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Counting the waves – An introduction to the EEG and how processed EEG devices generate their values

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INTRODUCTION

This article reviews the basis of the electroencephalogram (EEG), and the principles of operation of commercial processed EEG devices. The goal is to provide an intuitive understanding of how these devices work and to clarify the technical jargon.

As with any clinical device, knowing how these monitors function is crucial, not just to appreciate what they measure, but also to recognise when they might be misleading. A clear grasp of their underlying principles helps to avoid incorrect assumptions that could impact clinical decisions, and supports a more critical appraisal of both research findings and manufacturer claims.

Rationale

Since the brain is the primary target of general anaesthetic agents, monitoring the EEG is an appealing concept. Individual dose requirements vary significantly, and balancing drug administration against surgical stimulation and patient illness is challenging. A reliable monitor of anaesthetic effect on the brain could help guide dosing, much as adjusting the vasopressor dose can be used to maintain a target blood pressure. The hope is that this could help reduce under-dosing, thereby minimising the risk of intraoperative awareness, while also minimising the deleterious effects of excess hypnotic drug administration.

Terminology

The term "depth of anaesthesia" monitor is both imprecise and misleading, contributing to confusion in both clinical practice and research.[1] It conflates the EEG effects of anaesthesia with broader systemic changes, and implies that these devices are reliable measures of effects beyond the brain. Expecting a monitor of the brain to provide a definitive measure of a multifaceted, body-wide phenomenon such as surgical anaesthesia is not just implausible, it is conceptually flawed. For such a measure to accurately reflect the state of other organ systems, a fixed relationship between them would be required. This is clearly not the case, as drugs affect different systems independently. At best, cortical electrical activity may reflect aspects of consciousness, but it cannot account for anaesthesia as a whole.

A comparison of propofol and sevoflurane illustrates this point. A propofol dose sufficient to induce 50% burst suppression (e.g. effect-site: 8–10 µg/mL) results in a Bispectral Index (BIS) in the low 20s. Achieving the same with sevoflurane requires concentrations exceeding 1.5 minimum alveolar concentration (MAC). Yet despite similar EEG patterns, the systemic effects differ markedly.

In both cases the patient is unconscious, yet with propofol alone, attempts at intubation will trigger movement and a pronounced adrenergic response, leading to hypertension and tachycardia. Surgery will not be possible due to continual reflex movements. In contrast, with 1.5 MAC sevoflurane there is profound

muscle relaxation, no movement on incision, and because it is MAC-BAR, there will be minimal change in heart rate and blood pressure. The BIS index may be the same, but the overall state of anaesthesia is markedly different.

These devices, therefore, do not measure anaesthesia *as such*; they primarily reflect the brain's surface electrical activity. For this reason, we will use the term "processed EEG" devices. This avoids overstating their capabilities while accurately describing their function: they process the EEG and generate index values.

Accidental awareness

The primary goal of general anaesthesia is to prevent conscious experience during surgery. Yet despite having highly effective drugs to achieve this, inadequate anaesthesia remains a real risk, with an incidence an order of magnitude higher than death from anaesthetic causes.[2,3] Its causes are multifactorial, and no single intervention will prevent it. Monitoring the EEG for signs of unconsciousness can play an important role as part of a wider strategy, but relying solely on a processed index would require it to be 100% reliable, which no index currently is.[4,5]

THE SCALP EEG AND HOW IT CHANGES WITH ANAESTHESIA

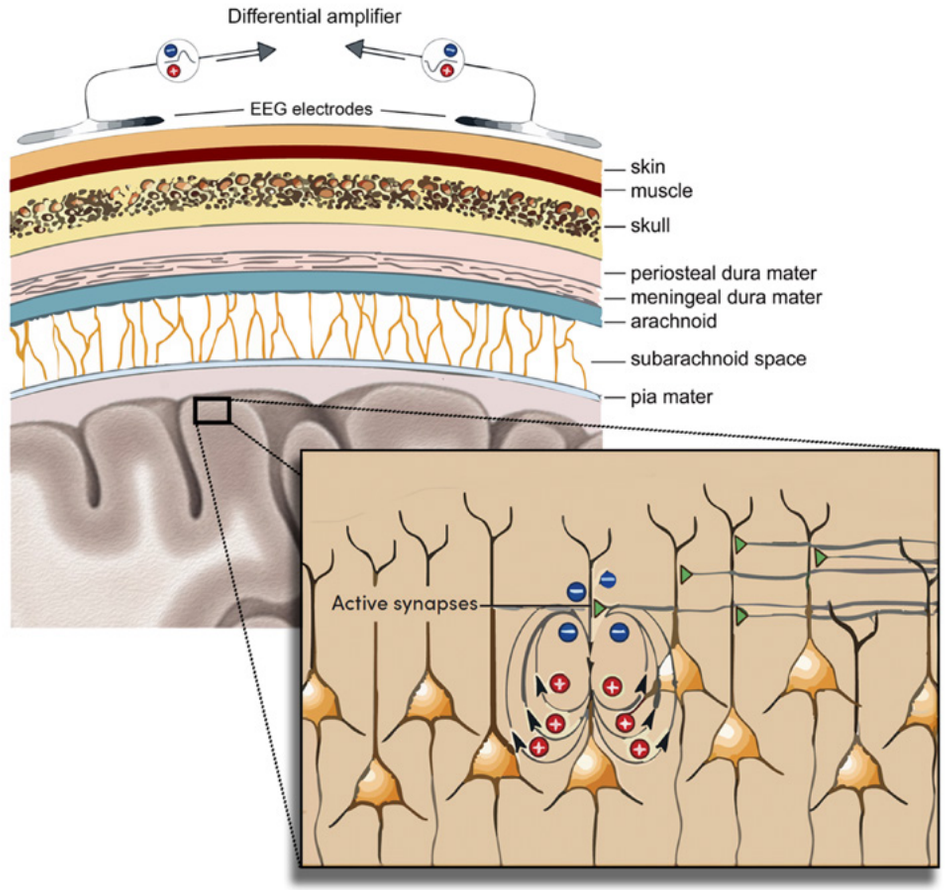
The scalp EEG = brain EEG + EMG + EOG

The "EEG" in routine practice refers to signals recorded from scalp electrodes, measuring voltage changes over time. Although "scalp EEG" would be more precise, the term "scalp" is omitted for convenience – since invasive intracortical recordings are seldom used outside research or special clinical settings. This omission can foster the misconception that "the EEG" reflects only brain activity. In reality, scalp electrodes also pick up signals from muscles (electromyogram, EMG) and eye movements (electrooculogram, EOG). This is especially relevant when considering the function of processed EEG monitors.

It might be assumed that cortical EEG signals arise directly from the depolarisation of individual neurons. However, because individual neurons are neither perfectly aligned nor synchronised, their electrical activity largely cancels out even a few millimetres away.

Instead, the EEG arises primarily from the summation of inhibitory and excitatory post-synaptic potentials. In particular regions of the cortex, large groups of neurons are arranged in columns that are innervated by ascending pyramidal axons. This columnar geometry means that the post-synaptic potentials add together in both time and space rather than cancel each other out. It is these post-synaptic potentials and their associated extracellular currents in the top few millimetres of the cortex that form the EEG (Figure 1).[6]

Figure 1. The origin of the electroencephalogram (EEG)



Post-synaptic potentials on pyramidal cells create electrical currents in the upper layers of the cortex, which are detectable at the scalp. The tissue layers between the brain and the skin decrease the voltage and spatially blur the signal, while muscle tissue contributes its own electrical activity.

Most EEG systems use at least three scalp electrodes (active, reference, and ground) for a single channel. A differential amplifier subtracts the reference signal from the active electrode, removing shared noise ("common-mode rejection"). Figure adapted from Siuly et al (2016).[6]

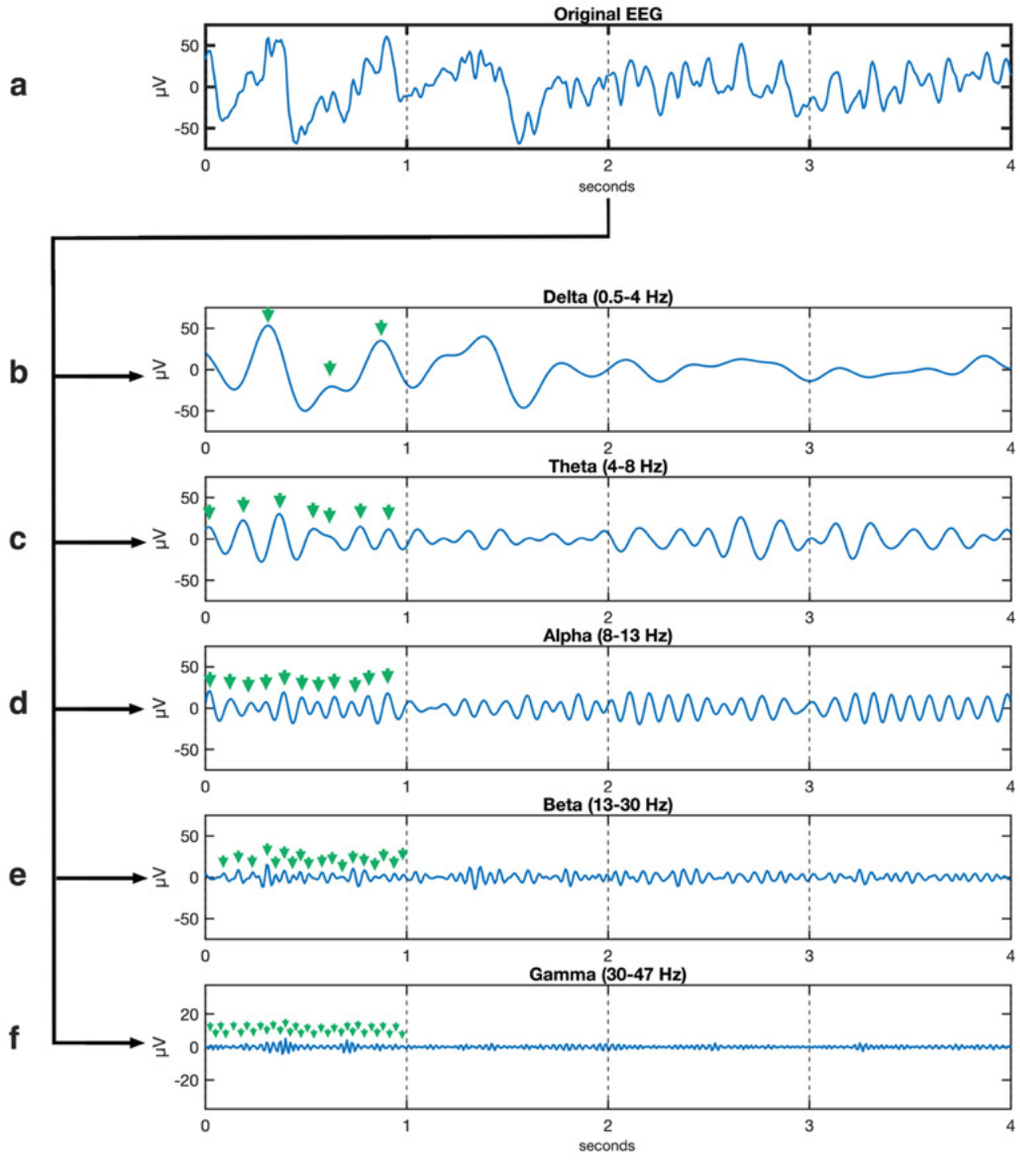
The signal that a single electrode detects arises from a few square centimetres of the top few millimetres of the surface of the brain. Deeper structures such as the thalamus and limbic system which are essential for consciousness, emotion, and the subjective experience of pain, do not produce measurable signals at the scalp directly, but they play a crucial role in shaping cortical EEG rhythms.[7]

Even when lying still with closed eyes, there is ongoing muscle tone which produces electrical activity (EMG). Although these muscles are small, they lie directly beneath the electrodes, so the signal from resting muscle can greatly exceed that of the brain, especially when the individual is awake.[8,9]

Features of the awake EEG

Traditionally, EEG activity has been classified into frequency bands based on empirical observations: delta (0.5–4 Hz), theta (4–8 Hz), alpha (8–13 Hz), beta (13–30 Hz), and gamma (30–80 Hz). Figure 2 illustrates how a typical EEG can be viewed via these frequency bands, which highlight the contribution of different components. These bands do not represent specific underlying physiology, but they are useful to describe broad changes in the EEG.

Figure 2. The raw EEG and its traditional frequency bands



a) Four seconds of raw EEG during sevoflurane anaesthesia; b–f) The same EEG filtered into conventional frequency bands. The x-axis represents time (seconds) and the y-axis voltage (μV).

The EEG always includes a mixture of frequency components, which can be seen more easily by filtering the EEG into bands of similar frequencies (b-f). Adding the filtered traces (b–f) together would reconstruct the original EEG (a).

Green arrows mark the wave peaks during the first second.

Note the large amplitude of the delta waves (up to ~60 μV), while alpha and beta are ~10–20 μV. Gamma waves are even smaller and are shown on a different scale. This is typical of the EEG – the lower frequencies have the highest amplitude, often described as greater "power." The gamma component is particularly small in this example because the patient is anaesthetised.

The Fourier transform takes this process a step further. Rather than filtering the waveform into just five frequency bands, it represents the original EEG as a sum of a large number of sine waves at frequency intervals of 0.5 or 1 Hz, each with a unique amplitude and phase (timing). See Figure 4.

In an awake individual, the EEG has a low amplitude (10–20 μV), with visible high-frequency components from muscle, which can give the waveform a "fuzzy" appearance.

When the eyes are closed, alpha waves become more prominent, a pattern referred to as the "posterior dominant rhythm" because it is maximal over the occipital cortex. Eye blinks produce large (100 μV), low-frequency deflections, which may appear as delta or theta waves. These arise from the potential difference between the cornea and retina as the eyeball rotates during blinking. Most of the high-frequency activity (>20 Hz) in the awake EEG arises from resting muscle tone.[8]

EEG changes due to anaesthesia

There are characteristic changes that occur in the EEG when anaesthetic drugs such as propofol or volatile agents are administered, and it is these changes that processed EEG devices rely on to generate their values (Figure 3). The following is a simplified account of typical changes, which omits many subtleties, drug-specific effects, and inter-individual variation. Still, it is sufficient to enable a basic understanding of how these devices work. There are several excellent review articles which offer a more detailed coverage.[4,10-12]

Broadly speaking, when propofol or volatile agents are administered, there is an increase in the power of low-frequency waves (<30 Hz) and a relative decrease in the power at higher frequencies. Most of the decrease in the higher frequencies is from loss of muscle tone, which occurs even without the use of muscle relaxants.

Once unconscious, a characteristic feature at lower hypnotic drug doses is the "spindle": a 1–2 second period of 8–14 Hz activity, which increases and then decreases in amplitude, giving it a distinctive shape. Spindles are associated with thalamic hyperpolarisation and are similar to those seen in natural sleep.

With a greater hypnotic dose, large "slow waves" become prominent (20–100 μV, 0.5–4 Hz). These are often accompanied by smaller alpha waves, giving rise to the classic "alpha-delta" description of the anaesthetised EEG. A notable feature is "phase-amplitude coupling", in which small alpha-band oscillations can be seen superimposed on the peaks or troughs of larger slow waves.[13] Painful stimuli may induce one or more of several EEG changes: increased beta activity, loss of alpha waves ("alpha dropout"), or even increased delta waves ("delta arousal"). These changes can be reversed by the administration of opioids.[14,15]

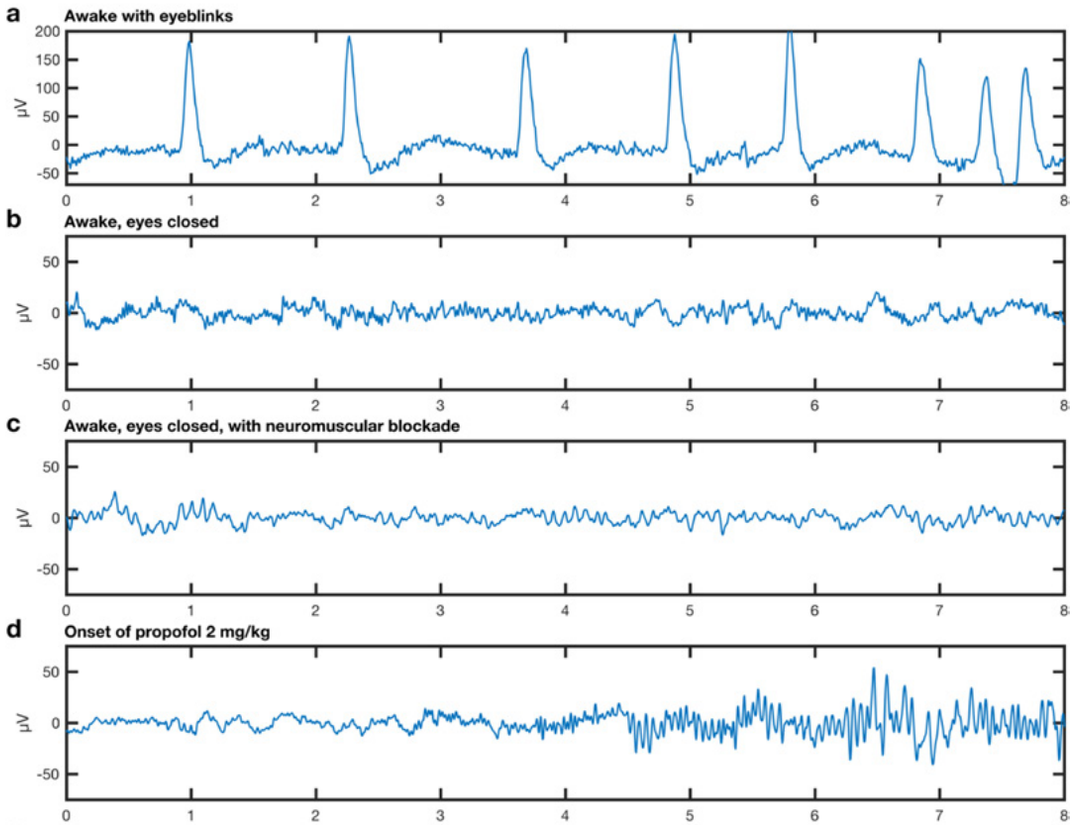
At even higher doses, "burst suppression" is induced. This is a pattern of brief periods of electrical silence ("suppression"), punctuated by large-amplitude flurries of EEG activity ("bursts"). Burst suppression does not occur during normal sleep. It can be quantified as the "suppression ratio" – the percentage of time that the EEG is suppressed over the preceding 30 or 60 seconds. When given as a sole agent in young adults, burst suppression starts to appear at propofol levels of ~5 mcg/mL and sevoflurane of ~1.5 MAC. In the elderly, or when used with other agents, it can occur at much lower concentrations. There is usually a profound reduction in brain metabolic activity when burst suppression is induced by anaesthesia, but it is not typically a desirable EEG state except in specific clinical circumstances.[4]

The relationship between these EEG changes and the level of consciousness is not precise, and the extent to which specific features appear varies between patients. EEG changes can be related to the dose of the drug ("drug-related"), or to the state of consciousness of the patient ("state-related"). Some elderly patients do not generate large slow waves, but transition to burst suppression with just a slight increase in drug dose.[16–18] Emergence from anaesthesia is not the opposite of induction, and can display a number of different patterns.[19]

Finally, this sequence of EEG changes occurs only with specific drugs. Agents such as ketamine and nitrous oxide produce different EEG patterns for the same level of responsiveness.[4,11] Ketamine can increase beta and gamma power, and reduce alpha power. In high doses, it can cause large delta waves.[4,20] Nitrous oxide has varied effects depending on dose, often increasing beta and gamma power or reducing alpha power. Large slow waves may appear during wash-in and even wash-out.[4,21,22] Neither agent causes burst suppression when given alone. Because processed EEG devices are not designed to track the sequence of EEG changes that these drugs induce, their values can be misleading when nitrous oxide or ketamine are used.

These drug-specific differences illustrate a fundamental limitation: the indices correspond to changes in cortical electrical activity, rather than a true physiological endpoint such as "level of consciousness."

Figure 3. The effect of anaesthetic drugs on the EEG

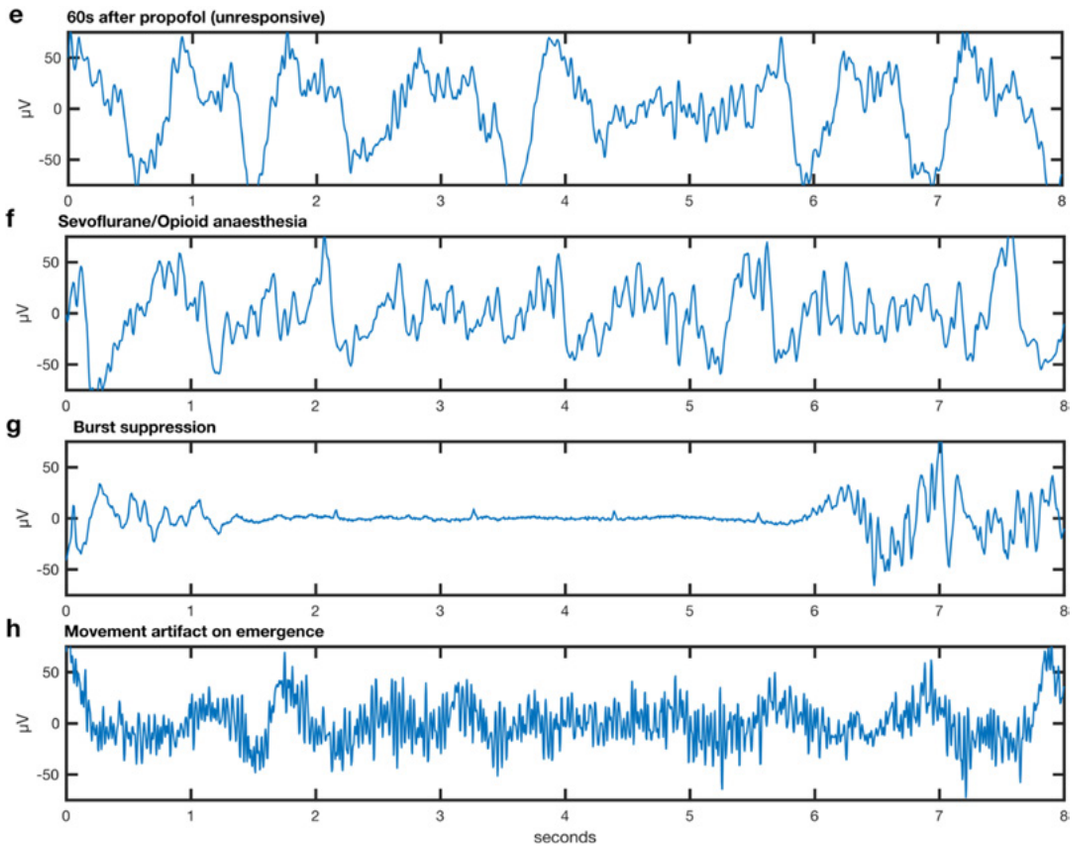


a) Awake patient with eyeblinks. The large eyeblink deflections frequently exceed the 100 μV display range. Between blinks the awake EEG resembles Panel B.

b) Awake patient with eyes closed. The typical high-frequency, low-amplitude pattern in an awake individual. The trace appears "fuzzy" in the first few seconds due to high-frequency gamma waves from muscle activity oscillating too rapidly to resolve.

c) Awake patient with neuromuscular blockade. The fuzziness from the gamma waves of EMG is absent, but the EEG is otherwise unchanged. Alpha waves are seen at second one and four.

d) Ten seconds after a bolus of 2 mg/kg propofol. At the third second, the moment of propofol onset, there is a sudden increase in alpha and beta activity; this increased beta activity is a transient phenomenon and can occur with sedation-level doses as well.



e) Sixty seconds after propofol. The EEG has "slowed" and now contains large delta waves with some superimposed alpha. Other frequencies are still present, but they can be difficult to see without a spectrogram.

f) Established sevoflurane/opioid anaesthesia. This shows the typical large amplitude alpha-delta pattern of anaesthesia.

g) Burst suppression. Four seconds show "suppressed" EEG, followed by a "burst" giving a "suppression ratio" of 50%. Small QRS complexes (ECG artefact) are visible during the suppressed period. QRS complexes are usually obscured by EEG activity and are most apparent during suppression. It is more commonly seen in patients with left ventricular hypertrophy, thick necks, and with left-sided electrode placement.

h) Muscle artefact from patient grimacing during emergence. Many vertical spikes are seen, typical of muscle twitches.

EEGs are eight seconds' duration, and were recorded with frontal bipolar electrodes from a BIS monitor. For more detailed discussion of the nuances of EEG changes during anaesthesia, see the excellent review articles by Sleigh,[4] Hight,[11] or Purdon.[22]

HOW PROCESSED EEG DEVICES GENERATE THEIR INDEX VALUES

This section describes the key principles of index generation, while avoiding undue technical detail. The goal is to establish a conceptual framework for understanding how the index values are generated rather than offer an exhaustive discussion of every device. We will concentrate on the BIS algorithm, because that is the most well-described and it serves as an excellent guide to understanding the principles of operation of other devices. The physical components of typical devices is summarised in Table 1.

Preliminary analysis

The very first step is to convert the tiny voltages detected at the scalp into numbers that can be analysed. This process, called analogue-to-digital conversion, also includes filtering to remove frequencies outside the range of interest (typically below ~0.5 Hz and above ~128 Hz). The signal is sampled about 256 times per second to create a stream of numbers suitable for automated computer analysis. The analysis itself begins by mathematically processing the raw EEG to determine its frequency composition. This is sometimes referred to as "converting the EEG waveform from the 'time domain' (how voltage changes over time) to the 'frequency domain' (how much of each frequency is present during a particular period)."

Epochs and artefact

The EEG is analysed stepwise, breaking it into small, overlapping segments called "epochs", usually around two seconds long. Epochs which contain unwanted signals, such as from diathermy, vibration, patient movement, or eye blinks, are classified as "artefact" and are discarded.

Accurate artefact detection – separating noise from meaningful data – is crucial, but not all interference is easily recognised. Signals that mimic EEG or which lack typical artefact signatures can evade detection, and because the device calculates its index from power ratios across multiple frequency bands, artefact at key frequencies can significantly distort results. This is why electrical noise from an operating table transformer or the fluttering of a forced-air warmer can alter index values.[24,25] Displaying the EEG waveform is therefore essential, to verify that the monitor is analysing a valid signal (Table 2). Manufacturers' default screen settings vary greatly, so it is important to check that the waveform is being presented with a familiar scale and sweep time.

The Fourier transform

In the previous section, we saw how the raw EEG can be divided into traditional frequency bands. The Fourier transform takes this a step further by representing the EEG during each epoch by a set of sine waves of different frequencies, typically spaced every 0.5 to 1 Hz. In addition to the frequency, each sine wave has an amplitude (size), and a phase (timing relative to other waves). If all of these sine waves were added together, the original waveform would be reconstructed.

A key concept is the "power" of each wave, which quantifies how much of that frequency is present in the EEG. Mathematically, power is proportional to the square of the wave's amplitude – twice the amplitude means four times the power. This is similar to ocean waves: a wave that is 10 times higher carries 100 times more energy. In EEG terms, when slow waves are large, we say there is a high level of "slow wave power." This is sometimes referred to as "slowing" of the EEG, but the EEG has not really "slowed down", it just has more low frequency waves and less high frequency waves.

To improve accuracy, results from a single two-second epoch are usually averaged over the preceding 20-30 seconds. The power at each frequency can now be plotted as a graph – the power spectrum – which shows how important each frequency is in shaping the original EEG (Figure 4b). By adding up the power of all the sine waves in a certain frequency range, the "total power" within that frequency band (the "band power") can be calculated. These bandpowers are the basis of most automated EEG analysis.

While few devices display the power spectrum, some newer models use this information to generate a "spectrogram". A sequence of spectra is plotted over time, with power represented usually by colour, but occasionally by height. In this form, it becomes easy to visualise how power in different frequency bands changes throughout the anaesthetic.[4] The spectrogram reveals patterns of brain activity that are lost when the EEG is reduced to a single numerical index. Spectrograms can vary significantly in appearance between manufacturers and between patients, partly due to technical differences in how power is mapped to colour, and partly due to individual differences in overall EEG power (Figure 4d).

Index calculation

The design of a useful processed index requires selecting features in the power spectrum that change with anaesthetic drug dosing in a way that aligns with changes in responsiveness. In a sense, the index is an estimate of how far the current EEG is along the sequence of waveforms: from the awake pattern, through slow waves, to burst-suppression (Figure 3).

All currently available monitors follow a similar approach: they calculate power within various frequency bands, derive ratios to quantify changes in the power spectrum, and then combine and scale these values to produce a final index.

The final scaling to a 0–100 range is based on calibration studies in which EEG recordings are aligned with patient responsiveness. Like any population-based model, the monitor only approximates the population average, and its accuracy depends on the quality of the validation studies and the specific drugs tested.

Importantly, the calibration studies from awake to loss of consciousness have typically been done without using neuromuscular blocking drugs.[5,26,27] This has obvious implications for whether these devices might reliably be used in paralysed patients.

Unlike physiological monitors such as pulse oximeters, which follow a clear chain from a physical principle, through signal processing, to a number with a clearly defined meaning, processed EEG indices are not direct measures of a defined physical quantity. Rather, they are real-time implementations of statistical models, which evaluate a set of summary descriptors of the EEG power spectrum and then combine them in a way that was found, experimentally, to best fit the original training population.

How the Bispectral Index (BIS) is calculated

BIS is the best-described of the processed EEG indices mainly because of Connor's reverse-engineering of the algorithm.[28,29] Examining its calculation provides insight into the type of analysis these devices use to generate their values.

The core components

Having digitised the electrode voltages, and discarded epochs which contain unwanted noise, analysis begins by calculating the power spectrum average over the last 30-60 seconds (Figure 4). From this it derives three band-power ratios.

Beta Ratio: the logarithm of power in the 30–47 Hz band relative to the 11–20 Hz band. The name is slightly misleading, it is really gamma (>30 Hz) relative to low beta (11–20 Hz), but we retain the conventional term. When awake, most of the power >30Hz is from muscle, and because muscle tone decreases with sedation and anaesthesia, power in the gamma range falls. At the same time, 11–20 Hz power rises, so the fall is even greater in relative terms. The Beta Ratio therefore decreases during sedation and induction of anaesthesia.

Normalised Gamma: the logarithm of power in the 40–47 Hz band relative to total EEG power (0.5–47 Hz). This is gamma power as a proportion of total EEG power. Like the Beta Ratio, it decreases with induction, because 40–47 Hz activity decreases and slower activity increases. However, unlike Beta Ratio, it is also affected by delta and theta power. Increases in these will reduce the Normalised Gamma because they add to the denominator. Therefore, Normalised Gamma is more responsive than the Beta Ratio during stages of anaesthesia when slow waves are prominent.

Delta Ratio: the logarithm of power in the 0.5–4 Hz band relative to the 11–20 Hz band. This is the mirror image of the Beta Ratio: instead of quantifying the proportional fall in higher activity, it quantifies the proportional rise in slow activity. Delta Ratio is used to determine how the two previously mentioned ratios are combined.

Rescaling and compression

The raw band-power ratios have values around -60 to 0 dB. To align them with the BIS scale, the algorithm applies non-linear transformations. These not only rescale the values, but compress them in some regions while expanding them in. The Beta Ratio is remapped to approximately 55-104, and the Normalised Gamma Ratio to about 20-105.

The rescaled ratios are then merged into a single "provisional" index. When the Beta Ratio is lower than the Normalised Gamma, the Beta Ratio is used. When the Normalised Gamma is lower, then Delta Ratio determines how the two measures are combined. If delta power is high, Normalised Gamma predominates; otherwise the Beta Ratio has greater influence. The combined value is then compressed to fit the 0-100 scale.

The effect of burst suppression

The final modification comes from burst suppression. BIS calculates the "suppression ratio" (SR): the percentage of time the EEG is suppressed during the preceding 63 seconds. When SR is between 10-50%, the provisional index is decreased slightly, producing values between 20-40. When SR exceeds 50%, the EEG band-power ratios no longer have any effect, and the index is determined solely by the equation:

BIS = 50 – (SR/2)

This generates a BIS of zero when the EEG is fully suppressed.[40,41]

The full BIS algorithm is more elaborate than the simplified account given here, but this description illustrates the main principles. The full algorithm calculates more than ten different band-power ratios and incorporates several time-dependent processes when blending them. In some cases, features from the preceding four minutes are used to decide which ratio to apply, while segments from up to 20 minutes earlier alter the threshold for burst suppression detection.

Relation of bandpowers to consciousness

It is clear from the above that BIS is far from a simple or transparent calculation. Rather than having a clear logic, the index is assembled from a sequence of physiologically-unmotivated adjustments of EEG band-power ratios.

At which point, it is reasonable to ask: *"What do all these ratios have to do with consciousness?"* A fair answer might be, "In a direct sense, not much." They are not derived from any model of the brain, or of consciousness, but are *proxies* – summaries of EEG changes that are seen experimentally during anaesthesia.

It is important to appreciate that a bandpower measure like Beta Ratio is not *in itself* a measure of consciousness. It can track the drug effect of propofol and sevoflurane only because these drugs change both consciousness and Beta Ratio at the same time. Neuromuscular blockade has no effect on consciousness but it does reduce Beta Ratio, showing that it is not a direct measure of cognitive state. This distinction is subtle but central, and it underlies the assumptions on which EEG indices are built.

The method used to blend the band-power ratios was chosen by selecting whichever gave the greatest apparent accuracy for the drugs and patients in the original development population. BIS is therefore best understood not as a mechanistic or personalised measure of cognitive state, but as a group-level "best fit."

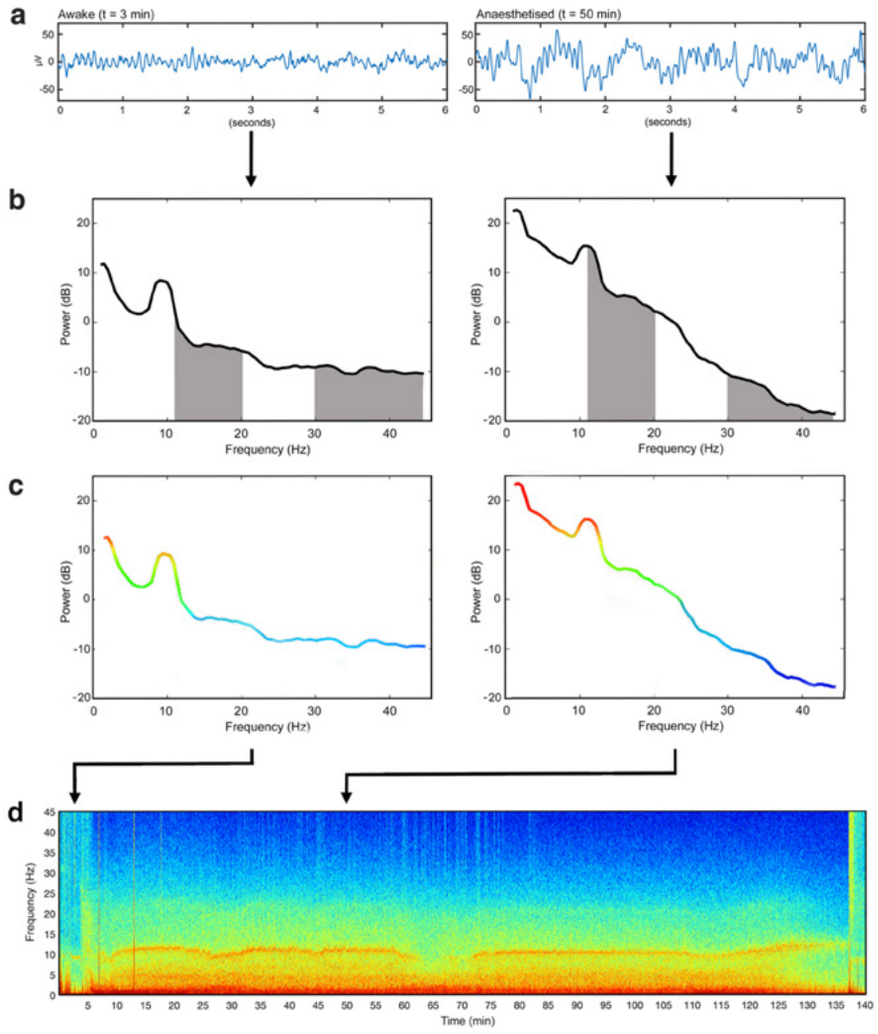
The limitation is not only statistical but also anatomical. Indices like BIS are derived entirely from frontal EEG, yet it is doubtful that consciousness resides in the frontal cortex. What these devices are really doing is evaluating is a set of drug-induced EEG changes in one brain region, under the assumption that these map in a reasonable way onto the neural processes that matter for awareness. That assumption may hold in broad terms, but it is indirect, drug-dependent, and incomplete.

The apparent precision of the final index, carefully sculpted to a 0–100 scale, conceals the blending of different measures, the statistical basis of its design, and the anatomical limitation. Recognising this is key to interpreting BIS intelligently and resisting the misplaced confidence that its scale can invite. The same logic applies to other processed EEG indices, which differ in detail but share the same empirical foundations and the same vulnerabilities.

Where is the "bispectral analysis"?

A follow-up question may arise: why has "bispectral analysis," from which the BIS index takes its name, not been discussed? Early studies suggested there was little advantage in computing the bispectrum,[31] and the recent reverse-engineering revealed that as soon as BIS calculates the Fourier transform, it discards the phase information required for bispectral analysis. It appears that there is no bispectral analysis in the "bispectral index" monitor.[32]

Figure 4. The BetaRatio before and after induction of anaesthesia



a) Two segments of EEG from before induction, and during sevoflurane anaesthesia; b) Power spectra at these two points, calculated over 20 seconds. Shaded areas indicate the frequencies used to calculate the BetaRatio; c) The power spectrum coloured to indicate power; d) The colour spectrogram for the entire anaesthetic, with the two timepoints indicated.

Before induction (a, left), the raw EEG is low amplitude with alpha waves. During anaesthesia (a, right), it has larger amplitude with prominent delta and alpha waves. The power spectrum (b) shows these changes more clearly, and demonstrates increased power at all frequencies below ~30 Hz, and decreased power above. The alpha rhythm is visible as a peak in the power spectrum, at ~9 Hz when awake, and 11 Hz during anaesthesia.

The shaded areas demonstrate how the BetaRatio will change due to anaesthesia. During anaesthesia, there is less power in the upper band, and more in the lower band, and so the BetaRatio decreases.

Panel c) shows the spectrum with colour used to indicate power. Blue is low power, red is high power, and yellow and green intermediate. The final spectrogram d) uses the data from a large number of successive power spectra remapped to colour, with time on the x-axis, and frequency on the y-axis. Anaesthesia was induced at minute 4. The increased delta and alpha power are shown by the red bands. Note the loss of alpha oscillations at minute 62. This "alpha dropout" occurred just before the patient became tachycardic and hypertensive. Opioids were administered, and a few minutes later the alpha rhythm returned.

Other processed indices: Entropy, Conox, SedLine

Other devices operate on similar principles, in that they evaluate the power in different frequency bands, then combine them and scale the results. While manufacturers emphasise unique features in their descriptions, these differences largely reflect variations in how the band-powers are combined, rather than being fundamentally distinct analytical methods.

For example, the GE Entropy monitor uses “spectral entropy” rather than “power” in different frequency bands, to generate two related indices: State Entropy (SE) and Response Entropy (RE), using frequency ranges of 0.8–32 Hz and 0.8–47 Hz respectively.[33,34] Despite common misconceptions, "spectral entropy" does not measure the “randomness” of the EEG but rather how evenly power is distributed across frequencies. Essentially, it is another way to quantify shifts in the power spectrum.[35,36] The developers proposed that RE would be more sensitive to EMG, while SE remained unaffected but in practice EMG affects both indices.[9,37]

The Conox monitor analyses power in six frequency bands across a similar range (1–44 Hz) and combines the results to generate an index of the "state of consciousness" (qCON) and a second index (qNOX) intended to indicate the "likelihood of response" to nociceptive stimuli.[26]

SedLine, originally marketed in 2001 by Physiometrix as the Patient State Index (PSI), and now integrated into Masimo’s Root device, takes a similar approach. It uses bifrontal electrodes and primarily relies on band-power analysis, although it claims to also evaluate the degree of EEG similarity on either side of the head.[38,39]

In all devices, burst suppression is calculated over the preceding 30-60 seconds, and, similarly to BIS, is used to decrease the index further towards zero.[33,40,41]

Table 3 lists features of devices available in Australia and New Zealand.

Spectral edge frequency (SEF95)

The SEF95 is an indication of the upper “edge” of the spectrum, defined as the frequency below which 95% of the total EEG power lies. Awake values are typically 20–25 Hz, and decrease during anaesthesia to ~8–14 Hz.[42] The SEF95 provides only a glimpse of the raw EEG data, and is sensitive to artefacts such as eye blinks or movement. It predates modern processed EEG devices, and while there has been a mild resurgence of interest in recent years, this appears to be driven primarily by marketing.[4,29]

Other displayed variables – EMG, SQI

Each manufacturer estimates EMG slightly differently, but the general principle is to assess the power in higher frequencies where EEG activity is minimal. BIS calculates EMG from power in the 70–100 Hz range over the preceding 10 seconds, displaying it as a bar graphic – an appropriate approach given the imprecise nature of the data. Conox evaluates power in the 30–42 Hz range over 30–60 seconds, displaying it on a 0–100 scale.[43] SedLine does not describe its method but it also displays EMG on a 0–100 scale. EMG values between devices are, therefore, not comparable.

Some monitors display a "Signal Quality Index" (SQI) or an "Artefact Index", which reflects a combination of electrode impedance and the proportion of artefact-affected epochs over the last ~60 seconds. While these values do not indicate the reliability of the index itself, most monitors stop displaying values when SQI falls below a certain threshold.[43,44]

LIMITATIONS OF COMMERCIAL PROCESSED EEG INDICES

There is no doubt that these indices generally decrease during induction of anaesthesia, and come back up again at emergence, but an appreciation of the role of any monitor requires an understanding of its weaknesses and when it may be misleading.

The problem of EMG

The electrical signal from muscle (primarily the forehead muscles) spans a broad frequency range, from below 1 Hz to over 200 Hz. This completely overlaps the EEG signal, making separation difficult. EMG is particularly prominent in frontal electrodes. In an awake individual, more than 90% of the power over 30 Hz originates from muscle rather than brain.[8,9]

EMG activity decreases in a dose-dependent manner in response to anaesthesia, even in the absence of neuromuscular blockade. During balanced anaesthesia, it is typically minimal or absent. As a result, EMG serves as a reasonable, although indirect, marker of anaesthetic effect. Processed EEG devices appear to rely on this relationship and incorporate EMG into their algorithms by using frequency bands in which muscle activity predominates.[5,26,27,33]

This introduces an obvious limitation: such systems will be prone to failure when neuromuscular blocking agents are used. This is why BIS and Entropy values drop to low levels in awake individuals given neuromuscular blocking drugs, despite the EEG clearly indicating they are awake.[9,45,46] The same effect has now also been demonstrated with Conox.[47] Other devices which rely on frequency bands in which EMG is predominant are likely to share this limitation.

The irony here is striking: the very situation in which processed EEG indices could be most useful – detecting awareness in the muscle-relaxed patient – is precisely where they may be least reliable. Of course, by the time the patient is fully awake, the horse has bolted. Ideally, an EEG monitor would identify inadequate anaesthesia early enough to alert the anaesthetist before that point is reached.

The problem of precision

Processed EEG monitors use a 0–100 scale, implying a high level of precision. However, studies consistently show wide variation in index values at the same level of patient responsiveness, with substantial overlap between responsive and unresponsive states.[5,48–52] The original BIS calibration study demonstrated some patients were non-responsive to voice at BIS 75, while others were still responsive at 51.[5]

This is not simply a reflection of the inter-patient drug variability that anaesthetists encounter daily. These indices purportedly represent a physiological endpoint, ostensibly helping to manage pharmacodynamic variability. Yet, if they were truly measuring a physiological state, we wouldn’t expect index values from different levels of consciousness to overlap. By analogy, it would be as if a blood pressure monitor showed significantly different readings in different patients despite their actual blood pressure being the same.

Another issue is the internal inconsistency of these devices. Identical BIS or Entropy monitors used simultaneously on the one patient can produce substantially different index values.[53,54] Entropy monitors have even been shown to generate different values when repeatedly presented with the exact same EEG.[9] If two identical monitors can differ by more than 10 or 20 points in the same patient, clearly a 0-100 scale grossly misrepresents the device’s accuracy, and suggests that the scale itself may owe more to marketing considerations than physiological reality.

Finally, different devices produce discordant results. When EEG recordings indicating inadequate anaesthesia were replayed to five different devices, clinically significant disagreements occurred in 70% of cases.[55] It seems perverse that the anaesthetic dose that a patient receives could ultimately depend on which monitor that particular hospital has purchased, a decision shaped partly by financial and marketing considerations, rather than by patient physiology.

The problem of the reference range

BIS values between 40 and 60 are often regarded as a definitive range for ensuring unconsciousness. However, not only is there a lack of strong evidence to support this range, but there is good evidence against it.

In the original BIS calibration study, approximately 15% of participants receiving low-dose infusions of propofol, midazolam, or isoflurane, were still responsive to voice at BIS values between 50 and 60.[5,56] The authors themselves stated that “BIS levels less than 50 indicate that a patient is *probably* unconscious.” This has been confirmed independently.[57]

Given that some conscious patients had BIS values below 60 in the very study used to calibrate the algorithm, it was inevitable that this would also occur in surgical patients, and indeed, it did. A few years later, the B-Aware study reported a patient who experienced full awareness with recall, despite a BIS value that never rose above 55. This prompted the authors to suggest that 55 might be a safer upper limit than 60.[58] Several large trials and case reports also documented intraoperative awareness with BIS values below

60.[59] The B-Unaware trial reported three cases, and the BAG-RECALL trial reported five.[60,61]

Although it may be true that most patients with adequate anaesthesia have a BIS between 40 and 60, it does not follow that a BIS between 40 and 60 means they are adequately anaesthetised. "All dogs have four legs. This has four legs, therefore..."

Other manufacturers have published little or no data to support their suggested reference ranges. None have claimed to calibrate their indices in paralysed patients. As a result, we do not know how reliable these ranges are in different anaesthetic scenarios – particularly those involving neuromuscular blockade. We are left with devices that are widely used, but poorly validated, and regulated to standards far below those applied to the drugs they are meant to guide.

The problem of generalisability

Most commercial processed EEG monitoring indices were calibrated using studies conducted on young, healthy volunteers, meaning their performance is largely unvalidated in older or medically complex patients.[62] In clinical settings, variability in anaesthetic regimens, pre-existing medical conditions, fluctuating surgical stimulation, and physiological perturbations further challenge their reliability.

A particularly vulnerable population is the elderly, where reducing anaesthetic dose may be desirable to minimise side effects on other organ systems. Ageing is associated with a number of neurophysiological changes, yet processed EEG algorithms do not account for age. This can lead to higher index values despite older patients being more sensitive to anaesthetic drugs.[16,18]

The problem of lacking a sound neurobiological foundation

The lack of a solid neurobiological foundation is possibly the root cause of many deficiencies in processed EEG devices. Consciousness itself is poorly understood, as are the mechanisms by which anaesthetics induce unconsciousness.[63,64] This is why devices rely on broad spectral changes and use frequency bands in which muscle activity predominates.

A fundamental challenge is that EEG changes can arise both directly from the drug itself ("drug-related" changes) and from the altered level of consciousness that the drug produces ("state-related" changes). Should an index primarily reflect drug-related changes (and so predict dose), or should it track state-related changes (predicting level of consciousness)? Some studies seem to assume that both can be achieved simultaneously, which overlooks pharmacodynamic variability. A stronger neurobiological foundation may help disentangle these influences, but even with greater understanding, they may remain intertwined.[65] Addressing this issue is essential for improving the reliability and clinical utility of EEG-derived indices.

THE ROLE OF PROCESSED EEG MONITORING: ENHANCING SAFETY WHILE RECOGNISING LIMITATIONS

The question is, are these devices so inaccurate that they should not be used at all? While their lack of reliability is a legitimate concern, they still serve an important role when used appropriately.

Broadly speaking, processed EEG monitors can be used in two distinct ways: as an alarm to identify potential problems or as a guide to dose titration. These two approaches have very different implications for clinical decision-making.

As an alarm

In this approach, anaesthesia is conducted using established dosing regimens that include a safety margin to account for patient variability. If the index shows an unexplained trend or falls outside expected limits, this should prompt checks of drug delivery systems and equipment, with values verified by looking at the raw EEG or its spectral display. Used in this way, processed EEG can provide an additional layer of safety, notwithstanding its inaccuracies.

The history of anaesthesia has demonstrated the benefits of engineering human error out of the system and introducing fail-safes and alarms wherever possible. Total intravenous anaesthesia, however, bypasses many of these safeguards, and processed EEG monitoring can help restore some of that protection, despite its limitations.[2,66–68] Even an alarm that works only part of the time may be preferable to no alarm at all.

As a guide to dose titration

Reducing anaesthetic doses to low levels based solely on processed index values is not a safe approach. The inaccuracies discussed earlier mean that at the same index value, some patients may be anaesthetised while others are not, even at BIS values less than 60. That said, very low index values are often a result of burst suppression, suggesting that the dose could be safely reduced. Again, examining the raw EEG or the spectrogram will help distinguish between situations in which the index is accurate or misleading.

Diagnosing the index

In both cases, it is important to display the raw EEG, to determine whether the signal is suitable for automated analysis, or has been distorted by artefact. Just as cardiovascular monitoring is not based solely on a numerical heart rate, and end-tidal CO_2 is not interpreted without examining the waveform, anaesthetic management should not rely purely on a processed index derived from an obscure algorithm.

CONCLUSION

Processed EEG monitors have made brain monitoring a routine part of anaesthetic practice. However, their index values oversimplify the complex interplay between patient physiology, anaesthetic regimen, and the risk of awareness.

Their indices are derived from evaluating broad spectral changes and therefore serve only as indirect proxies for consciousness. Reference ranges lack supporting evidence, and no device has been calibrated for use in paralysed patients. They are essentially statistical constructs, calibrated to fit their development population, and so although they may correlate with responsiveness at a group level, they can be misleading in individual patients. Finally, the indices are vulnerable to artefact and other technical or physiological interference.

For all these reasons, processed EEG monitors should not be used as the primary guide to anaesthetic dosing. In their current form, they are most useful when interpreted alongside a sound understanding of the raw EEG waveform and spectrogram. Anaesthetists should be familiar with the typical EEG effects of commonly used agents and remain aware of the limitations of processed indices.

APPENDICES

Table 1. Physical components of processed EEG devices

Component	Function	Comments
Electrodes	Electrodes convert electrical potentials on the skin into electronic currents within the device, enabling measurement and analysis of underlying ionic currents.	Most processed EEG devices use single or dual-channel frontal arrays with metal/gel electrodes. Low electrode-skin impedance is critical, ensured by design features like Ag/AgCl electrodes, conductive gels, or "tine pads" that slightly penetrate the stratum corneum to reduce impedance. Proper application, including skin preparation, pressure and consistent technique will minimise the impedance.
Amplifiers	Differential amplifiers in processed EEG devices magnify very small voltages (10-50 μV) for processing.	Differential amplifiers magnify the voltage difference between two electrodes while suppressing signals common to both, a process known as "common mode rejection". This selective amplification helps reduce interference from external electrical sources, such as those present in an operating theatre.
Computing device	The computing device performs analogue-to-digital conversion (ADC) of the waveform, digital filtering, artefact rejection and further processing and analysis. Some devices allow uploading of the EEG and processed indices for later analysis.	An analogue-to-digital converter (ADC) converts the continuous EEG signal to discrete digital values many times per second, enabling further digital processing. Digital filtering and artefact rejection help reduce effects of noise. However, excessive filtering can unintentionally remove physiologically relevant frequencies, potentially distorting the EEG.
Monitor	Display the various outputs of the system, allowing users to evaluate the patient data.	Most processed EEG systems display a combination of the following: raw EEG, processed indices, and a colour spectrogram.

Information sourced from Rampil (1998)[29]

Table 2. External factors affecting processed EEG values

Source of interference	Appearance in EEG waveform	Effect on processed index	Mechanism of effect
Physiological			
Blink artefact	High amplitude (~100-200 μV) deflections with each blink.	Contributes to low frequency band power (delta or theta bands).	Cornea is electropositive relative to the retina. As the eye blinks, the globe turns slightly upwards, resulting in deflections in the EEG waveform.
Muscle activity (most commonly frontalis/ temporalis muscles)	High-frequency and relatively high-amplitude activity (amplitude correlates with strength of muscle contraction). May appear as a "fuzzy" waveform, or as short vertical "spikes."	Contributes to higher frequency band power (typically beta and gamma). Index likely to be elevated when present, and reduced when absent.	High frequencies are used by devices to generate high index values. Therefore increased muscle tone during anaesthesia can increase index values. Conversely, loss of EMG can cause falsely low index values.
ECG artefact	Small QRS-like complexes, occurring regularly with the heart beat.	May increase or decrease processed index values, depending on the size and relative contribution to different frequency bands.	More pronounced in patients with LVH, and thick necks, which enable conduction of the ECG voltage.
Ventilation	Rhythmic, very slow gentle waves synchronised with ventilation pattern; often difficult to appreciate on the raw waveform unless viewing at a lower reading speed.	Contributes to lower frequency band power, theoretically may lower index value.	Overlap between frequency components of ventilation with low frequency bands.
Non-physiological (external)			
Electrode "pop"	Sharp steep, high-amplitude upstroke followed by downslope before return to baseline.	Often triggers artefact rejection as amplitude range typically exceeds that of the system.	Mechanical movement of electrodes and/ or associated cables produce distortions in scalp-electrode impedance.
Electrical interference (e.g. operating table transformer)	Bizarre-appearing waveform.	Can cause high or low index values.	Induced currents can generate frequencies in the signal that alter the frequency ratios but do not trigger artefact-rejection algorithms.
Forced-air warmer	High-frequency activity.	Inappropriately high BIS values although a low-frequency flutter may cause low values.	Mechanical vibrations of processed EEG system cables caused by air circulation.
Power supply	High-frequency (50 Hz) and continuous activity makes waveform appear "fuzzy". Most systems have a specific filter to remove this from the waveform.	Power spectra affected by a large spike at 50 Hz.	Poor electrical shielding, poor electrode contact, or lack of appropriate filtering.
Surgical diathermy	High-frequency, high-amplitude interference rendering the underlying EEG wave difficult to discern.	Increased BIS value during electrocautery if not excluded by artefact rejection.	Large amplitude contamination of EEG signal, from induced current and muscle stimulation. Usually results in rejection of epochs, but low levels may not trigger artefact-rejection.

Information sourced from Bennett et al. (2007)10 and Rampil (1998)[29]

Table 3. Summary details of processed EEG devices available in Australia and New Zealand

Monitor	Data and display features	Index ranges, manufacturer recommended target range*
Bispectral index	Raw EEG BIS EMG Suppression ratio Signal quality index Colour Spectrogram, SEF95	 0-100, Target 40-60 Bar graphic 0-100 0-100 Recent models only
Conox	Raw EEG qCON index qNOX index EMG Signal quality index Suppression ratio	 0-99, Target 40-60 0-99 0-100 0-100 0-100
Entropy	Raw EEG State Entropy Response Entropy Suppression ratio	 0-91, Target 40-60 0-100 0-100
SedLine	Raw EEG Patient state index (PSI) Artefact Suppression ratio Colour Spectrogram SEF95	 0-100, Target 25-50 0-100 0-100 0-30

*Numerical range associated with a “general anaesthetic” hypnotic state

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