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]