Altered Perfusion, Ventilation, Anesthesia and Lung-surface Forces in Dogs

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The combination of hyperventilation, lowered pulmonary blood flow and pentobarbital–O_2 anesthesia led to a progressive increase in retractive forces of the lung accompanied by a gradual reduction in inflatable volume in dogs studied for eight hours. After four hours of ventilation, significant changes began to appear at the higher transpulmonary pressures. Results after four hours of ventilation were similar in other dogs treated identically except for the addition of 1.2 per cent halothane to the inspired gas. Hyperventilation and the absence of pulmonary arterial blood flow led to significant ($P < 0.05$) reductions in percentages of maximum lung volumes during deflation pressure–volume loops in shorter periods of time regardless of anesthetic technique. The effect of anesthesia on lung-surface forces in living dogs appears small compared with the effects of hyperventilation and diminished blood flow. (Key words: Hyperventilation; Blood flow; Surfactant; Halothane; Barbiturate; Pressure–volume relationships.)

THE EFFECTS of halothane, chloroform and nitrous oxide on surface-active forces in excised unperfused dogs' lungs have been reported.[1] Changes in deflating pressure–volume slopes reflect changes in surfactant function.[2–6] With excised unperfused dogs' lungs percentages of maximum lung volume (%MLV) after ventilation with halothane and chloroform at 23 C were less than after ventilation with air alone, but only with halothane were the lung volumes significantly smaller at the end of the study.[1] This demonstration that lipid-soluble anesthetics could alter surfactant function during ventilation in freshly excised dogs' lungs prompted the question whether deleterious effects of anesthetics on surfactant performance could be detected in normothermic dogs after thoracotomy. To this purpose, the interactions of hyperventilation, pulmonary blood flow, anesthesia and lung-surface forces were investigated in intact dogs.

Methods

Fifty mongrel dogs (weighing 7.2–13.5 kg) were anesthetized with intravenous pentobarbital sodium, 30 mg/kg. Each dog was supine on a K-thermia blanket (model R.K. 101, Gorman-Rupp Industries, Inc., Belville, Ohio), and the esophageal temperature was monitored by an indwelling thermistor (Yellow Springs Co.). The temperature of the dog was kept between 35 and 38 C. To insure that esophageal temperatures accurately reflected body temperatures, rectal temperatures in ten dogs were recorded simultaneously. Both femoral arteries were cannulated for arterial sampling and for measurement of arterial pressure. A femoral vein was cannulated to allow for injections of 6 per cent dextran in saline solution to replace blood loss and removal. No other fluid replacement was given. A right atrial catheter was passed via a jugular vein. Tracheostomy was performed and a modified Carlens tube was placed on the carina; the modification involved the addition of metal connectors with machined three-way metal stopcocks. The intraluminal pressures were then monitored by attachment to a Hewlett-Packard 7718 system by Sanborn high-sensitivity transducers (series 26SB). Great

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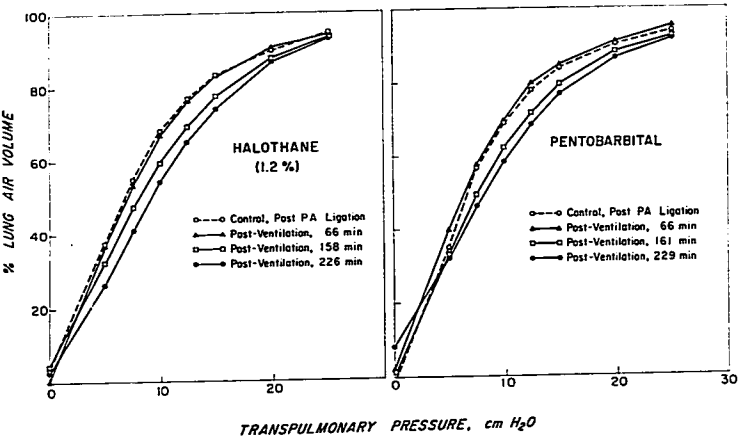

FIG. 1. Deflation pressure–volume curves. Means of measurements from ten dogs are given in each panel. Hyperventilation (50 ml/kg body weight, P_{aco_2} approximately 20 torr) of dogs anesthetized with pentobarbital–1.2 per cent halothane or pentobarbital alone led to a progressive increase in lung retractive forces after ligation of the pulmonary artery. Changes were significant at transpulmonary pressures 15–7.5 cm H_2O after three to four hours, $P < 0.05$ (tables 1 and 2).

care was taken to secure an airtight fit to each lung by inflation and maintenance of constant pressure. Succinylcholine HCl, 500 mg, was given intramuscularly initially and subsequently when needed. A right thoracotomy was performed in each dog. The right pulmonary artery (PA) was ligated in each of 20 dogs. All 50 dogs were ventilated at a rate of 20 breaths/min. A tidal volume of 50 ml/kg was produced by an Emerson postoperative ventilator; the inspiratory/expiratory ratio was 1:2. A Wright respirometer was used to check the tidal volume delivered. The inspired oxygen concentration was maintained between 85 and 90 per cent and monitored with a Beckman E2 paramagnetic oxygen analyzer.

The lung quasistatic volume–pressure relationships were studied in the following manner: The right lung was gently aspirated to minimum volume; dry USP helium (99.9 per cent pure) was injected into the right lung in 100-ml increments every 2–3 sec while the airway pressure was recorded with a calibrated Sanborn transducer. After an airway pressure of 30 cm H_2O had been reached and maintained for 5 sec, helium was aspirated in 50-ml increments until the airway pressure reached zero; after the withdrawal of each 50 ml, time was allowed for the pressure to stabilize before the next withdrawal (2–3 sec). With new helium the process was repeated twice more; the third determination was used to plot the volume–pressure diagrams. Gas volume in the lung was corrected for temperature, pressure, and water vapor according to the following formula:

$$V_C = (V_I \cdot P_B \cdot T_B)/T_R\,(P_A - P_W)$$

where V_C = corrected gas volume, V_I = volume of dry USP helium injected, P_B = barometric pressure, T_B = esophageal body temperature, T_R = room temperature, P_A = airway pressure, and P_W = water vapor pressure. The amount of helium and water vapor withdrawn during each deflation was subtracted from the total corrected lung volume. The amount of helium and water vapor remaining within the lung at the beginning of the third volume–pressure loop reflected the residual lung volume and was the starting point of inflation for the volume–pressure study. No correction for oxygen or carbon dioxide diffusion out of the

pulmonary arterial blood was made.[7] During determination of volume–pressure relationships, the left lung was ventilated at 20–25 ml/kg body weight and the right thoracotomy opened widely with rib retractors.

Anesthetic Technique

In 20 dogs (ten with PA ligation) anesthesia was maintained with 1.2 ± 0.04 (SE) per cent halothane in humidified 98.8 per cent oxygen. Vaporization of halothane was by a Fluotec vaporizer (Fraser Sweatman Inc., Hatfield, Pa.). The actual concentration of halothane delivered was checked by a gas–liquid chromatograph (Hewlett-Packard 5750) in conjunction with a gas-sampling valve and 0.5-ml gas loop.[1] The peak heights on the chromatogram were obtained from Ohio Calibration Standard halothane and were compared with those from the gas samples. The remaining 30 dogs (ten with PA ligation) were kept anesthetized with intermittent doses of 60 mg pentobarbital sodium.

In 40 dogs pressure–volume, cardiac output and blood-gas measurements were made at hourly intervals; during the longer eight-hour study (ten dogs) the measurements were made every two hours. Arterial blood gases were measured by standard Radiometer equipment maintained at 37 ± 0.1 C. For dogs whose temperatures differed from 37 C, standard blood-gas temperature-correction factors were applied.[8, 9] Immediately before arterial sampling cardiac output was determined by analysis of dye-dilution curves with a Gilson densitometer (model DTL) after injection of indocyanine green. Amplification and recording were via the Hewlett-Packard 7718 system. Cardiac outputs were calculated using the forward-triangle method.[10] Cardiac indices were calculated after obtaining the body surface area by the equation of Rubner.[11] For comparison of lung volumes, expressed as percentages of maximum lung volume (%MLV), a *t* test for paired variates was used when measurements from the same dog were obtained. Student's *t* test for unpaired variates was employed when we compared measurements obtained from different dogs. Significance values (*P*) for *t* were obtained from the table of Fisher and Yates.[12] All dogs were sacrificed at the end of the experiment.

Results

Dogs With Pulmonary-Artery Ligation

Hyperventilation and absence of pulmonary arterial blood flow for four hours led to a significant ($P < 0.05$) reduction of %MLV during deflation at transpulmonary pressures (P_{TP}) from 15 to 7.5 cm H_2O (fig. 1, tables 1 and 2). Mean %MLV decreased with time of ventilation after ligation of the pulmonary artery (fig. 1). The depression of %MLV was threefold

Table 1. Lung Pressure–Volume Changes during Deflation in Ten Dogs after Ligation of the Pulmonary Artery and Hyperventilation with 1.2 ± 0.06 Per Cent Halothane in O_2 for 3–4 Hours

	Control* (%MLV)		1–2 Hours (%MLV)			2–3 Hours (%MLV)			3–4 Hours (%MLV)		
	Mean	SD	Mean	SD	*P*†	Mean	SD	*P*†	Mean	SD	*P*†
P_{TP} (cm H_2O)											
25	94.4	4.1	94.1	4.0	NS‡	93.3	4.0	NS	92.6	4.8	NS
20	89.8	3.8	90.5	4.2	NS	87.5	6.8	NS	86.8	7.0	NS
15	83.3	3.1	83.2	5.2	NS	77.4	12.8	NS	73.8	12.9	<0.05
12.5	76.9	6.2	76.4	6.7	NS	69.3	15.0	NS	65.2	14.7	<0.05
10	68.2	5.2	66.9	8.5	NS	59.3	16.6	NS	54.3	16.9	<0.05
7.5	54.9	6.0	53.3	8.7	NS	47.4	16.2	NS	40.9	16.0	<0.05
5	37.4	7.8	37.0	8.5	NS	32.1	13.1	NS	26.1	12.6	<0.05
0	3.3	6.2	0.5	1.9	NS	4.2	6.9	NS	2.8	5.1	NS
MLV (ml)	541	170	522	148	NS	499	133	<0.1	438	127	<0.01

* Pressure–volume loop measured after PA ligation and before hyperventilation.
† In comparison with the control (paired variates).
‡ Not significant.

TABLE 2. Lung Pressure–Volume Changes during Deflation in Ten Dogs after Ligation of the Pulmonary Artery and Anesthesia with Pentobarbital and Hyperventilation for 3–4 Hours

	Control* (%MLV)		1–2 Hours (%MLV)			2–3 Hours (%MLV)			3–4 Hours (%MLV)		
	Mean	SD	Mean	SD	P†	Mean	SD	P†	Mean	SD	P†
P_{TP} (cm H_2O)											
25	93.5	2.8	94.9	3.5	NS‡	92.3	3.7	NS	91.6	3.6	<0.05
20	89.9	3.4	90.6	4.0	NS	87.8	4.3	<0.05	86.4	4.0	<0.01
15	83.8	5.2	84.6	6.0	NS	79.2	7.4	<0.02	76.6	7.8	<0.01
12.5	77.6	6.1	78.6	7.6	NS	71.4	8.2	<0.02	68.1	9.4	<0.01
10	68.8	6.5	69.5	8.0	NS	62.0	10.4	<0.05	58.3	10.1	<0.01
7.5	56.7	6.6	57.6	9.1	NS	49.3	10.4	<0.02	46.1	10.0	<0.01
5	35.1	7.6	38.6	14.4	NS	33.1	9.3	NS	31.8	7.7	NS
0	−1.2	2.9	2.0	4.9	<0.1	1.8	5.4	<0.1	8.0	6.7	<0.01
MLV (ml)	530	138	537	156	NS	520	134	NS	488	119	NS

* Pressure–volume loop measured after PA ligation and before hyperventilation.
† In comparison with the control (paired variates).
‡ Not significant.

greater after pulmonary arterial ligation than with intact pulmonary arteries, the anesthetic and pressure–volume histories being identical (tables 1–4). No significant difference in reduction of %MLV ascribable to differences in anesthetic technique (pentobarbital–halothane vs. pentobarbital) was found, although after PA ligation mean %MLV at P_{TP} 20–5 cm H_2O was always lower with pentobarbital–halothane 1.2 per cent anesthesia (fig. 1, tables 1 and 2); the maximum inflatable volumes were significantly less in dogs ventilated with halothane after three to four hours (table 1).

Dogs Without Pulmonary-Artery Ligation

For dogs without ligation of the pulmonary artery the combination of hyperventilation, diminished pulmonary blood flow and pentobarbital–halothane–O_2 anesthesia led to a significant increase in retractive forces of the lung at one transpulmonary pressure (P_{TP} 12.5 cm H_2O) in approximately four hours; at other transpulmonary pressures between 25 and 10 cm H_2O, mean %MLV was lower after three to four hours of ventilation, although these changes were not significant (table 3).

TABLE 3. Lung Pressure–Volume Changes during Deflation in Ten Dogs Hyperventilated with 1.2 ± 0.05 Per Cent Halothane in O_2 for 3–4 Hours after Thoracotomy Alone

	Control* (%MLV)		1–2 Hours (%MLV)			2–3 Hours (%MLV)			3–4 Hours (%MLV)		
	Mean	SD	Mean	SD	P†	Mean	SD	P†	Mean	SD	P†
P_{TP} (cm H_2O)											
25	94.9	2.9	94.2	2.7	NS‡	94.3	2.6	NS	93.4	2.8	<0.1
20	91.0	3.3	90.3	2.8	NS	90.4	3.2	NS	89.7	2.4	NS
15	84.5	4.6	84.0	5.1	NS	83.6	4.7	NS	82.2	4.7	NS
12.5	79.0	6.0	77.6	6.5	NS	77.4	6.5	<0.1	75.7	5.8	<0.05
10	69.6	7.6	68.2	8.2	NS	68.9	8.5	NS	67.9	7.2	NS
7.5	55.9	8.4	54.5	8.5	NS	56.0	8.7	NS	55.8	8.3	NS
5	36.1	9.5	37.6	8.6	NS	40.1	9.8	NS	40.2	8.4	NS
0	0.9	6.9	2.5	4.5	NS	7.0	8.4	<0.01	3.9	4.9	NS
MLV (ml)	581	196	571	207	NS	575	180	NS	554	206	NS

* Pressure–Volume loop measured after thoracotomy and before hyperventilation.
† In comparison with control (paired variates).
‡ Not significant.

TABLE 4. Lung Pressure–Volume Changes during Deflation in Ten Dogs Anesthetized with Pentobarbital and Hyperventilated for 3–4 Hours after Thoracotomy Alone

	Control* (%MLV)		1–2 Hours (%MLV)			2–3 Hours (%MLV)			3–4 Hours (%MLV)		
	Mean	SD	Mean	SD	P†	Mean	SD	P†	Mean	SD	P†
P_{TP} (cm H_2O)											
25	95.6	3.8	95.1	3.5	NS‡	95.0	4.1	NS	93.7	4.8	<0.05
20	91.8	4.0	91.4	3.7	NS	91.3	4.5	NS	89.4	4.7	<0.05
15	86.4	3.7	86.3	4.9	NS	84.3	5.8	<0.1	83.7	5.8	<0.1
12.5	80.8	5.2	80.6	6.6	NS	78.9	7.9	NS	79.0	7.4	NS
10	71.7	6.4	72.3	8.2	NS	70.7	9.6	NS	70.0	8.9	NS
7.5	57.8	8.0	59.7	9.2	NS	57.9	11.1	NS	58.1	10.0	NS
5	38.4	9.1	42.6	10.0	NS	41.0	10.0	NS	42.5	11.4	<0.1
0	1.8	5.3	3.4	4.7	NS	4.6	6.6	NS	12.0	7.2	<0.001
MLV(ml)	473	118	454	91	NS	441	97	<0.02	454	117	NS

* Pressure–Volume loop measured after thoracotomy and before hyperventilation.
† In comparison with control (paired variates).
‡ Not significant.

In ten dogs hyperventilated for three to four hours under pentobarbital anesthesia alone, significant changes were noted at the higher transpulmonary pressures (P_{TP} 20 and 25 cm H_2O) (table 4). The maximum inflatable volumes in both groups decreased with time, but the changes at four hours still were not significant.

In ten other dogs studied for eight hours under pentobarbital anesthesia, significant changes became apparent at the higher transpulmonary pressures at six and eight hours (fig. 2, table 5). The greatest decrease in %MLV was 12 per cent at P_{TP} 10 cm H_2O. Concomitant with the volume–pressure effect was an 18.5 per cent decrease in inflatable volume.

Cardiac indices and acid–base status are shown in table 6. No significant difference between esophageal and rectal temperatures (mean 37 C) was found.

Discussion

A reduction in pulmonary blood flow is associated with impairment of surface activity of the alveolar lining.[5, 6, 13–15] Following occlusion of the pulmonary artery, alveolar perfusion is partially maintained by the bronchial circulation; the contribution of this flow in providing substrate for surfactant formation was demonstrated by Naimark, who found a 90 per cent reduction in incorporation of palmitic acid, a precursor of surfactant, after ligation of the pulmonary artery in dogs.[16] In excised dogs' lungs devoid of pulmonary blood flow, surface activity is dependent on the de-

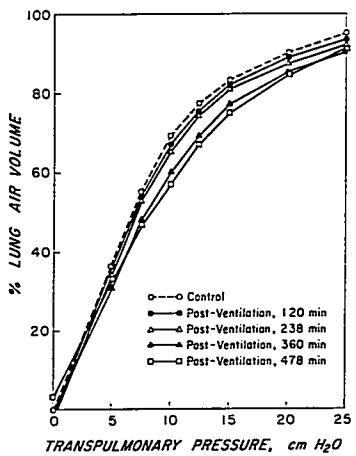

FIG. 2. Deflation pressure–volume curves for ten dogs hyperventilated for eight hours under pentobarbital anesthesia. In contrast to the experiments illustrated in figure 1, the right pulmonary artery was not ligated. Hyperventilation (50 ml/kg body weight, P_{aco_2} approximately 15 torr) led to a progressive increase in lung retractive forces. Changes were significant at transpulmonary pressures 10–25 cm H_2O after eight hours (table 5).

TABLE 5. Lung Pressure–Volume Changes during Deflation in Ten Dogs Anesthetized with Pentobarbital and Hyperventilated for 8 Hours after Thoracotomy Alone

	Control* (%MLV)		2 Hours (%MLV)			4 Hours (%MLV)			6 Hours (%MLV)			8 Hours (%MLV)		
	Mean	SD	Mean	SD	Pt	Mean	SD	Pt	Mean	SD	Pt	Mean	SD	Pt
P_{tp} (cm H_2O)														
25	95.4	3.8	93.6	2.4	<0.1	92.6	2.5	<0.1	90.6	3.3	<0.01	90.8	2.7	<0.01
20	90.1	4.0	89.4	3.5	NS‡	87.9	2.8	NS	85.9	5.3	<0.05	85.3	4.3	<0.05
15	83.1	6.9	82.3	7.1	NS	81.0	4.7	NS	77.4	9.5	NS	75.5	8.4	<0.05
12.5	77.6	8.4	75.9	8.6	NS	74.5	6.6	<0.1	69.4	13.1	<0.1	67.6	11.5	<0.05
10	69.3	10.2	67.3	9.5	NS	65.9	7.9	NS	60.2	14.5	<0.1	57.3	14.9	<0.05
7.5	55.3	11.7	54.1	11.0	NS	53.0	9.4	NS	48.2	14.8	NS	47.1	15.1	NS
5	36.5	11.9	36.5	9.3	NS	34.1	8.6	NS	31.8	12.1	NS	33.4	12.7	NS
0	−0.1	4.0	−2.3	1.3	<0.1	−3.0	0.8	<0.1	−1.4	2.9	NS	3.7	12.9	NS
MLV(Ml)	444	83.0	448	98.3	NS	442	100.7	NS	393	91.4	<0.05	382	99.1	<0.001

* Pressure–volume loop measured after thoracotomy and before hyperventilation.
† In comparison with control (paired variates).
‡ Not significant.

gree of stretching of the alveolar lining, adequate oxygenation, temperature and the presence of metabolic inhibitors such as anesthetic agents.[1, 17, 18] The greater the ventilation, the larger the consumption of oxygen and glycogen.[19] Consequently, the interaction of hyperventilation, diminished pulmonary blood flow and anesthesia can influence the rate of impairment of surface activity.

In this study, reduction in pulmonary blood flow was accomplished by various means: first, by mechanical occlusion of the right pulmonary artery; second, by decreasing cardiac output. The latter is a result of two effects, that of the anesthetic agent on myocardial function and that of diminished venous return produced by increased intrathoracic pressure. After ligation of the pulmonary artery, the increase in retractive forces of the lung is progressive and significant after two to three hours of hyperventilation even with an intact bronchial circulation.

When lungs are overinflated, pulmonary edema, atelectasis, hemorrhage and alterations in surface forces may ensue.[20–22] Gross atelectasis after prolonged hyperventilation was not apparent in this study, but maximum inflatable volume had decreased by 18.5 per cent after eight hours. However, this reduction in MLV, representing noninflatable areas, should not contribute to alterations in measurements of pulmonary volumes expressed as %MLV and, therefore, cannot fully account for any accompanying increase in retractive forces.[4] For instance, Edmunds and Huber found no change in helium or saline pressure–volume loops, expressed as %MLV, after ligation of the pulmonary arteries of dogs, although the maximum inflatable volumes were reduced.[5] Their surgical procedure for pulmonary arterial ligation lasted 20–40 min; the dogs were not hyperventilated and were allowed to awaken and breathe spontaneously until sacrificed for excised lung pressure–volume loop measurements. Gross pathologic changes were present a day after ligation of the artery, but only a few focal areas of blood-filled alveoli were present. These areas of hemorrhagic atelectasis tended to become confluent as time passed, but there was no change in the pulmonary static recoil of the inflatable portions of dogs'

TABLE 6. Acid-Base Measurements and Cardiac Indices

	Time of Ventilation (min)		P_{aCO_2} (torr)		pH_a		Cardiac Index (l/m²/min)	
	Mean	SE	Mean	SE	Mean	SE	Mean	SE
Pentobarbital-halothane, ten dogs	0		27.2	2.0	7.41	0.02	2.8	0.3
	65	2.7	12.4	1.1	7.62	0.02	1.8	0.2
	158	5.9	12.0	1.0	7.54	0.02	1.3	0.2
	219	7.7	11.5	1.0	7.55	0.02	1.2	0.2
Pentobarbital-halothane and PA ligation, ten dogs	0		29.3	1.9	7.42	0.03	3.1	0.2
	65	2.3	16.7	1.6	7.50	0.03	1.4	0.1
	158	4.4	17.3	1.6	7.45	0.02	1.3	0.2
	226	6.4	24.7	2.3	7.32	0.04	1.2	0.1
Pentobarbital, ten dogs	0		26.9	2.1	7.42	0.02	3.0	0.3
	63	2.6	12.7	1.4	7.57	0.03	1.6	0.2
	149	3.8	11.8	0.5	7.56	0.02	1.5	0.1
	227	8.0	14.7	1.2	7.52	0.02	1.5	0.1
Pentobarbital and PA ligation, ten dogs	0		30.6	2.8	7.44	0.03	3.2	0.4
	66	2.7	16.1	1.4	7.55	0.03	2.0	0.3
	161	4.3	18.1	1.6	7.50	0.04	1.7	0.3
	229	5.0	23.7	3.4	7.40	0.06	1.5	0.2
Pentobarbital, ten dogs	0		27.1	10.1	7.50	0.09	3.2	0.9
	120	2.0	12.8	2.0	7.59	0.08	2.6	0.6
	238	1.7	13.6	2.6	7.56	0.07	2.2	0.7
	360	2.6	13.1	2.6	7.51	0.07	2.3	1.1
	478	3.4	17.0	4.4	7.39	0.07	1.9	0.8

lungs for as much as a week after ligation of the pulmonary artery.[5] The lack of change in surface forces of inflated alveoli in their study can be attributed to the lower level of ventilation and, thus, lower demand for surfactant.

With intact but diminished pulmonary circulation (cardiac output approximately 50 per cent of normal), significant depressions in %MLV were seen at high pulmonary volumes after eight hours of hyperventilation. Such changes have been interpreted as early evidence of diminished surfactant function.[4] In the presence of marginal circulation the increase in retractive forces is accentuated by extreme hyperventilation. This maximal stretching and compression of the alveolar surface lining depletes the supply of surfactant and overcomes the rate of its synthesis and replacement.[22] Although the turnover rate of surfactant is unknown, the turnover rate of dipalmitoyl lecithin (a major component of surfactant) is estimated from studies in rats to be 14 hours under conditions of spontaneous normal ventilation and perfusion.[23]

Faridy has shown that ventilation of the left lower lobes of dogs with tidal volumes which are 30 per cent of maximum lobar air volumes does not affect surfactant function provided total blood flow is at least 15 per cent of the control value.[12] The rate of ventilation of Faridy's study was 12 breaths/min, whereas we ventilated at 20 with a tidal volume which was approximately 60 per cent of MLV. Consequently, it would seem that the increased ventilation we used stressed surfactant function to a point where an increase in perfusion from 15 to 50 per cent of normal was barely sufficient to allow for adequate metabolism and replacement.[13] This concept is supported by the effect of temperature on surfactant function. Ventilation of excised dogs' lungs at room temperature (24 C) with a tidal volume of 30 per cent MLV leads to significant depression of surfactant function after two hours, but no depression at 37 C.[17] The depression produced by ventilation at 24 C is reversed by incubation at 37 C.[17, 18] Initial attempts to lower body temperature to 32 C in our study

resulted in high mortality and were abandoned. Hypothermia by lowering surfactant metabolism would be expected to accentuate the changes we report.

The method we used to assess lung retractive forces is fraught with possible errors. Accurate measurement of quasistatic intratracheal pressure is difficult in the time allowed for pressure stabilization (2–3 sec); in addition, inaccuracies are contributed by the small quantities of helium that escape from the lungs and the CO_2 and O_2 that diffuse in. To minimize these problems the same point of the asymptotic lung-pressure curve was taken on all measurements. It might have been advantageous to have postmortem excised pressure–volume loops in order to determine surfactant function, but the lungs were biopsied for other studies.

In our previous study of excised unperfused dogs' lungs we found that the lipid-soluble anesthetics, halothane and chloroform, depressed surfactant function more than the barbiturate when other experimental conditions were identical.[1] In the present study we did not find any significant added effect on surfactant function of a lipid-soluble inhalation anesthetic (halothane 1.2 per cent in oxygen after pentobarbital induction) over continued barbiturate anesthesia, although changes were always greater with the addition of halothane. The effect of anesthesia on surfactant function appears small compared with the effects of hyperventilation and lowered blood flow.

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