Bispectral index monitoring during electroconvulsive therapy under propofol anaesthesia

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Background. The accuracy of the bispectral index (BIS) as a monitor of consciousness has not been well studied in patients who have abnormal electroencephalograms (EEG).

Methods. We studied the changes in BIS, its subparameters, and spectral entropy of the EEG during 18 electroconvulsive treatments under propofol and succinylcholine anaesthesia. A single bifrontal EEG, and second subocular channel (for eye movement estimation) was recorded.

Results. The median (interquartile range) BIS value at re-awakening was only 57 (47–78)—thus more than a quarter of the patients woke at BIS values of less than 50. The changes in spectral entropy values were similar: 0.84 (0.68–0.99) at the start, 0.65 (0.42–0.88) at the point of loss-of-consciousness, 0.63 (0.47–0.79) during the seizures, and 0.58 (0.31–0.85) at awakening.

Conclusions. Post-ictal slow-wave activity in the EEG (acting via the SynchFastSlow subparameter) may cause low BIS values that do not correspond to the patient’s clinical level of consciousness. This may be important in the interpretation of the BIS in other groups of patients who have increased delta-band power in their EEG.

Br J Anaesth 2002; 88: 184–7

Keywords: monitoring, bispectral index; anaesthesia, depth; brain, electroconvulsive therapy; monitoring, electroencephalography; brain, entropy

Accepted for publication: September 18, 2001

The bispectral index (BIS) is a processed electroencephalogram (EEG) parameter that purports to measure the level of hypnosis in anaesthetized patients. The BIS was formulated retrospectively using a large database of EEG recordings and clinical correlative data. A literature search did not reveal any studies specifically evaluating the accuracy of the BIS in predicting the hypnotic level in patients with underlying abnormal EEG patterns. Tanabe and colleagues observed in a single patient that electroconvulsive therapy (ECT) may decrease BIS during continuous propofol infusion. It has been observed that patients in the post-ictal state often display slow, delta, EEG waves even after they have regained consciousness. We performed an observational study on a group of patients undergoing ECT to evaluate the effect of seizure and post-ictal activity on the BIS and its ability to track the level of hypnosis.

Methods

After obtaining regional ethics committee approval and written patient consent, the BIS was recorded during 20 ECT treatments in 13 patients (aged 28–72 yr). Patients having compulsory treatment under the mental health act were excluded. Apart from the primary psychiatric illness requiring ECT, one patient had a previous history of childhood epilepsy but was currently symptom-free and not on anticonvulsants. No other patients had any neurological illness. EEG electrodes (Zipprep, Aspect Medical Systems, Newton, MA, USA) were attached before induction and impedance checked to be below 10 KΩ. One bipolar EEG channel was recorded from the forehead (F7–F8, on the International 10–20 system). Because eye movements may look very similar to slow delta waves in the frontal EEG, a second channel was recorded from the cheek with electrodes placed diagonally below the right eye to detect eye movement artifact. The eye movements (EYE) were quantified using the standard deviation (SD) of the amplitude from channel two.

Recording was commenced using an Aspect A-1000 EEG monitor (software version 3.31, Aspect Medical Systems), and data downloaded to a portable computer for storage and analysis. The sampling rate was 128/s, and the filters set at...
0.5 and 70 Hz. An anaesthetist uninvolved with the study gave their ‘usual’ anaesthetic consisting of: induction with propofol (70–170 mg i.v.) and neuromuscular block with succinylcholine (45–60 mg i.v.). Times were recorded for the following events: (1) start of induction (T1); (2) loss of response to verbal command (T2); (3) 10 s after the end of the electroconvulsive shock (T3); (4) first eye opening to verbal command (T4); and (5) 1 min after first eye opening (T5).

The data were analysed using Matlab (Matlab 5.3, The Mathworks Inc., Natick, MA, USA). The subparameters of the BIS (the BetaRatio and the SynchFastSlow) were calculated using published algorithms. Burst suppression was not observed during convulsions or in the post-ictal phase. Because there is some evidence that the spectral entropy of the EEG signal is a promising measure of depth of anaesthesia, this parameter was also calculated as described by Inoue. We used the frequency-band 2–40 Hz, and a resolution of 1 Hz.

**Results**

Two recordings had to be excluded because of technical faults in recording. Typical examples of the raw EEG waveform at various stages of the anaesthetic.

![Fig 1 Typical examples of the changes in the raw EEG waveform at various stages of the anaesthetic.](image)

![Fig 2 Changes in the BIS at various stages during the anaesthetic. The bars indicate the SD. Stage 1=T1, 2=T2, 3=T3, 4=T4, 5=T5.](image)

values are significantly lower ($P<0.01$) than those we obtained in a separate, previously published study, which involved similar dosages of propofol in patients having cardioversion. In this previous study the patients awoke at median (25–75th percentiles) BIS values of 79 (73–84), which increased 60 s later to 90 (85–95).

Figure 3 shows the changes in the two calculated subparameters of the BIS at each stage. It can be seen that the BetaRatio returns to near-pre-induction levels at the point of awakening, whereas the SynchFastSlow remains low—suggesting that the SynchFastSlow subparameter is predominant in depressing the BIS during the patient’s recovery phase. We conducted a backward-selection step-wise multiple linear regression of possible explanatory factors that may be correlated with the BIS at the point of awakening. The possible explanatory variables included in the initial regression model were: BetaRatio, SynchFastSlow, EYE, total spectral power, relative spectral power in the gamma (40–47 Hz), alpha (10–20 Hz), and delta (1–5 Hz) wavebands, and frontalis EMG activity – logarithmic power in 55–64 Hz band. Only SynchFastSlow and EYE remained in the final model as significant independent predictors of the BIS at the point of awakening (overall model $R^2=0.39$, $P=0.001$). The model was:

$$BIS_{T4}=118.1 \text{(SE}=14.3, P<0.0001) + 13.4 \text{(SE}=5.3, P=0.016) \times \text{SynchFastSlow} -8.6 \text{(SE}=2.2, P=0.007) \times \text{EYE}.$$

The median (25–75th percentiles) values of the spectral entropy at the start were 0.84 (0.68–0.99), dropping to 0.65 (0.42–0.88) at the point of loss-of-consciousness, and further decreasing to 0.63 (0.47–0.79) during the seizures. Spectral entropy did not increase at the point of awakening (0.58 (0.31–0.85)). The value at awakening was significantly different from the start value ($P=0.001$, paired $t$-test), but not different from the value during the seizures ($P=0.44$, $P=0.44$, respectively).
paired \( t \)-test). If the spectral entropy was calculated using only the higher frequencies of the EEG signal (20–40 Hz), the values at awakening (0.96 (0.86–0.99)) were significantly higher (\( P < 0.001, t \)-test) than those calculated using the whole range of frequencies (2–40 Hz).

**Discussion**

We have found that following ECT, over one-quarter of the patients re-awaken at a BIS value that is considered to reliably indicate unconsciousness (<50), when applied to the normal surgical population. This finding has two consequences. First, monitoring the BIS following ECT may not reliably correlate with the patient’s clinical level of consciousness. More importantly it suggests that, in other disease states in which there is increased delta EEG activity in conscious patients, the BIS may be falsely low. The question of whether this generalization is true warrants further investigation. Increased slower EEG activity (delta and theta) is common in both schizophrenic patients, and the elderly.\(^7\)–\(^10\)

Which subcomponent of the BIS was responsible for the falsely depressed values? The BetaRatio recovered towards pre-induction levels at the time of first eye-opening to verbal command and at 60 s after, but the SynchFastSlow remained low. This may be explained by the presence of low-frequency (delta) EEG activity in the post-ictal state. Because of the denominator used in the respective calculations, activity below 10 Hz will affect the calculation of the SynchFastSlow, but not the BetaRatio.\(^1\) The regression model was derived from only 18 data points, and therefore must be interpreted with caution, but supports the predominance of the SynchFastSlow in lowering the BIS during recovery.

The spectral entropy calculated at each stage showed changes that were similar to those shown by the BIS. Neither parameter increased reliably at the point of awakening. However, the behaviour of the spectral entropy is somewhat dependent on technicalities of the range of frequencies over which it is calculated, and the amount of pre-processing and filtering. If the spectral entropy was calculated over higher frequencies (e.g. 20–40 Hz) it is sensitive to increases in higher frequency activity that are characteristic of the return of consciousness.\(^11\) However it is difficult to be sure how much of this activity is frontal EMG, and how much is true return of high-frequency EEG ‘gamma’ rhythms at the point of awakening. Conversely, if the lower frequencies are excluded, then the spectral entropy will be less sensitive to the influence of the strong delta rhythms that indicate deep anaesthesia. One may speculate that in order to cover fully the range of anaesthetic concentrations and effects it would be logical to have a ‘meta-function’ that varied and combined the influence of different frequency-bands in the spectral entropy calculations. This is analogous to the construction of the BIS. If the value of the spectral entropy (20–40 Hz) was high (e.g. >0.9) it would predominate. As the high-frequency spectral entropy (20–40 Hz) fell, then the proportional influence of spectral entropies calculated using progressively lower frequencies (e.g. 2–20 Hz) would increase.

The interpretation of the EYE parameter is problematic. The diagonal placement of the eye electrodes should have been able to detect both horizontal and vertical eye movements. The EYE parameter decreased only minimally during the pre-ECT period when the patients were anaesthetized with propofol and paralysed with suxamethonium—as evidenced by minimal movements during the grand mal seizure. Because EYE still had activity when the patient was supposedly paralysed, this clearly indicates that the EYE parameter was not a pure estimate of eye-movements, but included an unknown and significant quantity of EEG signal and, possibly, frontalis EMG activity. Indeed, the BIS derived from the second (eye) channel correlated well with the true channel-one BIS (derived from the frontal montage) when the patient was anaesthetized—Pearson’s correlation coefficient (\( r \)): \( r_{T2T3}=0.99, r_{T3T4}=0.97, r_{T4T5}=0.95, r_{T5T6}=0.57.\)

In conclusion, our study suggests that a low BIS value does not correspond to a deep hypnotic level in patients after ECT. By inference, this may also be applicable to patients with other pre-existing EEG abnormalities. In our patient group, the recovery of spectral power in the higher frequencies (as estimated by the BetaRatio, or spectral entropy (20–40 Hz)) was more predictive than the BIS, and the broadband spectral entropy (2–40 Hz), of the recovery of consciousness.
References