responses are received while the operation is being performed. This technique, however, should not be used for patients with a cardiac pacemaker.

A decrease in the MEP responses or an increase in the stimulation threshold may occur due to a decrease in the patient's body temperature (29). On the other hand, hyperthermia may cause a decrease in latency or an increase in rate. The patient's body temperature must be tracked during surgery because of its affect on MEP and SEP responses, and the neurology team must be notified about critical limits. Care must be taken to keep the patient's body temperature at the base value $\pm$ 2-2.5 degrees C (13).

Somatosensory Evoked Potential

(SSEP) Monitoring

Inhalation agents have negative effects on SSEP, which is used to track the evoked potentials of auditory pathways (2). All halogenated inhalation agents decrease the latency and amplitude in a dose-dependent manner (11,16). In addition, cortical SSEP is more sensitive to these agents. It is possible to record cortical SSEP at levels of 0.1-1.0 MAC with halothane, enflurane or isoflurane. While desflurane and sevflurane allow SSEP recording, they increase the depressant effects of nitrous oxide volatile agents. Intravenous anesthetic agents (properidol, barbiturates, benzodiazepines, etomidate, and propofol) typically depress the SSEP but do so less than inhalation agents (11). Etomidate and ketamine increase the cortical SSEP amplitude. Propofol is preferred since it preserves the somatosensorial potentials better in comparison to all other anesthetic agents (6,7,11). Clinically unimportant changes can occur in the SSEP latency and amplitude after the administration of

opioids. Unlike MEP, SSEP is affected only minimally or not at all by muscle relaxants (23,24). When intraoperative motor tract stimulation is to be done, the use of a muscle relaxant may affect the recording negatively.

Although it is affected less by the use of muscle relaxants, SSEP is affected by changes in the patient's body temperature, the anesthesia depth, and changes in blood pressure. Stable and deep anesthesia, control of heart rate and blood pressure, and stable hemodynamics are essential during SSEP monitoring. Minor changes in the blood pressure may rapidly change the signal; thus, the perfusion pressure must be kept stable (12,16). Most studies show a relationship between the local cerebral blood flow and cortical evoked responses. Cortical SSEP remains steady with blood pressure as low as $\sim$ 20 ml/min/100g. But when the pressure drops to 18 or 15 ml/min/100g, SSEP is disrupted, even lost. The literature notes that the SSEP information acquired during this induced hypotension may be confused with motor function loss in the legs (10). Therefore, rapid changes in the blood pressure must be avoided and bolus dosages of the drugs can be administered to increase the depth of anesthesia. To keep the body temperature stable, the patient must be warmed actively (base temperature $\pm$ 2-2.5 degrees C).

Any hypoxemia occurring during the anesthesia may corrupt evoked potentials. Changes in the carbon dioxide level can decrease spinal cord and cortical blood flow, consequently changing the cortical SSEP. A further decrease in carbon dioxide levels (PaCO_2 20 mmHg) may cause vasoconstriction and ischemia and, again, changes in the SSEP. Therefore, existing evoked potentials must be considered once hyperventilation is started (14).

Although they are used together in most cases, MEP and SSEP can be monitored separately.

Notifying the neurology team about the additional anesthetic doses to be administered is crucial to ensure that the data is interpreted accurately and the surgical team is not misled.

Electroencephalography (EEG)

Inhalation agents decrease the EEG amplitude and reduce the post-initial wave interval in a dose-dependent fashion. While this effect is more apparent for cortically generated waves, it is more noticeable on subcortical, spinal and peripheral evoked potentials. Inhalation anesthetics cause burst suppression on EEG, although this phenomenon is diminished with propofol (10). Bursts are very slow negative waves. With isoflurane and sevoflurane, the bursts begin and end with an abrupt DC shift, whereas with propofol the burst onset and end are smooth. Etomidate and ketamine increase the EEG amplitude (3). Rapid changes in the depth of anesthesia and the use of volatile agents or bolus doses of intravenous agents must be avoided during surgery, especially during excision in the surgical area. During intraoperative EEG recording and before the surgical lesion is reached, the anesthesiologist may need to stop the drug infusion or, especially, decrease the propofol dose. In such cases, the remifentanyl dose may be increased to prevent the patient's waking.

Electrocorticography

Electrocorticography is used to determine the area of seizure activity. When surgery is done with general anesthesia, all the volatile agents, intravenous hypnotics, and benzodiazepines affect spikes negatively (32). Therefore, before the lesion is excised, the doses of anesthetic agents must be decreased and the patient should be under surface anesthesia, if possible. However, the patient's reflexes, such as moving, straining and coughing, must be suppressed with muscle relaxants. Studies show that methohexital, etomidate and alfentanil increase spikes (3,10,32), but

our knowledge regarding this subject is insufficient. To ensure safety, precautions must be taken in case the patient experiences seizures during direct cortical stimulation.

Complications related with Neuromonitoring

In the postoperative period, side effects related to neuromonitoring were rarely seen in our patients and any effects were due to traumatic results related to the needle electrodes. None of the patients complained about these effects, and none of the patients had hematoma or infection in the areas where the needle electrodes were inserted. In our series, no crucial complications resulted from needle electrodes being fixed on vocal cords. But it's difficult to determine whether the sore throat experienced by some patients was due to intubation, the transesophageal echocardiography probe used preoperatively, or needle electrodes fixed to the vocal cords.

Total intravenous anesthesia is the ideal type of anesthesia to use during neuromonitoring. However, balance anesthesia may be administered based on the joint decision of the surgeon and anesthesiologist, and nitrous oxide must be avoided (34). During such anesthesia, agents must be administered after the bolus dose as infusion and with stable doses. Rapid boluses must be avoided as much as possible and the neurology team must be notified. In addition, a neuromuscular block must be used during endotracheal intubation.

During the 3-year period, all of the patients in our clinic were given total intravenous anesthesia with propofol and remifentanyl or fentanyl. Hemodynamics were controlled during neuromonitoring and there were no problems in neuromonitoring due to anesthesia. All patients were extubated after surgery without problems except 3 patients who were intubated before surgery. Drug

doses were titrated to keep the patients hemodynamically stable during anesthesia and the neurology team was notified during the administration of additional bolus doses. Patients were occasionally warmed when required; otherwise, their body temperatures were kept stable.

Neuromonitoring methods are being used more often to protect the neurological status of patients during surgery. Consequently, there is an increase in the number of people in the operating room, the amount of equipment used, and even the number of surgical attempts as a result of the developments introduced by the technology. Neuromonitoring is being used increasingly in neurosurgery to ensure the best outcome possible without harming the patient. Since the techniques are not the sole responsibility of a single team, all teams in the operating room must work together to help the patient. The anesthesia team must work with the neurology and surgery teams in total harmony. Communication among these teams, especially the monitoring personnel, is essential. For the neurophysiologist and the anesthetist, there is a learning curve to master all the tricks of this complex technology and its surgical and anesthetic applications.

ACKNOWLEDGEMENTS

The authors would like to thank anesthesia technicians Hediye Kaya, Murat Şeker, Oğuzhan Yıldırım and neurophysiology technicians Ezgi Davulcu, Emine Sarıyıldız and Gülçin İrim for their contributions during the study period.

REFERENCES

- 1)Bala E, Sessler DI, Nair DR, McLain R, Dalton JE, Farag E. Motor and somatosensory evoked potentials are well maintained in patients given dexmedetomidine during spine surgery. *Anesthesiology* 2008; 109: 417-25.
- 2)Banoub M, Tetzlaff JE, Schubert A. Pharmacologic and physiologic influences affecting sensory evoked potentials: implications for perioperative monitoring. *Anesthesiology* 2003; 99: 716-37.

- 3)Gancher S, Laxer KD, Krieger W. Activation of epileptogenic activity by etomidate. *Anesthesiology* 1984; 61: 616-8.
- 4)Guérit JM. Neuromonitoring in the operating room: why, when, and how to monitor? *Electroencephalogr Clin Neurophysiol* 1998; 106: 1-21.
- 5)Jellinek D, Platt M, Jewkes D, Symon L. Effects of nitrous oxide on motor evoked potentials under total anesthesia with intravenously administered propofol. *Neurosurgery* 1991; 29: 558-560.
- 6)Kalkman CJ, Brink SA, Been HD, Bovill JG. Variability of somatosensory cortical evoked potentials during spinal surgery. Effects of anesthetic technique and high-pass digital altering. *Spine* 1991; 16: 924-9.
- 7)Kalkman CJ, Traast H, Zuurmond WW, Bovill JG. Differential effects of propofol and nitrous oxide on posterior tibial nerve somatosensory cortical evoked potentials during alfentanil anaesthesia. *Br J Anaesth* 1991; 66: 483-9.
- 8)Kallman CJ, Drummond JC, Ribbennick AA, Patel PM, Sano T, Bickford RG. Effects of propofol, etomidate, midazolam, and fentanyl on motor evoked responses to transcranial electrical and magnetic stimulation in humans. *Anesthesiology* 1992; 76: 502-9.
- 9)Karlıkaya G, Bingöl A: İntraoperatif nöromonitorizasyon. In: Bingöl CA, Çelik M, Gürtekin Y, ed, *Klinik nörofizyoloji laboratuvarları uygulama el kitabı*, İstanbul: Namas; 2006: 191-7.
- 10)Keene DL, Roberts D, Splinter WM, Higgins M, Ventureyra E. Alfentanil mediated activation of epileptiform activity in the electrocorticogram during resection of epileptogenic foci. *Can J Neurol Sci* 1997; 24: 37-9.
- 11)Ku AS, Hu Y, Irwin MG, Chow B, Gunawardene S, Tan EE, Luk KD. Effect of sevoflurane/nitrous oxide versus propofol anaesthesia on somatosensory evoked potential monitoring of the spinal cord during surgery to correct scoliosis. *Br J Anaesth* 2002; 88: 502-7.
- 12)Kunisawa T, Ito T, Omachi H, Ozaki Y, Nagashima M, Suzuki A, Takahata O, Iwasaki H. Anesthetic management of a case of craniotomy using TCI of remifentanyl with intraoperative monitoring of motor evoked potential. *Masui* 2008; 57: 467-70.
- 13)Lang M, Welte M, Syben R, Hansen D. Effects of hypothermia on median nerve somatosensory evoked potentials during spontaneous circulation. *J Neurosurg Anesthesiol* 2002; 14: 141-5.
- 14)Ledsome JR, Cole C, Sharp-Kehl JM. Somatosensory evoked potentials during hypoxia and hypocapnia in conscious humans. *Can J Anaesth* 1996; 43: 1025-9.

- 15)Liu EH, Wong HK, Chia CP, Lim HJ, Chen ZY, Lee TL. Effects of isoflurane and propofol on cortical somatosensory evoked potentials during comparable depth of anaesthesia as guided by bispectral index. *Br J Anaesth* 2005; 94: 193-7.
- 16)Lotto ML, Banoub M, Schubert A.Effects of anesthetic agents and physiologic changes on intraoperative motor evoked potentials. *J Neurosurg Anesthesiol* 2004; 16: 32-42.
- 17)Minahan RE. Intraoperative neuromonitoring. *Neurologist* 2002; 8: 209-26.
- 18)Nathan N, Tabaraud F, Lacroix F, Moulies D, Viviani X, Lansade A, Terrier G, Feiss P. Influence of propofol concentrations on multipulse transcranial motor evoked potentials. *Br J Anaesth* 2003; 1: 493-7.
- 19)Pajewski TN, Arlet V, Phillips LH. Current approach on spinal cord monitoring: the point of view of the neurologist, the anesthesiologist and the spine surgeon. *Eur Spine* 2007; 16: 115-29.
- 20)Pechstein U, Nadstawek J, Zentner J, Schramm J, Pechstein U, Nadstawek J. Isoflurane plus nitrous oxide versus propofol for recording of motor evoked potentials after high frequency repetitive electrical stimulation. *Electroencephalogr Clin Neurophysiol* 1998; 108: 175-81.
- 21)Pelosi L, Stevenson M, Hobbs GJ, Jardine A, Webb JK. Intraoperative motor evoked potentials to transcranial electrical stimulation during two anaesthetic regimens. *Clin Neurophysiol* 2001; 112: 1076-87.
- 22)Pollard BJ. The role of muscle relaxants in total intravenous anaesthesia. *Acta Anaesthesiol Scand* 1995; 106: 58-61.
- 23)Reinacher PC, Priebe HJ, Blumrich W, Zentner J, Scheufler KM. The effects of stimulation pattern and sevoflurane concentration on intraoperative motor-evoked potentials. *Anesth Analg* 2006; 102: 888-95.
- 24)Richard A, Girard F, Girard DC, Boudreault D, Chouinard P, Moumdjian R, Bouthillier A, Ruel M, Couture J, Varin F. Cisatracurium-induced neuromuscular blockade is affected by chronic phenytoin or carbamazepine treatment in neurosurgical patients. *Anesth Analg* 2005; 100: 538-44.
- 25)Scheufler KM, Reinacher PC, Blumrich W, Zentner J, Priebe HJ: The modifying effects of stimulation pattern and propofol plasma concentration on motor-evoked potentials. *Anesth Analg* 2005; 100: 440-7.
- 26)Sekimoto K, Nishikawa K, Ishizeki J, Kubo K, Saito S, Goto F.The effects of volatile anesthetics on intraoperative monitoring of myogenic motor-evoked potentials to transcranial electrical stimulation and on partial neuromuscular blockade during propofol/fentanyl/nitrous oxide anesthesia in humans. *J Neurosurg Anesthesiol* 2006; 18: 106-11.
- 27)Sloan, Tod B. Anesthetic Effects on Electrophysiologic Recordings. *Journal of Clinical Neurophysiology. Intraoperative Evoked Potentials* 15: 217-226, 1998.
- 28)Sloan TB, Heyer EJ.Anesthesia for intraoperative neurophysiologic monitoring of the spinal cord. *J Clin Neurophysiol* 2002; 19: 430-43.
- 29)Stecker MM, Kent G, Escherich A, Patterson T, Cheung AT: Anesthesia and temperature effects on somatosensory evoked potentials produced by train stimuli. *Int J Neurosci* 2002; 112: 349-69.
- 30)Svalov S, Khalil M, Merle JC, Agostini MM, Ruggier R, Duvaldestin P. Comparison of duration of neuromuscular blocking effect of atracurium and vecuronium in young and elderly patients. *BJA* 1995; 74: 709-11.
- 31)Tobias JD, Goble TJ, Bates G, Anderson JT, Hoernschemeyer DG.Effects of dexmedetomidine on intraoperative motor and somatosensory evoked potential monitoring during spinal surgery in adolescents. *Paediatr Anaesth* 2008; 18: 1082-8.
- 32)Wyler AR, Richey ET, Atkinson RA, Hermann BP. Methohexital activation of epileptogenic foci during acute electrocorticography. *Epilepsia* 1987; 28: 490-4.
- 33)Yamamoto Y, Kawaguchi M, Hayashi H, Horiuchi T, Inoue S, Nakase H, Sakaki T, Furuya H. The Effects of the Neuromuscular Blockade Levels on Amplitudes of Posttetanic Motor-Evoked Potentials and Movement in Response to Transcranial Stimulation in Patients Receiving Propofol and Fentanyl Anesthesia. *Anesth Analg* 2008; 106: 930-4.
- 34)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* 1989; 24: 253.
- 35)Zenter J, Albercht T, Heuser D. Influence of halothane, enflurane and isoflurane on motor evoked potentials. *Neurosurgery* 1992; 31: 298.