

Halothane and Isoflurane Only Slightly Impair Arterial Oxygenation during One-lung Ventilation in Patients Undergoing Thoracotomy

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Controversy exists as to whether the halogenated inhalation (IH) anesthetics impair arterial oxygenation during one-lung ventilation (1-LV). Accordingly, the authors have answered this question in 12 consenting patients who required 1-LV to facilitate the performance of thoracic surgery, by comparing arterial oxygenation during a prolonged period of IH anesthesia with arterial oxygenation during a prolonged period of intravenous (IV) anesthesia during stable 1-LV conditions. The patients were equally divided into halothane and isoflurane groups. Each patient in each IH anesthetic group underwent the following experimental sequence: step 1, two-lung ventilation (2-LV), 1 MAC IH anesthesia; step 2, 1-LV, 1 MAC IH anesthesia; step 3, 1-LV, iv anesthesia; step 4, 2-LV, iv anesthesia. Stable 1-LV conditions were proven by serial arterial blood gas measurement. Conversion from 2-LV to 1-LV during IH anesthesia (step 1 to step 2) caused a very large and significant decrease in P_{aO_2} (from 484 ± 49 to 116 ± 61 , and from 442 ± 58 to 232 ± 97 mmHg in the halothane and isoflurane groups, respectively) and increase in shunt (from 14 ± 4 to 44 ± 9 , and from 19 ± 5 to $31 \pm 8\%$ in the halothane and isoflurane groups, respectively). Conversion from 1 MAC halothane anesthesia to iv anesthesia during 1-LV caused a slight but significant decrease in shunt ($7 \pm 2\%$ of the cardiac output) and increase in P_{aO_2} (39 ± 29 mmHg), whereas conversion from 1 MAC isoflurane anesthesia to iv anesthesia caused a very slight and nonsignificant decrease in shunt ($2 \pm 2\%$ of the cardiac output) and increase in P_{aO_2} (13 ± 19 mmHg). Return to 2-LV (step 3 to step 4) caused a large and significant increase in P_{aO_2} (from 155 ± 84 to 411 ± 101 mmHg, and from 245 ± 97 to 431 ± 104 mmHg in the halothane and isoflurane groups, respectively) and a large decrease in shunt (from 37 ± 9 to 20 ± 8 , and from 36 ± 5 to $20 \pm 7\%$ in the halothane and isoflurane groups, respectively). The authors calculated that the atelectatic lung had hypoxic pulmonary vasoconstriction (HPV) and that the slight increase in shunt and decrease in P_{aO_2} caused by changing from IH to iv anesthesia was consistent with the finding in animal studies that 1 MAC IH anesthesia causes a 20% inhibition of HPV. In view of the usual efficacy of nondependent lung CPAP, the authors conclude that IH anesthetic drugs are safe to use in patients undergoing 1-LV. (Key words: Anesthesia; thoracic. Anesthetic techniques: one-lung ventilation. Anesthetics, volatile: halothane; isoflurane. Lung: atelectasis; blood flow; oxygen; shunting. Surgery: thoracic. Ventilation: one-lung.)

HYPoxic PULMONARY VASOCONSTRICTION (HPV) is considered to be an important mechanism by which

blood flow is diverted away from atelectatic or hypoxic regions of the lung to better ventilated normoxic regions. The blood flow diversion minimizes the amount of shunt flow that can occur through hypoxic regions, and thereby minimizes any decrease in arterial oxygen tension. Studies in animals indicate that the inhalational anesthetics halothane and isoflurane inhibit HPV.¹ Extrapolating from these studies, many authors have suggested that inhalational anesthetics are relatively contraindicated during use of one-lung ventilation.²⁻⁶

However, two recent studies have shown that administration of the inhalational anesthetics halothane⁷ and isoflurane^{7,8} to patients anesthetized with intravenous agents during a condition of stable one-lung hypoxia (atelectasis of one lung⁷ and ventilation of one lung with 8% oxygen in nitrogen⁸, did not change the arterial oxygen tension (P_{aO_2}). In the first study, end-tidal concentrations of halothane and isoflurane were held constant for approximately 20 min at 1.45 and 1.15 MAC, respectively.⁷ In the second study, isoflurane was administered at an end-tidal concentration of 1% for 15 min, followed by a 15-min period of administration at an end-tidal concentration of 1.5%.⁸ However, in view of the relatively short duration of administration of inhalation anesthetic in both studies, it is possible that clinically relevant tissue concentrations of these anesthetics were not achieved. The purpose of this study was to re-examine the question of the effect of inhalation anesthetics on P_{aO_2} and shunt by first establishing a long duration of inhalation anesthesia (end-tidal concentration equal to or greater than 1 MAC for 1 h) during the condition of stable one-lung atelectasis, and then, while maintaining one-lung atelectasis, eliminate the inhalation anesthesia by maintaining anesthesia with only intravenous drugs (end-tidal concentration of inhalation anesthetic closely approached 0% after approximately 50 min).

Methods

SUBJECTS

This study was approved by the University of California San Diego Human Subjects Committee, and all subjects gave their informed consent. The subjects were 12 adult patients who required thoracotomy and one-lung

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TABLE 1. Preoperative Demographic Patient Profile

Inhalational Anesthetic	Patient Number	Age (yr)	Weight (kg)	Height (cm)	RA ABG (mmHg)		Pre-induction Shunt, %	Surgical Procedure
					PaO ₂	PaCO ₂		
Halothane	1	69	65	173	80	38	9	Left upper lobectomy
	2	59	90	185	59	44	21	Right upper lobectomy
	3	71	82	188	76	43	8	Right upper lobectomy
	4	25	57	170	97	39	2	Nissen fundal plication
	5	33	58	167	65	51	19	Esophageal tear repair
	6	64	86	183	72	48	13	Right middle lobectomy
	Mean ± SD	54 ± 18	73 ± 13	178 ± 8	75 ± 12	44 ± 5	12 ± 7	—
Isoflurane	1	64	51	155	86	48	14	Right lower lobectomy
	2	48	50	165	96	40	3	Left upper lobectomy
	3	40	93	180	75	50	17	Nissen fundal plication
	4	58	111	168	76	38	10	Left lower lobectomy
	5	67	73	170	80	41	5	Right upper lobectomy
	6	63	65	180	92	40	3	Esophagogastrectomy
	Mean ± SD	57 ± 10	74 ± 22	170 ± 9	84 ± 9	43 ± 5	9 ± 6	—

RA = room air; ABG = arterial blood gases.

atelectasis for various surgical procedures (table 1). Subjects were alternatively assigned to either a halothane-receiving (n = 6) or an isoflurane-receiving (n = 6) anesthetic study group. There were no significant differences between the two groups with respect to age, weight, height, room air arterial blood gases, pre-induction shunt, and incidence of pulmonary resection *versus* non-pulmonary surgery.

MONITORING

Under local anesthesia, all patients had radial artery catheters placed for arterial blood pressure, blood gas, and hemoglobin measurements, and flow-directed pulmonary artery catheters inserted *via* the right internal jugular vein for right atrial and pulmonary artery pressure, mixed venous blood gas, and thermal dilution cardiac output measurements. The transducers attached to these two catheters were zeroed at the vertical level of the left atrium with patients in the lateral decubitus position, and the blood pressures were electronically meaned and recorded on paper (Hewlett-Packard®). In all patients, the end-tidal concentration of inhalation anesthetic was measured continuously by mass spectrometry (Chemtron). Additional monitoring in all patients included esophageal temperature, electrocardiogram, peak and plateau inspiratory airway pressure, and tidal volume (Wright Spirometer) measurements. Static compliance was calculated by dividing tidal volume by plateau airway pressure.

INDUCTION AND MAINTENANCE OF ANESTHESIA WITH INHALATIONAL ANESTHETICS

Subjects were either unpremedicated, or they received diazepam orally, 10 mg, or morphine intramus-

cularly, 0.1 mg/kg, 1 h before entering the operating room. Anesthesia was induced with sodium thiopental, 3–4 mg/kg *iv*. Inhalation anesthetics were then administered from a Drager flow-over vaporizer *via* a circle system and black rubber mask using a high fresh oxygen flow, and initial moderate over-pressure (2 MAC inspired concentration). As soon as possible, the end-tidal anesthetic concentration was held nearly constant for the next 1.0–1.2 h between 1.0 and 1.5 MAC. All subjects were paralyzed with a combination of pancuronium 0.04 mg/kg *iv*, and metocurine 0.15 mg/kg *iv*. During inhalation anesthesia and paralysis, intravenous fluids were administered so that neither systemic arterial, central venous, nor pulmonary artery diastolic pressures varied by more than 15% from pre-induction values. Additional doses of pancuronium were administered so as to achieve approximately a 90–95% motor blockade as indicated by a blockade monitor.

VENTILATION

After anesthetic induction, a left-sided double-lumen endotracheal tube (National Catheter Corp.) was inserted, and its proper position was confirmed initially by unilateral airway clamping and chest auscultation maneuvers. After the patient was turned into the lateral decubitus position, correct positioning of the double-lumen endotracheal tube was confirmed by fiberoptic bronchoscopy and by visual observation of lung collapse and mediastinal movement after the pleura was incised.

Two-lung ventilation conditions (steps 1 and 4, see experimental sequence section for description of the four steps) consisted of FI_O₂ = 1.0; tidal volume = 12 ml/kg; and respiratory rate adjusted to achieve a value of PaCO₂ between 35 and 40 mmHg. One-lung ventila-

tion conditions (steps 2 and 3) were $FI_{O_2} = 1.0$; tidal volume = 10 ml/kg; and respiratory rate adjusted so that Pa_{CO_2} was between 35 and 40 mmHg.

MAINTENANCE OF ANESTHESIA WITH INTRAVENOUS ANESTHETICS

Following 1 h of inhalation anesthesia and one-lung ventilation with patients in the lateral decubitus position, the inhalation anesthetics were discontinued and anesthesia was maintained with intravenously administered drugs. Intravenous anesthesia consisted of fentanyl 10–15 μ g/kg in several divided doses, diazepam 0.1 mg/kg in two to three divided doses, and 2–5 mg/kg of sodium thiopental in multiple divided doses. During intravenous anesthesia, intravenous fluids were administered so that neither systemic arterial, central venous, nor pulmonary artery diastolic pressures varied by more than 15% from pre-induction values.

EXPERIMENTAL SEQUENCE

Our experimental sequence consisted of four steps. Step 1 was two-lung ventilation during inhalation anesthesia. Final measurements made during step 1 were obtained after the pleura was incised (step 1 had a duration of 20–30 min). Step 2 was one-lung ventilation during inhalation anesthesia. One-lung ventilation conditions were initiated after the pleura was incised by opening one side of the double-lumen tube to atmosphere. To demonstrate achievement of stable one-lung ventilation conditions, arterial blood gas measurements (Pa_{O_2}) were obtained every 10 min until two consecutive Pa_{O_2} determinations were near equal (± 25 mmHg). Final measurements made during step 2 were obtained after 40 min of step 2 conditions. Step 3 was to discontinue inhalation anesthesia (which ended step 2), and to administer intravenous anesthesia (as described above) during one-lung ventilation. Final step 3 measurements were made after the end-tidal anesthetic concentration decreased to less than 0.1%. During steps 2 and 3, the surgeons gently dissected free the relevant artery, vein, and bronchus, but did not ligate them. Thus, Steps 2 and 3 were always completed before any pulmonary vessels were ligated, and all measurements were made when the surgeons were not compressing the non-dependent lung, blood loss was minimal, and the patients were hemodynamically stable. Step 4 was two-lung ventilation during intravenous anesthesia. We returned to two-lung ventilation conditions during intravenous anesthesia near the end of the surgical procedure, but before the pleura was closed. The experimental sequence and approximate time for each step is summarized below. Step 1—two-lung ventilation, inhalation anesthesia (20–30 min). Step 2—one-lung ventilation,

inhalation anesthesia (40 min). Step 3—one-lung ventilation, intravenous anesthesia (40–60 min). Step 4—two-lung ventilation, intravenous anesthesia (20–30 min).

CALCULATIONS

The following formulas were used to calculate venous admixture. Alveolar oxygen tension:

$$PA_{O_2} = FI_{O_2} \times (P_B - P_{H_2O}) - Pa_{CO_2}$$

Oxygen content (ml O_2 /100 ml blood):

$$C = (1.34 \times Hgb \times \% Sat) + (0.003 \times P_{O_2})$$

Venous admixture:

$$\dot{Q}_v/\dot{Q}_t = (Cc'O_2 - CaO_2)/(Cc'O_2 - C\bar{v}O_2)$$

Hemoglobin saturation was calculated from P_{O_2} corrected for P_{CO_2} , pH, and temperature, using the method of Kelman.⁹

STATISTICS

All results were analyzed by F-test and Student's paired *t* test, with $P < 0.05$ considered significant. Results are expressed as individual patient data and as mean \pm SD for each inhalational anesthetic group.

Results

Average values that characterized ventilation and gas exchange, hemodynamics, and inhalation anesthetic depth at each experimental step in each inhalation anesthetic group are presented in table 2.

VENTILATION DURING ALL FOUR EXPERIMENTAL SEQUENCE STEPS

There was no significant change in Pa_{CO_2} throughout the entire four-step experimental sequence (table 2). There was a significant increase in the plateau inspiratory airway pressure and a significant decrease in static compliance with the initiation of one-lung ventilation, but these airway pressure and compliance changes remained constant during one-lung ventilation (steps 2 and 3).

HEMODYNAMICS DURING ALL FOUR EXPERIMENTAL SEQUENCE STEPS

There was a significant decrease in systemic arterial blood pressure during inhalation anesthesia compared to intravenous anesthesia in both inhalation anesthetic groups (compare step 1 with 4 and step 2 with 3 in table 2). There was no significant change in the mixed venous oxygen tension associated with the administration of inhalational anesthetics (table 2).

TABLE 2. Average Values (+SD) Obtained at Each Experimental Step in Both Study Groups

Function	Variable	Anesthetic	Experimental Step			
			1 (2-LV, IH)	2 (1-LV, IH)	3 (1-LV, iv)	4 (2-LV, iv)
Ventilation and gas exchange	PaO ₂ (mmHg)	HAL	484 ± 49	116 ± 61*	155 ± 74*	411 ± 101*
		ISO	442 ± 58	232 ± 97*	245 ± 97	431 ± 104*
	PaCO ₂ (mmHg)	HAL	40 ± 3	41 ± 2	41 ± 2	39 ± 4
		ISO	40 ± 3	42 ± 3	41 ± 4	42 ± 6
	Q _s /Q _t (%)	HAL	14 ± 4	44 ± 9*	37 ± 9*	20 ± 8*
		ISO	19 ± 5	38 ± 8*	36 ± 5	20 ± 7*
	Pao plateau (cm H ₂ O)	HAL	24 ± 3	33 ± 4*	35 ± 4	26 ± 4*
		ISO	25 ± 4	31 ± 5*	32 ± 6	26 ± 6*
C _{STA} (ml/cm H ₂ O)	HAL	43 ± 12	27 ± 9*	26 ± 6	35 ± 7*	
	ISO	41 ± 10	25 ± 5*	24 ± 4	37 ± 11*	
Hemodynamics	SAP (mmHg)	HAL	84 ± 12	74 ± 6	91 ± 9*	88 ± 13
		ISO	78 ± 11	75 ± 6	92 ± 11*	92 ± 12
	CO (L · min ⁻¹)	HAL	4.2 ± 0.6	4.9 ± 1.1	4.4 ± 0.9	4.5 ± 1.1
		ISO	4.8 ± 0.9	5.6 ± 1.8	5.1 ± 1.4	5.2 ± 1.1
	PvO ₂ (mmHg)	HAL	52 ± 4	48 ± 5	45 ± 3	49 ± 6
		ISO	54 ± 4	54 ± 5	53 ± 5	56 ± 7
PAP (mmHg)	HAL	16 ± 6	19 ± 5*	18 ± 4	15 ± 5*	
	ISO	15 ± 5	18 ± 4*	18 ± 6	16 ± 5*	
Anesthetic depth	F _{ET} , % HAL or ISO	HAL	0.84 ± 0.10	1.08 ± 0.08*	0.08 ± 0.02*	0.03 ± 0.02*
		ISO	1.00 ± 0.20	1.17 ± 0.12*	0.08 ± 0.02*	0.04 ± 0.02*

2-LV = two-lung ventilation; 1-LV = one-lung ventilation; IH = inhalation anesthesia; iv = intravenous anesthesia; HAL = halothane; ISO = isoflurane; Q_s/Q_t = shunt; Pao = airway pressure; C_{STA} = static compliance; SAP = systemic arterial pressure (mean); CO = cardiac

output (mean); PAP = pulmonary artery pressure (mean); F_{ET} = fraction in end-tidal gas.

* P < 0.05 compared to previous experimental step.

INHALATION ANESTHETIC DEPTH DURING ALL FOUR EXPERIMENTAL SEQUENCE STEPS

Table 2 shows the end-tidal concentrations of the inhalational anesthetics at each of the four experimental steps. At the time of change from inhalation anesthesia to intravenous anesthesia, the end-tidal concentrations of halothane and isoflurane corresponded to 1.1 and 1.0 MAC, respectively. After 40–60 min of intravenous anesthesia, the end-tidal concentrations were below 0.1 MAC for both halothane and isoflurane, and still lower at the time of return to two-lung ventilation during intravenous anesthesia.

ARTERIAL OXYGENATION AND SHUNT DURING CONVERSION FROM TWO-LUNG VENTILATION TO ONE-LUNG VENTILATION (STEP 1 TO STEP 2)

Initiation of one-lung ventilation caused a significant decrease in PaO₂. Figure 1 shows serial PaO₂ determinations in individual patients during the transition from two-lung to one-lung ventilation during inhalation anesthesia (step 1 to 2). The last PaO₂ value in figure 1 was taken to represent the PaO₂ during stable one-lung ventilation conditions under inhalation anesthesia (step 2). The mean ± SD difference in PaO₂ between the last PaO₂ and the preceding PaO₂ for all patients was 13 ± 10 mmHg, with a range of 2–25 mmHg.

ARTERIAL OXYGENATION AND SHUNT DURING CHANGE FROM INHALATION ANESTHESIA TO INTRAVENOUS ANESTHESIA DURING STABLE ONE-LUNG VENTILATION CONDITIONS (STEP 2 TO STEP 3)

Figures 2 and 3 show the average and individual PaO₂ and shunt results, respectively, obtained at each experi-

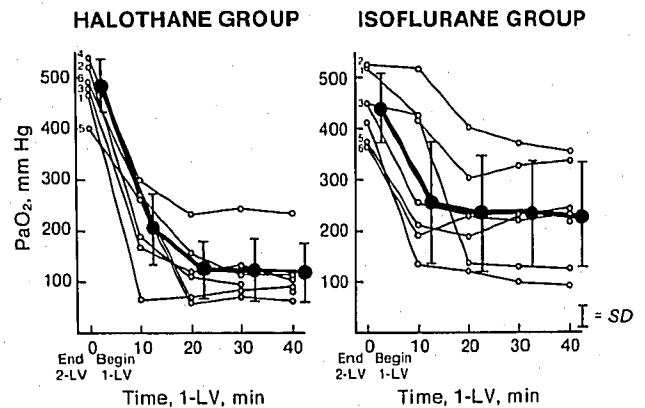


FIG. 1. Serial PaO₂ measurements during conversion from two-lung ventilation (2-LV) to one-lung ventilation (1-LV) in both the halothane and isoflurane groups. The mean PaO₂ value at the end of 2-LV and just before the beginning of 1-LV is the same as the first mean PaO₂ value in figure 2, and the last (40 min) mean 1-LV PaO₂ value is the same as the second mean PaO₂ value in figure 2. The small numbers to the left of the first data point refer to the patient number in table 1.

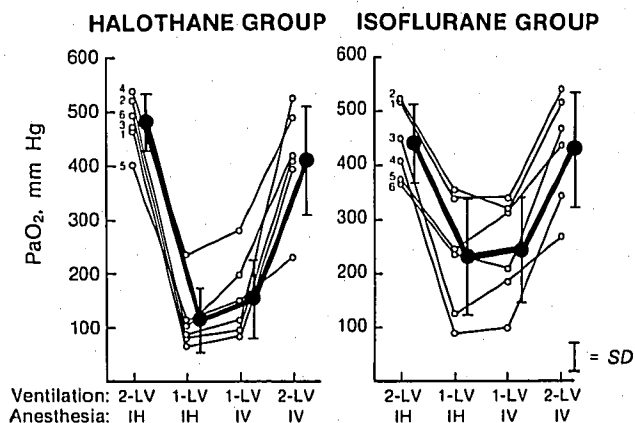


FIG. 2. P_{aO_2} values during the four experimental sequence steps. Conversion from two-lung ventilation (2-LV) to one-lung ventilation (1-LV) during inhalation anesthesia (IH) in both groups (step 1 to step 2) caused a very large and significant decrease in P_{aO_2} . Conversion from IH to intravenous anesthesia (iv) during 1-LV (step 2 to step 3) caused a slight but significant increase in P_{aO_2} in the halothane group, and a very slight and nonsignificant increase in P_{aO_2} in the isoflurane group. Conversion from 1-LV to 2-LV during iv anesthesia (step 3 to step 4) caused a very large and significant increase in P_{aO_2} in both the halothane and isoflurane groups. The small numbers to the left of the first data point refer to the patient number in table 1.

mental step in both inhalation anesthetic study groups. Discontinuing halothane during one-lung ventilation caused a slight, but statistically significant, increase in P_{aO_2} (39 ± 29 mmHg) and decrease in shunt ($7 \pm 2\%$ of

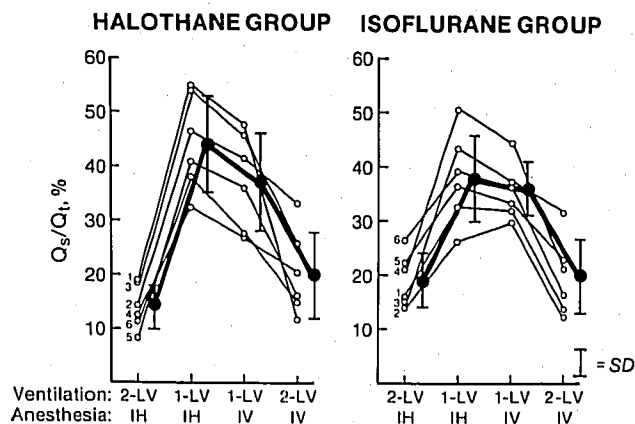


FIG. 3. Shunt (\dot{Q}_s/\dot{Q}_t) values during the four experimental sequence steps. Conversion from two-lung ventilation (2-LV) to one-lung ventilation (1-LV) during inhalation anesthesia (IH) in both groups (step 1 to step 2) caused a very large and significant increase in \dot{Q}_s/\dot{Q}_t . Conversion from IH anesthesia to intravenous anesthesia (iv) during 1-LV (step 2 to step 3) caused a slight but significant decrease in \dot{Q}_s/\dot{Q}_t in the halothane group, and a very slight and nonsignificant decrease in \dot{Q}_s/\dot{Q}_t in the isoflurane group. Conversion from 1-LV to 2-LV during iv anesthesia (step 3 to step 4) caused a very large and significant decrease in \dot{Q}_s/\dot{Q}_t in both the halothane and isoflurane groups. The small numbers to the left of the first data point refer to the patient number in table 1.

the cardiac output). Discontinuing isoflurane during one-lung ventilation caused a very slight, but nonsignificant, increase in P_{aO_2} (13 ± 19 mmHg) and decrease in shunt ($2 \pm 2\%$ of the cardiac output).

ARTERIAL OXYGENATION AND SHUNT DURING CONVERSION FROM ONE-LUNG TO TWO-LUNG VENTILATION (STEP 3 TO STEP 4)

Return to two-lung ventilation from one-lung ventilation during intravenous anesthesia caused a significant increase in P_{aO_2} and a significant decrease in shunt in both inhalation anesthetic groups. The P_{aO_2} and shunt values during two-lung ventilation and intravenous anesthesia were not significantly different from the P_{aO_2} and shunt values during two-lung ventilation and inhalation anesthesia.

Discussion

We found that during one-lung atelectasis, 1 MAC halothane anesthesia slightly but significantly increased shunt and decreased P_{aO_2} (compared to intravenous anesthesia), whereas 1 MAC isoflurane anesthesia very slightly and nonsignificantly increased shunt and decreased P_{aO_2} (compared to intravenous anesthesia). Consideration should be given to the stability of the one-lung atelectasis condition, whether hypoxic pulmonary vasoconstriction was present in the atelectatic non-dependent lung, and what effect halothane and isoflurane anesthesia might have had on non-dependent lung hypoxic pulmonary vasoconstriction.

Crucial to the interpretation of our findings of the effect of halothane and isoflurane on arterial oxygenation during one-lung atelectasis is evidence that we achieved stable gas exchange during the one-lung atelectatic condition in each patient. Our serial P_{aO_2} measurements during the transition from two-lung ventilation to one-lung ventilation indicate that stability of one-lung ventilation was reached as early as 20 min of one-lung ventilation. Nevertheless, we continued one-lung ventilation for 40 min so that the last two consecutive P_{aO_2} measurements differed by less than 25 mmHg and were separated by at least 10 min. Although major changes in cardiac output, pulmonary vascular pressures, and mixed venous oxygen tension can affect gas exchange during one-lung ventilation, the only significant hemodynamic change that occurred during conversion from inhalation anesthesia to intravenous anesthesia during one-lung ventilation was a moderate increase in systemic arterial pressure. The fact that the final two-lung ventilation shunt, P_{aO_2} , P_{aCO_2} , and compliance values were not significantly different than the initial two-lung ventilation values indicates that neither the non-dependent or dependent lung was damaged during the anesthesia and surgical experience.

Blood flow to the non-dependent lung was most probably acutely reduced when this lung was non-ventilated. The non-dependent lung, when ventilated, receives an average of 40% of the total blood flow (due to the effect of gravity) when the right and left lungs are non-dependent an equal number of times (as was the case in both our groups).¹⁰⁻¹² Our patients (both inhalation anesthetic groups together) had an average shunt of 16% during two-lung ventilation in the lateral decubitus position. If it is assumed that this shunt was equally distributed between the non-dependent and dependent lungs, and blood flow to the atelectatic non-dependent lung was not acutely decreased, then the shunt during non-dependent lung atelectasis would have been 48%. However, during intravenous anesthesia, we found a total shunt across the lungs of only 36% of the cardiac output (both inhalation anesthetic groups together) during non-dependent lung atelectasis, which is consistent with a major acute decrease in non-dependent lung blood flow.

The most likely mechanism of the acute decrease in blood flow to the atelectatic non-dependent lung was hypoxic pulmonary vasoconstriction. The finding of a 36% shunt during intravenous anesthesia (no inhibition of hypoxic pulmonary vasoconstriction) and one-lung ventilation is exactly what would be predicted in this situation (two-lung ventilation shunt of 16%) from a single-lung hypoxic pulmonary vasoconstrictor response that should decrease the blood flow to the hypoxic lung by 50%.¹³ Although surgical interference may be a cause of decreased non-dependent lung blood flow, we made our measurements when the lung was not being manipulated. Other possible, but unlikely, mechanisms for acute non-dependent lung blood flow reduction during clinical one-lung ventilation have been previously discussed.⁷

Data in animals indicate that 1 MAC halothane and isoflurane anesthesia should inhibit a one-lung hypoxic pulmonary vasoconstrictor response by 20%.⁶ In other words, instead of a single lung being able to decrease its blood flow by 50%, 1 MAC halothane and isoflurane anesthesia would cause a single hypoxic lung to be able to diminish its blood flow only by 40%. Using our average conditions outlined above, a 20% inhibition of the non-dependent lung hypoxic pulmonary vasoconstrictor response from a 50% blood flow reduction to a 40% blood flow reduction would cause an increase in total shunt across both lungs during one-lung atelectasis of 4% of the cardiac output. This is in close agreement with the finding of an increase of shunt of 7% and 2% of the cardiac output in the halothane and isoflurane anesthetized groups, respectively, compared to intravenous anesthesia. The lower Pa_{O_2} and higher shunt values in the halothane group during inhalation anesthesia and one-lung ventilation is consistent with a greater depres-

sion of hypoxic pulmonary vasoconstriction by halothane compared to isoflurane. Although a vessel-rich tissue such as the lung should washout inhalation anesthetic quickly, the same (but smaller) differences in Pa_{O_2} and shunt during intravenous anesthesia and one-lung ventilation may reflect failure to achieve full recovery of hypoxic pulmonary vasoconstriction.

In summary, we established stable one-lung ventilation conditions during inhalation anesthesia. The limited increase in shunt across both of the lungs during one-lung atelectasis and intravenous anesthesia was consistent with the presence of hypoxic pulmonary vasoconstriction in the non-dependent lung. Our data are also consistent with the notion that halothane and isoflurane had only a small inhibitory effect on the one-lung hypoxic pulmonary vasoconstrictor response. In view of the usual efficacy of non-dependent lung CPAP, we conclude that the use of halogenated drugs in patients undergoing one-lung ventilation is no longer a significant clinical issue.

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