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Function of Each Lung in Spontaneously Breathing Man Anesthetized with Thiopental–Meperidine

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Intrapulmonary gas distribution between individual lungs was studied in five healthy, anesthetized, spontaneously breathing adult volunteers in the supine and both lateral decubitus positions. Thiopental–meperidine anesthesia in spontaneously breathing subjects altered the regional distribution of the FRC/TLC ratio. This was associated with an alteration in intrapulmonary gas distribution; the distribution followed the predicted pattern seen in awake man with voluntarily induced reduction in the FRC/TLC ratio. In eight of ten observations, fractional nonshunted blood flow to the dependent lung decreased in the lateral decubitus positions, contrary to the findings in awake, spontaneously breathing man. The results suggest that thiopental–meperidine anesthesia *per se*, that is, without mechanical ventilation and muscle paralysis, can alter intrapulmonary gas distribution. The mechanism by which general anesthesia with thiopental–meperidine promotes a change in the FRC/TLC ratio remains to be clarified. (Key words: Spontaneous breathing; Thiopental–meperidine anesthesia; Body position; Differential lung function.)

We previously reported [1] a study of intrapulmonary gas distribution in man during anesthesia, muscle paralysis, and mechanical ventilation. We found that intrapulmonary gas distribution was more uniform under these conditions than in the conscious state. In another study,[2] we investigated the function of each lung in anesthetized, paralyzed, mechanically ventilated subjects in the supine and lateral positions. In the lateral positions the nondependent lung received a larger fraction of the tidal volume than the dependent lung. This result is contrary to the findings in studies showing preferential ventilation of the dependent lung in conscious, spontaneously breathing man.[3-5]

Thus far, our studies have not distinguished between the effects of mechanical ventilation with muscle paralysis and the effects of thiopental–meperidine anesthesia. One might consider the possibility that the decrease in FRC that occurs with the induction of general anesthesia might alter the regional FRC/TLC ratios, and that these in turn would be associated with an alteration in intrapulmonary gas distribution. If this were correct, anesthesia alone, that is, without mechanical ventilation and muscle paralysis, should alter gas distribution. Therefore, we decided to study the distribution of inspired gas between the left and right lungs in anesthetized man during spontaneous breathing, thereby eliminating the effects of mechanical ventilation and muscle paralysis.

The results of the study showed preferential ventilation of the nondependent lung in the lateral positions, similar to that observed in anesthetized, paralyzed, mechanically ventilated subjects,[2] and unlike ventilation in awake subjects breathing spontaneously.[4] Thus, it appears that thiopental–meperidine anesthesia alone, without muscle paralysis and mechanical ventilation, can alter intrapulmonary gas distribution. Possible mechanisms are discussed.

Methods

Five healthy unpremedicated male volunteers † (ages 24–31 years, weights 75–104 kg) were studied. Each was premedicated with scopolamine hydrobromide, 0.43 mg, iv, and

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† Physicians and nurse anesthetists only were permitted to participate in this study. The nature of the study, the manner in which it would be conducted, and its possible risks were outlined in a lengthy interview. Consent to participate was accepted only after the volunteers had had suitable time for consideration.

TABLE 1. Physical Characteristics and Lung Volumes* (Sitting, Awake)

	Physical Characteristics			Lung Volumes (liters)†			
	Age (Years)	Height (cm)	Weight (kg)	TLC	VC	FRC	RV
Subject 1	31	189	91	8.0	6.3	3.2	1.7
Subject 2	29	182	104	5.7	4.7	1.7	1.0
Subject 3	24	178	75	7.6	5.4	3.8	2.2
Subject 4	25	181	83	5.9	4.7	2.3	1.2
Subject 5	29	175	78	6.4	5.1	2.6	1.3
Mean	28	181	86	6.7	5.2	2.7	1.5

* Measurements made at body temperature and pressure saturated with water vapor (BTPS) in five male subjects.

† TLC = total lung capacity; VC = vital capacity; FRC = functional residual capacity; RV = residual volume.

then anesthetized with thiopental sodium (average dose, 15.1 mg/kg) and meperidine hydrochloride (average dose, 6.5 mg/kg). Muscle paralysis for intubation and for changes in body position was accomplished with intravenous injections of succinylcholine chloride (average total dose, 150 mg). After the trachea and larynx had been sprayed with 4 per cent lidocaine (Xylocaine), the trachea was intubated with a double-lumen cuffed endotracheal tube (Carlens; resistances of the tube were 5.7 cm H_2O/l/sec [left side] and 6.2 cm H_2O/l/sec [right side], at a flow rate of 0.5 l/sec). Following resumption of spontaneous breathing, the subjects were connected to a specially designed multichannel valve (deadspace 18 ml) to permit rapid changes of inspiratory gas mixtures for the two lungs simultaneously.

Inspiratory gas from cylinders containing either 66 per cent oxygen in nitrogen or 100 per cent oxygen flowed through large-bore, corrugated tubes to the multichannel valve and from there through the Carlens tube to the lungs. The gas expired from each lung flowed through the Carlens tube to the multichannel valve and on through heated pneumotachographs (Fleisch, No. 1, Instrumentation Associates, Inc., New York) and large-bore tubings into neoprene bags. Expired gases from each lung were collected separately in neoprene bags, which were evacuated just prior to gas collections. Oxygen uptake, carbon dioxide elimination, minute ventilation, and tidal volume of each lung were determined from measurement of volumes (gasometer) of expired gas collected over a 10-minute period, from respiratory frequencies, and from oxygen and carbon dioxide concentrations of inspired ($F_{I_{O_2}}$ 0.66) and expired gases (duplicate Haldane analyses). Functional residual capacity (FRC) of each lung was calculated from the volumes of expired gas and the concentrations of nitrogen in alveolar and expired gas after 7-minute nitrogen clearance periods. Alveolar nitrogen concentrations prior to nitrogen clearance ($F_{I_{O_2}}$ 0.66) were calculated by subtracting from barometric pressure: water vapor tension (P_{H_2O} at body temperature); Pa_{CO_2}, and $P_{A_{O_2}}$, determined by substitution of appropriate values in the alveolar gas equation. Alveolar nitrogen concentrations at the end of the nitrogen clearance periods were determined by nitrogen meters. Expired gases were analyzed for oxygen and carbon dioxide (duplicate Haldane analyses) and expired nitrogen concentrations were calculated from the difference. Expired nitrogen concentrations were corrected for nitrogen contained in inspired gas (<0.5 per cent) and for nitrogen eliminated from blood and tissue. The total correction factor for the nitrogen eliminated from blood and tissue ($F_{I_{N_2}}$ 0.34) was assumed to be 0.34/0.80 of that determined by Cournand *et al.*[6] for $F_{I_{N_2}}$ 0.80. Correction factors for nitrogen eliminated from blood and tissue through each lung were determined by multiplying the total correction factor with the fractional nonshunted blood flow (fractional oxygen uptake) of each lung.

TABLE 2. Effects of Position on Lung Function*

Ventilatory Component	No.	Supine Position (Mean ± SE)	No.	Lateral Decubitus Position† (Mean ± SE)
Ventilation (l/min, BTPS)	5	5.23 ± 0.51	10	5.48 ± 0.29
O_2 uptake (ml/min, STPD)	5	291 ± 13	10	293 ± 15
CO_2 elimination (ml/min, STPD)	5	191 ± 10	10	192 ± 5
Pa_{CO_2} (torr)	4	58 ± 4	9	54 ± 2
Pa_{O_2} (torr)				
$F_{I_{O_2}} = 0.66$	4	295 ± 24	9	306 ± 10
$F_{I_{O_2}} = 1.0$	4	490 ± 23	8	501 ± 10
V_D/V_T‡	4	0.46 ± 0.02	9	0.44 ± 0.01
FRC, anesthetized (liters, BTPS)	5	2.09 ± 0.26	10	2.43 ± 0.22
Body temperature (C)	5	36.8 ± 0.1	10	36.9 ± 0.1
Respiratory frequency (breaths/min)	5	8.4 ± 1.6	10	8.0 ± 0.8
FRC/TLC‡	5	0.31 ± 0.03	10	0.36 ± 0.02
Lung clearance ratio (LCR)	9	2.8 ± 0.3	9	3.2 ± 0.2

* Total measurements = left plus right lung.
† Combined values for left and right lateral decubitus positions.
‡ V_D = physiologic deadspace; V_T = tidal volume; FRC = functional residual capacity; TLC = total lung capacity.

TABLE 3. Function of the Right Lung as Percentage of Total: Mean Values and Ranges

Ventilatory Component	Position		
	Supine	Left Lateral Decubitus	Right Lateral Decubitus
Ventilation	52.1 (48.8–56.4)	56.4 (51.7–61.0)	44.7* (34.1–51.9)
Nonshunted blood flow	54.8 (50.2–61.1)	56.8 (46.4–63.1)	49.3 (41.7–61.1)
CO_2 elimination	53.5 (48.9–57.9)	59.0 (52.4–65.1)	44.1* (29.9–54.1)
FRC	56.8 (52.3–61.1)	68.8† (61.4–76.1)	43.6*‡ (37.6–48.8)

* Significant difference ($P < 0.05$) between left and right lateral positions.
† Significant difference ($P < 0.05$) between supine and left lateral positions.
‡ Significant difference ($P < 0.05$) between supine and right lateral positions.

TABLE 4. Mean of the Differences: Supine Minus Dependent Lung (Per Cent)

Ventilatory Component	Mean ± SE*
Ventilation	5.8 ± 1.9†
Nonshunted blood flow	3.7 ± 1.9
CO_2 elimination	7.5 ± 2.6†
FRC	12.6 ± 1.0†

* Number of observations, 10.
† Statistically significant, $P < 0.025$.

The cumulative volume of ventilation necessary to reduce end-tidal N_2 concentration to 5 per cent was determined and the ratio of this volume to FRC was designated the "lung clearance ratio."[1]

The relative oxygen uptake of each lung reflects the relative amount of nonshunted blood flow to each lung, since with $F_{I_{O_2}}$ 0.66, arterial oxygen tensions were approximately 300 torr and hemoglobin saturation in the arterial blood was likely to have been complete. Since these conditions existed, arterial–venous oxygen content differences across each lung should have been nearly identical, differing only in the amount of oxygen in physical solution. Arterial blood was sampled midway during the 10-minute gas collection period for the oxygen uptake determination ($F_{I_{O_2}}$ 0.66) and again at the end of the 7-minute nitrogen clearance period ($F_{I_{O_2}}$ 1.0). Arterial blood gas tensions and *p*H were measured by electrodes at a water-bath temperature of 37 C and corrected to the subjects' rectal temperatures (thermistor).

Total physiologic deadspace (left plus right lung) was calculated by the following equation:

$$V_D = V_{E_L} \times \frac{Pa_{CO_2} - P\bar{E}_{CO_{2L}}}{Pa_{CO_2}} + V_{E_R} \times \frac{Pa_{CO_2} - P\bar{E}_{CO_{2R}}}{Pa_{CO_2}}$$

where V_{E_L} and V_{E_R} are expiratory volumes and $P\bar{E}_{CO_{2L}}$ and $P\bar{E}_{CO_{2R}}$ are mean expired CO_2 tensions of the left and right lungs, respectively. The physiologic deadspace calculated by this equation is equivalent to that obtained by combining the two tidal volumes and using total expiratory volume and mixed CO_2 tension.[7]

A few days prior to the study, total lung capacity (TLC) and its subdivisions were measured spirometrically and FRC was obtained by the helium-dilution technique while the subjects were sitting, awake, and unmedicated.

Results

The physical characteristics and lung volumes (sitting, awake) of the subjects studied are shown in table 1.

Total mean values (left lung plus right lung) are summarized in table 2. The total values for the left and right lateral decubitus positions have been combined since no consistent difference between mean values was observed. During anesthesia and spontaneous ventilation there was no significant difference between mean values for the supine and lateral decubitus positions.

In the supine position, mean percentages of ventilation ($\dot{V}$), nonshunted blood flow ($\dot{V}_{O_2}$), CO_2 elimination ($\dot{V}_{CO_2}$), and FRC for the right lung were 52.1, 54.8, 53.5, and 56.8 per cent, respectively (table 3). In the left lateral decubitus position, FRC of the right lung (nondependent) was consistently increased; fractional ventilation and CO_2 elimination of the right lung increased in four of five instances; and mean percentages of $\dot{V}$, $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, and FRC for the right lung increased to 56.4, 56.8, 59.0, and 68.8 per cent, respectively. In the right lateral decubitus position, fractional ventilation, CO_2 elimination, and FRC of the right lung (dependent) decreased consistently, and the mean percentages of $\dot{V}$, $\dot{V}_{O_2}$, $\dot{V}_{CO_2}$, and FRC decreased to 44.7, 49.3, 44.1, and 43.6 per cent, respectively.

Table 4 shows the means of the differences between the percentage values for lungs in supine and dependent positions (that is, left lung in the supine position minus left lung in the left lateral position and right lung in the supine position minus right lung in the right lateral position). Fractional $\dot{V}$, $\dot{V}_{CO_2}$, and FRC of the dependent lung decreased significantly in the lateral position ($P < 0.025$). Fractional $\dot{V}_{O_2}$ of the dependent lung decreased in eight of ten observations; this difference did not achieve statistical significance.

Discussion

The effects of thiopental–meperidine anesthesia on intrapulmonary gas distribution have not been clarified; it is still unclear whether the altered gas distribution is caused by mechanical ventilation and muscle paralysis or anesthesia itself. The purpose of this study was to determine the effect of anesthesia apart from that of mechanical ventilation and muscle paralysis.

Kaneko and associates[8] observed that intrapulmonary gas distribution was dependent upon overall lung volume. At overall lung volumes of less than 50 per cent TLC they found that ventilation of the dependent lung was decreased in awake man in the lateral position. This represented an alteration in the intrapulmonary distribution of gas from that observed at lung volumes of more than 50 per cent TLC, at which the nondependent lung received a greater proportion of the inspired gas. Similarly, at overall lung volumes of less than 40 per cent TLC, dependent lung regions were less well ventilated in the supine position, which again represented a change from the observations made at lung volumes greater than 40 per cent TLC. These authors suggested this change in intrapulmonary gas distribution might be due to "airway closure" in the dependent lung or lung regions. To reopen such "closed airways," greater changes in transpulmonary pressures would be necessary, and if changes in transpulmonary pressures were uniform throughout, the dependent

TABLE 5. Mean Values of Total Lung Capacity and Subdivisions for Individual Lungs

Study	Position					
	Supine		Left Lateral Decubitus		Right Lateral Decubitus	
	Left Lung	Right Lung	Left Lung	Right Lung	Left Lung	Right Lung
Present study (subjects anesthetized and breathing spontaneously)						
FRC (liters)	0.91	1.18	0.79	1.71	1.32	1.03
TLC (liters)	2.89	3.81				
RV (liters)	0.64	0.84				
RV/TLC	0.22	0.22				
FRC/TLC	0.31	0.31	0.27	0.45	0.46	0.27
Total FRC/total TLC	0.31		0.37		0.35	
Svanberg[3] (subjects awake and breathing spontaneously)						
FRC (liters)	1.39	1.69	1.38	2.19	2.07	1.68
TLC (liters)	2.83	3.43				
RV (liters)	0.96	1.17				
RV/TLC	0.34	0.34				
FRC/TLC	0.49	0.49	0.49	0.64	0.73	0.49
Total FRC/total TLC	0.49		0.57		0.60	
Rehder *et al.*[2] (subjects anesthetized, paralyzed, and mechanically ventilated)						
FRC (liters)	1.16	1.36	1.12	2.29	2.21	1.33
TLC (liters)	3.41	3.99				
RV (liters)	0.97	1.13				
RV/TLC	0.28	0.28				
FRC/TLC	0.34	0.34	0.33	0.57	0.65	0.33
Total FRC/total TLC	0.34		0.46		0.48	

lung should receive a smaller fraction of the tidal volume. The decrease in FRC (with the accompanying element of "airway closure") which occurs during anesthesia[1,9] and which may encroach upon residual volume[10] might explain the altered intrapulmonary gas distribution.

In order to test this possibility, we tabulated (table 5) the mean values of FRC and of the ratio of FRC/TLC for individual lungs and total lungs, in all three of the body positions examined, for the following subjects: the five of the present study (anesthetized, breathing spontaneously), five other subjects studied earlier in this laboratory[2] (anesthetized, paralyzed, mechanically ventilated), and five subjects studied by Svanberg[3] (awake, breathing spontaneously).‡ We also tabulated the mean values of TLC, residual volume (RV), and RV/TLC ratio for individual lungs in the supine position. In our calculations we used the values for TLC and RV obtained in the sitting awake subjects and assumed that both remain unchanged in the supine and two lateral positions. This assumption appears to be valid for awake subjects.[11] We further assumed that the fractional distribution of TLC and RV be-

‡ We have used the data from Svanberg for this comparison, since the report of Lillington and associates,[6] which was used in an earlier comparison,[2] did not contain measurements of TLC and hence did not provide data that could be used to check the calculations. Calculations of the FRC/TLC ratios for Lillington and associates' subjects showed similar results.

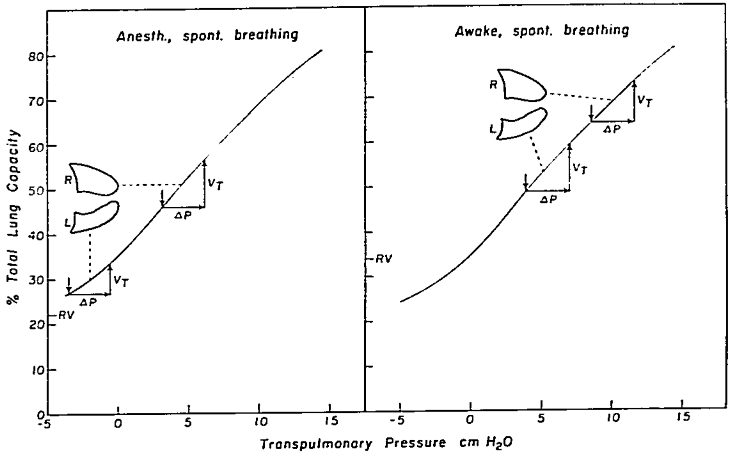

FIG. 1. Pressure–volume curves for both lungs of awake normal subjects in the left lateral decubitus position.[12] *Left,* mean dependent lung volume was 27 per cent and mean nondependent lung volume 45 per cent of TLC in anesthetized subjects breathing spontaneously. Let transpulmonary pressure during inspiration increase uniformly by 3 cm H_2O, the lung volume change of the nondependent lung is greater than that of the dependent lung (*longer upward arrow*). *Right,* mean dependent lung volume was 49 per cent, mean nondependent lung volume 64 per cent of TLC in awake subjects breathing spontaneously.[3] If transpulmonary pressure again increases uniformly by 3 cm H_2O, the volume change of the dependent lung is greater than that of the nondependent lung (*longer upward arrow*). (See text for further explanations.)

tween the two lungs equaled that of FRC in the supine position and that, unlike FRC, it remained unchanged in the lateral decubitus position. This assumption for conscious man seems reasonable, considering the results of the study by Svanberg.[3]

It is apparent from table 5 that the total FRC/total TLC ratios in both anesthetized, spontaneously breathing and anesthetized, paralyzed, mechanically ventilated subjects were less than 0.40 in the supine and less than 0.50 in both lateral decubitus positions. In contrast, in awake, spontaneously breathing subjects the ratio exceeded 0.40 in the supine position and exceeded 0.50 in the lateral positions. In order to determine what effect this difference in the FRC/TLC ratio might have on gas distribution between the two lungs in the lateral position, we replotted the mean pressure–volume curve for the total lungs obtained in a study of six awake men in the left lateral decubitus position by Milic-Emili and associates[12] (fig. 1). Assuming no change in the slope of the pressure–volume curve with anesthesia (an assumption which may, however, be incorrect[10]), and assuming equal lung pressure–volume curves for the dependent and nondependent lungs, one can determine the respective position of each lung on the pressure–volume curve at end-expiration (fig. 1, *downward arrow*). The mean end-expiratory lung volume of the dependent lung in anesthetized, spontaneously breathing subjects (left lateral position) was 27 per cent of TLC; that of the nondependent lung was 45 per cent. Assuming also equal stresses to both lungs (an assumption which may not be valid, particularly during mechanical ventilation[13,14]) and assuming an increase in transpulmonary pressure of 3 cm H_2O during inspiration (fig. 1, *horizontal arrow*), this will result in a larger volume change in the nondependent lung (fig. 1, *upward arrow* is longer) than in the dependent lung of the anesthetized subject, provided similar conditions for the chest wall prevail for the two hemithoraces. In conscious, spontaneously breathing man, the mean end-expiratory lung volume of the dependent lung was 49 per cent and that of the nondependent lung, 64 per cent of TLC in the left lateral position.[3] Given the same assumptions, one would expect a larger volume change of the dependent lung (fig. 1, *upward arrow* is longer) than of the nondependent lung in the awake subject.

The distribution of inspired gas is affected not only by the elastic and flow-resistive forces but also by stresses applied to the two lungs. Thus, gravity acting on the abdominal contents and on the mediastinum in the lateral decubitus position causes the end-expiratory lung volume of the dependent lung to be smaller than that of the nondependent lung. This volumetric difference between the two lungs is associated with a more sharply curved, high, dependent hemidiaphragm. It has been said that this permits the dependent hemidiaphragm to contract more effectively during active inspiration. However, this is not the only mechanism, because in patients with ipsilateral diaphragmatic paralysis a larger volume of gas ventilating the dependent lung has been observed.[15]

The lung clearance ratio (LCR) is independent of changes in FRC and alveolar ventilation rate, provided these factors change proportionately. In awake normal man, LCR increased when the subject was turned from the supine to the lateral position.[1] In contrast, in this study LCR did not increase significantly when the anesthetized subjects were turned. This indicates that the efficiency of utilization of total ventilation in nitrogen clearance (including V_D/V_T and uniformity of intrapulmonary gas distribution) was not changed significantly. This observation agrees with our findings in a study of anesthetized, paralyzed, mechanically ventilated man,[1] in which LCR also remained unchanged when the subjects were turned to the lateral position.

Since the hemoglobin of the peripheral arterial blood was saturated with oxygen, relative oxygen uptake of each lung represented the relative distribution of the nonshunted pulmonary blood flow (see Methods). Contrary to observations relating to conscious human subjects[6, 15, 16] and to anesthetized, paralyzed, mechanically ventilated dogs,[7] our observations showed increases in the relative oxygen uptake of the dependent lung in only two of ten instances.

Recently, Denlinger and associates[17] reported that the pulmonary capillary blood flow was greater to the nondependent lung in five of 11 anesthetized, paralyzed, mechanically ventilated subjects. These authors ascribed the larger nonshunted blood flow to the nondependent lung to preferential collapse of dependent portions of the lung. Such a hypothesis seems to be supported by the results of a study by Katori and associates[18] in anesthetized dogs; these authors observed significant blood flow to nonventilated alveoli (shunted blood flow) in dependent lung regions while the animals were breathing a gas mixture containing 99.6 per cent oxygen. They speculated that the dependent small airways and alveoli might decrease in size, or even completely collapse, so causing underventilated or nonventilated but perfused alveoli.

Another possible mechanism of altered distribution of nonshunted blood flow is reduction of end-expiratory lung volume associated with the induction of thiopental–meperidine anesthesia in subjects in supine and lateral positions. This change in lung volume may affect the pulmonary vascular resistances of the two lungs differently. Table 5 indicates that the relative volume of the dependent lung during anesthesia (with spontaneous breathing) was 27 per cent of its TLC (approximately RV in the supine position), whereas the nondependent lung volume was approximately 45 per cent of its TLC.

Hughes and associates,[19] studying healthy subjects sitting erect, demonstrated that the proportion of pulmonary blood flow per alveolus to the dependent regions decreased after maximal expiration to residual volume. At residual volume, apical blood flow exceeded basal blood flow, and when the lung volume was between RV and FRC, pulmonary blood flow per alveolus was uniform throughout the lung. These authors attributed the decreased blood flow to the dependent zones at low lung volumes to an increase in interstitial pressure, which affects the caliber of the extra-alveolar vessels. In agreement with these observations, Reed and Wood[20] have stressed that, in intact dogs, other factors—in addition to pulmonary arterial, pulmonary venous, and alveolar pressures—affect the vertical distribution of pulmonary blood flow. These authors pointed out that the position-dependent changes in blood-gas tensions which they had observed and the related changes in the *p*H of the pulmonary venous blood draining the dependent lung regions could be expected to be associated with increased tone of the muscles of the blood vessels in the dependent lung and thereby with an increase in the vascular resistance in the dependent lung.

Whatever the mechanism, the alterations in intrapulmonary gas distribution during anesthesia and spontaneous breathing in man are apparently associated with similar directional changes in the distribution of the nonshunted pulmonary blood flow. These similar directional changes in blood and gas flow distribution in the lateral position may tend to minimize the disturbances of pulmonary gas exchange during anesthesia and spontaneous breathing. Although these studies suggest that the reduction of end-expiratory lung volume following induction of anesthesia with thiopental and meperidine is probably a major factor in altering the distribution of intrapulmonary gas between the two lungs, the possible contribution of mechanical ventilation and muscle paralysis cannot be excluded.

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