Comparison of Bispectral Index and Entropy values with electroencephalogram during surgical anaesthesia with sevoflurane†


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Abstract

Background: Concomitantly recorded Bispectral Index® (BIS) and Entropy™ values sometimes show discordant trends during general anaesthesia. Previously, no attempt had been made to discover which EEG characteristics cause discrepancies between BIS and Entropy. We compared BIS and Entropy values, and analysed the changes in the raw EEG signal during surgical anaesthesia with sevoflurane.

Methods: In this prospective, open-label study, 65 patients receiving general anaesthesia with sevoflurane were enrolled. BIS, Entropy and multichannel digital EEG were recorded. Concurrent BIS and State Entropy (SE) values were selected. Whenever BIS and SE values showed ≥10-unit disagreement for ≥60 s, the raw EEG signal was analysed both in time and frequency domain.

Results: A ≥10-unit disagreement ≥60 s was detected 428 times in 51 patients. These 428 episodes accounted for 5158 (11%) out of 45 918 analysed index pairs. During EEG burst suppression, SE was higher than BIS in 35 out of 49 episodes. During delta-theta dominance, BIS was higher than SE in 141 out of 157 episodes. During alpha or beta activity, SE was higher than BIS in all 49 episodes. During electrocautery, both BIS and SE changed, sometimes in the opposite direction, but returned to baseline values after electrocautery. Electromyography caused index disagreement four times (BIS > SE).

Conclusions: Certain specific EEG patterns, and artifacts, are associated with discrepancies between BIS and SE. Time and frequency domain analyses of the original EEG improve the interpretation of studies involving BIS, Entropy and other EEG-based indices.


Key words: anaesthetics inhalation, sevoflurane; EEG; electromyography; monitoring, intraoperative
Editor’s key points

- Discrepancies are sometimes found between concomitantly recorded BIS and spectral entropy values during anaesthesia.
- The authors recorded BIS and spectral entropy values in 65 patients undergoing sevoflurane anaesthesia.
- Concomitantly recorded raw EEG signals were investigated for patterns associated with discrepancies.

**EEG** is the method of choice for assessing the effects of anaesthetics on the central nervous system. The behaviour of and the agreement between Bispectral Index® (BIS; Medtronic-Covidien, Dublin, Ireland) and Entropy® (GE Healthcare, Helsinki, Finland), two EEG-based depth of anaesthesia monitors, during different stages of clinical anaesthesia, have been studied very intensively.

It has been shown that these commercially available parameters do not change in parallel with each other during anaesthesia or sedation. Some studies have reported ‘paradoxical’ changes and inaccurate readings of index values. While complex factors, different anaesthetic agents produce EEG waves or sedation. Also, different anaesthetic agents produce EEG waveforms that are different in pattern and reactivity. In addition, the agreement between BIS and Entropy has been shown to deteriorate with increasing age.

The numerical comparison of index values alone is insufficient to reveal the electrophysiological reasons for disagreement between them. The aim of this study was to analyse which EEG characteristics or artifacts are associated with disagreement between the numerical values of BIS and Entropy during sevoflurane anaesthesia.

**Methods**

This study followed the design of a prospective, open-label clinical study. After the approval of the local Ethics Committee (R09183), the study was registered at ClinicalTrials.gov (Identifier NCT01077674). Written informed consent was obtained from all patients.

**Patients**

Sixty-five patients, aged 50–80 yr, and undergoing elective gynaecological surgery, were studied. Inclusion criteria were: ASPhysical status I–III, expected duration of operation >30 min, and general anaesthesia requiring endotracheal intubation. Exclusion criteria were: disease or previous trauma of the autonomic or central nervous system, family history of malignant hyperthermia, history of allergic reaction to planned study medication and an inability to communicate in Finnish language.

**Anaesthesia and study protocol**

All patients were premedicated (diazepam 10 mg and paracetamol 1 g orally) approximately 60 min before induction of anaesthesia. In the operating room, an i.v. route was established and infusion of isotonic saline was started. Non-invasive blood pressure (NIBP) was measured every 5 min. ECG, peripheral oxygen saturation ($Sp_O_2$), inspired fractions (Fi) and end-tidal (Et) concentrations of sevoflurane, oxygen and carbon dioxide (CO2) were continuously monitored (Datex-Ohmeda S/5™ Anesthesia Monitor, Datex-Ohmeda™, Helsinki, Finland). The baseline values for NIBP and heart rate (HR) were measured before anaesthetic induction.

The general anaesthesia was conducted according to the judgement of the anaesthetist in charge of the anaesthesia. The anaesthesia was induced with i.v. anaesthetics (propofol 1.5–3.0 mg kg$^{-1}$ and fentanyl 1–2 µg kg$^{-1}$) and maintained with sevoflurane. Neuromuscular blocking agent (rocuronium or cisatracurium) was given to facilitate endotracheal intubation; additional doses were given according to clinical needs. After endotracheal intubation, the lungs were ventilated with a tidal volume of 6–8 ml kg$^{-1}$. The respiratory rate was adjusted to maintain normocapnia (EtCO2 4.0–5.5). The anaesthetist in charge of the anaesthesia was blinded to raw EEG, BIS and Entropy information. To avoid awareness during anaesthesia, the sevoflurane concentration was kept at ≥0.5 minimum alveolar concentration (MAC) at all times. Furthermore, if the BIS value was below 30 for more than 120 s or if the BIS value exceeded 60, the principal investigator (KK) notified the anaesthetist to consider the adjustment of medication. Supplemental fentanyl doses were given according to clinical needs.

The anaesthetist guided the titration of general anaesthesia based on clinical signs as follows; in case of hypertension (>20% increase in mean arterial pressure (MAP) from baseline value), the sevoflurane concentration was increased or fentanyl 2 µg kg$^{-1}$ was administered. Tachycardia (HR >110 min$^{-1}$ or >20% increase from baseline value) was also treated with fentanyl 2 µg kg$^{-1}$. The anaesthetist used vasoconstrictors (etilefrine, noradrenaline) and crystalloids/colloids according to clinical needs.

**EEG acquisition**

BIS, Entropy and multichannel EEG monitoring were started before induction of anaesthesia and continued uninterrupted until the end of the operation. After the patient’s forehead had been wiped with an alcohol swab and then allowed to dry, the BIS (BIS Quatro™ sensor, Medtronic-Covidien, Dublin, Ireland) and Entropy (GE Healthcare, Helsinki, Finland) sensors were placed on the forehead as recommended by the manufacturers. The contralateral position of the two electrodes on the forehead (above and below, left and right) was randomly assigned. The BIS and Entropy plug-in modules were connected to the same anesthesia monitor. The sampling rate for the raw EEG was 400 Hz for Entropy and 256 Hz for BIS. The baseline concentrations of sevoflurane were continuously monitored (Datex-Ohmeda S/5™ Anesthesia Monitor, Datex-Ohmeda™, Helsinki, Finland) at 10-s intervals on a laptop computer for offline analysis.

Multichannel EEG was recorded with silver/silver chloride electrodes after careful skin preparation (LemonPrep™; Mavidon Medical Products, Lake Worth, FL). Adhesive silver/silver chloride gel-filled electrodes (Ambu® Neuroline 720; Ambu A/S, Ballerup, Denmark) were applied to a hairless skin area, and silver/silver chloride cup electrodes (Reusable Cup Electrodes; Technomed
Europe, Maastricht-Airport, Netherlands) filled with conductive paste (OL-eletroditapa; BERNER, Helsinki, Finland) were used for the scalp recording (Cz). According to the international 10–20 electrode placement system, Fp1, Fp2, and Cz, with added left outer canthus electrode were adopted. The reference electrode was attached to the left mastoid, and ground electrode was placed on the right clavicle. Submental EMG was collected with bipolar montage; the electrodes were placed 3 cm apart under mandible according to the standard location of the EEG monitoring in sleep studies. Two electrodes located vertically with a distance of 3 cm on masseter muscle below zygomatic arch were used to collect masseter EMG. The EEG and EMG were digitalised and continuously recorded using NicoletOne Monitor™ (CareFusion; San Diego, CA) digital EEG system, version 5.50. Electrode impedance was kept <10 kΩ, and band-pass was 0.03–300 Hz with a sampling rate of 1000 Hz. Continuous signals (EEG, ECG) were directly collected to the NicoletOne Monitor™. Power line artifact (50 Hz) was not filtered.

The clocks of NicoletOne Monitor™ and the laptop computer with SS Collect software were synchronised manually before anaesthetic induction. All significant anaesthesia- or surgery-related events were annotated on the computer hard disk by the principal investigator.

Analyses of the biosignals
All biosignals recorded after initial loss of consciousness to the end of operation were analysed off-line. Only concurrent pairs of BIS and SE values with BIS signal quality index >50 were included in the analysis. Earlier studies have shown a delay in the calculation of the numerical values of BIS and Entropy. This delay depends on the index used and on the morphology of EEG, and the delay is different for ascending and descending values. To minimize the effect of the time delay in the calculation of the numerical values, we decided that only disagreements lasting ≥60 s would be analysed.

The raw EEG signals at the time points of interest (≥10-unit disagreement lasting ≥60 s) were visually inspected by a clinical neurophysiologist with substantial experience on anaesthesia-related EEG (VJ), and analysed with the software of NicoletOne Monitor. EEG spectrogram was used to visualise different frequency band activities, and power spectra were calculated to help decision making, if necessary. The reasons for deviating values were analysed and classified into either artifacts or specific changes in EEG pattern.

Sample size
Sample size was not based on power analysis, as we did not compare parallel groups. In a recent publication, 58 patients with 7800 data pairs were sufficient to reveal the distribution of difference between BIS and Entropy. We enrolled 65 patients and collected 46724 BIS/Entropy data pairs.

Results
A total of 68 patients were enrolled in this study, of whom three were excluded as a result of technical problems in EEG recording. Patient characteristics and intraoperative data are shown in Table 1.

A total of 46724 concurrent index pairs were recorded during surgical period, of which 806 were excluded (BIS SQI ≤50), thus, 45918 index pairs were accepted for analysis. In only 14 patients the intraoperative BIS and SE readings did not differ (i.e. no ≥10-unit disagreements lasting ≥60 s were detected). In the remaining 51 patients, episodes where BIS was higher than SE were seen in 24 patients, and episodes where SE was higher than BIS were seen in 18 patients. In nine patients, sometimes BIS, sometimes SE was higher. Altogether 428 episodes (containing 5158 (11%) out of a total of 45918 concurrent pairs) of index disagreement were seen. In 231 episodes (2620 concurrent pairs) BIS was higher than SE, and in 197 episodes (2538 concurrent pairs) SE was higher than BIS.

The classification and prevalence of signal features associated with index disagreement are presented in Fig. 1. During EEG delta/theta dominance, the highest difference between BIS and SE was 41 units (BIS > SE). During delta activity with alpha/beta activity the highest difference was 43 units (SE > BIS). During fast EEG activity (alpha/beta) the highest difference was 30 units (SE > BIS). During EEG burst suppression, the highest difference was 43 units (SE > BIS). During electrocautery, the highest difference was 42 units (BIS > SE). The highest difference caused by EMG was 42 units (BIS > SE). Effects of some of these phenomena on the index values are illustrated in Fig. 2 (EEG delta/theta dominance and burst suppression), Fig. 3 (EMG activation) and Fig. 4 (electrocautery).

Episodes in which the median value of one index was >60 (i.e. above the threshold for surgical anaesthesia, while the other index was ≤60), were detected 29 times. During one episode of index disagreement, both indices showed values >60 (Fig. 1). Clinically extreme situations, where one of the studied indices showed low (<40) values, indicating deep anaesthesia, while the other index indicated insufficient hypnotic effect (>60), were seen in two patients. In one patient (three separate episodes) the BIS value was <40 and SE was >60. The raw EEG in this case showed burst suppression pattern. In another patient (one episode during EMG activity), the BIS value was >60 and SE was <40. The course of anaesthesia was uneventful in all 65 patients.

Discussion
Our results show that disagreement between numerical values of BIS and Entropy can be caused by both artifacts in recorded EEG signal and changes in the EEG pattern itself. BIS was higher than SE in 141 out 157 index discrepancies during slow EEG activity (delta-theta dominance). During EEG burst suppression, SE was higher than BIS 35 times, and BIS was higher than SE 14 times. Fast EEG activity (alpha and beta), either separately or with delta activity, resulted in SE values being higher than BIS values. Both BIS and SE values increased during electrocautery, but returned to baseline values after cessation of electrocautery. In four episodes during EMG activity, the BIS values were higher than SE.

| Table 1 Patient characteristics and intraoperative data. Data presented as mean (SD), or number |
|---------------------------------|---------------------------------|---------------------------------|
| **Patient Characteristics**     | **Intraoperative data**         | **Duration of operation (min)** |
| **Age (yr)**                    | 66 (11)                         | 119 (62)                        |
| **Height (cm)**                 | 165 (6)                         |                                 |
| **Weight (kg)**                 | 75 (14)                         |                                 |
| **ASA physical status (1/2/3)** | 14/16/35                        |                                 |
| **Laparoscopy/laparotomy**     | 28/37                           |                                 |

Discussion
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These findings offer electrophysiological explanation for the results of earlier studies, in which variable correlation between different index values during anaesthesia and surgery has been reported. To our knowledge, this is the first study where the raw EEG has been studied in both time and frequency domain and compared with numerical information of BIS and Entropy, in order to discover which EEG patterns or artifacts cause discrepancies in numerical values of BIS and Entropy.

Although an identical EEG signal was not used for BIS, Entropy and raw EEG, the fact that most of the time indices were comparable, shows that different electrode locations were not the reason for occasional differences in indices. Before exploring the reasons for discrepancies between the indices, it must be emphasized that discrepancies occurred in only 11% (5158/46 724) of the analysed concurrent pairs. This percentage is smaller than in an earlier study by Pilge and co-workers comparing BIS and Cerebral State Index; most likely because we analysed only episodes lasting ≥60 s. By analysing episodes lasting ≥60 s, the short-lasting discrepancies during transitions between two anaesthetic levels, because of time delays in index calculations were not included in the analyses. The most common EEG phenomena during general anaesthesia, that deviate from the ‘normal’ EEG pattern, are epileptiform EEG and EEG beta/delta arousals. The raw EEG in this study was carefully analysed visually, and we found no evidence of epileptiform activity or EEG beta/delta arousals. Using propofol as an induction agent instead of mask induction with sevoflurane (reducing the risk of epileptiform activity) and administering opioids (most likely reducing the likelihood EEG beta/delta arousals) may have been contributing factors to the acceptable correlation between BIS and Entropy.

The EEG changes during progressive administration of most anaesthetics can be summarized as follows: initially, during light sedation, there is an increase in higher (beta, 20–30 Hz) frequencies. Thereafter, the dominant frequency shifts towards lower frequencies (beta to alpha (8–13 Hz) to theta (4–8 Hz) to delta (<4 Hz). If the dose of anaesthetic is further increased, burst suppression activity ensues. During burst suppression, low voltage periods (suppressions) are followed by high amplitude activity (bursts). After burst suppression, the EEG will change to suppression. In addition to the changes in the frequency bands, anaesthesia also produces topographical changes: (1) anteriorisation of EEG power (i.e. EEG power shifts from posterior to anterior regions of the brain), and (2) a decrease in coherence of homologous regions of the two hemispheres. Our results show that during delta-theta dominance BIS is sometimes higher than SE, and during burst suppression, SE is sometimes higher than BIS. As both delta-theta dominance and burst suppression are signs of deep anaesthesia, it cannot be concluded that one index is superior to the other during deep anaesthesia, but instead it must be kept in mind that BIS and SE are calculated using different algorithms.

During EEG burst suppression, SE values were higher than BIS values in 14 patients (35 episodes). We hypothesise that the reason for this discrepancy is the occasional inability of the Entropy algorithm to recognize the burst suppression correctly. However, it is noteworthy that in 14 episodes during burst suppression
Fig 2 The opposite effect of two different EEG patterns on the numerical values of BIS (Bispectral Index®) and SE (State Entropy). The whole intraoperative data of one study patient. Panel A: Numerical values of BIS, SE and RE-SE. Panel B: BIS suppression ratio (BIS SR) and Entropy burst suppression ratio (Entropy BSR). Panel C: BIS EMG. Panels D and E: End-tidal (ET) concentration of sevoflurane and the degree of neuromuscular block (Train of four, TOF), respectively. Panel F: A 15-s sample of raw EEG (# 1 in Panel A, Fp1-LOC [left outer canthus], band pass 0.5–70 Hz) at 11:05. The EEG consists of mixed frequencies (delta, theta, alpha), which results in similar BIS and SE values. Panels G and H: Two 15-s samples of raw EEG at approximately 11:15 and 12:40 (# 2 and # 3 in Panel A, respectively, Fp1-LOC, band pass 0.5–70 Hz). At 11:10, the concentration of ET-sevoflurane is increased from 1.7 to 3.6%. At approximately 11:15, a discrepancy starts to be seen between BIS and SE. In raw EEG (Panel G), delta/theta dominance is seen, and sharp waves. The increasing sevoflurane concentration produces characteristic changes in EEG associated with deepening anaesthesia, but the BIS values remain unchanged. At 12:35–12:45, there is a pause in the operation, because the operating gynaecologist consults a GI surgeon. The cessation of surgery causes a change in the nociceptive/antinociceptive balance of the patient, and the EEG turns into burst suppression, shown in panel H. Accordingly, BIS value decreases below 30, but SE value remains around 50. Because of 0.5 Hz high pass filter the DC-shift at suppression onset is transformed to ‘slow wave’ and is not readily detected as part of the burst suppression pattern in Entropy monitoring.
suppression, BIS was higher than SE (Fig. 1). The fact that both indices sometimes fail to detect burst suppression, an EEG phenomenon that is relatively easy to interpret visually, highlights the importance of displaying the raw EEG signal on the anaesthesia monitor.

Electrocautery-induced artifacts produced discrepancies between BIS and SE in 35 patients (110 episodes). After cessation of electrocautery, both indices returned to values before electrocautery. It is theoretically possible that the discrepancy between BIS and SE during electrocautery was in fact caused by genuine changes in evoked responses and EEG arousal. But, because analysis of EEG was impossible as a result of a strong electrocautery artifact, the true origin of the discrepancy remains speculative. In earlier studies, it has been shown that Entropy is more resistant of electrocautery, both indices returned to values before electrocautery. It is theoretically possible that the discrepancy between BIS and SE during electrocautery was in fact caused by genuine changes in evoked responses and EEG arousal. But, because analysis of EEG was impossible as a result of a strong electrocautery artifact, the true origin of the discrepancy remains speculative. In earlier studies, it has been shown that Entropy is more resistant
Fig 4 The effect of electrocautery on BIS (Bispectral Index®) and SE (State Entropy). Before the beginning of electrocautery, BIS and SE values show similar values. Electrocautery begins shortly after 13:52 and is associated with a discrepancy between BIS and SE. After electrocautery both indices return to baseline level. Panel A: The numerical values of BIS, SE and RE-SE. Panel B: BIS signal quality index (BIS SQI). Panel C: BIS suppression ratio (BIS SR) and Entropy burst suppression ratio (Entropy BSR). Panel D: BIS EMG. Panel E: End-tidal (ET) concentration of sevoflurane. Panel F: 30-s sample of raw EEG at approximately 13:53:30 (#1 in Panel A, Fp1-LOC (left outer canthus), band pass 0.5–70 Hz). EEG signal is repeatedly distorted by electrocautery artifact.
to artifacts than BIS. It must be emphasized that during electrocautery, the numerical information produced by EEG-derived depth of anesthesia monitors must be interpreted with caution.

We would also like to point out that our material included only EEG segments during surgical anesthesia. The EEG during surgical anesthesia is not as artifact-contaminated as EEG during lighter levels of anesthesia.

There are limitations to our study. First, the contralateral placement of the BIS and Entropy sensors is potentially a confounding factor. Although the placement of BIS and Entropy sensors on the forehead was randomly assigned, the two sensors could register biosignals differently. The global brain activity, such as EEG burst suppression, and regional EEG and EMG activities in the frontal area, can be identically recorded by two different electrodes positioned on the opposite sides of the head. However, if the activated motor unit is directly under one of the EEG sensors, the EMG activity can affect one sensor more strongly than the other sensor, as EMG may lose its spikelike waveform when conducted a longer distance under skin. Second, it has been reported that the time delay in index calculations are different with BIS and Entropy, and that this time delay depends on the depth of anesthesia. Additionally, the different time delays during increasing and decreasing values have been reported, even when using the same depth of anesthesia monitor. Therefore, the results obtained in this study could be affected by the different latencies in index calculation. However, as the criteria for discrepancy in this study was ≥10 units for ≥60 s, the difference of 10–20 s in time delays cannot be the only explaining factor for the discrepancy between BIS and Entropy.

One may argue that the current study does not reflect routine clinical practice, as in the operating room, only one depth of anesthesia monitor is used at a time. We cannot fully contest that argument, but in our opinion, it is more relevant to study the raw EEG and to be more aware of the electrophysiology behind strangely behaving numerical values of depth of anesthesia monitors, than to perform various intricate statistical analyses (e.g. Bland-Altman plots), on the agreement/disagreement between different depths of anaesthesia monitors. The signal segments with EMG were identified using high resolution signal and logarithmic power spectrum (Fig. 3), and must be excluded from statistical analyses of BIS and Entropy values.

We chose to use a sevoflurane concentration of ≥0.5 MAC to avoid intraoperative awareness. It can be argued that a MAC level of ≥0.7 should have been implemented, owing to the fact that the original reports of Avidan and co-workers used ≥0.7 MAC. However, these studies consisted of patients with a high risk of intraoperative awareness. The study population in our study consisted of unselected surgical patients so, in our opinion, a lower MAC threshold was justified. A recent study by Maeshour and co-workers offers evidence that our approach was justified. Furthermore, the anaesthetist was immediately notified in case of BIS values >60, further reducing the risk of intraoperative awareness. It must be kept in mind that our study was not designed to study intraoperative awareness. Despite this, all patients were carefully interviewed postoperatively, and we found no evidence of intraoperative awareness in any patients.

In conclusion, we found that the discrepancies between the numerical values of BIS and Entropy can be caused by EEG burst suppression, EEG delta-theta dominance, EEG alpha/beta activity, EMG, or electrocautery artifacts. These findings emphasize the importance of displaying the EEG signal on the monitor instead of relying solely on the numerical information of BIS and Entropy. Furthermore, anaesthetists using EEG-based depth of anesthesia monitoring, should also have the skills to interpret the raw EEG signal correctly. In research, the depth of anesthesia indexes can be used only if the raw EEG signal is recorded and professionally analysed for artifacts like EMG, and for features deviating from the normal anaesthetic pattern, such as epileptiform spikes.

Authors’ contributions

A.J.A.: wrote the manuscript, recruited and anaesthetized patients for the study and helped to analyse the data. K.K.: helped to design the study and write first draft of the manuscript, set up the EEG registrations and collected all the data on computer. She is also responsible for archiving the study files. V.J.: helped to design the study, analysed all the EEG recordings, and helped to write the study. A.K.: helped to analyse the data, constructed the figures of the manuscript, and helped to write the manuscript. He has seen the original study data and reviewed the analysis of the data. S.H.: has helped in interpretation and analyses of the data and helped in the preparation of the manuscript. H.H.: has helped to interpret and analyse the data and provided valuable expertise in revising the manuscript. She has seen the original data. A.Y.-H.: designed the study, registered the study at ClinicalTrials.gov, recruited and anaesthetized patients for the study, helped in the analyses of the data and in the writing of the manuscript. He has seen the original data and reviewed the analyses of the data. All of the authors read and approved the final manuscript.

Declaration of interest

None declared.

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None declared.

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