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Nasal continuous positive airway pressure improves airway obstruction during midazolam-induced sedation under spinal or epidural anesthesia

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Patients under spinal or epidural anesthesia often request sedation during surgery. In these situations, we administer midazolam intravenously to obtain a sufficiently sedated condition; however, upper airway obstruction occurs occasionally in these patients. On the other hand, continuous positive airway pressure applied to the nose (nasal CPAP) has been reported to prevent snoring, restore hemoglobin oxygen saturation, and improve upper airway obstruction in obstructive sleep apnea syndromes [1]. We thus considered that nasal CPAP also would improve airway obstruction associated with midazolam-induced sedation. The present study was carried out to examine the effectiveness of nasal CPAP on airway obstruction in patients under midazolam sedation.

After approval of the institutional committee, 13 adult patients (ASA physical status 1–2) from lower extremital surgeries, who had requested sedation during surgery under spinal (n = 5) or epidural (n = 8) anesthesia and following intravenous administration of midazolam, and whose percutaneous hemoglobin oxygen saturation (Spo_2) decreased by more than 3% associated with upper airway obstruction, were subjected to this study. Persons with a history of sleep disturbance, gastroesophageal reflux, and upper airway pathology were excluded from the study. Their age (mean ± SD) was 47.8 ± 21.0 years (range 19–74 years), their weight was 66.8 ± 12.4 kg (range 51–85 kg), and their height was 158.6 ± 11.0 cm (range 145–174 cm). Four of the patients were men, and 9 were women. All patients provided informed prior consent.

The nasal CPAP device was made of a tight-fitting nasal mask (Respironics, Murrysville, PA, USA), Jackson-Rees circuit system with a 5-l bag, and a 5-cm H_2O or 10-cm H_2O threshold resistance expiratory pressure valve. Continuous air flow at 10 l·min^{-1} was delivered into the nasal CPAP device. Twenty minutes after spinal or epidural anesthesia and confirming that the range of hypesthesia was above the 12th thoracic dermatome, the patients received 5 or 10 mg midazolam intravenously in the supine position and with their heads in a neutral position until they slept. After depression of Spo_2 by more than 3% and recognizing the airway obstruction associated with snoring or an abnormal breathing pattern, the nasal mask was secured with straps that encircled the patient's head, and 5-cm H_2O nasal CPAP was applied with the patient's mouth closed. Fifteen minutes later, the pressure of nasal CPAP was increased to 10 cm H_2O. When the airway obstruction or the value of Spo_2 was not improved by 5-cm H_2O nasal CPAP, 10 cm H_2O was applied immediately. Mean blood pressure (MBP), heart rate (HR), respiratory rate (RR), Spo_2, partial pressure of arterial oxygen (Pao_2) and partial pressure of arterial carbon dioxide ($Paco_2$) were measured before and after administration of midazolam, and 5 min after each of 5-cm H_2O and 10-cm H_2O nasal CPAP. Arterial blood was obtained from the femoral artery. After these procedures, the surgery began. Data are presented as the mean ± SD. Statistical analysis was done with the Wilcoxon's signed-rank test (Stat View SE+, Abacus Concepts, Berkeley, CA, USA), and $P < 0.05$ was taken as the level of significance.

The upper border of anesthetized spinal segments was 8.0 ± 2.7th thoracic dermatome. The dose of midazolam required in this study was 7.7 ± 2.6 mg. Airway obstruction and depression of Spo_2 were not

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Table 1. Changes of mean blood pressure, heart rate, respiratory rate, percutaneous hemoglobin oxygen saturation, Pao_2, and $Paco_2$

	Administration of midazolam		Nasal CPAP	
	Before (n = 13)	After (n = 13)	5 cm H_2O (n = 12)	10 cm H_2O (n = 13)
MBP (mmHg)	90.7 ± 19.1*	85.2 ± 11.2	79.6 ± 12.2*	79.0 ± 10.1*
HR (beats/min)	73.9 ± 8.5	77.1 ± 11.3	70.4 ± 9.3*	70.0 ± 9.1*
RR (breaths/min)	16.6 ± 2.8	16.8 ± 3.8	17.8 ± 3.2	16.3 ± 2.4
Spo_2 (%)	95.2 ± 2.0*	88.6 ± 4.2	94.9 ± 2.5*	95.3 ± 1.9*
Pao_2 (mmHg)	78.6 ± 11.6*	59.2 ± 11.3	85.2 ± 17.1*	90.2 ± 14.2*
$Paco_2$ (mmHg)	42.7 ± 3.2*	44.5 ± 3.7	47.1 ± 2.8*	45.2 ± 2.3

Data are presented as mean ± SD.
Pao_2, partial pressure of arterial oxygen; $Paco_2$, partial pressure of arterial carbon dioxide; CPAP, continuous positive airway pressure; MBP, mean blood pressure; HR, heart rate; RR, respiratory rate; Spo_2, percutaneous hemoglobin oxygen saturation.
* $P < 0.05$ *vs* after administration of midazolam.

improved by 5-cm H_2O nasal CPAP in one patient who thereby immediately received 10-cm H_2O nasal CPAP. The patient subsequently recovered airway clearance and Spo_2. The data of 5-cm H_2O nasal CPAP in this patient was thus not obtained. The other 12 patients restored their Spo_2 and breathing conditions at 5 cm H_2O. The results are shown in Table 1. MBP decreased significantly after the administration of midazolam, and after nasal CPAP, MBP further decreased. HR did not change after midazolam, but significantly decreased after nasal CPAP. RR did not show significant changes. The midazolam-induced sedation decreased Spo_2 significantly, and nasal CPAP improved these depressions significantly. Pao_2 also showed similar changes of Spo_2. $Paco_2$ increased significantly after the administration of midazolam, and after 5-cm H_2O nasal CPAP, $Paco_2$ further increased: however, it returned to the CPAP 0-cm H_2O level after 10 cm H_2O.

The results of this clinical trial suggested that nasal CPAP improves upper airway obstruction and prevents hypoxia that is caused by the midazolam-induced sedation in patients under spinal or epidural anesthesia. Nozaki-Taguchi et al. [2] also examined the effect of nasal CPAP on midazolam-induced depression of Spo_2 under spinal anesthesia, and demonstrated that this depression contributes to upper airway obstruction. They also documented the minimally effective CPAP to prevent snoring to be around 5 cm H_2O. Upper airway obstruction in obstructive sleep apnea and during sedation or general anesthesia had been considered generally to be due to reduced genioglossus activity and the consequent relaxation of the tongue [3]. However, many reports have implicated the soft palate or epiglottis as the site of airway obstruction [4–7]. Several recent works documented the apparent occlusion of the upper airway at the level of the soft palate in patients anesthetized with thiopental [8] and propofol [9]. Nasal CPAP, on the other hand, has been shown to restore airway patency by creating a positive transmural pressure that functions as a pneumatic splint [10,11]. Mathru et al. [9] examined the effect of nasal CPAP on the upper airway using magnetic resonance imaging during propofol anesthesia, and demonstrated that the airway obstruction is restored by pushing the soft palate forward from the posterior pharyngeal wall. However, whether midazolam-induced sedation causes upper airway obstruction similar to that of propofol anesthesia is unclear.

Since nasal CPAP can be easily applied to sedated patients in some clinical situations, this procedure seems to be a promising method for airway management even in the operating room. However, potential complications include causing gastric distension, regurgitation, and aspiration of gastric contents. To prevent these complications, nasal CPAP should be indicated only for elective surgery patients. Although further examinations concerning nasal CPAP during anesthesia are needed, we consider that this simple and easy airway management technique may be utilized in sedated patients under spinal or epidural anesthesia.

References

1. Strohl KP, Redline S (1986) Nasal CPAP therapy, upper airway muscle activation, and obstructive sleep apnea. Am Rev Respir Dis 134:555–558
2. Nozaki-Taguchi N, Isono S, Nishino T, Numai T, Taguchi N (1995) Upper airway obstruction during midazolam sedation: modification by nasal CPAP. Can J Anaesth 42:685–690

3. Safar P, Escarraga LA, Chang F (1959) Upper airway obstruction in the unconscious patient. J Appl Physiol 14:760–764
4. Hudgel DW (1992) The role of upper airway anatomy and physiology in obstructive sleep apnea. Clin Chest Med 13:383–398
5. Boidin MP (1985) Airway patency in the unconscious patient. Br J Anaesth 57:306–310
6. Abernethy LJ, Allan PL, Drummond GB (1990) Ultrasound assessment of the position of the tongue during induction of anaesthesia. Br J Anaesth 65:744–748
7. Shorten GD, Opie NJ, Graziotti P, Khangure MM (1994) Assessment of upper airway anatomy in awake, sedated and anaesthetized patients using magnetic resonance imaging. Anaesth Intens Care 22:165–169
8. Nandi PR, Charlesworth CH, Taylor SJ, Nunn JF, Dore CJ (1991) Effect of general anaesthesia on the pharynx. Br J Anaesth 66:157–162
9. Mathru M, Esch O, Lang J, Herbert ME, Chaljub G, Goodacre B, van Sonnenberg E (1996) Magnetic resonance imaging of the upper airway: effects of propofol anesthesia and nasal continuous positive airway pressure in humans. Anesthesiology 84:273–279
10. Polo O, Berthon-Jones M, Douglas NJ, Sullivan CE (1994) Management of obstructive sleep apnoea/hypopnoea syndrome. Lancet 344:656–660
11. Douglas NJ, Polo O (1994) Pathogenesis of obstructive sleep apnoea/hypopnoea syndrome. Lancet 344:653–655