

Continuous positive airway pressure oxygenation during one-lung ventilation with 50% nitrous oxide and isoflurane in oxygen

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Key words: CPAP, One-lung ventilation, N₂O-isoflurane anesthesia

Introduction

Application of continuous positive airway pressure (CPAP) with an inspired oxygen concentration (F_{IO₂}) of 1.0 to the nondependent lung has been reported to be an effective method of improving P_{aO₂} during one-lung ventilation for elective thoracic surgery [1–5]. When nitrous oxide (N₂O)/oxygen/volatile anesthetics technique is employed during one-lung ventilation, the N₂O decreases the inspired oxygen concentration and increases the chance of hypoxemia. However, the use of N₂O allows maintenance of anesthesia with low concentrations of potent inhalational agents.

We have constructed a CPAP unit from equipment readily available in our department. The purpose of this study was to determine whether the CPAP oxygenation, using this unit, can prevent hypoxemia during one-lung ventilation with N₂O and isoflurane anesthesia (F_{IO₂} of 0.5).

Patients and methods

This study was approved by the Hospital Clinical Trial Committee, and informed consent was obtained from patients preoperatively. Twenty patients, 33–73 years of age (56.5 ± 11.5), were studied who were having elective thoracotomies (right: 8; left: 12) involving lung cancer in 14 patients, esophageal cancer in 3, giant bullae in

2, and mediastinal tumor in 1. Arterial blood gas values, forced expiratory volume in the first second (FEV_{1.0}), and vital capacity (VC) were measured on all patients the day before surgery. Preoperative medication consisted of atropine sulfate 0.5 mg and pentazocine 30 mg, given intramuscularly 1 h prior to induction of anesthesia. Following insertion of intravenous and intra-arterial cannulae, anesthesia was induced with intravenous thiopental (4 mg·kg⁻¹) followed by vecuronium (0.15 mg·kg⁻¹). The trachea was intubated with a double-lumen endotracheal tube (Bronch-Cath, size #37 to 39 Fr, Mallinckrodt, St. Louis, MO, USA). The position of the endotracheal tube was checked by auscultation and verified by fiberoptic bronchoscopy in the supine and lateral decubitus positions. Anesthesia was maintained with 50% N₂O, 0.5%–2% isoflurane in oxygen, and intermittent injection of vecuronium. The patient's lungs were ventilated with a volume ventilator (Exell 110 Ohmeda, Madison, WI, USA), using a tidal volume of 10–15 ml·kg⁻¹ for two-lung ventilation. The respiratory rate was adjusted to maintain a P_{aCO₂} at 36 ± 3 mmHg [1].

The preoperative arterial blood gas sample was obtained with the patient in the lateral decubitus position during two-lung ventilation 20 min after induction of anesthesia. After the chest was opened, one-lung ventilation was commenced and CPAP was applied to the nondependent lung at the level of 2.5–10 cmH₂O with pure oxygen via a CPAP system (Fig. 1). The dependent lung was ventilated at a rate of 15–20 min⁻¹, with a tidal volume of 8–10 ml·kg⁻¹ adjusted to maintain P_{aCO₂} at 36 ± 3 mmHg. Arterial blood gases were analyzed every 10 min after application of CPAP for 1 h, and at 90 min (Radiometer ABL505, Copenhagen, Denmark). Further intraoperative arterial blood gases were drawn 10 min after re-inflation of the nondependent lung, and 20 min after resumption of two-lung ventilation. End-tidal CO₂ (ETCO₂), end-tidal isoflurane concentration (ETiso) and arterial oxygen saturation (Sao₂) were

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Received for publication on October 14, 1994; accepted on February 4, 1995

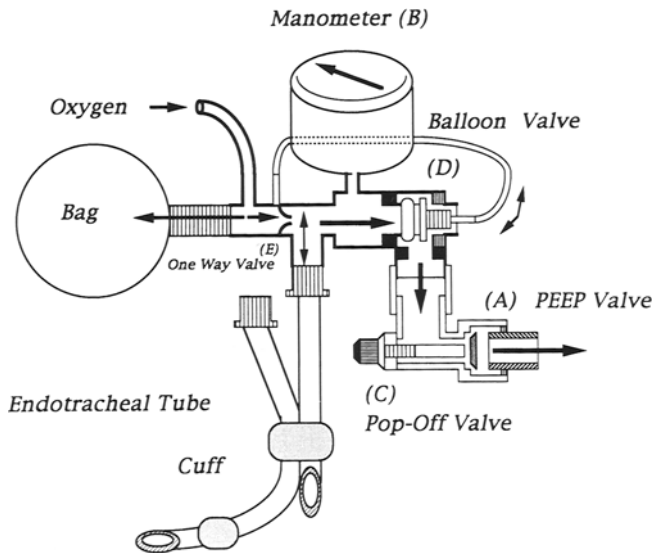


Fig. 1. Circuit for application of continuous positive airway pressure (CPAP) to the nondependent lung. The flexible positive end-expiratory pressure (PEEP) valve (A) with integral pop-off valve was placed on the circuit expiratory limb to produce CPAP to the nondependent lung. End-expiratory pressure was measured at the airway with an aneroid manometer (B). The oxygen flow rate and/or pop-off valve (C) is adjusted to provide the desired amount of CPAP as measured on the manometer. When re-inflation of the nondependent lung is necessary, the bag is squeezed and the circuit is closed by the stopper (balloon valve) (D) to avoid air leak. The one-way valve (E) is closed to produce PEEP without rebreathing during deflation of the lung, so that the nondependent lung can be ventilated without losing end-expiratory pressure

recorded continuously throughout the anesthesia. (Capnomac Ultima, Datex, Helsinki, Finland)

All data were expressed as means \pm SD. A two-tailed *t*-test was used to compare the various parameters. Differences with a statistical probability of less than 0.05 were considered to be significant.

Results

Table 1 indicates that the pulmonary function and blood gas data before surgery were within the normal ranges in all patients. One patient who had ischemic changes in the ECG immediately after the induction of anesthesia was dropped from the study.

The time course of Pao₂ during thoracotomy with one-lung ventilation is shown in Fig. 2. The mean values of Pao₂ generally declined to 100 mmHg within the 30–60 min of one-lung ventilation. After inflating the nondependent lung, the Pao₂ rose to near the control values quickly. No clinical problems were encountered with any of the patients. The lowest values of Pao₂ from each patient during one-lung ventilation was 94.2 ± 18.7 mmHg, ranging from 78 to 129 mmHg during one-lung ventilation with CPAP.

The individual changes in intraoperative end-tidal parameters and blood gas values during thoracotomy are shown in Table 2. ETIso was gradually decreased after the initiation of one-lung ventilation, and was significantly decreased after resumption of two-lung ventilation. Other parameters showed no statistically significant changes during thoracotomy.

Discussion

The dependent lung is usually ventilated mechanically with a mixture of 100% oxygen and inhaled anesthetics to avoid hypoxemia. However, there are some pathophysiologic problems. First, although a high F_{IO₂} in the single-ventilated lung may increase Pao₂ levels, absorption atelectasis and oxygen toxicity are possible

Table 1. Pulmonary function and blood gas data before surgery

| Parameters | Mean \pm SD | Range |
|--------------------------|-----------------|--------------|
| %VC (%) | 99.8 \pm 7.5 | 88.6 – 114.0 |
| FEV _{1.0} (%) | 82.2 \pm 11.0 | 75 – 106 |
| FEV/VC | 0.83 \pm 0.14 | 0.66 – 1.08 |
| pH | 7.43 \pm 0.04 | 7.35 – 7.49 |
| Paco ₂ (mmHg) | 40.3 \pm 2.7 | 36.7 – 44.4 |
| Pao ₂ (mmHg) | 83.3 \pm 8.1 | 71.5 – 97.6 |
| Sao ₂ (%) | 94.7 \pm 1.8 | 92 – 97 |
| BE (mmol/l) | 1.8 \pm 2.7 | –1.1 – 7.8 |

VC, vital capacity; FEV_{1.0}, forced expiratory volume in the first second; FEV, forced expiratory volume; Paco₂, partial arterial pressure of CO₂; Sao₂, arterial oxygen saturation; BE, base excess.

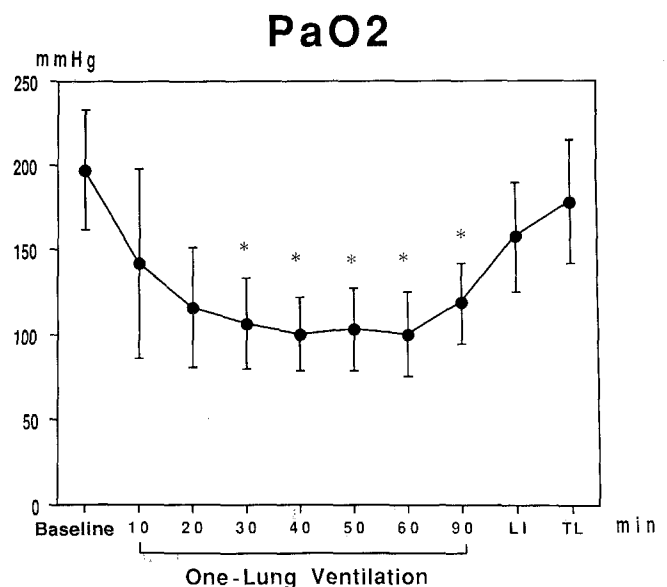


Fig. 2. The time course of Pao₂ during one-lung ventilation with 50% N₂O-isoflurane anesthesia. LI, inflation of nondependent lung; TL, two-lung ventilation. Values are expressed as mean \pm SD. **P* < 0.05 vs baseline

Table 2. Changes of end-tidal parameters and blood gas values during thoracotomy with one-lung ventilation

| | Control | 10 | 20 | 30 | 40 | 50 | 60 | 90 (min) | LI | TL |
|--------------------------|-------------|-------------|-------------|-------------|-------------|-------------|-------------|--------------|--------------|--------------|
| Paco ₂ (mmHg) | 38.8 (3.7) | 37.3 (10.9) | 40.5 (5.3) | 38.8 (4.9) | 40.2 (4.1) | 39.3 (5.2) | 40.1 (4.4) | 39.1 (3.7) | 38.8 (4.3) | 39.4 (5.4) |
| ETco ₂ (mmHg) | 34.9 (3.1) | 34.9 (3.4) | 35.3 (2.8) | 34.4 (2.4) | 35.4 (2.3) | 35.4 (3.1) | 34.5 (3.2) | 36.2 (5.6) | 35.0 (3.1) | 36.1 (4.1) |
| Sao ₂ (%) | 99.4 (0.7) | 97.8 (2.1) | 97.1 (1.7) | 97.2 (1.6) | 97.0 (1.3) | 96.9 (1.7) | 96.6 (2.2) | 97.4 (1.9) | 98.9 (0.7) | 99.1 (0.4) |
| SpO ₂ (%) | 98.7 (0.9) | 97.7 (2.8) | 97.1 (2.2) | 96.6 (2.7) | 97.1 (2.1) | 96.3 (2.6) | 96.7 (2.8) | 96.4 (2.2) | 99.1 (1.2) | 99.0 (1.2) |
| ETIso (%) | 1.21 (0.32) | 1.21 (0.27) | 1.01 (0.21) | 0.91 (0.24) | 0.89 (0.16) | 0.86 (0.10) | 0.83 (0.10) | 0.79 (0.10)* | 0.74 (0.11)* | 0.56 (0.11)* |
| pH | 7.40 (0.03) | 7.39 (0.04) | 7.39 (0.05) | 7.39 (0.05) | 7.39 (0.04) | 7.39 (0.04) | 7.39 (0.04) | 7.38 (0.05) | 7.39 (0.04) | 7.39 (0.05) |
| BE (mmol/l) | -0.5 (4.5) | -0.9 (2.2) | -0.7 (2.2) | -1.3 (2.0) | -0.9 (1.9) | -1.3 (1.9) | -0.8 (2.0) | -1.7 (2.4) | -1.2 (2.3) | -1.1 (2.3) |

Mean (\pm SD).

LI, lung inflation; TL, two-lung ventilation; Paco₂, partial arterial pressure of CO₂; ETco₂, end-tidal CO₂; Sao₂, arterial oxygen saturation; SpO₂, peripheral oxygen saturation; ETIso, end-tidal isoflurane concentration; BE, base excess.

* $P < 0.05$ vs control.

during one-lung ventilation with 100% oxygen [6]. Second, a higher MAC of inhaled anesthetics may be required to maintain hemodynamic stability during thoracic surgery. The high concentration of inhalation anesthetics required would inhibit hypoxic pulmonary vasoconstriction (HPV) in the non-ventilated lung, leading to an increase in transpulmonary shunt [7].

Nitrous oxide (N₂O) can be used during one-lung anesthesia [8]. The administration of N₂O may prevent the oxygen toxicity, and will permit the use of lower concentration of the potent inhalational agents, which may result in less hemodynamic depression. In this study, end-tidal isoflurane concentrations were maintained between 0.79%–1.2% during one-lung ventilation. In clinical doses near 1 MAC, the inhaled anesthetics provide a reasonable degree of cardiovascular stability [6]. The use of N₂O, however, necessitates a significant decrease in the inspired oxygen concentration, and increases the chance of hypoxemia when one-lung ventilation is employed [9]. Some investigators reported that an FIO₂ of 0.5 causes significant hypoxemia during one-lung ventilation [9–11]. In this study, however, we found that the application of CPAP to the nondependent lung produced acceptable oxygenation in all patients, resulting in a mean Pao₂ between 100.0 \pm 21.5 and 142.8 \pm 56.4 mmHg. Unfortunately, we could not complete a control study (measurement of oxygenation during one-lung ventilation with 50% N₂O without CPAP to the nondependent lung). The reasons for this are as follows: (1) informed consent was obtained from only nine patients; (2) these nine patients developed severe hypoxemia (Pao₂ less than 70 mmHg) within the first 30 min, and FIO₂ had to be changed from 0.5 to 1.0.

To improve oxygenation during one-lung ventilation, applying CPAP to the nondependent lung/or applying positive end-expiratory pressure (PEEP) to the ventilated lung has been studied. Capan et al. [1] found a

CPAP of 10 cmH₂O to be optimal for improving oxygenation. Brown et al. [12] ventilated patients with selective PEEP to the dependent lung in the lateral position during thoracotomy; this resulted in adequate arterial oxygenation with 30%–50% inspired oxygen. Cohen et al. [13] also reported that CPAP to the nondependent lung had the most beneficial effects on oxygenation and hemodynamics during one-lung ventilation. This maneuver may increase Pao₂ by keeping the alveoli in the nondependent lung distended, thus permitting oxygen uptake, and/or by diverting blood flow to the dependent, ventilated lung. Therefore, application of CPAP to the nondependent lung will increase arterial oxygenation during one-lung ventilation.

An undesirable property of general anesthesia is inhibition of HPV in the nondependent lung by anesthetic agents such as isoflurane and halothane [7,14,15]. If HPV is greatly inhibited in the nonventilated lung, pulmonary blood flow persists, resulting in a marked increase of venous admixture. However, it has been noted that inhalational anesthetics have less of an inhibitory effect on HPV in intact in vivo preparations as compared to in vitro studies [6]. In addition, approximately 1 MAC of isoflurane or halothane does not impair arterial oxygenation any more than intravenous anesthesia [6], suggesting that there is no inhibition of HPV by 1 MAC of these inhalational anesthetics during one-lung ventilation. In this study, using nitrous oxide allowed isoflurane to be decreased to 1 MAC or less. CPAP with 100% oxygen was applied to the nonventilated lung immediately after beginning one-lung ventilation. Therefore, it is believed, from the results of our study, that pulmonary capillary blood flow around the inflated alveoli with 100% oxygen could still take up oxygen, and pulmonary capillaries around the collapsed alveoli, which might still exist in spite of the application of CPAP, could undergo HPV to minimize venous admixture.

It has previously been shown that arterial oxygenation during one-lung ventilation has a negative correlation with the degree of obstructive lung disease [16]. The patient population in this study had no obstructive lung disease (Table 1). Further study is required to find the effect of CPAP to the nondependent lung on arterial oxygenation in the patients with obstructive lung disease during one-lung ventilation.

In conclusion, adequate oxygenation can be maintained by ventilating the dependent lung with 50% N₂O-O₂-isoflurane (<MAC) if CPAP with 100% oxygen is applied to the nondependent lung in patients who do not have obstructive pulmonary disease.

References

1. Capan LM, Turndorf H, Patel C, Ramanathan S, Acinarura A, Chanlon J (1980) Optimization of arterial oxygenation during one-lung anesthesia. *Anesth Analg* 59:847-851
2. Benumof JL (1982) One lung ventilation: Which lung should be PEEPed? *Anesthesiology* 56:161
3. Benumof JL, Alferg DD (1986) Anesthesia for thoracic surgery. In: Miller RD (ed) *Anesthesia*, 2nd edn. Churchill Livingstone, New York, pp 1371-1469
4. Benumof JL, Gaughan S, Ozaki GT (1992) Operative lung constant positive airway pressure with the Univent bronchial blocker tube. *Anesth Analg* 74:406-410
5. Slinger P, Triolet W, Wilson J (1988) Improving arterial oxygenation during one-lung ventilation. *Anesthesiology* 68:291-295
6. Benumof JL (1985) One-lung ventilation and hypoxic pulmonary vasoconstriction: Implication for anesthetic management. *Anesth Analg* 64:821-831
7. Bjertnass LJ, Hawge A, Torgrinsen T (1980) The pulmonary vasoconstriction response to hypoxia. The hypoxia-sensitive site studied with a volatile inhibitor. *Acta Physiol Scand* 109:447-162
8. Boutrous AR, Weisel MR (1968) Arterial blood oxygenation during thoracotomy using 70% nitrous oxide in oxygen. *Anesthesiology* 29:705-710
9. Bendixen HH (1968) Anesthesia for thoracic surgery. *Anesthesiology* 28:649-650
10. Torda TA, McCulloch CH, O'Brien HD, Wright JS, Horton DA (1974) Pulmonary venous admixture during one-lung anaesthesia. The effect of inhaled oxygen tension and respiration rate. *Anaesthesia* 29:272-279
11. Kerr JH, Smith AC, Prys-Roberts C, Meloche R, Foex P (1974) Observations during endobronchial anaesthesia. (II): Oxygenation. *Br J Anaesth* 41:84-92
12. Brown RD, Cafer RED, Robertson OV, Wilcox BR, Murray GF (1977) Improved oxygenation during thoracotomy with selective PEEP to the dependent lung. *Anesth Analg* 56:26-31
13. Cohen E, Eisenkraft JB, Thys DM, Kirshner PA, Kaplan JA (1988) Oxygenation and hemodynamic changes during one-lung ventilation: Effects of CPAP10, PEEP10, and CPAP10/PEEP10. *J Cardiothorac Anesth* 2:34-40
14. Benumof JL, Wahrenbrock EA (1975) Local effects of anesthetics on regional hypoxic pulmonary vasoconstriction. *Anesthesiology* 43:535-532
15. Mathers J, Benumof JL, Wahrenbrock EA (1977) General anesthetics and regional hypoxic pulmonary vasoconstriction. *Anesthesiology* 46:111-114
16. Katz JA, Laverne RG, Fairley HB, Thomas AN (1982) Pulmonary oxygen exchange during endobronchial anesthesia: Effect of tidal volume and PEEP. *Anesthesiology* 56:164-170