Case Report

Transient decreases in Bispectral Index without associated changes in the level of consciousness during photic stimulation in an epileptic patient

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This case report describes a patient with a history of epileptic seizures who showed unusual decreases in the Bispectral Index (BIS) attributable to the induction of abnormal slow electroencephalographic (EEG) waves by photic stimulation, without any associated decrease in his level of consciousness. After starting anticonvulsive therapy, photic stimulation no longer induced abnormal EEG activity nor decreased BIS values. These findings suggest that BIS values may not accurately reflect a patient’s actual level of consciousness in the presence of epilepsy-related abnormal EEG activity and that the BIS monitor may be able to track such EEG changes.

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The electroencephalographic (EEG) Bispectral Index (BIS) is used as an indicator of the level of consciousness during anaesthesia.¹² Although BIS values are reported to change abruptly upon the appearance of epileptiform EEG activity during general anaesthesia,³⁴ it remains unknown whether such changes in the BIS reflect actual changes in the level of consciousness. We present a patient with epilepsy whose BIS values decreased because of abnormal EEG activity induced by photic stimulation, without any associated decrease in his level of consciousness.

Case report

A 30-yr-old male treated with oral risperidone (6 mg day⁻¹) for schizophrenia developed generalized epileptic seizures. Extensive physical and radiological examinations failed to identify the source of his seizures.

Before starting anticonvulsive therapy, two EEG studies incorporating photic stimulation were performed, the first with a standard EEG monitor using the 10-20 international electrode placement system (10-20EEG) (Synafit1000, NEC, Tokyo, Japan), and the second with both the 10-20EEG monitor and the BIS EEG monitor (A-2000, Aspect Medical Systems, Natick, MA, USA). The BIS electrodes (BIS sensor plus, Aspect Medical Systems) were placed on the forehead and temple according to the manufacturer’s recommendation. The smoothing rate for BIS reports was set at 30 s. Standard intermittent photic stimulation was conducted with a flicker device (PS lamp house unit, NEC, Tokyo, Japan) using a white light placed at a distance of 30 cm from the eye while the awake patient was lying on his back with his eyes closed lightly.⁵⁶ The patient received 10 cycles of 10 s photic stimulation spaced at 10 s intervals. The flicker frequency was set to 1 Hz for the first cycle, increased to 4 Hz for the second cycle and then increased by 2-Hz increments for succeeding cycles to 20 Hz for the 10th cycle.

In both studies, photic stimulation induced the same changes in raw EEG waves. Before photic stimulation, baseline 10-20EEG showed dominant fast low-amplitude waves (8–9 Hz 30–40 μV α waves) and intermittent slow high-voltage waves (3–4 Hz 50–100 μV δ or θ waves) (Fig. 1A). During photic stimulation, frequent slow-high-voltage waves 2–4 s
in duration were seen, predominantly in frontal areas, while the patient continued to reply promptly to voice without showing any convulsive movements (Fig. 1B). These slow waves disappeared almost completely after photic stimulation ended.

Raw EEG waves displayed on the BIS monitor had a similar appearance to the above-described waves. Before photic stimulation, BIS values ranged between 90 and 98 (Fig. 2D), probably reflecting dominant fast waves intermingled with slow high-voltage waves (Fig. 2A). Forty-five seconds after starting photic stimulation, BIS values decreased rapidly to a minimum of 63 (Fig. 2E), probably reflecting dominance of the slow high-voltage waves (Fig. 2B). After photic stimulation ended, BIS values returned to baseline levels over the next 30 s (Fig. 2E).

During the period of intermittent photic stimulation, the patient replied promptly to voice every 10 s. After the EEG examination ended, he had an explicit memory of the flickering light, seen through the closed eyelids, throughout the photic stimulation period over 190 s.

Subsequently, an oral anticonvulsant, sodium valproate, 800 mg day\(^{-1}\) was added to the patient’s schizophrenia treatment regimen. Two weeks later, when the plasma valproate concentration had reached a therapeutic value (78.9 µg ml\(^{-1}\)), we conducted a third EEG study using both BIS and 10-20EEG monitors. The baseline EEG was almost identical to the baseline EEGs of the earlier studies, except that fewer slow high-voltage waves were present (Fig. 1C). BIS values ranged between 94 and 98 (Fig. 2C). Photic stimulation only minimally increased the number of slow high-voltage waves (Figs 1D and 2C) and minimally decreased BIS values (92 at a minimum) (Fig. 2C). A follow-up EEG conducted 6 months later, with the patient still treated with valporate, detected no baseline or photic stimulation-induced abnormalities in raw EEG waves or BIS values.

**Discussion**

Untreated epileptic patients are reported to exhibit significant slowing of background EEG with increased δ and θ power, which is reduced with treatment with valproate.\(^7,8\) In the present case, slow EEG waves in the δ or θ band were occasionally seen even during wakefulness before institution of anticonvulsive therapy. Bispectral analysis and power spectral analyses are major components of the BIS algorithm,\(^2,9\) and therefore the appearance of a δ or θ waves during wakefulness might have resulted in a slight decrease in baseline BIS values (90–98) initially seen in the present case. During photic stimulation, BIS values decreased further to a minimum of 63, probably reflecting a photic stimulation-induced predominance of slow δ or θ activity, although the patient remained quite alert to our calls during the examination, and afterwards, he had a thorough memory of the examination. This case suggests that BIS values may not accurately reflect the actual level of consciousness when abnormal EEG activity is evoked in epileptic patients.

The search for a reliable depth of anaesthesia monitor is ongoing. The BIS algorithm is not fully known, and high BIS values associated with low level of consciousness and low BIS values associated with intraoperative awareness have been reported.\(^7\) The present case report describes a false low BIS reading in an epileptic patient during photic stimulation, once again showing that this depth of anaesthesia monitor has some limitations. It is quite conceivable, however, that BIS values do not always reflect the actual
Fig 2 Photic stimulation (PS)-induced changes in Bispectral Index (BIS) and raw electroencephalogram (EEG) waves displayed on the A-2000 BIS monitor, before and after starting anticonvulsant treatment with valproate. (A and B) portray raw EEG waves recorded before and during PS, respectively, from the second EEG examination, before the initiation of anticonvulsant therapy. (C) portrays raw EEG waves recorded during PS from the third EEG examination, 2 weeks after the initiation of anticonvulsant therapy. (D and E) portray BIS values before and during PS, respectively, from the second EEG examination. (F) portrays BIS values during photic stimulation from the third EEG examination. In panels (E and F), the labelled box identifies the period of photic stimulation.
Reduced BIS values during epileptiform EEG

level of consciousness in the presence of neurological diseases and/or abnormal EEG activity, given that the BIS algorithm has been derived empirically from a sizeable database of anaesthetized volunteers without neurological diseases. It is known that some neurological diseases are associated with slowing of background EEG waves. For example, a significant proportion of patients with dementia exhibits low baseline BIS values, reflecting increased δ and θ power with a parallel decrease in α and β power. Similarly, frontal intermittent rhythmic delta activity (FIRDA) is detected principally in awake patients having some structural brain lesions (e.g. hemispheric tumours and old ischaemic lesions) or metabolic encephalopathy. Such pathological EEG changes may intermittently decrease BIS values in awake patients, as would be the case in the present report.

Three case reports have documented abnormal BIS changes associated with the development of epilepsy-related EEG activity. In a patient with a recent history of epileptic seizure, intermittent changes in BIS values between 40 and 20 occurred repeatedly at intervals of a few minutes during abdominal surgery under combined general (sevoflurane 0.6–1% and nitrous oxide 67% in oxygen) and epidural anaesthesia, reflecting sudden transitions between baseline EEG (fast 10–14 Hz low-voltage waves) and abnormal epileptiform EEG (slow 1.5–2.5 Hz high-voltage spike-and-wave complex). In two human volunteers out of eight given the two minimum alveolar concentration (MAC) of sevoflurane, this dose of sevoflurane evoked three episodes of epileptiform discharges, immediately after inducing burst suppression EEG and isoelectrical silence. In these cases, BIS values increased markedly, from 0 to 6 during the burst suppression and isoelectrical silence to 44–73 during the epileptiform discharges. In a seizing patient suffering from status epilepticus unresponsive to i.v. phenytoin and benzodiazepines, a high BIS value (94) was observed with typical epileptiform EEG waves despite the fact that at no point was the patient rousable or awake. The previous and present case reports suggest that depending on waveform characteristics of both baseline and epileptiform EEG waves, the development of epileptiform discharges can result in sudden, unusual increases or decreases in BIS values irrespective of the actual anaesthetic depth or level of consciousness. Therefore, raw EEG signal should be checked if BIS values behave inconsistently during anaesthesia, especially in patients with a history of epilepsy and those receiving high-dose anaesthetics with proconvulsive property.

Although BIS monitors primarily detect signals from the frontal regions of the brain, BIS monitoring should be able to detect abnormal EEG waves elsewhere if such EEG activity propagates to frontal regions. Furthermore, the BIS monitor is much simpler to use than conventional EEG monitors. Our psychiatrists have used the monitor in some patients with psychiatric disorders and possible EEG abnormality. Previous reports have also shown that the BIS monitor can trace the development of epileptiform EEG activity and its immediate disappearance in response to i.v. injection of anticonvulsants. The BIS monitor thus may be capable of conveniently detecting and following epilepsy-related abnormal EEG activity in some patients with epilepsy. Clearly, the BIS monitor is not intended as a diagnostic tool, but a monitoring tool and further studies would be required to assess the adequacy of its use for indications other than measuring depth of anaesthesia. A number of works have suggested, however, that the BIS monitoring can be conveniently used to detect and follow cerebral hypoperfusion, to detect brain death, to make a diagnosis of pseudoseizures, to assess the level of consciousness in brain-injured patients with and without sedation, and to predict recovery of consciousness in patients with severe brain injury, although some controversy has continued as to the reliability of the BIS monitoring in brain-injured patients.

In conclusion, we have presented a patient with a history of epileptic seizures who showed an unusual decrease in BIS values attributable to the induction of abnormal slow EEG waves by photic stimulation, without any associated decrease in his level of consciousness. Our findings suggest that BIS values may not accurately reflect a patient’s actual level of consciousness in the presence of epilepsy-related abnormal EEG activity, and that the BIS monitor may conveniently track such EEG changes.

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