J Anesth (2005) 19:311–314
DOI 10.1007/s00540-005-0333-1

Journal of
Anesthesia

Combined spinal-propofol anesthesia with noninvasive positive-pressure ventilation

Hiroshi Ohmizo[1], Tomoko Morota[1], Yasuhiro Seki[1], Takahisa Miki[1], and Hiroshi Iwama[2]

[1]Department of Anesthesiology, Central Aizu General Hospital, Aizuwakamatsu, Japan
[2]Trauma and Critical Care Center, Teikyo University School of Medicine, 2-11-1 Kaga, Itabashi-ku, Tokyo 173-8606, Japan

Abstract
Twenty-three adult patients undergoing repair of inguinal hernia under spinal anesthesia received propofol infusion for sedation with the assist of noninvasive positive-pressure ventilation (NPPV). Circulatory and respiratory parameters, such as percutaneous oxygen saturation, transcutaneous carbon dioxide tension, respiratory rate, tidal volume, blood pressure, and heart rate, were maintained within physiological ranges during the anesthesia. There were no adverse effects. These findings suggest that the application of NPPV in patients receiving propofol infusion for sedation is clinically practicable during anesthesia.

Key words Spinal anesthesia · Propofol anesthesia · Airway · Noninvasive positive-pressure ventilation · Transcutaneous carbon dioxide tension

Introduction

Patients undergoing relatively minor surgery under spinal or epidural anesthesia alone occasionally request sedation or sleep during surgery, and certain sedatives such as midazolam or propofol are administered intravenously [1]. However, sometimes a sufficiently sedated condition is not obtained because of upper airway obstruction or intraoperative awareness. In recent years, we have determined an infusion dosage of propofol that achieves preservation of spontaneous breathing [2,3] and have developed a minimally invasive technique of airway maintenance [4] for surgical patients under epidural anesthesia. An infusion rate of 5 mg kg^{-1} h^{-1} for propofol (P5), giving a blood concentration of 2.0–2.5 μg ml^{-1}, is clinically sufficient to preserve spontaneous breathing; and noninvasive positive-pressure ventilation (NPPV) via the nose can be applied as airway maintenance. In this report, various clinical parameters were examined in patients undergoing "combined spinal-propofol anesthesia with NPPV."

Address correspondence to: H. Iwama
Received: January 14, 2005 / Accepted: April 30, 2005

Patients and methods

This study was approved by the institutional committee of our hospital. A total of 23 adult patients, classified as American Society of Anesthesiologists (ASA) physical status 1 or 2 with no evidence of upper airway, esophageal, or gastric abnormality, and who underwent unilateral or bilateral repair of an inguinal hernia under spinal anesthesia and requested sleep during surgery provided informed consent to undergo our anesthetic protocol.

The anesthetic protocol was based on our previous reports [4,5] as follows: Premedication consisted of atropine 0.008 mg kg^{-1} and butorphanol 0.5–1.0 mg intramuscularly 30 min before entering the operating theater. A physiological monitor with an autosphygmomanometer, percutaneous oxygen saturation (Sp_{O_2}) and cardiograph (BP-680EV; Colin, Komaki, Japan) was attached. The sensor probe was put on the medial upper arm at a sensor temperature of 43.5°C (Cutaneous P_{O_2}/P_{CO_2} Monitor 9000; Kohken Medical, Tokyo, Japan) for measurement of transcutaneous carbon dioxide tension (TcP_{CO_2}). Subsequently, spinal anesthesia was initiated by injecting 0.5% hyperbaric bupivacaine via a 25-gauge spinal needle into the L4–5 or L3–4 interspace. Ten minutes later, when the TcP_{CO_2} values were stable, the upper anesthetized dermatome level of thermohypesthesia was assessed, and the values of Sp_{O_2}, TcP_{CO_2}, respiratory rate, systolic blood pressure, and heart rate were recorded. Tidal volume was also measured using a spirometer (Respirometer; Kawaguchiko Seimitsu, Yamanashi, Japan). Then a nasal strip (Breathe Right; CNS, Chanhassen, MN, USA) was attached to the bridge of the nose, and a nasal mask (Simplicity Nasal

Mask; Respironics, Murrysville, PA, USA), not connected to a ventilator, was secured on the nose. The NPPV ventilator (BiPAP Vision; Respironics) was set at an inspiratory positive airway pressure (IPAP) and expiratory positive airway pressure (EPAP) of 14 cm H_2O and 8 cm H_2O, respectively, at a respiratory rate of 10 breaths min^{-1}, a timed inspiration of 2 s, a rise time of inspiratory positive pressure of 0.4 s, and an inspiratory oxygen fraction of 0.35. The flow trigger sensitivity level to detect spontaneous breathing was fixed to autotrack sensitivity. This NPPV ventilator shows an estimated tidal volume calculated by leak and inspiratory flow volumes. During delivery of 6 $l \cdot min^{-1}$ oxygen via the nasal mask, 1% propofol 1.5–2.0 $mg \cdot kg^{-1}$ was infused as a bolus dose at a rate of 200 $mg \cdot min^{-1}$, followed by P5 (5 $mg \cdot kg^{-1} \cdot h^{-1}$ propofol) as an infusion dose. After patients fell unconscious, the ventilator was connected to the nasal mask with the patient's head in a neutral or slight extension position. If hypotensive reactions occurred, the vasopressor agent ephedrine was injected intravenously. When the patient moved during anesthesia, propofol 0.5 $mg \cdot kg^{-1}$ at a rate of 200 $mg \cdot min^{-1}$ was injected additionally. After completion of surgery, propofol infusion was discontinued. When the patients opened their eyes to verbal commands, the nasal mask was removed. The values of Sp_{O_2}, TcP_{CO_2}, respiratory rate, tidal volume, systolic blood pressure, and heart rate were recorded 5, 15, 30, 45, and 60 min after initiation of propofol infusion and 5 min after removal of the nasal mask.

The time course of Sp_{O_2}, TcP_{CO_2}, respiratory rate, tidal volume, systolic blood pressure, and heart rate were analyzed statistically by repeated measures one-way analysis of variance, followed by Fisher's protected least significant difference for multicomparison, in which the preinduction value was compared with other values. $P < 0.05$ was considered significant.

Results

The age, weight, height, body mass index, male/female, and ASA physical status 1/2 of the patients were 52 ± 18 years (22–81 years), 62 ± 9 kg (46–82 kg), 162 ± 10 cm (148–182 cm), 23.6 ± 2.6 $kg \cdot m^{-2}$ (19.0–27.9 $kg \cdot m^{-2}$), 18/5, and 16/7, respectively, shown as the mean ± SD and range or as numbers. The puncture site for spinal anesthesia was in the L3–4 interspace in 11 patients and the L4–5 interspace in 12 patients. The volume of 0.5% hyperbaric bupivacaine injected was 2.4 ± 0.3 ml (1.8–3.0 ml), and the upper anesthetized dermatome level was T9 (T4–10), shown as the mean ± SD and range or median and range, respectively. Of these 23 patients, 8 required an ephedrine injection owing to the occurrence of hypotension during anesthesia; seven of these patients were given 5 mg ephedrine, and one patient was given a total of 15 mg ephedrine. Ten patients were given additional propofol injection due to slight body movement; one injection was needed for six patients and two injections were needed for four.

The Sp_{O_2} increased significantly after propofol induction and showed an increased value after the completion of anesthesia. The TcP_{CO_2} increased gradually and showed an increased value after 30 min. Although the respiratory rate decreased significantly after propofol induction, the tidal volume did not change throughout. Systolic blood pressure decreased significantly after propofol induction and showed a decreased value after the completion of anesthesia. The heart rate decreased gradually, showed a decreased value after 15 min, and then returned to the preinduction level (Fig. 1).

All patients examined underwent our anesthetic protocol successfully without complications (e.g., regurgitation, aspiration, aerophagia). The duration of surgery, fluid volume during anesthesia, time of emergence from anesthesia, and time of conversation from anesthesia were 64 ± 24 min (35–134 min), 1163 ± 347 ml (600–2000 ml), 6 ± 3 min (2–15 min), and 8 ± 3 min (4–17 min), respectively, shown as the mean ± SD and range. After the completion of anesthesia, 17 of 23 patients informed us of good feelings, and others informed us of normal feelings. Only two patients reported mild pain at the surgery site, and one patient had slight intranasal pain. No patients experienced intraoperative awareness.

Discussion

The principles of our anesthetic regimen are spinal anesthesia of a local area, sleep induction by sole propofol infusion, and airway maintenance by NPPV. The procedure of emergence from anesthesia involves simply the discontinuity of propofol infusion and removing the nasal mask. The results obtained from the present study revealed that the time course of Sp_{O_2}, TcP_{CO_2}, respiratory rate, tidal volume, systolic blood pressure, and heart rate during anesthesia were clinically reasonable and safe.

It has been demonstrated that upper airway obstruction during propofol anesthesia is a result of occlusion at the level of the soft palate [6]. NPPV restores the airway by creating a positive transmural pressure that functions as a pneumatic splint, pushing the soft palate forward [6–8]. Judging from our experience, care should be taken not to block any leak from the mouth completely because there are some patients for whom nasal inspiration is sufficient but insufficient volume can be expired nasally. Mouth leak volumes vary accordingly in each patient, and completely sealing the mouth

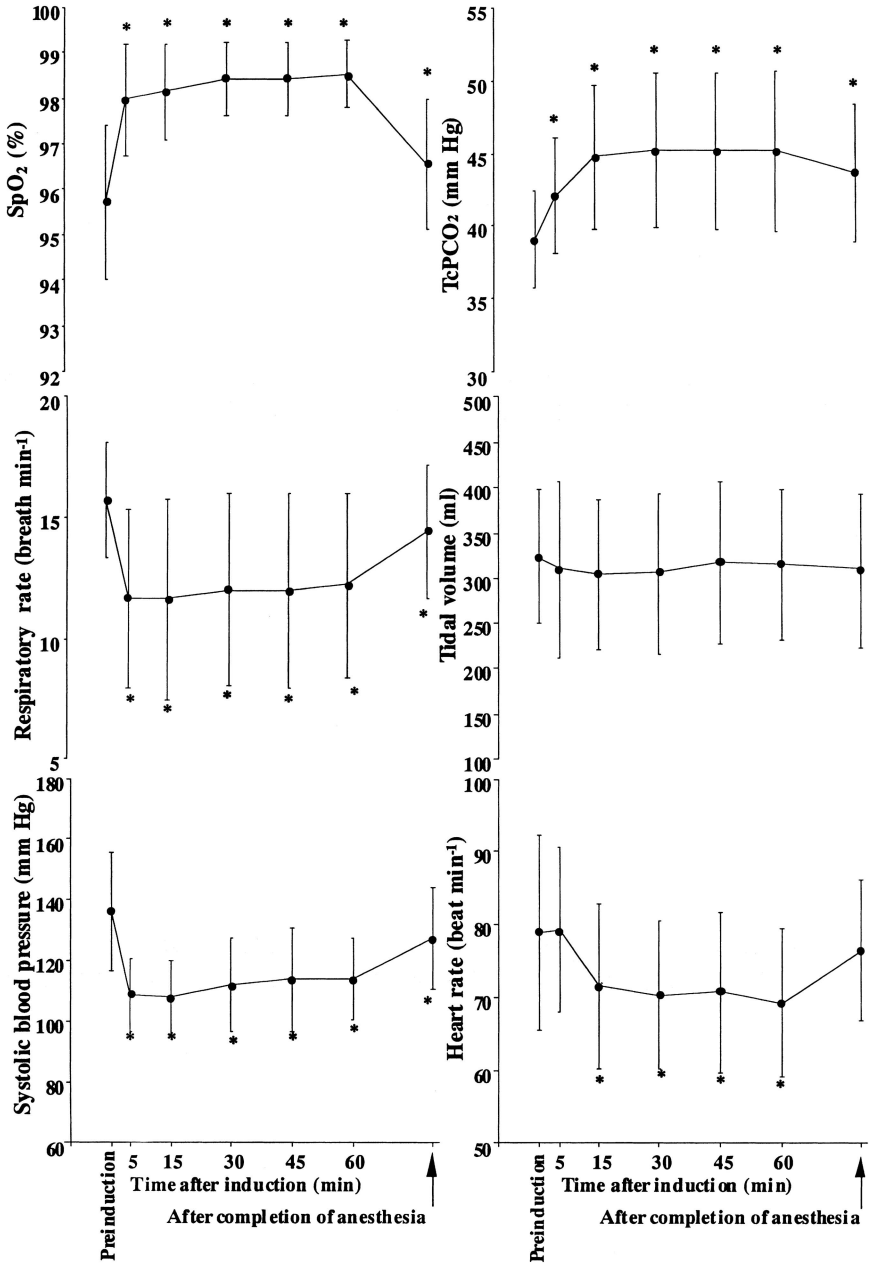

Fig. 1. Time course of percutaneous oxygen saturation (Sp_{O_2}), transcutaneous carbon dioxide tension (TcP_{CO_2}), respiratory rate, tidal volume, systolic blood pressure, and heart rate. Values are the mean ± SD ($n = 23$). *$P < 0.05$ vs. preinduction value

potentially causes extensive airway pressure. It is thought that the mouth leak acts as a safety valve to maintain the predetermined pressures. However, this makes monitoring of the end-tidal carbon dioxide impossible, which may be a serious disadvantage of this anesthetic protocol. To resolve this problem, the application of a TcP_{CO_2} monitor was considered useful because it has been reported that TcP_{CO_2} estimates a reliable value of Pa_{CO_2}, even in adults [5].

Various parameters of circulation and respiration during combined spinal-propofol anesthesia with NPPV were maintained within physiological ranges. We therefore concluded that airway maintenance with NPPV during propofol infusion is clinically applicable to spinal anesthesia.

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