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A Simple Device for Oxygen Insufflation with Continuous Positive Airway Pressure during One-lung Ventilation

To the Editor:—Hypoxemia is a significant clinical problem in approximately 20% of patients for whom a one-lung ventilation (OLV) anesthetic technique is utilized. Oxygen insufflation to the upper, nonventilated lung with continuous positive airway pressure (CPAP) is reported to increase PaO_2 during OLV.^{1,2} We have constructed an inexpensive, reusable, and readily available device that provides O_2 insufflation with variable CPAP to the upper, nonventilated lung during OLV (fig. 1).

A modified Ayre's T-piece (essentially a Mapleson E system) is connected to the lumen of a double-lumen endotracheal tube that supplies the nonventilated lung. The fresh gas port of the T-piece is connected to an

oxygen source, either a tank or a wall outlet, via an appropriate flowmeter. A reusable valve designed to produce positive end-expiratory pressure (Boehringer Laboratories, Wynewood, Pennsylvania) is connected to the corrugated tubing at the end of the T-piece assembly.³ Using this system clinically, we have observed

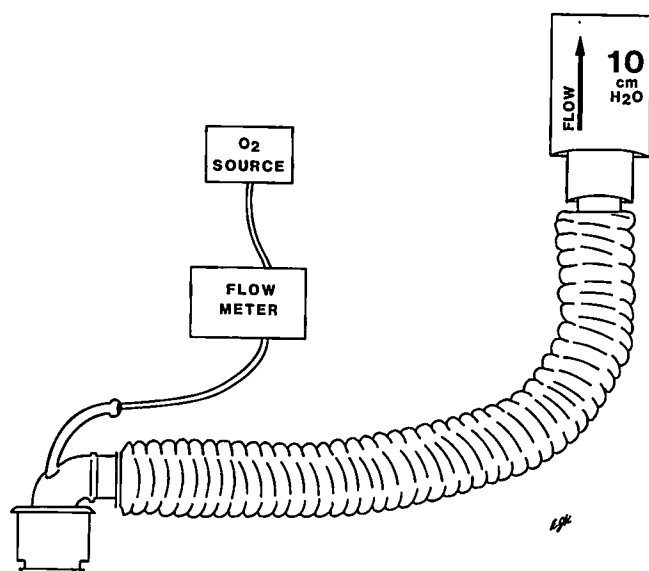


FIG. 1. A modified Mapleson "E" open-ended anesthesia circuit is connected to an oxygen source via a flow meter, and to a Boehringer "PEEP" valve at the distal end. When the system is connected to the nonventilated lung with an oxygen flow rate of $8 \text{ l} \cdot \text{min}^{-1}$, this circuit will provide oxygen insufflation; the level of CPAP is indicated on the valve, provided the valve is fixed in the vertical position.

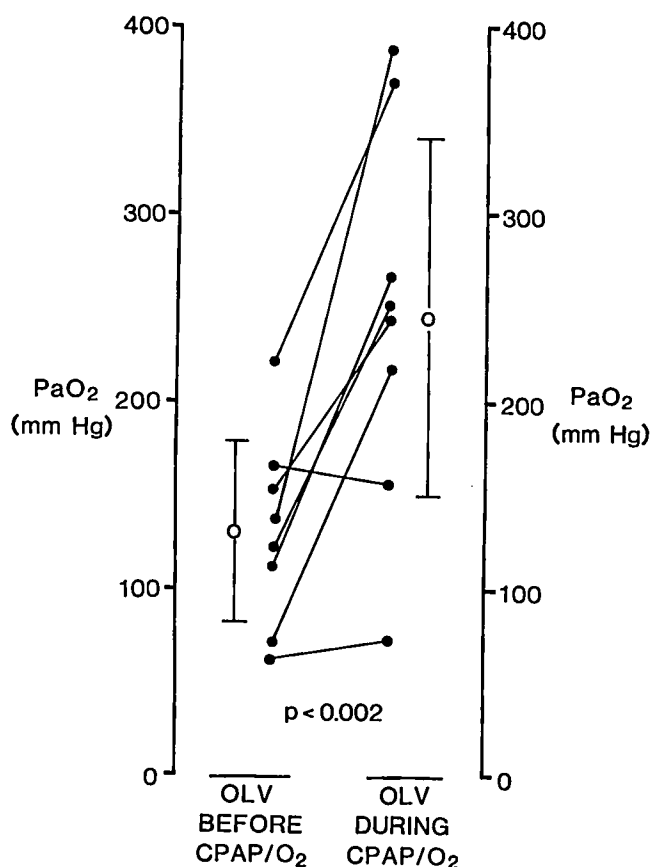


FIG. 2. Arterial oxygen tension (PaO_2) measured after 20 min of (OLV) without CPAP/ O_2 is shown compared with similar data obtained 20 min after starting CPAP/ O_2 to the nonventilated lung. Open symbols show mean values for each group with brackets to indicate one standard deviation.

airway pressure in the nonventilated lung to vary less than 1 cmH₂O from the 10 cmH₂O indicated on the valve (with an 8 l·min⁻¹ oxygen flow rate). Other O₂ flow rates were not tested clinically; however, bench testing of the circuit with O₂ flow rates varying from 1 to 10 l·min⁻¹ produced less than 10% (1 cmH₂O) variation from the indicated pressure at all O₂ flow rates tested. Clinically, use of this circuit to provide CPAP and O₂ insufflation to the nonventilated lung during continuous OLV significantly improved PaO₂ in patients undergoing thoracotomy, as shown in figure 2.

Capan *et al.*¹ have described a similar system to provide CPAP and O₂ insufflation to the nonventilated lung during which they used an adjustable pressure relief valve to limit the escape of oxygen from the insufflation circuit. This system has the disadvantage that an inadvertent occlusion of the valve or an increase in flow rate could create high airway pressures. The system we describe utilizes reusable materials that are readily available within most anesthesiology departments, which gives it the combined advantages of availability and simplicity. Over-pressure is highly unlikely, since 10 cmH₂O pressure relief (independent of O₂ flow rate from 1 to 10 l·min⁻¹) is designed into the system. We

observed no difficulty with the circuit; furthermore, 10 cmH₂O CPAP does not appear to interfere with surgical exposure and may even facilitate intralobar dissection.

DAVID L. BROWN, M.D.
Staff Anesthesiologist
Lackland Air Force Base
Texas

RICHARD F. DAVIS, M.D.
Assistant Professor
Department of Anesthesiology
University of Florida College of Medicine
Gainesville, Florida 32610

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Does Isoflurane Really Preserve Baroreflex Responsiveness Better than Halothane or Enflurane?

To the Editor:—Kotrly *et al.* assert that "the data indicate isoflurane preserves baroreflex regulation of heart rate better than either halothane or enflurane" and suggest that isoflurane may be the preferred inhalational anesthetic in clinical situations where baroreflex responsiveness would be important, *e.g.*, acute hypovolemia or position changes under anesthesia.¹ There are four reasons why I believe these conclusions to be premature.

First, if the pressor slopes (R-R interval *vs.* blood pressure) for isoflurane from Duke *et al.*² are plotted *versus* MAC multiples as in figure 6 of reference 1, the line for isoflurane can be superimposed on the halothane-N₂O one and is parallel to those for halothane, enflurane, and enflurane-N₂O. Even if the concentration of isoflurane is reduced but the level of anesthesia maintained with N₂O, the isoflurane-N₂O line can be superimposed on the isoflurane one. Thus, the data of Duke *et al.* do not show the degree of sparing of baroreflex responsiveness that Kotrly observed.

Given the above, the question is raised as to how comparable are the experimental methods from the two

different laboratories. Duke controlled ventilation to keep the arterial P_{CO₂} about 35 mmHg.³ It is not clear from Kotrly's article whether ventilation was controlled and what the actual arterial P_{CO₂}s were. A statement is made that "all subjects resumed spontaneous ventilation prior to the tests carried out during anesthesia."¹ Thus, the arterial P_{CO₂} may have been 50 mmHg.⁴ An elevated CO₂ tension could increase sympathetic outflow from the vasomotor center, one component of the baroreflex arc.

Thirdly, in two elegant studies in dogs, Kampine's group determined that both halothane and isoflurane, with a thiopental background, attenuated in a dose-related manner all the individual components of the baroreceptor reflex arc except the baroreceptor itself, which was sensitized.^{5,6} When the intact reflex arc was studied, isoflurane at 1 MAC appeared to be less depressant than halothane at 1 MAC. However, the control slopes (R-R interval in ms *vs.* mean BP in mmHg) in the conscious dogs are so different (12.38 ± 3.6 ms/mmHg for halothane and 59.4 ± 16.5 ms/mmHg for isoflurane). If the halothane data were normalized to the same