The effect of propofol infusion on minimum alveolar concentration of sevoflurane for smooth tracheal intubation

TSUYOSHI SATSUMAE1, SEJI WATANABE1, and HIROSHI YAMAGUCHI2

1Department of Anesthesia, Pain Clinic, and Clinical Toxicology, Mito Saiseikai General Hospital, Mito, Ibaraki, Japan
2Department of Anesthesia and Critical Care Medicine, Iwaki Kyoritsu General Hospital, Iwaki, Fukushima, Japan

Introduction

The plasma propofol concentration that resulted in a 95% probability of no response during tracheal intubation (Cp95 intubation) to be 34.8\( \mu \text{g} \cdot \text{ml}^{-1}\). However, with this concentration, patients experienced severe hypotension and their systolic blood pressure decreased to 70.3 mmHg [1]. Moreover, a very large dose of propofol appeared to be required, which did not seem to be practical. It is assumed that smooth tracheal intubation cannot be achieved with propofol alone at a clinically acceptable dose.

In contrast, sevoflurane alone makes smooth tracheal intubation possible, without a muscle relaxant, at a clinically acceptable dose. Previously, we reported the minimum alveolar concentration necessary for smooth tracheal intubation (MACEI) of sevoflurane to be 4.52% [2]. However, with a high concentration of sevoflurane, it takes a long time to reach equilibrium between cerebral and alveolar gas tensions.

In terms of upper airway integrity, propofol is an excellent agent [3]. However, tracheal intubation with a combination of sevoflurane and propofol, without a muscle relaxant, has not yet been tried. We hypothesized that a combination of sevoflurane plus propofol would allow smoother tracheal intubation without a muscle relaxant than either agent alone. In this study, we tested the effect of propofol infusion on the MACEI of sevoflurane in patients undergoing elective surgery under general anesthesia.

Subjects and methods

The study protocol was approved by the Ethics Committee of Mito Saiseikai General Hospital, and written informed consent was obtained from each patient. Sixty-nine adult patients (American Society of Anesthesiologists [ASA] physical status I; age range, 30 to 49 years) were randomly assigned to one of three groups according to the agents used for tracheal intubation (n = 23 for each group): the SP group, in whom the intubation was attempted under sevoflurane plus propofol infusion; the S group, tracheal intubation under sevoflurane alone; and the P group, tracheal intubation under propofol infusion alone. Anesthesia was induced with propofol 2.5 mg·kg\(^{-1}\) i.v. bolus. Prior to the tracheal intubation attempt, propofol infusion, 10 mg·kg\(^{-1}\)·h\(^{-1}\), was given for 15 min in the SP and P groups, and sevoflurane equilibration was established in the SP and S groups. All tracheal intubation attempts were made 15 min after anesthetic induction. The end-tidal sevoflurane concentration at which tracheal intubation was attempted was predetermined by the up-and-down method (with 0.5% as a step size). MACEI was determined using a logistic regression test.

Results

The MACEI of sevoflurane was 1.73% in the SP group, and 2.99% in the S group. Laryngoscopy was not possible in the P group patients.

Conclusion

Propofol infusion reduced sevoflurane MACEI. This finding suggests that propofol would be an excellent adjuvant to use for tracheal intubation.

Key words
Anesthetic techniques: tracheal intubation · Anesthetics, intravenous: propofol · Anesthetics, volatile: sevoflurane
years) who were undergoing elective surgery under general anesthesia were enrolled in this study. The patients were randomly assigned to one of three groups according to the agents used for tracheal intubation (n = 23 for each group): the SP group, in whom tracheal intubation was attempted under sevoflurane plus propofol infusion; the S group, in whom the intubation was attempted under sevoflurane alone; and the P group, in whom the intubation was attempted under propofol infusion alone.

Blood pressure was determined indirectly, and electrocardiogram, arterial hemoglobin oxygen saturation (SpO2), and rectal temperature were continuously monitored, using a patient monitor system (BP-508; Colin, Komaki City, Aichi, Japan). Acetated Ringer’s solution was infused intravenously, at the rate of 10 ml·kg⁻¹·h⁻¹ as a maintenance fluid during the study period. In all the patients, anesthesia was induced using propofol, 2.5 mg·kg⁻¹, i.v. bolus, and a laryngeal mask airway (LMA) was inserted. In the SP and S groups, the patients were connected to an anesthesia ventilator, which delivered a tidal volume of 10 ml·kg⁻¹ at 12 bpm in a controlled ventilation mode, and they inhaled sevoflurane of a predetermined concentration in pure oxygen. The P group patients inhaled pure oxygen under manually assisted ventilation, which was provided because of the possibility of the patient fighting against the mechanical ventilator. Breath-by-breath inspired/end-tidal sevoflurane and carbon dioxide concentrations were measured with a gas monitor (RASCAL-1, Albion Instruments, Salt Lake City, UT, USA). We employed a semiclosed anesthetic breathing system with a fresh gas flow of 61·min⁻¹. For the measurement of anesthetic concentration, respiratory gases were sampled from an angle piece fitted at the distal end of the LMA. End-tidal sevoflurane concentration at laryngoscopy and tracheal intubation was predetermined by an up-and-down method (0.5% as the step size), starting with 2% (SP group) or 4% (S group). End-tidal CO₂ and rectal temperature were maintained at around 30 mmHg and 35.5°C or above, respectively.

In the SP and P groups, in addition to the induction dose, propofol, 10 mg·kg⁻¹·h⁻¹, was infused for 15 min prior to the tracheal intubation attempt. In the SP and S groups, when 90% or more of the predetermined end-tidal sevoflurane concentration had been achieved and maintained for at least 10 min under mechanical ventilation, the LMA was removed, and tracheal intubation, using a cuffed tracheal tube (Portex, 7.0-mm internal diameter [ID]), was attempted by conventional indirect laryngoscopy. The P group patients inhaled pure oxygen under manually assisted ventilation for the same period prior to the intubation attempt. All tracheal intubation attempts were made 15 min after anesthetic induction, using bolus propofol.

The patient were described as either “unresponsive” or “responsive” to laryngoscopy and tracheal intubation. When laryngoscopy was uneventful, and coughing and bucking did not occur after tracheal cuff inflation, this was considered “unresponsive”. When we encountered difficulty in mouth-opening, gross purposeful muscular movements, vocal cord movements during laryngoscopy, or bucking after cuff inflation, this was considered “responsive”. A single anesthesiologist, who was not blinded to the grouping, attempted all laryngoscopies and tracheal intubations, while another anesthesiologist, who was blinded to the grouping, determined the presence or absence of any responses.

Arterial blood samples were taken from the radial artery, just after the intubation attempt, for the determination of blood propofol concentration in the ten “unresponsive” patients in the SP and S groups, and in ten patients who were randomly selected from the P group. Blood samples were stored at 5°C until extraction and assay. Blood propofol concentration was determined using high-performance liquid chromatography.

Statistical analyses were done using analysis of variance (ANOVA) for differences in demographic data. Analysis of the probability of unresponsiveness versus end-tidal sevoflurane concentration, the maximum likelihood estimators of the model parameters, and goodness of fit were tested using a logistic regression test (SAS proprietary software; SAS Institute, Chicago, IL, USA), which provided the best fitting sigmoid curve. A probit test (SAS proprietary software, SAS Institute) was used to obtain 95% confidence limits. Intraoperative awareness was recorded. P < 0.05 was considered statistically significant. All values were expressed as means ± SD.

**Results**

There were no significant differences in the demographic data among the three groups (Table 1). LMA was inserted smoothly in all the patients after induction with propofol, 2.5 mg·kg⁻¹ (i.v.). Arterial blood pressure and heart rate before induction, and before and after tracheal intubation attempts, in the SP and S groups, are shown in Fig. 1. The mean end-tidal CO₂ value for all patients was 30 ± 2 mmHg. Mean body temperature for all patients during the study was 36.1 ± 0.6°C.

The total amount of propofol administered before intubation attempts was 5.0 mg·kg⁻¹ in the SP and P groups, and 2.5 mg·kg⁻¹ in the S group. The responses in the 23 consecutive patients in each of the SP and S groups and the end-tidal sevoflurane concentration in oxygen are shown in Fig. 2. A finding of “responsive” was observed in all the patients in the P group. The reasons for responsiveness are listed in Table 1.