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Continuous-flow Apneic Ventilation during Thoracotomy

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Continuous-flow apneic ventilation (CFAV) by endobronchial insufflation of conditioned gas was evaluated in dogs during thoracotomy. In Group 1 (n = 6), dogs were anesthetized with pentobarbital (25 mg/kg). An endobronchial catheter (2.5 mm ID) was introduced into each mainstem bronchus using a fiberoptic bronchoscope and held in place by an endotracheal tube. Before the onset of CFAV (total flow 1.0 $l \cdot kg^{-1} \cdot min^{-1}$, the animals were paralyzed with pancuronium bromide and muscle relaxation was monitored with a peripheral nerve stimulator. The CFAV delivery system consisted of a flow meter, air/oxygen blender, oxygen analyzer, heated humidifier, and ultrasonic spirometer. Blood gas values were measured after 30 min of spontaneous ventilation, and CFAV with: 1) closed chest, fractional inspired O_2 concentration (FI_{O_2}) 0.21; 2) open chest, FI_{O_2} 0.21; 3) open chest, FI_{O_2} 0.21, continuous positive airway pressure (CPAP) 5 mmHg; and 4) open chest FI_{O_2} 0.4, CPAP 5 mmHg. This last combination resulted in a mean Pa_{O_2} of 113.1 ± 5.5 (SEM) mmHg and a Pa_{CO_2} of 35.0 ± 2.1 (SEM) mmHg. In Group 2 (n = 6), animals with open chests were ventilated with CFAV (FI_{O_2} 0.4 and CPAP 5 mmHg) for 5 h. Adequate oxygenation and ventilation were achieved. Pa_{CO_2} after 5 h of CFAV was 41.8 ± 1.9 (SEM) mmHg compared with 40.8 ± 1.9 (SEM) mmHg during spontaneous breathing. Pa_{O_2} after 5 h of CFAV was 138.1 ± 11.7 (SEM) mmHg. There were no significant changes observed in vascular pressures. Significant differences in other hemodynamic parameters were probably due to pentobarbital anesthesia. Adequate gas exchange can be achieved during CFAV in dogs with open chests for 5 h. (Key words: Anesthetic techniques; continuous flow apneic ventilation; endobronchial insufflation. Surgery; thoracotomy. Ventilation; artificial; apnea.)

IN A PREVIOUS STUDY of gas exchange during continuous–flow apneic ventilation (CFAV),[1] a constant air flow of 1 $l \cdot kg^{-1} \cdot min^{-1}$ through endobronchial catheters was required to maintain normal arterial oxygen (Pa_{O_2}) and arterial carbon dioxide (Pa_{CO_2}) tensions. Adequate gas exchange was maintained for 5 h in all animals, and no changes in vascular pressures and shunt fraction were observed.

In this same study the importance of accurate placement of the endobronchial catheters was pointed out. With the tips of the catheters placed above the carina, gas distribution, as evaluated by Xenon[133] studies, was limited to the large airways with no peripheral distribution, resulting in low Pa_{O_2} levels and increased Pa_{CO_2} levels. Endobronchial catheters permitted gas distribution to the peripheral airways, and oxygenation and ventilation were normal.

However, the efficacy of this method of ventilation has never been evaluated under open-chest conditions.[1–4] The reasons why the technique may not work under open-chest conditions include atelectasis with open pleura and a possible reduction in cardiogenic oscillations, resulting in reduced convective mixing. The present study was designed to determine if CFAV could be used as a method of ventilation in experimental animals during thoracotomy.

Materials and Methods

Twelve mongrel dogs of both sexes (average body weight 21.5 kg) were fasted overnight and divided into two experimental groups of six dogs each.

GROUP 1

Unpremedicated dogs were anesthetized with sodium pentobarbital (25 mg/kg) intravenously and placed in the supine position. Supplementary doses of anesthetic were given intermittently during the experiment, 100 mg every 30 min. Heating blankets were used to maintain body temperature. Balanced electrolyte solution was infused at the rate of 3 $ml \cdot kg^{-1} \cdot h^{-1}$. Each mainstem bronchus was cannulated with a polyethylene catheter (2.5 mm ID and 3.5 mm OD) using a fiberoptic bronchoscope. The endobronchial catheters were angled approximately 30° from their axis with the trachea. The catheter placed in the left mainstem bronchus extended approximately 3 cm below the carina, and the catheter in the right mainstem bronchus extended approximately 2.5 cm below the carina. Following the placement of the catheters, the trachea was then intubated with a 9 mm ID Hi-Lo Jet® (NCC, Division Mallinckrodt, Inc.) tracheal tube, and its cuff inflated to hold the endobronchial catheters in place. Accurate positioning of the endobronchial catheters was again verified by fiberoptic bronchoscopy. Animals were permitted to breathe spontaneously, and after a 1-h period, arterial blood gas samples were drawn for control values. Prior to the onset of CFAV, each endobronchial catheter was connected to the gas delivery system, which consisted of an air/oxygen blender, calibrated flow meter, oxygen analyzer, heated humidifier (MR 500 Duo Servo® Controlled Heated Humidifier, Fisher-Paykel Medical, Inc.) and connecting tubing. The animals were paralyzed with pancuronium bromide (0.15 mg/kg), and complete neuromuscular blockade was monitored with a peripheral

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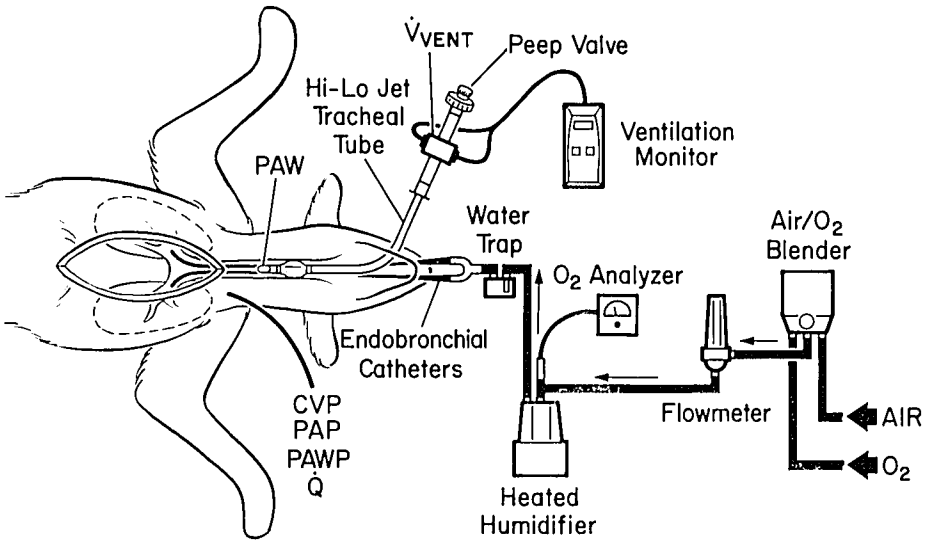

FIG. 1. Schematic diagram of the experimental design of CFAV during thoracotomy.

nerve stimulator. CFAV was started by turning on the flow meter in the gas delivery system, and total flow was calculated as 1 $l \cdot kg^{-1} \cdot min^{-1}$. It was assumed that gas flow would be equally divided between the lungs. No manual or mechanical hyperinflation of the lungs was applied during the periods of CFAV. After 1 h of closed-chest CFAV (total flow 1.0 $l \cdot kg^{-1} \cdot min^{-1}$), a second arterial blood gas sample was analyzed, and physiologic parameters were recorded. Under sterile conditions a median sternotomy was then performed, both lungs were exposed, and CFAV was continued. Arterial blood gases were measured after 30 min of: 1) CFAV with open chest with air as the ventilating gas; 2) open chest with air and continuous positive airway pressure (CPAP) of 5 mmHg (6.8 cmH_2O); and 3) with open chest, fractional inspired O_2 concentration ($F_{I_{O_2}}$) of 0.4 and CPAP of 5 mmHg (6.8 cmH_2O). Throughout the experiment, ECG, arterial blood pressure (BP), heart rate (HR), and temperature were measured and recorded. Airway pressure (P_{aw}) was monitored through a Statham® P23 ID transducer connected by water-filled tubing to the pressure port of the Hi-Lo Jet® tracheal tube. This port opened at the tip of the tracheal tube. Accuracy of the delivery of gas flow was checked by connecting the tracheal tube to an ultrasonic spirometer (Bourns® LS 75). CPAP was achieved by connecting the tracheal tube to a spring-loaded, diaphragm-type PEEP valve. A schematic of the experimental design is shown in figure 1.

TABLE 1. Mean Values of Pa_{O_2} and Pa_{CO_2} during CFAV in Group 1 (n = 6)

	Pa_{O_2}	Pa_{CO_2}
Spontaneous Respiration Fi_{O_2} 0.21	81.1 ± 6.7	35.7 ± 1.8
CFAV/closed-chest Fi_{O_2} 0.21	68.6 ± 5.2	34.2 ± 1.1
CFAV/open-chest Fi_{O_2} 0.21	48.2 ± 3.8*	39.2 ± 1.8
CFAV/open-chest Fi_{O_2} 0.21 CPAP 5 mmHg	50.35 ± 4.5*	33.5 ± 1.2
CFAV/open-chest Fi_{O_2} 0.4 CPAP 5 mmHg	113.1 ± 5.5*	35.0 ± 2.1

Spontaneous respiration group was used as control.
Values (±SEM) in mmHg.
* Significantly different compared with control ($P < 0.05$).

GROUP 2

Anesthetic management was the same as Group 1. A femoral artery was catheterized for measurement of arterial pressure and arterial blood sampling. A triple-lumen thermodilution catheter (Swan-Ganz 7 French, Instrumentation Laboratory) was advanced to the pulmonary artery through the external jugular vein for determination of cardiac output (CO), continuous pulmonary artery pressure (PAP), central venous pressure (CVP), and pulmonary arterial wedge pressure (PAWP) recording. The measurements were made and blood samples were drawn at the time of spontaneous ventilation, 1 h after CFAV with closed chest, and subsequently every h for a period of 5 h with CFAV and open chest. In this second group, CFAV was carried out with a heated and humidified air/oxygen mixture, $F_{I_{O_2}}$ of 0.4, and CPAP of 5 mmHg (6.8 cmH_2O). Total flow of ventilating gas was calculated as 1.0 $l \cdot kg^{-1} \cdot min^{-1}$. P_{aw} was measured in a similar fashion as in Group 1. Statistical significance was established by

TABLE 2. Mean Values of Blood Gases during Spontaneous Respiration (Fi_{O_2} 0.21), CFAV with Closed Chest (Fi_{O_2} 0.4), and during a 5-h Period of CFAV with Open Chest (Fi_{O_2} 0.4) in Group 2

			CFAV Open Chest				
	Control	CFAV Closed Chest	1 h	2 h	3 h	4 h	5 h
*p*H	7.39 (±0.02)	7.40 (±0.03)	7.41 (±0.04)	7.40 (±0.03)	7.42 (±0.03)	7.42 (±0.02)	7.41 (±0.01)
Pa_{O_2}	93.5 (±1.4)	146.9* (±7.3)	126.4* (±9.3)	138.0* (±5.0)	136.4* (±7.7)	140.2* (±10.9)	138.1* (±11.7)
$P\bar{v}_{O_2}$	55.5 (±3.1)	58.0 (±4.1)	49.1† (±4.2)	45.7*† (±4.3)	42.6*† (±4.1)	43.5*† (±1.6)	39.9*† (±3.1)
Pa_{CO_2}	40.8 (±1.9)	45.2 (2.8)	42.2 (±1.0)	41.3 (±3.0)	40.0 (±1.8)	39.0 (±1.8)	41.8 (±1.9)

Spontaneous respiration was used as control.
Values (±SEM) in mmHg and *p*H units (±SEM).
* Significantly different compared with control ($P < 0.05$).
† Significantly different compared with CFAV with closed chest ($P < 0.05$).

two-way analysis of variance followed by the Dunnett *t* test. $P < 0.05$ was considered significant.

Results

GROUP 1

Table 1 shows Pa_{O_2} and Pa_{CO_2} values obtained during spontaneous ventilation and during CFAV in animals with closed chest and open chest. There were no significant differences in Pa_{CO_2} between the control condition (spontaneous respiration) and CFAV during closed chest and thoracotomy.

Pa_{O_2} fell significantly during CFAV after median sternotomy (81.1 ± 6.7 *vs.* 48.2 ± 3.8, mmHg ± SEM), and a CPAP of 5 mmHg during thoracotomy did not affect oxygenation significantly. After an increase in FI_{O_2} to 0.4, Pa_{O_2} increased to 113.1 + 5.5 (mmHg ± SEM). There were no significant changes in HR or BP throughout the experiment, and the temperature remained normal in all groups. P_{aw} with CFAV without CPAP was in the range of 2–3 mmHg.

GROUP 2

Table 2 shows blood gas results during 5 h of CFAV in animals with open chests. All CFAV/open-chest values were compared with the control mode (spontaneous respiration), and to the values obtained from animals during CFAV/closed-chest. Pa_{O_2} increased during CFAV as a consequence of a higher FI_{O_2}. There was also a significant increase in the alveolar–arterial oxygen difference [$C(a\text{-}v)_{O_2}$] during CFAV. There was no statistically significant increase in Pa_{CO_2} values during 5 h of CFAV/open-chest.

Table 3 shows the results of vascular pressures during 5 h of CFAV in animals with open chests as compared with CFAV/closed-chest and spontaneous respiration. There were no significant changes in MAP, CVP, PAP, and PCWP.

TABLE 3. Vascular Pressures during Spontaneous Respiration, CFAV with Closed Chest and during a 5-h Period of CFAV with Open Chest in Group 2

			CFAV Open Chest				
	Control	CFAV Closed Chest	1 h	2 h	3 h	4 h	5 h
MAP	185.2 (±8.4)	185.3 (±8.0)	188.2 (±9.7)	182.8 (±7.2)	179.3 (±9.4)	176.3 (±9.5)	168.3 (±14.2)
CVP	5.5 (±1.7)	3.6 (±0.8)	4.8 (±0.7)	4.8 (±0.8)	5.2 (±0.8)	4.6 (±0.9)	5.3 (±1.3)
PAP	26.5 (±4.7)	24.4 (±1.1)	27.9 (±2.0)	25.1 (±1.9)	29.5 (2.3)	27.9 ±(2.3)	26.9 (±2.4)
PCWP	9.2 (±1.1)	9.8 (±1.3)	12.5 (±0.5)	11.3 (±0.6)	11.8 (±1.2)	10.8 (±0.6)	10.8 (±0.7)

See text for abbreviations.
Spontaneous respiration was used as control.
Values (±SEM) in mmHg.

TABLE 4. Hemodynamic Variables and Body Temperature during Spontaneous Respiration, CFAV with Closed Chest and during a 5-hr Period of CFAV with Open Chest

	Control	CFAV Closed Chest	CFAV Open Chest				
			1 h	2 h	3 h	4 h	5 h
CO l/min	6.25 (±0.7)	4.94* (±0.5)	3.82*† (±0.4)	3.37*† (±0.4)	2.95*† (±0.4)	2.80*† (±0.3)	2.55*† (±0.2)
CI $l \cdot min^{-1} \cdot m^{-2}$	8.35 (±1.5)	6.54* (±1.0)	5.15*† (±0.9)	4.63*† (±1.1)	3.99*† (±0.9)	3.74*† (±0.6)	3.36*† (±0.5)
SV ml	33.9 (±4.0)	27.2* (±2.7)	21.6*† (±1.9)	19.0*† (±2.7)	16.8*† (±2.0)	15.9*† (±2.1)	14.7*† (±1.6)
SI $ml \cdot min^{-1} \cdot m^{-2}$	45.2 (±8.2)	35.8 (±5.4)	28.9* (±4.9)	26.1* (±6.2)	22.7*† (±4.6)	21.2*† (±3.7)	19.2*† (±2.6)
HR beats/min	185 (±12.0)	181 (±1.6)	1.76 (±5.6)	181 (±7.9)	175 (±5.1)	178 (±6.1)	176 (±6.2)
SVR $dyn \cdot cm^{-5} \cdot s^{-1}$	2509.8 (±369.3)	3138.2 (±410.3)	4084.3*† (±511.7)	4992.6*† (±573.4)	5057.2*† (±585.6)	5133.3*† (±529.6)	5241.8*† (±500.5)
PVR $dyn \cdot cm^{-5} \cdot s^{-1}$	206.7 (±31.5)	236.7 (±21.5)	349.4 (±75.6)	351.1 (±57.3)	509.0*† (±83.7)	495.8*† (±71.7)	501.6*† (±36.3)
RVWI units	3.67 (±1.3)	2.33* (±0.4)	2.03* (±0.4)	1.67* (±0.4)	1.69* (±0.3)	1.54* (±0.3)	1.34* (±0.2)
LVWI units	21.4 (±3.0)	17.0* (±2.2)	12.9*† (±2.1)	11.3*† (±2.4)	10.1*† (±1.8)	9.3*† (±1.4)	8.1*† (±1.2)
$C(a\text{-}v)_{O_2}$ mmHg	1.77 (±0.35)	1.77 (±0.36)	2.42 (±0.37)	3.06 (±0.51)	3.80*† (±0.72)	3.08 (±0.66)	4.06*† (±0.53)
Q_S/Q_t %	11.0 (±0.2)	11.1 (±0.3)	11.8 (±0.4)	12.4 (±0.6)	13.1*† (±0.8)	12.3 (±0.5)	13.4*† (±0.7)
Temperature ° C	37.3 (±0.2)	37.3 (±0.3)	36.8 (±0.3)	36.8 (±0.3)	36.7 (±0.3)	36.6† (±0.3)	36.5*† (±0.3)

See text for abbreviations.
Spontaneous respiration was used as control.
Values (±SEM) in mmHg, ° C (±SEM), and units (±SEM).
* Significantly different compared with control ($P < 0.05$).
† Significantly different compared with CFAV with closed chest ($P < 0.05$).

Hemodynamic variables $C(a\text{-}v)_{O_2}$, and temperature are shown in table 4. All values were compared with the CFAV/closed-chest and control group (spontaneous respiration). No significant changes were observed in HR. CO, CI, stroke volume (SV), stroke index (SI), intrapulmonary shunt (Q_S/Q_t), right ventricular work index (RVWI), and left ventricular work index (LVWI) all decreased during the course of the experiment. Systemic vascular resistance (SVR), peripheral vascular resistance (PVR), and $C(a\text{-}v)_{O_2}$ increased significantly. Body temperature decreased during 5 h from the control value of 37.3 to 36.5° C ($P < 0.05$), but was not clinically significant.

Discussion

Several previous studies have shown that adequate ventilation can be obtained during endobronchial insufflation in anesthetized and paralyzed dogs.[1-3] Effective CO_2 elimination occurred only when the ventilatory gas was delivered into the lumen of the main bronchi. A marked hypercarbia followed withdrawal of the catheter tips above the carina. Oxygenation was usually adequate and depended on the FI_{O_2}. The possible mechanism of gas transport during CFAV is related to the convective gas movement, effects of cardiogenic oscillations, and molecular diffusion. Conceptually, the lungs can be divided into two zones: zone 1, affected by the jet of gas; and zone 2, independent of the jet.[5] Because of the high flow rates used during CFAV, bidirectional streaming will take place in the area closest to the insufflation jet. Gas will enter and exit the same airway simultaneously. The jet of insufflating gas will generate turbulence below the tips of the catheters, and the turbulent diffusivity will be a part of the gas transport mechanism. The higher the flow of insufflated gas, the further into the periphery of the lungs this turbulence will reach. This may, perhaps, partially explain why patients subjected to CFAV retain some CO_2, but dogs do not. The flow used in the animal experiments was calculated as 1 $l \cdot kg^{-1} \cdot min^{-1}$, whereas such high flows were thought to be potentially dangerous in humans and only 0.6 to 0.7 $l \cdot kg^{-1} \cdot min^{-1}$ was used.[4]

Effectiveness of cardiogenic oscillation during CFAV is strongly supported in the literature, but is still controversial. West and Hugh-Jones, measuring the air flow in

the lobar and segmental bronchi of humans during bronchoscopy, observed that pulsations clearly augmented the expiratory flow.[6] The authors suggested that the oscillations might enhance mixing between dead space and alveolar gas. Studies by Engel *et al.* suggested that cardiac action and the resulting flow pulsations might increase the effective diffusion coefficient on the airways by five-fold.[7,8] Similar results and conclusions were presented by Fukuchi *et al.*[9]

Chakrabarti *et al.*, by measuring mixing efficiency in the lungs of dogs by helium and hexafluoride washin technique and using cardiopulmonary bypass, were unable to demonstrate any effect on gas mixing attributable to the heart beat.[10] This was also confirmed by the study of Horsfield *et al.*[11] This controversial subject was again studied recently by Burwen *et al.* during tracheal insufflation of oxygen in anesthetized and paralyzed dogs.[5] By analyzing continuous N_2 washout curves in alive and dead animals, the authors found that cardiogenic oscillation increased gas mixing four-fold and that this value was independent of the insufflation flow rate.

Augmented diffusion in the central airways combined with molecular diffusion in the periphery of the lungs can account for the gas transport during high-frequency oscillation but, during CFAV, it probably has a minimal effect.[12]

This study was designed to find out if opening of the chest will affect CO_2 elimination during CFAV. In spontaneously breathing subjects, opening of the pleura will result in severe ventilatory impairment. Usually negative intrapleural pressure becomes equal to atmospheric pressure, elastic recoil of the lung tissue is no longer opposed, and the lung tends to collapse. Respiratory muscles become ineffective in moving gases in and out of the lungs, leading to acute hypoxia and hypercarbia. Early reports described development of respiratory acidosis in patients undergoing thoracotomy under general endotracheal anesthesia with unassisted or manually assisted respiration.[13-17] This was obviously due to the inefficient controlled ventilation during anesthesia.[18] Adequate oxygenation was insured by delivering a high $F_{I_{O_2}}$.[13,14,16]

The respiratory consequences of the open pneumothorax can be corrected by controlled ventilation and adequate $F_{I_{O_2}}$. Our findings indicate that adequate CO_2 elimination can be achieved without any respiratory movement for at least 5 h in animals with open chest by using continuous endobronchial insufflation of conditioned gas. Thoracotomy, by itself, did not affect ventilation but, as might be expected, $P_{a_{O_2}}$ markedly decreased and was corrected by an increase in the $F_{I_{O_2}}$.

It might be postulated that some CO_2 was diffused through the pleural space, as the chest was widely open with both pleural spaces exposed. This phenomenon probably occurs, but we are not aware of any reference on the subject.

In the first group of animals, the application of 5 mmHg of CPAP during CFAV and open chest did not affect oxygenation. However, the $F_{I_{O_2}}$ was 0.21 and an increase in $P_{a_{O_2}}$ might not be observed because because under these conditions, the shunt lines as presented on the iso-shunt diagram (fig. 1) are close together.[19] CPAP of 5 mmHg (6.8 cmH_2O) seems to be relatively small, since the P_{aw} values were in the range of 2–3 mmHg. Mean alveolar pressures ($P_{\bar{A}}$), however, could be substantially higher. Our technique of P_{aw} measurements was by means of the pressure port of the tracheal tube connected to the pressure transducer. It is known that P_{aw} significantly underestimates the P_A, at least during high-frequency ventilation.[20] Simon *et al.*, using the clamping technique of P_{aw} measurements, found in dogs that elevation of P_A above the P_{aw} was seen to be a function of mean flow and largely independent of the frequency–tidal–volume combination that produced the flow.[20] Saari *et al.*, while studying the relationship between P_{aw} and lung volume during high-frequency ventilation, found a substantial increase in lung volume, despite constant P_{aw}.[21]

Adequate CO_2 elimination and oxygenation was achieved during 5 h of experiments with the animals studied while in the supine position so as to be comparable to earlier experiments. Gas exchange might have been different had the animals been studied while in the lateral position.

High flow rates of ventilatory gases during CFAV could possibly cause damage to the bronchial mucosa on lung parenchyma. We did not examine the lungs microscopically after the experiments, as macroscopic examination revealed no evidence of damage to the bronchial mucosa. Several authors investigated this problem during high-frequency ventilation when flow rates and velocities of ventilating gases are even higher than during CFAV. Smith *et al.* reported no damage to the airway or lung parenchyma in one dog after 24 h of high-frequency jet ventilation.[22] No apparent parenchymal lung damage was found by Keszler *et al.* during use of high-frequency jet ventilation in dogs for a period of 24 h.[23] Nordin *et al.* performed electron microscopic studies of tracheal muscosa after high-frequency jet ventilation. Severe damage was observed only when dry gases were used, but with humidification no significant changes compared with normal mucosa were found.§ No pathologic evidence of mucosal damage was observed by Doyle *et al.* during 72 hours of continuous ventilation with high-frequency jet ventilation and different humidification systems.[24]

During 5-h CFAV experiments in animals with closed chests, a marked decrease in body temperature has been

§ Nordin U, Klain M, Keszler H: Electron-microscopic studies of tracheal mucosa after high frequency jet ventilation (abstract). Crit Care 10:211, 1981.

observed,[1] which was thought to be the cause of significant differences in HR, CO, and vascular resistances between the beginning and end of the experiments. A more effective heated humidifier and a warming mattress prevented hypothermia in the present studies. Changes in cardiovascular parameters similar to the closed-chest CFAV experiments were, however, observed in animals with open chests. Even though vascular pressures remained unchanged during 6 h of CFAV, a decrease in CO, CI, SV, SI, and increase in SVR and PVR, as well as decreases in RVWI and LVWI were noted. This progressive deterioration in the cardiovascular status of animals is probably related to the pentobarbital anesthesia rather than the method of ventilation or decreased temperature. Several previous studies clearly demonstrated the profound cardiovascular effect of this anesthetic agent.[25–29] Nash *et al.* showed that pentobarbital produces a progressive decrease in canine CO during the first 3 h of anesthesia.[27] The maximum decrease exceeded 44% of control value, and was statistically significant from the 1-h observation throughout the remainder of the 7-h experimental period. As in our studies, a decrease in mean BP was not observed. The results of their studies and ours indicate that a different type of anesthetic agent, such as a potent narcotic, should be used for future studies investigating the cardiovascular response to various techniques or procedures.

This method of ventilation may provide adequate gas exchange during thoracotomy in human subjects. Possible clinical applications include surgery of major airways and for periods of apnea to facilitate closed-heart surgery. Bilateral or unilateral endobronchial insufflation in combination with conventional intermittent positive–pressure or high–frequency ventilation might prove useful in preventing intraoperative hypoxia. Further studies and modifications of this technique may be needed before it can be tested clinically.

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