Correspondence

Hyperventilation increases the induction dose of propofol

To the Editor:
The induction dose of propofol may be affected by many factors such as age, American Society of Anesthesiologists' (ASA) classification, plasma protein level, ect.[1,2] but the effect of hyperventilation has not been established. After approval from the Human Research Committee of our hospital and informed consent from each patient was obtained, 17 adult patients scheduled for thyroid adenoma or breast mass surgery (ASA physical status I–II) were randomly divided into a hyperventilation group (n = 8) and a normoventilation group (n = 9). Patients were premedicated with sodium phenobarbital 100 mg and atropine 0.5 mg intramuscularly 60 min before anesthesia. None of the patients had any cardiovascular, central nervous system, or metabolic disease. End-tidal CO_2 ($PETCO_2$) was monitored during induction. As the propofol was infused at a rate of 33.3 $mg{\cdot}min^{-1}$ with a micro-infusion pump via the main saphenous vein and continuously flushed with lactated Ringer's solution, the patient was asked to count in the normoventilation group, and to hyperventilate for 90 sec before beginning to count in the hyperventilation group. The propofol dose required for the patient to cease counting was recorded as the minimum induction dose.

Age, weight, gender, ASA classification, preoperative albumin, globulin, total protein, urea, hemoglobin concentration[1,2] and baseline $PETCO_2$ (37.3 ± 2.4 mmHg *vs* 38.2 ± 1.5 mmHg) were comparable between groups (P > 0.05). The $PETCO_2$ decreased to 25.9 ± 2.3 mmHg as the patients hyperventilated. The minimum induction dose of propofol was 1.66 ± 0.24 $mg{\cdot}kg^{-1}$ in the hyperventilation group and 1.19 ± 0.42 $mg{\cdot}kg^{-1}$ in the normoventilation group (P < 0.01).

Hyperventilation results in hypocapnia, which may decrease cerebral blood flow.[3] Correspondingly, a smaller fraction of the infused propofol is transported to the central nervous system. On the other hand, voluntary hyperventilation may increase cardiac output.[4] This decreases the peak arterial and peak brain propofol levels,[5] and more propofol is needed to reach the brain concentration at which the patient loses consciousness. The change of protein binding of propofol resulting from hyperventilation could also be part of the explanation.

We conclude that hyperventilation increases the induction dose of propofol.

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Continuous epidural infusion of ropivacaine with sufentanil 1.5 $\mu g{\cdot}mL^{-1}$ for postoperative analgesia after total knee replacement

To the Editor:
We report a prospective, randomized, double-blinded pilot study on the epidural combination of ropivacaine with 1.5 $\mu g{\cdot}mL^{-1}$ sufentanil for postoperative analgesia after total knee replacement (TKR).

Despite the limited number of patients (n = 10), we present our results as they support the continuous epidural infusion of ropivacaine 0.2% with 1.5 $\mu g{\cdot}mL^{-1}$ sufentanil at a time when continuous three-in-one block is a popular method for postoperative analgesia after TKR.[1]

Our study was designed to assess the clinical efficacy of postoperative continuous epidural infusion of ropivacaine 0.1% *vs* 0.2% both combined with 1.5 $\mu g \cdot mL^{-1}$ sufentanil. After written informed consent, ten patients ASA I–III undergoing elective TKR were enrolled in the investigation. Lumbar epidural anesthesia using 0.75% ropivacaine was combined with either propofol sedation or general anesthesia for surgery. On arrival in the recovery room, five patients received ropivacaine 0.1% with 1.5 $\mu g \cdot mL^{-1}$ sufentanil (Group A), and five patients received ropivacaine 0.2% with 1.5 $\mu g \cdot mL^{-1}$ sufentanil (Group B) at a rate of 5–9 $mL \cdot hr^{-1}$. All patients had access to ***iv*** piritramide via a patient-controlled analgesia device. Patients were examined eight hours, 20 hr, 32 hr, and 44 hr postoperatively by the same anesthesiologist blinded to group assignment. Repeated measurement ANOVA was performed for pain scores and opioid consumption. Data are presented as mean ± SEM.

Cumulative opioid rescue medication was tenfold less in Group B than in Group A (6 ± 3 *vs* 65 ± 23 mg, $P = 0.001$). Patients in Group B had lower visual analogue scale scores on a scale from 0–100 mm at rest (4 ± 6 mm *vs* 38 ± 6 mm, $P = 0.007$) and on movement (9 ± 7 mm ***vs*** 53 ± 7 mm, $P = 0.003$) than patients in Group A. Motor block was negligible in both groups. Three patients (two in Group A, one in Group B) experienced nausea, one patient in Group A experienced vomiting and itching. This patient rated quality of pain management as fair, the other nine patients rated quality of pain management as excellent or good. No severe side effects, such as respiratory depression were observed over our study period of 44 hr.

Our pilot data indicate that ropivacaine 0.2% with 1.5 $\mu g \cdot mL^{-1}$ sufentanil seems to be more effective than 0.1% ropivacaine with 1.5 $\mu g \cdot mL^{-1}$ sufentanil for preventing pain after TKR.

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Increased S-100 B protein levels in a patient undergoing Cesarean delivery in the presence of prolonged hemorrhagic shock

To the Editor:

Serum S-100 B protein is an early and sensitive marker of hypoxic brain damage.[1,2] Consequently, levels of this protein may be correlated to neurological outcome after severe bleeding and anemia that decrease cerebral oxygen delivery to critical levels. Since the peak levels of S-100 B protein occur on the third day after a stroke,[3,4] we measured S-100 B protein levels three days after severe hemorrhagic shock and immediately thereafter. Two women (one with an anterior placenta previa and the other with anterior vasa previa) at risk from hemorrhage were scheduled for Cesarean delivery under combined spinal-epidural anesthesia. On admission, their hemoglobin concentrations were 11.1 and 9.6 $g \cdot dL^{-1}$, respectively. In both patients, massive bleeding started immediately after amniotomy. The patient with a placenta previa suffered from decreased systolic blood pressure in the range of 35–55 mmHg, which persisted for 125 min. In contrast, the systolic blood pressure of the patient with vasa previa decreased to 65 mmHg for only two minutes, followed by rapid recovery to 80 mmHg. Although blood had been cross-matched prior to the operation, the hemoglobin level immediately before blood transfusion in both patients was similarly very low (39 $g \cdot L^{-1}$) because the blood was sent for irradiation after massive bleeding was observed. When hemorrhagic shock occurred, both cases were intubated and ventilated mechanically, and hysterectomy was performed because the uterus failed to contract. In the patient with prolonged shock, S-100 B protein levels analyzed by chemiluminescent immunoassay (SRL Inc., Tokyo Japan) immediately after the operation and on the third postoperative day were 0.24 and 1.04 $ng \cdot mL^{-1}$, whereas in the patient without shock, these levels were 0.1 and 0.07 $ng \cdot mL^{-1}$, respectively. The S-100 B protein level in the patient with prolonged shock was above that reported in patients with unilateral supratentorial cerebral infarction (0.5 $ng \cdot mL^{-1}$).[2] Slight extrapyramidal symptoms were noted postoperatively only in the patient with prolonged shock. Brain damage was undetectable by computerized tomography or by magnetic resonance imaging. Our preliminary observation warrants further studies to clarify the significance of increased levels of serum S-100 B protein in severe shock.