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Basic Principles of Processed Electroencephalography for Neuroanesthesiologists

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Abstract

Processed electroencephalography (pEEG) has become an integral tool in modern anesthesia and critical care, enhancing the precision of anesthesia depth monitoring, reducing the risk of accidental awareness under general anesthesia (AAGA), and postoperative cognitive issues. Unlike raw EEG, which records cortical electrical activity directly, pEEG applies mathematical and algorithmic analyses, such as spectral analysis and Fourier transformation, to generate numerical indices that are more interpretable for clinicians. Several commercial systems, including the Bispectral Index (BIS), Entropy, Conox, and SedLine, are widely available. For neuroanesthesiologists, understanding core EEG principles and advanced metrics, such as the Density Spectral Array (DSA), Spectral Edge Frequency (SEF), and Burst Suppression Ratio (BSR), is crucial for accurate interpretation. Moreover, recognizing EEG patterns characteristic of various anesthetic agents, including propofol, inhaled agents, dexmedetomidine, ketamine, and opioids, further refines clinical decision-making. Mastery of EEG interpretation ultimately supports better safety, individualized neuroanesthesia practice aligned with the principles of precision medicine.

Keywords: Density spectral array, EEG monitoring, neuroanesthesia, processed EEG

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Introduction

In recent years, the use of processed electroencephalography (pEEG) has increased substantially in anesthetic and intensive care practice. This trend parallels grow evidence that accurate monitoring of sedation and anesthesia depth can reduce perioperative complications, particularly in patients with complex comorbidities.[1,2] Processed EEG enables anesthesiologists to directly assess patients' level of sedation while providing more objective and quantifiable information compared to conventional clinical assessment methods.

Nevertheless, knowledge of and familiarity with pEEG and electroencephalography (EEG) in general remain limited among Indonesian anesthesiologists. This gap largely stems from the absence of EEG training in the current anesthesiology residency curriculum in Indonesia. The use of EEG as a tool for brain function monitoring offers significant advantages, particularly in enhancing the precision of anesthetic administration and minimizing the risk of postoperative neurological complications. At present, several commercial pEEG systems are available, each employing distinct algorithms and features.[3] These devices possess unique strengths and limitations in terms of cost, availability, and available parameters. For neuroanesthesiologists who frequently manage patients with neurological disorders, proficiency in EEG principles and interpretation is becoming

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an essential competency. As neuroanesthesia continues to evolve and greater attention is paid to personalized anesthetic dosing, effective utilization and understanding of EEG data have emerged as crucial clinical competencies.

Literature Review

Physiological Basis and Clinical Applications

Electroencephalography (EEG) is a widely used neurophysiological monitoring modality that records the brain's electrical activity non-invasively through electrodes placed on the scalp. First described by Hans Berger in 1929, the EEG revealed spontaneous electrical oscillations in the human brain.[3] These electrical signals represent the collective activity of large populations of pyramidal neurons within the cerebral cortex.[3,4] EEG provides real-time information about the brain's functional dynamics, allowing rapid detection of changes in neural activity.[5] This neuronal activity is influenced by both intrinsic factors (such as the sleep–wake cycle) and extrinsic factors (including anesthetic pharmacology).

Traditionally, EEG activity is classified into several frequency bands that correlate with distinct neurophysiological states. Delta waves (0.5–4 Hz) are prominent during deep sleep and deep anesthesia, while theta waves (4–8 Hz) appear during drowsiness or light sleep. Alpha waves (8–13 Hz) are observed in relaxed wakefulness with closed eyes and diminish during mental activity. Beta waves (13–30 Hz) are associated with alertness and active cognition, whereas gamma waves (>30 Hz) reflect complex sensory processing and higher consciousness.[3-5] In deep anesthesia, EEG may exhibit burst suppression patterns, characterized by alternating periods of high-amplitude activity and isoelectric silence.[3]

Within neuroanesthesia practice, EEG serves multiple critical clinical applications. One major use is monitoring the depth of anesthesia, for which several processed EEG (pEEG) systems have been developed to quantify EEG activity into a numerical index representing the patient's level of consciousness. In addition, real-time changes in EEG patterns may indicate inadequate cerebral perfusion in surgeries with a high risk of cerebral ischemia, such as carotid endarterectomy or aneurysm clipping.[3,6] In the intensive care unit (ICU), EEG is used to monitor brain activity in patients with altered consciousness, detect non-convulsive status epilepticus (NCSE), and assess sedation depth in mechanically ventilated patients. Among patients with traumatic brain injury or post–cardiac arrest, EEG patterns such as burst suppression or isoelectric tracing can serve as prognostic indicators or therapeutic guides.[3]

Definition, Mechanism, and Clinical Evidence

Processed electroencephalography (pEEG) refers to EEG monitoring that undergoes mathematical and algorithmic processing to generate numerical indices or specific parameters intended to facilitate clinical interpretation of a patient's level of consciousness.[2-4] Unlike raw EEG, which displays direct electrical brain activity, pEEG presents more simplified data. This approach can be particularly advantageous for non-neurophysiologist clinicians, such as neuroanesthesiologists and neurointensivists, as it enables rapid and practical interpretation in the operating theatre and the intensive care unit (ICU), supporting real-time clinical decision-making in dynamic settings.

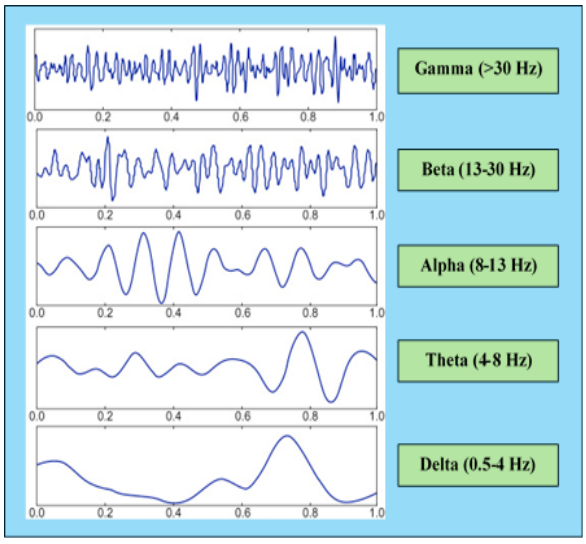

Figure 1. Brain waves divided by their frequency bands. Adapted from: Abhang PA, Gawali BW, Mehrotra SC. Introduction to EEG and Speech-Based Emotion Recognition. Academic Press; 2016

pEEG signals are derived from raw EEG recordings obtained via scalp electrodes. These signals are then processed using mathematical techniques such as spectral analysis, Fourier transformation, and proprietary algorithms to produce single numerical parameters representing anesthesic depth or sedation level.[3,4] The first widely recognized pEEG technology was the Bispectral Index (BIS), developed in the late 1980s by Aspect Medical Systems (USA) and introduced commercially in 1994.[3] BIS utilizes bispectral analysis, a complex statistical method

Table 1. Incidence of AAGA in Several Clinical Trials

Study (Year)	Device Studied	Patient Population	AAGA Incidence (Device vs Control)	Outcome
B-AWARE (2004)	BIS vs Standard Care	2,463 high-risk adult sur-gical patients (RCT)	0.16% vs 0.89% (2 vs 11 cases	BIS ↓ awareness (p = 0.022)
B-UNAWARE (2008)	BIS vs ETAC (0.7–1.3 MAC)	1,941 high-risk adult sur-gical patients (RCT)	1,941 high-risk adult sur-gical patients (RCT)	No difference
BAG-RECALL (2011)	BAG-RECALL (2011)	±5,700 high-risk adult surgical patients (RCT)	0.66% vs 0.28% (19 vs 8 cases)	No BIS benefit
Mashour (2012)	BIS vs ETAC	18,836 general adult surgical patients (RCT)	0.08% vs 0.12% (estimated)	No difference
Zhang TIVA (2011)	BIS vs Routine TIVA	5,228 adult surgical patients (RCT, TIVA	0.14% vs 0.65% (4 vs 15 cases)	BIS reduced incidence of awareness (p = 0.002)

BIS: Bispectral Index; ETAC: End-Tidal Anesthetic Concentration; TIVA: Total Intravenous Anesthesia; AAGA: Accidental Awareness during General Anesthesia; RCT: Randomized Controlled Trial; MAC: Minimum Alveolar Concentration

Table 2. Incidence of Postoperative Delirium in Several Clinical Trials

Study (Year)	Depth Monitor	Patient Population	Delirium Incidence (Intervention vs Control)	Cognitive Outcomes
CODA (2013)	BIS vs Standard Care	921 adult patients ≥60 years (RCT)	15.6% vs 24.1% (POD by CAM, p = 0.01)	3-month POCD: 10.2% vs 14.7%, p = 0.025
ENGAGES (2019)	Processed EEG vs Standard Care	1,232 adult patients ≥60 years (RCT)	26.0% vs 23.0% (POD by CAM, p = 0.22)	30-day cognition not different; 30-day mortality lower in EEG group (0.65% vs 3.07%, not a primary endpoint)
Balanced-Delirium	BIS 50 vs BIS 35	655 adult patients ≥60 years	19% vs 28% (POD, days 1–5, p = 0.010	Early postoperative cognition: lower delirium risk in BIS 50 group

POD: Postoperative Delirium; CAM: Confusion Assessment Method; POCD: Postoperative Cognitive Dysfunction; RCT: Randomized Controlled Trial

that evaluates phase relationships between EEG frequencies, resulting in an index value ranging from 0 (isoelectric EEG, no cortical activity) to 100 (fully awake).[3,6] A BIS range of 40–60 is generally considered optimal for surgical anesthesia.

Beyond BIS, several other pEEG systems are now commercially available, including Entropy (GE Healthcare), Patient State Index (PSI; Masimo SedLine), Narcotrend, and Conox (Fresenius Kabi). Each device employs different mathematical algorithms but shares the same objective: to provide a quantitative, real-time representation of consciousness level. Many also offer additional visualizations, such as spectrograms or Density Spectral Array (DSA) plots, that display the continuous frequency distribution of EEG activity during anesthesia or sedation, as further discussed in a later section.[3]

Initially, the use of pEEG in anesthesia was closely associated with total intravenous anesthesia (TIVA). This was primarily influenced by findings from the National Audit Project 5 (NAP5) in the United Kingdom (2014), which identified accidental awareness during general anesthesia (AAGA) as most commonly associated with TIVA with neuromuscular blockade and inadequate depth of anesthesia monitoring.[2,3,7] NAP5 recommends that pEEG monitoring should be considered during TIVA to ensure optimal dosing and prevent AAGA.[3,7] Several large-scale clinical trials have since evaluated the benefits of pEEG monitoring, particularly in preventing AAGA and optimizing anesthetic management. The B-Aware Trial, a double-blind study involving 2,463 high-risk patients, demonstrated that BIS monitoring significantly reduced the incidence of AAGA compared with conventional clinical monitoring.[3,8] However, subsequent trials such as the B-Unaware Trial and the BAG-RECALL Trial found that BIS monitoring was not superior to monitoring end-tidal anesthetic agent concentration (ETAC) in preventing AAGA, showing equivalent efficacy under rigorous ETAC-based protocols.[7-9] A Cochrane meta-analysis concluded that pEEG monitoring was associated with a reduced risk of AAGA, particularly among high-risk populations. However, this reduction did not consistently translate into improved long-term outcomes such as mortality or postoperative neurological complications.[1]

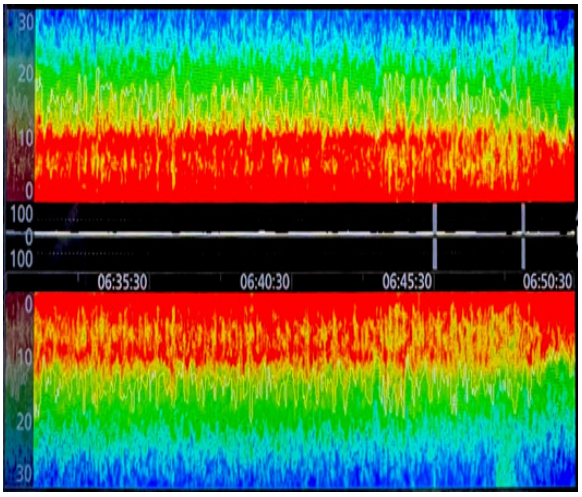

Figure 2. Spectrogram of a patient under sevoflurane anesthesia characterized by increased power in the delta, theta, and alpha frequency bands

Beyond AAGA prevention, recent studies have highlighted the potential of pEEG to reduce postoperative delirium (POD) and postoperative cognitive dysfunction (POCD). The CODA Trial demonstrated that pEEG-guided anesthesia significantly decreased the incidence of postoperative delirium.[9,10] This effect is attributed to reduced anesthetic overexposure, minimizing neurotoxicity and preserving brain function. While evidence for long-term cognitive protection remains inconsistent, emerging data suggest that precision-guided anesthetic management may offer neuroprotective benefits, particularly in vulnerable populations such as the elderly and patients with pre-existing neurological conditions (e.g., traumatic brain injury, stroke, neurodegenerative disease).[6] Several professional societies have issued recommendations on the use of pEEG. The American Society of Anesthesiologists (ASA), Association of Anaesthetists of Great Britain and Ireland (AAGBI), and European Society of Anaesthesiology and Intensive Care (ESAIC) recommend selective use of pEEG, particularly in high-risk patients or during TIVA.[10] Meanwhile, the Society of Critical Care Medicine (SCCM)

supports the use of pEEG in the ICU to assess sedation depth in mechanically ventilated patients, detect non-convulsive status epilepticus (NCSE), and monitor burst suppression in patients with elevated intracranial pressure.[3]

Advantages and Limitations

Anesthetic depth indices represent a key component of processed EEG (pEEG) monitoring in clinical practice. These indices are dimensionless numerical values, typically scaled from 0 to 100, reflecting the patient's level of consciousness or hypnotic depth; higher values indicate wakefulness, whereas lower values correspond to more profound anesthesia. The primary objective of these indices is to simplify complex raw EEG signals and quantitative parameters into a single, easily interpretable value that can guide anesthetic titration.[3] This practicality has facilitated the widespread clinical adoption of pEEG.

However, despite their clinical usefulness, pEEG indices have notable limitations. In a recent study comparing five pEEG systems simultaneously, the same EEG signal produced five different index values, underscoring substantial inter-device variability.[10] Moreover, external factors such as electromyographic (EMG) activity and the use of neuromuscular blocking agents can significantly influence index readings. These limitations highlight the importance of interpreting pEEG indices within a broader clinical and neurophysiological context rather than relying solely on numerical values.[11-13]

Spectrogram, Spectral Edge Frequency (SEF), and Burst Suppression Ratio (BSR)

A spectrogram is a visual representation of EEG activity that illustrates changes in signal frequency and amplitude over time. It provides a dynamic view of the continuously evolving oscillatory EEG patterns during anesthesia. The spectrogram is generated through a mathematical process known as the Fourier transformation, which decomposes raw EEG signals into its fundamental frequency components. Each spectrum is typically calculated over a 3-second time window, with a 0.5-second overlap between segments. Sequential arrangement of these spectra across the EEG recording produces a spectrogram that reflects changes in frequency power distribution.[3,4] Two common visual formats are frequently described in the literature. The first, the Compressed Spectral Array (CSA), presents the spectrogram as a three-dimensional plot, where the x-axis represents time, the y-axis frequency, and the z-axis (often color or height) indicates signal power. The second, the Density Spectral Array (DSA), displays a two-dimensional representation, with time on the horizontal axis, frequency on the vertical axis, and power intensity represented by color gradients.[3,4] Most modern pEEG devices have incorporated DSA as a standard feature because it provides an intuitive and rapidly interpretable visualization of dominant frequency shifts during anesthesia.

An additional parameter derived from the spectrogram is the Spectral Edge Frequency (SEF). SEF represents the frequency below which a specified proportion (typically 90–95%) of the total EEG power is contained.[3,4] For instance, SEF-95 denotes the upper frequency limit that encompasses 95% of the total EEG power at a given moment. SEF serves as a valuable indicator of anesthetic depth, as the dominant EEG frequency generally decreases with increasing depth of anesthesia. High SEF values (e.g., >15–20 Hz) are typically observed in awake or lightly sedated patients, whereas values around 8–12 Hz indicate adequate surgical anesthesia. A further reduction below 8 Hz may indicate profound anesthesia.[3] However, in very deep anesthesia where slow-wave power also diminishes, SEF may paradoxically increase.

Another commonly available EEG parameter is the Burst Suppression Ratio (BSR). Burst suppression refers to a distinct EEG pattern characterized by alternating periods of high-amplitude activity (bursts) and very low or isoelectric activity (suppressions) (Figure 3C). Figure. BSR quantifies this phenomenon as the percentage of time during which the EEG remains suppressed over a defined period. This pattern typically emerges under conditions of profound anesthesia or pathological states, such as severe

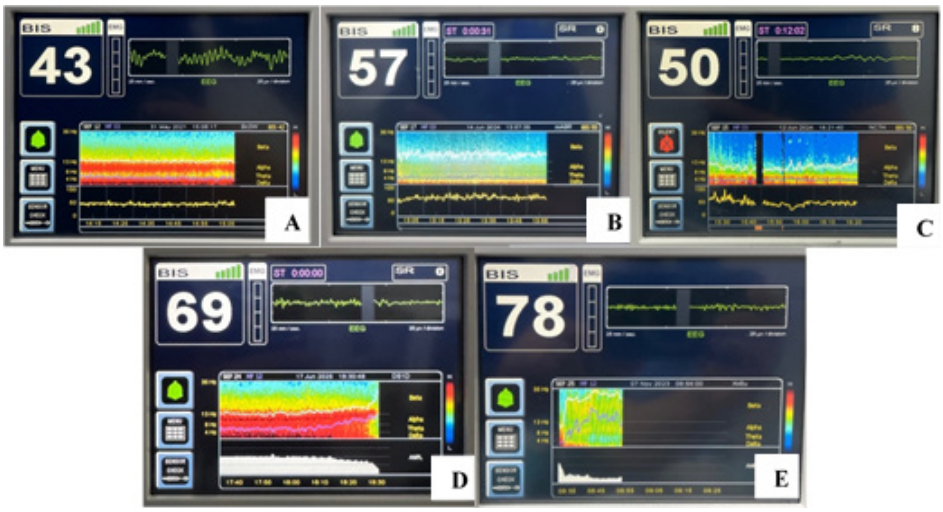

Figure 3. (A)The Bispectral Index of a patient undergoing propofol maintenance demonstrates an SEF of 12, which aligns closely with the EEG trace, index, and spectrogram findings. (B) Deep anesthesia is indicated by a low-power EEG tracing and corresponding spectrogram; however, both the index and SEF presented unexpectedly elevated values. (C) Patient under deep anesthesia. Suppression ratio (SR) is 8, meaning that 8% of the last 63s of EEG consisted of suppressed or isoelectric EEG. Suppression time (ST) shows the total cumulative time spent in suppression. (D) The patient was under sevoflurane anesthesia. From 17:30 to 18:10, alpha, theta, and delta power remained uniform. During awakening, a zipper pattern appears as brain sevoflurane levels decrease. (E) Ketamine sedation in an elderly patient. Note the activation at a higher frequency around 8:35.
Original figures by the author

brain injury, cerebral hypoxia, deep hypothermia, or as a pharmacologic effect of high doses of anesthetic agents like propofol or barbiturates.[3,4]

Clinically, anesthesiologists should generally avoid burst suppression during intraoperative anesthesia, as it indicates excessive anesthetic depth. The presence of burst suppression has been associated with hemodynamic instability and an increased risk of postoperative cognitive dysfunction, underscoring the importance of continuous EEG monitoring as part of balanced anesthetic management.

Effects of Propofol on EEG

Propofol acts primarily through GABAA receptors, inducing neuronal hyperpolarization within the cerebral cortex. This results in a characteristic EEG pattern dominated by frontal alpha oscillations (8–12 Hz) and slow delta oscillations (0.1–4 Hz) (Figure 3A). During induction, propofol produces a rapid transition from the awake EEG pattern, characterized by low-amplitude beta and gamma activity, to a pattern of high-amplitude delta and slow oscillations. This transition reflects reduced thalamocortical activity due to enhanced inhibition of thalamic neurons mediated by propofol.[3,4]

At deeper anesthetic levels, propofol may induce a burst suppression (BS) pattern, characterized by alternating periods of high-amplitude bursts and isoelectric suppression. This pattern indicates profound anesthesia, with significantly reduced cerebral metabolic activity. Clinically, the EEG signature of propofol is notably consistent and regular, making it easily recognizable on spectrograms (Figure 2 left).[3,4]

Effects of Inhalational Anesthetic Agents (Sevoflurane, Isoflurane, and Desflurane) on EEG

At sub-Minimum Alveolar Concentration (MAC) levels, all three volatile anesthetics (sevoflurane, isoflurane, and desflurane) produce EEG patterns characterized by prominent frontal alpha oscillations (8–12 Hz) accompanied by slow delta waves (0.5–4 Hz). This pattern closely resembles that produced by propofol, reflecting enhanced thalamocortical GABAergic inhibition.[3,4] As the concentration increases to MAC or higher, theta

oscillations (4–7 Hz) emerge between the delta and alpha bands, resulting in a more uniform power spectrum extending from low to alpha frequencies (Figure 2 and Figure 3D). This EEG pattern is typically observed during the maintenance phase of anesthesia at higher gas concentrations. The presence of theta oscillations is associated with deep unconsciousness and immobility, serving as a useful clinical indicator of anesthesia depth.[3,4] During emergence, theta oscillations disappear first, followed by the attenuation of alpha and delta activity (zipper-like appearance) (Figure 3D). The EEG then transitions to low-amplitude beta–gamma activity, resembling that of an awake patient.[3,4] Similar to propofol, very high concentrations of inhalational agents can induce burst suppression (BS). However, the BS pattern produced by volatile anesthetics is often more variable and less regular than that observed with propofol.[4] This variability is attributed to the broader effects of volatile agents on multiple neuronal circuits, including both thalamocortical and cortical inhibitory neurons, leading to a more heterogeneous EEG appearance.[4]

Effects of Dexmedetomidine on EEG

Dexmedetomidine, an anesthetic and sedative agent acting as a selective α_2-adrenergic receptor agonist, produces a distinctive EEG pattern that closely resembles natural non–rapid eye movement (non-REM) sleep. EEG recordings under dexmedetomidine are characterized by dominant slow delta waves of moderate amplitude, periodically interspersed with spindle-like bursts (9–15 Hz). These spindles represent activity within thalamocortical circuits, similar to those observed during physiological sleep.[3,4] This EEG pattern reflects the lighter sedative state produced by dexmedetomidine compared with propofol or volatile anesthetics. Patients typically remain easily arousable with mild stimulation, despite being clearly sedated. At higher doses, dexmedetomidine enhances the dominance of slow delta activity while diminishing the spindle features, resulting in an EEG pattern closely resembling deep slow-wave sleep (stage N3 of non-REM sleep).[3,4]

Effects of Ketamine on EEG

Ketamine produces a markedly different EEG pattern compared with other anesthetic agents, primarily due to its mechanism of action as an N-methyl-D-aspartate (NMDA) receptor antagonist. At both sedative and anesthetic doses, ketamine induces a prominent increase in high-frequency activity (25–32 Hz) within the beta–gamma range, reflecting enhanced cortical neuronal activity secondary to disinhibition of excitatory neurons (Figure 3E).[3,4] This phenomenon helps explain why ketamine often elicits dissociative states, hallucinations, or euphoria, representing paradoxical cortical hyperactivation rather than suppression, as observed with most other anesthetic agents. The EEG pattern associated with ketamine is also less organized and more variable compared with the characteristic, regular patterns seen with propofol or inhalational anesthetics.[3,4]

Effects of Opioids on EEG

Opioids generally exert minimal effects on the EEG when administered at standard analgesic doses. The typical EEG change observed is a slight slowing of frequency toward low-amplitude theta and delta waves, indicating that opioids alone rarely induce a state of deep anesthesia. However, at very high doses, opioids can induce marked EEG slowing, reflecting profound central nervous system depression.4 Clinically, this deep EEG suppression may be accompanied by severe respiratory depression and hemodynamic instability, underscoring the narrow therapeutic margin of opioids when used beyond their analgesic range.

pEEG Electrode Placement and Alternative Sites

In processed EEG (pEEG) monitoring systems such as BIS, SedLine, and Entropy, the standard electrode placement is typically located in the frontal region. This location is chosen because it effectively captures the phenomenon of anteriorization—a shift of dominant EEG activity toward the frontal cortex that occurs during anesthesia.[3,4] Frontal placement also provides a stable and easily interpretable signal, making it the preferred configuration in most surgical and intensive care settings. However, in neurosurgical procedures, particularly supratentorial surgeries

or bifrontal craniotomies, the frontal area is often inaccessible due to the surgical field or interference from equipment such as Mayfield head pins or neuronavigation devices. In such cases, anesthesiologists must identify alternative electrode positions that can still yield reliable EEG signals for intraoperative monitoring.

Several studies over the past decade have evaluated alternative electrode sites, including:

1. Supralabial region – evaluated by Dubey et al.[14]
2. Postauricular area – studied by Akavipat et al. and Abdelrahman et al.[15,16]
3. Nasal dorsum – proposed by Hajiyeva et al. and Nelson et al.[17,18]
4. Temporal and lateral placements – reported to maintain correlation with frontal readings during the maintenance phase, but less consistent during emergence.[19]
5. Infraorbital, lateral, and semilateral placements – assessed by Isik et al. using the SedLine system.[20]

These alternative placements provide potential solutions when standard frontal sites are unavailable, though signal reliability and inter-device correlation may vary depending on the monitoring system and the phase of anesthesia. Continued research is warranted to determine the optimal electrode configurations for neurosurgical applications without compromising data accuracy or patient safety.

Conclusion

The use of processed electroencephalography (pEEG) continues to expand in the era of personalized medicine, driven by increasing emphasis on patient safety and precision anesthesia. With the growing variety of monitoring devices available on the market, selecting and interpreting pEEG appropriately has become an integral component of modern anesthetic practice. For neuroanesthesiologists, a solid understanding of EEG fundamentals is essential. Furthermore, familiarity with additional features such as the Density Spectral Array (DSA), Spectral Edge Frequency (SEF), and Burst Suppression Ratio (BSR) enhances the accuracy and clinical utility of pEEG interpretation. Mastery of these tools not only supports safer intraoperative management but also advances the broader goal of individualized, neuroprotective anesthesia care.

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