Intraoperative EEG monitoring

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Intraoperative electroencephalographic (EEG) monitoring has become technically feasible in everyday clinical practice. Anesthesiologists should become familiar with the utility and indications for intraoperative EEG monitoring. An understanding of the EEG and the various descriptors of the EEG, including spectral edge frequency, density spectral array, and other parameters, is readily obtained. Clinicians can adapt these EEG parameters to their decision making process to guide therapeutic interventions or follow the course of pharmacologic manipulations. This article presents a discussion of the clinical application of routine intraoperative EEG monitoring with clinical examples and comparison of two commercially available monitors.

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Routine intraoperative electroencephalographic (EEG) monitoring of the effects of anesthetics on the brain has been available to anesthesiologists for more than 20 years.1-3 Various monitors have been developed commercially to provide continuous and real time EEG monitoring data in the operating room (OR).4-6 These monitors have met with variable success and acceptance by the anesthesia community. Early monitors had problems with data acquisition and placement of electrodes and electrode arrays, cables, and cumbersome space-occupying cabinetry, data displays, and ease of data interpretation.7 As each generation of new monitors and devices was introduced, anesthesiologists were presented with new methodologies and information to incorporate (or not) into their anesthetic management decision making processes.8 Often, the introduction of new methodologies was met not only with healthy skepticism,9 but with intransigent resistance to the need for yet another monitor in the OR (“I’ve done fine without this monitor for years, why do I need it now?”).

However, much of the reluctance to incorporate new brain monitoring technologies in the OR was based on more substantial concerns: familiarity with the neurophysiology of the EEG and an appreciation of the highly variable effects of pharmacologic anesthetic agents on the brain.10 Simplistic descriptors of anesthetic effects on the EEG (eg, frequency, amplitude, spectral edge) provided only limited information correlated to depth of anesthesia and failed to differentiate pharmacologic effects from physical or physiological.11,12 (Are the observed EEG changes due to a drug effect, surgical compression, temperature, oxygenation, perfusion, or underlying disease state pathophysiology?).

Significant concerns about the reproducibility and validity of analytic parameters used to describe the EEG and proprietary algorithms employed by these commercial monitors13,14 limited early acceptance of descriptive EEG analysis as applied to the intraoperative clinical setting. These topics have been discussed elegantly by Rampil15 and others.16 From these early experiences and concerns, new questions and avenues for investigation in anesthetic EEG neuropsychopharmacology continue to provide ample material for debate.17

Whether sophisticated EEG analysis paradigms will eventually satisfy the above concerns remains to be determined. Despite these limitations, routine use of EEG-based
intraoperative brain monitors has become commonplace as commercial products have facilitated data acquisition. Although not yet a “standard of practice,” the intraoperative monitoring of brain function as evidenced by the EEG has been encouraged by regulatory bodies, professional organizations, and eventually will be established by legal precedent. The dilemma for the clinician is not whether to use an EEG monitor, but when, and then what to make of this information and how to incorporate it into the decision making process. Intraoperative EEG monitors provide a window into the effects of anesthetic agents on the brain and an adjunct information source to aid in anesthetic case management. This paper deals with the practical application of intraoperative EEG monitoring data as obtained from two commonly available (commercial) machines: the Bispectral Index, BIS XP monitor (Aspect Medical, Newton, MA) and the Patient State Array, PSA 4000 monitor (Physiometrix, N. Billerica, MA), and a “head to head” comparison with clinical examples.

The EEG

The brain or central nervous system (CNS), produces electrical signals at a variety of frequencies, ranging from DC (direct current) potential shifts to AC (alternating current) frequencies. Early electroencephalographic researchers were limited in their ability to record EEG signals by AC current artifacts and the inability to filter out 60 Hz noise precisely. Thus, the EEG was recorded by using simple “band pass” filters that allowed relatively clean signal detection only below 30 Hz, while “ignoring” signals above that frequency. The EEG is classically composed of four frequency bands: delta (0-3 Hz), theta (4-7 Hz), alpha (8-12 Hz), and beta (13-24 Hz). One can easily remember these bands with a simple pneumonic: “Diet TAB” (and remembering only delta and alpha frequencies, allows one to fit the other frequency bands to the pneumonic). Although the EEG is classically described as occurring between 0 and 30 Hz, recent work (and precise “notch” digital filtering) has defined a gamma frequency greater than 24 Hz extending to 50-60 Hz. However, since most of the literature and all current monitors deal with EEG between 0 and 30 Hz, gamma frequency EEG, as well as DC (“field potential” currents), higher frequencies, or other modalities of neuronal function are of little concern to the clinician (so far). Sophisticated functional neuroimaging monitors have already been proposed to measure brain function and cognitive awareness in astronauts; applications to the OR will inevitably follow.

Most anesthesiologists recognize that the EEG slows under deepening planes of inhalation anesthesia (following an initial increase in frequency and decreased amplitude, “beta activation,” excitement phase, during stage II). Although the EEG is not a good predictor of depth of anesthesia, this is somewhat dependent on anesthetic agents/techniques, the age and physiology of the patient, and the EEG descriptors employed in the analysis. Surgical planes of anesthesia are correlated with slower EEG frequencies, such that there is a predominance of delta and theta activity under deep volatile agent inhalation general anesthesia. Conversely, emergence from general inhalation anesthesia is characterized by a return to pre-anesthetic (higher frequency: alpha and beta) EEG frequencies.

In addition to a number of other modifying factors (eg, extremes of age, disease states), the EEG varies in frequency and amplitude with changes in metabolic state as induced by temperature, perfusion, or pharmacologic agents. Hypothermia and hypoperfusion predictably slow the EEG, as do agents such as barbiturates, propofol, or etomidate. Slowing of the EEG is correlated with a decrease in cerebral metabolic oxygen requirements (CMRO2) and may be “neuroprotective” when induced electively by either hypothermia or pharmacologic interventions (burst suppression). In contrast, EEG slowing due to a low blood pressure, low flow, or hypoperfusion, although a “normal” physiologic CNS response, is indicative of early ischemia and is not desirable.

The propensity of the EEG to slow with decreased blood pressure/perfusion emphasizes the importance of the EEG as a measure of “functional cerebral perfusion” and the adequacy of blood flow during periods of controlled hypotension. Indeed, recent work by Monk et al. has correlated brief periods of hypotension and lowered EEG frequencies under anesthesia with increased morbidity and subtle changes in personality traits or cognitive function in elderly patients. Familiarity with the EEG as a monitor of “functional cerebral perfusion” is required of the diligent anesthesiologist during challenging clinical situations. An understanding of data acquisition, signal analysis, limitations, and artifacts of the EEG is also necessary in order to avoid errors in clinical judgment or too great a reliance on highly variable descriptive parameters of the processed EEG when making clinical decisions.

EEG signal processing

Raw EEG

In the OR, raw EEG data using these monitors are usually obtained from scalp electrodes placed on the forehead overlying the frontal cortex (an area of the CNS known to be involved in perception and memory/amnesia formation). The raw EEG signal is filtered and amplified within each monitor at default settings that can be modified by user accessible screens. The BIS monitor employs a single strip array of four electrodes placed from midline to right or left side of the forehead. Raw signal EEG is filtered and displayed on the monitor as a single channel, monitoring one
hemisphere of the underlying frontal cortex. The PSA monitor uses a bilateral hemispheric (frontal lobe) array of six electrodes. Raw EEG is filtered and displayed as two channels per hemisphere or four channels of raw EEG data, when that screen display is selected. When comparing the raw EEG between hemispheres, only the PSA monitor provides simultaneous bilateral frontal lobe recordings. To do the same with the BIS monitor, one would need to add another electrode strip and monitor on the opposite hemisphere. The BIS monitor does provide for two-channel recording; however, the data are obtained from only one hemisphere. A two-hemisphere BIS monitor with individual hemisphere display of processed EEG and BIS numbers is in development and awaiting FDA approval (Kelley S, Medical Director, Aspect Medical, Newton, MA, Personal Communication).

Comparing amplitude and frequency of raw EEG between two hemispheres is of practical clinical application when there is need to follow functional cerebral perfusion differences between hemispheres. For example, when carotid endarterectomy is complicated by an incomplete or compromised anterior communicating (“a com”) artery at the Circle of Willis, monitoring functional cerebral perfusion by following changes in amplitude and frequency between hemispheres may guide the need for shunting (incomplete “a com”) or need to increase blood pressure to perfuse across a constricted anterior communicating artery. Theoretically, this can be done with the PSA monitor or similarly accomplished by placing the BIS monitor strip over the hemisphere at risk. The advantage of the PSA monitor, in this setting, is that EEG decreases in both channels, theoretically indicating global, bilateral, hypoperfusion ischemia changes, whereas changes in only one channel, on the hemisphere at risk, indicate compromised perfusion (as above). Placing the single BIS strip over the hemisphere at risk leaves in question the adequacy of perfusion to the contralateral frontal lobe when perfusion-related EEG changes are observed (ie, is the EEG change observed due to local or global hypoperfusion?).

Visual inspection of raw EEG data is difficult for the trained eye, interpretation even more so. When looking at raw EEG recordings on these monitors, brief episodic attenuation of amplitude or decreased frequency can be missed, as the display is short-lived (4 second sweep). Capturing the raw EEG (“freezing” the display) is not possible with either monitor, unfortunately. More important is the recognition of trends in other descriptive parameters of the EEG: spectral edge, dominant frequencies, and suppression ratio. Each monitor displays these descriptive parameters differently (or not at all), and each provides an additional descriptive parameter (PSI or BIS number) unique to the monitor. Since raw EEG visual inspection is problematic, most clinicians rely on these mathematically derived descriptive parameters to reveal EEG trends and guide decision making.

Spectral edge frequency

The spectral edge frequency (SEF) is defined as two standard deviations above the total power frequency or that frequency beneath which 95% of the EEG falls. It is essentially the highest frequency recorded or the upper limit of EEG obtained during a recording period (“epoch”). An epoch is merely a brief period of time during which the EEG is analyzed (usually 2-4 seconds).

As the EEG is dynamic and changing with time, so the SEF changes with the EEG and follows changes of activation (arousal) or depression of the EEG during anesthesia. The SEF has been suggested to be an easily recognized parameter that follows depth of anesthesia. The SEF is displayed as a number by the PSA monitor and as both a number and a trend on the BIS monitor display. Trending the SEF provides easily recognized changes in SEF and quicker recognition of activation or depression of the EEG when using the BIS monitor. Changes in SEF also appear faster than changes in either BIS or PSI numbers as these proprietary descriptors require more time for data acquisition and algorithm calculations prior to display on the monitors.

The SEF may rapidly change in response to surgical stimulation or level of analgesia/anesthesia without apparent change in either dominant frequency or BIS number. As such, SEF can be used as an “early warning” indicator of impending changes in depth of anesthesia or BIS number. Transient changes in SEF may not be clinically significant or due to artifact; sustained changes are followed by changes in the BIS or PSI numbers and deserve attention.

Artifact recognition

The raw EEG and SEF are subject to artifact induced by extraneous signal contamination produced by electrocautery (Bovie), vibration (drilling), evoked potential stimulation (facial nerve stimulation), and/or muscle twitch (neuromuscular blockade) monitor, despite artifact filters built into each monitor’s circuitry. During periods of artifact noise, each monitor may display contaminated, unreliable information that should not be used to interpret EEG changes or guide clinical decisions. On the BIS and PSA monitors, artifact noise appears as an increase in the EMG (electromyogram) signal strength bar. Noise interference recognition has been reported to be better with the PSA monitor (PSArray2) as compared with the BIS monitor (XP array). The EMG will also increase as neuromuscular blockade diminishes, and may be used as a “rich man’s” twitch monitor to indicate early return of facial muscle activity. Although this is not adequate to guide reversal decision, it may alert to the need for supplemental blockade when profound neuromuscular paralysis is required.

Artifact may also be generated by improper electrode contact as indicated by impedance measurement on each monitor (SQI: signal quality indicator on the BIS monitor).
Each monitor self checks electrode contact impedance on start up and alerts the user as to the location of loose electrodes. Intraoperative disruption of electrode contact also will generate artifact and error messages. Taping the electrode array to the forehead minimizes the chance of loose electrode contact. Tapping rhythmically on the electrode array (surgeon’s hands) will not only generate artifact, but also will generate artifact and error messages. Taping the start up and alerts the user as to the location of loose electrode array to the forehead minimizes the chance of fusion and alert the anesthesiologist to an impending isch-

**Suppression ratio**

The suppression ratio (SR) is defined as the percent time of flat line EEG in a given interval (approximately 60 seconds). Thus, 30 seconds of cumulative 0 amplitude EEG would yield an SR value of 50. Since this is a summed ratio, the SR value of 50 may represent a full 30-second flat line EEG, or two 15-second episodes, or three 10-second episodes, etc. It is important when evaluating SR numbers to observe the raw EEG during each sweep to determine the apparent duration of flat line EEG. The BIS monitor displays SR as a number, making quantification of burst suppression simple. The PSA-4000 monitor displays SR as a linear bar graph, without a number, making SR evaluation problematic (software upgrades to give a numerical SR number are available). Both monitors display intervals of burst suppression as breaks in data displays. The PSI (Patient State Index) display on the PSA-4000 is color coded to show burst suppression. The BIS monitor trends SR and BIS as a screen selection linear or tabular display choice.

The SR is used clinically to quantify the duration of flat line EEG (burst suppression) during attempts to provide pharmacologic brain protection with propofol, barbiturates, or etomidate and a concomitant reduction in CMRO2 (silencing the EEG for brief periods is analogous to lowering the heart rate to reduce myocardial oxygen requirements). Since approximately 60% of CMRO2 is relegated to producing EEG, significant theoretical reductions in CMRO2 may be obtained when burst suppression is twice the duration of active EEG. This 2:1 (suppression:burst) ratio may reduce CMRO2 by 40% (2/3 of 60), provided the suppression duration is at least 10 seconds. At the author’s institution, we strive to maintain a burst suppression duration of 10-20 seconds with a 5-second interval of active EEG. This is usually accomplished with etomidate (20-40 mg IV bolus) to establish the burst suppression, followed by propofol infusions at 100-150 ug/kg/min (0.6 cc/kg/hour) titrated to maintain an SR value of 66 or higher. Etomidate potentiates the propofol effects, while maintaining blood pressure (BP). Occasionally vasopressors may be required to control hypotension induced by propofol. Using these parameters and doses, a clinical benefit in surgical outcome has been demonstrated for open craniotomy aneurysm clipping surgery with intraoperative pharmacologic burst suppression.

Conversely, during periods of controlled hypotension or in trauma surgery, the appearance of elevated SR values (“burst suppression”) may indicate inadequate cerebral perfusion and alert the anesthesiologist to an impending isch-

**Density spectral array**

Perhaps a more useful clinical application of EEG processing is obtained by mathematical analysis of the EEG which converts the raw EEG into a comprehensible display. Rampil has exhaustively and elegantly discussed the process of EEG analysis. The interested reader is referred to his and other publications.15,26 The density spectral array (DSA) provides for recognition of dominant frequencies over time, displayed as increased density of data points during each epoch (2-4 seconds) of raw EEG analysis. The display updates over time so that one may follow changes in dominant frequencies throughout the course of an anesthetic, surgical, or pharmacologic manipulation.

The BIS monitor presents DSA obtained from one hemisphere, although a two-hemisphere, bifrontal separate DSA (and BIS) display monitor upgrade is in the process of FDA approval (Kelley S, Medical Director, Aspect Medical, Newton, MA). The PSA-4000 monitor does not display DSA, a major disadvantage to EEG interpretation. This flaw has been corrected by the evolution of the PSA monitor to the Sedline Monitor (Hospira, Inc., Lake Forest, IL), providing bispectral DSA displays with SEF trending and burst suppression (Sedline Monitor, Hosperia, Inc., Lake Forrest, IL). The monitor lacks individual hemisphere PSI numbers (a single PSI is calculated and displayed). The SR is presented in trend and as a numerical value, similar to the display on the BIS monitor. The BIS monitor DSA display will be presented as Sedline clinical examples are lacking.

The awake EEG can be characterized as “unorganized” with no dominant frequency patterns appearing on the DSA. With induction of anesthesia, the EEG becomes more “organized” with the appearance of frequency bands on the DSA. The transition from awake EEG to the anesthetized state is rapid26 and readily apparent on the DSA display (Figure 1). Light general inhalation anesthesia may often be accompanied by the appearance of alpha and delta bands, with an SEF in the low teens. Deeper levels of inhalation general anesthesia display predominant theta and delta bands with SEF below 12 (Figure 2). Narcotic infusions combined with inhalation general anesthesia (“balanced anesthesia”) tend to produce a predominant alpha band with delta activity and a slightly higher SEF (Figure 3).

These DSA trends are highly variable between patients and should not be used as a measure of depth or adequacy of anesthesia. It is falsely reassuring to view an expected...
DSA display and expect that a given patient is amnestic or will not move to surgical stimulation. As with any other single modality, the DSA can be an aid to observing anesthetic drug effects when used with reservation and caution.

The utility of the DSA display is most often as an indicator of change from one dominant frequency pattern to another requiring correlation with ongoing clinical processes or manipulations. For example, the sudden appearance of slow frequency dominance coupled with hypotension may precede or indicate ischemia or guide shunting interventions to offset ischemia during balloon occlusion and carotid endarterectomy. Hypotension in the geriatric patient may lead to cerebral ischemia (see above); the DSA may help visualize changes in functional cerebral perfusion in these patients at risk by virtue of age, disease, or operative position. These interpretations are further limited by the electrode placement over the frontal cortex; one cannot extrapolate changes to another, differentially perfused brain region.

Emergence from anesthesia correlates with a disappearance of dominant frequency patterns. The DSA changes on emergence are less rapid than induction changes. Patients may respond to verbal stimuli and follow commands while displaying DSA consistent with light general anesthesia (as above). Except in cases of elective postoperative sedation, a recovered patient should not display dominant delta or theta activity on the DSA. Delayed emergence from anesthesia may be followed in the PACU (post-anesthesia care unit) using the DSA in an attempt to differentiate cerebral hypoperfusion states from individual sensitivity to anesthesia.

Because of individual patient variations in DSA and EEG responses to anesthetics, one cannot determine whether a given patient is analgesic, anesthetic, or amnestic from a single descriptor. In order to provide this information, proprietary algorithms, incorporating a variety of mathematical treatments of the EEG, have been developed. Their utility, reliability, and clinical application as predictors of depth of anesthesia and/or amnesia or recall continue to generate investigations and debate.

### Proprietary algorithm displays

Each monitor (Aspect BIS or Physiometrix PSA-4000) employs a proprietary algorithm to analyze the raw EEG signal and display that information as a numerical descriptor (PSI or BIS number) that may be correlated to awareness, recall, and depth of anesthesia. These algorithms are based on mathematical transformations of raw EEG into various descriptors of the EEG: mean power frequency, fast Fourier power spectrum, suppression ratio, frequency band dominance or ratios, and other descriptive parameters.
(bispectral coherence or temporal dominance). The exact algorithm or mathematical formulae used to generate the PSI or BIS number remain a proprietary secret, although generalized discussions of their workings have been made available in the literature. Again, the reader is referred to Rampil’s publication for more detail on the derivation of the BIS (Aspect monitor) and the work of Drover et al. for the PSI (PSA monitor). These monitors were originally intended to measure the probability of recall. They have since morphed into presumed monitors of depth of anesthesia, an entirely different entity.

Each EEG descriptor (PSI or BIS) has been scaled from 100 to 0, such that 100 indicates an awake state EEG and 0 indicates electrical silence. At a BIS level of 60 to 20, the probability of recall is substantially reduced and patients achieve surgical planes of anesthesia when BIS levels are near 40. BIS levels below 20 are rare and occur primarily with burst suppression. BIS levels at 20 may be indicative of excessive anesthetic, individual sensitivity or hypotension. Emerging evidence of increased morbidity associated with intraoperative hypotension and low BIS values warrant the careful titration of anesthetics to more than just BP.

The PSA monitor uses a different scale from 50 to 30 as a desired target PSI level during surgical planes of anesthesia. The scale can be adjusted (widened or narrowed) by the user. PSI data is displayed in color-coded histogram format to reveal PSI values falling within the desired range as green, outside the range as orange, and burst suppression as blue (artifact as white, no data black). The Sedline monitor display is more polychromatic and requires training the eye to appreciate the multiple parameters (SR, SEF, burst suppression, and DSA frequencies, etc.) displayed within its colorful montage.

Much of the literature deals with a validation of the methodologies of each monitor and their clinical application. The volume of reports and investigations number in the thousands for the BIS and hundreds for the PSA monitor. There is general agreement that the BIS and PSI descriptors follow hypnotic state under anesthesia. There is concern regarding the utility and reliability of these parameters in various clinical settings, as investigators have published conflicting accounts.

The BIS has been proposed to follow changes in functional cerebral perfusion in head trauma as a predictor of outcome, onset in syncope, in cervical hematoma, during surgical planes of anesthesia, and during cardiac arrest resuscitation or recovery from cardiac arrest outside the hospital.

Of interest is the utility of the BIS (as opposed to raw EEG inspection, SEF, or DSA, discussed above) to follow changes in functional cerebral perfusion during carotid endarterectomy (CEA). Under general anesthesia CEA, the BIS falls and follows changes in evoked potentials, but it is unclear whether this is reliable as BIS has been reported a poor predictor of ischemia in awake CEA or outcome under general anesthesia CEA. In isolated internal carotid injections of barbiturate, the BIS failed to elucidate decreased cerebral function in the isolated hemisphere.

Obviously, there are limitations to the BIS algorithm (and its clinical application). The BIS algorithm is weighted to higher (beta) frequencies at higher BIS numbers and is sensitive to the contribution of EMG and muscle artifact, falling under neuromuscular blockade or rising on reversal. Awake CEA may predispose the BIS to higher numbers, but this doesn’t explain a poor predictor of outcome under general anesthesia. Ischemic stroke may be better revealed by other parametric descriptors: bilateral symmetry or spectral entropy or other time domain frequency analyses.

The BIS algorithm is somewhat bound by the pharmacologic agents employed. General inhalation anesthesia produced by a combination of substituted ethers, nitrous oxide, oxygen, opioid narcotic, and benzodiazepine produce replicable changes in BIS correlated to amnesia/recall and depth of anesthesia. However, a single dose of Midazolam (10 mg IV) produces a BIS reading in the low 80s, with little or no chance of recall. The algorithm may fail or be less reliable when the pharmacologic paradigm under which it was developed is breached.

For this reason, one must consider anesthetic technique and the effects of drugs when interpreting the BIS. Nitrous oxide alone at 50% FIO2 does not change the BIS from baseline, although EEG frequency decreased with sedation. Ketamine increases the BIS. Vasoactive calcium channel blockers, nicardipine or diltiazem, have no effect. Propofol alone produces wide individual variations in awareness and recall, suggesting caution when interpreting BIS values with propofol as a sole anesthetic. EMG artifact is significant in producing high BIS values as the BIS algorithm is weighted to higher EEG frequencies.

Fewer clinical investigations have been reported with the PSI. The PSI has been used to titrate levels of anesthesia and recovery. The PSI falls with cardiac arrest and returns with resuscitation following effective cerebral perfusion. The PSI also falls under spinal anesthesia in awake patients, perhaps through inhibition of ascending reticular activating system input to thalamic and cerebral EEG generators. The PSI has been suggested as a monitor of adequacy pain management and for the assessment of sedation in intensive care unit (ICU) patients. The effects on the PSI of sole agent anesthetic techniques and the impact of vasoactive pharmacologic agents await continued clinical investigation.

Clinical case: Awake internal carotid intravascular stent placement with PSI monitoring.

The subject is a 56-year-old, male with bilateral internal carotid stenosis (>80%). He presents with baseline BP
172/86, P 86; incomplete anterior communicating artery, HTN, CAD, vasculopathy; awake at baseline BP, describes “curtain over eyes” and decreased consciousness at low BPs; MAC anesthesia with 5 mg Midazolam and 50 mg meperidine IV titrated slowly to maintain awake and “normal” BP; intravascular approach via femoral artery under local anesthesia.

Figures 4 and 5 show right internal carotid stenosis before and after balloon dilation and stent placement.

Figure 6 shows PSI trends throughout case. Note initial low baseline PSI number (50s) prior to medication, and improved elevated PSI (70s) following successful stent placement.

This is the first published case of bilateral internal carotid stenosis with low baseline PSI values demonstrating a change in PSI following stent placement. Low initial BIS numbers have been reported in patients with dementia and in genetically determined low voltage EEG. Conclusions

Both PSI and BIS seem to follow functional cerebral perfusion during periods of hypotension and resuscitation, as well as the level of hypnosis and/or depth of anesthesia. Clinical use of the BIS or PSI should include observations of the DSA and SEF to follow changes in underlying EEG frequencies contributing to these descriptors. Such changes need to be correlated with ongoing surgical, anesthetic, pharmacologic, and/or physiologic manipulations. All interpretations of monitored parameters require inspection and consideration of potential artifacts prior to making any clinical decisions.

Despite these caveats, the clinical application of routine intraoperative EEG monitoring to everyday anesthetic management is facilitated by these monitors. Refinements and technologic advances in computation EEG signal processing are already being applied to EEG analysis and continued experimental validation of the BIS and PSI. Thirty years of improvements in intraoperative EEG monitoring and enhancements to data display have provided a readily accessible window into the final common pathway and target organ of anesthetics. The next 30 years should be equally productive.

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Figure 5  Internal carotid after stent placement. Note increased vessel diameter compared with Figure 4.

Figure 6  PSI trends during MAC anesthesia for awake intravascular internal carotid stent placement. Note baseline low PSI numbers (50s) prior to medication and improved PSI (70s) following carotid stent.
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