Journal of Clinical Monitoring and Computing (2009) 23:369–390
DOI: 10.1007/s10877-009-9191-y

GUIDELINES FOR INTRAOPERATIVE NEUROMONITORING USING RAW (ANALOG OR DIGITAL WAVEFORMS) AND QUANTITATIVE ELECTROENCEPHALOGRAPHY: A POSITION STATEMENT BY THE AMERICAN SOCIETY OF NEUROPHYSIOLOGICAL MONITORING

Michael R. Isley, PhD, DABNM, FASNM[1], Harvey L. Edmonds Jr., PhD, FASNM[2] and Mark Stecker, MD, PhD, DABNM, FASNM[3]

Published by the Education Committee of the American Society of Neurophysiological Monitoring.

From the [1]Intraoperative Neuromonitoring Department, Orlando Regional Medical Center, Orlando, FL 32806, USA; [2]Department of Anesthesiology and Perioperative Medicine, University of Louisville School of Medicine, Louisville, KY 40207-3633, USA; [3]Neuroscience Department, Marshall University School of Medicine, Huntington, WV 25705, USA.

Received 2 July 2009. Accepted for publication 2 July 2009.

Address correspondence to M. R. Isley, Intraoperative Neuromonitoring Department, Orlando Regional Medical Center, Orlando, FL 32806, USA.
E-mail: Michael.Isley@OrlandoHealth.com

Isley MR, Edmonds HL, Stecker M. Guidelines for intraoperative neuromonitoring using raw (analog or digital waveforms) and quantitative electroencephalography a position statement by the American society of neurophysiological monitoring.
J Clin Monit Comput 2009; 23:369–390

ABSTRACT. Background context. Electroencephalography (EEG) is one of the oldest and most commonly utilized modalities for intraoperative neuromonitoring. Historically, interest in the EEG patterns associated with anesthesia is as old as the discovery of the EEG itself. The evolution of its intraoperative use was also expanded to include monitoring for assessing cortical perfusion and oxygenation during a variety of vascular, cardiac, and neurosurgical procedures. Furthermore, a number of quantitative or computer-processed algorithms have also been developed to aid in its visual representation and interpretation. The primary clinical outcomes for which modern EEG technology has made significant intraoperative contributions include: (1) recognizing and/or preventing perioperative ischemic insults, and (2) monitoring of brain function for anesthetic drug administration in order to determine depth of anesthesia (and level of consciousness), including the tailoring of drug levels to achieve a predefined neural effect (e.g., burst suppression). While the accelerated development of microprocessor technologies has fostered an extraordinarily rapid growth in the use of intraoperative EEG, there is still no universal adoption of a monitoring technique(s) or of criteria for its neural end-point(s) by anesthesiologists, surgeons, neurologists, and neurophysiologists. One of the most important limitations to routine intraoperative use of EEG may be the lack of standardization of methods, alarm criteria, and recommendations related to its application. Lastly, refinements in technology and signal processing can be expected to advance the usefulness of the intraoperative EEG for both anesthetic and surgical management of patients. **Objective.** This paper is the position statement of the American Society of Neurophysiological Monitoring. It is the practice guidelines for the intraoperative use of raw (analog and digital) and quantitative EEG. **Methods.** The following recommendations are based on trends in the current scientific and clinical literature and meetings, guidelines published by other organizations, expert opinion, and public review by the members of the American Society of Neurophysiological Monitoring. This document may not include all possible methodologies and interpretative criteria, nor do the authors and their sponsor intentionally exclude any new alternatives. **Results.** The use of the techniques reviewed in these guidelines may reduce perioperative neurological morbidity and mortality. **Conclusions.** This position paper summarizes commonly used protocols for recording and interpreting the intraoperative use of EEG. Furthermore, the American Society of Neurophysiological Monitoring recognizes this as primarily an educational service.

KEY WORDS. American society of neurophysiological monitoring, electroencephalography, intraoperative neuromonitoring.

INTRODUCTION: RAW AND QUANTITATIVE ELECTROENCEPHALOGRAPHY (REEG AND QEEG, RESPECTIVELY)

This document outlines the guidelines and recommendations for the application and interpretation of intraoperative neuromonitoring using ''raw'' (analog or digital) and quantitative EEG (rEEG and qEEG, respectively) as an index of cerebral function during particular vascular, neurosurgical and cardiac procedures. The following recommendations are based on trends in the current scientific and clinical literature, and guidelines published by other organizations (e.g., American Electroencephalographic Society (AEEGS) [1], American Society of Electroneurodiagnostic Technologists (ASET) [2]), the American Clinical Neurophysiology Society (ACNS, formerly the AEEGS) [3, 4]). This document may not include all possible methodologies and interpretative criteria, nor do the authors and their sponsor intentionally exclude any new alternatives. Furthermore, the American Society of Neurophysiological Monitoring (ASNM) recognizes this as primarily an educational service. The use of electrocorticography for functional mapping is covered under a separate set of intraoperative neuromonitoring guidelines to be published by the ASNM. Furthermore, digital EEG will only be addressed as it relates to the use of computer-processed algorithms and display techniques for enhancing the interpretation and display of the analog EEG. Lastly, for a current and comprehensive review of intraoperative neuromonitoring using rEEG and qEEG, see Freye and Levy [5].

Intraoperative monitoring using rEEG is one of the most traditional methods used to assess cortical function, including perfusion, oxygenation, and anesthetic effects [6]. Typically, artifact-minimized, pre- and post-induction baselines are established, and efforts are made to preserve those baseline waveforms. As stated in the *Guidelines on Intraoperative Electroencephalography for Technologists* established by the ASET, the term EEG ''monitoring'' should be applied ''when the EEG is used to establish if there is a change in the baseline of ongoing brain activity as a result of the surgeon's activity…'' (p. 206) [2]. It is the recommendation of the ASNM that this definition should be more encompassing, including any expected or unexpected anesthetic- or surgically-induced alteration from pre- and/or post-induction baseline waveforms.

INSTRUMENTATION AND ACQUISITION PARAMETERS

The primary purposes of this document are: (1) to recommend intraoperative recording protocols for rEEG and qEEG, and anesthetic regimes that optimize the detection of ischemia, and (2) to outline interpretative criteria and interventional strategies at an advanced level for the detection of ischemia and anesthetic effects. Since detailed guidelines for patient preparation, selection of electrode montage, instrumentation, acquisition parameters, as well as safety and technical considerations have been generated in previous publications, only selective attention will be paid to these particular items. Most of this section is summarized from three sources: (1) *Guidelines in Electroencephalography, Evoked Potentials and Polysomnography* published by the AEEGS [1], (2) the ACNS revisions in 2006 [4], and (3) *Guidelines on Intraoperative Electroencephalography for Technologists* published by the ASET, Inc. [2]. In addition, this section includes the consensus of neuromonitoring experts from within the ASNM, as well as input from experts in related disciplines who have been solicited by the public review process established by the ASNM for the generation of neuromonitoring guidelines.

Patient preparation

Electrode montage

For recording the rEEG and qEEG, a ''full array of scalp electrodes is recommended whenever possible,'' and should be applied using the measurements of the International 10–20 System as recommended by the International Federation of Societies for EEG and Clinical Neurophysiology [1, (p. 207) 2–4]. In particular, standard 8-, 10-, 16-, and 18-channel montages for diagnostic rEEG using transverse bipolar and referential derivations are listed in *Guideline Seven: A Proposal for Standard Montages to Be Used in Clinical EEG* (pp. 30–36) [1]. These guidelines further state ''that no less than 8 channels of simultaneous recording be used, and that a larger number of channels be encouraged,'' and ''that the full 21 electrode placements of the 10–20 System be used.'' (p. 30) [1]. The intraoperative guidelines first established by the ASET declared that ''a minimum of 16 channels should be used whenever possible;'' however, 21 or more channels are optimal for ''EEG surgical testing'' since it allows recording of additional parameters such as electrocardiographic and muscle potentials (p. 208) [4]. The ACNS (2006) revisions of the original AEEGS (1994) diagnostic guidelines still recommended 21 channels [1, 4]. Specifically addressing intraoperative monitoring, only 4 channels may be adequate if a ''lateralize change'' or interhemispheric asymmetry is the ''only desired information'' (p. 209) [2]. If any electrode placement is altered because of the surgical site, this should be documented. For direct cortical monitoring, the position of the ''strip'' or grid electrode array will be determined by the area of cortical surface under study. It should also be noted that

the adequate number of channels deemed necessary for intraoperative monitoring remains controversial and unresolved, and has been based primarily on the recommendations used in the diagnostic setting. These issues, as well as specific montages using fewer channels that have been purported to optimize the detection of ischemia during carotid endarterectomy and cardiac surgeries, are discussed in detail in Section Electrode montages for CEA surgery.

Impedance of recording electrodes

Electrode impedances should be less than 5,000 Ω and relatively equal or balanced (i. e., the inter-electrode differences should not exceed 2,000 Ω) [1–4]. This reduces the risk for internal and external noise interference, and distorted signals [7–9]. Great care should be paid to achieving low electrode impedances prior to the start of surgery and should be rechecked whenever there is any artifact present in the signal.

Electrode types and applications

Metal disk or ''cup'' electrodes (gold, silver, or tin) applied using the method of collodion-soaked gauze and filled with a conductive gel is the preferred application, particularly for long-term recordings [1–4]. In addition, this allows for routine acquisition of pre-surgical baselines where detection of pre-existing asymmetries or interhemispheric differences may be important in the intraoperative management of vascular (e.g., carotid endaterectomy) and cardiac (e.g., particularly cardiopulmonary bypass procedures) surgeries. However, in recent years the use of subdermal needle electrodes has been adopted by many clinicians for use with anesthetized patients, mostly due to practical considerations (e.g., such as in the case of cerebral aneurysm surgery where sterility is a requirement).

Along with the careful selection of the type of recording electrodes from vendors, the following tips may be of value when performing intraoperative neuromonitoring using rEEG and qEEG:

1. The recording electrode montage should consist of one type of electrode, with no mismatching of metals (e.g., silver disk, gold disk, tin disk, stainless steel needles, platinum-iridium needles, or transcutaneous, silver-silver, chloride electrodes)
2. All disk electrodes must be of good quality with intact surfaces (i. e., there is no visible pitting on the surface). Reusable electrodes with inconsistent surfaces can create uneven current densities.
3. All surface electrodes should be applied with sufficient conductive gel to ensure low impedances, and the impedances should be checked if artifact is present.
4. The method used to adhere surface electrodes should result in a secure placement (e.g., collodion-soaked gauze).
5. Electrode leads and cables used for the neuromonitoring system should not be bundled with the cables used for any other device. Separating the leads and cables will reduce the chance of electrical coupling between adjacent lines.
6. Shielded leads may be used. The length of the leads to the preamplifier should be minimized and the leads braided in order to reduce electromagnetic contamination.

The use of needle electrodes may be necessary if sterile technique is required, the preoperative setup is precluded, or if the size of the surface electrode encumbers the surgical procedure. It is generally acknowledged that the use of needle electrodes presents a set of special considerations, including infection control, electrical safety, and higher impedance values resulting in higher levels of electrical noise [1, 2, 4]. Any routine use has been discouraged [1, 2, 4]. If needle electrodes are required, several considerations and requirements should be met:

1. The use of disposable, sterile, subdermal, needle electrodes is strongly recommended.
2. The electrode site should be prepared in an antiseptic fashion.
3. A strict adherence to infection control, and sterilization policies and procedures of the clinical institution must be met.
4. Needle electrode application should involve a subcutaneous insertion by well-trained personnel.
5. The needle shaft should not be bent before or after insertion.
6. Skin stapling is one simple method to prevent dislodging.
7. The use of needle electrodes increases the risk of electrical burn, and biological and electrical noise interference.
8. Orientation of needle insertion should involve a parallel, anteroposterior alignment since misalignment may cause artifactual amplitude asymmetries or distortions.

For intraoperative use, platinum–iridium needles are recommended because of lower impedances than stainless steel [2]. An alternative is spiral needles which are not as easily dislodged [2].

Instrumentation

Equipment selection for intraoperative neuromonitoring

Although different neuromonitoring techniques (e.g., rEEG and qEEG, transcranial and microvascular Dopplers,

sensory and motor evoked potentials, and cerebral oximetry) offer unique information about cerebral function and perfusion, no single technique or corresponding alarm criteria have proven entirely reliable for detection of both ischemia and embolism. Whenever possible, a multimodality neuromonitoring protocol should be utilized, since this would potentially afford a better neurological outcome associated with anesthesia and surgery [10, 11]. When considering what neuromonitoring technique(s) to employ, the choice will largely depend on a clinical center's equipment, personnel availability, and level of expertise.

Filter settings

As outlined in the Sections Filter settings–2.2.2.3 of the *Guidelines on Intraoperative Electroencephalography for Technologists* established by the ASET [2], filter settings: (1) ''will depend on the frequency of the waveforms of importance,'' (2) should be determined by the ''judicious use'' which ''will allow emphasis on a particular event as it occurs,'' and (3) should be used to acquire and save data with a wider bandpass than those used for on-line or off-line digital filtering (pp. 209–210). A high frequency setting of 70 Hz or higher is considered optimal (if available), but not lower than 35 Hz since significant distortion and attenuation of spikes or anesthetic-induced, higher frequency activity can occur. However, exceptional cases where sources of noise cannot be eliminated (e.g., jaw clenching in an awake patient) and rEEG cannot be easily visualized, a lower high-frequency filter setting may be required. A low frequency filter of 0.3–1.0 Hz is recommended, as this setting allows the display of slow frequency activity without significant baseline variability and loss of sensitivity for the detection of ischemic events or anesthetic-induced, slow, frequency activity. A greater than 1 Hz setting should be restricted to brief periods when viewing low-voltage beta or spike activity. It should be noted that the setting of the hardware low frequency filter greatly affects the recovery of the amplifier after the application of electrocautery, If this filter is set too low, there may be prolonged recovery and blocking. Lastly, a 60-Hz notch filter is often necessary to eliminate extraneous, irreducible noise in the operating room (OR) arena, such as line frequency artifact. However, this should only be used if other measures against 60 Hz interference fails, since this notch filter can distort or attenuate spikes, and other faster frequencies. In addition, great care should be exercised to reduce excessive noise by trouble-shooting which may include removing, replacing, or unplugging any unwanted current source (e.g., OR table, blood and body warmers, microscope, extraneous power supply, etc.) which does not interfere with normal clinical practice or distraction from neuromonitoring.

Safety and technical considerations in the operating room

Electrical safety and maintenance

The selection and operation of any neuromonitor for rEEG and qEEG should conform to the recommendations set forth by the AEEGS [1], ASET [2] and ACNS [3, 4] and the reader is encouraged to review the appropriate sections contained in these documents. Routine maintenance, evaluation of leakage current and inspection of the overall electrical integrity of the equipment should be completed 2–3 times a year (or as per the biomedical engineering protocol at your institution), or at any time faulty or malfunctioning equipment is suspected.

In particular, one item of interest involved the use of safety connectors for the recording leads, as the pin-style connectors pose the risk of inadvertently being connected to a voltage source. This issue was addressed in Section 2.4.1.1 of the *Guidelines on Intraoperative Electroencephalography for Technologists* established by the ASET [2].

> In 1997 the Food and Drug Administration (FDA) issued mandatory standards for electrode lead wires and patient cables. By May 9, 2000 all electrodes used in the O.R. must comply with Subclause 56.3 (c) of the International Electrotechnical Commission (IEC) standard 6 (Federal Register 1997). The 1.5 mm or 1.0 mm covered connector electrode (DIN safety connectors or female electrode) meets this standard. If 2 mm unprotected lead wires (male electrodes) are still in use, they must utilize adapters that are not readily detached (p. 211).

Disinfection procedures and general infection control guidelines

Disinfection procedures for personnel, equipment and electrodes are recommended to be consistent with those detailed in and endorsed by the Board of Trustees of the ASET: (1) *Infection Control and the Electroneurodiagnostic Department: 1994 Guidelines* [12], and (2) *Infection Control: 2000 Review and Update for Electroneurodiagnostic Technologists* [13], as well as the policy and procedures of the individual institution. In particular:

1. Sterile areas should always be respected and non-sterile personnel should minimize their activity around those areas.
2. Proper surgical attire should be worn, including scrubs, hat, mask, appropriate eye care and shoe covers.
3. Neuromonitors and ancillary equipment such as cables and the electrode jackbox should be cleaned with a high-level disinfectant after each case.
4. All equipment used in the OR should be properly isolated electrically and protected from contamination or exposure to body fluids.

5. Gloves should be routinely worn in high-risk areas such as the ICU and OR arenas, particularly when touching patients with wounds, bloody areas, and other secretions or excretions present.
6. Gloves should also be worn when handling any neuromonitoring item soiled by bodily fluids (e.g., electrodes, patient cables).
7. Hand washing before and after patient contact should be done with a hospital-approved antimicrobial preparation.
8. Disposable, subdermal, needle electrodes when used should be disposed in the appropriate manner for sharp objects.
9. Reusable, needle electrodes should be washed, soaked in Clorox (1:10 solution) for 10–15 min, packaged, and taken to sterile processing for steam sterilization, typically for 1 h at 120°at 15 psi.
10. Intraoperative neuromonitoring personnel should adhere to standard precautions which guard against the risk of accidental exposure to blood and body fluids, and be informed about contraction of and inoculation against Hepatitis B.
11. Any and all institutional or manufacturer's requirements should be respected.

DOCUMENTATION OF INTRAOPERATIVE REEG AND QEEG

In general, an intraoperative neuromonitoring log and patient protocol forms should be completed for each case. These records should include detailed information such as: demographic data, diagnosis and type of surgery, equipment and neuromonitoring procedures, neuromonitoring personnel, intraoperative events, and clinical outcome, if available. In particular, great care should be exercised to produce artifact-minimized, hardcopy, samples of both the continuous rEEG and qEEG (if employed) before, during, and after the various routine and critical anesthetic and surgical events of any surgery (e.g., pre- versus post-induction, surgical exposure, cross-clamping of the carotid arteries, carotid artery shunting, selective shunting, heart cannulation, onset and offset of cardiopulmonary bypass procedure, aneurysm clipping, skin closure, etc.,). In addition, all vital signs (e.g., heart rate, blood pressure, temperature, etc.), anesthetic agents and levels, any notable intraoperative changes in the rEEG or qEEG, any critical alerts or alarms to the surgeon and anesthesiologist, and any interventions or corrections in surgical or anesthetic care based on intraoperative neuromonitoring should all be appropriately documented on the pertinent hardcopy of neural activity and/or neuromonitoring log for a given patient. Most importantly, any change in the neuromonitoring data, regardless of its etiology, should be reported to the surgeon and anesthesia staff in a timely fashion. Lastly, requirements for storage of these data are dictated by state law and the policies of the individual institution.

CREDENTIALS FOR AND INTERPRETATION OF INTRAOPERATIVE NEUROMONITORING

Information contained in this section may be helpful in decision-making processes for granting privileges concerning the staff performing and supervising intraoperative neuromonitoring. This section does not address local, state or federal laws nor does it necessarily account for community standards of acceptable practice. The reader is encouraged to review the ASNM's Credentialing and Competency Policy Statement for Intraoperative Neuromonitoring Staff) [14]. Over the past decade, there have been a number of guidelines developed by various professional societies for intraoperative neuromonitoring: (1) International Federation of Clinical Neurophysiology (IFCN), 1993; (2) ACNS, 1994; (3) ASET, 1998; (4) International Organization of Societies for Electrophysiology Technologist (OSET), 1999; (5) ACNS, 2000; and (6) ASNM, 2001 [14]. Only those guidelines for staffing and interpretation (i. e., the professional and technical levels) are summarized in this section, however, it is strongly recommended that all of the guidelines should be reviewed and considered [14].

Clinical levels and credentialing organizations

There are two levels of clinical services related to performing intraoperative neuromonitoring: a supervisory/interpretative level and a technical level. There are several organizations which offer credentials at the professional/supervisory level which specifically qualify competency for interpretation of intraoperative neuromonitoring assessment: (1) The American Board of Neurophysiological Monitoring (ABNM) grants recognition as a Diplomate (DABNM), (2) the American Board of Psychiatry and Neurology (ABPN) grants a status as ''Certification in the Subspecialty of Clinical Neurophysiology,'' (3) the American Board of Clinical Neurophysiology (ABCN) grants a certification ''with special competency in intraoperative neuromonitoring,'' and (4) the American Board of Electrodiagnostic Medicine (ABEM) provides a Diplomate certification in neurophysiology concentrating on EMG and evoked potentials. At a technical level, the American Board of Registration of Electroencephalographic and Evoked Potential Technologists, Inc. (ABRET) offers a Certification in Neurophysiologic Intraoperative Monitoring (CNIM). The websites for

these organizations and their specific requirements for certification are listed: (1) ABNM: www.abnm.info (2) ABPN: www.abpn.com (3) ABCN www.abcn.org (4) ABEM: www.abemexam.net and (5) ABRET: www.abret.org [14].

Professional level

Interpretation of intraoperative neuromonitoring data and any recommendations regarding the consequences or intervention are the responsibility of a qualified physician or clinical neurophysiologist. The educational degree of said physician or clinical neurophysiologist was originally defined as "M.D., Ph.D., or D.O." as established in *Guideline 11: Guidelines for Intraoperative Monitoring of Sensory Evoked Potentials* published in the *Journal of Clinical Neurophysiology*, 11: 77–87 (1994), and was originally restricted to "experienced in EPs" [1]. The requirements were expanded to include any doctoral level in a physical science, life science, or clinical allied health profession from an accredited institution, as well as other neuromonitoring modalities by the ABNM in 2010 and 1999, respectively. "…[T]he ACNS Guidelines [2000]… endorse its previously established principle: the clinical neurophysiologist is responsible for the conduct and interpretation of all intraoperative clinical neurophysiology procedures" (p. 3) [3]. There are a number of appropriate qualifications for the supervision and interpretation of intraoperative neuromonitoring protocols [14]. In the above Section Clinical levels and credentialing organizations, the various credentials required of an appropriate "clinical neurophysiologist" are listed [14].

Technical level

Administered by ABRET, CNIM certification is intended for the monitoring personnel involved with the technical aspects of monitoring. The primary role of the technologists is implementation of procedures related to: (1) patient preparation, (2) operation of instrumentation for continuous neuromonitoring, (3) recognition and correction of artifact, (4) establishing appropriate baselines, (5) recognition of critical periods during anesthesia and surgery, (6) detailed and accurate documentation of waveforms, anesthetic and surgical events, vital signs, and any deviations from baseline data and interventions taken, and (7) alerting the appropriate intraoperative neurophysiologist for interpretation of the waveforms.

According to the original AEEGS's (1994) and ACNS's (2006) revisions for *Guideline Four: Standards of Practice in Clinical Electroencephalography:* "Under no circumstances should a technologist, however well-qualified and experienced, have primary responsibility for clinical interpretation of EEGs (p. 14) [1] and (p. 105) [4], respectively. "Therefore, at no time is the EEG technologist allowed to provide the surgeon with an interpretation, discuss what the waveforms mean, or direct the surgeon's action (p. 217) [2].

Likewise, according to the *Code of Ethics and Standards of Practice* outlined by the ABRET [15], an ABRET registered technologist or certified individual shall: (Item 7) "Refuse primary responsibility for interpretation of testing or monitoring of Electroencephalograms, Evoked Potentials, or Neurophysiologic Intraoperative Monitoring. Individuals who are licensed or otherwise authorized by practice standards to provide interpretation are excluded" (p. 1). As stated in the current *Guidelines on Intraoperative Electroencephalography for Technologists* established by the ASET [2]:

> For monitoring procedures when the clinical neurophysiologist is not present physically nor present by means of remote mechanism and when the technologist is providing the waveform description, the surgeon or anesthesiologist is responsible for ascertaining the interpretation, diagnosis, and a course of action; with the medical director of the electroneurodiagnostic department or an experienced clinical neurophysiologist bearing the responsibility for educating the surgeon or anesthesiologist regarding possible interpretation and potential consequences of the waveforms identified by the technologist…… [T]he EEG technologist….. may furnish the surgeon a description of the waveforms being recorded….. The term 'description' is not synonymous with the term 'interpretation' (p. 216).

Guidelines of the ACNS regarding interpretation and communication related to intraoperative neuromonitoring

The ACNS's (2000) guidelines [3] for the use of intraoperative rEEG and qEEG stated that:

> ….. The clinical neurophysiologist interpreting each monitoring study is responsible for determining the degree to which the ENDT [electroneurodiagnostic technologist] conducting the study is qualified.
>
> ….. The clinical neurophysiologist has the overall responsibility for the conduct of the intraoperative monitoring, including control of its technical quality as well as responsibility for the interpretation of the intraoperative EEG. ….. For all monitoring, a qualified neurophysiologist should be available, in a timely fashion to offer interpretation advice, including during critical periods of monitoring ….. When not providing

contemporaneous (real-time) interpretation, the clinical neurophysiologist, must establish procedures to be reached rapidly by the ENDT ….., review specific aspects of the recording and communicate with the surgeon if the occasion occurs. …. [I]f during a period in which the clinical neurophysiologist is not available for contemporaneous EEG interpretation and the ENDT believes EEG changes are present, the technologist should notify the surgeon of a reason for concern then contact the neurophysiologist. The specific method of communication between the clinical neurophysiologist and the ENDT ….. may vary ….. These methods may be based upon, but are not limited to: on-site interpretation in the operating room or on-line interpretation at a remote site.

Methods which include off-site interpretation must provide for direct and contemporaneous (real-time) communication between the clinical neurophysiologist, the ENDT and if necessary, the surgeon and other members of the ….. operative teams during the surgical procedure (pp. 4–7).

SURGICAL PROCEDURES

Neuromonitoring for carotid endarterectomy (CEA) surgery

CEA is a surgical procedure designed to prevent ischemic stroke by removing the atheromatous lesion at the carotid bifurcation (a high-grade stenosis of 70–99%), and restoring the patency of the carotid vessels to an almost normal level. CEA surgery remains the most commonly performed non-cardiac, vascular surgery in the United States (US), with more than 1 million performed in the last 40 years at a current annual cost of $1.2 billion [16]. ''Further increase has been prompted by the Asymptomatic Carotid Atherosclerosis Study (ACAS) finding of a 50% risk reduction of ipsilateral stroke in asymptomatic patients with greater than 60% carotid narrowing'' (p. 42) [5, 17]. Currently, it is estimated that 170,000 CEAs are performed per year in the US and rising due to the aging of our society [18]. Concern about adequate cerebral perfusion and embolism during CEA surgery has made intraoperative neuromonitoring more common during this surgery than with any other type of cerebrovascular procedure [11]. A variety of techniques have been utilized to monitor for adequate cerebral circulation and neural function, and detection of embolism, particularly for routine and selective shunting during CEA surgery [11]. These options include the following: (1) electrical activity of the brain (e.g., rEEG and qEEG, and median nerve somatosensory evoked potentials (MN SSEPs)), (2) intracranial, cerebral circulation measures (e.g., subjective estimation of carotid artery back-bleeding, internal carotid artery ''stump'' pressure, regional cerebral blood flow (rCBF) using ''washout'' techniques, and cerebral blood flow velocity measurements of the middle cerebral artery (MCA) using transcranial Doppler (TCD)), and (3) oxygen saturation evaluation (e.g., jugular bulb oxygenation and noninvasive cerebral oximetry). Although each technique offers unique information, no single method has proven entirely reliable and flawless for detecting both cerebral ischemia and embolization. However, a multimodality neuromonitoring strategy which combines several of these techniques affords a better neurological outcome [11, 19]. In a survey of anesthesiologists, some form of intraoperative neuromonitoring was performed during CEA surgery in almost 90% of the cases, with EEG being the most commonly performed type of neuromonitoring in 67.5% of the cases (Table 1) [20].

Perioperative stroke rate for CEA surgery

One of the most serious perioperative complications associated with CEA surgery is stroke [21, 22]. The literature has indicated that 2–21% of patients having CEA surgery experienced a stroke [22]. However, the Ad Hoc Committee on Carotid Surgery Standards of the Stroke Council of the American Heart Association has set standards for the stroke morbidity/mortality rates associated with CEA surgery [23]. The committee recommended that the ''30-day mortality rate from all causes for all carotid endarterectomies should not exceed 2%.'' In addition, the recommendations for the combined morbidity and mortality stroke rates during and after CEA surgery, which should not prompt individual peer review, ranged from 3 to 10%, contingent on clinical conditions (Table 2). In particular, CEA surgery for asymptomatic

Table 1. Current types of intraoperative neuromonitoring performed for CEA surgery (adapted from Cheng et al. [20])

Modalities of neuromonitoring during cea surgery	% of cases
No neuromonitoring	10.2
EEG	67.5
Awake, sedated patient	19.6
SSEPs	13.9
Carotid stump pressure	11.3
Transcranial Doppler	8.2
Cerebral oximetry	1.0
Cerebral function monitor	0.5
XENON 133	0.5

Table 2. Recommendations of the Stroke Council of the American Heart Association for acceptable stroke morbidity and mortality rates associated with CEA surgery based on clinical conditions [23]

Clinical indication	Stroke morbidity/ mortality rate (%)
Asymptomatic stenosis	<3
TIA	<5
Ischemic stroke	<7
Recurrent stenosis	<10

disease should produce a stroke rate of 3% or less, whereas operations performed for transient ischemic attack (TIA) and ischemic stroke should be associated with a stroke morbidity of 5% or less and 7% or less, respectively. Lastly, operative repair of recurrent symptomatic carotid stenosis should yield the highest morbidity of approximately 10%.

The main factors contributing to the above outcomes include: (1) hypoperfusion during cross-clamping, (2) air or particulate emboli occurring during shunting procedures and reperfusion of the carotid, (3) reperfusion cerebral hyperemia, (4) postoperative emboli (debris remaining in the vessel or clot formation at the operative site), and (5) postoperative hypotension and hypertension [11]. Studies evaluating the operative morbidity and mortality rates associated with CEA surgery are usually retrospective, and the differentiation between intraoperative and postoperative stroke is often unclear. In fact, many authors simply describe a neurological deficit in relation to CEA surgery as perioperative. However, Jansen et al. [19] have categorized the time of stroke. Sixty-eight percent of perioperative strokes occurred intraoperatively. Fifty percent of the intra-operative strokes were probably caused by hemodynamic factors. The remaining half was probably of thrombo-embolic origin and primarily occurred during surgical manipulation of the carotid arteries.

Electrode montages for CEA surgery

Over the years, much controversy has ensued over the electrode montage used (bipolar versus referential) and the number of channels (2, 4, 8, 16, or 21) deemed adequate for intraoperative monitoring of the rEEG and qEEG [1–4, 24–28]. To date, no randomized, prospective studies have been published addressing this issue. Furthermore, no studies have been published that determine such requirements for intraoperative EEG monitoring when using multimodality protocols that included TCD, MN SSEPs, or cerebral oximetry. Since this issue remains unresolved, this section represents the spectrum of recommendations/opinions that currently exists.

The primary argument for multi-channel recordings (greater than 8–12) in the diagnostic setting is the need "…to ensure that EEG activity having a small area of representation on the scalp is recorded and to analyze accurately the distribution of more diffuse activity" (p. 3) [1] and (p. 87) [4]. This premise underlies the concepts and proposals advocated by others for intraoperative neuromonitoring [2, 3, 24, 25]. Originally in 1994, as stated in *Guideline One: Minimum Technical Requirements for Performing Clinical Electroencephalography*, the AEEGS concluded that a "minimum" of 8 channels of simultaneous recordings are required to show cortical areas which produce "most normal and abnormal EEG patterns," however, "16 channels are now found to be necessary…" (p. 2) [1]. Recently published revisions contained in: *Guideline 1: Minimum Technical Requirements for Performing Clinical Electroencephalography* by the ACNS [4] are the same as those proposed in the earlier AEEG's (1994) guidelines: "[a]ll 21 electrodes and placements recommended by the International Federation of Clinical Neurophysiology (IFCN; Jasper HH, 1958, 1983) should be used. The 10–12 System is the only one officially recommended…" (p. 87). Traditionally for intraoperative recordings, a bipolar, anterior-posterior, 16-channel, montage was proposed since this derivation "is less prone to artifact and electrical interference, and gives easily appreciated inter-hemispheric comparative data" (p. 748) [25]. The Therapeutic and Technology Assessment Subcommittee of the American Academy of Neurology (1990) stated that "…monitoring should be carried out at least at the anterior and the posterior regions over each hemisphere. Sixteen channels are preferable to identify occasional embolic complications" (p. 1) [24]. According to the ACNS's (2000) intraoperative guidelines, "[u]sing less than 8–12 channels is insufficient. Displaying the EEG in the form of bipolar montages is preferred in most cases" (p. 8) [4].

The *Guidelines on Intraoperative Electroencephalography for Technologists* established by the ASET stated that [2]:

> ….. for EEG surgical monitoring (e.g., carotid endarterectomy) where a baseline has been obtained and outcomes at critical times are compared to that baseline, there is some evidence that 4-channel recordings may be adequate if a lateralized change is the only desired information. When only 4 channels are visualized, it is imperative to understand that the selection of electrode sites is crucial, and that localized changes may not be detected. The surgical monitoring policy regarding the number of recording channels utilized and the montage selection must be established by the medical director in concert with the surgical monitoring staff or technologists, and interpreting clinical

neurophysiologist. This policy must allow the surgical monitoring staff to feel secure in the adequacy of displayed information (p. 209).

A recent evaluation of 2-, 4-, and 16-channel monitoring using rEEG and qEEG for detection of cerebral ischemia led Edmonds et al. [28] to conclude that 8 scalp-recording electrodes may be sufficient to detect cortical ischemia in both the anterior and posterior watershed areas. A previous report by some of the same proponents who have traditionally advocated many channels (>8) have suggested that a minimum of two may be appropriate, yielding a high degree of sensitivity and specificity if the appropriate montage is used [26]. This particular montage selectively recorded rEEG from the area of cortical hemisphere where blood supply is most compromised by CEA surgery, specifically the middle cerebral artery (MCA) distribution [26]. Significant changes in rEEG were defined as a >50% decrease in amplitude of 8–15 Hz activity. Using this alarm criteria, the channel pair combinations shown in the Table 3. yielded 100% sensitivity and 100% specificity for detection of an ischemic episode within the cortical areas perfused by the MCA. The channel pair combinations shown in Table 3. provide a frontoparietal plus a frontotemporal coverage, which correlates with the distribution of the blood supply of the superior and inferior M2 branches of the MCA, respectively. However, it should be noted that using combinations of these montages, even with experienced personnel, does not necessarily guarantee perfect sensitivity and specificity, and an uneventful outcome.

Shunting and rEEG

One of the most controversial issues associated with CEA surgery, and its mortality and morbidity rates, is what clinically optimal event should occur following carotid cross-clamping and during the period of plaque removal: (1) the use of universal temporary carotid shunting, (2) selective shunting, or (3) no shunting. The potential consequences of cerebral ischemia and/or embolism appear unavoidable regardless of the technique employed, and thus can only be minimized at best. Although in principle, the routine use of shunting may eliminate the need for neuromonitoring and measurement of collateral cerebral circulation, the risk of iatrogenic problems associated with shunting ranged from 0.5 to 3% [29, 30]. The inherent risks associated with shunting can be attributed to: (1) technical problems which limit the surgeons ability to expose and dissect the atheroma, especially the distal segment, (2) shunt kinking or occlusion due to improper placement which results in ischemia, (3) intraoperative thrombosis, (4) increased risk of cerebral embolization of atherosclerotic debris and air into the distal cerebral circulation, and (5) potential intimal damage resulting in postoperative thrombosis at the operative site.

Selective shunting is considered by many to offer the optimal surgical management of CEA surgery, tailoring to the needs of the individual patient and thus minimizing the above risks [31]. However, there remains a lack of universal selection criteria for its use and the type(s) of neuromonitoring required. Although the routine practice of shunting varies among surgeons, intraoperative cerebral ischemia is an inevitable consequence of temporary mechanical carotid occlusion, requiring shunting in at least 9–20% of patients based on rEEG criteria [32, 33]. Others have reported a higher incidence of 20–35% for the occurrence of ipsilateral ischemia that required shunting [34]. Lastly, selective shunting based on neuromonitoring, particularly major changes in the rEEG, may reduce the incidence of stroke 10-fold [6], and even "close to an irreducible minimum" (0, 0.3, and 1.1%) [35–37, respectively]. However, it should be noted that some authors have reported good outcomes when all patients were shunted without the use of intraoperative neuromonitoring [38].

Table 3. Bipolar montages for CEA surgical monitoring which yielded a 100% sensitivity and 100% specificity for detection of cerebral ischemia [26]

Channel pair combinations for rEEG with 100% sensitivity and 100% specificity					
F3-C3	F7-T3	T3-T5	C3-P3	F7-T3	T3-T5
and	or		and	or	
F4-C4	F8-T4	T4-T6	C4-P4	F8-T4	T4-T6

Incidence of ischemia detected by rEEG during CEA surgery

When considering the methods for assessing brain function and ischemic insult during CEA surgery under general anesthesia, it seems clear that the traditional "gold standard" is conventional, multi-channel rEEG. Changes in the rEEG are typically characterized by alterations in both frequency and amplitude. These changes have been reported in 9.8–35% of patients after mechanical occlusion of the carotid artery prior to arteriotomy, with the majority of these clamp-related changes occurring within 1 min following cross-clamping [25, 34]. Of patients exhibiting any clamp-related changes in the rEEG, 80% appear within the first minute with 69% appearing within 20 s. Major changes begin earlier, with more than 80% of these occurring within the first 20 s [25].

Establishing intraoperative baselines and recording strategies for optimizing the detection of cerebral ischemia

Firstly, artifactual contamination must always be a concern in interpretation of the intraoperative rEEG and qEEG. Intraoperative monitoring using rEEG and qEEG should also be continuously recorded throughout all anesthetic and surgical events. A preinduction, premedicated baseline should be recorded in order to assess any pre-existing asymmetries or abnormalities. In addition for intraoperative rEEG, it has been recommended that "at least a 10-min baseline pre-clamp recording while anesthetized is essential to appreciate any clamp-associated changes" (p. 748) [25]. Similarly, a 10-min period following restoration of blood flow upon clamp release is also required to ensure that any intraoperative changes have resolved. Despite these statements, it is important to record the rEEG continuously during the procedure. In particular for patients with significant stenosis of the carotid arteries, the development of ischemia as a result of minor blood pressure fluctuations may occur prior to clamping and the endarterectomy.

For rEEG, recording strategies altering the display sensitivity and/or paper speed (if digital EEG, the computer display time base or sweep speed) have been suggested in order to optimize the visual detection of an ischemic event. Following carotid artery cross-clamping, one of the most common initial changes in the rEEG is a reduction of relatively low-amplitude, beta activity. A sensitivity change to 3 or 5 μV/mm [25], or a pre-clamp sensitivity setting which achieves an average pen deflection of at least 1 cm aids in the detection of such amplitude decreases [40]. Decreasing paper speeds (or time bases) to 5, 10 or 15 mm/s for intraoperative monitoring of the rEEG may also visually enhance the detection of an ischemic episode by accentuating slow-wave asymmetries and voltage changes [25, 40]. However, it should be noted that these time bases (or paper speeds) will most often make it difficult to appreciate the complete morphology of the pre-event, baseline rEEG.

The type of anesthetic agent, as well as controlling its maintenance level to a steady-state, are also important for optimizing conditions for the detection of cerebral ischemia during routine and critical anesthetic and surgical events. For example, a bolus of intravenous (IV) drugs such as barbiturates, propofol, or etomidate, or increasing inhalational agents at critical times during surgery (e.g., before cross-clamping of the carotid arteries) should be avoided. Such maneuvers can cause moderate to severe rEEG depression, making detection of ischemia impossible or difficult at best. These anesthetic concerns and desirable protocols are discussed in more detail in Section Anesthetic effects on the rEEG during CEA surgery.

rEEG: Ischemic effects associated with CEA surgery

The characteristic alterations in rEEG due to ischemia range from subtle changes, such as a mild loss of beta/alpha activity and a minimal increase in slower frequencies, to a complete loss of all detectable electrical activity. The most common and sensitive analog EEG change is attenuation of anesthetic-induced, fast activity (low beta, high alpha), which occurs in 14–47% of the patients following carotid cross-clamping [41, 42]. Increased delta activity is almost always associated with decreased amplitudes of higher frequency activity. The amplitude of the rEEG may increase (e.g., high-amplitude, slowing phenomenon) or decrease. A major loss of amplitude and the appearance of delta waves for longer than 30 min have been associated with postoperative deficits [42]. Frequency and amplitude changes are usually ipsilateral to the occlusion, although bilateral changes may occur with severely compromised collateral circulation [25, 41]. Unilateral changes occur more than twice as often as bilateral changes [25]. After shunt placement, focal changes in the rEEG typically resolve in 2–7 min, although longer times may be required. In the event of amplitude and frequency changes, it has been suggested that an abrupt change, particularly focal, may be associated with embolic causes, whereas a more gradual decline is probably due to hemodynamic causes [11]. Furthermore, severe changes not associated with cross-clamping during conditions of stable anesthesia and blood pressure control are likely a result of embolic complications.

Alarm criteria for ischemic thresholds using rEEG

Various alarm criteria or guidelines for intervention have been suggested to characterize the severity of changes in the rEEG, particularly those associated with carotid cross-clamping and shunting (Table 4). As a general rule for a major change prompting immediate notification of the surgeon for intervention, Jenkins et al. [39] proposed that a loss of 75–80% or more in amplitude should be treated as a complete loss of all electroencephalographic activity. At the Mayo Clinic, major clamp-related changes were defined as changes producing at least a 75% alteration of all activity, and/or a two-fold or greater increase of ≤ 1 Hz delta activity [25]. A moderate change was attenuation of non-delta activity to about 50% of pre-clamp levels, and/or an obvious and persistent increase of delta activity at >1 Hz. Although gradations of changes in the rEEG occur, augmentation of delta activity reflects a less severe ischemic episode than does attenuation of all encephalographic activity. Others have defined significant changes as a $>50\%$ decrease in the amplitude of the 8–15 Hz bandwidth (fast alpha/slow beta) [26].

One of the more detailed alarm criteria for classifying electroencephalogically-determined ischemia during CEA

Table 4. Analog EEG criteria for determination of critical, cerebral, ischemic thresholds associated with CEA surgery, particularly during carotid artery cross-clamping

Reference	Severe or major analog EEG changes
Jenkins et al. [39]	Loss of 75–80% or more in amplitude is the same as a complete loss of all EEG activity
Blume and Sharbrough [25] (Mayo clinic)	>75% reduction in all activity, particularly the 8- to 15-Hz fast activity, and/or a two-fold or greater increase of ≤1 Hz delta activity
Kearse et al. [43]	Marked loss or complete absence of alpha and beta frequencies, a predominance of delta activity with little or no theta frequencies, and an increase or decrease in amplitude
Craft et al. [26]	>50% decrease in the amplitude of the 8–15 Hz bandwidth (fast alpha/slow beta)
Nuwer [6]	>50% loss of overall EEG amplitude or fast activity, or >50% increase in slow activity
Mizrahi et al. [3] (ACNS)	All EEG activity progressively diminished in amplitude and approaching isoelectricity

surgery was outlined by Kearse and colleagues [43]. These authors defined three distinct categories which included mild, moderate, and severe ischemic changes in the rEEG from an anesthetic-induced baseline established 5 min before carotid artery cross-clamping. Each category was defined by three components. Electroencephalographic changes were classified as mild if there was a minimal diminution of alpha (8–13 Hz) and beta (14–30 Hz) activities, a less than 50% increase in theta activity (4–7 Hz), and no detectable change in amplitude or increase in delta activity (0.5–3 Hz). Moderate ischemia was defined by easily detectable loss or absence of fast activity, a more than 50% increase in theta and/or delta activity, and a 30% or less increase or decrease in amplitude. Severe or major ischemia was characterized by a marked loss or complete absence of alpha and beta frequencies, a predominance of delta activity with little or no theta frequencies, and a greater than 30% increase or decrease in amplitude. In each severity category, the ischemic pattern could be focal or generalized. Others have classified a major change in the analog EEG using a 50% criterion: a >50% loss of overall amplitude or fast activity, or >50% increase in slow activity [6]. Using such criteria, the incidence of major changes in rEEG has been reported to range from 3 to 12.5% [25, 39]. These latter changes have been typically reported when regional rCBF decreased below 10–15 mL/100 gm/min [44, 45]. The critical rCBF threshold needed to maintain a relatively normal EEG is 18–20 mL/100 g/min (about 35–40% of normal [46]. However, these thresholds may vary with the type of maintenance anesthesia: the threshold is least under isoflurane (about 10 mL/100 g/min) and most under halothane (20 mL/100 g/min) [47].

Lastly, the ACNS's intraoperative EEG guidelines (2000) defined three degrees of EEG changes caused by ischemia: (1) the first degree–a decrease in background fast activity, most apparent when using anesthetic agents that generate such fast activity (the diminution is considered significant if it exceeds 50–60% of baseline), (2) the second degree–an increase in slow (delta–theta) which should be considered clinically significant if it exceeds 50% of baseline (a decrease in fast activity may be simultaneous), and (3) the third degree–all rEEG activity progressively diminishes in amplitude and approaches bioelectricity [3].

Controlled intraoperative hypertension is commonly used during the anesthetic and surgical management of CEA surgery in order to prevent cerebral ischemic insult (an incidence of 61%) [20]. Although target blood pressures (e.g., preoperative baseline mean arterial pressure (MAP), 10 or 20% above preoperative baseline MAP, or MAP = 90 or 100 mm Hg) have been commonly used, these strategies can be tailored to the individual patient using neuromonitoring [20]. Changes in rEEG and other neural measures (e.g., cerebral blood flow velocity of the MCA as measured by TCD and cerebral oximetry) have been utilized to determine the appropriate blood pressure requirement [20].

Multimodality neuromonitoring: other computer-processed modalities

Other forms of intraoperative neuromonitoring for CEA surgery, such as MN SSEPs, TCD of the MCA on the operative side, or cerebral oximetry are strongly recommended to be performed in conjunction with the rEEG and qEEG [11]. Multi-channel, rEEG is considered by most as the gold standard for intraoperative neuromonitoring during CEA surgery; however, several disadvantages should be recognized. Conventional rEEG can be technically laborious, difficult to interpret, requires experienced personnel, does not provide direct information about subcortical structures, and, as the only intraoperative monitoring modality, can have a lower sensitivity (50%) and specificity (92%) as compared to MN SSEPs in detecting postoperative neurological deficits [48]. In comparison, Lam et al. [48] reported sensitivity and specificity rates of 100 and 94%, respectively, for MN

SSEPs. For example, neuromonitoring using TCD of the MCA provides a beat-by-beat detection of cerebral blood flow velocity, which can be used for the detection of ischemia, and air or particulate embolism. Cerebral oximetry is a very inexpensive, noninvasive technique, although currently the critical ischemic thresholds are not firmly established. The reader is encouraged to review the ASNM's guidelines for the use of intraoperative SSEPs, TCD and cerebral oximetry.

Anesthetic effects on the rEEG during CEA surgery

Historically, interest in the patterns of the rEEG associated with anesthesia is as old as the discovery of the analog EEG, itself. However, there is still no universal adoption of a monitoring technique or criteria for its neural endpoint(s) by anesthesiologists and neurophysiologist. The normal practice of modern anesthesia involves an almost universal use of ''polypharmacy,'' or the use of a combination of anesthetic agents for premedication and ''balanced'' anesthesia producing hypnosis, analgesia, amnesia, and muscle relaxation [49]. Thus, the electroencephalographic effects often observed and assessed are not necessarily the effects on background rhythms typically associated with each drug alone, but often in combination. Selection of anesthetic agents for induction and maintenance of a steady-state are important for optimizing the detection of cerebral ischemia.

A brief coverage of anesthetic effects on the rEEG during CEA surgery is presented in this section. For a more extensive review of anesthetic effects on the rEEG and qEEG, see the following references [5, 50–53]. Knowledge of such influences is a prerequisite for the interpretation of ischemic changes. The effects of premedication and anesthetic induction are typically bilateral and symmetrical. Induction is usually with IV drugs such as thiopental, propofol, or etomidate, and then steady-state anesthesia is maintained by inhalational agents, most commonly halogenated agents (e.g., isoflurane) and nitrous oxide.

The reader is also encouraged to review Blume and Sharbrough's [25] characterization of typical rEEG patterns prevalent during sub-minimal alveolar concentrations (sub-MAC concentrations) of anesthetic agents, particularly during steady-state anesthesia. These include:

Widespread anteriorly maximum rhythm (WAR) - This pattern is characterized by a rhythmic lower beta or alpha (8–14 Hz) activity which appears as the dominant activity over the anterior hemispheric region with induction using inhalational agents such as halothane, enflurane, and isoflurane, and IV drugs such as thiopental. During lighter levels of steady state anesthesia the WAR pattern becomes widespread and is essentially generalized. This pattern does slow with increasing level of anesthetic agent. In addition, the EEG typically shows intermittent delta wave (usually 1 sec or less in duration), which is often sharply contoured and commonly biphasic, and best expressed as transients or in a brief train.

Frontal intermittent rhythmic delta activity (FIRDA) - This pattern is characterized by a high-amplitude, intermittent, rhythmic, delta activity which is usually maximal frontally. It is typically seen with a rapid induction with thiopental as a burst intermixed with the faster alpha/beta activity.

Anterior intermittent slow waves (AIS) - This pattern is characterized by an anteriorly, maximum, intermittent, slow-wave activity which is commonly diphasic, triangular in morphology, and may occur either singly or in brief trains lasting about 1 second.

Widespread persistent slow activity (WPS) - This polymorphic pattern is characterized by a widespread, persistent, slow-wave activity with low amplitude, expressed maximally over the temporal and posterior regions, and lasts about 1 second. This pattern is more prevalent when higher concentrations of isoflurane anesthesia are used, particularly in conjunction with 50–60% nitrous oxide.

At sub-MAC concentrations of the inhalational anesthetics or at lighter levels of steady-state anesthesia, the dominant rEEG activity is characteristized by the WAR pattern, as described above. This is a preferred, anesthetic-induced, rEEG pattern which would optimize the detection of cerebral ischemia. At supra-MAC concentrations of the inhalational anesthetics, unique rEEG patterns may develop. Since isoflurane is typically used, its patterns will be further reviewed. At sufficiently higher levels approaching about 1.0–1.5 MAC for this inhalational agent, the rEEG emerges into a burst-suppression pattern, or becomes isoelectric. Frequently, all activity is lost between 2.0–3.0 MAC. This is clearly a pattern of activity which is undesirable for evaluating ischemia, particularly during carotid cross-clamping. In addition, during inhalational anesthesia with nitrous oxide, a WPS activity with lower amplitude may become more prominent. Again, this is not preferred during neuromonitoring since it typically produces a pattern that is not optimal for the detection of ischemia.

Although most patients undergoing CEA surgery show diffuse anesthetic-related EEG changes, one-third may show focal abnormalities in their pre-clamp rEEG [46]. These abnormalities consist of unilateral attenuation of

WAR patterns and prominent polymorphic delta on the same side. In many cases, these abnormalities seen under anesthesia are present in the waking traces, and probably reflect a pre-existing focal area that is ischemic and dysfunctional, such as an infarction or an area of vascular insufficiency [54]. Furthermore, anesthesia may influence the detection of preoperative focal abnormalities in the rEEG by either activating or obscuring them.

Neuromonitoring during cardiac surgery utilizing cardiopulmonary bypass procedures

Background

In 2006, an estimated 7 million inpatient cardiovascular operations and procedures were performed in the US alone of which 448,000 involved coronary artery bypass graft (CABG) surgery [55]. Cardiac surgery utilizing cardiopulmonary bypass procedures (CPB) consists of a cascade of dynamic surgical and anesthetic events. The incidence rates of early postoperative neurological and/or neuropsychological complications have ranged from 0 to 100% with the most commonly reported ranging between 35 and 50% [56, 57]. In particular, impaired cognitive function can persist in up to 35% of patients for 12 months [56]. A recent postoperative study by Newman and colleagues [58] found that 42% of bypass patients still experienced cognitive decline 5 years later. Both the rEEG and qEEG have been used to ''provide a sensitive measure of synaptic activity within the cortical mantel'' (p. 148) [59]. Although, intraoperative neuromonitoring of the rEEG during CPB is nearly as old as extracorporeal circulation, brain monitoring has not yet become a routine tool in the repertoire of surgical monitoring for these procedures [10, 59–61]. However, is should be noted that in certain studies the use of intraoperative rEEG and qEEG has significantly lowered neurological deficits, shortened postoperative recovery, and reduced hospital costs [10, 62–64].

Anesthetic effects on the rEEG during cardiac surgery

The use of rapid-acting synthetic opioids (such as fentanyl and sufentanil) during cardiac surgery is a well-established practice for cardiac anesthesia. For rEEG, anesthetic induction begins with the appearance of diffuse theta and some delta which is maximal frontally. Within 1–2 min following the emergence of an irregular bifrontal delta, global and more synchronous, monomorphic, delta activity may prevail, depending upon the dose and the individual. Over the next 2–5 min the global rEEG pattern evolves and stabilizes into a more polymorphic or irregular, slow-wave activity, comprised mostly of delta waves [65, 66]. Although the level of anesthesia may be maintained in a range that permits a stable baseline for the rEEG, alterations due to ischemia are easily masked by high-dose anesthesia and thus may go undetected. One method for detecting ischemic changes during this type of anesthetic regime, which is typically even more complicated by induced-hypothermia during CPB, involves the use of relative brain power in the delta frequency bandwidth (see Section Alarm criteria for ischemic thresholds using qEEG during cardiac surgery). Lastly, it should be noted that many centers currently use anesthetic protocols combing inhalational and intravenous agents similar to those used for CEA surgery.

rEEG and qEEG changes associated with cardiac surgery

Like neuromonitoring for CEA surgery, changes in the rEEG and qEEG have been used for the detection of cerebral ischemia, objective administration of anesthesia, blood pressure control, and ''cerebral protection'' during cardiac surgery. Increasing neural activity (i. e., augmentation of higher frequency components or decreased lower frequency activity in the analog signal, and increased mean and median frequencies, and a higher SEF95 index) which is non-pathologic, probably signifies decreasing anesthetic effects [63]. Conversely in the presence of stable anesthesia, slowing of the rEEG with a progressive loss of amplitude and frequency content, burst-suppression, and even electrocortical silence (isoelectric waveforms) are indications of pathological synaptic depression. Although, the rEEG can be well-suited for the detection and correction of anesthetic imbalance or intraoperative seizure activity, electroencephalographic depression caused by pathologic factors such as ischemia or hypoxia may not be easily discriminated from non-pathologic influences such as hypothermia or deepening anesthesia (e.g., induced-hypothermia with onset of bypass or a bolus of high-dose narcotics, respectively) [63, 67].

As stated by Edmonds et al. [10], one major function of electroencephalographically-based interventions using rEGG and qEEG during cardiac surgery is to optimally match perfusion with metabolic demand during the critical periods of surgery. Possible conditions or times when this relationship is compromised are listed below.

1. Marked slowing of the rEEG associated with cannulation and onset of bypass related to the low-oxygen, priming volume of the bypass machine. The magnitude and duration of the rEEG changes are directly related to the priming volume and inversely related to the size of the patient. Thus, smaller patients are more likely to exhibit marked slowing of the rEEG at cannulation.

2. Release of the aortic cross-clamp with its attendant transient hypotension is often coupled with slowing of the rEEG that may persist until effective pulsatile perfusion is re-established.
3. Depression of the rEEG may also be expected at the completion of rapid rewarming. Because of the demand of a hypermetabolic state induced by 39°– 40° C, blood outpaces the delivery of the mechanical or recovery of the cardiac pump. This may be viewed as a 'cerebral anginal attack.'

Lastly, another important use for monitoring rEEG and qEEG during cardiac surgery is to provide objective maintenance and documentation of burst-suppression and isoelectric patterns during deep hypothermia and/or barbiturate protection prior to the initiation of circulatory arrest [68, 69].

Neuromonitoring during cerebral vascular surgery: clipping of intracranial aneurysms and arteriovenous malformations

Intraoperative neuromonitoring using rEEG and qEEG during the clipping of cerebral aneurysms has traditionally been used for two purposes. The first is the detection of cerebral ischemia during the dissection and clipping of an intracranial aneurysm, which is of paramount importance. Compromised cerebral perfusion can occur during either placement of retractors for surgical exposure and placement of the aneurysm clip, or both. Both the rEEG and qEEG are neuromonitoring modalities which can be used, although may not necessarily be the primary ones. In addition, other neuromodalities are also strongly recommended: (1) upper and lower extremity SSEPs for aneuryms of the anterior vessels of the Circle of Willis (e.g., internal carotid, middle and anterior cerebral arteries), and (2) microvascular Doppler for any cerebral aneurysm for evaluation of cerebral blood flow velocity of the arterial branches and its perforators, and to confirm the absence of pulsatile flow in the "dome" of the clipped aneurysm [70].

The second use of rEEG and qEEG monitoring during intracranial aneurysm surgery is for cerebral protection. Pharmacological protection of the brain is a relatively common practice during surgical manipulation and clipping of a cerebral aneurysm, although the efficacy of a barbiturate coma to produce cerebral protection is still debated. In general, barbiturates and other IV drugs (e.g., etomidate) are often titrated to produce a burst-suppression pattern or even isoelectric waveforms prior to aneurysm clipping in order to reduce the cerebral metabolic rate by about 50%. Most feel barbiturates are the preferred drug. The "typical" dose of barbiturates necessary to induce burst suppression varies greatly among individuals, ranging from as little as 2 mg/kg to as much as 25 mg/kg [71]. "With such a wide range of dosage necessary for the desired effect, is clear that the only rational way to administer barbiturates…is using EEG" (p. 566) [71]. Lastly, titration of these agents to a specific degree of burst suppression can be quantified by the burst suppression ratio (BSR) which is available on virtually all commercially-available neuromonitoring systems. One method used to calculate the BSR is the sum of the intervals of suppression (voltages <5 μV) that last at least 0.5 s divided by the epoch length [72].

The alarm criteria for detection of a cerebral ischemic event are listed in Sects. Alarm criteria for ischemic thresholds using rEEG and Alarm criteria for ischemia using qEEG During CEA surgery. Obviously, if the rEEG is pharmacologically depressed, detection of ischemia is compromised. Thus, other neuromonitoring modalities, such as SSEPs, motor evoked potentials or microvascular Doppler may be more efficacious during such anesthetic maneuvers.

INTRAOPERATIVE COMPUTER-PROCESSED OR QUANTITATIVE EEG (QEEG)

Background

Clearly, assessment of the frequency and amplitude of the rEEG is crucial for rapid and accurate interpretation; however, such assessment is quite difficult sometimes using the raw signal and naked eye alone. Over the past several decades, a number of computer-processed algorithms and display techniques have been developed to make easier the recording and interpretation of the rEEG. The primary advantages of qEEG include: (1) enhanced visual graphics for easier on-line interpretation, (2) the ability to determine pre-selected baselines in order to evaluate deviations during critical anesthetic and surgical manipulations, (3) the ability to quantify and statistically evaluate the rEEG, and (4) to develop a parameter(s) that would allow intraoperative monitoring of depth of anesthesia [60, 61]. Although qEEG has been used to assist in the analysis of the analog signal and to quantify intraoperative ischemia and depth of anesthesia, in the final analysis, the visual inspection of an artifact-free, contemporaneous, raw signal is still deemed by many as clinically the most critical and superior technique for interpretation. In 1987, the AEEGS supported the position that "the clinical application of quantitative EEG analysis is considered to be limited and adjunctive" (p. 87) [73]. Thus, a real-time view of the rEEG must always be available whenever qEEG is used.

Since the analog EEG is an alternating voltage which changes over time, some of the first methods for computer-processing or quantification involved a time domain analysis such as zero-crossing (aperiodic analysis) [72, 74, 75]. An alternative approach to statistical examination of the EEG is frequency domain analysis which is based on signal activity as a function of frequency (power spectral analysis based on Fast-Fourier transformation (FFT) [60, 61, 72, 76–78]. This it typically done using the Fourier transform which is most commonly implemented using FFT. Essentially, the Fourier transform decomposes the analog EEG (a complex waveform) into its component sine waves. The power spectrum is then calculated by squaring the amplitudes of the individual frequency components. Thus, the analog EEG signals which were recorded on the time axis are transformed and displayed on the frequency axis as the amount of power or energy in user-defined bandwidths, typically delta, theta, alpha, and beta.

There are a number of properties of the power spectrum that are important to understand. First, the power in any signal is related to the square of its amplitude of the signal so that doubling the amplitude of the signal quadruples the power. This means that small amplitude components in the EEG can be obscured by higher amplitude components. One means of reducing this problem is by plotting the logarithm of the power, a method that is occasionally used in the display of EEG power spectra. Second, the highest frequency in the power spectrum is related to the rate at which the analog EEG is sampled according to the Nyquist relation: $f_{max} = 0.5^{*} f_{sample}$ where f_{max} is the maximum frequency in the power spectrum and f_{sample} is the rate at which the raw EEG is sampled. Thus, if the raw EEG is sampled at 250 Hz, then the highest frequency in the power spectrum is 125 Hz. If the input signal contains frequencies greater than f_{max}, the power at these frequencies is falsely represented (or aliased) at a frequency in the range from 0 to f_{max}. Thus, it is important for the sample frequency to exceed twice the frequency of any significant frequency in the input signal. Third, the smallest difference in frequencies that can be resolved is $1/T$ where T is the size of the segment of EEG analyzed in seconds. Thus, if power spectral analysis is performed in 1 s epochs then the resolution is only 1 Hz. If, however, 10 s epochs are used the resolution is 0.1 Hz. Typically, an epoch length of 2–2.5 s is employed.

Several different display formats have been developed for computer-enhanced imaging of the power spectral analysis of the rEEG: (1) the compressed-spectral array (CSA; a pseudo-three-dimensional topographic plot) [76], (2) dot-density spectral array (DSA; a gray- or color-scaled two dimensional contour plot) [77] and color-scaled topographic brain mapping [78]. These computer-enhanced images can literally give the user an impression that individual anesthetic agents and surgical events may produce their own neural ''signature or fingerprint'' [51, 53, 60, 61]. In addition, derived measures such as spectral edge frequency (SEF; e.g., SEF95 is the frequency below which 95% of the total spectral power is contained), mean and median frequency, absolute and relative power frequency bandwidths, coherence, burst-suppression ratio, and asymmetry indices have also been used to simplify, and assist in the display and interpretation of the rEEG. Perhaps the most informative and widely used computer-processed display technique in routine clinical practice are the CSA and DSA formats with derived indices such as SEF, power frequency bandwidths of the traditionally-defined bandwidths, and median power and peak power frequencies.

Alarm criteria for ischemia using qEEG during CEA surgery

In general, ischemia is associated with a shift of the power spectrum to the lower frequency range, and concomitant loss of amplitude in the power spectrum for selected frequencies or across the entire frequency spectrum. As stated above, proper interpretation of any computer-processed display or derived measures of the rEEG should always include selected segments of concurrent analog or digitally-recorded EEG to verify the validity of the interpretation of the reduced and simplified computer-processed data [79]. Like anesthetic effects, the display techniques for power spectral analysis using either the CSA or DSA can afford the user a remarkable neural fingerprint of the effects of cerebral ischemia during CEA surgery [80–82]. One major advantage of these computer-processed transformations of the rEEG is that the alarm criteria for intervention during ischemia can be more easily quantified. For example, several quantitative criteria have been applied which have accurately predicted post-operative neurological outcome.

1. Rampil et al. [83] defined a significant ischemic period as a rapid (<1 min) decrease in SEF to $\leq 50\%$ of the prior baseline, which persisted for longer than 10 min.
2. Using power spectral analysis, Ivanovic et al. [80] defined three broad frequency bandwidths: low (delta and theta, 0.25–6.0 Hz, middle (alpha, 6.0–10.5 Hz), and high (sigma to beta, 10.5–16.0 Hz). Changes in the qEEG during carotid cross-clamping were assigned to one of three categories based on the magnitude of the changes in the power spectrum of each bandwidth: (1) mild or no power reduction in which the changes in the power spectrum did not exceed 50% for any frequency bandwidth, (2) marked power reduction

characterized by a >50% reduction in one or two frequency bandwidths, and (3) global or profound reduction that reflected at least a 50% reduction in the power of the qEEG in all three frequency bands. In their series, the percentage of patients falling into each category was 78%, 11%, and 11%, respectively.

3. Tempelhoff et al. [84] used the criterion of either a decrease in SEF ≥50% or a decrease in total spectral power >30%.

In contrast, however, others have criticized these computer-enhanced techniques for their unreliability in detecting mild ischemia, which was claimed to be more easily recognized with greater sensitivity and specificity when using conventional, analog, multi-channel EEG recordings [43].

Alarm criteria for ischemic thresholds using qEEG during cardiac surgery

The benefits of neural monitoring using qEEG for the detection of cerebral ischemia have been discussed by various authors. As a matter of historical interest, two studies will be reviewed that utilized commerically-available software packages which represented a pioneering effort for neuromonitoring using qEEG during cardiac surgeries involving CPB. In particular, Arom et al. [62] demonstrated that patients who were ''brain-monitored'' with interventional criteria implemented as indicated below, presented postoperatively with only 5% new, global, neurological deficits as compared to an incidence of 40% in patients ''brain-monitored'' without interventions based on neural events. A power drop index (PDI) was calculated and compared to baseline measures. The PDI is a numerical indicator of the severity and duration of the decrease from baseline power level for each channel. The greater the decrease in power and/or the longer it lasts, the higher the PDI. The PDI would accumulate when the power level dropped to less than 40% of baseline level for a given channel.

The criteria for intervention CPB were:

- Any drop in power to 25% of baseline activity during CBP
- Any asymmetry or lateralized drop in power during pump CBP

The methods of intervention were:

- Increase cerebral perfusion (Mean Arterial Pressure−Central Venous Pressure = Cerebral Perfusion Pressure; MAP−CVP = CPP) to 60–65 mmHg
- Increase CPB pump flow
- Increase MAP using vasopressor (in this case, neosynephrine)
- Readjust the venous cannula
- Increase blood CO_2
- Readjust the arterial cannula for a lateralized deficit

Another study also employed a proprietary software package to evaluate cerebral ischemia during cardiac surgery using CPB [63]. In particular, a drop in the relative low-frequency power (1.5–3.5 Hz) was chosen as a single, quantitative, electroencephalographic descriptor using Cerebrovascular Intraoperative Monitor (CIMON; Cadwell Laboratories, Inc., Kennewick, WA)). Relative delta power appeared to be insensitive to moderate changes in body temperature (deliberate-induced hypothermia) and level of opioid anesthesia, but was a statistically significant indicator of cortical dysfunction when using standard deviations in *z*-scores from an individualized reference or self-normative data. Despite the advantages of EEG monitoring in some studies, other studies have found no relation between rEEG changes and neurophysiologic outcome after cardiac surgery due to the wide variability in the rEEG seen during these procedures [85].

Although displays of the qEEG and derived indices can be very useful for the detection of cerebral ischemia as summarized above, great care and caution should be paid to its exclusive use. qEEG is subject to a variety of unpredictable influences of sampling error, environmental, and statistical artifact [59]. High-dose narcotics and hypothemic suppression of the activity of the rEEG can produce artifactually-induced increases in relative delta, theta, and beta activity. In addition, during ischemic suppression of the rEEG, the SEF may remain unchanged in instances where imperceptibly small amounts of high frequency artifact contaminate the signal.

qEEG as a ''depth of anesthesia'' indicator

Much effort has been devoted to the development and study of the rEEG and qEEG as a measure of depth of anesthesia or consciousness. Not only was Hans Berger the first to record the analog EEG in man (1924, [86]), but he was also the first to perform analog EEG recordings under anesthesia (chloroform) [87]. This led to the development of the first EEG-controlled dosage machines [88–90], and multiple classification schemes for defining the clinical stages of anesthesia based on the analog EEG [91–94]. Historically, the term, depth of anesthesia, has been considered quite nebulous. Furthermore, there is still no exact definition of what is meant by depth of anesthesia since its measurement is still considered an unsolved problem [95].

Traditionally, the anesthetic state has been quantified by a single end-point: the lack of somatic motor responses to surgical incision [75]. This single reference point was termed the Mean Alveolar Concentration (MAC), which is the concentration of inhaled anesthetic agent that provides surgical immobility in 50% of the patients. In general, anesthesia has been defined as a behavioral state that must have at least two components: (1) oblivion (amnesia or depressed level of consciousness) and (2) unresponsiveness (surgical somatic immobility or MAC testing, and hemodynamic changes) [75]. Although traditionally, depth of anesthesia has been measured using lack of acute hemodynamic changes, lack of somatic motor responses, and cerebral depression using rEEG and qEEG, anesthesia is now understood to be a result of ''heterogeneous actions at specific [multiple] sites within the central nervous system…'' (p. 650) [75].

At this writing, the incidence of awareness or recall is of public concern. In a huge, multi-center, randomized, double-blinded, prospective trial (n = 2463) sponsored by Aspect Medical Systems (Newton, MA), qEEG using bispectral analysis was shown to reduce awareness. [96]. However, a comparable study by Avidan and colleagues concluded that a BIS-guided protocol did not reduce the frequency of definite or possible awareness [97]. Such concern over depth of anesthesia or awareness was framed as a ''sentinel event alert'' in an initiative to mandate depth of anesthesia monitoring by the Joint Commission on October 6, 2004 under the presidency of Dr. Dennis O'Leary [98]. The current literature has indicated a range of $<$0.03–0.2% for the incidence of awareness [96, 99–101]. In particular, the American Society of Anesthesiology (ASA) reported the incidence of intraoperative awareness is 1–2 cases per 1000 surgeries under general anesthesia. Assuming that approximately 20 million anesthetics are administered in the US annually, Sebel et al. [102] calculated an expected incidence of awareness involving approximately 26,000 cases per year. In a recent article on neuromonitoring for determining depth of anesthesia, Rampil described the evolution of devices and algorithms developed for such monitoring as ''mov[ing] from wishful thinking to competitive commercialization with broad public interest'' (p. 649) [75].

According to the ASA's Closed Claims Project database in 1996, awareness/recall was responsible for an estimated 0.2–7% of the lawsuits against anesthesiologists [103, 104]. Of 3,533 closed claims, 69 (2%) were for awareness. Of those, 54 (1.5%) were for patient recall while under anesthesia and 15 (0.5%) were for paralysis while awake. In particular, awareness was estimated to occur at a higher incidence for certain surgeries: 11–43% of all trauma cases and 2.5–4% of obstetric cases. Cardiac surgery also typically yields a higher incidence of awareness. The rate of payment for awareness-related suits was 57% with an average payment of $18,000 because these ''injuries'' were judged to be minor or temporary. However, higher awards have occurred (as much as $600,000 due to complications).

At its annual meeting in 2005, the House of Delegates of the ASA approved their final report of the ASA's Task Force on Intraoperative Awareness, which had been appointed the preceding year. Their report titled, ''Practice Advisory for Intraoperative Awareness and Brain Function Monitoring,'' represents the most thorough document to date to assist anesthesiologists and hospitals in minimizing the risks of awareness under general anesthesia. The goals of this task force were to: (1) identify risk factors associated with intraoperative awareness, (2) provide decision tools to enable the clinician to reduce the incidence of awareness, (3) stimulate the pursuit and evaluation of strategies to prevent/reduce its incidence, and (4) provide guidance for the intraoperative use of ''brain function monitors'' as they relate to this phenomenon.

Furthermore, their report on brain function monitoring ''recognizes [these] devices as a possible tool for monitoring selected patients, but concludes that the decision to use this emerging technology should be made on a case-by-case basis by the individual practitioner …brain function monitors are an option to be used when the anesthesiologist deems it appropriate, just as he or she makes choices about specific drugs, dosages,…and other types of monitors depending on the individual patient.'' In conclusion, the ASA's position is consistent with their historical perspective that language encouraging the use of brain function monitoring (or any monitoring device) in the ASA standards and guidelines ''did not happen overnight,'' and will be ''strengthened gradually as these devices' usefulness, reported by anesthesiologists and researches, become more evident.''

Early attempts used power spectral analysis displayed as CSAs or DSAs, and derived measures such as SEF [51, 53, 76, 77, 105]. Some of these early studies suggested that changes in these neural measures correlated in a rather orderly and predictive fashion with concentration of the agent for a variety of drugs (e.g., halothane [106]; serum concentrations of thiopental, fentanyl and alfentanil [107, 108]). Others have tailored induction doses of narcotics to a predefined neural fingerprint in an attempt to ensure clinical efficacy and cost effectiveness [60, 61, 109]. However, these measures have not proven to be an adequate indicator of depth of anesthesia [5, 110]. The most current attempts using computer-processed algorithms have been used to evaluate ''level of consciousness'' which includes bispectral analysis (the bispectral index, BIS®), patient state index (PS1™), and spectral entropy (SE) [5, 72, 75, 110].

The BIS® is a complex measure of EEG activity that employs a number of different measures of the EEG including the burst-suppression ratio, some elements of the power spectrum, as well as a new technology known as high-order spectral analysis. This high-order spectral analysis analyzes the phase coupling (or correlations) between pairs of frequencies found in the raw waveforms as a measure of the hypnotic component of the anesthetic state [5, 72, 111–113]. The BIS® is a multivariate parameter since it measures changes in the interfrequency coupling or harmonic relations at different EEG frequencies. The BIS index ranges from 0 to 100, with 100 indicating that the patient is alert and awake, and zero indicating the absence of brain activity. A BIS® level of 60 or lower correlates with a 95% chance that a patient is unconscious. The 40–60 range is recommended for surgical anesthesia. In particular, by using BIS® monitoring, the control of the maintenance level and emergence from anesthesia can be improved, actually resulting in indirect-cost savings [101, 112, 114].

It should be noted, however, that exactly what all these phase relationships means physiologically is still uncertain [72]. In addition, one should be aware that the algorithm for the BIS® can be compromised in detecting and mitigating artifacts and inconsistencies. For example, excessive facial muscle tone will increase the BIS® index, whereas a subdural hematoma may reduce the scalp voltage and thus decrease the computed index [75]. Another possible shortcoming in ''interpretation'' by qEEG is related to a poorly understood phenomenon know as paradoxical arousal. Typically during noxious stimulation, the rEEG is characterized by acceleration and desynchronization. However, in some instances, the rEEG will become highly synchronized, producing high-amplitude delta waves. Unfortunately, this pattern may not be easily distinguished from deep anesthesia or cerebral ischemia [75].

The PSI™ is also a multivariate qEEG index derived to measure the continuum of sedation and hypnosis [5, 110]. Like BIS®, Fourier spectral analysis is used to compute the power in each of the standard frequency bandwidths of the rEEG. The PSI™ is a complex computation reflecting several dimensions of brain activity including changes in power within the various EEG bandwidths, changes in symmetry and synchronization between critical brain regions, and inhibition of regions of the frontal cortex. The 0–100 scale for the PSI represents the probability of a patient responding to voice commands. An index of 25–50 is recommended for surgical anesthesia.

Lastly, the SE algorithm transforms the time-domain signal of rEEG into discrete frequency-domain epochs [110]. These calculations are used to describe the ''EEG complexity.'' For example, a perfect sine wave has very low entropy, while white noise has maximal entropy. Most hypnotic anesthetics decrease the rEEG complexity in a dose-related fashion, thus producing a small index value.

Entropy is based on processing the rEEG and facial electromyographic signals by using the Entropy Algorithm originally developed by Datex–Ohmeda (Helsinki, Finland) [5, 110]. Further development and marketing by General Electric has made commercially available the M-Entropy module. The Entropy index is based on the concept that anesthetic-induced unconsciousness and amnesia are cortical functions reflected by the EEG and subcortical function is reflected by facial electromyography (FEMG). The M-entropy module uses a single sensor to provide two separate readings: (1) one for the spectral entropy of the ''pure'' EEG signal (state entropy), and (2) the other for the spectral entropy of the combined EEG-FEMG waveforms (response entropy).

Thus, SE is postulated to be an index of the hypnotic state of the anesthetic agent, while RE would indicate insufficient analgesia when nociceptive stimulation increases frontal muscle activity. Entropy values of 40–60 reflect adequate anesthesia.

Currently, these quantitative measures are regarded as promising. Of the three, BIS® has received the most commercialization. Based upon data provided by the manufacturer, Orser [115] reported that 23 million patients have been monitored using the BIS worldwide, and in the US, 60% of all ORs use BIS technology. In conclusion, the generalized use of computer-processed EEG for monitoring depth of anesthesia remains an important clinical goal ''based on ample peer-reviewed data'' (p. 1190) [115].

DEFINITIONS

Based upon scientific studies, case studies and the expert opinion of those in the intraoperative field, these techniques are given evidence ratings and a strength-of-practice rating. The definitions and layout of this section are taken from Leppanen [116].

Quality of evidence ratings

Class I. Evidence provided by one or more well-designed, prospective, blinded, controlled studies.

Class II. Evidence provided by one or more well-designed, clinical studies such as case control, cohort studies, etc.

Class III. Evidence provided by expert opinion, non-randomized historical controls or case reports of one or more.

Strength-of-recommendation ratings

Type A. Strong positive recommendation, based on Class I evidence, or overwhelming Class II evidence.
Type B. Positive recommendation, based on Class II evidence.
Type C. Positive recommendation, based on strong consensus Class III evidence.
Type D. Negative recommendation, based on inconclusive or conflicting Class II evidence.
Type E. Negative recommendation, based on evidence of ineffectiveness or lack of efficacy.
Type U. No recommendation, based on divided expert opinion or insufficient data.

Standard

Standards are generally-accepted principles for patient management that reflect a high degree of clinical certainty.

Guidelines

Guidelines are recommendations for patient management that may identify a particular strategy or range of management strategies that reflect moderate clinical certainty.

Practice options or advisories

Practice options or advisories are other strategies for patient management for which there is some favorable evidence, but for which the community still considers this an option to be decided upon by individual practitioners.

Practice parameters

Practice parameters are results in the form of one or more specific recommendations from a scientifically-based analysis of a specific clinical problem.

SUMMARY

1. rEEG monitoring during CEA surgery using selective shunting is a standard (Class II and III evidence, strong Type A recommendation).
2. rEEG monitoring during CEA surgery using routine shunting is a practice option (Class II and III evidence, Type B recommendation).
3. qEEG monitoring is a practice option for level of consciousness (Class II and III evidence, Type B recommendation).
4. rEEG monitoring for cerebral aneurysms is a practice option (Class II and III evidence, Type C recommendation)
5. qEEG monitoring CEA surgery using routine shunting is a practice option (Class III evidence, Type D recommendation).
6. qEEG monitoring CEA surgery using selective shunting is a practice option (Class III evidence, Type D recommendation).
7. rEEG monitoring is a practice option for depth of anesthesia is (Class II and III evidence, Type D recommendation).
8. qEEG monitoring is a practice option for depth of anesthesia is a practice option (Class II and III evidence, Type D recommendation).
9. qEEG monitoring for cerebral aneurysms is a practice option (Class III evidence, Type E recommendation)
10. rEEG monitoring during cardiac surgery using cardiopulmonary bypass is a practice option (Class II and III evidence, Type U recommendation).
11. qEEG monitoring during cardiac surgery using cardiopulmonary bypass is a practice option (Class II and III evidence, Type U recommendation).
12. Different types of electrodes may be used for EEG recording, but the standard metal, disc (cup), surface electrodes are preferred to subdermal needle electrodes when this is practical (Class III evidence, Type C recommendation).

REFERENCES

1. American Electroencephalographic Society. Guidelines in electroencephalography evoked potentials and polysomnography. J Clin Neurophysiol 1994; 11: 1–147.
2. Cummings M, Ahn-Ewing J, Brouwer M, et al. Guidelines on intraoperative electroencephalography for technologists. Am J END Technol 1998; 38: 204–225.
3. Mizrahi EM, Chatrain G-E, Byrum W, et al. (2000) American clinical guidelines on intraoperative electroencephalography. American Clinical Neurophysiology Society Council 1–21.
4. American Clinical Neurophysiology Society. Guidelines in electroencephalography and evoked potentials. J Clin Neurophysiol 2006; 23: 85–179.
5. Freye E, Levy JV. Cerebral monitoring in the operating room and the intensive care unit: an introductory for the clinician and a guide for the novice wanting to open a window to the brain. Part I: the electroencephalogram. J Clin Monit Comput 2005; 19: 1–76.
6. Nuwer MR. Intraoperative electroencephalography. J Clin Neurophysiol 1993; 10: 437–444.

7. Seaba P. The importance of measuring electrode impedance, Oxford Observer, Spring, Oxford Medical Systems: Clearwater, FL, 1985.
8. Stecker MM, Patterson T. Electrode impedance in neurophysiologic recordings: 1. Theory and intrinsic contributions to noise. Am J END Technol 1998; 38: 174–198.
9. Stecker MM, Patterson T. Electrode impedance in neurophysiologic recordings: 2. Role in electromagnetic interference. Am J END Technol 1999; 39: 34–51.
10. Edmonds HL Jr, Rodriguez RA, Audenaert SM, et al. The role of neuromonitoring in cardiovascular surgery. J Cardiothorac Vasc Anesth 1996; 10: 15–23.
11. Isley MR, Cohen MJ, Wadsworth JS, et al. Multimodality neuromonitoring for carotid endarterectomy surgery: determination of critical cerebral ischemic thresholds. Am J END Technol 1998; 38: 65–122.
12. Altman CL. Infection control and the electroneurodiagnostic department: 1994 guidelines. Am J EEG Technol 1995; 35: 3–36.
13. Altman CL. Infection control: 2000 review and update for electroneurodiagnostic technologists. Am J EEG Tech 2000; 40: 73–97.
14. Isley MR, Pearlman RC, Credentialing and competency policy statement for intraoperative neuromonitoring staff: American society of neurophysiological monitoring position statement. Syngery July/August. 2006; 34: 38–41.
15. American Board of Registration of Electroencephalographic and Evoked Potential Technologists, Inc. Code of ethics and standards of practice. 1996: 1–2.
16. Luna G, Adye B. Cost-effective carotid endarterectomy. Am J Surg 1995; 169: 516–518.
17. Executive Committee for the Asymptomatic Carotid Atherosclerois Study. Endarterectomy for asymptomatic carotid artery stenosis. JAMA 1995; 273: 1421–1428.
18. Cowan JA, Dimick JB, Thompson BG, et al. Surgeon volume as an indicator of outcomes after carotid endarterectomy: an effect independent of speciality practice and hospital volume. J Am Coll Surg 2002; 195: 814–821.
19. Jansen C, Vriens EM, Eikelboom BC, et al. Carotid endarterectomy with transcranial doppler and electroencephalographic monitoring: a prospective study in 130 operations. Stroke 1993; 24: 665–669.
20. Cheng MA, Theard MA, Templehoff R. Anesthesia for carotid endarterectomy: a survey. J Neurosurg Anesth 1997; 9: 211–216.
21. Fode NC, Sundt TS, Robertson JT, et al. Multicenter retrospective review of results and complications of carotid endarterectomy in 1981. Stroke 1986; 17: 370–376.
22. Crosby G. CNS dysfunction in the perioperative period: causes and solutions. Course review lecture. Am Soc Anesthesiol 1990; 42: 1–7.
23. Beebe HG, Clagett GP, DeWeese JA, et al. Assessing risk associated with carotid endarterectomy. A statement for health professionals by ad hoc committee on carotid surgery standards of the stroke council american heart association. Circulation 1989; 79: 472–473.
24. American Academy of Neurology. Assessment. Intraoperative neurophysiology. report of the therapeutics and technology assessement, Executive Office: Minneapolis,MN, 1990.
25. Blume WT, Sharbrough FW. EEG monitoring during carotid endarterectomy and open heart surgery. In: Niedermeyer, E, da Silva, FL, eds, Electroencephalography: basic principles, clinical applications, and related fields. Urban and Schwarzenberg, Baltimore, 1993: 747–763.
26. Craft RM, Losasso TJ, Perkins WJ, et al. EEG monitoring for cerebral ischemia during carotid endarterectomy (CEA): how much is enough?. Anesthesiology 1994; 81: A213.
27. Rampil IJ. Electrophysiologic monitoring. Probl Anesth 2000; 12: 401–409.
28. Edmonds HL Jr, Sehic A, Gruenthal M. Comparison of 2-, 4- and 16-channel EEG for.detection of cerebral ischemia. Anesthesiology 2000; 97(3A): A-305.
29. Sundt TM Jr, Ebersold MJ, Sharbrough FW, et al. The risk-benefit ratio of intraoperative shunting during carotid endarterectomy: relevancy of operative and post-operative results and complications. Ann Surg 1986; 203: 196–204.
30. Ahn S, Concepcion B. Intraoperative monitoring during carotid endarterectomy. Semin Vasc Surg 1995; 8: 29–37.
31. McGrail KM. Intraoperative use of electroencephalography as an assessment of cerebral blood flow. Neurosurg Clin N Am 1996; 7: 685–692.
32. Gewertz BL, McCaffrey M. Recognition of cerebral ischemia during carotid artery reconstruction. In: Ernst, CB, Stanley, JC, eds, Current therapy in vascular surgery. 2nd edn. B. C. Decker Inc, Philadelphia, 1991: 76–81.
33. LoGerfo FW, Jepsen SJ. Role of carotid shunting during carotid endarterectomy. In: Ernst, CB, Stanley, JC, eds, Current therapy in vascular surgery. 2nd edn. B. C. Decker Inc, Philadelphia, 1991: 81–84.
34. Malone JM, Lalka SG. The placement of a carotid artery shunt: argument for its routine use. In: Moore, WS, ed., Surgery for cerebrovascular disease. 1st edn. Churchill Livingstone, New York, NY, 1987.
35. Bloom MJ, Schwartz DM, Berkowitz HD, Pratt RE. DSA processing of eeg is an effective monitor in cea. J Neurosurg Anesth 1990; 2: S13 .
36. Plestis KA, Loubser P, Mizrahi EM, et al. Continuous electroencephalographic monitoring and selective shunting reduces neurologic morbidity rates in carotid endarterectomy. J Vasc Surg 1997; 25: 620–628.
37. Cho I, Smullen SN, Streletz LJ, Fariello RG. The value of intraoperative eeg monitoring during carotid endarterectomy. Ann Neurol 1986; 20: 508–512.
38. Javid H, Julian OC, Dye WS, et al. Seventeen-year experience with routine shunting in carotid artery surgery. World J Surg 1979; 3(2): 167–177.
39. Jenkins GM, Chiappa KH, Young RR. Practical aspects of EEG monitoring during carotid endarterectomies. Am J EEG Technol 1983; 23: 191–203.
40. Sharbrough FW. EEG monitoring: II. Intraoperative recording. American EEG Society Course, American EEG Society, 1983.
41. Chiappa KH, Burke SR, Young RR. Results of electroencephalographic monitoring during 367 carotid endarterectomies: use of a dedicated minicomputer. Stroke 1979; 10: 381–388.
42. Faught E. Current role of electroencephalography in cerebral ischemia. Stroke 1993; 24: 609–613.
43. Kearse LA, Martin D, McPeck K, Lopez-Bresnahan M. Computer derived density spectral array in detection of mild analog electroencephalographic ischemic pattern changes during carotid endarterectomy. J Neurosurg 1993; 78: 884–890.

44. Sharbrough FW, Messick JM, Sundt TM. Correlation of continuous electroencephalograms with cerebral blood flow measurements during carotid endarterectomy. Stroke 1973; 4: 674–683.
45. Astrup J, Siesjo BK, Symon L. Thresholds in cerebral ischemia-the ischemia penumbra. Stroke 1981; 12: 723–725.
46. Sundt TM Jr, Sharbrough FW, Piepgras DG, et al. Correlation of cerebral blood flow and electroencephalographic changes during carotid endarterectomy with results of surgery and hemodynamics of cerebral ischemia. Mayo Clin Proc 1981; 56: 533–541.
47. Michenfelder JD, Sundt TM Jr, Fode NC, Sharbrough FW. Isoflurane when compared to enflurane and halothane decreases the frequency of cerebral ischemia during carotid endarterectomy. Anesthesiology 1987; 67: 336–340.
48. Lam AM, Manninen PH, Ferguson GG, Nantau W. Monitoring electrophysiologic function during carotid endarterectomy: a comparison of somatosensory evoked potentials and conventional electroencephalogram. Anesthesiology 1991; 75: 15–21.
49. Mahla ME. Anesthetic effects on the electroencephalogram. ASNM Monit: Am Soc Neurophysiol Monit Newsl 1992; 3: 2–7.
50. Brechner VL, Walter RD, Dillon JB. Practical electroencephalography for the anesthesiologist, Thomas: Springfield, IL, 1962.
51. Pichlmayr I, Lips U, Künkel H. The electroencephalogram in anesthesia: fundamentals, practical applications, examples, Springer-Verlag: New York NY, 1984.
52. Donegan JH. The electroencephalogram. In: Blitt, CD, ed., Monitoring anesthesia and critical care medicine. Churchill Livingstone, New York NY, 1985: 323–343.
53. Pichlmayr I. EEG atlas for anesthesiologists, Springer-Verlag: New York NY, 1987.
54. Markand ON. Continuous assessment of cerebral function with EEG and somatosensory evoked potential techniques during extracranial vascular reconstruction. In: Loftus, CM, Traynelis, VC, eds, Intraoperative monitoring techniques in neurosurgery. McGraw-Hill Inc, New York, NY, 1994: 19–31.
55. Heart Disease and Stroke Statistics. Update At-A-Glance. Statistical fact sheets: 13 medical procedures. American Heart Association, 2009.
56. Hammeke TA, Hastings JE. Neuropscyhologic alterations after cardiac surgery. J Thorac Cardiovasc Surg 1988; 96: 326–331.
57. Reves JG, Newman MF. The brain and cardiac surgery. Anesth Analg IARS Review Course Lectures 1996.
58. Newman MF, Kirchner JL, Phillips-Bute B, et al. Longitudinal assessment of neurocognitive function after coronary-artery bypass surgery. N Engl J Med 2001; 6: 395–402.
59. Edmonds HL Jr, Pollock SB Jr, et al. Neuromonitoring for cardiac and vascular surgery. In: Newman, SP, Harrison, MJG, eds, The brain and cardiac surgery. Harwood Publishers, London, 2000: 145–152.
60. Isley MR, Kafer ER, Bloom MJ. Anesthesia monitoring during surgery: intraoperative cerebral monitoring during cardiac surgery using computer-processed eeg. Med Electron 1990; 122: 82–94.
61. Isley MR. Intraoperative brain monitoring using analog and computer-processed eeg. biophysical measurements: electromyography/electroencephalography, SpaceLabs Medical: Seattle WA, 1993, pp. 158–196.
62. Arom KV, Cohen DE, Strobl FT. Effect of intraoperative intervention on neurological outcome based on electroencephalographic monitoring during cardiopulmonary bypass. Ann Thorac Surg 1989; 48: 476–483.
63. Edmonds HL Jr, Griffiths LK, van der Laken J, et al. Quantitative electroencephalogragphic monitoring during myocardial revascularization predicts postoperative disorientation and improves outcome. J Thorac Cardiovasc Surg 1992; 3: 555–563.
64. Edmonds HL Jr, Toney KA, Thomas MH, Pollock SB Jr. Neuromonitoring reduces cardiac surgery cost. Anesthesiology 1997; 87(3A): A-42–A-46.
65. Sebel PS, Bovill JG, Waquier A, Rog P. The effects of high-dose fentanyl on the electroencephalogram. Anesthesiology 1981; 55: 203–211.
66. Waquier A, Bovill JG, Sebel PS. Electroencephalographic effects on fentanyl-, sufentanil-, and alfentanil anesthesia in man. Neuropsychobiology 1984; 11: 203–206.
67. Stecker MM, Cheung AT, Pochettino A, et al. Deep hypothermic circulatory arrest: I. Effects of cooling on the electroencephalogram and evoked potentials. Ann Thorac Surg 2001; 71: 14–21.
68. Nussmeir NA, Arlund C, Slogoff S. Neuropsychiatric complications after cardiopulmonary bypass: cerebral protection by a barbiturate. Anesthesiology 1986; 64: 165–170.
69. Todd MM. A comfortable hypothesis reevaluated: cerebral metabolic depression and brain protection during ischemia (Editorial). Anesthesiology 1992; 76: 161–164.
70. Bailes JE, Lokesh ST, Fukushima T, et al. Intraoperative microvascular Doppler sonography in aneurysm surgery. Neurosurgery 1997; 40: 965–972.
71. Bloom MJ. EEG monitoring: intraoperative application. Anesth Clin N Am 1997; 15: 551–571.
72. Rampil IJ. A primer for eeg signal processing in anesthesia. Anesthesiology 1998; 89: 980–1002.
73. American Electroencephalographic Society. Statement on the clinical use of quantitative eeg. J Clin Neurophysiol 1987; 4: 87.
74. Levy WJ, Shapiro HM, Maruchak G, et al. Automated eeg processing for intraoperative monitoring: a comparison of techniques. Anesthesiology 1980; 53: 223–236.
75. Rampil IR. Monitoring depth of anesthesia. Curr Opin Anaesthesiol 2001; 14: 649–653.
76. Bickford RG, Fleming NI, Billinger TW. Compression of EEG data. Transact Am Neurological Assoc 1971; 98: 118–122.
77. Fleming RA, Smith NT. An inexpensive device for analyzing and monitoring the electroencephalogram. Anesthesiology 1979; 50: 456–460.
78. Duffy FN. Topographic mapping of brain electrical activity, Butterworth Publishers: Boston MA, 1986.
79. Fisher RS, Raudzens P, Nunemacher M. Efficacy of intraoperative neurophysiological monitoring. J Clin Neurophysiol 1995; 12: 97–109.
80. Ivanovic LV, Rosenberg RS, Towle VL, et al. Spectral analysis of eeg during carotid endarterectomy. Ann Vasc Surg 1986; 1: 112–117.
81. Archibald JE. Changes in eeg/csa seen during carotid endarterectomy intraoperative monitoring: EEG. American Society of Electroneurodiagnostic Technologist, Inc 1996; 2: 20–42.
82. Beachman SG, Frye D. EEG monitoring during carotid endarterectomy surgery: a tutorial. Intraoperative monitoring:

EEG. American Society of Electroneurodiagnostic Technologist, Inc 1996; 2: 43–63.
83. Rampil IJ, Holzer JA, Quest DO, et al. Prognostic value of computerized eeg analysis during carotid endarterectomy. Anesth Analg 1983; 62: 186–192.
84. Tempelhoff R, Modica PA, Grubb J, et al. Selective shunting during carotid endarterectomy based on two-channel computerized electroencephalographic/compressed spectral array analysis. Neurosurgery 1989; 24: 339.
85. Bashein G, Nessly ML, Bledsoe SW, et al. Electroencephalography during surgery with cardiopulmonary bypass and hypothermia. Anesthesiology 1992; 76: 878–891.
86. Berger H. Über das elektroenkephalogramm des menschen. Arch Paychiatr Nervenkr 1929; 87: 527–570.
87. Berger H. Über das elektroenkephalogramm des menschen: VI. Mitteilung. Arch Psychiatr Nervenkr 1933; 100: 301–321.
88. Bickford RG. Neurophysiology applications of automatic anesthesia-regulator controlled by brain potentials. J Physiol 1949; 159: 562–563.
89. Bickford RG. Automatic electroencephalographic control of general anesthesia. Electroencephalogr Clin Neurophysiol 1950; 2: 93–96.
90. Bickford RG. Use of frequency discrimination in the automatic eeg-control of anesthesia. Electroencephalogr Clin Neurophysiol 1951; 3: 81–85.
91. Gibbs FA, Gibbs EL. Atlas of electroencephalography1–3Addison- Wessly: MA, 1951.
92. Schneider J, Thomalske G. Betrachtungen über den narkosemechanismus unter besonderer berücksichtigung des hirnstammes. Zentralbl Neurochic 1956; 16: 185–202.
93. Kubicki S. Elektroenzephalographische aspekte der narkose. Berl Med 1968; 19: 4–12.
94. Kugler J, Elektroenzephalographie in klinik and praxis, eine einführung, 3rd Edition, Thieme Stuttgart, 1981.
95. Schmidt GN, Bischoff P, Standl T, et al. Narocrend^{R} and bispectral index^{R} monitors are superior to classic electroencephalographic parameters for the assessment of anesthetic states during propofol-remifentanil anesthesia. Anesthesia 2003; 99: 1072–1077.
96. Myles PS, Leslie K, McNeil J, et al. Bispectral index monitoring to prevent awareness during anaesthesai: B-Aware randomised controlled trial. Lancet 2004; 363: 1757–1763.
97. Avidan MS, Zhang L, Burnside BA, et al. Anesthesia awareness and the bispectral index. N Engl J Med 2008; 358: 1097–1108.
98. The Joint Commission. Sentinel Event Alert: Preventing, and managing the impact of anesthesia, Issue 32, 2004.
99. Liu WH, Thorp TA, Graham SG, Aitkenhead AR. Incidence of awareness with recall during general anesthesia. Anaesthesia 1991; 46: 435–437.
100. Jones JG. Perception and memory during general anaesthesia. Br J Anaesth 1994; 73: 31–37.
101. Ranta S, Jussila J, Hynynen M. Recall awareness during cardiac anaesthesia: influence.of feedback information to the anaesthesiologist. Acta Anaesthesiol Scand 1996; 40: 554–560.
102. Sebel PS, Bowdle TA, Ghoneim MM, et al. The incidence of awareness during anesthesia: a multicenter united states study. Anesth Analg 2004; 99: 833–839.
103. Domino KB. Closed malpractice claims for awareness during anesthesia. ASA Newsl 1996; 60(6): 45.
104. Macready N (Editor). Handling the difficult dilemma of awareness under anesthesia. Anesthesia Malpractice Prevention. 1997;2:89–96.
105. Rampil IJ, Sasse FJ, Smith NT, et al. Spectral edge frequency: a new correlate of anesthetic depth. Anesthesiology 1980; 53: S4.
106. Rampil IJ, Smith NT. Comparison of EEG indices during halothane anesthesia. J Clin Monit 1985; 1: 89–90.
107. Scott JC, Poganis KV, Stanski DR. EEG quantification narcotic effect: the comparative pharmacodynamics of fentanyl and alfentanil. Anesthesiology 1985; 62: 234–241.
108. Scott JC, Stanski DR. Decreased fentanyl and alfentanil dose requirements with age. A simultaneous pharmacokinetic and pharmacodynamic evaluation. J Pharmacol Exp Ther 1987; 240: 159–166.
109. Bowdle TA, Ward RJ. Induction of anesthesia with small doses of sufentanil or fentanyl: dose versus eeg response, speed of onset, and thiopental requirement. Anesthesiology 1989; 707: 26–30.
110. Gugino LD, Aglio LS, Yli-Hankala A. Monitoring the electroencephalogram during bypass procedures. Semin Cardiothorac Vasc Anesth 2004; 8: 61–83.
111. Sigl JC, Chamoun NG. An introduction to bispectral analysis for the electro- encephalogram. J Clin Monit 1994; 10: 392–404.
112. Gan TJ, Glass PS, Windsor A, et al. Bipsectral index monitoring allows faster emergence and improved recovery from propofol, alfentanil, and nitrous oxide anesthesia. Anesthesiology 1997; 87: 808–815.
113. Rosow C, Manberg PJ. Bispectral index monitoring. Anesth Clin North Am Annual Anesth Pharm 1998; 2: 89–107.
114. Johansen JW, Sigl JC. Bispectral index (bis) monitoring: cost analysis and anesthetic outcome. Anesthesiology 1997; 87: 3A.
115. Orser BA. Depth-of-anesthesia monitor and the frequency of intraoperative awareness. N Engl J Med 2008; 358: 1189–1191.
116. Leppanen RE. Intaoperative monitoring of segmental spinal nerve root function with free-run and electrically-triggered electromyography and spinal cord function with reflexes and F-responses. J Clin Monit Comput 2005; 19: 437–461.