J Anesth (2007) 21:516–518
DOI 10.1007/s00540-007-0540-z

Journal of
Anesthesia

Short communication

Infusion rate of propofol and jugular venous oxygen saturation

Yasunori Haranishi[1], Yasuhiro Kuroda[2], Yasutoshi Matayoshi[1], Hisashi Tamura[1], Hideo Oka[1], Kumiko Nakamura[1], Toru Gohara[1], Yoshiaki Nagusa[1], and Kaori Yoshitomi[1]

[1]Department of Anesthesia and Intensive Care Medicine, Yamaguchi Grand Medical Center, Yamaguchi, Japan
[2]Department of Emergency and Critical Care Medicine, Kagawa University Hospital, 1750-1 Miki, Kida, Kagawa 761-0793, Japan

Abstract
We compared jugular venous blood oxygen saturation (Sj_{O_2}) and the arterial-to-jugular-bulb venous oxygen content difference (AjD_{O_2}) between bispectral index (BIS) values of 40 and 60, adjusted by the infusion rate of propofol. Eighteen postoperative neurosurgical patients (Glasgow Coma Scale [GCS] scores, 11–15) were enrolled. Normocapnia, normothermia, and a mean arterial blood pressure greater than 70 mmHg were maintained. At BIS values of 40 and 60, hemoglobin, oxygen saturation, and the oxygen partial pressure of arterial and jugular venous blood were measured. Sj_{O_2} at BIS40 (58 ± 9%) was significantly ($P < 0.01$) lower than that at BIS60 (63 ± 10%), and AjD_{O_2} at BIS40 (6.3 ± 1.5 ml·dl^{-1}) was significantly ($P < 0.01$) higher than that at BIS60 (5.7 ± 1.5 ml·dl^{-1}; mean ± SD). At BIS40, status defined as Sj_{O_2} less than 50% was observed in 3 patients, while this status was observed in 1 patient at BIS60. In conclusion, in patients with postoperative neurosurgical surgery (GCS scores, 11–15), decreases of propofol infusion to adjust the BIS value from 40 to 60 increase the cerebral oxygen balance.

Key words Propofol · Jugular venous oxygen saturation · Bispectral index

Propofol reduces cerebral blood flow (CBF) and/or the cerebral metabolic rate for oxygen (CMR_{O_2}) in humans [1–5]. These reports suggested that the cerebral oxygen balance (CBF divided by CMR_{O_2}) was decreased [1–3] or was not changed by propofol [4,5]. Iwata et al. [6] reported that no changes in hemoglobin oxygen saturation (S_{O_2}) in the jugular venous blood (Sj_{O_2}) or in the incidence of desaturation were observed between different bispectral index (BIS) values (43 ± 12 vs 57 ± 9) during propofol infusion. A BIS value of less than 40 reflects a deep hypnotic level, while a value of 60 reflects a moderate hypnotic level in critically ill patients [7]. In the present study, we measured Sj_{O_2} and the arterial-to-jugular-bulb venous oxygen content difference (AjD_{O_2}) in postneurosurgical patients sedated to adjust BIS value 40 or 60 by propofol infusion.

This study protocol was approved by the Ethics Committee for Human Study of the Yamaguchi Grand Medical Center, and informed consent was obtained from each patient or relative. Eighteen elective neurosurgical patients with an American Society of Anesthesiologists physical status of 1–3 were enrolled. Patients were excluded from this study if they had renal, hepatic, or cardiac dysfunction, or hypertension. Anesthesia was maintained with isoflurane, fentanyl, and vecuronium bromide. Once the removed cranium was replaced, propofol infusion was started at a rate of 6 mg·kg^{-1}·h^{-1} and the isoflurane was stopped. At the end of surgery, the end-tidal isoflurane percentage was 0%.

After transfer to the intensive care unit (ICU), continuous sedation with propofol and artificial ventilation were performed from 12 to 24 h after the end of the operation. A single-lumen catheter (Medicut LCV-UK Kit; Tyco Healthcare, Tokyo, Japan) was inserted into the internal jugular vein on the same side as the surgical procedure, with the tip of the catheter positioned in the jugular bulb. BIS monitoring was then started (BIS A-1050; software ver.3.4; Aspect Medical Systems, Norwood, MA, USA). The rate of propofol infusion was titrated, aiming for an initial BIS value of between 38 and 43 (BIS40). The rate of propofol infusion was then decreased until a BIS value of between 58 and 63 (BIS60) was reached. For each BIS value, both arterial and jugular bulb venous blood samples were obtained after a 15-min stabilization period. These samples were analyzed for partial pressure of oxygen (P_{O_2}) and of carbon dioxide (P_{CO_2}), using a blood gas analyzer (ABL505; Radiometer, Copenhagen, Denmark) at 37.0°C. S_{O_2} and hemoglobin concentrations were

Address correspondence to: Y. Kuroda
Received: September 7, 2006 / Accepted: May 17, 2007

Table 1. Demographic data

Patient no.	Age (years)	Sex	Diagnosis	GCS (EVM)
1	55	F	Tumor (rt temporal lobe)	15 (E4V5M6)
2	78	F	Tumor (rt c-p angle)	13 (E3V5M5)
3	65	F	Tumor (rt c-p angle)	15 (E4V5M6)
4	61	F	Tumor (rt occipital lobe)	15 (E4V5M6)
5	74	M	Tumor (rt cerebellum)	15 (E4V5M6)
6	62	M	Tumor (sella turcica)	13 (E3V4M6)
7	61	F	Unruptured aneurysm (lt MCA)	15 (E4V5M6)
8	58	F	Unruptured aneurysm (lt MCA)	15 (E4V5M6)
9	66	F	Unruptured aneurysm (rt ICA)	15 (E4V5M6)
10	50	F	Unruptured aneurysm (lt MCA)	15 (E4V5M6)
11	48	F	Unruptured aneurysm (lt MCA)	15 (E4V5M6)
12	64	M	Unruptured aneurysm (a-com)	15 (E4V5M6)
13	30	M	Subarachnoid hemorrhage (a-com)	12 (E3V4M5)
14	65	M	Rt cerebral hemorrhage	13 (E4V4M5)
15	85	F	Rt acute subdural hematoma	11 (E3V4M4)
16	61	F	Lt acute epidural hematoma	13 (E3V5M5)
17	60	F	Brain abscess (lt temporal lobe)	15 (E4V5M6)

c-p angle, cerebellopontine angle; MCA, middle cerebral artery; rt, right; lt, left; ICA, internal carotid artery; a-com, anterior communicating artery; GCS, Glasgow Coma Scale (after tracheal extubation)

Table 2. Infusion rate of propofol and physiological variables during BIS measurements in the ICU

	BIS40	BIS60
Propofol ($mg \cdot kg^{-1} \cdot h^{-1}$)	6.1 ± 2.2	3.6 ± 1.4*
Mean arterial blood pressure (mmHg)	87 ± 11	93 ± 15
Heart rate (min^{-1})	72 ± 13	71 ± 13
Body temperature (°C)	36.2 ± 0.2	36.5 ± 0.2
Arterial pH	7.45 ± 0.02	7.43 ± 0.03
Pa_{O_2} (mmHg)	169 ± 22	165 ± 17
Pa_{CO_2} (mmHg)	38 ± 2	38 ± 2
Hemoglobin ($g \cdot dl^{-1}$)	10.8 ± 1.6	10.8 ± 1.6

*$P < 0.01$, BIS40 vs BIS60
Data values are presented as means ± SD
Pa_{O_2}, arterial partial pressure of oxygen; Pa_{CO_2}, arterial partial pressure of carbon dioxide

measured spectrophotometrically (OSM3; Radiometer). The AjD_{O_2} values were calculated using the following equation:

$$AjD_{O_2} = 1.34 \times Hb \times (Sa_{O_2} - Sj_{O_2}) + 0.0031 \times (Pa_{O_2} - Pj_{O_2})$$

where Sa_{O_2} is the arterial S_{O_2} and Pj_{O_2} is the jugular venous P_{O_2}. Mean arterial blood pressure was maintained at a value greater than 70 mmHg, using a continuous infusion of phenylephrine ($0.1–1.0\,\mu g \cdot kg^{-1} \cdot min^{-1}$), if necessary. Pa_{O_2} (100–150 mmHg), normocapnia (Pa_{CO_2} 35–45 mmHg), and normothermia (36.0°C–37.0°C) were maintained. The hemoglobin (Hb) concentration was preserved at over 9.0 g/dl. Comparisons of the parameters at BIS40 and BIS60 were assessed using a paired *t*-test. A *P* value of less than 0.05 was considered statistically significant. Values are expressed as means ± SD.

One patient was excluded from this study because retrograde displacement of the catheter inserted into the internal jugular vein was confirmed by radiography. Demographic data are shown in Table 1. The Glasgow coma scale (GCS) scores after tracheal extubation were between 11 and 15.

The infusion rate of propofol and the physiological variables of the patients during the BIS measurements are shown in Table 2. No significant differences between these values at BIS40 and BIS60 were noted, except for the infusion rate of propofol. None of the patients received phenylephrine. The Sj_{O_2} and AjD_{O_2} values of the patients are shown in Fig. 1. Sj_{O_2} at BIS40 (58 ± 9%) was significantly ($P < 0.01$) lower than that at BIS60 (63 ± 10%), and AjD_{O_2} at BIS40 ($6.3 \pm 1.5\,ml \cdot dl^{-1}$) was significantly ($P < 0.01$) higher than that at BIS60 ($5.7 \pm 1.5\,ml \cdot dl^{-1}$). At BIS40, three patients had an Sj_{O_2} value of less than 50%, while one patient had an Sj_{O_2} value of

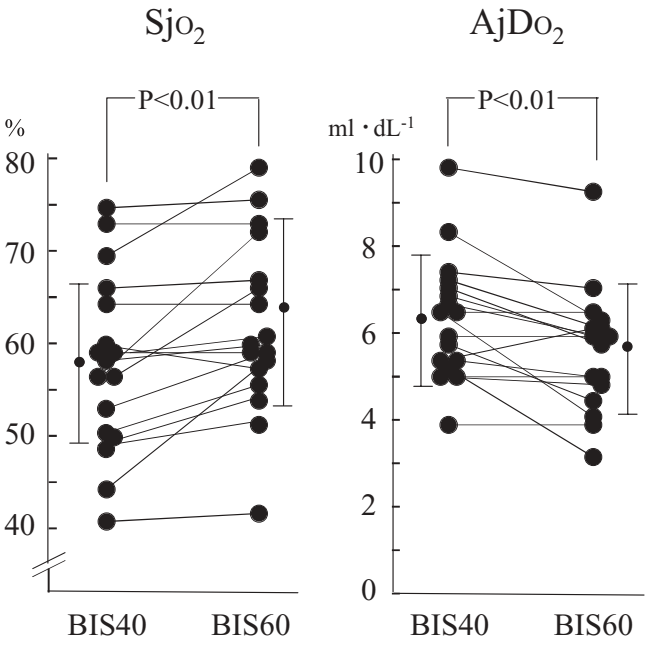

Fig. 1. Jugular venous blood oxygen saturation (S_{jO_2}) and arterial-to-jugular-bulb venous oxygen content difference ($A_jD_{O_2}$) at *BIS40* or *BIS60*. By reducing the hypnotic level, S_{jO_2} increased and $A_jD_{O_2}$ decreased significantly ($P < 0.01$). Values are expressed as means ± SD

less than 50% at BIS60. None of the patients showed status defined as an S_{jO_2} of less than 40% at either BIS value. Patient no. 15 exhibited right-side hemiplegia after tracheal extubation in the ICU.

Increases of S_{jO_2} and/or decreases of $A_jD_{O_2}$ suggest increased cerebral oxygen balance [8]. Our results agreed with those of several studies demonstrating that the reduction in CBF was greater than the reduction in CMR_{O_2} during propofol infusion [1–3]. Stephan et al. [1] demonstrated that CBF and CMR_{O_2} were decreased by 51% and 36%, respectively, after induction of propofol at $12\,mg\cdot kg^{-1}\cdot h^{-1}$ in patients without neurologic disorders. Vandesteene et al. [2] reported that three-step infusion of propofol (21, 12, then $6\,mg\cdot kg^{-1}\cdot h^{-1}$) decreased CBF but not CMR_{O_2} in patients without neurologic disorders. Jansen et al. [3] showed that S_{jO_2} values of less than 50% were observed in half of the patients with brain tumors (normal intracranial pressure [ICP]), and three of ten patients had an S_{jO_2} of less than 40% under propofol anesthesia ($6\,mg\cdot kg^{-1}\cdot h^{-1}$). However, other reports suggested that the cerebral oxygen balance was not changed by propofol infusion at $6\,mg\cdot kg^{-1}\cdot h^{-1}$ [4] in patients without neurologic disorders, or at $9\,mg\cdot kg^{-1}\cdot h^{-1}$ in comatose patients [5]. Iwata et al. [6] reported that no changes of S_{jO_2} or in the incidence of desaturation were observed between different BIS values (43 ± 12 vs 57 ± 9) during propofol anesthesia with craniotomy. Relatively large doses of analgesies (epidural anesthesia [4], phenoperidine [5], fentanyl [6]) were added as basal anesthesia in those reports, suggesting no change of cerebral oxygen balance by propofol, in comparison with the reports suggesting decreased cerebral oxygen balance by propofol [1–3]. We speculate that the large dose (5 mg) of fentanyl used by Iwata et al. [6] may have affected the BIS value, which would have produced the discrepancy in BIS values between their results and our results.

In conclusion, in patients with postoperative neurosurgical surgery (GCS scores, 11–15), decreases of propofol infusion to adjust the BIS value from 40 to 60 increase the cerebral oxygen balance.

References

1. Stephan H, Sonntag H, Schenk HD, Kohlhausen S (1987) Effects of "Disoprivan" on cerebral blood flow, cerebral oxygen consumption and cerebral vascular reactivity. Anaesthesist 36:60–65
2. Vandesteene A, Trempont V, Engelman E, Deloof T, Focroul M, Schoutens A, de Rood M (1988) Effect of propofol on cerebral blood flow and metabolism in man. Anaesthesia 1988; 43(Suppl):42–43
3. Jansen GF, van Praagh BH, Kedaria MB, Odoom JA (1999) Jugular bulb oxygen saturation during propofol and isoflurane/nitrous oxide anesthesia in patients undergoing brain tumor surgery. Anesth Analg 89:358–363
4. Oshima T, Karasawa F, Satoh T (2002) Effects of propofol on cerebral blood flow and the metabolic effect of oxygen in humans. Acta Anaesthesiol Scand 46:831–835
5. Pinaud M, Lelausque JN, Chetanneau A, Fauchoux N, Menegalli D, Souron R (1990) Effects of propofol on cerebral hemodynamics and metabolism in patients with brain trauma. Anesthesiology 73:404–409
6. Iwata M, Kawaguchi M, Inoue S, Takahashi M, Horiuchi T, Sakaki T, Furuya H (2006) Effects of increasing concentrations of propofol on jugular venous bulb oxygen saturation in neurosurgical patients under normothermic and mildly hypothermic conditions. Anesthesiology 104:33–38
7. Nasraway SA Jr, Wu EC, Kelleher RM, Yasuda CM, Donnelly AM (2002) How reliable is the bispectral index in critically ill patients? A prospective, comparative, single-blinded observer study. Crit Care Med 30:1483–1487
8. Robertson CS, Gopinath SP, Goodman JC, Contant CF, Valadka AB, Narayan RK (1995). Sjv_{O_2} monitoring in head-injured patients. J Neurotrauma 12:891–896