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Bispectral index is related to the spread of spinal sensory block in patients with combined spinal and general anaesthesia

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Editor's key points

- Previous publications described a relationship between the depth of sedation as measured by the bispectral index (BIS) and spinal sensory block height in patients with light to no additional sedation.
- In this study, BIS significantly correlates with the spread of spinal sensory block under conditions of identical predicted effect-site concentration of propofol.

Background. A relationship between the depth of sedation as measured by the bispectral index (BIS) and spinal sensory block height in patients with light to no additional sedation has been described previously. The present study was designed to investigate the hypothesis that BIS values closely correlate with the spread of spinal sensory block in patients deeply sedated with an i.v. target-controlled infusion of propofol.

Methods. Subjects comprised 100 patients aged 20–64 yr and undergoing arthroscopic knee surgery. Patients were given spinal anaesthesia with bupivacaine 0.5% (3 ml). Propofol was administered to achieve a target effect-site concentration of 3.0 μg ml^{-1}. The relationship between the spinal sensory level at 15 min after spinal anaesthesia and BIS values during 1–5, 6–10, 11–15, and 16–20 min time intervals after the estimated effect-site concentration reached 3.0 μg ml^{-1} was evaluated.

Results. The sensory level of spinal analgesia significantly and strongly correlated with BIS values during each time period after the estimated effect-site concentration remained at 3.0 μg ml^{-1} ($P<0.0001$). The correlation coefficient values were 0.8 during 1–5 min, 0.844 during 6–10 min, 0.801 during 11–15 min, and 0.804 during 16–20 min time periods.

Conclusions. We demonstrated that BIS values significantly correlate with the level of spinal sensory block under deep sedation with propofol. The depth of sedation induced by spinal anaesthesia depends on the spread of spinal sensory block.

Keywords: anaesthesia, depth; anaesthetic techniques, subarachnoid; anaesthetics i.v., propofol; monitoring, bispectral index

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Spinal anaesthesia has been noted to exert sedative effects.[1–4] Clinically, patients given high spinal anaesthesia frequently exhibit a decrease in alertness, with drowsiness becoming more frequent and pronounced as the spread of spinal block becomes higher. It has also been reported that the extent of spinal anaesthesia influences the depth of sedation, measured by the bispectral index (BIS) and previously reported to be in the range of 65–75.[5 6] However, BIS is sensitive to internal or external circumstances surrounding the patient and can be affected by abrupt arousal, movement, coughing, or noise in patients with light or no additional sedation.[7–9] There have not been enough data fully quantifying the relationship between BIS and the spread of spinal block during effect compartmental controlled propofol sedation. The present study aimed at investigating the hypothesis that BIS values closely correlate with the spread of spinal anaesthesia when patients are under deep sedation.

Methods

The study was approved by the Ethics Committee of Nihon University School of Medicine (Ref: 06/0903) and written informed consent was obtained from all patients. Subjects comprised 100 patients aged 20–64 yr [mean age, 37.8 (10.9) yr] with ASA physical status I and with a BMI between 18.5 and 30 who were undergoing elective arthroscopic knee surgery without tourniquets under spinal anaesthesia combined with general anaesthesia with a duration of $<$60 min. Exclusion criteria included any history of substance abuse, known allergic disorders, current prescriptions, psychological, cardiovascular, or neurological diseases, regular consumption of alcohol, cigarettes, or both, the use

of any psychoactive medicines such as benzodiazepines, antidepressants, anticonvulsants, antihistamines, opiates, or recreational drugs during the 10 yr before the day of surgery, and the use of any medicines for common cold including antihistamines during the 3 months before the day of surgery that would be expected to affect the EEG response. The interspaces through which the spinal anaesthetic was administered (L2–3, L3–4, or L4–5) were randomly selected using sealed envelopes. Patients were also randomly allocated by selection of sealed envelopes to tilting the bed upwards, horizontal, or downwards during and for 1 min after receiving spinal anaesthesia. The angle at which the bed was tilted was left to the discretion of the attending anaesthetist to provide an adequate spinal level for the surgery. The aim of the randomization was to produce various levels of spinal anaesthesia.

No patients received any premedications. I.V. access was established in a forearm vein before arrival at the operating theatre. The operating theatre was warmed to prevent an increase in EMG activity due to shivering. Standard monitoring devices (Bedside Station, BSS-9800, Nihon Kohden Corporation, Tokyo, Japan) including ECG, non-invasive arterial pressure measurement (NIAP), and arterial oxygen saturation via pulse oximetry (Sp_{O_2}) were applied and baseline values of vital signs were obtained. All patients received an i.v. colloid solution before initiation of the spinal anaesthesia at a rate of 20 ml kg^{-1} h^{-1} and at a rate of 30 ml kg^{-1} h^{-1} after spinal injection of the local anaesthetics until completion of data collection to prevent cardiovascular depression. Thereafter, additional hydration was done by administration of a Ringer's lactate solution according to the discretion of the attending anaesthesiologists. Heart rate, NIAP, and Sp_{O_2} were continuously monitored and recorded every 2.5 min using an electronic anaesthesia chart. Before spinal anaesthesia, the BIS electrodes were placed in the fronto-temporal regions as recommended by the manufacturer (Aspect Medical Systems, Norwood, MA, USA) and connected to an EEG monitor (*A-2000 ver. 2.1*, Aspect Medical Systems) for BIS measurement. Smoothening rate was set at 15 s. To reduce skin/electrode impedance, the skin over the forehead was cleaned with an alcohol-impregnated skin wipe. The attending anaesthesiologists could view BIS and SQI values throughout the study. The BIS values were only considered valid when SQI was above 50%. If SQI was below 50% for 1 min, BIS values for that minute were excluded from data analysis. If SQI was $<$50% for longer than 20% of the total study period, all data for the patient were excluded from analysis. All data were retrieved from the monitors after completion of each anaesthesia and stored for later analysis.

All anaesthetic procedures were conducted by a board-certified anaesthetist. Once BIS readings were stable, the patient was positioned in the lateral decubitus position with his/her surgical leg dependent. Bed tilting (upwards, horizontal, or downwards) was performed before subarachnoid puncture. Subarachnoid puncture was performed with a 25 G Sprotte needle (Spinocan, B. Braun Melsungen AG, Melsungen, Germany) at the L2–3, L3–4, or L4–5 space. After injection of intradermal local anaesthesia with mepivacaine 1% (2 ml) at the puncture site, plain hyperbaric bupivacaine 0.5% (3 ml) (Marcaine 0.5%, AstraZeneca, Osaka, Japan) (15 mg) was administered into the subarachnoid space. Cerebrospinal fluid aspiration (0.1 ml) was done to confirm correct needle placement before and after spinal drug administration. The bed tilting was maintained until 1 min after administration of the anaesthetic agent, whereafter the patient was turned to the supine position. Sensory block height was evaluated bilaterally using a pinprick test with the sharp tip of a safety pin every 1 min until 15 min after the initiation of the spinal anaesthesia. Bilateral sensory block level was segmentally confirmed to remain at the same level with three consecutive evaluations at 15 min after the administration. Complete motor block of the lower limbs was also confirmed at 15 min after subarachnoid drug administration. If the patients were able to flex either knees or ankles or the sensory block did not extend rostral to the operative site, spinal anaesthesia was readministered and the patient was excluded. Arterial pressure was measured every minute after spinal administration. Hypotension and bradycardia were defined as systolic arterial pressure below 80 mm Hg and heart rate below 45 beats min^{-1}, respectively, according to the definition by Reich and colleagues.[10] If hypotension or bradycardia persisted for more than 1 min, ephedrine or atropine, respectively, was administered i.v. and the patient was excluded from the study since these drugs affect the central nervous system. In addition, all patients had previously been informed that the spread of spinal anaesthesia could reach thoracic or cervical levels due to the bed tilting. If patients complained of any symptoms due to spinal anaesthesia, for example, nausea or dyspnoea, they were scheduled to be immediately sedated and excluded from the study. After checking the adequacy of spinal anaesthesia, a urinary catheter and rectal thermometer was inserted. The rectal temperature was maintained at 36.0–37.0°C using a forced-air warming blanket.

Patients were sedated with i.v. administration of propofol after confirmation of the level of the sensory block. All patients received plasma target-controlled infusion using the Diprifusor syringe pump (TERUMO Inc., Tokyo, Japan).

General anaesthesia was induced with i.v. propofol and vecuronium after preoxygenation. The target plasma concentration of propofol was initially set at 6.0 μg ml^{-1}. After loss of consciousness and confirmation of the absence of a difficult airway, vecuronium bromide was administered i.v. at a dose of 1 mg kg^{-1} to facilitate the insertion of a laryngeal mask airway (LMA) and controlled ventilation of the lungs. No further doses of vecuronium were administered. The LMA was inserted 2.5 min after the administration of propofol, and the plasma target concentration of propofol was subsequently reduced to 3.0 μg ml^{-1}. If LMA insertion could not be successfully completed at the first attempt, the target concentration of propofol was maintained at 6.0 μg ml^{-1} until successful insertion was achieved and the patient was excluded. The patient's lungs were ventilated